Circulation
JOURNAL OF THE AMERICAN HEART ASSOCIATION

Heart Disease and Stroke Statistics 2009 Update. A Report From the American Heart Association Statistics Committee and Stroke Statistics Subcommittee

Donald Lloyd-Jones, Robert Adams, Mercedes Carnethon, Giovanni De Simone, T. Bruce Ferguson, Katherine Flegal, Earl Ford, Karen Furie, Alan Go, Kurt Greenlund, Nancy Haase, Susan Hailpern, Michael Ho, Virginia Howard, Brett Kissela, Steven Kittner, Daniel Lackland, Lynda Lisabeth, Ariane Marelli, Mary McDermott, James Meigs, Dariush Mozaffarian, Graham Nichol, Christopher O'Donnell, Veronique Roger, Wayne Rosamond, Ralph Sacco, Paul Sorlie, Randell Stafford, Julia Steinberger, Thomas Thom, Sylvia Wasserthiel-Smoller, Nathan Wong, Judith Wylie-Rosett, Yuling Hong and American Heart Association Statistics Committee and Stroke Statistics Subcommittee

Circulation published online Dec 15, 2008;
DOI: 10.1161/CIRCULATIONAHA.108.191261
Circulation is published by the American Heart Association. 7272 Greenville Avenue, Dallas, TX 72514
 Print ISSN: 0009-7322. Online ISSN: 1524-4539

Heart Disease and Stroke Statistics—2009 Update

A Report From the American Heart Association Statistics Committee and Stroke Statistics Subcommittee

WRITING GROUP MEMBERS

Donald Lloyd-Jones, MD, ScM, FAHA; Robert Adams, MD, FAHA; Mercedes Carnethon, PhD, FAHA; Giovanni De Simone, MD; T. Bruce Ferguson, MD; Katherine Flegal, PhD*; Earl Ford, MD, MPH*; Karen Furie, MD; Alan Go, MD; Kurt Greenlund, PhD*; Nancy Haase; Susan Hailpern, DPH; Michael Ho, MD, PhD; Virginia Howard, PhD, FAHA; Brett Kissela, MD; Steven Kittner, MD; Daniel Lackland, PhD, FAHA; Lynda Lisabeth, PhD; Ariane Marelli, MD; Mary McDermott, MD; James Meigs, MD; Dariush Mozaffarian, MD, PhD, FAHA; Graham Nichol, MD, FAHA; Christopher O'Donnell, MD, MPH, FAHA; Veronique Roger, MD, FAHA; Wayne Rosamond, PhD, FAHA; Ralph Sacco, MD, FAHA; Paul Sorlie, PhD; Randell Stafford, MD; Julia Steinberger, MD, MSC, FAHA; Thomas Thom; Sylvia Wasserthiel-Smoller, PhD; Nathan Wong, PhD; Judith Wylie-Rosett, EdD; Yuling Hong, MD, PhD, FAHA; for the American Heart Association Statistics Committee and Stroke Statistics Subcommittee

Table of Contents

Acknowledgments

We thank Drs Sean Coady, Eric L. Ding, Brian Eigel, Gregg C. Fonarow, Linda Geiss, Cherie James, and Michael Wolz for their valuable comments and contributions. We acknowledge Tim Ander-

*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. The 2009 Update is available online at ●●●●●●●●.

The American Heart Association makes every effort to avoid any actual or potential conflicts of interest that may arise as a result of an outside relationship or a personal, professional, or business interest of a member of the writing panel. Specifically, all members of the writing group are required to complete and submit a Disclosure Questionnaire showing all such relationships that might be perceived as real or potential conflicts of interest.

A single reprint is available by calling 800-242-8721 (US only) or writing the American Heart Association, Public Information, 7272 Greenville Ave, Dallas, TX 75231-4596. Ask for reprint No. ●●●●●●●●. To purchase additional reprints, call 843-216-2533 or e-mail kelle.ramsay@wolterskluwer.com.

(*Circulation.* 2009;119:e1-e161.)

***Circulation* is available at http://circ.ahajournals.org** **DOI: 10.1161/CIRCULATIONAHA.108.191261**

son and Tom Schneider for their editorial contributions, and Karen Modesitt for her administrative assistance.

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. This year's edition includes several areas not covered in previous editions. Below are a few highlights from this year's Update.

Death rates from cardiovascular disease have declined, yet the burden of disease remains high.

- The 2005 overall death rate from cardiovascular disease (CVD) (*International Classification of Diseases 10*, I00–I99) was 278.9 per 100 000. The rates were 324.7 per 100 000 for white males, 438.4 per 100 000 for black males, 230.4 per 100 000 for white females, and 319.7 per 100 000 for black females. From 1995 to 2005, death rates from CVD declined 26.4%. Preliminary mortality data for 2006 show that CVD (I00–I99; Q20–Q28) accounted for 34.2% (829 072) of all 2 425 900 deaths in 2006, or 1 of every 2.9 deaths in the United States.
- On the basis of 2005 mortality rate data, nearly 2400 Americans die of CVD each day—an average of 1 death every 37 seconds. The 2006 overall preliminary death rate from CVD was 262.9. More than 150 000 Americans killed by CVD (I00–I99) in 2005 were <65 years of age. In 2005, 32% of deaths from CVD occurred before the age of 75 years, which is well before the average life expectancy of 77.9 years.
- Coronary heart disease (CHD) caused about 1 of every 5 deaths in the United States in 2005. CHD mortality in 2005 was 445 687. In 2009, an estimated 785 000 Americans will have a new coronary attack, and about 470 000 will have a recurrent attack. It is estimated that an additional 195 000 silent first myocardial infarctions occur each year. About every 25 seconds, an American will have a coronary event, and about every minute someone will die from one.
- Each year, about 795 000 people experience a new or recurrent stroke. About 610 000 of these are first attacks, and 185 000 are recurrent attacks. Preliminary data from 2006 indicate that stroke accounted for about 1 of every 18 deaths in the United States. On average, every 40 seconds someone in the United States has a stroke. From 1995 to 2005, the stroke death rate fell 29.7%, and the actual number of stroke deaths declined 13.5%.
- In 2005, 1 in 8 death certificates (292 214 deaths) in the United States mentioned heart failure.

Control of risk factors remains an issue for many Americans.

- Data from the National Health and Nutrition Examination Survey 2005–2006 found that between 1999–2000 and 2005–2006, mean serum total cholesterol levels in adults ≥20 years of age declined from 204 mg/dL 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 previous 5 years. In 2005–2006, 16% of adults had serum total cholesterol levels of ≥240 mg/dL.
- Despite recommendations that some proportion of activity be vigorous (activity that causes heavy sweating and a large increase in breathing and/or heart rate), 62% of adults >18 years of age who responded to the 2006 National Health Interview Survey reported no vigorous activity lasting >10 minutes per session.
- On the basis of data from the National Health and Nutrition Examination Survey (National Center for Health Statistics), the prevalence of overweight (body mass index–for–age values at or above the 95th percentile) in children 6 to 11 years of age increased from 4.0% in 1971–1974 to 17.0% in 2003–2006. The prevalence of body mass index–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. Among infants and children between the ages of 6 and 23 months, the prevalence of high weight-for-age was 7.2% in 1976–1980 and 11.5% in 2003–2006 (National Health and Nutrition Examination Survey, National Center for Health Statistics).
- Just over 12% of preschool children 2 to 5 years of age were overweight in 2003–2006.

The 2009 Update expands data coverage of congenital cardiovascular defects and nutritional/dietary intake and adds a new chapter on epidemiology and statistics of subclinical atherosclerosis and a subsection on family history of CVD.

Several chapters and sections that have been added or revised for this year's Update merit specific mention. First, we have added a new chapter (Chapter 3) that describes the epidemiology of subclinical atherosclerosis. It has been known for decades that atherosclerosis, the underlying cause of the majority of clinical CVD events, is typically present for decades before the onset of a clinical CVD event or symptoms. As discussed in Chapters 2 and 4, the initial manifestation of clinical atherosclerotic CVD too often is a fatal event, such as sudden cardiac death, or a devastating nonfatal event, such as a large nonfatal myocardial infarction or a disabling stroke. Advances in imaging technology over the past several decades have made it possible to detect and evaluate the burden of subclinical atherosclerosis in a variety of different vascular beds. Two modalities, ultrafast computed tomography for imaging of coronary artery calcification (CAC) and B-mode ultrasound for measurement of carotid intima-media thickness (IMT), have been studied widely in diverse population samples and have greatly enhanced our understanding of the development and progression of subclinical atherosclerosis, as well as its relationship

to subsequent clinical events. The American Heart Association Statistics Committee felt that, given the extensive literature in this area and the increasing consideration of use of these modalities in clinical practice, it was time to provide a review of the epidemiological data from representative, nonreferral population samples to provide a measure of context for the data on subclinical atherosclerosis in the scientific and lay media.

For example, the National Heart, Lung, and Blood Institute's Coronary Artery Risk Development in Young Adults (CARDIA) study and Multi-Ethnic Study of Atherosclerosis (MESA) have helped to define age-, sex-, and race-specific levels of CAC in a diverse population. In younger adults in CARDIA, 33 to 45 years of age, 15.0% of men and 5.1% of women already had CAC, and 1.6% had a CAC score >100. Among older adults in MESA, the prevalence and 75th percentile levels of CAC were highest in white men and lowest in black and Hispanic women, as shown in Table 3-1 in Chapter 3. Significant ethnic differences persisted after adjustment for risk factors, with the relative risk of having CAC being 22% lower in blacks, 15% lower in Hispanics, and 8% lower in Chinese, as compared with whites. Longitudinal data from MESA also highlight the risks associated with the presence and extent of CAC. Chart 3-3 in Chapter 3 shows the relative risks or hazard ratios 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 were approximately 4 times more likely and those with CAC scores >100 were 7 to 10 times more likely to suffer a coronary event than those without CAC.

Carotid IMT, in the absence of frank atherosclerotic plaque, is thought to represent an earlier and more continuous manifestation of atherosclerosis than CAC. Analyses from the Bogalusa Heart Study, CARDIA, MESA, and the Cardiovascular Health Study have helped to describe the epidemiology of carotid IMT across the spectra of age, sex, and race. Concurrent levels of risk factors in young adulthood and early levels of risk factors, even those measured in people 4 to 17 years of age, were significantly associated with carotid IMT at a mean age of 32 years. Higher body mass index and low-density lipoprotein cholesterol levels measured at 4 to 17 years of age were associated with increased risk for being above the 75th percentile for carotid IMT later on in young adulthood. Higher systolic blood pressure and low-density lipoprotein cholesterol and lower high-density lipoprotein cholesterol in young adulthood were also associated with having high carotid IMT. These data highlight the importance of adverse risk factor levels and obesity in early childhood and young adulthood in the early development of atherosclerosis. In the Cardiovascular Health Study, among older Americans, 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 that of 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. These data should help to provide some context for physicians and patients to help understand the evolving roles of subclinical atherosclerosis imaging in research and clinical practice.

As in prior years, we continue to highlight (in Chapter 2) the importance of maintaining low risk factor burden through young adulthood to middle and older ages. An extensive body of literature has demonstrated that individuals who survive to middle age (eg, age 50) without developing traditional CVD risk factors, such as hypercholesterolemia, hypertension, diabetes, or smoking, enjoy a broad array of health benefits, including substantially greater longevity, substantially reduced short- and long-term and remaining lifetime risks for CVD events even in the face of greater longevity, lower risks for both CVD death and non-CVD death, better health-related quality of life in older age, and substantially reduced total and annual Medicare expenditures.

A new section in Chapter 2 also highlights some of the increasing knowledge available about the complex association between family history of CVD and future risk for CVD among offspring and siblings. In future updates, we anticipate including greatly expanded information and discussion of results from genetic studies that may help elucidate novel underlying mechanisms and pathways of atherosclerosis and CVD development.

The chapter on congenital cardiovascular disease (Chapter 7) has been completely revised to provide updated and more useful information. Whereas surveillance for congenital heart defects is incomplete, these data reflect more contemporary estimates and represent the best available data. For example, on the basis of present estimates, 9 congenital heart defects per 1000 live births, or 36 000 infants born with congenital heart defects, are expected in the United States per year. 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.

We have substantially revised and updated the chapter (Chapter 17) describing current nutritional intake data, trends and changes in intakes, estimated effects on cardiovascular risk factors and cardiovascular outcomes, and current costs and trends for all foods. New tables and charts added to the chapter this year include: Table 17-1, on dietary consumption by US adults (>20 years of age) of selected foods and nutrients related to cardiometabolic health; Table 17-2, on dietary consumption by US children and teenagers of selected foods and nutrients related to cardiometabolic health; Chart 17-1, on age-adjusted trends in macronutrients and total calories consumed by US adults (20 to 74 years of age); Chart 17-2, on per capita calories consumed from different beverages by US adults (≥19 years of age); and Chart 17-3, on total US food expenditures away from home and at home.

Reporting and monitoring quality-of-care measures stratified by patient's race/ethnicity and sex are important steps toward addressing disparities in health care through organizational quality improvement. In Chapter 18, new data on quality of care and quality-of-care measures stratified by race/ethnicity and sex, are reported for hospitals participating in Get With The Guidelines from January 1, 2007, through December 31, 2007 (Tables 18-3, 18-9, and 18-10) for the first time in our annual Statistics Update.

Other new data that are of note in this year's Update include:

- The 10 leading diagnoses from the National Hospital Discharge Survey (Chapter 2).
- Extent of awareness, treatment, and control of high blood pressure, by race/ethnicity and sex (Chapter 6).
- Trends in the prevalence of total serum cholesterol in adults ≥20 years of age, by sex and race/ethnicity (Chapter 11).
- Prevalence of students in grades 9 through 12 who did not meet currently recommended levels of moderate-to-vigorous physical activity during the past 7 days, by race/ethnicity and sex (Chapter 12).
- Prevalence of children 6 to 19 years of age who attained sufficient moderate-to-vigorous physical activity to meet public health recommendations of ≥60 minutes per day on ≥5 of 7 days, by sex and age (Chapter 12).
- Trends in diabetes prevalence in adults ≥20 years of age, by sex (Chapter 14).
- Number of surgical procedures in the 10 leading diagnostic groups (Chapter 19).
- Direct costs of the 10 leading diagnostic groups (Chapter 20).

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 2006 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/nvsr56/nvsr56_16.pdf.

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

Donald Lloyd-Jones, MD, ScM, FAHA
Nancy Haase
Yuling Hong, MD, PhD, FAHA
On behalf of the American Heart Association Heart Disease and Stroke Statistics Writing Group

Disclosures

Writing Group Disclosures

Writing Group Member	Employment	Research Grant	Other Research Support	Speakers' Bureau/Honoraria	Expert Witness	Ownership Interest	Consultant/ Advisory Board	Other
Donald Lloyd Jones	Northwestern	NIH/NHLBI†	None	Pfizer* (educational honoraria)	None	None	Abbott*	None
Robert Adams	Medical University South Carolina	NHLBI†	Duke Endowment, Health Sciences South Carolina*	Boehringer Ingleheim†; Genentech*; sanofi-aventis*	None	REACHCAll Inc Telemedicine System†	Boehringer Ingelheim*	None
Mercedes Carnethon	Northwestern University	None	None	Community Health Plan of Seattle*	None	None	None	None
Giovanni de Simone	Federico II University Hospital	Ministry of Research, Italy*	None	None	None	None	None	None
T. Bruce Ferguson	Brody School of Medicine at ECU	Brody School of Medicine at ECU†	None	None	None	None	None	None
Katherine Flegal	Centers for Disease Control and Prevention	None	None	None	None	None	None	None
Earl Ford	Centers for Disease Control and Prevention	None	None	None	None	None	None	None
Karen Furie	Massachusetts General Hospital	NINDS*; AHA*; Bugher*	Deane Institute for Integrative Research in Stroke and Atrial Fibrillation*	None	None	None	GE Healthcare*; Novartis Advisory Board*	None
Alan Go	The Permanente Medical Group	Amgen†; Site PI for a clinical trial sponsored by Johnson & Johnson†	None	None	None	None	None	None
Kurt Greenlund	Centers for Disease Control and Prevention	None	None	None	None	None	None	None
Nancy Haase	American Heart Association	None	None	None	None	None	None	None
Susan Hailpern	Northrop Grumman	None	None	None	None	None	None	None

(*Continued*)

Writing Group Disclosures, *Continued*

Writing Group Member	Employment	Research Grant	Other Research Support	Speakers' Bureau/Honoraria	Expert Witness	Ownership Interest	Consultant/ Advisory Board	Other
P. Michael Ho	Denver VA Medical Center/University of Colorado Denver Medical School and Healthcare	American Heart Association†; Colorado Department of Public Health and Environment†; NHLBI†; VA Research and Development†	None	Novartis*	None	None	None	None
Yuling Hong	American Heart Association	None	None	None	None	None	None	None
Virginia Howard	University of Alabama at Birmingham	Co-investigator, Etiology of Geographic and Racial Differences in Stroke (REGARDS) NIH/NINDS U01 NS041588†; Carotid Revascularization Endarterectomy vs. Stenting Trial (CREST) NIH/NINDS R01 NS 38384†	None	None	None	None	None	None
Brett Kissela	University of Cincinnati	NIH-NINDS R-01 NS30678, "Hemorrhagic and Ischemic Strokes Among Blacks and Whites"†; NIH-NINDS R-01 NS039987, "Siblings with Ischemic Stoke Study (SWISS)" (James Meschia, PI)†; NIH-NINDS U-01 NS041588, "Etiology of Geographic and Racial Differences in Stroke"; (REasons for Geographic and Racial Differences in Stroke, or REGARDS Study) (George Howard, PI)†	None	Boehringer-Ingelheim†	Has served as an expert witness and performed record review for medicolegal cases related to stroke*	None	Advisor to Northstar Neuroscience, Inc (without pay)*	None
Steven Kittner	University of Maryland School of Medicine/Baltimore Department of Veterans Affairs Medical Center	AHA Grant-in-Aid†; NIH grant†	None	Grand Rounds presentations at a variety of medical institutions on topics relating to stroke epidemiology and prevention*	None	None	None	None
Daniel Lackland	Medical University of South Carolina	NHLBI*; DOE*; Health Science South Carolina*	None	Merck*; Novartis*; sanofi-aventis*	None	None	None	None
Lynda Lisabeth	University of Michigan	None	None	None	None	None	None	None
Ariane Marelli	McGill University Health Center	Heart and Stroke Foundation of Canada†	None	None	None	None	None	None
Mary McDermott	Northwestern University's Feinberg School of Medicine	ALL NIH/NHLBI R01-HL073351-01-A1†; R01-HL076298-01†; R01-HL073912-01A2†; K12- HL083790-01†; R01 HL083064†; R01 HL088589† (PI on all); CO-I N01-HC-65236* (PI Daviglus)	None	None	None	None	None	None
James Meigs	Massachusetts General Hospital	None	None	None	None	None	None	None

(*Continued*)

Writing Group Disclosures, *Continued*

Writing Group Member	Employment	Research Grant	Other Research Support	Speakers' Bureau/Honoraria	Expert Witness	Ownership Interest	Consultant/ Advisory Board	Other
Dariush Mozaffarian	Brigham and Women's Hospital, Harvard Medical School	NHLBI† and NIEHS† (K08 HL 075628-01, R01 HL 085710-01, R01 ES 014433-01A2†; Searle Scholar Award grant from the Searle Funds at The Chicago Community Trust†; Genes and Environment Initiative from the Harvard School of Public Health†; the Gates Foundation/World Health Organization Global Burden of Diseases, Injuries, and Risk Factors Study†; GlaxoSmithKline†; Sigma Tau†; Pronova for an investigator-initiated trial†	None	Associations and universities for speaking and reviewing on topics related to diet and cardiovascular disease, including from the US Food and Drug Administration*; Food and Agriculture Organization of the United Nations*; World Health Organization*; American Diabetes Association*; American Dietetic Association*; American Oil Chemists Society*; National Lipid Association*; Institute of Food Technologists*; International Life Sciences Institute*; Medical Society of Delaware*; Johns Hopkins University*; Columbia University*; University of New Hampshire*; University of Guelph*; and Washington University*	None	None	None	None
Graham Nichol	University of Washington	NHLBI,† Bethesda, Md, Grantee, co-PI, Resuscitation Outcomes Consortium Data Coordinating Center; Canadian Institutes of Health Research*; Medtronic Inc.,* Grantee, Co-investigator, Resynchronization in Advanced Failure Trial (RAFT); Asmund S. Laerdal Foundation for Acute Medicine,* Stavanger, Norway, PI, Randomized Trial of CPR Training Aid in Community	Equipment donation of training aids for overseas medical mission, Laerdal Inc. (2006)*; equipment donation of monitors/ defibrillators for overseas medical mission, Physio-Control Inc. (2007)*; Equipment donation of training materials for overseas medical mission, Channing-Bete Inc. (2007)*	None	None	None	Consultant, Northfield Laboratories*; Consultant, Paracor Medical Inc.*; Member of Board of Directors, Medic One Foundation*	None
Christopher O'Donnell	National Heart, Lung, and Blood Institute; Massachusetts General Hospital	None	None	None	None	None	None	None
Veronique Roger	Mayo Clinic Health Care Center	None	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	Boehringer Ingelheim*; sanofi-aventis*	None	None	Boehringer Ingelheim for design of clinical trial on stroke prevention†; GlaxoSmithKline*; sanofi-aventis*	None
Paul Sorlie	National Heart, Lung, and Blood Institute, NIH	None	None	None	None	None	None	None
Randell Stafford	Stanford University	Procter & Gamble†	None	Bayer*	None	None	None	None
Julia Steinberger	University of Minnesota	None	None	None	None	None	None	None Thom

(*Continued*)

Writing Group Disclosures, *Continued*

Writing Group Member	Employment	Research Grant	Other Research Support	Speakers' Bureau/Honoraria	Expert Witness	Ownership Interest	Consultant/ Advisory Board	Other
Thomas Thom	National Heart, Lung, and Blood Institute, NIH, DHHS, US Government	None	None	None	None	None	None	None
Sylvia Wasserthiel-Smoller	Albert Einstein College of Medicine	NIH/NHLBI Hispanic Community Health Study†; Women's Health Initiative†; Women's Health Initiative Memory Study†	None	None	None	None	None	None
Nathan Wong	University of California, Irvine	Merck†; Pfizer†	None	Novartis*; Takeda†	None	None	Merck*	None
Judith Wylie-Rosett	Albert Einstein College of Medicine	None	None	VA, 1199*	None	None	Mt. Sinai Medical Center Diabetes Prevention*; Yale School of Nursing*	None

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

*Modest.

†Significant.

1. About These Statistics

The American Heart Association (AHA) works with the Centers for Disease Control and Prevention's (CDC's) National Center for Health Statistics (NCHS); the National Heart, Lung, and Blood Institute (NHLBI); the National Institute of Neurological Disorders and Stroke (NINDS); and other government agencies to derive the annual statistics in this 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 Manhattan Stroke Study (GCNKSS)—stroke incidence rates and outcomes within a biracial population
- Medical Expenditure Panel Survey (MEPS)—data on specific health services that Americans use, how frequently they use them, the cost of these services, and how the costs are paid
- National Health and Nutrition Examination Survey (NHANES)—disease and risk factor prevalence and nutrition statistics
- National Health Interview Survey (NHIS)—disease and risk factor prevalence
- National Hospital Discharge Survey (NHDS)—hospital inpatient discharges and procedures (discharged alive, dead, or status unknown)
- National Ambulatory Medical Care Survey (NAMCS)—physician office visits
- National Hospital Ambulatory Medical Care Survey (NHAMCS)—hospital outpatient and emergency department visits
- National Inpatient Sample (NIS) of the Agency for Health Research and Quality (AHRQ)—hospital inpatient discharges, procedures, and charges
- National Nursing Home Survey (NNHS)—nursing home visits
- National Vital Statistics—national and state mortality data
- Youth Risk Behavior Surveillance (YRBS) (CDC)—trends for 6 categories of priority health-risk behaviors in youth and young adults
- World Health Organization (WHO)—mortality rates by country

Abbreviations Used in Chapter 1

AHA	American Heart Association
AHRQ	Agency for Health 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
FHS	Framingham Heart Study
GCNKSS	Greater Cincinnati/Northern Kentucky Stroke Study
HF	Heart Failure
ICD	International Classification of Diseases
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
WHO	World Health Organization
YRBS	Youth Risk Behavior Surveillance

See Glossary (Chapter 22) for explanation of terms.

Disease Prevalence

Prevalence is an estimate of how many people have a disease at a given point or period in time. The NCHS conducts health examination and health interview surveys that provide estimates of the prevalence of diseases and risk factors. In this Update, the health interview part of the NHANES is used for the prevalence of cardiovascular diseases (CVD). 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 of total CVD to include 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 more realistic estimate of burden. Most estimates based on NHANES prevalence rates are based on data collected from 2005 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 using more recent census population estimates cannot be used to evaluate possible trends in prevalence. Trends can only be evaluated by comparing prevalence rates estimated from surveys conducted in different years.

Risk Factor Prevalence

The NHANES 2005–2006 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 Youth Risk Factor Surveillance System.

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, the Cardiovascular Health Study (CHS), all conducted by the NHLBI, and the Greater Cincinnati/Northern Kentucky Stroke Study (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. "Total-mention" mortality is the number of death certificates in a year that mention the given disease classification either as the underlying cause or as a contributing cause. For many deaths classified as attributable to CVD, selection of the most likely single underlying cause can be difficult when several major comorbidities are present, as is often the case in the elderly population. It is, therefore, useful to know the extent of mortality from a given cause, regardless of whether it is the underlying cause or a contributing cause—ie, its "total mentions." The number of total-mention deaths in 2005 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. That number is referred to as "mortality." Mortality is followed by the number for "total-mention mortality." All other numbers or rates of deaths in the Update refer to the given disease as the underlying cause. One exception is Chapter 9, where total-mention HF mortality statistics are presented.

National and state mortality data presented by the underlying cause of death were computed from the Data Warehouse mortality tables of the NCHS Web site or the CDC compressed file. Total-mention numbers of deaths were tabulated from the electronic mortality files of the NCHS Web site. Note that any mortality data for 2006 are preliminary.

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 2005 were used in the computation of death rate data. The Census Bureau 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 the all-listed procedures (primary plus secondary). These estimates are from the NHDS of the NCHS unless otherwise noted. Ambulatory care visit data from NHAMCS include patient visits to physicians' offices and hospital emergency and outpatient departments. Ambulatory care visit data reflect the first-listed (primary) diagnosis. These estimates are from the 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 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 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 2005. For disease and risk factor *prevalence*, most rates in this report are calculated from the 2005–2006 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 relatively high prevalence in the institutionalized population. The numbers and rates of *hospital inpatient discharges* for the United States are for 2005 and 2006. Numbers of visits to *physician offices*, *hospital emergency departments*, and *out-*

patient departments are for 2006. Except as noted, *economic cost* estimates are projected to 2009.

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 and 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 Web site, http://www.americanheart.org/statistics, and in the journal *Circulation*.

References

1. US Census Bureau population estimates. Available at: http://www.census.gov/popest/national/asrh/2006_nat_res.html. Accessed April 16, 2008.
2. National Center for Health Statistics. *Health, United States, 2007, With Chartbook on Trends in the Health of Americans*. Hyattsville, Md: National Center for Health Statistics; 2007. Available at: http://www.cdc.gov/nchs/hus.htm. Accessed May 5, 2008.
3. National Center for Health Statistics, Centers for Medicare and Medicaid Services. *International Classification of Diseases, Ninth Revision. Clinical Modification (ICD 9 CM)*. Hyattsville, Md: National Center for Health Statistics; 1978.
4. Anderson RN, Rosenberg HM. Age standardization of death rates: implementation of the year 2000 standard. *Natl Vital Stat Rep.* 1998;47:1–16, 20.
5. World Health Organization. *World Health Statistics Annual*. Geneva, Switzerland: World Health Organization; 1998.

2. Cardiovascular Diseases

ICD-9 390–459, 745–747, ICD-10 I00–I99, Q20–Q28; see Glossary (Chapter 21) for details and definitions. See Tables 2-1 through 2-4 and Charts 2-1 through 2-21.

Abbreviations Used in Chapter 2

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)
CDC	Centers for Disease Control and Prevention
CHD	coronary heart disease
CHF	congestive heart failure
CHS	Cardiovascular Health Study
CLRD	chronic lower respiratory disease
CVD	cardiovascular disease
DM	diabetes mellitus
ED	emergency department
EMS	emergency medical services
FHS	Framingham Heart Study
HBP	high blood pressure
HD	heart disease
HF	heart failure
HIV	human immunodeficiency virus
ICD	International Classification of Diseases
kg/m2	kilograms/meter2
MEPS	Medical Expenditure Panel Survey
MI	myocardial infarction
mg/dL	milligrams per deciliter
mm Hg	millimeter of mercury
MRFIT	Multiple Risk Factor Intervention Trial
NAMCS	National Ambulatory Medical Care Survey
NCHS	National Center for Health Statistics
NH	non-Hispanic
NHAMCS	National Hospital Ambulatory Medical Care Survey
NHANES	National Health and Nutrition Examination Survey
NHDS	National Hospital Discharge Survey
NHES	National Health Examination Survey
NHIS	National Health Interview Survey
NHLBI	National Heart, Lung, and Blood Institute
NIS	Nationwide Inpatient Sample
NNHS	National Nursing Home Survey
PA	physical activity
VF	ventricular fibrillation

Prevalence

An estimated 80 000 000 American adults (approximately 1 in 3) have 1 or more types of CVD. Of these, 38 100 000 are estimated to be ≥60 years of age (extrapolated to 2006 from NCHS/NHANES 2005–2006 data). 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 blood pressure (HBP)—73 600 000. (Defined as systolic pressure ≥140 mm Hg or diastolic pressure ≥90 mm Hg, use of antihypertensive medication, or being told at least twice by a physician or other health professional that one has HBP.)
- Coronary heart disease (CHD)—16 800 000.
  - — Myocardial infarction (MI; heart attack)—7 900 000.
  - — Angina pectoris (AP; chest pain)—9 800 000.
- Heart failure (HF)—5 700 000.
- Stroke—6 500 000.
- Congenital cardiovascular defects—650 000 to 1 300 000 (see Chapter 7).

The following prevalence estimates are for 2007 from NHIS, NCHS for people ≥18 years of age[1]:

- Among whites only, 11.4% have heart disease (HD), 6.1% have CHD, 22.2% have hypertension, and 2.2% have had a stroke.
- Among blacks or African Americans, 10.2% have HD, 6.0% have CHD, 31.7% have hypertension, and 3.7% have had a stroke.
- Among Hispanics or Latinos, 8.8% have HD, 5.7% have CHD, 20.6% have hypertension, and 2.5% have had a stroke.
- Among Asians, 6.9% have HD, 4.3% have CHD, 19.5% have hypertension, and 2.6% have had a stroke.
- Among Native Hawaiians or other Pacific Islanders, HD, CHD, and stroke numbers are suppressed owing to large relative standard error, and 28.5%* have hypertension. Among American Indians or Alaska Natives, 10.5% have HD, 5.6%* have CHD, and 25.5% have hypertension, and stroke numbers are suppressed owing to large relative standard error.
- Asian Indian adults (9%) are approximately 2 times as likely as Korean adults (4%) to have ever been told they have HD.[2]

Incidence

- On the basis of the NHLBI's Framingham Heart Study (FHS) original and offspring cohort data from 1980 to 2003[3]:
  - — The average annual rates of first cardiovascular events rise from 3 per 1000 men at 35 to 44 years of age to 74 per 1000 men at 85 to 94 years of age. For women,

*Figure considered unreliable.

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 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 CVD 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).

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 (includes congenital cardiovascular defects) accounted for 35.3% (864 480) of all 2 448 017 deaths in 2005 or 1 of every 2.8 deaths in the United States. CVD total mentions (1 372 000 deaths in 2005) constituted approximately 56% of all deaths that year (NHLBI; NCHS public use data files).[5] Preliminary 2006 mortality (I00–I99, Q20–Q28) was 829 072. The preliminary death rate was 262.9 (NCHS).
- In every year since 1900, except 1918, CVD accounted for more deaths than any other major cause of death in the United States.[6,7]
- Nearly 2400 Americans die of CVD each day, an average of 1 death every 37 seconds. CVD claims approximately as many lives each year as cancer, CLRD, accidents, and diabetes mellitus (DM) combined.[5]
- The 2005 overall death rate due to CVD (I00–I99) was 278.9. The rates were 324.7 for white males, 438.4 for black males, 230.4 for white females, and 319.7 for black females. From 1995 to 2005, death rates due to CVD (ICD-10 I00–I99) declined 26.4%. In the same 10-year period, the actual number of CVD deaths per year declined 9.6%.[5]
- Among other causes of death in 2005, cancer caused 559 312 deaths; accidents, 117 809; Alzheimer's disease, 71 599; and HIV (human immunodeficiency virus)/AIDS (acquired immune deficiency syndrome), 12 543.[5]
- The 2005 CVD (I00–I99) death rates were 331.1 for males and 237.1 for females. Death rates for cancer (malignant neoplasms) were 225.1 for males and 155.6 for females. Breast cancer claimed the lives of 41 116 females in 2005; lung cancer claimed 69 105. Death rates for females were 24.1 for breast cancer and 40.5 for lung cancer. One in 30 female deaths was of breast cancer, whereas 1 in 6 was of CHD. For comparison, 1 in 4.6 females died of cancer, whereas 1 in 2.7 died of CVD (I00–I99, Q20–Q28). On the basis of 2005 mortality data, CVD caused approximately 1 death per minute among females, or approximately 455 000 female deaths in 2005. That represents nearly as many female lives as were claimed by cancer, CLRD, Alzheimer's disease, accidents, and DM combined.[5]
- Nearly 151 000 Americans died of CVD (I00–I99) in 2005 who were <65 years of age, and 32% of deaths due to CVD occurred before the age of 75 years, which is well before the average life expectancy of 77.8 years.[5] Preliminary data for 2006 gave an estimated 78.1 average years of life expectancy.[8]
- In 2005, death rates for diseases of the heart in American Indians or Alaska Natives were 173.2 for males and 115.9 for females; for Asians or Pacific Islanders, they were 141.1 for males and 91.9 for females; and for Hispanics or Latinos, they were 192.4 for males and 129.1 for females.[9]
- 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 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 DM, and 0.7% for HIV.[10]
- In 2005, 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).[11]
- 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.[12]
- Between 1980 and 2002, death rates due to HD 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 broken down 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.[13]

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 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.[14] Available epidemiological databases do not adequately

characterize cardiac arrest or the subset of cases that occur with sudden onset. The following information summarizes representative data from several sources in an attempt to characterize the incidence and outcome of out-of-hospital cardiac arrest.

- 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 294 851 (quasi confidence intervals 236 063, 325 007) emergency medical services (EMS)–treated out-of-hospital cardiac arrests annually in the United States (unpublished data, Graham Nichol, MD, May 25, 2008).
- Extrapolation of data from ARIC, CHS, and Framingham suggests that there are 138 000 CHD deaths within 1 hour of symptom onset (personal communication with NHLBI, May 20, 2008).
- Only 33% of those with EMS-treated out-of-hospital cardiac arrest have symptoms within 1 hour of death.[15]
- Approximately 60% of out-of-hospital cardiac deaths are treated by EMS personnel.[16]
- From 20% to 38% of out-of-hospital cardiac arrests have ventricular fibrillation (VF) or ventricular tachycardia as the first recorded rhythm.[17,18]
- 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.[18]
- The median reported survival to hospital discharge after out-of-hospital cardiac arrest with any first recorded rhythm is 7.9%.[19,20]
- The average proportion of cases of out-of-hospital cardiac arrest that receive bystander cardiopulmonary resuscitation is 31.4%[19] (personal communication with Graham Nichol, MD).
- The incidence of lay-responder defibrillation is low (2.05% in 2002) but is increasing over time.[21]
- In 2005, 5003 people died of unintentional choking or suffocation (NCHS).[22]
- A study conducted in New York City found the age-adjusted incidence per 10 000 adults of out-of-hospital cardiac arrest 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.[23]
- Approximately 80% of out-of-hospital cardiac arrests occur in private or residential settings.[24]
- 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.[25–28]

Out-of-Hospital Cardiac Arrest: Children

- The reported incidence of out-of-hospital pediatric cardiac arrest varies widely (from 2.6 to 19.7 annual cases per 100 000).[29]
- There are more than 72 million individuals <18 years of age in the United States[30]; this implies that there are from 1900 to 14 200 pediatric out-of-hospital cardiac arrests annually of all causes (including trauma, sudden infant death syndrome, respiratory causes, cardiovascular causes, and submersion).
- VF is an uncommon cause of cardiac arrest in children but is observed in approximately 5% to 15% of children with out-of-hospital cardiac arrest.[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.[32,33] 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.[34]
- The reported average rate of survival to hospital discharge after pediatric out-of-hospital cardiac arrest is 6.7%.[29]

In-Hospital Cardiac Arrest

- The rates of survival to discharge after in-hospital cardiac arrest are 27% among children and 18% among adults.[34]
- A total of 303 facilities reported 21 748 events to the National Registry for Cardiopulmonary Resuscitation in 2007.
  - Of these, 93% were monitored or witnessed.
  - 17.9% had VF or pulseless ventricular tachycardia as the first recorded rhythm. Of these, 79% received a defibrillation attempt within 3 minutes.

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 automated electrical defibrillator 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.[35]

Awareness of Warning Signs and Risk Factors for CVD

- Surveys conducted by the American Heart Association in 1997, 2000, 2003, and 2006 to evaluate trends in women's awareness, knowledge, and perceptions related to CVD found that in 2006, awareness of HD as the leading cause of death among women was 57%, significantly higher than in prior surveys. Awareness was lower among black and Hispanic women than among white women, and the racial/ethnic difference has not changed appreciably over time. In 2006, more than twice as many women felt uninformed about stroke compared with HD. Hispanic women were more likely than white women to report that there is nothing they can do to keep themselves from getting CVD. The majority of respondents reported confusion related to basic CVD prevention strategies.[36]

- Nearly 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.[37]
- 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 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.[38]

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 increased among successive 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.[39]
- Data from the Chicago Heart Association Detection Project (1967–1973, 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], diabetes, 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.[40,41]
- 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.[42]
- 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.[43]
- Analysis of 5 cross-sectional, nationally representative surveys from NHES 1960–1962 to NHANES 1999–2000 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.[44]
- 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 (see Table 2-4).[45]
- Analysis of >14 000 middle-aged subjects in the ARIC study of 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.[46]

- Data from the Medical Expenditure Panel Survey (MEPS) 2004 Full Year Data File showed that nearly 26 million US adults ≥18 years of age were told by a doctor that they had HD, stroke, or any other heart-related disease[47]:
  - 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).
    - Those ≥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).
- Forty-four percent of 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.[48]

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[49] or a sibling[50] 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 containing traditional risk factors provides modest improvement in the area under the receiver operating curve in the FHS.[49] 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[51] and men.[52]
- Parental history of premature CHD is associated with increased burden of atherosclerosis in the coronary arteries and the abdominal aorta.[53,54]
- In the FHS, a parental history of validated heart failure is associated with a 1.7-fold elevated risk of HF in offspring, after multivariable adjustment.[55]
- A family history of early-onset sudden cardiac death in a first-degree relative is associated with a more than doubled

risk for sudden cardiac death in available case-control studies.[56]

- A recent survey of persons in the United States indicated that most respondents believe that knowing their family history is important for their own health, but few are aware of the specific health information from relatives necessary to develop a family history.[57]
- 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.
- Numerous genomewide genetic association studies are under way to determine the specific genetic variants that may underlie a family history.

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.[45]
- 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.[58]
  - 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) Study and Chicago cohorts 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 risk for CVD mortality, 40% to 60% lower total mortality rates, and 6 to 10 years' greater life expectancy.[41]
- A study of 84 129 women enrolled in the Nurses' Health Study identified 5 healthy lifestyle factors, including absence of current smoking, drinking ½ glass or more of wine per day (or equivalent alcohol consumption), ½ hour or more per day of moderate or vigorous PA, BMI <25 kg/m^2, and dietary score in the top 40% (including 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, risk for CHD over a 14-year period was reduced by 57%; when 4 were present, risk was reduced by 66%; and when all 5 factors were present, risk was reduced by 83%.[59]
- 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.[60]
- 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.[61]
- Seventeen-year mortality data from the NHANES II Mortality Follow-Up Study indicated that the risk 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.[62]
- 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.[63]
— 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).[64]

Hospital Discharges, Ambulatory Care Visits, and Nursing Home Visits

- 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 2005, CVD ranked highest among all disease categories in hospital discharges.[65]
- In 2006, there were 72 151 000 physician office visits, hospital ED visits, and outpatient department visits with a primary diagnosis of CVD (NCHS, NAMCS, and NHAMCS).[66]
- In 2006, there were 4 378 000 visits to EDs with a primary diagnosis of CVD (NCHS, NHAMCS).[67]
- In 2004, 24.7% of nursing home residents ≥65 years of age had a primary diagnosis of CVD at admission. This was the highest disease category for these residents (NCHS, NNHS).[68]
- In 2006, there were 6 633 000 outpatient department visits with a primary diagnosis of CVD (NHAMCS).[69] 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%.[70]
- In 2004, coronary atherosclerosis was 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.[71]
- 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, congestive HF, and stroke were for women. There was no difference between men and women in hospitalizations for cardiac dysrhythmias.[72]
- 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, which was ranked first, the most common heart-related conditions were CHF (No. 2), chest pain (No. 3), hardening of the arteries (No. 4), and heart attack (No. 5), which together accounted for >15% of all admissions through the ED. Stroke and irregular heart beat ranked seventh and eighth, respectively.[73]

Cost

The estimated direct and indirect cost of CVD for 2009 is $475.3 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.[74]

Operations and Procedures

- In 2006, an estimated 7 095 000 inpatient cardiovascular operations and procedures were performed in the United States; 4.0 million were performed on males, and 3.1 million were performed on females (NHDS, NCHS, and NHLBI).

References

1. Pleis JR, Lucus JW. Summary health statistics for U.S. adults: National Health Interview Survey, 2007. *Vital Health Stat 10*. In press. No. 240; provisional report.
2. Barnes PM, Adams PF, Powell-Griner E. *Health Characteristics of the Asian Adult Population: United States, 2004–2006.* Advance Data From Vital and Health Statistics; No. 394. Hyattsville, Md: National Center for Health Statistics; January 22, 2008.
3. National Institutes of Health, National Heart, Lung, and Blood Institute. *Incidence and Prevalence: 2006 Chart Book on Cardiovascular and Lung Diseases.* Bethesda, Md: National Heart, Lung, and Blood Institute; 2006. Available at: http://www.nhlbi.nih.gov/resources/docs/06a_ip_chtbk.pdf. Accessed October 17, 2007.
4. Ali T, Jarvis B, O'Leary M. *Strong Heart Study Data Book: A Report to American Indian Communities.* Rockville, Md: National Institutes of Health, National Heart, Lung, and Blood Institute; November 2001. NIH publication No. 01-3285. Available at: http://www.nhlbi.nih.gov/resources/docs/shs_db.pdf. Accessed October 17, 2007.
5. National Center for Health Statistics, Centers for Disease Control and Prevention. Compressed mortality file: underlying cause of death, 1979 to 2005. Available at: http://wonder.cdc.gov/mortSQL.html. Accessed September 15, 2008.
6. National Center for Health Statistics. HIST290A: deaths for selected causes by 10-year age groups, race, and sex: death registration states, 1900–32, and United States, 1933–98. Available at: http://www.cdc.gov/nchs/datawh/statab/unpubd/mortabs/hist290a.htm. Accessed October 17, 2007.
7. National Center for Health Statistics. GMWK292F: deaths for 358 selected causes by 5-year age groups, race, and sex: United States, 1999–2004. Available at: http://www.cdc.gov/nchs/datawh/statab/unpubd/mortabs/gmwk292_10.htm. Accessed June 15, 2008.

8. Heron MP, Hoyert DL, Xu J, Scott C, Tejada-Vera B. *Deaths: Preliminary Data for 2006.* National Vital Statistics Reports; Vol. 56, No. 16; June 11, 2008. Hyattsville, Md: National Center for Health Statistics; 2008.
9. *Health, United States, 2007: With Chartbook on Trends in the Health of Americans.* Hyattsville, Md: National Center for Health Statistics; 2007. Available at: http://www.cdc.gov/nchs/hus.htm. Accessed June 24, 2008.
10. Anderson RN. *U.S. Decennial Life Tables for 1989–91, Vol. 1, No. 4, United States Life Tables Eliminating Certain Causes of Death.* Hyattsville, Md: National Center for Health Statistics; 1999. Available at: http://www.cdc.gov/nchs/data/lifetables/life89_1_4.pdf. Accessed October 17, 2007.
11. Centers for Disease Control and Prevention, National Center for Injury Prevention and Control. WISQARS leading causes of death reports, 1999–2005. Available at: http://webapp.cdc.gov/sasweb/ncipc/leadcaus10.html. Accessed June 4, 2008.
12. Ford ES, Ajani UA, Croft JB, Critchley JA, Labarthe DR, Kottke TE, Giles WH, Capewell S. Explaining the decrease in U.S. deaths from coronary disease, 1980–2000. *N Engl J Med*. 2007;356:2388–2398.
13. Ford ES, Capewell S. Coronary heart disease mortality among young adults in the U.S. from 1980 through 2002: concealed leveling of mortality rates. *J Am Coll Cardiol*. 2007;50:2128–2132.
14. Jacobs I, Nadkarni V, Bahr J, Berg RA, Billi JE, Bossaert L, Cassan P, Coovadia A, D'Este K, Finn J, Halperin H, Handley A, Herlitz J, Hickey R, Idris A, Kloeck W, Larkin GL, Mancini ME, Mason P, Mears G, Monsieurs K, Montgomery W, Morley P, Nichol G, Nolan J, Okada K, Perlman J, Shuster M, Steen PA, Sterz F, Tibballs J, Timerman S, Truitt T, Zideman D; International Liaison Committee on Resuscitation; American Heart Association; European Resuscitation Council; Australian Resuscitation Council; New Zealand Resuscitation Council; Heart and Stroke Foundation of Canada; InterAmerican Heart Foundation; Resuscitation Councils of Southern Africa; ILCOR Task Force on Cardiac Arrest and Cardiopulmonary Resuscitation Outcomes. Cardiac arrest and cardiopulmonary resuscitation outcome reports: update and simplification of the Utstein templates for resuscitation registries: a statement for healthcare professionals from a task force of the International Liaison Committee on Resuscitation (American Heart Association, European Resuscitation Council, Australian Resuscitation Council, New Zealand Resuscitation Council, Heart and Stroke Foundation of Canada, InterAmerican Heart Foundation, Resuscitation Councils of Southern Africa). *Circulation.* 2004;110:3385–3397.
15. Müller D, Agrawal R, Arntz HR. How sudden is sudden cardiac death? *Circulation*. 2006;114:1146–1150.
16. Chugh SS, Jui J, Gunson K, Stecker EC, John BT, Thompson B, Ilias N, Vickers C, Dogra V, Daya M, Kron J, Zheng ZJ, Mensah G, McAnulty J. Current burden of sudden cardiac death: multiple source surveillance versus retrospective death certificate–based review in a large U.S. community. *J Am Coll Cardiol*. 2004;44:1268–1275.
17. Vaillancourt C, Stiell IG; Canadian Cardiac Outcomes Research Team. Cardiac arrest care and emergency medical services in Canada. *Can J Cardiol*. 2004;20:1081–1090.
18. Cobb LA, Fahrenbruch CE, Olsufka M, Copass MK. Changing incidence of out-of-hospital ventricular fibrillation, 1980–2000. *JAMA*. 2002;288:3008–3013.
19. Nichol G, Stiell IG, Laupacis A, Pham B, De Maio V, Wells GA. A cumulative meta-analysis of the effectiveness of defibrillator-capable emergency medical services for victims of out-of-hospital cardiac arrest. *Ann Emerg Med*. 1999;34(pt 1):517–5525.
20. Nichol G, Thomas E, Callaway CW, Hedges J, Powell JL, Aufderheide TP, Rea T, Lowe R, Brown T, Dreyer J, Davis D, Idris A, Stiell I; Resuscitation Outcomes Consortium Investigators. Regional variation in out-of-hospital cardiac arrest incidence and outcome [published correction appears in *JAMA*. 2008;300:1763]. *JAMA*. 2008;300:1423–1431.
21. Culley LL, Rea TD, Murray JA, Welles B, Fahrenbruch CE, Olsufka M, Eisenberg MS, Copass MK. Public access defibrillation in out-of-hospital cardiac arrest: a community-based study. *Circulation*. 2004;109:1859–1863.
22. Deleted in proof.
23. Galea S, Blaney S, Nandi A, Silverman R, Vlahov D, Foltin G, Kusick M, Turnik M, Richmond N. Explaining racial disparities in incidence of and survival from out-of-hospital cardiac arrest. *Am J Epidemiol*. 2007;166:534–543.
24. Becker L, Eisenberg M, Fahrenbruch C, Cobb L. Public locations of cardiac arrest: implications for public access defibrillation. *Circulation*. 1998;97:2106–2109.
25. Larsen MP, Eisenberg MS, Cummins RO, Hallstrom AP. Predicting survival from out-of-hospital cardiac arrest: a graphic model. *Ann Emerg Med*. 1993;22:1652–1658.
26. Valenzuela TD, Roe DJ, Cretin S, Spaite DW, Larsen MP. Estimating effectiveness of cardiac arrest interventions: a logistic regression survival model. *Circulation*. 1997;96:3308–3313.
27. Swor RA, Jackson RE, Cynar M, Sadler E, Basse E, Boji B, Rivera-Rivera EJ, Maher A, Grubb W, Jacobson R, et al. Bystander CPR, ventricular fibrillation, and survival in witnessed, unmonitored out-of-hospital cardiac arrest. *Ann Emerg Med*. 1995;25:780–784.
28. Holmberg M, Holmberg S, Herlitz J. Incidence, duration and survival of ventricular fibrillation in out-of-hospital cardiac arrest patients in Sweden. *Resuscitation*. 2000;44:7–17.
29. Donoghue A, Nadkarni V, Berg RA, Osmond MH, Wells GA, Nesbitt L, Stiell IG; CanAm Pediatric Cardiac Arrest Investigators. Out-of-hospital pediatric cardiac arrest: an epidemiologic review and assessment of current knowledge. *Ann Emerg Med.* 2005;46:512–522.
30. Monthly Postcensal Resident Population: U.S. Census data. Available at: http://www.census.gov. Accessed June 27, 2007.
31. Mogayzel C, Quan L, Graves JR, Tiedeman D, Fahrenbruch C, Herndon P. Out-of-hospital ventricular fibrillation in children and adolescents: causes and outcomes. *Ann Emerg Med*. 1995;25:484–491.
32. Luckstead EF, Patel DR. Catastrophic pediatric sports injuries. *Pediatr Clin North Am.* 2002;49:581–591.
33. Maron BJ, Gohman TE, Aeppli D. Prevalence of sudden cardiac death during competitive sports activities in Minnesota high school athletes. *J Am Coll Cardiol*. 1998;32:1881–1884.
34. Lotfi K, White L, Rea T, Cobb L, Copass M, Yin L, Becker L, Eisenberg M. Cardiac arrest in schools. *Circulation.* 2007;116:1374–1379.
35. ECC Harris Interactive Poll.
36. Christian AH, Rosamond W, White AR, Mosca L. Nine-year trends and racial and ethnic disparities in women's awareness of heart disease and stroke: an American Heart Association national study. *J Womens Health (Larchmt)*. 2007;16:68–81.
37. Vanhecke TE, Miller WM, Franklin BA, Weber JE, McCullough PA. Awareness, knowledge, and perception of heart disease among adolescents. *Eur J Cardiovasc Prev Rehabil*. 2006;13:718–723.
38. Mosca L, Mochari H, Christian A, Berra K, Taubert K, Mills T, Burdick KA, Simpson SL. National study of women's awareness, preventive action, and barriers to cardiovascular health. *Circulation.* 2006;113:525–534.
39. Centers for Disease Control and Prevention (CDC). Racial/ethnic and socioeconomic disparities in multiple risk factors for heart disease and stroke: United States, 2003. *MMWR Morb Mortal Wkly Rep*. 2005;54:113–117.
40. Daviglus ML, Stamler J, Pirzada A, Yan LL, Garside DB, Liu K, Wang R, Dyer AR, Lloyd-Jones DM, Greenland P. Favorable cardiovascular risk profile in young women and long-term risk of cardiovascular and all-cause mortality. *JAMA*. 2004;292:1588–1592.
41. Stamler J, Stamler R, Neaton JD, Wentworth D, Daviglus ML, Garside D, Dyer AR, Liu K, Greenland P. Low risk-factor profile and long-term cardiovascular and noncardiovascular mortality and life expectancy: findings for 5 large cohorts of young adult and middle-aged men and women. *JAMA*. 1999;282:2012–2018.
42. Mensah GA, Mokdad AH, Ford ES, Greenlund KJ, Croft JB. State of disparities in cardiovascular health in the United States. *Circulation*. 2005;111:1233–1241.
43. Reeves MJ, Rafferty AP. Healthy lifestyle characteristics among adults in the United States, 2000. *Arch Intern Med*. 2005;165:854–857.
44. Gregg EW, Cheng YJ, Cadwell BL, Imperatore G, Williams DE, Flegal KM, Narayan KM, Williamson DF. Secular trends in cardiovascular disease risk factors according to body mass index in US adults [published correction appears in *JAMA*. 2005;294:182]. *JAMA*. 2005;293:1868–1874.
45. Lloyd-Jones DM, Leip EP, Larson MG, D'Agostino RB, Beiser A, Wilson PW, Wolf PA, Levy D. Prediction of lifetime risk for cardiovascular disease by risk factor burden at 50 years of age. *Circulation*. 2006;113:791–798.
46. Hozawa A, Folsom AR, Sharrett AR, Chambless LE. Absolute and attributable risks of cardiovascular disease incidence in relation to optimal and borderline risk factors: comparison of African American with white subjects: Atherosclerosis Risk in Communities Study. *Arch Intern Med*. 2007;167:573–579.
47. Soni A. *Personal Health Behaviors for Heart Disease Prevention Among the U.S. Adult Civilian Noninstitutionalized Population, 2004.* MEPS

Statistical Brief No. 165. Rockville, Md: Agency for Healthcare Research and Quality; March 2007. Available at: http://www.meps.ahrq.gov/mepsweb/data_files/publications/st165/stat165.pdf. Accessed October 17, 2007.
48. Carnethon MR, Lynch EB, Dyer AR, Lloyd-Jones DM, Wang R, Garside DB, Greenland P. Comparison of risk factors for cardiovascular mortality in black and white adults. *Arch Intern Med*. 2006;166:1196–1202.
49. Lloyd-Jones DM, Nam B-H, D'Agostino RB Sr, Levy D, Murabito JM, Wang RJ, Wilson PWF, O'Donnell CJ. Parental cardiovascular disease as a risk factor for cardiovascular disease in middle-aged adults: a prospective study of parents and offspring. *JAMA*. 2004;291:2204–2211.
50. Murabito JM, Pencina MJ, Nam BH, D'Agostino RB Sr, Wang TJ, Lloyd-Jones D, Wilson PW, O'Donnell CJ. Sibling cardiovascular disease as a risk factor for cardiovascular disease in middle-aged adults. *JAMA*. 2005;294:3117–3123.
51. Ridker PM, Buring JE, Rifai N, Cook NR. Development and validation of improved algorithms for the assessment of global cardiovascular risk in women: the Reynolds Risk Score [published correction appears in *JAMA*. 2007;297:1433]. *JAMA*. 2007;297:611–619.
52. Assmann G, Cullen P, Schulte H. Simple scoring scheme for calculating the risk of acute coronary events based on the 10-year follow-up of the Prospective Cardiovascular Munster (PROCAM) Study. *Circulation*. 2002;105:310–315.
53. Parikh NI, Hwang SJ, Larson MG, Cupples LA, Fox CS, Manders ES, Murabito JM, Massaro JM, Hoffmann U, O'Donnell CJ. Parental occurrence of premature cardiovascular disease predicts increased coronary artery and abdominal aortic calcification in the Framingham Offspring and Third Generation cohorts. *Circulation*. 2007;116:1473–1481.
54. Nasir K Budoff MJ, Wong ND, Scheuner M, Herrington D, Arnett DK, Szklo M, Greenland P, Blumenthal RS. Family history of premature coronary heart disease and coronary artery calcification: Multi-Ethnic Study of Atherosclerosis (MESA). *Circulation*. 2007;116:619–626.
55. Lee DS, Pencina MJ, Benjamin EJ, Wang TJ, Levy D, O'Donnell CJ, Nam BH, Larson MG, D'Agostino RB, Vasan RS. Association of parental heart failure with risk of heart failure in offspring. *N Engl J Med*. 2006;355:138–147.
56. Friedlander Y, Siscovick DM, Arbogast P, Psaty BM, Weinmann S, Lemaitre RN, Raghunathan TE, Cobb LA. Sudden death and myocardial infarction in first degree relatives as predictors of primary cardiac arrest. *Atherosclerosis*. 2002;162:211–216.
57. Centers for Disease Control and Prevention (CDC). Awareness of family health history as a risk factor for disease: United States, 2004. *MMWR Morb Mortal Wkly Rep*. 2004;53:1044–1047.
58. Terry DF, Pencina MJ, Vasan RS, Murabito JM, Wolf PA, Hayes MK, Levy D, D'Agostino RB, Benjamin EJ. Cardiovascular risk factors predictive for survival and morbidity-free survival in the oldest-old Framingham Heart Study participants. *J Am Geriatr Soc*. 2005;53:1944–1950.
59. Stampfer MJ, Hu FB, Manson JE, Rimm EB, Willett WC. Primary prevention of coronary heart disease in women through diet and lifestyle. *N Engl J Med*. 2000;343:16–22.
60. Lloyd-Jones DM, Dyer AR, Wang R, Daviglus ML, Greenland P. Risk factor burden in middle age and lifetime risks for cardiovascular and non-cardiovascular death (Chicago Heart Association Detection Project in Industry). *Am J Cardiol*. 2007;99:535–540.
61. Knoops KT, de Groot LC, Kromhout D, Perrin AE, Moreiras-Varela O, Menotti A, van Staveren WA. Mediterranean diet, lifestyle factors, and 10-year mortality in elderly European men and women: the HALE project. *JAMA*. 2004;292:1433–1439.
62. Mensah GA, Brown DW, Croft JB, Greenlund KJ. Major coronary risk factors and death from coronary heart disease: baseline and follow-up mortality data from the Second National Health and Nutrition Examination Survey (NHANES II). *Am J Prev Med*. 2005;29(suppl 1):68–74.
63. Daviglus ML, Liu K, Pirzada A, Yan LL, Garside DB, Feinglass J, Guralnik JM, Greenland P, Stamler J. Favorable cardiovascular risk profile in middle age and health-related quality of life in older age. *Arch Intern Med*. 2003;163:2460–2468.
64. Daviglus ML, Liu K, Greenland P, Dyer AR, Garside DB, Manheim L, Lowe LP, Rodin M, Lubitz J, Stamler J. Benefit of a favorable cardiovascular risk-factor profile in middle age with respect to Medicare costs. *N Engl J Med*. 1998;339:1122–1129.
65. DeFrances CJ, Lucas CA, Buie VC, Golosinskiy A. *2006 National Hospital Discharge Survey*. Hyattsville, Md: National Center for Health Statistics; 2008. National Health Statistics Reports, No. 5.
66. Cherry DK, Hing E, Woodwell DA, Rechtsteiner EA. *National Ambulatory Medical Care Survey: 2006 Summary*. Hyattsville, Md: National Center for Health Statistics; 2008. National Health Statistics Reports, No. 3.
67. Pitts SR, Niska RW, Xu J, Burt CW. *National Hospital Ambulatory Medical Care Survey: 2006 Emergency Department Summary*. Hyattsville, Md: National Center for Health Statistics; 2008. National Health Statistics Reports; No. 7.
68. Jones A. National Nursing Home Survey (NNHS): 2004 current resident tables. Available at: http://www.cdc.gov/nchs/about/major/nnhsd/ResidentTables.htm. National Center for Health Statistics; 2008. Accessed September 15, 2008.
69. Hing E, Hall MJ, Xu J. *National Hospital Ambulatory Medical Care Survey: 2006 Outpatient Department Summary*. Hyattsville, Md: National Center for Health Statistics; 2008. National Health Statistics Reports; No. 4.
70. Russo CA, Ho K, Elixhauser A. *Hospital Stays for Circulatory Diseases, 2004*. Rockville, Md: Agency for Healthcare Research and Quality; February 2007. Available at: http://www.hcup-us.ahrq.gov/reports/statbriefs/sb26.pdf. Accessed October 17, 2007. HCUP Statistical Brief No. 26.
71. Russo CA, Andrews RM. *The National Hospital Bill: The Most Expensive Conditions, by Payer, 2004*. Rockville, Md: Agency for Healthcare Research and Quality; September 2006. Available at: http://www.hcup-us.ahrq.gov/reports/statbriefs/sb13.pdf. Accessed October 17, 2007. HCUP Statistical Brief No. 13.
72. Elixhauser A, Jiang HJ. *Hospitalizations for Women With Circulatory Disease, 2003*. Rockville, Md: Agency for Healthcare Research and Quality; May 2006. Available at: http://www.hcup-us.ahrq.gov/reports/statbriefs/sb5.pdf. Accessed October 17, 2007. HCUP Statistical Brief No. 5.
73. Elixhauser A, Owens P. *Reasons for Being Admitted to the Hospital Through the Emergency Department, 2003*. Rockville, Md: Agency for Healthcare Research and Quality; February 2006. Available at: http://www.hcup-us.ahrq.gov/reports/statbriefs/sb2.pdf. Accessed October 17, 2007. HCUP Statistical Brief No. 2.
74. Centers for Medicare & Medicaid Services. *Health Care Financing Review: Medicare & Medicaid Statistical Supplement*. Table 5.5: Discharges, Total Days of Care, and Program Payments for Medicare Beneficiaries Discharged from Short-Stay Hospitals, by Principal Diagnoses Within Major Diagnostic Classifications (MDCs): Calendar Year 2006. Baltimore, Md: Centers for Medicare and Medicaid Services; 2005. Available at: http://www.cms.hhs.gov/MedicareMedicaidStatSupp/downloads/2007Table5.5b.pdf. Accessed August 28, 2008.
75. D'Agostino RB Sr, Vasan RS, Pencina MJ, Wolf PA, Cobain M, Massaro JM, Kannel WB. General cardiovascular risk profile for use in primary care: the Framingham Heart Study. *Circulation*. 2008;117:743–753.Center for Health Statistics; 2008. National Health Statistics Reports; No. 4.
70. Russo CA, Ho K, Elixhauser A. *Hospital Stays for Circulatory Diseases, 2004*. Rockville, Md: Agency for Healthcare Research and Quality; February 2007. Available at: http://www.hcup-us.ahrq.gov/reports/statbriefs/sb26.pdf. Accessed October 17, 2007. HCUP Statistical Brief No. 26.
71. Russo CA, Andrews RM. *The National Hospital Bill: The Most Expensive Conditions, by Payer, 2004*. Rockville, Md: Agency for Healthcare Research and Quality; September 2006. Available at: http://www.hcup-us.ahrq.gov/reports/statbriefs/sb13.pdf. Accessed October 17, 2007. HCUP Statistical Brief No. 13.
72. Elixhauser A, Jiang HJ. *Hospitalizations for Women With Circulatory Disease, 2003*. Rockville, Md: Agency for Healthcare Research and Quality; May 2006. Available at: http://www.hcup-us.ahrq.gov/reports/statbriefs/sb5.pdf. Accessed October 17, 2007. HCUP Statistical Brief No. 5.
73. Elixhauser A, Owens P. *Reasons for Being Admitted to the Hospital Through the Emergency Department, 2003*. Rockville, Md: Agency for Healthcare Research and Quality; February 2006. Available at: http://www.hcup-us.ahrq.gov/reports/statbriefs/sb2.pdf. Accessed October 17, 2007. HCUP Statistical Brief No. 2.
74. Centers for Medicare & Medicaid Services. *Health Care Financing Review: Medicare & Medicaid Statistical Supplement*. Table 5.5: Discharges, Total Days of Care, and Program Payments for Medicare Beneficiaries Discharged from Short-Stay Hospitals, by Principal Diagnoses Within Major Diagnostic Classifications (MDCs): Calendar Year 2006. Baltimore, Md: Centers for Medicare and Medicaid Services; 2005. Available at: http://www.cms.hhs.gov/MedicareMedicaidStatSupp/downloads/2007Table5.5b.pdf. Accessed August 28, 2008.
75. D'Agostino RB Sr, Vasan RS, Pencina MJ, Wolf PA, Cobain M, Massaro JM, Kannel WB. General cardiovascular risk profile for use in primary care: the Framingham Heart Study. *Circulation*. 2008;117:743–753.

Table 2-1. Cardiovascular Disease

Population Group	Prevalence, 2006 Age ≥20 y	Mortality, 2005 All Ages*	Hospital Discharges, 2006 All Ages	Cost, 2009
Both sexes	80 000 000 (36.3%)	864 480	7 095 000	$475.3 billion
Males	38 700 000 (37.6%)	409 867 (47.4%)†	4 038 000	. . .
Females	41 300 000 (34.9%)	454 613 (52.6%)†	3 057 000	. . .
NH white males	37.8%	329 607	. . .	. . .
NH white females	33.3%	372 191	. . .	. . .
NH black males	45.9%	47 384	. . .	. . .
NH black females	45.9%	52 401	. . .	. . .
Mexican American males	26.1%	. . .	. . .	. . .
Mexican American females	32.5%	. . .	. . .	. . .

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 2005–2006, NCHS and NHLBI. Percentages for racial/ethnic groups are age-adjusted for Americans ≥20 years of age. These data are based on self-reports. Estimates from NHANES 2005–2006 (NCHS) are applied to 2006 population estimates ≥20 years of age. 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 2009.

