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Heart Disease and Stroke Statistics 2010 Update: A Report From the American Heart Association

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Heart Disease and Stroke Statistics—2010 Update

A Report From the American Heart Association

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TABLE OF CONTENTS

Summary e47

1. About These Statistics. e53
2. Cardiovascular Diseases. e56
3. Subclinical Atherosclerosis e80
4. Coronary Heart Disease, Acute Coronary Syndrome, and Angina Pectoris e86
5. Stroke (Cerebrovascular Disease). e99
6. High Blood Pressure e115
7. Congenital Cardiovascular Defects e124
8. Cardiomyopathy and Heart Failure e129
9. Other Cardiovascular Diseases e134
   — Rheumatic Fever/Rheumatic Heart Disease . . .e134
   — Pulmonary Embolism. e134
   — Bacterial Endocarditis e135
   — Valvular Heart Disease e135
   — Aortic Valve Disorders e135
   — Mitral Valve Disorders e135
   — Arrhythmias (Disorders of Heart Rhythm) . . .e136
   — Arteries, Diseases of (including Peripheral Arterial Disease) e137
   — Venous Thromboembolism. e137
   — Peripheral Arterial Disease e138
10. Risk Factor: Smoking/Tobacco Use. e143
11. Risk Factor: High Blood Cholesterol and Other Lipids. e148
12. Risk Factor: Physical Inactivity e153
13. Risk Factor: Overweight and Obesity. e159
14. Risk Factor: Diabetes Mellitus e166
15. End-Stage Renal Disease and Chronic Kidney Disease e174
16. Metabolic Syndrome. e179
17. Nutrition e183
18. Quality of Care. e195
19. Medical Procedures e202
20. Economic Cost of Cardiovascular Diseases. e206

*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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21. At-a-Glance Summary Tables e208
— Men and Cardiovascular Diseases e209
— Women and Cardiovascular Diseases e210
— Ethnic Groups and Cardiovascular Diseases. . .e211
— Children, Youth and Cardiovascular Diseases. .e212
22. Glossary e213

Appendix I: List of Statistical Fact Sheets. URL:
http://www.americanheart.org/presenter.jhtml?identifier=2007

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Summary

Each year, the American Heart Association, 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 2000, the Statistical Update has been cited more than 6500 times in the literature (including citations of all annual versions). In 2008 alone, the various Statistical Updates were cited approximately 1300 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 American Heart Association, updated all of the current chapters with the most recent nationally representative data and inclusion of relevant papers from the literature over the past year. In future years, the Committee plans for the Statistical Update to be a major source for monitoring both cardiovascular health and disease in the population, with a focus on progress toward achievement of the American Heart Association's 2020 Impact Goals. In addition, future Statistical Updates will begin to incorporate the vast amounts of data becoming available from large population-based efforts to study the genetics of cardiovascular disease (CVD). Below are a few highlights from this year's Update.

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

- The 2006 overall death rate from CVD (*International Classification of Diseases 10*, I00–I99) was 262.5 per 100 000. The rates were 306.6 per 100 000 for white males, 422.8 per 100 000 for black males, 215.5 per 100 000 for white females, and 298.2 per 100 000 for black females. From 1996 to 2006, death rates from CVD declined 29.2%. Mortality data for 2006 show that CVD (I00–I99; Q20–Q28) accounted for 34.3% (831 272) of all 2 426 264 deaths in 2006, or 1 of every 2.9 deaths in the United States.
- On the basis of 2006 mortality rate data, nearly 2300 Americans die of CVD each day, an average of 1 death every 38 seconds. The 2007 overall preliminary death rate from CVD was 250.4. More than 151 000 Americans killed by CVD (I00–I99) in 2006 were <65 years of age. In 2006, nearly 33% of deaths due to CVD occurred before the age of 75 years, which is well before the average life expectancy of 77.7 years.
- Coronary heart disease caused approximately 1 of every 6 deaths in the United States in 2006. Coronary heart disease mortality in 2006 was 425 425. In 2010, an estimated 785 000 Americans will have a new coronary attack, and approximately 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, approximately 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 2006 indicate that stroke accounted for approximately 1 of every 18 deaths in the United States. On average, every 40 seconds, someone in the United States has a stroke. From 1996 to 2006, the stroke death rate fell 33.5%, and the actual number of stroke deaths declined 18.4%.
- In 2006, 1 in 8.6 death certificates (282 754 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) 2003–2006 indicate that 33.6% of US adults ≥20 years of age have hypertension (Table 6-1). This amounts to an estimated 74 500 000 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 >43%. Among hypertensive adults, approximately 78% are aware of their condition, 68% are using antihypertensive medication, and only 44% of those treated had their hypertension controlled.
- Despite 4 decades of progress, in 2008, among Americans ≥18 years of age, 23.1% of men and 18.3% of women continued to be cigarette smokers. In grades 9 through 12, 21.3% of male students and 18.7% of female students reported current tobacco use. The percentage of the nonsmoking population with detectable serum cotinine (indicating exposure to secondhand smoke) was 46.4% in 1999–2004 and was highest for those 4 to 11 years of age (60.5%) and those 12 to 19 years of age (55.4%).
- An estimated 35 700 000 adults ≥20 years of age have total serum cholesterol levels ≥240 mg/dL, with a prevalence of 16.2% (Table 11-1).
- In 2006, an estimated 17 200 000 Americans had diagnosed diabetes, representing 7.7% of the adult population. A further 6 100 000 had undiagnosed diabetes, and 29% had prediabetes, with abnormal fasting glucose levels. African-Americans,

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Mexican-Americans, Hispanic/Latino individuals, and other ethnic minorities bear a strikingly disproportionate burden of diabetes in the United States (Table 14-1).

The 2010 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 144 100 000, which represents 66.3% of this group in 2006. Fully 32.9% 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 13-1).
- Among children 2 to 19 years of age, 31.9% are overweight and obese (which represents 23 500 000 children), and 16.3% are obese (12 000 000 children). Mexican-American boys and girls and African-American girls are disproportionately affected. Over the last 3 decades, the prevalence of obesity in children 6 to 11 years of age has increased from approximately 4% to more than 17%.
- Although there is some debate regarding the amount of excess mortality associated with overweight, it is clear that 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, 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 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 2007, among adolescents in grades 9 through 12, 31.8% of females and 18% of males 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.
- Fifty-nine percent of adults who responded to the 2008 National Health Interview Survey reported engaging in no vigorous activity (activity that causes heavy sweating and a large increase in breathing or heart rate).
- 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 17-1).
- The increases in calories consumed during this time period are attributable primarily to greater average carbohydrate intake, particularly 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 2010 Update Provides Critical Data Regarding 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 Update provides these critical data in several sections.

Quality-of-Care Metrics for CVDs

Chapter 18 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 American Heart Association's "Get With the Guidelines" programs for acute coronary syndromes and heart failure and the American Stroke Association/American Heart Association's "Get With the Guidelines" program for acute stroke. Similar data from the Veterans Healthcare Administration, national Medicare and Medicaid data, and NCDR ACTION 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 19 provides data on trends and current usage of cardiovascular surgical and invasive procedures. For example, from 1996 to 2006, the total number of inpatient cardiovascular operations and procedures increased 33%, from 5 444 000 to 7 235 000 annually (American Heart Association computation based on National Center for Health Statistics annual data).

Chapter 20 reviews trends and current projections of direct and indirect healthcare costs related to CVDs, stroke, and related conditions. The total direct and indirect cost of CVD and stroke in the United States for 2010 is estimated to be $503.2 billion. This figure includes health expenditures (direct costs, which include the cost of physicians and other professionals, hospital and nursing home services, prescribed medications, home health care, and other medical durables) and lost productivity resulting from morbidity and mortality (indirect costs). Total hospital costs (inpatients, outpatients, and emergency department patients) projected for the year 2010 are estimated to be $155.7 billion. 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 American Heart Association, 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. The 2007 preliminary mortality data have been released. More information can be found at the National Center for Health Statistics Web site, http://www.cdc.gov/nchs/data/nvsr/nvsr58/nvsr58_01.pdf.

Finally, it must be noted that this annual Statistical Update is the product of an entire year's worth of effort by dedicated

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professionals, volunteer physicians and scientists, and outstanding American Heart Association staff members, without whom publication of this valuable resource would be impossible. Their contributions are gratefully acknowledged.

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Nancy Haase
On behalf of the American Heart Association Heart Disease and Stroke Statistics Writing Group

Note: Population data used in the compilation of NHANES prevalence estimates will now agree with the latest year of the NHANES survey being used. Extrapolations for NHANES prevalence estimates are based on the census resident population for 2006 because this is the most recent year of NHANES data used in the Statistical Update. An exception is the provisional smoking data from the 2008 National Health Interview Survey.

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Véronique L. Roger	Mayo Clinic Health Care Center	NIH/NHLBI†	None	None	None	None	None	None
Wayne Rosamond	University of North Carolina	None	None	None	None	None	None	None
Ralph Sacco	University of Miami Medical School	NINDS R37 29993 Northern Manhattan Study†; NINDS R01 040807 Family Study of Stroke Risk and Carotid Atherosclerosis†	None	None	None	None	Boehringer Ingelheim for design of clinical trial on stroke prevention*; GlaxoSmithKline*; Sanofi-Aventis*	None
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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.

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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 Update. This chapter describes the most important sources and the types of data we use from them. For more details, see Chapter 22 of this document, the Glossary.

The surveys used are:

- Behavioral Risk Factor Surveillance Survey (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

Abbreviations Used in Chapter 1

AHA	American Heart Association
AHRQ	Agency for Healthcare Research and Quality
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
ED	emergency department
FHS	Framingham Heart Study
GCNKSS	Greater Cincinnati/Northern Kentucky Stroke Study
HF	heart failure
ICD	International Classification of Diseases
ICD-9-CM	International Classification of Diseases, Clinical Modification, 9th 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
NHIS	National Health Interview Survey
NHLBI	National Heart, Lung, and Blood Institute
NINDS	National Institute of Neurological Disorders and Stroke
NIS	National Inpatient Sample
NNHS	National Nursing Home Survey
OPD	outpatient department
WHO	World Health Organization
YRBS	Youth Risk Behavior Surveillance

See Glossary (Chapter 22) for explanation of terms.

- 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 Hospital Ambulatory Medical Care Survey (NHAMCS)—hospital outpatient and emergency department visits
- National Inpatient Sample (NIS) of the Agency for Healthcare Research and Quality (AHRQ)—hospital inpatient discharges, procedures, and charges
- National Nursing Home Survey (NNHS)—nursing home residents
- National Vital Statistics—national and state mortality data
- World Health Organization (WHO)—mortality rates by country
- Youth Risk Behavior Surveillance (YRBS) (CDC)—trends for 6 categories of priority health-risk behaviors in youth and young adults

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 Update is to present the latest estimates of the number of persons 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 2003 to 2006 (in most cases, these are the latest published figures). These are applied to census population estimates for 2006. 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 2003 to 2006 data are used in this Update to present estimates of the percentage of persons with high lipid values, diabetes, 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 YRBS.

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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 due 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 2006 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 (Hypertension) and Chapter 9 (Heart Failure). Hypertension increases the mortality risks of CVD and other diseases, and HF is 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 Data Warehouse mortality tables of the NCHS World Wide Web site, the Health Data Interactive data system of the NCHS, or the CDC compressed 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 2006 in the computation of morbidity data. NCHS population estimates for 2006 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 (OPDs) and emergency departments (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 2006 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 2006. For disease and risk factor *prevalence*, most rates in this report are calculated from the 2003 to 2006 NHANES. Rates by age and sex are also applied to the US population in 2006 to estimate the numbers of persons with the disease or risk factor in that year. Because NHANES is conducted only in the noninstitutionalized population, we extrapolated the rates to the total US population in 2006, recognizing that this probably underestimates the total prevalence, given the rela-

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tively high prevalence in the institutionalized population. The numbers and rates of *hospital inpatient discharges* for the United States are for 2006. Numbers of visits to *physician offices*, *hospital EDs*, and *hospital OPDs* are for 2007. Except as noted, *economic cost* estimates are projected to 2010.

Cardiovascular Disease

For data on hospitalizations, physician office visits, and mortality, CVD is defined according to ICD codes given in Chapter 22 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.

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 Biostatistics Program Coordinator at the American Heart Association National Center (e-mail nancy.haase@heart.org, phone 214-706-1423). 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.americanheart.org/statistics, and in the journal *Circulation*.

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2. Cardiovascular Diseases

ICD-9 390–459, 745–747, ICD-10 I00–I99, Q20–Q28; see Glossary (Chapter 22) for details and definitions. See Tables 2-1 through 2-5 and Charts 2-1 through 2-21.

Abbreviations Used in Chapter 2

AED	automated external defibrillator
AHA	American Heart Association
AHRQ	Agency for Healthcare Research and Quality
AIDS	acquired immune deficiency syndrome
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)
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
CPR	cardiopulmonary resuscitation
CVD	cardiovascular disease
DM	diabetes mellitus
ED	emergency department
EMS	emergency medical services
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
NHLBI	National Heart, Lung, and Blood Institute
NIS	National Inpatient Sample
NNHS	National Nursing Home Survey
OPD	outpatient department
PA	physical activity
RR	relative risk
VF	ventricular fibrillation

Prevalence

An estimated 81 100 000 American adults (more than 1 in 3) have 1 or more types of CVD. Of these, 38 100 000 are estimated to be ≥60 years of age. Total CVD includes diseases listed in the bullet points below, except for congenital CVD. Because of overlap, it is not possible to add these conditions to arrive at a total.

- High BP (HBP)—74 500 000 (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)—17 600 000.
  - — MI (heart attack)—8 500 000.
  - — AP (chest pain)—10 200 000.
- Heart failure (HF)—5 800 000.
- Stroke—6 400 000.
- Congenital cardiovascular defects—650 000 to 1 300 000 (see Chapter 7).

The following age-adjusted prevalence estimates from the NHIS, NCHS are for diagnosed conditions for people ≥18 years of age in 2008[1]:

- Among whites only, 12.1% have heart disease (HD), 6.5% have CHD, 23.3% have hypertension, and 2.7% have had a stroke.
- Among blacks or African Americans, 10.2% have HD, 5.6% have CHD, 31.8% have hypertension, and 3.6% have had a stroke.
- Among Hispanics or Latinos, 8.1% have HD, 5.7% have CHD, 21.0% have hypertension, and 2.6% have had a stroke.
- Among Asians, 5.2% have HD, 2.9% have CHD, 21.0% have hypertension, and 1.8% have had a stroke.
- Among American Indians or Alaska Natives, 12.1% have HD, 6.6%* have CHD, 25.3% have hypertension, and 3.9% have had a stroke.*
- Among Native Hawaiians or other Pacific Islanders, HD, CHD, and stroke numbers are not reported because of large relative standard errors; 19.7%* have hypertension.
- Asian Indian adults (9%) are approximately 2-fold more likely than Korean adults (4%) to have ever been told they have HD.[2]

Incidence

- On the basis of the NHLBI's FHS original and offspring cohort data from 1980 to 2003[3]:
  - — The average annual rates of first cardiovascular (CVD) 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 due to CHD occur in men than in women, and a higher

*Figure considered unreliable.

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proportion of events due 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.[4]
- Data from the FHS indicate that the lifetime risk for all CVD in recipients free of disease is 2 in 3 for men and more than 1 in 2 for women at 40 years of age (personal communication, Donald Lloyd-Jones, MD, Northwestern University, Chicago, Ill) (see Table 2-4).
- 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.[5]

Mortality

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

- Mortality data show that CVD (I00–I99, Q20–Q28) as the underlying cause of death (including congenital cardiovascular defects) accounted for 34.3% (831 272) of all 2 426 264 deaths in 2006, or 1 of every 2.9 deaths in the United States. CVD any-mentions (1 347 000 deaths in 2006) constituted approximately 56% of all deaths that year (NHLBI; NCHS public-use data files).[6] Preliminary 2007 mortality (I00–I99) was 807 485. The preliminary death rate was 250.4 (NCHS).[7] In every year since 1900 except 1918, CVD accounted for more deaths than any other major cause of death in the United States.[6–11]
- Nearly 2300 Americans die of CVD each day, an average of 1 death every 38 seconds. CVD claims more lives each year than cancer, CLRD, and accidents combined.[6]
- The 2006 overall death rate due to CVD (I00–I99) was 262.5. The rates were 306.6 for white males, 422.8 for black males, 215.5 for white females, and 298.2 for black females. From 1996 to 2006, death rates due to CVD (ICD-10 I00–I99) declined 29.2%. In the same 10-year period, the actual number of CVD deaths per year declined 12.9%.[6] (Appropriate comparability ratios were applied.)
- Among other causes of death in 2006, cancer caused 559 888 deaths; accidents, 121 599; Alzheimer's disease, 72 432; and HIV (human immunodeficiency virus)/AIDS (acquired immune deficiency syndrome), 12 113.[6]
- The 2006 CVD (I00–I99) death rates were 313.3 for males and 221.6 for females. Death rates for cancer (malignant neoplasms) were 220.1 for males and 153.6 for females. Breast cancer claimed the lives of 40 821 females in 2006; lung cancer claimed 69 385. Death rates for females were 23.5 for breast cancer and 40.0 for lung cancer. One in 30 female deaths was due to breast cancer, whereas 1 in 6 was due to CHD. For comparison, 1 in 4.5 females died of cancer, whereas 1 in 2.8 died of CVD (I00–I99, Q20–Q28). On the basis of 2006 mortality data, CVD caused approximately 1 death per minute among females, or 432 709 female deaths in 2006. That represents more female lives than were claimed by cancer, CLRD, Alzheimer's disease, and accidents combined.[6]
- More than 151 000 Americans died of CVD (I00–I99) in 2006 who were <65 years of age, and nearly 33% of deaths due to CVD occurred before the age of 75 years, which is well before the average life expectancy of 77.7 years.[6]
- In 2006, death rates for diseases of the heart in American Indians or Alaska Natives were 170.2 for males and 113.2 for females; for Asians or Pacific Islanders, they were 136.3 for males and 87.3 for females; and for Hispanics or Latinos, they were 175.2 for males and 118.9 for females.[11]
- According to the NCHS, if all forms of major CVD were eliminated, life expectancy would rise by almost 7 years. If all forms of cancer were eliminated, the estimated gain would 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 diabetes mellitus (DM), and 0.7% for HIV.[12]
- In 2006, 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).[6,13]
- A recent study of the decrease in US deaths due to CHD from 1980 to 2000 suggests that approximately 47% of the decrease was attributable to evidence-based medical therapies and 44% to changes in risk factors in the population due to lifestyle and environmental changes.[14]
- 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 high BP were responsible for an estimated 467 000 deaths, accounting for approximately 1 in 5 or 6 deaths among US adults. Overweight/obesity and physical inactivity were each 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 mortality effects.[15]

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.[16]

Out-of-Hospital Cardiac Arrest

There is a wide variation in the reported incidence of and outcome 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

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the onset of cardiac arrest. Cardiac arrest is defined as cessation of cardiac mechanical activity and is confirmed by the absence of signs of circulation.[17]

- Extrapolation of the mortality rate observed in the Resuscitation Outcomes Consortium to the total population of the United States suggests that each year, there are 295 000 (quasi confidence intervals 236 000 to 325 000) emergency medical services (EMS)–assessed out-of-hospital cardiac arrests in the United States.[18]
- Approximately 60% of out-of-hospital cardiac deaths are treated by EMS personnel.[19]
- Only 33% of those with EMS-treated out-of-hospital cardiac arrest have symptoms within 1 hour of death.[20]
- Among EMS-treated out-of-hospital cardiac arrests, 23% have an initial rhythm of ventricular fibrillation (VF), ventricular tachycardia, or shockable by automated external defibrillator (AED); 31% receive bystander cardiopulmonary resuscitation (CPR).[18]
- 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.[21]
- The incidence of lay-responder defibrillation is low (2.05% in 2002) but is increasing over time.[22]
- If bystander CPR is not provided, a sudden cardiac arrest victim's chances of survival fall 7% to 10% for every minute of delay until defibrillation.[23–27]
- The median survival rate to hospital discharge after EMS-treated out-of-hospital cardiac arrest with any first recorded rhythm is 7.9%.[18]
- The median survival rate after VF is 21%.[18]
- Extrapolation of data from ARIC, CHS, and Framingham suggests that there are 138 000 CHD deaths within 1 hour of symptom onset (Thomas Thom, NHLBI, written communication, May 20, 2008).
- 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. 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.[30]

Out-of-Hospital Cardiac Arrest: Children

- The reported incidence of out-of-hospital pediatric cardiac arrest varies widely (approximately 8 per 100 000).[31]
- There are more than 72 million individuals <18 years of age in the United States[32]; this implies that there are about 5760 pediatric out-of-hospital cardiac arrests annually of all causes (including trauma, sudden infant death syndrome, respiratory causes, cardiovascular causes, and submersion).
- Thirty-five percent of EMS-treated pediatric cardiac arrest patients had an initial rhythm of VF, ventricular tachycardia, or shockable by AED; 35% received bystander CPR.[31]
- Studies that document voluntary reports of deaths among high school athletes suggest that the incidence of out-of-hospital cardiac arrest ranges from 0.28 to 1.0 deaths per 100 000 high school athletes annually nationwide.[33,34] Although incomplete, these numbers provide a basis for estimating the number of deaths in this age range.
- One report describes the incidence of nontraumatic pediatric cardiac arrest (among students 3 to 18 years of age) that occurs in schools and estimates rates (per 100 000 person-school-years) for elementary, middle, and high schools to be 0.18, 0.19, and 0.15, respectively, for the geographic area (King County, Washington) and time frame (January 1, 1990, to December 31, 2005) studied.[35]
- The reported average rate of survival to hospital discharge after pediatric out-of-hospital cardiac arrest is 6%.
- Most sudden deaths in athletes were due 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 increased over time.[36]

In-Hospital Cardiac Arrest

- A total of 292 facilities reported 20 913 events to the National Registry for Cardiopulmonary Resuscitation from August 1, 2007, to July 31, 2008.
  - The rates of survival to discharge after in-hospital cardiac arrest were 35% among children and 19% among adults. Of these, 95% were monitored or witnessed.
  - Eighteen percent had VF or pulseless ventricular tachycardia as the first recorded rhythm. Of these, 78% received a defibrillation attempt within 3 minutes.
- Patients who experience cardiac arrest during the weekday have an absolute 5.6% greater survival than those who experience cardiac arrest during the night or on weekends.

Awareness of CPR

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

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

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from getting CVD. The majority of respondents reported confusion related to basic CVD prevention strategies.[38]

- 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 physical activity (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.[39]
- 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.[40]

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 Indians/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). Persons 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 persons (45.1%), unemployed adults (43.4%), homemakers (34.3%), and employed persons (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.[41]
- Data from the Chicago Heart Association Detection Project (1967 to 1973, with an average follow-up of 31 years) showed that in younger women (18 to 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.[42,43]
- 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 was greater among men, but hospitalization for congestive heart failure (CHF) and stroke was greater among women. Among Medicare enrollees, CHF hospitalization was higher in blacks, Hispanics, and American Indians/Alaska Natives than among whites, and stroke hospitalization was highest in 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 approximately 5 years. CVD mortality at all ages tended to be highest in blacks.[44]
- In respondents 18 to 74 years of age, data from the 2000 BRFSS (CDC) showed the prevalence of healthy lifestyle characteristics was as follows: No smoking, 76.0%; healthy weight, 40.1%; consumption of 5 fruits and vegetables per day, 23.3%; and regular PA, 22.2%. The overall prevalence of the healthy lifestyle indicators (ie, having all 4 healthy lifestyle characteristics) was only 3%, with little variation among subgroups.[45]
- 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 diabetes) decreased over time across all BMI groups, with the greatest reductions observed among overweight and obese groups. Total diabetes 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.[46]
- 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 approximately 70% in white subjects, were 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 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. The primary prevention of elevated risk factors might largely eliminate the incidence of CVD, and these beneficial effects would be applicable not only for white but also for black subjects.[47]
- Data from the MEPS 2004 Full-Year Data File showed that nearly 26 million US adults ≥18 years of age were told by a

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doctor that they had HD, stroke, or any other heart-related disease[48]:

— 56.6% of those surveyed said they engaged in moderate-to-vigorous PA 3 times per week; 57.9% of those surveyed who had not been told they had HD engaged in regular PA, more than those who had been told they had HD (46.3%).
— 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 to 44 years of age) were more likely (59.9%) than those who were older (45 to 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).
  - Persons ≥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 to 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, not the direction, of association.[49]
- A study of nearly 1500 participants in the Multi-Ethnic Study of Atherosclerosis (MESA) found that Hispanics with hypertension, hypercholesterolemia, and/or diabetes who speak Spanish at home and/or have spent less than half a year in the United States have higher systolic BP, 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.[50]

Family History of Premature-Onset CVD

- 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[51] or a sibling[52] was associated with an approximately 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.[51] 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[53] and men.[54]
- Parental history of premature CHD is associated with increased burden of atherosclerosis in the coronary arteries and the abdominal aorta.[55,56]
- 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.[57]
- 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.[58]
- A recent survey of persons in the United States indicated that most respondents believe that knowing their family

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history is important for their own health, but few are aware of the specific health information from relatives necessary to develop a family history.[59]

- An accurate and complete family history may identify rare mendelian conditions such as hypertrophic cardiomyopathy, long-QT syndrome, or familial hypercholesterolemia. However, in most persons 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 persons 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 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 intensive prevention efforts at younger and middle ages once risk factors develop to improve healthy longevity.

- The lifetime risk for CVD and median survival were highly associated with risk factor 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 diabetes, and absence of smoking. Elevated risk factors were defined as stage 1 hypertension or borderline high cholesterol (200 to 239 mg/dL). Major risk factors were defined as stage 2 hypertension, elevated cholesterol (≥240 mg/dL), current smoking, and diabetes. 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.[5]
- 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.[60]
- 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 predicted overall survival and morbidity-free survival to 85 years of age or more.[61]
  - — 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 systolic BP, lower total cholesterol, 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, HF, stroke, dementia, and cancer were nearly identical.
  - — When adverse levels of 4 of these factors were present in middle age, fewer than 5% of men and approximately 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 diabetes, 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 total mortality rates, and 6 to 10 years' longer life expectancy.[43]
- 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 reduced by 57%; when 4 were present, RR was reduced by 66%; and when all 5 factors were present, RR was reduced by 83%.[62]
- 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 to 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.[63]
- Among individuals 70 to 90 years of age, adherence to a Mediterranean-style diet and greater PA are associated with 65% to 73% lower rates of all-cause mortality, as well as lower mortality rates due to CHD, CVD, and cancer.[64]

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- 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 or more risk factors. Had all 3 major risk factors not occurred, it is estimated that 64% of all CHD deaths among women and 45% of CHD deaths in men could have been avoided.[65]
- 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.[66]
  - 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).[67]
  - Data from NHANES 1999 to 2002 showed that about one third of adults complied with 6 or more of the recommended heart-healthy behaviors. Dietary recommendations in general and daily fruit intake recommendations in particular were least likely to be followed.[68]

Hospital Discharges, Ambulatory Care Visits, and Nursing Home Residents

- From 1996 to 2006, the number of inpatient discharges from short-stay hospitals with CVD as the first-listed diagnosis increased from 6 107 000 to 6 161 000 (NCHS, NHDS). In 2006, CVD ranked highest among all disease categories in hospital discharges.[69]
- In 2007, there were 79 697 000 physician office visits with a primary diagnosis of CVD (NCHS, NAMCS).[70]
- In 2007, there were 4 048 000 ED visits with a primary diagnosis of CVD (NCHS, NHAMCS).[71]
- In 2007, there were 7 929 000 hospital OPD visits with a primary diagnosis of CVD (NHAMCS).[72] In 2005, approximately 1 of every 6 hospital stays, or almost 6 million, resulted from CVD (AHRQ, 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 approximately 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 overall rate of 43.1%; 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%.[73]
- In 2004, coronary atherosclerosis was estimated to be responsible for 1.2 million hospital stays and was the most expensive condition treated. This condition resulted in more than $44 billion in expenses. More than half of the hospital stays for coronary atherosclerosis were among patients who also received percutaneous coronary intervention or cardiac revascularization (coronary artery bypass graft [CABG]) during their stay. Acute MI 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.[74]
- In 2003, approximately 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 acute MI and coronary atherosclerosis 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.[75]
- 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.[76]
- In 2004, nursing home residents had a primary diagnosis of CVD at admission (23.7%) and at the time of interview (25%). This was the leading primary diagnosis for these residents (NCHS, NNHS).[77]

Operations and Procedures

- In 2006, an estimated 7 235 000 inpatient cardiovascular operations and procedures were performed in the United States; 4.1 million were performed on males, and 3.1 million were performed on females (NHDS, NCHS, and NHLBI).

Cost

- The estimated direct and indirect cost of CVD for 2010 is $503.2 billion.
- 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.[78]

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Table 2-1. Cardiovascular Disease

Population Group	Prevalence, 2006 Age ≥20 y	Mortality, 2006 All Ages*	Hospital Discharges, 2006 All Ages	Cost, 2010
Both sexes	81 100 000 (36.9%)	831 272	6 161 000	$503.2 Billion
Males	39 000 000 (37.9%)	398 563 (47.9%)†	3 121 000	. . .
Females	42 100 000 (35.7%)	432 709 (52.1%)†	3 040 000	. . .
NH white males	38.1%	340 594	. . .	. . .
NH white females	34.4%	372 764	. . .	. . .
NH black males	44.6%	47 956	. . .	. . .
NH black females	46.9%	50 798	. . .	. . .
Mexican American males	28.5%	. . .	. . .	. . .
Mexican American females	34.5%	. . .	. . .	. . .

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 CVD mortality that is attributable to males vs females.

Sources: Prevalence: NHANES 2003–2006, NCHS and NHLBI. Percentages for racial/ethnic groups are age-adjusted for Americans ≥20 years of age. Age-specific percentages are extrapolated to the 2006 US population estimates. Mortality: NCHS. These data represent underlying cause of death only. Data include congenital CVD mortality. Hospital discharges: NHDS, NCHS. Data include those inpatients discharged alive, dead, or of unknown status. Cost: NHLBI. Data include estimated direct and indirect costs for 2010.

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Table 2-2. 2006 Age-Adjusted Death Rates for CVD, CHD, and Stroke by State (Includes District of Columbia and Puerto Rico)

	CVD*			CHD†			Stroke‡		
State	Rank§	Death Rate	% Change‖ 1996–2006	Rank§	Death Rate	% Change‖ 1996–2006	Rank§	Death Rate	% Change‖ 1996–2006
Alabama	51	330.9	−17.2	25	121.7	−32.4	51	55.5	−18.8
Alaska	11	227.5	−28.5	4	87.4	−38.2	34	46.8	−31.9
Arizona	5	215.4	−28.9	24	120.8	−31.1	3	34.5	−39.6
Arkansas	48	311.0	−23.8	47	160.1	−22.0	52	58.8	−35.1
California	29	257.3	−27.8	34	139.0	−36.3	29	44.9	−32.3
Colorado	4	212.8	−29.2	6	96.3	−35.9	13	38.7	−35.2
Connecticut	18	232.3	−35.1	13	110.0	−42.3	8	36.5	−37.7
Delaware	27	255.4	−26.1	37	140.8	−31.4	18	41.8	−24.0
District of Columbia	50	325.7	−19.1	52	193.5	7.0	10	37.6	−45.9
Florida	10	227.4	−30.1	28	129.2	−37.2	4	35.3	−33.3
Georgia	41	288.8	−28.2	12	108.7	−41.5	43	51.4	−33.6
Hawaii	2	206.2	−30.9	3	85.2	−40.2	22	43.2	−32.9
Idaho	20	238.5	−25.3	14	110.2	−34.0	44	51.6	−27.3
Illinois	33	268.2	−29.8	31	134.8	−39.4	31	45.4	−33.0
Indiana	40	288.7	−27.7	35	139.7	−36.0	39	49.1	−34.8
Iowa	22	246.7	−29.6	39	141.6	−36.2	20	42.9	−31.6
Kansas	28	255.4	−26.1	17	114.1	−35.0	33	46.7	−28.0
Kentucky	44	307.7	−25.6	42	148.6	−32.2	42	50.5	−30.4
Louisiana	46	308.4	−22.4	33	138.3	−32.4	46	52.1	−24.7
Maine	17	232.2	−33.1	15	112.2	−43.3	17	41.3	−28.6
Maryland	32	266.6	−25.4	40	141.7	−29.7	23	43.6	−31.6
Massachusetts	8	224.0	−31.3	9	105.6	−39.9	11	37.7	−28.2
Michigan	42	291.7	−27.8	45	156.6	−35.2	28	44.5	−34.5
Minnesota	1	190.9	−35.9	2	79.7	−45.5	14	39.3	−40.1
Mississippi	52	348.8	−23.4	41	146.8	−38.1	49	53.7	−25.7
Missouri	43	293.2	−27.4	44	155.2	−34.2	41	49.4	−27.3
Montana	7	223.3	−30.2	7	99.0	−36.1	16	41.2	−33.9
Nebraska	13	228.8	−34.5	5	89.9	−44.0	25	43.9	−29.5
Nevada	39	287.7	−22.0	23	119.5	−38.5	15	39.7	−33.8
New Hampshire	16	230.1	−34.4	21	116.3	−42.7	5	35.4	−47.4
New Jersey	26	254.1	−30.1	38	141.2	−36.1	6	35.9	−33.8
New Mexico	9	224.0	−24.4	18	114.6	−30.8	9	37.5	−35.9
New York	37	278.6	−30.9	51	181.2	−32.9	1	29.7	−37.2
North Carolina	34	268.2	−30.4	27	126.1	−39.3	47	52.4	−36.0
North Dakota	23	246.7	−28.8	30	133.7	−26.6	40	49.2	−29.8
Ohio	38	283.8	−28.0	43	154.0	−32.6	30	45.2	−28.1
Oklahoma	49	322.0	−21.2	50	177.4	−23.2	48	53.3	−23.0
Oregon	14	228.8	−29.6	8	99.2	−40.2	36	48.0	−38.8
Pennsylvania	35	268.8	−29.9	32	136.0	−37.4	24	43.6	−30.0
Puerto Rico¶	6	219.4	−27.5	10	106.6	−23.7	26	43.9	−25.0
Rhode Island	24	249.8	−25.7	48	162.4	−27.3	2	31.4	−38.4
South Carolina	36	270.5	−33.1	22	119.2	−43.0	45	51.6	−41.8
South Dakota	19	235.6	−30.0	36	140.0	−27.7	19	42.4	−30.9
Tennessee	45	307.7	−25.1	49	167.8	−30.0	50	54.6	−31.2
Texas	31	262.8	−28.6	29	132.2	−37.4	37	48.3	−30.5

(*Continued*)

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Table 2-2. Continued

	CVD*			CHD†			Stroke‡		
State	Rank§	Death Rate	% Change‖ 1996–2006	Rank§	Death Rate	% Change‖ 1996–2006	Rank§	Death Rate	% Change‖ 1996–2006
Utah	3	208.2	−28.0	1	77.5	−44.0	7	36.2	−40.7
Vermont	15	229.3	−33.0	26	124.5	−37.8	12	37.8	−39.7
Virginia	30	258.1	−31.1	20	115.6	−36.8	38	49.0	−33.5
Washington	12	228.0	−28.7	19	114.7	−31.7	21	42.9	−39.0
West Virginia	47	309.2	−27.4	46	158.7	−35.8	35	47.6	−21.7
Wisconsin	21	241.8	−30.9	16	113.9	−39.2	27	44.3	−38.9
Wyoming	25	250.1	−26.6	11	107.1	−36.5	32	45.4	−37.2
Total United States		262.5	−29.5		135.0	−35.9		43.6	−32.7

*CVD is defined here as ICD-10 I00–I99.

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

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

§Rank is lowest to highest.

‖Percent change is based on log-linear slope of rates for each year, 1996–2006. For stroke, the death rates in 1996–1998 were comparability modified with the ICD-10 to ICD-9 comparability ratio of 1.0502.

¶Percent changes for Puerto Rico are for 1996–1998 (averaged) to 2006 and are not based on a log-linear slope.

Source: NCHS compressed mortality file 1979–2006. Data provided by personal communication with NHLBI.

The AHRQ 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 NCHS 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. BRFSS data are also collected within each state (www.cdc.gov/brfss). The CDC has the Geographic Information Systems (GIS), which provides mortality rates down to the county level, by gender 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/library/stroke_hospitalization_atlas.htm).

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Table 2-3. International Death Rates (Revised 2009): Death Rates (Per 100 000 Population) for Total CVD, CHD, Stroke, and Total Deaths in Selected Countries (Most Recent Year Available)

	ICD Revision 9 or 10	CVD Deaths	CHD Deaths	Stroke Deaths	Total Deaths
Men Ages 35–74 y					
Russian Federation (2002)	10	1555.2	835.0	452.8	3186.9
Bulgaria (2004)*	9	915.6	273.3	227.2	1610.0
Hungary (2005)	10	709.7	384.7	140.8	1818.0
Romania (2007)	10	692.5	287.6	216.4	1534.8
Poland (2006)	10	510.0	198.5	104.8	1447.6
Czech Republic (2007)	10	416.5	223.9	69.0	1128.7
Argentina (2001)	10	405.9	119.8	102.6	1261.7
China–urban (2000)*	9	374.8	108.3	160.1	976.8
Colombia (1999)	10	331.3	168.2	94.7	1021.0
Finland (2007)	10	298.4	180.1	47.4	868.4
Scotland (2007)	10	294.8	195.3	46.1	936.1
Greece (2007)*	9	281.1	148.3	58.4	762.3
United States (2006)	10	272.6	162.2	32.3	885.6
Belgium (1999)	10	267.5	133.0	46.4	963.5
China–rural (2000)*	9	265.3	41.6	364.5	828.0
Portugal (2003)	10	252.8	96.6	96.1	965.5
Germany (2006)	10	242.1	125.2	34.5	788.5
Mexico (2001)	10	235.0	129.9	54.7	1055.8
Northern Ireland (2007)	10	232.4	161.9	29.6	781.2
Ireland (2006)	10	231.3	141.8	29.1	694.6
England/Wales (2007)	10	219.7	138.3	32.7	702.2
New Zealand (2005)	10	206.8	138.4	30.3	646.1
Denmark (2006)	10	206.6	84.8	45.6	865.6
Austria (2007)	10	202.4	119.1	27.8	731.2
Sweden (2006)	10	201.5	118.7	33.3	620.9
Canada (04)	10	198.3	130.8	24.2	705.3
Spain (2005)	10	191.2	92.1	39.2	786.3
Netherlands (2007)	10	176.5	71.7	28.8	660.2
Norway (2006)	10	176.1	97.1	28.3	642.2
Republic of Korea (2006)	10	175.6	51.4	93.1	966.5
Australia (2005)	10	172.4	113.5	25.2	614.6
Italy (2006)	10	167.5	80.1	31.9	641.2
Israel (2005)	10	159.3	78.8	34.7	680.3
Switzerland (2006)	10	153.5	79.9	19.3	587.9
Japan (2007)	10	151.6	47.7	56.3	630.4
France (2006)	10	149.8	58.4	27.7	793.6
Women ages 35–74 y					
Russian Federation (2002)	10	659.2	288.1	257.0	1192.4
Bulgaria (2004)*	9	434.6	100.4	133.1	745.9
Romania (2007)	10	347.7	117.4	134.0	720.8
Hungary (2005)	10	291.1	144.6	67.0	780.4
China–urban (2000)*	9	258.9	71.9	103.1	637.7
Colombia (1999)	10	229.9	94.7	70.7	640.1
Poland (2006)	10	193.1	57.5	56.1	583.4
China–rural (2000)*	9	184.6	29.1	239.1	528.1
Czech Republic (2007)	10	176.8	78.4	38.3	532.3

(*Continued*)

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Table 2-3. Continued

	ICD Revision 9 or 10	CVD Deaths	CHD Deaths	Stroke Deaths	Total Deaths
Argentina (2001)	10	174.2	35.2	55.4	617.2
Mexico (2001)	10	166.0	69.0	46.6	713.2
United States (2006)	10	138.3	65.1	25.3	559.4
Scotland (2007)	10	130.9	67.5	33.0	590.0
Portugal (2003)	10	123.2	34.8	54.6	448.7
Belgium (1999)	10	120.4	41.8	29.6	480.2
Greece (2007)*	9	105.5	36.9	33.6	339.3
Northern Ireland (2007)	10	101.4	51.5	28.1	470.7
Denmark (2006)	10	100.0	32.4	32.1	557.8
Germany (2006)	10	97.8	38.2	20.1	402.4
England/Wales (2007)	10	97.1	43.4	25.4	450.4
New Zealand (2005)	10	97.0	47.2	27.1	425.3
Ireland (2006)	10	90.4	40.1	22.6	422.5
Republic of Korea (2006)	10	89.0	20.0	50.8	359.3
Finland (2007)	10	88.4	41.2	25.3	380.2
Netherlands (2007)	10	84.2	25.3	22.8	423.8
Canada (04)	10	83.1	42.8	17.3	432.7
Sweden (2006)	10	83.1	39.1	21.2	388.5
Austria (2007)	10	77.6	32.1	17.1	368.1
Norway (2006)	10	74.0	32.3	19.9	395.0
Spain (2005)	10	73.5	22.9	21.0	325.9
Australia (2005)	10	72.2	34.1	18.3	359.6
Israel (2005)	10	71.4	26.3	19.2	406.4
Italy (2006)	10	70.0	23.1	19.3	327.5
Japan (2007)	10	59.6	14.2	25.3	281.6
Switzerland (2006)	10	54.6	20.1	13.4	333.1
France (2006)	10	54.3	13.0	15.2	358.4

Rates are adjusted to the European Standard population. 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.

*Countries using ICD-9.

Sources: The World Health Organization, NCHS, and NHLBI.

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Table 2-4. 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[5]	1 in 2	1 in 3	1 in 3	1 in 4
AF[47]	1 in 4	1 in 4	1 in 4	1 in 4
CHF[48]	1 in 5	1 in 5	1 in 5	1 in 5
Stroke[49]	1 in 6†	1 in 5†	1 in 6	1 in 5
Dementia[49]	. . .	. . .	1 in 7	1 in 5
Hip fracture[61]	1 in 20	1 in 6	. . .	. . .
Breast cancer[62,64]	1 in 1000	1 in 8	. . .	1 in 14
Prostate cancer[62]	1 in 6	. . .	. . .	. . .
Lung cancer[62]	1 in 12	1 in 17	. . .	. . .
Colon cancer[62]	1 in 16	1 in 17	. . .	. . .
Diabetes[65]	1 in 3	1 in 3	1 in 9	1 in 7
Hypertension[66]	9 in 10†	9 in 10†	9 in 10‡	9 in 10‡
Obesity[67]	1 in 3	1 in 3	. . .	. . .

Ellipses (zelb) indicate not estimated.
*Personal communication from Donald Lloyd-Jones, based on FHS data.
†Age 55 years.
‡Age 65 years.

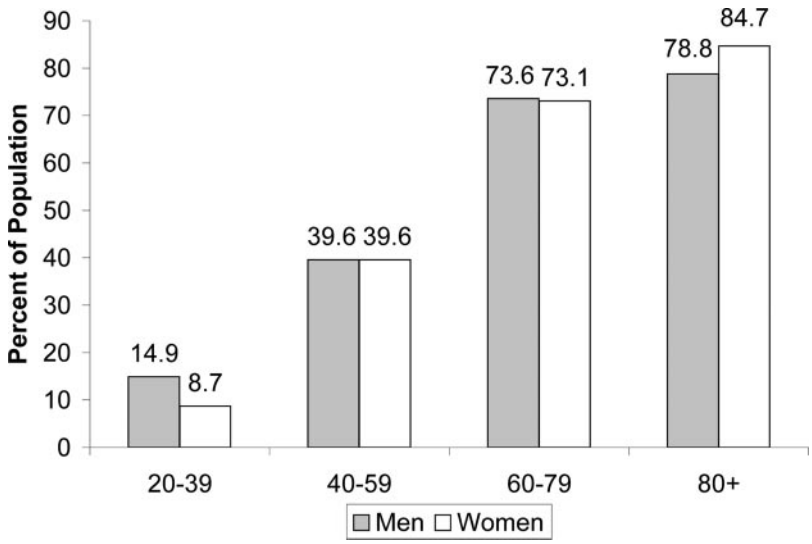

Chart 2-1. Prevalence of CVD in adults ≥20 years of age by age and sex (NHANES: 2003–2006). Source: NCHS and NHLBI. These data include CHD, HF, stroke, and hypertension.

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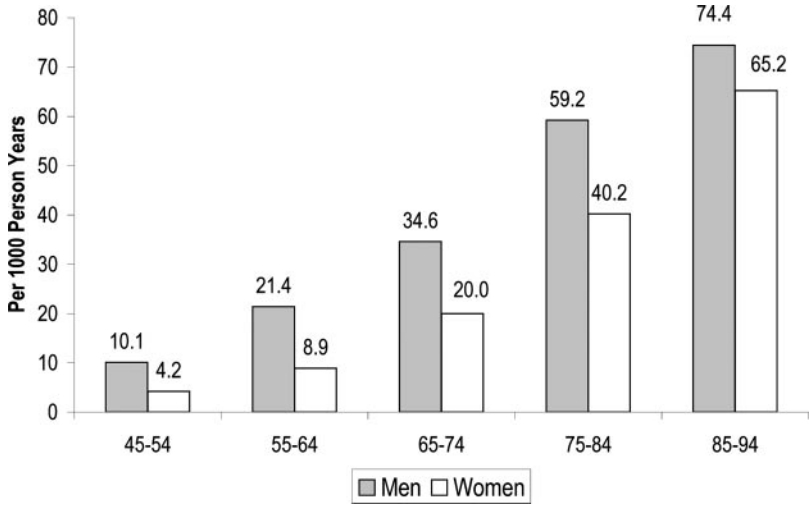

Chart 2-2. Incidence of CVD* by age and sex (FHS, 1980–2003). *CHD, HF, stroke, or intermittent claudication. Does not include hypertension alone. Source: NHLBI.[3]

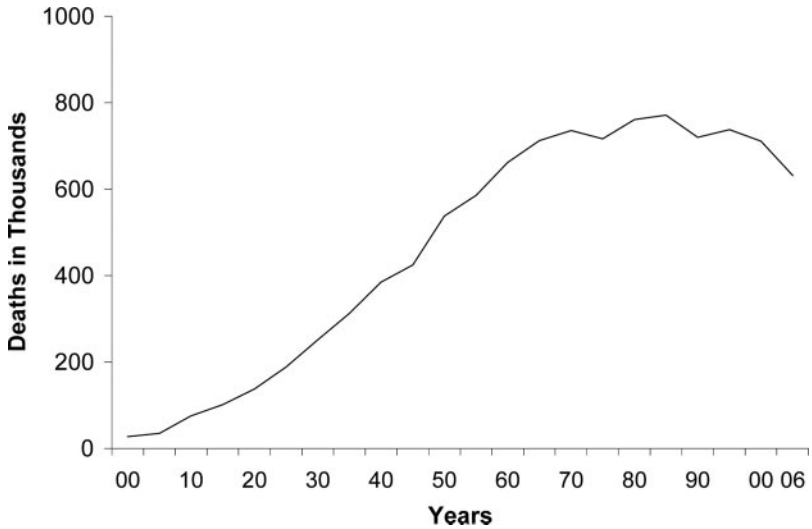

Chart 2-3. Deaths due to diseases of the heart (United States: 1900–2006). See Glossary for an explanation of "diseases of the heart." Source: NCHS.

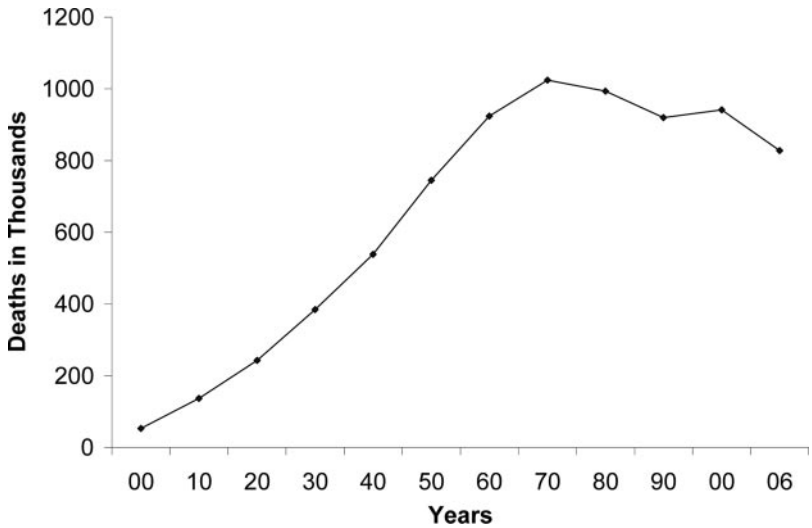

Chart 2-4. Deaths due to CVD (United States: 1900–2006). CVD does not include congenital CVD. Source: NCHS.

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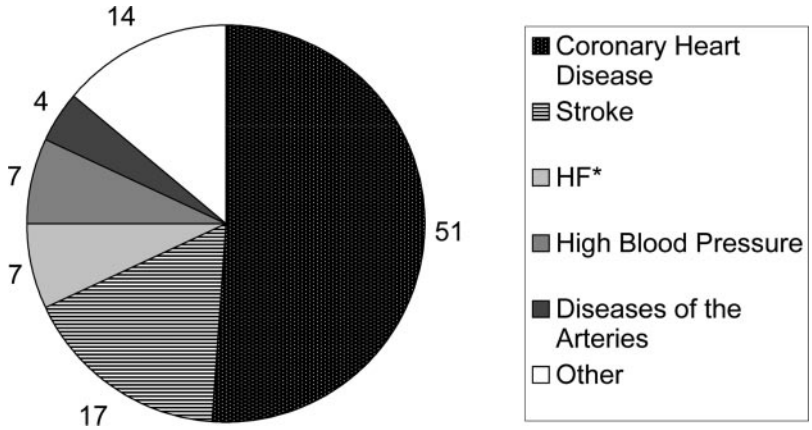

Chart 2-5. Percentage breakdown of deaths due to CVD (United States: 2006). Source: NCHS. *Not a true underlying cause. May not add to 100 because of rounding.

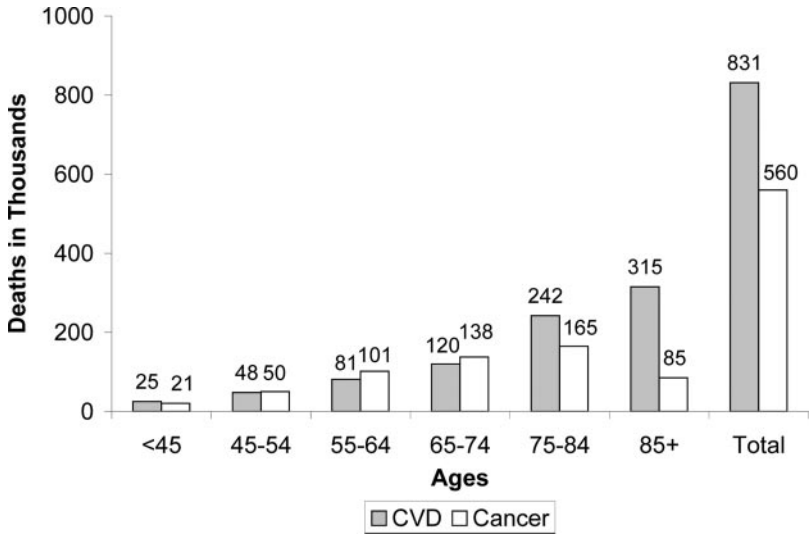

Chart 2-6. CVD deaths vs cancer deaths by age (United States: 2006). Source: NCHS.

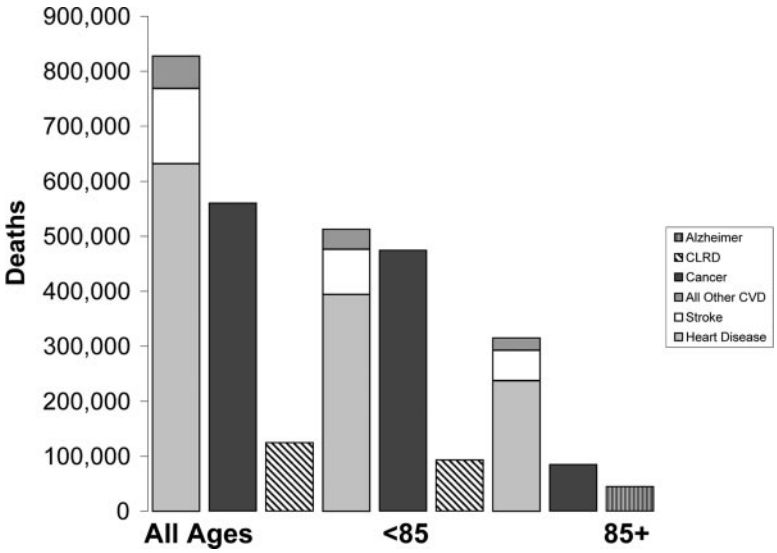

Chart 2-7. CVD and other major causes of death: total, <85 years of age, and ≥85 years of age. Deaths among both sexes, United States, 2006. Source: NCHS and NHLBI.