Table 2-2. 2005 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‖ 1995 to 2005	Rank§	Death Rate	% Change‖ 1995 to 2005	Rank§	Death Rate	% Change‖ 1995 to 2005
Alabama	51	349.4	−13.9	17	122.8	−29.5	52	60.9	−12.0
Alaska	6	232.8	−30.4	4	94.0	−37.5	43	53.6	−32.9
Arizona	9	235.9	−25.3	26	133.5	−28.9	8	38.6	−32.5
Arkansas	45	322.7	−20.9	46	169.4	−20.8	50	58.6	−31.5
California	26	267.7	−25.3	33	145.4	−34.3	28	47.9	−27.1
Colorado	4	231.4	−25.7	8	104.2	−33.9	12	42.0	−28.4
Connecticut	12	239.0	−33.7	13	119.0	−39.3	2	36.7	−34.5
Delaware	34	288.3	−22.5	42	161.1	−27.4	14	42.9	−21.7
District of Columbia	44	315.7	−17.4	49	180.7	11.9	7	38.5	−47.3
Florida	15	245.5	−25.9	29	139.9	−33.2	6	38.0	−29.1
Georgia	40	303.6	−24.6	12	117.0	−38.1	42	52.9	−28.8
Hawaii	3	220.5	−27.3	2	84.2	−39.0	20	46.1	−26.2
Idaho	20	248.2	−22.9	10	115.9	−32.2	41	52.5	−23.6
Illinois	32	285.4	−28.8	31	144.2	−38.7	29	48.0	−30.3
Indiana	38	298.6	−26.4	30	142.7	−35.4	35	50.8	−32.3
Iowa	25	265.8	−27.7	35	148.1	−35.6	30	48.3	−25.9
Kansas	24	264.9	−24.5	15	122.0	−33.8	33	49.4	−23.8
Kentucky	46	324.0	−22.1	43	161.1	−29.7	36	51.0	−25.6
Louisiana	49	332.4	−20.2	38	153.4	−30.4	45	56.7	−21.5
Maine	19	247.0	−30.3	16	122.7	−40.9	16	43.4	−21.2
Maryland	31	283.6	−22.7	37	152.3	−26.6	22	46.7	−26.9
Massachusetts	5	232.2	−29.3	9	109.7	−38.7	10	39.1	−24.8
Michigan	43	310.0	−26.0	45	166.4	−33.9	31	48.3	−31.4
Minnesota	1	208.0	−34.8	3	88.2	−45.3	15	42.9	−38.3
Mississippi	52	373.3	−20.4	44	162.6	−33.5	44	55.5	−22.5

(Continued)

Table 2-2. Continued

State	CVD* Rank§	CVD* Death Rate	CVD* % Change‖ 1995 to 2005	CHD† Rank§	CHD† Death Rate	CHD† % Change‖ 1995 to 2005	Stroke‡ Rank§	Stroke‡ Death Rate	Stroke‡ % Change‖ 1995 to 2005
Missouri	41	304.4	−24.5	40	158.5	−32.6	38	51.5	−24.1
Montana	8	235.4	−27.4	5	99.5	−35.5	27	47.8	−29.9
Nebraska	16	246.1	−32.4	6	102.0	−42.0	26	47.5	−26.0
Nevada	42	308.5	−21.0	21	124.0	−39.0	21	46.3	−25.5
New Hampshire	13	241.6	−32.0	25	130.7	−39.0	3	36.7	−42.7
New Jersey	28	271.7	−28.1	39	153.5	−33.7	5	37.8	−32.4
New Mexico	10	237.8	−23.7	18	122.8	−29.4	9	38.6	−33.2
New York	36	293.0	−29.8	52	192.8	−32.1	1	31.1	−34.7
North Carolina	33	287.0	−27.1	28	137.4	−36.4	47	57.4	−31.3
North Dakota	14	242.3	−29.5	27	135.0	−26.4	13	42.4	−30.1
Ohio	39	301.9	−25.9	41	160.3	−32.6	32	49.3	−22.8
Oklahoma	50	344.8	−15.9	51	190.8	−16.7	49	58.2	−18.0
Oregon	21	249.7	−26.6	7	104.1	−39.3	46	56.7	−30.9
Pennsylvania	35	291.6	−27.5	36	149.4	−35.3	23	47.0	−26.2
Puerto Rico¶	11	238.8	−19.5	14	121.7	−12.6	17	44.2	−17.6
Rhode Island	29	281.6	−23.7	50	186.0	−25.2	11	39.5	−33.0
South Carolina	37	296.4	−30.3	24	129.1	−40.9	48	57.6	−35.9
South Dakota	22	254.8	−28.6	32	145.0	−31.0	37	51.4	−26.3
Tennessee	48	330.2	−21.3	48	178.1	−26.9	51	60.8	−28.0
Texas	30	281.9	−23.8	34	147.0	−32.6	34	50.0	−26.2
Utah	2	220.2	−23.8	1	81.8	−42.0	19	44.5	−30.2
Vermont	7	234.5	−33.9	22	124.6	−40.0	4	37.3	−37.1
Virginia	27	270.6	−29.3	19	122.9	−34.9	40	52.3	−29.5
Washington	17	246.5	−25.2	23	125.7	−28.0	24	47.1	−31.0
West Virginia	47	327.7	−23.4	47	171.4	−31.7	39	51.8	−16.6
Wisconsin	23	257.6	−28.6	20	123.8	−37.3	25	47.3	−34.5
Wyoming	18	246.8	−25.9	11	116.9	−33.4	18	44.2	−38.1
Total United States		278.8	−27.0		144.4	−33.7		46.6	−28.3

Empty cells indicate data not available.

*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, 1995–2005. For stroke, the death rates in 1995–1998 were comparability modified using the ICD-10 to ICD-9 comparability ratio of 1.0502.

¶Percent changes for Puerto Rico are for 1996–1998 (averaged) to 2004 and are not based on a log linear slope.

Source: NCHS compressed mortality file 1979–2005. 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) 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 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 NCHS has "Health Data for All Ages by State" at http://www.cdc.gov/nchs/health_data_for_all_ages.htm. 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/. In addition, in the 2008 Atlas of Stroke Hospitalizations Among Medicare Beneficiaries (CDC, 2008), a new resource, data are available down to the county level, by sex and race: http://www.cdc.gov/dhdsp/library/stroke_hospitalization_atlas.htm.

Table 2-3. International Death Rates (Revised 2008): Death Rates (Per 100 000 Population) for Total Cardiovascular Disease, Coronary Heart Disease, Stroke, and Total Deaths in Selected Countries (Most Recent Year Available)

	ICD Version	CVD Deaths	CHD Deaths	Stroke Deaths	Total Deaths
Men, ages 35 to 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
Romania (2004)	10	770.0	314.3	250.9	1652.1
Hungary (2005)	10	709.7	384.7	140.8	1818.0
Poland (2005)	10	517.3	201.3	109.2	1457.0
Czech Republic (2005)	10	454.7	224.8	87.9	1204.7
China—Rural (1999)*	9	413.4	64.3	243.1	1260.2
Argentina (2001)	10	405.9	119.8	102.6	1261.7
China—Urban (1999)*	9	389.0	105.7	217.1	1002.6
Colombia (1999)	10	331.3	168.2	94.7	1021.0
Scotland (2004)	10	327.3	220.6	53.0	995.9
Finland (2005)	10	311.2	193.9	50.2	898.8
Greece (2004)*	9	306.7	164.8	69.0	778.7
Belgium (1997)*	9	289.3	143.0	49.8	990.9
Denmark (2001)	10	285.7	141.8	52.2	955.6
United States (2005)	10	283.3	169.4	33.1	908.6
Northern Ireland (2004)	10	281.7	194.4	42.0	827.8
New Zealand (2001)	10	273.6	181.2	41.6	783.3
Germany (2004)	10	270.8	142.2	38.6	845.9
England/Wales (2004)	10	262.8	168.9	41.3	756.7
Portugal (2003)	10	252.8	96.6	96.1	966.5
Ireland (2005)	10	249.7	161.8	31.8	721.5
Mexico (2001)	10	235.0	129.9	54.7	1055.8
Netherlands (2004)	10	222.0	95.5	36.8	758.5
Sweden (2004)	10	218.8	132.6	37.0	655.4
Italy (2002)*	9	218.4	101.1	41.0	743.7
Austria (2005)	10	217.7	124.6	31.7	789.0
Canada (2002)	10	212.3	141.7	27.7	741.4
Norway (2004)	10	205.5	117.7	35.9	692.8
Republic of Korea (2004)	10	203.6	52.1	117.2	1003.9
Spain (2004)	10	192.6	92.6	39.6	788.9
Israel (2003)	10	179.9	95.1	38.0	717.0
Australia (2003)	10	179.6	117.2	26.8	636.4
Japan (2004)	10	163.1	50.4	62.3	674.7
Switzerland (2004)	10	162.5	83.1	22.7	636.0
France (2004)	10	161.5	64.3	29.3	820.7
Women, ages 35 to 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 (2004)	10	403.1	134.3	165.6	786.8
Hungary (2005)	10	291.1	118.1	67.0	780.4
China—Rural (1999)*	9	279.3	40.9	151.7	798.7
China—Urban (1999)*	9	273.4	71.1	146.9	662.7
Colombia (1999)	10	229.9	94.7	70.7	640.1
Poland (2005)	10	201.8	59.6	60.6	589.9
Czech Republic (2005)	10	200.0	82.3	49.2	577.9

(*Continued*)

Table 2-3. Continued

	ICD 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
Scotland (2004)	10	153.9	80.6	40.7	606.6
United States (2005)	10	145.3	69.6	26.1	572.3
New Zealand (2001)	10	135.2	74.9	34.3	497.1
Northern Ireland (2004)	10	129.2	66.0	34.1	509.7
Denmark (2001)	10	127.3	51.0	37.0	642.0
Belgium (1997)*	9	126.4	44.0	35.0	494.0
Greece (2004)*	9	125.5	43.1	41.2	354.6
Portugal (2003)	10	123.2	35.0	55.0	449.0
England/Wales (2004)	10	117.0	55.4	30.9	478.0
Germany (2004)	10	111.3	45.0	23.0	426.0
Republic of Korea (2004)	10	109.8	21.0	68.4	404.6
Netherlands (2004)	10	102.0	34.0	26.0	466.0
Ireland (2005)	10	99.6	49.4	20.1	451.3
Finland (2005)	10	98.0	45.2	28.7	409.6
Canada (2002)	10	92.1	48.0	20.0	452.0
Italy (2002)*	9	92.1	29.0	25.0	372.0
Sweden (2004)	10	89.9	42.3	23.9	408.3
Austria (2005)	10	89.6	39.2	20.1	394.2
Israel (2003)	10	83.3	31.0	22.0	431.0
Norway (2004)	10	81.3	31.1	25.3	408.4
Australia (2003)	10	78.6	37.4	20.1	368.4
Spain (2004)	10	73.9	23.9	20.8	327.9
Japan (2004)	10	66.4	14.9	29.6	298.8
Switzerland (2004)	10	60.9	22.1	13.5	340.0
France (2004)	10	58.0	14.3	16.0	367.8

Rates adjusted to the European Standard population.

*Countries using ICD-9. 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.

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

Table 2-4. Remaining 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		Remaining Lifetime Risk at Age 70	
Diseases	Men	Women	Men	Women
Any CVD*	2 in 3	>1 in 2	>1 in 2	1 in 2
CHD[45]	1 in 2	1 in 3	1 in 3	1 in 4
AF[46]	1 in 4	1 in 4	1 in 4	1 in 4
CHF[47]	1 in 5	1 in 5	1 in 5	1 in 5
Stroke[48]	1 in 6†	1 in 5†	1 in 6	1 in 5
Dementia[48]	. . .	. . .	1 in 7	1 in 5
Hip fracture[58]	1 in 20	1 in 6	. . .	. . .
Breast cancer[59,61]	1 in 1000	1 in 8	. . .	1 in 14
Prostate cancer[59]	1 in 6	. . .	. . .	. . .
Lung cancer[59]	1 in 12	1 in 17	. . .	. . .
Colon cancer[59]	1 in 16	1 in 17	. . .	. . .
Diabetes[62]	1 in 3	1 in 3	1 in 9	1 in 7
Hypertension[63]	9 in 10†	9 in 10†	9 in 10‡	9 in 10‡
Obesity[64]	1 in 3	1 in 3	. . .	. . .

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

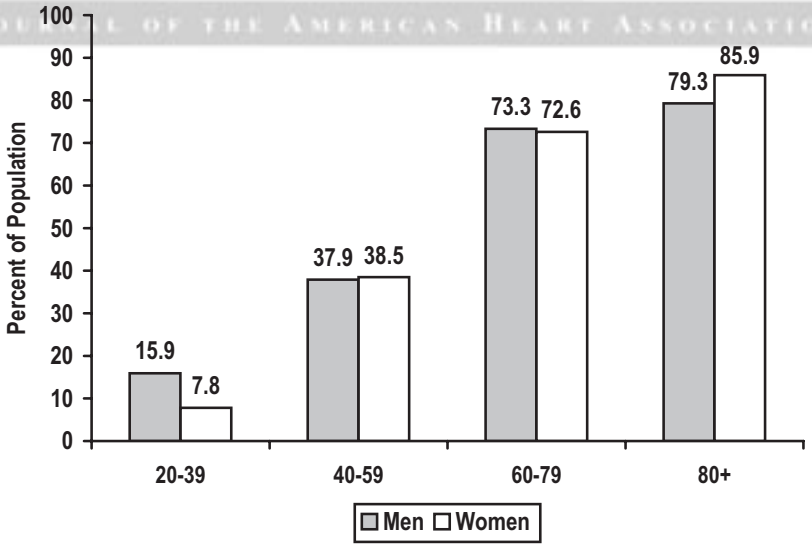


Chart 2-1. Prevalence of CVD in adults ≥20 years of age by age and sex (NHANES: 2005–2006). Source: NCHS and NHLBI. These data include CHD, HF, stroke, and hypertension.

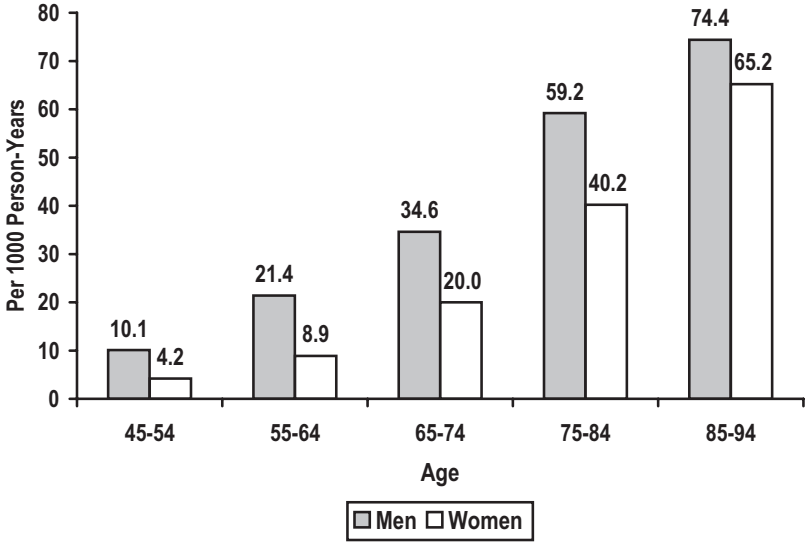


Chart 2-2. Incidence of CVD by age and sex (FHS, 1980–2003). CVD includes CHD, HF, stroke, or intermittent claudication but does not include hypertension alone. Source: NHLBI.[3]

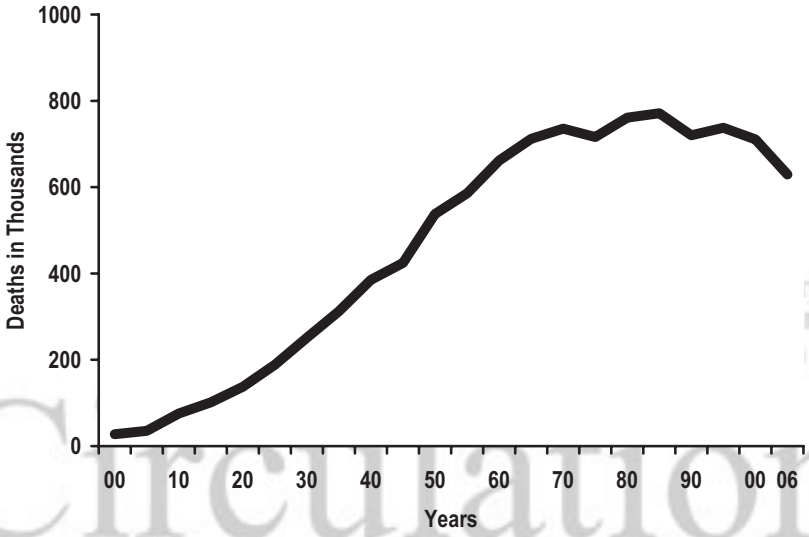


Chart 2-3. Deaths due to diseases of the heart (United States: 1900–2006). See Glossary for an explanation of "diseases of the heart." Source: NCHS.

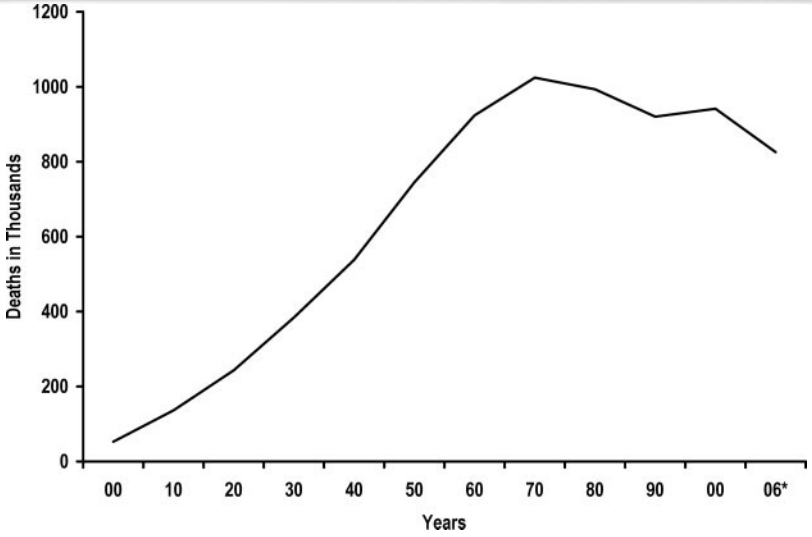


Chart 2-4. Deaths due to CVD (United States: 1900–2006). CVD does not include congenital CVD. Source: NCHS. *Preliminary.

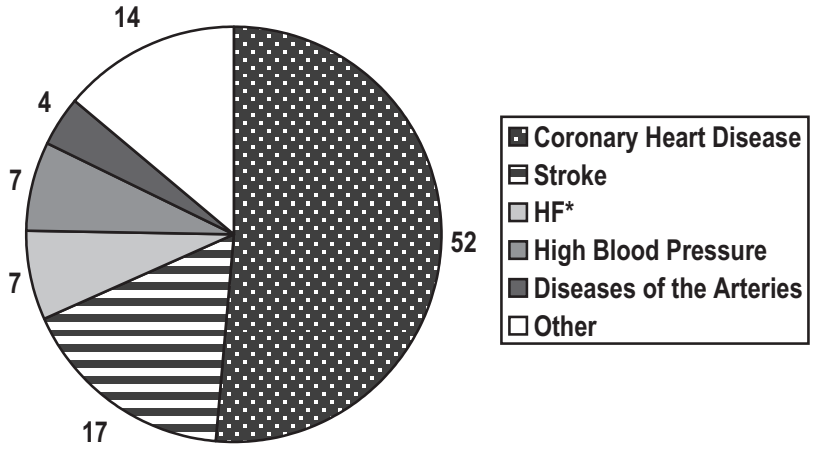


Chart 2-5. Percentage breakdown of deaths due to CVD (United States: 2006, preliminary). Source: NCHS. May not add to 100 owing to rounding. *Not a true underlying cause.

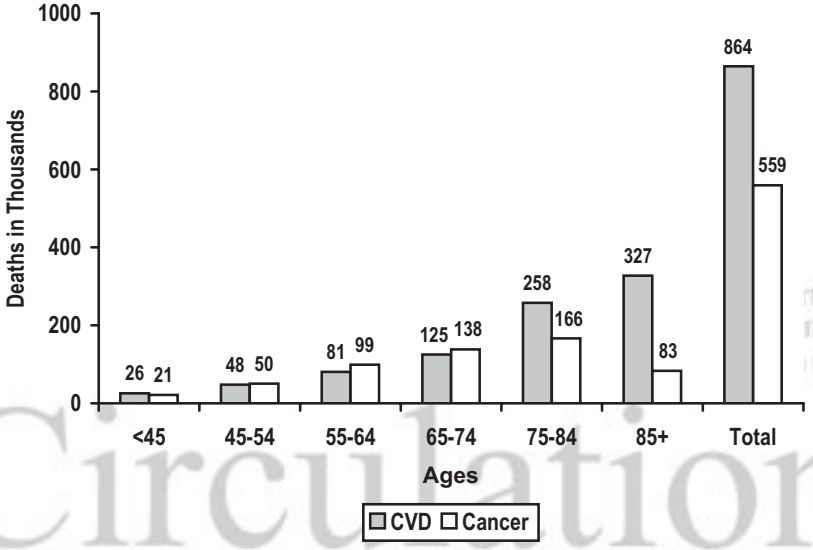


Chart 2-6. CVD deaths vs cancer deaths by age (United States: 2005). Source: NCHS.

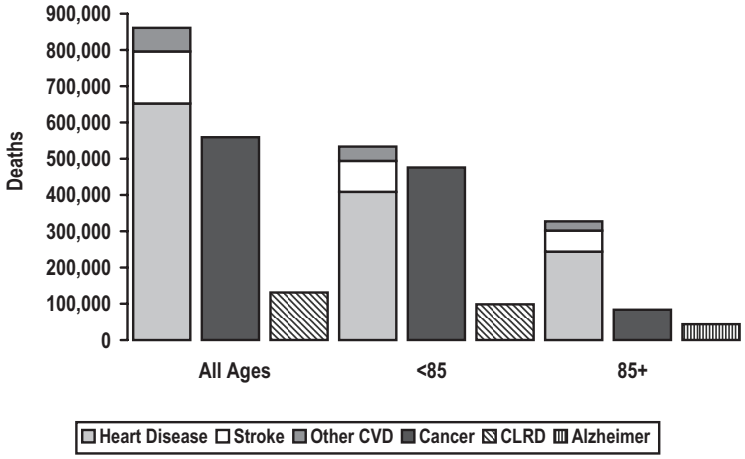


Chart 2-7. CVD and other major causes of death: total, <85 years of age, and ≥85 years of age. Deaths among both sexes, United States, 2005. Source: NCHS and NHLBI.

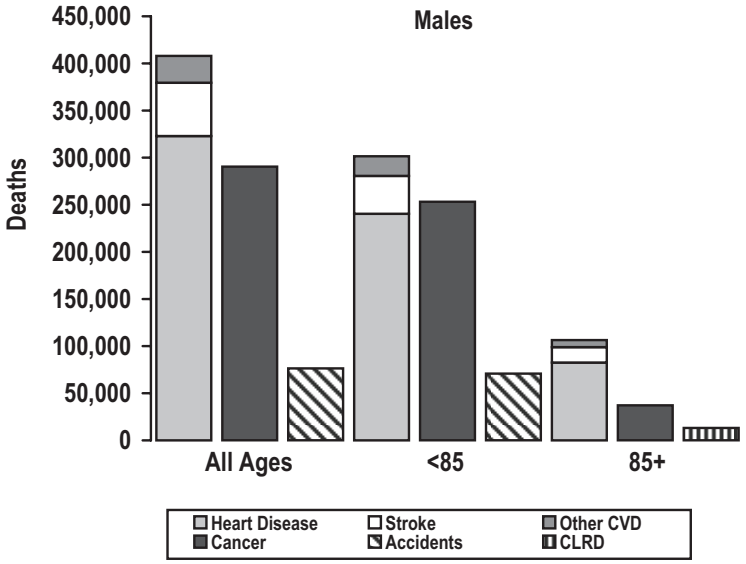


Chart 2-8. CVD and other major causes of death: total, <85 years of age, and ≥85 years of age. Deaths among males, United States, 2005. Source: NCHS and NHLBI.

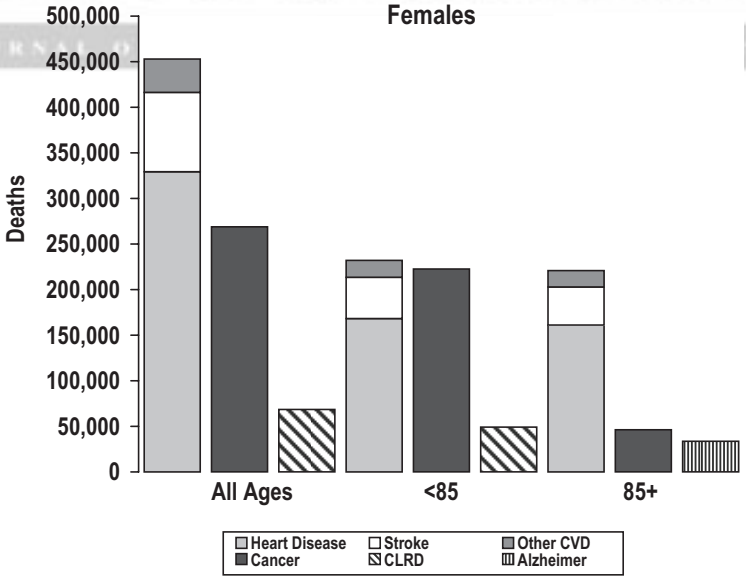


Chart 2-9. CVD and other major causes of death: total, <85 years of age, and ≥85 years of age. Deaths among females, United States, 2005. Source: NCHS and NHLBI.

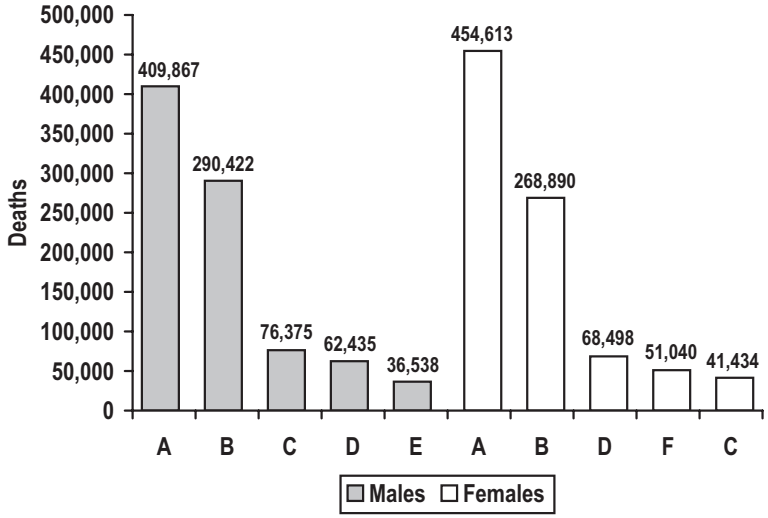


Chart 2-10. CVD and other major causes of death for all males and females (United States: 2005). Source: NCHS and NHLBI. A indicates CVD plus congenital CVD; B, cancer; C, accidents; D, CLRD; E, diabetes; and F, Alzheimer's disease.

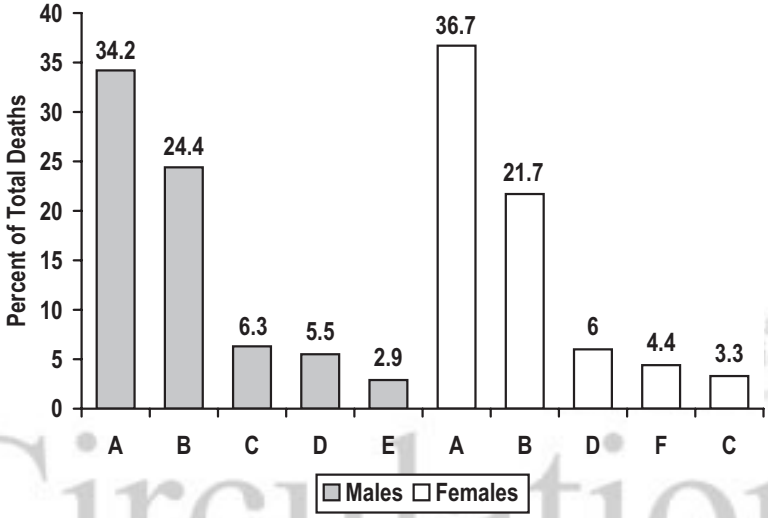


Chart 2-11. CVD and other major causes of death for white males and females (United States: 2005). Source: NCHS. Abbreviations as in Chart 2-10. Note: Using the combined category of "diseases of the heart and stroke," which do not constitute total CVD, the percentages were 31.8 for males and 33.7 for females.

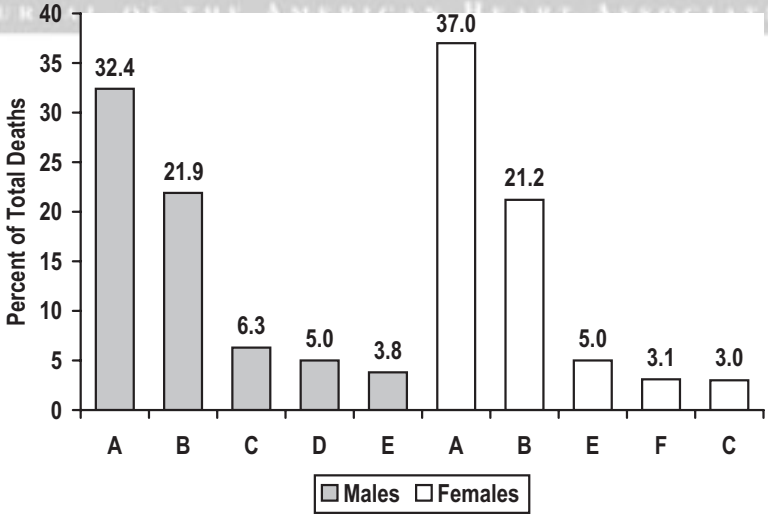


Chart 2-12. CVD and other major causes of death for black males and females (United States: 2005). Source: NCHS. A indicates CVD plus congenital CVD; B, cancer; C, accidents; D, diabetes; E, assault (homicide); and F, nephritis. Note: Using the combined category of "diseases of the heart and stroke," which do not constitute total CVD, the percentages were 29.4 for males and 33.3 for females.

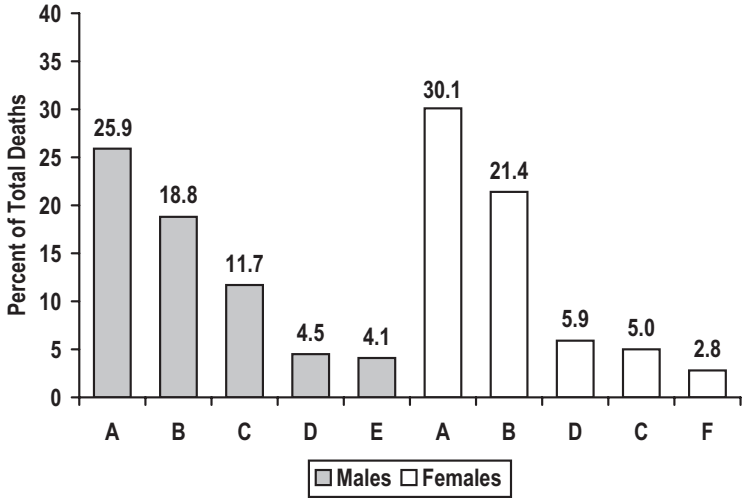


Chart 2-13. Diseases of the heart and stroke and other major causes of death for Hispanic or Latino males and females (United States: 2005). Data for total CVD are not readily available. Source: NCHS. A indicates diseases of the heart and stroke; B, cancer; C, accidents; D, DM; E, assault (homicide); and F, CLRD.

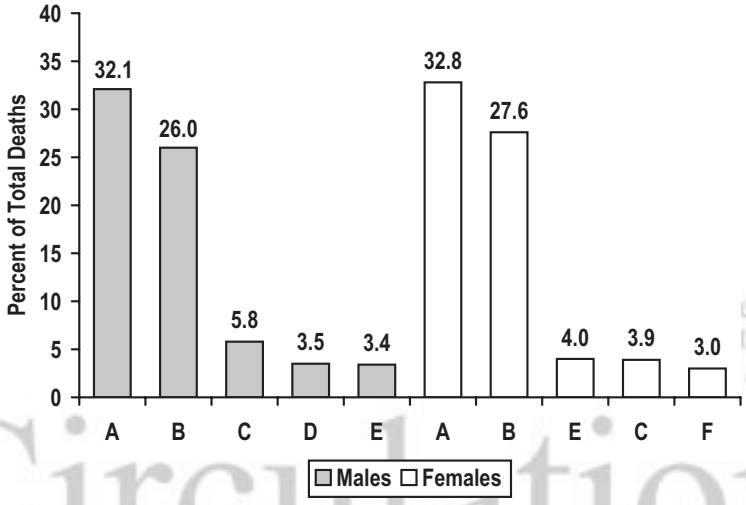


Chart 2-14. Diseases of the heart and stroke and other major causes of death for Asian or Pacific Islander males and females (United States: 2005). "Asian or Pacific Islander" is a heterogeneous category that includes people at high CVD risk (eg, South Asian) and people at low CVD risk (eg, Japanese). More specific data on the groups are not available. Mortality data for total CVD are not readily available. Source: NCHS. A indicates diseases of the heart/stroke; B, cancer; C, accidents; D, CLRD; E, diabetes; and F, influenza and pneumonia.

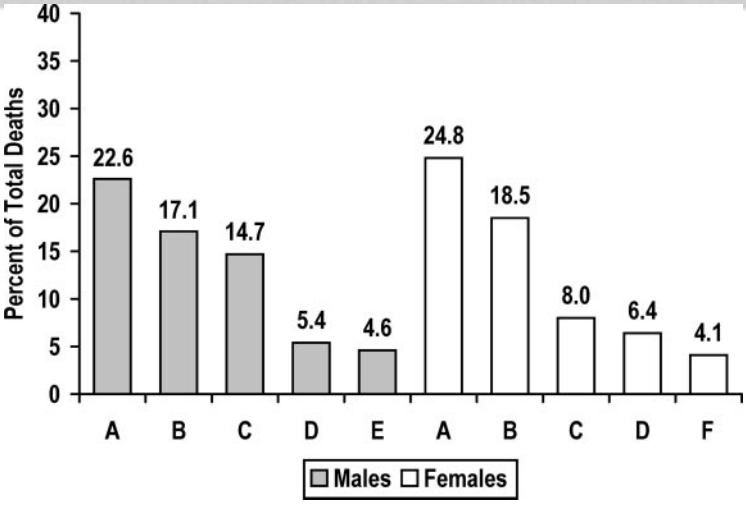


Chart 2-15. Diseases of the heart and stroke and other major causes of death for American Indian or Alaska Native males and females (United States: 2005). Data for total CVD are not readily available. Source: NCHS. A indicates diseases of the heart/stroke; B, cancer; C, accidents; D, diabetes; E, chronic liver disease and cirrhosis; and F, CLRD.

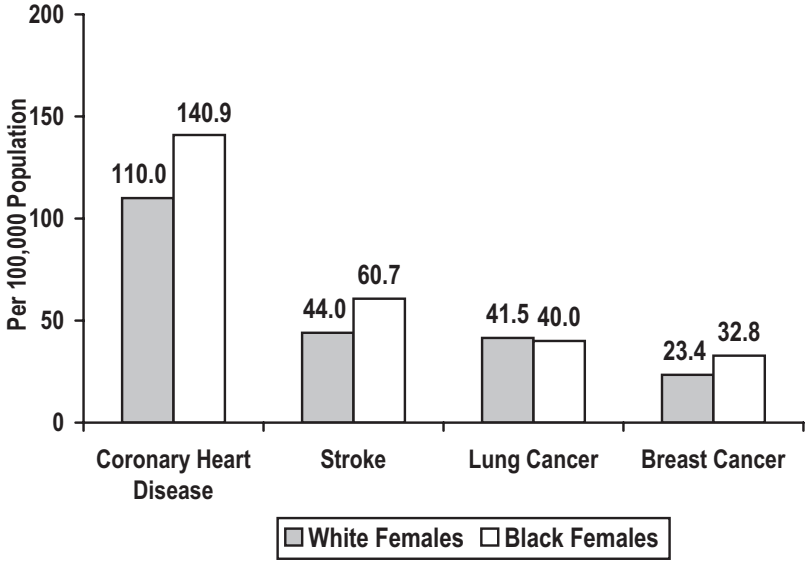


Chart 2-16. Age-adjusted death rates for CHD, stroke, and lung and breast cancer for white and black females (United States: 2005). Source: NCHS.

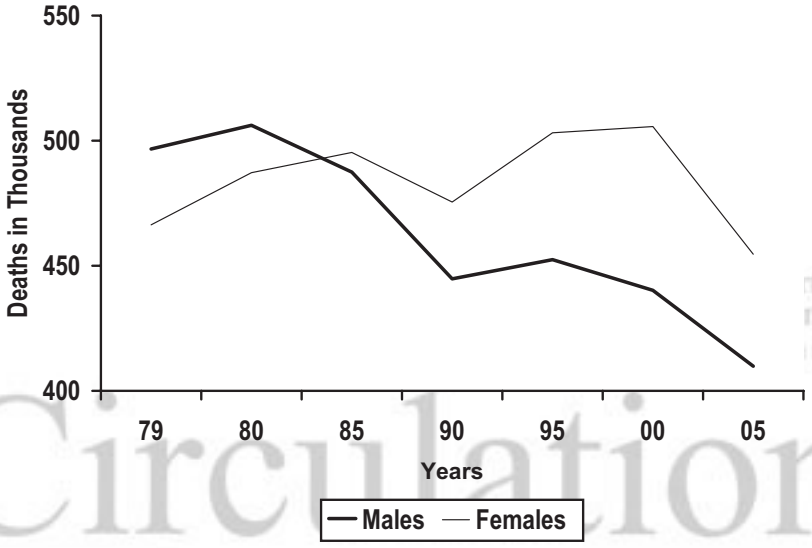


Chart 2-17. CVD mortality trends for males and females (United States: 1979–2005). Source: NCHS. The overall comparability for CVD between the ICD-9 (1979–1998) and ICD-10 (1999–2005) is 0.9962. No comparability ratios were applied.

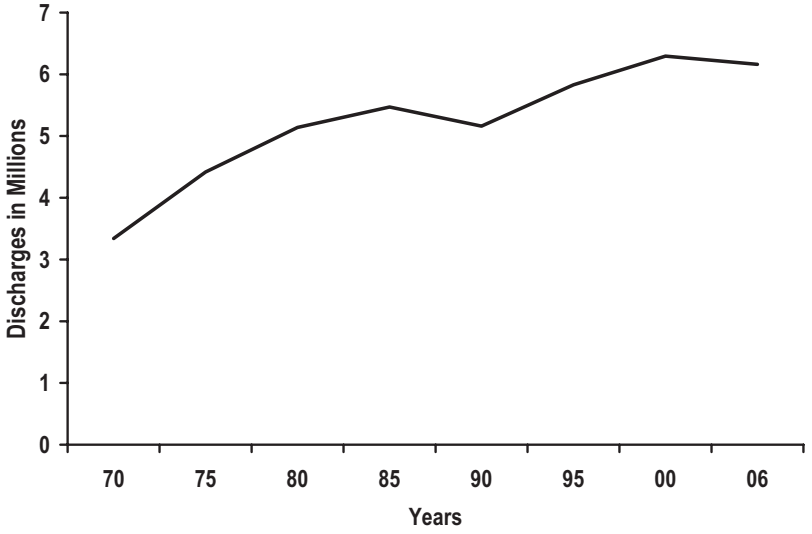


Chart 2-18. Hospital discharges for CVD (United States: 1970–2006). Hospital discharges include people discharged alive, dead, and "status unknown." Source: NCHS and NHLBI.

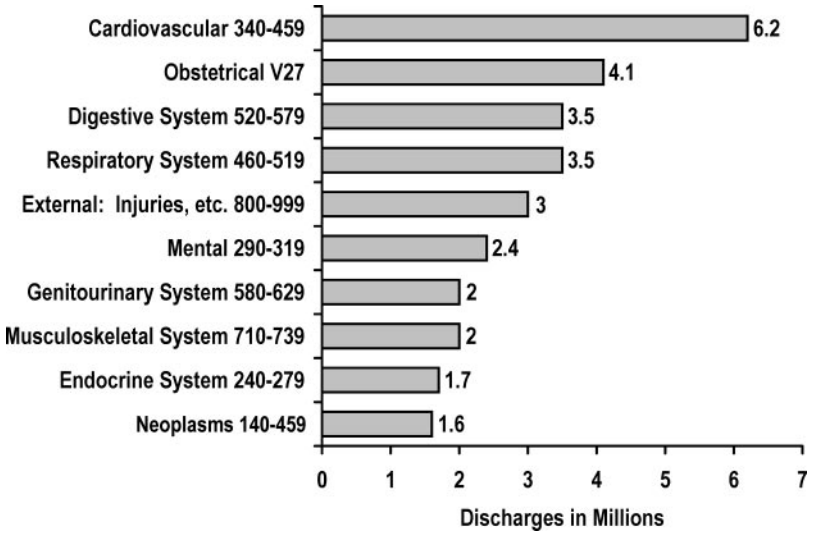
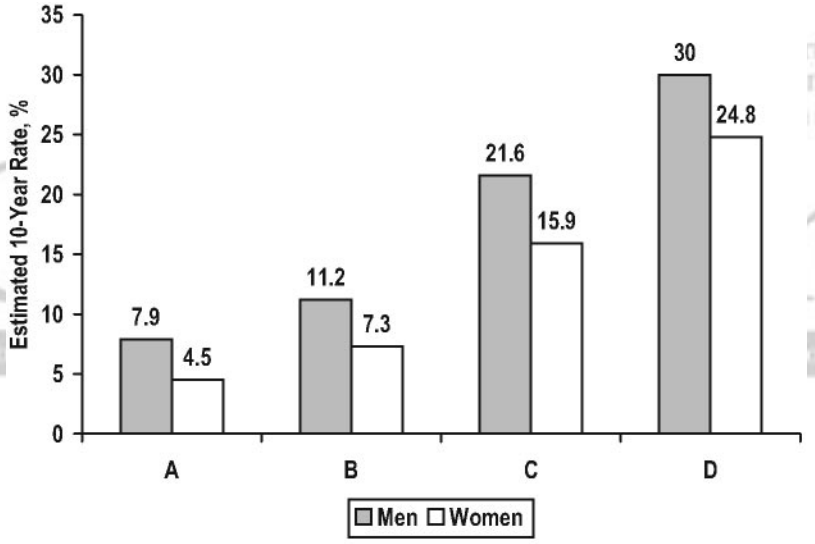


Chart 2-19. Hospital discharges for the 10 leading diagnostic groups (United States: 2006). Source: NHDS/NCHS and NHLBI.

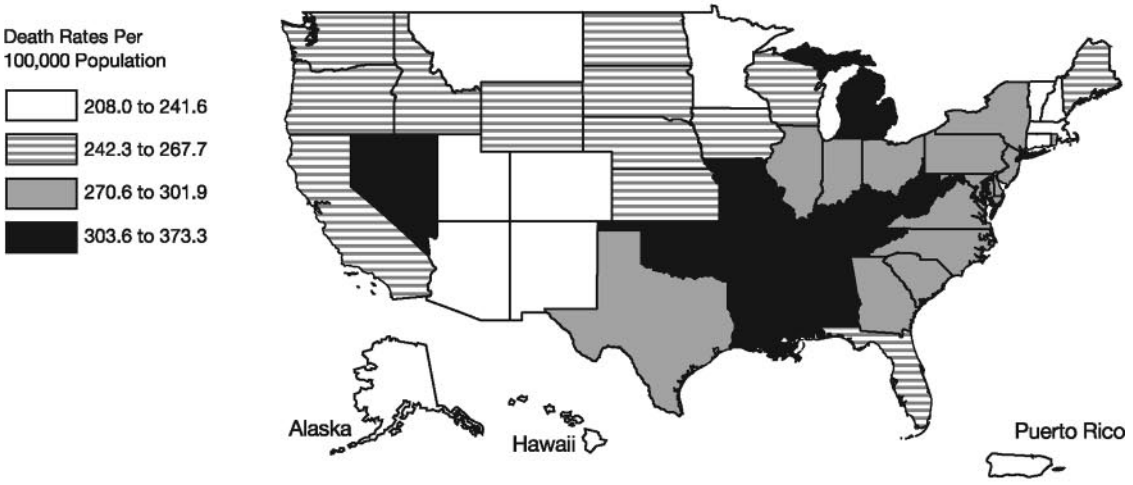


	A	B	C	D
Age	50-54	50-54	50-54	50-54
HDL cholesterol, mg/dL	45-49	45-49	35-34	35-34
Total cholesterol, mg/dL	160-199	200-239	200-239	200-239
Systolic BP, mm Hg, no treatment	120-129	130-139	130-139	130-139
Smoker	No	No	No	Yes
Diabetes	No	No	Yes	Tes

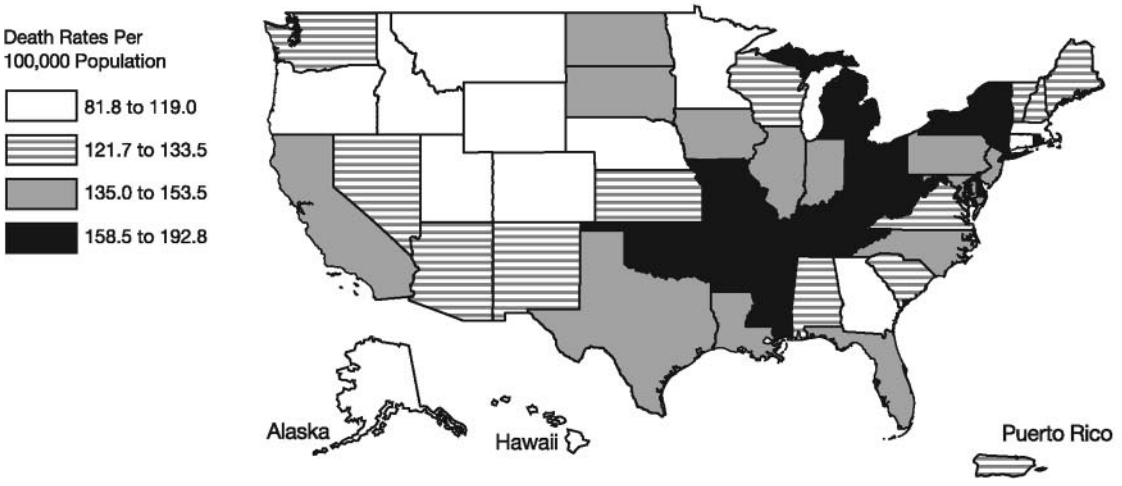
Chart 2-20. Estimated average 10-year CVD risk in adults 50 to 54 years of age according to levels of various risk factors (Framingham Heart Study). Source: D'Agostino et al.[75]

Death Rates by State — Statistics
(Includes District of Columbia)

2005 Total Cardiovascular Disease Age-Adjusted Death Rates by State



2005 Coronary Heart Disease Age-Adjusted Death Rates by State



2005 Stroke Age-Adjusted Death Rates by State

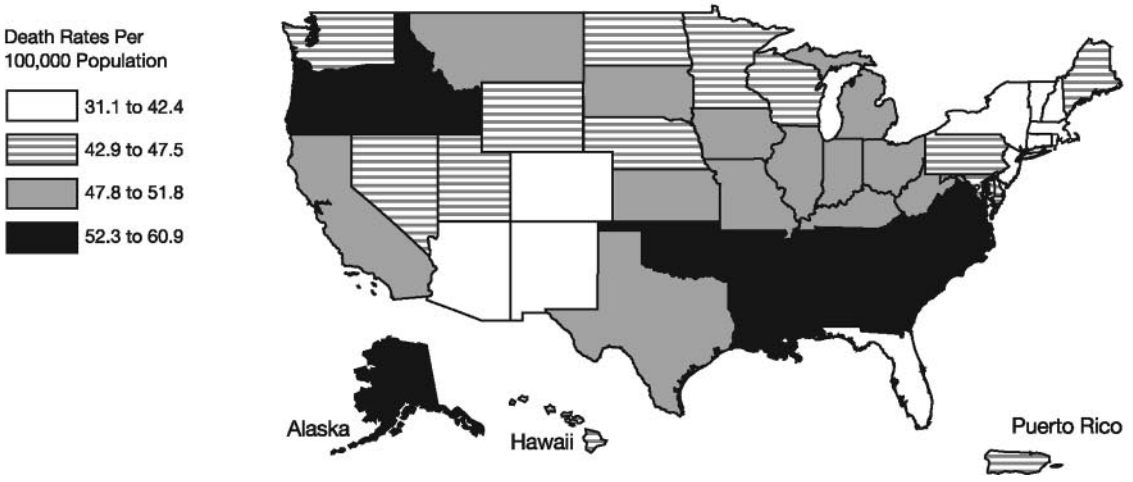


Chart 2-21. US maps corresponding to state death rates.

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 to 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.

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, often 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 for coronary artery disease (eg, exercise testing or myocardial perfusion imaging).

Abbreviations Used in Chapter 3

BMI	body mass index
CAC	coronary artery calcification
CARDIA	Coronary Artery Risk Development in Young Adults
CHD	coronary heart disease
CT	computed tomography
CVD	cardiovascular disease
DBP	diastolic blood pressure
FRS	Framingham Risk Score
HDL	high-density lipoprotein
HR	hazard ratio
IMT	intima-media thickness
LDL	low-density lipoprotein
MESA	Multiethnic Study of Atherosclerosis
mg/dL	milligrams per deciliter
MI	myocardial infarction
NHLBI	National Heart, Lung, and Blood Institute
SBP	systolic blood pressure
SD	standard deviation

Prevalence

- The NHLBI's Coronary Artery Risk Development in Young Adults (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 Multiethnic Study of Atherosclerosis (MESA) 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 relative risk 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 cutpoints 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).

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 relative risks 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.

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.[7]

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.[8]

— 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 and bulb IMTs than whites.
— Even at this young age, after adjustment for age, race, and sex, carotid IMT was associated significantly and positively with waist circumference, SBP, DBP, and LDL cholesterol. Carotid IMT was inversely correlated with HDL cholesterol levels. Participants with greater numbers of adverse risk factor levels (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.[9] 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 Cardiovascular Health Study reported follow-up of 4476 men and women ≥65 years of age (mean age 72 years) who were free of CVD at baseline.[10]

— 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.

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.[11]

— 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 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.[12]

— 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.

References

1. Budoff MJ, Achenbach S, Blumenthal RS, Carr JJ, Goldin JG, Greenland P, Guerci AD, Lima JA, Rader DJ, Rubin GD, Shaw LJ, Wiegers SE; American Heart Association Committee on Cardiovascular Imaging and Intervention; American Heart Association Council on Cardiovascular Radiology and Intervention; American Heart Association Committee on Cardiac Imaging, Council on Clinical Cardiology. Assessment of coronary artery disease by cardiac computed tomography: a scientific statement from the American Heart Association Committee on Cardiovascular Imaging and Intervention, Council on Cardiovascular Radiology and Intervention, and Committee on Cardiac Imaging, Council on Clinical Cardiology. *Circulation*. 2006;114:1761–1791.
2. Greenland P, Bonow RO, Brundage BH, Budoff MJ, Eisenberg MJ, Grundy SM, Lauer MS, Post WS, Raggi P, Redberg RF, Rodgers GP, Shaw LJ, Taylor AJ, Weintraub WS; American College of Cardiology Foundation Clinical Expert Consensus Task Force (ACCF/AHA Writing Committee to Update the 2000 Expert Consensus Document on Electron Beam Computed Tomography); Society of Atherosclerosis Imaging and Prevention; Society of Cardiovascular Computed Tomography. ACCF/AHA 2007 clinical expert consensus document on coronary artery calcium scoring by computed tomography in global cardiovascular risk assessment and in evaluation of patients with chest pain: a report of the American College of Cardiology Foundation Clinical Expert Consensus Task Force (ACCF/AHA Writing Committee to Update the 2000 Expert Consensus Document on Electron Beam Computed Tomography) Developed in Collaboration With the Society of Atherosclerosis Imaging and Prevention and the Society of Cardiovascular Computed Tomography. *J Am Coll Cardiol*. 2007;49:378–402.
3. Loria CM, Liu K, Lewis CE, Hulley SB, Sidney S, Schreiner PJ, Williams OD, Bild DE, Detrano R. Early adult risk factor levels and subsequent coronary artery calcification: the CARDIA Study. *J Am Coll Cardiol*. 2007;49:2013–2020.
4. Bild DE, Detrano R, Peterson D, Guerci A, Liu K, Shahar E, Ouyang P, Jackson S, Saad MF. Ethnic differences in coronary calcification: the Multi-Ethnic Study of Atherosclerosis (MESA). *Circulation*. 2005;111:1313–1320.
5. Detrano R, Guerci AD, Carr JJ, Bild DE, Burke G, Folsom AR, Liu K, Shea S, Szklo M, Bluemke DA, O'Leary DH, Tracy R, Watson K, Wong ND, Kronmal RA. Coronary calcium as a predictor of coronary events in four racial or ethnic groups. *N Engl J Med*. 2008;358:1336–1345.
6. Greenland P, LaBree L, Azen SP, Doherty TM, Detrano RC. Coronary artery calcium score combined with Framingham score for risk prediction in asymptomatic individuals [published correction appears in *JAMA*. 2004;291:563]. *JAMA*. 2004;291:210–215.
7. Smith SC Jr, Greenland P, Grundy SM. Prevention Conference V: beyond secondary prevention : identifying the high-risk patient for primary prevention: executive summary. *Circulation*. 2000;101:111–116.
8. Urbina EM, Srinivasan SR, Tang R, Bond MG, Kieltyka L, Berenson GS. Impact of multiple coronary risk factors on the intima-media thickness of different segments of carotid artery in healthy young adults (the Bogalusa Heart Study). *Am J Cardiol*. 2002;90:953–958.
9. Li S, Chen W, Srinivasan SR, Bond MG, Tang R, Urbina EM, Berenson GS. Childhood cardiovascular risk factors and carotid vascular changes in adulthood: the Bogalusa Heart Study [published correction appears in *JAMA*. 2003;290:2943]. *JAMA*. 2003;290:2271–2276.
10. O'Leary DH, Polak JF, Kronmal RA, Manolio TA, Burke GL, Wolfson SK Jr; Cardiovascular Health Study Collaborative Research Group. Carotid-artery intima and media thickness as a risk factor for myocardial infarction and stroke in older adults. *N Engl J Med*. 1999;340:14–22.
11. Manolio TA, Arnold AM, Post W, Bertoni AG, Schreiner PJ, Sacco RL, Saad MF, Detrano RL, Szklo M. Ethnic differences in the relationship of carotid atherosclerosis to coronary calcification: the Multi-Ethnic Study of Atherosclerosis. *Atherosclerosis*. 2008;197:132–138.
12. Folsom AR, Kronmal RA, Detrano RC, O'Leary DH, Bild DE, Bluemke DA, Budoff MJ, Liu K, Shea S, Szklo M, Tracy RP, Watson KE, Burke GL. Coronary artery calcification compared with carotid intima-media thickness in the prediction of cardiovascular disease incidence: the Multi-Ethnic Study of Atherosclerosis (MESA). *Arch Intern Med*. 2008;168:1333–1339.

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).

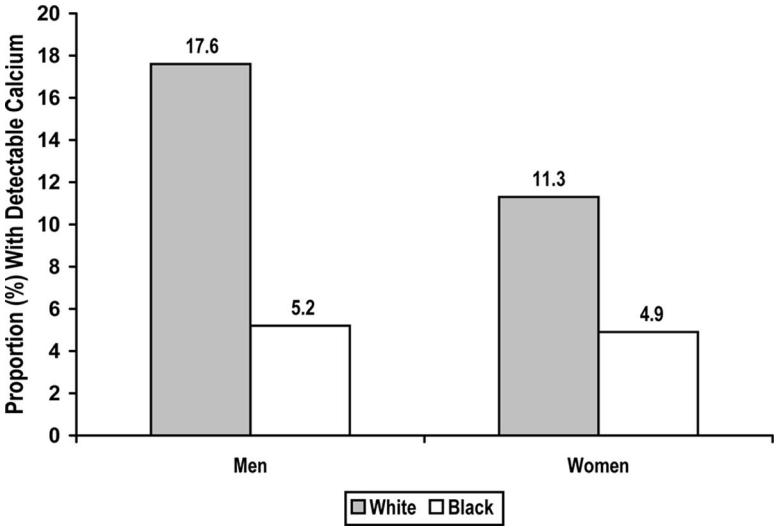


Chart 3-1. Prevalence (%) of coronary calcium: US adults 33 to 45 years of age. Source: Reprinted from Loria et al,[3] with permission from Elsevier. Copyright 2007. $P<0.0001$ across race–sex groups.

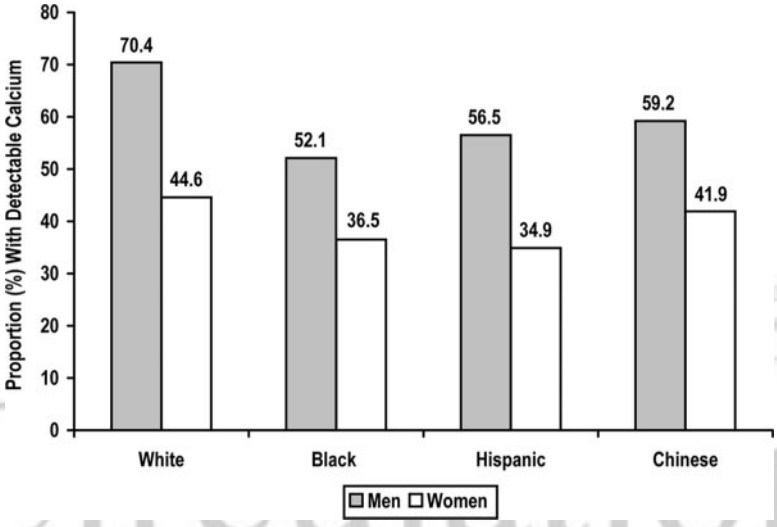


Chart 3-2. Prevalence (%) of coronary calcium: US adults 45 to 84 years of age. Source: Bild et al.[4] $P<0.0001$ across ethnic groups in both men and women.

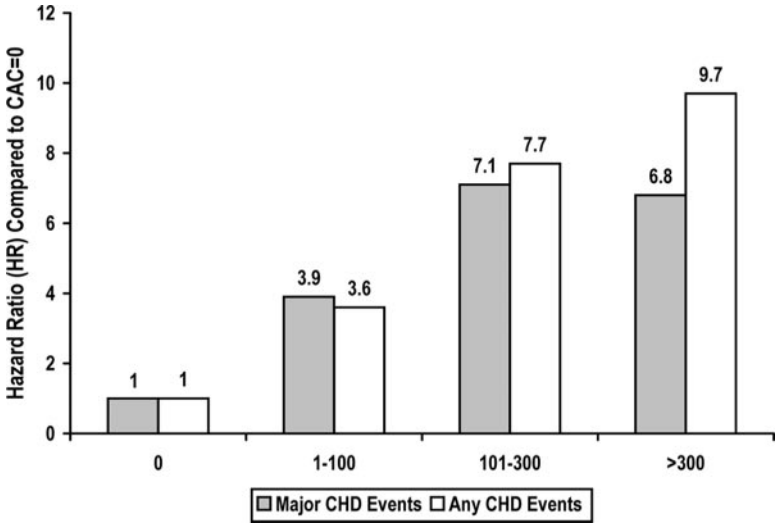


Chart 3-3. HRs for CHD events associated with coronary calcium scores: US adults 45 to 84 years of age (reference group CAC=0). Source: Data derived from Detrano et al.[5] All HRs $P<0.0001$. Major CHD events included MI and death due to CHD; any CHD events included major CHD events plus definite angina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

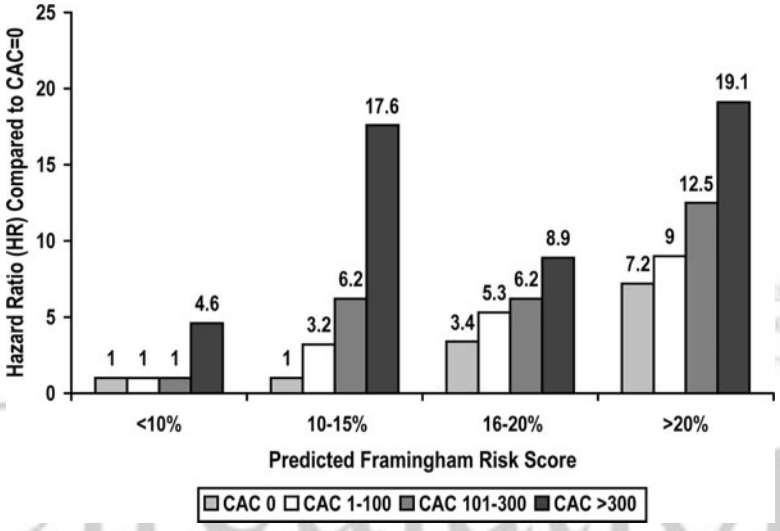


Chart 3-4. HRs for CHD events associated with coronary calcium scores: US adults (reference group CAC=0 and FRS <10%). CHD events included nonfatal MI and death due to CHD. Source: Reprinted from Greenland et al,[6] with permission. Copyright © 2004, American Medical Association. All rights reserved.

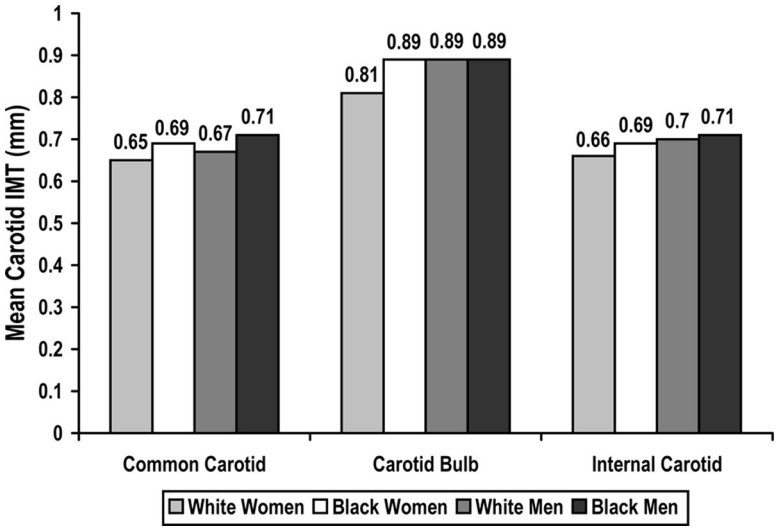


Chart 3-5. Mean values of carotid IMT for different carotid artery segments in younger adults by race and sex. Source: Reprinted from Urbina et al,[8] with permission from Elsevier. Copyright 2002.

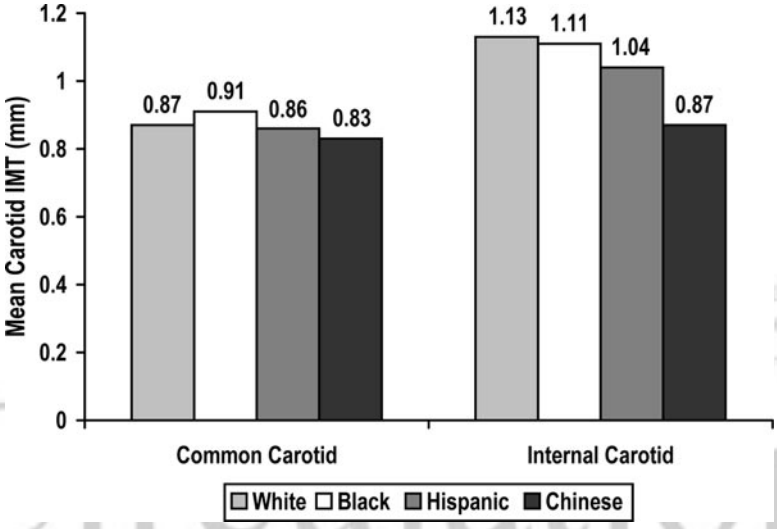


Chart 3-6. Mean values of carotid IMT for different carotid artery segments in older adults, by race. Source: Data derived from Manolio et al.[11]

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

Coronary Heart Disease

ICD-9 410-414, 429.2; ICD-10 I20-I25; see Glossary (Chapter 21) for details and definitions. See Tables 4-1 and 4-2. See Charts 4-1 through 4-8.

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
BMI	body mass index
BP	blood pressure
BRFSS	Behavioral Risk Factor Surveillance System
CAD	coronary artery disease
CDC	Centers for Disease Control and Prevention
CHD	coronary heart disease
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
ECG	electrocardiogram
ED	emergency department
EMS	emergency medical services
FHS	Framingham Heart Study
GRACE	Global Registry of Acute Coronary Events
HF	heart failure
HMO	health maintenance organization
ICD	*International Classification of Diseases*
MET	metabolic equivalent
MI	myocardial infarction
NAMCS	National Ambulatory Medical Care Survey
NCHS	National Center for Health Statistics
NH	non-Hispanic
NHAMCS	National Hospital Ambulatory Medical Care Survey
NHANES	National Health and Nutrition Examination Survey
NHDS	National Hospital Discharge Survey
NHIS	National Health Interview Study
NHLBI	National Heart, Lung, and Blood Institute
NSTE ACS	non–ST-segment–elevation acute coronary syndromes
OB/GYN	obstetrics/gynecology
PA	physical activity
PCI	percutaneous coronary intervention
STEMI	ST-segment–elevation MI
UA	unstable angina
WISE	Women's Ischemia Syndrome Evaluation

Prevalence

- Among American Indians/Alaska Natives ≥18 years of age, it is estimated that 5.6% have CHD (estimate considered unreliable). Among blacks, the rate was 6.0%; among whites, it was 6.1%; and among Asians, it was 4.3% (NHIS, NCHS).[1]
- Data from 2007 from the BRFSS survey of the CDC found that 4.2% of respondents had been told that they had had MI. The highest prevalence was in Kentucky and West Virginia (6.0%). The lowest prevalence was in Alaska (2.3%). In the same survey, 4.1% of respondents were told that they had angina or CHD. The highest prevalence was in West Virginia (7.6%), and the lowest was in Utah (2.4%).[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 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 hazard rate ratios (95% 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). Diabetes mellitus was somewhat more predictive in white women than in other groups. Hazard rate ratios 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]
- 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]

- 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.[8]
- Analysis of data from NHANES III and NHANES 1999–2002 (NCHS) showed that in adults 20 to 74 years of age, the overall distribution of 10-year risk of developing CHD changed little during this time. Among the 3 racial/ethnic groups, blacks had the highest proportion of participants in the high-risk group.[9]

Mortality

CHD caused ≈1 of every 5 deaths in the United States in 2005. CHD mortality was 445 687.[10] CHD total-mention mortality was 607 000. MI mortality was 151 004. MI total-mention mortality was 191 000 (NHLBI; NCHS public use data files). Preliminary 2006 mortality was 424 892. The preliminary death rate was 134. CHD is the largest major killer of American males and females.[11] Approximately every 25 seconds, an American will suffer a coronary event, and approximately every minute, someone will die from one. Approximately 37% of the people who experience a coronary attack in a given year will die from it, and ≈16% who experience a heart attack (MI) will die from it (AHA computation). Approximately every 34 seconds, an American will suffer an MI. The percentage of CHD out-of-hospital deaths in 2005 was 69%.