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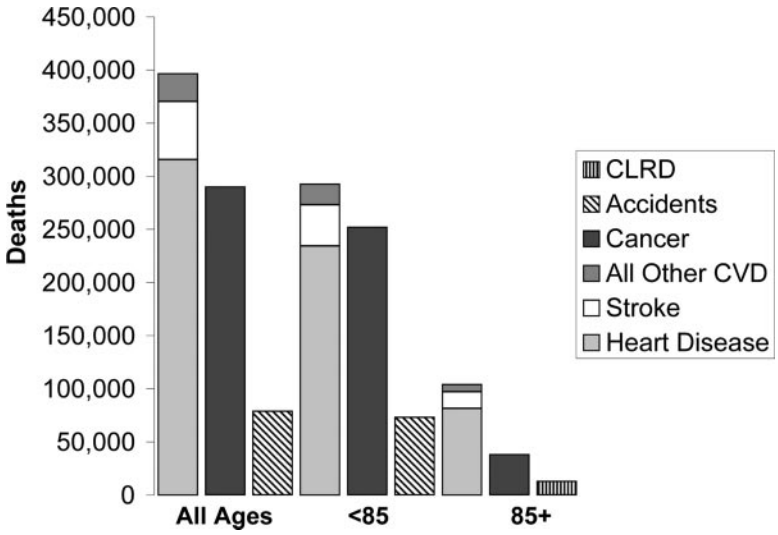

Chart 2-8. CVD and other major causes of death: total, <85 years of age, and ≥85 years of age. Deaths among males, United States, 2006. Source: NCHS and NHLBI.

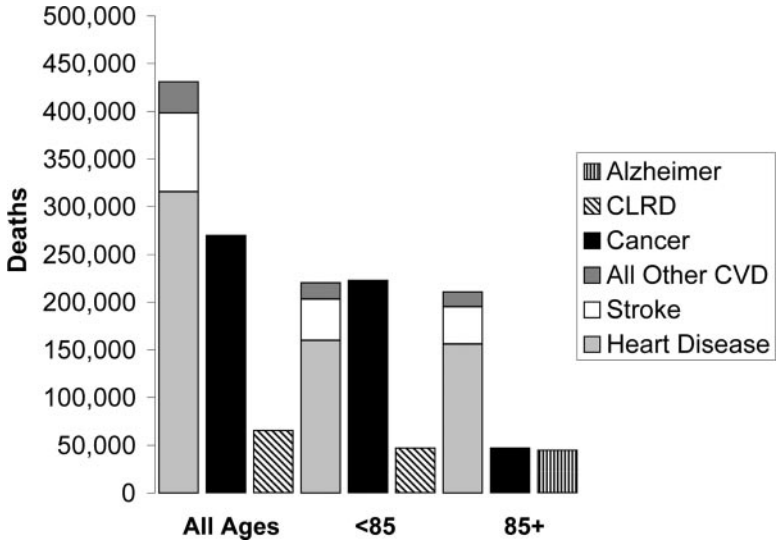

Chart 2-9. CVD and other major causes of death: total, <85 years of age, and ≥85 years of age. Deaths among females, United States, 2006. Source: NCHS and NHLBI.

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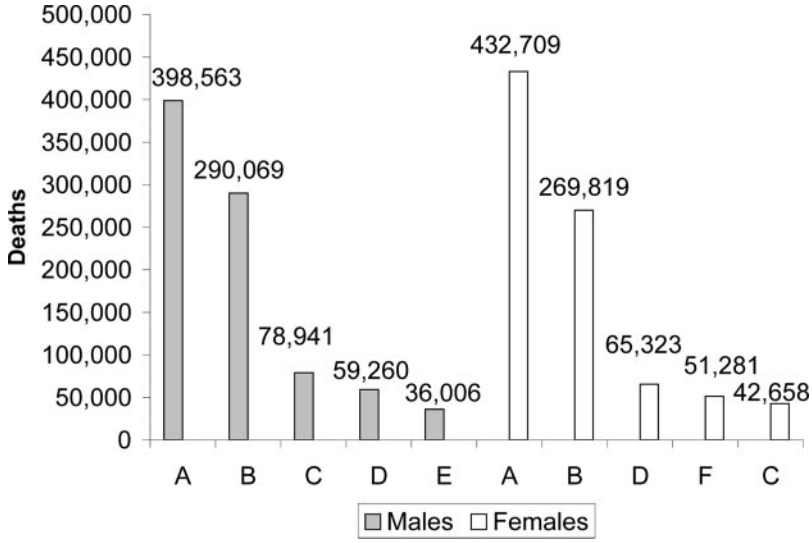

Chart 2-10. CVD and other major causes of death for all males and females (United States: 2006). Source: NCHS and NHLBI. A indicates CVD plus congenital CVD; B, cancer; C, accidents; D, CLRD; E, diabetes; and F, Alzheimer's disease.

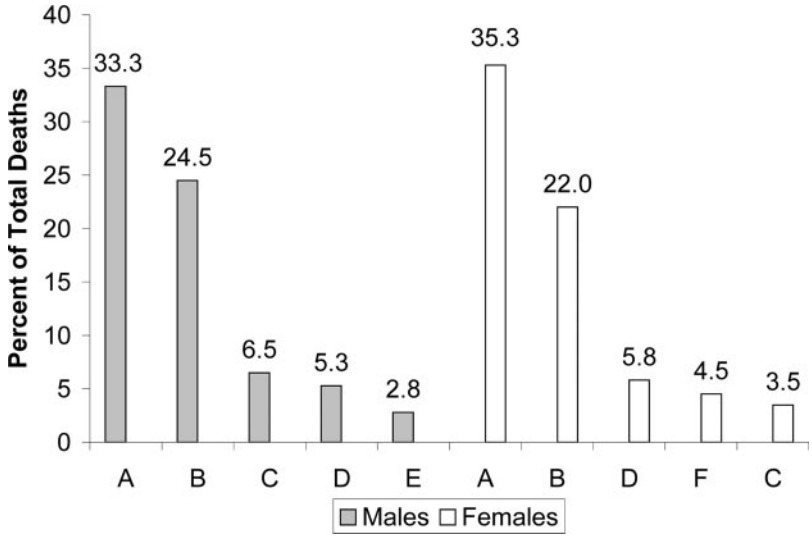

Chart 2-11. CVD and other major causes of death for white males and females (United States: 2006). Source: NCHS. A indicates CVD plus congenital CVD; B, cancer; C, accidents; D, CLRD; E, diabetes; and F, Alzheimer's disease.

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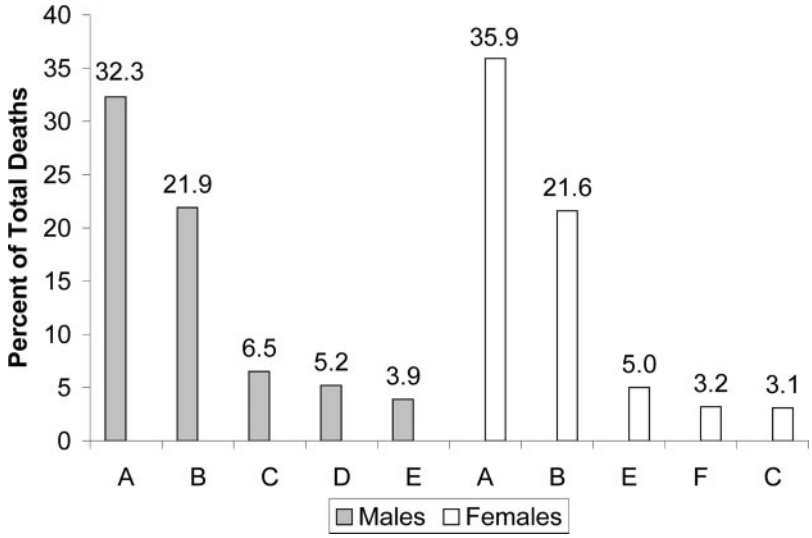

Chart 2-12. CVD and other major causes of death for black males and females (United States: 2006). Source: NCHS and NHLBI. A indicates CVD plus congenital CVD; B, cancer; C, accidents; D, assault (homicide); E, diabetes; and F, nephritis.

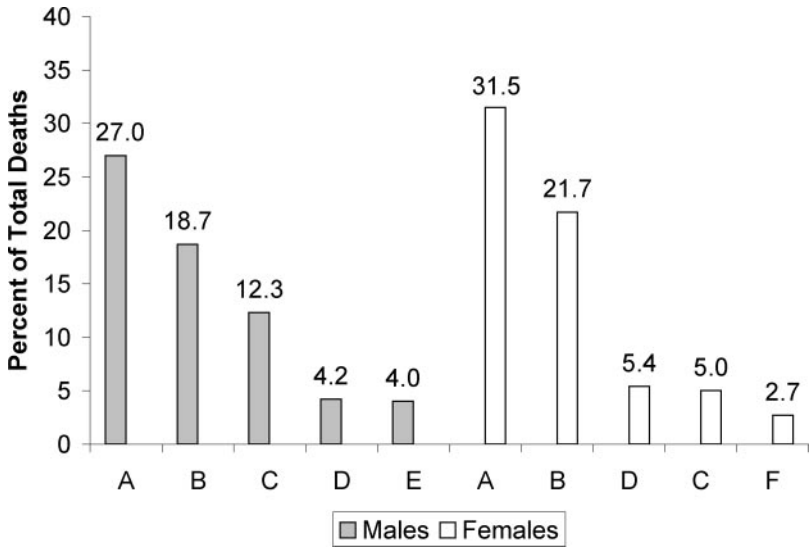

Chart 2-13. CVD and other major causes of death for Hispanic or Latino males and females (United States: 2006). Source: NCHS and NHLBI. A indicates CVD (I00–I99); B, cancer; C, accidents; D, diabetes mellitus; E, assault (homicide); and F, CLRD.

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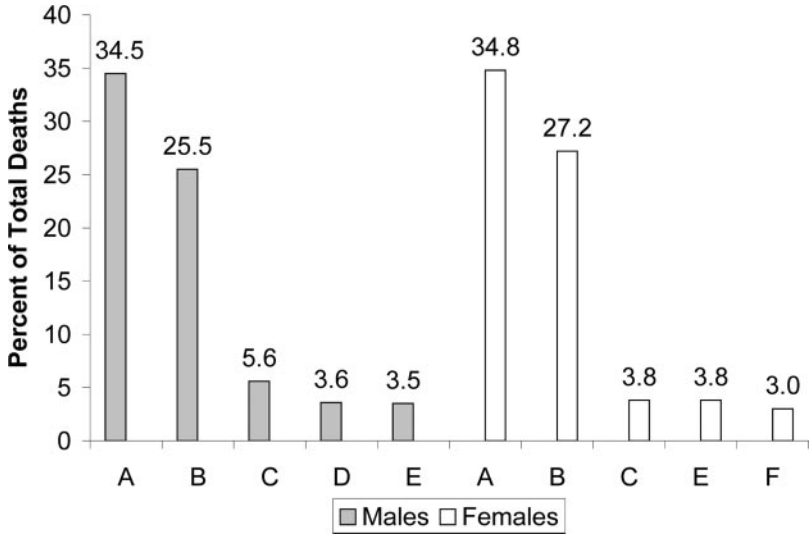

Chart 2-14. CVD and other major causes of death for Asian or Pacific Islander males and females (United States: 2006). Source: NCHS and NHLBI. "Asian or Pacific Islander" is a heterogeneous category that includes people at high CVD risk (eg, South Asian) and people at low CVD risk (eg, Japanese). More specific data on these groups are not available. A indicates CVD (I00–I99); B, cancer; C, accidents; D, CLRD; E, diabetes; and F, influenza and pneumonia.

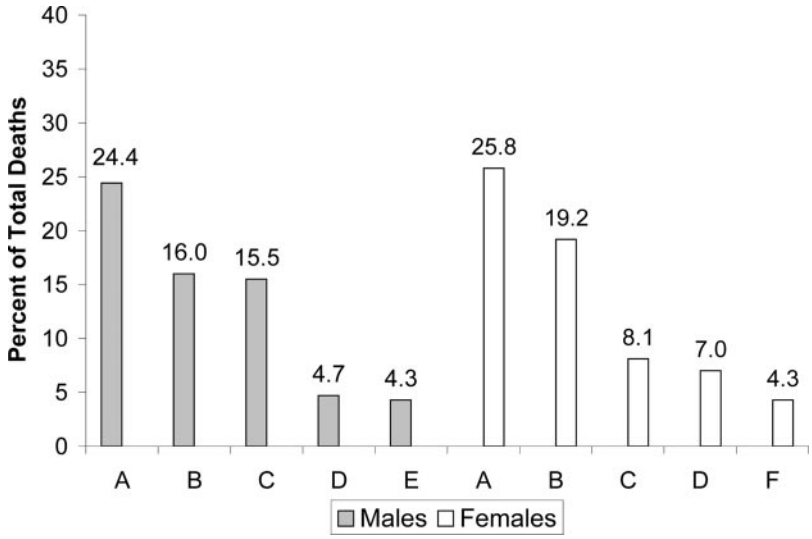

Chart 2-15. CVD and other major causes of death for American Indian or Alaska Native males and females (United States: 2006). Source: NCHS and NHLBI. A indicates CVD (I00–I99); B, cancer; C, accidents; D, diabetes mellitus; E, chronic liver disease; and F, CLRD.

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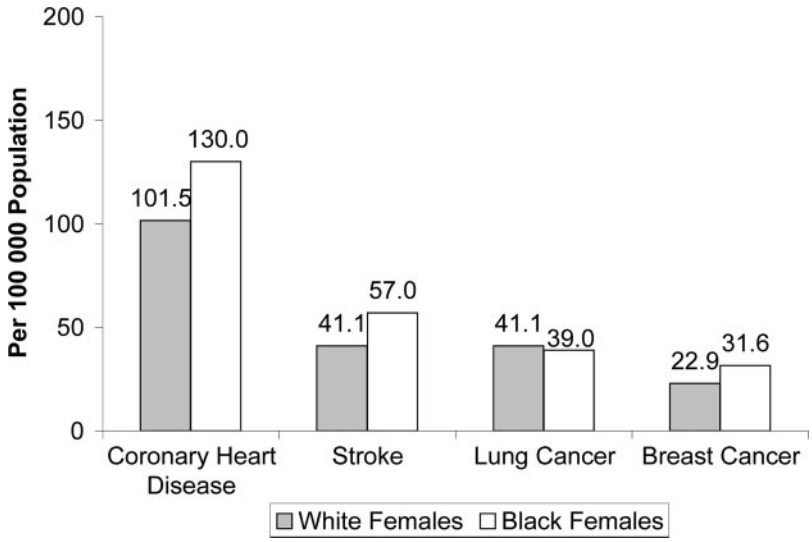

Chart 2-16. Age-adjusted death rates for CHD, stroke, and lung and breast cancer for white and black females (United States: 2006). Source: NCHS.

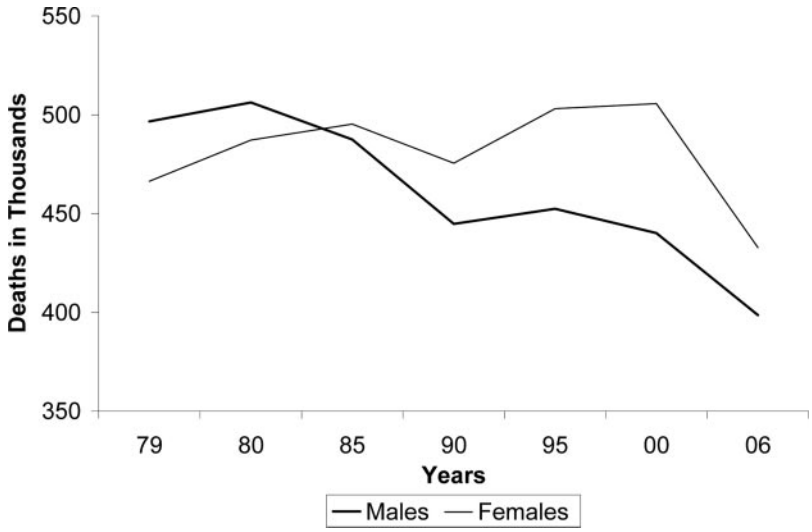

Chart 2-17. CVD mortality trends for males and females (United States: 1979–2006). Source: NCHS. The overall comparability for CVD between the ICD/9 (1979–1998) and ICD/10 (1999–2006) is 0.9962. No comparability ratios were applied.

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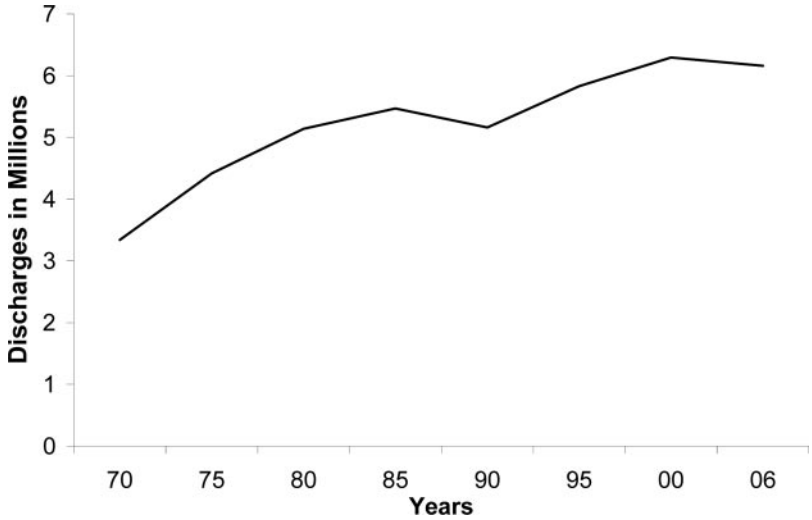

Chart 2-18. Hospital discharges for CVD (United States: 1970–2006). Hospital discharges include people discharged alive, dead, and status unknown. Source: NCHS and NHLBI.

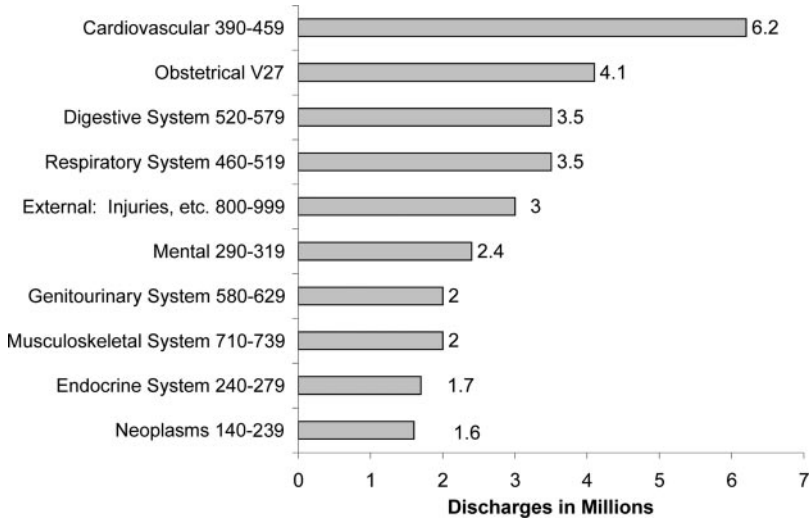

Chart 2-19. Hospital discharges for the 10 leading diagnostic groups (United States: 2006). Source: NHDS/NCHS and NHLBI.

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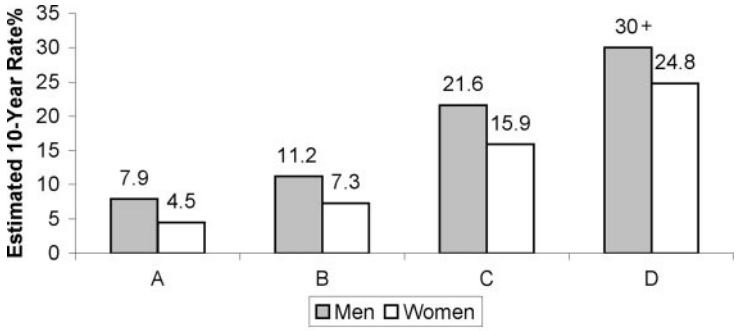

	A	B	C	D
Age	50-54	50-54	50-54	50-54
HDL Cholesterol, mg/dL	45-49	45-49	35-34	35-34
Total Cholesterol, mg/dL	160-199	200-239	200-239	200-239
Systolic BP mm/Hg, no treatment	120-29	130-139	130-139	130-139
Smoker	No	No	No	Yes
Diabetes	No	No	Yes	Yes

Chart 2-20. Estimated average 10-year CVD risk in adults 50 to 54 years of age according to levels of various risk factors (FHS). Source: D'Agostino et al.[79]

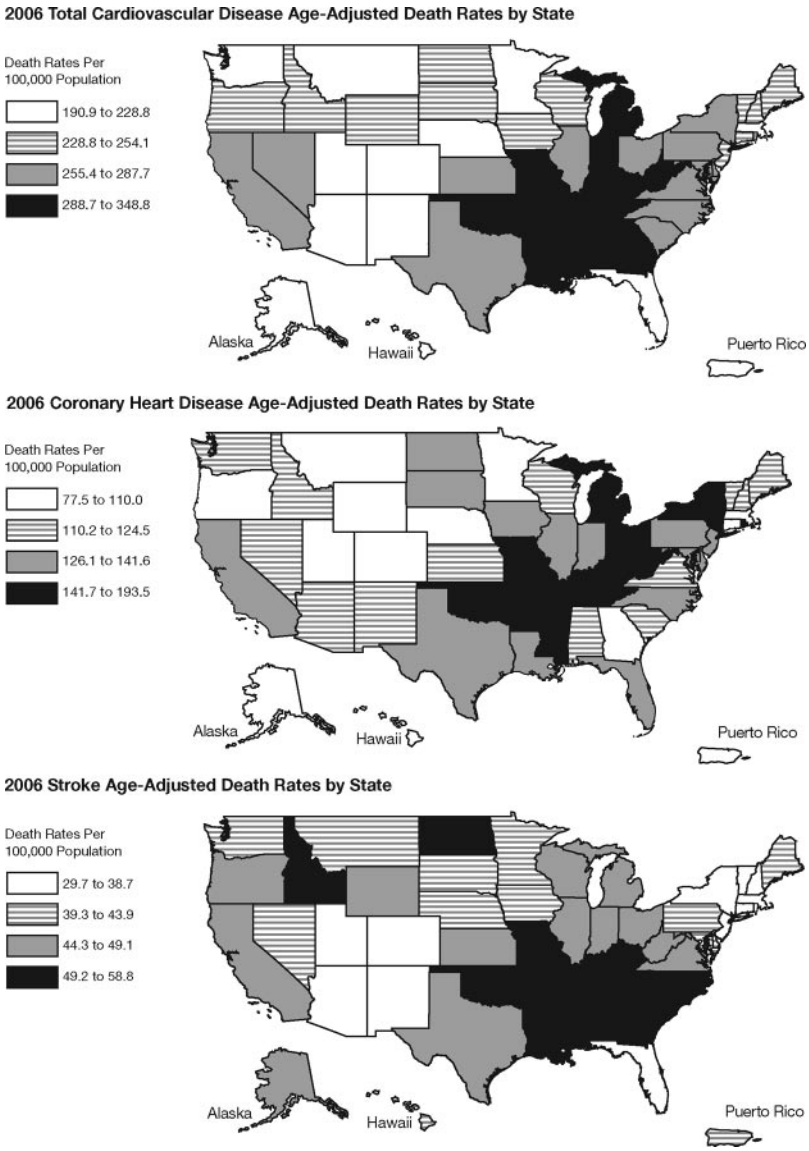

Chart 2-21. US maps corresponding to state death rates (including the District of Columbia).

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3. Subclinical Atherosclerosis

See Table 3-1 and Charts 3-1 through 3-6.

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 artery types 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, which is discussed in Chapter 9. Data on cardiovascular outcomes are more limited (or nonexistent) and/or standards for abnormal tests are less well defined for other modalities for measuring subclinical disease, including brachial artery reactivity testing, aortic and carotid magnetic resonance imaging (MRI), 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. We have therefore chosen to limit our discussion here to CAC and IMT.

Abbreviations Used in Chapter 3

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
FRS	Framingham Risk Score
HDL	high-density lipoprotein
HR	hazard ratio
IMT	intima-media thickness
LDL	low-density lipoprotein
MESA	Multi-Ethnic Study of Atherosclerosis
mg/dL	milligrams per deciliter
MRI	magnetic resonance imaging
NHLBI	National Heart, Lung, and Blood Institute
RR	relative risk
SBP	systolic blood pressure
SD	standard deviation

Coronary Artery Calcification

Background

- CAC is a measure of the burden of atherosclerosis in the heart arteries and is measured by CT. Other parts 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.
- Several guidelines and consensus statements have suggested that screening for CAC may be appropriate in persons at intermediate risk for heart disease (eg, 10-year estimated risk of 10% to 20%) but not for lower-risk general population screening or for persons with preexisting heart disease, diabetes mellitus, or other high-risk conditions.[1,2]
- 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 a score ≥100 or a score ≥75th percentile for one's age and sex. A score ≥400 has been noted to be an indication for further diagnostic evaluation (eg, exercise testing or myocardial perfusion imaging) for coronary artery disease (CAD).

Prevalence

- 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).[3]
  - — 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 subjects had a score that exceeded 100.
  - — Chart 3-1 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 subjects 45 to 84 years of age, including white (n=2619), black (n=1898), Hispanic (n=1494), and Chinese (n=803) men and women.[4]
  - — Chart 3-2 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 3-1 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 blood pressure) could be implemented and/or at which treatment goals might be more aggressive (eg, LDL cholesterol <100 mg/dL instead of <130 mg/dL).

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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).[5]
  - Chart 3-3 shows the RRs or hazard ratios (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. Persons with CAC scores of 1 to 100 had approximately 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 to 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.[6]
  - Chart 3-4 shows the HRs associated with increasing CAC scores (relative to CAC=0 and <10% risk category) in low- (<10%), intermediate- (10% to 15% and 16% to 20%), and high-risk (>20%) Framingham Risk Score (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.[7]

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 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 that they may cause. Epidemiological data, including the data discussed below, have indicated high-risk levels 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), there are not yet any guidelines for the screening of asymptomatic people for 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.[8]

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.[9]
  - The mean values of carotid IMT for the different segments are shown in Chart 3-5 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, systolic BP (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.[10] Higher BMI and LDL cholesterol levels measured at 4 to 7 years of age were associated with increased risk for being above the 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, particularly in the internal carotid, of the 4 ethnic groups (Chart 3-6).
- 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.[11]
  - 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 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.

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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.[12]
  - — 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 to 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.[13]
  - — 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 (SD) increment of log-transformed CAC score versus 1.3-fold for each 1-SD increment of the maximum carotid IMT.
  - — For CHD events, the HRs per 1-SD 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 young adults 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.[14]

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Table 3-1. 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

*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: MESA CAC Tools Web site: http://www.mesa-nhlbi.org/Calcium/input.aspx).

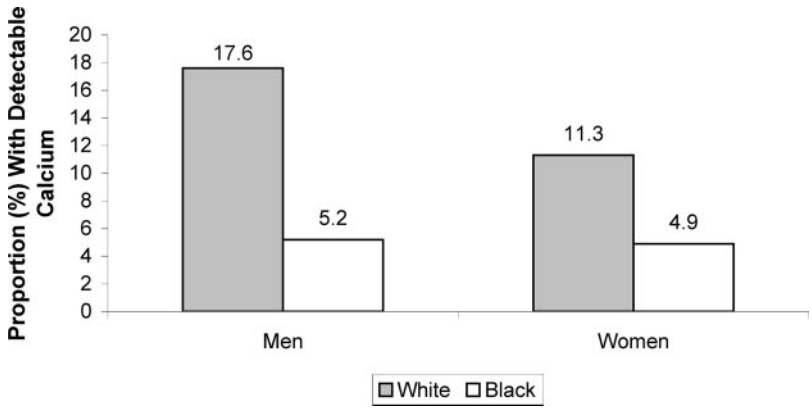

Chart 3-1. Prevalence (%) of coronary calcium: US adults 33 to 45 years of age. Source: Loria et al.[3] $P<0.0001$ across race-sex groups.

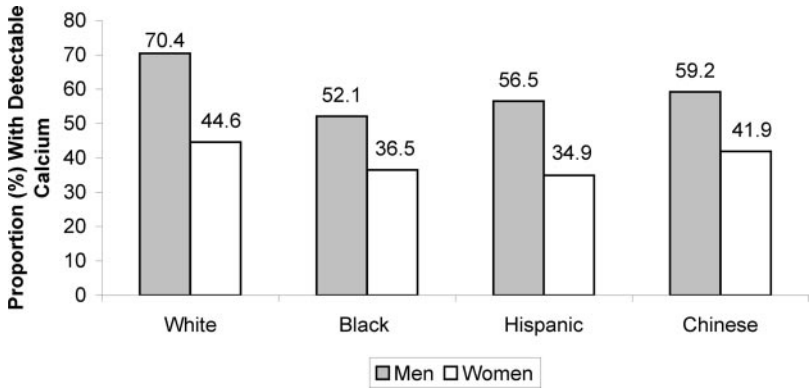

Chart 3-2. Prevalence (%) of coronary calcium: US adults 45 to 84 years of age. Source: Bild et al.[4] $P<0.0001$ across ethnic groups in both men and women.

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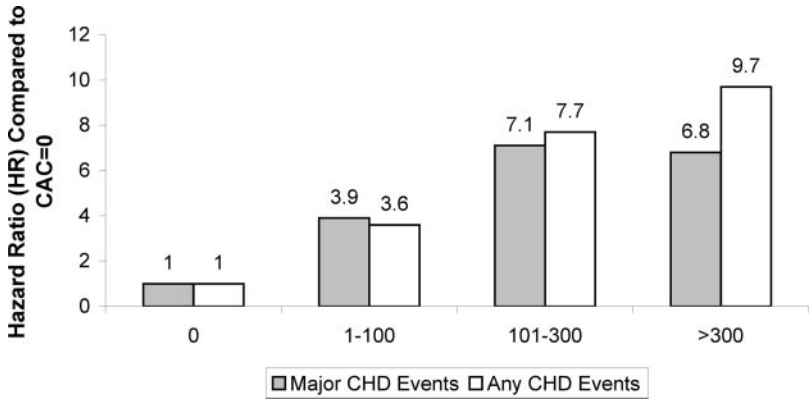

Chart 3-3. HRs for CHD events associated with coronary calcium scores: US adults 45 to 84 years of age (reference group CAC=0). Source: Detrano et al.[5] All HRs $P<0.0001$. Major CHD events included MI and death due to CHD; any CHD events included major CHD events plus definite angina or definite or probable angina followed by revascularization.

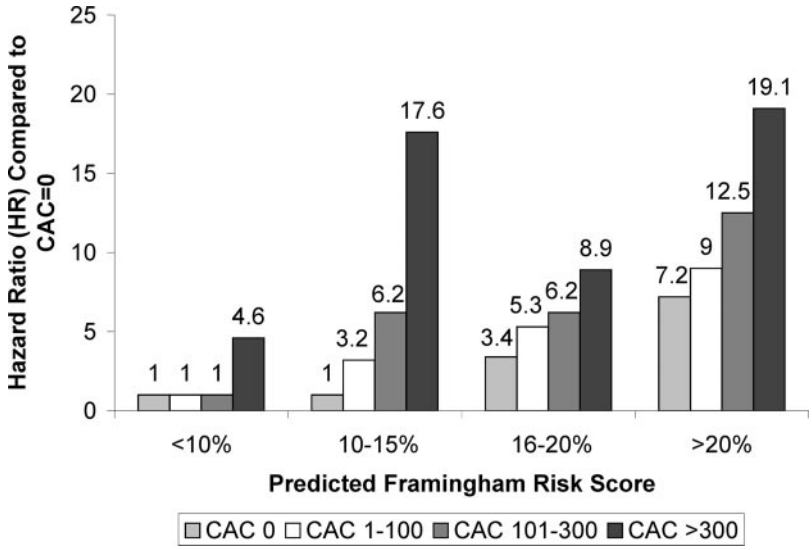

Chart 3-4. HRs for CHD events associated with coronary calcium scores: US adults (reference group CAC=0 and FRS <10%). CHD events included nonfatal MI and death due to CHD. Source: Greenland et al.[6]

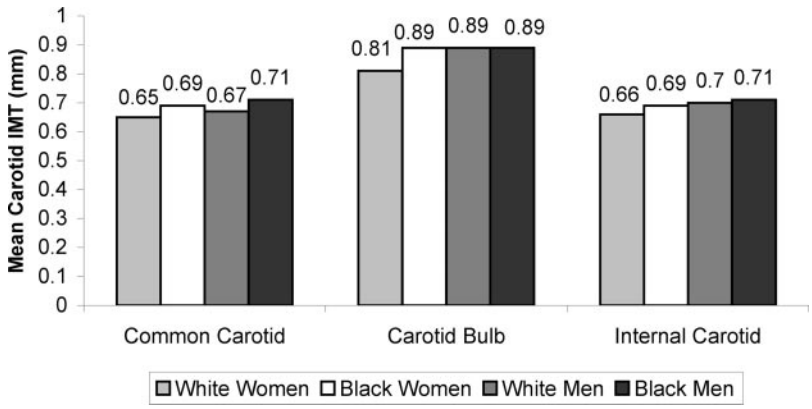

Chart 3-5. Mean values of carotid IMT for different carotid artery segments in younger adults by race and sex (Bogalusa Heart Study). Source: Urbina et al.[9]

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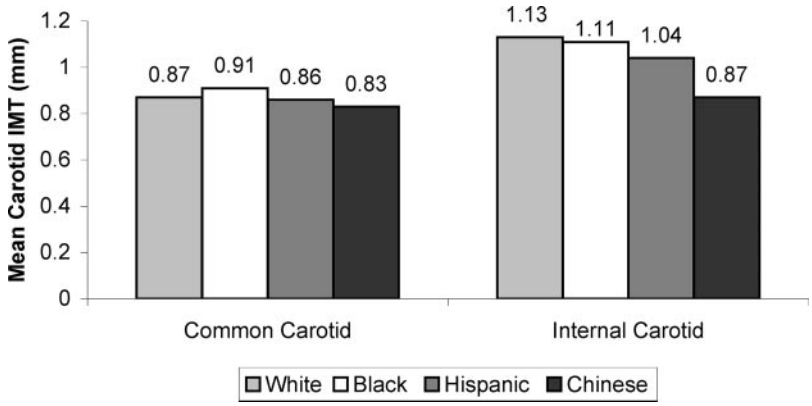

Chart 3-6. Mean values of carotid IMT for different carotid artery segments in older adults, by race. Source: Manolio et al.[12]

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

Coronary Heart Disease

ICD-9 410 to 414, 429.2; ICD-10 I20–I25; see Glossary (Chapter 22) for details and definitions. See Tables 4-1 and 4-2. See Charts 4-1 through 4-8.

Prevalence

- On the basis of data from NHANES 2003 to 2006 (NCHS; Table 4-1; Chart 4-1), an estimated 17 600 000 Americans ≥20 years of age have CHD:
  - Total CHD prevalence is 7.9% in US adults ≥20 years of age. CHD prevalence is 9.1% for men and 7.0% for women.
  - Among non-Hispanic whites, CHD prevalence is 9.4% for men and 6.9% for women.
  - Among non-Hispanic blacks, CHD prevalence is 7.8% for men and 8.8% for women.
  - Among Mexican Americans, CHD prevalence is 5.3% for men and 6.6% for women.
  - Among Hispanic or Latino individuals ≥18 years of age, CHD prevalence is 5.7% (NHIS, NCHS).[1]
  - Among American Indians/Alaska Natives ≥18 years of age, it is estimated that 6.6% have CHD (estimate considered unreliable), and among Asians ≥18 years of age, it is 2.9% (NHIS, NCHS).[1]
- According to data from NHANES 2003 to 2006 (NCHS) the overall prevalence for MI is 3.6% in US adults ≥20 years of age. MI prevalence is 4.7% for men and 2.6% for women.[1]
  - Among non-Hispanic whites, MI prevalence is 5.1% for men and 2.6% for women.
  - Among non-Hispanic blacks, MI prevalence is 3.6% for men and 2.9% for women.
  - Among Mexican Americans, MI prevalence is 2.6% for men and 2.0% for women.
- Data from 2008 from the BRFSS survey of the CDC found that 4.2% of respondents had been told that they had had an MI. The highest prevalence was in Alabama (5.8%) and West Virginia (7.6%). The lowest prevalence was in the District of Columbia (2.2%). In the same survey, 4.1% of respondents were told that they had angina or CHD. The highest prevalence was in West Virginia (8.1%), and the lowest was in the District of Columbia (2.5%).[2]

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

Abbreviations Used in Chapter 4

ACC	American College of Cardiology
ACS	acute coronary syndrome
AHA	American Heart Association
AMI	acute myocardial infarction
AP	angina pectoris
ARIC	Atherosclerosis Risk in Communities study
ATP III	Adult Treatment Panel III
BMI	body mass index
BP	blood pressure
BRFSS	Behavioral Risk Factor Surveillance System
CABG	Coronary artery bypass graft
CAD	coronary artery disease
CDC	Centers for Disease Control and Prevention
CHD	coronary heart disease
CHS	Cardiovascular Health Study
CI	confidence interval
CRUSADE	Can Rapid stratification of Unstable angina patients Suppress ADverse outcomes with Early implementation of the ACC/AHA guidelines
CVD	cardiovascular disease
DM	diabetes mellitus
ECG	electrocardiogram
ED	emergency department
EHS-ACS-II	Second Euro Heart Survey on ACS
EMS	emergency medical services
FHS	Framingham Heart Study
GRACE	Global Registry of Acute Coronary Events
HD	heart disease
HF	heart failure
HMO	Health maintenance organization
HR	hazard ratio
ICD	International Classification of Diseases
INTERHEART	Interheart Study
MET	metabolic equivalent
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
NHIS	National Health Interview Study
NHLBI	National Heart, Lung, and Blood Institute
NRMI	National Registry of Myocardial Infarction
NSTE ACS	non–ST-segment–elevation acute coronary syndromes
NSTEMI	non–ST-segment–elevation myocardial infarction
OR	odds ratio
PA	physical activity
PCI	percutaneous coronary intervention
PREMIER	Prospective Registry Evaluating Myocardial Infarction: Events and Recovery
STEMI	ST-segment–elevation MI
UA	unstable angina
WISE	Women's Ischemia Syndrome Evaluation

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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.[3,4]
— 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.[3]
  — The lifetime risk of developing CHD after 40 years of age is 49% for men and 32% for women.[5]
  — 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.[3]
- 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. In a multivariable analysis, hypertension was a particularly strong risk factor in black women, with HR ratios (95% confidence interval [CI]) as follows: Black women, 4.8 (2.5 to 9.0); white women, 2.1 (1.6 to 2.9); black men, 2.0 (1.3 to 3.0); and white men, 1.6 (1.3 to 1.9). DM was somewhat more predictive in white women than in other groups. HR ratios (95% CI) were as follows: Black women, 1.8 (1.2 to 2.8); white women, 3.3 (2.4 to 4.6); black men, 1.6 (1.1 to 2.5); and white men, 2.0 (1.6 to 2.6).[6]
- Incidence rates for MI in the NHLBI-sponsored ARIC study are displayed in Charts 4-3 and 4-4, stratified by age, race, and gender. 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.[7]
- Analysis of more than 40 years of physician-validated acute MI (AMI) data in the FHS study of the NHLBI found that AMI rates diagnosed by electrocardiographic (ECG) criteria declined approximately 50% with a concomitant 2-fold increase in rates of AMI diagnosed by blood markers. National MI trend data may be biased by a diagnostic drift that has resulted from the advent of diagnostic biomarker tests for AMI; investigators were able to identify and quantify the possible magnitude of this effect within the study setting. These findings may explain the paradoxical stability of AMI rates in the United States despite concomitant improvements in CHD risk factors.[8]
- 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.[9] Analysis of data from NHANES III (1988–1994) and NHANES 1999 to 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.[10]

Mortality

- CHD caused ≈1 of every 6 deaths in the United States in 2006. CHD mortality was 425 425.[11] CHD any-mention mortality was 587 000. MI mortality was 141 462. MI any-mention mortality was 181 000 (NHLBI; NCHS public-use data files). Preliminary 2007 CHD mortality was 403 741. The preliminary 2007 CHD death rate was 125.2.[12] CHD is the largest major killer of American males and females.[13] Approximately every 25 seconds, an American will suffer 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 2006 was 70%. According to NCHS Data Warehouse mortality data, 309 000 CHD deaths occur out of the hospital or in hospital EDs annually (2005, ICD-10 codes I20 to I25).[14]
- A study of 1275 health maintenance organization (HMO) 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]

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.[16]
- From 1996 to 2006, the annual death rate due to CHD declined 36.4%, and the actual number of deaths declined 21.9%. (Appropriate comparability ratios were applied.) In 2006, the overall CHD death rate was 134.9 per 100 000 population. The death rates were 176.3 for white males and 206.4 for black males; for white females, the rate was 101.5, and for black females, it was 130.0.[11,13]
  — 2006 Age-adjusted death rates for CHD were 132.8 for Hispanic or Latino males and 85.4 for females, 122.4 for American Indian or Alaska Native males and 76.4 for females, and 101.3 for Asian or Pacific Islander males and 58.9 for females.[11]
- 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 15.[17]
- On the basis of data from the FHS of the NHLBI[3]:

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— 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.

- According to data from the National Registry of Myocardial Infarction[18]:

  — 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.

- 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 due 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[19]:

  — Secondary preventive therapies after MI or revascularization (11%);
  — initial treatments for AMI or unstable angina (UA; 10%);
  — treatments for HF (9%);
  — revascularization for chronic angina (5%); and
  — 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[19]:

  — Lower total cholesterol (24%);
  — lower SBP (20%);
  — lower smoking prevalence (12%); and
  — decreased physical inactivity (5%).
  — Nevertheless, these favorable improvements in risk factors were offset in part by increases in BMI and in diabetes prevalence, which accounted for an increased number of deaths (8% and 10%, respectively).

- Between 1980 and 2002, death rates due 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 to 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.[20]
- 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.[21]

Risk Factors

- Risk factors for CHD act synergistically to increase CHD risk, as shown in the example in Chart 4-6.
- A study of men and women in 3 prospective cohort studies found that antecedent major CHD risk factor exposures were very common among those who developed CHD. Approximately 90% of CHD patients 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 diabetes.[22]
- 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, diabetes, abdominal obesity, a lack of PA, low daily fruit and vegetable consumption, alcohol overconsumption, and psychosocial index.[23]
- 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 (MET) increment in exercise capacity reduced risk by 13%.[24]
- A study of non-Hispanic white persons 35 to 74 years of age in the FHS (NHLBI) and the NHANES III (NCHS) studies showed that 26% of men and 41% of women had at least 1 borderline risk factor in NHANES III. It is estimated that >90% of CHD events will occur in individuals with at least 1 elevated risk factor and that ≈8% will occur in people with only borderline levels of multiple risk factors. Absolute 10-year CHD risk exceeded 10% both in men >45 years of age who had 1 elevated risk factor and ≥4 borderline risk factors and in those who had ≥2 elevated risk factors. In women, absolute CHD risk was >10% only in those >55 years of age who had ≥3 elevated risk factors.[25]
- A recent analysis examined the number and combination of risk factors necessary to exceed Adult Treatment Panel III (ATP III) treatment thresholds. In this analysis, relatively high risk factor levels were required to exceed ATP III treatment thresholds in men <45 years of age and women <65 years of age, which suggests that alternative means of

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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.[26]

- Analysis of data from the CHS study (NHLBI) among participants ≥65 years of age at entry into the study showed that subclinical CVD is very 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.[27]
- 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.[28]
- 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.[29]

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.[30]
- 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%).[31]
- 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.[32]
- A recent community surveillance study in 4 US communities reported that in 2000, the overall proportion of persons 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 MI patients who arrived at the hospital by 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.[33]
- 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.[34]
- 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.[35]
- Data from Worcester, Mass, 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. Compared with those arriving within 2 hours of symptom onset, those with prolonged prehospital delay were less likely to receive thrombolytic therapy and percutaneous coronary intervention (PCI) within 90 minutes of hospital arrival.[36]
- In an analysis from ARIC, low neighborhood household income (OR 1.46, 95% CI 1.09 to 1.96) and being a Medicaid recipient (OR 1.87, 95% CI 1.10 to 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.[37]

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,

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sudden death, AP, HF, and stroke—for both men and women—is substantial (FHS, NHLBI).[3]

- 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.[38]
- On the basis of pooled data from the FHS, ARIC, and CHS studies of the NHLBI, within 1 year after a first MI:
  - At ≥40 years of age, 18% of men and 23% of women will die.
  - At 40 to 69 years of age, 8% of white men, 12% of white women, 14% of black men, and 11% of black women will die.
  - At ≥70 years of age, 27% of white men, 32% of white women, 26% of black men, and 28% 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 ≥40 years of age, 33% of men and 43% of women will die.
  - At 40 to 69 years of age, 15% of white men, 22% of white women, 27% of black men, and 32% of black women will die.
  - At ≥70 years of age, 50% of white men, 56% of white women, 56% of black men, and 62% 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 40 to 69 years of age, 16% of men and 22% of women.
  - At 40 to 69 years of age, 14% of white men, 18% of white women, 27% of black men, and 29% of black women.
  - At ≥70 years of age, 24% of white men and women, 30% of black men, and 32% of black women.
- The percentage of persons with a first MI who will have HF in 5 years is:
  - At 40 to 69 years of age, 7% of men and 12% of women.
  - At ≥70 years of age, 22% of men and 25% of women.
  - At 40 to 69 years of age, 7% of white men, 11% of white women, 11% of black men, and 14% of black women.
  - At ≥70 years of age, 21% of white men, 25% of white women, 29% of black men, and 24% of black women.
- The percentage of persons with a first MI who will have a stroke within 5 years is:
  - At 40 to 69 years of age, 4% of men and 6% of women.
  - At ≥70 years of age, 6% of men and 11% of women.
  - At 40 to 69 years of age, 3% of white men, 5% of white women, 8% of black men, and 9% of black women.
  - At ≥70 years of age, 6% of white men, 10% of white women, 7% of black men, and 17% of black women.
- The percentage of persons with a first MI who will experience sudden death in 5 years is:
  - At 40 to 69 years of age, 1.1% of white men, 1.9% of white women, 2.5% of black men, and 1.4% of black women.
  - At ≥70 years of age, 6.0% of white men, 3.5% of white women, 14.9% of black men, and 4.8% of black women.
- The median survival time (in years) after a first MI is:
  - At 60 to 69 years of age, data not available for men and 7.4 for women.
  - At 70 to 79 years of age, 7.4 for men and 10.4 for women.
  - At ≥80 years of age, 2.0 for men and 6.4 for women.
- 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.[39]
- 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 persons, women, nonwhites, and individuals with comorbidities were less likely to enroll in cardiac rehabilitation programs.[40]

Hospital Discharges and Ambulatory Care Visits

(See Table 4-1 and Chart 4-7.)

- From 1996 to 2006, the number of inpatient discharges from short-stay hospitals with CHD as the first-listed diagnosis decreased from 2 272 000 to 1 760 000 (NHDS, NCHS).
- In 2007, there were 14 722 000 ambulatory care visits with CHD as the first-listed diagnosis (NCHS, NAMCS, NHAMCS). The majority of these visits (69.0%) were for coronary atherosclerosis.[41]
- 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 nonspecific chest pain stays among patients ≥85 years of age, higher than for any other condition examined. For AMI, one third

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more women than men died in the hospital: 9.3% of women died in the hospital compared with 6.2% of men.[42]

Operations and Procedures

- In 2006, an estimated 1 313 000 inpatient PCI procedures, 448 000 inpatient bypass procedures, 1 115 000 inpatient diagnostic cardiac catheterizations, 114 000 inpatient implantable defibrillators, and 418 000 pacemaker procedures were performed for inpatients in the United States.[43]

Cost

- The estimated direct and indirect cost of CHD for 2010 is $177.1 billion.
- 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).[35,44]

Acute Coronary Syndrome

ICD-9 codes 410, 411.

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, non–ST-segment–elevation MI (NSTEMI), and ST-segment–elevation MI (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 2006 is 733 000. Of these, an estimated 401 000 are male and 332 000 are female. This estimate is derived by adding the first-listed inpatient hospital discharges for MI (647 000) to those for UA (86 000; NHDS, NCHS).
- When secondary discharge diagnoses in 2006 were included, the corresponding number of inpatient hospital discharges was 1 365 000 unique hospitalizations for ACS; 765 000 were male, and 600 000 were female. Of the total, 810 000 were for MI alone, and 537 000 were for UA alone (18 000 hospitalizations received both diagnoses; NHDS, NCHS).

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 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 MI patients are STEMI patients.[45] The AHA Get With the Guidelines project found that 32% of the MI patients in the CAD module are STEMI patients (personal communication from AHA Get With the Guidelines staff, October 1, 2007). The study of the Global Registry of Acute Coronary Events (GRACE), 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 ACS patients have STEMI.[46]

- 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 (NSTE ACS). These changes have been accompanied by significant decreases in the rates of in-hospital death, cardiogenic shock, and new MI among patients with NSTE 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.[47]
- A study of patients with NSTE 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.[48]
- 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.[48]

Angina Pectoris

ICD-9 413; ICD-10 I20. See Table 4-2; and Chart 4-5.

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.[49]

Incidence

- Only 18% of coronary attacks are preceded by long-standing AP (NHLBI computation of FHS follow-up since 1986).

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- 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).[7]
- 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.[7]

Mortality

A small number of deaths resulting from CHD are coded as being due to AP. These are included as a portion of total deaths from 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.[50]

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Table 4-1. Coronary Heart Disease

Population Group	Prevalence, CHD, 2006 Age ≥20 y	Prevalence, MI, 2006 Age ≥20 y	New and Recurrent MI and Fatal CHD, Age ≥35 y	New and Recurrent MI, Age ≥35 y	Mortality,* CHD, 2006, All Ages	Mortality,* MI, 2006, All Ages	Hospital Discharges, CHD, 2006, All Ages	Cost, CHD, 2010
Both sexes	17 600 000 (7.9%)	8 500 000 (3.6%)	1 255 000	935 000	425 425	141 462	1 760 000	$177.1 Billion
Males	9 200 000 (9.1%)	5 000 000 (4.7%)	740 000	565 000	224 510 (52.8%)‡	76 089 (53.8%)‡	1 056 000	…
Females	8 400 000 (7.0%)	3 500 000 (2.6%)	515 000	370 000	200 915 (47.2%)‡	65 373 (46.2%)‡	704 000	…
NH white males	9.4%	5.1%	675 000§	…	196 396	66 946	…	…
NH white females	6.9%	2.6%	445 000§	…	175 049	56 603	…	…
NH black males	7.8%	3.6%	70 000§	…	22 713	7392	…	…
NH black females	8.8%	2.9%	65 000§	…	21 817	7384	…	…
Mexican American males	5.3%	2.6%	…	…	…	…	…	…
Mexican American females	6.6%	2.0%	…	…	…	…	…	…
Hispanic or Latino,† age ≥18 y	5.7%	…	…	…	…	…	…	…
Asian,† age ≥18 y	2.9%	…	…	…	…	…	…	…
American Indian/ Alaska Native,† age ≥18 y	6.6%	…	…	…	…	…	…	…

NH indicates non-Hispanic.

CHD includes acute MI (I21, I22), other acute ischemic (coronary) heart disease (I24), AP (I20), atherosclerotic CVD (I25.0), and all other forms of ischemic CHD (I25.1–I25.9). Ellipses indicate data not available. Sources: Prevalence: NHANES 2003–2006 (NCHS) and NHLBI. Percentages for racial/ethnic groups are age adjusted for Americans ≥20 years of age. Age-specific percentages are extrapolated to the 2006 US population estimates. These data are based on self-reports. Incidence: ARIC (1987–2004), NHLBI. Mortality: NCHS (these data represent underlying cause of death only). Hospital discharges: NHDS, NCHS (data include those inpatients discharged alive, dead, or status unknown). Cost: NHLBI; data include estimated direct and indirect costs for 2009.

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

†NHIS, NCHS 2008; data are weighted percentages for Americans ≥18 years of age.[1]

‡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.

Table 4-2. Angina Pectoris

Population Group	Prevalence, 2006 Age ≥20 y	Incidence of Stable AP, Age ≥45 y	Hospital Discharges, 2006,* All Ages
Both sexes	10 200 000 (4.6%)	500 000	41 000
Males	4 700 000 (4.6%)	320 000	17 000
Females	5 500 000 (4.6%)	180 000	24 000
NH white males	4.7%	…	…
NH white females	4.5%	…	…
NH black males	4.0%	…	…
NH black females	5.4%	…	…
Mexican American males	2.9%	…	…
Mexican American females	4.8%	…	…

NH indicates non-Hispanic.

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 MI. Ellipses indicate data not available.

Sources: Prevalence: NHANES 2003–2006 (NCHS) and NHLBI; percentages for racial/ethnic groups are age adjusted for Americans ≥20 years of age. The prevalence of AP is based on responses to the Rose angina questionnaire and the question, "Have you ever been told of having angina?" Estimates from NHANES 2003–2006 (NCHS) were applied to 2006 population estimates (≥20 years of age). Incidence: AP uncomplicated by an MI or with no MI (FHS 1980 to 2001–2003 of the original cohort and 1980 to 1998–2001 of the Offspring Cohort, NHLBI). Hospital discharges: NHDS, NCHS; data include those inpatients discharged alive, dead, or status unknown.

*There were 86 000 days of care for discharges with AP from short-stay hospitals in 2006.

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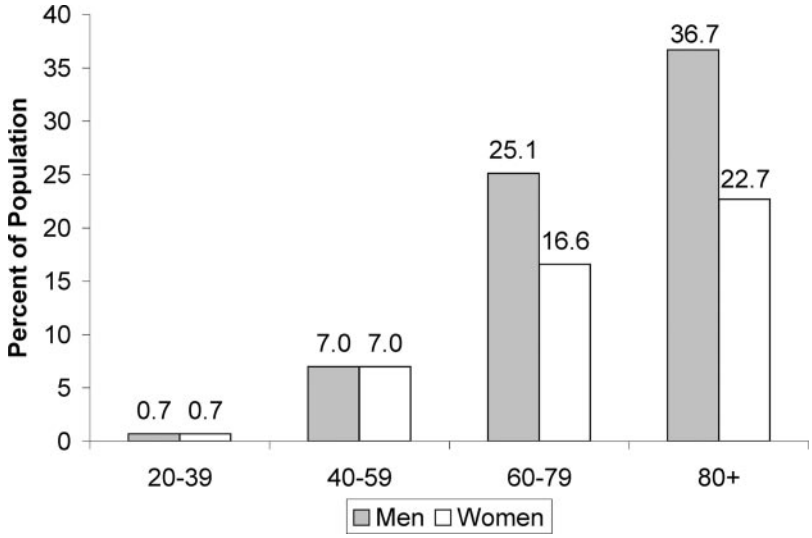

Chart 4-1. Prevalence of CHD by age and sex (NHANES: 2003–2006). Source: NCHS and NHLBI.