- According to NCHS Data Warehouse mortality data, 309 000 CHD deaths occur out of hospital or in hospital EDs annually (2005, *ICD-10* codes I20 to I25).[12]
- A study of 1275 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 heart disease, compared with 0.8/1000 subject-years in subjects without heart disease. In subgroups with heart disease, incidence was 13.6/1000 subject-years in subjects with prior MI and 21.9/1000 subject-years in subjects with HF.[13]
- 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.[14]
- From 1995 to 2005, the annual death rate from CHD declined 34.3%, but the actual number of deaths declined only 19.4%. In 2005, the overall CHD death rate was 144.4 per 100 000 population. The death rates were 187.7 for white males and 213.9 for black males; for white females, the rate was 110.0, and for black females it was 140.9.[13]
  - 2005 age-adjusted death rates for CHD were 118.0 for Hispanics or Latinos, 96.2 for American Indians or Alaska Natives, and 81.0 for Asians or Pacific Islanders.[15]
- Approximately 82% 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.[16]
- On the basis of data from the FHS of the NHLBI[3]:
  - 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[17]:
  - 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 from 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[18]:
  - secondary preventive therapies after MI or revascularization (11%),
  - initial treatments for AMI or unstable angina (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[18]:
  - lower total cholesterol (24%),
  - lower systolic BP (20%),
  - lower smoking prevalence (12%),
  - and increased physical inactivity (5%).
- Nevertheless, these favorable improvements in risk factors were partially offset by increases in BMI and in diabetes prevalence, which accounted for an increased number of deaths (8% and 10%, respectively).
- Analysis of CHD mortality data among US adults 35 to 54 years of age showed that the annual percent change in (age-adjusted) mortality slowed markedly from 1980 to 2002 in both men and women. Particularly noteworthy is that the mortality rate among women 35 to 44 years of age has been increasing on average by 1.3% per year since 1997.[19]

Risk Factors

- 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 the 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.[20]
- 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.[21]
- 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 MET increment in exercise capacity reduced risk by 13%.[22]
- 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% 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.[23]
- 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 diabetes mellitus.[24]
- 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.[25]

Awareness of Warning Signs and Risk Factors for Heart Disease

- Data from the Women Veteran Cohort showed that 42% of women ≥35 years of age were concerned about heart disease. Only 8% to 20% were aware that CAD is the major cause of death for women.[26]
- 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 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 calling 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%).[27]
- A 2004 national study of physician awareness and adherence to CVD prevention guidelines showed that <1 in 5 physicians knew that more women than men die each year from CVD.[28]
- 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 delaying ≥4 hours, which indicates that there has been little improvement in the speed at which patients with MI symptoms arrive at the hospital after 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.[29]
- A survey of >500 internists and OB/GYNs attending presentations developed for the NY State Women and Heart Disease Physician Education Initiative found that 71.5% correctly responded to 13 questions assessing knowledge of coronary risk prevention. Of the attendees, 71.5% were internists, and 42.7% were women. Almost one third of internists and half of OB/GYNs did not know that tobacco use was the leading cause of MI in young women. For patients who smoked tobacco, only two thirds of internists and 55.4% of OB/GYNs reported suggesting a quit date.[30]
- A study of the perceptions of susceptibility and seriousness of heart disease and the relationships between socioeconomic status, age, and knowledge of heart disease and its risk factors was conducted among 194 educated black women. Participants did not perceive themselves to be at high risk for developing heart disease, although they did perceive heart disease as serious. Black women who were older perceived heart disease to be more serious than did their younger counterparts. Older women and those with higher socioeconomic status knew more about heart disease and risk

factors. Neither socioeconomic status nor age moderated the relationship between knowledge and perceived susceptibility or seriousness.[31]

- According to 2003 data from the BRFSS (CDC), 36.5% of all women surveyed had multiple risk factors for heart disease 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 compared with 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.[32]
- In an effort to understand why women delay seeking treatment for symptoms of an AMI, 30 interviews were conducted to determine black, Hispanic, and white women's perceptions of heart disease risk and whether differences existed on the basis of the participants' race or ethnicity. Perceptions of heart disease risk were similar between groups, with women generally believing that they were at risk for heart disease because of family history, diet, and obesity. Racial and ethnic differences were noted, however, in risk reduction and anticipated treatment-seeking behaviors.[33]
- 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.[34]

Aftermath

- Depending on their sex and clinical outcome, people who survive the acute stage of an MI have a chance of illness and death 1.5 to 15 times higher than that of the general population. Among these people, the risk of another MI, sudden death, AP, HF, and stroke—for both men and women—is substantial (FHS, NHLBI).[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 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.[35]
- 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 from 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 than women, among Hispanics, among those married, among those with higher education, and among those with higher levels of household income.[36]

Hospital Discharges and Ambulatory Care Visits

- From 1996 to 2006, the number of inpatient discharges from short-stay hospitals with CHD as the first-listed diagnosis decreased from 2 263 000 to 1 760 000 (NHDS, NCHS).
- Data from Ambulatory Medical Care Utilization Estimates for 2006 showed the number of visits for CHD as 11 371 000 (NAMCS, NHAMCS).[37]
- 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 more women than men died in the hospital: 9.3% of women died in the hospital compared with 6.2% of men.[38]

Cost

- The estimated direct and indirect cost of CHD for 2009 is $165.4 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 acute MI, $12 977 per discharge for coronary atherosclerosis, and $10 630 per discharge for other ischemic heart disease).[31,39]

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.[40]

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, NSTEMI, and STEMI share common pathophysiological origins related to coronary plaque progression, instability, or rupture with or without luminal thrombosis and vasospasm.)

- A conservative estimate for the number of discharges with ACS from hospitals in 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 are included, the corresponding numbers of inpatient hospital discharges were 1 365 000 unique hospitalizations for ACS; 765 000 are male and 600 000 are 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 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.[41] The AHA Get With the Guidelines project found that 32% of the MI patients in the CAD module are STEMI patients (AHA Get With the Guidelines Staff, personal communication, 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.[42]

- Analysis of data from the GRACE multinational observational cohort study of patients with ACS found evi-

dence of a change in practice for both pharmacological and interventional treatments in patients with either STEMI or NSTE ACS. These changes are 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.[43]

- A study of patients with NSTE ACS treated at 350 US hospitals found that up to 25% of opportunities to provide ACC/AHA guideline–recommended care were missed in current practice. Composite guideline adherence rate was significantly associated with in-hospital mortality.[44]
- A study of hospital process performance in 350 centers of nearly 65 000 patients enrolled in the CRUSADE National Quality Improvement Initiative found that ACC/AHA guideline–recommended treatments were adhered to in 74% of eligible instances.[44]

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.[45]

Incidence

- Only 18% of coronary attacks are preceded by long-standing AP (NHLBI computation of FHS follow-up since 1986).
- The annual rates per 1000 population of new episodes of AP for nonblack men are 28.3 for those 65 to 74 years of age, 36.3 for those 75 to 84 years of age, and 33.0 for those ≥85 years of age. For nonblack women in the same age groups, the rates are 14.1, 20.0, and 22.9, respectively. For black men, the rates are 22.4, 33.8, and 39.5; for black women, the rates are 15.3, 23.6, and 35.9, respectively (CHS, NHLBI).[7]
- On the basis of 1987–2001 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; 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 from AP. These are included as a portion of total deaths from CHD.

Cost

For women with nonobstructive CHD enrolled in the 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.[46]

References

1. Pleis JR, Lucus JW. Summary health statistics for U. S. adults: National Health Interview Survey, 2007. *Vital Health Stat 10*. In press.
2. CDC. 2007 Behavioral Risk Factor Surveillance System. Available at: http://www.cdc.gov/brfss. Accessed September 15, 2008.
3. Thom TJ, Kannel WB, Silbershatz H, D'Agostino RB. Cardiovascular disease in the United States and preventive approaches. In: Fuster V, Alexander RW, O'Rourke RA, eds. *Hurst's The Heart, Arteries and Veins*. 10th ed. New York, NY: McGraw-Hill; 2001: 3–7.
4. Boland LL, Folsom AR, Sorlie PD, Taylor HA, Rosamond WD, Chambless LE, Cooper LS. Occurrence of unrecognized myocardial infarction in subjects aged 45 to 65 years: the ARIC Study. *Am J Cardiol*. 2002;90:927–931.
5. Lloyd-Jones DM, Larson MG, Beiser A, Levy D. Lifetime risk of developing coronary heart disease. *Lancet*. 1999;353:89–92.
6. Jones DW, Chambless LE, Folsom AR, Heiss G, Hutchinson RG, Sharrett AR, Szklo M, Taylor HA Jr. Risk factors for coronary heart disease in African Americans: the Atherosclerotic Risk in Communities Study, 1987–1997. *Arch Intern Med*. 2002;162:2565–2571.
7. National Heart, Lung, and Blood Institute. *Incidence and Prevalence: 2006 Chart Book on Cardiovascular and Lung Diseases*. Bethesda, Md: National Institutes of Health; 2006.
8. Ali T, Jarvis B, O'Leary M. *Strong Heart Study Data Book: A Report to American Indian Communities*. Rockville, Md: National Institutes of Health, National Heart, Lung, and Blood Institute; 2001.
9. Ajani UA, Ford ES. Has the risk for coronary heart disease changed among U.S. adults? *J Am Coll Cardiol*. 2006;48:1177–1182.
10. Heron MP, Hoyert DL, Xu J, Scott C, Tejada-Vera B. Deaths: preliminary data for 2006. *Natl Vital Stat Rep*. 2008;56(16):1–52.
11. National Center for Health Statistics, Centers for Disease Control and Prevention. Compressed mortality file: underlying cause of death, 1979 to 2005. Atlanta, Ga: Centers for Disease Control and Prevention. Available at: http://wonder.cdc.gov/mortSQL.html. Accessed May 29, 2008.
12. National Center for Health Statistics. Vital Statistics of the United States, Data Warehouse. Available at: http://www.cdc.gov/nchs/datawh.htm. Accessed May 25, 2008.
13. Rea TD, Pearce RM, Raghunathan TE, Lemaitre RN, Sotoodehnia N, Jouven X, Siscovick DS. Incidence of out-of-hospital cardiac arrest. *Am J Cardiol*. 2004;93:1455–1460.
14. Fox CS, Evans JC, Larson MG, Kannel WB, Levy D. Temporal trends in coronary heart disease mortality and sudden cardiac death from 1950–1999: the Framingham Heart Study. *Circulation*. 2004;110:522–527.
15. National Center for Health Statistics. *Health, United States, 2007: With Chartbook on Trends in the Health of Americans*. Hyattsville, Md: National Center for Health Statistics; 2007. Available at: http://www.cdc.gov/nchs/hus.htm. Accessed September 15, 2008.
16. Kung HC, Hoyert DL, Xu JQ, Murphy SL. Deaths: final data for 2005. *Natl Vital Stat Rep*. 2008;56(10):1–120.
17. National Registry of Myocardial Infarction. Available at: www.nrmi.org/nrmi_data.html. Accessed February 20, 2008.
18. Ford ES, Ajani UA, Croft JB, Critchley JA, LaBarthe DR, Kottke TE, Giles WH, Capewell S. Explaining the decrease in U.S. deaths from coronary disease, 1980–2000. *N Engl J Med*. 2007;356:2388–2398.
19. Ford ES, Capewell S. Coronary heart disease mortality among young adults in the U.S. from 1980 through 2002: concealed leveling of mortality rates. *J Am Coll Cardiol*. 2007;50:2128–2132.
20. Greenland P, Knoll MD, Stamler J, Neaton JD, Dyer AR, Garside DB, Wilson PW. Major risk factors as antecedents of fatal and nonfatal coronary heart disease events. *JAMA*. 2003;290:891–897.
21. Yusuf S, Hawken S, Ounpuu S, Dans T, Avezum A, Lanas F, McQueen M, Budaj A, Pais P, Varigos J, Lisheng L, for the INTERHEART Study Investigators. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. *Lancet*. 2004;364:937–952.

22. Balady GJ, Larson MG, Vasan RS, Leip EP, O'Donnell CJ, Levy D. Usefulness of exercise testing in the prediction of coronary disease risk among asymptomatic persons as a function of the Framingham risk score. *Circulation*. 2004;110:1920–1925.
23. Vasan RS, Sullivan LM, Wilson PW, Sempos CT, Sundstrom J, Kannel WB, Levy D, D'Agostino RB. Relative importance of borderline and elevated levels of coronary heart disease risk factors [published correction appears in *Ann Intern Med*. 2005;142:681]. *Ann Intern Med*. 2005;142: 393–402.
24. Kuller LH, Arnold AM, Psaty BM, Robbins JA, O'Leary DH, Tracy RP, Burke GL, Manolio TA, Chaves PH. 10-year follow-up of subclinical cardiovascular disease and risk of coronary heart disease in the Cardiovascular Health Study. *Arch Intern Med*. 2006;166:71–78.
25. Zhao G, Ford ES, Li C, Mokdad AH. Are United States adults with coronary heart disease meeting physical activity recommendations? *Am J Cardiol*. 2008;101:557–561.
26. Biswas MS, Calhoun PS, Bosworth HB, Bastian LA. Are women worrying about heart disease? *Womens Health Issues*. 2002;12: 204–211.
27. Centers for Disease Control and Prevention (CDC). Disparities in adult awareness of heart attack warning signs and symptoms—14 states, 2005. *MMWR Morb Mortal Wkly Rep*. 2008;57:175–179.
28. Mosca L, Linfante AH, Benjamin EJ, Berra K, Hayes SN, Walsh BW, Fabunmi RP, Kwan J, Mills T, Simpson SL. National study of physician awareness and adherence to cardiovascular disease prevention guidelines. *Circulation*. 2005;111:499–510.
29. McGinn AP, Rosamond WD, Goff DC Jr, Taylor HA, Miles JS, Chambless L. Trends in prehospital delay time and use of emergency medical services for acute myocardial infarction: experience in 4 US communities from 1987–2000. *Am Heart J*. 2005;150:392–400.
30. Barnhart J, Lewis V, Houghton JL, Charney P. Physician knowledge levels and barriers to coronary risk prevention in women survey results from the women and heart disease physician education initiative. *Womens Health Issues*. 2007;17:93–100.
31. Jones DE, Weaver MT, Grimley D, Appel SJ, Ard J. Health belief model perceptions, knowledge of heart disease, and its risk factors in educated African-American women: an exploration of the relationships of socioeconomic status and age. *J Natl Black Nurses Assoc*. 2006;17:13–23.
32. Hayes DK, Denny CH, Keenan NL, Croft JB, Sundaram AA, Greenlund KJ. Racial/ethnic and socioeconomic differences in multiple risk factors for heart disease and stroke in women: behavioral risk factor surveillance system, 2003. *J Womens Health (Larchmt)*. 2006;15:1000–1008.
33. Arslanian-Engoren C. Black, Hispanic and white women's perception of heart disease. *Prog Cardiovasc Nurs*. 2007;22:13–19.
34. Dracup K, McKinley S, Doering LV, Riegel B, Meischke H, Moser DK, Pelter M, Carlson B, Aitken L, Marshall A, Cross R, Paul SM. Acute coronary syndrome: what do patients know? *Arch Intern Med*. 2008;168: 1049–1054.
35. Witt BJ, Jacobsen SJ, Weston SA, Killian JM, Meverden RA, Allison TG, Reeder GS, Roger VL. Cardiac rehabilitation after myocardial infarction in the community. *J Am Coll Cardiol*. 2004;44:988–996.
36. Centers for Disease Control and Prevention (CDC). Receipt of outpatient cardiac rehabilitation among heart attack survivors—United States, 2005. *MMWR Morb Mortal Wkly Rep*. 2008;57:89–94.
37. Schappert SM, Rechsteiner EA. Ambulatory medical care utilization estimates for 2006. *Natl Health Stat Rep*. 2008;8:1–29.
38. Elixhauser A, Jiang HJ. Hospitalizations for women with circulatory disease, 2003. Rockville, Md: Agency for Healthcare Research and Quality; May 2006. HCUP Statistical Brief #5. Available at: http://www.hcup-us.ahrq.gov/reports/ statbriefs/sb5.pdf. Accessed August 2007. Accessed September 15, 2008.
39. Centers for Medicare & Medicaid Services. *Health Care Financing Review: Medicare & Medicaid Statistical Supplement*. Table 5.5: Discharges, Total Days of Care, and Program Payments for Medicare Beneficiaries Discharged from Short-Stay Hospitals, by Principal Diagnoses Within Major Diagnostic Classifications (MDCs): Calendar Year 2006. Baltimore, Md: Centers for Medicare and Medicaid Services; 2005. Available at: http://www.cms.hhs.gov/MedicareMedicaidStatSupp/. Accessed August 28, 2008.
40. DeFrances CJ, Lucas Ca, Buie VC, Golosinskiy A. 2006 National Hospital Discharge Survey. *Natl Health Stat Rep*. 2008;5:1–20.
41. Roe MT, Parsons LS, Pollack CV Jr, Canto JG, Barron HV, Every NR, Rogers WJ, Peterson ED, for the National Registry of Myocardial Infarction Investigators. Quality of care by classification of myocardial infarction. *Arch Intern Med*. 2005;165:1630–1636.
42. Mandelzweig L, Battler A, Boyko V, Bueno H, Danchin N, Filippatos G, Gitt A, Hasdai D, Hasin Y, Marrugat J, Van de Werf F, Wallentin L, Behar S, for the Euro Heart Survey Investigators. The second Euro Heart Survey on acute coronary syndromes: characteristics, treatment, and outcome of patients with ACS in Europe and the Mediterranean Basin in 2004. *Eur Heart J*. 2006;27:2285–2293.
43. Fox KAA, Steg PG, Eagle KA, Goodman SG, Anderson FA Jr, Granger CB, Flather MD, Budaj A, Quill A, Gore JM. Decline in rates of death and heart failure in acute coronary syndromes, 1999–2006. *JAMA*. 2007; 297:1892–1900.
44. Peterson ED, Roe MT, Mulgund J, DeLong ER, Lytle BL, Brindis RG, Smith SC Jr, Pollack CV Jr, Newby LK, Harrington RA, Gibler WB, Ohman EM. Association between hospital process performance and outcomes among patients with acute coronary syndromes. *JAMA*. 2006; 295:1912–1920.
45. Ford ES, Giles WH. Changes in preference of nonfatal coronary heart disease in the United States from 1971–1994. *Ethn Dis*. 2003;13: 85–93.
46. Shaw LJ, Merz CN, Pepine CJ, Reis SE, Bittner V, Kip KE, Kelsey SF, Olson M, Johnson BD, Mankad S, Sharaf BL, Rogers WJ, Pohost GM, Sopko G, for the Women's Ischemia Syndrome Evaluation (WISE) Investigators. The economic burden of angina in women with suspected ischemic heart disease: results from the National Institutes of Health—National Heart, Lung, and Blood Institute–sponsored Women's Ischemia Syndrome Evaluation. *Circulation*. 2006;114:894–904.
47. Wilson PW, D'Agostino RB, Levy D, Belanger AM, Silbershatz H, Kannel WB. Prediction of coronary heart disease using risk factor categories. *Circulation*. 1998;97:1837–1847.

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, 2005 All Ages	Mortality,* MI, 2005 All Ages	Hospital Discharges, CHD, 2006 All Ages	Cost, CHD, 2009
Both sexes	16 800 000 (7.6%)	7 900 000 (3.6%)	1 255 000	935 000	445 687	151 004	1 760 000	$165.4 billion
Males	8 700 000 (8.6%)	4 700 000 (4.7%)	740 000	565 000	232 115 (52.1%)‡	80 079 (53.0%)‡	1 056 000	...
Females	8 100 000 (6.8%)	3 200 000 (2.7%)	515 000	370 000	213 572 (47.9%)‡	70 925 (47.0%)‡	704 000	...
NH white males	8.8%	4.9%	675 000§	...	203 924	70 791	...	...
NH white females	6.6%	3.0%	445 000§	...	186 497	61 573	...	...
NH black males	9.6%	5.1%	70 000§	...	22 933	7527	...	...
NH black females	9.0%	2.2%	65 000§	...	23 094	8009	...	...
Mexican American males	5.4%	2.5%	...	...	...	...	...	...
Mexican American females	6.3%	1.1%	...	...	...	...	...	...
Hispanic or Latino,† age ≥18 y	5.7%	...	...	...	...	...	...	...
Asian,† age ≥18 y	4.3%	...	...	...	...	...	...	...
American Indian/Alaska Native,† age ≥18 y	5.6%	...	...	...	...	...	...	...

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 2005–2006 (NCHS) and NHLBI. Total data are for Americans ≥20 years of age; percentages for racial/ethnic groups are age adjusted for ≥20 years of age. These data are based on self-reports. Estimates from NHANES 2005–2006 (NCHS) applied to 2006 population estimates (≥20 years of age). 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 2007—data are weighted percentages for Americans ≥18 years of age. Estimates for American Indians/Alaska Natives are considered unreliable.[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	9 800 000 (4.4%)	500 000	41 000
Males	4 300 000 (4.3%)	320 000	17 000
Females	5 500 000 (4.5%)	180 000	24 000
NH white males	4.1%	...	...
NH white females	4.3%	...	...
NH black males	4.4%	...	...
NH black females	6.7%	...	...
Mexican American males	3.5%	...	...
Mexican American females	4.5%	...	...

AP is chest pain or discomfort resulting 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 2005–2006 (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 2005–2006 (NCHS) 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.

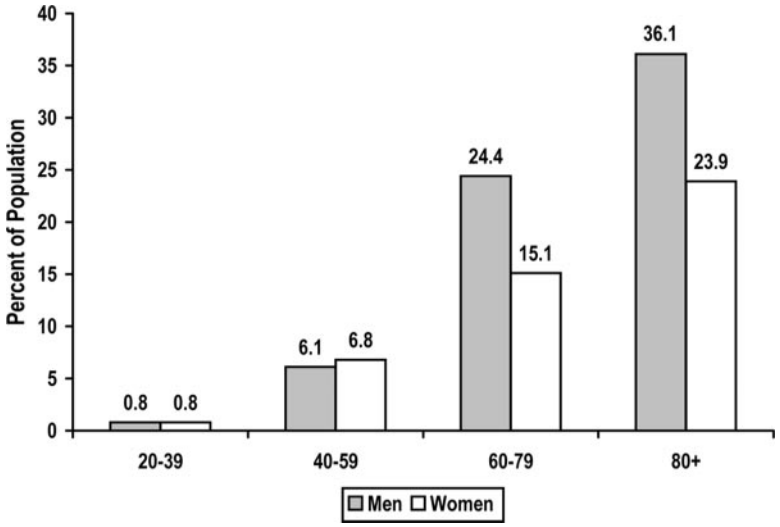


Chart 4-1. Prevalence of CHD by age and sex (NHANES: 2005–2006). Source: NCHS and NHLBI.

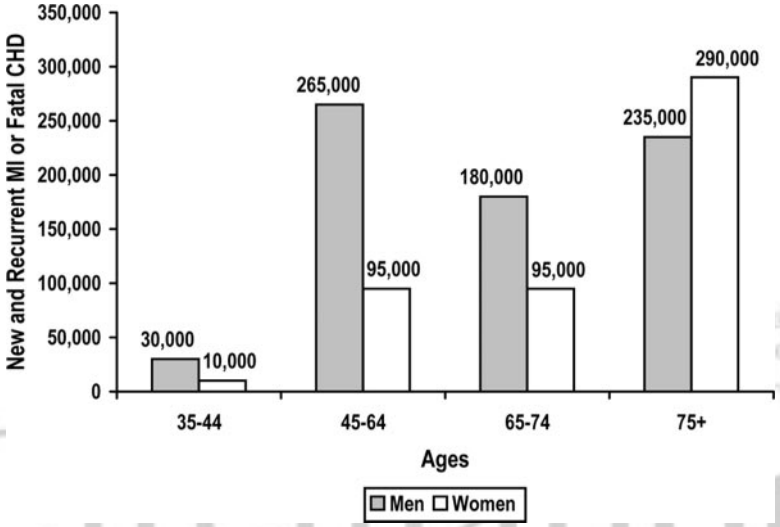


Chart 4-2. Annual number of adults having diagnosed heart attack by age and sex (ARIC Surveillance: 1987–2004 and CHS: 1989–2004). Source: NHLBI. These data include MI and fatal CHD but not silent MI.

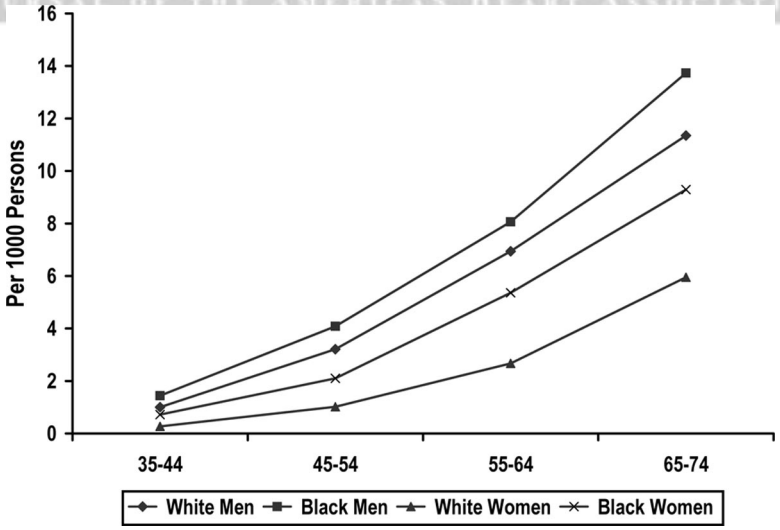


Chart 4-3. Annual rate of first heart attacks by age, sex, and race (ARIC Surveillance: 1987–2004). Source: NHLBI.

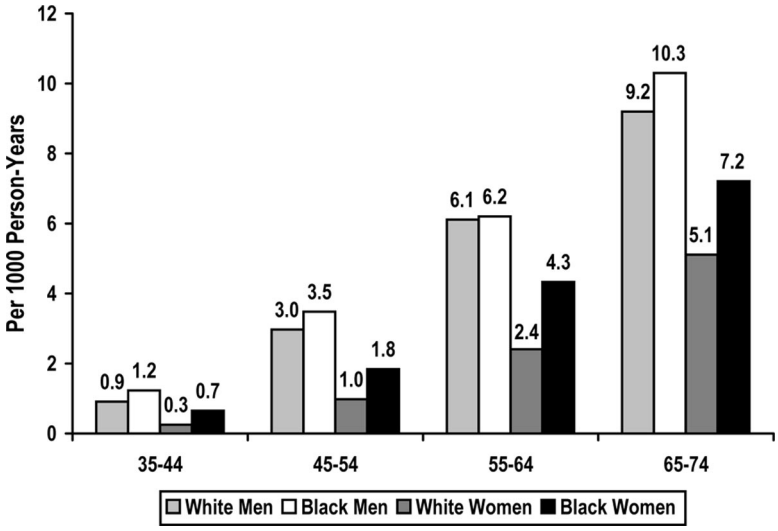


Chart 4-4. Incidence of MI* by age, race, and sex (ARIC Surveillance, 1987–2004). *MI diagnosis by expert committee based on review of hospital records. Source: Unpublished data from ARIC, NHLBI.

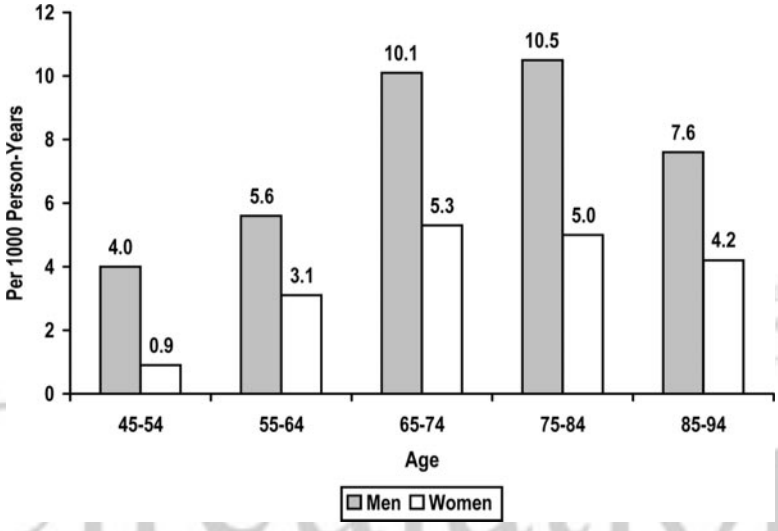
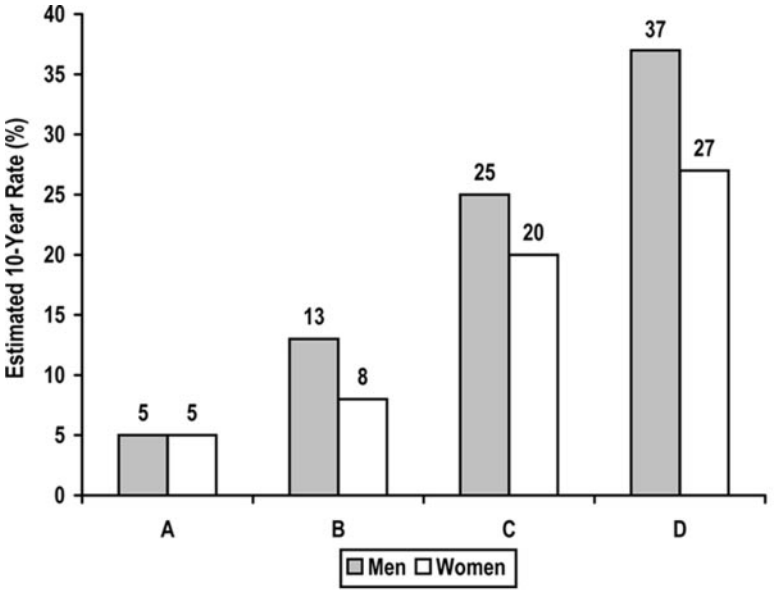


Chart 4-5. Incidence of AP* by age, race, and sex (FHS 1980–2002/2003). *AP uncomplicated based on physician interview of patient. (Rate for women 45 to 54 years of age considered unreliable.) Source: NHLBI.[7]



	A	B	C	D
BP	120/80	140/90	140/90	140/90
Cholesterol	200	240	240	240
HDL cholesterol	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.[47]

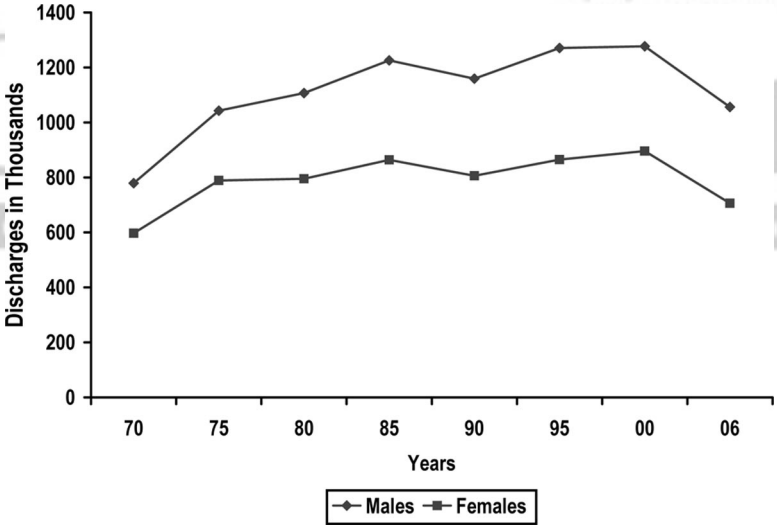


Chart 4-7. Hospital discharges for CHD by sex (United States: 1970–2006). Note: Hospital discharges include people discharged alive, dead, and "status unknown." Source: NHDS/NCHS.

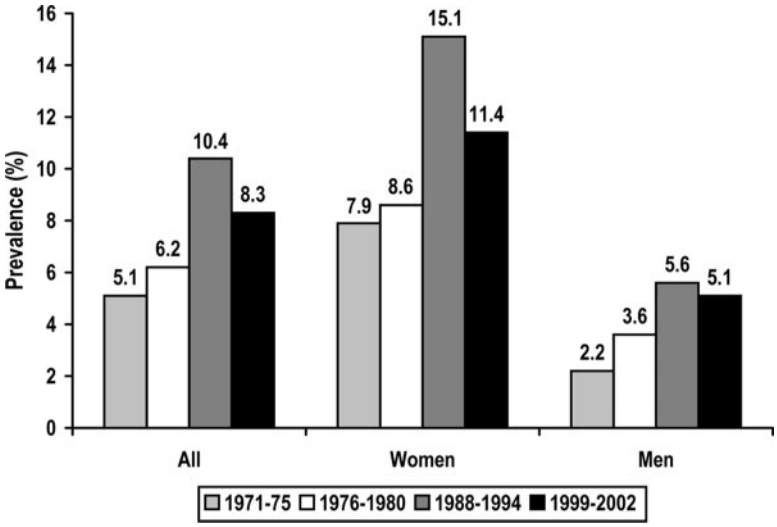


Chart 4-8. Prevalence of low CHD risk, overall and by sex (NHANES: 1971–2002). Source: Personal communication with NHLBI, June 28, 2007. Low risk is defined as systolic BP <120 mm Hg and diastolic BP <80 mm Hg; cholesterol <200 mg/dL; BMI <25 kg/m^2; currently not smoking cigarettes; and no prior MI or DM.

5. Stroke (Cerebrovascular Disease)

ICD-9 430-438, 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
CVD	cardiovascular disease
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
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
SIPP	Survey of Income and Program Participation
STOP	Stroke Prevention Trial in Sickle Cell Anemia
TIA	transient ischemic attack
WEST	Women's Estrogen for Stroke Trial

Prevalence

- 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 (might be of any race), 6.0% were American Indian/Alaska Native, and 4.6% were multiracial (see Table 5-2).[1]
- Data from the 2007 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 Missouri (3.7%), and the lowest was in Utah (1.6%).[2]
- Among American Indians/Alaska Natives ≥18 years of age, the estimated prevalence of stroke is considered unreliable. Among blacks, the prevalence was 3.7%; among whites, it was 2.2%; and among Asians, it was 2.6% (NHIS, NCHS).[3]
- 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]
- Data from the Strong Heart Study show that the prevalence of stroke in American Indian men 45 to 74 years of age ranges from 0.2% to 1.4%. Among American Indian women in the same age group, the prevalence ranges from 0.2% to 0.7%.[6]
- The prevalence of stroke 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 less education, 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).[7]

Transient Ischemic Attack

- The prevalence of transient ischemic attack (TIA; a mini-stroke with symptoms that last <24 hours) increases with age.[8]
- Approximately 15% of all strokes are heralded by a TIA.[8]
- One third of spells characterized as TIAs according to the classic definition (focal neurological deficits that resolve within 24 hours) would be considered infarctions on the basis of diffusion-weighted magnetic resonance imaging findings.[9]
- In population-based studies, the age- and gender-adjusted incidence rates for TIA range from 68.2 to 83 per 100 000. Men and blacks have higher rates of TIA.[10,11]
- Approximately half of all patients who experience a TIA fail to report it to their healthcare providers.[12]

- After TIA, the 90-day risk of stroke is 3% to 17.3% and is highest within the first 30 days.[11–14]
- Within 1 year of TIA, up to one fourth of patients will die.[11,15]
- 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%/y).[16]
- 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.[17]

Incidence

Each year, ≈795 000 people experience a new or recurrent stroke. Approximately 610 000 of these are first attacks, and 185 000 are recurrent attacks (GCNKSS, NINDS, and NHLBI; GCNKSS, NINDS data for 1999 provided July 9, 2008; estimates compiled by NHLBI).

- On average, every 40 seconds, someone in the United States has a stroke (AHA computation based on latest available data).
- Each year, ≈55 000 more women than men have a stroke (GCNKSS, NINDS).
- Men's stroke incidence rates are greater than women's 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).[18]
- Data from the GCNKSS, NINDS show that the annual incidence of first-ever hospitalized stroke did not change significantly between study periods: 158 per 100 000 in both 1993–1994 and 1999. Blacks continue to have a higher stroke incidence than whites, especially among the young. Despite advances in stroke prevention treatments during the 1990s, the incidence of hospitalized stroke did not decrease within the population being studied. Case fatality also did not change between study periods. Excess stroke mortality rates seen in blacks nationally are likely the result of excess stroke incidence and not case fatality, and the racial disparity in stroke incidence did not change over time.[19]
- 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).[18] On the basis of 1987–2001 data from the ARIC study of 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.[18]
- Of all strokes, 87% are ischemic, 10% are intracerebral hemorrhage, and 3% are subarachnoid hemorrhage strokes (GCNKSS, NINDS 1999).[18]
- 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: risk ratio, 2.04; 95% CI, 1.55 to 2.69; 60 to 74 years of age: risk ratio, 1.58; 95% CI, 1.31 to 1.91) but not at older ages (≥75 years of age: risk ratio, 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.[20]
- Among American Indians 65 to 74 years of age, the annual rates per 1000 population of new and recurrent strokes are 6.1 for men and 6.6 for women.[6]
- 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.[21]
- 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%).[22]
- 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.[23]

Mortality

- Stroke accounted for ≈1 of every 17 deaths in the United States in 2005. Approximately 53% of stroke deaths in 2005 occurred out of the hospital.[24] Stroke mortality in 2005 was 143 579; total-mention mortality in 2005 was ≈242 000 (NHLBI; NCHS public use data files).
- Preliminary stroke mortality in 2006 was 137 265, and the preliminary death rate was 43.6.[25]
- 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).

- On average, every 3 to 4 minutes, someone dies of a stroke (NCHS, NHLBI).
- 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.[26]
- 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.[27]
- From 1995 to 2005, the annual stroke death rate fell 29.7%, and the actual number of stroke deaths declined 13.5%.[28]
- 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.[28]
- The 2005 overall death rate for stroke was 46.6 per 100 000. Death rates were 44.7 for white males, 70.5 for black males, 44.0 for white females, and 60.7 for black females.[28]
- In 2005, death rates for stroke were 38.0 for Hispanic or Latino men and 33.5 for women, 41.5 for Asian or Pacific Islander men and 36.3 for women, and 31.3 for American Indian/Alaska Native men and 37.1 for women.[29]
- Because women live longer than men, more women than men die of stroke each year. Women accounted for 60.6% of US stroke deaths in 2005 (AHA computation).
- 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.[30]
- In 2002, death certificate data showed that the mean age at stroke death was 79.6 years; however, men 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.[31]
- 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 men did 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.[32]
- 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.[33]

Stroke Risk Factors

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

- BP is a powerful determinant of stroke risk. Subjects with BP <120/80 mm Hg have approximately half the lifetime risk of stroke of subjects with hypertension.
- TIAs confer a substantial short-term risk of stroke, hospitalization for cardiovascular events, and death. Of 1707 TIA patients evaluated in the ED of a large healthcare plan, 180, or 10%, developed stroke within 90 days. Ninety-one patients, or 5%, did so within 2 days. Predictors of stroke included age >60 years, diabetes mellitus, focal symptoms of weakness or speech impairment, and TIA that lasted >10 minutes.[34]
- 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).
- AF is an independent risk factor for stroke, increasing risk ≈5-fold. 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.[35,36]
- 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.[37]
- 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. 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%).[38]
- 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).[39]
- In the FHS of the NHLBI, in participants <65 years of age, the risk of developing stroke/TIA was 4.21 times greater 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. Identifica-

tion of depressive symptoms at younger ages may have an impact on the primary prevention of stroke.[40]

- 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.[41]

Stroke Risk in Women

- Analysis of NHANES 1999–2004 data found that women 45 to 54 years of age are more than twice as likely as men to suffer a stroke. Women in the 45- to 54-year age group had a >4-fold higher likelihood of having a stroke than women 35 to 44 years of age.[42]
- 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.[43]
- Among postmenopausal women who were generally healthy, the Women's Health Initiative, 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.[44]
- In the Women's Health Initiative 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.[45]
- 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.[46]
- The Women's Estrogen for Stroke Trial (WEST) found that estrogen alone (1 mg 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 rise in overall stroke rate in the first 6 months.[47]
- 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.[44,48]
- A 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.[49]

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 postpartum. Intracerebral hemorrhage showed a small relative risk (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.[50]
- With Swedish administrative data, it was found that ischemic stroke and intracerebral hemorrhage, including subarachnoid hemorrhage, are increased in association with pregnancy. Compared with the risk of stroke among women who were not pregnant or who were in early pregnancy (up to the first 27 gestational weeks), women in the peripartum (from 2 days before to 1 day after delivery) and the puerperium (from 2 days before to 6 complete weeks after delivery) periods were at increased risk for all 3 major stroke types. The 3 days surrounding delivery were the time of highest risk.[51]
- 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, or 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.[52]

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 lower stroke risk associated with vigorous exercise among men (total stroke RR, 0.86 for exercise ≥5 times per week).[53] The Harvard Alumni Study showed a decrease in total stroke risk in men who were highly physically active (RR, 0.82).[54] 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% risk reduction in the most active category. In sex-stratified analysis, the trend was not significant in women.[55]
- 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.[56]
- 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).[57]
- PA—be it in sports, during leisure time, or at work—was related to reduced risk of ischemic stroke according to a follow-up of the ARIC/NHLBI cohort.[58]
- The association between type of PA and stroke risk has been investigated in several studies. In an evaluation of

walking and sports participation in a cohort of 73 265 men and women in Japan, the risks of stroke death for those in the highest category of walking and sports participation were 29% and 20% lower, respectively.[59] In a study of 47 721 men and women in Finland, the effect of leisure-time, occupational, and commuting PA on incident stroke was investigated. Significant trends toward lower stroke risk were associated with moderate and high levels of leisure-time activity and active commuting, with the strongest trend seen for ischemic stroke; a smaller but still significant benefit was seen with occupational activity.[60] A meta-analysis of reports of 31 observational studies conducted mainly in the United States and Europe found that moderate and high levels of leisure-time and occupational PA protected against total stroke, hemorrhagic stroke, and ischemic stroke.[61]

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 (41.3%), women (41.5%), and persons at higher education levels (47.6% for persons with a college degree or more) than among blacks and Hispanics (29.5% and 26.8%, respectively), men (34.5%), and persons at lower education levels (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).[62]
- 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.[63]
- 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).[64,65] In the 1995 survey,[65] 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.[66]
- 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 racial/ethnic and age groups.[67]
- 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, nonwhites, and those with lower income and fewer years of education were less likely to be knowledgeable about TIA.[10]
- Participants in the 1999 World Senior Games received 1 or more free screening tests and completed an awareness questionnaire. Results indicate that stroke education should be targeted at the very elderly, those who have less than a college education, and those who do not have a history of chronic disease. It also may be effectively directed toward those with higher cholesterol.[68]
- 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 and that 97% required the study of cardiology. Underrepresentation of neurology in internal medicine residency programs may contribute to stroke outcome.[69]
- 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 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.[70]
- A telephone survey of adults ≥45 years of age in 2 Montana counties showed that >70% were able to correctly name ≥2 warning signs for stroke. More than 45% were able to name ≥2 risk factors. Respondents 45 to 64 years of age, women, those with ≥12 years of education, and those with high cholesterol were more likely to correctly identify ≥2 warning signs than were those without these characteristics. Women and respondents 45 to 64 years of age also were more likely than men or older respondents to correctly identify ≥2 stroke risk factors.[71]
- 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.[72]
- 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. This finding highlights the need for educational intervention about cardiovascular emergencies targeted to Spanish-speaking communities.[73]

- 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 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.[74]

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).[75]

- 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.[76]
- On the basis of pooled data from the FHS, ARIC, and CHS studies of the NHLBI:
  - The percentages 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 percentages 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 percentages with a recurrent stroke in 5 years are 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 are:
    - 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.[77]
- In the NHLBI's FHS, among ischemic stroke survivors who were ≥65 years of age, these disabilities were observed at 6 months after stroke[78]:
  - 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 (2000–2001, NCHS) as analyzed by the CDC.[79]
- 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 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.[80–83]

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.[84]

- 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 fell ≈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.[85]
- 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.[86]
- In 2006, the number of ambulatory care visits for stroke was 3 982 000 (NAMCS, NHAMCS/NCHS).[86]
- 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 65- to 84-year-olds, 54.5% of stroke patients were women, whereas among the oldest age group, women constituted 69.7% of all stroke patients.[87]
- A first-ever county-level *Atlas of Stroke Hospitalizations Among Medicare Beneficiaries* was released 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).[33]

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.[88]

- 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 the Kaiser Permanente Medical Care Program. The risk 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.[89]
- 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 would have a stroke in the United States in 2004.[90]
- 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.[91]
- 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. Boys have a 1.28-fold higher risk of stroke than girls. There are no ethnic differences in stroke severity or case fatality, but boys have a higher case-fatality rate for ischemic stroke. 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.[92]
- Despite current treatment, 1 of 10 children with ischemic stroke will have a recurrence within 5 years.[93]
- 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.[94]
- From 1979 to 1998 in the United States, childhood mortality resulting from stroke declined by 58% overall, with reductions in all major subtypes.[95]
  - 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 <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 1991–1998 compared with 0.50 in 1999 and 0.17 in 2000 ($P<0.005$ for trend).[96]

Access to Stroke Care

- In 2006, there were 378 diplomates certified in Vascular Neurology by the American Board of Psychiatry and Neurology.[97]

- 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.[98]
- 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.[99]
- 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.[100]
- 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.[101]
- 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.[102]
- 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.[103]
- 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.[104]

Cost

The estimated direct and indirect cost of stroke for 2009 is $68.9 billion.

- In 2006, $3.9 billion ($7449 per discharge) was paid to Medicare beneficiaries discharged from short-stay hospitals for stroke.[105]
- 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.)[106]
- In a population study of stroke costs within 30 days of an acute event, 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).[107]
- Inpatient hospital costs for an acute stroke event account for 70% of first-year poststroke costs.[106]
- The largest components of acute-care costs were room charges (50%), medical management (21%), and diagnostic costs (19%).[108]
- 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.[107]
- 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.[108,109] 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.[94]

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).

References

1. Centers for Disease Control and Prevention (CDC). Prevalence of stroke: United States, 2005. *MMWR Morb Mortal Wkly Rep*. 2007;56:469–474.

2. CDC. Behavioral Risk Factor Surveillance System: turning information into health data. Available at: http://www.cdc.gov/brfss. Accessed September 15, 2008.
3. Pleis JR, Lucas JW. Summary health statistics for U. S. adults: National Health Interview Survey, 2007. *Vital Health Stat 10*. In press.
4. Howard G, Wagenknecht LE, Cai J, Cooper L, Kraut MA, Toole JF. Cigarette smoking and other risk factors for silent cerebral infarction in the general population. *Stroke*. 1998;29:913–917.
5. Bryan RN, Wells SW, Miller TJ, Elster AD, Jungreis CA, Poirier VC, Lind BK, Manolio TA. Infarctlike lesions in the brain: prevalence and anatomic characteristics at MR imaging of the elderly: data from the Cardiovascular Health Study. *Radiology*. 1997;202:47–54.
6. Tauqeer Ali T, Jarvis B, O'Leary M. *Strong Heart Study Data Book: A Report to American Indian Communities*. Rockville, Md: National Institutes of Health, National Heart, Lung, and Blood Institute; 2001.
7. Howard VJ, McClure LA, Meschia JF, Pulley L, Orr SC, Friday GH. High prevalence of stroke symptoms among persons without a diagnosis of stroke or transient ischemic attack in a general population: the Reasons for Geographic And Racial Differences in Stroke (REGARDS) Study. *Arch Intern Med*. 2006;166:1952–1958.
8. Hankey GJ. Impact of treatment of people with transient ischemic attack on stroke incidence and public health. *Cerebrovasc Dis*. 1996;6(suppl 1):26–33.
9. Ovbiagele B, Kidwell CS, Saver JL. Epidemiological impact in the United States of a tissue-based definition of transient ischemic attack. *Stroke*. 2003;34:919–924.
10. Hill MD, Yiannakoulias N, Jeerakathil T, Tu JV, Svenson LW, Schopflocher DP. The high risk of stroke immediately after transient ischemic attack: a population-based study. *Neurology*. 2004;62:2015–2020.
11. Kleindorfer D, Panagos P, Pancioli A, Khoury J, Kissela B, Woo D, Schneider A, Alwell K, Jauch E, Miller R, Moomaw C, Shukla R, Broderick JP. Incidence and short-term prognosis of transient ischemic attack in a population-based study. *Stroke*. 2005;36:720–723.
12. Johnston SC, Fayad PB, Gorelick PB, Hanley DF, Shwayder P, van Husen D, Weiskopf T. Prevalence and knowledge of transient ischemic attack among US adults. *Neurology*. 2003;60:1429–1434.
13. Lisabeth LD, Ireland JK, Risser JM, Brown DL, Smith MA, Garcia NM, Morgenstern LB. Stroke risk after transient ischemic attack in a population-based setting. *Stroke*. 2004;35:1842–1846.
14. Coull AJ, Lovett JK, Rothwell PM, for the Oxford Vascular Study. Population based study of early risk of stroke after transient ischemic attack or minor stroke: implications for public education and organization of services. *BMJ*. 2004;328:326.
15. Sherman DG. Reconsideration of TIA diagnostic criteria. *Neurology*. 2004;62(suppl 6):S20–S21.
16. Clark TG, Murphy MF, Rothwell PM. Long term risks of stroke, myocardial infarction, and vascular death in "low-risk" patients with a non-recent transient ischaemic attack. *J Neurol Neurosurg Psychiatry*. 2003;74:577–580.
17. Eliasziw M, Kennedy J, Hill MD, Buchan AM, Barnett HJ, for the North American Symptomatic Carotid Endarterectomy Trial Group. Early risk of stroke after a transient ischemic attack in patients with internal carotid artery disease. *CMAJ*. 2004;170:1105–1109.
18. *Incidence and Prevalence: 2006 Chart Book on Cardiovascular and Lung Diseases*. Bethesda, Md: National Heart, Lung, and Blood Institute; 2006.
19. Kleindorfer D, Broderick J, Khoury J, Flaherty M, Woo D, Alwell K, Moomaw CJ, Schneider A, Miller R, Shukla R, Kissela B. The unchanging incidence and case-fatality of stroke in the 1990s: a population-based study. *Stroke*. 2006;37:2473–2478.
20. Morgenstern LB, Smith MA, Lisabeth LD, Risser JM, Uchino K, Garcia N, Longwell PJ, McFarling DA, Akuwumi O, Al-Wabil A, Al-Senani F, Brown DL, Moye LA. Excess stroke in Mexican Americans compared with non-Hispanic whites: the Brain Attack Surveillance in Corpus Christi Project. *Am J Epidemiol*. 2004;160:376–383.
21. White H, Boden-Albala B, Wang C, Elkind MS, Rundek T, Wright CB, Sacco RL. Ischemic stroke subtype incidence among whites, blacks, and Hispanics: the Northern Manhattan Study. *Circulation*. 2005;111:1327–1331.
22. Carandang R, Seshadri S, Beiser A, Kelly-Hayes M, Kase CS, Kannel WB, Wolf PA. Trends in incidence, lifetime risk, severity, and 30-day mortality of stroke over the past 50 years. *JAMA*. 2006;296:2939–2946.
23. Rich DQ, Gaziano JM, Kurth T. Geographic patterns in overall and specific cardiovascular disease incidence in apparently healthy men in the United States. *Stroke*. 2007;38:2221–2227.
24. National Center for Health Statistics. Vital statistics of the United States, data warehouse. Available at: http://www.cdc.gov/nchs/data/dvs/MortFinal2003_WorkTable307.pdf. Accessed Spring/Summer 2008.
25. Heron MP, Hoyert DL, Xu J, Scott C, Tejada-Vera B. *Preliminary Data for 2006*. Hyattsville, Md: National Center for Health Statistics; 2008. National Vital Statistics Reports, Vol. 56, No. 16.
26. Rosamond WD, Folsom AR, Chambless LE, Wang CH, McGovern PG, Howard G, Copper LS, Shahar E. Stroke incidence and survival among middle-aged adults: 9-year follow-up of the Atherosclerotic Risk in Communities (ARIC) cohort. *Stroke*. 1999;30:736–743.
27. El-Saed A, Kuller LH, Newman AB, Lopez O, Costantino J, McTigue K, Cushman M, Kronmal R. Geographic variations in stroke incidence and mortality among older populations in four US communities. *Stroke*. 2006;37:1975–1979.
28. National Center for Health Statistics, Centers for Disease Control and Prevention. *Compressed Mortality File: Underlying Cause of Death, 1979 to 2005*. Atlanta, Ga: Centers for Disease Control and Prevention; 2008. Available at: http://wonder.cdc.gov/mortSQL.html. Accessed June 2008.
29. National Center for Health Statistics. *Health, United States, 2007*. Hyattsville Md; National Center for Health Statistics; 2007. Available at: http://www.cdc.gov/nchs/hus.htm. Accessed June 24,2008.
30. Ayala C, Greenlund KJ, Croft JB, Keenan NL, Donehoo RS, Giles WH, Kittner SJ, Marks JS. Racial/ethnic disparities in mortality by stroke subtype in the United States, 1995–1998. *Am J Epidemiol*. 2001;154:1057–1063.
31. Centers for Disease Control and Prevention. Disparities in deaths from stroke among persons aged <75 years: United States, 2002. *MMWR Morb Mortal Wkly Rep*. 2005;54:477–481.
32. Luepker RV, Arnett DK, Jacobs DR Jr, Duval SJ, Folsom AR, Armstrong C, Blackburn H. Trends in blood pressure, hypertension control, and stroke mortality: the Minnesota Heart Survey. *Am J Med*. 2006;119:42–49.
33. Casper ML, Nwaise IA, Croft JB, Nilasena DS. *Atlas of Stroke Hospitalizations Among Medicare Beneficiaries*. Atlanta, Ga: US Department of Health and Human Services, Centers for Disease Control and Prevention; 2008.
34. Johnston SC, Gress DR, Browner WS, Sidney S. Short-term prognosis after emergency department diagnosis of TIA. *JAMA*. 2000;284:2901–2906.
35. Wolf PA, Abbott RD, Kannel WB. Atrial fibrillation as an independent risk factor for stroke: the Framingham Study. *Stroke*. 1991;22:983–988.
36. Wang TJ, Massaro JM, Levy D, Vasan RS, Wolf PA, D'Agostino RB, Larson MG, Kannel WB, Benjamin EJ. A risk score for predicting stroke or death in individuals with new-onset atrial fibrillation in the community: the Framingham Heart Study. *JAMA*. 2003;290:1049–1056.
37. Kissela BM, Khoury J, Kleindorfer D, Woo D, Schneider A, Alwell K, Miller R, Ewing I, Moomaw CJ, Szaflarski JP, Gebel J, Shukla R, Broderick JP. Epidemiology of ischemic stroke in patients with diabetes: the greater Cincinnati/Northern Kentucky Stroke Study. *Diabetes Care*. 2005;28:355–359.
38. Ohira T, Shahar E, Chambless LE, Rosamond WD, Mosley TH Jr, Folsom AR. Risk factors for ischemic stroke subtypes: the Atherosclerosis Risk in Communities study. *Stroke*. 2006;37:2493–2498.
39. Das RR, Seshadri S, Beiser AS, Kelly-Hayes M, Au R, Himali JJ, Kase CS, Benjamin EJ, Polak JF, O'Donnell CJ, Yoshita M, D'Agostino RB Sr, Decarli C, Wolf PA. Prevalence and correlates of silent cerebral infarcts in the Framingham Offspring Study. *Stroke*. 2008;39:2929–2935.
40. Salaycik KJ, Kelley-Hayes M, Beiser A, Nguyen AH, Brady SM, Kase CS, Wolf PA. Depressive symptoms and risk of stroke: the Framingham Study. *Stroke*. 2007;38:16–21.
41. Curb JD, Abbott RD, Rodriguez BL, Masaki KH, Chen R, Popper JS, Petrovitch H, Ross GW, Schatz IJ, Belleau GC, Yano K. High density lipoprotein cholesterol and the risk of stroke in elderly men: the Honolulu Heart Program. *Am J Epidemiol*. 2004;160:150–157.
42. Towfighi A, Saver JL, Engelhardt R, Ovbiagele B. A midlife surge among women in the United States. *Neurology*. 2007;69:1898–1904.
43. Bousser MG. Stroke in women: the 1997 Paul Dudley White International Lecture. *Circulation*. 1999;99:463–467.
44. Wassertheil-Smoller S, Hendrix SL, Limacher M, Heiss G, Kooperberg C, Baird A, Kotchen T, Curb JD, Black H, Rossouw JE, Aragaki A, Safford M, Stein E, Laowattana S, Mysiw WJ, for the WHI Investigators. Effect of estrogen plus progestin on stroke in postmenopausal

women: the Women's Health Initiative: a randomized trial. *JAMA*. 2003;289:2673–2684.
45. Hendrix SL, Wassertheil-Smoller S, Johnson KC, Howard BV, Kooperberg C, Rossouw JE, Trevisan M, Aragaki A, Baird AE, Bray PF, Buring JE, Criqui MH, Herrington D, Lynch JK, Rapp SR, Torner J, for the WHI Investigators. Effects of conjugated equine estrogen on stroke in the Women's Health Initiative. *Circulation*. 2006;113: 2425–2434.
46. Simon JA, Hsia J, Cauley JA, Richards C, Harris F, Fong J, Barrett-Connor E, Hulley SB. Postmenopausal hormone therapy and risk of stroke: the Heart and Estrogen-Progestin Replacement Study (HERS). *Circulation.* 2001;103:638–642.
47. Viscoli CM, Brass LM, Kernan WN, Sarrel PM, Suissa S, Horwitz RI. A clinical trial of estrogen-replacement therapy after ischemic stroke. *N Engl J Med*. 2001;345:1243–1249.
48. Rossouw JE, Anderson GL, Prentice RL, LaCroix AZ, Kooperberg C, Stefanick ML, Jackson RD, Beresford SA, Howard BV, Johnson KC, Kotchen JM, Ockene J, for the Writing Group for the Women's Health Initiative Investigators. Risks and benefits of estrogen plus progestin in healthy postmenopausal women: principal results from the Women's Health Initiative randomized controlled trial. *JAMA*. 2002;288:321–333.
49. Kurth T, Moore SC, Gaziano JM, Kase CS, Stampfer MJ, Berger K, Buring JE. Healthy lifestyle and the risk of stroke in women. *Arch Intern Med*. 2006;166:1403–1409.
50. Kittner SJ, Stern BJ, Feeser BR, Hebel R, Nagey DA, Buchholz DW, Earley CJ, Johnson CJ, Macko RF, Sloan MA, Wityk RJ, Wozniak MA. Pregnancy and the risk of stroke. *N Engl J Med*. 1996;335:768–774.
51. Salonen Ros H, Lichtenstein P, Bellocco R, Petersson G, Cnattingius S. Increased risks of circulatory diseases in late pregnancy and puerperium. *Epidemiology*. 2001;12:456–460.
52. James AH, Bushnell CD, Jamison MG, Myers ER. Incidence and risk factors for stroke in pregnancy and the puerperium. *Obstet Gynecol*. 2005;106:509–516.
53. Lee IM, Hennekens CH, Berger K, Buring JE, Manson JE. Exercise and risk of stroke in male physicians. *Stroke*. 1999;30:1–6.
54. Lee IM, Paffenbarger RS Jr. Physical activity and stroke incidence: the Harvard Alumni Health Study. *Stroke.* 1998;29:2049–2054.
55. Myint PK, Luben RN, Wareham NJ, Welch AA, Bingham SA, Day NE, Khaw K-T. Combined work and leisure physical activity and risk of stroke in men and women in the European Prospective Investigation Into Cancer–Norfolk Prospective Population Study. *Neuroepidemiology*. 2006;27:122–129.
56. Hu FB, Stampfer MJ, Colditz GA, Ascherio A, Rexrode KM, Willett WC, Manson JE. Physical activity and risk of stroke in women. *JAMA*. 2000;283:2961–2967.
57. Sacco RL, Gan R, Boden-Albala B, Lin IF, Kargman DE, Hauser WA, Shea S, Paik MC. Leisure-time physical activity and ischemic stroke risk: the Northern Manhattan Stroke Study. *Stroke*. 1998;29:380–387.
58. Evenson KR, Rosamond WD, Cai J, Toole JF, Hutchinson RG, Shahar E, Folsom AR. Physical activity and ischemic stroke risk: the Atherosclerosis Risk in Communities study. *Stroke*. 1999;30:1333–1339.
59. Noda H, Iso H, Toyoshima H, Date C, Yamamoto A, Kikuchi S, Koizumi A, Kondo T, Watanabe Y, Wada Y, Inaba Y, Tamakoshi A, for the JACC Study Group. Walking and sports participation and mortality from coronary heart disease and stroke. *J Am Coll Cardiol*. 2005;46: 1761–1767.
60. Hu G, Sarti C, Jousilahti P, Silventoinen K, Barengo NC, Tuomilehto J. Leisure time, occupational, and commuting physical activity and the risk of stroke. *Stroke*. 2005;36:1994–1999.
61. Wendel-Vos GC, Schuit AJ, Feskens EJ, Boshuizen HC, Verschuren WM, Saris WH, Kromhout D. Physical activity and stroke: a meta-analysis of observational data. *Int J Epidemiol*. 2004;33:787–798.
62. Centers for Disease Control and Prevention (CDC). Awareness of stroke warning symptoms: 13 states and the District of Columbia, 2005. *MMWR Morb Mortal Wkly Rep*. 2008;57:481–485.
63. Kothari R, Sauerbeck L, Jauch E, Broderick J, Brott T, Khoury J, Liu T. Patient's awareness of stroke signs, symptoms, and risk factors. *Stroke*. 1997;28:1871–1875.
64. Schneider AT, Pancioli AM, Khoury JC, Rademacher E, Tuchfarber A, Miller R, Woo D, Kissela B, Broderick JP. Trends in community knowledge of the warning signs and risk factors for stroke. *JAMA*. 2003;289:343–346.
65. Pancioli AM, Broderick J, Kothari R, Brott T, Tuchfarber A, Miller R, Khoury J, Jauch E. Public perception of stroke warning signs and knowledge of potential risk factors. *JAMA*. 1998;279:1288–1292.
66. Samsa GP, Cohen SJ, Goldstein LB, Bonito AJ, Duncan PW, Enarson C, DeFriese GH, Horner RD, Matchar DB. Knowledge of risk among patients at increased risk for stroke. *Stroke*. 1997;28:916–921.
67. Ferris A, Robertson RM, Fabunmi R, Mosca L, for the American Heart Association, American Stroke Association. American Heart Association and American Stroke Association national survey of stroke risk awareness among women. *Circulation*. 2005;111:1321–1326.
68. Robinson KA, Merrill RM. Relation among stroke knowledge, lifestyle, and stroke-related screening results. *Geriatr Nurs*. 2003;24:300–305.
69. Maron BA, Dansereau LM, Maron BJ, Easton JD. Impact of post-graduate medical education on recognition of stroke. *Cardiol Rev*. 2005; 13:73–75.
70. Harwell TS, Blades LL, Oser CS, Dietrich DW, Okon NJ, Rodriguez DV, Burnett AM, Russell JA, Allen MJ, Fogle CC, Helgerson SD, Gohdes D. Perceived risk for developing stroke among older adults. *Prev Med*. 2005;41:791–794.
71. Blades LL, Oser CS, Dietrich DW, Okon NJ, Rodriguez DV, Burnett AM, Russell JA, Allen MJ, Fogle CC, Helgerson SD, Gohdes D, Harwell TS. Rural community knowledge of stroke warning signs and risk factors. *Prev Chronic Dis*. 2005;2:A14.
72. Zerwic J, Hwang SY, Tucco L. Interpretation of symptoms and delay in seeking treatment by patients who have had a stroke: exploratory study. *Heart Lung*. 2007;36:25–34.
73. DuBard CA, Garrett J, Gizlice Z. Effect of language on heart attack and stroke awareness among U.S. Hispanics. *Am J Prev Med*. 2006;30: 189–196.
74. Howard G, Prineas R, Moy C, Cushman M, Kellum M, Temple E, Graham A, Howard V. Racial and geographic differences in awareness, treatment, and control of hypertension: the Reasons for Geographic and Racial Differences in Stroke Study. *Stroke*. 2006;37:1171–1178.
75. Centers for Disease Control and Prevention (CDC). Prevalence of disabilities and associated health conditions among adults: United States, 1999. *MMWR Morb Mortal Wkly Rep*. 2001;50:120–125.
76. Centers for Disease Control and Prevention. Outpatient rehabilitation among stroke survivors: 21 states and the District of Columbia, 2005. *MMWR Morb Mortal Wkly Rep*. 2007;56:504–507.
77. Asplund K, Stegmayr B, Peltonen M. From the twentieth to the twenty-first century: a public health perspective on stroke. In: Ginsberg MD, Bogousslavsky J, eds. *Cerebrovascular Disease Pathophysiology, Diagnosis, and Management*. Malden, Mass: Blackwell Science; 1998; 2:chap 64.
78. Kelley-Hayes M, Beiser A, Kase CS, Scaramucci A, D'Agostino RB, Wolf PA. The influence of gender and age on disability following ischemic stroke: the Framingham study. *J Stroke Cerebrovasc Dis*. 2003;12:119–126.
79. Centers for Disease Control and Prevention (CDC). Differences in disability among black and white stroke survivors: United States, 2000–2001. *MMWR Morb Mortal Wkly Rep*. 2005;54:3–6.
80. Roquer J, Campello AR, Gomis M. Sex differences in first-ever acute stroke. *Stroke*. 2003;34:1581–1585.
81. Gargano JW, Reeves MJ, for the Paul Coverdell National Acute Stroke Registry Michigan Prototype Investigators. Sex differences in stroke recovery and stroke-specific quality of life: results from a statewide stroke registry. *Stroke*. 2007;38:2541–2548.
82. Lai SM, Duncan PW, Dew P, Keighley J. Sex differences in stroke recovery. *Prev Chronic Dis*. 2005;2:A13.
83. Kelly-Hayes M, Beiser A, Kase CS, Scaramucci A, D'Agostino RB, Wolf PA. The influence of gender and age on disability following ischemic stroke: the Framingham study. *J Stroke Cerebrovasc Dis*. 2003;12:119–126.
84. Fang J, Alderman MH, Keenan NL, Croft JB. Declining US stroke hospitalization since 1997: National Hospital discharge Survey, 1988–2004. *Neuroepidemiology*. 2007;29:243–249.
85. Russo CA, Andrews RM. *Hospital Stays for Stroke and Other Cerebrovascular Diseases, 2005*. Rockville, Md: Agency for Healthcare Research and Quality; May 2008. HCUP Statistical Brief No. 51. Available at: http://www.hcup-us.ahrq.gov/reports/statbriefs/sb51.pdf. Accessed September 15, 2008.
86. DeFrances CJ, Lucas CA, Buie VC, Golosinskiy A. *2006 National Hospital Discharge Survey*. Hyattsville, Md: National Center for Health Statistics; 2008. National Health Statistics Reports, No. 5.
87. Elixhauser A, Jiang HJ. *Hospitalizations for Women With Circulatory Disease, 2003*. Rockville, Md: Agency for Healthcare Research and Quality; May 2006. HCUP Statistical Brief No. 5.