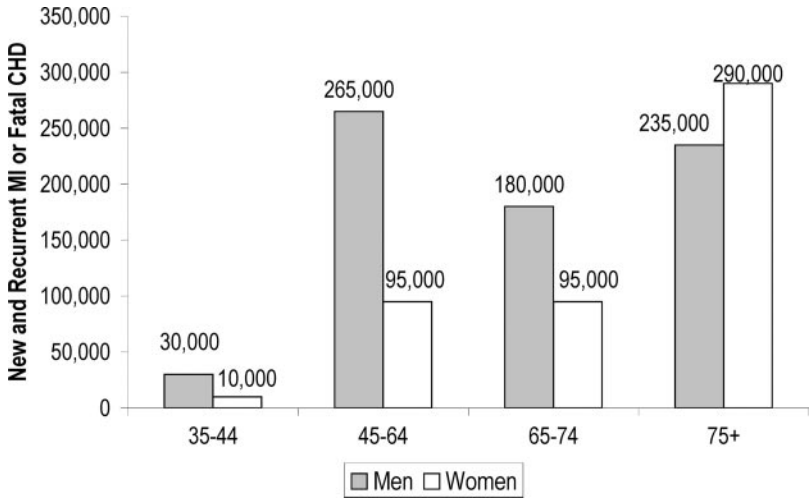

Chart 4-2. Annual number of adults having diagnosed heart attack by age and sex (ARIC Surveillance: 1987–2004 and CHS: 1989–2004). These data include MI and fatal CHD but not silent MI. Source: NHLBI.

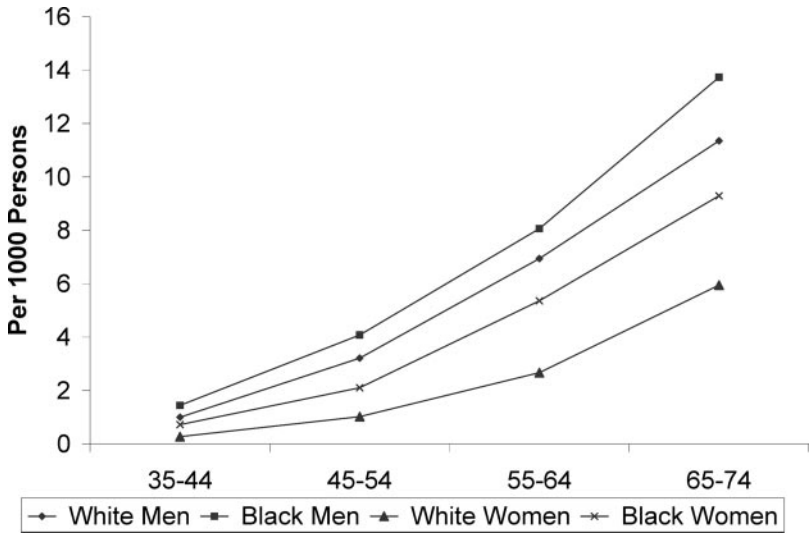

Chart 4-3. Annual rate of first heart attacks by age, sex, and race (ARIC Surveillance: 1987–2004). Source: NHLBI.

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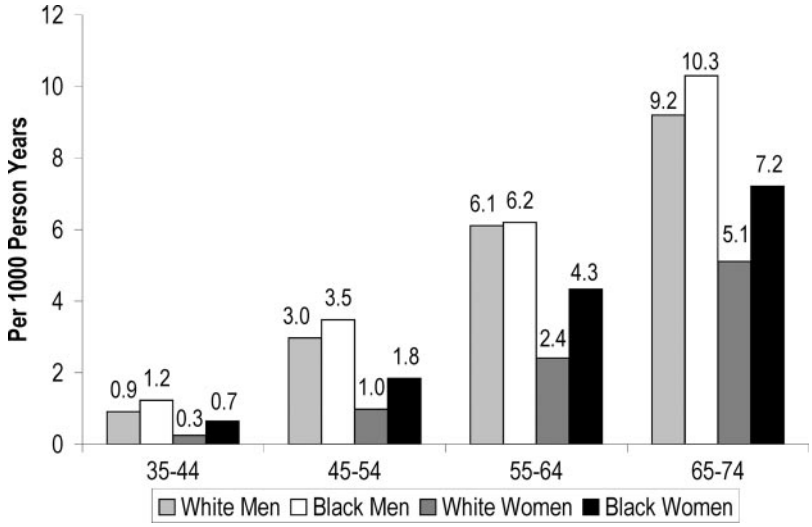

Chart 4-4. Incidence of MI* by age, race, and sex (ARIC Surveillance, 1987–2004). *MI diagnosis by expert committee based on review of hospital records. Source: Unpublished data from ARIC, NHLBI.

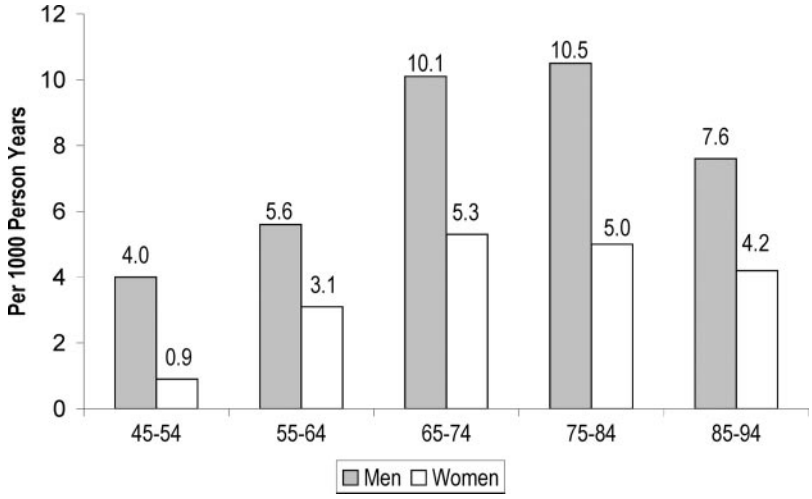

Chart 4-5. Incidence of AP* by age, race, and sex (FHS 1980–2002/2003). *AP uncomplicated based on physician interview of patient. (Rate for women 45–54 years of age considered unreliable.) Source: NHLBI.[7]

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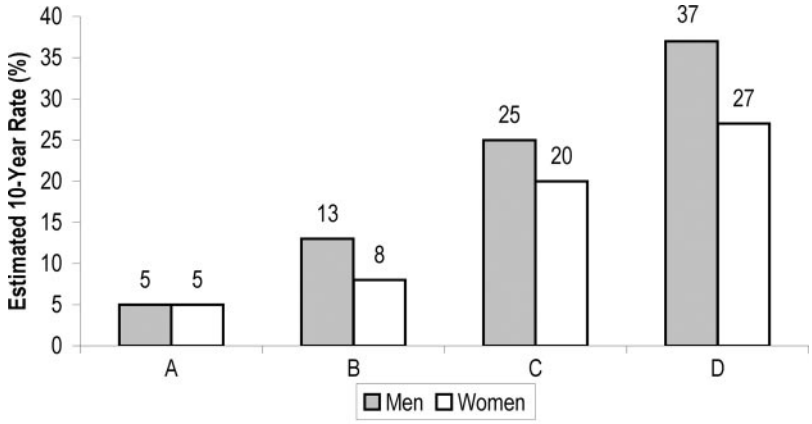

	A	B	C	D
Blood pressure	120/80	140/90	140/90	140/90
Cholesterol	200	240	240	240
HDL-C	50	50	40	40
Diabetes	No	No	Yes	Yes
Cigarettes	No	No	No	Yes

Chart 4-6. Estimated 10-year CHD risk in adults 55 years of age according to levels of various risk factors (Framingham Heart Study). Source: Wilson et al.[51]

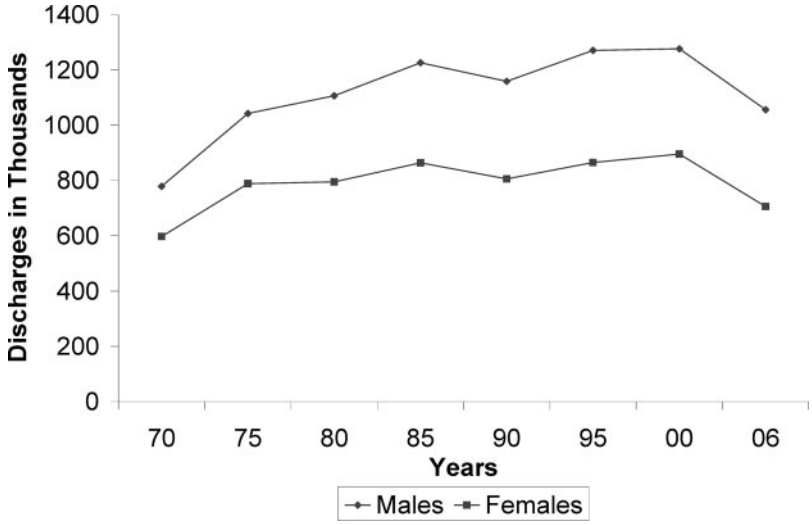

Chart 4-7. Hospital discharges for CHD by sex (United States: 1970–2006). Hospital discharges include people discharged alive, dead, and status unknown. Source: NHDS/NCHS and NHLBI.

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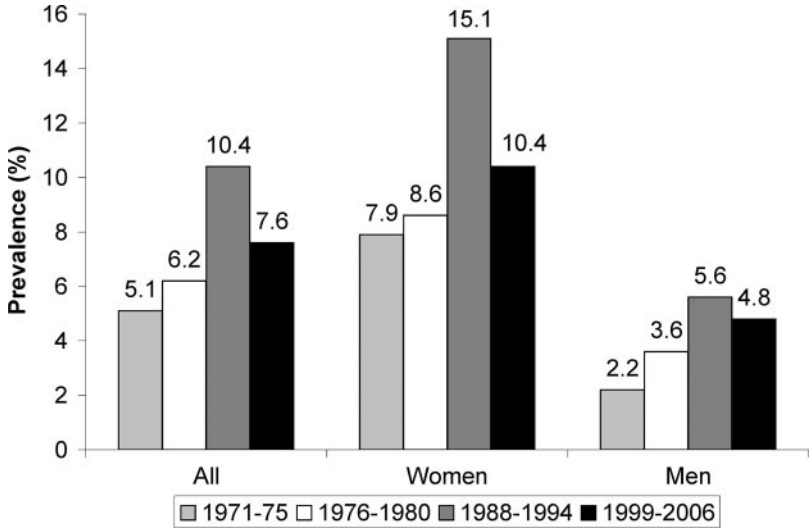

Chart 4-8. Prevalence of low CHD risk, overall and by sex, ages 25 to 74 years (NHANES: 1971–2006). Source: Personal communication with NHLBI, June 28, 2007. Low risk is defined as SBP <120 mm Hg and DBP <80 mm Hg; cholesterol <200 mg/dL; BMI <25 kg/m^2; currently not smoking cigarettes; and no prior MI or DM.

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5. Stroke (Cerebrovascular Disease)

ICD-9 430 to 438, ICD-10 I60-I69. See Tables 5-1 and 5-2 and Charts 5-1 through 5-6.

Abbreviations Used in Chapter 5

AF	atrial fibrillation
ADL	activities of daily living
AHA	American Heart Association
ARIC	Atherosclerosis Risk in Communities study
BASIC	Brain Attack Surveillance in Corpus Christi
BI	Barthel Index
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
CMS	Center for Medicare and Medicaid Services
CVD	cardiovascular disease
ED	emergency department
FHS	Framingham Heart Study
GCNKSS	Greater Cincinnati/Northern Kentucky Stroke Study
HDL	high-density lipoprotein
HERS	Heart and Estrogen/progestin Replacement Study
HHP	Honolulu Heart Program
ICD	*International Classification of Diseases*
MI	myocardial infarction
mm Hg	millimeters of mercury
MRI	magnetic resonance imaging
mRS	modified Rankin Scale
NAMCS	National Ambulatory Medical Care Survey
NASCET	North American Symptomatic Carotid Endarterectomy
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
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
rtPA	recombinant tissue plasminogen activator
SCI	silent cerebral infarct

(*Continued*)

Abbreviations Used in Chapter 5, Continued

SIPP	Survey of Income and Program Participation
STOP	Stroke Prevention Trial in Sickle Cell Anemia
TIA	transient ischemic attack
WASID	Warfarin–Aspirin Symptomatic Intracranial Disease Trial
WEST	Women's Estrogen for Stroke Trial
WHI	Women's Health Initiative

Prevalence

- An estimated 6 400 000 Americans ≥20 years of age have had a stroke (extrapolated to 2006 using NCHS/NHANES 2003 to 2006 data). Overall stroke prevalence during this period is an estimated 2.9% (see Table 5-1).
- According to data from the 2005 BRFSS (CDC), 2.7% of men and 2.5% of women ≥18 years of age had a history of stroke. Among these, 2.3% were non-Hispanic white, 4.0% were non-Hispanic black, 1.6% were Asian/Pacific Islander, 2.6% were Hispanic (of any race), 6.0% were American Indian/Alaska Native, and 4.6% were admixed.[1]
- Data from the 2008 survey of the CDC/BRFSS found that, overall, 2.6% of respondents had been told that they had a stroke. The highest prevalence was in Alabama and West Virginia (4.2%) and the lowest was in Colorado (1.8%).[2]
- Among American Indians/Alaska Natives ≥18 years of age, the estimated prevalence of stroke based on the 2008 NHIS was 3.9% (estimate considered unreliable). Among blacks, the prevalence was 3.6%; among whites, it was 2.7%; and among Asians, it was 1.8% (NHIS, NCHS).[3]
- Among American Indians/Alaska Natives ≥18 years of age, the estimated prevalence of stroke is considered unreliable in available data sources.
- The prevalence of silent cerebral infarction between 55 and 64 years of age is ≈11%. This prevalence increases to 22% between 65 and 69 years of age, 28% between 70 and 74 years of age, 32% between 75 and 79 years of age, 40% between 80 and 85 years of age, and 43% at ≥85 years of age. Application of these rates to 1998 US population estimates results in an estimated 13 million people with prevalent silent stroke.[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. 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 (REGARDS, NINDS).[6]

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

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NHLBI; GCNKSS and NINDS data for 1999 provided July 9, 2008; estimates compiled by NHLBI). Of all strokes, 87% are ischemic, 10% are intracerebral hemorrhage, and 3% are subarachnoid hemorrhage strokes (GCNKSS, NINDS, 1999).[7]

- On average, every 40 seconds, someone in the United States has a stroke. (AHA computation based on latest available data.).
- A study of nearly 18 000 middle-aged, predominantly white male participants in the Physicians' Health Study found that the Southeast and Midwest had higher crude and age-standardized major CVD, total stroke, ischemic stroke, coronary revascularization, and CVD death incidence rates compared with the Northeast.[8]
- Each year, ≈55 000 more women than men have a stroke (GCNKSS, NINDS).
- The stroke incidence rate is higher for men compared with women at younger ages, but not at older ages. The male-to-female incidence ratio was 1.25 in those 55 to 64 years of age, 1.50 in those 65 to 74 years of age, 1.07 in those 75 to 84 years of age, and 0.76 in those ≥85 years of age (ARIC and CHS studies, NHLBI).[7]
- Data from the GCNKSS and NINDS show that the annual incidence of first-ever hospitalized stroke did not change significantly between 1993 to 1994 and 1999: 158 per 100 000 blacks continue to have a higher stroke incidence than whites, especially among young adults.[9]
- Blacks have a risk of first-ever stroke that is almost twice that of whites. The age-adjusted stroke incidence rates in people 45 to 84 years of age are 6.6 per 1000 population in black men, 3.6 in white men, 4.9 in black women, and 2.3 in white women (ARIC, NHLBI).[7] On the basis of 1987 to 2001 data from the ARIC study sponsored by the NHLBI, stroke/TIA incidence rates (per 1000 person-years) are 2.4 for white men 45 to 54 years of age, 6.1 for white men 55 to 64 years of age, and 12.2 for white men 65 to 74 years of age. For white women in the same age groups, the rates are 2.4, 4.8, and 9.8, respectively. For black men in the same age groups, the rates are 9.7, 13.1, and 16.2, and for black women, the rates are 7.2, 10.0, and 15.0, respectively.[7]
- National statistics from death certificate data have long shown an increase in deaths attributed to stroke for blacks because of a higher stroke incidence compared with whites, although the case-fatality rate is similar between the 2 racial groups. Blacks <55 years of age seem to be at particularly high risk (2- to 5-fold higher than age-matched white subjects), but in elderly ages, this racial disparity is attenuated. This racial disparity in stroke incidence does not seem to be changing over time. Community socioeconomic status appeared to explain 39% of the excess stroke incidence risk in blacks in this study.[10]
- The Brain Attack Surveillance in Corpus Christi (BASIC, NINDS) demonstrated an increased incidence of stroke among Mexican Americans compared with non-Hispanic whites in this community. The crude cumulative incidence was 168 per 10 000 in Mexican Americans and 136 per 10 000 in non-Hispanic whites. Specifically, Mexican Americans have a higher cumulative incidence for ischemic stroke at younger ages (45 to 59 years of age: relative risk [RR] 2.04; 95% CI, 1.55 to 2.69; 60 to 74 years of age: RR 1.58; 95% CI, 1.31 to 1.91), but not at older ages (≥75 years of age: RR 1.12; 95% CI, 0.94 to 1.32). Mexican Americans also have a higher incidence of intracerebral hemorrhage and subarachnoid hemorrhage than non-Hispanic whites, adjusted for age, as well as a higher incidence of ischemic stroke and TIA at younger ages than non-Hispanic whites.[11]
- 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.[12]
- The age-adjusted incidence of first ischemic stroke per 100 000 was 88 in whites, 191 in blacks, and 149 in Hispanics, according to data from the Northern Manhattan Study (NOMAS, NINDS). Among blacks, compared with whites, the relative rate of intracranial atherosclerotic stroke was 5.85; extracranial atherosclerotic stroke, 3.18; lacunar stroke, 3.09; and cardioembolic stroke, 1.58. Among Hispanics (primarily Cuban and Puerto Rican), compared with whites, the relative rate of intracranial atherosclerotic stroke was 5.00; extracranial atherosclerotic stroke, 1.71; lacunar stroke, 2.32; and cardioembolic stroke, 1.42.[13]
- Analysis of data from the FHS study of the NHLBI, 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 at 65 years of age decreased significantly, from 19.5% to 14.5% in men and from 18.0% to 16.1% in women. Age-adjusted stroke severity did not vary across periods; however, 30-day mortality rate decreased significantly in men (from 23% to 14%), but not in women (from 21% to 20%).[14]
- Analysis of black and white patients in the WASID trial found that blacks were significantly more likely to have an ischemic stroke, brain hemorrhage or vascular death, or ischemic stroke alone than whites.[15]
- 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 elderly people, many of whom lack adequate blood pressure control, and the increasing use of anticoagulants, thrombolytics, and antiplatelet agents. Mexican Americans, Latin Americans, African Americans, Native Americans, Japanese people, and Chinese people have higher incidences than do white Americans.[16]

Transient Ischemic Attack

- The prevalence of transient ischemic attack (TIA)—a temporary episode of neurologic dysfunction caused by reduced blood flow to the brain, spinal cord, or retina, without permanent death of brain tissue—increases significantly with older age.[17]
- The incidence of TIA in the United States has been estimated to be ≈200 000 to 500 000 per year, with a

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population prevalence of 2.3%, which translates into ≈5 million people.[18]

- Approximately half of all patients who experience a TIA fail to report it to their healthcare providers.[19]
- Approximately 15% of all strokes are heralded by a TIA.[17]
- One third of episodes characterized as TIAs 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.[20]
- In population-based studies, the age- and gender-adjusted incidence rates for TIA range from 68.2 to 83.0 per 100 000. Men and blacks have higher rates of TIA.[21,22]
- After TIA, the 90-day risk of stroke is 3.0% to 17.3% and is highest within the first 30 days, with half occurring within the first 48 hours after a TIA.[22–25]
- Within 1 year of TIA, up to one fourth of patients will die.[22,26]
- Individuals who have a TIA have a 10-year stroke risk of 18.8% and a combined 10-year stroke, MI, or vascular death risk of 42.8% (4%/year).[27]
- In the North American Symptomatic Carotid Endarterectomy Trial (NASCET) study, patients with a first-ever hemispheric TIA had a 90-day stroke risk of 20.1%. The risk of stroke after TIA exceeded the risk after hemispheric stroke.[28]

Mortality

- On average, every 4 minutes, someone dies of a stroke (NCHS, NHLBI).
- Stroke accounted for ≈1 of every 18 deaths in the United States in 2006. Approximately 53% of stroke deaths in 2006 occurred out of the hospital.[29] Stroke mortality in 2006 was 137 119; any-mention mortality in 2006 was ≈232 000 (NHLBI; NCHS public use data files).[30]
- Preliminary stroke mortality in 2007 was 133 990, and the preliminary death rate was 41.6.[31]
- When considered separately from other CVDs, stroke ranks No. 3 among all causes of death, behind diseases of the heart and cancer (NCHS mortality data).
- Among persons 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 the ARIC study of the NHLBI.[32]
- In a study of persons ≥65 years of age recruited from a random sample of Health Care Financing Administration Medicare Part B eligibility lists in 4 US communities, the 1-month case fatality rate was 12.6% for all strokes, 8.1% for ischemic strokes, and 44.6% for hemorrhagic strokes.[33]
- From 1996 to 2006, the annual stroke death rate decreased 33.5%, and the actual number of stroke deaths declined 18.4%.(Appropriate comparability ratios were applied).[30]
- Conclusions about changes in stroke death rates from 1980 to 2005:
  - — 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 at ≥65 years of age in men than in women compared with younger ages.[30]
- More women than men die of stroke each year due to the larger number of elderly women. Women accounted for 60.6% of US stroke deaths in 2006. (AHA computation).
- The 2006 overall death rate for stroke was 43.6 per 100 000. Death rates were 41.7 for white males, 67.1 for black males, 41.1 for white females, and 57.0 for black females.[30]
- In 2006, death rates for stroke were 35.9 for Hispanic or Latino males and 32.3 for females, 39.8 for Asian or Pacific Islander males and 34.9 for females, and 25.8 for American Indian/Alaska Native males and 30.9 for females.[34]
- From 1995 to 1998, age-standardized mortality rates for ischemic stroke, subarachnoid hemorrhage, and intracerebral hemorrhage were higher among blacks than whites. Death rates from intracerebral hemorrhage also were higher among Asians/Pacific Islanders than among whites. All minority populations had higher death rates from subarachnoid hemorrhage than did whites. Among adults 25 to 44 years of age, blacks and American Indians/Alaska Natives had higher risk ratios than did whites for all 3 stroke subtypes.[35]
- 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 Indians/Alaska Natives, and Asians/Pacific Islanders had younger mean ages than whites, and the mean age at stroke death was also younger among Hispanics than non-Hispanics.[36]
- 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.[37]
- A report released by the CDC in collaboration with the Centers for Medicare and Medicaid Services (CMS), the *Atlas of Stroke Hospitalizations Among Medicare Beneficiaries*, found that in Medicare beneficiaries, 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.[38]

Stroke Risk Factors

(See Table 5-2 for data on modifiable stroke risk factors.)

- 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, diabetes mellitus, focal

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symptoms of weakness or speech impairment, and TIA that lasted >10 minutes.[39]

- BP is a powerful determinant of risk for both ischemic stroke and intercranial hemorrhage. Subjects with BP <120/80 mm Hg have approximately half the lifetime risk of stroke of subjects with hypertension.
- 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.[40,41]
- The risk of ischemic stroke associated with current cigarette smoking has been shown to be approximately double that of nonsmokers after adjustment for other risk factors (FHS, CHS, HHP, NHLBI).
- Age-specific incidence rates and rate ratios show that diabetes 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.[42]
- In a recent ARIC/NHLBI study of a biracial population 45 to 64 years of age, with an average follow-up of 13.4 years, researchers found that blacks had a 3-fold higher multivariate-adjusted risk ratio of lacunar stroke than whites. In this middle-aged population, the top 3 risk factors based on the population-attributable fraction for lacunar stroke were hypertension (population-attributable fraction, 33.9%), diabetes mellitus (26.3%), and current smoking (22.0%).[43]
- In the Framingham Offspring Study, 2040 individuals free of clinical stroke had an MRI scan to detect silent cerebral infarct (SCI). Prevalent SCI was associated with the Framingham Stroke Risk Profile score (OR, 1.27; 95% CI, 1.10 to 1.46), hypertension (OR, 1.56; 95% CI, 1.15 to 2.11), elevated plasma homocysteine (OR, 2.23; 95% CI, 1.42 to 3.51), AF (OR, 2.16; 95% CI, 1.07 to 4.40), carotid stenosis >25% (OR, 1.62; 95% CI, 1.13 to 2.34), and increased carotid intimal-medial thickness (OR, 1.65; 95% CI, 1.22 to 2.24).[44]
- In the FHS of the NHLBI, in participants <65 years of age, the risk of developing stroke/TIA was 4.2-fold higher in those with symptoms of depression. After adjustment for components of the Framingham Stroke Risk Profile and education, similar results were obtained. In subjects ≥65 years of age, use of antidepressant medications did not alter the risk associated with depressive symptoms. Identification of depressive symptoms at younger ages may have an impact on the primary prevention of stroke.[45]
- Data from the HHP/NHLBI found that in Japanese men 71 to 93 years of age, low concentrations of high-density lipoprotein (HDL) cholesterol were more likely to be associated with a future risk of thromboembolic stroke than were high concentrations.[46]

Female Sex as a Risk Factor for Stroke

- Analysis of NHANES 1999 to 2004 data found that women 45 to 54 years of age are more than twice as likely as men to have suffered a stroke. Women in the 45- to 54-year age group had a >4-fold higher likelihood of having had a stroke than women 35 to 44 years of age.[47]
- Stroke is a major health issue for women, particularly for postmenopausal women, which raises the question of whether increased incidence is due to aging or to hormone status and whether hormone therapy affects risk.[48]
- 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. The excess risk was apparent in all age groups, in all categories of baseline stroke risk, and in women with and without hypertension or prior history of CVD.[49]
- 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. The excess risk of total stroke conferred by estrogen alone was 12 additional strokes per 10 000 person-years.[50]
- In postmenopausal women with known CHD, the Heart and Estrogen/Progestin Replacement Study (HERS), a secondary CHD prevention trial, found that a combination of estrogen plus progestin (conjugated equine estrogen [0.625 mg] and medroxyprogesterone acetate [2.5 mg]) hormone therapy did not reduce stroke risk.[51]
- The Women's Estrogen for Stroke Trial (WEST) found that estrogen alone (1 mg of 17β-estradiol) in women with a mean age of 71 years also had no significant overall effect on recurrent stroke or fatality, but there was an increased rate of fatal stroke and an early increase in overall stroke rate in the first 6 months of therapy.[52]
- 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 women with established heart disease.[49,53]
- An observational study of >37 000 women ≥45 years of age participating in the Women's Health Study suggests that a healthy lifestyle that consists of abstinence from smoking, low BMI, moderate alcohol consumption, regular exercise, and a healthy diet was associated with a significantly reduced risk of total and ischemic stroke, but not of hemorrhagic stroke.[54]
- 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 compared with those with menopause <42 years of age, even after adjustment for potential confounders. Women with menopause before 42 years of age had twice the stroke risk compared with all other women in different age groups.[55]

Pregnancy as a Risk Factor for Stroke

- The risk of ischemic stroke or intracerebral hemorrhage during pregnancy and the first 6 weeks postpartum 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 6 weeks postpar-

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tum. Intracerebral hemorrhage showed a small RR of 2.5 during pregnancy but increased dramatically to an RR of 28.3 in the 6 weeks postpartum. The excess risk of stroke (all types except subarachnoid hemorrhage) attributable to the combined pregnancy/postpregnancy period was 8.1 per 100 000 pregnancies.[56]

- 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.[57]

Physical Inactivity as a Risk Factor for Stroke

- Higher levels of PA are associated with lower stroke risk. Results from the Physicians' Health Study showed a 14% lower RR of stroke associated with vigorous exercise (exercise ≥5 times per week) among men.[58] The Harvard Alumni Study showed that men who were highly physically active had an 18% lower RR of total stroke.[59] More recently, a clear inverse relationship between stroke incidence and increasing levels of combined work and leisure activity was shown in the EPIC-Norfolk study of 22 602 men and women, with a nearly 40% RR reduction in the most active category. In sex-stratified analysis, the trend was not significant in women.[60]
- For women in the Nurses' Health Study, RRs for total stroke from the lowest to the highest PA levels were 1.00, 0.98, 0.82, 0.74, and 0.66, respectively.[61]
- NOMAS (NINDS), which included white, black, and Hispanic men and women in an urban setting, showed a decrease in ischemic stroke risk associated with PA levels across all racial/ethnic and age groups and for each gender (OR 0.37).[62]
- PA—whether in sports, during leisure time, or at work—was related to lower risk of ischemic stroke during follow-up of the ARIC/NHLBI cohort.[63]
- A meta-analysis including 31 observational studies conducted mainly in the United States and Europe found that moderate and high levels of leisure-time and occupational PA was associated with lower risks of total stroke, hemorrhagic stroke, and ischemic stroke.[64]

Awareness of Stroke Warning Signs and Risk Factors

- 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 versus blacks and Hispanics (41.3%, 29.5%, and 26.8% respectively), women versus men (41.5% versus 34.5%), and persons with higher versus lower educational attainment (47.6% for persons with a college degree or more versus 22.5% for those who had not received a high school diploma). Among states, the same measure ranged from 27.9% (Oklahoma) to 49.7% (Minnesota).[65]
- 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.[66]
- A study of >2100 respondents to a random-digit telephone survey in Cincinnati, Ohio, in 2000 showed that 70% of respondents correctly named at least 1 established stroke warning sign (versus 57% in 1995), and 72% correctly named at least 1 established risk factor (versus 68% in 1995).[67,68] In the 1995 survey,[68] respondents ≥75 years of age were less likely to correctly list 1 warning sign and to list 1 risk factor.
- Among patients recruited from the Academic Medical Center Consortium, the CHS, and United HealthCare, only 41% were aware of their increased risk for stroke. Approximately 74% recalled being told of their increased stroke risk by a physician, compared with 28% who did not recall this. Younger patients, depressed patients, those in poor current health, and those with a history of TIA were most likely to be aware of their risk.[69]
- An AHA-sponsored random-digit dialing telephone survey was conducted in mid-2003. Only 26% of women >65 years of age reported being well informed about stroke. Correct identification of the warning signs of stroke was low among all age and racial/ethnic groups.[70]
- Among participants in a study by the National Stroke Association, 2.3% reported having been told by a physician that they had had a TIA. Of those with a TIA, only 64% saw a physician within 24 hours of the event, only 8.2% correctly related the definition of TIA, and 8.6% could identify a typical symptom. Men, persons of color, and those with lower income and fewer years of education were less likely to be knowledgeable about TIA.[21]
- Insufficient awareness persists in the general medical community with regard to risk factors, warning signs, and prevention strategies for stroke. A survey of 308 internal medicine residency programs showed that only 46% required the study of neurology, whereas 97% required the study of cardiology. Under-representation of neurology training in internal medicine residency programs may lead to an under-recognition of stroke signs and symptoms by physicians and affect stroke outcome.[71]
- 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 diabetes, high BP, high cholesterol, heart disease, and stroke/TIA were independently associated with perceived risk for stroke. Respondents with AF were no more likely

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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.[72]

- 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.[73]
- Spanish-speaking Hispanics are far less likely to know all heart attack symptoms and 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 heart attack and stroke knowledge among Hispanics.[74]
- In the Reasons for Geographic and Racial Differences in Stroke Study (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. There was 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.[75]

Aftermath

Stroke is a leading cause of serious, long-term disability in the United States (Survey of Income and Program Participation [SIPP], a survey of the US Bureau of the Census).[76]

- 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. Increasing the number of stroke survivors who receive needed outpatient rehabilitation might lead to better functional status and quality of life in this population.[77]
- On the basis of pooled data from the FHS, ARIC, and CHS studies of the NHLBI:
  - The proportions of patients dead 1 year after a first stroke were as follows:
    - At ≥40 years of age: 21% of men and 24% of women.
    - At 40 to 69 years of age: 14% of white men, 20% of white women, 19% of black men, and 19% of black women.
    - At ≥70 years of age: 24% of white men, 27% of white women, 25% of black men, and 22% of black women.
  - The proportions of patients dead within 5 years after a first stroke were as follows:
    - At ≥40 years of age: 47% of men and 51% of women.
    - At 40 to 69 years of age: 32% of white men, 32% of white women, 34% of black men, and 42% of black women.
    - At ≥70 years of age: 58% of white men, 58% of white women, 49% of black men, and 54% of black women.
  - Of those who have a first stroke, the proportions with a recurrent stroke in 5 years were as follows:
    - At 40 to 69 years of age: 13% of men and 22% of women.
    - At ≥70 years of age: 23% of men and 28% of women.
    - At 40 to 69 years of age: 15% of white men, 17% of white women, 10% of black men, and 27% of black women.
    - At ≥70 years of age: 23% of white men, 27% of white women, 16% of black men, and 32% of black women.
  - The median survival times after a first stroke were:
    - At 60 to 69 years of age: 6.8 years for men and 7.4 years for women.
    - At 70 to 79 years of age: 5.4 years for men and 6.4 years for women.
    - At ≥80 years of age: 1.8 years for men and 3.1 years for women.
- The length of time to recover from a stroke depends on its severity. Between 50% and 70% of stroke survivors regain functional independence, but 15% to 30% are permanently disabled, and 20% require institutional care at 3 months after onset.[78]
- In the NHLBI's FHS, among ischemic stroke survivors who were ≥65 years of age, these disabilities were observed at 6 months after stroke[79]:
  - 50% had some hemiparesis.
  - 30% were unable to walk without some assistance.
  - 26% were dependent in ADL.
  - 19% had aphasia.
  - 35% had depressive symptoms.
  - 26% were institutionalized in a nursing home.
- Black stroke survivors had greater activity limitations than did white stroke survivors, according to data from the NHIS (2000 to 2001, NCHS) as analyzed by the CDC.[80]
- After stroke, women have greater disability than men. A Michigan-based stroke registry found that 33% of women had moderate to severe disability (mRS ≥4) at discharge, compared with 27% of men. In a study of 108 stroke survivors from FHS, 34% of women were disabled at 6

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months (BI <60), compared with 16% of men. In the Kansas City Stroke Study, women had a 30% lower probability of achieving independence (BI ≥95) by 6 months compared with men. In the Michigan registry, women had a 63% lower probability of achieving ADL independence (BI ≥95) 3 months after discharge.[81–84]

Hospital Discharges/Ambulatory Care Visits

- From 1996 to 2006, the number of inpatient discharges from short-stay hospitals with stroke as the first listed diagnosis declined from 956 000 to 889 000 (NHDS, NCHS). The decrease was observed in men and women ≥65 years of age.[85]
- In 2005, there was a hospitalization rate of 77.3 stays per 10 000 persons >45 years of age for cerebrovascular disease. There has been a decline in the hospitalization rate for different types of cerebrovascular disease between 1997 and 2005, with the exception of hemorrhagic stroke. Between 1997 and 2005, the hospitalization rate for ischemic stroke decreased by 34%, from 54.4 to 35.9 stays per 10 000 persons. The hospitalization rate for transient cerebral ischemia also decreased ≈23% during this period. Similarly, the hospitalization rate for occlusion or stenosis of precerebral arteries steadily decreased by 30% between 1997 and 2005, from 18.4 to 12.8 stays per 10 000 persons. In contrast, the hospitalization rate for hemorrhagic stroke remained relatively stable during this period.[86]
- Data from 2006 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 4.9 days.[87]
- In 2007, the number of ambulatory care visits with stroke as the first-listed diagnosis was 3 764 000 (NAMCS, NHAMCS/NCHS).[88]
- In 2003, men and women accounted for roughly the same number of hospital stays for stroke in the 18- to 44-year age group. After 65 years of age, women were the majority. Among persons 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.[89]
- 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 and 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 age group (65 to 74 years of age).[38]

Stroke in Children

Stroke in children peaks in the perinatal period. In the NHDS/NCHS, from 1980 to 1998, the rate of stroke for infants <30 days old (per 100 000 live births per year) was 26.4, with rates of 6.7 for hemorrhagic stroke and 17.8 for ischemic stroke.[90]

- A history of infertility, preeclampsia, prolonged rupture of membranes, and chorioamnionitis were found to be independent risk factors for radiologically confirmed perinatal arterial ischemic stroke in Kaiser Permanente of Northern California, a large integrated healthcare delivery system. The RR of perinatal stroke increased ≈25-fold, with an absolute risk of 1 per 200 deliveries, when ≥3 of the following antenatally determined risk factors were present: infertility, preeclampsia, chorioamnionitis, prolonged rupture of membranes, primiparity, oligohydramnios, decreased fetal movement, prolonged second stage of labor, and fetal heart rate abnormalities.[91]
- The overall incidence rate of all strokes in children <15 years of age was 6.4 per 100 000 in 1999, a nonsignificant increase compared with 1988. The 30-day case fatality rates were 18% in 1988 to 1989, 9% in 1993 to 1994, and 9% in 1999. The incidence of stroke in children has been stable over the past 10 years. The previously reported nationwide decrease in overall stroke mortality in children might be due to decreasing case fatality after stroke and not decreasing stroke incidence. It was conservatively estimated that ≈3000 children and adults <20 years of age had a stroke in the United States in 2004.[92]
- Stroke in childhood and young adulthood has a disproportionate impact on the affected patients, their families, and society compared with stroke at older ages. Outcome of childhood stroke was a moderate or severe deficit in 42% of cases.[93]
- Boys have a 1.28-fold higher risk of stroke and a higher case-fatality rate for ischemic stroke than girls. Compared with the stroke risk of white children, black children have a higher RR of 2.12, Hispanics have a lower RR of 0.76, and Asians have a similar risk. 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.[94]
- Despite current treatment, 1 of 10 children with ischemic stroke will have a recurrence within 5 years.[95]
- Cerebrovascular disorders are among the top 10 causes of death in children, with rates highest in the first year of life. Stroke mortality in children <1 year of age has remained the same over the past 40 years.[96]
- From 1979 to 1998 in the United States, childhood mortality resulting from stroke declined by 58% overall, with reductions in all major subtypes.[97]
  - Ischemic stroke decreased by 19%, subarachnoid hemorrhage by 79%, and intracerebral hemorrhage by 54%.
  - Black ethnicity was a risk factor for death resulting from all stroke types.
  - Male sex was a risk factor for death caused by subarachnoid hemorrhage and intracerebral hemorrhage, but not for death resulting from ischemic stroke.
- Sickle cell disease is the most important cause of ischemic stroke among black children. The Stroke Prevention Trial in Sickle Cell Anemia (STOP) demonstrated the efficacy of blood transfusions for primary stroke prevention in high-risk children with sickle cell disease in 1998. First-admission rates for stroke in California among persons

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<20 years of age with sickle cell disease showed a dramatic decline subsequent to the publication of the STOP study. For the study years 1991 to 1998, 93 children with sickle cell disease were admitted to California hospitals with a first stroke; 92.5% of these strokes were ischemic, and 7.5% were hemorrhagic. The first-stroke rate was 0.88 per 100 person-years during 1991 to 1998 compared with 0.50 in 1999 and 0.17 in 2000 ($P<0.005$ for trend).[98]

Access to Stroke Care

- In 2008, there were 322 diplomates receiving initial certification in Vascular Neurology by the American Board of Psychiatry and Neurology.[99]
- A 2004 survey conducted by the American Academy of Neurology revealed that 40% of the 6298 US neurologists responding considered cerebrovascular disease a focus practice area.[100]
- 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.[101]
- The median time from stroke onset to arrival in an ED is between 3 and 6 hours, according to a study of at least 48 unique reports of prehospital delay time for patients with stroke, TIA, or stroke-like symptoms. The study included data from 17 countries, including the United States. Improved clinical outcome at 3 months was seen for patients with acute ischemic stroke when intravenous thrombolytic treatment was started within 3 hours of symptom onset.[102]
- 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 optimally treated, 57% could have received thrombolytic treatment.[103]
- Data from the Paul Coverdell National Acute Stroke Registry were analyzed from the 142 hospitals that participated in the 4 registry states. Among the >17 600 patients in the study, 66.1% were ≥65 years of age. Women were older than men, and whites were older than blacks. Ischemic stroke (65%) was the most common subtype, followed by TIA (24%) and hemorrhagic stroke (9.7%). 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%). The median arrival time for all patients with known time of onset was 2.0 hours. Sixty-five percent of patients who arrived at the ED within 2 hours of onset received imaging within 1 hour of ED arrival. Significantly fewer women (62%) received imaging within 1 hour of ED arrival than men.[104]
- Patients with a discharge diagnosis of ischemic stroke were identified in 7 California hospitals participating in the California Acute Stroke Pilot Registry. Six points of care were tracked: thrombolysis, receipt of antithrombotic medications within 48 hours, prophylaxis for deep vein thrombosis, smoking cessation counseling, and prescription of lipid-lowering and antithrombotic medications at discharge. Overall, rates of optimal treatment improved for patients treated in year 2 versus year 1, with 63% receiving a perfect score in year 2 versus 44% in year 1. Rates improved significantly in 4 of the 6 hospitals and for 4 of the 6 interventions. A seventh hospital that participated in the registry but did not implement standardized orders showed no improvement in optimal treatment.[105]
- A population-based study performed in a biracial population of 1.3 million in Ohio in 1993 and 1994 showed that 8% of all ischemic stroke patients presented to an ED within 3 hours and met other eligibility criteria for treatment with recombinant tissue plasminogen activator (rtPA). Even if time were not an exclusion criterion for use of rtPA, only 29% of all ischemic strokes in the population would have otherwise been eligible for rtPA.[106]

Operations and Procedures

In 2006, an estimated 99 000 inpatient endarterectomy procedures were performed in the United States. Carotid endarterectomy is the most frequently performed surgical procedure to prevent stroke. (NHDS, NCHS)

Cost

The estimated direct and indirect cost of stroke for 2010 is $73.7 billion.

- In 2006, $3.9 billion ($7449 per discharge) was paid to Medicare beneficiaries discharged from short-stay hospitals for stroke.[107]
- 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.)[108]
- In a study of stroke costs within 30 days of an acute event between 1987 to 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).[109]
- Inpatient hospital costs for an acute stroke event account for 70% of first-year poststroke costs.[108]
- The largest components of acute-care costs were room charges (50%), medical management (21%), and diagnostic costs (19%).[110]
- 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.[109]

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- Demographic variables (age, sex, and insurance status) are not associated with stroke cost. Severe strokes (NIHSS score >20) cost twice as much as mild strokes, despite similar diagnostic testing. Comorbidities such as ischemic heart disease and AF predict higher costs.[110,111] 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.[96]

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Table 5-1. Stroke

Population Group	Prevalence, 2006 Age ≥20 y	New and Recurrent Attacks All Ages	Mortality, 2006 All Ages*	Hospital Discharges, 2006 All Ages	Cost, 2010
Both sexes	6 400 000 (2.9%)	795 000	137 119	889 000	$73.7 billion
Males	2 500 000 (2.5%)	370 000 (46.5%)†	54 524 (39.8%)†	404 000	...
Females	3 900 000 (3.2%)	425 000 (53.5%)†	82 595 (60.2%)†	486 000	...
NH white males	2.3%	325 000‡	45 198	...	...
NH white females	3.1%	365 000‡	70 666	...	...
NH black males	3.8%	45 000‡	7424	...	...
NH black females	4.3%	60 000‡	9621	...	...
Mexican-American males	2.8%	...	...	...	...
Mexican-American females	3.1%	...	...	...	...
Hispanic or Latino age ≥18 y§	2.6%	...	...	...	...
Asian age ≥18 y§	1.8%	...	...	...	...
American Indian/Alaska Native age ≥18 y§	3.9%‖	...	...	...	...

Ellipses (…) indicate data not available.

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

†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.

§NHIS 2008 (NCHS): data are weighted percentages for Americans ≥18 years of age.[3]

‖Estimates are considered unreliable.

Sources: Prevalence: NHANES 2003 to 2006, NCHS and NHLBI. Percentages for racial/ethnic groups are age-adjusted for Americans ≥20 years of age. Age-specific percentages are extrapolated to the 2006 US population. Prevalence data for the Hispanic, Asian, and American Indian/Alaska Native populations, ≥18 years of age, are from NHIS/NCHS. Estimate for American Indians/Alaska Natives is considered unreliable.[3] Incidence: GCNKSS/NINDS data for 1999 provided on August 1, 2007. US estimates compiled by NHLBI. See also Kissela et al.[112] 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: NHDS, NCHS. Data include those inpatients discharged alive, dead, or status unknown. Cost: NHLBI. Data include estimated direct and indirect costs for 2010.

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Table 5-2. Modifiable Stroke Risk Factors

Factor	Prevalence, %	Population-Attributable Risk, %*	RR
CVD			
CHD[113]			
Men	8.4	5.8†	1.73 (1.68–1.78)[130]
Women	5.6	3.9†	1.55 (1.17–2.07)[131]
Heart failure[113]			
Men	2.6	1.4†	
Women	2.1	1.1†	
Peripheral arterial disease	4.9	3.0†	
Hypertension[114]			
Age 50 y	20	40	4.0
Age 60 y	30	35	3.0
Age 70 y	40	30	2.0
Age 80 y	55	20	1.4
Age 90 y	60	0	1.0
Cigarette smoking	25	12–18	1.8
Diabetes	7.3	5–27	1.8–6
Asymptomatic carotid stenosis	2–8[117–123]	2–7‡	2.0[131]
Atrial fibrillation (nonvalvular)[115,116]			
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
Sickle cell disease	0.25 (of blacks)[124]	. . .	200–400[132]§
Dyslipidemia			
High total cholesterol	25[125]	15	2.0 for men and for women <55 y of age
Low HDL cholesterol	25[125]	10	1.5–2.5 for men
Dietary factors			
Na intake >2300 mg	75–90	Unknown	Unknown
K intake <4700 mg	90–99[126]	Unknown	Unknown
Obesity	17.9[127]	12–20†	1.75–2.37[133,134]
Physical inactivity[62]	25	30	2.7‡
Postmenopausal hormone therapy	20[128] (women 50–74 y of age)[129]	7	1.4[135]

Data derived from Hart et al[136,137] and van Walraven et al.[138] Stroke includes both ischemic and hemorrhagic stroke. Cardiovascular disease includes coronary heart disease, heart failure, and peripheral arterial disease.

*Population-attributable risk is the proportion of ischemic stroke in the population that can be attributed to a particular risk factor (see text for formula).

†Calculated on the basis of point estimates of referenced data provided in the table. For peripheral arterial disease, calculation was based on average RR 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.

‖For high-risk patients treated with transfusion.

Adapted from Goldstein LB, Adams R, Alberts MJ, Appel LJ, Brass LM, Bushnell CD, Culebras A, DeGraba TJ, Gorelick PB, Guyton JR, Hart RG, Howard G, Kelly-Hayes M, Nixon JVI, Sacco RL. Primary prevention of ischemic stroke: a guideline from the American Heart Association/American Stroke Association Stroke Council: cosponsored by the Atherosclerotic Peripheral Vascular Disease Interdisciplinary Working Group; Cardiovascular Nursing Council; Clinical Cardiology Council; Nutrition, Physical Activity, and Metabolism Council; and the Quality of Care and Outcomes Research Interdisciplinary Working Group. *Stroke*. 2006;37:1583–1633.

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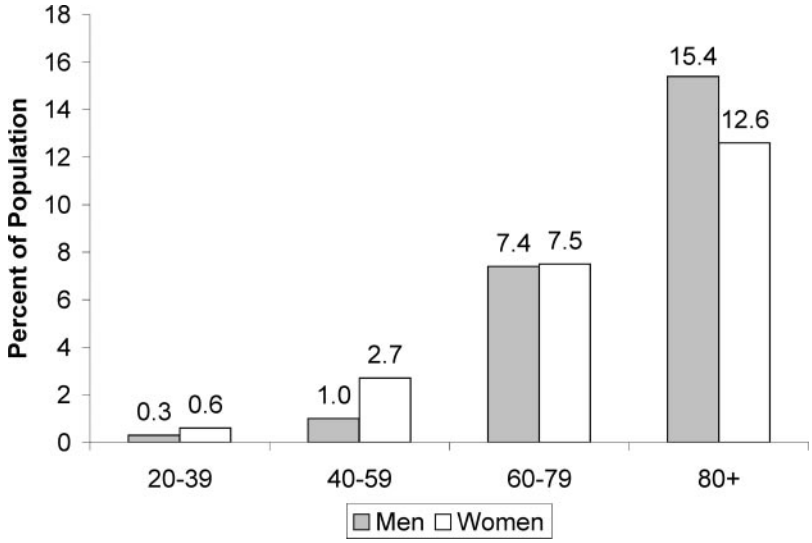

Chart 5-1. Prevalence of stroke by age and sex (NHANES: 2003–2006). Source: NCHS and NHLBI.

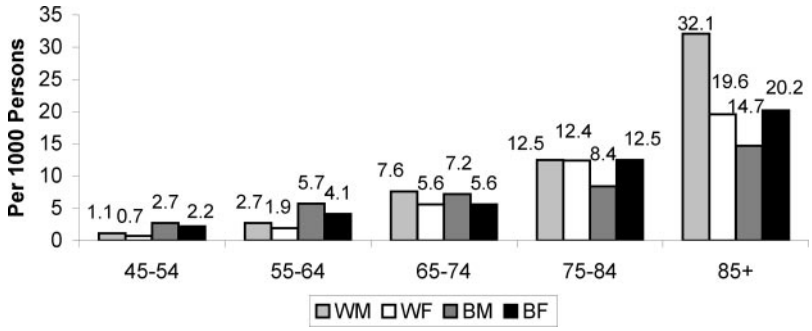

Chart 5-2. Annual rate of first cerebral infarction by age, sex, and race (GCNKSS: 1999). Note: Rates for black men and women 45 to 54 years of age and for black men ≥75 years of age are considered unreliable. An estimated 15 000 people have first cerebral infarctions before 45 years of age. Source: Unpublished data from the GCNKSS.

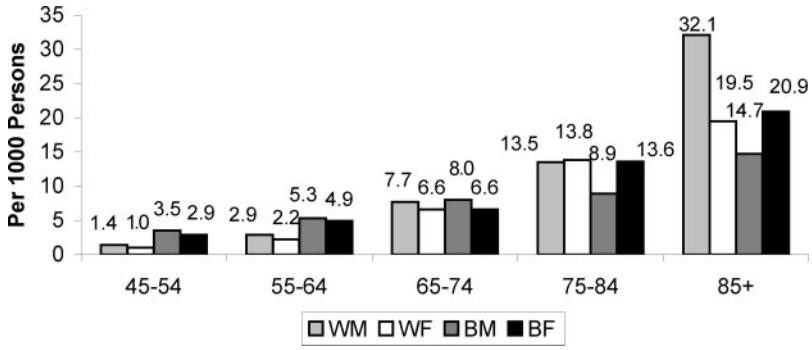

Chart 5-3. Annual rate of all first-ever strokes by age, sex, and race (GCNKSS: 1999). Note: Rates for black men and women 45 to 54 years of age and for black men ≥75 years of age are considered unreliable. Source: Unpublished data from the GCNKSS.

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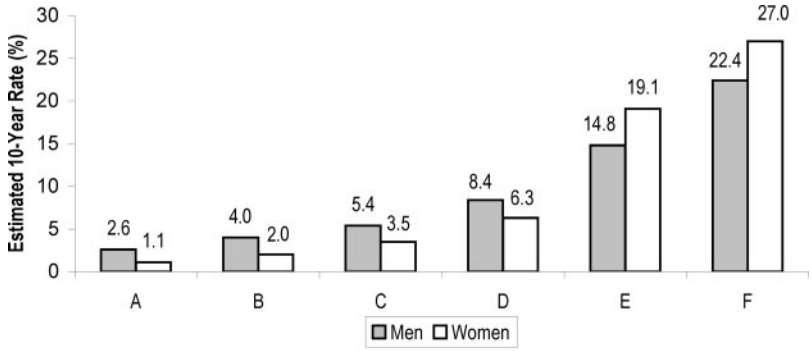

	A	B	C	D	E	F
Blood pressure*	95--105	138--148	138--148	138--148	138--148	138--148
Diabetes	No	No	Yes	Yes	Yes	Yes
Cigarette smoking	No	No	No	Yes	Yes	Yes
Prior AF	No	No	No	No	Yes	Yes
Prior CVD	No	No	No	No	No	Yes

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

Chart 5-4. Estimated 10-year stroke risk in adults 55 years of age according to levels of various risk factors (Framingham Heart Study). Source: Wolf et al.[139]

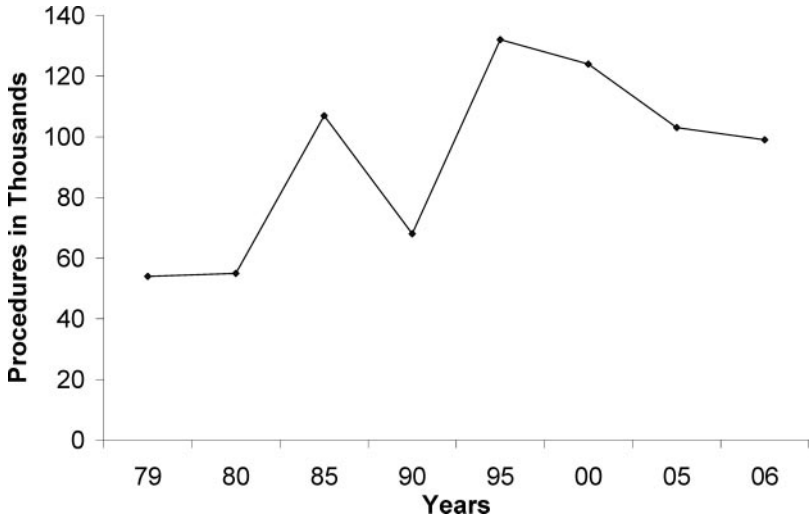

Chart 5-5. Trends in carotid endarterectomy procedures (United States: 1979–2006). Source: NHDS/NCHS and NHLBI.