88. Lynch JK, Hirtz DG, DeVeber G, Nelson KB. Report of the National Institute of Neurological Disorders and Stroke workshop on perinatal and childhood stroke. *Pediatrics*. 2002;109:116–123.
89. Lee J, Croen LA, Backstrand KH, Yoshida CK, Henning LH, Lindan C, Ferriero DM, Fullerton HJ, Barkovich AJ, Wu YW. Maternal and infant characteristics associated with perinatal arterial stroke in the infant. *JAMA*. 2005;293:723–729.
90. Kleindorfer D, Khoury J, Kissela B, Alwell K, Woo D, Miller R, Schneider A, Moomaw C, Broderick JP. Temporal trends in the incidence and case fatality of stroke in children and adolescents. *J Child Neurol*. 2006;21:415–418.
91. deVeber GA, MacGregor D, Curtis R, Mayank S. Neurologic outcome in survivors of childhood arterial ischemic stroke and sinovenous thrombosis. *J Child Neurol*. 2000;15:316–324.
92. Fullerton HJ, Wu YW, Zhao S, Johnston SC. Risk of stroke in children: ethnic and gender disparities. *Neurology*. 2003;61:189–194.
93. Strater R, Becker S, von Eckardstein A, Heinecke A, Gutsche S, Junker R, Kurnik K, Schobess R, Nowak-Gottl U. Prospective assessment of risk factors for recurrent stroke during childhood: a 5-year follow-up study. *Lancet*. 2002;360:1540–1545.
94. Brown DL, Boden-Albala B, Langa KM, Lisabeth LD, Fair M, Smith MA, Sacco RL, Morgenstern LB. Projected costs of ischemic stroke in the United States. *Neurology*. 2006;67:1390–1395.
95. Fullerton HJ, Chetkovich DM, Wu YW, Smith WS, Johnston SC. Deaths from stroke in US children, 1979 to 1998. *Neurology*. 2002;59:34–39.
96. Fullerton HJ, Adams RJ, Zhao S, Johnston SC. Declining stroke rates in Californian children with sickle cell disease. *Blood*. 2004;104:336–339.
97. American Board of Psychiatry and Neurology. www.abpn.com. Accessed September 15, 2008.
98. American Academy of Neurology. www.aan.com. Accessed September 15, 2008.
99. Centers for Disease Control and Prevention. First-ever county level report on stroke hospitalizations. CDC Press Release. March 28, 2008. Available at: http://www.cdc.gov/od/oc/media/pressrel/2008/r080328.htm. Accessed April 3, 2008.
100. Evenson KR, Rosamond WD, Morris DL. Prehospital and in-hospital delays in acute stroke care. *Neuroepidemiology*. 2001;20:65–76.
101. California Acute Stroke Pilot Registry (CASPR) Investigators. Prioritizing interventions to improve rates of thrombolysis for ischemic stroke. *Neurology*. 2005;64:654–659.
102. Centers for Disease Control and Prevention (CDC). Prehospital and hospital delays after stroke onset: United States, 2005–2006. *MMWR Morb Mortal Wkly Rep*. 2007;56:474–478.
103. California Acute Stroke Pilot Registry Investigators. The impact of standardized stroke orders on adherence to best practices. *Neurology*. 2005;65:360–365.
104. Kleindorfer D, Kissela B, Schneider A, Woo D, Khoury J, Miller R, Alwell K, Gebel J, Szaflarski J, Pancioli A, Jauch E, Moomaw C, Shukla R, Broderick JP, for the Neuroscience Institute. Eligibility for recombinant tissue plasminogen activator in acute ischemic stroke: a population-based study. *Stroke*. 2004;35:e27–e29.
105. Centers for Medicare & Medicaid Services. *Health Care Financing Review: Medicare & Medicaid Statistical Supplement*. Table 5.5: Discharges, Total Days of Care, and Program Payments for Medicare Beneficiaries Discharged from Short-Stay Hospitals, by Principal Diagnoses Within Major Diagnostic Classifications (MDCs): Calendar Year 2006. Baltimore, Md: Centers for Medicare and Medicaid Services; 2005. Available at: http://www.cms.hhs.gov/MedicareMedicaidStatSupp/. Accessed August 28, 2008.
106. Taylor TN, Davis PH, Torner JC, Holmes J, Meyer JW, Jacobson MF. Lifetime cost of stroke in the United States. *Stroke*. 1996;27:1459–1466.
107. Leibson CL, Hu T, Brown RD, Hass SL, O'Fallon WM, Whisnant JP. Utilization of acute care services in the year before and after first stroke: a population-based study. *Neurology*. 1996;46:861–869.
108. Diringer MN, Edwards DF, Mattson DT, Akins PT, Sheedy CW, Hsu CY, Dromerick AW. Predictors of acute hospital costs for treatment of ischemic stroke in an academic center. *Stroke*. 1999;30:724–728.
109. Metz R. Cost-effective, risk-free, evidence-based medicine. *Arch Intern Med*. 2003;163:2795.
110. Kissela B, Schneider A, Kleindorfer D, Khoury J, Miller R, Alwell K, Woo D, Szaflarski J, Gebel J, Moomaw C, Pancioli A, Jauch E, Shukla R, Broderick J. Stroke in a biracial population: the excess burden of stroke among blacks. *Stroke*. 2004;35:426–431.
111. Howard G, Wagenknecht LE, Burke GL, Diez-Roux A, Evans GW, McGovern P, Nieto FJ, Tell GS. Cigarette smoking and progression of atherosclerosis: the Atherosclerosis Risk in Communities (ARIC) Study. *JAMA*. 1998;279:119–124.
112. Whisnant JP, Wiebers DO, O'Fallon WM, Sicks JD, Frye RL. A population-based model of risk factors for ischemic stroke: Rochester, Minnesota. *Neurology*. 1996;47:1420–1428.
113. Bonow RO, Carabello B, de Leon AC Jr, Edmunds LH Jr, Fedderly BJ, Freed MD, Gaasch WH, McKay CR, Nishimura RA, O'Gara PT, O'Rourke RA, Rahimtoola SH, Ritchie JL, Cheitlin MD, Eagle KA, Gardner TJ, Garson A Jr, Gibbons RJ, Russell RO, Ryan TJ, Smith SC Jr. ACC/AHA guidelines for the management of patients with valvular heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Management of Patients With Valvular Heart Disease). *J Am Coll Cardiol*. 1998;32:1486–1588.
114. Go AS, Hylek EM, Phillips KA, Chang Y, Henault LE, Selby JV, Singer DE. Prevalence of diagnosed atrial fibrillation in adults: national implications for rhythm management and stroke prevention: the Anticoagulation and Risk Factors in Atrial Fibrillation (ATRIA) Study. *JAMA*. 2001;285:2370–2375.
115. O'Leary DH, Polak JF, Kronmal RA, Kittner SJ, Bond MG, Wolfson SK Jr, Bommer W, Price TR, Gardin JM, Savage PJ. Distribution and correlates of sonographically detected carotid artery disease in the Cardiovascular Health Study: the CHS Collaborative Research Group. *Stroke*. 1992;23:1752–1760.
116. Fine-Edelstein JS, Wolf PA, O'Leary DH, Poehlman H, Belanger AJ, Kase CS, D'Agostino RB. Precursors of extracranial carotid atherosclerosis in the Framingham Study. *Neurology*. 1994;44:1046–1050.
117. Yin D, Carpenter JP. Cost-effectiveness of screening for asymptomatic carotid stenosis. *J Vasc Surg*. 1998;27:245–255.
118. Colgan MP, Strode GR, Sommer JD, Gibbs JL, Sumner DS. Prevalence of asymptomatic carotid disease: results of duplex scanning in 348 unselected volunteers. *J Vasc Surg*. 1988;8:674–678.
119. Prati P, Vanuzzo D, Casaroli M, Di Chiara A, De Biasi F, Feruglio GA, Touboul PJ. Prevalence and determinants of carotid atherosclerosis in a general population. *Stroke*. 1992;23:1705–1711.
120. Pujia A, Rubba P, Spencer MP. Prevalence of extracranial carotid artery disease detectable by echo-Doppler in an elderly population. *Stroke*. 1992;23:818–822.
121. Ramsey DE, Miles RD, Lambeth A, Sumner DS. Prevalence of extracranial carotid artery disease: a survey of an asymptomatic population with noninvasive techniques. *J Vasc Surg*. 1987;5:584–588.
122. Ahmed A, Adams RJ. Sickle cell disorders and cerebrovascular disease. In: Gillum RF, Gorelick PB, Cooper ES, eds. *Stroke in Blacks: A Guide to Management and Prevention*. Basel, Switzerland: Karger; 1999:62–69.
123. National Institutes of Health. *Adult Treatment Panel III: Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults*. Bethesda, Md: National Institutes of Health; 2002.
124. Institute of Medicine. *Dietary Reference Intakes: Water, Potassium, Sodium, Chloride, and Sulfate*. Washington, DC: National Academies Press; 2004.
125. Boushey CJ, Beresford SA, Omenn GS, Motulsky AG. A quantitative assessment of plasma homocysteine as a risk factor for vascular disease: probable benefits of increasing folic acid intakes. *JAMA*. 1995;274:1049–1057.
126. Majumdar SR, Almasi EA, Stafford RS. Promotion and prescribing of hormone therapy after report of harm by the Women's Health Initiative. *JAMA*. 2004;292:1983–1988.
127. Haas JS, Kaplan CP, Gerstenberger EP, Kerlikowske K. Changes in the use of postmenopausal hormone therapy after the publication of clinical trial results. *Ann Intern Med*. 2004;140:184–188.
128. D'Agostino RB, Wolf PA, Belanger AJ, Kannel WB. Stroke risk profile: adjustment for antihypertensive medication: the Framingham Study. *Stroke*. 1994;25:40–43.
129. Wilterdink JL, Easton JD. Vascular event rates in patients with atherosclerotic cerebrovascular disease. *Arch Neurol*. 1992;49:857–863.
130. Adams RJ, McKie VC, Hsu L, Files B, Vichinsky E, Pegelow C, Abboud M, Gallagher D, Kutlar A, Nichols FT, Bonds DR, Brambilla D. Prevention of a first stroke by transfusions in children with sickle cell anemia and abnormal results on transcranial Doppler ultrasonography. *N Engl J Med*. 1998;339:5–11.

131. Welin L, Svardsudd K, Wilhelmsen L, Larsson B, Tibblin G. Analysis of risk factors for stroke in a cohort of men born in 1913. *N Engl J Med*. 1987;317:521–526.
132. Strauss RS, Pollack HA. Epidemic increase in childhood overweight, 1986–1998. *JAMA*. 2001;286:2845–2848.
133. Rossouw JE, Anderson GL, Prentice RL, LaCroix AZ, Kooperberg C, Stefanick ML, Jackson RD, Beresford SA, Howard BV, Johnson KC, Kotchen JM, Ockene J, for the Writing Group for the Women's Health Initiative Investigators. Risks and benefits of estrogen plus progestin in healthy postmenopausal women: principal results from the Women's Health Initiative randomized controlled trial. *JAMA*. 2002;288:321–333.
134. Hart RG, Benavente O, McBride R, Pearce LA. Antithrombotic therapy to prevent stroke in patients with atrial fibrillation: a meta-analysis. *Ann Intern Med*. 1999;131:492–501.
135. Hart RG. Intensity of anticoagulation to prevent stroke in patients with atrial fibrillation. *Ann Intern Med*. 1998;128:408.
136. van Walraven C, Hart RG, Singer DE, Laupacis A, Connolly S, Petersen P, Koudstaal PJ, Chang Y, Hellemons B. Oral anticoagulants vs aspirin in nonvalvular atrial fibrillation: an individual patient meta-analysis. *JAMA*. 2002;288:2441–2448.
137. Wolf PA, D'Agostino RB, Belanger AJ, Kannel WB. Probability of stroke: a risk profile from the Framingham Study. *Stroke*. 1991;22:312–318.

Table 5-1. Stroke

Population Group	Prevalence, 2006 Age ≥20 y	New and Recurrent Attacks All Ages	Mortality, 2005 All Ages*	Hospital Discharges, 2006 All Ages	Cost, 2009
Both sexes	6 500 000 (2.9%)	795 000	143 579	889 000	$68.9 billion
Males	2 600 000 (2.6%)	370 000 (46.5%)†	56 586 (39.4%)†	404 000	. . .
Females	3 900 000 (3.2%)	425 000 (53.5%)†	86 993 (60.6%)†	486 000	. . .
NH white males	2.3%	325 000‡	47 194	. . .	. . .
NH white females	3.2%	365 000‡	74 674	. . .	. . .
NH black males	3.9%	45 000‡	7519	. . .	. . .
NH black females	4.1%	60 000‡	10 022	. . .	. . .
Mexican-American males	2.1%	. . .	. . .	. . .	. . .
Mexican-American females	3.8%	. . .	. . .	. . .	. . .
Hispanic or Latino age ≥18 y§	2.5%	. . .	. . .	. . .	. . .
Asian age ≥18 y§	2.6%	. . .	. . .	. . .	. . .
American Indian/Alaska Native age ≥18 y§	‖	. . .	. . .	. . .	. . .

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 2007 (NCHS): data are weighted percentages for Americans ≥18 years of age.[3]

‖Estimates are considered unreliable; figure is suppressed.

Sources: Prevalence (total, males, females, whites, blacks, Mexican Americans) is based on NHLBI computations of NHANES 2005 to 2006, NCHS (≥20 years of age). Age-adjusted rates are extrapolated to the US population ≥20 years of age, 2006. Prevalence data for the Hispanic, Asian, and American Indian/Alaska Native populations, ≥18 years of age, are from NHIS/NCHS.[1] Incidence: GCNKSS/NINDS data for 1999 provided on August 1, 2007. US estimates compiled by NHLBI. See also Kissela et al.[110] 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 2009.

Table 5-2. Modifiable Stroke Risk Factors

Factor	Prevalence, %	Population-Attributable Risk, %*	RR
CVD			
CHD[111]			
Men	8.4	5.8†	1.73 (1.68–1.78)[128]
Women	5.6	3.9†	1.55 (1.17–2.07)[128]
Heart failure[111]			
Men	2.6	1.4†	
Women	2.1	1.1†	
Peripheral arterial disease	4.9	3.0†	
Hypertension[112]			
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[115-121]	2–7‡	2.0[129]
Atrial fibrillation (nonvalvular)[113,114]			
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)[122]	. . .	200–400[130]§
Dyslipidemia			
High total cholesterol	25[123]	15	2.0 for men and for women <55 y of age
Low HDL cholesterol	25[123]	10	1.5–2.5 for men
Dietary factors			
Na intake >2300 mg	75–90	Unknown	Unknown
K intake <4700 mg	90–99[124]		Unknown
Obesity	17.9[125]	12–20†	1.75–2.37[131,132]
Physical inactivity[57]	25	30	2.7‡
Postmenopausal hormone therapy	20[126] (women 50–74 y of age)[127]	7	1.4[133]

Data derived from Hart et al[134,135] and van Walraven et al.[136] 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 JV, Sacco RL, for the American Heart Association/American Stroke Association Stroke Council; Atherosclerotic Peripheral Vascular Disease Interdisciplinary Working Group; Cardiovascular Nursing Council; Clinical Cardiology Council; Nutrition, Physical Activity, and Metabolism Council; Quality of Care and Outcomes Research Interdisciplinary Working Group; and American Academy of Neurology. 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: the American Academy of Neurology affirms the value of this guideline. *Stroke.* 2006;37:1583-1633.

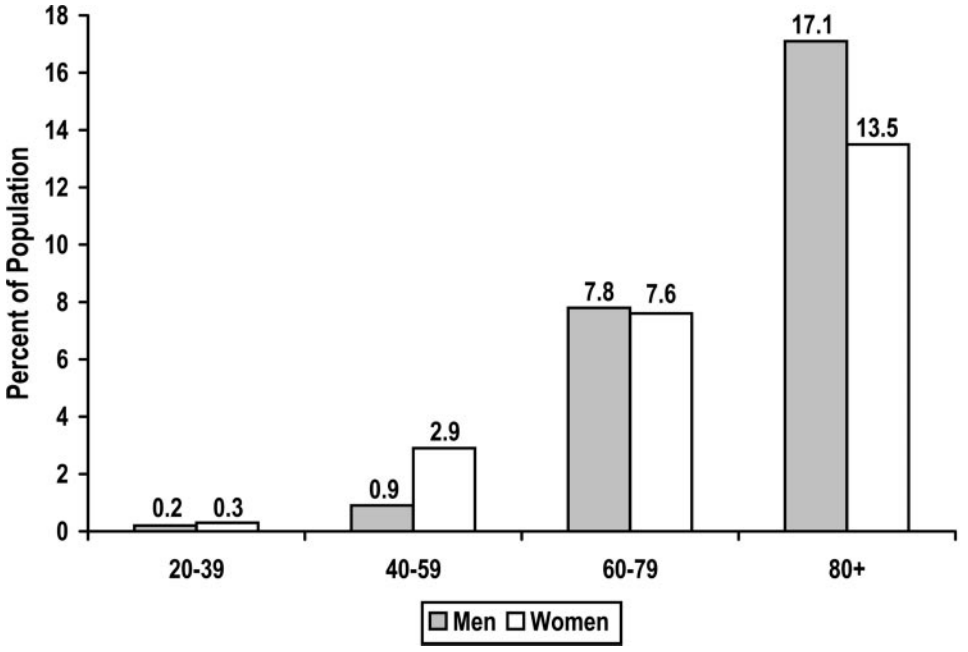


Chart 5-1. Prevalence of stroke by age and sex (NHANES: 2005 to 2006). Source: NCHS and NHLBI.

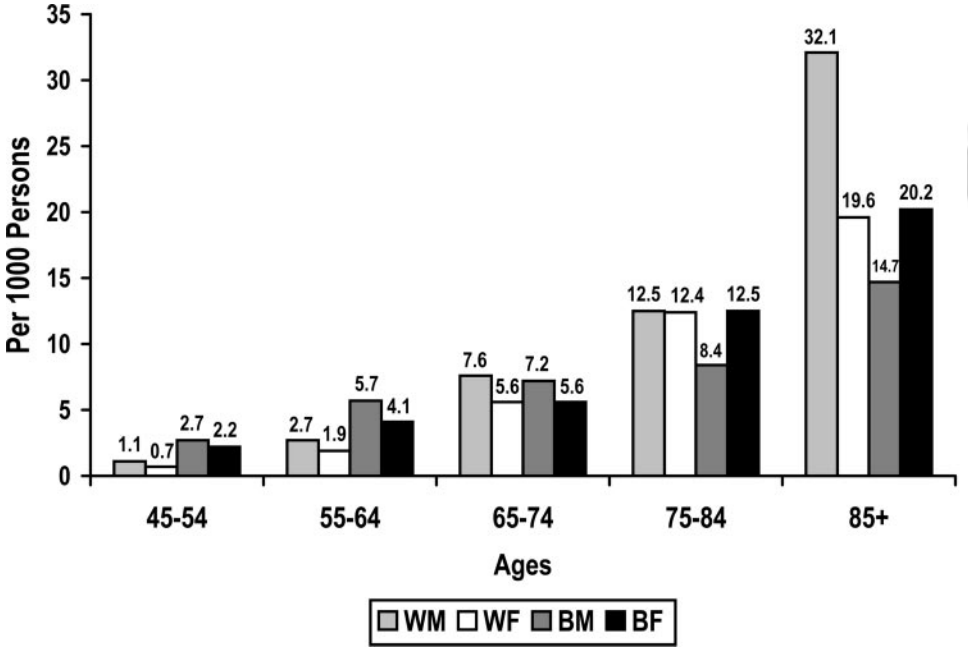


Chart 5-2. Annual rate of first cerebral infarction by age, sex, and race (GCNKSS: 1999). Source: Unpublished data from the GCNKSS. Note: Rates for black men and women 45 to 54 years of age and for black men ≥75 years of age are considered unreliable. An estimated 15 000 people have first cerebral infarctions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

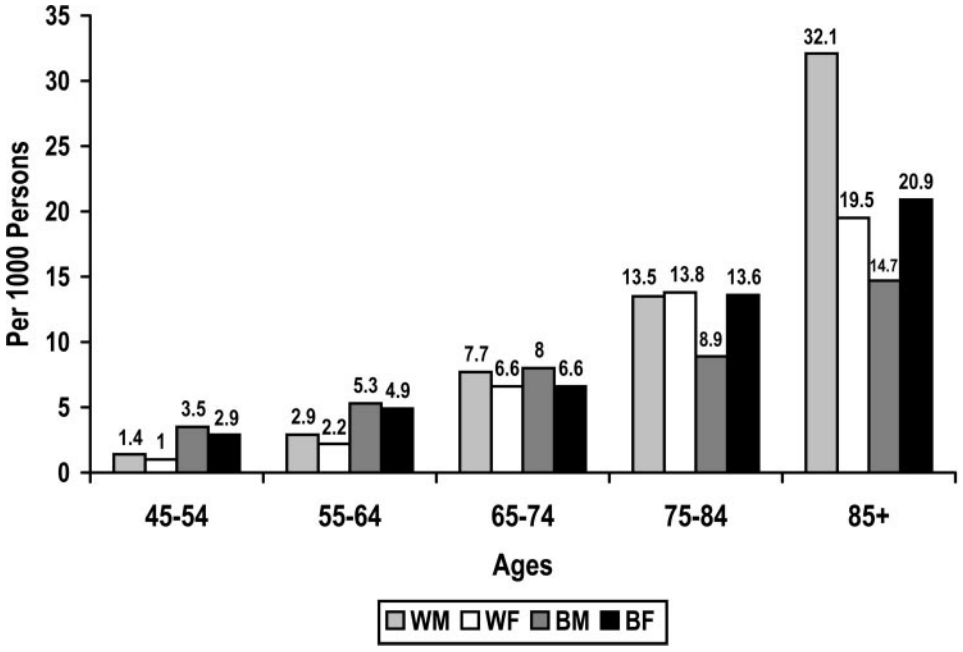
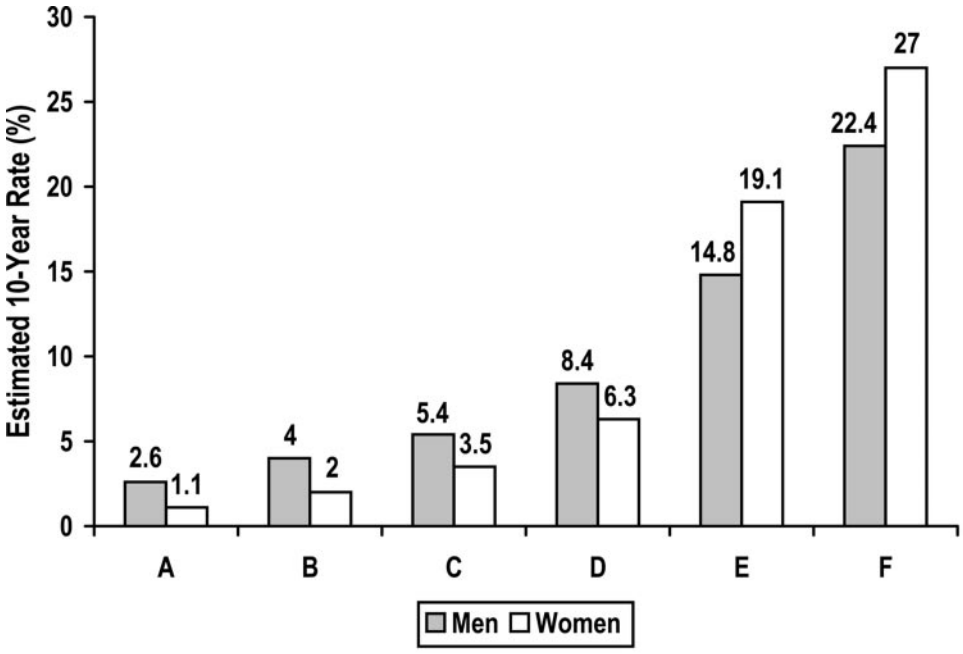


Chart 5-3. Annual rate of first-ever strokes by age, sex, and race (GCNKSS: 1999). Note: Rates for black men and women 45 to 54 years of age and for black men ≥75 years of age are considered unreliable. Source: Unpublished data from the GCNKSS.



	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 55-year-old adults according to levels of various risk factors (Framingham Heart Study). Source: Wolf et al.[137]

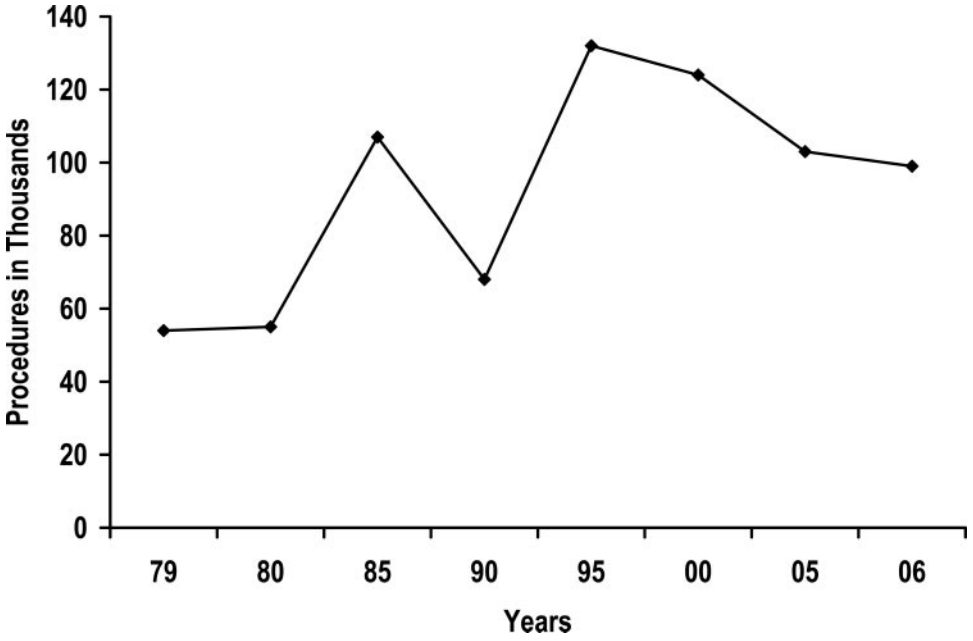


Chart 5-5. Trends in carotid endarterectomy procedures (United States: 1979 to 2006). Source: NHDS/NCHS and NHLBI.

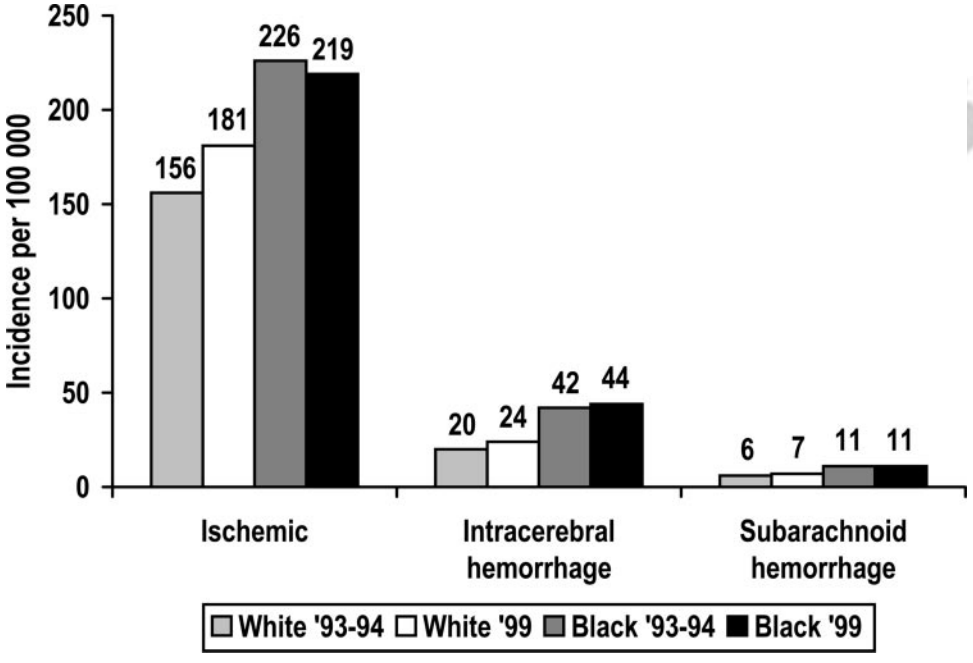


Chart 5-6. Annual age-adjusted incidence of first-ever stroke by race. Inpatient plus out-of-hospital ascertainment, 1993 to 1994 and 1999. Source: Kleindorfer et al.[19]

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]
- A higher percentage of men than women have HBP until 45 years of age. From 45 to 54 and 55 to 64 years of age, the percentages of men and women with HBP are similar. After that, a much higher percentage of women have HBP than men.[2]
- HBP is 2 to 3 times more common in women taking oral contraceptives, especially in obese and older women, than in women not taking them.[3]

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
FHS	Framingham Heart Study
HBP	high blood pressure
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
NH	non-Hispanic
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
HHANES	Hispanic Health and Nutrition Examination Survey
NINDS	National Institute of Neurological Disorders and Stroke
REGARDS	Reasons for Geographic and Racial Differences in Stroke study
SBP	systolic blood pressure

- Data from NHANES 2005–2006 found that 29% of US adults ≥18 years of age were hypertensive. The prevalence of hypertension was nearly equal between men and women. An additional 37% 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 were 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]

Older Adults

- Age-adjusted estimates show that in 2004–2005, 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 heart disease (33% for men, 26% for women) and diabetes (17% for men, 15% for women).[6]
- The age-adjusted prevalence of hypertension (both diagnosed and undiagnosed) in 1999–2002 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 this study, HBP in children and adolescents is defined as SBP and/or DBP that is, on repeated measurement, ≥95th percentile.[7]
- A study in Ohio of >14 000 children and adolescents 3 to 18 years of age observed ≥3 times between 1999 and 2006 found that 3.6% had hypertension. Of these, 26% had been diagnosed and 74% were undiagnosed. In addition, 3% of those with hypertension had stage 2 hypertension, and 41% of those with stage 2 hypertension were undiagnosed. Criteria for prehypertension were met by 485 children. Of these, 11% were diagnosed. In this study, HBP in children and adolescents is defined as SBP and/or DBP that is, on repeated measurement, ≥95th percentile.[8]
- A study from 1988–1994 through 1999–2000 of children and adolescents 8 to 17 years of age showed that among non-Hispanic blacks, mean SBP levels increased by 1.6 mm Hg among girls and 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 1988–1994 through 1999–2002, 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 heart disease death, 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 on antihypertensive medication tend to be male, to be younger, and to have infrequent contact with a physician.
- Analysis from the REGARDS study of the NINDS suggests that efforts to raise awareness of prevalent hypertension among blacks have apparently 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 in the control of BP (SBP $<$140 mm Hg, DBP $<$90 mm Hg), with the odds of control 27% lower in blacks relative to whites. In contrast, geographic disparities in hypertension awareness, treatment, and control were minimal.[12]
- Data from the 2007 NHIS survey showed that American Indian/Alaska Native adults $\geq$18 years of age were less likely (25.5%) than black adults (31.7%) and more likely than white adults (22.2%) and Asian adults (19.5%) to have been told on $\geq$2 occasions that they had hypertension.[13]
- The CDC analyzed death certificate data from 1995 to 2002. 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 female rates 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 in black women.[16]
- Data from the Multi-Ethnic Study of Atherosclerosis (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 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 2005—57 356. Total-mention mortality in 2005 was ≈319 000. Preliminary 2006 mortality—56 121. The 2005 death rate was 17.7.[19]

- From 1995 to 2005, the death rate from HBP increased 25.2%, and the actual number of deaths rose 56.4% (NCHS and NHLBI; 1995 rate modified by appropriate comparability ratio).
- The 2005 overall death rate from HBP was 18.4. Death rates were 15.8 for white males, 52.1 for black males, 15.1 for white females, and 40.3 for black females.[20] Using total-mention mortality for 2005, the overall death rate was 70.0. Death rates were 73.0 for white males, 180.8 for black males, 52.3 for white females, and 128.5 for black females.

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 physical activity, 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.[21]

Aftermath

- About 69% of people who have a first heart attack, 77% who have a first stroke, and 74% 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 current BP level.[22]
- 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.[23]
  - — 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 10 644 000[24] (unpublished data from the National Hospital Discharge Survey, 2006).
- Data from Ambulatory Medical Care Utilization Estimates for 2006 showed that the number of visits for essential hypertension was 44 879 000.[25]
- 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.[25]

Awareness, Treatment, and Control

- Data from NHANES/NCHS 2005–2006 showed that of those with hypertension ≥20 years of age, 78.7% were aware of their condition, 69.1% were under current treatment, 45.4% had it under control, and 54.6% did not have it controlled (NCHS and NHLBI).
- Analysis of NHANES/NCHS data from 1999–2004 through 2005–2006 found that there were substantial increases in awareness and treatment rates of hypertension. The control rates increased in both sexes, non-Hispanic blacks, and Mexican Americans. Among the group ≥60 years of age, awareness, treatment, and control rates of hypertension increased significantly.[4,26]
- 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%.[27]
- In NHANES/NCHS 2005–2006, rates of control were lower in Mexican Americans (35.2%) than in non-Hispanic whites (46.1%) and non-Hispanic blacks (46.5%).[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%.[28]
- 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.[29]
- 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.[30]
- A study of >300 women in Wisconsin showed a need for significant improvement in BP and low-density lipoprotein (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.[31]
- In 2005, a survey of people in 20 states conducted by the BRFSS of the CDC found that 19.4% of respondents had been told on ≥2 visits to a health professional that they had HBP. Of these, 70.9% reported changing their eating habits; 79.5% reduced the use of or were not using salt; 79.2% reduced the use of or eliminated alcohol; 68.8% were exercising; and 73.4% were taking antihypertensive medication.[32]
- On the basis of NHANES 2003–2004 data, it was found that nearly three fourths of adults with CVD comorbidities have hypertension. Poor control rates of systolic hypertension remain a principal problem that further compromises their already high CVD risk.[33]

Cost

- The estimated direct and indirect cost of HBP for 2009 is $73.4 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 2005–2006 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). Two published sources have a higher estimate, 37% overall, in part because the sources did not exclude persons within the prehypertension BP cut points who were told on 2 occasions by a doctor or other health professional of having hypertension. Those persons are part of the 73.6 million persons with hypertension.[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.[34]
- 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 prehypertensives <60 years of age, 4.9% in those 60 to 79 years of age, and 19.8% in those ≥80 years of age.[29]
- In a study of NHANES 1999–2000 (NCHS), people with prehypertension were more likely than those with normal BP levels to have above-normal cholesterol levels, overweight/obesity, and diabetes mellitus, whereas the probability of currently smoking was lower. Persons with prehypertension were 1.65 times more likely to have ≥1 of these adverse risk factors than were those with normal blood pressure.[35]

References

1. Fields LE, Burt VL, Cutler JA, Hughes J, Roccella EJ, Sorlie P. The burden of adult hypertension in the United States 1999–2000: a rising tide. *Hypertension*. 2004;44:398–404.
2. *Health, United States, 2007 With Chartbook on Trends in the Health of Americans*. Hyattsville Md: National Center for Health Statistics; 2007. Available at: http://www.cdc.gov/nchs/hus.htm. Accessed April 29, 2008.
3. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr, Jones DW, Materson BJ, Oparil S, Wright JT Jr, Roccella EJ, for the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure, National Heart, Lung, and Blood Institute, National High Blood Pressure Education Program Coordinating Committee. Seventh report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure. *Hypertension*. 2003;42:1206–1252.
4. Ostchega Y, Yoon SS, Hughes J, Louis T. *Hypertension Awareness, Treatment, and Control—Continued Disparities in Adults: United States, 2005–2006*. Hyattsville, Md: National Center for Health Statistics; 2008. NCHS Data Brief No. 3.
5. Centers for Disease Control and Prevention: BRFSS: turning information into health. Available at: http://www.cdc.gov/brfss/index.htm. Accessed September 15, 2008.
6. Robinson K. *Trends in Health Status and Health Care Use Among Older Women*. Hyattsville, Md: National Center for Health Statistics; 2007. Aging Trends No 7.
7. Din-Dzietham R, Liu Y, Bielo M-V, Shamsa F. High blood pressure trends in children and adolescents in national surveys, 1963 to 2002. *Circulation*. 2007;116:1488–1496.
8. Hansen ML, Gunn PW, Kaelber DC. Underdiagnosis of hypertension in children and adolescents. *JAMA*. 2007;298:874–879.
9. Muntner P, He J, Cutler JA, Wildman RP, Whelton PK. Trends in blood pressure among children and adolescents. *JAMA*. 2004;291:2107–2113.
10. Hertz RP, Unger AN, Cornell JA, Saunders E. Racial disparities in hypertension prevalence, awareness and management. *Arch Intern Med*. 2005;165:2098–2104.
11. Collins R, Winkleby MA. African American women and men at high and low risk for hypertension: a signal detection analysis of NHANES III, 1988–1994. *Prev Med*. 2002;35:303–312.
12. Howard G, Prineas R, Moy C, Cushman M, Kellum M, Temple E, Graham A, Howard V. Racial and geographic differences in awareness, treatment, and control of hypertension: the Reasons for Geographic and Racial Differences in Stroke Study. *Stroke*. 2006;37:1171–1178.
13. Pleis JR, Lucas JW. Summary health statistics for U.S. adults: National Health Interview Survey, 2007: National Center for Health Statistics. *Vital Health Stat 10*. In press.
14. Centers for Disease Control and Prevention (CDC). Hypertension-related mortality among Hispanic subpopulations: United States, 1995–2002. *MMWR Morb Mortal Wkly Rep*. 2006;55:177–180.
15. Borrell LN. Self-reported hypertension and race among Hispanics in the National Health Interview Survey. *Ethn Dis*. 2006;16:71–77.
16. Jones DW, Chambless LE, Folsom AR, Heiss G, Hutchinson RG, Sharrett AR, Szklo M, Taylor HA Jr. Risk factors for coronary heart disease in African Americans: the Atherosclerotic Risk in Communities Study, 1987–1997. *Arch Intern Med*. 2002;162:2565–2571.
17. Moran A, Roux AV, Jackson SA, Kramer H, Manolio T, Shrager S, Shea S. Acculturation is associated with hypertension in a multiethnic sample. *Am J Hypertens*. 2007;20:354–363.
18. Barnes PM, Adams PF, Powell-Griner E. *Health Characteristics of the Asian Adult Population: United States, 2004–2006: Advance Data From Vital and Health Statistics; No. 394*. Hyattsville, Md: National Center for Health Statistics; 2008.
19. Heron MP, Hoyert DL, Xu J, Scott C, Tejada-Vera B. Preliminary data for 2006. *National Vital Statistics Reports; Vol. 56; No 16*. Hyattsville, Md: National Center for Health Statistics; 2008.
20. National Center for Health Statistics, Centers for Disease Control and Prevention. Compressed mortality file: underlying cause of death, 1979 to 2005. Atlanta, Ga: Centers for Disease Control and Prevention. Available at: http://wonder.cdc.gov/mortSQL.html. Accessed June 2008.
21. Levy D, DeStefano AL, Larson MG, O'Donnell CJ, Lifton RP, Gavras H, Cupples LA, Myers RH. Evidence for a gene influencing blood pressure on chromosome 17: genome scan linkage results for longitudinal blood pressure phenotypes in subjects from the Framingham Heart Study. *Hypertension*. 2000;36:477–483.
22. Vasan RS, Massaro JM, Wilson PW, Seshadri S, Wolf PA, Levy D, D'Agostino RB, for the Framingham Heart Study. Antecedent blood pressure and risk of cardiovascular disease: the Framingham Heart Study. *Circulation*. 2002;105:48–53.
23. Franco OH, Peeters A, Bonneux L, de Laet C. Blood pressure in adulthood and life expectancy with cardiovascular disease in men and women: life course analysis. *Hypertension*. 2005;46:280–286.
24. Graves EJ, Kozak LJ. Detailed diagnoses and procedures: National Hospital Discharge Survey, 1996. *Vital Health Stat 13*. 1998:i–iii, 1–151.

25. Schappert SM, Rechsteiner EA. *Ambulatory Medical Care Utilization Estimates for 2006*. Hyattsville, Md: National Center for Health Statistics; 2008. National Health Statistics Reports No. 8.
26. Ong KL, Cheung BM, Man YB, Lau CP, Lam KS. Prevalence, awareness, treatment and control of hypertension among United States adults 1999–2004. *Hypertension*. 2007;49:69–75.
27. Centers for Disease Control and Prevention. BRFSS: prevalence and trends data. Available at: http://apps.nccd.cdc.gov/brfss/index.asp. Accessed May 19, 2008.
28. Psaty BM, Manolio TA, Smith NL, Heckbert SR, Gottdiener JS, Burke GL, Weissfeld J, Enright P, Lumley T, Powe N, Furberg CD, for the Cardiovascular Health Study. Time trends in high blood pressure control and the use of antihypertensive medications in older adults: the Cardiovascular Health Study. *Arch Intern Med*. 2002;162:2325–2332.
29. Lloyd-Jones DM, Evans JC, Levy D. Hypertension in adults across the age spectrum: current outcomes and control in the community. *JAMA*. 2005;294:466–472.
30. Wassertheil-Smoller S, Anderson G, Psaty BM, Black HR, Manson J, Wong N, Francis J, Grimm R, Kotchen T, Langer R, Lasser N. Hypertension and its treatment in postmenopausal women: baseline data from the Women's Health Initiative. *Hypertension*. 2000;36:780–789.
31. Sanchez RJ, Khalil L. Badger Heart Program: health screenings targeted to increase cardiovascular awareness in women at four northern sites in Wisconsin. *WMJ*. 2005;104:24–29.
32. Centers for Disease Control and Prevention (CDC). Prevalence of actions to control high blood pressure: 20 states, 2005. *MMWR Morb Mortal Wkly Rep*. 2007;56:420–423.
33. Wong ND, Lopez VA, L'Italien G, Chen R, Kline SE, Franklin SS. Inadequate control of hypertension in US adults with cardiovascular disease comorbidities in 2003–2004. *Arch Intern Med*. 2007;167:2431–2436.
34. Vasan RS, Larson MG, Leip EP, Kannel WB, Levy D. Assessment of frequency of progression to hypertension in non-hypertensive participants in the Framingham Heart Study: a cohort study. *Lancet*. 2001;358:1682–1686.
35. Greenlund KJ, Croft JB, Mensah GA. Prevalence of heart disease and stroke risk factors in persons with prehypertension in the United States, 1999–2000. *Arch Intern Med*. 2004;164:2113–2118.
36. Cutler JA, Sorlie P, Wolz M, Thom T, Fields LE, Rocella EJ. Trends in hypertension prevalence, awareness, treatment, and control rates in the United States adults between 1988–94 and 1999–2004. *Hypertension*. 2008;52:818–827.

Table 6-1. High Blood Pressure

Population Group	Prevalence, 2006 Age ≥20 y	Mortality,* 2005 All Ages	Hospital Discharges, 2006 All Ages	Estimated Cost, 2009
Both sexes	73 600 000 (33.3%)	57 356	514 000	$73.4 billion
Males	35 300 000 (34.1%)	24 046 (41.9%)†	204 000	. . .
Females	38 300 000 (32.1%)	33 310 (58.1%)†	309 000	. . .
NH white males	34.1%	17 312	. . .	. . .
NH white females	30.3%	25 814	. . .	. . .
NH black males	44.4%	6019	. . .	. . .
NH black females	43.9%	6746	. . .	. . .
Mexican American males	23.1%	. . .	. . .	. . .
Mexican American females	30.4%	. . .	. . .	. . .
Hispanic or Latino‡ ≥18 y	20.6%	. . .	. . .	. . .
Asian‡ ≥18 y	19.5%	. . .	. . .	. . .
American Indians/Alaska Natives‡ ≥18 y	25.5%	. . .	. . .	. . .

Ellipses (. . .) indicate data not available.

*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 (2007), NCHS; data are weighted percentages for Americans ≥18 years of age.[13]

Sources: Prevalence: NHANES (2005–2006, NCHS) and NHLBI; percentages for racial/ethnic groups are age adjusted for Americans ≥20 years of age. Estimates from NHANES 2005–2006 (NCHS) applied to 2006 population estimates ≥20 years of age. 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.

Note: 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 2005–2006 (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% according to the more complete definition, a difference of 8 million persons.

Table 6-2. Hypertension Awareness, Treatment, and Control: NHANES 1988–1994 and 1999–2004, by Race[36]

	Awareness		Treatment		Control	
	1988–1994	1999–2004	1988–1994	1999–2004	1988–1994	1999–2004
NH white male	63.0%	70.4%	46.2%	60.0%	22.0%	39.3%
NH white female	74.7%	73.4%	61.6%	64.0%	32.2%	34.5%
NH black male	62.5%	67.8%	42.3%	56.4%	16.6%	29.9%
NH black female	77.8%	81.8%	64.6%	71.7%	30.0%	36.0%
Mexican American male	47.8%	55.9%	30.9%	40.4%	13.5%	21.4%
Mexican American female	69.3%	66.9%	47.8%	54.9%	19.4%	27.4%

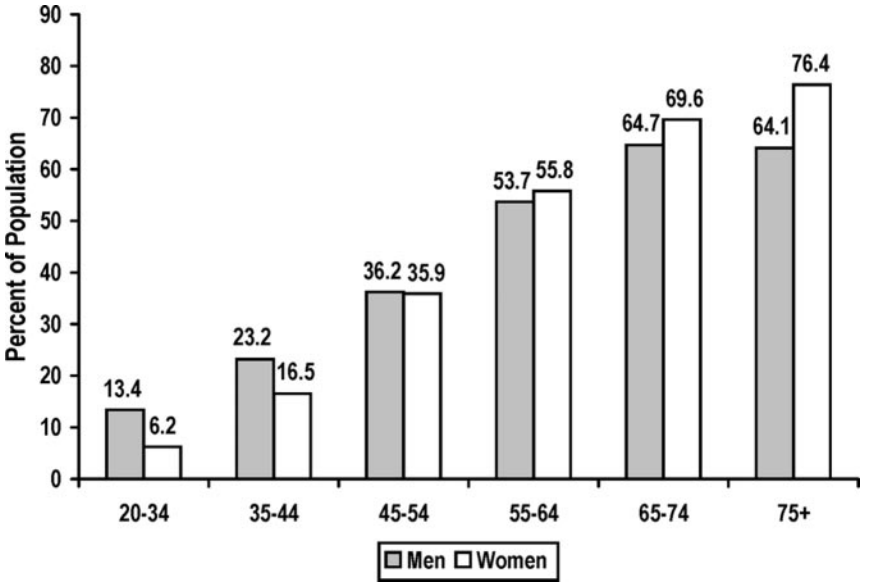


Chart 6-1. Prevalence of HBP in adults ≥20 years by age and sex (NHANES: 2005 to 2006). Source: NCHS and NHLBI. Hypertension is defined as SBP ≥140 mm Hg or DBP ≥90 mm Hg, taking antihypertensive medication, or being told twice by a physician or other professional that one has hypertension.

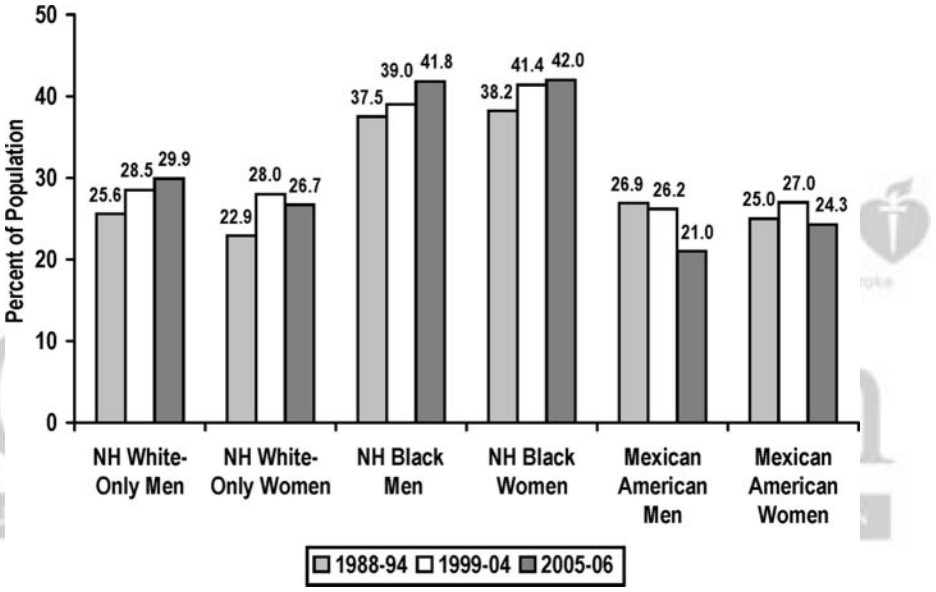


Chart 6-2. Age-adjusted prevalence trends for HBP in adults ≥20 years of age by race/ethnicity, sex, and survey (NHANES: 1988 to 1994, 1999 to 2004, and 2005 to 2006). Source: NCHS and NHLBI.

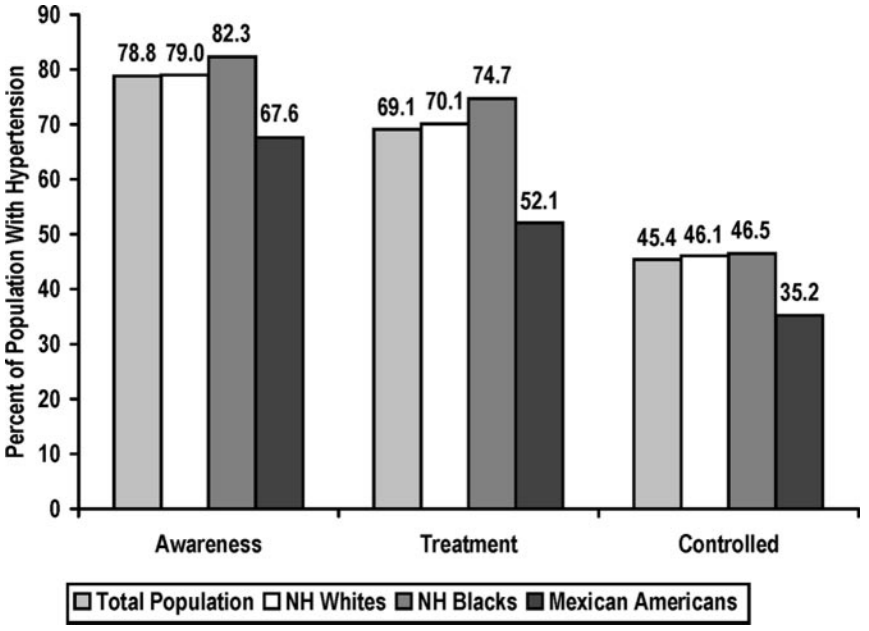


Chart 6-3. Extent of awareness, treatment, and control of HBP by race/ethnicity (NHANES: 2005 to 2006). Source: NCHS and NHLBI.

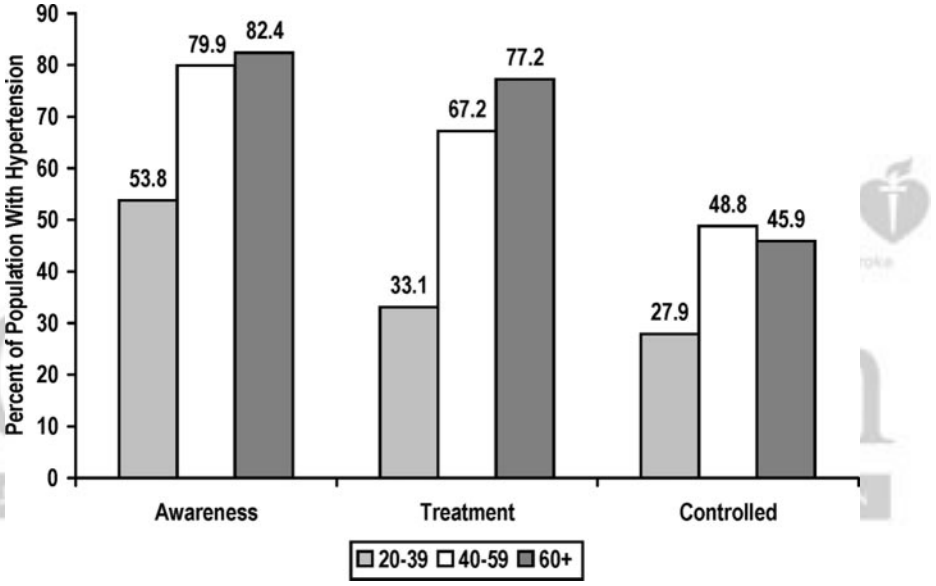


Chart 6-4. Extent of awareness, treatment, and control of HBP by age (NHANES: 2005 to 2006). Source: NCHS and NHLBI.

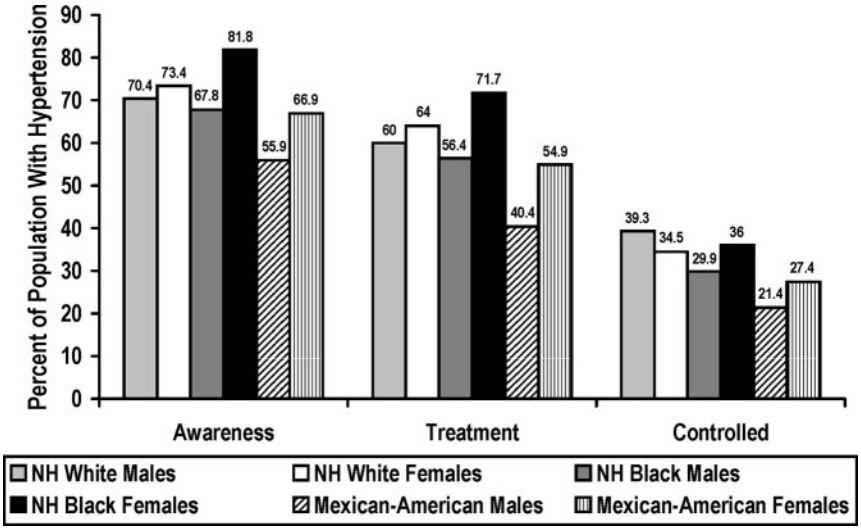


Chart 6-5. Extent of awareness, treatment, and control of HBP by race/ethnicity and sex (NHANES: 1999 to 2004). Source: Cutler et al.[36]

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 arising from abnormal formation of the heart or major blood vessels. At least 18 distinct types of congenital heart defects are recognized, with many additional anatomic variations.

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

VSD is the most common defect. Many close spontaneously, but VSDs still account for 14% to 16% of defects requiring an invasive procedure within the first year of life.[1]

Prevalence

As of 2002, the prevalence of congenital cardiovascular defects in the United States varied between 4 and 10 per 1000 live births and was estimated to range from 650 000 to 1.3 million people.[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 authors estimate their survival to 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]

Abbreviations Used in Chapter 7

ASD	atrial septal defect
CDC	Centers for Disease Control and Prevention
CHD	coronary heart disease
ICD	*International Classification of Diseases*
KID	Kids' Inpatient Database
MACDP	Metropolitan Atlanta Congenital Defects Program
NCHS	National Center for Health Statistics
NHDS	National Hospital Discharge Survey
VSD	ventricular septal defect

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. A recent report 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; atrial septal defect (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 tetralogy of Fallot.[3]

Incidence

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 presenting 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 shown 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 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.[6]
- Estimates also are available for bicuspid aortic valves, occurring in 13.7 per 1000 people; these defects may not require treatment in infancy but can cause problems later in adulthood.[7]
- 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.[8,9] 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. These data indicated that there are >6500 estimated annual cases of 5 cardiovascular defects: truncus arteriosus, transposition of the great arteries, tetralogy of Fallot, atrioventricular septal defect, and hypoplastic left heart syndrome.[10]

- Data from the MACDP showed that the overall prevalence of major defects was stable from 1978 to 2005. The prevalence of defects was generally lower among births to black and Hispanic mothers than to white mothers. During this period, the number of births in the metropolitan Atlanta area more than doubled.[11]

Risk Factors

- A recent 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]
- 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.[13]
- Pregestational diabetes mellitus was significantly associated with cardiac defects, both isolated and multiple. Gestational diabetes mellitus was associated with a limited group of birth defects.[14]

Mortality

Congenital cardiovascular defects mortality in 2005—3637.
Total-mention mortality in 2005—5510.

- Congenital cardiovascular defects are the most common cause of infant death resulting from birth defects; >29% of infants who die of a birth defect have a heart defect (National Vital Statistics System, final data for 2005).
- The 2005 death rate for congenital cardiovascular defects was 1.2. Death rates were 1.3 for white males, 1.4 for black males, 1.1 for white females, and 1.4 for black females. Crude infant death rates (<1 year of age) were 39.0 for white infants and 47.7 for black infants.[15]
- In 2005, 192 000 life-years were lost before 55 years of age because of deaths from congenital cardiovascular defects. This is about the same as the life-years lost from leukemia, prostate cancer, and Alzheimer's disease combined.[15]
- The mortality rate from congenital defects has been declining. From 1979 to 1997, age-adjusted death rates from 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. Mortality rate varies considerably according to type of defect.[16]
- From 1995 to 2005, death rates for congenital cardiovascular defects declined 42.1%, whereas the actual number of deaths declined 27.3%.[15]
- 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%.[17]

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.[18]

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 Surveillance Report, it was found that the most expensive average neonatal hospital charges were for 2 congenital heart defects: hypoplastic left heart syndrome ($199 597) and common truncus arteriosus ($192 781). Two other cardiac defects, coarctation of the aorta and transposition of the great arteries, 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.[19]
- 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.[18]

References

1. Moller JH. Prevalence and incidence of cardiac malformation. In: *Perspectives in Pediatric Cardiology*. Armonk, NY: Futura Publishing Co; 1998;6:19–26.
2. Hoffman JI, Kaplan S, Liberthson RR. Prevalence of congenital heart disease. *Am Heart J*. 2004;147:425–439.
3. Warnes CA, Liberthson R, Danielson GK, Dore A, Harris L, Hoffman JI, Somerville J, Williams RG, Webb GD. Task force 1: the changing profile of congenital heart disease in adult life. *J Am Coll Cardiol*. 2001;37:1170–1175.
4. Marelli AJ, Mackie AS, Ionescu-Ittu R, Rahme E, Pilote L. Congenital heart disease in the general population: changing prevalence and age distribution. *Circulation*. 2007;115:163–172.

5. Botto LD, Correa A, Erickson JD. Racial and temporal variations in the prevalence of heart defects. *Pediatrics*. 2001;107:E32.
6. Prevalence and incidence of cardiac malformations. In: Moller JH, ed. *Surgery of Congenital Heart Disease: Pediatric Cardiac Care Consortium, 1984–1995*. Armonk, NY: Futura Publishing Co; 1998:20.
7. Hoffman JI, Kaplan S. The incidence of congenital heart disease. *J Am Coll Cardiol*. 2002;39:1890–1900.
8. Roguin N, Du ZD, Barak M, Nasser N, Hershkowitz S, Milgram E. High prevalence of muscular ventricular septal defect in neonates. *J Am Coll Cardiol*. 1995;26:1545–1548.
9. Sands AJ, Casey FA, Craig BG, Dornan JC, Rogers J, Mulholland HC. Incidence and risk factors for ventricular septal defect in "low-risk" neonates. *Arch Dis Child Fetal Neonatal Ed*. 1999;81:F61–F63.
10. Centers for Disease Control and Prevention (CDC). Improved national prevalence estimates for 18 selected major birth defects: United States, 1999–2001. *MMWR Morb Mortal Wkly Rep*. 2006;54:1301–1305.
11. Centers for Disease Control and Prevention (CDC). Update on overall prevalence of major birth defects: Atlanta, Georgia, 1978–2005. *MMWR Morb Mortal Wkly Rep*. 2008;57:1–5.
12. Malik S, Cleves MA, Honein MA, Romitti PA, Botto LD, Yang S, Hobbs CA, for the National Birth Defects Prevention Study. Maternal smoking and congenital heart defects. *Pediatrics*. 2008;121:e810–e816.
13. Waller DK, Shaw GM, Rasmussen SA, Hobbs CA, Canfield MA, Siega-Riz AM, Gallaway MS, Correa A, for the for the National Birth Defects Prevention Study. Prepregnancy obesity as a risk factor for structural birth defects. *Arch Pediatr Adolesc Med*. 2007;161:745–750.
14. Correa A, Gilboa SA, Besser LM, Botto LD, Moore CA, Hobbs CA, Cleves MA, Riehle-Colarusso TJ, Waller DK, Reece A. Diabetes mellitus and birth defects. *Am J Obstet Gynecol*. 2008;199:237.e1–237.e9.
15. Centers for Disease Control and Prevention. Compressed mortality file: underlying cause of death, 1979 to 2005. Atlanta, Ga: Centers for Disease Control and Prevention. Available at: http://wonder. cdc.gov/mortSQL.html. Accessed August 11, 2008.
16. Boneva RS, Botto LD, Moore CA, Yang Q, Correa A, Erickson JD. Mortality associated with congenital heart defects in the United States: trends and racial disparities, 1979–1997. *Circulation*. 2001;103:2376–2381.
17. Jacobs JP, Jacobs ML, Mavroudis C, Lacour-Gayet F, Tchervenkov CI. Executive summary: the Society of Thoracic Surgeons Congenital Heart Surgery Database: Eighth Harvest (2004–2007): The Society of Thoracic Surgeons (STS) and Duke Clinical Research Institute (DCRI), Duke University Medical Center, Durham, North Carolina, United States, Spring 2008 Harvest. Available at: http://www.sts.org/documents/pdf/ndb/Spring_2008_STSCONG-ALLPatientsSUMMARY.pdf. Accessed November 18, 2008.
18. Russo CA, Elixhauser A. *Healthcare Cost and Utilization Project (HCUP) Statistical Brief #24: Hospitalizations for Birth Defects, 2004*. Rockville, Md: US Agency for Healthcare Research and Quality; January 2007. Available at: http://www.hcupdoc.net/reports/statbriefs/sb24.pdf. Accessed October 23, 2007.
19. Centers for Disease Control and Prevention. Hospital stays, hospital charges, and in-hospital deaths among infants with selected birth defects: United States, 2003. *MMWR Morb Mortal Wkly Rep*. 2007;56:25–29.
20. Larson EW, Edwards WD. Risk factors for aortic dissection: a necropsy study of 161 cases. *Am J Cardiol*. 1984;53:849–855.

Table 7-1. Congenital Cardiovascular Defects

Population Group	Estimated Prevalence All Ages	Incidence in Infants	Mortality, 2005 All Ages	Hospital Discharges, 2006 All Ages
Both sexes	650 000 to 1.3 million[2]	36 000[6]	3637	70 000
Males	...	...	1931 (54.1%)*	30 000
Females	...	...	1706 (45.9%)*	40 000
White males	...	...	1564	...
White females	...	...	1320	...
Black males	...	...	291	...
Black females	...	...	309	...

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[6–9,20]

Type of Presentation	Rate per 1000 Live Births	No.
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			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
Tetralogy of Fallot	61	32	28	6.1	7	5.4
Atrioventricular septal defect	31	18	13	3.1	3.9	2.5
Transposition of great arteries	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
Hypoplastic left heart syndrome	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. Copyright 2004. Average of the low and high estimates, two thirds from low estimate.[2]

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 gender (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 for hypoplastic left heart syndrome	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 hypoplastic left heart syndrome. 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% versus 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.

8. Heart Failure

ICD-9 428, ICD-10 I50. See Table 8-1 and Charts 8-1 through 8-3.

Incidence

- Data from the NHLBI-sponsored FHS[1] 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).[2]
- 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).[3]
- In Olmsted County, Minn, the incidence of HF (ICD-9 428) did not decline during 2 decades, but the survival rate improved overall, with less improvement, however, among women and elderly persons.[4]
- 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—ie, 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.[5]
- 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.[6]
- 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%.[7]
- 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.[6]

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.[8]
- In a 1993–2000 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.[9]
- 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

*Unreliable estimate.