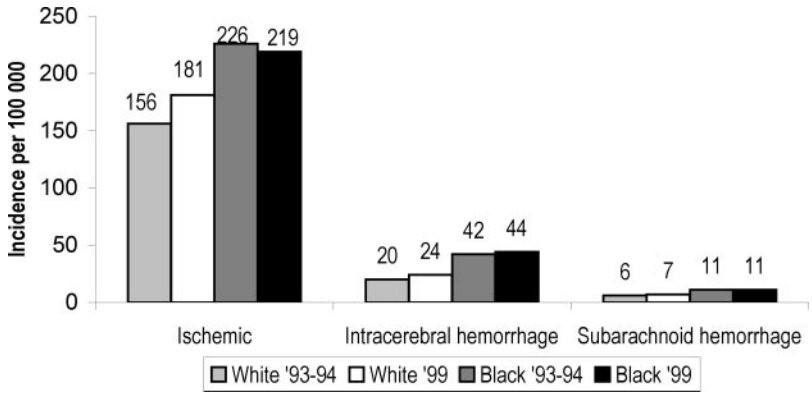

Chart 5-6. Annual age-adjusted incidence of first-ever stroke by race. Inpatient plus out-of-hospital ascertainment, 1993–1994 and 1999. Source: Kleindorfer et al.[22]

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6. High Blood Pressure

ICD-9 401–404, ICD-10 I10–I15. See Tables 6-1 and 6-2 and Charts 6-1 through 6-5.

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]
- An estimated 74 500 000 adults ≥20 years of age have HBP, extrapolated to 2006 with NHANES 2003 to 2006 data (Table 6-1).
- 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 much higher percentage of women have hypertension than men.[2]
- 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.[3]
- Data from NHANES 2005 to 2006 found that 29% of US adults ≥18 years of age were hypertensive. The prevalence of hypertension was nearly equal between men and women. An additional 28% of US adults had prehypertension, and 7% of adults with hypertension 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.[4]
- Data from the 2007 BRFSS/CDC study indicate that the percentage of adults ≥18 years of age who had been told that they had HBP ranged from 19.7% in Utah to 33.8% in Tennessee. The median percentage was 27.8%.[5]

Abbreviations Used in Chapter 6

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-CM	International Classification of Diseases, ninth revision, clinical modification
JNC	Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure
LDL	low-density lipoprotein
MESA	Multi-Ethnic Study of Atherosclerosis
mm Hg	millimeter of mercury
NCHS	National Center for Health Statistics
NHANES	National Health and Nutrition Examination Survey
NHES	National Health 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
PA	physical activity
REGARDS	Reasons for Geographic and Racial Differences in Stroke study
SBP	systolic blood pressure

Older Adults

- Age-adjusted estimates show that in 2004 to 2005, diagnosed chronic conditions that were more prevalent among older women than men included hypertension (51% for women, 45% for men). Ever-diagnosed conditions that were more prevalent among older men than older women included HD (33% for men, 26% for women) and DM (17% for men, 15% for women).[6]
- The age-adjusted prevalence of hypertension (both diagnosed and undiagnosed) in 1999 to 2002 was 78% for older women and 64% for older men on the basis of data from NHANES/NCHS.[6]

Children and Adolescents

- Analysis of NHES, HHANES, and 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%, respectively, 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 gender 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 males than in females. In that study, HBP in children and adolescents was defined as SBP or DBP that was, on repeated measurement, ≥95th percentile.[7]
- A study in Ohio of more than 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

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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.[8]

- A study from 1988 to 1994 through 1999 to 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.[9]

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%.[10]
- 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 due to HD, and a 4.2-times greater rate of end-stage kidney disease (Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure [JNC] 5 and 6).
- Within the black community, rates of hypertension vary substantially.[10,11]
  - — 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 diabetes.
  - — 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 Reasons for Geographic and Racial Differences in Stroke Study (REGARDS) 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 whites. In contrast, geographic disparities in hypertension awareness, treatment, and control were minimal.[12]
- Data from the 2008 NHIS survey showed that black adults ≥18 years of age were more likely (31.8%) to have been told on ≥2 occasions that they had hypertension than American Indian/Alaska Native adults (25.3%), white adults (23.3%), and Asian adults (21.0%).[13]
- The CDC analyzed death certificate data from 1995 to 2002 (any-mention mortality; ICD-9 codes 401 to 404 and ICD-10 codes I10 to 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 women (118.3) was substantially lower than that observed for Hispanic men (135.9). Male hypertension-related mortality rates 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).[14]
- 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 surveys 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.[15] Data from the NHLBI's ARIC study found that hypertension was a particularly powerful risk factor for CHD in black persons, especially black women.[16]
- 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.[17]
- 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.[18]

Mortality

HBP mortality in 2006 was 56 561. Any-mention mortality in 2006 was ≈326 000.[19] Preliminary 2007 mortality was 57 077. The preliminary 2007 death rate was 17.5.[20]

- From 1996 to 2006, the death rate due to HBP increased 19.5%, and the actual number of deaths rose 48.1% (NCHS and NHLBI; appropriate comparability ratios were applied).

The 2006 overall death rate due to HBP was 17.8. Death rates were 15.6 for white males, 51.1 for black males, 14.3 for white females, and 37.7 for black females. When any-mention mortality for 2006 was used, the overall death rate was 107.6. Death rates were 106.3 for white males, 231.2 for

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black males, 90.2 for white females, and 176.5 for black females.[21]

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 estimated that when measured at a single examination, BP levels are ≈40% heritable; when measured across multiple examinations, long-term BP trends are ≈55% heritable.[22]

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.[23]
- 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.[24]
  - Total life expectancy was 5.1 years longer for normotensive men and 4.9 years longer for normotensive women than for hypertensives 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 1996 to 2006, the number of inpatient discharges from short-stay hospitals with HBP as the first-listed diagnosis increased from 417 000 to 514 000 (NCHS, NHDS). The number of all-listed discharges increased from 6 163 000 to 11 180 000[25,26] (unpublished data from the NHDS, 2006).
- Data from ambulatory medical care utilization estimates for 2007 showed that the number of visits for essential hypertension was 46 284 000.[27]
- In 2006, there were 293 000 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 9 057 000 times for hospitalized inpatients[26] (unpublished data from the NHDS, 2006).

Awareness, Treatment, and Control

- Data from NHANES/NCHS 2003 to 2006 showed that of those with hypertension who were ≥20 years of age, 77.6% were aware of their condition, 67.9% were under current treatment, 44.1% had their hypertension under control, and 55.9% did not have it controlled (NCHS and NHLBI).
- Analysis of NHANES/NCHS data from 1999 to 2004 through 2005 to 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.[4,28]
- Data from the 2007 BRFSS/CDC survey indicate that the percentage of adults ≥18 years of age who had been told that they had HBP ranged from 19.7% in Utah to 33.8% in Tennessee. The median percentage among states was 27.8%.[29]
- In NHANES/NCHS 2005 to 2006, rates of control were lower in Mexican Americans (35.2%) than in non-Hispanic whites (46.1%) and non-Hispanic blacks (46.5%).[4]
- 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. Prevalences 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%.[30]
- Data from the FHS study 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.[31]
- Data from the Women's Health Initiative 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.[32]

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- A study of more than 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.[33]
- 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 or more 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.[34]
- On the basis of NHANES 2003 to 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.[35]

Cost

- The estimated direct and indirect cost of HBP for 2010 is $76.6 billion.

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 doctor or other health professional that one has hypertension.
- On the basis of NHANES 2005 to 2006 data, it is estimated that ≈25% of the US population ≥20 years of age has prehypertension, including 32 400 000 men and 21 200 000 women (estimated by NHLBI).[4]
- 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.[36]
- 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 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 persons <60 years of age, 4.9% in those 60 to 79 years of age, and 19.8% in those ≥80 years of age.[31]
- In a study of NHANES 1999 to 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. Persons with prehypertension were 1.65 times more likely to have 1 or more of these adverse risk factors than were those with normal BP.[37]

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Table 6-1. High Blood Pressure

Population Group	Prevalence, 2006, Age ≥20 y	Mortality,* 2006, All Ages	Hospital Discharges, 2006, All Ages	Estimated Cost, 2010
Both sexes	74 500 000 (33.6%)	56 561	514 000	$76.6 Billion
Males	35 700 000 (34.4%)	24 382 (43.1%)†	204 000	. . .
Females	38 800 000 (32.6%)	32 179 (56.9%)†	309 000	. . .
NH white males	34.3%	17 581	. . .	. . .
NH white females	31.1%	24 888	. . .	. . .
NH black males	43.04%	6089	. . .	. . .
NH black females	44.8%	6480	. . .	. . .
Mexican American males	25.9%	. . .	. . .	. . .
Mexican American females	31.6%	. . .	. . .	. . .
Hispanic or Latino‡ ≥18 y	21.0%	. . .	. . .	. . .
Asian‡ ≥18 y	21.0%	. . .	. . .	. . .
American Indians/Alaska Natives‡ ≥18 y	25.3%	. . .	. . .	. . .

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 HBP mortality that is for males vs females.

‡NHIS (2008), NCHS; data are weighted percentages for Americans ≥18 years of age.[13]

Sources: Prevalence: NHANES (2003–2006, NCHS) and NHLBI; percentages for racial/ethnic groups are age-adjusted for Americans ≥20 years of age. Age-specific percentages are extrapolated to the 2006 US population estimates. Mortality: NCHS; these data represent underlying cause of death only. Hospital discharges: NHDS, NCHS; data include those discharged alive, dead, or status unknown. Cost: NHLBI; data include estimated direct and indirect costs for 2009.

Sources: Prevalence: NHANES (2003–2006, NCHS) and NHLBI. Percentages for racial/ethnic groups are age- adjusted for Americans ≥20 years of age. Age-specific percentages are extrapolated to the 2006 US population estimates. Mortality: NCHS. These data represent underlying cause of death only. Hospital discharges: NHDS, NCHS; data include those discharged alive, dead, or status unknown. Cost: NHLBI; data include estimated direct and indirect costs for 2009.

Hypertension is defined as SBP ≥140 mm Hg or DBP ≥90 mm Hg, taking antihypertensive medication, or being told twice by a physician or other professional that one has hypertension. The NHLBI computed the numbers and rates on the basis of NHANES 2005–2006 (NCHS). Many studies define hypertension as BP ≥140/90 mm Hg or taking antihypertensive medication. Under this definition, extrapolation of NHANES 2003–2006 (NCHS) data to the US population in 2006 gives an estimated prevalence of 65.6 million. That is 30% of the population ≥20 years of age compared with 33.6% according to the more complete definition, a difference of almost 9 million persons.

Table 6-2. Hypertension Awareness, Treatment, and Control: NHANES 1988–1994 and 1999–2006, by Race and Sex

	Awareness, %		Treatment, %		Control, %	
	1988–1994	1999–2006	1988–1994	1999–2006	1988–1994	1999–2006
NH white male	63.0	71.8	46.2	61.8	22.0	41.9
NH white female	74.7	76.9	61.6	68.1	32.2	40.0
NH black male	62.5	70.1	42.3	59.6	16.6	34.1
NH black female	77.8	85.3	64.6	76.6	30.0	43.8
Mexican American male	47.8	57.7	30.9	41.8	13.5	25.6
Mexican American female	69.3	69.9	47.8	57.9	19.4	31.9

NH indicates non-Hispanic.

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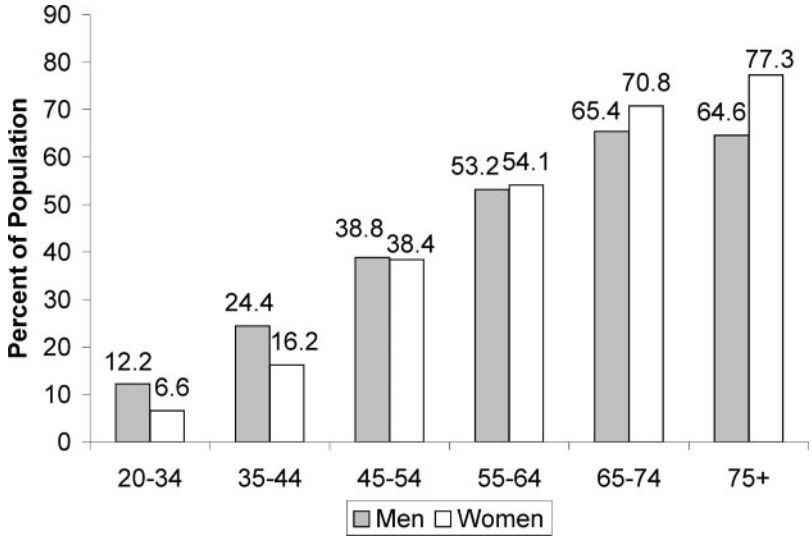

Chart 6-1. Prevalence of HBP in adults ≥20 years of age by age and sex (NHANES: 2003–2006). Source: NCHS and NHLBI. Hypertension is defined as SBP ≥140 mm Hg or DBP ≥90 mm Hg, taking antihypertensive medication, or being told twice by a physician or other professional that one has hypertension.

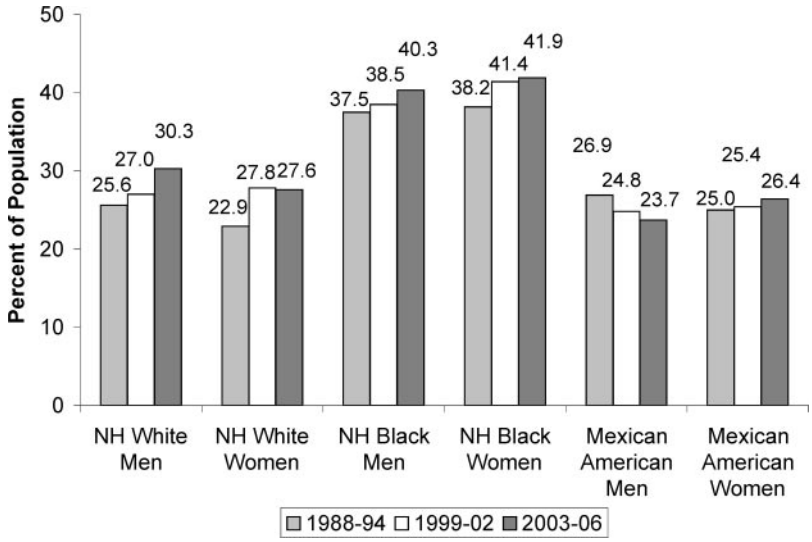

Chart 6-2. Age-adjusted prevalence trends for HBP in adults ≥20 years of age by race/ethnicity, sex, and survey (NHANES: 1988–1994, 1999–2002, and 2003–2006). Source: NCHS and NHLBI. NH indicates non-Hispanic.

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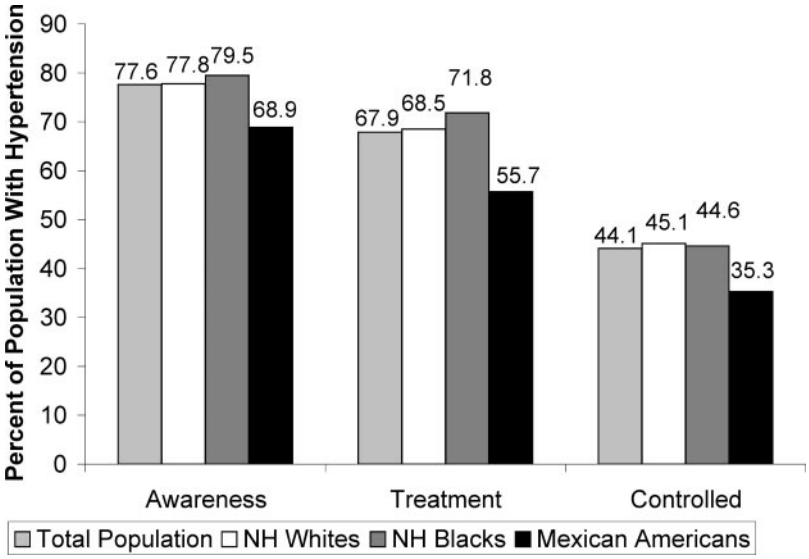

Chart 6-3. Extent of awareness, treatment, and control of HBP by race/ethnicity (NHANES: 2003–2006). Source: NCHS and NHLBI. NH indicates non-Hispanic.

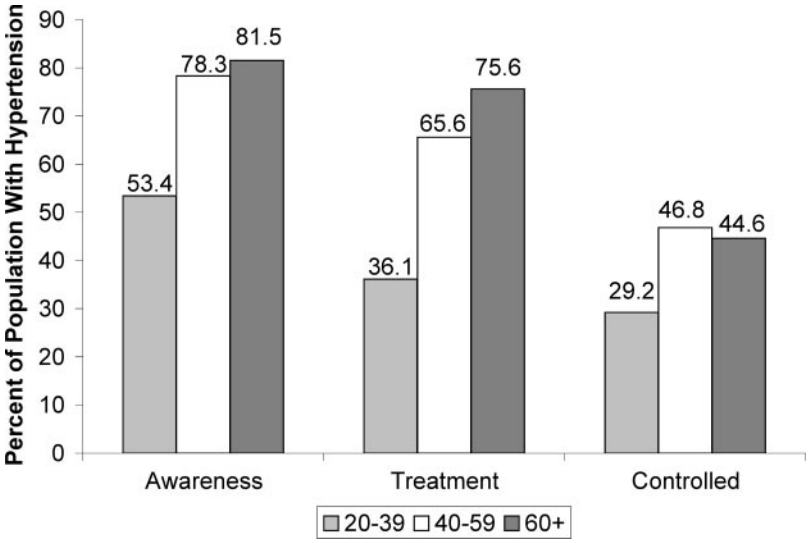

Chart 6-4. Extent of awareness, treatment, and control of HBP by age (NHANES: 2003–2006). Source: NCHS and NHLBI.

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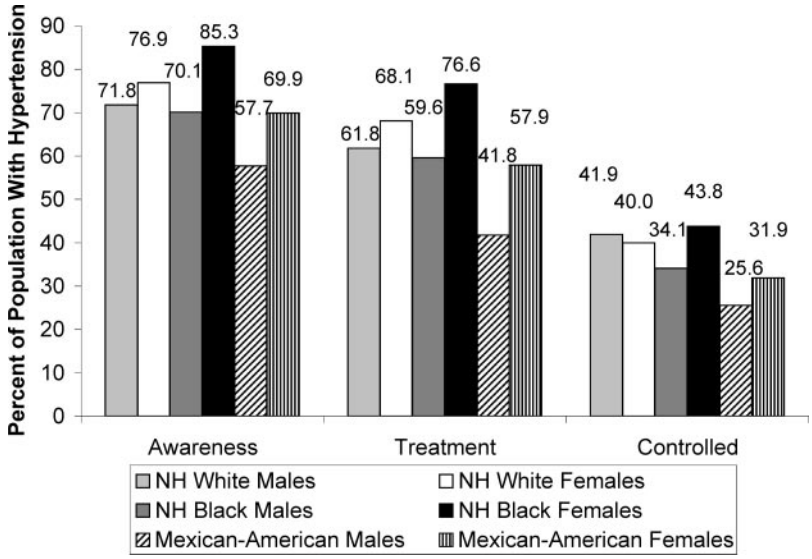

Chart 6-5. Extent of awareness, treatment, and control of HBP by race/ethnicity and sex (NHANES: 1999–2006). Source: NCHS and NHLBI. NH indicates non-Hispanic.

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7. Congenital Cardiovascular Defects

ICD-9 745–747, ICD-10 Q20–Q28. See Tables 7-1 through 7-4.

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 and/or hemodynamic lesions.

Defects range in severity from tiny pinholes between chambers, which are nearly irrelevant and often 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:

- Tetralogy of Fallot (TOF; 9% to 14%)
- Transposition of the great arteries (TGA; 10% to 11%)
- Atrioventricular septal defects (4% to 10%)
- Coarctation of the aorta (8% to 11%)
- Hypoplastic left heart syndrome (HPLHS; 4% to 8%)
- Ventricular septal defects (VSDs)

Although VSDs may close spontaneously, these lesions are the most prevalent in childhood and still account for 14% to 16% of defects that require an invasive procedure within the first year of life.[1] Atrial septal defects (ASDs) are the most common defects seen in adults.[2]

Prevalence

The estimated number of adults with congenital heart defects ranges from 650 000 to 1.3 million.[1,2] From 1940 to 2002, ≈2 million patients with congenital cardiovascular defects were born in the United States, ≈1 million with simple lesions and 0.5 million each with moderate and complex lesions. Using available data to estimate the prevalence of congenital cardiovascular defects at birth and in adults in year 2000, the survival of these patients is estimated to be 2000 assuming no treatment (the low estimate) and full treatment (the high estimate). If all 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 bicuspid aortic valves. 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.[2] The 32nd Bethesda Conference estimated that the total number of adults living with congenital heart disease in the United States in 2000 was 787 800.[3] Currently, no measured data are available in the United States for the prevalence of congenital cardiovascular defects in adults. Population data from Quebec, Canada, measured a prevalence of congenital cardiac defects of 11.89 per 1000 children and 4.09 per 1000 adults.[4] The most common types of defects in children are as follows: VSD, 620 000 people; ASD, 235 000 people; valvular pulmonary stenosis, 185 000 people; and patent ductus arteriosus, 173 000 people.[2] The most common lesions seen in adults are ASD and TOF.[3]

Abbreviations Used in Chapter 7

ASD	atrial septal defect
CDC	Centers for Disease Control and Prevention
CHD	coronary heart disease
CI	confidence interval
DM	diabetes mellitus
HPLHS	hypoplastic left heart syndrome
ICD	*International Classification of Diseases*
KID	Kids' Inpatient Database
MACDP	Metropolitan Atlanta Congenital Defects Program
TGA	transposition of the great arteries
TOF	tetralogy of Fallot
VSD	ventricular septal defect

Incidence

As of 2002, the most commonly reported incidence of congenital cardiovascular defects in the United States is between 4 and 10 per 1000, clustering around 8 per 1000 live births.[5] Major defects are usually apparent in the neonatal period, but minor defects may not be detected until adulthood. Thus, true measures of the incidence of congenital heart disease would need to record new cases of defects that present any time in fetal life through adulthood; however, estimates are available only for new cases detected between birth and 30 days of life, known as birth prevalence, or for new cases detected in the first year of life only. Both of 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 7-1.

- According to the CDC, 1 in every 110 infants in the metropolitan Atlanta, Ga, area is born with a congenital heart defect, including some infants with tiny defects that resolve without treatment. Some defects occur more commonly in males or females or in whites or blacks.[5]
- Nine (9.0) defects per 1000 live births, or 36 000 affected infants per year, are expected in the United States. Of these, several studies suggest that 9200, or 2.3 per 1000 live births, require invasive treatment or result in death 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.[6]
- Some studies suggest that as many as 5% of newborns, or 200 000 per year, are born with tiny muscular VSDs, almost all of which close spontaneously.[7,8] These defects almost never require treatment, so they are not included in Table 7-1.
- 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.

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These data indicated that there are >6500 estimated annual cases of 5 cardiovascular defects: Truncus arteriosus, TGA, TOF, atrioventricular septal defect, and HPLHS.[9]

Risk Factors

- Numerous intrinsic and extrinsic nongenetic risk factors contribute to CHD.[10]
- 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 HPLHS (4.6%).[11]
- 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.[12]
- Associations between exposure to air pollutants during first-trimester pregnancy and risks of congenital heart defects were documented from 1986 to 2003 by the Metropolitan Atlanta Congenital Defects Program (MACDP) that related carbon monoxide, nitrogen dioxide, and sulfur dioxide measurements to the risk of ASD, VSD, TGA, and TOF.[13]
- 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.[14]
- Although folic acid supplementation is recommended during pregnancy to potentially reduce the risk of congenital heart defects,[10] 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.[15] A study from Quebec 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.[16]
- Pregestational DM was significantly associated with cardiac defects, both isolated and multiple. Gestational DM was associated with a limited group of birth defects.[17]

Mortality

Congenital cardiovascular defects mortality in 2006 was 3531. Any-mention mortality related to congenital cardiovascular defects in 2006 was 6883.

- Congenital cardiovascular defects are the most common cause of infant death resulting from birth defects; more than 29% of infants who die of a birth defect have a heart defect (National Vital Statistics System, final data for 2005).
- The 2006 death rate for congenital cardiovascular defects was 1.2. Death rates were 1.3 for white males, 1.3 for black males, 1.0 for white females, and 1.7 for black females. Crude infant mortality rates (<1 year of age) were 36.5 for white infants and 52.5 for black infants.[18]
- In 2005, 192 000 life-years were lost before 55 years of age because of deaths due to congenital cardiovascular defects. This is about the same as the life-years lost from leukemia, prostate cancer, and Alzheimer's disease combined.[18]
- The mortality rate attributable to congenital defects has been declining. From 1979 to 1997, age-adjusted death rates due to all defects declined 39%, and deaths tended to occur at progressively older ages. Nevertheless, 45% of deaths still occurred in infants <1 year of age. The mortality rate varies considerably according to type of defect.[19]
- From 1996 to 2006, death rates for congenital cardiovascular defects declined 33.3%, whereas the actual number of deaths declined 26.7%.[18]
- Data analysis from the Society of Thoracic Surgeons, a voluntary registry with self-reported data for a 4-year cycle (2004 to 2007) from 68 centers performing congenital heart surgery (67 from the United States and 1 from Canada) showed that of 61 410 total operations, the overall aggregate hospital discharge mortality rate was 3.7%; specifically, for neonates (0 to 30 days of age), the mortality rate was 10.7%; for infants (31 days to 1 year of age), it was 2.6%; for children (>1 year to 18 years of age), it was 1.2%; and for adults (>18 years of age), it was 1.9%.[20]
- Using the Nationwide Inpatient Sample 1988 to 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%; $P<0.0001$).[21]

Hospitalizations

In 2004, birth defects accounted for >139 000 hospitalizations, representing 47.4 stays per 100 000 persons. Cardiac and circulatory congenital anomalies, which include ASDs and VSDs, accounted for more than one third of all hospital stays for birth defects and had the highest in-hospital mortality rate. Between 1997 and 2004, hospitalization rates increased by 28.5% for cardiac and circulatory congenital anomalies. For almost 86 300 hospitalizations, ASD was noted as the principal reason for the hospital stay or as a coexisting or secondary condition.[22]

Cost

- From 2003 data from the Healthcare Cost and Utilization Project 2003 Kids' Inpatient Database (KID) and information on birth defects in the Congenital Malformations

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Surveillance Report, it was found that the most expensive average neonatal hospital charges were for 2 congenital heart defects: HPLHS ($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.[23]

- 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.[22]

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Table 7-1. Congenital Cardiovascular Defects

Population Group	Estimated Prevalence Adults	Mortality, 2006, All Ages	Hospital Discharges, 2006, All Ages
Both sexes	650 000 to 1.3 million[2]	3531	70 000
Males	. . .	1951 (55.3%)*	30 000
Females	. . .	1580 (44.7%)*	40 000
White males	. . .	1510	. . .
White females	. . .	1216	. . .
Black males	. . .	356	. . .
Black females	. . .	296	. . .

Ellipses (…) indicate data not available.

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

Sources: Mortality: NCHS. These data represent underlying cause of death only; data for white and black males and females include Hispanics. Hospital discharges: NHDS, NCHS; data include those inpatients discharged alive, dead, or status unknown.

Table 7-2. Annual Incidence of Congenital Cardiovascular Defects[1,5–8,24]

Type of Presentation	Rate per 1000 Live Births	n
Fetal loss	Unknown	Unknown
Invasive procedure during the first year	2.3	9200
Detected during first year*	9	36 000
Bicuspid aortic valve	13.7	54 800
Other defects detected after first year	Unknown	Unknown
Total	Unknown	Unknown

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

Table 7-3. 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

*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,[2] with permission from Elsevier. Average of the low and high estimates, two thirds from low estimate.[2]

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Table 7-4. 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)		
Male	8127	14 109
Deaths	420	714
Mortality rate, %	5.2	5.1
Female	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

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 KID, HCUPnet, Healthcare Cost and Utilization Project, Agency for Healthcare Research and Quality (http://www.hcup-us.ahrq.gov), and personal communication with Kathy Jenkins, MD, Children's Hospital of Boston, October 1, 2006.

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8. Cardiomyopathy and Heart Failure

ICD-9 425; ICD-10 I42. See Table 8-1 and Charts 8-1 through 8-3.

Cardiomyopathy

ICD-9 425; ICD-10 I42.

Mortality—24 703. Any-mention mortality—49 700. Hospital discharges—43 000.

- Mortality from cardiomyopathy is highest in older persons, men, and blacks (CDC compressed file).
- 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.[1]
- 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).[2]
  - The overall incidence of cardiomyopathy is 1.13 cases per 100 000 in children <18 years of age.
  - In children <1 year of age, the incidence is 8.34, and in 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).
- Studies show that 36% of young athletes who die suddenly have probable or definite hypertrophic cardiomyopathy.[3]
- Hypertrophic cardiomyopathy is the leading cause of sudden cardiac death in young people, including trained athletes. Hypertrophic cardiomyopathy is the most common inherited heart defect, occurring in 1 of 500 individuals. In the United States, ≈500 000 people have hypertrophic cardiomyopathy, yet most are unaware of it.[4]
- In a recent report of the Pediatric Cardiomyopathy Registry, the overall annual incidence of hypertrophic cardiomyopathy 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.[5]
- 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 per year 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%).[6]

Abbreviations Used in Chapter 8

ARIC	Atherosclerosis Risk in Communities Study
BMI	body mass index
BP	blood pressure
CARDIA	Coronary Artery Risk Development In Young Adults Study
CDC	Centers for Disease Control and Prevention
CHD	coronary heart disease
CHS	Cardiovascular Health Study
EF	ejection fraction
EPHESUS	Eplerenone Post-Acute Myocardial Infarction Heart Failure Efficacy and Survival Study
FHS	Framingham Heart Study
HF	heart failure
ICD	International Classification of Diseases
MI	myocardial infarction
mm Hg	millimeters of mercury
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

Heart Failure

ICD-9 428, ICD-10 I50.

Incidence

- Data from the NHLBI-sponsored FHS[7] indicate that:
  - 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]
- Among 21 906 white male physicians in the Physicians Health Study I, there was no significant change in the age-adjusted incidence of confirmed, self-reported HF between 1985 and 1989 (1.75 per 1000 person-years) and 2000 and 2004 (1.96 per 1000 person-years).[9]
- In Olmsted County, Minn, the incidence of HF (ICD-9 428) did not decline during 2 decades, but the survival rate

*Unreliable estimate.

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improved overall, with less improvement, however, among women and elderly persons.[10]

- Data from the ARIC study of the NHLBI found the age-adjusted incidence rate per 1000 person-years to be 3.4 for white women, significantly less than 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 that of 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 and improved survival rate among the elderly, with both of these effects being greater in men.[12]
- Data from hospitals in Worcester, Mass, 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]
- A retrospective study of a well-defined population of older persons provides further insight into the epidemic increase in HF observed in the United States and elsewhere between the 1970s and 1990s. The epidemic increase in HF among the older population is associated with increased incidence and improved survival rate, with both of these effects being greater in men than in women.[12]
- Data from the CARDIA study indicate that HF before age 50 years is more common among blacks than whites. Hypertension, obesity, and systolic dysfunction are important risk factors that may be targets for prevention.[14]

Risk Factors

- In the NHLBI-sponsored FHS, hypertension is a common risk factor for HF that contributed to a large proportion of HF cases, followed closely by antecedent MI.[15]
- In a 1993 to 2000 study of 2763 postmenopausal women with established coronary disease, diabetes was the strongest risk factor for HF. Diabetic women with elevated BMI or reduced creatinine clearance were at highest risk, with annual incidence rates of 7% and 13%, respectively. Among nondiabetic women with no risk factors, the annual incidence of HF was 0.4%. HF incidence increases with each additional risk factor, and nondiabetic women with ≥3 risk factors had an annual incidence of 3.4%. Among diabetic persons with no additional risk factors, the annual incidence of HF was 3.0%, compared with 8.2% among diabetics with ≥3 additional risk factors.[16]
- The prevalence of diabetes is increasing among older persons with HF, and diabetes is a risk factor for death in these individuals. Between 1979 and 1999, among subjects in Olmsted, Minn, with a first diagnosis of HF, data indicate that the prevalence of diabetes increased 3.8% every year. The odds of having diabetes for those first diagnosed with HF in 1999 were nearly 4 times higher than for those diagnosed 20 years earlier. The 5-year survival rate was 46% for those with HF alone, but only 37% for those with HF and diabetes mellitus.[17]
- In the Framingham Offspring Study, among 2739 participants, increased circulating concentrations of resistin were associated with incident HF, independently of prevalent coronary disease, obesity, insulin resistance, and inflammation.[18]

Left Ventricular Function

- Data from Olmsted County, Minn, 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. Altogether, 6% had moderate or severe diastolic dysfunction with normal ejection fraction (EF). 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.[19]
  - Among individuals with symptomatic HF, the prevalence rates of left ventricular diastolic dysfunction were 6% for mild diastolic dysfunction and 75% for moderate or severe diastolic dysfunction. Isolated diastolic dysfunction (diastolic dysfunction with preserved EF) was present in 44% of persons presenting with HF. The prevalence of systolic dysfunction was 45%.[20]
  - The proportion of persons with HF and preserved EF increased over time. The survival rate improved over time among individuals with reduced EF but not among those with preserved EF.[21]

Mortality

- In 2006, HF any-mention mortality was 282 754 (159 167 males and 123 587 females). HF was mentioned on 282 754 US death certificates and was selected as the underlying cause in 60 337 of those deaths.[22] In preliminary 2007 mortality, HF was selected as the underlying cause in 57 235 deaths.[23] Unlike other cardiovascular diseases, HF is the end stage of a cardiac disease. It is most often a consequence of hypertension, CHD, valve deformity, diabetes, or cardiomyopathy. There are other less common causes of HF. For each of the 60 337 deaths, the true underlying cause—that is, the origin of HF—is not known. The certifier of the cause of death either failed to report the underlying cause or had insufficient information to do so. In those cases, HF must be nominally coded as the underlying cause. Table 8-1 contains the total-mention numbers of deaths from HF, with a footnote giving the numbers of these deaths that are coded to HF as the underlying cause.
- The 2006 overall any-mention death rate for HF was 89.2. Any-mention death rates were 103.7 for white males, 105.9 for black males, 80.3 for white females, and 84.4 for black females (NCHS, NHLBI).
- One in 8 deaths has HF mentioned on the death certificate (NCHS, NHLBI).

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- The number of any-mention deaths from HF was about as high in 1995 (287 000) as it was in 2006 (283 000) (NCHS, NHLBI).
- On the basis of follow-up of the original and offspring FHS cohorts (NHLBI) 65 to 74 years of age for selected time periods from the 1950s to the 1990s:
  - The 1-year mortality rate for HF is high, with 1 in 5 dying.
  - After HF is diagnosed, survival is lower in men than in women: 59% of men and 45% of women that age who have HF diagnosed will die within 5 years.
  - Even after surviving at least 30 days after onset, 54% of men and 40% of women will die within 5 years.
  - Although these percentages are appreciable, rates of death after onset of HF declined by approximately one third from the 1950s to the 1990s in both sexes.[24,25]

Hospital Discharges/Ambulatory Care Visits

- Hospital discharges for HF increased from 877 000 in 1996 to 1 106 000 in 2006 (unpublished data from the NHDS 2006, NCHS).[26]
- Data from Ambulatory Medical Care Utilization Estimates for 2007 showed the number of visits for HF was 3 434 000.[27]

Cost

- The estimated direct and indirect cost of HF in the United States for 2010 is $39.2 billion.[28] (See Chapter 20.) This estimate is likely greatly understated because it is based on data for HF as the primary diagnosis or underlying cause of death.
- Cost utilization data from 1516 outpatients enrolled in the EPHESUS study found that health status assessments can identify HF outpatients with left ventricular dysfunction after MI who are likely to have high resource use over the following year despite excellent medical therapy.[29]

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Table 8-1. Heart Failure

Population Group	Prevalence, 2006 Age ≥20 y	Incidence (New Cases) Age ≥45 y	Mortality (Any Mention), 2006 All Ages*	Hospital Discharges, 2006 All Ages	Cost, 2010
Both sexes	5 800 000 (2.6%)	670 000	282 754	1 106 000	$39.2 billion
Males	3 100 000 (3.1%)	350 000	123 600 (43.7%)†	523 000	. . .
Females	2 700 000 (2.1%)	320 000	159 167 (56.3%)†	583 000	. . .
NH white males	3.2%	. . .	110 250	. . .	. . .
NH white females	2.1%	. . .	142 378	. . .	. . .
NH black males	3.0%	. . .	10 926	. . .	. . .
NH black females	3.6%	. . .	14 151	. . .	. . .
Mexican American males	1.7%	. . .	. . .	. . .	. . .
Mexican American females	1.8%	. . .	. . .	. . .	. . .

Ellipses (…) indicate data not available.

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

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

Sources: Prevalence: NHANES 2003–2006 (NCHS) and NHLBI. Percentages are age adjusted for Americans ≥20 years of age. Age-specific percentages are extrapolated to the 2006 US population estimates. These data are based on self-reports. Incidence: FHS, 1980–2003 from NHLBI Incidence and Prevalence Chart Book, 2006. Mortality: NCHS. HF as an underlying cause of death accounted for 60 337 of the any-mention deaths in 2006: 23 918 males and 36 419 females. Hospital discharges: NHDS, NCHS; data include those inpatients discharged alive, dead, or "status unknown." Cost: NHLBI; data include estimated direct and indirect costs for 2010.

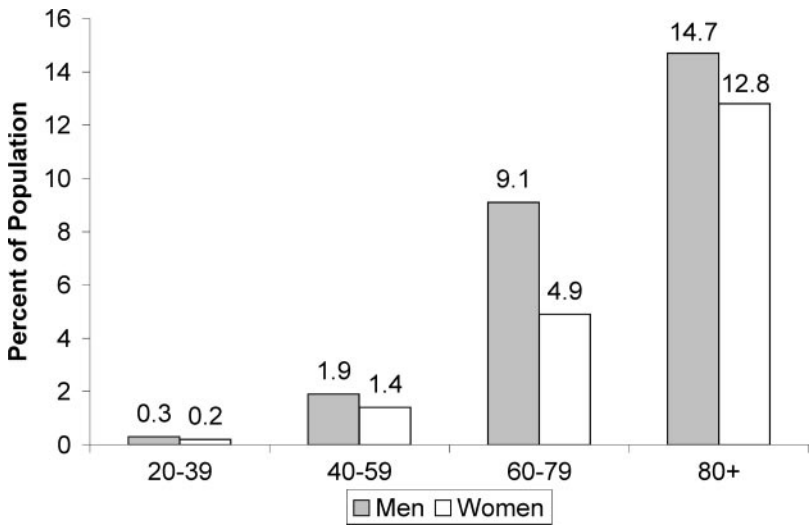

Chart 8-1. Prevalence of HF by sex and age (NHANES: 2003–2006). Source: NCHS and NHLBI.

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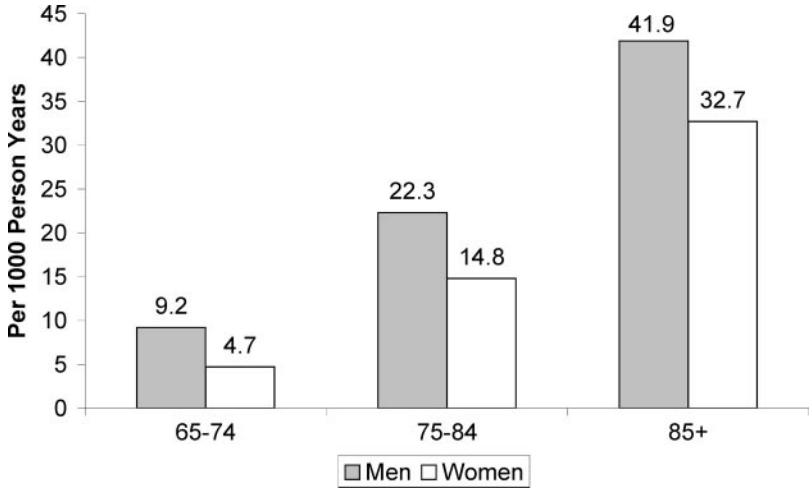

Chart 8-2. Incidence of HF (HF based on physician review of medical records and strict diagnostic criteria) by age and sex (FHS 1980–2003). Source: NHLBI.

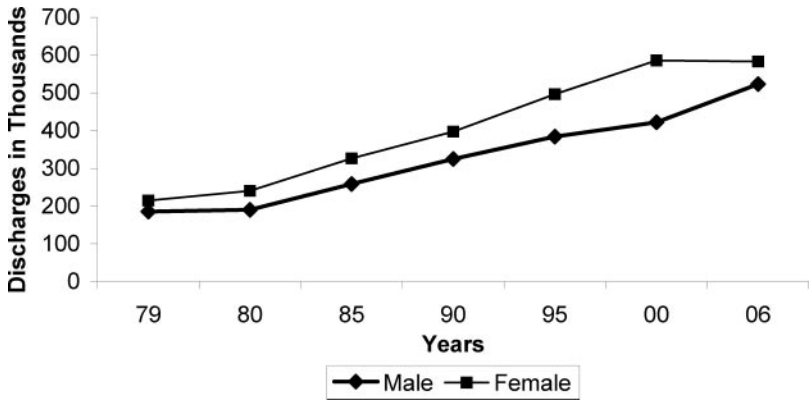

Chart 8-3. Hospital discharges for HF by sex (United States: 1979–2006). Note: Hospital discharges include people discharged alive, dead, and status unknown. Source: NHDS/NCHS and NHLBI.

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9. Other Cardiovascular Diseases

See Table 9-1.

Mortality and any mention mortality in this section are for 2006. "Mortality" is the number of deaths in 2006 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 2006 are based on *ICD-9* codes.

Rheumatic Fever/Rheumatic Heart Disease

ICD-9 390 to 398; ICD-10 I00-I09.
Mortality—3257. Any-mention mortality—6033.

- The incidence of acute rheumatic fever has decreased in the United States.[1]

Abbreviations Used in Chapter 9

AAA	abdominal aortic aneurysm
ABI	ankle brachial index
AF	atrial fibrillation
AHA	American Heart Association
ARIC	Atherosclerosis Risk in Communities study
BMI	body mass index
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
CI	confidence interval
CVD	cardiovascular disease
DVT	deep vein thrombosis
FHS	Framingham Heart Study
HBP	high blood pressure
HF	heart failure
HLA	human leukocyte antigen
ICD	*International Classification of Diseases*
IE	infective endocarditis
KD	Kawasaki disease
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
PAD	peripheral arterial disease
PE	pulmonary embolism
REACH	Reduction of Atherothrombosis for Continued Health
RR	relative risk
VTE	venous thromboembolism
WHO	World Health Organization
WRIGHT	WHO Research Into Global Hazards of Travel

- Although localized outbreaks have occurred, the overall incidence of acute rheumatic fever remains very low in most areas of the United States.[2,3]
- The incidence of rheumatic fever remains high in blacks, Puerto Ricans, Mexican Americans, and American Indians.[4]
- In 1950, ≈15 000 Americans (adjusted for changes in ICD codes) died of rheumatic fever/rheumatic heart disease, compared with ≈3300 today (NCHS/NHLBI).
- From 1996 to 2006, the death rate from rheumatic fever/rheumatic heart disease fell 8.3%, and actual deaths declined 26.2% (NCHS/NHLBI).
- The 2006 overall death rate for rheumatic fever/rheumatic heart disease was 1.1. Death rates were 0.8 for white males, 0.7 for black males, 1.3 for white females, and 0.9 for black females (NCHS/NHLBI).
- Immune risk factors have been linked with rheumatic heart disease. HLA typing was performed in 120 black patients with severe chronic rheumatic heart disease 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, suggesting that genetically determined immune response factors may play a role in the pathogenesis of severe chronic rheumatic heart disease.[5]

Pulmonary Embolism

ICD-9 415.1; ICD-10 I26.
Mortality—6924. Any-mention mortality—27 755. Hospital discharges—145 000.

- In the Nurses' Health Study, nurses ≥60 years of age in the highest BMI quintile had the highest rates of PE. Heavy cigarette smoking and HBP were also identified as risk factors for PE.[6]
- Death occurs in ≈12% of recognized PE cases within 1 month of diagnosis.[7]
- A study of Medicare recipients ≥65 years of age reported 30-day case fatality rates in patients with PE. Overall, men had higher fatality rates than women (13.7% versus 12.8%), and blacks had higher fatality rates than whites (16.1% versus 12.9%).[6]
- In the International Cooperative Pulmonary Embolism Registry, the 3-month mortality rate was 17.5%. In contrast, the overall 3-month mortality rate in the Prospective Investigation of Pulmonary Embolism Diagnosis was 15%, but only 10% of deaths during 1 year of follow-up were ascribed to PE.[6]
- The age-adjusted rate of deaths from pulmonary thromboembolism decreased from 191 per million in 1979 to 94 per million in 1998 overall, decreasing 56% for men and 46% for women. During this time, the age-adjusted mortality rates for blacks were consistently 50% higher than those for whites, and those for whites were 50% higher than those for people of other races (eg, Asian, American Indian). Within racial strata, mortality rates were consistently 20% to 30% higher among men than among women.[8]

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Bacterial Endocarditis

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

Any-mention mortality—2554. Hospital discharges—35 000, primary plus secondary diagnoses.

- The 2007 AHA Guidelines on Prevention of Infective Endocarditis[9] 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.[10]
- Transient bacteremia is common with manipulation of the teeth and periodontal tissues, and reported frequencies of bacteremia due to dental procedures vary widely: tooth extraction (10% to 100%), periodontal surgery (36% to 88%), scaling and root planing (8% to 80%), teeth cleaning (up to 40%), rubber dam matrix/wedge placement (9% to 32%), and endodontic procedures (up to 20%). Transient bacteremia also occurs frequently during routine daily activities unrelated to dental procedures: tooth brushing and flossing (20% to 68%), use of wooden toothpicks (20% to 40%), use of water irrigation devices (7% to 50%), and chewing food (7% to 51%). When it is considered that the average person living in the United States has <2 dental visits per year, the frequency of bacteremia from routine daily activities is far greater than that associated with dental procedures.[11]
- 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[9]:

  — Mitral valve prolapse: 1 per 1.1 million procedures;
  — CHD: 1 per 475 000;
  — Rheumatic heart disease: 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.[9]

Valvular Heart Disease

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

Mortality—21 386 Any-mention mortality—43 700. Hospital discharges—93 000.

- Echocardiographic data from the CARDIA Study (4351), the ARIC Study (2435), and the CHS (5125) were pooled to assess the prevalence of valve disease. The prevalence increased with age, from 0.7% (95% CI, 0.5 to 1.0) in participants 18 to 44 years of age to 13.3% (95% CI, 11.7 to 15.0) in participants ≥75 years of age ($P<0.0001$). The prevalence of valve disease, adjusted to the US 2000 population, was 2.5% (95% CI, 2.2 to 2.7). The adjusted mortality risk ratio associated with valve disease was 1.36 (95% CI, 1.15 to 1.62; $P=0.0005$).[11]
- Doppler echocardiography data in 1696 men and 1893 women (54±10 years of age) attending a routine examination at the Framingham Study 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.[12]

Aortic Valve Disorders

ICD-9 424.1; ICD-10 I35.

Mortality—13 752. Any-mention mortality—27 380. Hospital discharges—49 000.

- Calcific aortic stenosis on a trileaflet valve or bicuspid aortic valve are the most common causes of aortic stenosis.[13]
- In the Euro Heart Survey, which included 4910 patients in more than 25 countries, aortic stenosis was the most frequent lesion, accounting for 43% of all patients having valvular heart disease.[14]
- 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 from cardiovascular causes and the risk of MI.[15] Clinical factors associated with aortic sclerosis and stenosis were similar to risk factors for atherosclerosis.[16] These data largely exclude congenital heart disease patients, a group that is expected to increasingly contribute to the prevalence of valve disease.

Mitral Valve Disorders

ICD-9 424.0; ICD-10 I34.

Mortality—2581. Any-mention mortality—≈5975. Hospital discharges—41 000.

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 in a frequency of 1.7% as adjusted to the US adult population of 2000, increasing from 0.5% to 9.3% between 18 and ≥75 years of age.[11]

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- Isolated mitral stenosis is more common in women and occurs in 40% of all patients presenting with rheumatic heart disease.[17]
- 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.[18]
- 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 prolapse and those without prolapse.[18]

Pulmonary Valve Disorders
ICD-9 424.3; ICD-10 I37.
Mortality—12. Any-mention mortality—28.

Tricuspid Valve Disorders
ICD-9 424.2; ICD-10 I36.
Mortality—12 Any-mention mortality—93.

Endocarditis, Valve Unspecified
ICD-9 424.9; ICD-10 I38.
Mortality—5029 Any-mention mortality—10 186.

Arrhythmias (Disorders of Heart Rhythm)

ICD-9 426, 427; ICD-10 I46-I49.
Mortality—36 860. Any-mention mortality—461 016. Hospital discharges—835 000.

- In 2006, $3.1 billion ($7783 per discharge) was paid to Medicare beneficiaries for cardiac dysrhythmias.[19]

Atrial Fibrillation and Flutter
ICD-9 427.3; ICD-10 I48.
Mortality—11 438. Any-mention mortality—90 000. Prevalence —>2.2 million, projected to 2.66 million in 2010.[20,21] Incidence—>75 000.[21] Hospital discharges—461 000.

- 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%.[22]
- Data from a large community-based population suggest that AF is less prevalent in blacks than in whites, both overall and in the setting of CHF.[20,23]
- Data from the NHDS/NCHS (1996–2001) on cases that included AF as a primary discharge diagnosis found the following[24]:
  - — 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.
  - — The incidence in men ranged from 20.58/100 000 persons per year for patients between 15 and 44 years of age to 1077.39/100 000 persons per year for patients ≥85 years of age. In women, the incidence ranged from 6.64/100 000 persons per year for patients between 15 and 44 years of age to 1203.7/100 000 persons per year for those ≥85 years of age.
  - — From 1996 to 2001, hospitalizations with AF as the first-listed diagnosis increased 34%.
- In 1999, the CDC analyzed data from national and state multiple-cause mortality statistics and Medicare hospital claims for persons with AF. The most common disease listed as the primary diagnosis for persons hospitalized with AF was HF (11.8%), followed by AF (10.9%), CHD (9.9%), and stroke (4.9%).[25] In Olmsted County, Minnesota, the age-adjusted incidence of clinically recognized AF in a white population increased by 12.6% between 1980 and 2000.[26,27]
  - — The incidence of AF was greater in men (incidence ratio for men over women, 1.86) and increased markedly with older age.[26]
  - — If incidence estimates are applied to US population projections from the Census Bureau, the projected number of persons with AF may exceed 12 million by 2050.[26]
  - — 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 subsets of the study population. Stroke rates per 1000 patient-years declined from 46.7 in 1992 to 19.5 in 2002 for ischemic stroke but remained fairly steady for hemorrhagic stroke (1.6 to 2.9).[28]
  - — AF independently increases the risk of ischemic stroke by 4- to 5-fold.[29]
  - — AF is responsible for at least 15% to 20% of all ischemic strokes.[21]
  - — Paroxysmal, persistent, and permanent AF all appear to increase the risk of ischemic stroke to a similar degree.[30]
  - — AF is also an independent risk factor for ischemic stroke severity and recurrence. In one study, persons who have AF and are 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.[31]
  - — Isolated chronic atrial flutter is uncommon but is associated with a high risk of developing AF,[32] and data from a sample of 191 patients with chronic atrial flutter revealed a risk of ischemic stroke that was similar to that for AF.[33]
  - — 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

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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.[27]

Other Arrhythmias

Tachycardia
ICD-9 427.0, 1, 2; ICD-10 I47.0, 1, 2, 9.
Mortality—575. Any-mention mortality—6000. Hospital discharges—80 000.

Paroxysmal Supraventricular Tachycardia
ICD-9 427.0; ICD-10 I47.1.
Mortality—125. Any-mention mortality—1294. Hospital discharges—29 000.

Ventricular Fibrillation
ICD-9 427.4; ICD-10 I49.0.
Mortality—1014 Any-mention mortality—9 800. Hospital discharges—7000.

Ventricular fibrillation is listed as the cause of relatively few deaths, but the overwhelming majority of sudden cardiac deaths from coronary disease (estimated at ≈310 000 per year) are thought to be from ventricular fibrillation.

- In Olmsted County, Minn, the incidence of out-of-hospital treated ventricular fibrillation decreased from 1985 to 2002[34]:
  - 1985 to 1989: 26.3/100 000 (95% CI, 21.0 to 32.6)
  - 1990 to 1994: 18.2/100 000 (95% CI, 14.1 to 23.1)
  - 1995 to 1999: 13.8/100 000 (95% CI, 10.4 to 17.9)
  - 2000 to 2002: 7.7/100 000 (95% CI, 4.7 to 11.9).