Abbreviations Used in Chapter 8

ARIC	Atherosclerosis Risk in Communities study
BMI	Body mass index
BP	Blood pressure
CHD	Coronary heart disease
CHS	Cardiovascular Health Study
EF	Ejection fraction
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

rate was 46% for those with HF alone but only 37% for those with HF and diabetes mellitus.[10]

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.[11]
  - — 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%.[12]
  - — 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.[13]

Mortality

In 2005, HF total-mention mortality was 292 214. HF was mentioned on 292 214 US death certificates and was selected as the "underlying cause" in 58 933 of those deaths.[14] In preliminary 2006 mortality, HF was selected as the "underlying cause" in 60 315 deaths. 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 58 933 deaths, the true underlying cause—ie, the "etiology" 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 2005 overall total-mention death rate for HF was 52.3. Total-mention death rates were 62.1 for white males, 81.9 for black males, 43.2 for white females, and 58.7 for black females (NCHS, NHLBI).
- One in 8 deaths has HF mentioned on the death certificate (NCHS, NHLBI).
- The number of total-mention deaths from HF was nearly as high in 1995 (287 000) as it was in 2005 (292 000) (NCHS, NHLBI).
- On the basis of the 44-year follow-up of the original FHS cohort (NHLBI) and 20-year follow-up of the offspring cohort:
  - — Eighty percent of men and 70% of women <65 years of age who have HF will die within 8 years.
  - — After HF is diagnosed, the survival rate is lower in men than in women, but <15% of women survive more than 8 to 12 years. The 1-year mortality rate is high, with 1 in 5 dying.
  - — In people diagnosed with HF, sudden cardiac death occurs at 6 to 9 times the rate of the general population.[15]

Hospital Discharges

- Hospital discharges for HF rose from 877 000 in 1996 to 1 106 000 in 2006 (unpublished data from the NHDS 2006, NCHS).[16]
- Data from Ambulatory Medical Care Utilization Estimates for 2006 showed the number of visits for HF was 3 390 000.[17]

Cost

- The estimated direct and indirect cost of HF in the United States for 2009 is $37.2 billion.[18] (See Chapter 20).

References

1. Lloyd-Jones DM, Larson MG, Leip EP, Beiser A, D'Agostino RB, Kannel WB, Murabito JM, Vasan RS, Benjamin EJ, Levy D, for the Framingham Heart Study. Lifetime risk for developing congestive heart failure: the Framingham Heart Study. *Circulation*. 2002;106:3068–3072.
2. *Incidence and Prevalence: 2006 Chart Book on Cardiovascular and Lung Diseases*. Bethesda, Md: National Heart, Lung, and Blood Institute; 2006.
3. Djoussé L, Kochar J, Gaziano JM. Secular trends of heart failure among US male physicians. *Am Heart J*. 2007;154:855–860.
4. Roger VL, Weston SA, Redfield MM, Hellermann-Homan JP, Killian J, Yawn BP, Jacobsen SJ. Trends in heart failure incidence and survival in a community-based population. *JAMA*. 2004;292:344–350.
5. Loehr LR, Rosamond WD, Chang PP, Folsom AR, Chambless LE. Heart failure incidence and survival (from the Atherosclerosis Risk in Communities study). *Am J Cardiol*. 2008;101:1016–1022.
6. Barker WH, Mullooly JP, Getchell W. Changing incidence and survival for heart failure in a well-defined older population, 1970–1974 and 1990–1994. *Circulation*. 2006;113:799–805.
7. Goldberg RJ, Spencer FA, Farmer C, Meyer TE, Pezzella S. Incidence and hospital death rates associated with heart failure: a community-wide perspective. *Am J Med*. 2005;118:728–734.
8. Levy D, Larson MG, Vasan RS, Kannel WB, Ho KK. The progression from hypertension to congestive heart failure. *JAMA*. 1996;275:1557–1562.
9. Bibbins-Domingo K, Lin F, Vittinghoff E, Barrett-Connor E, Hulley SB, Grady D, Shlipak MG. Predictors of heart failure among women with coronary disease. *Circulation*. 2004;110:1424–1430.
10. From AM, Leibson CL, Bursi F, Redfield MM, Weston SA, Jacobsen SJ, Rodeheffer RJ, Roger VL. Diabetes in heart failure: prevalence and impact on outcome in the population. *Am J Med*. 2006;119:591–599.
11. Redfield MM, Jacobsen SJ, Burnett JC Jr, Mahoney DW, Bailey KR, Rodeheffer RJ. Burden of systolic and diastolic ventricular dysfunction in the community: appreciating the scope of the heart failure epidemic. *JAMA*. 2003;289:194–202.
12. Bursi F, Weston SA, Redfield MM, Jacobsen SJ, Pakhomov S, Nkomo VT, Meverden RA, Roger VL. Systolic and diastolic heart failure in the community. *JAMA*. 2006;296:2209–2216.
13. Owan TE, Hodge DO, Herges RM, Jacobsen SJ, Roger VL, Redfield MM. Trends in prevalence and outcome of heart failure with preserved ejection fraction. *N Engl J Med*. 2006;355:251–259.

14. National Center for Health Statistics. Centers for Disease Control and Prevention. *Compressed Mortality File: Underlying Cause of Death*. Atlanta, Ga: Centers for Disease Control and Prevention. Available at: http://wonder.cdc.gov/mortSQL.html. Accessed August 11, 2008.
15. Thom TJ, Kannel WB, Silbershatz H, D'Agostino RB Sr. Cardiovascular diseases in the United Sates and prevention approaches. In: Fuster V, Alexander RW, O'Rourke RA, eds. *Hurst's The Heart, Arteries and Veins*. 10th ed. New York, NY: McGraw-Hill; 2001:3–17.
16. Graves EJ, Kozak LJ. Detailed diagnoses and procedures: National Hospital Discharge Survey, 1996. *Vital Health Stat 13*. 1998;i–iii: 1–151.
17. Schappert SM, Rechsteiner EA. *Ambulatory Medical Care Utilization Estimates for 2006*. Hyattsville, Md: National Center for Health Statistics; 2008. National Health Statistics Reports No. 8.
18. Centers for Medicare & Medicaid Services. *Health Care Financing Review: Medicare & Medicaid Statistical Supplement*. Table 5.5: Discharges, Total Days of Care, and Program Payments for Medicare Beneficiaries Discharged from Short-Stay Hospitals, by Principal Diagnoses Within Major Diagnostic Classifications (MDCs): Calendar Year 2006. Baltimore, MD: Centers for Medicare and Medicaid Services; 2005. Available at: http://www.cms.hhs.gov/MedicareMedicaidStatSupp/. Accessed August 28, 2008.

Table 8-1. Heart Failure

Population Group	Prevalence, 2006 Age ≥20 y	Incidence (New Cases) Age ≥45 y	Mortality (Total Mentions), 2004 All Ages*	Hospital Discharges, 2006 All Ages	Cost, 2009
Both sexes	5 700 000 (2.5%)	670 000	292 214	1 106 000	$37.2 billion
Males	3 200 000 (3.2%)	350 000	126 163 (43.2%)†	523 000	. . .
Females	2 500 000 (2.0%)	320 000	166 051 (56.8%)†	583 000	. . .
NH white males	3.1%	. . .	112 550	. . .	. . .
NH white females	1.8%	. . .	148 582	. . .	. . .
NH black males	4.2%	. . .	11 276	. . .	. . .
NH black females	4.2%	. . .	14 928	. . .	. . .
Mexican American males	2.1%	. . .	. . .	. . .	. . .
Mexican American females	1.4%	. . .	. . .	. . .	. . .

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 2005–2006 (NCHS) and NHLBI; percentages are age adjusted for Americans ≥20 years of age. These data are based on self-reports. Estimates from NHANES 2005–2006 applied to 2006 population estimates ≥20 years of age. Incidence: FHS, 1980–2003 from NHLBI Incidence and Prevalence Chart Book, 2006. Mortality: NCHS. HF as an underlying cause of death accounted for 58 933 of the total-mention deaths in 2005: 23 026 males and 35 907 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 2009.

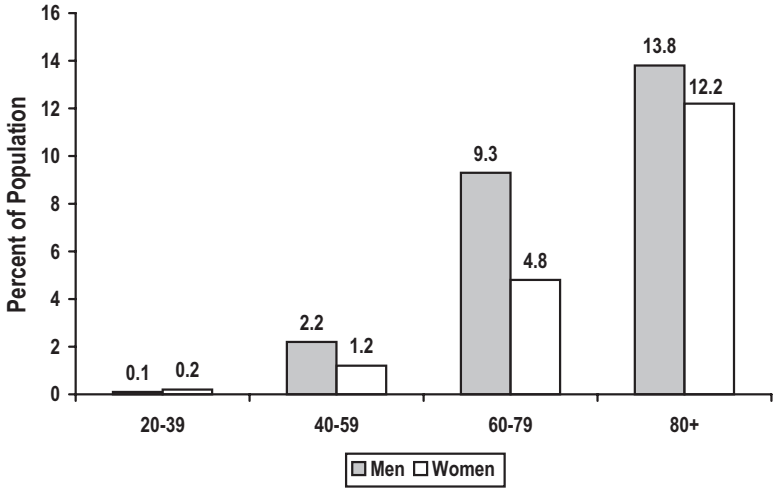


Chart 8-1. Prevalence of HF by sex and age (NHANES: 2005–2006). Source: NCHS and NHLBI.

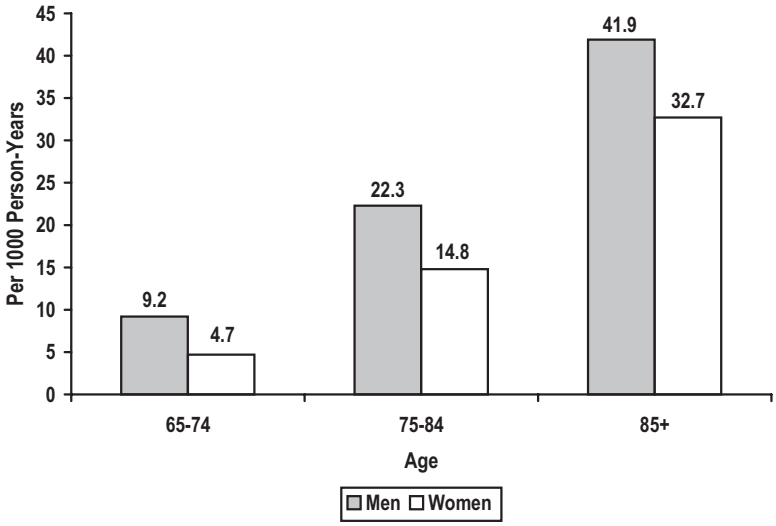


Chart 8-2. Incidence of HF (HF based on physician review of medical records and strict diagnostic criteria) by age and sex (FHS 1980–2003). Source: NHLBI.

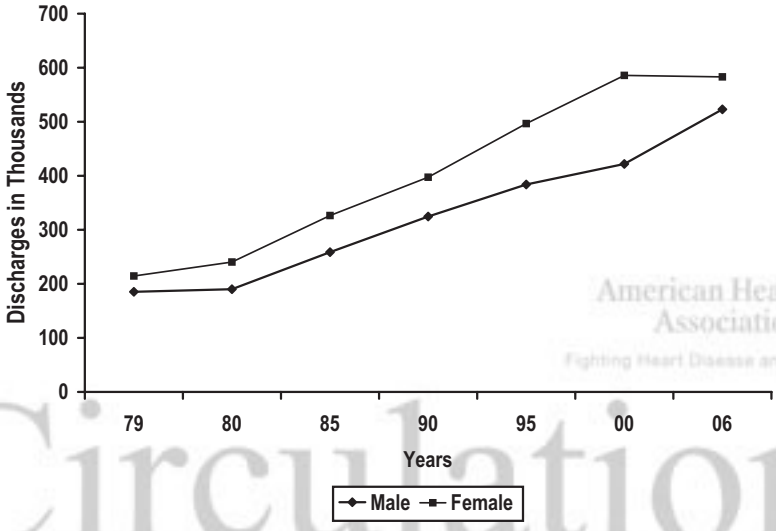


Chart 8-3. Hospital discharges for HF by sex (United States: 1979–2006). Note: Hospital discharges include people discharged alive, dead, and "status unknown." Source: NHDS/NCHS, and NHLBI.

9. Other Cardiovascular Diseases

See Table 9-1.

Mortality and total mentions in this section are for 2005. "Mortality" is the number of deaths in 2005 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-9 390-398; ICD-10 I00-I09. See Table 9-1.

Mortality—3365. Total-mention mortality—6188.

- The incidence of rheumatic fever remains high in blacks, Puerto Ricans, Mexican Americans, and American Indians.[1]

Abbreviations Used in Chapter 9

AAA	abdominal aortic aneurysm
AF	atrial fibrillation
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
COPD	chronic obstructive pulmonary disease
CVD	cardiovascular disease
DVT	deep vein thrombosis
FHS	Framingham Heart Study
HBP	high blood pressure
HCUP	Healthcare Cost and Utilization Project
HF	heart failure
NIS	Nationwide Inpatient Sample
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
PHD	pulmonary heart disease
REACH	Reduction of Atherothrombosis for Continued Health
VTE	venous thromboembolism
WHO	World Health Organization
WRIGHT	WHO Research Into Global Hazards of Travel

- In 1950, ≈15 000 Americans (adjusted for changes in *ICD* codes) died of rheumatic fever/rheumatic heart disease, compared with ≈3400 today.
- From 1995 to 2005, the death rate from rheumatic fever/rheumatic heart disease fell 38.9%, and actual deaths declined 25.8%.
- The 2005 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 0.8 for black females.

Pulmonary Heart Disease

ICD-9 415-417; ICD-10 I26-I28.

Mortality in 2005—13 504. Total-mention mortality in 2005—44 012.

Data from the HCUP 2005 NIS[2] found the following:

- The number of PHD-related all-listed-diagnosis hospital stays increased by >50% from 1997 to 2005, from 301 400 to 456 500.
- PHD was listed in ≈20 000 in-hospital deaths. The in-hospital mortality rate for patients with PHD was >2 times greater than for all patients being cited.
- The mean age for patients hospitalized with PHD was 69 years.
- From 1997 to 2005, heart conditions were recorded as the principal reason for admission in 7 of the 10 stays that noted PHD as a secondary condition. Respiratory conditions were the principal reason in 3 of 10 PHD-related stays. In 2005, the largest share of PHD-related stays was for treatment of CHF (21%), followed by COPD (7.9%) and pneumonia (6.7%).

Pulmonary Embolism

ICD-9 415.1; ICD-10 I26.

Mortality—8283. Total-mention mortality—27 304. 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.[3]
- Death occurs in ≈12% of recognized PE cases within 1 month of diagnosis.[4]
- 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%).[3]
- 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.[3]
- 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.[5]

Bacterial Endocarditis

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

Total-mention mortality—2487. Hospital discharges—30 000, primary plus secondary diagnoses.

- The 2007 AHA Guidelines on Prevention of Infective Endocarditis[6] 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.[7]
- 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.[8]
- 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[6]:

  — 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.[6]

Valvular Heart Disease

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

Mortality—20 891. Total-mention mortality—43 900. 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; $P=0.0005$).[8]
- 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.[9]

Aortic Valve Disorders

ICD-9 424.1; ICD-10 I35.

Mortality—13 137. Total-mention mortality—27 390. Hospital discharges—49 000.

- 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.[10] Clinical factors associated with aortic sclerosis and stenosis were similar to risk factors for atherosclerosis.[11] 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—2605. Total-mention mortality—about 6210. Hospital discharges—41 000.

- The NHLBI-sponsored FHS reports that among people 26 to 84 years of age, prevalence of mitral valve disorders is ≈1% to 2% and equal between women and men.[12]
- The prevalence of mitral valve prolapse in the general population was evaluated with the use of echocardiograms of 1845 women and 1646 men who participated in the fifth examination of the Offspring Cohort of the FHS. The prevalence of mitral valve prolapse was 2.4%. The frequencies of chest pain, dyspnea, and ECG abnormalities were similar among subjects with prolapse and those without prolapse.[12]

Pulmonary Valve Disorders
ICD-9 424.3; ICD-10 I37.
Mortality—20. Total-mention mortality—45.

Tricuspid Valve Disorders
ICD-9 424.2; ICD-10 I36.
Mortality—20. Total-mention mortality—114.

Endocarditis, Valve Unspecified
ICD-9 424.9; ICD-10 I38.
Mortality—5109. Total mention mortality—10 120.

Cardiomyopathy
ICD-9 425; ICD-10 I42.
Mortality—25 505. Total-mention mortality—51 100. 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.[13]
- 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).[14]
  - 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.[15]
- 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, some 500 000 people have hypertrophic cardiomyopathy, yet most are unaware of it.[16]
- 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.[17]
- 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%).[18]

Arrhythmias (Disorders of Heart Rhythm)
ICD-9 426, 427; ICD-10 I46-I49.
Mortality—37 633. Total-mention mortality—466 750. 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—11 555. Total-mention mortality—88 000. Prevalence—>2 200 000.[20] 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]
— People who have ischemic strokes caused by AF have been reported to be 2.23 times more likely to be bedridden than those who have strokes from other causes.[32]
— Isolated chronic atrial flutter is uncommon but is associated with a high risk of developing AF,[33] 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.[34]
— A study of >4600 patients diagnosed with first AF showed that risk of death within the first 4 months after the AF diagnosis was high. The most common causes of CVD death were CAD, HF, and ischemic stroke, accounting for 22%, 14%, and 10% respectively, of the early deaths (within the first 4 months) and 15%, 16%, and 7%, respectively, of the late deaths.[27]

Other Arrhythmias

Tachycardia

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

Mortality—586. Total-mention mortality—6300. Hospital discharges—80 000.

Paroxysmal Supraventricular Tachycardia
ICD-9 427.0; ICD-10 I47.1.

Mortality—118. Total-mention mortality—1368. Hospital discharges—29 000.

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

Mortality—1076. Total-mention mortality—10 600. 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, Minnesota, the incidence of out-of-hospital treated ventricular fibrillation decreased from 1985 to 2002[35]:

— 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-9 440-448; ICD-10 I70-I79. Includes peripheral arterial disease (PAD).

Mortality—35 458. Total-mention mortality—102 200. Hospital discharges—284 000.

Aortic Aneurysm

ICD-9 441; ICD-10 I71.

Mortality—13 843. Total-mention mortality—19 600. 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.[36]
- The prevalence of AAAs 2.9 to 4.9 cm in diameter ranges from 1.3% in men 45 to 54 years of age to 12.5% in men 75 to 84 years of age. For women, the prevalence ranges from 0% in the youngest to 5.2% in the oldest age groups.[36]
- Factors associated with increased prevalence of AAA include older age, male sex, family history of AAA, tobacco use, hypertension, dyslipidemia, and manifest atherosclerotic disease in other vascular beds.[36]
- Large AAAs tend to expand more rapidly than small AAAs, and large AAAs are at substantially higher risk for rupture.[36]

— Average annual expansion rates are ≈1 to 4 mm for aneurysms <4.0 cm in diameter, 4 to 5 mm for AAAs 4.0 to 6.0 cm in diameter, and as much as 7 to 8 mm for AAAs >6.0 cm in diameter.
— Absolute risk for eventual rupture is ≈20% for AAAs >5.0 cm, ≈40% for AAAs >6.0 cm, and >50% for AAAs >7.0 cm in diameter.
— Rupture of an AAA may be associated with death rates as high as 90%.

Atherosclerosis

ICD-9 440; ICD-10 I70.

Mortality—11 841. Total-mention mortality—54 413. 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[37] 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.[38]

Other Diseases of Arteries

ICD-9 442-448; ICD-10 I72-I78.

Mortality—9774. Total-mention mortality—432 484. 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.[4]
- Whites and blacks have a significantly higher incidence than Hispanics and Asians or Pacific Islanders.[4]
- In studies in Worcester, Massachusetts, and Olmsted County, Minnesota, 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.[3]
- 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.[39]
- 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.[40]
- 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%.[41]
- 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.[42]
- 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.[43]
- 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.[44] This is because the risk of VTE remains elevated for ≈4 weeks.

Deep Vein Thrombosis

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

Mortality—2779. Total-mention mortality—11 600.

- 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.[45]
- Death occurs in ≈6% of DVT cases within 1 month of diagnosis.[4]

Kawasaki Disease

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

Mortality—7. Total-mention mortality—11. Hospital discharges—9000, primary plus secondary diagnoses.

- An estimated 5300 cases of KD were diagnosed in 2003. KD occurs more often among boys (63%) and among those of Asian ancestry[37] (Jane W. Newburger and Kimberlee Gauvreau of Children's Hospital of Boston, Mass; written communication, August 15, 2007).
- 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).[46] 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.[47]

Peripheral Arterial Disease

- PAD affects ≈8 million Americans and is associated with significant morbidity and mortality.[48] Recently published data from multiple epidemiological studies demonstrate that ≈8 million men and women ≥40 years of age have PAD.[49] Prevalence increases dramatically with age, and PAD disproportionately affects blacks.[50]
- PAD affects 12% to 20% of Americans ≥65 years of age.[51] 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.[52]

- 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.[48,53] In an older, disabled population of women, however, as many as two thirds of individuals with PAD had no exertional leg symptoms.[54]
- Intermittent claudication is present in <1% of individuals <50 years of age and ≈≥5% of those >80 years of age.[36]
- 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 rose 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.[55]
- The risk factors for PAD are similar to those for CHD, although diabetes and cigarette smoking are particularly strong risk factors for PAD.[36] 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.[56]
- 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.[57] 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.[58] 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.[59,60]
- 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.[61,62]
- 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.[63]
- Data from NHANES 1999–2000 (NCHS) show that high blood levels of lead and cadmium may increase the risk 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.[64]
- Results from NHANES 1999–2000 (NCHS) showed a remarkably high prevalence of PAD among patients with renal insufficiency.[65]
- 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.[49,66]
- Recent studies indicate an association of elevated ankle-brachial index levels with increased risk of all-cause and cardiovascular death.[67,68]
- Among patients with established PAD, higher physical activity levels during daily life are associated with better overall survival rate and a lower risk of death from CVD.[69]
- A cross-sectional, population-based telephone survey of >2500 adults ≥50 years of age, with oversampling of blacks and Hispanics, found that 26% expressed familiarity with PAD. Of these, half were not aware that 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.[70]
- 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).[71]

References

1. Fuster V. *Hurst's The Heart, Arteries and Veins*. 10th ed. New York, NY: McGraw-Hill; 2001.
2. Merrill CT, Nagamine M, Elixhauser A. *Hospital Stays Involving Pulmonary Heart Disease, 2005*. Rockville, Md: Agency for Healthcare Research and Quality; December 2007. HCUP Statistical Brief 43. Available at: http://www.hcup-us.ahrq.gov/reports/statbriefs/sb43.pdf.
3. Goldhaber SZ. Pulmonary embolism. *N Engl J Med*. 1998;339:93–104.
4. White RH. The epidemiology of venous thromboembolism. *Circulation*. 2003; 107(23 suppl 1):I4–I8.
5. Horlander KT, Mannino DM, Leeper KV. Pulmonary embolism mortality in the United States, 1979–1998: an analysis using multiple-cause mortality data. *Arch Intern Med*. 2003;163:1711–1717.
6. Wilson W, Taubert KA, Gewitz M, Lockhart PB, Baddour LM, Levison M, Bolger A, Cabell CH, Takahashi M, Baltimore RS, Newburger JW, Strom BL, Tani LY, Gerber M, Bonow RO, Pallasch T, Shulman ST, Rowley AH, Burns JC, Ferrieri P, Gardner T, Goff D, Durack DT; American Heart Association Rheumatic Fever, Endocarditis, and Kawasaki Disease Committee; American Heart Association Council on Cardiovascular Disease in the Young; American Heart Association Council on Clinical Cardiology; American Heart Association Council on Cardiovascular Surgery and Anesthesia; Quality of Care and Outcomes Research Interdisciplinary Working Group. Prevention of infective endocarditis: guidelines from the American Heart Association: a guideline from the American Heart Association Rheumatic Fever, Endocarditis, and Kawasaki Disease Committee, Council on Cardiovascular Disease in the Young, and the Council on Clinical Cardiology, Council on Cardiovascular Surgery and Anesthesia, and the Quality of Care and Outcomes Research Interdisciplinary Working Group. *Circulation*. 2007;116:1736–1754.
7. Fowler VG, Scheld WM, Bayer AS. Endocarditis and intravascular infections. In: Mandell GL, Douglas RG, Bennett JE, Dolin R, eds. *Principles and Practices of Infectious Diseases*. 6th ed. New York, NY: Elsevier/Churchill Livingstone; 2005:975–1021.

8. Nkomo VT, Gardin JM, Skelton TN, Gottdiener JS, Scott CG, Enriquez-Sarano M. Burden of valvular heart diseases: a population-based study. *Lancet*. 2006;368:1005–1011.
9. Singh JP, Evans JC, Levy D, Larson MG, Freed LA, Fuller DL, Lehman B, Benjamin EJ. Prevalence and clinical determinants of mitral, tricuspid, and aortic regurgitation (the Framingham Heart Study) [published correction appears in *Am J Cardiol*. 1999;84:1143]. *Am J Cardiol*. 1999;83:897–902.
10. Otto CM, Lind BK, Kitzman DW, Gersh BJ, Siscovick DS. Association of aortic-valve sclerosis with cardiovascular mortality and morbidity in the elderly. *N Engl J Med*. 1999;341:142–147.
11. Stewart BF, Siscovick D, Lind BK, Gardin JM, Gottdiener JS, Smith VE, Kitzman DW, Otto CM. Clinical factors associated with calcific aortic valve disease: Cardiovascular Health Study. *J Am Coll Cardiol*. 1997;29:630–634.
12. Freed LA, Levy D, Levine RA, Larson MG, Evans JC, Fuller DL, Lehman B, Benjamin EJ. Prevalence and clinical outcome of mitral-valve prolapse. *N Engl J Med*. 1999;341:1–7.
13. Nerheim P, Birger-Botkin S, Piracha L, Olshansky B. Heart failure and sudden death in patients with tachycardia-induced cardiomyopathy and recurrent tachycardia. *Circulation*. 2004;110:247–252.
14. Lipshultz SE, Sleeper LA, Towbin JA, Lowe AM, Orav EJ, Cox GF, Lurie PR, McCoy KL, McDonald MA, Messere JE, Colan SD. The incidence of pediatric cardiomyopathy in two regions of the United States. *N Engl J Med*. 2003;348:1647–1655.
15. Maron BJ, Shirani J, Poliac LC, Mathenge R, Roberts WC, Mueller FO. Sudden death in young competitive athletes: clinical, demographic, and pathological profiles. *JAMA*. 1996;276:199–204.
16. Maron BJ, McKenna WJ, Danielson GK, Kappenberger LJ, Kuhn HJ, Seidman CE, Shah PM, Spencer WH 3rd, Spirito P, Ten Cate FJ, Wigle ED; Task Force on Clinical Expert Consensus Documents, American College of Cardiology; Committee for Practice Guidelines, European Society of Cardiology. American College of Cardiology/European Society of Cardiology clinical expert consensus document on hypertrophic cardiomyopathy: a report of the American College of Cardiology Foundation Task Force on Clinical Expert Consensus Documents and the European Society of Cardiology Committee for Practice Guidelines. *J Am Coll Cardiol.* 2003;42:1687–1713.
17. Colan SD, Lipshultz SE, Lowe AM, Sleeper LA, Messere J, Cox GF, Lurie PR, Orav EJ, Towbin JA. Epidemiology and cause-specific outcome of hypertrophic cardiomyopathy in children: findings from the Pediatric Cardiomyopathy Registry. *Circulation*. 2007;115:773–781.
18. Towbin JA, Lowe AM, Colan SD, Sleeper LA, Orav EJ, Clunie S, Messere J, Cox GF, Lurie PR, Hsu D, Canter C, Wilkinson JD, Lipshultz SE. Incidence, causes, and outcomes of dilated cardiomyopathy in children. *JAMA*. 2006;296:1867–1876.
19. Centers for Medicare & Medicaid Services. *Health Care Financing Review: Medicare & Medicaid Statistical Supplement*. Table 5.5: Discharges, Total Days of Care, and Program Payments for Medicare Beneficiaries Discharged from Short-Stay Hospitals, by Principal Diagnoses Within Major Diagnostic Classifications (MDCs): Calendar Year 2006. Baltimore, Md: Centers for Medicare and Medicaid Services; 2005. Available at: http://www.cms.hhs.gov/MedicareMedicaidStatSupp/. Accessed August 28, 2008.
20. Go AS, Hylek EM, Phillips KA, Chang Y, Henault LE, Selby JV, Singer DE. Prevalence of diagnosed atrial fibrillation in adults: national implications for rhythm management and stroke prevention: the AnTicoagulation and Risk Factors in Atrial Fibrillation (ATRIA) Study. *JAMA*. 2001;285:2370–2375.
21. Go AS. The epidemiology of atrial fibrillation in elderly persons: the tip of the iceberg. *Am J Geriatr Cardiol*. 2005;14:56–61. Review.
22. Lloyd-Jones DM, Wang TJ, Leip EP, Larson MG, Levy D, Vasan RS, D'Agostino RB, Massaro JM, Beiser A, Wolf PA, Benjamin EJ. Lifetime risk for development of atrial fibrillation: the Framingham Heart Study. *Circulation*. 2004;110:1042–1046.
23. Ruo B, Capra AM, Jensvold NG, Go AS. Racial variation in the prevalence of atrial fibrillation among patients with heart failure: the Epidemiology, Practice, Outcomes, and Costs of Heart Failure (EPOCH) study. *J Am Coll Cardiol*. 2004;43:429–435.
24. Khairallah F, Ezzedine R, Ganz LI, London B, Saba S. Epidemiology and determinants of outcome of admissions for atrial fibrillation in the United States from 1996 to 2001. *Am J Cardiol*. 2004;94:500–504.
25. Centers for Disease Control and Prevention (CDC). Atrial fibrillation as a contributing cause of death and Medicare hospitalization–United States, 1999. *MMWR Morb Mortal Wkly Rep*. 2003;52:128, 130–131.
26. Miyasaka Y, Barnes ME, Gersh BJ, Cha SS, Bailey KR, Abhayaratna WP, Seward JB, Tsang TS. Secular trends in incidence of atrial fibrillation in Olmsted County, Minnesota, 1980 to 2000, and implications on the projections for future prevalence [published correction appears in *Circulation*. 2006;114:e498]. *Circulation*. 2006;114:119–125.
27. Miyasaka Y, Barnes ME, Bailey KR, Cha SS, Gersh BJ, Seward JB, Tsang TS. Mortality trends in patients diagnosed with first atrial fibrillation: a 21-year community-based study. *J Am Coll Cardiol*. 2007;49:986–992.
28. Lakshminarayan K, Solid CA, Collins AJ, Anderson DC, Herzog CA. Atrial fibrillation and stroke in the general Medicare population: a 10-year perspective (1992 to 2002). *Stroke*. 2006;37:1969–1974.
29. Wolf PA, Abbott RD, Kannel WB. Atrial fibrillation as an independent risk factor for stroke: the Framingham Study. *Stroke*. 1991;22:983–988.
30. Hart RG, Pearce LA, Rothbart RM, McAnulty JH, Asinger RW, Halperin JL; Stroke Prevention in Atrial Fibrillation Investigators. Stroke with intermittent atrial fibrillation: incidence and predictors during aspirin therapy. *J Am Coll Cardiol*. 2000;35:183–187.
31. Penado S, Cano M, Acha O, Hernandez JL, Riancho JA. Atrial fibrillation as a risk factor for stroke recurrence. *Am J Med*. 2003;114:206–210.
32. Dulli DA, Stanko H, Levine RL. Atrial fibrillation is associated with severe acute ischemic stroke. *Neuroepidemiology*. 2003;22:118–123.
33. Halligan SC, Gersh BJ, Brown RD Jr, Rosales AG, Munger TM, Shen WK, Hammill SC, Friedman PA. The natural history of lone atrial flutter. *Ann Intern Med*. 2004;140:265–268.
34. Seidl K, Hauer B, Schwick NG, Zellner D, Zahn R, Senges J. Risk of thromboembolic events in patients with atrial flutter. *Am J Cardiol*. 1998;82:580–583.
35. Bunch TJ, White RD, Friedman PA, Kottke TE, Wu LA, Packer DL. Trends in treated ventricular fibrillation out-of-hospital cardiac arrest: a 17-year population-based study. *Heart Rhythm*. 2004;1:255–259.
36. Hirsch AT, Haskal ZJ, Hertzer NR, Bakal CW, Creager MA, Halperin JL, Hiratzka LF, Murphy WR, Olin JW, Puschett JB, Rosenfield KA, Sacks D, Stanley JC, Taylor LM Jr, White CJ, White J, White RA, Antman EM, Smith SC Jr, Adams CD, Anderson JL, Faxon DP, Fuster V, Gibbons RJ, Hunt SA, Jacobs AK, Nishimura R, Ornato JP, Page RL, Riegel B; American Association for Vascular Surgery; Society for Vascular Surgery; Society for Cardiovascular Angiography and Interventions; Society for Vascular Medicine and Biology; Society of Interventional Radiology; ACC/AHA Task Force on Practice Guidelines Writing Committee to Develop Guidelines for the Management of Patients With Peripheral Arterial Disease; American Association of Cardiovascular and Pulmonary Rehabilitation; National Heart, Lung, and Blood Institute; Society for Vascular Nursing; TransAtlantic Inter-Society Consensus; Vascular Disease Foundation. ACC/AHA 2005 Practice Guidelines for the management of patients with peripheral arterial disease (lower extremity, renal, mesenteric, and abdominal aortic): a collaborative report from the American Association for Vascular Surgery/Society for Vascular Surgery, Society for Cardiovascular Angiography and Interventions, Society for Vascular Medicine and Biology, Society of Interventional Radiology, and the ACC/AHA Task Force on Practice Guidelines (Writing Committee to Develop Guidelines for the Management of Patients With Peripheral Arterial Disease): endorsed by the American Association of Cardiovascular and Pulmonary Rehabilitation; National Heart, Lung, and Blood Institute; Society for Vascular Nursing; TransAtlantic Inter-Society Consensus; and Vascular Disease Foundation. *Circulation*. 2006;113:e463–e654.
37. Healthcare Cost and Utilization Project. Overview of the Kids' Inpatient Database (KID). Available at: http://www.hcup-us.ahrq.gov/kidoverview.jsp. Accessed October 24, 2007.
38. Steg PG, Bhatt DL, Wilson PW, D'Agostino R Sr, Ohman EM, Rother J, Liau CS, Hirsch AT, Mas JL, Ikeda Y, Pencina MJ, Goto S; REACH Registry Investigators. One-year cardiovascular event rates in outpatients with atherothrombosis. *JAMA*. 2007;297:1197–1206.
39. Tsai AW, Cushman M, Rosamond WD, Heckbert SR, Tracy RP, Aleksic N, Folsom AR. Coagulation factors, inflammation markers, and venous thromboembolism: the Longitudinal Investigation of Thromboembolism Etiology (LITE). *Am J Med*. 2002;113:636–642.
40. Heit JA. Venous thromboembolism epidemiology: implications for prevention and management. *Semin Thromb Hemost*. 2002;28(suppl 2):3–13.
41. Cushman M, Tsai AW, White RH, Heckbert SR, Rosamond WD, Enright P, Folsom AR. Deep vein thrombosis and pulmonary embolism in two cohorts: the Longitudinal Investigation of Thromboembolism Etiology. *Am J Med*. 2004;117:19–25.

42. Heit JA, Kobbervig CE, James AH, Petterson TM, Bailey KR, Melton LJ 3rd. Trends in the incidence of venous thromboembolism during pregnancy or postpartum: a 30-year population-based study. *Ann Intern Med*. 2005;143:697–706.
43. Steffen LM, Folsom AR, Cushman M, Jacobs DR Jr, Rosamond WD. Greater fish, fruit, and vegetable intakes are related to lower incidence of venous thromboembolism: the Longitudinal Investigation of Thromboembolism Etiology. *Circulation*. 2007;115:188–195.
44. World Health Organization. Study results released on travel and blood clots: WHO project finds VTE risk higher after long travel, but still relatively low [press release]. June 29, 2007. Available at: http://www.who.int/mediacentre/news/releases/2007/pr35/en/print.html. Accessed October 1, 2007.
45. Fowkes FJ, Price JF, Fowkes FG. Incidence of diagnosed deep vein thrombosis in the general population: systematic review. *Eur J Vasc Endovasc Surg*. 2003;25:1–5.
46. Newburger JW, Takahashi M, Gerber MA, Gewitz MH, Tani LY, Burns JC, Shulman ST, Bolger AF, Ferrieri P, Baltimore RS, Wilson WR, Baddour LM, Levison ME, Pallasch TJ, Falace DA, Taubert KA, Committee on Rheumatic Fever, Endocarditis and Kawasaki Disease; Council on Cardiovascular Disease in the Young, American Heart Association; American Academy of Pediatrics. Diagnosis, treatment, and long-term management of Kawasaki disease: a statement for health professionals from the Committee on Rheumatic Fever, Endocarditis and Kawasaki Disease, Council on Cardiovascular Disease in the Young, American Heart Association. *Circulation*. 2004;110:2747–2771.
47. Chang RK. The incidence of Kawasaki disease in the United States did not increase between 1988 and 1997. *Pediatrics*. 2003;111(5 pt 1):1124–1125.
48. Hirsch AT, Criqui MH, Treat-Jacobson D, Regensteiner JG, Creager MA, Olin JW, Krook SH, Hunninghake DB, Comerota AJ, Walsh ME, McDermott MM, Hiatt WR. Peripheral arterial disease detection, awareness, and treatment in primary care. *JAMA*. 2001;286:1317–1324.
49. Allison MA, Ho E, Denenberg J, Langer R, Newman A, Fabsitz R, Criqui M. Ethnic-specific prevalence of peripheral arterial disease in the United States. *Am J Prev Med*. 2007;32:328–333.
50. Selvin E, Erlinger TP. Prevalence of and risk factors for peripheral arterial disease in the United States: results from the National Health and Nutrition Examination Survey, 1999–2000. *Circulation*. 2004;110:738–743.
51. Ostchega Y, Paulose-Ram R, Dillon CF, Gu Q, Hughes JP. Prevalence of peripheral arterial disease and risk factors in persons aged 60 and older: data from the National Health and Nutrition Examination Survey 1999–2004. *J Am Geriatr Soc*. 2007;55:583–589.
52. Bhatt DL, Steg PG, Ohman EM, Hirsch AT, Ikeda Y, Mas JL, Goto S, Liau CS, Richard AJ, Rother J, Wilson PW; REACH Registry Investigators. International prevalence, recognition, and treatment of cardiovascular risk factors in outpatients with atherothrombosis. *JAMA*. 2006;295:180–189.
53. McDermott MM, Greenland P, Liu K, Guralnik JM, Criqui MH, Dolan NC, Chan C, Celic L, Pearce WH, Schneider JR, Sharma L, Clark E, Gibson D, Martin GJ. Leg symptoms in peripheral arterial disease: associated clinical characteristics and functional impairment. *JAMA*. 2001;286:1599–1606.
54. McDermott MM, Fried L, Simonsick E, Ling S, Guralnik JM. Asymptomatic peripheral arterial disease is independently associated with impaired lower extremity functioning: the women's health and aging study [published correction appears in *Circulation*. 2001;104:504]. *Circulation*. 2000;101:1007–1012.
55. Murabito JM, Evans JC, D'Agostino RB Sr, Wilson PW, Kannel WB. Temporal trends in the incidence of intermittent claudication from 1950 to 1999. *Am J Epidemiol*. 2005;162:430–437.
56. Norgren L, Hiatt WR, Dormandy JA, Nehler MR, Harris KA, Fowkes FG; TASC II Working Group. Inter-Society Consensus for the Management of Peripheral Arterial Disease (TASC II). *J Vasc Surg*. 2007;45(suppl S):S5–S67.
57. Ridker PM, Cushman M, Stampfer MJ, Tracy RP, Hennekens CH. Plasma concentration of C-reactive protein and risk of developing peripheral vascular disease. *Circulation*. 1998;97:425–428.
58. Pradhan AD, Shrivastava S, Cook NR, Rifai N, Creager MA, Ridker PM. Symptomatic peripheral arterial disease in women: nontraditional biomarkers of elevated risk. *Circulation*. 2008;117:823–831.
59. Owens CD, Ridker PM, Belkin M, Hamdan AD, Pomposelli F, Logerfo F, Creager MA, Conte MS. Elevated C-reactive protein levels are associated with postoperative events in patients undergoing lower extremity vein bypass surgery. *J Vasc Surg*. 2007;45:2–9.
60. Vidula H, Tian L, Liu K, Criqui MH, Ferrucci L, Pearce WH, Greenland P, Green D, Tan J, Garside DB, Guralnik J, Ridker PM, Rifai N, McDermott MM. Biomarkers of inflammation and thrombosis as predictors of near-term mortality in patients with peripheral arterial disease: a cohort study. *Ann Intern Med*. 2008;148:85–93.
61. McDermott MM, Greenland P, Liu K, Guralnik JM, Celic L, Criqui MH, Chan C, Martin GJ, Schneider J, Pearce WH, Taylor LM, Clark E. The ankle brachial index is associated with leg function and physical activity: the Walking and Leg Circulation Study [published correction appears in *Ann Intern Med.* 2003;139:306]. *Ann Intern Med*. 2002;136:873–883.
62. McDermott MM, Liu K, Greenland P, Guralnik JM, Criqui MH, Chan C, Pearce WH, Schneider JR, Ferrucci L, Celic L, Taylor LM, Vonesh E, Martin GJ, Clark E. Functional decline in peripheral arterial disease: associations with the ankle brachial index and leg symptoms. *JAMA*. 2004;292:453–461.
63. Heald CL, Fowkes FG, Murray GD, Price JF; Ankle Brachial Index Collaboration. Risk of mortality and cardiovascular disease associated with the ankle-brachial index: systematic review. *Atherosclerosis*. 2006;189:61–69.
64. Navas-Acien A, Selvin E, Sharrett AR, Calderon-Aranda E, Silbergeld E, Guallar E. Lead, cadmium, smoking, and increased risk of peripheral arterial disease. *Circulation*. 2004;109: 3196–3201.
65. O'Hare AM, Glidden DV, Fox CS, Hsu CY. High prevalence of peripheral arterial disease in persons with renal insufficiency: results from the National Health and Nutrition Examination Survey 1999–2000. *Circulation.* 2004;109:320–323.
66. Criqui MH, Vargas V, Denenberg JO, Ho E, Allison M, Langer RD, Gamst A, Bundens WP, Fronek A. Ethnicity and peripheral arterial disease: the San Diego Population Study. *Circulation*. 2005;112:2703–2707.
67. O'Hare AM, Katz R, Shlipak MG, Cushman M, Newman AB. Mortality and cardiovascular risk across the ankle-arm index spectrum: results from the Cardiovascular Health Study. *Circulation*. 2006;113:388–393.
68. Resnick HE, Lindsay RS, McDermott MM, Devereux RB, Jones KL, Fabsitz RR, Howard BV. Relationship of high and low ankle brachial index to all-cause and cardiovascular disease mortality: the Strong Heart Study. *Circulation*. 2004;109:733–739.
69. Garg PK, Tian L, Criqui MH, Liu K, Ferrucci L, Guralnik JM, Tan J, McDermott MM. Physical activity during daily life and mortality in patients with peripheral arterial disease. *Circulation*. 2006;114:242–248.
70. Hirsch AT, Murphy TP, Lovell MB, Twillman G, Treat-Jacobson D, Harwood EM, Mohler ER 3rd, Creager MA, Hobson RW 2nd, Robertson RM, Howard WJ, Schroeder P, Criqui MH; for the Peripheral Arterial Disease Coalition. Gaps in public knowledge of peripheral arterial disease: the First National PAD Public Awareness Survey. *Circulation*. 2007;116:2086–2094.
71. Wilson AM, Kimura E, Harada RK, Nair N, Narasimhan B, Meng XY, Zhang F, Beck KR, Olin JW, Fung ET, Cooke JP. Beta2-microglobulin as a biomarker in peripheral arterial disease: proteomic profiling and clinical studies. *Circulation*. 2007;116:1396–1403.

Table 9-1. Rheumatic Fever/Rheumatic Heart Disease

Population Group	Mortality, 2005 All Ages*	Hospital Discharges, 2006 All Ages
Both sexes	3365	59 000
Males	1044 (31.0%)†	22 000
Females	2321 (69.0%)†	36 000
White males	926	. . .
White females	2103	. . .
Black males	81	. . .
Black females	146	. . .

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. 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 vs females.

10. Risk Factor: Smoking/Tobacco Use

See Tables 10-1 and 10-2 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 tobacco 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 2006, the percentage of high school seniors who reported smoking in the previous month decreased 29.2%. Smoking decreased by 16.4% in male students, 39.8% in female students, 20.3% in whites, and 56.3% in blacks.[2]
- Among youths 12 to 17 years of age in 2006, 3.3 million (12.9%) used a tobacco product in the past month, and 2.6 million (10.4%) used cigarettes. The rate of cigarette use in the past month declined from 13.0% in 2002 to 10.4% in 2006. Cigar use in the past month declined to 4.1% in 2006 from 4.8% in 2004. Smokeless tobacco use was reported by 2.4% of youths in 2006, higher than any estimates since 2002.[3]
- Results from the 2007 Monitoring the Future survey of the NIH showed a considerable drop in lifetime, past-month, and daily smoking among eighth graders. From 2006 to 2007, it dropped from 4% to 3%, down from its 10.4% peak in 1996.[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.

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
GED	General Educational Development
HF	heart failure
MI	myocardial infarction
NIH	National Institutes of Health
NCHS	National Center for Health Statistics
NH	non-Hispanic
NHANES	National Health and Nutrition Examination Survey
NHIS	National Health Interview Survey
SHS	secondhand smoke
YRBS	Youth Risk Behavior Surveillance

Adults

- From 1965 to 2006, smoking in the United States declined by 50.4% among people ≥18 years of age (NCHS).[2]
- In 2007, among Americans ≥18 years of age, 22.0% of men and 17.5% of women were cigarette smokers, putting them at increased risk of heart attack and stroke.[6]
- Rates of use of any tobacco product in 2005, among persons ≥12 years of age, were 31.2% for non-Hispanic whites only, 28.4% for non-Hispanic blacks only, 41.7% for non-Hispanic American Indians or Alaska Natives only, 14.6% for non-Hispanic Asians only, and 24.5% for Hispanics or Latinos of any race (NCHS).[2]
- In 2006, non-Hispanic American Indian or Alaska Native adults ≥18 years of age were more likely (32.4%) to be current smokers than were non-Hispanic white adults (21.9%), non-Hispanic black adults (23.0%), and non-Hispanic Asian adults (10.4%).[2]
- BRFSS/CDC 2007 data showed that among adults ≥18 years of age, the median percentage of current smokers among the states was 19.8%. The highest percentage was in Kentucky (28.2%), and the lowest was in Utah (11.7%).[7]
- According to combined data from 2005–2006, among women 15 to 44 years of age, rates of past-month cigarette smoking were lower for pregnant (16.5%) women than for nonpregnant (29.5%) women; however, among those 15 to 17 years of age, the smoking rate for pregnant women was higher than for nonpregnant women (23.1% versus 17.1%).[3]
- Between 1965 and 2004–2005, the age-adjusted prevalence of noninstitutionalized women ≥65 years of age who were current smokers increased from 8% in 1965 to 13% in the mid-1980s and then decreased back to 8% in 2004–2005. In 2004–2005, 28% of women and 49% of men ≥65 years of age (age adjusted) had previously smoked cigarettes.[8]
- According to 2004–2006 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.[9]

Incidence

- In 2007, 1.0 million people started smoking cigarettes daily within the prior 12 months. Of these daily smokers, 40.7%, or 0.4 million (an average of ≈1100 initiates per day), were <18 years of age when they started smoking daily.[3] In 2007, over 3500 people initiated cigarette smoking before age 18.[10]
- Data from 2002–2004 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.[11]

- 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.[12]

Mortality

- From 1997 to 2001, an estimated 438 000 Americans died each year of smoking-related illnesses, and ≈38 000 of these deaths were from SHS. A total of 34.7% of these deaths were related to CVD.[13,14]
- Each year from 1997 to 2001, smoking caused 3.3 million years of potential life lost for men and 2.2 million years for women.[14]
- From 1997 to 2001, smoking during pregnancy resulted in an estimated 910 infant deaths annually.[14]
- Cigarette smoking kills an estimated 178 000 women in the United States annually. Of these, ≈40 000 deaths are from heart disease.[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 September 2006 stated that:
  - — Cigarette smoking results in a 2- to 3-fold increased risk of dying of CHD.
  - — On average, adults who smoke cigarettes die 14 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 1988–1994 to 1999–2004 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:
  - — Levels of cotinine, a biomarker of SHS, fell by 70% from 1988–1991 to 2001–2002.
  - — Almost 60% of US children 3 to 11 years of age, or almost 22 million, are exposed to SHS.
  - — Nonsmokers who are exposed to SHS at home or at work increase their risk of developing heart disease 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 a heart attack.
- Healthcare costs associated with exposure to SHS average $10 billion annually.[13]

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.[23]
- The CDC "Health Effects of Cigarette Smoking" fact sheet[24] 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 risk of abdominal aortic aneurysm.

- According to the 2007 National Healthcare Quality Report, in 2004, 63.7% of smokers with routine office visits during the preceding year reported that they had been advised to quit, an increase from 61.9% in 2000. Smokers 18 to 44 years of age were less likely than the other age groups to be advised to quit smoking.[25]

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,[26] based on the results of the 2005 National Survey on Drug Use and Health, indicate:
  - — 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 associated with smoking ($97 billion) total an estimated $193 billion per year.[13]

References

1. Eaton DK, Kann L, Kinchen S, Shanklin S, Ross J, Hawkins J, Harris WA, Lowry R, McManus T, Chyen D, Lim C, Brener ND, Wechsler H; Centers for Disease Control and Prevention (CDC). Youth risk behavior surveillance–United States, 2007. *MMWR Surveill Summ*. 2008;57:1–131.
2. National Center for Health Statistics. *Health, United States, 2007 With Chartbook on Trends in the Health of Americans*. Hyattsville, Md: National Center for Health Statistics; 2007. Available at: http://www.cdc.gov/nchs/hus.htm. Accessed May 29, 2008.
3. Substance Abuse and Mental Health Services Administration. *Results From the 2006 National Survey on Drug Use and Health: National Findings*. Rockville, Md: US Dept of Health and Human Services, Office of Applied Studies; 2007. DHHS publication SMA 07-4293; NSDUH series H-32.
4. National Institute on Drug Abuse, University of Michigan. *Monitoring the Future Study, 2007*. Bethesda, Md: National Institute on Drug Abuse; 2007. Table 1: Trends on cigarette smoking and smokeless tobacco. Available at: http://www.monitoringthefuture.org/data/07data/pr07cig1.pdf. Accessed June 10, 2008.
5. Centers for Disease Control and Prevention. YRBBS: national trends in risk behaviors. Available at: http://www.cdc.gov/HealthyYouth/yrbs/trends.htm. Accessed June 10, 2008.
6. Pleis JR, Lucus JW. Summary health statistics for U.S. adults: National Health Interview Survey, 2007. *Vital Health Stat 10*. In press.
7. Centers for Disease Control and Prevention (CDC). Prevalence data, tobacco use. In: *Behavioral Risk Factor Surveillance System Survey Data*. Atlanta, Ga: US Dept of Health and Human Services, Centers for Disease Control and Prevention; 2007. Available at: http://www.cdc.gov/brfss/index.htm. Accessed April 28, 2008.
8. Robinson K. *Trends in Health Status and Health Care Use Among Older Women: Aging Trends, No. 7*. Hyattsville, Md: National Center for Health Statistics; 2007.
9. Barnes PM, Adams PF, Powell-Griner E. *Health Characteristics of the Asian Adult Population: United States, 2004–2006: Advance Data From Vital and Health Statistics; No. 394*. Hyattsville, Md: National Center for Health Statistics; 2008.
10. Substance Abuse and Mental Health Services Administration. *Results From the 2007 National Survey on Drug Use and Health: National Findings*. Rockville, Md: US Department of Health and Human Services, Office of Applied Studies; 2008. DHHS publication SMA 08-4343; NSDUH Series H-34.
11. Centers for Disease Control and Prevention (CDC). Racial/ethnic differences among youths in cigarette smoking and susceptibility to start smoking: United States, 2002–2004. *MMWR Morb Mortal Wkly Rep*. 2006;55:1275–1277.
12. US Department of Health and Human Services. *Preventing Tobacco Use Among Young People: A Report of the Surgeon General: Executive Summary*. Atlanta, Ga: US Dept of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health; 1994.
13. Centers for Disease Control and Prevention (CDC). Smoking & tobacco use: fast facts. Available at: http://www.cdc.gov/tobacco/data_statistics/fact_sheets/fast_facts/index.htm#toll. Accessed September 16, 2008.
14. Centers for Disease Control and Prevention (CDC). Annual smoking-attributable mortality, years of potential life lost, and productivity losses: United States, 1997–2001. *MMWR Morb Mortal Wkly Rep*. 2005;54:625–628.
15. US Department of Health and Human Services. *Fact Sheet: Women and Tobacco*. Atlanta, Ga: US Dept of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health; updated November 2006. Available at: http://www.cdc.gov/tobacco/data_statistics/fact_sheets/populations/women_tobacco.htm. Accessed March 14, 2008.
16. US Department of Health and Human Services. *2004 Surgeon General's Report—The Health Consequences of Smoking*. Atlanta, Ga: US Dept of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health; 2004. Available at: www.cdc.gov/tobacco/sgr/sgr_2004/index.htm. Accessed October 29, 2006.
17. Goldenberg I, Jonas M, Tenenbaum A, Boyko V, Matetzky S, Shotan A, Behar S, Reicher-Reiss H; Bezafibrate Infarction Prevention Study Group. Current smoking, smoking cessation, and the risk of sudden cardiac death in patients with coronary artery disease. *Arch Intern Med*. 2003;163:2301–2305.
18. Anthonisen NR, Skeans MA, Wise RA, Manfreda J, Kanner RE, Connett JE; Lung Health Study Research Group. The effects of a smoking cessation intervention on 14.5-year mortality: a randomized clinical trial. *Ann Intern Med*. 2005;142:233–239.
19. US Department of Health and Human Services. *Fact Sheet: Tobacco-Related Mortality*. Atlanta, Ga: US Dept of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health; updated September 2006. Available at: http://www.cdc.gov/tobacco/data_statistics/fact_sheets/health_effects/tobacco_related_mortality.htm. Accessed June 10, 2008.
20. Centers for Disease Control and Prevention (CDC). State-specific prevalence of smoke-free home rules: United States, 1992–2003. *MMWR Morb Mortal Wkly Rep*. 2007;56:501–504.
21. Centers for Disease Control and Prevention (CDC). Disparities in secondhand smoke exposure: United States, 1988–1994 and 1999–2004. *MMWR Morb Mortal Wkly Rep*. 2008;57:744–747.
22. US Department of Health and Human Services. *The Health Consequences of Involuntary Exposure to Tobacco Smoke: A Report of the Surgeon*

General. Atlanta, Ga: US Dept of Health and Human Services, Centers for Disease Control and Prevention, Coordinating Center for Health Promotion, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health; 2006.
23. John U, Meyer C, Hanke M, Völzke H, Schumann A. Relation between awareness of circulatory disorders and smoking in a general population health examination. *BMC Public Health*. 2006;6:48.
24. US Department of Health and Human Services. *Fact Sheet: Health Effects of Cigarette Smoking*. Atlanta, Ga: US Dept of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health; updated January 2008. Available at: http://www.cdc.gov/tobacco/data_statistics/fact_sheets/health_effects/health_effects.htm. Accessed March 13, 2008.
25. Agency for Healthcare Research and Quality. *2007 National Healthcare Quality Disparities Reports*. Rockville, Md: US Dept of Health and Human Services, Agency for Healthcare Research and Quality; February 2008. AHRQ publication 08-0040.
26. US Department of Health and Human Services. *Fact Sheet: Smokeless Tobacco*. Atlanta, Ga: US Dept of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health; updated April 2007. Available at: http://www.cdc.gov/tobacco/data_statistics/fact_sheets/smokeless/index.htm. Accessed October 25, 2007.
27. Centers for Disease Control and Prevention (CDC). Current smoking among adults: United States, 2006. *MMWR Morb Mortal Wkly Rep*. 2007;56:1157–1161.

Table 10-1. Cigarette Smoking

Population Group	Prevalence, 2006 Age ≥18 y	Cost[13]
Both sexes	47 100 000 (20.8%)*	$193 billion per year
Males	26 200 000 (23.5%)*	. . .
Females	20 900 000 (18.1%)*	. . .
White males	23.5%	. . .
White females	18.8%	. . .
Black males	26.1%	. . .
Black females	18.5%	. . .
Hispanic males[27]	20.1%	. . .
Hispanic females[27]	10.1%	. . .
NH Asian-only males[27]	16.8%	. . .
NH Asian-only females[27]	4.6%	. . .
NH American Indian/Alaska Native males[27]	35.6%	. . .
NH American Indian/Alaska Native females[27]	29.0%	. . .

Ellipses indicate data not available.

*Data are for 2006 for Americans ≥18 years of age. NHIS/NCHS percentages applied to 2006 population estimates.[27]

Table 10-2. Cigarette Smoking in the Past Month by Race/Ethnicity, Age, and Sex in the United States, 2006

Demographic Characteristic	Ages 12 to 17 y	Age ≥18 y
Total	10.4	26.7
Male	10.0	30.0
Female	10.7	23.6
NH white	12.4	27.5
NH black	6.0	27.2
NH American Indian or Alaska Native	21.2	40.1
NH Asian	5.2	15.6
Hispanic or Latino	8.2	24.7
NH white male	11.8	NR
NH white female	13.0	NR
NH black male	5.9	NR
NH black female	6.2	NR
Hispanic male	8.6	NR
Hispanic female	7.7	NR

NR indicates data not provided. Values are percentages. Source: Percentage of persons between 12 and 17 years of age and ≥18 years of age reporting cigarette use during the past month, by race/ethnicity and sex.[3]

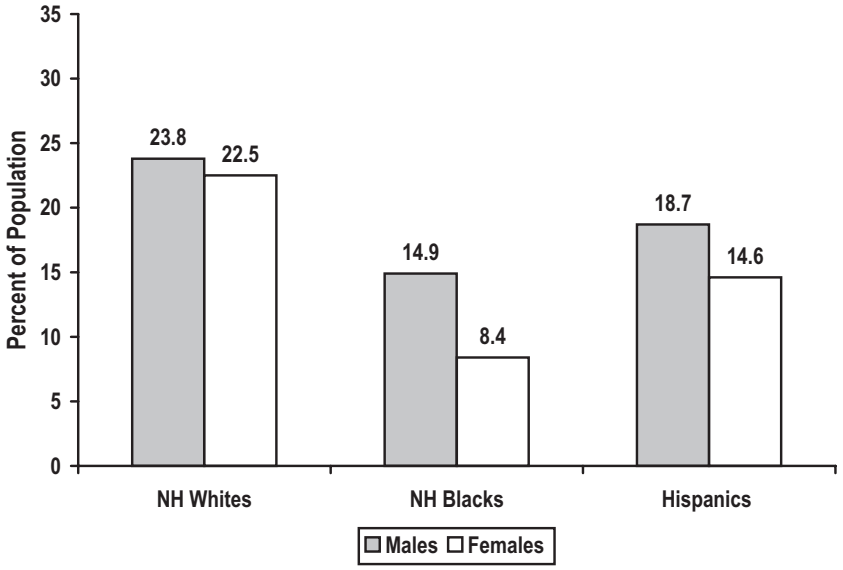


Chart 10-1. Prevalence of students in grades 9 to 12 reporting current cigarette use by sex and race/ethnicity (YRBS, 2007). Source: *MMWR*.[1]

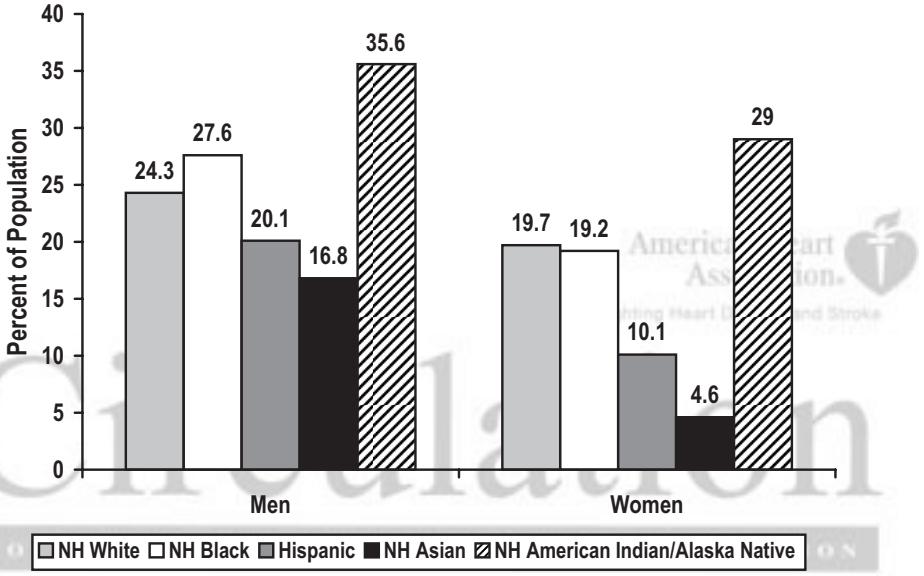


Chart 10-2. Prevalence of current smoking for adults ≥18 years of age by race/ethnicity and sex (NHIS, 2006). Source: *MMWR*.[26]

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 165.8 mg/dL. For boys, it is 165.4 mg/dL; for girls, it is 166.3 mg/dL. The racial/ethnic breakdown is as follows (NHANES 2005–2006, NCHS and NHLBI; unpublished analysis):
  - — For non-Hispanic whites, 166.5 mg/dL for boys and 165.9 mg/dL for girls.
  - — For non-Hispanic blacks, 166.5 mg/dL for boys and 165.1 mg/dL for girls.
  - — For Mexican Americans, 162.3 mg/dL for boys and 160.8 mg/dL for girls.
- Among adolescents 12 to 19 years of age, the mean total blood cholesterol level is 160.4 mg/dL. For boys, it is 156.8 mg/dL; for girls, it is 164.2 mg/dL. The racial/ethnic breakdown is as follows (NHANES 2005–2006, NCHS and NHLBI; unpublished analysis):
  - — For non-Hispanic whites, 154.5 mg/dL for boys and 165.0 mg/dL for girls.
  - — For non-Hispanic blacks, 161.7 mg/dL for boys and 162.8 mg/dL for girls.
  - — For Mexican Americans, 158.2 mg/dL for boys and 163.1 mg/dL for girls.
- Approximately 9.6% of adolescents 12 to 19 years of age have total cholesterol levels ≥200 mg/dL (NHANES 2005–2006, NCHS and NHLBI; unpublished analysis).

Adults

- 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 2001–2004 (NCHS) showed the serum total crude mean cholesterol level in adults ≥20 years of age was 201 mg/dL for men and 203 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, 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
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

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 87.9 mg/dL. For boys, it is 85.4 mg/dL, and for girls, it is 90.5 mg/dL. The racial/ethnic breakdown is as follows (NHANES 2005–2006, NCHS and NHLBI; unpublished analysis):

  — Among non-Hispanic whites, 84.0 mg/dL for boys and 91.2 mg/dL for girls.
  — Among non-Hispanic blacks, 90.2 mg/dL for boys and 91.4 mg/dL for girls.
  — Among Mexican Americans, 87.6 mg/dL for boys and 91.2 mg/dL for girls.

Adults

- The mean level of LDL cholesterol for American adults ≥20 years of age is 115.0 mg/dL (NHANES 2005–2006, 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 2005–2006 (NCHS and NHLBI; unpublished data):

  — Among non-Hispanic whites, mean LDL cholesterol levels were 113.9 mg/dL for men and 116.0 mg/dL for women.
  — Among non-Hispanic blacks, mean LDL cholesterol levels were 115.1 mg/dL for men and 109.7 for women.
  — Among Mexican Americans, mean LDL cholesterol levels were 123.2 mg/dL for men and 110.3 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 56.3 mg/dL. For boys, it is 57.4 mg/dL, and for girls, it is 55.3 mg/dL. The racial/ethnic breakdown is as follows (NHANES 2005–2006, NCHS and NHLBI; unpublished analysis):

  — Among non-Hispanic whites, 57.5 mg/dL for boys and 54.9 mg/dL for girls.
  — Among non-Hispanic blacks, 62.2 mg/dL for boys and 59.2 mg/dL for girls.
  — Among Mexican Americans, 54.5 mg/dL for boys and 51.9 mg/dL for girls.

- Among adolescents 12 to 19 years of age, the mean HDL cholesterol level is 52.2 mg/dL. For boys, it is 49.8 mg/dL, and for girls, it is 54.7 mg/dL. The racial/ethnic breakdown is as follows (NHANES 2005–2006, NCHS and NHLBI; unpublished analysis):

  — Among non-Hispanic whites, 48.2 mg/dL for boys and 53.8 mg/dL for girls.
  — Among non-Hispanic blacks, 55.3 mg/dL for boys and 57.7 mg/dL for girls.
  — Among Mexican Americans, 49.8 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 heart disease and stroke. The mean level of HDL cholesterol for American adults ≥20 years of age is 54.6 mg/dL (NHANES 2005–2006, NCHS and NHLBI; unpublished analysis).
- According to NHANES 2005–2006 (NCHS and NHLBI; unpublished analysis):

  — Among non-Hispanic whites, mean HDL cholesterol levels were 48.5 mg/dL for men and 60.3 mg/dL for women.
  — Among non-Hispanic blacks, mean HDL cholesterol levels were 52.1 mg/dL for men and 62.1 mg/dL for women.
  — Among Mexican Americans, mean HDL cholesterol levels were 47.0 mg/dL for men and 55.5 mg/dL for women.

Triglycerides

Youth

- Among adolescents 12 to 19 years of age, the mean triglyceride level is 90.6 mg/dL. For boys, it is 88.0 mg/dL, and for girls, it is 93.2 mg/dL. The racial/ethnic breakdown is as follows (NHANES 2005–2006, NCHS and NHLBI; unpublished analysis):

  — Among non-Hispanic whites, 93.6 mg/dL for boys and 94.2 mg/dL for girls.

— Among non-Hispanic blacks, 68.0 mg/dL for boys and 67.4 mg/dL for girls.
— Among Mexican Americans, 87.9 mg/dL for boys and 94.8 mg/dL for girls.

Adults

- A triglyceride level >150 mg/dL in adults is considered elevated and is a risk factor for heart disease and stroke. The mean level of triglycerides for American adults ≥18 years of age is 146.0 mg/dL (NHANES 2005–2006, NCHS and NHLBI; unpublished analysis).

  — Among men, the mean triglyceride level is 157.7 mg/dL (NHANES 2005–2006, NCHS and NHLBI; unpublished analysis).

    ○ 163.8 mg/dL for white men.
    ○ 121.0 mg/dL for black men.
    ○ 165.2 mg/dL for Mexican American men.

  — Among women, the mean triglyceride level is 135.0 mg/dL.

    ○ 138.5 mg/dL for white women.
    ○ 104.6 mg/dL for black women.
    ○ 155.6 mg/dL for Mexican American women.

References

1. Centers for Disease Control and Prevention. *Behavioral Risk Factor Surveillance System: Prevalence and Trends Data.* Atlanta, Ga: US Department of Health and Human Services, Centers for Disease Control and Prevention; 2007. Available at: http://apps.nccd.cdc.gov/brfss/index.asp. Accessed September 15, 2008.
2. Centers for Disease Control and Prevention (CDC). State-specific cholesterol screening trends: United States, 1991–1999. *MMWR Morb Mortal Wkly Rep*. 2000;49:750–755.
3. Centers for Disease Control and Prevention (CDC). Disparities in screening for and awareness of high blood cholesterol: United States, 1999–2002. *MMWR Morb Mortal Wkly Rep*. 2005;54:117–119.
4. Carroll MD, Lacher DA, Sorlie PD, Cleeman JI, Gordon DJ, Wolz M, Grundy SM, Johnson CL. Trends in serum lipids and lipoproteins of adults, 1960–2002. *JAMA*. 2005;294:1773–1781.
5. National Center for Health Statistics. *Health, United States, 2007: With Chartbook on Trends in the Health of Americans.* Hyattsville, Md: National Center for Health Statistics; 2007. Available at: http://www.cdc.gov/nchs/hus.htm. Accessed September 15, 2008.
6. Arnett DK, Jacobs DR Jr, Luepker RV, Blackburn H, Armstrong C, Claas SA. Twenty-year trends in serum cholesterol, hypercholesterolemia, and cholesterol medication use: the Minnesota Heart Survey, 1980–1982 to 2000–2002. *Circulation*. 2005;112:3884–3891.
7. Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion. Behavioral Risk Factor Surveillance System: Prevalence Data: Cholesterol Awareness. Available at: http://apps.nccd.cdc.gov/brfss/page.asp?yr=2007&state=All&cat=CA#CA. Accessed April 28, 2008.
8. Schober SE, Carroll MD, Lacher DA, Hirsch R. High serum total cholesterol: an indicator for monitoring cholesterol lowering efforts: U.S. adults, 2005–2006. NCHS Data Brief No. 2. Hyattsville, Md: National Center for Health Statistics; December 2007.
9. National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. *Circulation*. 2002;106:3143–3421.
10. Hyre AD, Muntner P, Menke A, Raggi P, He J. Trends in ATP-III-defined high blood cholesterol prevalence, awareness, treatment and control among U.S. adults. *Ann Epidemiol*. 2007;17:548–555.
11. Centers for Disease Control and Prevention (CDC). Trends in cholesterol screening and awareness of high blood cholesterol: United States, 1991–2003. *MMWR Morb Mortal Wkly Rep*. 2005;54:865–870.

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*	98 600 000 (45.1%)	34 400 000 (15.7%)	71 800 000 (32.8%)	33 900 000 (15.5%)
Males*	45 000 000 (42.6%)	14 600 000 (13.8%)	35 800 000 (33.8%)	26 300 000 (24.9%)
Females*	53 600 000 (47.1%)	19 800 000 (17.3%)	36 000 000 (31.7%)	7 500 000 (6.7%)
NH white males, %	42.1	14.3	31.0	24.9
NH white females, %	47.7	18.1	33.7	6.5
NH black males, %	35.6	7.9	36.2	13.5
NH black females, %	41.4	13.4	27.4	6.1
Mexican-American males, %	52.1	17.5	45.0	30.6
Mexican-American females, %	48.0	14.5	30.3	10.5
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	. . .	. . .

NH indicates non-Hispanic. Ellipses (. . .) indicate data not available. 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 (2005–2006), NCHS, and NHLBI. Estimates from NHANES 2005–2006 (NCHS) applied to 2006 population estimates.

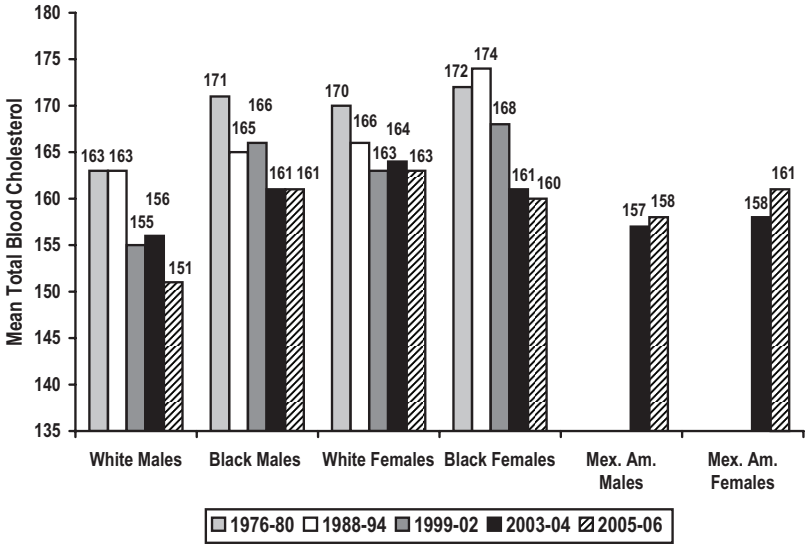


Chart 11-1. Trends in mean total serum cholesterol among adolescents 12 to 17 years of age by race, sex, and survey (NHANES: 1976–1980, 1988–1994, 1999–2002, 2003–2004, and 2005–2006). Source: NCHS and NHLBI. Mex. Am. indicates Mexican American.

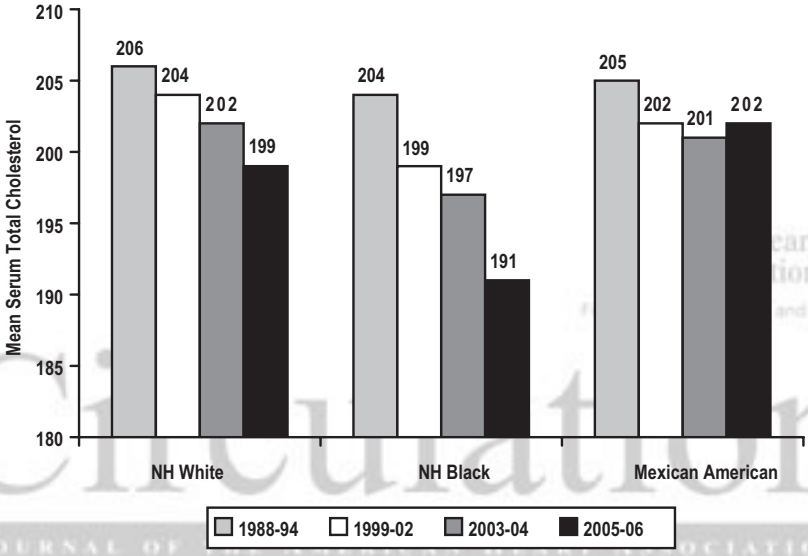


Chart 11-2. Trends in mean total serum cholesterol among adults by race and survey (NHANES: 1988–1994, 1999–2002, 2003–2004, and 2005–2006). Source: NCHS and NHLBI. NH indicates non-Hispanic.

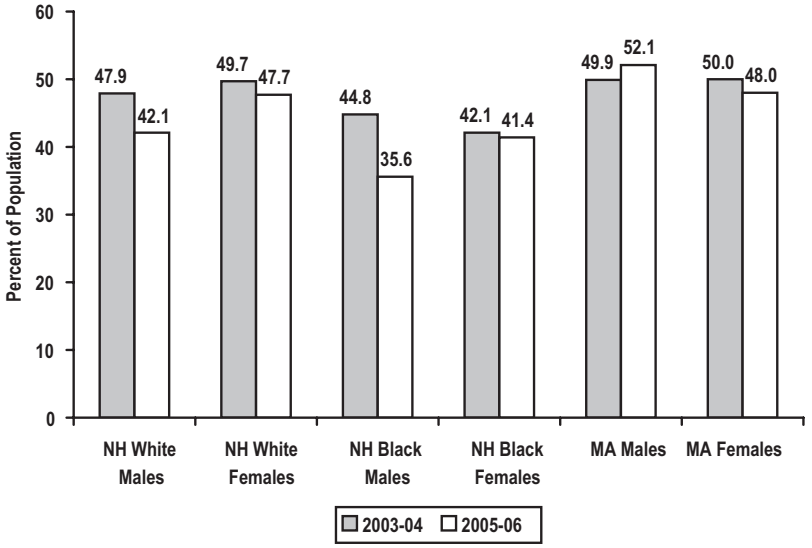


Chart 11-3. Trends in the prevalence of total serum cholesterol (≥200 mg/dL) in adults ≥20 years of age by sex and race/ethnicity (NHANES 2003–2004 and 2005–2006). NH indicates non-Hispanic; MA, Mexican American.

12. Risk Factor: Physical Inactivity

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

Prevalence

Youth

- As reported in the 2007 YRBS survey of adolescents in grades 9 through 12, 31.8% of females and 18% of males did not engage 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, despite recommendations that children engage in such activity ≥5 days per week.
  - — Rates of inactivity were highest among black (42.1%) and Hispanic (35.2%) females compared with white females (28.2%).
  - — Among males, blacks were also the least likely to engage in MVPA at current recommended levels (21.8%), followed by Hispanic (18.8%) and white (16.7%) males.[1]
- As reported in the 2007 YRBS survey of adolescents in grades 9 through 12, more than one fourth of all students spent ≥3 hours per day using computers outside of school time (24.9%) or watching television (35.4%).
  - — 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%).
  - — A greater proportion of black and Hispanic students used computers or watched television >3 hours per day than white students.[1]
- Among children 9 to 13 years of age, 61.5% do not participate in any organized PA during nonschool hours, and 22.6% do not engage in any free-time PA, according to 2002 data from the Youth Media Campaign Longitudinal Study (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.[2]
- By the age of 16 or 17 years, 31% of white girls and 56% of black girls report no habitual leisure-time PA.[3]
  - — 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.
  - — Cigarette smoking is associated with a decline in PA among white girls. Pregnancy is associated with a decline in PA among black girls but not among white girls.
  - — A higher BMI is associated with a greater decline in PA among girls of both races.
- 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:
  - — In the 2007 YRBS, 43.7% of male and 25.6% of female students in grades 9 through 12 self-reported that they met currently recommended levels of PA.
  - — Among these students, 33.2% of males and 27.3% of females attended physical education classes daily.
  - — Physical education class participation declined from the 9th through the 12th grades in males and females.[1]
  - — The proportion of boys and girls who actually met activity recommendations according to accelerometry declined with age in both sexes.
  - — Forty-two percent of 6- to 11-year-olds accumulated ≥60 minutes of MVPA (based on counts per minute >2020) 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.
  - — More boys than girls met recommendations as measured by accelerometry.[4]

Adults

- According to 2007 BRFSS/CDC data, the percentage of adults ≥18 years of age with ≥30 minutes of moderate PA ≥5 days per week or >20 minutes of vigorous PA ≥3 days per week ranged from 38.6% in Louisiana to 60.8% in Alaska. The median percentage among states was 49.5%. The percentage of adults with ≥20 minutes of vigorous PA ≥3 days per week ranged from 18.5% in Tennessee to 39.5% in Alaska (median among states 28.3%).[5]
- In 2005, 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[6]:
  - — Inactivity in 2005 was higher among females (12%) than males (8.4%).

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
CVD	cardiovascular disease
HBP	high blood pressure
MI	myocardial infarction
MVPA	moderate-to-vigorous physical activity
NCHS	National Center for Health Statistics
NHANES	National Health and Nutrition Examination Survey
NHIS	National Health Interview Survey
PA	physical activity
RR	relative risk
YMCLS	Youth Media Campaign Longitudinal Study
YRBS	Youth Risk Behavior Surveillance

— Inactivity in 2005 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.
— Non-Hispanic black and Hispanic adults were more likely to report inactivity (16.7% and 10.7%, respectively) than were non-Hispanic white adults (10.7%).

- Despite recommendations that some proportion of activity be vigorous (activity that causes heavy sweating and a large increase in breathing and/or heart rate),[7] 62% of adults >18 years of age who responded to the 2007 NHIS survey reported no vigorous activity that lasted >10 minutes per session.[8]

— Women (66.3%) were more likely than men (56.0%) to report never engaging in vigorous PA.
— Of the 11.4% of adults who engaged in vigorous activity for ≥5 days/week, the proportion was higher among men (13.1%) than women (9.8%).
— The proportion of respondents who did not participate in any vigorous activity increased with age from 52.4% in 18- to 44-year-olds to 88.7% in adults ≥75 years of age.
— American Indians (72.7%) and blacks (69.3%) were more likely to report not engaging in vigorous activity than Asians (62.9%), Native Hawaiians or other Pacific Islanders (61.2%), and white participants (60.0%).
— Hispanic or Latino adults were more likely not to engage in vigorous activity (71.8%) than non-Hispanic or non-Latino adults (59.5%).
— A lack of vigorous leisure-time activity was inversely associated with educational attainment: 83.6%, 72.7%, 61.3%, and 46.4% of respondents with less than a high school education, a high school diploma, some college or bachelor's degree or higher, respectively, reported no vigorous leisure-time activity.

- Adherence to PA recommendations was much lower when based on PA measured by accelerometer in NHANES 2003–2004[4]:

— 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.

Physical Inactivity 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.[9]
- 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.[10]
- A 2.3% decline in physical inactivity between 1980 and 2000 prevented or postponed approximately 17 445 deaths (≈5%) due to CHD in the United States.[11]
- A 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.[12]
- Data from the 2003 BRFSS (CDC) found that 53.2% of respondents with heart disease were told to be more physically active, 32% met recommended PA levels, and 30.8% were sedentary.[13]

References

1. Eaton DK, Kann L, Kinchen S, Shanklin S, Ross J, Hawkins J, Harris WA, Lowry R, McManus T, Chyen D, Lim C, Brener ND, Wechsler H; Centers for Disease Control and Prevention (CDC). Youth Risk Behavior Surveillance: United States, 2007. *MMWR Surveill Summ*. 2008;57:1–131.
2. Centers for Disease Control and Prevention (CDC). Physical activity levels among children aged 9–13 years: United States, 2002. *MMWR Morb Mortal Wkly Rep*. 2003;52:785–788.
3. Kimm SY, Glynn NW, Kriska AM, Barton BA, Kronsberg SS, Daniels SR, Crawford PB, Sabry ZI, Liu K. Decline in physical activity in black girls and white girls during adolescence. *N Engl J Med*. 2002;347:709–715.
4. Troiano RP, Berrigan D, Dodd KW, Masse LC, Tilert T, McDowell M. Physical activity in the United States measured by accelerometer. *Med Sci Sports Exerc*. 2008;40:181–188.
5. Behavioral Risk Factor Surveillance System. Prevalence and trends data, physical activity, 2007. Available at: http://apps.nccd.cdc.gov/brfss/list.asp?cat=PA&yr=2007&qkey=4418&state=All. Accessed April 22, 2008.
6. National Center for Health Statistics. Physical activity among adults: United States, 2000 and 2005. Available at: http://www.cdc.gov/nchs/products/pubs/pubd/hestats/physicalactivity/physicalactivity.htm. Accessed September 15, 2008.
7. Haskell WL, Lee IM, Pate RR, Powell KE, Blair SN, Franklin BA, Macera CA, Heath GW, Thompson PD, Bauman A. Physical activity and public health: updated recommendation for adults from the American College of Sports Medicine and the American Heart Association. *Circulation*. 2007;116:1081–1093.
8. Pleis JR, Lucus JW. Summary health statistics for U.S. adults: National Health Interview Survey, 2007. *Vital Health Stat 10*. In press. No. 240; provisional report.
9. Pate RR, Pratt M, Blair SN, Haskell WL, Macera CA, Bouchard C, Buchner D, Ettinger W, Heath GW, King AC, Kriska A, Leon AS, Marcus BH, Morris J, Paffenbarger RS Jr, Patrick K, Pollock ML, Rippe JM, Sallis J, Wilmore JH. Physical activity and public health: a recommendation from the Centers for Disease Control and Prevention and the American College of Sports Medicine. *JAMA*. 1995;273:402–407.
10. Yusuf S, Hawken S, Ôunpuu S, Dans T, Avezum A, Lanas F, McQueen M, Budaj A, Pais P, Varigos J, Lisheng L; INTERHEART Study Investigators. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. *Lancet*. 2004;364:937–952.
11. Ford ES, Ajani UA, Croft JB, Critchley JA, Labarthe DR, Kottke TE, Giles WH, Capewell S. Explaining the decrease in U.S. deaths from coronary disease, 1980–2000. *N Engl J Med*. 2007;356:2388–2398.
12. Hu FB, Stampfer MJ, Colditz GA, Ascherio A, Rexrode KM, Willett WC, Manson JE. Physical activity and risk of stroke in women. *JAMA*. 2000;283:2961–2967.
13. Wofford TS, Greenlund KJ, Croft JB, Labarthe DR. Diet and physical activity of U.S. adults with heart disease following preventive advice. *Prev Med*. 2007;45:295–301.
14. National Center for Health Statistics. Early release of selected estimates based on data from the 2007 National Health Interview Survey. Available at: http://www.cdc.gov/nchs/about/major/nhis/released200806.htm. Accessed June 25, 2008.
15. Centers for Disease Control and Prevention (CDC). Prevalence of regular physical activity among adults: United States, 2001 and 2005. *MMWR Morb Mortal Wkly Rep*. 2007;56:1209–1212.

Table 12-1. Regular Leisure-Time PA

Population Group	Prevalence, 2007 (Age ≥18 y)
Both sexes	30.80%
Males	28.90%
Females	28.90%
NH white only	33.90%
NH black only	22.90%
Hispanic or Latino	23.80%

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 2007 (NCHS).[14]

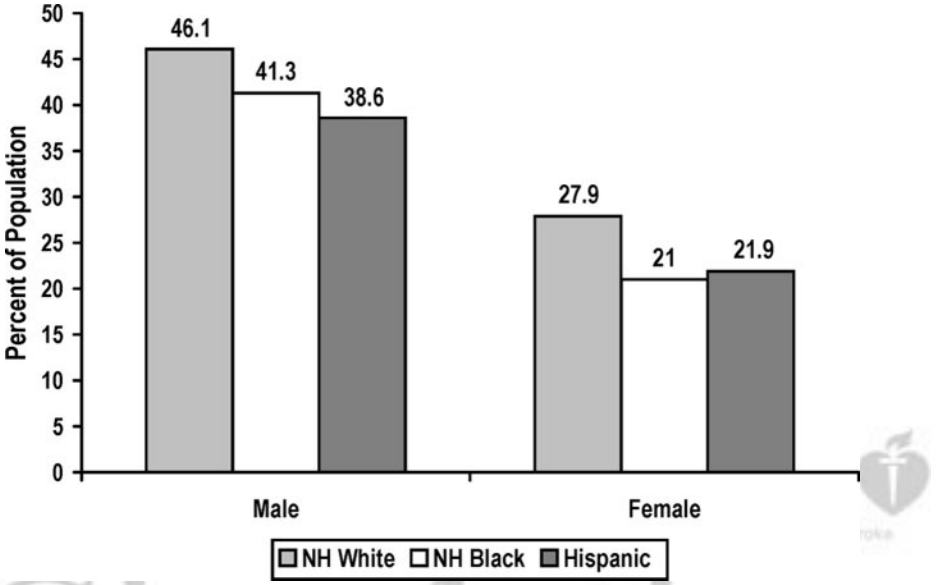


Chart 12-1. Prevalence of students in grades 9 through 12 who met currently recommended levels of PA during the past 7 days by race/ethnicity and sex (YRBS: 2007). "Currently recommended levels" is defined as activity that increased their heart rate and made them breathe hard some of the time for a total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

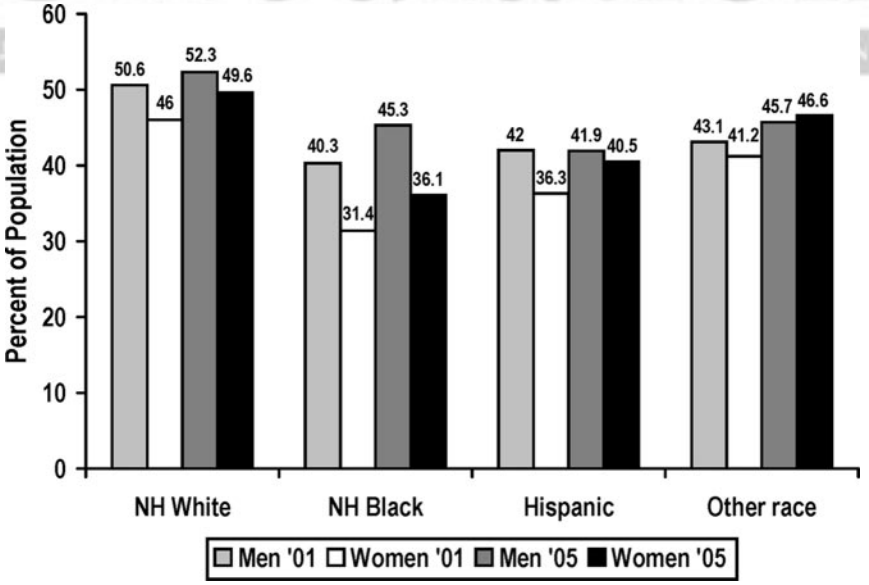


Chart 12-2. Prevalence of regular PA among adults ≥18 years of age by race/ethnicity and sex (BRFSS: 2001 and 2005). Source: MMWR.[15] NH indicates non-Hispanic.

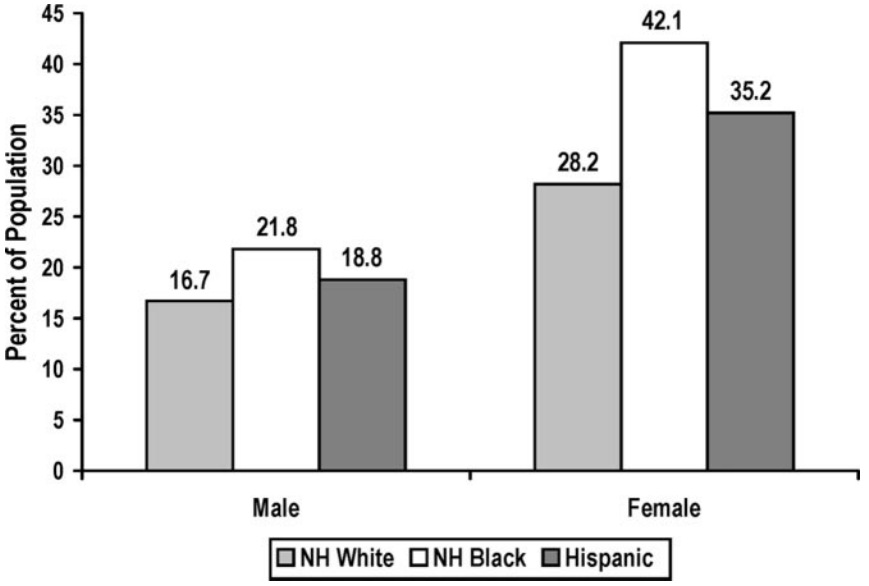


Chart 12-3. Prevalence of students in grades 9 through 12 who did not meet currently recommended levels of MVPA during the past 7 days by race/ethnicity and sex (YRBS: 2007). Source: Troiano et al.[4] "Currently recommended levels" are defined as activity that increased heart rate and made them breathe hard some of the time for a total of ≥60 minutes per day on ≥5 of the 7 days preceding the survey. NH indicates non-Hispanic.

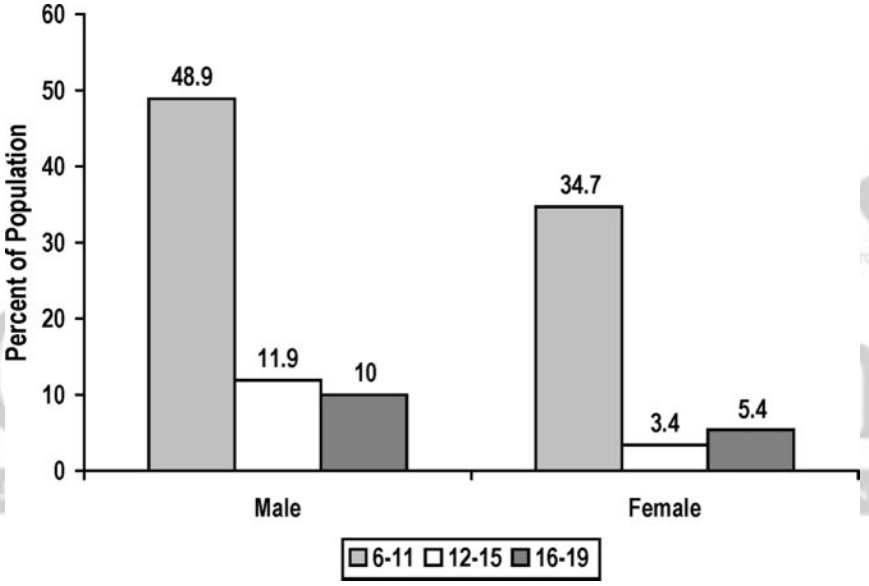


Chart 12-4. Prevalence of children 6 to 19 years of age who attained sufficient MVPA to meet public health recommendations of ≥60 minutes per day on ≥5 of the 7 days preceding the survey by sex and age (NHANES 2003–2004). Source: Troiano et al.[4]

13. Risk Factor: Overweight and Obesity

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

Prevalence

Youth

- On the basis of 2003–2006 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 2003–2006 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 1971–1974 to 17.0.% in 2003–2006. 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 1976–1980 and 11.5% in 2003–2006 (NHANES, NCHS).[3]
- Just over 12% of preschool children 2 to 5 years of age were overweight in 2003–2006.[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 2003–2006 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 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-Hispanic white male (15.7%) students. The prevalence of being obese 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

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
FHS	Framingham Heart Study
HHP	Honolulu Heart Program
kg/m^2	kilograms per meter squared
MESA	Multiethnic Study of Atherosclerosis
NCHS	National Center for Health Statistics
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

male (20.3%) and non-Hispanic black male (18.9%) than non-Hispanic white male (14.6%) students.[5]

- Data from the 2007 National Healthcare Quality Report found that:
  - — During 1999–2004, 38.8% of overweight children and teens 2 to 19 years of age were told by a doctor or health professional that they were overweight.
  - — During 1999–2004, overweight children 2 to 5 years of age (19.8%) and 6 to 11 years old (35.0%) were less likely than overweight children 12 to 19 years of age (47.6%) to be told by a provider that they were overweight.[6]

Adults

- Analysis of the FHS, 1971–2001 (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.[7]
- The age-adjusted prevalence of overweight and obesity (BMI $\geq$25 kg/m^2) increased from 64.5% in NHANES 1999–2000 (NCHS) to 66.3% in NHANES 2003–2004 (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]
- According to 2007 data from the BRFSS/CDC survey, based on self-reported height and weight, the prevalence of obesity ranged from 19.3% in Colorado to 32.6% in Mississippi. The median percentage by state was 26.3%.[8]
- 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).[9]
- 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%.[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 2007 NHIS study of the NCHS showed that blacks $\geq$18 years of age were less likely (28.1%) than American Indians or Alaska Natives (32.7%), whites (37.4%), and Asians (57.4%) to be at a healthy weight.[13]
- Data from the 2007 NHIS study of the NCHS showed that blacks $\geq$18 years of age were more likely (35.1%) to be obese than American Indians or Alaska Natives (32.4%), whites (25.4%), and Asians (8.9%).[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 2001–2002 (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]
- On the basis of NHANES/NCHS data, in 2003–2004, 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 1988–1994, when 27% of women 65 to 74 years of age and 19% of women $\geq$75 years of age were obese. For men, from 1988–1994, 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 2003–2004.[16]
- A 1997–2002 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.[17]
- 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 as Asian Indian (6%), Vietnamese (5%), or Chinese (4%) adults.[18]
- Data from NHANES 2005–2006 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.[19]
- From 1999 to 2004, obese adults 45 to 64 years of age (73%) and $\geq$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 $\geq$65 years of age were more likely to receive advice about exercise than those 18 to 44 years of age.[6]

- Data from the 2007 National Healthcare Disparities Report found that approximately 66.2% of obese adults were told by a doctor or health professional that they were overweight.[20]
- The proportion of obese adults told that they were overweight was significantly lower for blacks (61.1%) and Mexican Americans (56.5%) than for whites (68.8%); for middle-income people than for high-income people (64.2% versus 69.8%); and for adults with less than a high school education than for those with any college education (62.7% versus 70.7%).[20]
- 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.[21]

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,[22] overweight (BMI 25 to <30 kg/m^2) was not associated with excess deaths.[23]
- 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.[24]

Cost

- Among children and adolescents, annual hospital costs related to obesity were $127 million between 1997 and 1999.[25]
- According to one study, annual medical spending due to overweight and obesity could be as high as $92.6 billion in 2002 dollars, which represents 9.1% of US health expenditures.[26] According to another estimate, the annual cost of overweight and obesity, in 2001 dollars, is $117 billion. The direct annual cost is $61 billion, and the indirect cost is $56 billion. The cost of lost productivity related to obesity among Americans 17 to 64 years of age is $3.9 billion annually (1994).[27]

References

1. Ogden CL, Carroll MD, Flegal KM. High body mass index for age among US children and adolescents, 2003–2006. *JAMA*. 2008;299:2401–2405.
2. National Center for Health Statistics. *Health, United States, 2007: With Chartbook on Trends in the Health of Americans.* Hyattsville, Md: US Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Health Statistics; 2007. Available at: http://www.cdc.gov/nchs/hus.htm. Accessed September 15, 2008.
3. US Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Health Statistics. *Prevalence of Overweight, Infants and Children Less Than 2 Years of Age: United States, 2003–2004.* Hyattsville, Md: US Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Health Statistics; 2007. Available at: http://www.cdc.gov/nchs/products/pubs/pubd/hestats/overweight/overwght_child_under02.htm. Accessed October 26, 2007.
4. US Department of Health and Human Services. *The Surgeon General's Call to Action to Prevent Overweight and Obesity: Overweight in Children and Adolescents.* Washington, DC: US Department of Health and Human Services; 2007. Available at: http://www.surgeongeneral.gov/topics/obesity/calltoaction/fact_adolescents.htm. Accessed October 26, 2007.
5. Eaton DK, Kann L, Kinchen S, Shanklin S, Ross J, Hawkins J, Harris WA, Lowry R, McManus T, Chyen D, Lim C, Brener ND, Wechsler H; Centers for Disease Control and Prevention. Youth risk behavior surveillance: United States, 2007. *MMWR Surveill Summ*. 2008;57:1–131.
6. Agency for Healthcare Research and Quality. *2007 National Healthcare Quality Report.* Rockville, Md: US Department of Health and Human Services, Agency for Healthcare Research and Quality; February 2008. AHRQ publication No. 08-0040.
7. Vasan RS, Pencina MJ, Cobain M, Freiberg MS, D'Agostino RB. Estimated risks for developing obesity in the Framingham Heart Study. *Ann Intern Med*. 2005;143:473–480.
8. Centers for Disease Control and Prevention (CDC). State-specific prevalence of obesity among adults: United States, 2007. *MMWR Morb Mortal Wkly Rep*. 2008;57:765–768.
9. Suk SH, Sacco RL, Boden-Albala B, Cheun JF, Pittman JG, Elkind MS, Paik MC; Northern Manhattan Stroke Study. Abdominal obesity and risk of ischemic stroke: the Northern Manhattan Stroke Study. *Stroke*. 2003;34:1586–1592.
10. Rodriguez BL, D'Agostino R, Abbott RD, Kagan A, Burchfiel CM, Yano K, Ross GW, Silbershatz H, Higgins MW, Popper J, Wolf PA, Curb JD. Risk of hospitalized stroke in men enrolled in the Honolulu Heart Program and the Framingham Study: a comparison of incidence and risk factor effects. *Stroke*. 2002;33:230–236.
11. Centers for Disease Control and Prevention (CDC). State-specific prevalence of obesity among adults with disabilities: eight states and the District of Columbia, 1998–1999. *MMWR Morb Mortal Wkly Rep*. 2002;51:805–808.
12. Peeters A, Barendregt JJ, Willekens F, Mackenbach JP, Al Mamun A, Bonneux L; NEDCOM, the Netherlands Epidemiology and Demography Compression of Morbidity Research Group. Obesity in adulthood and its consequences for life expectancy: a life-table analysis. *Ann Intern Med*. 2003;138: 24–32.
13. Pleis JR, Lucus JW. Summary health statistics for U.S. adults: National Health Interview Survey, 2007. *Vital Health Stat 10*. In press.
14. World Health Organization. *Obesity and Overweight.* Fact Sheet No. 311. Geneva, Switzerland: World Health Organization; September 2006. Available at: www.who.int/mediacentre/factsheets/fs311/en/print.html/. Accessed October 26, 2007.
15. Seo DC, Torabi MR. Racial/ethnic differences in body mass index, morbidity and attitudes toward obesity among U.S. adults. *J Natl Med Assoc*. 2006;98:1300–1308.
16. Robinson K. *Trends in Health Status and Health Care Use Among Older Women.* Aging Trends, No. 7. Hyattsville, Md: National Center for Health Statistics; March 2007.
17. Doshi JA, Polsky D, Chang VW. Prevalence and trends in obesity among aged and disabled U.S. Medicare beneficiaries, 1997–2002. *Health Affairs (Millwood)*. 2007;26:1111–1117.
18. Barnes PM, Adams PF, Powell-Griner E. *Health Characteristics of the Asian Adult Population: United States, 2004–2006.* Advance Data From Vital and Health Statistics, No. 394. Hyattsville, Md: National Center for Health Statistics; January 22, 2008.
19. Ogden CL, Carroll MD, McDowell MA, Flegal KM. *Obesity Among Adults in the United States: No Statistically Significant Change Since 2003–2004.* NCHS Data Brief No. 1. Hyattsville, Md: US Department of

Health and Human Services, Centers for Disease Control and Prevention, National Center for Health Statistics; 2007.
20. Agency for Healthcare Research and Quality. *2007 National Healthcare Disparities Report.* Rockville, Md: US Department of Health and Human Services, Agency for Healthcare Research and Quality; February 2008. AHRQ publication No. 08-0041.
21. Burke GL, Bertoni AG, Shea S, Tracy R, Watson KE, Blumenthal RS, Chung H, Carnethon MR. The impact of obesity on cardiovascular disease risk factors and subclinical vascular disease: the Multi-Ethnic Study of Atherosclerosis. *Arch Intern Med*. 2008;168:928–935.
22. McGee DL; Diverse Populations Collaboration. Body mass index and mortality: a meta-analysis based on person-level data from twenty-six observational studies. *Ann Epidemiol*. 2005;15:87–97.
23. Flegal KM, Graubard BI, Williamson DF, Gail MH. Excess deaths associated with underweight, overweight, and obesity. *JAMA*. 2005;293:1861–1867.
24. Flegal KM, Graubard BI, Williamson DF, Gail MH. Cause-specific excess deaths associated with underweight, overweight, and obesity. *JAMA*. 2007;298:2028–2037.
25. Centers for Disease Control and Prevention. *Preventing Obesity and Chronic Diseases Through Good Nutrition and Physical Activity.* Atlanta, Ga: Centers for Disease Control and Prevention; 2005. Available at: http://www.cdc.gov/nccdphp/publications/factsheets/Prevention/obesity.htm. Accessed October 30, 2006.
26. Finkelstein EA, Fiebelkorn IC, Wang G. National medical spending attributable to overweight and obesity: how much, and who's paying? *Health Aff (Millwood)*. 2003;Suppl Web Exclusives:W3-219–W3-226. Available at: http://content.healthaffairs.org/cgi/content/full/hlthaff.w3.219v1/DC1. Accessed October 26, 2007.
27. Weight-Control Information Network. Statistics related to overweight and obesity. Available at: http://www.win.niddk.nih.gov/statistics/index.htm#econ. Accessed February 5, 2008.

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, 2002*
Both sexes, n (%)	145 000 000 (66.7)	74 100 000 (33.9)	23 400 000 (31.9)	12 000 000 (16.3)	$117 billion
Males, n (%)	76 900 000 (73.0)	34 700 000 (32.7)	12 300 000 (32.7)	6 400 000 (17.1)	. . .
Females, n (%)	68 100 000 (60.5)	39 400 000 (35.0)	11 100 000 (31.0)	5 600 000 (15.5)	. . .
NH white males, %	72.4	32.3	31.9	15.6	. . .
NH white females, %	57.5	32.7	29.5	13.6	. . .
NH black males, %	73.7	36.8	30.8	17.4	. . .
NH black females, %	77.7	52.9	39.2	24.1	. . .
Mexican American males, %	74.8	26.8	40.8	23.2	. . .
Mexican American females, %	73.0	41.9	35.0	18.5	. . .
Hispanic or Latino age ≥18 y†, %	67.8	27.5	. . .	. . .	. . .
Asian-only, age ≥18 y†, %	38.1	8.9	. . .	. . .	. . .
American Indian/Alaska Native, age ≥18 y†, %	67.1	32.4	. . .	. . .	. . .

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 NIDDK.[27]

†NHIS (2007), NCHS; data 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 2005–2006 (NCHS), NHLBI and unpublished data. Data for adults are for age ≥20 years. Estimates from NHANES 2005–2006 (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]

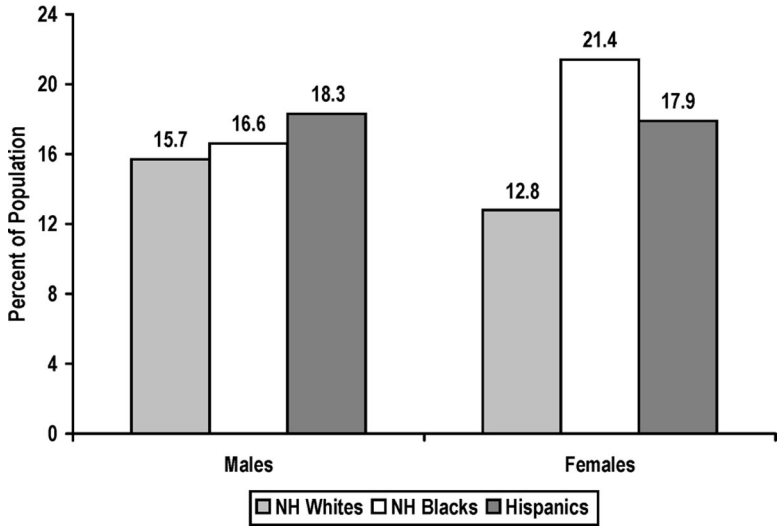


Chart 13-1. Prevalence of overweight among students in grades 9 through 12 by sex and race/ethnicity (YRBS: 2007). Source: BMI 95th percentile or higher by age and sex of the CDC 2000 growth chart.[5] NH indicates non-Hispanic.

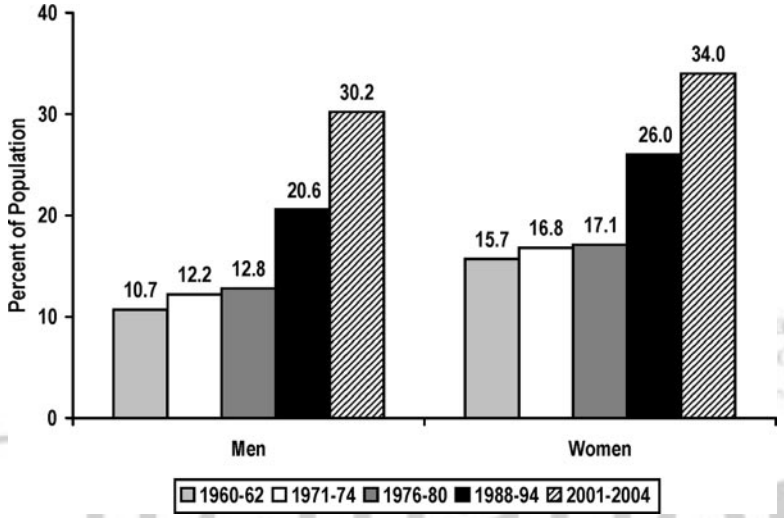


Chart 13-2. Age-adjusted prevalence of obesity in adults 20 to 74 years of age, by sex and survey (NHES: 1960–1962; NHANES: 1971–1974, 1976–1980, 1988–1994, and 2001–2004). Obesity is defined as a BMI ≥30.0. Source: *Health, United States, 2007* (NCHS).[2]

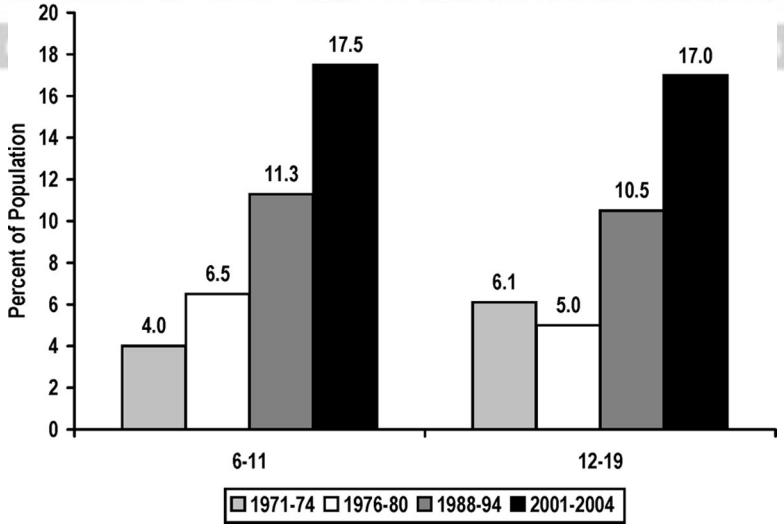


Chart 13-3. Trends in the prevalence of overweight among US children and adolescents by age and survey (NHANES: 1971–1974, 1976–1980, 1988–1994, and 2001–2004). Source: *Health, United States, 2007* (NCHS).[2]

14. Risk Factor: Diabetes Mellitus

ICD-9 250; ICD-10 E10–E14. See Table 14-1 and Charts 14-1 through 14-3.

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.
- 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]

Adults

- Data from NHANES 1999–2002 (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.[4]
- 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.[5]
- 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.[6]
- The prevalence of diabetes was more than twice as high for Asian Indian adults (14%) as for Chinese (6%) or Japanese (5%) adults.[7]
- Type 2 DM accounts for 90% to 95% of all diagnosed cases of DM in adults.[8] In Framingham, Mass, 99% of DM is type 2.[9]
- 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%.[10]
- On the basis of 2007 BRFSS (CDC) data, the prevalence of adults who reported ever having been told by a doctor that they had DM ranged from 5.7% in Minnesota to 11.9% in Tennessee. The median percentage among states was 8.0%.[11]
- The CDC analyzed data from 1994 to 2004 collected by the Indian Health Service that indicated that the age-adjusted

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
CI	confidence interval
CVD	cardiovascular disease
DM	diabetes mellitus
FHS	Framingham Heart Study
HbA_{1c}	glycosylated hemoglobin
HR	hazard ratio
ICD	International Classification of Diseases
kg/m^2	kilograms per meter squared
LDL	low-density lipoprotein
mg/dL	milligrams per deciliter
MI	myocardial infarction
mm Hg	millimeter of mercury
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
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

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.[12]

- The prevalence of DM for all age groups worldwide was estimated to be 2.8% in 2000 and is projected to be 4.4% in 2030. The total number of people with DM is projected to rise from 171 million in 2000 to 366 million in 2030.[13]
- 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%).[14]

Incidence

Youths

- In the SEARCH study, the incidence of DM in youths overall was 24.3 per 100 000 person-years. Among children <10 years of age, most had type 1 DM, regardless of race/ethnicity. The highest rates of incident type 1 DM were observed in non-Hispanic white youths (18.6, 28.1, and 32.9 per 100 000 person-years for age groups of 0 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.[15]

Adults

- One and a half million new cases of DM were diagnosed in people ≥20 years of age in 2005.[5]
- 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).[16]

Mortality

DM mortality in 2005 was 75 119. Total-mention mortality in 2005 was 233 600; 2006 preliminary mortality was 72 507, and the death rate was 23.3. (Source: NCHS and NHLBI)

- The 2005 overall underlying-cause death rate due to DM was 24.6. Death rates per 100 000 persons were 26.5 for white males, 50.8 for black males, 19.3 for white females, and 43.8 for black females.[17]
- According to data from the National Diabetes Information Clearinghouse, NIDDK, 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.[18]
- FHS/NHLBI data show that having DM significantly increased the risk of developing CVD (HR 2.5 for women and 2.4 for men) and of dying when CVD was present (HR 2.2 for women and 1.7 for men). Diabetic men and women ≥50 years of age lived an average of 7.5 and 8.2 years less than their nondiabetic equivalents. The differences in life expectancy free of CVD were 7.8 and 8.4 years, respectively.[19]
- Analysis of data from NHANES 1971–2000 found that men with DM experienced a 43% relative reduction in the age-adjusted mortality rate, which 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.[20]
- During 1979–2004, diabetes death rates for black youths 1 to 19 years of age were approximately twice those for white youths. During 2003–2004, the annual average diabetes death rate per 1 million youths was 2.46 for black youths and 0.91 for white youths.[21]

Awareness

- The NIDDK estimates that 20.8 million Americans (7% of the population) have DM and that ≈30% are unaware of the diagnosis.[5]
- Analysis of NHANES/NCHS data from 1988–1994 to 1999–2002 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 the past decade, the prevalences of undiagnosed DM and impaired fasting glucose have remained relatively stable. Minority groups remain disproportionately affected.[22]

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.[23]
- DM increases the risk of stroke, with the RR ranging from 1.8 to almost 6.0.[24]
- 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.[25]

- On the basis of data from the NCHS/NHIS, 1997–2005[26]:
  - — During 1997–2005, 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 1997–2005, 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 fell 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).[27] 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.[28]
- 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%).[29]
- Data from the ARIC study of the NHLBI found that DM was a weaker predictor of CHD in blacks than in whites.[30]
- 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 1950–1966 ("earlier" time period) and 1977–1995 ("later" time period) were followed up for incident MI, CHD death, and stroke. Among participants with DM, the age- and sex-adjusted CVD incidence rate was 286.4 per 10 000 person-years in the earlier period and 146.9 per 10 000 person-years in the later period, a 35.4% decline. HRs for DM as a predictor of incident CVD were not significantly different in the earlier (risk factor–adjusted HR 2.68, 95% CI 1.88 to 3.82) versus later (HR 1.96, 95% CI 1.44 to 2.66) periods.[31] 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.[31]
  - — 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 ($P=0.12$).[32]
  - — 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.[33]

- 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 1989–1991 and 1999–2001 and hospital discharge data for 1988–2002 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.[34]

- 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 fell 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%.[35]
- 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 risen, so has the absolute burden of CVD, especially among middle-aged and older individuals.[36]

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 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.[37]
  - 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 interventions in 2001.[37]
- Between NHANES III 1988–1994 (NCHS) and NHANES 1999–2002 (NCHS), considerable differences were found among ethnic groups in glycemic control rates among adults with type 2 DM. Among non-Hispanic whites, the controlled rates were 43.8% in 1988–1994 and 48.4% in 1999–2002. 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%.[38]
- In 1 large academic medical center, outpatients with type 2 DM were observed during an 18-month period for proportions of patients who had HbA_{1c} levels, BP, or total cholesterol levels measured; who had been prescribed any drug therapy if HbA_{1c} levels, SBP, or LDL cholesterol levels exceeded recommended treatment goals; and who had been prescribed greater-than-starting-dose therapy if these values were above treatment goals. Patients were less likely to have cholesterol levels measured (76%) than HbA_{1c} levels (92%) or BP (99%; $P<0.0001$ for either comparison). The proportion of patients who received any drug therapy was greater for above-goal HbA_{1c} (92%) than for above-goal SBP (78%) or LDL cholesterol (38%; $P<0.0001$ for each comparison). Similarly, patients whose HbA_{1c} levels were above the treatment goal (80%) were more likely to receive greater-than-starting-dose therapy than were those who had above-goal SBP (62%) and LDL cholesterol levels (13%; $P<0.0001$).[39]
  - 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, $P=0.006$).[40]
- In 2001–2002, 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.[41]

Hospitalizations

Youth

- National Inpatient Sample data from 1993–2004 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.[42]

Cost

In 2007, the direct ($116 billion) and indirect ($58 billion) cost attributable to DM was $174 billion.[43]

References

1. SEARCH for Diabetes in Youth Study Group. The burden of diabetes mellitus among US youth: prevalence estimates from the SEARCH for Diabetes in Youth Study. *Pediatrics*. 2006;118:1510–1518.
2. Centers for Disease Control and Prevention. National Diabetes Fact Sheet 2007. Available at: http://www.searchfordiabetes.org/public/documents/CDCFact2008.pdf. Accessed December 5, 2008.
3. The Writing Group for the SEARCH for Diabetes in Youth Study Group. Incidence of diabetes in youth in the United States [published correction appears in *JAMA*. 2007;298:627]. *JAMA*. 2007;297:2716–2724.
4. Selvin E, Coresh J, Brancati FL. The burden and treatment of diabetes in elderly individuals in the U.S. *Diabetes Care*. 2006;29:2415–2419.
5. National Institute of Diabetes and Digestive and Kidney Diseases. *National Diabetes Statistics Fact Sheet: General Information and National Estimates on Diabetes in the United States, 2005.* Bethesda, Md: US Department of Health and Human Services, National Institutes of Health; 2005.
6. Centers for Disease Control and Prevention (CDC). Prevalence of diabetes and impaired fasting glucose in adults: United States, 1999–2000. *MMWR Morb Mortal Wkly Rep*. 2003;52:833–837.
7. Barnes PM, Adams PF, Powell-Griner E. *Health Characteristics of the Asian Adult Population: United States, 2004–2006.* Advance Data From Vital and Health Statistics; No. 394. Hyattsville, Md: National Center for Health Statistics; January 22, 2008.
8. National Diabetes Information Clearinghouse. National diabetes statistics. Available at: http://www.diabetes.niddk.nih.gov/dm/pubs/statistics/index.htm. Accessed November 1, 2007.
9. Meigs JB, Cupples LA, Wilson PW. Parental transmission of type 2 diabetes mellitus: the Framingham Offspring Study. *Diabetes*. 2000;49:2201–2207.
10. Mokdad AH, Ford ES, Bowman BA, Dietz WH, Vinicor F, Bales VS, Marks JS. Prevalence of obesity, diabetes, and obesity-related health risk factors, 2001. *JAMA*. 2003;289:76–79.
11. Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion. Behavioral Risk Factor Surveillance System, Prevalence Data, Diabetes: 2007. Have you ever been told by a doctor that you have diabetes? Available at: http://apps.nccd.cdc.gov/brfss/list.asp?cat=DB&yr=2007&qkey=1363&state=All. Accessed April 28, 2008.
12. Centers for Disease Control and Prevention (CDC). Diagnosed diabetes among American Indians and Alaska Natives aged <35 years: United States, 1994–2002. *MMWR Morb Mortal Wkly Rep*. 2006;55:1201–1203.
13. Wild S, Roglic G, Green A, Sicree R, King H. Global prevalence of diabetes: estimates for the year 2000 and projections for 2030. *Diabetes Care*. 2004;27:1047–1053.
14. Narayan KM, Boyle JP, Geiss LS, Saaddine JB, Thompson TJ. Impact of recent increase in incidence on future diabetes burden: U.S., 2005–2050. *Diabetes Care*. 2006;29:2114–2116.
15. Writing Group for the SEARCH for Diabetes in Youth Study Group; Dabelea D, Bell RA, D'Agostino RB Jr, Imperatore G, Johansen JM, Linder B, Liu LL, Loots B, Marcovina S, Mayer-Davis EJ, Pettitt DJ, Waitzfelder B. Incidence of diabetes in youth in the United States [published correction appears in *JAMA*. 2007;298:627]. *JAMA*. 2007;297:2716–2724.
16. Fox CS, Pencina MJ, Meigs JB, Vasan RS, Levitzky YS, D'Agostino RB Sr. Trends in the incidence of type 2 diabetes mellitus from the 1970s to the 1990s: the Framingham Heart Study. *Circulation*. 2006;113:2914–2918.
17. Kung H-C, Hoyert DL, Xu JQ, Murphy SL. Deaths: final data for 2005. *Natl Vital Stat Rep*. 2008;56;1–120.
18. National Diabetes Information Clearinghouse (a service of the National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health). Diabetes Across the United States. Available at: http://www.diabetes.niddk.nih.gov/populations/index.htm. Accessed November 1, 2007.
19. Franco OH, Steyerberg EW, Hu FB, Mackenbach J, Nusselder W. Associations of diabetes mellitus with total life expectancy and life expectancy with and without cardiovascular disease. *Arch Intern Med*. 2007;167:1145–1151.
20. Gregg EW, Gu Q, Cheng YJ, Narayan KM, Cowie CC. Mortality trends in men and women with diabetes, 1971 to 2000. *Ann Intern Med*. 2007;147:149–155.
21. Centers for Disease Control and Prevention (CDC). Racial disparities in diabetes mortality among persons aged 1–19 years: United States, 1979–2004. *MMWR Morb Mortal Wkly Rep*. 2007;56:1184–1187.
22. Cowie CC, Rust KF, Byrd-Holt DD, Eberhardt MS, Flegal KM, Engelgau MM, Saydah SH, Williams DE, Geiss LS, Gregg EW. Prevalence of diabetes and impaired fasting glucose in adults in the U.S. population: National Health and Nutrition Examination Survey 1999–2002. *Diabetes Care*. 2006;29:1263–1268.
23. Fox CS, Sullivan L, D'Agostino RB Sr, Wilson PW; for the Framingham Heart Study. The significant effect of diabetes duration on coronary heart disease mortality: the Framingham Heart Study. *Diabetes Care*. 2004;27:704–708.
24. Goldstein LB, Adams R, Becker K, Furberg CD, Gorelick PB, Hademenos G, Hill M, Howard G, Howard VJ, Jacobs B, Levine SR, Mosca L, Sacco RL, Sherman DG, Wolf PA, del Zoppo GJ. Primary prevention of ischemic stroke: a statement for healthcare professionals from the Stroke Council of the American Heart Association. *Stroke*. 2001;32:280–299.
25. Kissela BM, Khoury J, Kleindorfer D, Woo D, Schneider A, Alwell K, Miller R, Ewing I, Moomaw CJ, Szaflarski JP, Gebel J, Shukla R, Broderick JP. Epidemiology of ischemic stroke in patients with diabetes: the greater Cincinnati/Northern Kentucky Stroke Study. *Diabetes Care*. 2005;28:355–359.
26. Centers for Disease Control and Prevention (CDC). Prevalence of self-reported cardiovascular disease among persons aged ≥35 years with diabetes: United States, 1997–2005. *MMWR Morb Mortal Wkly Rep*. 2007;56:1129–1132.
27. Ford ES, Ajani UA, Croft JB, Critchley JA, Labarthe DR, Kottke TE, Giles WH, Capewell S. Explaining the decrease in U.S. deaths from coronary disease, 1980–2000. *N Engl J Med*. 2007;356:2388–2398.
28. Lyerly GW, Sui X, Church TS, Lavie CJ, Hand GA, Blair SN. Maximal exercise electrocardiography responses and coronary heart disease mortality among men with diabetes mellitus. *Circulation*. 2008;117:2734–2742.
29. Donahoe SM, Stewart GC, McCabe CH, Mohanavelu S, Murphy SA, Cannon CP, Antman EM. Diabetes and mortality following acute coronary syndromes. *JAMA*. 2007;298:765–775.
30. Jones DW, Chambless LE, Folsom AR, Heiss G, Hutchinson RG, Sharrett AR, Szklo M, Taylor HA Jr. Risk factors for coronary heart disease in African Americans: the Atherosclerotic Risk in Communities Study, 1987–1997. *Arch Intern Med*. 2002;162:2565–2571.
31. Fox CS, Coady S, Sorlie PD, Levy D, Meigs JB, D'Agostino RB Sr, Wilson PW, Savage PJ. Trends in cardiovascular complications of diabetes. *JAMA*. 2004;292:2495–2499.
32. Fox CS, Coady S, Sorlie PD, D'Agostino RB Sr, Pencina MJ, Vasan RS, Meigs JB, Levy D, Savage PJ. Increasing cardiovascular disease burden due to diabetes mellitus: the Framingham Heart Study. *Circulation*. 2007;115:1544–1550.
33. Fox CS, Pencina MJ, Wilson PW, Paynter NP, Vasan RS, D'Agostino RB Sr. Lifetime risk of cardiovascular disease among individuals with and without diabetes stratified by obesity status in the Framingham Heart Study. *Diabetes Care*. 2008;31:1582–1584.
34. Fang J, Alderman MH. Impact of the increasing burden of diabetes on acute myocardial infarction in New York City: 1990–2000. *Diabetes*. 2006;55:768–773.
35. Booth GL, Kapral MK, Fung K, Tu JV. Recent trends in cardiovascular complications among men and women with and without diabetes. *Diabetes Care*. 2006;29:32–37.

36. Booth GL, Kapral MK, Fung K, Tu JV. Relation between age and cardiovascular disease in men and women with diabetes compared with non-diabetic people: a population-based retrospective cohort study. *Lancet*. 2006;368:29–36.
37. US Department of Health & Human Services, Agency for Healthcare Research and Quality. *National Healthcare Disparities Report, 2004*. Rockville, Md: Agency for Healthcare Research and Quality; 2004. AHRQ publication No. 05-0014. Available at: http://www.ahrq.gov/qual/nhdr04/nhdr04.htm. Accessed November 1, 2007.
38. Fan T, Koro CE, Fedder DO, Bowlin SJ. Ethnic disparities and trends in glycemic control among adults with type 2 diabetes in the U.S. from 1988 to 2002. *Diabetes Care*. 2006;29:1924–1925.
39. Grant RW, Cagliero E, Murphy-Sheehy P, Singer DE, Nathan DM, Meigs JB. Comparison of hyperglycemia, hypertension, and hypercholesterolemia management in patients with type 2 diabetes. *Am J Med*. 2002;112:603–609.
40. Wexler DJ, Grant RW, Meigs JB, Nathan DM, Cagliero E. Sex disparities in treatment of cardiac risk factors in patients with type 2 diabetes. *Diabetes Care*. 2005;28:514–520.
41. Malik S, Lopez V, Chen R, Wu W, Wong ND. Undertreatment of cardiovascular risk factors among persons with diabetes in the United States. *Diabetes Res Clin Pract*. 2007;77:126–133.
42. Lee JM, Okumura MJ, Freed GL, Menon RK, Davis MM. Trends in hospitalizations for diabetes among children and young adults: United States, 1993–2004. *Diabetes Care*. 2007;30:3035–3039.
43. National Institute of Diabetes and Digestive and Kidney Diseases. *National Diabetes Statistics, 2007 Fact Sheet*. Bethesda, Md: US Department of Health and Human Services, National Institutes of Health; 2008.
44. Pleis JR, Lucas JW. Summary health statistics for U.S. adults: National Health Interview Survey, 2007. National Center for Health Statistics. *Vital Health Stat 10*. In press. No. 240; provisional report.

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), 2005‡ All Ages	Hospital Discharges, 2006 All Ages	Cost, 2007§
Both sexes	17 000 000 (7.7%)	6 400 000 (2.9%)	57 000 000 (25.9%)	1 600 000§	75 119	584 000	$174 billion
Males	7 500 000 (7.4%)	3 900 000 (3.8%)	34 000 000 (31.7%)	. . .	36 538 (48.6%)*	283 000	. . .
Females	9 500 000 (8.0%)	2 500 000 (2.1%)	23 000 000 (19.9%)	. . .	38 581 (51.4%)*	301 000	. . .
NH white males	5.8%	3.6%	32.0%	. . .	29 628	. . .	. . .
NH white females	6.1%	2.2%	18.7%	. . .	30 127	. . .	. . .
NH black males	14.9%	4.7%	22.9%	. . .	5730	. . .	. . .
NH black females	13.1%	3.1%	19.0%	. . .	7240	. . .	. . .
Mexican American males	11.3%	6.0%	28.5%	. . .	. . .	. . .	. . .
Mexican American females	14.2%	1.9%	23.6%	. . .	. . .	. . .	. . .
Hispanic or Latino,† age ≥18 y	11.1%	. . .	. . .	. . .	. . .	. . .	. . .
Asian,† age ≥18 y	8.9%	. . .	. . .	. . .	. . .	. . .	. . .
AI/AN,† age ≥18 y	17.2%	. . .	. . .	. . .	. . .	. . .	. . .

Ellipses (. . .) indicate data not available; 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.[44] 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: NHLBI computations from NHANES 2003–2006; extrapolation to the 2006 US population. Prevalence of prediabetes: CDC Fact Sheet.[43] 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.

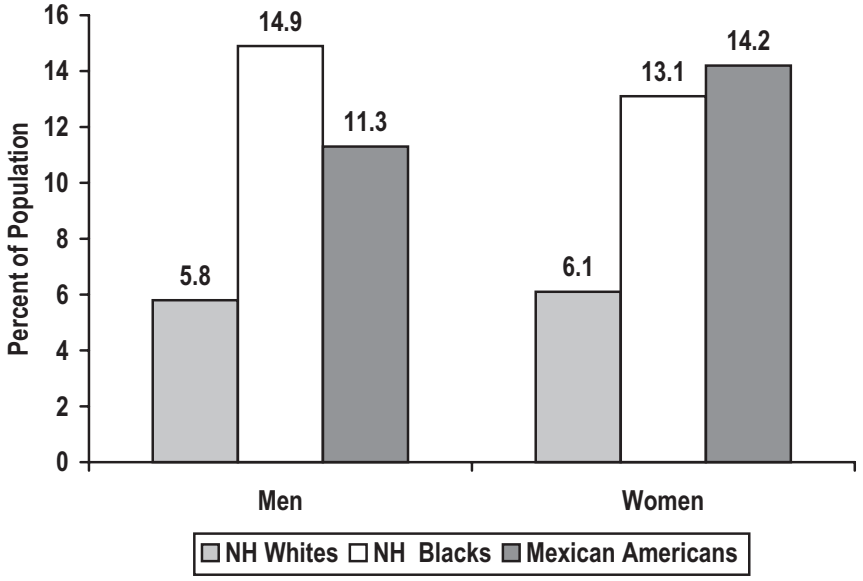


Chart 14-1. Prevalence of physician-diagnosed diabetes in adults ≥20 years of age by race/ethnicity and sex (NHANES: 2005–2006). Source: NCHS and NHLBI. NH indicates non-Hispanic.

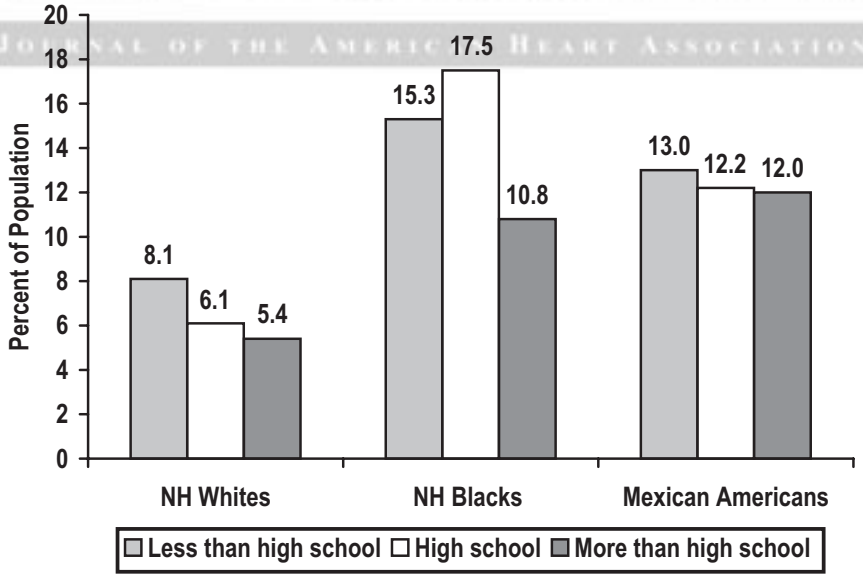


Chart 14-2. Prevalence of physician-diagnosed type 2 diabetes in adults ≥20 years of age by race/ethnicity and years of education (NHANES: 2005–2006). Source: NCHS and NHLBI. NH indicates non-Hispanic.