Arteries, Diseases of

ICD-9 440 to 448; ICD-10 I70-I79. Includes peripheral arterial disease (PAD).
Mortality—31 136. Any-mention mortality—96 900. Hospital discharges—284 000.

Aortic Aneurysm

ICD-9 441; ICD-10 I71.
Mortality—13 238. Any-mention mortality—18 800. Hospital discharges—57 000.

- Although the definition varies somewhat by age and body surface area, generally, an AAA is considered to be present when the anteroposterior diameter of the aorta reaches 3.0 cm.[35]
- 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 groups.[35]
- 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.[35,36] The association of dyslipidemia with AAA is mixed.[37]
- Patients with diabetes mellitus are approximately half as likely as patients without diabetes to have AAA.[38,39]
- Male sex, older age, and smoking are important risk factors for incident AAA in the next 7 years.[40]
- Large AAAs tend to expand more rapidly than small AAAs, and large AAAs are at substantially higher risk for rupture.[35]
  - 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%.

Atherosclerosis

ICD-9 440; ICD-10 I70.
Mortality—8652. Any-mention mortality—50 600. Hospital discharges—129 000.

Atherosclerosis is a process that leads to a group of diseases characterized by a thickening of artery walls. Atherosclerosis causes many deaths from heart attack and stroke and accounts for nearly three fourths of all deaths from CVD (FHS, NHLBI).

Analysis of data from the REACH Registry[41] showed that atherothrombosis (CAD, CVD, and PAD) is associated with the main causes of death on a worldwide scale. Despite decreases in age-adjusted death rates, the absolute number of deaths from these conditions continues to increase, and prevalence is increasing sharply in other parts of the world. Atherothrombotic diseases are projected to be the leading cause of death worldwide in 2020. In the REACH study, outpatients with established atherosclerotic arterial disease or at risk of atherothrombosis experienced relatively high annual cardiovascular event rates. Multiple disease locations increased the 1-year risk of cardiovascular events.[42]

Other Diseases of Arteries

ICD-9 442 to 448; ICD-10 I72-I78.
Mortality—9246. Any-mention mortality—31 571 Hospital discharges—97 000.

Venous Thromboembolism

- VTE occurs for the first time in ≈100 per 100 000 persons each year in the United States. Approximately one third of patients with symptomatic VTE manifest PE, whereas two thirds manifest DVT alone.[7]
- Whites and blacks have a significantly higher incidence than Hispanics and Asians or Pacific Islanders.[7]
- In studies in Worcester, Mass, and Olmsted County, Minn, the incidence of VTE was ≈1 in 1000. In both studies, VTE was more common in men; for each 10-year increase in age, the incidence doubled. By extrapolation, it is estimated that >250 000 patients are hospitalized annually with VTE.[6]

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- The crude incidence rate per 1000 person-years was 0.80 in the ARIC study, 2.15 in the CHS, and 1.08 in the combined cohort. Half of the participants who developed incident VTE were women, and 72% were white.[43]
- A recent clinical trial (JUPITER) of individuals at risk for arterial CVD events examined venous thromboembolism (including PE) as an end point as well. Of note, the incidence of venous thromboembolic events was identical to the incidence rates for stroke and fatal/nonfatal MI.[44]
- More than 200 000 new cases of VTE occur annually. Of these, 30% die within 30 days, one fifth suffer sudden death due to PE, and ≈30% develop recurrent VTE within 10 years. Independent predictors for recurrence include increasing age, obesity, malignant neoplasm, and extremity paresis.[45]
- Data from the ARIC study of the NHLBI showed that the 28-day fatality rate from DVT is 9%; from PE, 15%; from idiopathic DVT or PE, 5%; from secondary non–cancer-related DVT or PE, 7%; and from secondary cancer-related DVT or PE, 25%.[46]
- The RR of VTE among pregnant or postpartum women was 4.29, and the overall incidence of VTE (absolute risk) was 199.7 per 100 000 woman-years. The annual incidence was 5 times higher among postpartum women than pregnant women, and the incidence of DVT was 3 times higher than that of PE. PE was relatively uncommon during pregnancy versus the postpartum period. Over the 30-year period, the incidence of VTE during pregnancy remained relatively constant, whereas the postpartum incidence of PE decreased >2-fold.[47]
- On the basis of a prospective study of black and white middle-aged adults in the ARIC study of the NHLBI, it was found that consumption of ≥4 servings of fruit and vegetables per day or ≥1 serving of fish per week was associated with lower incidence of VTE. In a comparison of the highest quintile of intake with the lowest, red and processed meat and a Western diet pattern were positively associated with incident VTE.[48]
- Results from phase I of the WHO WRIGHT project found that the risk of developing VTE approximately doubles after travel lasting ≥4 hours. Nevertheless, the absolute risk of developing VTE if seated and immobile for >4 hours remains relatively low, at ≈1 in 6000. Other risk factors that increase the risk of VTE during travel are obesity, being very tall or very short, use of oral contraceptives, and inherited blood disorders that lead to increased clotting tendency. One study within the project examining flights in particular found that those taking multiple flights over a short period of time are also at higher risk.[49] This is because the risk of VTE remains elevated for ≈4 weeks.

Deep Vein Thrombosis

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

Mortality—2328. Any-mention mortality—12 100.

A review of 9 studies conducted in the United States and Sweden showed that the mean incidence of first DVT in the general population was 5.04 per 10 000 person-years. The incidence was similar in males and females and increased dramatically with age from ≈2 to 3 per 10 000 person-years at 30 to 49 years of age to 20 at 70 to 79 years of age.[50]

- Death occurs in ≈6% of DVT cases within 1 month of diagnosis.[7]

Kawasaki Disease

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

Mortality—3. Any-mention mortality—8. Hospital discharges—5000 (figure considered unreliable), primary plus secondary diagnoses.

- There were an estimated 5600 admissions for KD in 2006. KD occurs more often among boys (60%) and among those of Asian ancestry[41] (Jane W. Newburger and Kimberlee Gauvreau of Children's Hospital of Boston, Mass; written communication, June 19, 2009).
- An estimated 4248 hospitalizations for KD occurred in the United States in 2000, with a median patient age of 2 years. Race-specific incidence rates indicate that KD 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).[51] In the United States, KD 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 are <5 years of age.[52]

Peripheral Arterial Disease

- PAD affects ≈8 million Americans and is associated with significant morbidity and mortality.[53] Prevalence increases dramatically with age, and PAD disproportionately affects blacks.[54]
- PAD affects 12% to 20% of Americans ≥65 years of age.[55] Despite its prevalence and cardiovascular risk implications, only ≈20% to 30% of patients with PAD are on recommended antiplatelet therapy and/or lipid-lowering therapy.[56]
- In the general population, only ≈10% of persons 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.[53,57] In an older, disabled population of women, however, as many as two thirds of individuals with PAD had no exertional leg symptoms.[58]
- Intermittent claudication is present in <1% of individuals <50 years of age and ≈≥5% of those >80 years of age.[35]
- In the FHS (NHLBI), the incidence of PAD was based on symptoms of intermittent claudication in subjects 29 to 62 years of age. Annual incidence of intermittent claudication per 10 000 subjects at risk increased from 6 in men and 3 in women between the ages of 30 and 44 years to 61 in men and 54 in women between the ages of 65 and 74 years. The incidence of intermittent claudication has declined since 1950, but survival among persons with intermittent claudication has remained low.[59]

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- The risk factors for PAD are similar but not identical to those for CHD. Diabetes and cigarette smoking are stronger risk factors for PAD than for CHD.[35] ORs for associations of diabetes 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.[60]
- A recent meta-analysis of 24 955 men and 23 339 women demonstrated that the association of the ankle brachial index (ABI) with mortality demonstrates a reverse J-shaped distribution in which participants with an ABI of 1.11 to 1.40 are at lowest risk for mortality.[61] Furthermore, an ABI <0.90 added meaningfully to the Framingham Risk Score 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 Framingham Risk Score category.[61]
- Among 508 patients (449 men) identified from two vascular laboratories in San Diego, California, a decline in ABI of more than 0.15 within a 10-year period was associated with an increased risk of all-cause mortality (RR=2.4) and CVD mortality (RR of 2.8) at 3 years' follow-up.[62]
- 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.[63] 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, 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 for soluble intercellular adhesion molecule-1, 12 years after soluble intercellular adhesion molecule-1 measurement.[64] Among individuals with PAD, higher levels of inflammatory biomarkers are associated with increased all-cause and cardiovascular mortality rate and increased risk of failure of lower-extremity revascularization procedures.[65,66]
- Persons with PAD have impaired function and quality of life. This is true even for persons who do not report leg symptoms. Furthermore, PAD patients, including those who are asymptomatic, experience a significant decline in lower-extremity functioning over time.[67,68]
- Pooled data from 11 studies in 6 countries found that PAD is a marker for systemic atherosclerotic disease. The age- and sex-adjusted relative risk of all-cause death was 2.35; for CVD mortality, 3.34; and for CHD fatal and nonfatal events combined, 2.13. The findings for stroke were slightly weaker but still significant, with a pooled relative risk of 1.86 for fatal and nonfatal events combined.[69]
- Data from NHANES 1999 to 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.[70]
- Results from NHANES 1999 to 2000 (NCHS) showed a remarkably high prevalence of PAD among patients with renal insufficiency.[71]
- Available evidence suggests that the prevalence of PAD in persons of Hispanic origin is similar to or slightly higher than that in non-Hispanic whites.[72,73]
- Recent studies indicate an association of elevated ankle-brachial index levels with increased risk of all-cause and cardiovascular death.[74,75]
- Among patients with established PAD, higher physical activity levels during daily life are associated with better overall survival rate, a lower risk of death from CVD, and slower rates of functional decline.[76,77]
- 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 diabetes 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.[78]
- A recent study of proteomic profiling identified that the protein β-2 microglobulin is elevated in patients with PAD. In unadjusted analyses of 20 men and women with PAD and 20 without PAD, β-2 microglobulin levels were highly correlated with the ankle-brachial index (r=0.727).[79]

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Table 9-1. Rheumatic Fever/Rheumatic Heart Disease

Population Group	Mortality, 2006 All Ages*	Hospital Discharges, 2006 All Ages
Both sexes	3257	59 000
Males	1008 (30.9%)†	22 000
Females	2249 (69.1%)†	36 000
White males	887	. . .
White females	2016	. . .
Black males	87	. . .
Black females	162	. . .

Ellipses (. . .) indicate that data are not available.

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

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

Sources: Mortality: NCHS; data represent underlying cause of death only. Hospital discharges: NHDS, NCHS, and NHLBI; data include those inpatients discharged alive, dead, or of unknown status.

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10. Risk Factor: Smoking/Tobacco Use

See Table 10-1 and Charts 10-1 and 10-2.

Prevalence

Youth

- In 2007, in grades 9 through 12, 21.3% of male students and 18.7% of female students reported current cigarette use, 19.4% of male students and 7.6% of female students reported current cigar use, and 13.4% of male students and 2.3% of female students reported current smokeless tobacco use. Overall, 30.3% of male students and 21% of female students reported any current tobacco use.[1]
- From 1980 to 2007, the percentage of high school seniors who reported smoking in the previous month decreased 29.2%. Smoking decreased by 13.8% in male students, 41.3% in female students, 18.7% in whites, and 57.9% in blacks.[2]
- Among youths 12 to 17 years of age in 2007, 3.1 million (12.4%) used a tobacco product in the past month, 2.5 million (9.8%) used cigarettes, and 1.1 million (4.2%) used cigars, which represents a decrease in use in all categories compared with 2006. Cigarette use in the past month declined from 13.0% in 2002 to 9.8% in 2007. Smokeless tobacco use in the past month was 2.4% in 2007, which was the same as in 2006 but persistently higher than any estimates since 2002 (2.0%).[3] The 2008 Monitoring the Future survey showed that ever use of cigarettes among 8th graders dropped from 44% in 1991 to 20.5% in 2008, whereas ever use of any tobacco dropped from 55.1% to 31.7% among 10th graders and from 63.1% to 44.7% among 12th graders.[4]
- Data from the YRBS[5] among high school students indicated that:
  - — The percentage of students ever trying cigarettes declined from 70.4% in 1999 to 50.3% in 2007.
  - — The percentage who smoked in the prior 30 days declined from 36.4% in 1997 to 20% in 2007.
  - — The percentage who smoked on ≥20 of the prior 30 days declined from 16.8% in 1999 to 8.1% in 2007.
  - — The percentage of current tobacco users (cigarettes, cigars, smokeless tobacco) declined from 43.5% in 1997 to 25.7% in 2007.
- Data from the YRBS of the CDC found that overall, 60.9% of students in grades 9 to 12 who ever smoked cigarettes daily tried to quit smoking cigarettes. The prevalence of this behavior did not vary by grade but was higher among female students (67.3%) than male students (55.5%) and higher among black students (68.1%) than Hispanic students (54.1%). No other differences were found by race/ethnicity. Overall, 12.2% of students who ever smoked cigarettes daily tried to quit smoking cigarettes and were successful.[6]

Abbreviations Used in Chapter 10

BRFSS	Behavioral Risk Factor Surveillance System
CDC	Centers for Disease Control and Prevention
CHD	coronary heart disease
CVD	cardiovascular disease
HD	heart disease
HF	heart failure
MEPS	Medical Expenditure Panel Survey
MI	myocardial infarction
NCHS	National Center for Health Statistics
NH	non-Hispanic
NHANES	National Health and Nutrition Examination Survey
NHIS	National Health Interview Survey
NHLBI	National Heart, Lung, and Blood Institute
SHS	secondhand smoke
YRBS	Youth Risk Behavior Surveillance

Adults

- From 1965 to 2007, smoking in the United States declined by 50.4% among people ≥18 years of age (NCHS).[2]
- In 2008, among Americans ≥18 years of age, 23.1% of men and 18.3% of women were cigarette smokers, putting them at increased risk of heart attack and stroke.[7]
- From 1998 to 2007, BRFSS data indicated that smoking prevalence decreased in 44 states, the District of Columbia, and Puerto Rico. Six states had no substantial changes in prevalence after controlling for age, sex, and race/ethnicity. However, only Utah and the US Virgin Islands met the Healthy People 2010 target for reducing adult smoking prevalence to 12%.[8]
- BRFSS/CDC 2008 data showed that among adults ≥18 years of age, the median percentage of current smokers among the states was 18.3%. The highest percentage was in West Virginia (26.5%), and the lowest was in Utah (9.3%).[9]
- Rates of use of any tobacco product among persons 12 years of age and older in 2006 were 31.4% for non-Hispanic whites only, 29.1% for non-Hispanic blacks only, 42.3% for non-Hispanic American Indians or Alaska Natives only, 16% for non-Hispanic Asians only, and 24.4% for Hispanics or Latinos of any race (NCHS).[2]
- In 2005 to 2007, Asian adults ≥18 years of age (men 17.2%, women 4.8%) were less likely to be current smokers than American Indian or Alaska Native adults (men 30.9%, women 24.3%), white adults (men 23.0%, women 18.8%), and black adults (men 25.1%, women 17.1%).[2]
- Among women 15 to 44 years of age, combined data for 2006 and 2007 indicated that the rate of past-month cigarette use was lower among those who were pregnant (16.4%) than it was among those who were not pregnant (28.4%). This pattern was evident among women 18 to 25 years of age (23.3% versus 33.9% for pregnant and nonpregnant women, respectively) and among women 26 to 44 years of age (11.6% versus 28.3%, respectively). However, among those 15 to 17 years of age, the rate of cigarette smoking for pregnant women was higher than for nonpregnant women (24.3% versus 16.0%, respectively).

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A similar pattern in cigarette smoking was observed in the combined 2004 to 2005 data, although the difference among those 15 to 17 years of age was not statistically significant in the data for those years.[3]

- Between 1965 and 2004 to 2005, the age-adjusted prevalence of noninstitutionalized women ≥65 years of age (age adjusted) who were current smokers increased from 8% in 1965 to 13% in the mid-1980s and then decreased back to 8% in 2004 to 2005. In 2004 to 2005, 28% of women and 49% of men ≥65 years of age (age adjusted) had previously smoked cigarettes.[10]
- According to 2004 to 2006 data, most Asian adults had never smoked, with rates ranging from 65% of Korean adults to 84% of Chinese adults. Korean adults (22%) were ≈2 to 3 times as likely to be current smokers as were Japanese (12%), Asian Indian (7%), or Chinese (7%) adults.[11]

Incidence

- In 2007, ≈2.2 million persons ≥12 years of age smoked cigarettes for the first time within the past 12 months. The 2007 estimate averages out to ≈6100 new cigarette smokers every day. Most new smokers (59.7%) in 2007 were <18 years of age when they first smoked cigarettes.[3]
- Data from 2002 to 2004 from the National Survey on Drug Use and Health suggest that ≈1 in 5 nonsmokers 12 to 17 years of age is likely to start smoking. Youths in 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 smoking between male and female youths in any of the major populations or subpopulations.[12]
- Approximately 80% of people who use tobacco began at <18 years of age, according to a report from the Surgeon General of the United States. The most common age of initiation is 14 to 15 years.[13]

Mortality

- During 2000 to 2004, cigarette smoking resulted in an estimated 443 000 premature deaths each year of smoking-related illnesses, and ≈49 000 of these deaths were from SHS. 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.[14]
- From 2000 to 2004, smoking during pregnancy resulted in an estimated 776 infant deaths annually.[14]
- Cigarette smoking kills an estimated 178 000 women in the United States annually. Of these, ≈40 000 deaths are due to HD.[15]
- On average, male smokers die 13.2 years earlier than male nonsmokers, and female smokers die 14.5 years earlier than female nonsmokers.[16]
- Current cigarette smoking is a powerful independent predictor of cardiac arrest in patients with CHD.[17]
- After up to 14.5 years of follow-up of participants in the Lung Health Study of the NHLBI, the all-cause death rate among participants in a smoking-cessation intervention was significantly lower (15%) than among those given usual care.[18]
- The CDC fact sheet on tobacco-related mortality[19] dated May 2009 stated that:
  - — Cigarette smoking results in a 2- to 3-fold increased risk of dying of CHD.
  - — On average, adults who smoke cigarettes die 13 to14 years earlier than nonsmokers.
  - — Cigarette smoking kills an estimated 259 500 men and 178 000 women in the United States each year.

Secondhand Smoke

- Data from the "Tobacco Use Supplement" to the "Current Population Survey" from 1992 to 2003 showed that the national prevalence of households with smoke-free–home rules increased from 43.2% during 1992–1993 to 72.2% in 2003. 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 SHS in the United States as of 1999–2002.[20]
- Analysis of data from NHANES found that the percentage of the US nonsmoking population ≥4 years of age with self-reported home SHS exposure declined from 20.9% in 1988–1994 to 10.2% in 1999–2004. The percentage of the nonsmoking population with detectable serum cotinine declined from 83.9% in 1988–1994 to 46.4% in 1999–2004. The percentage of nonsmokers with detectable serum cotinine decreased for all age groups during 1999–2004 and remained highest for those 4 to 11 years of age (60.5%) and those 12 to 19 years of age (55.4%) compared with those ≥20 years of age (42.2%). By 1999–2004, the gap increased between non-Hispanic blacks with detectable serum cotinine (70.5%) and non-Hispanic whites (43.0%) and Mexican Americans (40.0%). During both periods, prevalence of SHS exposure in the home was highest among non-Hispanic blacks and persons with lower incomes. For both periods, self-reported home SHS exposure was not significantly different in males than in females, but a higher percentage of males had detectable serum cotinine than did females.[21]
- Data from a 2006 report of the US Surgeon General on the consequences of involuntary exposure to tobacco smoke[22] indicate the following:
  - — Exposure among nonsmokers based on detectable levels of cotinine, a biomarker of SHS, fell from 88% in 1988–1991 to 43% in 2001–2002.
  - — Almost 60% of US children 3 to 11 years of age, or almost 22 million children, are exposed to SHS.

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— Nonsmokers who are exposed to SHS at home or at work increase their risk of developing CHD by 25% to 30%.
— Short exposures to SHS 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 acute MI.

- Healthcare costs associated with exposure to SHS average $10 billion annually.[23]

Aftermath

- Among ever-smokers who had 1 circulatory disorder, 52.1% were current smokers, and among those who reported that they had ≥3 circulatory disorders, 28% were current smokers at the time of the interview. The adjusted odds of being a current smoker were lower for individuals who had ever smoked in life and had ≥2 central circulatory disorders, such as MI, HF, or stroke, than for ever-smokers without a central circulatory disorder.[24]
- The CDC "Health Effects of Cigarette Smoking" fact sheet[25] provides the following information:

— Cigarette smokers are 2 to 4 times more likely to develop CHD than are nonsmokers.
— Cigarette smoking approximately doubles a person's risk for stroke.
— Cigarette smokers are >10 times as likely as nonsmokers to develop peripheral vascular disease.
— Smoking increases the risk of abdominal aortic aneurysm.

- According to data from the MEPS in the 2008 National Healthcare Quality Report, in 2005, 64.5% of smokers with routine office visits during the preceding year reported that they had been advised to quit, about the same percentage as in 2002 (63.5%). Smokers 18 to 44 years of age were less likely than the other age groups to be advised to quit smoking.[26]

Smokeless Tobacco

- In 2006, an estimated 8.2 million Americans ≥12 years of age (3.3%) used smokeless tobacco.[3]
- Data from the CDC fact sheet on smokeless (oral) tobacco,[27] based on the results of the 2005 National Survey on Drug Use and Health, indicate the following:

— Nationally, an estimated 3% of adults are current smokeless tobacco users. Approximately 6% of men and 0.4% of women use smokeless tobacco.
— Nine percent of American Indian/Alaska Natives, 4% of whites, 2% of blacks, 1% of Hispanics, and 0.6% of Asian American adults are current smokeless tobacco users.
— Eight percent of high school students are current smokeless tobacco users. Smokeless tobacco use is more common among male (13.6%) than female (2.2%) high school students. Estimates by race/ethnicity are 10.2% among whites, 5.1% for Hispanics, and 1.7% for blacks.
— An estimated 3% of middle school students are current smokeless tobacco users. Smokeless tobacco is more common among male (4%) than female (2%) middle school students. Estimates by race/ethnicity are 3% for white, 1% for Asian, 2% for black, and 4% for Hispanic middle school students.

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.[23]

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Table 10-1. Cigarette Smoking

Population Group	Prevalence, 2008 Age ≥18 y*	Cost[23]
Both sexes	46 000 000 (20.6%)	$193 Billion per year
Males	24 800 000 (23.1%)	. . .
Females	21 100 000 (18.3%)	. . .
White males	23.5%	. . .
White female	20.6%	. . .
Black males	25.6%	. . .
Black females	17.8%	. . .
Hispanic or Latino males	20.7%	. . .
Hispanic or Latino females	10.7%	. . .
NH Asian only (both sexes)	9.9%	. . .
NH American Indian/Alaska Native only (both sexes)	24.3%	. . .

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

*Data are provisional for 2008 for Americans ≥18 years of age; NHIS/NCHS.[7]

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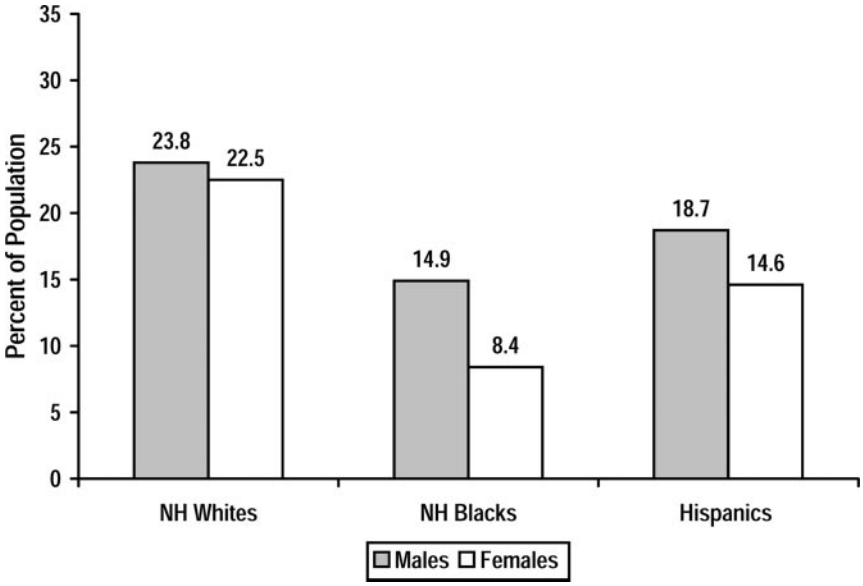

Chart 10-1. Prevalence of students in grades 9 to 12 reporting current cigarette use by sex and race/ethnicity (YRBS, 2007). Source: *MMWR: Morbidity and Mortality Weekly Report*.[1] NH indicates non-Hispanic.

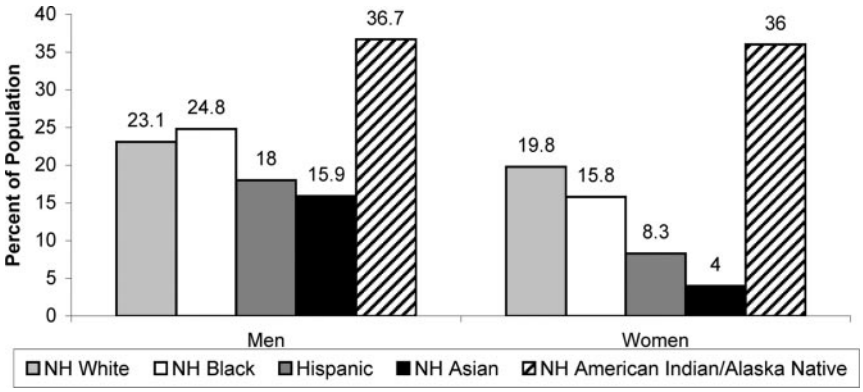

Chart 10-2. Prevalence of current smoking for adults ≥18 years of age by race/ethnicity and sex (NHIS: 2007). Source: *MMWR: Morbidity and Mortality Weekly Report*.[28] NH indicates non-Hispanic.

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11. Risk Factor: High Blood Cholesterol and Other Lipids

See Table 11-1 and Charts 11-1 through 11-3.

Prevalence

For information on dietary cholesterol, total fat, saturated fat, and other factors that affect blood cholesterol levels, see Chapter 17 (Nutrition).

Youth

- Among children 4 to 11 years of age, the mean total blood cholesterol level is 165.1 mg/dL. For boys, it is 164.6 mg/dL; for girls, it is 165.6 mg/dL. The racial/ethnic breakdown is as follows (NHANES 2003–2006, NCHS and NHLBI; unpublished analysis):
  - For non-Hispanic whites, 165.2 mg/dL for boys and 166.1 mg/dL for girls.
  - For non-Hispanic blacks, 165.6 mg/dL for boys and 164.9 mg/dL for girls.
  - For Mexican Americans, 161.7 mg/dL for boys and 163.1 mg/dL for girls.
- Among adolescents 12 to 19 years of age, the mean total blood cholesterol level is 161.1 mg/dL. For boys, it is 157.5 mg/dL; for girls, it is 164.8 mg/dL. The racial/ethnic breakdown is as follows (NHANES 2003–2006, NCHS and NHLBI; unpublished analysis):
  - For non-Hispanic whites, 155.8 mg/dL for boys and 166.3 mg/dL for girls.
  - For non-Hispanic blacks, 161.3 mg/dL for boys and 162.9 mg/dL for girls.
  - For Mexican Americans, 158.9 mg/dL for boys and 162.3 mg/dL for girls.
- Approximately 10.2% of adolescents 12 to 19 years of age have total cholesterol levels ≥200 mg/dL (NHANES 2003–2006, NCHS and NHLBI; unpublished analysis).

Adults

- An estimated 35 700 000 adults ≥20 years of age have total serum cholesterol levels ≥240 mg/dL (extrapolated to 2006 using NCHS/NHANES 2003–2006 data), with a prevalence of 16.2% (Table 11-1).[11]
- Data from the BRFSS study of the CDC in 2007 showed that the percentage of adults who had been screened for high blood cholesterol in the preceding 5 years ranged from 65.9% in Utah to 85% in the District of Columbia. The median percentage among states was 74.8%.[1]
- A 10% (population-wide) decrease in total cholesterol levels may result in an estimated 30% reduction in the incidence of CHD.[2]
- 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.[3]
- Between the periods 1988–1994 and 1999–2002 (NHANES/NCHS), the age-adjusted mean total serum cholesterol level of adults ≥20 years of age decreased from 206 to 203 mg/dL, HDL levels increased from 50.7 to 51.3 mg/dL, and LDL cholesterol levels decreased from 129 to 123 mg/dL.[4]
- Data from NHANES 2003–2006 (NCHS) showed the serum total crude mean cholesterol level in adults ≥20 years of age was 198 mg/dL for men and 202 mg/dL for women.[5]
- 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 in 1980–1982 to 5.16 and 5.09 mmol/L, respectively, in 2000–2002; 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 between 35 and 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.[6]
- Data from the BRFSS (CDC) survey in 2007 showed that among adults screened for high blood cholesterol, the percentage who had been told that they had high blood cholesterol ranged from 32.4% in Minnesota to 42.4% in West Virginia. The median percentage among states was 37.6%.[7]
- According to data from NHANES 2005–2006, between the periods 1999–2000 and 2005–2006, 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 2005–2006, approximately 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.[8]

Abbreviations Used in Chapter 11

BRFSS	Behavioral Risk Factor Surveillance System
CDC	Centers for Disease Control and Prevention
CHD	coronary heart disease
HD	heart disease
HDL	high-density lipoprotein
LDL	low-density lipoprotein
mg/dL	milligrams per deciliter
mmol/L	millimoles per liter
NCHS	National Center for Health Statistics
NHANES	National Health and Nutrition Examination Survey
NHLBI	National Heart, Lung, and Blood Institute

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Adherence

- On the basis of data from the Third Report of the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults[9]:
  - — Fewer than half of persons who qualify for any kind of lipid-modifying treatment for CHD risk reduction are receiving it.
  - — Fewer than half of even the highest-risk persons (those with symptomatic CHD) are receiving lipid-lowering treatment.
  - — Only about one third of treated patients are achieving their LDL goal; fewer than 20% of CHD patients are at their LDL goal.

Lipid Levels

LDL (Bad) Cholesterol

Youth

- Among adolescents 12 to 19 years of age, the mean LDL cholesterol level is 89.2 mg/dL. For boys, it is 87.5 mg/dL, and for girls, it is 90.9 mg/dL. The racial/ethnic breakdown is as follows (NHANES 2003–2006, NCHS and NHLBI; unpublished analysis):
  - — Among non-Hispanic whites, 87.1 mg/dL for boys and 91.5 mg/dL for girls.
  - — Among non-Hispanic blacks, 89.0 mg/dL for boys and 91.5 mg/dL for girls.
  - — Among Mexican Americans, 88.7 mg/dL for boys and 91.6 mg/dL for girls.

Adults

- The mean level of LDL cholesterol for American adults ≥20 years of age is 115.0 mg/dL (NHANES 2003–2006, NCHS and NHLBI; unpublished analysis). 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 2003–2006 (NCHS and NHLBI; unpublished data):
  - — Among non-Hispanic whites, mean LDL cholesterol levels were 115.6 mg/dL for men and 116.6 mg/dL for women.
  - — Among non-Hispanic blacks, mean LDL cholesterol levels were 115.1 mg/dL for men and 111.6 mg/dL for women.
  - — Among Mexican Americans, mean LDL cholesterol levels were 123.4 mg/dL for men and 111.6 mg/dL for women.
- The age-adjusted prevalence of high LDL cholesterol in US adults was 26.6% in 1988–1994 and 25.3% in 1999–2004 (NHANES/NCHS). Between 1988–1994 and 1999–2004, 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 cholesterol. In 1999–2004, 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 males than among females (22.6% versus 26.9%, respectively).[10]

HDL (Good) Cholesterol

Youth

- Among children 4 to 11 years of age, the mean HDL cholesterol level is 55.7 mg/dL. For boys, it is 56.7 mg/dL, and for girls, it is 54.7 mg/dL. The racial/ethnic breakdown is as follows (NHANES 2003–2006, NCHS and NHLBI; unpublished analysis):
  - — Among non-Hispanic whites, 55.9 mg/dL for boys and 54.0 mg/dL for girls.
  - — Among non-Hispanic blacks, 60.9 mg/dL for boys and 58.0 mg/dL for girls.
  - — Among Mexican Americans, 54.5 mg/dL for boys and 52.9 mg/dL for girls.
- Among adolescents 12 to 19 years of age, the mean HDL cholesterol level is 52.4 mg/dL. For boys, it is 49.4 mg/dL, and for girls, it is 55.6 mg/dL. The racial/ethnic breakdown is as follows (NHANES 2003–2006, NCHS and NHLBI; unpublished analysis):
  - — Among non-Hispanic whites, 47.6 mg/dL for boys and 55.2 mg/dL for girls.
  - — Among non-Hispanic blacks, 54.8 mg/dL for boys and 57.7 mg/dL for girls.
  - — Among Mexican Americans, 49.6 mg/dL for boys and 53.8 mg/dL for girls.

Adults

- An HDL cholesterol level below 40 mg/dL in adults 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 54.3 mg/dL (NHANES 2003–2006, NCHS and NHLBI; unpublished analysis).
- According to NHANES 2003–2006 (NCHS and NHLBI; unpublished analysis):
  - — Among non-Hispanic whites, mean HDL cholesterol levels were 48.3 mg/dL for men and 60.1 mg/dL for women.
  - — Among non-Hispanic blacks, mean HDL cholesterol levels were 52.4 mg/dL for men and 61.3 mg/dL for women.
  - — Among Mexican Americans, mean HDL cholesterol levels were 47.1 mg/dL for men and 55.4 mg/dL for women.

Triglycerides

Youth

- Among adolescents 12 to 19 years of age, the mean triglyceride level is 92.4 mg/dL. For boys, it is 92.4 mg/dL, and for girls, it is 92.4 mg/dL. The racial/ethnic breakdown

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is as follows (NHANES 2003–2006, NCHS and NHLBI; unpublished analysis):

— Among non-Hispanic whites, 98.1 mg/dL for boys and 95.4 mg/dL for girls.
— Among non-Hispanic blacks, 69.7 mg/dL for boys and 68.8 mg/dL for girls.
— Among Mexican Americans, 93.1 mg/dL for boys and 97.3 mg/dL for girls.

Adults

- A 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 144.2 mg/dL (NHANES 2003–2006, NCHS and NHLBI; unpublished analysis).

  — Among men, the mean triglyceride level is 156.5 mg/dL (NHANES 2003–2006, NCHS and NHLBI; unpublished analysis).

    ○ 158.3 mg/dL for white men.
    ○ 126.5 mg/dL for black men.
    ○ 172.5 mg/dL for Mexican American men.

  — Among women, the mean triglyceride level is 132.1 mg/dL.

    ○ 135.0 mg/dL for white women.
    ○ 102.4 mg/dL for black women.
    ○ 152.2 mg/dL for Mexican American women.

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Table 11-1. High Total and LDL Cholesterol and Low HDL Cholesterol

Population Group	Prevalence of Total Cholesterol ≥200 mg/dL, 2006 Age ≥20 y	Prevalence of Total Cholesterol ≥240 mg/dL, 2006 Age ≥20 y	Prevalence of LDL Cholesterol ≥130 mg/dL, 2006 Age ≥20 y	Prevalence of HDL Cholesterol <40 mg/dL, 2006 Age ≥20 y
Both sexes*	102 200 000 (46.8%)	35 700 000 (16.2%)	71 200 000 (32.6%)	35 100 000 (16.2%)
Males*	47 700 000 (45.2%)	15 900 000 (15.0%)	34 900 000 (33.1%)	26 400 000 (25.0%)
Females*	54 500 000 (47.9%)	19 700 000 (17.2%)	36 300 000 (32.0%)	8 700 000 (7.9%)
NH white males, %	45.0	15.3	31.5	25.4
NH white females, %	48.7	18.1	33.8	7.9
NH black males, %	40.2	10.9	34.4	14.7
NH black females, %	41.8	13.1	28.6	6.5
Mexican-American males, %	51.1	16.8	42.7	29.3
Mexican-American females, %	49.0	14.3	30.4	11.7
Total Hispanics† ≥20 y of age, %	. . .	29.9	. . .	. . .
Total Asian/Pacific Islanders† ≥20 y of age, %	. . .	29.2	. . .	. . .
Total American Indians/Alaska Natives† ≥20 y of age, %	. . .	31.2	. . .	. . .

Ellipses (. . .) indicate data not available; 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 years of age. Data for LDL cholesterol, HDL cholesterol, and all racial/ethnic groups are age adjusted for age ≥20 years.

†BRFSS (1991–2003, CDC), *MMWR*[11]; data are self-reported data for Americans ≥20 years of age.

Source for total cholesterol ≥200 mg/dL, ≥240 mg/dL, LDL, and HDL: NHANES (2003–2006), NCHS, and NHLBI. Estimates from NHANES 2003–2006 (NCHS) applied to 2006 population estimates.

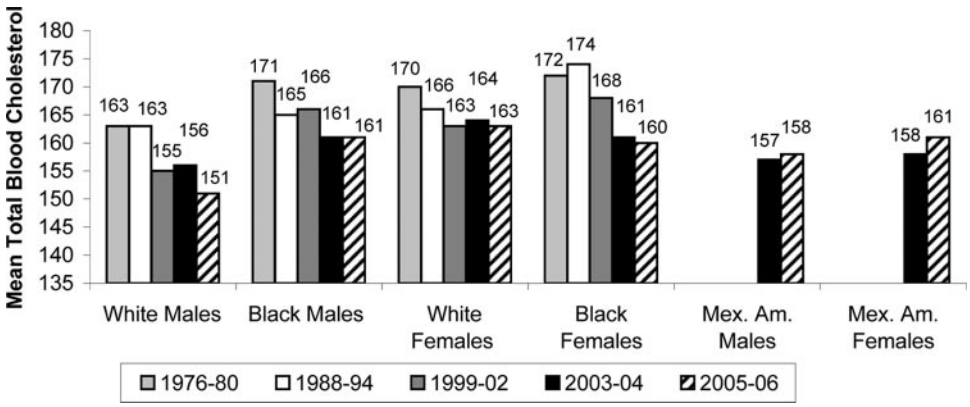

Chart 11-1. Trends in mean total serum cholesterol among adolescents 12 to 17 years of age by race, sex, and survey (NHANES: 1976–1980, 1988–1994, 1999–2002, 2003–2004, and 2005–2006). Source: NCHS and NHLBI. Mex. Am. indicates Mexican American.

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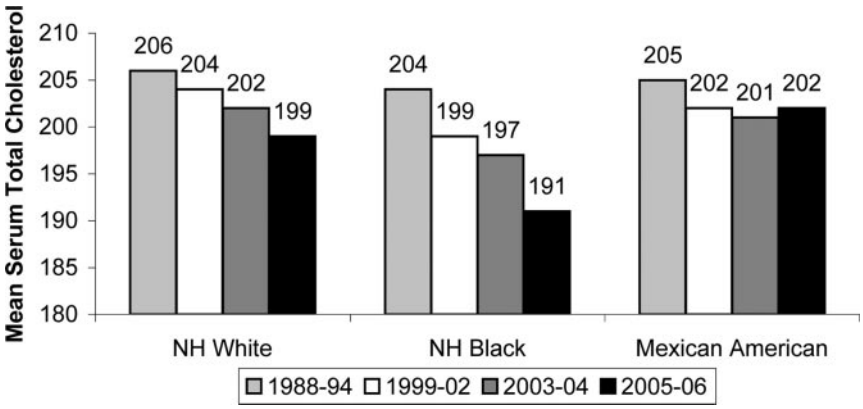

Chart 11-2. Trends in mean total serum cholesterol among adults by race and survey (NHANES: 1988–1994, 1999–2002, 2003–2004, and 2005–2006). Source: NCHS and NHLBI. NH indicates non-Hispanic.

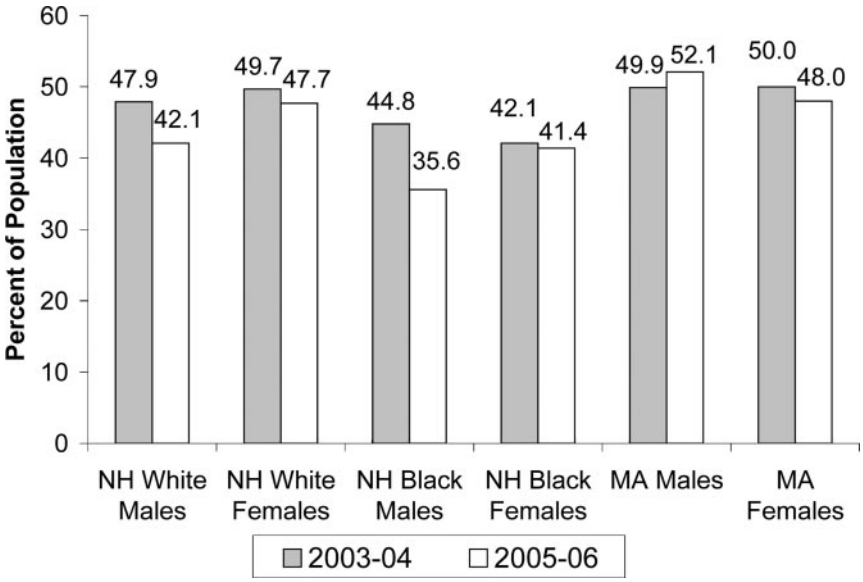

Chart 11-3. Trends in the prevalence of total serum cholesterol (≥200 mg/dL) in adults ≥20 years of age by sex and race/ethnicity (NHANES 2003–2004 and 2005–2006). NH indicates non-Hispanic; MA, Mexican American.

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12. Risk Factor: Physical Inactivity

See Table 12-1 and Charts 12-1 through 12-4.

Prevalence

Youth

Inactivity

- The proportion of adolescents who report engaging in no regular PA is high, and the proportion increases with age:
  - — In the 2007 YRBS of adolescents in grades 9 through 12, 31.8% of females and 18% of males had not engaged in 60 minutes of moderate-to-vigorous physical activity (MVPA), defined as any activity that increased heart rate or breathing rate, even once in the previous 7 days.[1]
  - — By the age of 16 or 17 years, 31% of white girls and 56% of black girls reported no habitual leisure-time PA.[2]
  - — Rates of inactivity were highest among black (42.1%) and Hispanic (35.2%) females compared with white females (28.2%).[1]
  - — Among males, blacks were also the least likely to engage in MVPA 5 or more days per week (21.8%), followed by Hispanic (18.8%) and white (16.7%) males.[1]
- More than one fourth of all adolescents in grades 9 through 12 reported spending ≥3 hours per day using computers outside of school time (24.9%) or watching television (35.4%) in the 2007 YRBS.[1]
- The proportion of males who spent >3 hours using computers (29.1%) or watching television (37.5%) was higher than that of females (computers 20.6% and television 33.2%).[1]
- A greater proportion of black and Hispanic students than white students used computers or watched television >3 hours per day.[1]

Abbreviations Used in Chapter 12

BMI	body mass index
BRFSS	Behavior Risk Factor Surveillance System
CDC	Centers for Disease Control and Prevention
CHD	coronary heart disease
CI	confidence interval
CVD	cardiovascular disease
DM	diabetes mellitus
HBP	high blood pressure
HD	heart disease
HDL	high-density lipoprotein
HF	heart failure
HR	hazard ratio
MEPS	Medical Expenditure Panel Survey
MI	myocardial infarction
MVPA	Moderate-to-vigorous physical activity
mm Hg	millimeters of mercury
mmol/L	millimoles per liter
NHANES	National Health and Nutrition Examination Survey
NHIS	National Health Interview Survey
PA	physical activity
RR	relative risk
WHO	World Health Organization
YMCLS	Youth Media Campaign Longitudinal Study
YRBS	Youth Risk Behavior Surveillance

Self-Reported and Measured Activity

- There is a marked discrepancy between the proportion of youth who report meeting PA guidelines (≥60 minutes of MVPA on most days of the week) and those who met guidelines when activity was measured objectively with accelerometers (portable motion censors that record and quantify movements) in the NHANES 2003–2004 survey[3]:
  - — In the 2007 YRBS, 34.7% of students in grades 9 through 12 reported that they met current recommendations for activity. The proportion was higher in males (43.7%) than in females (25.6%).[1]
  - — The proportion of students meeting recommendations declined from 9th (38.1%) to 12th (29.5%) grades, and the proportion was again higher in males than in females.[1]
  - — Forty-two percent of 6- to 11-year-olds accumulated ≥60 minutes of MVPA (based on counts per minute >2020 with an accelerometer) 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 met activity guidelines.[3]
  - — More boys than girls met recommendations as measured by accelerometry.[3]

Correlates of Activity Behaviors

- Lower levels of parental education are associated with greater decline in PA for white girls at both younger and older ages. For black girls, this association is seen only at older ages.[2]
- Cigarette smoking is associated with lower levels of PA among white girls.[2]
- Pregnancy is associated with a lower level of PA among black girls but not among white girls.[2]
- A higher BMI is associated with lower levels of PA.[1]
  - — Sixty percent of students reported engaging in PA to lose weight or keep from gaining weight in the 30 days before the 2007 YRBS.
  - — Females were more likely to report exercising to avoid weight gain (67.0%) than were males (55%).
  - — White (71.5%) and Hispanic (66.4%) females were more likely to report exercising for weight control than were black females (50.7%).

Organized Activities

- Physical education class participation declined from the 9th through the 12th grades among males and females.[1]

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— A total of 30.3% of students attended physical education classes in school daily (33.2% of males and 27.3% of females).

- More than half (56.3%) 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 59.2% in the 9th grade to 49% in the 12th grade.[1]
- Among children 9 to 13 years of age, 61.5% do not participate in any organized PA during nonschool hours.[4]

— A total of 22.6% do not engage in any free-time PA, according to 2002 data from the Youth Media Campaign Longitudinal Study (YMCLS) of the CDC.
— Non-Hispanic black and Hispanic children are significantly less likely than non-Hispanic white children to report involvement in organized activities, as are children whose parents have lower incomes and education levels.

Adults

Inactivity

- The age-adjusted proportion of adults who reported engaging in no MVPA in leisure time, as part of their occupation, or for transportation was 10.3% in 2005[5]:

— Inactivity in 2005 was higher among females (12%) than males (8.4%) and increased with age from 5.5% to 6.1%, 10.2%, and 24% among adults 18 to 24, 25 to 44, 45 to 64, and ≥65 years of age, respectively.

- A total of 59% of adults who responded to the 2008 NHIS survey reported no vigorous activity (activity that causes heavy sweating and a large increase in breathing or heart rate).[6]

— Women (63.8%) were more likely than men (52.8%) to report never engaging in vigorous PA, and the proportion of respondents who did not participate in any vigorous activity increased with age from 49.4% in 18- to 44-year-olds to 86.0% in adults ≥75 years of age.[6]

Self-Reported and Measured Activity

- The proportion of adults reporting that they meet current guidelines for regular PA of at least 30 minutes of moderate PA 5 or more days per week or >20 minutes of vigorous activity on 3 or more days per week has declined over time.[7]

— According to 2007 BRFSS/CDC data, 64.5% of adults met PA guidelines (68.9% of men and 60.4% of women).[8]
— Nearly three quarters (74%) of 18- to 24-year-olds reported being active, whereas only half (51.2%) of adults ≥65 years of age were active.[9]

- Adherence to PA recommendations was much lower when based on PA measured by accelerometer in NHANES 2003–2004[3]:

— Of those 20 to 59 years of age, 3.8% of males and 3.2% of females met recommendations to engage in MVPA (accelerometer counts >2020/min) for 30 minutes (in sessions of ≥10 minutes) on ≥5 of 7 days.
— Among persons ≥60 years of age, adherence was 2.5% in males and 2.3% in females.

Correlates of Activity

- The proportion of adults reporting 30 minutes of moderate activity 5 times per week in the 2008 NHIS was positively associated with education level; 45.4% of persons with a college degree or higher were regularly active compared with 17.2% of adults with a high school education or less in 2008.[6]
- Data from the 2008 NHIS show that non-Hispanic black and Hispanic adults were more likely to report inactivity (47.7% and 47.5%, respectively) than were non-Hispanic white adults (32.1%; "inactive" refers to no sessions of light/moderate PA of at least 10 minutes' duration).[6]

— American Indians (67.5%) and blacks (66.2%) were more likely than white respondents (57.2%) to report not engaging in vigorous activity. Asians (59.9%) and Native Hawaiians or other Pacific Islanders (60.5%) were as likely as white respondents to report not engaging in vigorous activity.
— Hispanic or Latino adults were more likely not to engage in vigorous activity (69.1%) than non-Hispanic or non-Latino adults (56.7%).
— A total of 80.0%, 69.3%, 57.7%, and 42.8% of respondents with less than a high school education, a high school diploma, some college, or a bachelor's degree or higher, respectively, did not report engaging in any vigorous PA.[6]

Physical Inactivity and CHD

Activity and CHD Risk Factors

- In the Diabetes Prevention Project randomized trial, intensive lifestyle modification, which included dietary modification and a goal of 150 minutes of PA per week, was associated with a lower rate of diabetes over 3.9 years (4.8 cases per 100 person-years) than metformin use (7.8 per 100 person-years) or placebo (11 per 100 person-years); these findings persisted after adjustment for known risk factors.[10]
- In a Cochrane review of 2 studies of exercise-only interventions (as opposed to exercise plus diet) for the prevention of type 2 DM, exercise therapy showed a trend toward protection, but the findings were not statistically significant (RR 0.69, 95% CI 0.29 to 1.65).[11]
- According to the "Physical Activity for Everyone" guidelines, up to 150 minutes per week of moderate-intensity aerobic activity, 75 minutes of vigorous-intensity activity, or an equivalent mix of the 2 is important for weight maintenance.[12]
- As a weight-loss intervention, exercise alone was associated with significant reductions in diastolic blood pressure

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(−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).[13]

- One hundred twenty to 150 minutes per week of moderate-intensity activity can reduce the risk of development of metabolic syndrome and its individual components (eg, abdominal adiposity, HBP, low HDL cholesterol, high triglycerides, or high glucose).[12]

Inactivity and CVD (Stroke and CHD)

- The RR of CHD associated with physical inactivity ranges from 1.5 to 2.4, an increase in risk comparable to that observed for high blood cholesterol, HBP, or cigarette smoking.[14]
- Physical inactivity is responsible for 12.2% of the global burden of MI after accounting for other CVD risk factors such as cigarette smoking, diabetes, hypertension, abdominal obesity, lipid profile, no alcohol intake, and psychosocial factors.[15]
- A 2.3% decline in physical inactivity between 1980 and 2000 prevented or postponed ≈17 445 deaths (≈5%) due to CHD in the United States.[16]
- 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 to 0.91) and moderate (RR 0.91, 95% CI 0.80 to 1.05) levels of activity were inversely associated with the likelihood of developing total stroke (ischemic and hemorrhagic).[17]

Secondary Prevention

- Data from the 2003 BRFSS (CDC) found that 53.2% of respondents with HD were told to be more physically active, 32% met recommended PA levels, and 30.8% were sedentary.[18]
- Analysis of 2005 and 2007 data from the BRFSS study of the CDC found that during these 2 years combined, doctor-diagnosed arthritis affected 57.4% of adults with HD (heart attack, angina, or CHD) compared with 27.4% of adults in the general population. In this group, the adjusted likelihood of PA was 30% greater than that of persons with HD but without arthritis (adjusted for age, sex, race/ethnicity, education level, and BMI).[19]
- PA improves inflammatory markers in persons with existing stable CHD. After a 6-week training session, C-reactive protein levels declined by 23.7% ($P<0.001$), and plasma vascular 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.[20]
- In a randomized trial of patients with peripheral arterial disease, 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 flow-mediated dilation, whereas resistance training was associated with stair-climbing ability versus control.[21]
- The benefit of intense exercise training for cardiac rehabilitation in persons 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 ejection fraction 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.[22]

Primary Prevention

- The "Physical Activity for Everyone" PA guidelines for adults cite evidence that getting ≈150 minutes per week of moderate-intensity aerobic activity can reduce the risk of CVD.[12]
- 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.[23]
- 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 to 39.3, 35.9, 32.2, and 25.8 according to quintiles of activity. The adjusted HR comparing the uppermost quintile of activity with the lowest was 0.72 (95% CI 0.61 to 0.85).[24]

Economic Consequences of Inactivity

- The economic consequences of physical inactivity are substantial. In a summary of WHO 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.[25]
- 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.[26]
- Total costs are even higher when the expenses attributable to obesity, a primary consequence of inactivity, are taken into account. In 1995, 9.4% of total US health expenditures were attributable to physical inactivity and obesity combined.[27]

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Table 12-1. Regular Leisure-Time PA

Population Group	Prevalence, 2008 (Age ≥18 y), %
Both sexes	32.5
Males	34.8
Females	30.6
NH white only	35.9
NH black only	24.8
Hispanic or Latino	25.2

NH indicates non-Hispanic.

Regular leisure-time PA is defined as light to moderate activity for ≥30 minutes, 5 times per week, or vigorous activity for ≥20 minutes, ≥3 times per week.