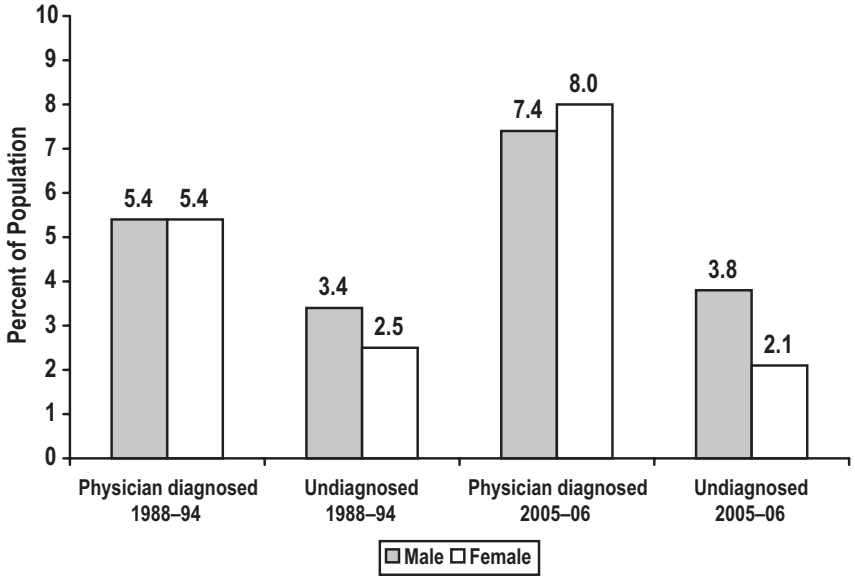


Chart 14-3. Trends in diabetes prevalence in adults ≥20 years of age, by sex (NHANES: 1988–1994 and 2005–2006). Source: NCHS and NHLBI.

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. 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.

- The incidence of reported ESRD has increased ≈40% in the past 10 years.[1]
- In 2005, 106 912 new cases of ESRD were reported.[1]
- The number of persons treated for ESRD increased from 68 757 in 1994 to 102 356 in 2004; this translates to an increase of 261.3 per 1 million population in 1994 to 348.6 per 1 million population in 2004.[2]
- Data from the US Renal Data System show that in 2005, 85 790 patients died of ESRD.[1]
- In 2004, mortality rates for those ≥65 years of age receiving dialysis were 7 times greater than those of the general Medicare population.[2]

Abbreviations Used in Chapter 15

ARF	acute renal failure
BMI	body mass index
BP	blood pressure
CHF	congestive heart failure
CI	confidence interval
CKD	chronic kidney disease
CKF	chronic kidney failure
CVD	cardiovascular disease
DM	diabetes mellitus
eGFR	estimated glomerular filtration rate
ESRD	end-stage renal disease
HBP	high blood pressure
HDL	high-density lipoprotein
HMO	health maintenance organization
JNC	Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure
kg/m^2	kilograms per square meter
K/DOQI	Kidney Disease Outcome Quality Initiative
LDL	low-density lipoprotein
mL/min per 1.73 m^2	first morning urine protein/creatinine ratio
NCHS	National Center for Health Statistics
NHANES	National Health and Nutrition Examination Survey
NKF	National Kidney Foundation
RR	relative risk
USDHHS	US Department of Health and Human Services

- More than 17 400 kidney transplantations were performed in 2005.[1]
- Diabetes continues to be the most common reported cause of ESRD, followed by hypertension and glomerulonephritis.[2] These 3 diseases accounted for 80% of cases of ESRD between 1994 and 2004.[2]
- From 1994 to 2004, ESRD attributed to glomerulonephritis decreased among all races analyzed.[2]
- From 1994 to 2004, ESRD attributed to glomerulonephritis was highest among blacks.[2]
- 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.[2] This decrease is particularly impressive given the increasing prevalence of diabetes among American Indians/Alaska Natives.
- The CDC analyzed 1990–2002 data from the US Renal Data System, which showed that diabetes was the leading cause of ESRD, accounting for 44% of new cases in 2002. Although new cases of ESRD-attributed diabetes mellitus (DM) increased overall, the incidence of ESRD-attributed diabetes 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.[3]
- Between 1996 and 1997, 3.2% of the Medicare population had a diagnosis of CKD, representing 63.6% of people who progressed to ESRD after 1 year.[4]
- Data from a large HMO population reveal that among adults with a GFR >60 mL/min per 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[5] (see also Table 15-1).
- Results from a large, community-based population showed that higher BMI also independently increased the risk of ESRD. The higher risk of ESRD with overweight and obesity was consistent across age, sex, race, and the presence or absence of diabetes, hypertension, or known baseline kidney disease[6] (see also 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 acute myocardial infarction 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 National Hospital Discharge survey, 2006).

Age, Sex, Race, and Ethnicity

- Children with pediatric ESRD have high transplantation rates. Time to first transplantation appears to be increasing. From 1996 to 2000, 75% of children ≤10 years of age and 90.3% of those >10 years of age received a transplant

within 5 years of initiation; from 2001 to 2005, the numbers fell to 70% and 79%, respectively.[1]

- The median age of the population with ESRD is 57.9 years (58.8 years for whites, 56.3 years for blacks, 55.4 years for Hispanics, 58.3 years for Asians, and 56.5 years for Native Americans).[1] Treatment of ESRD is more common in men than in women.
- Blacks and Native Americans have much higher rates of ESRD than do whites and Asians. Blacks represent nearly 29% of treated ESRD patients.[1]
- Without treatment, ESRD is fatal. Even with dialysis treatment, 18% of ESRD patients die yearly.[7]
- The percentage of hemodialysis patients with a urea reduction ratio ≥65 increased from 84% in 2001 to 88% in 2005.[7]

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 our healthcare system. Controversy exists over 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 CVD risk. The US Renal Data System estimates that by 2010, 650 000 Americans will require treatment for kidney failure,[8,9] representing a 60% increase from those who received such treatment in 2001.[10]
- The NKF K/DOQI developed guidelines providing a standardized definition for CKD in 2002. 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.[11]
- The prevalence of CKD (stages I to V)[1] is 16.8%. This represents an increase from the 14.5% prevalence estimate from NHANES 1988–1994 (NCHS) (recalculated).
- The prevalence of GFR ≥90 mL/min per 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 per 1.73 m^2 with kidney damage) is 5.4%.
- The prevalence of stage III CKD (eGFR 30 to 59 mL/min per 1.73 m^2) is 5.4%.
- The prevalence of stages IV and V CKD (eGFR <29 mL/min per 1.73 m^2) is 0.4%.
- Nearly 26 million people in the United States have CKD, and another 20 million are at increased risk for CKD.[12]
- Self-reported awareness of poor kidney function is associated with the degree of CKD; in 1999–2000, 24.3% were aware of their disease with an eGFR of 15 to 59 mL/min per 1.73 m^2 and albuminuria, whereas 1.1% were aware of decreased kidney function with an eGFR ≥90 mL/ min per 1.73 m^2 and no albuminuria.[9]

Demographics

- The prevalence of CKD rose as age increased as follows[11]:
  - 8.5% for those 20 to 39 years of age;
  - 12.6% for those 40 to 59 years of age; and
  - 39.4% 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%).[11]
- CKD prevalence was greater among those with diabetes (40.2%), hypertension (24.6%), and CVD (28.2%) than among those without these chronic conditions.[11]
- 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.[11]

Risk Factors

- Many traditional CVD risk factors are also risk factors for CKD, including older age, male sex, hypertension, diabetes, 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.[13]
- Many clinical risk factors for CKD are the same as those for CVD.
- Proteinuria is a strong independent risk factor for decline in eGFR, regardless of diabetes status, and is associated with many of the same CVD risk factors as those for CKD.[14,15]

ESRD/CKD and CVD

- CVD is the leading cause of death for 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.[16,17]
  - Individuals with less severe forms of kidney disease are also at significantly increased risk.[16]
  - CKD is a risk factor for recurrent cardiovascular events.[18]
  - Management of CVD differs and is more complex in patients with CKD.[19]
- Studies from a broad range of cohorts demonstrate an association between reduced eGFR and elevated risk of CVD, CVD outcomes, and all-cause death,[20–26] 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, and all-cause death in a wide variety of cohorts.[27–31]
- A number of consensus documents, including statements from the NKF Task Force[32] and American Heart Association (2003),[16] 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.[12]
- 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 1980–2005, from 25.0 to 66.6 per 10 000 in men and from 17.8 to 45.8 per 10 000 in women.[12]

Cost–ESRD

- The total annual cost of treating ESRD in the United States was approximately $33 billion in 2005.[12]

References

1. US Renal Data System. *USRDS 2007 Annual Data Report: Atlas of Chronic Kidney Disease and End-Stage Renal Disease in the United States*. Bethesda, Md: National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases; 2007. Available at: http://www.usrds.org/adr.htm. Accessed August 20, 2008.
2. Centers for Disease Control and Prevention (CDC). Racial differences in trends of end-stage renal disease, by primary diagnosis: United States, 1994–2004. *MMWR Morb Mortal Wkly Rep*. 2007;56:253–256.
3. Centers for Disease Control and Prevention (CDC). Incidence of end-stage renal disease among persons with diabetes: United States, 1990–2002. *MMWR Morb Mortal Wkly Rep*. 2005;54:1097–1100.
4. Collins AJ, Li S, Gilbertson DT, Liu J, Chen SC, Herzog CA. Chronic kidney disease and cardiovascular disease in the Medicare population. *Kidney Int Suppl*. 2003;87:S24–S31.
5. Hsu CY, McCulloch CE, Darbinian J, Go AS, Iribarren C. Elevated blood pressure and risk of end-stage renal disease in subjects without baseline kidney disease. *Arch Intern Med*. 2005;165:923–928.
6. Hsu CY, McCulloch CE, Iribarren C, Darbinian J, Go AS. Body mass index and risk for end-stage renal disease. *Ann Intern Med*. 2006;144:21–28.
7. Agency for Healthcare Research and Quality. 2007 National healthcare quality and disparities reports. Available at: http://www.ahrq.gov/qual/qrdr07.htm#toc. Accessed September 1, 2008.
8. Xue JL, Ma JZ, Louis TA, Collins AJ. Forecast of the number of patients with end-stage renal disease in the United States to the year 2010. *J Am Soc Nephrol*. 2001;12:2753–2758.
9. Coresh J, Byrd-Holt D, Astor BC, Briggs JP, Eggers PW, Lacher DA, Hostetter TH. Chronic kidney disease awareness, prevalence, and trends among U.S. adults, 1999 to 2000. *J Am Soc Nephrol*. 2005;16:180–188.
10. United Sates Renal Data System. *USRDS 2003 Annual Data Report: Atlas of End-Stage Renal Disease in the United States*. Bethesda, Md: National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases; 2003. Available at: http://www.usrds.org/adr_2003.htm. Accessed November 2, 2007.
11. Centers for Disease Control and Prevention (CDC). Prevalence of chronic kidney disease and associated risk factors: United States, 1999–2004. *MMWR Morb Mortal Wkly Rep*. 2007;56:161–165.
12. Centers for Disease Control and Prevention (CDC). Hospitalization discharge diagnoses for kidney disease–United States, 1980–2005. *MMWR Morb Mortal Wkly Rep*. 2008;57:309–312.
13. Coresh J, Astor B, Sarnak MJ. Evidence for increased cardiovascular disease risk in patients with chronic kidney disease. *Curr Opin Nephrol Hypertens*. 2004;13:73–81.
14. Sarnak MJ, Coronado BE, Greene T, Wang SR, Kusek JW, Beck GJ, Levey AS. Cardiovascular disease risk factors in chronic renal insufficiency. *Clin Nephrol*. 2002;57:327–335.
15. Bianchi S, Bigazzi R, Campese VM. Microalbuminuria in essential hypertension: significance, pathophysiology, and therapeutic implications. *Am J Kidney Dis*. 1999;34:973–995.
16. Sarnak MJ, Levey AS, Schoolwerth AC, Coresh J, Culleton B, Hamm LL, McCullough PA, Kasiske BL, Kelepouris E, Klag MJ, Parfrey P, Pfeffer M, Raij L, Spinosa DJ, Wilson PW, for the American Heart Association Councils on Kidney in Cardiovascular Disease, High Blood Pressure Research, Clinical Cardiology, and Epidemiology and Prevention. Kidney disease as a risk factor for development of cardiovascular disease: a statement from the American Heart Association Councils on Kidney in Cardiovascular Disease, High Blood Pressure Research, Clinical Cardiology, and Epidemiology and Prevention. *Circulation*. 2003;108:2154–2169.
17. Weiner DE, Tabatabai S, Tighiouart H, Elsayed E, Bansal N, Griffith J, Salem DN, Levey AS, Sarnak MJ. Cardiovascular outcomes and all-cause mortality: exploring the interaction between CKD and cardiovascular disease. *Am J Kidney Dis*. 2006;48:392–401.
18. Weiner DE, Tighiouart H, Stark PC, Amin MG, MacLeod B, Griffith JL, Salem DN, Levey AS, Sarnak MJ. Kidney disease as a risk factor for recurrent cardiovascular disease and mortality. *Am J Kidney Dis*. 2004;44:198–206.
19. Gupta R, Birnbaum Y, Uretsky BF. The renal patient with coronary artery disease: current concepts and dilemmas. *J Am Coll Cardiol*. 2004;44:1343–1353.
20. Mann JF, Gerstein HC, Pogue J, Bosch J, Yusuf S. Renal insufficiency as a predictor of cardiovascular outcomes and the impact of ramipril: the HOPE randomized trial. *Ann Intern Med*. 2001;134:629–636.
21. Fried LF, Shlipak MG, Crump C, Bleyer AJ, Gottdiener JS, Kronmal RA, Kuller LH, Newman AB. Renal insufficiency as a predictor of cardiovascular outcomes and mortality in elderly individuals. *J Am Coll Cardiol*. 2003;41:1364–1372.
22. Shlipak MG, Fried LF, Cushman M, Manolio TA, Peterson D, Stehman-Breen C, Bleyer A, Newman A, Siscovick D, Psaty B. Cardiovascular mortality risk in chronic kidney disease: comparison of traditional and novel risk factors. *JAMA*. 2005;293:1737–1745.
23. Ruilope LM, Salvetti A, Jamerson K, Hansson L, Warnold I, Wedel H, Zanchetti A. Renal function and intensive lowering of blood pressure in hypertensive participants of the Hypertension Optimal Treatment (HOT) study. *J Am Soc Nephrol*. 2001;12:218–225.
24. Manjunath G, Tighiouart H, Ibrahim H, MacLeod B, Salem DN, Griffith JL, Coresh J, Levey AS, Sarnak MJ. Level of kidney function as a risk factor for atherosclerotic cardiovascular outcomes in the community. *J Am Coll Cardiol*. 2003;41:47–55.
25. Hailpern SM, Cohen HW, Alderman MH. Renal dysfunction and ischemic heart disease mortality in hypertensive population. *J Hypertens*. 2005;23:1809–1816.
26. Culleton BF, Larson MG, Wilson PW, Evans JC, Parfrey PS, Levy D. Cardiovascular disease and mortality in a community-based cohort with mild renal insufficiency. *Kidney Int*. 1999;56:2214–2219.
27. Arnlöv J, Evans JC, Meigs JB, Wang TJ, Fox CS, Levy D, Benjamin EJ, D'Agostino RB, Vasan RS. Low-grade albuminuria and incidence of cardiovascular disease events in nonhypertensive and nondiabetic individuals: the Framingham Heart Study. *Circulation*. 2005;112:969–975.
28. Klausen K, Borch-Johnsen K, Feldt-Rasmussen B, Jensen G, Clausen P, Scharling H, Appleyard M, Jensen JS. Very low levels of microalbuminuria are associated with increased risk of coronary heart disease and death independently of renal function, hypertension, and diabetes. *Circulation*. 2004;110:32–35.
29. Gerstein HC, Mann JF, Yi Q, Zinman B, Dinneen SF, Hoogwerf B, Hallé JP, Young J, Rashkow A, Joyce C, Nawaz S, Yusuf S, for the HOPE Study Investigators. Albuminuria and risk of cardiovascular events, death, and heart failure in diabetic and nondiabetic individuals. *JAMA*. 2001;286:421–426.
30. Hillege HL, Fidler V, Diercks GF, van Gilst WH, de Zeeuw D, van Veldhuisen DJ, Gans RO, Janssen WM, Grobbee DE, de Jong PE, for the Prevention of Renal and Vascular End Stage Disease (PREVEND) Study Group. Urinary albumin excretion predicts cardiovascular and noncardiovascular mortality in general population. *Circulation*. 2002;106:1777–1782.
31. Yuyun MF, Adler AI, Wareham NJ. What is the evidence that microalbuminuria is a predictor of cardiovascular disease events? *Curr Opin Nephrol Hypertens*. 2005;14:271–276.
32. National Kidney Foundation. K/DOQI clinical practice guidelines for chronic kidney disease: evaluation, classification, and stratification. *Am J Kidney Dis*. 2002;39(suppl 1):S1–S266.

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)

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)

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 National Cholesterol Education Program Adult Treatment Panel III (ATP III) definition and its 2 subsequent revisions have been most commonly used. By this definition, MetS is diagnosed when ≥3 of the following 5 risk factors are present[1]:
  - Fasting plasma glucose ≥100 mg/dL or on drug treatment for elevated glucose.
  - HDL cholesterol <40 mg/dL in men or <50 mg/dL in women or on drug treatment for reduced HDL cholesterol.
  - Triglycerides ≥150 mg/dL or on 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 drug treatment for hypertension or on antihypertensive drug treatment in a patient with a history of hypertension.
- The clinical utility of MetS continues to be the subject of vigorous debate.[2,3]

Adults

- On the basis of NHANES 1999–2002 data, the age-adjusted prevalence of MetS according to the 2004 AHA/NHLBI revision of the 2001 ATP III definition for adults[2] was 34.6%.[3]
  - Applying the unadjusted prevalence of 34.5% to the 2007 population estimates for US adults ≥20 years of age yields an estimated 76 million US residents with MetS.

ABBREVIATIONS USED IN CHAPTER 16

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
CI	confidence interval
cm	centimeter
CVD	cardiovascular disease
DM	diabetes mellitus
MetS	metabolic syndrome
mg/dL	milligrams per deciliter
mm Hg	millimeter of mercury
NHANES	National Health and Nutrition Examination Survey
NHLBI	National Heart, Lung, and Blood Institute
OR	odds ratio
RR	relative risk

  - The age-adjusted prevalence was similar for men (34.4%) and women (34.5%).
  - Among men, the age-specific prevalence ranged from 14.9% among people 20 to 29 years of age to 51.6% for people 60 to 69 years of age and 46.6% for those ≥70 years of age. Among women, the age-specific prevalence ranged from 12.1% among people 20 to 29 years of age to 60.9% for people 60 to 69 years of age and 57.8% for those ≥70 years of age.
  - The age-adjusted prevalences of people with MetS were 35.4%, 24.5%, and 40.3% for white, black, and Mexican American men, respectively. For women, the percentages were 31.5%, 36.4%, and 44.0%, respectively.
  - The age-adjusted prevalence was ≈49% higher among black women than men and ≈9% higher among Mexican American women than men.

Children/Adolescents

- On the basis of NHANES 1999–2002 data, the prevalence of the MetS in adolescents 12 to 19 years of age was 9.4%, representing ≈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.[4]
- In 1999–2004, ≈4.5% of United States adolescents 12 to 17 years of age had the MetS according to the definition developed by the International Diabetes Federation.[5] In 2006, this prevalence would have represented ≈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 the MetS. The prevalence of MetS remained relatively stable during successive 2-year periods: 4.5% for 1999 to 2000, 4.4% to 4.5% for 2001 to 2002, and 3.7% to 3.9% for 2003 to 2004.
- Among overweight or obese adolescents, 44% had the MetS.[4] Two thirds of all adolescents had at least 1 metabolic abnormality.[6]
- MetS categorization in adolescents is not stable. Approximately half of 1098 adolescent participants in the Princeton School District Study diagnosed with pediatric ATP III MetS lost the diagnosis over 3 years of follow-up.[7]
- Of 31 participants in the National Heart, Lung, and Blood Institute Lipid Research Clinics Princeton Prevalence Study and the Princeton Follow-up Study who had MetS at baseline, 21 (68%) had MetS 25 years later.[8] After adjustment for age, sex, and race, the baseline status of MetS was significantly associated with an increased risk of having the 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 high-density lipoprotein cholesterol, and mean arterial pressure) considered to be part of the MetS clustered together in blacks and whites and in children and adults.[9] 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.

Risk

Adults

- Consistent with 2 earlier meta-analyses, a recent meta-analysis of prospective studies concluded that the MetS increased the risk of developing CVD (summary RR, 1.78; 95% CI, 1.58 to 2.00).[10] The risk of CVD tended to be higher in women (summary RR, 2.63) than men (summary RR, 1.98) ($P=0.09$). On the basis of results from 3 studies, the 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 Framingham Risk Score is a better predictor of incident CVD than the MetS.[11–13] In the San Antonio Heart Study, the area under the receiver-operating characteristic curve (aROC) was 0.816 for the Framingham Risk Score and 0.811 for the Framingham Risk Score plus the MetS.[11] Furthermore, the sensitivity for CVD at a fixed specificity was significantly higher for the Framingham Risk Score than MetS. In the Atherosclerosis Risk in Communities Study, the MetS did not improve the risk prediction achieved by the Framingham Risk Score.[12] In the British Regional Heart Study, the aROC for the Framingham Risk Score was 0.73 for incident coronary heart disease during 10 years of follow-up, and the aROC for the number of MetS components was 0.63.[13] For coronary heart disease events during 20 years of follow-up, the aROCs were 0.68 for the Framingham Risk Score 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.[13,14] Compared with men without an abnormal component in the Framingham Offspring Study, the hazard ratios 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.[14] Among women, the hazard ratios 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 hazard ratios 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.[13]

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 National Heart, Lung, and Blood Institute 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 the MetS than among those without this syndrome (OR, 14.6; 95% CI, 4.8 to 45.3) who were followed up for 25 years.[8]
- Another analysis of 814 participants of this cohort showed that those 5 to 19 years of age who had the 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).[15]

Risk Factors

- In prospective or retrospective cohort studies, the following factors have been reported as being directly associated with incident MetS defined using one of the major definitions: age,[15–18] low educational attainment,[16,19] smoking,[19,20,21] low levels of physical activity,[19–24] low levels of physical fitness,[22,25–27] intake of soft drinks,[28] intake of diet soda,[29] magnesium intake,[30] energy intake,[24] carbohydrate intake,[16,20,31] total fat intake,[16,31] Western dietary pattern,[29] meat intake,[29] intake of fried foods,[29] heavy alcohol consumption,[32] abstention from alcohol use,[16] parental history of diabetes,[15] chronic stress at work,[33] pediatric MetS,[15] obesity or BMI,[16,17,20,24,34] childhood obesity,[35] waist circumference,[18,31,36–39] intraabdominal fat,[40] gain in weight or BMI,[16,41] change in weight or BMI,[18,20,42] weight fluctuation,[43] blood pressure,[18,31,38,44] heart rate,[45] homeostasis model assessment,[39,46] fasting insulin,[36] 2-hour insulin,[36] proinsulin,[36] fasting glucose or hyperglycemia,[18,36,38] 2-hour glucose,[36] impaired glucose tolerance,[36] triglycerides,[18,31,34,36–38] low high-density lipoprotein cholesterol,[18,31,34,36,38] oxidized low-density lipoprotein,[47] uric acid,[42,48] gamma-glutamyltransferase,[42,49,50] alanine transaminase,[42,49,51,52] plasminogen activator inhibitor-1,[53] aldosterone,[53] leptin,[54] C-reactive protein,[55,56] adipocyte–fatty acid binding protein,[46] and free testosterone index.[57]
- The following factors have been reported as being inversely associated with incident MetS defined using one of the major definitions in prospective or retrospective cohort studies: muscular strength,[58] change in physical activity or fitness,[20,25] alcohol intake,[19,24] Mediterranean diet,[59] dairy consumption,[29] insulin sensitivity,[36] ratio of aspartate aminotransferase to alanine transaminase,[51] total testosterone,[57,60,61] sex hormone–binding globulin,[57,60,61] and delta(5)-desaturase activity.[62]
- Furthermore, men were more likely to develop the MetS than were women,[16,18] and blacks were shown to be less likely to develop the MetS than were whites.[16]

References

1. Grundy SM, Cleeman JI, Daniels SR, Donato KA, Eckel RH, Franklin BA, Gordon DJ, Krauss RM, Savage PJ, Smith SC Jr, Spertus JA, Costa F, for the American Heart Association; National Heart, Lung, and Blood Institute. Diagnosis and management of the metabolic syndrome: an American Heart Association/National Heart, Lung, and Blood Institute Scientific Statement [published corrections appear in *Circulation*. 2005; 112:e297 and *Circulation*. 2005;112:e298]. *Circulation*. 2005;112: 2735–2752.
2. Grundy SM, Brewer HB Jr, Cleeman JI, Smith SC Jr, Lenfant C, for the American Heart Association; National Heart, Lung, and Blood Institute. Definition of metabolic syndrome: report of the National Heart, Lung, and Blood Institute/American Heart Association conference on scientific issues related to definition. *Circulation*. 2004;109:433–438.
3. Ford ES. Prevalence of the metabolic syndrome defined by the International Diabetes Federation among adults in the U.S. *Diabetes Care*. 2005;28:2745–2749.

4. Cook S, Auinger P, Li C, Ford ES. Metabolic syndrome rates in United States adolescents, from the National Health and Nutrition Examination Survey, 1999–2002. *J Pediatr*. 2008;152:165–170.
5. Ford ES, Li C, Zhao G, Pearson WS, Mokdad AH. Prevalence of metabolic syndrome among U.S. adolescents using the definition from the International Diabetes Federation. *Diabetes Care*. 2008;31:587–589.
6. de Ferranti SD, Gauvreau K, Ludwig DS, Neufeld EJ, Newburger JW, Rifai N. Prevalence of the metabolic syndrome in American adolescents: findings from the Third National Health and Nutrition Examination Survey. *Circulation*. 2004;110:2494–2497.
7. Goodman E, Daniels SR, Meigs JB, Dolan LM. Instability in the diagnosis of metabolic syndrome in adolescents. *Circulation*. 2007;115:2316–2322.
8. Morrison JA, Friedman LA, Gray-McGuire C. Metabolic syndrome in childhood predicts adult cardiovascular disease 25 years later: the Princeton Lipid Research Clinics Follow-Up Study. *Pediatrics*. 2007;120:340–345.
9. Chen W, Srinivasan SR, Li S, Xu J, Berenson GS. Clustering of long-term trends in metabolic syndrome variables from childhood to adulthood in blacks and whites: the Bogalusa Heart Study. *Am J Epidemiol*. 2007;166:527–533.
10. Gami AS, Witt BJ, Howard DE, Erwin PJ, Gami LA, Somers VK, Montori VM. Metabolic syndrome and risk of incident cardiovascular events and death: a systematic review and meta-analysis of longitudinal studies. *J Am Coll Cardiol*. 2007;49:403–414.
11. Stern MP, Williams K, Gonzalez-Villalpando C, Hunt KJ, Haffner SM. Does the metabolic syndrome improve identification of individuals at risk of type 2 diabetes and/or cardiovascular disease? *Diabetes Care*. 2004;27:2676–2681.
12. McNeill AM, Rosamond WD, Girman CJ, Golden SH, Schmidt MI, East HE, Ballantyne CM, Heiss G. The metabolic syndrome and 11-year risk of incident cardiovascular disease in the Atherosclerosis Risk in Communities study. *Diabetes Care*. 2005;28:385–390.
13. Wannamethee SG, Shaper AG, Lennon L, Morris RW. Metabolic syndrome vs Framingham Risk Score for prediction of coronary heart disease, stroke, and type 2 diabetes mellitus. *Arch Intern Med*. 2005;165:2644–2650.
14. Wilson PW, D'Agostino RB, Parise H, Sullivan L, Meigs JB. Metabolic syndrome as a precursor of cardiovascular disease and type 2 diabetes mellitus. *Circulation*. 2005;112:3066–3072.
15. Morrison JA, Friedman LA, Wang P, Glueck CJ. Metabolic syndrome in childhood predicts adult metabolic syndrome and type 2 diabetes mellitus 25 to 30 years later. *J Pediatr*. 2008;152:201–206.
16. Carnethon MR, Loria CM, Hill JO, Sidney S, Savage PJ, Liu K, for the Coronary Artery Risk Development in Young Adults study. Risk factors for the metabolic syndrome: the Coronary Artery Risk Development in Young Adults (CARDIA) study, 1985–2001. *Diabetes Care*. 2004;27:2707–2715.
17. Albareda M, Caballero A, Badell G, Rodriguez-Espinosa J, Ordonez-Llanos J, de Leiva A, Corcoy R. Metabolic syndrome at follow-up in women with and without gestational diabetes mellitus in index pregnancy. *Metabolism*. 2005;54:1115–1121.
18. Cheung BM, Wat NM, Tam S, Thomas GN, Leung GM, Cheng CH, Woo J, Janus ED, Lau CP, Lam TH, Lam KS. Components of the metabolic syndrome predictive of its development: a 6-year longitudinal study in Hong Kong Chinese. *Clin Endocrinol (Oxf)*. 2008;68:730–737.
19. Wilsgaard T, Jacobsen BK. Lifestyle factors and incident metabolic syndrome: the Tromso Study 1979–2001. *Diabetes Res Clin Pract*. 2007;78:217–224.
20. Wannamethee SG, Shaper AG, Whincup PH. Modifiable lifestyle factors and the metabolic syndrome in older men: effects of lifestyle changes. *J Am Geriatr Soc*. 2006;54:1909–1914.
21. Holme I, Tonstad S, Sogaard AJ, Larsen PG, Haheim LL. Leisure time physical activity in middle age predicts the metabolic syndrome in old age: results of a 28-year follow-up of men in the Oslo study. *BMC Public Health*. 2007;7:154.
22. Laaksonen DE, Lakka HM, Salonen JT, Niskanen LK, Rauramaa R, Lakka TA. Low levels of leisure-time physical activity and cardiorespiratory fitness predict development of the metabolic syndrome. *Diabetes Care*. 2002;25:1612–1618.
23. Ekelund U, Brage S, Franks PW, Hennings S, Emms S, Wareham NJ. Physical activity energy expenditure predicts progression toward the metabolic syndrome independently of aerobic fitness in middle-aged healthy Caucasians: the Medical Research Council Ely Study. *Diabetes Care*. 2005;28:1195–1200.
24. Ferreira I, Twisk JW, van MW, Kemper HC, Stehouwer CD. Development of fatness, fitness, and lifestyle from adolescence to the age of 36 years: determinants of the metabolic syndrome in young adults: the Amsterdam Growth and Health Longitudinal Study. *Arch Intern Med*. 2005;165:42–48.
25. Carnethon MR, Gidding SS, Nehgme R, Sidney S, Jacobs DR Jr, Liu K. Cardiorespiratory fitness in young adulthood and the development of cardiovascular disease risk factors. *JAMA*. 2003;290:3092–3100.
26. LaMonte MJ, Barlow CE, Jurca R, Kampert JB, Church TS, Blair SN. Cardiorespiratory fitness is inversely associated with the incidence of metabolic syndrome: a prospective study of men and women. *Circulation*. 2005;112:505–512.
27. Ferreira I, Henry RM, Twisk JW, van Mechelen W, Kemper HC, Stehouwer CD, for the Amsterdam Growth and Health Longitudinal Study. The metabolic syndrome, cardiopulmonary fitness, and subcutaneous trunk fat as independent determinants of arterial stiffness: the Amsterdam Growth and Health Longitudinal Study. *Arch Intern Med*. 2005;165:875–882.
28. Dhingra R, Sullivan L, Jacques PF, Wang TJ, Fox CS, Meigs JB, D'Agostino RB, Gaziano JM, Vasan RS. Soft drink consumption and risk of developing cardiometabolic risk factors and the metabolic syndrome in middle-aged adults in the community. *Circulation*. 2007;116:480–488.
29. Lutsey PL, Steffen LM, Stevens J. Dietary intake and the development of the metabolic syndrome: the Atherosclerosis Risk in Communities study. *Circulation*. 2008;117:754–761.
30. He K, Liu K, Daviglus ML, Morris SJ, Loria CM, Van Horn L, Jacobs DR Jr, Savage PJ. Magnesium intake and incidence of metabolic syndrome among young adults. *Circulation*. 2006;113:1675–1682.
31. Mirmiran P, Noori N, Azizi F. A prospective study of determinants of the metabolic syndrome in adults. *Nutr Metab Cardiovasc Dis*. 2008;18:567–573.
32. Baik I, Shin C. Prospective study of alcohol consumption and metabolic syndrome. *Am J Clin Nutr*. 2008;87:1455–1463.
33. Chandola T, Brunner E, Marmot M. Chronic stress at work and the metabolic syndrome: prospective study. *BMJ*. 2006;332:521–525.
34. Lim HS, Lip GY, Beevers DG, Blann AD. Factors predicting the development of metabolic syndrome and type II diabetes against a background of hypertension. *Eur J Clin Invest*. 2005;35:324–329.
35. Sun SS, Liang R, Huang TT, Daniels SR, Arslanian S, Liu K, Grave GD, Siervogel RM. Childhood obesity predicts adult metabolic syndrome: the Fels Longitudinal Study. *J Pediatr*. 2008;152:191–200.
36. Palaniappan L, Carnethon MR, Wang Y, Hanley AJ, Fortmann SP, Haffner SM, Wagenknecht L, for the Insulin Resistance Atherosclerosis Study. Predictors of the incident metabolic syndrome in adults: the Insulin Resistance Atherosclerosis Study. *Diabetes Care*. 2004;27:788–793.
37. Morrison JA, Friedman LA, Harlan WR, Harlan LC, Barton BA, Schreiber GB, Klein DJ. Development of the metabolic syndrome in black and white adolescent girls: a longitudinal assessment. *Pediatrics*. 2005;116:1178–1182.
38. Sheu WH, Chuang SY, Lee WJ, Tsai ST, Chou P, Chen CH. Predictors of incident diabetes, metabolic syndrome in middle-aged adults: a 10-year follow-up study from Kinmen, Taiwan. *Diabetes Res Clin Pract*. 2006;74:162–168.
39. Onat A, Uyarel H, Hergenc G, Karabulut A, Albayrak S, Can G. Determinants and definition of abdominal obesity as related to risk of diabetes, metabolic syndrome and coronary disease in Turkish men: a prospective cohort study. *Atherosclerosis*. 2007;191:182–190.
40. Tong J, Boyko EJ, Utzschneider KM, McNeely MJ, Hayashi T, Carr DB, Wallace TM, Zraika S, Gerchman F, Leonetti DL, Fujimoto WY, Kahn SE. Intra-abdominal fat accumulation predicts the development of the metabolic syndrome in non-diabetic Japanese-Americans. *Diabetologia*. 2007;50:1156–1160.
41. Lloyd-Jones DM, Liu K, Colangelo LA, Yan LL, Klein L, Loria CM, Lewis CE, Savage P. Consistently stable or decreased body mass index in young adulthood and longitudinal changes in metabolic syndrome components: the Coronary Artery Risk Development in Young Adults Study. *Circulation*. 2007;115:1004–1011.
42. Ryu S, Song J, Choi BY, Lee SJ, Kim WS, Chang Y, Kim DI, Suh BS, Sung KC. Incidence and risk factors for metabolic syndrome in Korean male workers, ages 30 to 39. *Ann Epidemiol*. 2007;17:245–252.
43. Vergnaud AC, Bertrais S, Oppert JM, Maillard-Teyssier L, Galan P, Hercberg S, Czernichow S. Weight fluctuations and risk for metabolic syndrome in an adult cohort. *Int J Obes (Lond)*. 2008;32:315–321.

44. Sun SS, Grave GD, Siervogel RM, Pickoff AA, Arslanian SS, Daniels SR. Systolic blood pressure in childhood predicts hypertension and metabolic syndrome later in life. *Pediatrics*. 2007;119:237–246.
45. Tomiyama H, Yamada J, Koji Y, Yambe M, Motobe K, Shiina K, Yamamoto Y, Yamashina A. Heart rate elevation precedes the development of metabolic syndrome in Japanese men: a prospective study. *Hypertens Res*. 2007;30:417–426.
46. Xu A, Tso AW, Cheung BM, Wang Y, Wat NM, Fong CH, Yeung DC, Janus ED, Sham PC, Lam KS. Circulating adipocyte–fatty acid binding protein levels predict the development of the metabolic syndrome: a 5-year prospective study. *Circulation*. 2007;115:1537–1543.
47. Holvoet P, Lee DH, Steffes M, Gross M, Jacobs DR Jr. Association between circulating oxidized low-density lipoprotein and incidence of the metabolic syndrome. *JAMA*. 2008;299:2287–2293.
48. Sui X, Church TS, Meriwether RA, Lobelo F, Blair SN. Uric acid and the development of metabolic syndrome in women and men. *Metabolism*. 2008;57:845–852.
49. Andre P, Balkau B, Vol S, Charles MA, Eschwege E, for the DESIR Study Group. Gamma-glutamyltransferase activity and development of the metabolic syndrome (International Diabetes Federation Definition) in middle-aged men and women: data from the Epidemiological Study on the Insulin Resistance Syndrome (DESIR) cohort. *Diabetes Care*. 2007;30:2355–2361.
50. Lee DS, Evans JC, Robins SJ, Wilson PW, Albano I, Fox CS, Wang TJ, Benjamin EJ, D'Agostino RB, Vasan RS. Gamma glutamyl transferase and metabolic syndrome, cardiovascular disease, and mortality risk: the Framingham Heart Study. *Arterioscler Thromb Vasc Biol*. 2007;27:127–133.
51. Hanley AJ, Williams K, Festa A, Wagenknecht LE, D'Agostino RB Jr, Haffner SM. Liver markers and development of the metabolic syndrome: the Insulin Resistance Atherosclerosis study. *Diabetes*. 2005;54:3140–3147.
52. Schindhelm RK, Dekker JM, Nijpels G, Stehouwer CD, Bouter LM, Heine RJ, Diamant M. Alanine aminotransferase and the 6-year risk of the metabolic syndrome in Caucasian men and women: the Hoorn Study. *Diabet Med*. 2007;24:430–435.
53. Ingelsson E, Pencina MJ, Tofler GH, Benjamin EJ, Lanier KJ, Jacques PF, Fox CS, Meigs JB, Levy D, Larson MG, Selhub J, D'Agostino RB Sr, Wang TJ, Vasan RS. Multimarker approach to evaluate the incidence of the metabolic syndrome and longitudinal changes in metabolic risk factors: the Framingham Offspring Study. *Circulation*. 2007;116:984–992.
54. Galletti F, Barbato A, Versiero M, Iacone R, Russo O, Barba G, Siani A, Cappuccio FP, Farinaro E, della Valle E, Strazzullo P. Circulating leptin levels predict the development of metabolic syndrome in middle-aged men: an 8-year follow-up study. *J Hypertens*. 2007;25:1671–1677.
55. Laaksonen DE, Niskanen L, Nyyssonen K, Punnonen K, Tuomainen TP, Valkonen VP, Salonen R, Salonen JT. C-reactive protein and the development of the metabolic syndrome and diabetes in middle-aged men. *Diabetologia*. 2004;47:1403–1410.
56. Hassinen M, Lakka TA, Komulainen P, Gylling H, Nissinen A, Rauramaa R. C-reactive protein and metabolic syndrome in elderly women: a 12-year follow-up study. *Diabetes Care*. 2006;29:931–932.
57. Rodriguez A, Muller DC, Metter EJ, Maggio M, Harman SM, Blackman MR, Andres R. Aging, androgens, and the metabolic syndrome in a longitudinal study of aging. *J Clin Endocrinol Metab*. 2007;92:3568–3572.
58. Jurca R, Lamonte MJ, Barlow CE, Kampert JB, Church TS, Blair SN. Association of muscular strength with incidence of metabolic syndrome in men. *Med Sci Sports Exerc*. 2005;37:1849–1855.
59. Tortosa A, Bes-Rastrollo M, Sanchez-Villegas A, Basterra-Gortari FJ, Nunez-Cordoba JM, Martinez-Gonzalez MA. Mediterranean diet inversely associated with the incidence of metabolic syndrome: the SUN prospective cohort. *Diabetes Care*. 2007;30:2957–2959.
60. Laaksonen DE, Niskanen L, Punnonen K, Nyyssonen K, Tuomainen TP, Valkonen VP, Salonen R, Salonen JT. Testosterone and sex hormone-binding globulin predict the metabolic syndrome and diabetes in middle-aged men. *Diabetes Care*. 2004;27:1036–1041.
61. Kupelian V, Page ST, Araujo AB, Travison TG, Bremner WJ, McKinlay JB. Low sex hormone-binding globulin, total testosterone, and symptomatic androgen deficiency are associated with development of the metabolic syndrome in nonobese men. *J Clin Endocrinol Metab*. 2006;91:843–850.
62. Warensjo E, Riserus U, Vessby B. Fatty acid composition of serum lipids predicts the development of the metabolic syndrome in men. *Diabetologia*. 2005;48:1999–2005.

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 Dariush Mozaffarian.*

Abbreviations Used in Chapter 17

AHEI	Alternative Health Eating Index
ALA	alpha-linoleic acid
apo	apolipoprotein
BMI	body mass index
BRFSS	Behavioral Risk Factor Surveillance System
CDC	Centers for Disease Control and Prevention
CHD	coronary heart disease
CHS	Cardiovascular Health Study
CI	confidence interval
CPI	Consumer Price Index
CVD	cardiovascular disease
DART	Diet and Reinfarction Trial
DASH	Dietary Approaches to Stop Hypertension
DGA	Dietary Guidelines for Americans
DHA	docosahexaenoic acid.
EPA	eicosapentaenoic acid
g	gram
HOMA	homeostasis model assessment
HDL	high-density lipoprotein
HEI	Healthy Eating Index
ISSFAL	International Society for the Study of Fatty Acids and Lipids
kcal	kilocalories
LDL	low-density lipoprotein
mg	milligram
Mm Hg	millimeter of mercury
NCHS	National Center for Health Statistics
NHANES	National Health and Nutrition Examination Survey
PA	physical activity
pmol/L	picomoles per liter
PUFA	polyunsaturated fat
RR	relative risk
TOHP	Trials of Hypertension Prevention
μg	microgram
USDA	US Department of Agriculture
YRBS	Youth Risk Behavioral Surveillance

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. Including 100% fruit juices, servings consumed and 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. Including 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 <2 servings per week. About 6% of whites, 7% of blacks, and 6% to 7% of Mexican Americans consumed ≥500 mg/d of EPA+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. About 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. About 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). About two thirds of white and black men and women; and half of 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/d of dietary cholesterol.

- About 3% to 7% of whites, 2% to 3% of blacks, and 11% to 12% of Mexican Americans consumed ≥28 g/d of dietary fiber.
- About 7% to 13% of whites, 9% to 10% of blacks, and 17% to 24% of Mexican Americans consumed <2.3 g/d of sodium.

Foods and Nutrients: Children and Teenagers

See Table 17-2*; NHANES 2005–2006; personal communication with Dariush Mozaffarian.*

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, servings consumed approximately doubled or tripled, and proportions consuming ≥4 servings per day were 18% to 19% in those 5 to 9 years of age, 16% in those 10 to 14 years of age, and 10% to 14% in 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 compared with girls and was about 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. About one fifth to one third of children consumed <10% energy from saturated fat; and ≈80% consumed <300 mg/d of dietary cholesterol.
- 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, one individual may consume relatively high calories but have negative energy balance (as a result of even greater calories expended), whereas another individual may consume relatively few calories but have positive energy balance (because of low calories expended). Given such variation, the most practical and reasonable method to assess energy balance is to assess changes in weight over time (see Trends below).

- Average total caloric intake in the United States is ≈2500 calories in adult men and 1800 calories in adult women (see Table 17-1). In children and teenagers, average caloric intake is higher in boys versus girls and increases with age in boys (see Table 17-2).
- Individual nutritional determinants of positive energy balance (more calories consumed than expended), as determined by adiposity or weight gain, include larger portion sizes,[1,2] higher intake of sugar-sweetened beverages,[3,4] and greater consumption of fast food and commercially prepared meals.[5–9]
- Each of these dietary factors has multiple influences; eg, preferences for portion size are related to body mass index, socioeconomic status, eating in fast food restaurants, and television watching.[10,11] Portion sizes are largest at fast food restaurants compared with home or other restaurants.[12]
- Between 1999 and 2004, 53% of Americans consumed an average of 1 to 3 restaurant meals per week, and 23% consumed ≥4 restaurant meals per week.[13] In 1999–2000, 41% of US adults consumed ≥3 commercially prepared meals per week.[6] In 2004, spending on food away from

home, including restaurant meals, catered affairs, and food on out-of-town trips, accounted for 42% of average annual food expenditures.[13]

- 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.[14–16] Preliminary evidence suggests that aspects of dietary quality, rather than composition, such as extent of processing of carbohydrates consumed,[16] consumption of trans fat,[17–19] and energy density[20–22] 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 focusing on dietary quality and whole foods may be most successful.[23–25]
- Other individual factors associated with positive energy balance (weight gain) include greater television watching (particularly as related to greater food consumption)[26–31] and lower average sleep duration, particularly among children.[32]
- Societal and environmental factors related to energy imbalance (weight gain), as mediated by either caloric consumption or 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.[33–35] 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 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.[36]
- 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.[37]

Dietary Supplements

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

- Half (52%) of US adults in 1999–2000 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 body mass index, and white race.[38]
- One third (32%) of US children (birth to 18 years of age) used dietary supplements in 1999–2002, with highest use (48.5%) among 4- to 8-year-olds. The most common supplements were multivitamins and multiminerals (58% of supplement users). The primary nutrients supplemented included vitamin C (29%), 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 body mass index, and less screen time (television, video games, computers).[39]
- 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 more common in women (44.9%) versus men (19.8%) and in blacks (48.7%) or Hispanics (41.6%) versus whites (31.2%); in those with high school education or less (38.4%) versus some college or more (31.1%); and in those with household income less than $40 000/y (41.8%) versus higher (30.3%).[40]
- Multiple trials of most dietary supplements, including folate, vitamin C, and vitamin E, have generally shown no significant effect on CVD risk. The major exceptions are long-chain omega-3 fatty acids, for which 3 large randomized controlled trials, including populations with and without established heart disease, have shown significant reductions in risk of CVD events at doses of 1 to 2 g/d (GISSI-Prevenzione, JELIS, GISSI-HF).[41–43]

Trends

Energy Balance

Energy balance, or consumption of total calories appropriate for needs, has been steadily worsening in the United States over the past several decades, as evidenced by the dramatic increases in overweight and obesity among both children and adults across broad cross sections of sex, race/ethnicity, geographic residence, and socioeconomic status.[44,45]

- 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) (see 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).[12] The increase in calories consumed is attributable primarily to greater average carbohydrate intake, particularly starches, grains, and sweetened beverages (see Foods and Nutrients below).
- The increases in caloric consumption in the United States relate to changes in several specific dietary factors, includ-

ing larger portion sizes, greater food quantity and calories per meal, and increased consumption of sugar-sweetened beverages, snacks, commercially prepared (especially fast food) meals, and higher energy-density foods.[6,12,46–50]

- 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).[12]
- Among US children 2 to 7 years of age, an estimated energy imbalance of only 110 to 165 kcal/d (the equivalent of one 12- to 16-oz bottle of soda/cola) was sufficient to account for the excess weight gain between 1988–1994 and 1999–2002.[51]
- Spending on food away from home, including restaurant meals, catered foods, and food on out-of-town trips, increased from 26% of average annual food expenditures in 1970 to 42% in 2004.[13] Half of US per capita food expenditures were spent on meals outside the home in 2007.[52]

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.[50] 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 (see Chart 17-1).
- Dietary guidelines during this time also emphasized carbohydrate consumption (eg, as the base of the Food Guide Pyramid),[53] which increased from 42.4% to 48.2% of calories in men and from 45.4% to 50.6% of calories in women (see 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.[54,55]

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, representing an overall absolute increase of 222 cal/d per person.[49] 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 (see 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.[12]
- 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.[50]

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.[56]

Morbidity and Mortality

Effects on Cardiovascular Risk Factors

In randomized controlled trials, dietary habits affect multiple cardiovascular risk factors, including both established risk factors (systolic and diastolic blood pressures, 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 systolic blood pressure by 7.1 mm Hg in adults without hypertension and by 11.5 mm Hg in adults with hypertension.[57]
- 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 varying (27% to 37%) total fat and focused on whole foods such as fruits, vegetables, whole grains, and fish; potassium and other minerals; and low sodium, reduced systolic blood pressure by 8 to 10 mm Hg, diastolic blood pressure by 4 to 5 mm Hg, and LDL cholesterol by 12 to 14 mg/dL. The diets that had higher levels of protein and unsaturated fat also lowered triglyceride levels by 16 and 9 mg/dL, respectively.[58]
- In a meta-analysis of randomized controlled trials, consuming 1% of calories from trans fat in place of saturated fat, monounsaturated fat, or polyunsaturated fat increased the ratio of total to HDL cholesterol by 0.031, 0.054, and 0.67; increased 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, respectively.[59]
- In meta-analyses of randomized controlled trials, consumption of EPA+DHA for ≥12 weeks lowered systolic blood pressure by 2.1 mm Hg[60] and resting heart rate by 2.5 bpm.[61]
- 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 systolic blood pressure 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 ratio by 0.38 and 0.26; and raised HDL cholesterol by 2.9 and 1.6 mg/dL, respectively.[62] 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.[62]

Effects on Cardiovascular Outcomes

Because dietary habits affect a broad range of established and novel risk factors, estimating the impact of nutritional factors on cardiovascular health by considering only a limited number of pathways (eg, only effects on lipids, blood pressure, 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.[63] 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.[64–73]
- In a meta-analysis of randomized controlled trials, increased polyunsaturated fat consumption, in place of saturated fat, reduced CHD risk by 24%.[74]
- 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 (heart disease, 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).[75]
- In a pooled analysis of prospective cohort studies, each 2 servings per day of whole grain consumption were associated with a 21% lower risk of developing type 2 diabetes (RR, 0.79; 95% CI, 0.72 to 0.87).[76]
- Compared with little or no consumption, modest consumption of fish or fish oil (250 mg/d EPA+DHA, the equivalent of 1 to 2 servings per week of oily fish) was associated with a 36% lower risk of cardiac mortality (RR, 0.64; 95% CI, 0.50 to 0.80) in a pooled analysis of prospective cohort studies and randomized controlled trials.[77]
- In a meta-analysis of prospective cohort studies, each 2% of calories from trans fat was associated with 23% higher risk of CHD (RR, 1.23; 95% CI, 1.11 to 1.37).[78]
- Each additional 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 5% lower risk of stroke (RR, 0.95; 95% CI, 0.92 to 0.97) in meta-analyses of prospective cohort studies.[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 resulting from underlying risk (reverse causation). In a posthoc analysis of the Trials of Hypertension Prevention (TOHP) trials, 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]
- 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).[83] 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 22% higher cardiovascular mortality (RR, 1.22; 95% CI, 1.01 to 1.48).[84] Similar findings have been seen in other cohorts and for other outcomes, including development of diabetes and metabolic syndrome.[85–89]

Cost

The US Department of Agriculture forecasts that the Consumer Price Index (CPI) for all food is forecast to increase 4.5% to 5.5% in 2008 as retailers continue 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[53] (see 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.[52]
- 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.[52]
- Among 154 forms of fruits and vegetables priced using ACNeilsen 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.[52]
- 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.[90]

- Each year, more than $33 billion in medical costs and $9 billion in lost productivity resulting from heart disease, cancer, stroke, and diabetes are attributed to poor nutrition.[91–96]

References

1. Ello-Martin JA, Ledikwe JH, Rolls BJ. The influence of food portion size and energy density on energy intake: implications for weight management. *Am J Clin Nutr*. 2005;82:236S–241S.
2. Fisher JO, Kral TV. Super-size me: portion size effects on young children's eating. *Physiol Behav*. 2008;94:39–47.
3. Malik VS, Schulze MB, Hu FB. Intake of sugar-sweetened beverages and weight gain: a systematic review. *Am J Clin Nutr*. 2006;84:274–288.
4. Sichieri R, Paula Trotte A, de Souza RA, Veiga GV. School randomised trial on prevention of excessive weight gain by discouraging students from drinking sodas. *Public Health Nutr*. 2008;18:1–6.
5. Bowman, Vinyard BT. Fast food consumption of U.S. adults: impact on energy and nutrient intakes and overweight status. *J Am Coll Nutr*. 2004;23:163–168.
6. Kant AK, Graubard BI. Eating out in America, 1987–2000: trends and nutritional correlates. *Prev Med*. 2004;38:243–249.
7. Duerksen SC, Elder JP, Arredondo EM, Ayala GX, Slymen DJ, Campbell NR, Baquero B. Family restaurant choices are associated with child and adult overweight status in Mexican American families. *J Am Diet Assoc*. 2007;107:849–853.
8. Duffey KJ, Gordon-Larsen P, Jacobs DR Jr, Williams OD, Popkin BM. Differential associations of fast food and restaurant food consumption with 3-y change in body mass index: the Coronary Artery Risk Development in Young Adults Study. *Am J Clin Nutr*. 2007;85:201–208.
9. Rosenheck R. Fast food consumption and increased caloric intake: a systematic review of a trajectory towards weight gain and obesity risk. *Obes Rev*. March 14, 2008. DOI: 10.1111/j.1467-789X.2008.00477.x. Available at: http://www3.interscience.wiley.com/journal. Accessed March 14, 2008.
10. Burger KS, Kern M, Coleman KJ. Characteristics of self-selected portion size in young adults. *J Am Diet Assoc*. 2007;107:611–618.
11. Colapinto CK, Fitzgerald A, Taper LJ, Veugelers PJ. Children's preference for large portions: prevalence, determinants, and consequences. *J Am Diet Assoc*. 2007;107:1183–1190.
12. Nielsen SJ, Popkin BM. Patterns and trends in food portion sizes, 1977–1998. *JAMA*. 2003;289:450–453.
13. National Center for Health Statistics. *Health, United States 2007, With Chartbook on Trends in the Health of Americans*. Hyattsville, Md: National Center for Health Statistics; 2007.
14. Willett WC, Leibel RL. Dietary fat is not a major determinant of body fat. *Am J Med*. 2002;113(suppl 9B):47S–59S.
15. Brehm BJ, D'Alessio DA. Weight loss and metabolic benefits with diets of varying fat and carbohydrate content: separating the wheat from the chaff. *Nat Clin Pract Endocrinol Metab*. 2008;4:140–146.
16. van Dam RM, Seidell JC. Carbohydrate intake and obesity. *Eur J Clin Nutr*. 2007 ;61(suppl 1):S75–S99.
17. Koh-Banerjee P, Chu NF, Spiegelman D, Rosner B, Colditz G, Willett W, Rimm E. Prospective study of the association of changes in dietary intake, physical activity, alcohol consumption, and smoking with 9-y gain in waist circumference among 16 587 US men. *Am J Clin Nutr*. 2003;78:719–727.
18. Field AE, Willett WC, Lissner L, Colditz GA. Dietary fat and weight gain among women in the Nurses' Health Study. *Obesity (Silver Spring)*. 2007;15:967–976.
19. Kavanagh K, Jones KL, Sawyer J, Kelley K, Carr JJ, Wagner JD, Rudel LL. Trans fat diet induces abdominal obesity and changes in insulin sensitivity in monkeys. *Obesity (Silver Spring)*. 2007;15:1675–1684.
20. Rolls BJ, Roe LS, Beach AM, Kris-Etherton PM. Provision of the foods differing in energy density affects long-term weight loss. *Obes Res*. 2005;13:1052–1056.
21. Ledikuie JH, Rolls BL, Smiciklas-Wright H, Mitchell DC, Ard JD, Champagne C, Karanja N, Lin PH, Stevens VJ, Appel LJ. Reductions in dietary energy density are associated with weight loss in overweight and obese participants in the PREMIER trial. *Am J Clin Nutr*. 2007;85:1212–1221.
22. Ello-Martin JA, Roe LS, Ledikwe JH, Beach AM, Roll BJ. Dietary energy density in the treatment of obesity: a year-long trial comparing 2 weight-loss diets. *Am J Clin Nutr*. 2007;85:1465–1477.
23. Nordmann AJ, Nordmann A, Briel M, Keller U, Yancy WS Jr, Brehm BJ, Bucher HC. Effects of low-carbohydrate vs low-fat diets on weight loss and cardiovascular risk factors: a meta-analysis of randomized controlled trials. *Arch Intern Med*. 2006;166:285–293.
24. Gardner CD, Kiazand A, Alhassan S, Kim S, Stafford RS, Balise RR, Kraemer HC, King AC. Comparison of the Atkins, Zone, Ornish, and LEARN diets for change in weight and related risk factors among overweight premenopausal women: the A TO Z Weight Loss Study: a randomized trial. *JAMA*. 2007;297:969–977.
25. Shai I, Schwarzfuchs D, Henkin Y, Shahar DR, Witkow S, Greenberg I, Golan R, Fraser D, Bolotin A, Vardi H, Tangi-Rozental O, Zuk-Ramot R, Sarusi B, Brickner D, Schwartz Z, Sheiner E, Marko R, Katorza E, Thiery J, Fiedler GM, Blüher M, Stumvoll M, Stampfer MJ, for the Dietary Intervention Randomized Controlled Trial (DIRECT) Group. Weight loss with a low-carbohydrate, Mediterranean, or low-fat diet. *N Engl J Med*. 2008;359:229–241.
26. Gortmaker SL, Must A, Sobol AM, Peterson K, Colditz GA, Dietz WH. Television viewing as a cause of increasing obesity among children in the United States, 1986–1990. *Arch Pediatr Adolesc Med*. 1996;150:356–362.
27. Robinson TN. Reducing children's television viewing to prevent obesity: a randomized controlled trial. *JAMA*. 1999;282:1561–1567.
28. Gable S, Chang Y, Krull JL. Television watching and frequency of family meals are predictive of overweight onset and persistence in a national sample of school-aged children. *J Am Diet Assoc*. 2007;107:53–61.
29. Temple JL, Giacomelli AM, Kent KM, Roemmich JN, Epstein LH. Television watching increases motivated responding for food and energy intake in children. *Am J Clin Nutr*. 2007;85:355–361.
30. Dubois L, Farmer A, Girard M, Peterson K. Social factors and television use during meals and snacks is associated with higher BMI among pre-school children. *Public Health Nutr*. 2008:11:1267–1279.
31. Epstein LH, Roemmich JN, Robinson JL, Paluch RA, Winiewicz DD, Fuerch JH, Robinson TN. A randomized trial of the effects of reducing television viewing and computer use on body mass index in young children. *Arch Pediatr Adolesc Med*. 2008;162:239–245.
32. Patel SR, Hu FB. Short sleep duration and weight gain: a systematic review. *Obesity (Silver Spring)*. 2008;16:643–653.
33. Kumanyika S, Grier S. Targeting interventions for ethnic minority and low-income populations. *Future Child*. 2006;16:187–207.
34. Sallis JF, Glanz K. The role of built environments in physical activity, eating, and obesity in childhood. *Future Child*. 2006;16:89–108.
35. Li F, Harmer PA, Cardinal BJ, Bosworth M, Acock A, Johnson-Shelton D, Moore JM. Built environment, adiposity, and physical activity in adults aged 50–75. *Am J Prev Med*. 2008;35:38–46.
36. Mellen PB, Gao SK, Vitolins MZ, Goff DC Jr. Deteriorating dietary habits among adults with hypertension: DASH dietary accordance, NHANES 1988–1994 and 1999–2004. *Arch Intern Med*. 2008;168:308–314.
37. Ervin RB. Healthy Eating Index scores among adults, 60 years of age and over, by sociodemographic and health characteristics: United States, 1999–2002. *Advance Data from Vital and Health Statistics*. May 20, 2008. No. 395.
38. Radimer K, Bindewald B, Hughes J, Ervin B, Swanson C, Picciano MF. Dietary supplement use by US adults: data from the National Health and Nutrition Examination Survey, 1999–2000. *Am J Epidemiol*. 2004;160:339–349.
39. Picciano MF, Dwyer JT, Radimer KL, Wilson DH, Fisher KD, Thomas PR, Yetley EA, Moshfegh AJ, Levy PS, Nielsen SJ, Marriott BM. Dietary supplement use among infants, children, and adolescents in the United States, 1999–2002. *Arch Pediatr Adolesc Med*. 2007;161:978–985.
40. Pillitteri JL, Shiffman S, Rohay JM, Harkins AM, Burton SL, Wadden TA. Use of dietary supplements for weight loss in the United States: results of a national survey. *Obesity (Sliver Spring)*. 2008;16:790–796.
41. Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto miocardico. Dietary supplementation with n-3 polyunsaturated fatty acids and vitamin E after myocardial infarction: results of the GISSI-Prevenzione trial: Gruppo Italiano per lo Studio della Sopravvivenze nell'Infarto miocardico. *Lancet*. 1999;354:447–455.
42. Yokoyama M, Origasa H, Matsuzaki M, Matsuzawa Y, Saito Y, Ishikawa Y, Oikawa S, Sasaki J, Hishida H, Itakura H, Kita T, Kitabatake A, Nakaya N, Sakata T, Shimada K, Shirato K, for the Japan EPA Lipid Intervention Study (JELIS). Effects of eicosapentaenoic acid on major

coronary events in hypercholesterolaemic patients (JELIS): a randomised open-label, blinded endpoint analysis. *Lancet*. 2007;369:1090–1098.
43. GISSI-HF Investigators. Effect of n-3 polyunsaturated fatty acids in patients with chronic heart failure (the GISSI-HF trial): a randomised, double-blind, placebo-controlled trial. *Lancet*. 2008;372:1223–1230.
44. Mokdad AH, Serdula MK, Dietz WH, Bowman BA, Marks JS, Koplan JP. The spread of the obesity epidemic in the United States, 1991–1998. *JAMA*. 1999;282:1519–1522.
45. Flegal KM, Carroll MD, Ogden CL, Johnson CL. Prevalence and trends in obesity among US adults, 1999–2000. *JAMA*. 2002;288:1723–1727.
46. Briefel RR, Johnson CL. Secular trends in dietary intake in the United States. *Annu Rev Nutr*. 2004;24:401–431.
47. Kant AK, Graubard BI. Secular trends in patterns of self-reported food consumption of adult Americans: NHANES 1971–1975 to NHANES 1999–2002. *Am J Clin Nutr*. 2006;84:1215–1223.
48. Popkin BM, Armstrong LE, Bray GM, Caballero B, Frei B, Willett WC. A new proposed guidance system for beverage consumption in the United States. *Am J Clin Nutr*. 2006;83:529–542.
49. Duffey KJ, Popkin BM. Shifts in patterns and consumption of beverages between 1965 and 2002. *Obesity (Silver Spring)*. 2007;15:2739–2747.
50. Wang YC, Bleich SN, Gortmaker SL. Increasing caloric contribution from sugar-sweetened beverages and 100% fruit juices among US children and adolescents, 1988–2004. *Pediatrics*. 2008;121: e1604–e1614.
51. Wang YC, Gortmaker SL, Sobol AM, Kuntz KM. Estimating the energy gap among US children: a counterfactual approach. *Pediatrics*. 2006;118: e1721–e1733.
52. United States Department of Agriculture. Food CPI, prices and expenditures. Available at: www.ers.usda.gov/Briefing/CPIFoodAndExpenditures/Data/. Accessed July 17, 2008.
53. Davis C, Saltos E, for the US Department of Agriculture, Economic Research Service. Dietary recommendations and how they have changed over time. Available at: http://www.ers.usda.gov/publications/aib750/aib750b.pdf. Accessed July 16, 2008. Accessed July 17, 2008.
54. Centers for Disease Control and Prevention (CDC). Trends in intake of energy and macronutrients: United States, 1971–2000. *MMWR Morb Mortal Wkly Rep*. 2004;53:80–82.
55. Egan SK, Bolger PM, Carrington CD. Update of US FDA's Total Diet Study food list and diets. *J Expo Sci Environ Epidemiol*. 2007;17: 573–582.
56. Blanck HM, Gillespie C, Kimmons JE, Seymour JD, Serdula MK. Trends in fruit and vegetable consumption among U.S. men and women, 1994–2005. *Prev Chronic Dis*. 2008;5:A35.
57. Sacks FM, Svetkey LP, Vollmer WM, Appel LJ, Bray GA, Harsha D, Obarzanek E, Conlin PR, Miller ER 3rd, Simons-Morton DG, Karanja N, Lin PH, for the DASH-Sodium Collaborative Research Group. Effects on blood pressure of reduced dietary sodium and the Dietary Approaches to Stop Hypertension (DASH) diet: DASH-Sodium Collaborative Research Group. *N Engl J Med*. 2001;344:3–10.
58. Appel LJ, Sacks FM, Carey VJ, Obarzanek E, Swain JF, Miller ER 3rd, Conlin PR, Erlinger TP, Rosner BA, Laranjo NM, Charleston J, McCarron P, Bishop LM, for the OmniHeart Collaborative Research Group. Effects of protein, monounsaturated fat, and carbohydrate intake on blood pressure and serum lipids: results of the OmniHeart randomized trial. *JAMA*. 2005;294:2455–2464.
59. Mozaffarian D, Clarke R. WHO Scientific Update: Effects of replacing trans fatty acids with other fats and oils on blood lipids and coronary heart disease. *Eur J Clin Nutr*. In press.
60. Geleijnse JM, Giltay EJ, Grobbee DE, Donders AR, Kok FJ. Blood pressure response to fish oil supplementation: metaregression analysis of randomized trials. *J Hypertens*. 2002;20:1493–1499.
61. Mozaffarian D, Geelen A, Brouwer IA, Geleijnse JM, Zock PL, Katan MB. Effect of fish oil on heart rate in humans: a meta-analysis of randomized controlled trials. *Circulation*. 2005;112:1945–1952.
62. Estruch R, Martínez-González MA, Corella D, Salas-Salvadó J, Ruiz-Gutiérrez V, Covas MI, Fiol M, Gómez-Gracia E, López-Sabater MC, Vinyoles E, Arós F, Conde M, Lahoz C, Lapetra J, Sáez G, Ros E, for the PREDIMED Study Investigators. Effects of a Mediterranean-style diet on cardiovascular risk factors: a randomized trial. *Ann Intern Med*. 2006; 145:1–11.
63. Howard BV, Van Horn L, Hsia J, Manson JE, Stefanick ML, Wassertheil-Smoller S, Kuller LH, LaCroix AZ, Langer RD, Lasser NL, Lewis CE, Limacher MC, Margolis KL, Mysiw WJ, Ockene JK, Parker LM, Perri MG, Phillips L, Prentice RL, Robbins J, Rossouw JE, Sarto GE, Schatz IJ, Snetselaar LG, Stevens VJ, Tinker LF, Trevisan M, Vitolins MZ, Anderson GL, Assaf AR, Bassford T, Beresford SA, Black HR, Brunner RL, Brzyski RG, Caan B, Chlebowski RT, Gass M, Granek I, Greenland P, Hays J, Heber D, Heiss G, Hendrix SL, Hubbell FA, Johnson KC, Kotchen JM. Low-fat dietary pattern and risk of cardiovascular disease: the Women's Health Initiative Randomized Controlled Dietary Modification Trial. *JAMA*. 2006;295:655–666.
64. Low-fat diet in myocardial infarction: a controlled trial. *Lancet*. 1965;2: 501–504.
65. European collaborative trial of multifactorial prevention of coronary heart disease: final report on the 6-year results: World Health Organisation European Collaborative Group. *Lancet*. 1986;1:869–872.
66. Mortality rates after 10.5 years for participants in the Multiple Risk Factor Intervention Trial: findings related to a priori hypotheses of the trial: the Multiple Risk Factor Intervention Trial Research Group. *JAMA*. 1990; 263:1795–1801.
67. Burr ML, Fehily AM, Gilbert JF, Rogers S, Holliday RM, Sweetnam PM, Elwood PC, Deadman NM. Effects of changes in fat, fish, and fibre intakes on death and myocardial reinfarction: Diet and Reinfarction Trial (DART). *Lancet*. 1989;2:757–761.
68. Ascherio A, Rimm EB, Giovannucci EL, Spiegelman D, Stampfer M, Willett WC. Dietary fat and risk of coronary heart disease in men: cohort follow up study in the United States. *BMJ*. 1996;313:84–90.
69. Pietinen P, Ascherio A, Korhonen P, Hartman AM, Willett WC, Albanes D, Virtamo J. Intake of fatty acids and risk of coronary heart disease in a cohort of Finnish men: the Alpha-Tocopherol, Beta-Carotene Cancer Prevention Study. *Am J Epidemiol*. 1997;145:876–887.
70. Gillman MW, Cupples LA, Millen BE, Ellison RC, Wolf PA. Inverse association of dietary fat with development of ischemic stroke in men. *JAMA*. 1997;278:2145–2150.
71. Hu FB, Stampfer MJ, Manson JE, Rimm E, Colditz GA, Rosner BA, Hennekens CH, Willett WC. Dietary fat intake and the risk of coronary heart disease in women. *N Engl J Med*. 1997;337:1491–1499.
72. He K, Merchant A, Rimm EB, Rosner BA, Stampfer MJ, Willett WC, Ascherio A. Dietary fat intake and risk of stroke in male US healthcare professionals: 14 year prospective cohort study. *BMJ*. 2003;327:777–782.
73. Oh K, Hu FB, Manson JE, Stampfer MJ, Willett WC. Dietary fat intake and risk of coronary heart disease in women: 20 years of follow-up of the Nurses' Health Study. *Am J Epidemiol*. 2005;161:672–679.
74. Gordon DS. Lowering cholesterol and total mortality. In: Rifkin BM, ed. *Lowering Cholesterol in High-Risk Individuals and Populations*. New York, NY: Marcel Dekker, Inc; 1995:33–48.
75. Mellen PB, Walsh TF, Herrington DM. Whole grain intake and cardiovascular disease: a meta-analysis. *Nutr Metab Cardiovasc Dis*. 2008;18: 283–290.
76. de Munter JS, Hu FB, Spiegelman D, Franz M, van Dam RM. Whole grain, bran, and germ intake and risk of type 2 diabetes: a prospective cohort study and systematic review. *PLoS Med*. 2007;4:e261.
77. Mozaffarian D, Rimm EB. Fish intake, contaminants, and human health: evaluating the risks and the benefits. *JAMA*. 2006;296:1885–1899.
78. Mozaffarian D, Katan MB, Ascherio A, Stampfer MJ, Willett WC. Trans fatty acids and cardiovascular disease. *N Engl J Med*. 2006;354: 1601–1613.
79. Dauchet L, Amouyel P, Hercberg S, Dallongeville J. Fruit and vegetable consumption and risk of coronary heart disease: a meta-analysis of cohort studies. *J Nutr*. 2006;136:2588–2593.
80. Dauchet L, Amouyel P, Dallongeville J. Fruit and vegetable consumption and risk of stroke: a meta-analysis of cohort studies. *Neurology*. 2005; 65:1193–1197.
81. Cohen HW, Hailpern SM, Alderman MH. Sodium intake and mortality follow-up in the Third National Health and Nutrition Examination Survey (NHANES III). *J Gen Intern Med*. 2008;23:1297–1302.
82. Cook NR, Cutler JA, Obarzanek E, Buring JE, Rexrode KM, Kumanyika SK, Appel LJ, Whelton PK. Long term effects of dietary sodium reduction on cardiovascular disease outcomes: observational follow-up of the Trials of Hypertension Prevention (TOHP). *BMJ*. 2007;334:885–888.
83. Mitrou PN, Kipnis V, Thiébaut AC, Reedy J, Subar AF, Wirfält E, Flood A, Mouw T, Hollenbeck AR, Leitzmann MF, Schatzkin A. Mediterranean dietary pattern and prediction of all-cause mortality in a US population: results from the NIH-AARP Diet and Health Study. *Arch Intern Med*. 2007;167:2461–2468.
84. Heidemann C, Schulze MB, Franco OH, van Dam RM, Mantzoros CS, Hu FB. Dietary patterns and risk of mortality from cardiovascular disease, cancer, and all causes in a prospective cohort of women. *Circulation*. 2008;118:230–237.