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

Source: NHIS 2008 (NCHS).[6]

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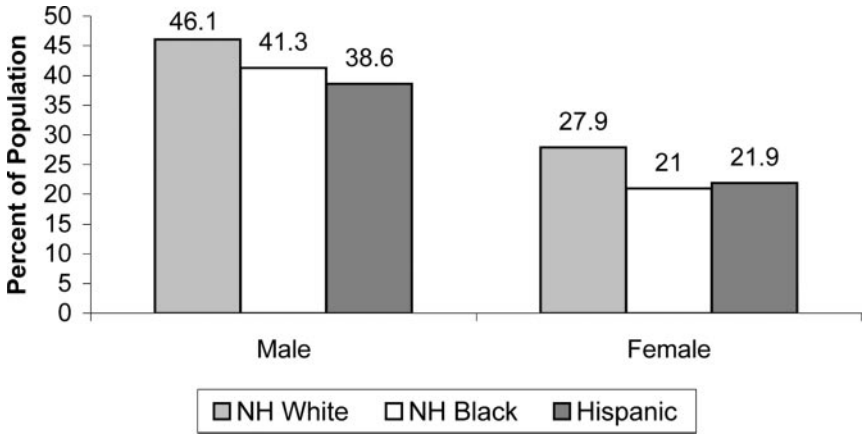

Chart 12-1. Prevalence of students in grades 9 through 12 who met currently recommended levels of PA during the past 7 days by race/ethnicity and sex (YRBS: 2007). *Currently recommended levels* is defined as activity that increased their heart rate and made them breathe hard some of the time for a total of at least 60 minutes per day on ≥5 of the 7 days preceding the survey. Source: *MMWR Surveillance Summaries*.[1] NH indicates non-Hispanic.

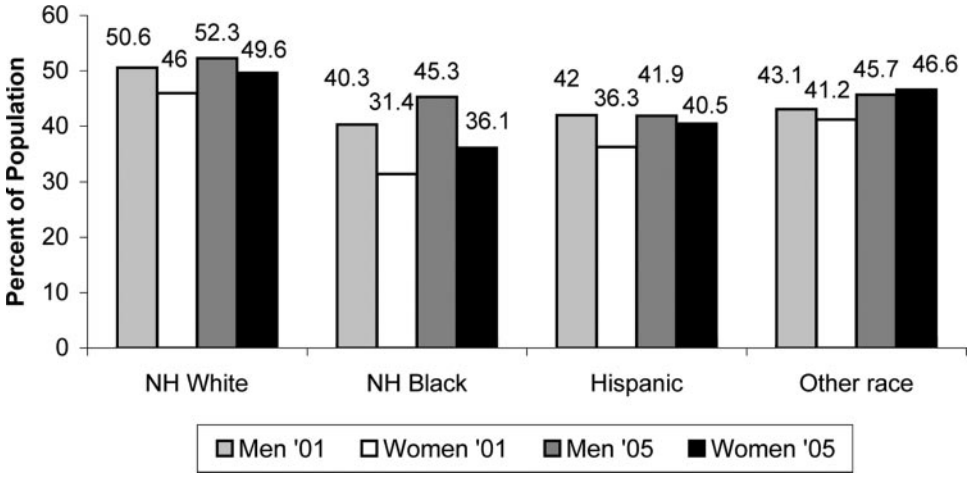

Chart 12-2. Prevalence of regular PA among adults ≥18 years of age by race/ethnicity and sex (BRFSS: 2001 and 2005). Source: Pleis et al.[28] NH indicates non-Hispanic.

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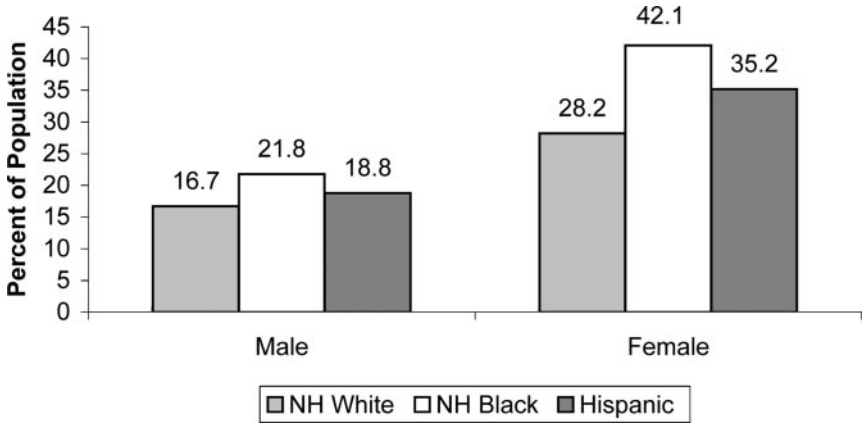

Chart 12-3. Prevalence of students in grades 9 through 12 who did not meet currently recommended levels of moderate-to-vigorous PA during the past 7 days by race/ethnicity and sex (YRBS: 2007). Source: *MMWR Surveillance Summaries*.[1] *Currently recommended levels* is defined as activity that increased 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.

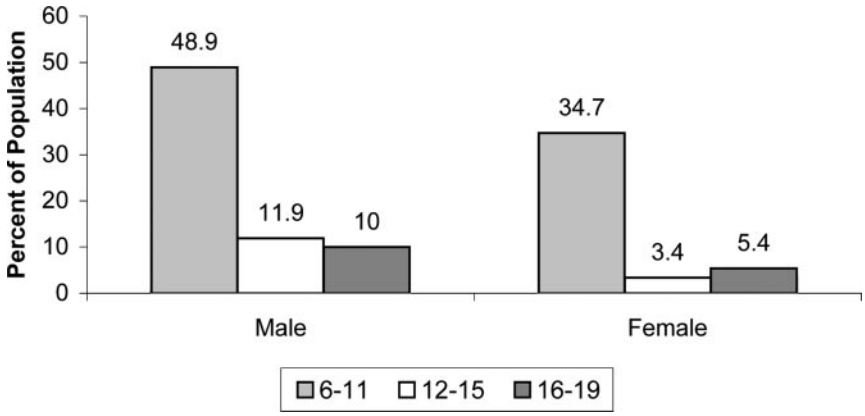

Chart 12-4. Prevalence of children 6 to 19 years of age who attained sufficient MVPA to meet public health recommendations (≥60 minutes per day on 5 or more of the 7 days preceding the survey), by sex and age (NHANES 2003–2004). Source: Troiano et al.[3]

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13. Risk Factor: Overweight and Obesity

See Table 13-1 and Charts 13-1 through 13-3.

Prevalence

Youth

- On the basis of 2003 to 2006 data from NHANES (NCHS), the prevalence of overweight and obesity in children 2 to 5 years of age, based on a BMI-for-age value at or above the 85th percentile of the 2000 CDC growth charts, was 25.4% for non-Hispanic white boys and 20.9% for non-Hispanic white girls, 23.2% for non-Hispanic black boys and 26.4% for non-Hispanic black girls, and 32.4% for Mexican American boys and 27.3% for Mexican American girls. In children 6 to 11 years of age, the prevalence was 31.7% for non-Hispanic white boys and 31.5% for non-Hispanic white girls, 33.8% for non-Hispanic black boys and 40.1% for non-Hispanic black girls, and 47.1% for Mexican American boys and 38.1% for Mexican American girls. In children 12 to 19 years of age, the prevalence was 34.5% for non-Hispanic white boys and 31.7% for non-Hispanic white girls, 32.1% for non-Hispanic black boys and 44.5% for non-Hispanic black girls, and 40.5% for Mexican American boys and 37.1% for Mexican American girls.[1]
- On the basis of 2003 to 2006 data from NHANES (NCHS), the prevalence of obesity in children 2 to 5 years of age, based on BMI-for-age values at or above the 95th percentile of the 2000 CDC growth charts, was 11.1% for non-Hispanic white boys and 10.2% for non-Hispanic white girls, 13.3% for non-Hispanic black boys and 16.6% for non-Hispanic black girls, and 18.8% for Mexican American boys and 14.5% for Mexican American girls. In children 6 to 11 years of age, the prevalence was 15.5% for non-Hispanic white boys and 14.4% for non-Hispanic white girls, 18.6% for non-Hispanic black boys and 24.0% for non-Hispanic black girls, and 27.5% for Mexican American boys and 19.7% for Mexican American girls. In children 12 to 19 years of age, the prevalence was 17.3% for non-Hispanic white boys and 14.5% for non-Hispanic white girls, 18.5% for non-Hispanic black boys and 27.7% for non-Hispanic black girls, and 22.1% for Mexican American boys and 19.9% for Mexican American girls.[1]
- Nearly 10 million children and adolescents 6 to 19 years of age have BMI-for-age values at or above the 95th percentile of the 2000 CDC growth charts for the United States (NHANES [2003–2006], NCHS).[1]
- On the basis of data from NHANES (NCHS), the prevalence of BMI-for-age values at or above the 95th percentile of the 2000 CDC growth charts in children 6 to 11 years of age increased from 4.0% in 1971 to 1974 to 17.0.% in 2003 to 2006. The prevalence of BMI-for-age values at or above the 95th percentile in adolescents 12 to 19 years of age increased from 6.1% to 17.6% in that same time frame.[1,2]
- Among infants and children between the ages of 6 and 23 months, the prevalence of high weight for age was 7.2% in 1976 to 1980 and 11.5% in 2003 to 2006 (NHANES, NCHS).[3]
- Data from the NHANES of the NCHS found that just over 12% of preschool children 2 to 5 years of age were overweight in 2003 to 2006.[1]
  - — Among preschool children, the following were overweight: 10.7% of non-Hispanic whites, 14.9% of non-Hispanic blacks, and 16.7% of Mexican Americans.
  - — Among children 6 to 11 years of age, the following were overweight: 15.0% of non-Hispanic whites, 21.3% of non-Hispanic blacks, and 23.8% of Mexican Americans.
  - — Among adolescents 12 to 19 years of age, the following were overweight: 16.0% of non-Hispanic whites, 22.9% of non-Hispanic blacks, and 21.1% of Mexican Americans.
- Data from NHANES 2003 to 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 at or above the 95th percentile, and 31.9% were at or above the 85th percentile.[1]
- Overweight adolescents have a 70% chance of becoming overweight adults. This increases to 80% if 1 or both parents are overweight or obese.[4]
- Data from the CDC's YRBS 2007 survey showed that the prevalence of being overweight (≥85th and <95th percentile of the 2000 BMI-for-age growth chart) was higher among non-Hispanic black (19.0%) and Hispanic (18.1%) students than among non-Hispanic white students (14.3%), higher among non-Hispanic black female (21.4%) and Hispanic female (17.9%) than non-Hispanic white female (12.8%) students, and higher among non-Hispanic black male (16.6%) and Hispanic male (18.3%) than non-

Abbreviations Used in Chapter 13

BMI	body mass index
BRFSS	Behavioral Risk Factor Surveillance System
CDC	Centers for Disease Control and Prevention
CI	confidence interval
CVD	cardiovascular disease
DM	diabetes mellitus
FHS	Framingham Heart Study
HHP	Honolulu Heart Program
kg/m^2	kilograms per square meter
MESA	Multiethnic Study of Atherosclerosis
NCHS	National Center for Health Statistics
NH	non-Hispanic
NHANES	National Health and Nutrition Examination Survey
NHIS	National Health Interview Survey
NHLBI	National Heart, Lung, and Blood Institute
NINDS	National Institute of Neurological Diseases and Stroke
NOMAS	Northern Manhattan Study
OR	odds ratio
WHO	World Health Organization
YRBS	Youth Risk Behavior Surveillance

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Hispanic white male (15.7%) students. The prevalence of being obese (≥95th percentile of the 2000 BMI-for-age growth chart) was higher among non-Hispanic black (18.3%) and Hispanic (16.6%) students than among non-Hispanic white students (10.8%), higher among non-Hispanic black female (17.8%) than non-Hispanic white female (6.8%) and Hispanic female (12.7%) students, and higher among Hispanic male (20.3%) and non-Hispanic black male (18.9%) than non-Hispanic white male (14.6%) students.[5]

- Data from the NHANES in the 2008 National Healthcare Quality Report found that:
  - 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 old (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.[6]
- A study of more than 8500 4-year-olds in the Early Childhood Longitudinal Study, Birth Cohort (National Center for Education Statistics), found that 1 in 5 are 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, based on 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.[7]
- Overweight adolescents have a 70% chance of becoming overweight adults. This increases to 80% if 1 or both parents are overweight or obese.[4]
- Childhood sociodemographic factors may contribute to gender disparities in obesity prevalence. A study of data from the National Longitudinal Study of Adolescent Health found that parental education consistently modified gender disparity in blacks. The gender 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 gender difference in obesity prevalence.[8]

Adults

- According to 2008 data from the BRFSS/CDC survey, based on self-reported height and weight, the prevalence of obesity ranged from 19.1% in Colorado to 33.3% in Mississippi. The median percentage by state was 26.6%. Additionally, no state met the Healthy People 2010 goal of reducing obesity to 15% of adults.[9]
- Data from NHANES 2005 to 2006, based on measured weight and height, found that 34% of US adults were obese (33.3% of men and 35.3% of women). Non-Hispanic black and Mexican-American women were more likely to be obese than non-Hispanic white women.[10]
- 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.[11]
- Analysis of data (FHS, NHLBI) showed that overweight and obesity were associated with large decreases in life expectancy. 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. In 40-year-old nonsmokers, females lost 7.1 years and males 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.[12]
- Data from the 2008 NHIS showed that blacks ≥18 years of age (29.3%), American Indians or Alaska Natives (29.2%), and whites (36.9%) were less likely than Asians (54.5%) to be at a healthy weight.[13]
- Data from the 2008 NHIS, based on self-reported weights and heights, showed that blacks ≥18 years of age (36.1%) and American Indians or Alaska Natives (42.1%) were more likely to be obese than were whites (26.5%), and Asians (9.4%)[13]
- The WHO 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.[14]
- In NHANES 2001 to 2002 (NCHS), racial disparities were observed among women but not among men: 68.6% of black women were overweight or obese, compared with 56.0% of white women and 54.5% of Hispanic women. Race-based differences in obesity were more pronounced among women: 41.5% of black women were obese, compared with 19.3% of white women and 26.2% of Hispanic women.[15]
- 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.[16]
- 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.[6]
- Data from the 2003 to 2006 NHANES in the 2008 National Healthcare Disparities Report found that approximately 64.8% of obese adults were told by a doctor or health professional that they were overweight.[17]
- The proportion of obese adults told that they were overweight was significantly lower for non-Hispanic blacks

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(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%).[17] Analysis of data from the MESA study found that 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%). These findings may be indicators of potential future increases in vascular disease burden and healthcare costs associated with the obesity epidemic.[18]

Trends

Youth

- On the basis of data from NHANES (NCHS), the prevalence of BMI-for-age values at or above the 95th percentile of the 2000 CDC growth charts in children 6 to 11 years of age increased from 4.0% in 1971 to 1974 to 17.0.% in 2003 to 2006. The prevalence of BMI-for-age values at or above the 95th percentile in adolescents 12 to 19 years of age increased from 6.1% to 17.6% in that same time frame.[1,2]
- Among infants and children between the ages of 6 and 23 months, the prevalence of high weight for age was 7.2% in 1976 to 1980 and 11.5% in 2003 to 2006 (NHANES, NCHS).[3]

Adults

- Analysis of the FHS, 1971 to 2001 (NHLBI), showed that among normal-weight white adults between the ages of 30 and 59 years, 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 persons, 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.[19]
- The age-adjusted prevalence of overweight and obesity (BMI $\geq$25 kg/m^2) increased from 64.5% in NHANES 1999 to 2000 (NCHS) to 66.3% in NHANES 2003 to 2004 (NCHS). The prevalence of obesity (BMI $\geq$30 kg/m^2) increased during this period from 30.5% to 34.3%. Extreme obesity (BMI $\geq$40.0 kg/m^2) increased from 4.7% to 5.9%.[1]
- On the basis of NHANES/NCHS data, in 2003 to 2004, 36% of noninstitutionalized women 65 to 74 years of age and 24% of women $\geq$75 years of age were obese. 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, from 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 33% of those 65 to 74 years of age and 23% of those $\geq$75 years of age in 2003 to 2004.[20]
- A 1997 to 2002 study of Medicare beneficiaries found the prevalence of obesity increased by 5.6%, or $\approx$2.7 million 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.[21]
- The WHO 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.[14]
- Using NHANES data collected from the 1970s to 2004, if current trends in the growth of obesity in the United States continue, by 2030, approximately 86% of US adults will be overweight or obese, and 51% will be obese. By 2048, all US adults will be overweight or obese.[22]

Morbidity

- Overweight children and adolescents are at increased risk for future adverse health effects, including[23]:
  - — Increased prevalence of traditional cardiovascular risk factors such as hypertension, hyperlipidemia, and diabetes.
  - — Poor school performance, tobacco use, alcohol use, premature sexual behavior, poor diet, and diabetes.
  - — Other associated health conditions, such as asthma, hepatic steatosis, sleep apnea, stroke, some cancers (breast, colon, and kidney), musculoskeletal disorders, and gallbladder disease.
- The increasing prevalence of obesity is driving an increased incidence of type 2 diabetes. 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.[24]
- In the Nurses' Health Study, obesity was the most powerful predictor of diabetes. Women with a BMI of $\geq$35 kg/m^2 had an RR for diabetes of 38.8 when compared with women with a BMI of <23 kg/m^2.[25]
- An analysis from the FHS showed that overweight and obesity were associated with increased risk for cardiovascular disease. The age-adjusted relative risk for cardiovascular disease was increased by 21% in men and 20% in women among those who were overweight and 46% in men and 64% in women among those who were obese.[26]
- 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).[27]
- A recent comparison of risk factors in both the HHP and FHS (NHLBI) showed that a BMI increase of $\approx$3 kg/m^2 raised the risk of hospitalized thromboembolic stroke by 10% to 30%.[28]
- Obesity is also a strong predictor of sleep-disordered breathing, itself strongly associated with the development

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of cardiovascular disease, as well as with a myriad of other health conditions including numerous cancers, nonalcoholic fatty liver disease, gallbladder disease, musculoskeletal disorders, and reproductive abnormalities.[29]

Mortality

- Among adults, obesity was associated with nearly 112 000 excess deaths (95% CI, 53 754 to 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 to 68 220) and grade II to III obesity (BMI ≥35 kg/m^2) with >82 000 (95% CI 44 843 to 119 289). Underweight was associated with nearly 34 000 excess deaths (95% CI 15 726 to 51 766). As other studies have found,[30] overweight (BMI 25 to <30 kg/m^2) was not associated with excess deaths.[31]
- Analysis of data from NHANES found that in 2004, overweight was associated with significantly increased mortality due to diabetes or kidney disease and was not associated with increased mortality due to cancer or CVD. Obesity was associated with significantly increased mortality due to CVD, some cancers, and diabetes or kidney disease. Obesity was associated with 13% of CVD deaths in 2004.[32]
- Data from NHANES 1988 to 1994 was 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.[33]
- In a collaborative analysis of data from almost 900 000 adults in 57 prospective studies, mostly in western Europe and North America, it was found that overall mortality was lowest at ≈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 ≈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 due to strong inverse associations for smoking-related respiratory disease, and the only clearly positive association was for ischemic heart disease.[34]
- Analysis of data (FHS, NHLBI) showed that overweight and obesity were associated with large decreases in life expectancy. 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. In 40-year-old nonsmokers, females lost 7.1 years and males 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.[12]

Cost

- Among children and adolescents, annual hospital costs related to obesity were $127 million between 1997 and 1999.[35]
- 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).[36]
- If current trends in the growth of obesity continue, total health care costs attributable to obesity could reach $861 to 957 billion by 2030, which would account for 16% to 18% of US health expenditures.[22]

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Table 13-1. Overweight and Obesity

Population Group	Prevalence of Overweight and Obesity in Adults, 2006 Age ≥20 y	Prevalence of Obesity in Adults, 2006 Age ≥20 y	Prevalence of Overweight and Obesity in Children, 2006 Ages 2–19 y	Prevalence of Obesity in Children, 2006 Ages 2–19 y	Cost, 2008*
Both sexes, n (%)	144 100 000 (66.3)	71 600 000 (32.9)	23 500 000 (31.9)	12 000 000 (16.3)	$147 billion
Males, n (%)	75 500 000 (71.7)	33 600 000 (31.8)	12 300 000 (32.7)	6 400 000 (17.1)	. . .
Females, n (%)	68 600 000 (61.0)	38 000 000 (34.0)	11 200 000 (31.0)	5 600 000 (15.5)	. . .
NH white males, %	71.4	31.6	31.9	15.6	. . .
NH white females, %	57.5	31.3	29.5	13.6	. . .
NH black males, %	71.4	35.2	30.8	17.4	. . .
NH black females, %	79.6	53.2	39.2	24.1	. . .
Mexican American males, %	75.1	29.1	40.8	23.2	. . .
Mexican American females, %	74.1	41.8	35.0	18.5	. . .
Hispanic or Latino age ≥18 y,† %	70.3	31.3	. . .	. . .	. . .
Asian-only, age ≥18 y,† %	40.7	9.4	. . .	. . .	. . .
American Indian/Alaska Native, age ≥18 y,† %	69.6	42.1	. . .	. . .	. . .

NH indicates non-Hispanic. Ellipses (. . .) indicate data not available. Data for white, black, and Asian or Pacific Islander males and females are for non-Hispanics. Overweight and obesity in adults is BMI ≥25 kg/m^2. Obesity in adults is ≥BMI 30 kg/m^2.

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 (available at http://www.ama-assn.org/ama1/pub/upload/mm/433/ped_obesity_recs.pdf). However, statistics based on this new definition are not yet available.

*Data from *Health Affairs*.[36]

†NHIS (2008), NCHS (provisional); data are based on self-reported height and weight and are age adjusted for Americans ≥18 years old. Overweight is BMI ≥25 kg/m^2 and <30.0 kg/m^2. Obese is BMI ≥30.0 kg/m^2.[13]

Sources: Age-adjusted NHANES 2003–2006 (NCHS), NHLBI and unpublished data. Data for adults are for ≥20 years of age. Estimates from NHANES 2003–2006 (NCHS) were applied to 2006 population estimates. In children, age-adjusted NHANES 2003–2006 data were applied to 2006 population estimates. Overweight and obesity are based on BMI-for-age values at or above the 85th percentile of the 2000 CDC growth charts. Obesity is based on BMI-for-age values at or above the 95th percentile of the CDC growth charts.[1]

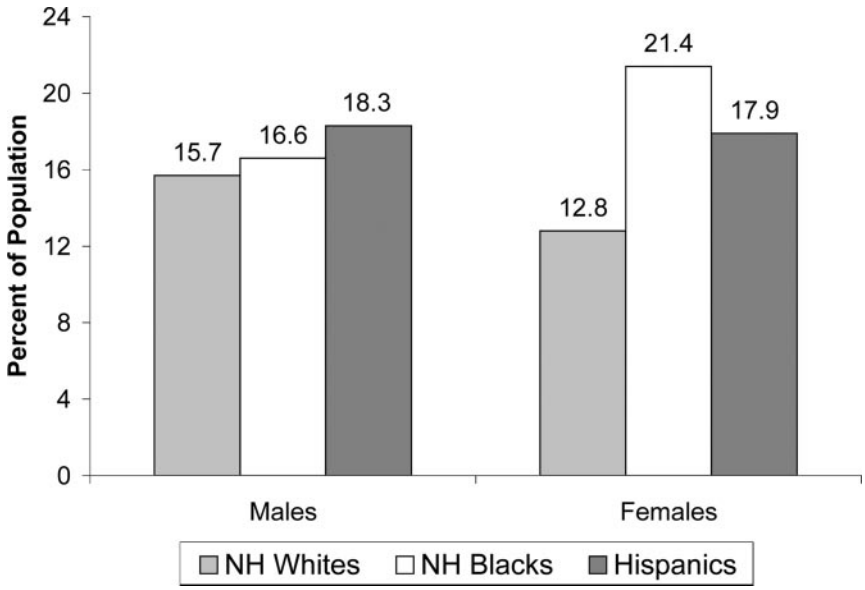

Chart 13-1. Prevalence of overweight among students in grades 9 through 12 by sex and race/ethnicity (YRBS: 2007). BMI ≥95th percentile by age and sex of the CDC 2000 growth chart. Source: YRBS: 2007.[5]

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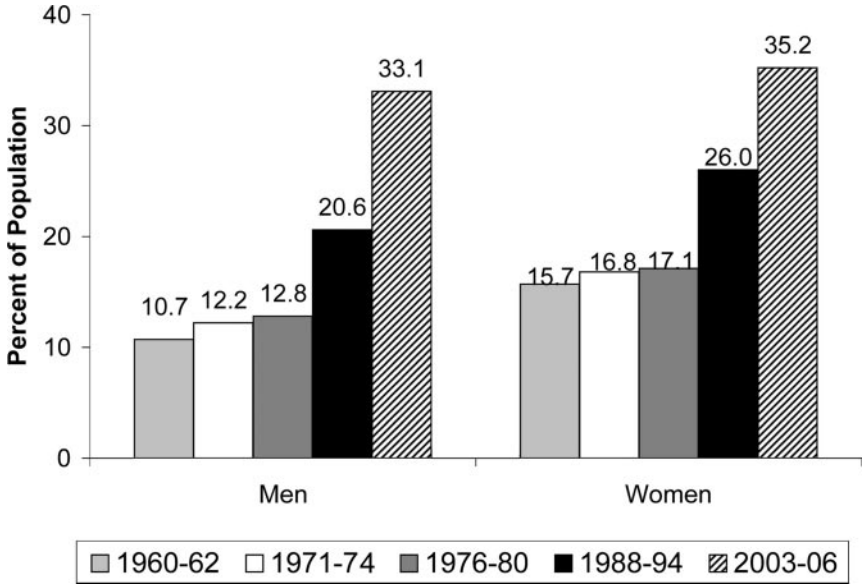

Chart 13-2. Age-adjusted prevalence of obesity in adults 20 to 74 years of age, by sex and survey (NHES: 1960–1962; NHANES: 1971–1974, 1976–1980, 1988–1994, and 2003–2006). Note: Obesity is defined as a BMI of ≥30.0 kg/m^2. Source: Health, United States, 2008 (NCHS).[2]

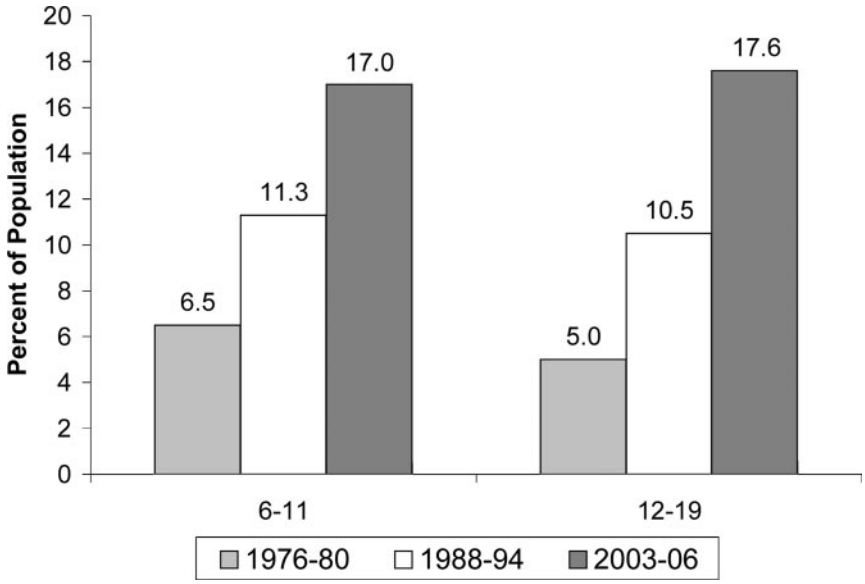

Chart 13-3. Trends in the prevalence of overweight among US children and adolescents by age and survey (NHANES: 1976–1980, 1988–1994, and 2003–2006). Source: Health, United States, 2008 (NCHS).[2]

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14. Risk Factor: Diabetes Mellitus

ICD-9 250; ICD-10 E10–E14. See Table 14-1 and Charts 14-1 through 14-4.

Prevalence

Youth

- In the Search for Diabetes in Youth Study (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 to 9 years of age and 2.80 per 1000 among youths 10 to 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 approximately 0.18%.[1]
- Approximately 186 000 people <20 years of age have diabetes. Each year, ≈15 000 people <20 years of age are diagnosed with type 1 diabetes. Healthcare providers are finding more and more children with type 2 diabetes, a disease usually diagnosed in adults ≥40 years of age. Children who develop type 2 diabetes 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 diabetes, 57.8% of blacks were diagnosed with type 2 versus type 1 diabetes 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 youth aging into adulthood, youth who were prediabetic or who had diabetes are more likely to have a constellation of metabolic disorders in young adulthood (19 to 44 years of age), including obesity, hypertension, dyslipidemia, and metabolic syndrome—all of which predisposes to CHD.[4]

Adult

- Data from NHANES 1999 to 2002 (NCHS) showed the prevalence of diagnosed DM in adults ≥65 years of age to be 15.3%. The prevalence of undiagnosed DM was 6.9%. This represents ≈5.4 million and 2.4 million older individuals, respectively.[5]
- Among Americans ≥20 years of age, 9.6% have DM, and among those ≥60 years of age, 21% have DM. Men ≥20 years of age have a slightly higher prevalence (11%) than women (9%). Among non-Hispanic whites ≥20 years of age, 9% have DM; the prevalence of DM among non-Hispanic blacks in this age range is 1.8 times higher; among Mexican Americans, it is 1.7 times higher; and among American Indians and Alaska Natives, it is 1.5 to 2.2 times higher.[6]
- Data from NHANES (NCHS) show a disproportionately high prevalence of DM in non-Hispanic blacks and Mexican Americans compared with non-Hispanic whites, as shown in Table 14-1.[7]
- The prevalence of diabetes was more than twice as high for Asian Indian adults (14%) as for Chinese (6%) or Japanese (5%) adults.[8]
- Type 2 DM accounts for 90% to 95% of all diagnosed cases of DM in adults.[9] In Framingham, Mass, 99% of DM is type 2.[10]

Abbreviations Used in Chapter 14

ACS	acute coronary syndrome
AHRQ	Agency for Healthcare Research and Quality
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	electrocardiogram
FHS	Framingham Heart Study
HbA_{1c}	glycosylated hemoglobin
HR	hazard ratio
ICD	International Classification of Diseases
kg/m^2	kilograms per square meter
LDL	low-density lipoprotein
mg/dL	milligrams per deciliter
MI	myocardial infarction
mm Hg	millimeter of mercury
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
NIDDK	National Institute of Diabetes and Digestive and Kidney Diseases
NIH	National Institutes of Health
NSTEMI	non–ST-segment–elevation myocardial infarction
OR	odds ratio
RR	relative risk
SBP	systolic blood pressure
SEARCH	Search for Diabetes in Youth Study
STEMI	ST-segment–elevation myocardial infarction
TIMI	Thrombolysis In Myocardial Infarction
UA	unstable angina

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- 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%.[11]
- On the basis of 2008 BRFSS (CDC) data, the prevalence of adults who reported ever having been told by a doctor that they had DM ranged from 5.9% in Minnesota to 11.9% in West Virginia. The median percentage among states was 8.2%.[12]
- 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.[13]
- 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.[14]
- 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 to 74 years of age and by 449% among those 75 years of age or older). 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%).[15]

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 to 4, 5 to 9, and 10 to 14 years, respectively). Overall, type 2 DM was relatively infrequent, with the highest rates (17.0 to 49.4 per 100 000 person-years) seen among 15- to 19-year-old minority groups.[16]

Adults

- A total of 1 600 000 new cases of DM were diagnosed in people ≥20 years of age in 2006.[6]
- Data from Framingham, Mass, 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 $=0.03$).[17]
- Diabetes incidence in adults also varies markedly by race. Over 5 years of follow-up in 45- to-84-year-olds in the Multi-Ethnic Study of Atherosclerosis (MESA), 8.2% of the cohort developed diabetes. The cumulative incidence was highest in Hispanics (11.3%), followed by black (9.5%), Chinese (7.7%), and white (6.3%) participants.[18]

Mortality

DM mortality in 2006 was 72 449. Any-mention mortality in 2006 was 231 000. The 2007 preliminary mortality was 70 905, and the death rate was 22.4.[19] (Source: NCHS and NHLBI).

- The 2006 overall underlying-cause death rate due to DM was 23.3. Death rates per 100 000 persons were 25.4 for white males, 49.7 for black males, 17.9 for white females, and 41.6 for black females.[20]
- According to data from the National Diabetes Information Clearinghouse, NIDDK, and NIH:
  - At least 65% of people with DM die of some form of heart disease or stroke.
  - Heart disease death rates among adults with DM are 2 to 4 times higher than the rates for adults without DM.[21]
- 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.[22]
- Analysis of data from NHANES 1971 to 2000 found that men with DM experienced a 43% relative reduction in the age-adjusted mortality rate, which is 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.[23]
- During 1979 to 2004, diabetes 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 diabetes death rate per 1 million youths was 2.46 for black youths and 0.91 for white youths.[24]
- Analysis of data from the FHS, 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 approximately 2-fold higher compared with individuals without DM.[25]

Awareness

- The NIDDK estimates that 20.8 million Americans (7% of the population) have DM and that ≈30% are unaware of the diagnosis.[6]
- Analysis of NHANES/NCHS data from 1988 to 1994 to 1999 to 2002 in adults ≥20 years of age showed that one third of those with DM did not know they had it. Although the prevalence of diagnosed DM has increased significantly over

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the past decade, the prevalences of undiagnosed DM and impaired fasting glucose have remained relatively stable. Minority groups remain disproportionately affected.[26]

- Analysis of NHANES/NCHS data collected during 2003 to 2006 indicated that the prevalence of diabetes was 10.4% among persons ≥20 years of age. Prevalence of diabetes was defined as persons who (1) were told by a physician or other health professional that they have diabetes, (2) reported current use of insulin or oral agents for diabetes, or (3) were not told of having diabetes and not on treatment, but with a fasting plasma glucose (FPG) ≥126 mg/dL. Of the estimated 21 186 000 adults with diabetes, 73.3% were told or were on 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 of them (2 604 000) were controlled (ie, on treatment with fasting plasma glucose <126 mg/dL) and 25.0% (5 300 000) were treated and uncontrolled (fasting plasma glucose ≥126 mg/dL). An estimated 13 300 000 individuals with diabetes are not treated. The untreated and unaware population (5 600 000) was 26.7% of the diabetic population (Source: NHLBI tabulation of NHANES 2003 to 2006) (see Chart 14-4).

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, Mass, suggest that the risk factor–adjusted relative risk of CHD was 1.38 (95% CI 0.99 to 1.92) times higher and the risk for CHD death was 1.86 times higher (95% CI 1.17 to 2.93) for each 10-year increase in duration of DM.[27]
- DM increases the risk of stroke, with the RR ranging from 1.8 to almost 6.0.[28]
- 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. 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.[29]
- On the basis of data from the NCHS/NHIS, 1997 to 2005[30]:
  - During 1997 to 2005, the estimated number of persons ≥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 persons 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 persons with diabetes 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 persons with diabetes 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).[31] An analysis from the Cooper Clinic in Dallas, Tex, of exercise ECG responses and CVD mortality in 2854 men with diabetes reported 441 deaths (210 CVD and 133 CHD) over 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 (95% CI) were 1.00, 1.68 (1.01 to 2.77), and 2.21 (1.41 to 3.46; P for trend <0.001), respectively.[32]
- A subgroup analysis was conducted of patients with diabetes enrolled in randomized clinical trials that evaluated ACS therapies. The data included 62 036 patients from TIMI studies (46 577 with ST-segment elevation MI [STEMI] and 15 459 with unstable angina/non-STEMI [UA/NSTEMI]). Of these, 17.1% had diabetes. Modeling showed that mortality at 30 days was significantly higher among patients with diabetes than among those without diabetes 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 diabetes versus no diabetes of 1.78 for UA/NSTEMI (95% CI 1.24 to 2.56) and 1.40 (95% CI 1.24 to 1.57) for STEMI. Diabetes was also associated with significantly higher mortality 1 year after UA/NSTEMI or STEMI. By 1 year after ACS, patients with diabetes presenting with UA/NSTEMI had a risk of death that approached that of patients without diabetes presenting with STEMI (7.2% versus 8.1%).[33]
- Data from the ARIC study of the NHLBI found that DM was a weaker predictor of CHD in blacks than in whites.[34]
- Data from Framingham, Mass, 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

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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 to 3.82) versus later (HR 1.96, 95% CI 1.44 to 2.66) periods.[34] 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 persons without DM.[35]

— 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 population-attributable risk 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 ($P=0.12$).[36]

— Other data from Framingham show that over 30 years, CVD among women with diabetes was 54.8% among normal-weight women but 78.8% among obese women. Among normal-weight men with diabetes, the lifetime risk of CVD was 78.6%, whereas it was 86.9% among obese men.[37]

- 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 acute MI (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.[38]
- 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 similar reductions in case fatality rates related to AMI and stroke as those without DM (−44.1% versus −33.2%, $P=0.1$; −17.1% versus −16.6%, $P=0.9$, respectively) and similarly comparable declines 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%.[39]
- 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 due to 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 persons, but as the prevalence of DM has increased, so has the absolute burden of CVD, especially among middle-aged and older individuals.[40]

Risk Factors

- Data from the 2004 National Healthcare Disparities Report (AHRQ, 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.[41]

  — In multivariate models that controlled for age, gender, 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.[41]

- Between NHANES III 1988 to 1994 (NCHS) and NHANES 1999 to 2002 (NCHS), considerable differences were found among ethnic groups in glycemic control rates among adults with type 2 DM. Among non-Hispanic whites, the controlled 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%.[42]
- 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%;

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$P<0.0001$ 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%; $P<0.0001$).[43]

— 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, $P=0.006$).[44]

- In 2001 to 2002, among adults ≥18 years of age with diabetes, 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.[45]
- Analysis of data from the CHS study of the NHLBI found that lifestyle risk factors including physical activity level, dietary habits, smoking habits, alcohol use, and adiposity measures, assessed late in life, were each independently associated with risk of new-onset diabetes. Participants whose physical activity level and dietary, smoking, and alcohol habits were all in the low-risk group had an 82% lower incidence of DM, compared with all other participants. When absence of adiposity was added to the other 4 low-risk lifestyle factors, incidence of DM was 89% lower.[46]
- Aggressive treatment of hypertension is recommended for adults with diabetes to prevent cardiovascular complications. Between NHANES III (1984 to 1992) and NHANES 1999 to 2004, the proportion of patients with diabetes whose BP was treated increased from 76.5% to 87.8%, and the proportion whose blood pressure was controlled nearly doubled (15.9% to 29.6%).[47]

Hospitalizations

Youth

- National Inpatient Sample data from 1993 to 2004 were analyzed for individuals 0 to 29 years of age with a diagnosis of diabetes. Rates of hospitalizations increased by 38%. Hospitalization rates were higher for females (42%) than for males (29%). Inflation-adjusted total charges for diabetes hospitalizations increased 130%, from $1.05 billion in 1993 to $2.42 billion in 2004.[48]

Cost

In 2007, the direct ($116 billion) and indirect ($58 billion) cost attributable to DM was $174 billion.[49] These estimates include not just diabetes as a primary diagnosis, but diabetes-related chronic complications that are attributed to diabetes.[50]

A study of data from NHANES 2003 to 2006, Ingenix Research DataMart, 2003 to 2005 National Ambulatory Medical Care Survey, the 2003 to 2005 National Hospital Ambulatory Medical Care Survey, the 2004 to 2005 Nationwide Inpatient Sample, and the 2003 to 2005 Medical Expenditure Panel Survey 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.[51]

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Table 14-1. Diabetes

Population Group	Prevalence of Physician-Diagnosed DM, 2006 Age ≥20 y	Prevalence of Undiagnosed DM, 2006 Age ≥20 y	Prevalence of Prediabetes, 2006 Age ≥20 y	Incidence of Diagnosed DM Age ≥20 y	Mortality (DM), 2006‡ All Ages	Hospital Discharges, 2006 All Ages	Cost, 2007§
Both sexes	17 200 000 (7.7%)	6 100 000 (2.8%)	63 200 000 (29.0%)	1 600 000§	72 449	584 000	$174 billion
Males	7 900 000 (7.6%)	3 800 000 (3.8%)	37 500 000 (35.9%)	. . .	36 006 (49.7%)*	283 000	. . .
Females	9 300 000 (7.9%)	2 300 000 (1.9%)	25 700 000 (22.2%)	. . .	36 443 (50.3%)*	301 000	. . .
NH white males	6.4%	3.7%	35.9%	. . .	29 060	. . .	. . .
NH white females	6.4%	1.8%	21.7%	. . .	28 144	. . .	. . .
NH black males	12.8%	3.8%	26.4%	. . .	5772	. . .	. . .
NH black females	13.0%	2.3%	22.3%	. . .	7041	. . .	. . .
Mexican American males	11.8%	3.2%	33.3%	. . .	. . .	. . .	. . .
Mexican American females	13.1%	3.8%	26.6%	. . .	. . .	. . .	. . .
Hispanic or Latino,† age ≥18 y	11.0%	. . .	. . .	. . .	. . .	. . .	. . .
Asian,† age ≥18 y	8.0%	. . .	. . .	. . .	. . .	. . .	. . .
AI/AN,† age ≥18 y	15.0%	. . .	. . .	. . .	. . .	. . .	. . .

Ellipses (. . .) indicate data not available; NH, non-Hispanic; and AI/AN, American Indian/Alaska Native.

Undiagnosed DM is defined here 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.

†NHIS.[52] Data are age-adjusted estimates for Americans ≥18 years of age.

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

§CDC; National Diabetes Fact Sheet, 2007. Accessed June 24, 2008.

Sources: Prevalence: Prevalence of diagnosed and undiagnosed diabetes: NHANES 2003–2006, NCHS, and NHLBI. Percentages for racial/ethnic groups are age-adjusted for Americans ≥20 years of age. Age-specific percentages are extrapolations to the 2006 US population estimates. Prevalence of prediabetes: CDC Fact Sheet.[49] CDC computations are from NHANES 2003–2006; extrapolation to the 2007 US population. Percentages for racial/ethnic groups are age adjusted for Americans ≥20 years of age. Incidence: NIDDK estimates. Mortality: NCHS. These data represent underlying cause of death only. Hospital discharges: NHDS, NCHS; data include those inpatients discharged alive, dead, or status unknown.

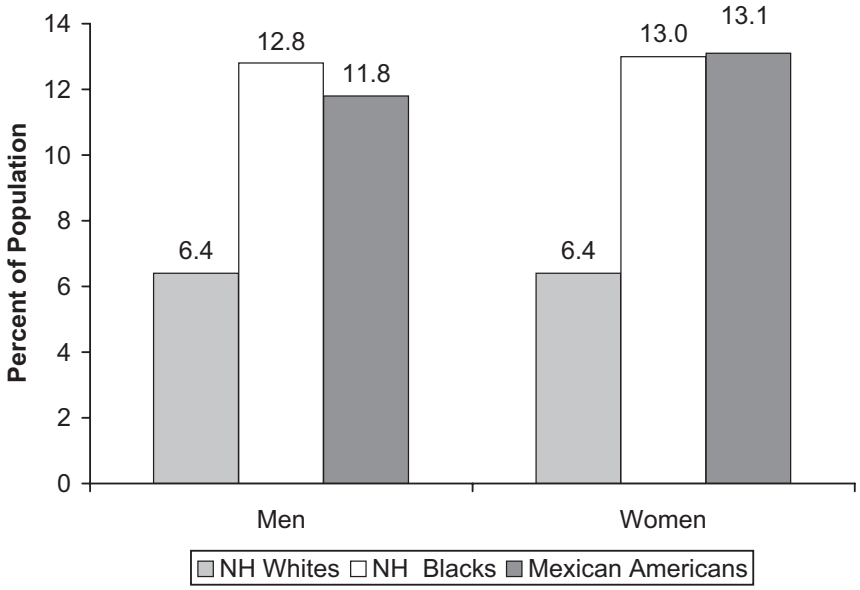

Chart 14-1. Prevalence of physician-diagnosed diabetes in adults ≥20 years of age by race/ethnicity and sex (NHANES: 2003–2006). NH indicates non-Hispanic. Source: NCHS and NHLBI.

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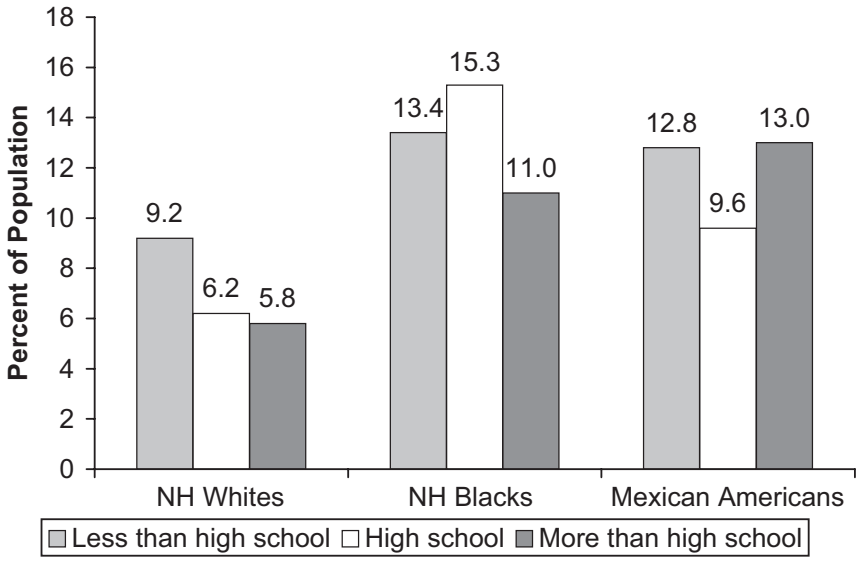

Chart 14-2. Prevalence of physician-diagnosed type 2 diabetes in adults ≥20 years of age by race/ethnicity and years of education (NHANES: 2003–2006). NH indicates non-Hispanic. Source: NCHS and NHLBI.

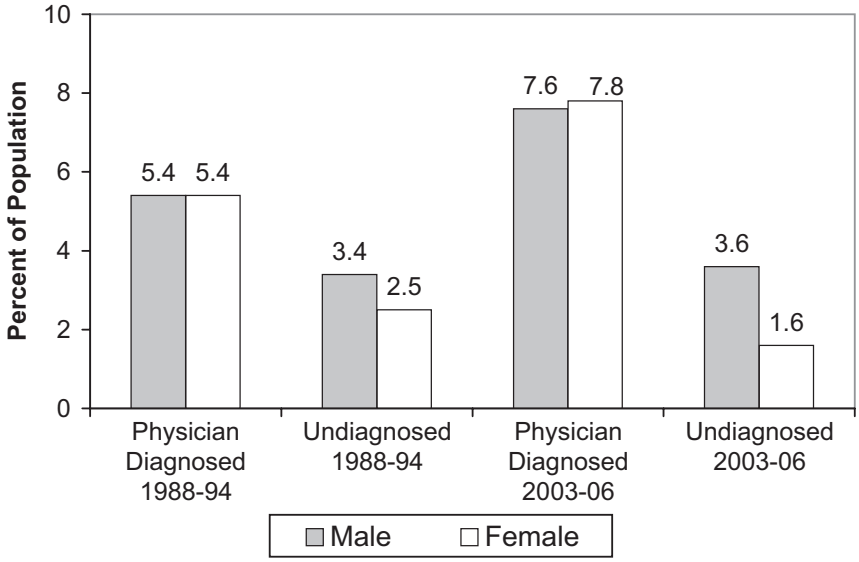

Chart 14-3. Trends in diabetes prevalence in adults ≥20 years of age, by sex (NHANES: 1988–1994 and 2003–2006). Note: "Undiagnosed 2003–2006" data are unreliable for females. Source: Health, United States, 2008 (NCHS).

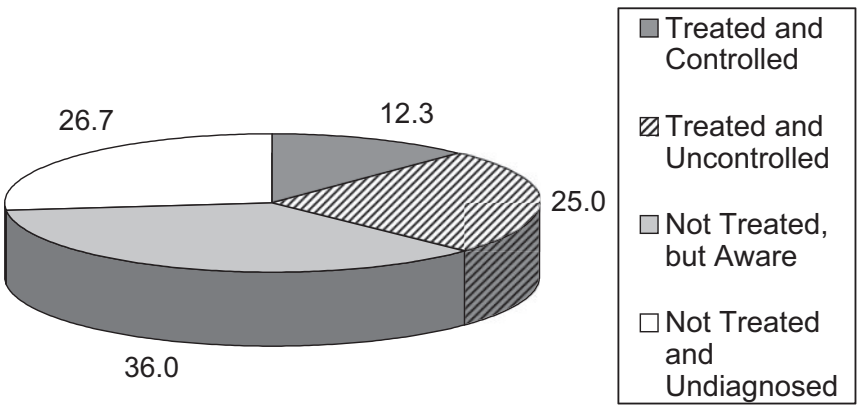

Chart 14-4. Diabetes awareness, treatment, and control (NHANES: 2003–2006). Source: NHLBI.

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15. End-Stage Renal Disease and Chronic Kidney Disease

ICD-10 N18.0. See Tables 15-1 and 15-2.

End-stage renal disease (ESRD) is a condition that is most commonly associated with diabetes and/or HBP and occurs when the kidneys can no longer function normally on their own. When this happens, patients are required to undergo treatment such as hemodialysis, peritoneal dialysis, or kidney transplantation. The ESRD population is increasing in size and cost as those with chronic kidney disease (CKD) transition to ESRD. ESRD morbidity rates vary dramatically among different age, race, ethnicity, and sex population groups. Morbidity rates tend to increase with age and then fall off for the oldest group. The age group with the highest incidence rate is 75 to 79 years of age; the age group with the highest prevalence rate is 70 to 74 years of age.

- Data from the 2008 annual report of the US Renal Data System (USRDS) stated that in 2006, the prevalence of ESRD was 506 256, with 65% of these prevalent cases being treated with hemodialysis.[1]

Abbreviations Used in Chapter 15

AHA	American Heart Association
AMI	acute myocardial infarction
ARF	acute renal failure
BMI	body mass index
BP	blood pressure
CDC	Centers for Disease Control and Prevention
CHD	coronary heart disease
CHF	congestive heart failure
CKD	chronic kidney disease
CKF	chronic kidney failure
CVD	cardiovascular disease
DM	diabetes mellitus
eGFR	estimated glomerular filtration rate
ESRD	end-stage renal disease
GFR	glomerular filtration rate
HBP	high blood pressure
HDL	high-density lipoprotein
HF	heart failure
HMO	health maintenance organization
kg/m^2	kilograms per square meter
K/DOQI	Kidney Disease Outcome Quality Initiative
LDL	low-density lipoprotein
$mL \cdot min^{-1} \cdot 1.73\ m^{-2}$	first morning urine protein/creatinine ratio
NCHS	National Center for Health Statistics
NHANES	National Health and Nutrition Examination Survey
NHDS	National Hospital Discharge Survey
NKF	National Kidney Foundation
PAD	peripheral arterial disease
RR	relative risk
USRDS	United States Renal Data System

- In 2006, 110 854 new cases of ESRD were reported.[1]
- By 2015, it is anticipated that there will be approximately 712 000 prevalent patients with ESRD or a requirement for chronic dialysis or kidney transplantation in the United States.[2]
- The number of persons treated for ESRD increased from 68 757 in 1994 to 102 356 in 2004.[3]
- Data from the USRDS show that in 2006, 87 654 patients died of ESRD.[1]
- In 2004, mortality rates for those ≥65 years of age who were receiving dialysis were 7 times greater than those of the general Medicare population.[3]
- More than 18 000 kidney transplantations were performed in 2006.[1]
- Diabetes continues to be the most commonly reported cause of ESRD, followed by hypertension and glomerulonephritis.[3] These 3 diseases accounted for 80% of all cases of ESRD between 1994 and 2004.[3] Of the more than 100 000 persons who initiated therapy for kidney failure, more than 70% did so because of diabetes and HBP.[4]
- Data from a large HMO cohort 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.[5]
- 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.[6,7]
- From 1994 to 2004, ESRD attributed to glomerulonephritis decreased among all races analyzed.[3]
- From 1994 to 2004, ESRD attributed to glomerulonephritis was highest among blacks.[3]
- ESRD attributed to diabetes or hypertension decreased for American Indians/Alaska Natives and Asians/Pacific Islanders but not for whites or blacks from 1999 to 2004.[3] This decrease is particularly impressive given the increasing prevalence of diabetes among American Indians/Alaska Natives.
- The CDC analyzed 1990–2002 data from the USRDS that showed that diabetes was the leading cause of ESRD, accounting for 44% of new cases in 2002. Although new cases of DM-attributed ESRD increased overall, the incidence of DM-attributed ESRD is not increasing among blacks, Hispanics, men, and people 65 to 74 years of age, and it is declining in people <65 years of age, women, and whites.[8]
- Between 1996 and 1997, 3.2% of the Medicare population had a diagnosis of CKD, which represents 63.6% of people who progressed to ESRD after 1 year.[9]
- Data from a large HMO population reveal that among adults with a GFR >60 $mL \cdot min^{-1} \cdot 1.73\ m^{-2}$ 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[10] (see Table 15-1).
- Results from a large community-based population showed that higher BMI also independently increased the risk of

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ESRD. The higher risk of ESRD with overweight and obesity was consistent across age, sex, and race and in the presence or absence of diabetes, hypertension, or known baseline kidney disease[11] (see Table 15-2).

- Among persons with a reported hospitalization for acute renal failure (ARF) in 2005, 23.1% had ARF as their first-listed diagnosis, whereas 6.9% had septicemia, 6.4% had CHF, and 5.9% had AMI as their first-listed diagnosis. In 1980, DM was reported as an additional discharge diagnosis for 23.4% of kidney disease hospitalizations. This proportion peaked at 39.0% in 1996; DM was associated with 27.0% of kidney disease hospitalizations in 2005. The proportion of kidney disease hospitalizations with hypertension listed among discharge diagnoses increased from 19.6% in 1980 to 41.1% in 2005 (unpublished data from the NHDS, 2006).
- Without treatment, ESRD is fatal. Even with dialysis treatment, mortality rates are higher than those of the non-ESRD population, although they have been falling for the past 5 years. Overall adjusted mortality rates for those on dialysis are 8.2%.[1]
- CVD mortality rates within the first few months after initiation of dialysis have declined in the past 5 years.[1]
- There was no significant difference between the percentage of hemodialysis patients with a urea reduction ratio ≥65 in 2002 (86%) and 2006 (87%).[12]

Age, Sex, Race, and Ethnicity

- Pediatric transplantation rates for children 0 to 17 years of age are higher than those of adults, with wait times for pediatric transplantation candidates less than 1 year (median 163 days). There is no substantial variation in time to transplantation by race among pediatric candidates.[1]
- The median age of the population with ESRD is 58.8 years and varies little by race/ethnicity, at 60.0 years for whites, 56.9 years for blacks, 57.2 years for Hispanics, 59.1 years for Asians, and 57.9 years for Native Americans.[1]
- Treatment of ESRD is more common in men than in women.[1]
- Blacks and Native Americans have much higher rates of ESRD than do whites and Asians. Blacks represent nearly 28% of treated ESRD patients.[1]

Chronic Kidney Disease

Prevalence

- CKD 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 very high cost to the US healthcare system. Controversy exists regarding whether CKD is itself an independent risk factor for incident CVD, but it is clear that persons with CKD, as well as those with ESRD, represent a population at very high risk for CVD. In fact, individuals with CKD are more likely to die of CVD than to transition to ESRD. The USRDS estimates that by 2020, more than 700 000 Americans will have ESRD, with more than 500 000 requiring dialysis and more than 250 000 receiving a transplant.[1]
- The NKF K/DOQI developed guidelines that provided a standardized definition for CKD in 2002. Prevalence estimates may differ depending on assumptions used in obtaining estimates.[13] The most recent US prevalence estimates of CKD, with the use of K/DOQI guidelines, come from NHANES 1999–2004 (NCHS) in adults ≥20 years of age[14]:
  - The prevalence of CKD (stages I to V)[15] is 16.8%.[14] This represents an increase from the 14.5% prevalence estimate from NHANES 1988–1994 (NCHS; recalculated).[14]
  - 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 (eGFR 60 to 89 mL · min^{-1} · 1.73 m^{-2} with kidney damage) is 5.4%.
  - The prevalence of stage III CKD (eGFR 30 to 59 mL · min^{-1} · 1.73 m^{-2}) is 5.4%.
  - The prevalence of stages IV and V CKD (eGFR <29 mL · min^{-1} · 1.73 m^{-2}) is 0.4%.
- Nearly 26 million people (13%) in the United States have CKD, and most are undiagnosed.[16] Another 20 million are at increased risk for CKD.[17]
- Self-reported awareness of poor kidney function is associated with the degree of CKD. In 1999 to 2000, 24.3% were aware of their disease with an eGFR of 15 to 59 mL · min^{-1} · 1.73 m^{-2} and albuminuria, whereas only 1.1% were aware of decreased kidney function with an eGFR ≥90 mL · min^{-1} · 1.73 m^{-2} and no albuminuria.[18]

Demographics

- The prevalence of CKD increased with advancing age as follows[1]:
  - 5.7% for those 20 to 39 years of age;
  - 5.7% for those 40 to 59 years of age; and
  - 37.8% for those ≥60 years of age.
- CKD was more prevalent among those with less than a high school education (22.1%) than among those with at least a high school education (15.7%).[14]
- CKD prevalence was greater among those with DM (43.7%) and hypertension (28.0%) than among those without these chronic conditions.[1]
- The prevalence of CKD was 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.[14]

Risk Factors

- Many traditional CVD risk factors are also risk factors for CKD, including older age, male sex, hypertension, DM,

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elevated LDL, low levels of HDL, smoking, physical inactivity, menopause, and family history of CVD.

- 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.[19]
- Many clinical risk factors for CKD are the same as those for CVD.
- Proteinuria is a strong independent risk factor for a decline in eGFR, regardless of diabetes status, and is associated with many of the same CVD risk factors as for CKD.[20,21]

ESRD/CKD and CVD

- CVD is the leading cause of death among those with ESRD.
  - — 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.[22,23]
  - — Individuals with less severe forms of kidney disease are also at significantly increased risk.[22]
  - — CKD is a risk factor for recurrent cardiovascular events.[24]
  - — Management of CVD differs and is more complex in patients with CKD.[25]
- Studies from a broad range of cohorts demonstrate an association between reduced eGFR and elevated risk of CVD, CVD outcomes, and all-cause death,[26–32] but data are inconsistent with regard to whether these elevated risks are independent of other known major CVD risk factors.
- Any degree of albuminuria, starting below the microalbuminuria cut point, 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.[33–35,37–39]
- A number of consensus documents, including statements from the NKF Task Force[40] and AHA (2003),[22] have indicated that persons with CKD should be considered part of the highest-risk group for CVD.

Hospitalizations

- In 2006, an estimated 315 000 hospitalizations with a first-listed discharge diagnosis of ARF and 35 000 with a first-listed discharge diagnosis of chronic kidney failure (CKF) occurred in the United States.[17]
- From 1980 to 2005, kidney disease was listed as a diagnosis in ≈10 million hospitalizations. The annual number of hospitalizations with a recorded diagnosis of kidney disease quadrupled during this period, from ≈416 000 in 1980 to 1 646 000 in 2005. Age-adjusted hospitalization rates per 10 000 population increased from 20.6 in 1980 to 54.6 in 2005. Kidney disease hospitalization rates were consistently 30% to 40% higher among men than among women. The rates for both sexes increased during 1980 to 2005, from 25.0 to 66.6 per 10 000 in men and from 17.8 to 45.8 per 10 000 in women.[17]

Cost–ESRD

- The total annual cost of treating ESRD in the United States was approximately $33 billion in 2005.[17]

Cystatin C: Kidney Function and HD

Serum cystatin C is a novel marker of kidney function and has been proposed to be a more sensitive indicator of kidney function than serum creatinine and creatinine-based estimating formulas. It is a low-molecular-weight protein produced at a constant rate by all nucleated cells independent of age, sex, and muscle mass. Cystatin C is excreted by the kidneys, filtered through the glomerulus, and nearly completely reabsorbed by proximal tubular cells.[41] Several equations have been proposed using cystatin C alone and in combination with serum creatinine to estimate kidney function.[42,43]

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.[44–46]

Cardiovascular Disease

- Data from a large national cohort found higher values of cystatin C to be associated with prevalent stroke, angina, and MI,[47] as well as higher BMI.[48]
- Elevated cystatin C was an independent risk factor for HF,[49,50] PAD events,[51] clinical atherosclerosis, and subclinical measures of CVD in older adults,[52] as well as for cardiovascular events among those with CHD.[44,53]
- In several diverse cohorts, elevated cystatin C has been found to be associated with cardiovascular mortality.[46,54,55]

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Table 15-1. 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)

JNC V indicates fifth report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure.

Table 15-2. 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)

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16. Metabolic Syndrome

- The term metabolic syndrome (MetS) refers to a cluster of risk factors for CVD and type 2 DM. Several different definitions for MetS are in use; in the United States, the NCEP Adult Treatment Panel III (ATP III) definition and its 2 subsequent revisions have been used most commonly. By this definition, MetS is diagnosed when ≥3 of the following 5 risk factors are present[1]:
  - 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.
  - 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.

Adults

- On the basis of NHANES 2003–2006 data and NCEP/ATP III guidelines, ≈34% of adults ≥20 years of age met the criteria for MetS.[4]

Abbreviations Used in Chapter 16

AHA	American Heart Association
ARIC	Atherosclerosis Risk In Communities
ATP III	Adult Treatment Panel III of the National Cholesterol Education Program
aROC	area under the receiver-operating characteristic curve
BMI	body mass index
BP	blood pressure
CHD	coronary heart disease
CI	confidence interval
cm	centimeter
CVD	cardiovascular disease
DM	diabetes mellitus
FRS	Framingham risk score
HDL	high-density lipoprotein
HR	hazard ratio
MetS	metabolic syndrome
mg/dL	milligrams per deciliter
mm Hg	millimeters of mercury
NCEP	National Cholesterol Education Program
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

- 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 MetS 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 approximately 30% higher among non-Hispanic black women than among non-Hispanic black men and approximately 22% higher among Mexican American women than among Mexican American men.
- The prevalence of MetS is also high among immigrant Asian Indians, ranging between 26.8% and 38.2% depending on the definition used.[5]
- The prevalence of MetS among pregnant women increased to 26.5% during 1999–2004 from 17.8% during 1988–1994.[6]
- However, the public's recognition of MetS is limited.[7]

Children/Adolescents

- An AHA scientific statement about MetS in children and adolescents was released in 2009.[8]
- MetS should be diagnosed with caution in children and adolescents, because MetS categorization in adolescents is not stable. Approximately half of the 1098 adolescent participants in the Princeton School District Study diagnosed with pediatric ATP III MetS lost the diagnosis over 3 years of follow-up.[8a]
- On the basis of NHANES 1999–2002 data, the prevalence of MetS in adolescents 12 to 19 years of age was 9.4%, which represents ≈2.9 million persons. It was 13.2% in males, 5.3% in females, 10.7% in whites, 5.2% in blacks, and 11.1% in Mexican Americans.[9]
- In 1999–2004, approximately 4.5% of United States adolescents 12 to 17 years of age had MetS according to the definition developed by the International Diabetes Federation.[10] In 2006, this prevalence would have represented approximately 1.1 million adolescents 12 to 17 years of age with MetS. 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 males (6.7%) than females (2.1%). Furthermore, 4.5% of white adolescents, 3.0% of black adolescents, and 7.1% of Mexican American adolescents had MetS. The prevalence of MetS remained relatively stable during successive 2-year periods: 4.5% for 1999–2000, 4.4% to 4.5% for 2001–2002, and 3.7% to 3.9% for 2003–2004.

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- In 1999–2002, among overweight or obese adolescents, 44% had MetS.[9] In 1988–1994, two thirds of all adolescents had at least 1 metabolic abnormality.[11]
- Of 31 participants in the NHLBI Lipid Research Clinics Princeton Prevalence Study and the Princeton Follow-Up Study who had MetS at baseline, 21 (68%) had MetS 25 years later.[12] After adjustment for age, sex, and race, the baseline status of MetS was significantly associated with an increased risk of having MetS during adulthood (OR 6.2, 95% CI 2.8 to 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 MetS clustered together in blacks and whites and in children and adults.[13] The degree of clustering was stronger among adults than children. The clustering of rates of change in the components of the MetS in blacks exceeded that in whites.
- Cardiovascular abnormalities are associated with MetS in children and adolescents.[14,15]

Risk

Adults

- Consistent with 2 earlier meta-analyses, a recent meta-analysis of prospective studies concluded that MetS increased the risk of developing CVD (summary RR 1.78, 95% CI 1.58 to 2.00).[16] 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, MetS remained a predictor of cardiovascular events after adjustment for the individual components of the syndrome (summary RR 1.54, 95% CI 1.32 to 1.79).
- Several studies suggest that the FRS is a better predictor of incident CVD than MetS.[17–19] In the San Antonio Heart Study, the area under the receiver operating characteristic curve (aROC) was 0.816 for the FRS and 0.811 for the FRS plus the MetS.[17] Furthermore, the sensitivity for CVD at a fixed specificity was significantly higher for the FRS than for the MetS. In ARIC, MetS did not improve the risk prediction achieved by the FRS.[18] In the British Regional Heart Study, the aROC for the FRS was 0.73 for incident CHD during 10 years of follow-up, and the aROC for the number of MetS components was 0.63.[19] For CHD events during 20 years of follow-up, the aROCs were 0.68 for the FRS and 0.59 for the number of MetS components.
- Estimates of relative risk for CVD generally increase as the number of components of MetS increases.[19,20] Compared with men without an abnormal component in the Framingham Offspring Study, the HRs for CVD were 1.48 (95% CI 0.69 to 3.16) for men with 1 or 2 components and 3.99 (95% CI 1.89 to 8.41) for men with ≥3 components.[20] Among women, the HRs were 3.39 (95% CI 1.31 to 8.81) for 1 or 2 components and 5.95 (95% CI 2.20 to 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 to 2.39) for 1 component, 2.34 (95% CI 1.65 to 3.32) for 2 components, 2.88 (95% CI 2.02 to 4.11) for 3 components, and 3.44 (95% CI 2.35 to 5.03) for 4 or 5 components.[19]
- 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.[21]

Children

- Few prospective pediatric studies have examined the future risk for CVD or diabetes according to baseline MetS status. Data of 771 participants 6 to 19 years of age from the NHLBI's Lipid Research Clinics Princeton Prevalence Study and the Princeton Follow-Up Study found that the risk of developing CVD was substantially higher among those with MetS than among those without this syndrome (OR 14.6, 95% CI 4.8 to 45.3) who were followed up for 25 years.[12]
- Another analysis of 814 participants of this cohort showed that those 5 to 19 years of age who had MetS at baseline had an increased risk of having diabetes 25 to 30 years later compared with those who did not have the syndrome at baseline (OR 11.5, 95% CI 2.1 to 63.7).[22]

Risk Factors

- In prospective or retrospective cohort studies, the following factors have been reported as being directly associated with incident MetS, defined by 1 of the major definitions: Age,[22–25] low educational attainment,[23,26] smoking,[26–28] low levels of PA,[26–31] low levels of physical fitness,[29,32–34] intake of soft drinks,[35] intake of diet soda,[36] magnesium intake,[37] energy intake,[31] carbohydrate intake,[23,27,38] total fat intake,[23,38] Western dietary pattern,[36] meat intake,[36] intake of fried foods,[36] heavy alcohol consumption,[39] abstention from alcohol use,[23] parental history of diabetes,[22] chronic stress at work,[40] pediatric MetS,[22] obesity or BMI,[23,24,27,31,41] childhood obesity,[42] waist circumference,[25,38,43–46] intra-abdominal fat,[47] gain in weight or BMI,[23,48] change in weight or BMI,[25,27,49] weight fluctuation,[50] BP,[25,38,45,51] heart rate,[52] homeostasis model assessment,[46,53] fasting insulin,[43] 2-hour insulin,[43] proinsulin,[43] fasting glucose or hyperglycemia,[25,43,45] 2-hour glucose,[43] impaired glucose tolerance,[43] triglycerides,[25,38,41,43–45] low HDL cholesterol,[25,38,42,43,45] oxidized LDL,[53] uric acid,[49,54] γ-glutamyltransferase,[49,55,56] alanine transaminase,[49,55,57,58] plasminogen activator inhibitor-1,[59] aldosterone,[59] leptin,[60] C-reactive protein,[61,62] adipocyte–fatty acid binding protein,[63] and free testosterone index.[64]
- The following factors have been reported as being inversely associated with incident MetS, defined by 1 of the major definitions, in prospective or retrospective cohort studies: Muscular strength,[65] change in PA or physical fitness,[27,32] alcohol intake,[26,31] Mediterranean diet,[66] dairy

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consumption,[36] insulin sensitivity,[43] ratio of aspartate aminotransferase to alanine transaminase,[57] total testosterone,[64,67,68] sex hormone–binding globulin,[64,67,68] and Δ^5-desaturase activity.[69]

- Furthermore, men were more likely than women to develop MetS,[23,25] and blacks were shown to be less likely to develop MetS than whites.[23]

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17. Nutrition

See Tables 17-1 and 17-2 and Charts 17-1 through 17-3.

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 17-1; NHANES 2005–2006; personal communication with D. Mozaffarian (December 2008).

Abbreviations Used in Chapter 17

AHEI	Alternative Health Eating Index
apo	apolipoprotein
BMI	body mass index
BP	blood pressure
Cal/d	calories per day
CHD	coronary heart disease
CI	confidence interval
CPI	Consumer Price Index
CVD	cardiovascular disease
DASH	Dietary Approaches to Stop Hypertension
DBP	diastolic blood pressure
DHA	docosahexaenoic acid.
DM	diabetes mellitus
EPA	eicosapentaenoic acid
g	gram
HD	heart disease
HDL	high-density lipoprotein
HEI	Healthy Eating Index
HOMA	homeostasis model assessment
ICAM-1	intercellular adhesion molecule-1
kcal	kilockalories
LDL	low-density lipoprotein
MetS	metabolic syndrome
mg	milligram
mg/dL	milligrams per deciliter
mg/L	milligrams per liter
mm Hg	millimeters of mercury
NHANES	National Health and Nutrition Examination Survey
PA	physical activity
pmol/L	picomoles per liter
RR	relative risk
SBP	systolic blood pressure
SD	standard deviation
TOHP	Trials of Hypertension Prevention
USDA	US Department of Agriculture
USDHHS	US Department of Health and Human Services

The dietary consumption by US adults of selected foods and nutrients related to cardiometabolic health is detailed in Table 17-1, according to sex and ethnic subgroups:

- Average consumption of whole grains by white and black men and women was between 0.5 and 0.7 servings per day, with only between 3% and 5% of white and black adults consuming ≥3 servings per day. Average whole grain consumption by Mexican Americans was ≈2 servings per day, with 22% to 28% consuming ≥3 servings per day.
- Average fruit consumption ranged from 1.1 to 1.8 servings per day in these sex and ethnic subgroups; 8% to 11% of whites, 6% to 9% of blacks, and 6% to 10% of Mexican Americans consumed ≥4 servings per day. When 100% fruit juices were included, the number of servings consumed and the proportions of adults consuming ≥4 servings per day approximately doubled.
- Average vegetable consumption ranged from 1.2 to 2.1 servings per day; 11% to 14% of whites, 5% to 10% of blacks, and 3% to 5% of Mexican Americans consumed ≥5 servings 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 (1.4 servings per week) and highest among black and Mexican American men (1.7 servings per week); between 75% and >80% of adults in each sex and ethnic subgroup consumed fewer than 2 servings per week. Approximately 6% of whites, 7% of blacks, and 6% to 7% of Mexican Americans consumed ≥500 mg/d of eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA).
- Average consumption of nuts, legumes, and seeds was ≈2 servings per week among black women, black men, and white women; 3 servings per week among white men; and 6 and 8 servings per week among Mexican American women and men, respectively. Approximately 18% of whites, 14% to 17% of blacks, and 36% to 46% of Mexican Americans consumed ≥4 servings per week.
- Average consumption of processed meats was lowest among Mexican American women (1.5 servings per week) and highest among black men (3.7 servings per week). Between 40% (Mexican American women) and 68% (black men) of adults consumed ≥1 serving per week.
- Average consumption of sugar-sweetened beverages ranged from ≈6 servings per week among white women to 18 servings per week among Mexican American men. Approximately 51% and 32% of white men and women, 76% and 66% of black men and women, and 78% and 61% of Mexican American men and women, respectively, consumed >36 oz (4.5 eight-ounce servings) per week.
- Average consumption of sweets and bakery desserts ranged from ≈4 servings per day (Mexican American men) to 8 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 >25 servings per week.
- Between 33% and 54% of adults in each sex and ethnic subgroup consumed <10% of total calories from saturated fat, and between 59% and 69% consumed <300 mg of dietary cholesterol per day.

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- Only 3% to 7% of whites, 2% to 3% of blacks, and 11% to 12% of Mexican Americans consumed ≥28 g of dietary fiber per day.
- Only 7% to 13% of whites, 9% to 10% of blacks, and 17% to 24% of Mexican Americans consumed <2.3 g of sodium per day. In 2005, the USDHHS and USDA recommended that adults in specific groups, including people with hypertension, all middle-aged and older adults, and all blacks, should consume no more than 1.5 g of sodium per day. Overall in 2005–2006, the majority (69.2%) of US adults belonged to 1 or more of these specific groups in whom sodium consumption should be ≤1.5 g/d.[1]

Foods and Nutrients: Children and Teenagers

See Table 17-2; NHANES 2005–2006; personal communication with D. Mozaffarian (December 2008).

The dietary consumption by US children and teenagers of selected foods and nutrients related to cardiometabolic health is detailed in Table 17-2:

- Average whole grain consumption was low, ranging from 0.4 to 0.5 servings per day, with ≤4% of children in different age and sex subgroups consuming ≥3 servings per day.
- Average fruit consumption was low: 1.5 and 1.3 servings per day in younger boys and girls (5 to 9 years of age), 1.3 servings per day in adolescent boys and girls (10 to 14 years of age), and 0.8 servings per day in teenage boys and girls (15 to 19 years of age). The proportion consuming ≥4 servings per day was low and decreased with age: 6% in those 5 to 9 years of age, 6% to 8% in those 10 to 14 years of age, and 3% to 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 ≥4 servings per day were 18% to 19% of those 5 to 9 years of age, 16% of those 10 to 14 years of age, and 10% to 14% of those 15 to 19 years of age.
- Average vegetable consumption was low, ranging from 0.8 to 0.9 servings per day, with only up to 2% of children in different age and sex subgroups consuming ≥5 servings per day.
- Average consumption of fish and shellfish was low, ranging from between 0.6 and 0.8 servings per week in 5- to 9-year-olds, 0.4 to 1.1 servings per week in 10- to 14-year-olds, and 0.6 to 0.7 servings per week in 15- to 19-year-olds. Among all ages, ≤15% of children and teenagers consumed ≥2 servings per week.
- Average consumption of nuts, legumes, and seeds ranged from 1.0 to 1.2 servings per week among 15- to 19-year-olds to 1.4 to 1.7 servings per week at younger ages. Between 9% and 13% of children in different age and sex subgroups consumed ≥4 servings per week.
- Average consumption of processed meats ranged from 2.1 to 3.4 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 42% and 60% 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 14 servings per week in 10- to 14-year-olds, and 15 to 23 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 13% (boys 15 to 19 years of age) and 40% (boys and girls 5 to 9 years of age) of children consumed ≤4.5 servings per week.
- Average consumption of sweets and bakery desserts was ≈10 servings per week in 5- to 9-year-olds and 10- to 14-year-olds and 6 to 9 servings per week in 15- to 19-year-olds. From 82% (5 to 9 years of age) to 59% (15 to 19 years of age) of youths consumed >2.5 servings per week.
- Average consumption of EPA+DHA was low, ranging from ≈40 to 80 mg/d in boys and girls at all ages. Only between 0.4% and 2.5% of children and teenagers at all ages consumed ≥500 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 11 to 14 g/d. Less than 2% of children in different age and sex subgroups consumed ≥28 g/d.
- Average consumption of sodium ranged from 3.0 to 3.4 g/d. Between 6% 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, 1 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 (see "Trends" below).

- Average daily caloric intake in the United States is ≈2500 calories in adult men and 1800 calories in adult women (Table 17-1). In children and teenagers, average caloric intake is higher in boys than in girls and increases with age in boys (Table 17-2). Trends in energy balance are described below.
- Individual nutritional determinants of positive energy balance (more calories consumed than expended), as determined by adiposity or weight gain, include larger portion sizes,[2,3] higher intake of sugar-sweetened beverages,[4,5] and greater consumption of fast food and commercially prepared meals.[6–10]

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- Each of these dietary factors has multiple influences; eg, preferences for portion size are associated with BMI, socioeconomic status, eating in fast food restaurants, and television watching.[11,12] Portion sizes are larger at fast food restaurants than at home or at other restaurants.[13]
- In 1999–2000, 41% of US adults consumed ≥3 commercially prepared meals per week.[7] 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.[14] 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.[14]
- Macronutrient composition of the diet, such as percent calories from total fat or total carbohydrate, does not appear to be strongly associated with energy balance as ascertained by weight gain or loss.[15–17] Preliminary evidence suggests that aspects of dietary quality rather than composition, such as extent of processing of carbohydrates consumed,[17] consumption of trans fat,[18–20] and energy density,[21–23] may be associated with energy imbalance as assessed by changes in adiposity or weight, but such data are still emerging. Randomized controlled trials 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.[24–26]
- Other individual factors associated with positive energy balance (weight gain) include greater television watching (particularly as related to greater food consumption)[27–32] and lower average sleep duration, particularly among children.[33]
- 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.[34–36] PA is covered in a separate chapter 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 Health Eating Index (AHEI), Western versus prudent dietary patterns, Mediterranean dietary pattern, and DASH-type diet.

- In 1999–2004, 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 1988–1994.[37]
- Among older US adults (≥60 years of age) in 1999–2002, 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.[38]
- 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.[39]

Dietary Supplements

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

- Half (53%) of US adults in 2001–2004 used dietary supplements, with the most common supplement being multivitamins and multiminerals (67% of supplement users). Most supplements were 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.[14,40]
- One third (32%) of US children (birth to 18 years of age) used dietary supplements in 1999–2002, 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).[41]
- In a 2005–2006 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 less than $40 000 per year (41.8%) than in those with higher incomes (30.3%).[42]
- 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 ω-3 fatty acids, 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 EPA Lipid Intervention Study, and GISSI-HF).[43–45]

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

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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 to 1886 kcal/d) and by 10% in men (from 2450 to 2693 kcal/d; Chart 17-1). These increases are supported by data from the Nationwide Food Consumption Survey (1977–1978) and the Continuing Surveys of Food Intake (1989–1998).[13]
- The increases in calories consumed during this time period are attributable primarily to greater average carbohydrate intake, particularly of starches, refined grains, and sugars (see "Foods and Nutrients" below). 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.[7,13,48–52]
- Between 1977–1978 and 1994–1996, 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).[13]
- 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 1988–1994 and 1999–2002.[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 17-1).
- 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 17-1). 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 due largely to 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 17-2).
- 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.[13]
- Among children and teenagers (2 to 19 years of age), the largest increases in consumption of sugar-sweetened beverages between 1988–1994 and 1999–2004 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 and 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% to 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

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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, respectively, increased the ratio of total to HDL cholesterol by 0.031, 0.054, and 0.67; increased apoB levels by 3, 10, and 11 mg/L; decreased apoA-1 levels by 7, 5, and 3 mg/L; and increased lipoprotein(a) levels by 3.8, 1.4, and 1.1 mg/L.[60]
- In meta-analyses of randomized controlled trials, consumption of EPA+DHA for ≥12 weeks lowered SBP by 2.1 mm Hg[61] and resting heart rate by 2.5 bpm.[62]
- 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 HOMA 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 (ICAM-1), and vascular cell adhesion molecule-1.[63]

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 the total impact on health. Randomized controlled trials and prospective observational studies can better quantify the effect of dietary habits on clinical outcomes:

- In the Women's Health Initiative 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 to 1.09), stroke (RR 1.02, 95% CI 0.90 to 1.15), or total CVD (RR 0.98, 95% CI 0.92 to 1.05) over a mean of 8.1 years.[64] This was consistent with null results of 4 prior randomized clinical trials (see below) and multiple large prospective cohort studies (see below) that indicated little effect of total fat consumption on risk of CVD.[65–74]
- In a meta-analysis of randomized controlled trials, increased polyunsaturated fat consumption in place of saturated fat reduced CHD risk by 24%.[75]
- 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 to 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 to 1.22).[76]
- 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% energy of polyunsaturated fat consumption in place of saturated fat was associated with a 13% lower risk of CHD (RR 0.87, 95% CI 0.77 to 0.97). In contrast, each 5% energy of carbohydrate consumption in place of saturated fat was associated with a 7% higher risk of CHD (RR 1.07, 95% CI 1.01 to 1.14), and each 5% energy of monounsaturated fat consumption in place of saturated fat was not significantly associated with CHD risk.[77]
- 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 to 1.37).[78]
- 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 to 0.99) and a 5% lower risk of stroke (RR 0.95, 95% CI 0.92 to 0.97).[79,80]
- Higher estimated consumption of dietary sodium was not associated with lower CVD mortality in NHANES,[81] 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 (TOHP), participants randomized to low-sodium interventions had a 25% lower risk of CVD (RR 0.75, 95% CI 0.57 to 0.99) after 10 to 15 years of follow-up after the original trials.[82]
- 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/d, respectively, compared with <1/mo.[83]
- 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 to 0.87).[84] 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 to 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 to 1.48).[85] Similar findings have been seen in other cohorts and for other outcomes, including development of diabetes and MetS.[86–90]
- In 1 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 ω-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.[91]

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Cost

The USDA forecast that the Consumer Price Index (CPI) 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 CPI 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 17-3).
- 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.[92]
- 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.[92]
- Among 154 forms of fruits and vegetables priced using 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.[92]
- 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 or more for nurse screening and lifestyle advice, $500 to $1250 for smoking cessation, and $20 to $900 for population-based healthy eating.[93]
- Each year, more than $33 billion in medical costs and $9 billion in lost productivity resulting from HD, cancer, stroke, and diabetes are attributed to poor nutrition.[94–99]

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Table 17-1. Dietary Consumption in 2005 to 2006 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 (Mean±SD)	% Meeting Guidelines*	Average Consumption (Mean±SD)	% Meeting Guidelines*	Average Consumption (Mean±SD)	% Meeting Guidelines*	Average Consumption (Mean±SD)	% Meeting Guidelines*	Average Consumption (Mean±SD)	% Meeting Guidelines*	Average Consumption (Mean±SD)	% Meeting Guidelines*
Foods												
Whole grains, servings/d	0.7±0.6	4.6	0.7±0.6	5.0	0.5±0.3	3.6	0.5±0.6	4.4	2.2±1.6	28.2	1.6±1.5	22.0
Fruits, servings/d	1.2±1.3	7.5	1.6±1.5	11.0	1.2±1.3	8.6	1.1±1.3	5.8	1.3±1.5	5.9	1.8±1.3	10.2
Fruits including 100% juices, servings/d	2.0±1.8	16.0	2.1±1.6	17.0	2.3±1.8	21.9	2.1±1.6	14.3	2.0±1.8	13.8	2.8±2.1	23.7
Vegetables, servings/d	1.8±1.1	10.7	2.1±1.1	14.3	1.3±0.8	5.1	1.7±1.2	9.5	1.2±0.6	3.6	1.4±0.7	4.6
Vegetables including juices/sauces, servings/d	2.0±1.2	13.4	2.2±1.2	16.0	1.4±0.7	5.2	1.8±1.2	10.2	1.4±0.6	4.3	1.6±0.6	5.5
Fish and shellfish, servings/wk	1.6±1.4	22.3	1.4±1.1	19.7	1.7±1.2	24.2	1.7±1.1	24.4	1.7±2.0	18.5	1.5±1.1	19.2
Nuts, legumes, and seeds, servings/wk	2.5±1.6	18.2	2.3±1.6	18.2	2.2±0.4	16.6	1.5±0.6	14.0	7.6±6.9	45.9	5.9±3.7	36.2
Processed meats, servings/wk	3.2±1.8	46.3	1.9±1.1	61.2	3.7±1.9	42.3	2.2±1.3	56.6	1.9±1.1	66.8	1.5±1.1	69.8
Sugar-sweetened beverages, servings/wk	10.5±11.4	48.7	6.0±10.2	68.2	15.6±8.6	23.8	12.5±8.2	35.6	17.7±10.8	21.8	10.6±8.2	38.9
Sweets and bakery desserts, servings/wk	7.6±4.9	33.1	7.3±3.7	34.9	7.1±4.9	41.0	7.2±1.8	40.5	4.3±2.9	50.6	6.6±3.0	47.3
Nutrients												
Total calories, kcal/d	2587±667	NA	1750±454	NA	2425±608	NA	1742±603	NA	2441±692	NA	1853±546	NA
EPA+DHA, g/d	0.126±0.134	5.8	0.124±0.134	5.8	0.164±0.168	7.6	0.153±0.125	6.7	0.138±0.134	7.4	0.123±0.134	5.5
ALA, g/d	1.34±0.27	25.4	1.54±0.51	72.1	1.28±0.34	20.1	1.43±0.44	67.2	1.17±0.26	15.6	1.27±0.32	57.9
n-6 PUFA, % energy	7.0±1.2	NA	7.4±1.6	NA	7.2±1.4	NA	7.5±2.0	NA	6.5±1.1	NA	6.6±1.7	NA
Saturated fat, % energy	11.5±2.3	32.7	11.5±2.3	33.5	11.0±1.9	36.4	10.6±2.3	40.1	9.9±2.1	54.1	10.3±1.7	49.3
Dietary cholesterol, mg/d	270±91	68.8	279±93	67.7	298±108	65.1	308±91	59.2	304±138	62.7	280±97	65.5
Total fat, % energy	34.1±5.3	55.4	34.1±4.9	55.2	34.1±4.8	46.3	33.2±5.4	52.9	31.2±5.2	66.4	31.2±5.3	66.1
Carbohydrate, % energy	47.3±7.7	NA	49.0±6.6	NA	48.8±6.2	NA	51.1±6.7	NA	50.9±6.9	NA	53.6±6.8	NA
Dietary fiber, g/d	14.8±4.6	3.2	17.1±5.7	6.8	12.9±3.8	2.0	14.0±5.0	3.3	18.0±6.7	11.7	19.1±4.6	10.7
Sodium, g/d	3.3±0.8	13.1	3.6±0.5	7.2	3.2±0.4	10.2	3.4±0.6	8.7	3.0±0.8	24.4	3.2±0.6	17.4

NH indicates non-Hispanic; ALA, α-linoleic acid; n-6-PUFA, ω-6-polyunsaturated fatty acid; and NA, not available.

Based on data from NHANES 2005 to 2006 (two 24-hour dietary recalls per person, with SDs adjusted for within- and between-person variation). All values are energy adjusted, and for comparability, means and proportions are reported for a 2000-kcal/d diet. To obtain actual mean consumption levels, multiply group means by group-specific total caloric consumption divided by 2000.

*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); fish or shellfish, 2 or more 100-g (3.5-oz) servings per week[96]; fruits, 4 or more 1/2-cup servings per day[97]; vegetables, 5 or more 1/2-cup servings per day, including up to 3 cups per week of starchy vegetables[97]; nuts, legumes, and seeds, 4 or more 50-g servings per week[96]; 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)[97]; sugar-sweetened beverages (defined as ≥50 cal/8 oz, excluding whole juices), 36 oz or less per week (≈1/4 of discretionary calories)[96,97]; sweets and bakery desserts, 2.5 or fewer 50-g servings per week (≈1/4 of discretionary calories)[96,97]; EPA+DHA, ≥0.5 g/d[98]; ALA, ≥1.6/1.1 g/d (men/women)[99]; saturated fat, <10% energy[97]; dietary cholesterol, <300 mg/d[97]; total fat, 20% to 35% energy[97]; dietary fiber, ≥28/d[97]; and sodium, <2.3 g/d.[97]

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Table 17-2. Dietary Consumption in 2005 to 2006 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 (Mean±SD)	% Meeting Guidelines*	Average Consumption (Mean±SD)	% Meeting Guidelines*	Average Consumption (Mean±SD)	% Meeting Guidelines*	Average Consumption (Mean±SD)	% Meeting Guidelines*	Average Consumption (Mean±SD)	% Meeting Guidelines*	Average Consumption (Mean±SD)	% Meeting Guidelines*
Foods												
Whole grains, servings/d	0.5±0.4	0.9	0.5±0.2	0.8	0.5±0.5	4.0	0.5±0.4	2.6	0.4±0.4	2.0	0.5±0.4	2.5
Fruits, servings/d	1.5±0.6	6.2	1.3±0.8	6.2	1.3±0.4	8.4	1.3±0.4	5.9	0.8±0.6	3.2	0.8±0.8	4.2
Fruits including 100% juices, servings/d	2.6±1.6	18.7	2.3±1.3	17.7	2.0±1.1	15.6	2.2±1.1	15.8	1.7±1.4	14.2	1.7±1.3	10.3
Vegetables, servings/d	0.8±0.5	1.4	1.9±0.6	2.1	0.8±0.5	2.2	0.9±0.5	2.2	0.8±0.5	1.2	0.9±0.5	2.3
Vegetables including juices/sauces, servings/d	0.9±0.5	1.8	1.0±0.6	1.7	0.9±0.8	2.2	1.0±0.5	2.3	1.0±0.8	1.5	1.0±0.5	2.4
Fish and shellfish, servings/wk	0.6±0.3	11.7	0.8±0.3	13.8	1.1±0.4	15.2	0.4±0.4	9.2	0.6±0.4	10.3	0.7±0.4	12.2
Nuts, legumes, and seeds, servings/wk	1.5±2.8	13.0	1.7±2.8	12.9	1.4±2.3	8.8	1.5±2.3	11.2	1.2±2.1	9.2	1.0±1.8	8.7
Processed meats, servings/wk	2.2±1.0	60.0	2.1±1.1	59.0	2.5±1.1	57.0	2.3±1.2	54.8	3.4±1.7	41.8	2.3±1.7	58.6
Sugar-sweetened beverages, servings/wk	7.8±5.5	40.6	8.0±3.7	39.7	14.2±6.2	19.9	10.9±5.6	31.6	22.5±8.7	12.9	15.3±8.7	27.2
Sweets and bakery desserts, servings/wk	10.2±4.1	18.2	9.8±4.1	18.4	9.5±4.1	24.0	8.4±4.0	28.0	6.5±3.3	41.2	8.5±1.5	32.6
Nutrients												
Total calories, kcal/d	2010±278	NA	1777±292	NA	2210±423	NA	1901±483	NA	2809±477	NA	1901±457	NA
EPA+DHA, g/d	0.048±0.025	NA	0.063±0.025	NA	0.081±0.030	NA	0.044±0.030	NA	0.064±0.022	NA	0.068±0.021	NA
ALA, g/d	1.14±0.17	11.1	1.13±0.25	42.6	1.13±0.17	11.2	1.23±0.25	49.4	1.12±0.20	12.5	1.33±0.20	56.7
n-6 PUFA, % energy	6.4±0.8	NA	6.3±1.0	NA	6.5±0.8	NA	6.9±1.0	NA	6.3±1.1	NA	6.9±1.1	NA
Saturated fat, % energy	11.9±1.5	21.9	12.0±1.1	20.2	11.7±1.7	24.3	11.5±1.5	28.6	11.8±1.2	25.6	11.7±2.0	29.5
Dietary cholesterol, mg/d	220±72	85.0	250±72	75.2	230±86	79.2	218±115	85.1	239±48	75.7	222±64	81.4
Total fat, % energy	33.3±3.5	63.8	33.3±2.5	67.9	33.4±3.3	61.9	33.3±4.1	62.3	33.5±3.0	57.6	33.4±5.6	56.8
Carbohydrate, % energy	54.0±4.7	NA	53.9±3.5	NA	53.1±4.9	NA	53.8±5.0	NA	51.5±3.6	NA	53.0±4.2	NA
Dietary fiber, g/d	13.6±2.1	0.1	13.7±2.2	1.3	13.0±3.6	1.8	13.8±3.2	0.8	11.5±2.3	0.7	12.8±1.9	0.7
Sodium, g/d	3.0±0.3	10.4	3.2±0.4	6.8	3.2±0.4	8.4	3.4±0.4	6.1	3.2±0.4	12.4	3.3±0.4	10.0

ALA indicates α-linoleic acid; n-6-PUFA, ω-6-polyunsaturated fatty acid; and NA, not available.

Based on data from NHANES 2005 to 2006 (two 24-hour dietary recalls per person, with SDs adjusted for within- and between-person variation). All values are energy adjusted, and for comparability, means and proportions are reported for a 2000-kcal/d diet. To obtain actual mean consumption levels, multiply group means by group-specific total caloric consumption divided by 2000. Each of these guidelines is age-appropriately adjusted to a 2000-kcal/d diet, as for adults.

*See Table 17-1 for food group, serving size, and guideline definitions.

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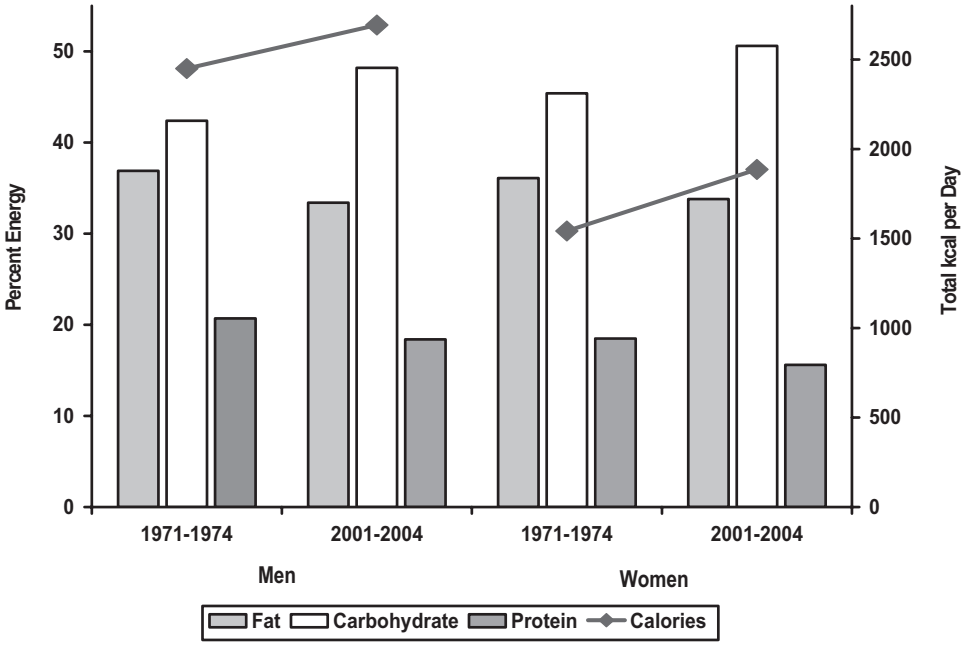

Chart 17-1. Age-adjusted trends in macronutrients and total calories consumed by US adults (20 to 74 years of age), 1971–2004. Source: National Center for Health Statistics. *Health, United States 2007, With Chartbook on Trends in the Health of Americans*.[14]

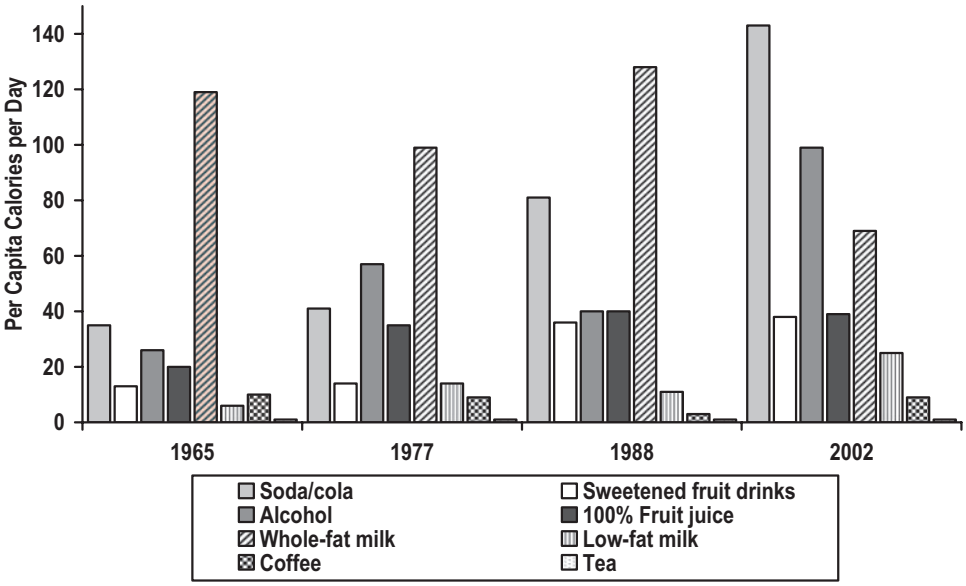

Chart 17-2. Per capita calories consumed from different beverages by US adults (≥19 years of age), 1965–2002. Source: Nationwide Food Consumption Surveys (1965, 1977–1978) and NHANES (1988–1994, 1999–2002); Duffey and Popkin.[51]

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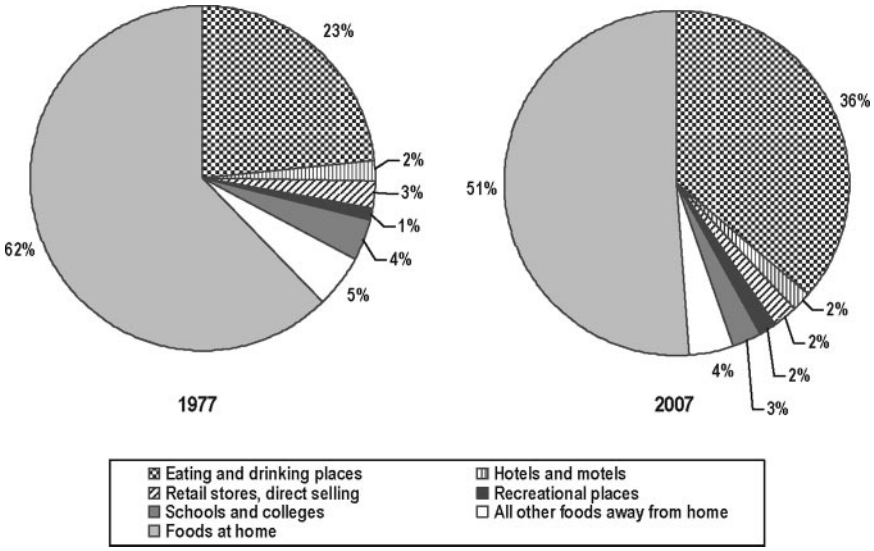

Chart 17-3. Total US food expenditures away from home and at home, 1977 and 2007. Source: USDA Economic Research Service.[54]

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18. Quality of Care

See Tables 18-1 through 18-8.

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" (p 232). This chapter of the update highlights national data on quality of care for several cardiovascular conditions. It is intended to serve as a benchmark for current care and to stimulate efforts to improve the quality of cardiovascular care nationally. Where possible, data are reported from standardized quality indicators (ie, those consistent with the methods for quality performance measures endorsed by the American College of Cardiology and the AHA).[2] Additional data on aspects of quality of care, such as adherence with American College of Cardiology/AHA clinical practice guidelines, are also included to provide a spectrum of quality-of-care data.

Abbreviations Used in Chapter 18

ACS	acute coronary syndrome
ACTION	Acute Coronary Treatment and Intervention Outcomes Network
AHA	American Heart Association
AMI	acute myocardial infarction
AOR	adjusted odds ratio
BP	blood pressure
CAD	coronary artery disease
CI	confidence interval
CMS	Centers for Medicare and Medicaid Services
CRUSADE	Can Rapid Risk Stratification of Unstable Angina Patients Suppress Adverse Outcomes With Early Implementation of the ACC/AHA Guidelines
CVD	cardiovascular disease
DBP	diastolic blood pressure
ED	emergency department
ETT	exercise tolerance test
GWTG	Get With The Guidelines
HBA_{1c}	glycosylated hemoglobin
HF	heart failure
ICD	implantable cardioverter defibrillator
IU/L	international units per liter
LDL	low-density lipoprotein
mg/dL	milligrams per deciliter
mm Hg	millimeters of mercury
NAMCS	National Ambulatory Medical Care Survey
NCDR	National Cardiovascular Data Registry
NHAMCS	National Hospital Ambulatory Medical Care Survey
NRMI	National Registry of Myocardial Infarction
NSTEMI	non–ST-elevation myocardial infarction
PCI	percutaneous coronary intervention
RR	relative risk
SBP	systolic blood pressure
STEMI	ST-elevation myocardial infarction
TIA	transient ischemic attack
USDHHS	US Department of Health and Human Services
VHA	Veterans Health Administration

In addition to describing gaps in care, there have been a number of studies that have implemented interventions to improve the quality of care provided to patients with CVDs. Some of these quality-improvement interventions are highlighted below.

- A randomized trial showed that a clinical reminder, embedded in echocardiography reports, about the use of β-blockers in patients with reduced left ventricular systolic function improved the prescription of β-blockers in the 9 months that followed the echocardiogram (74% versus 64%; $P=0.002$).[3]
- In a quasi-experimental study, there was a strong association between hospitals that used an explicit "discharge contract" with patients and lower 1-year mortality after AMI hospital discharge among hospitals participating in the Michigan "Guidelines Applied in Practice" program. After multivariable adjustment, hospitals in the higher 2 tertiles of hospital discharge contract use were associated with lower 1-year mortality than those in the lowest tertile (tertile 2 [OR 0.43, 95% CI 0.22 to 0.84]) and tertile 3 (OR 0.45, 95% CI 0.27 to 0.75).[4]
- In a statewide quasi-experimental study in North Carolina, the implementation of a coronary reperfusion plan at hospitals (10 PCI hospitals and 55 non-PCI hospitals) improved median reperfusion times significantly: First door-to-device time (presenting to PCI hospital; from 85 to 74 minutes, $P<0.001$), transferred to PCI hospital (from 165 to 128 minutes, $P<0.001$), door-to-needle time in non-PCI hospitals (from 35 to 29 minutes, $P=0.002$), and door-in to door-out time for patients transferred from non-PCI hospitals (from 120 to 71 minutes, $P<0.001$). Nonreperfusion rates were unchanged (15%) in non-PCI hospitals and decreased from 23% to 11% in the PCI hospitals.[5]
- In a before-and-after study, implementation of ED physician activation of the catheterization laboratory and immediate transfer of the patient to an available catheterization laboratory improved median door-to-balloon time (113.5 versus 75.5 minutes, $P<0.0001$). The percentage of patients treated within 90 minutes increased from 28% to 71% ($P<0.0001$). Mean infarct size decreased (peak creatinine kinase 2623 ± 3329 versus 1517 ± 1556 IU/L, $P=0.0089$), as did hospital length of stay (5 ± 7 versus 3 ± 2 days, $P=0.0097$) and total hospital costs per admission ($26 826±29 497 versus $18 280±8943, $P=0.0125$).[6]
- In a randomized controlled trial, an intervention that consisted of home BP monitoring and secure patient Web site training plus pharmacist care management delivered through Web communications significantly improved BP control compared with usual care in patients with essential hypertension. Patients in the intervention had a greater net reduction in SBP (−13.2 mm Hg, 95% CI −19.2 to 7.1 mm Hg, $P<0.001$) and DBP (−4.6 mm Hg, 95% CI −8.0 to −1.2 mm Hg, $P<0.001$) and improved BP control (RR 3.32, 95% CI 1.86 to 5.94, $P<0.001$) compared with patients given usual care.[7]
- At a tertiary care academic hospital, use of a 3-member rapid response team to evaluate, treat, and triage inpatients

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with evidence of acute physiological decline was not associated with a reduction in the primary end point of hospital-wide code rates (adjusted OR [AOR] 0.76, 95% CI 0.57 to 1.01, $P=0.06$) or in-hospital mortality (3.22 versus 3.09 per 100 admissions).[8]

Quality of Care by Race/Ethnicity and Sex

Racial/ethnic, sex-based, and socioeconomic disparities in healthcare quality have been well documented. Elimination of disparities in health care is a critical goal and has become the focus of a number of national initiatives. Reporting and monitoring of quality-of-care measures stratified by race/ethnicity and patient sex are important steps toward addressing disparities in health care through organizational quality improvement. Quality-of-care measures stratified by race/ethnicity and sex have been reported for hospitals participating in "Get With The Guidelines" (GWTG) from January 1, 2008, through December 31, 2008, for stroke, CAD, and HF (Tables 18-3, 18-7, and 18-8).

- In a retrospective cohort study of ≈1.2 million black and white Medicare beneficiaries ≥68 years of age admitted with AMI between January 1, 2000, and June 30, 2005, to 4627 US hospitals, black patients admitted to hospitals with or without revascularization services were less likely to undergo revascularization than white patients (34.3% versus 50.2% and 18.3% versus 25.9%, respectively; $P<0.001$) and had higher 1-year mortality rates (35.3% versus 30.2% and 39.7% versus 37.6%, respectively; $P<0.001$), which persisted after multivariable adjustment.[9]
- In a nested case-control study of Medicare beneficiaries ≥66 years of age by 1999–2001, nonblack men were more likely to be given a stress test than women or black men (OR for nonblack men compared with black women 1.71), even after adjustment for patient characteristics and physician visit frequency.[10] In a separate study, there were no gender differences in subsequent diagnostic testing (coronary angiography or additional stress testing) in the 6 months after an initial positive ETT; however, women were less likely to undergo angiography (OR 0.63, 95% CI 0.47 to 0.83), with a trend toward more subsequent stress testing.[11]
- On the basis of data from the NCDR ICD registry of patients undergoing first-time ICD implantation between January 2006 and December 2007, women were more likely than men to experience any in-hospital adverse event (4.4% versus 3.3%, $P<0.001$) and major in-hospital adverse events (2.0% versus 1.1%, $P<0.001$) after ICD implantation.[12]
- Using data from the 2005 NAMCS and the NHAMCS, among patients with hypertension, women 65 to 80 years of age were less likely than men to have controlled hypertension (OR 0.62, 95% CI 0.45 to 0.85), and women were also less likely than men to be given aspirin (OR 0.43, 95% CI 0.27 to 0.67) and β-blockers (OR 0.60, 95% CI 0.36 to 0.99) for secondary prevention of CVD.[13]
- Data from the US National Hospital Ambulatory Health Care Survey of EDs demonstrated that African American men had a lower likelihood of receiving diagnostic testing when they presented with chest pain. African American men had the lowest probabilities of undergoing electrocardiography and chest radiography (74.3% and 62%, respectively, compared with 81.1% and 70.3%, respectively, among non-African American men). Only 37.5% of African American women received cardiac monitoring, compared with 54.5% of non-African American men. Similarly, African American women were significantly less likely than non-African American men to have their oxygen saturation measured. Patients who were uninsured or self-paying, as well as patients with "other" insurance, also had a lower probability than insured persons of having these tests ordered.[14]

ACS Quality-of-Care Measures

Table 18-1 summarizes quality-of-care indicators as measured by different national organizations or registries. Each of the organization/registries focuses on specific populations among patients hospitalized for an ACS:

- Aggregate data related to CVD from 158 Veterans Administration hospitals for the period between January 2008 and December 2008 are listed in Table 18-1 (Office of Quality and Performance, VHA). Only patients who were candidates for each quality indicator were considered (ie, patients with contraindications to a given therapy were not considered).
- The data shown in Table 18-1 were collected by the Centers for Medicare and Medicaid Services (CMS)/Joint Commission on quality-of-care indicators including acute MI from eligible patients for hospital admissions from October 1, 2007, through September 30, 2008. Additional data were obtained from the USDHHS Hospital Compare Web site. Additional data can be obtained from http://www.hospitalcompare.hhs.gov/hospital/home2.asp.
- The ACTION Registry (Acute Coronary Treatment and Intervention Outcomes Network) is a national risk-adjusted, outcomes-based quality-improvement program. The ACTION Registry measures outcomes of STEMI and NSTEMI patients and combines the data collection and quality reporting features of the former NRMI and CRUSADE registries. By participating in the ACTION Registry, enrolled hospitals can measure their performance in treating patients with AMI against national benchmarks. Listed in Table 18-1 are aggregate data from 52 707 qualifying patients (20 982 STEMI and 31 725 NSTEMI) discharged in 2008 by 268 facilities.
- GWTG-CAD is a national quality-improvement initiative of the AHA to help hospitals redesign systems of care to improve adherence to guidelines in patients admitted with a cardiovascular event. Table 18-1 summarizes performance with regard to the selected quality-of-care indicators for CAD events. These were collected from 72 910 patients who were admitted to 317 hospitals participating in the GWTG-CAD program from January 1, 2008, through December 31, 2008.

HF Quality-of-Care Measures

GWTG-HF is a national quality-improvement initiative of the AHA to help hospitals redesign systems of care to improve adherence to guidelines in patients admitted with HF. Table 18-2 summarizes performance with regard to the selected quality-of-care indicators for HF hospitalizations. These were collected from 66 259 patients who were admitted to 310 hospitals

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participating in the GWTG-HF program from January 1, 2008, through December 31, 2008.

AHA/American Stroke Association GWTG-Stroke Program

GWTG-Stroke is a national quality-improvement initiative of the AHA/American Stroke Association to help hospitals redesign systems of care to improve adherence to guidelines in patients admitted with an ischemic stroke or transient ischemic attack (TIA). Table 18-3 summarizes performance with regard to the selected treatment and quality-of-care indicators for acute stroke and secondary prevention. There were 275 728 clinically identified patients who were admitted to 1155 hospitals participating in the GWTG-Stroke program from January 1, 2008, through December 31, 2008.

Society of Thoracic Surgeons National Database

The Society of Thoracic Surgeons National Database is a national quality-improvement initiative of the Society of Thoracic Surgeons designed to improve the quality of care for patients undergoing cardiothoracic surgery. Table 18-4 summarizes aggregate data for 270 012 procedures performed at 859 participating sites in 2008.

National Committee for Quality Assurance Health Plan Employer Data and Information Set Measures of Care

The National Committee for Quality Assurance is a not-for-profit organization dedicated to improving healthcare quality. The clinical data for 2006 were based on voluntary reporting by >500 health plans. All clinical data were rigorously audited. The Health Plan Employer Data and Information Set measures reported in Table 18-5 are a tool used by 90% of America's managed healthcare plans to measure performance on important dimensions of care and service. More information can be obtained at http://web.ncqa.org.

Data From 2006 NAMCS on Hypertension Control

NAMCS is a national probability sample survey of visits to nonfederal office-based physicians in the United States. Sample data are weighted to produce annual national estimates of physician visits. Table 18-6 summarizes data from the 2006 NAMCS.

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Table 18-1. ACS Quality-of-Care Measures

Quality-of-Care Measure	VHA*	National Medicare and Medicaid†	AHA GWTG-CAD‡	NCDR-ACTION§
Aspirin within 24 h of admission	98	98	91‖	97
Aspirin at discharge	99	97	94‖	98
β-Blockers within 24 h of admission, among AMI and angina patients	97	95	71	93
β-Blockers at discharge	99	97	94‖	96
Lipid-lowering medication at discharge	NM	NM	82	90
Lipid therapy at discharge if LDL >100 mg/dL	96¶	NM	92‖	92
ARB/ACEI at discharge for patients with LVEF <40%	95	93	92‖	85
ACEI at discharge for AMI patients	NM	NM	67	68
Adult smoking cessation advice/counseling	99	99	98‖	96
Fibrinolytic therapy within 30 minutes	53	49	NM	58
PCI within 90 minutes	NM	79	72	82
Cardiac rehabilitation referral for AMI patients	NM	NM	53	77

NM indicates not measured; ARB, angiotensin receptor blocker; ACEI, angiotensin-converting enzyme inhibitor; and LVEF, left ventricular ejection fraction.

Values are percentages.

*VHA: AMI patients.

†National Medicare and Medicaid: AMI patients.

‡AHA GWTG-CAD: Patients admitted with a cardiovascular event. In the GWTG-CAD registry, the in-hospital mortality rate was 4.3% (Excludes transfer outpatients. If discharge status is missing, assumed "no."). The mean length of hospital stay was 5.2 days (median 3.0 days). Length of stay (LOS) was defined previously as follows: LOS=[discharge date−arrival date]+1. Currently, same-day or next-day discharge is defined as LOS=1, and subsequent discharges are defined as LOS=[discharge date−arrival date].

§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 the 6 key achievement measures targeted in GWTG-CAD. The composite quality-of-care measure was 93.7%. The composite quality-of-care measure indicates performance with regard to 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).

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

Table 18-2. HF Quality-of-Care Measures

Quality-of-Care Measure	National Medicare and Medicaid	AHA-GWTG-HF	VA
LVEF assessment	96	97*	100
ARB/ACEI at discharge for patients with left ventricular systolic dysfunction	92	92*	95
Complete discharge instructions	81	89*	95
Adult smoking cessation advice/counseling	97	97*	97
β-Blockers at discharge for patients with LVSD, no contraindications	NM	93*	NM
Anticoagulation for AF or atrial flutter, no contraindications	NM	60	NM

LVEF indicates 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 2.1% of patients. In-hospital mortality rate was 2.9%, and mean length of hospital stay was 5.6 days (median 4.0 days).

*Indicates the 5 key performance measures targeted in GWTG-HF. The composite quality-of-care measure was 93.3%. The composite quality-of-care measure indicates performance with regard to 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).

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Table 18-3. AHA/American Stroke Association GWTG-Stroke Program

Quality-of-Care Measure	Overall	White	Black	Hispanic	Men	Women
IV tPA within 3 h in patients who arrived <2 h after symptom onset*	62.3	61.8	62.0	62.5	63.9	60.8
IV tPA within 3 h in patients who arrived <3 h after symptom onset	49.0	48.9	48.2	48.6	50.7	47.3
Antithrombotics <48 h after admission*	95.9	96.1	95.5	95.0	96.4	95.6
DVT prophylaxis by second hospital day*	92.2	92.1	92.5	91.2	92.5	92.0
Antithrombotics at discharge*	96.9	97.2	96.0	96.2	97.2	96.7
Anticoagulation for atrial fibrillation at discharge*	93.1	93.2	92.9	91.2	93.5	92.8
Therapy at discharge if LDL >100 mg/dL or LDL not measured or on therapy when admitted*	82.1	82.0	82.4	82.1	85.0	79.6
Counseling for smoking cessation*	94.2	94.8	93.3	92.6	94.3	94.1
Stroke education provided	65.5	65.5	67.4	63.4	66.4	64.7
Stroke rehabilitation referral	94.2	94.1	94.9	93.5	93.8	94.6
Composite quality-of-care measure	91.7	91.7	91.5	90.9	92.6	90.9

IV tPA indicates intravenous tissue plasminogen activator; DVT, deep venous thrombosis.
Values are percentages.
In-hospital mortality for the overall patient population was 7.03%, and mean length of hospital stay was 5.31 days (median 4.00 days).
*Indicates the 7 key performance measures targeted in GWTG-Stroke.

Table 18-4. The Society of Thoracic Surgeons National Database

Measure	Society of Thoracic Surgeons 2008 Data
No. of isolated coronary artery bypass procedures	158 750
No. of aortic valve procedures	19 830
No. of mitral valve procedures	4513
Unadjusted isolated coronary artery bypass operative mortality rate, %	1.9
Unadjusted aortic valve operative mortality rate, %	3.1
Unadjusted mitral valve operative mortality rate, %	6.0
Mean postprocedure length of stay for isolated coronary artery bypass procedures, d	7.0
Mean postprocedure length of stay for aortic valve procedures, d	8.1
Mean postprocedure length of stay for mitral valve procedures, d	10.4

Table 18-5. National Committee for Quality Assurance Health Plan Employer Data and Information Set Measures of Care

	Commercial, %	Medicare, %	Medicaid, %
AMI			
β-Blocker persistence*	71.9	75.5	62.0
Cholesterol management for patients with CAD			
Cholesterol screening	88.2	87.9	76.3
LDL control (<100 mg/dL)	58.7	55.9	38.3
Hypertension			
BP <140/90 mm Hg	62.2	57.7	53.4
Diabetes			
HbA_{1c} testing	88.1	88.1	77.3
HbA_{1c} >9.0%	29.4	29.0	47.9
HbA_{1c} <7.0%	42	46	30
Eye examination performed	55.1	62.7	49.9
LDL cholesterol screening	83.9	85.7	70.8
LDL cholesterol <100 mg/dL	43.8	46.8	3.3
Monitoring for nephropathy	80.6	85.7	74.4
BP <130/80 mm Hg	32.1	31.7	29.5
BP <140/90 mm Hg	63.9	58.9	55.5
Medical assistance with smoking cessation	75.8	75.4	69.5

*β-blocker persistence: Received persistent β-blocker treatment for 6 months after AMI hospital discharge.

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Table 18-6. Percent Distribution of Initial BP Measurements for Adults ≥18 Years of Age at Physician Office Visits Where BP Was Taken, With Corresponding Standard Errors, by Selected Patient Characteristics: United States, 2006

			Initial BP*									
			Percent Distribution					Standard Error of Percent				
Patient Characteristics	No. of Visits in Thousands	Total	Low	Normal	Mildly High	Moderately High	Severely High	Low	Normal	Mildly High	Moderately High	Severely High
All visits†	476 335	100.0	4.5	23.4	46.0	19.8	6.3	0.3	0.8	0.8	0.7	0.4
Age, y												
18–24	33 916	100.0	9.4	43.2	39.5	6.3	1.5‡	1.5	2.4	2.4	0.9	0.6
25–44	127 926	100.0	6.3	33.6	43.5	12.4	4.1	0.6	1.3	1.2	0.9	0.5
45–64	167 378	100.0	2.4	20.3	48.2	22.3	6.9	0.3	1.0	1.2	1.1	0.5
65–74	71 316	100.0	3.8	13.3	48.5	26.6	7.7	0.6	1.0	1.6	1.4	0.8
≥75	75 799	100.0	4.7	13.6	46.0	26.3	9.4	0.6	1.0	1.7	1.2	0.8
Sex												
Female	297 728	100.0	5.5	27.0	44.2	17.8	5.4	0.4	1.0	0.8	0.8	0.4
Male	178 608	100.0	2.9	17.3	49.0	23.1	7.7	0.3	0.8	1.3	1.0	0.6
Race§												
White	404 852	100.0	4.5	23.5	46.7	19.6	5.8	0.3	0.8	0.8	0.7	0.4
Black	47 367	100.0	4.8	21.3	42.5	22.3	9.1	1.1	1.6	1.8	1.6	1.2
Asian	18 455	100.0	4.8	26.5	40.3	18.7	9.7	1.4	2.6	2.7	2.7	1.9
Other	5662	100.0	4.2§	23.9	45.5	17.8	8.6§	1.9	5.0	5.5	3.3	3.3
Ethnicity												
Hispanic or Latino	58 351	100.0	6.0	28.1	41.6	17.6	6.6	1.0	1.9	1.8	1.7	1.0
Not Hispanic or Latino	417 985	100.0	4.3	22.7	46.6	20.1	6.2	0.3	0.8	0.8	0.7	0.4

Numbers may not add to totals because of rounding.

*BP levels were categorized with the following hierarchical definitions. "Severely high" BP is defined as 160 mm Hg systolic or above, or 100 mm Hg diastolic or above. "Moderately high" BP is defined as 140–150 mm Hg systolic or 90–99 mm Hg diastolic. "Mildly high" BP is defined as 120–139 mm Hg systolic or 80–89 mm Hg diastolic. "Low" BP is defined as less than 100 mm Hg systolic or less than 60 mm Hg diastolic. "Normal" BP is defined as 100–119 mm Hg systolic and 60–79 mm Hg diastolic. BP classification was based on the *Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure* (JNC-7).[14] "Mildly high" BP corresponds to the JNC-7 prehypertensive range; "moderately high" BP corresponds to the JNC-7 stage 1 hypertensive range; and "severely high" BP corresponds to the JNC-7 stage 2 hypertensive range.

†Visits during which BP was taken represent 66.2% (SE=1.7) of all office visits made by adults (≥18 years of age). In 25.8% (SE=1.6) of visits by children (0–17 years of age), a BP was recorded.

‡Figure does not meet standards of reliability or precision.

§Other race includes visits by Native Hawaiian or other Pacific Islander, American Indian or Alaska Native, and multiple races. All race categories include visits by persons of Hispanic or non-Hispanic origin. Starting with data year 1999, race- and ethnicity-specific estimates have been tabulated according to 1997 Standards for Federal Data on Race and Ethnicity and are not strictly comparable to estimates from earlier years. The percentage of visit records with multiple races indicated is smaller and lower than in household surveys.

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Table 18-7. Quality of Care by Race/Ethnicity and Sex in the GWTG-CAD Program

Quality-of-Care Measure	White	Black	Hispanic	Men	Women
Aspirin at admission*	97.6	97.2	97.9	98.0	96.7
Aspirin at discharge*	94.8	92.9	90.8	95.3	92.6
β-Blocker at discharge*	95.1	95.2	92.0	92.0	95.4
ACE inhibitor at discharge	63.2	67.5	64.3	65.3	59.1
ACE inhibitor at discharge for AMI patients	66.2	69.7	70.8	68.4	62.1
ACE inhibitor in LVSD patients	83.5	84.8	81.5	84.5	80.4
ACE inhibitor/ARB for LVSD patients at discharge*	92.7	93.7	90.1	92.2	92.8
Lipid therapy at discharge	84.2	78.0	71.9	85.0	78.7
Lipid therapy at discharge if LDL >100 mg/dL*	92.5	91.7	84.7	93.5	88.3
Patients with last BP <140/90 mm Hg	81.4	72.3	77.7	82.3	76.5
Smoking cessation counseling*	98.4	98.4	98.3	98.6	98.1
Referral to cardiac rehabilitation for AMI patients	55.8	56.4	64.3	53.8	50.8
Composite quality-of-care measure†	95.5	95.0	92.6	95.9	94.2

ACE indicates angiotensin-converting enzyme; LVSD, left ventricular systolic dysfunction; and ARB, angiotensin receptor blocker.

Values are percentages.

*Indicates the 5 key achievement measures targeted in GWTG-CAD.

†The composite quality-of-care measure indicates performance with regard to 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 18-8. 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*	88.1	90.3	90.2	89.1	85.4
Measure of LV function*	97.0	97.9	94.9	97.3	96.8
ACE or ARB at discharge for patients with LVSD, no contraindications*	90.5	92.9	92.4	91.4	91.2
Smoking cessation counseling, current smokers*	96.7	98.0	97.6	97.2	96.9
β-blockers at discharge for patients with LVSD, no contraindications*	93.3	92.7	90.1	93.3	92.6
Hydralazine/nitrates at discharge for patients with LVSD, no contraindications	NM	15.7	NM	17.7†	12.9†
Anticoagulation for atrial fibrillation or atrial flutter, no contraindications	60.5	58.8	57.4	61.3	52.3
Composite quality-of-care measure	93.0	94.2	92.5	93.4	93.1

LV indicates left ventricular; ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; LVSD, left ventricular systolic dysfunction; and NM, not measured.

*Indicates the 5 key achievement measures targeted in GWTG-HF.

†For black patients only.

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19. Medical Procedures

See Tables 19-1 and 19-2 and Charts 19-1 through 19-3.

- From 1996 to 2006, the total number of inpatient cardiovascular operations and procedures increased 33%, from 5 444 000 to 7 235 000 annually (AHA 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 due to PCI and CABG by sex.[1]
  - — Discharge rates (per 10 000 population) for PCI increased 58%, from 37.2 in 1990–1992 to 59.2 in 2002–2004.
  - — Discharge rates for CABG increased from 34.1 in 1990–1992 to 38.6 in 1996–1998, then declined to 25.2 in 2002–2004.
  - — In 1990–1992, 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 2002–2004. 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 2002–2004, discharge rates for males and females 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 2002–2004, despite an increase in Charlson comorbidity index. The mortality rate declined in all age and sex subsets, but especially in women.
  - — PCI mortality remained stable over the 15-year interval.
- 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 2002–2005, 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 19

AHA	American Heart Association
ICD-9-CM	International Classification of Diseases, 9th Revision, Clinical Modification
CABG	coronary artery bypass graft
D2B	door-to-balloon
GWTG-CAD	Get With The Guidelines–Coronary Artery Disease
HDL	high-density lipoprotein
NCHS	National Center for Health Statistics
NHDS	National Hospital Discharge Survey
NHLBI	National Heart, Lung, and Blood Institute
PCI	percutaneous coronary intervention
PTCA	percutaneous transluminal coronary angioplasty
STEMI	ST-elevation myocardial infarction
STS	Society of Thoracic Surgeons
STS NCD	Society of Thoracic Surgeons' National Adult Cardiac Database
UNOS	United Network for Organ Sharing

Cardiac Catheterization and PCI

- From 1996 to 2006, the number of cardiac catheterizations decreased slightly, from 1 161 000 to 1 115 000 annually.
- In 2006, an estimated 1 313 000 PCI (previously referred to as percutaneous transluminal coronary angioplasty, or PTCA) procedures were performed in the United States (NHDS, NCHS).
- In 2006, approximately 65% of PCI procedures were performed on men, and approximately 50% were performed on people ≥65 years of age (NHDS, NCHS).
- The mortality rate for PCI has remained stable, despite an increase in risk.[1]
- In 2006, approximately 76% of stents implanted during PCI were drug-eluting, compared with 24% of 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 percent 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. A study of patients undergoing PCI at Emory University Hospital from January 2001 to December 2004 found that a baseline HDL cholesterol level <35 mg/dL was an important prognostic indicator. Baseline HDL cholesterol levels <33 mg/dL for men and <38 mg/dL for women were associated with higher 1-year mortality after PCI.[5,6]

Cardiac Surgery

The NHDS (NCHS) estimates that in 2006, in the United States, 253 000 patients underwent a total of 448 000 coro-

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nary 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 (STS NCD), which voluntarily collects data from ≈80% of all hospitals performing CABG in the United States, indicate that a total of 176 138 procedures involved CABG in 2007.[7]
- Data from the STS NCD document a >50% decline in risk-adjusted mortality rate, despite a significant increase in preoperative surgical risk.[8]

Congenital Heart Surgery 2005–2008 (STS)

There were 87 271 procedures performed from January 2005 to December 2008. The in-hospital mortality rate was 3.8% in 2008. The 5 most common diagnoses were the following: Patent ductus arteriosus (7.9%); hypoplastic left heart syndrome (6.8%); ventricular septal defect, type 2 (5.4%); tetralogy of Fallot (5.3%); and cardiac, other (4.6%).[9]

Heart Transplantations

In 2008, 2163 heart transplantations were performed in the United States. There are 251 transplant hospitals in the United States, 126 of which perform heart transplantations (personal communication with the United Network for Organ Sharing [UNOS], June 19, 2009).

- Of the recipients, 72.4% are male, and 65.5% are white; 26.4% are <35 years of age, 19.4% are 35 to 49 years of age, and 54.2% are ≥50 years of age.
- As of June 5, 2009, 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 73.1% for males and 69.0% for females.
- As of June 5, 2009, 2791 heart patients were on the transplant waiting list.

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.[10]

- Ischemic heart disease, congestive heart failure, and cerebrovascular disease 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 19-1. 2007 National Healthcare Cost and Utilization Project Statistics: Mean Hospital Charges and In-Hospital Death Rates for Various Procedures

Procedure	Mean Hospital Charges, $	In-Hospital Death Rate, %
Total vascular and cardiac surgery and procedures	58 559	3.34
CABG	112 377	1.95
PCI	51 445	0.80
Diagnostic cardiac catheterization	31 181	0.79
Pacemaker	51 188	1.15
Implantable defibrillator	115 763	0.49
Endarterectomy	28 584	0.37
Valves	157 888	4.77

Source: Agency for Healthcare Research and Quality, Healthcare Cost and Utilization Project.[11]

Table 19-2. Estimated* Inpatient Cardiovascular Operations, Procedures, and Patient Data by Sex, Age, and Region: United States, 2006 (in Thousands)

Operation/Procedure/ Patients	ICD-9-CM Code(s)	Sex			Age, y				Region†			
		All	Males	Females	<15	15–44	45–64	≥65	Northeast	Midwest	South	West
Valves	35.1, 35.2, 35.99	104	61	43	‡	8‡	30	63	24	24	30	27
Angioplasty	36.0, 00.66	1314	855	459	‡	66	595	652	232	372	461	249
Total PCI§‖	36.06, 36.07, 00.66	1313	854	459	‡	66	595	651	232	371	460	249
Patients	36.06, 36.07, 00.66 (any)	700	453	247	‡	35	317	348	122	207	241	129
PCI	0.66	661	429	232	‡	33	301	327	123	168	239	130
PCI w/stents	36.06, 36.07	652	425	227	‡	33	294	324	109	203	221	119
Cardiac revascularization (bypass)#	36.1–36.3	448	323	125	‡	16	192	240	65	124	182	77
Cardiac revascularization (bypass) (patients)	36.1–36.3 (any)	253	181	73	‡	8‡	105	139	37	69	103	44
Cardiac catheterization	37.21–37.23	1115	666	450	12	87	487	529	201	258	458	199
Pacemakers	37.7, 37.8, 00.50, 00.53	418	198	219	. . .	9‡	46	361	103	94	147	73
Pacemaker devices	(37.8, 00.53)	195	92	103	. . .	4	19	171	49	44	67	35
Pacemaker leads	(37.7, 00.50)	223	106	116	. . .	5	27	190	54	50	80	38
Implantable defibrillators	37.94–37.99, 00.51, 00.54	114	80	34	‡	11	36	68	24	28	40	23
Endarterectomy	38.12	99	55	44	. . .	‡	22	77	13	25	44	18
Total vascular and cardiac surgery and procedures**††	35–39, 00.50–00.51, 00.53–00.55, 00.61–00.66	7235	4116	3119	210	734	2635	3658	1367	1620	2816	1432

Ellipses (. . .) indicate 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 the Table probably reflect this trend. Outpatient procedure data were not available in time to be included in this report.

*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 fewer than 5000. Categories of such small numbers are considered unreliable by NCHS and in some cases may have been omitted.

†Regions: Northeast—Connecticut, Maine, Massachusetts, New Hampshire, New Jersey, New York, Pennsylvania, Rhode Island, Vermont; Midwest—Illinois, Indiana, Iowa, Kansas, Michigan, Minnesota, Missouri, Nebraska, North Dakota, Ohio, South Dakota, Wisconsin; South—Alabama, Arkansas, Delaware, District of Columbia, Florida, Georgia, Kentucky, Louisiana, Maryland, Mississippi, North Carolina, Oklahoma, South Carolina, Tennessee, Texas, Virginia, West Virginia; and West—Alaska, Arizona, California, Colorado, Hawaii, Idaho, Montana, Nevada, New Mexico, Oregon, Utah, Washington, Wyoming.

‡Estimate should be used with caution because it may be unreliable or does not meet standards of reliability or precision.

§Data are for procedures with a PCI listed anywhere on the medical record. Procedures with a PCI listed were counted twice if they also had a code for insertion of stent: code 36.06, "insertion of non–drug-eluting stents," and 36.07, "insertion of drug-eluting stents."

‖Ninety-one percent of discharges with PCI were reported to have a stent inserted (personal communication with NCHS, June 15, 2007).

#Because 1 or more 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.

Source: NHDS, NCHS, 2006. Estimates are based on a sample of inpatient records from short-stay hospitals in the United States.

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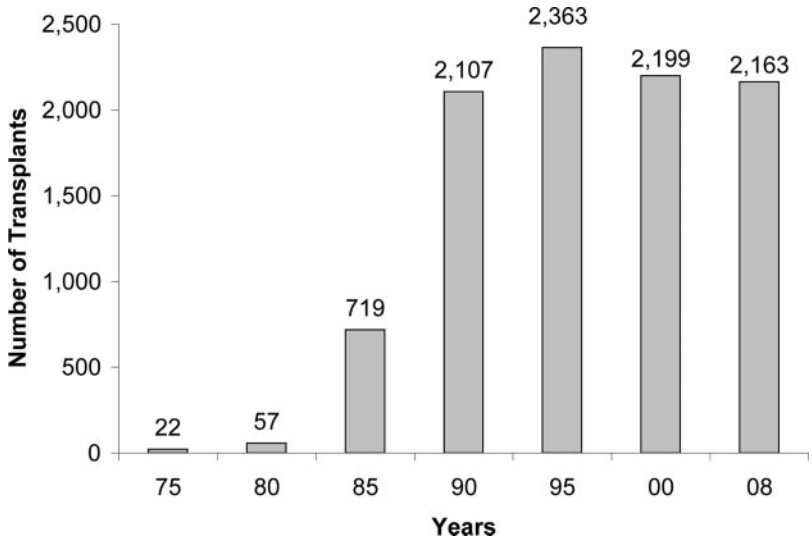

Chart 19-1. Trends in heart transplantations (UNOS: 1975–2008). Source: UNOS, scientific registry data.

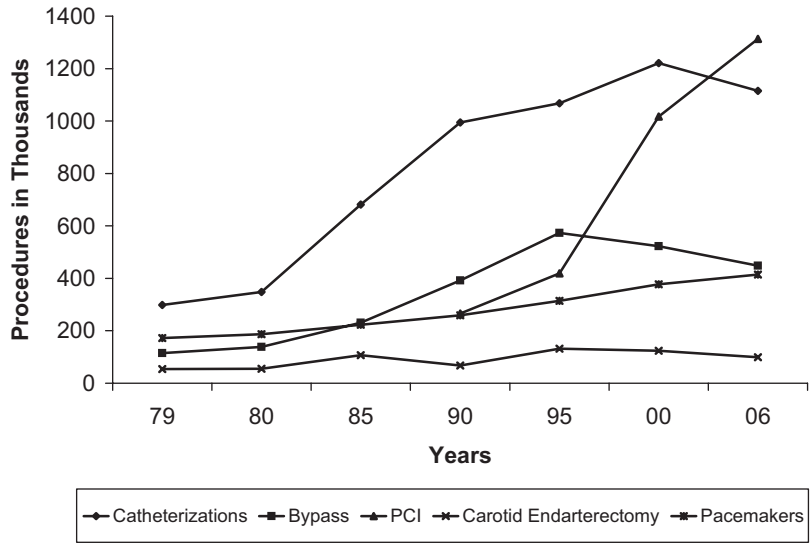

Chart 19-2. Trends in cardiovascular inpatient operations and procedures (United States: 1979–2006). Source: NHDS, NCHS, and NHLBI. Note: In-hospital procedures only.

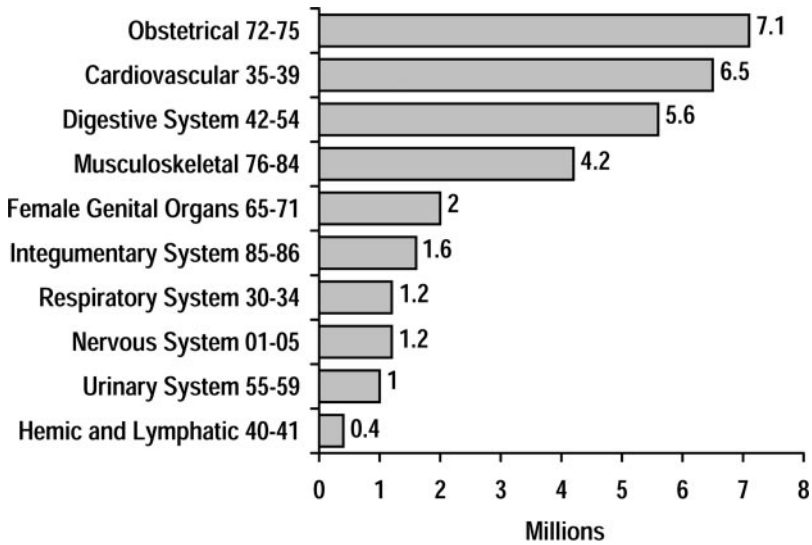

Chart 19-3. Number of surgical procedures in the 10 leading diagnostic groups (United States: 2006). Source: NHDS/NCHS and NHLBI.

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20. Economic Cost of Cardiovascular Diseases

See Table 20-1 and Charts 20-1 and 20-2.[1–5]

The total direct and indirect cost of CVD and stroke in the United States for 2010 is estimated at $503.2 billion. This figure includes health expenditures (direct costs, which include the cost of physicians and other professionals, hospital and nursing home services, prescribed medications, home health care, and other medical durables) and lost productivity resulting from morbidity and mortality (indirect costs). Total hospital costs (inpatients, outpatients, and ED patients) projected for the year 2010 are estimated to be $155.7 billion. 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 (unpublished data, D.P. Rice, W. Max, M. Michel, and H.-Y. Sung, Institute for Health and Aging, University of California, San Francisco, 2009). All estimates for a given disease are limited to that disease as the primary diagnosis.

Abbreviations Used in Chapter 20

CHD	coronary heart disease
CVD	cardiovascular disease
ED	emergency department
HF	heart failure
NHLBI	National Heart, Lung, and Blood Institute

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Table 20-1. Estimated Direct and Indirect Costs (in Billions of Dollars) of CVD and Stroke: United States: 2010[1–5]

	Heart Diseases*	CHD	Stroke	Hypertensive Disease	HF	Total CVD†
Direct costs						
Hospital	$110.2	$56.6	$21.0	$8.5	$20.9	$155.7
Nursing home	$24.7	$13.0	$17.1	$5.1	$4.7	$50.8
Physicians/other professionals	$24.7	$13.9	$3.8	$13.9	$2.5	$48.1
Drugs/other						
Medical durables	$21.5	$10.0	$1.3	$24.7	$3.2	$50.7
Home health care	$8.3	$2.5	$5.0	$2.7	$3.8	$18.8
Total expenditures†	$189.4	$96.0	$48.2	$54.9	$35.1	$324.1
Indirect costs						
Lost productivity/morbidity	$25.6	$11.3	$7.5	$9.0	. . .	$41.7
Lost productivity/mortality‡	$101.4	$69.8	$18.0	$12.7	$4.1	$137.4
Grand totals†	$316.4	$177.1	$73.7	$76.6	$39.2	$503.2

Ellipses (. . .) indicate data not available.

All estimates prepared by Thomas Thom, NHLBI.

*This category includes CHD, HF, part of hypertensive disease, cardiac dysrhythmias, rheumatic heart disease, cardiomyopathy, pulmonary heart disease, and other or ill-defined "heart" diseases.

†Totals do not add up because of rounding and overlap.

‡Lost future earnings of persons who will die in 2010, discounted at 3%.

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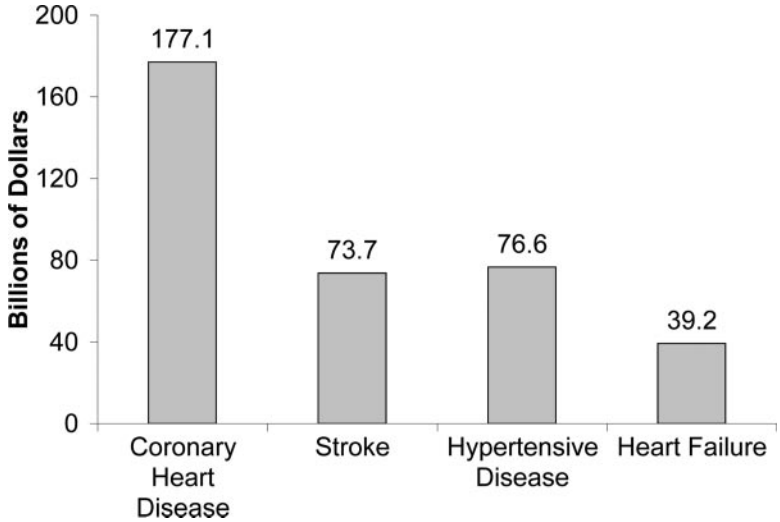

Chart 20-1. Estimated direct and indirect costs (in billions of dollars) of major CVDs and stroke (United States: 2010). Source: Personal communication, Thomas Thom, NHLBI.

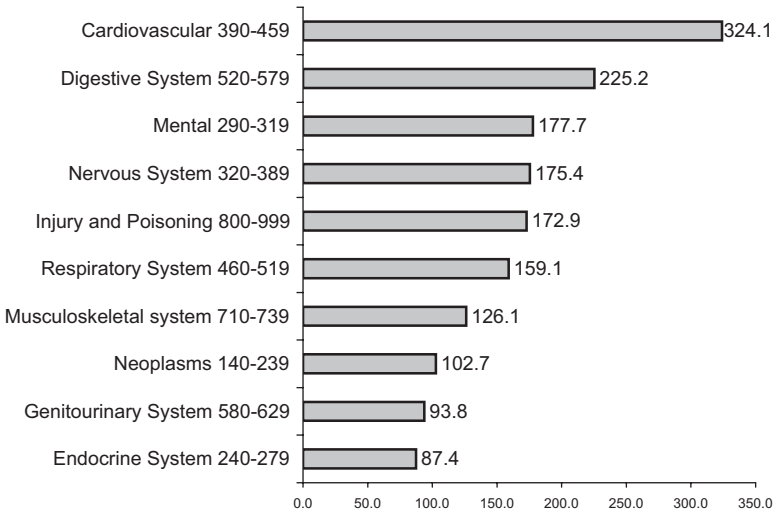

Chart 20-2. Direct costs (in billions of dollars) of the 10 leading diagnostic groups (United States: 2010). Estimated by NHLBI.[1,2] Source: NHLBI.

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21. At-a-Glance Summary Tables

See Tables 21-1 through 21-4.[1–6]

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Table 21-1. 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, 2006*	81.1 M (36.9%)	39.0 M (37.9%)	38.1%	44.6%	28.5%
Mortality, 2006†	831.3 K	398.6 K	340.6 K	48.0 K	N/A
CHD					
Prevalence, CHD, 2006*	17.6 M (7.9%)	9.2 M (9.1%)	9.4%	7.8%	5.3%
Prevalence, MI, 2006*	8.5 M (3.6%)	5.0 M (4.7%)	5.1%	3.6%	2.6%
Prevalence, AP, 2006*	10.2 M (4.6%)	4.7 M (4.6%)	4.7%	4.0%	2.9%
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, 2006 CHD†	425.4 K	224.5 K	196.4 K	22.7 K	N/A
Mortality, 2006 MI†	141.5 K	76.1 K	66.9 K	7.4 K	N/A
Stroke					
Prevalence, 2006*	6.4 M (2.9%)	2.5 M (2.5%)	2.3%	3.8%	2.8%
New and recurrent strokes†	795.0 K	370.0 K	325.0 K	45.0 K	N/A
Mortality, 2006†	137.1 K	54.5 K	45.2 K	7.4 K	N/A
HBP					
Prevalence, 2006*	74.5 M (33.6%)	35.7 M (34.4%)	34.3%	43.0%	25.9%
Mortality, 2006†	56.6 K	24.4 K	17.6 K	6.1 K	N/A
HF					
Prevalence, 2006*	5.8 M (2.6%)	3.1 M (3.1%)	3.2%	3.0%	1.7%
Mortality, 2006†¶	282.8 K	123.6 K	110.3 K	10.9 K	N/A
Smoking					
Prevalence, 2008#	46.0 M (20.6%)	24.8 M (23.1%)	23.5%	25.6%	N/A
Blood cholesterol					
Prevalence, 2006					
Total cholesterol ≥200 mg/dL*	102.2 M (46.8%)	47.7 M (45.2%)	45.0%	40.2%	51.1%
Total cholesterol ≥240 mg/dL*	35.7 M (16.2%)	15.9 M (15.0%)	15.3%	10.9%	16.8%
LDL cholesterol ≥130 mg/dL*	71.2 M (32.6%)	34.9 M (33.1%)	31.5%	34.4%	42.7%
HDL cholesterol <40 mg/dL*	35.1 M (16.2%)	26.4 M (25.0%)	25.4%	14.7%	29.3%
PA**					
Prevalence, 2008#	32.5%	34.8%	N/A	N/A	N/A
Overweight and obesity					
Prevalence, 2006					
Overweight and obesity, BMI ≥25.0 kg/m^2*	144.1 M (66.3%)	75.5 M (71.7%)	71.4%	71.4%	75.1%
Obesity, BMI ≥30.0 kg/m^2*	71.6 M (32.9%)	33.6 M (31.8%)	31.6%	35.2%	29.1%
Diabetes mellitus					
Prevalence, 2006					
Physician-diagnosed diabetes*	17.2 M (7.7%)	7.9 M (7.6%)	6.4%	12.8%	11.8%
Undiagnosed diabetes*	6.1 M (2.8%)	3.8 M (3.8%)	3.7%	3.8%	3.2%
Prediabetes*	63.2 M (29.0%)	37.5 M (35.9%)	35.9%	26.4%	33.3%
Incidence, diagnosed diabetes*	1.6 M	N/A	N/A	N/A	N/A
Mortality, 2006†	72.4 K	36.0 K	29.1 K	5.8 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; mg/dL, milligrams per deciliter; LDL, low-density lipoprotein; HDL, high-density lipoprotein; PA, physical activity; BMI, body mass index; and kg/m^2, kilograms per meter squared.

*Age ≥20 years.

†All ages.

‡New and recurrent MI and fatal CHD.

§Age ≥35 years.

‖Age ≥45 years.

¶Any mentions.

#Age ≥18 years.

**Regular leisure-time PA.

††Hispanic.

Sources: See summary tables for each chapter in this update. For data on men in other ethnic groups, see other chapters and Statistical Fact Sheets.[1]

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Table 21-2. 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, 2006*	81.1 M (36.9%)	42.1 M (35.7%)	34.4%	46.9%	34.5%
Mortality, 2006†	831.3 K	432.7 K	372.8 K	50.8 K	N/A
CHD					
Prevalence, CHD, 2006*	17.6 M (7.9%)	8.4 M (7.0%)	6.9%	8.8%	6.6%
Prevalence, MI, 2006*	8.5 M (3.6%)	3.5 M (2.6%)	2.6%	2.9%	2.0%
Prevalence, AP, 2006*	10.2 M (4.6%)	5.5 M (4.6%)	4.5%	5.4%	4.8%
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, 2006 CHD†	425.4 K	200.9 K	175.0 K	21.8 K	N/A
Mortality, 2006 MI†	141.5 K	65.4 K	56.6 K	7.4 K	N/A
Stroke					
Prevalence, 2006*	6.4 M (2.9%)	3.9 M (3.2%)	3.1%	4.3%	3.1%
New and recurrent strokes†	795.0 K	425.0 K	365.0 K	60.0 K	N/A
Mortality, 2006†	137.1 K	82.6 K	70.7 K	9.6 K	N/A
HBP					
Prevalence, 2006*	74.5 M (33.6%)	38.8 M (32.6%)	31.1%	44.8%	31.6%
Mortality, 2006†	56.6 K	32.2 K	24.9 K	6.5 K	N/A
HF					
Prevalence, 2006*	5.8 M (2.6%)	2.7 M (2.1%)	2.1%	3.6%	1.8%
Mortality, 2006†¶	282.8 K	159.2 K	142.4 K	14.2 K	N/A
Smoking					
Prevalence, 2008#	46.0 M (20.6%)	21.1 M (18.3%)	20.6%	17.8%	N/A
Blood cholesterol					
Prevalence, 2006					
Total cholesterol ≥200 mg/dL*	102.2 M (46.8%)	54.5 M (47.9%)	48.7%	41.8%	49.0%
Total cholesterol ≥240 mg/dL*	35.7 M (16.2%)	19.7 M (17.2%)	18.1%	13.1%	14.3%
LDL cholesterol ≥130 mg/dL*	71.2 M (32.6%)	36.3 M (32.0%)	33.8%	28.6%	30.4%
HDL cholesterol <40 mg/dL*	35.1 M (16.2%)	8.7 M (7.9%)	7.9%	6.5%	11.7%
PA**					
Prevalence, 2008#	32.5%	30.6%	N/A	N/A	N/A
Overweight and obesity					
Prevalence, 2006					
Overweight and obesity, BMI ≥25.0 kg/m^2*	144.1 M (66.3%)	68.6 M (61.0%)	57.5%	79.6%	74.1%
Obesity, BMI ≥30.0 kg/m^2*	71.6 M (32.9%)	38.0 M (34.0%)	31.3%	53.2%	41.8%
Diabetes mellitus					
Prevalence, 2006					
Physician-diagnosed diabetes*	17.2 M (7.7%)	9.3 M (7.9%)	6.4%	13.0%	13.1%
Undiagnosed diabetes*	6.1 M (2.8%)	2.3 M (1.9%)	1.8%	2.3%	3.8%
Prediabetes*	63.2 M (29.0%)	25.7 M (22.2%)	21.7%	22.3%	26.6%
Incidence, diagnosed diabetes*	1.6 M	N/A	N/A	N/A	N/A
Mortality, 2006†	72.4 K	36.4 K	28.1 K	7.0 K	N/A

Abbreviations as in Table 21-1.
*Age ≥20 years.
†All ages.
‡New and recurrent MI and fatal CHD.
§Age ≥35 years.
‖Age ≥45 years.
¶Any mentions.
#Age ≥18 years.
**Regular leisure-time PA.
††Hispanic.
Sources: See summary tables for each chapter in this update. For data on women in other ethnic groups, see other chapters and Statistical Fact Sheets.[2]

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Table 21-3. Ethnic Groups and CVD: At-a-Glance Table

		Whites		Blacks		Mexican Americans		Hispanics/ Latinos		Asians	American Indians/Alaska Natives
Diseases and Risk Factors	Both Sexes	Males	Females	Males	Females	Males	Females	Males	Females	Both Sexes	Both Sexes
Total CVD											
Prevalence, 2006*	81.1 M (36.9%)	38.1%	34.4%	44.6%	46.9%	28.5%	34.5%	N/A	N/A	N/A	N/A
Mortality, 2006†	831.3 K	340.6 K	372.8 K	48.0 K	50.8 K	N/A	N/A	N/A	N/A	N/A	N/A
CHD											
Prevalence, CHD, 2006*	17.6 M (7.9%)	9.4%	6.9%	7.8%	8.8%	5.3%	6.6%	5.7%‖††		2.9‖††	6.6‖††
Prevalence, MI, 2006*	8.5 M (3.6%)	5.1%	2.6%	3.6%	2.9%	2.6%	2.0%	N/A	N/A	N/A	N/A
Prevalence, AP, 2006*	10.2 M (4.6%)	4.7%	4.5%	4.0%	5.4%	2.9%	4.8%	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, 2006†	425.4 K	196.4 K	175.0 K	22.7 K	21.8 K	N/A	N/A	N/A	N/A	N/A	N/A
Mortality, MI, 2006†	141.5 K	66.9 K	56.6 K	7.4 K	7.4 K	N/A	N/A	N/A	N/A	N/A	N/A
Stroke											
Prevalence, 2006*	6.4 M (2.9%)	2.3%	3.1%	3.8%	4.3%	2.8%	3.1%	2.6‖††		1.8%‖††	3.9%‖††
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, 2006†	137.1 K	45.2 K	70.7 K	7.4 K	9.6 K	N/A	N/A	N/A	N/A	N/A	N/A
HBP											
Prevalence, 2006*	74.5 M (33.6%)	34.3%	31.1%	43.0%	44.8%	25.9%	31.6%	21.0%‖††		21.0%‖††	25.3%‖††
Mortality, 2006†	56.6 K	17.6 K	24.9 K	6.1 K	6.5 K	N/A	N/A	N/A	N/A	N/A	N/A
HF											
Prevalence, 2006*	5.8 M (2.6%)	3.2%	2.1%	3.0%	3.6%	1.7%	1.8%	N/A	N/A	N/A	N/A
Mortality, 2006†¶	282.8 K	110.3 K	142.4 K	10.9 K	14.2 K	N/A	N/A	N/A	N/A	N/A	N/A
Smoking											
Prevalence, 2008‖	46.0 M (20.6%)	23.5%	20.6%	25.6%	17.8%	14.9%		20.7%	10.7%	9.9%	24.3%
Blood cholesterol											
Prevalence, 2006											
Total cholesterol 200 ≥mg/dL*	102.2 M (46.8%)	45.0%	48.7%	40.2%	41.8%	51.1%	49.0%	N/A	N/A	N/A	N/A
Total cholesterol ≥240 Mg/dL*	35.7 M (16.2%)	15.3%	18.1%	10.9%	13.1%	16.8%	14.3%	29.9%#		29.2	31.2
LDL cholesterol ≥130 Mg/dL*	71.2 M (32.6%)	31.5%	33.8%	34.4%	28.6%	42.7%	30.4%	N/A	N/A	N/A	N/A
HDL cholesterol <40 Mg/dL*	35.1 M (16.2%)	25.4%	7.9%	14.7%	6.5%	29.3%	11.7%	N/A	N/A	N/A	N/A
PA**											
Prevalence, 2008‖	32.5%	35.9%		24.8%		N/A	N/A	25.2%		N/A	N/A
Overweight and obesity											
Prevalence 2006											
Overweight and obesity, BMI ≥25.0 kg/m^2*	144.1 M (66.3%)	71.4%	57.5%	71.4%	79.6%	75.1%	74.1%	70.3%‖††		40.7%‖††	69.6%‖††
Obesity, BMI ≥30.0 kg/m^2*	71.6 M (32.9%)	31.6%	31.3%	35.2%	53.2%	29.1%	41.8%	31.3%‖††		9.4%‖††	42.1%‖††
Diabetes Mellitus											
Prevalence, 2006											
Physician-diagnosed diabetes*	17.2 M (7.7%)	6.4%	6.4%	12.8%	13.0%	11.8%	13.1%	11.0%‖††		8.0%‖††	15.0%‖††
Undiagnosed diabetes*	6.1 M (2.8%)	3.7%	1.8%	3.8%	2.3%	3.2%	3.8%	N/A	N/A	N/A	N/A
Prediabetes*	63.2 M (29.0%)	35.9%	21.7%	26.4%	22.3%	33.3%	26.6%	N/A	N/A	N/A	N/A
Incidence, diagnosed diabetes*	1.6 M	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Mortality, 2006†	72.4 K	29.1 K	28.1 K	5.8 K	7.0 K	N/A	N/A	N/A	N/A	N/A	N/A

Abbreviations as in Table 21-1.
*Age ≥20 years.
†All ages.
‡New and recurrent MI and fatal CHD.
§Age ≥35 years.
‖Age ≥18 years.
¶Any mention.
#Behavioral Risk Factor Surveillance System.[3]
**Regular leisure-time PA.
††2008, NHIS
Sources: See summary tables for each chapter in this update. For data on other ethnic groups, see other chapters and Statistical Fact Sheets.[4]

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Table 21-4. Children, Youth, and CVD: At-a-Glance Table

				Whites		Blacks		Mexican Americans	
Diseases and Risk Factors	Both Sexes	Total Males	Total Females	Males	Females	Males	Females	Males	Females
Congenital cardiovascular defects									
Mortality, 2006*	3.5 K	2.0 K	1.6 K	1.5 K	1.2 K	0.4 K	0.3 K	N/A	N/A
Mortality, 2006 (age <15 y)	1.9 K	1.1 K	0.9 K	0.8 K	0.6 K	0.2 K	0.2 K	N/A	N/A
Smoking									
High school students, grades 9 to 12									
Current cigarette smoking, 2007	20.0%	21.3%	18.7%	23.8%	22.5%	14.9%	8.4%	18.7%†	14.6%†
Current cigar smoking, 2007	13.6%	19.4%	7.6%	22.0%	7.4%	13.2%	6.7%	16.3%†	9.0%†
Smokeless tobacco use, 2007	7.9%	13.4%	2.3%	18.0%	2.5%	2.0%	0.5%	6.7%†	2.7%†
Blood cholesterol									
Mean total cholesterol, mg/dL									
Ages 4 to 11 y	165.1	164.6	165.6	165.2	166.1	165.6	164.9	161.7	163.1
Ages 12 to 19 y	161.1	157.5	164.8	155.8	166.3	161.3	162.9	158.9	162.3
Mean HDL cholesterol, mg/dL									
Ages 4 to 11 y	55.7	56.7	54.7	55.9	54.0	60.9	58.0	54.5	52.9
Ages 12 to 19 y	52.4	49.4	55.6	47.6	55.2	54.8	57.7	49.6	53.8
Mean LDL cholesterol, mg/dL									
Ages 12 to 19 y	89.2	87.5	90.9	87.1	91.5	89.0	91.5	88.7	91.6
PA‡									
Prevalence, grades 9 to 12, 2007§									
Met currently recommended levels of PA	34.7%	43.7%	25.6%	46.1%	27.9%	41.3%	21.0%	38.6%†	21.9%†
Overweight and obesity									
Prevalence, 2006									
Children and adolescents, ages 2 to 19 y (overweight or obese)	23.5 M (31.9%)	12.3 M (32.7%)	11.2 M (31.0%)	31.9%	29.5%	30.8%	39.2%	40.8%	35.0%
Students in grades 9 to 12§ (overweight only)	15.8%	15.1%	9.6%	15.7%	12.8%	16.6%	21.4%	18.3%†	17.9%†

CVD indicates cardiovascular disease; K, thousands; N/A, data not available; mg/dL, milligrams per deciliter; 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.

§Centers for Disease Control and Prevention.[5]

Sources: See summary tables for related chapters in this update. For more data on congenital defects, see Chapter 7, and our Statistical Fact Sheets.[6]

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22. 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 (2000), so these rates are not affected by changes or differences in the age composition of the population. Unless otherwise noted, all death rates in this publication are age adjusted per 100 000 population and are based on underlying cause of death.
- *Agency for Healthcare Research and Quality (AHRQ)*—A part of the US Department of Health and Human Services, this is the lead agency charged with supporting research designed to improve the quality of health care, 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.
- *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)*—An agency within the US Department of Health and Human Services. The CDC conducts the Behavioral Risk Factor Surveillance System (BRFSS), an ongoing study. The NCHS also conducts or has conducted these studies (among others):
  - National Health Examination Survey (NHES I, 1960 to 1962; NHES II, 1963 to 1965; NHES III, 1966 to 1970)
  - National Health and Nutrition Examination Survey I (NHANES I, 1971 to 1974)
  - National Health and Nutrition Examination Survey II (NHANES II, 1976 to 1980)
  - National Health and Nutrition Examination Survey III (NHANES III, 1988 to 1994)
  - National Health and Nutrition Examination Survey (NHANES, 1999 to . . .) (ongoing)
  - National Health Interview Survey (NHIS) (ongoing)
  - National Home and Hospice Care Survey (ongoing)
  - National Hospital Discharge Survey (NHDS) (ongoing)
  - National Nursing Home Survey (periodic)
- *Centers for Medicare and 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 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 coronary heart disease (I25.1–I25.9).
- *Death rate*—The relative frequency with which death occurs within some specified interval of time in a population. National death rates are computed per 100 000 population. Dividing the total number of deaths by the total population gives a crude death rate for the total population. Rates calculated within specific subgroups, such as age-specific or sex-specific rates, are often more meaningful and informative. They allow well-defined subgroups of the total population to be examined. Unless otherwise stated, all death rates in this publication are age adjusted and are per 100 000 population.
- *Diseases of the circulatory system (ICD codes I00–I99)*—Included as part of what the AHA calls "cardiovascular disease." (See "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), coronary heart disease (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 and Medicaid Services (CMS).
- *Hispanic origin*—In US government statistics, "Hispanic" includes persons 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.

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- *Hospital discharges*—The number of inpatients 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–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 HDL (high-density lipoprotein) 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 World Wide Web site (http://cdc.gov/nchs/), by direct communication with the CDC/NCHS, or from the AHA's National Center Biostatistics Program Coordinator on request. The total number of deaths due 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 approximately 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 to 1997)
  — Cardiovascular Health Study (CHS) (1988 to . . .) (ongoing)
  — Atherosclerosis Risk in Communities (ARIC) study (1985 to . . .) (ongoing)
  — Strong Heart Study (SHS) (1989 to 1992; 1991 to 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

- *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 persons 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 persons

*According to criteria established by the American Heart Association/National Heart, Lung, and Blood Institute and published in *Circulation* (*Circulation*. 2005;112:2735–2752).

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include all persons of Hispanic origin of any race. See "Hispanic origin" in this Glossary.

- *Stroke (ICD-10 codes I60–I69)*—This category includes subarachnoid hemorrhage (I60); intracerebral hemorrhage (I61); other nontraumatic intracranial hemorrhage (I62); cerebral infarction (I63); stroke, not specified as hemorrhage or infarction (I64); occlusion and stenosis of precerebral arteries not resulting in cerebral infarction (I65); occlusion and stenosis of cerebral arteries not resulting in cerebral infarction (I66); other cerebrovascular diseases (I67); cerebrovascular disorders in diseases classified elsewhere (I68); and sequelae of cerebrovascular disease (I69).
- *Total cardiovascular disease (ICD-10 codes I00–I99, 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 contributing 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.

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Correction

In the article by Lloyd-Jones et al, "Heart Disease and Stroke Statistics—2010 Update: A Report From the American Heart Association," which published ahead of print on December 17, 2009, and appears in the February 23, 2010, issue of the journal (*Circulation.* 2010;121:e46–e215), several corrections were needed.

1. On page e46, in the author list, and on page e52, in the Writing Group Disclosure Table, Dr Stafford's name should have read Randall Stafford and Dr Roger's name should have read Véronique L. Roger. We regret these errors.

2. On page e143, in the right column, under the heading "Adults," the fifth bullet, the beginning of the sentence, "Rates of use of any tobacco product among persons 12 years of age in 2006 . . .." should have read, "Rates of use of any tobacco product among persons 12 years of age and older in 2006 . . .."

These corrections have been made to the current online version of the article, which is available at http://circ.ahajournals.org/cgi/reprint/121/7/e46.

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