85. Osler M, Heitmann BL, Gerdes LU, Jørgensen LM, Schroll M. Dietary patterns and mortality in Danish men and women: a prospective observational study. *Br J Nutr*. 2001;85:219–225.
86. van Dam RM, Rimm EB, Willett WC, Stampfer MJ, Hu FB. Dietary patterns and risk for type 2 diabetes mellitus in U.S. men. *Ann Intern Med*. 2002;136:201–209.
87. Heidemann C, Hoffmann K, Spranger J, Klipstein-Grobusch K, Möhlig M, Pfeiffer AF, Boeing H, for the European Prospective Investigation Into Cancer and Nutrition (EPIC)–Potsdam Study Cohort. A dietary pattern protective against type 2 diabetes in the European Prospective Investigation Into Cancer and Nutrition (EPIC)–Potsdam Study cohort. *Diabetologia*. 2005;48:1126–1134.
88. Brunner EJ, Mosdøl A, Witte DR, Martikainen P, Stafford M, Shipley MJ, Marmot MG. Dietary patterns and 15-y risks of major coronary events, diabetes, and mortality. *Am J Clin Nutr*. 2008;87:1414–1421.
89. Lutsey PL, Steffen LM, Stevens J. Dietary intake and the development of the metabolic syndrome: the Atherosclerosis Risk in Communities study. *Circulation*. 2008;117:754–761.
90. Brunner E, Cohen D, Toon L. Cost effectiveness of cardiovascular disease prevention strategies: a perspective on EU food based dietary guidelines. *Public Health Nutr*. 2001;4:711–715.
91. Centers for Disease Control and Prevention. *Preventing Chronic Diseases: Investing Wisely in Health: Preventing Obesity and Chronic Diseases Through Good Nutrition and Physical Activity*. Atlanta, Ga: US Department of Health and Human Services; 2005. Available at: http://www.cdc.gov/nccdphp/publications/factsheets/Prevention/obesity.htm. Accessed November 1, 2006.
92. The Steps to a Healthier US Cooperative Agreement Program. *The Power of Prevention*. Rockville, Md: United States Department of Health and Human Services, Office of Public Health and Science, Office of Disease Prevention and Health Promotion; 2003. Available at: www.healthierus.gov/STEPS/summit/prevportfolio/power/index.html#pop. Accessed November 1, 2006.
93. American Heart Association Nutrition Committee, Lichtenstein AH, Appel LJ, Brands M, Carnethon M, Daniels S, Franch HA, Franklin B, Kris-Etherton P, Harris WS, Howard B, Karanja N, Lefevre M, Rudel L, Sacks F, Van Horn L, Winston M, Wylie-Rosett J. Diet and lifestyle recommendations revision 2006: a scientific statement from the American Heart Association Nutrition Committee. *Circulation*. 2006;114:82–96.
94. U.S. Department of Health and Human Services. *Dietary Guidelines for Americans*. Available at: http://www.health.gov/dietaryguidelines/dga2005/report/. Accessed August 15, 2008.
95. International Society for the Study of Fatty Acids and Lipids. *Recommendations for Dietary Intake of Polyunsaturated Fatty Acids in Healthy Adults*. Devon, UK: International Society for the Study of Fatty Acids and Lipids; 2004.
96. Institute of Medicine. *2005 Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids (Macronutrients)*. Washington, DC: Institute of Medicine, National Academies Press; 2005.

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[93–96]

	Non-Hispanic White Men		Non-Hispanic White Women		Non-Hispanic Black Men		Non-Hispanic Black Women		Mexican American Men		Mexican American Women	
	Average Consumption (mean±SD)	Percent Meeting Guidelines*	Average Consumption (mean±SD)	Percent Meeting Guidelines*	Average Consumption (mean±SD)	Percent Meeting guldelines*	Average Consumption (mean±SD)	Percent Meeting Guidelines*	Average Consumption (mean±SD)	Percent Meeting Guidelines*	Average Consumption (mean±SD)	Percent 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

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 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 fiber per 10 g carbohydrate), 3 or more 1-ounce equivalent (1 oz bread; 1 cup dry cereal; 1/2 cup cooked rice, pasta, or cereal) servings per day (DGA); fish or shellfish, 2 or more 100 g (3.5 oz) servings per week[93]; fruits, 4 or more 1/2-cup servings per day[94]; vegetables, 5 or more 1/2-cup servings per day including up to 3 cups per week of starchy vegetables[94]; nuts, legumes, and seeds, 4 or more 50-g servings per week[93]; 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)[94]; sugar-sweetened beverages (defined as ≥50 cal/8 oz, excluding whole juices), 36 oz or less per week (≈1/4 of discretionary calories)[93,94]; sweets and bakery desserts, 2.5 or fewer 50-g servings per week (≈1/4 of discretionary calories)[93,94]; EPA+DHA, ≥0.5 g/d[95]; ALA, ≥1.6/1.1 g/d (men/women)[96]; saturated fat, <10% energy[94]; dietary cholesterol, <300 mg/d[94]; total fat, 20% to 35% energy[94]; dietary fiber, ≥28/d[94]; sodium, <2.3 g/d[94].

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)	Percent Meeting Guidelines*	Average Consumption (mean±SD)	Percent Meeting Guidelines*	Average Consumption (mean±SD)	Percent Meeting Guidelines*	Average Consumption (mean±SD)	Percent Meeting Guidelines*	Average Consumption (mean±SD)	Percent Meeting Guidelines*	Average Consumption (mean±SD)	Percent 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

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 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 appropriate adjusted to a 2000-kcal/d diet, as for adults.

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

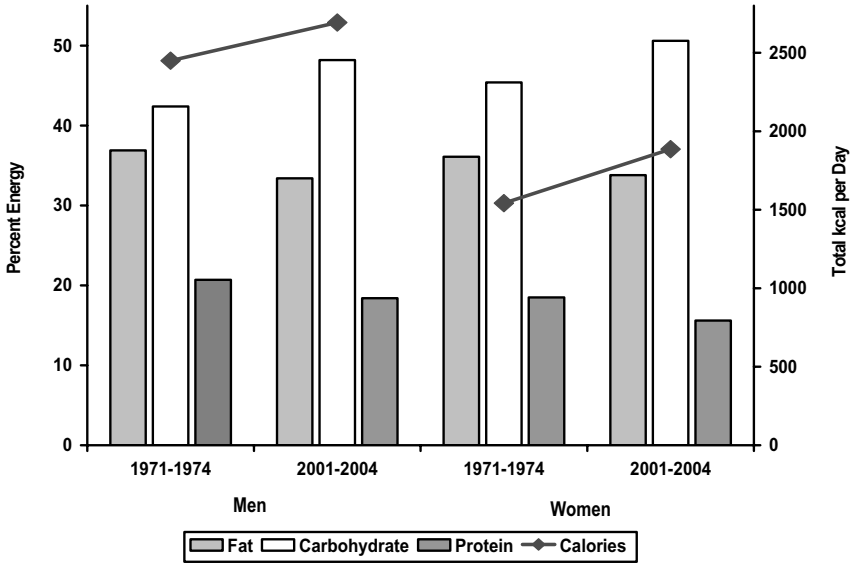


Chart 17-1. Age-adjusted trends in macronutrients and total calories consumed by US adults (20 to 74 years of age), 1971 to 2004. Source: National Center for Health Statistics. *Health, United States 2007, With Chartbook on Trends in the Health of Americans*.[13]

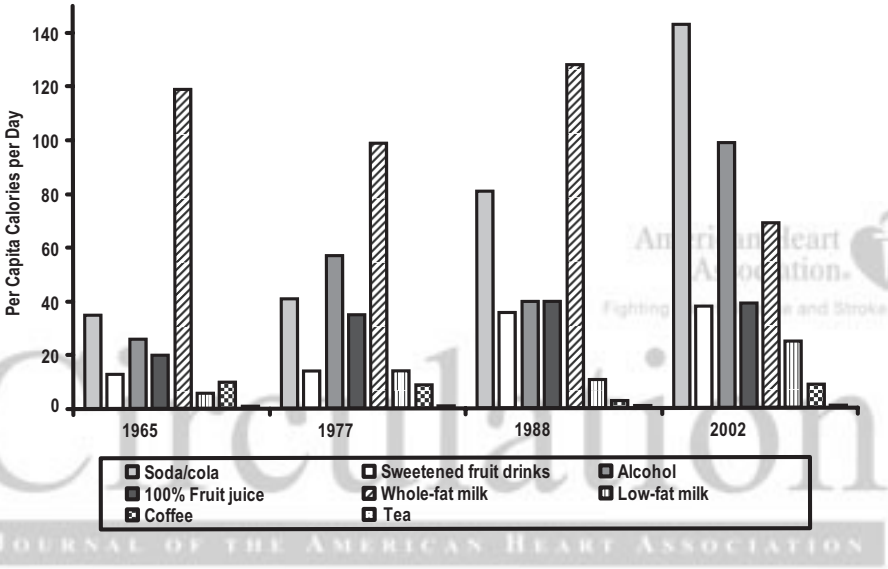


Chart 17-2. Per capita calories consumed from different beverages by US adults (≥19 years of age), 1965 to 2002. Source: Nationwide Food Consumption Surveys (1965, 1977–1978) and NHANES (1988–1994, 1999–2002); Duffey and Popkin.[49]

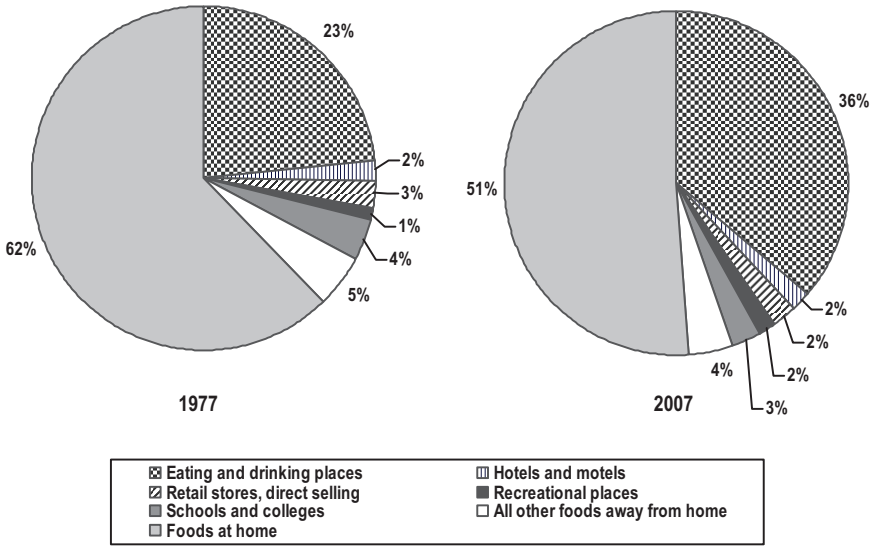


Chart 17-3. Total US food expenditures away from home and at home, 1977 and 2007. Source: US Department of Agriculture Economic Research Service.[53]

18. Quality of Care

See Tables 18-1 through 18-10.

The Institute of Medicine defines quality of care as "the degree to which health services for individuals and populations increase the likelihood of desired health outcomes and are consistent with current professional knowledge."[1]

Abbreviations Used in Chapter 18

ACEI	angiotensin-converting enzyme inhibitor
ACS	acute coronary syndrome
ACTION	Acute Coronary Treatment and Intervention Outcomes Network
AF	atrial fibrillation
AHA	American Heart Association
ARB	angiotensin receptor blocker
BMI	body mass index
BP	blood pressure
CABG	coronary artery bypass graft
CAD	coronary artery disease
CARE	Carotid Artery Revascularization and Endarterectomy data
CAS	carotid artery stenting
CMS	Centers for Medicare and Medicaid Services
CRUSADE	Can Rapid Stratification of Unstable Angina Patients Suppress ADverse Outcomes with Early Implementation of the ACC/AHA Guidelines
CVD	cardiovascular disease
D2B	door-to-balloon
DVT	deep vein thrombosis
EMR	electronic medical record
GWTG	Get With The Guidelines
HbA_{1c}	glycosylated hemoglobin
HF	heart failure
ICD	*International Classification of Diseases*
kg/m^2	kilograms/meter^2
LDL	low-density lipoprotein
LOS	length of stay
LVEF	left ventricular ejection fraction
LVSD	left ventricular systolic dysfunction
mm/dL	milligrams per deciliter
MI	myocardial infarction
NA	not applicable
NAMCS	National Ambulatory Medical Care Survey
NCDR	National cardiovascular Data Registry
NM	not measured
NSTEMI	non–ST-segment–elevation MI
PCI	percutaneous coronary intervention
STEMI	ST-segment–elevation MI
tPA	tissue plasminogen activator
TIA	transient ischemic attack
VHA	Veterans Health Administration

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.

Several studies that have identified potential opportunities to further improve the quality of care for patients with CVD are highlighted below.

- On the basis of annual reports submitted to the US Food and Drug Administration by manufacturers from 1990 to 2002, 17 323 devices were explanted because of confirmed malfunction. The annual implantable cardioverter-defibrillator malfunction replacement rate was 20.7 (11.6) per 1000 implants, compared with a rate of 4.6 (2.2) per 1000 implants for pacemakers.[3] On the basis of pacemaker and implantable cardioverter-defibrillator registries, the mean annual implantable cardioverter-defibrillator malfunction rate was 26.5 (3.8) per 1000 person-years versus 1.3 (0.1) for pacemakers. Battery malfunctions were the most common cause of device failure.[4]
- From the AHA's Get With The Guidelines (GWTG)–Heart Failure quality improvement program, less than 40% of potentially eligible patients hospitalized with HF received implantable cardioverter-defibrillator therapy, and rates of use were lower among eligible women and black patients than among white men.[5] Similarly, in a Medicare cohort, women were less likely than men to receive implantable cardioverter-defibrillator therapy for primary or secondary prevention of sudden cardiac death.[6]
- From the CRUSADE registry of non–ST-elevation MI (NSTEMI) patients, women had higher rates of bleeding regardless of whether they were treated with glycoprotein IIb/IIIa inhibitors during the hospitalization. Among patients treated with IIb/IIIa inhibitors, women were more likely than men to receive excess dosing. The bleeding risk attributable to excess dosing was much higher in women (25.0% versus 4.4%) than in men.[7]
- In a population-based registry of patients with newly diagnosed AF, approximately three fourths (71%) of patients received antithrombotic therapy in the 6 months after the diagnosis. However, among patients at high risk for stroke on the basis of the American College of Chest Physicians stroke risk score, 24% were not prescribed either aspirin or warfarin.[8]
- After AMI hospitalization, 23% and 18% of patients had not filled their discharge cardiac medications by day 7 and day 120, respectively, in a population-based study from Ontario, Canada.[9] On the basis of health plan records from members of 11 health plans, only 45% of patients were adherent to β-blockers in the year after AMI hospitalization, with the biggest drop in adherence between 30 and 90 days after discharge.[10] In addition, in a 19-center prospec-

tive registry of patients discharged on aspirin, β-blockers, and statins after AMI hospitalization, ≈12% discontinued all 3 medications within 1 month of hospital discharge.[11]

- According to data from the 2007 National Healthcare Quality Report, all 6 of the component measures recommended for care for Medicare MI patients showed improvement, including aspirin within 24 hours of admission (from 85.1% to 95.3%), aspirin at discharge (from 85.9% to 95.6%), counseling for smoking cessation (from 42.7% to 90.9%), β-blocker within 24 hours of admission (from 69.3% to 91.5%), and β-blocker at discharge (from 78.5% to 94.5%).[12]
- Between 1994 and 2004, the overall inpatient mortality rate for Medicare inpatients declined from 124.9 to 81.7 deaths per 1000 admissions with MI.[12]
- The overall HF composite showed improvement in the provision of recommended care for Medicare patients with HF, from 68.5% in 2000–2001 to 86.9% in 2005.[12]

Electronic Medical Record Use

- A study of office-based physicians in 2006 by NAMCS found that 29.2% were using full or partial EMR systems. This represents a 60% increase since 2001. These estimates show recent progress toward the goal of universal electronic health records. Features within the EMR system may be most likely to result in improved management and quality of care.[13]

Quality of Care by Race/Ethnicity and Sex

- Racial/ethnic, sex, 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 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 are reported for hospitals participating in GWTG from January 1, 2007, through December 31, 2007 (Tables 18-3, 18-9, and 18-10).

ACS Quality-of-Care Measures

The following are quality-of-care indicators as measured by different national organizations or registries. The quality indicators that are similar across organizations/registries have been summarized in Table 18-1. Each of the organization/registries focus on specific populations among patients hospitalized for an ACS:

- The Veterans Health Administration (VHA) collects national quality performance data related to CVD, including acute MI and HF. Aggregate data from 158 Veterans Administration hospitals for the period between March 2007 and February 2008 are listed (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).
- As part of the Hospital Quality Alliance Program, data are collected by the Centers for Medicare and Medicaid Services (CMS) on quality-of-care indicators for conditions including acute MI and HF. The data were collected from eligible patients for hospital admissions from July 1, 2007, through September 30, 2007. Additional data obtained from the US Department of Health & Human Services Hospital Compare Web site: 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 the table are aggregated data from 50 517 qualifying patients (19 481 STEMI and 31 036 NSTEMI) discharged in 2007 by ≈301 facilities.
- GWTG–Coronary Artery Disease (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 on the selected quality-of-care indicators for CAD events. These were collected from 61 543 patients who were admitted to 295 hospitals participating in the GWTG-CAD program from January 1, 2007, through December 31, 2007.

HF Quality-of-Care Measures

GWTG–Heart Failure (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 on the selected quality-of-care indicators for HF hospitalizations. These were collected from 45 307 patients who were admitted to 257 hospitals participating in the GWTG-HF program from January 1, 2007, through December 31, 2007. The VHA and the CMS data have been previously described.

American Stroke Association GWTG-Stroke Program

GWTG-Stroke is a national quality-improvement initiative of the AHA and American Stroke Association to help hospitals redesign systems of care to improve adherence to guidelines in patients admitted with an ischemic stroke or TIA. Table 18-3 summarizes performance on the selected treatment and quality-of-care indicators for acute stroke and secondary prevention. There were 184 437 clinically identified patients who were admitted to 917 hospitals participating in the GWTG-Stroke program from January 1, 2007, through December 31, 2007.

The 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 256 748 procedures performed at 815 participating sites in 2007.

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 are based on voluntary reporting by >500 health plans. All clinical data are 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.

National Cardiovascular Data Registry Cardiac Catheterization and PCI Data

The National Cardiovascular Data Registry (NCDR) CathPCI Registry, a partnership of the American College of Cardiology and Society of Coronary Angiography and Intervention, is composed of diagnostic cardiac catheterizations and interventional (PCI) procedures harvested from participating facilities across the United States. Listed in Table 18-6 are aggregated data from 593 116 diagnostic cardiac catheterizations (without PCI at same lab visit) and 468 143 PCI procedures performed on patients discharged in 2007 from 822 participating facilities. Only records with valid responses to indicators were considered, and not all procedures qualify for every indicator.

NCDR Implantable Cardioverter Defibrillator Data

In response to the CMS mandate to collect nationwide data on implantable cardioverter-defibrillator implantation, the Implantable Cardioverter-Defibrillator Registry, a partnership of the American College of Cardiology and Heart Rhythm Society, was developed. Facilities may choose whether to submit all implantable cardioverter-defibrillator procedures or a limited submission of CMS-mandated primary prevention procedures. Listed in Table 18-7 are aggregated data from 106 079 implantable cardioverter-defibrillator procedures submitted by 977 facilities where the patient was discharged in 2007 and the submitting facility has chosen to report all their implantable cardioverter-defibrillator procedures (ie, both primary and secondary prevention, Medicare and non-Medicare). Only records with valid responses to indicators were considered. These data are intended only for descriptive purposes; these measures are not intended as quality performance measures.

NCDR Carotid Artery Revascularization and Endarterectomy Data

The CARE Registry, a partnership of the American College of Cardiology, the Society for Cardiovascular Angiography and Interventions, the Society of Interventional Radiology, the American Academy of Neurology, the Society for Vascular Medicine, the American Association of Neurological Surgeons, and the Congress of Neurological Surgeons, was launched in September 2006 to collect and analyze data on patients undergoing carotid artery stenting (CAS) or carotid endarterectomy. The CARE Registry is the NCDR's first registry collecting data on procedures performed by multiple physician specialists. Embedded in the CARE Registry data set are the elements required for hospitals to submit to the CMS to maintain their CMS certification.

Table 18-8 contains aggregated data from 2031 CAS procedures submitted by 80 facilities where the patient was discharged in 2007. Only records with valid responses to indicators were considered, and not all procedures qualify for every indicator.

References

1. Institute of Medicine, Committee on Quality of Health Care in America. *Crossing the Quality Chasm: A New Health System for the 21st Century*. Washington, DC: National Academy Press; 2001:232.
2. Spertus JA, Eagle KA, Krumholz HM, Mitchell KR, Normand SL, American College of Cardiology, American Heart Association Task Force on Performance Measures. American College of Cardiology and American Heart Association methodology for the selection and creation of performance measures for quantifying the quality of cardiovascular care. *Circulation*. 2005;111:1703–1712.
3. Maisel WH, Moynahan M, Zuckerman BD, Gross TP, Tovar OH, Tillman DB, Schultz DB. Pacemaker and ICD generator malfunctions: analysis of Food and Drug Administration annual reports. *JAMA*. 2006;295:1901–1906.
4. Maisel WH. Pacemaker and ICD generator reliability: meta-analysis of device registries. *JAMA*. 2006;295:1929–1934.
5. Hernandez AF, Fonarow GC, Liang L, Al-Khatib SM, Curtis LH, LaBresh KA, Yancy CW, Albert NM, Peterson ED. Sex and racial differences in the use of implantable cardioverter-defibrillators among patients hospitalized with heart failure. *JAMA*. 2007;298:1525–1532.
6. Curtis LH, Al-Khatib SM, Shea AM, Hammill BG, Hernandez AF, Schulman KA. Sex differences in the use of implantable cardioverter-defibrillators for primary and secondary prevention of sudden cardiac death. *JAMA*. 2007;298:1517–1524.
7. Alexander KP, Chen AY, Newby LK, Schwartz JB, Redberg RF, Hochman JS, Roe MT, Gibler WB, Ohman EM, Peterson ED; CRUSADE (Can Rapid risk stratification of Unstable angina patients Suppress ADverse outcomes with Early implementation of the ACC/AHA guidelines) Investigators. Sex differences in major bleeding with glycoprotein IIb/IIIa inhibitors: results from the CRUSADE (Can Rapid risk stratification of Unstable angina patients Suppress ADverse outcomes with Early implementation of the ACC/AHA guidelines) initiative. *Circulation*. 2006;114:1380–1387.
8. Glazer NL, Dublin S, Smith NL, French B, Jackson LA, Hrachovec JB, Siscovick DS, Psaty BM, Heckbert SR. Newly detected atrial fibrillation and compliance with antithrombotic guidelines. *Arch Intern Med*. 2007;167:246–252.
9. Jackevicius CA, Li P, Tu JV. Prevalence, predictors, and outcomes of primary nonadherence after acute myocardial infarction. *Circulation*. 2008;117:1028–1036.
10. Kramer JM, Hammill B, Anstrom KJ, Fetterolf D, Snyder R, Charde JP, Hoffman BS, Allen LaPointe N, Peterson E. National evaluation of adherence to beta-blocker therapy for 1 year after acute myocardial infarction in patients with commercial health insurance. *Am Heart J*. 2006;152:454.e1–e8.

11. Ho PM, Spertus JA, Masoudi FA, Reid KJ, Peterson ED, Magid DJ, Krumholz HM, Rumsfeld JS. Impact of medication therapy discontinuation on mortality after myocardial infarction. *Arch Intern Med*. 2006; 166:1842–1847.
12. Agency for Healthcare Research and Quality. *2007 National Healthcare Quality Report.* Rockville, Md: US Department of Health and Human Services, Agency for Healthcare Research and Quality; February 2008. AHRQ Pub. No. 08-0040.
13. Hing ES, Burt CW, Woodwell DA Electronic medical record use by office-based physicians and their practices: United States, 2006. Advance data from vital and health statistics; No 393. Hyattsville, Md: National Center for Health Statistics; 2007.

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

Quality-of-Care Measure	VHA*	National Medicare and Medicaid†	AHA GWTG-CAD‡	ACTION-STEMI§	ACTION-NSTEMI§
Aspirin within 24 hours of admission, %	97	97	92	98	97
Aspirin at discharge, %	99	97	96‖	98	97
β-Blockers within 24 hours of admission, %	96	95	92	95	93
β-Blockers at discharge, %	98	97	94‖	97	95
Lipid-lowering medication at discharge, %	NM	NM	86	91	92
Lipid therapy at discharge if LDL >100 mg/dL, %	96¶	NM	94‖	NM	NM
ARB/ACEI at discharge for patients with LVEF <40%, %	92	93	82	86	74
ACEI at discharge for AMI patients, %	NM	NM	72‖	78#	70#
Adult smoking cessation advice/counseling, %	97	98	97‖	96	94
Fibrinolytic therapy within 30 minutes, %	NM	51	41	39	NA
Percutaneous coronary intervention within 90 minutes, %	NM	74	67	67	NA
Cardiac rehabilitation referral, %	NM	NM	72	78	68

NM indicates not measured; NA, not applicable.

*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% and mean length of hospital stay 5.2 days (median 3.0 days). (Length of stay previously defined as: LOS=[discharge date−arrival date]+1. Currently, same-day or next-day discharge: LOS=1. Subsequent discharges: 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 5 key performance measures targeted in GWTG-CAD. The composite quality-of-care measure was 90.4%. The composite quality-of-care measure indicates performance on the provision of several elements of care. It is computed by summing the numerators for each key performance measure across the population of interest to create a composite numerator (all the care that was given), summing the denominators for each measure to form a composite denominator (all the care that should have been given), and reporting the ratio (the percentage of all the needed care that was given).

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

#Data from October 1, 2007, through December 31, 2007 only.

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

Quality-of-Care Measure	VHA	National Medicare and Medicaid	AHA-GWTG-HF
LVEF assessment, %	99	94	96*
ARB/ACEI at discharge for patients with left ventricular systolic dysfunction, %	93	91	89*
Complete discharge instructions, %	92	77	85*
Adult smoking cessation advice/counseling, %	93	96	95*
β-Blockers at discharge for patients with LVSD, no contraindications, %	NM	NM	90*
Anticoagulation for AF or atrial flutter, no contraindications, %	NM	NM	67

NM indicates not measured.

In the GWTG registry, mechanical ventilation was required in 2.1% of patients. In-hospital mortality rate was 3.1% and mean length of hospital stay 5.6 days (median 4.0 days).

*Indicates the 5 key performance measures targeted in GWTG-HF. The composite quality-of-care measure was 90.8%. The composite quality-of-care measure indicates performance on the provision of several elements of care. It is computed by summing the numerators for each key performance measure across the population of interest to create a composite numerator (all the care that was given), summing the denominators for each measure to form a composite denominator (all the care that should have been given), and reporting the ratio (the percentage of all the needed care that was given).

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

Quality-of-Care Measure	Overall	White	Black	Hispanic	Male	Female
Intravenous tPA in patients who arrived <2 hours after symptom onset,* %	70.0	70.8	65.6	67.9	71.8	68.0
Intravenous tPA in patients who arrived <3 hours after symptom onset, %	57.4	58.4	52.2	56.0	59.6	55.4
Documentation of ineligibility (why no tPA), %	94.5	94.7	93.2	94.2	94.4	94.5
Rate of symptomatic brain hemorrhage after tPA,† %	5.2	4.9	6.9	5.8	5.1	5.4
Antithrombotics <48 hours after admission,* %	96.8	96.9	96.6	96.5	96.9	96.4
DVT prophylaxis by second hospital day,* %	87.2	86.9	88.8	86.4	88.1	86.7
Antithrombotics at discharge,* %	98.9	98.8	98.9	98.8	98.8	98.7
Anticoagulation for AF at discharge,* %	98.0	98.0	97.2	99.4	98.3	97.8
Therapy at discharge if LDL >100 mg/dL or on therapy at admission,* %	84.8	84.6	85.0	85.7	87.0	83.0
Counseling for smoking cessation,* %	91.3	91.5	91.4	88.7	91.1	91.1
Lifestyle changes recommended for BMI >25 kg/m^2, %	46.1	45.1	49.5	49.5	46.4	44.9
Composite quality-of-care measure, %	92.7	92.7	92.8	92.4	93.3	92.1

In-hospital mortality rate for the overall patient population was 6.97% and mean length of hospital stay 5.25 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 2007 Data
No. of isolated coronary artery bypass procedures	154 188
No. of aortic valve procedures	17 592
No. of mitral valve procedures	4251
Unadjusted isolated coronary artery bypass operative mortality rate	2%
Unadjusted aortic valve operative mortality rate	3%
Unadjusted mitral valve operative mortality rate	6%
Mean postprocedure length of stay for isolated coronary artery bypass procedures	7.0 days
Mean postprocedure length of stay for aortic valve procedures	8.1 days
Mean postprocedure length of stay for mitral valve procedures	10.6 days

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

	Commercial, %	Medicare, %	Medicaid, %
Acute MI			
β-Blocker prescription at discharge	98	94	88
β-Blocker persistence*	73	70	68
Cholesterol management for patients with CAD			
Cholesterol screening	88	88	76
LDL control (<100 mg/dL)	57	56	36
Hypertension			
BP <140/90 mm Hg	60	57	53
Diabetes			
HbA_{1c} testing	88	87	78
HbA_{1c} >9.0%	30	27	49
HbA_{1c} <7.0%	42	46	30
Eye exam performed	55	62	51
LDL cholesterol screening	83	85	71
LDL cholesterol <100 mg/dL	43	47	31
Monitoring nephropathy	80	85	75
BP <130/80 mm Hg	30	30	30
BP <140/90 mm Hg	61	58	57

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

Table 18-6. National Cardiovascular Data Registry Cardiac Catheterization and PCI Data

	Overall (Mean)	Highest Quartile	Lowest Quartile
Diagnostic cardiac catheterization (without PCI in same lab visit)			
In-lab mortality rate*	0.05%	0.0%	0.2% (90th percentile)
Major complications†	1.3%	0.2%	1.8%
PCI			
Major complications†	2.4%	0.97%	3.2%
Vascular complications‡	1.9%	0.86%	2.5%
Antiplatelet drug administration§	97%	99%	96%
Statin drug administration‖	85%	92%	81%
Emergency CABG¶	0.4%	0.0%	0.6%
Average D2B (door-to-balloon time)#	101.5 min	66.7 min	108.1 min
Patients with D2B ≥90 minutes**	69%	82%	60%
Patients with D2B ≥120 minutes††	86%	94%	81%
Risk-adjusted mortality rate‡‡	1.2%	0.9%	1.6%

*Mortality rate in lab.

†Contrast media reaction, cardiogenic shock, cerebrovascular accident, congestive heart failure, cardiac tamponade, renal failure.

‡Bleeding at entry site (femoral approach), retroperitoneal bleeding, vascular access occlusion at entry site, peripheral embolization, vascular dissection, psuedoaneurysm, arteriovenous fistula.

§Proportion of PCI patients with stent receiving antiplatelet therapy such as clopidogrel or ticlopidine during admission.

‖Proportion of PCI patients who received statin medication during admission.

¶Proportion of PCI patients requiring emergency coronary artery bypass surgery.

#Often called "door-to-balloon time" or D2B, this is the elapsed time between entry to facility and reperfusion of the affected coronary vessel for patients with acute MI treated with primary percutaneous intervention (primary PCI).

**Proportion of primary PCI patients with coronary reperfusion within 90 minutes of entry to facility.

††Proportion of primary PCI patients with coronary reperfusion within 120 minutes of entry to facility.

‡‡PCI mortality rate adjusted by NCDR Risk Adjustment Algorithm.

Table 18-7. NCDR Implantable Cardioverter Defibrillator Data

Implantable Cardioverter-Defibrillator Procedures (Facilities That Submit All Procedures)	Overall (Mean)	Highest Quartile	Lowest Quartile
Any adverse event*	3.2%	0.0%	4.4%
Lead dislodgement†	0.9%	0.0%	1.2%
β-Blocker medication during admission‡	89%	95%	83%
ACEI/ARB medication during admission§	79%	86%	73%
Single-chamber device‖	25%	...	...
Dual-chamber device¶	42%	...	...
Biventricular device#	34%	...	...
Total length of stay**	4.2 days	...	...
Postprocedure length of stay††	1.8 days	...	...

*Proportion of patients who had any adverse event including death in hospital, cardiac arrest, drug reaction, cardiac perforation, cardiac valve injury, conduction block, coronary venous dissection, hematoma, lead dislodgement, hemothorax, pneumothorax, peripheral nerve injury, peripheral embolus, deep phlebitis, TIA, stroke, or MI.

†No. of lead dislodgements per procedure (may record >1 event per procedure).

‡Proportion of patients with LVEF ≥40% admitted only for the procedure with any β-blocker prescribed at discharge, excluding patients with contraindications.

§Proportion of patients with LVEF ≥40% with any ACEI or ARB prescribed at discharge, excluding patients with contraindications.

‖Proportion of patients receiving single-chamber implantable cardioverter-defibrillator device implantation.

¶Proportion of patients receiving dual-chamber implantable cardioverter-defibrillator device implantation.

#Proportion of patients receiving biventricular implantable cardioverter-defibrillator device implantation.

**Total hospital length of stay in days.

††Postprocedure length of stay in days.

Table 18-8. NCDR Carotid Artery Revascularization and Endarterectomy Data

Carotid Stent Procedures (CAS)	Overall (Mean)
Major adverse events, symptomatic* patients†	3.1%
Major adverse events, asymptomatic* patients‡	1.9%
Incidence of stroke for symptomatic* patients§	2.0%
Incidence of stroke for asymptomatic* patients‖	1.6%
Procedures with patients at high surgical risk¶	74%
Embolic protection successfully deployed#	97%
Postprocedure length of stay**	2.6 days

**Symptomatic* is defined in the CMS National Coverage Decision as (a) carotid TIA: distinct focal neurological dysfunction persisting <24 hours; or (b) nondisabling stroke: Modified Rankin Scale <3 with symptoms for ≥24 hours; or (c) transient monocular blindness: amaurosis fugax.

†The proportion of symptomatic patients who die or experience a new stroke or MI from the time of the CAS procedure through discharge.

‡The proportion of asymptomatic patients who die or experience a new stroke or MI from the time of the CAS procedure through discharge.

§The proportion of procedures with symptomatic patients who experience a new stroke from the time of the CAS procedure through discharge

‖The proportion of procedures with asymptomatic patients who experience a new stroke from the time of the CAS procedure through discharge.

¶Proportion of patients with ≥1 condition that qualifies the patient to be at high surgical risk, as defined in the CMS National Coverage Decision, which lists 15 qualifying conditions. More information can be found on the CMS Web site at http://www.cms.hhs.gov/MedicareApprovedFacilitie/03_CASrecert.asp.

#Proportion of procedures in which a stent was implanted and embolic protection deployed successfully.

**Mean postprocedure length of hospital stay (in days) for patients undergoing a CAS procedure.

Table 18-9. 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, %	97.3	96.8	97.9	97.9	96.5
Aspirin at discharge,* %	96.2	94.9	94.7	96.5	94.5
β-Blocker at discharge,* %	95.6	95.8	94.0	95.9	94.4
ACEI at discharge, %	67.0	71.5	63.7	68.6	63.0
ACEI at discharge for AMI patients,* %	71.5	73.6	74.4	73.7	67.9
ACEI in LVSD patients, %	83.7	85.7	82.9	84.3	80.6
Lipid therapy at discharge, %	86.9	83.5	74.6	88.2	81.5
Lipid therapy at discharge if LDL >100 mg/dL,* %	90.6	90.7	87.8	92.6	86.4
BP control (to <140/90 mm Hg) at discharge, %	53.0	47.1	47.6	53.2	50.2
Smoking cessation counseling,* %	97.6	97.7	97.2	97.7	97.0
Referral to cardiac rehabilitation, %	75.2	76.2	68.0	71.1	69.0
Composite quality-of-care measure,† %	90.8	91.3	90.4	91.7	88.8

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

†The composite quality-of-care measure indicates performance on the provision of several elements of care. It is computed by summing the numerators for each key quality 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 18-10. 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,* %	85.4	84.9	83.6	85.3	83.7
Measure of LV function,* %	96.0	97.4	96.2	97.0	95.4
ACE or ARB at discharge for patients with LVSD, no contraindications,* %	88.7	91.2	85.9	89.3	89.1
Smoking cessation counseling, current smokers,* %	94.6	94.9	95.7	94.9	94.6
β-Blockers at discharge for patients with LVSD, no contraindications,* %	91.2	87.6	82.3	89.8	88.7
Hydralazine/nitrates at discharge for patients with LVSD, no contraindications, %	NM	7.7	NM	8.8†	6.2†
Anticoagulation for AF or atrial flutter, no contraindications, %	69.1	64.1	58.2	69.3	65.0
Composite quality-of-care measure, %	91.2	91.1	88.7	91.2	90.0

NM indicates not measured.

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

†For black patients only.

19. Medical Procedures

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

From 1996 to 2006, the total number of inpatient cardiovascular operations and procedures increased 30%, from 5 444 000 to 7 191 000 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 39.1 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. Discharge rates for males and females 65 to 74 years of age were 135.1 and 64.0, respectively. These declined slightly in those >75 years of age, to 128.7 and 69.0, respectively.
  - — In-hospital mortality rate (deaths/100 CABG discharges) declined from 4.3 to 3.5 in 2002–2004, despite an increase in Charlson comorbidity index. 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.

Abbreviations Used in Chapter 19

AHA	American Heart Association
ICD	International Classification of Diseases
CABG	coronary artery bypass graft
MI	myocardial infarction
NCHS	National Center for Health Statistics
NHDS	National Hospital Discharge Survey
PCI	percutaneous coronary intervention
STS NCD	Society of Thoracic Surgeons' National Adult Cardiac Database

- 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. 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.

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 perioperative risk.[1]
- By 2006, it was estimated that >70% of PCIs were performed with drug-eluting as opposed to bare-metal stents.[4]

Coronary Artery Bypass Surgery

The NHDS (NCHS) estimates that in 2006, in the United States, 253 000 patients underwent a total of 448 000 coronary artery bypass procedures (defined by procedure codes). CABG volumes have declined nationally since 1998. Risk-adjusted mortality for CABG has declined significantly over the past decade:

- Data from the Society of Thoracic Surgeons' National Adult Cardiac Database (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.[5]
- Data from the STS NCD document a >50% decline in risk-adjusted mortality rate, despite a significant increase in preoperative surgical risk.[6]

Heart Transplantations

In 2007, 2210 heart transplantations were performed in the United States. There are 254 transplant hospitals in the United States, 130 of which perform heart transplantations.[7]

- Of these recipients, 73.7% are male, and 67.6% are white; 25.4% are <35 years of age, 19.9% are 35 to 49 years of age, and 54.7% are ≥50 years of age.
- As of May 30, 2008, the 1-year survival rate for males was 87.5%, and for females, it was 85.5%; the 3-year rates were

78.8% for males and 76.0% for females; and the 5-year rates were 72.3% for males and 67.4% for females.
- As of June 13, 2008, 2607 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.[8]

- 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.

References

1. Holmes JS, Kozak LJ, Owings MF. Use and in-hospital mortality associated with two cardiac procedures, by sex and age: National Trends, 1990–2004. *Health Aff (Millwood)*. 2007;26:169–177.
2. Nallamothu BK, Young J, Gurm HS, Pickens G, Safavi K. Recent trends in hospital utilization for acute myocardial infarction and coronary revascularization in the United States. *Am J Cardiol*. 2007; 99:749–753.
3. Jha AK, Fisher ES, Li Z, Orav EJ, Epstein AM. Racial trends in the use of major procedures among the elderly. *N Engl J Med*. 2005;353: 683–691.
4. US Food and Drug Administration, Circulatory System Devices Panel. Meeting minutes, December 8, 2006, Washington, DC. Available at: http://www.fda.gov/ohrms/dockets/ac/06/transcripts/2006-4253t2.rtf. Accessed June 30, 2008.
5. STS Adult Cardiac Surgery Database: Period Ending 12/31/2007 Executive Summary Contents. Available at: http://www.sts.org/documents/pdf/ndb/2008_1stHarvest_Executive_Summary.pdf. Accessed June 30, 2008.
6. Ferguson TB Jr, Hammill BD, Peterson ED, DeLong EL, Grover FL; STS National Database Committee. A decade of change: risk profiles and outcomes for isolated coronary artery bypass grafting procedures, 1990–1999: a report from the STS National Database Committee and the Duke Clinical Research Institute. *Ann Thorac Surg*. 2002;73: 480–489.
7. The Organ Procurement and Transplantation Network. National data. Available at: http://www.optn.org/latestData/step2.asp. Accessed November 7, 2007.
8. Riley GF. Long-term trends in the concentration of Medicare spending. *Health Aff (Millwood)*. 2007;26:808–816.
9. Agency for Healthcare Research and Quality, Healthcare Cost and Utilization Project. HCUPnet. Available at: http://www.hcup.ahrq.gov/HCUPnet.jsp. Accessed August 13, 2008.

Table 19-1. 2006 National Healthcare Cost and Utilization Project Statistics: Mean Charges and In-Hospital Death Rates for Various Procedures

Procedure	Mean Charges	In-Hospital Death Rate, %
CABG	$99 743	1.94
PCI	$48 399	0.71
Diagnostic cardiac catheterization	$28 835	0.77
Pacemaker	$47 081	0.90
Implantable defibrillator	$104 743	0.64
Endarterectomy	$25 658	0.38
Valves	$141 120	4.98

Source: Agency for Healthcare Research and Quality, Healthcare Cost and Utilization Project.[9]

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

		Sex			Age, y				Region†			
Operation/Procedure/Patients	ICD-9-CM Code(s)	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
Open-heart surgery procedures‡‡	35 [less 35.4, 35.96], 36 [less 36.0], 37.1, 37.3–37.5	694	463	232	31	45	265	353	126	175	252	141
Total vascular and cardiac surgery and procedures§§	35–39, 00.50–00.51, 00.53–00.54, 00.66	7191	4088	3104	210	733	2622	3627	1358	1610	2799	1424

Ellipses (. . .) indicate data not available.

*Breakdowns are not available for some procedures, so entries for some categories do not add to totals. These data include codes where the estimated No. 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.

‡Open heart valvuloplasty without replacement, replacement of heart valve, other operations on heart valves.

§Estimate should be used with caution as it may be unreliable.

‖Previously referred to as percutaneous transluminal coronary angioplasty or PTCA.

¶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 angioplasty were reported to have a stent inserted (personal communication with NCHS, June 15, 2007).

**Because ≥1 procedure codes are required to describe the specific bypass procedure performed, it is impossible from these (mixed) data to determine the average number of grafts per patient.

††There are additional insertions, revisions, and replacements of pacemaker leads, including those associated with temporary (external) pacemakers.

‡‡Includes valves, bypass and "other" open-heart procedures (codes 35 [less 35.4, 35.96], 36 [less 36.0], 37.1, 37.3–37.5). There were 194 000 other open-heart procedures in 2005.

§§Totals include procedures not shown here.

Source: National Hospital Discharge Survey, NCHS. Unpublished data, 2006. Estimates are based on a sample of inpatient records from short-stay hospitals in the United States.

Note: 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 are not available at this time.

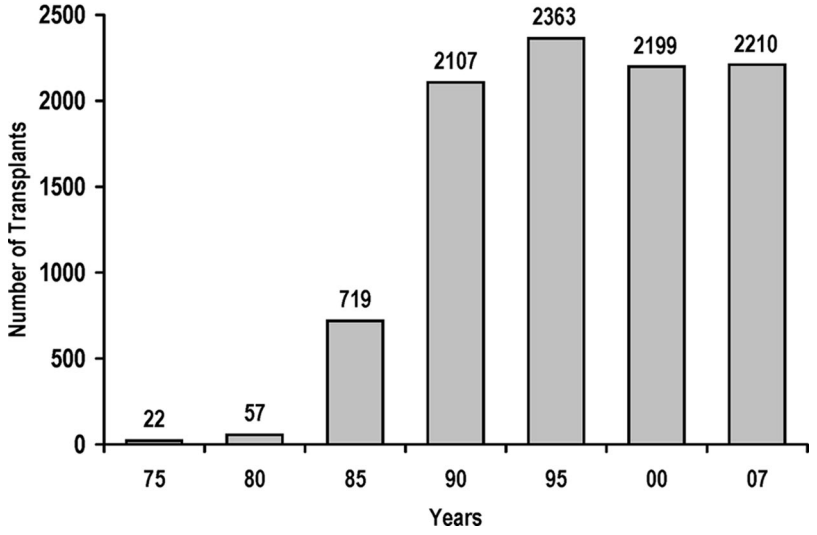


Chart 19-1. Trends in heart transplantations (UNOS: 1975–2007). Source: United Network for Organ Sharing (UNOS), scientific registry data.

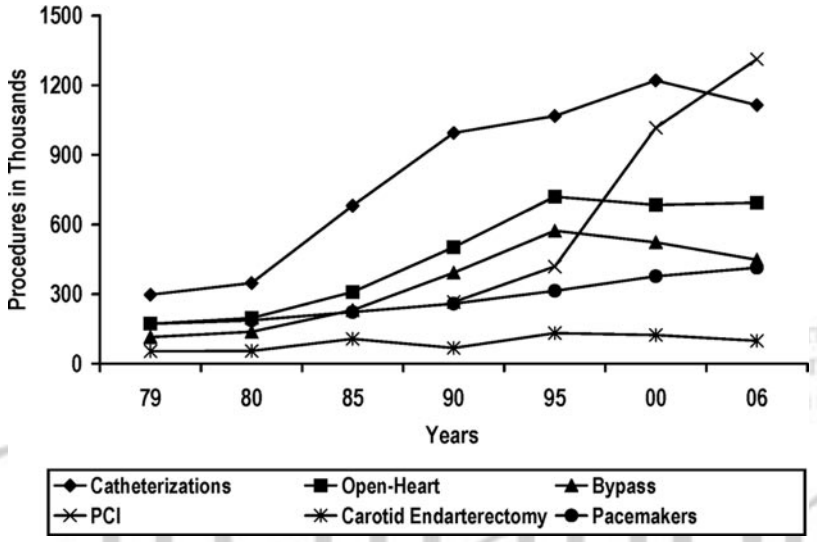


Chart 19-2. Trends in cardiovascular inpatient operations and procedures (United States: 1979–2006). Source: NHDS, NCHS and NHLBI. Note: In-hospital procedures only.

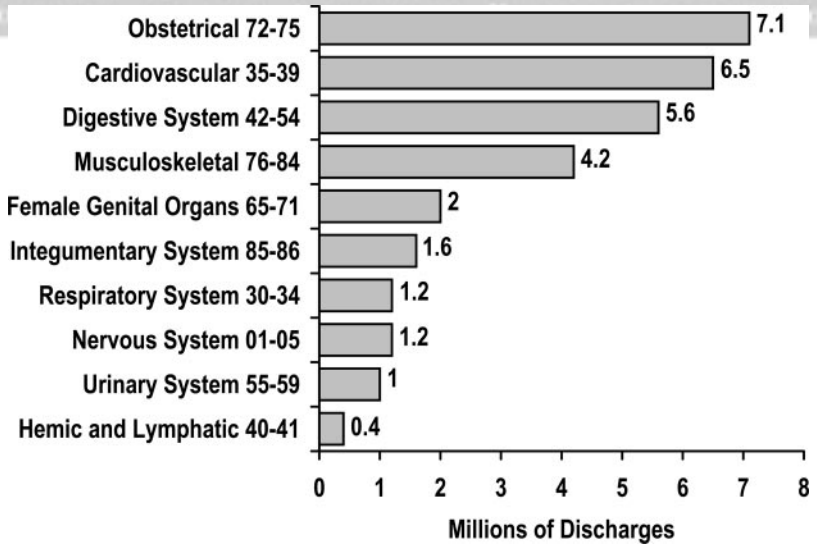


Chart 19-3. Number of surgical procedures in the 10 leading diagnostic groups (United States: 2006). Source: NHDS/NCHS and NHLBI.

20. Economic Cost of Cardiovascular Diseases

See Chart 20-1, Chart 20-2, and Table 20-1.[1-5]

The total direct and indirect cost of CVD and stroke in the United States for 2009 is estimated at $475.3 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 2009 are estimated to be $150.1 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.[6]

Abbreviations Used in Chapter 20

CHD	coronary heart disease
CVD	cardiovascular disease
HF	heart failure
NCHS	National Center for Health Statistics
NHLBI	National Heart, Lung, and Blood Institute

References

1. Hodgson TA, Cohen AJ. Medical care expenditures for selected circulatory diseases: opportunities for reducing national health expenditures. *Med Care*. 1999;37:994–1012.
2. Centers for Medicare and Medicaid Services, Office of the Actuary. National Health Expenditure Projections 2007–2017: Table 2: National health expenditure amounts, and annual percent change by type of expenditure: calendar years 2002–2017. Baltimore, Md: Centers for Medicare and Medicaid Services, 2008. Available at: http://www.cms.hhs.gov/NationalHealthExpendData/downloads/proj2007.pdf. Accessed June 9, 2008.
3. Rice DP, Hodgson TA, Kopstein AN. The economic costs of illness: a replication and update. *Health Care Financ Rev*. 1985;7:61–80.
4. US Census Bureau. Historical income tables: people: table p 39: full-time, year-round all workers by mean income and sex: 1960 to 2007. Washington, DC: Income Surveys Branch, Housing & Household Economic Statistics Division, US Census Bureau; 2007. Available at: http://www.census.gov/hhes/www/income/histinc/p09ar.html. Accessed June 9, 2008.
5. Data Warehouse, Mortality Statistics Branch, National Center for Health Statistics. Worktable 291F: Deaths from 113 selected causes, alcohol-induced causes, drug-induced causes, and injury by firearms, by 5-year age groups, race, and sex: United States, 1999–2005. Hyattsville, Md: Centers for Disease Control and Prevention, US Dept of Health and Human Services; 2007. Available at: http://www.cdc.gov/nchs/data/statab/mortfinal2005_worktable_291F.pdf. Accessed June 9, 2008.
6. National Heart, Lung, and Blood Institute. *Fact Book, Fiscal Year 2007*. Bethesda, Md: National Institutes of Health, National Heart, Lung, and Blood Institute; February 2008. Available at: www.nhlbi.nih.gov/about/factbook/FactBookFinal.pdf. Accessed August 13, 2008.
7. DeFrances CJ, Cullen KA, Kozak LJ. National Hospital Discharge Survey: 2005 annual summary with detailed diagnosis and procedure data. *Vital Health Stat 13*. 2007;(165):1–209.

Table 20-1. Estimated Direct and Indirect Costs (in Billions of Dollars) of CVD and Stroke: United States: 2009[1–5]

	Heart Diseases*	CHD	Stroke	Hypertensive Disease	HF	Total CVD†
Direct costs						
Hospital	$106.3	$54.6	$20.2	$8.2	$20.1	$150.1
Nursing home	$23.4	$12.3	$16.2	$4.8	$4.5	$48.2
Physicians/other professionals	$23.8	$13.4	$3.7	$13.4	$2.4	$46.4
Drugs/other						
Medical durables	$22.1	$10.3	$1.4	$25.4	$3.3	$52.3
Home health care	$7.4	$2.2	$4.4	$2.4	$3.4	$16.8
Total expenditures†	$183.0	$92.8	$45.9	$54.2	$33.7	$313.8
Indirect costs						
Lost productivity/morbidity	$24.0	$10.6	$7.0	$8.4	...	$39.1
Lost productivity/mortality‡	$97.6	$62.0	$16.0	$10.8	$3.5	$122.4
Grand totals†	$304.6	$165.4	$68.9	$73.4	$37.2	$475.3

Ellipses (. . .) indicate data not available.

*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 2009, discounted at 3%.

All estimates prepared by Thomas Thom, NHLBI.

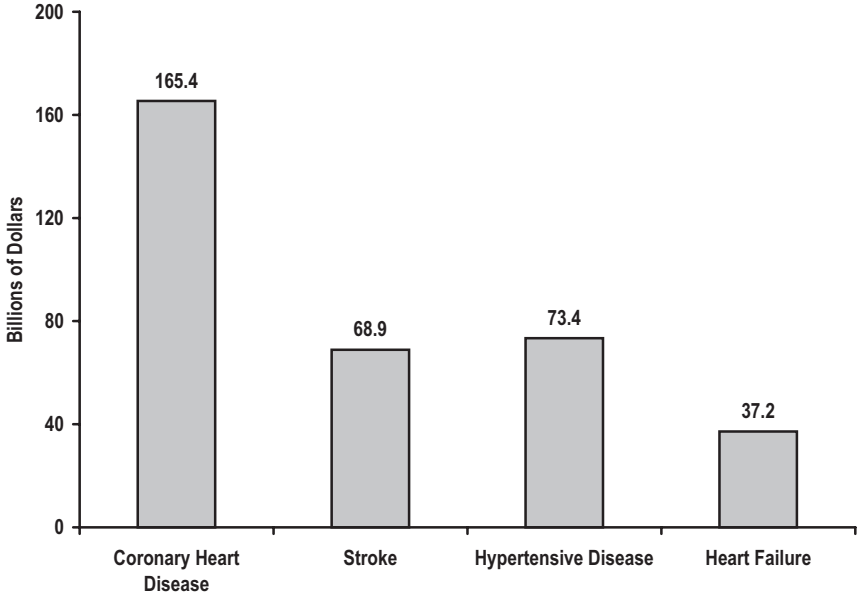


Chart 20-1. Estimated direct and indirect costs (in billions of dollars) of major cardiovascular diseases and stroke (United States: 2009). Source: NHLBI.[7]

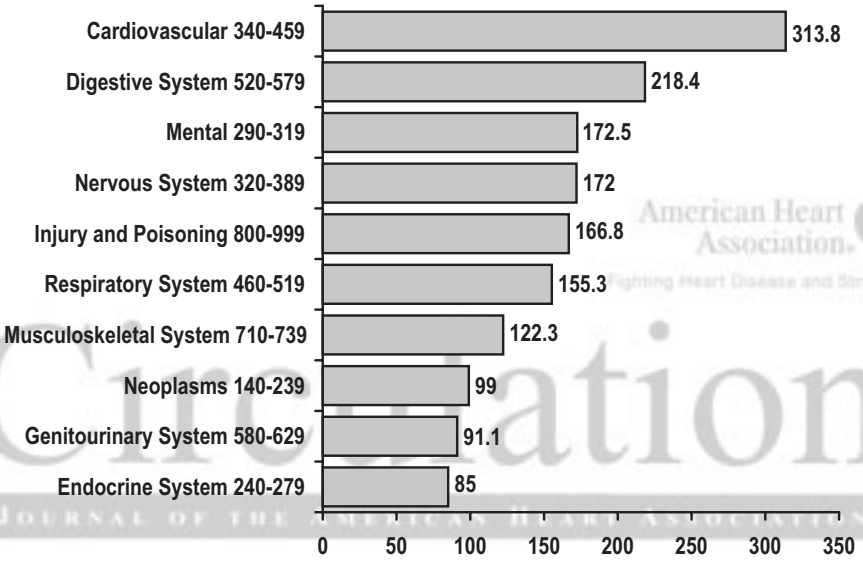


Chart 20-2. Direct costs of the 10 leading diagnostic groups (United States: 2009). Source: NHLBI.

21. At-a-Glance Summary Tables

See Tables 21-1 through 21-4.[1–6]

References

1. American Heart Association. Men and cardiovascular disease: statistics. Available at: http://www.americanheart.org/presenter.jhtml?identifier=3000935. Accessed September 15, 2008.
2. American Heart Association. Women and cardiovascular disease: statistics. Available at: http://www.americanheart.org/presenter.jhtml?identifier=3000941. Accessed September 15, 2008.
3. Bolen JC, Rhodes L, Powell-Griner EE, Bland SD, Holtman D. State-specific prevalence of selected health behaviors, by race and ethnicity: Behavioral Risk Factor Surveillance System, 199. *MMWR Morbid Mortal Wkly Rep*. 2000;49(SS-2):1–60.
4. American Heart Association. Statistical fact sheets: populations. Available at: http://www.americanheart.org/presenter.jhtml?identifier=2011. Accessed September 15, 2008.
5. Eaton DE, Kann L, Kinchen S, Shanklin S, Ross J, Hawkins J, Harris WA, Lowry R, McManus T, Chyen D, Lim C, Brener ND, Wechsler H. Youth risk behavior surveillance: United States, 2007. *MMWR Morbid Mortal Wkly Rep*. 2008;57(SS-4):1–131.
6. American Heart Association. Congenital cardiovascular defects: statistics. Available at: http://www.americanheart.org/presenter.jhtml?identifier=3020898. Accessed September 15, 2008.

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†	80.0 M (36.3%)	38.7 M (37.6%)	37.8%	45.9%	26.1%
Mortality, 2005§	864.5 K	409.9 K	329.6 K	47.4 K	N/A
CHD					
Prevalence, CHD, 2006†	16.8 M (7.6%)	8.7 M (8.6%)	8.8%	9.6%	5.4%
Prevalence, MI, 2006†	7.9 M (3.6%)	4.7 M (4.7%)	4.9%	5.1%	2.5%
Prevalence, AP, 2006†	9.8 M (4.4%)	4.3 M (4.3%)	4.1%	4.4%	3.5%
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, 2005 CHD§	445.7 K	232.1 K	203.9 K	22.9 K	N/A
Mortality, 2005 MI§	151.0 K	80.1 K	70.8 K	7.5 K	N/A
Stroke					
Prevalence, 2006†	6.5 M (2.9%)	2.6 M (2.6%)	2.3%	3.9%	2.1%
New and recurrent strokes§	795.0 K	370.0 K	325.0 K	45.0 K	N/A
Mortality, 2005§	143.6 K	56.6 K	47.2 K	7.5 K	N/A
HBP					
Prevalence, 2006†	73.6 M (33.3%)	35.3 M (34.1%)	34.1%	44.4%	23.1%
Mortality, 2005§	57.4 K	24.0 K	17.3 K	6.0 K	N/A
HF					
Prevalence, 2006†	5.7 M (2.5%)	3.2 M (3.2%)	3.1%	4.2%	2.1%
Mortality, 2005§††	292.2 K	126.2 K	112.6 K	11.3 K	N/A
Tobacco					
Prevalence, 2006‡	47.1 M (20.8%)	26.2 M (23.5%)	23.5%	26.1%	20.1#
Blood cholesterol					
Prevalence, 2006					
Total cholesterol ≥200 mg/dL†	98.6 M (45.1%)	45.0 M (42.6%)	42.1%	35.6%	52.1%
Total cholesterol ≥240 mg/dL†	34.4 M (15.7%)	14.6 M (13.8%)	14.3%	7.9%	17.5%
LDL cholesterol ≥130 mg/dL†	71.8 M (32.8%)	35.8 M (33.8%)	31.0%	36.2%	45.0%
HDL cholesterol <40 mg/dL†	33.9 M (15.5%)	26.3 M (24.9%)	24.9%	13.5%	30.6%
PA**					
Prevalence, 2007‡	30.8%	33.9%	N/A	N/A	N/A
Overweight and obesity					
Prevalence, 2006					
Overweight and obesity, BMI ≥25.0†	145.0 M (66.7%)	76.9 M (73.0%)	72.4%	73.7%	74.8%
Obesity, BMI ≥30.0†	74.1 M (33.9%)	34.7 M (32.7%)	32.3%	36.8%	26.8%
Diabetes mellitus					
Prevalence, 2006					
Physician-diagnosed diabetes†	17.0 M (7.7%)	7.5 M (7.4%)	5.8%	14.9%	11.3%
Undiagnosed diabetes†	6.4 M (2.9%)	3.9 M (3.8%)	3.6%	4.7%	6.0%
Prediabetes†	57.0 M (25.9%)	34.0 M (31.7%)	32.0%	22.9%	28.5%
Incidence, diagnosed diabetes†	1.6 M	N/A	N/A	N/A	N/A
Mortality, 2005§	75.1 K	36.5 K	29.6 K	5.7 K	N/A

AP indicates angina pectoris (chest pain); BMI, body mass index; CHD, coronary heart disease (includes heart attack, angina pectoris [chest pain] or both); CVD, cardiovascular disease; K, thousands; M, millions; MI, myocardial infarction (heart attack); mg/dL, milligrams per deciliter; and N/A, data not available.

*New and recurrent MI and fatal CHD.

†Age ≥20 years.

‡Age ≥18 years.

§All ages.

‖Age ≥45 years.

¶Age ≥35 years.

#Hispanic.

**Regular leisure-time physical activity.

††Total mentions.

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]

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†	80.0 M (36.3%)	41.3 M (34.9%)	33.3%	45.9%	32.5%
Mortality, 2005§	864.5 K	454.6 K	372.2 K	52.4 K	N/A
CHD					
Prevalence, CHD, 2006†	16.8 M (7.6%)	8.1 M (6.8%)	6.6%	9.0%	6.3%
Prevalence, MI, 2006†	7.9 M (3.6%)	3.2 M (2.7%)	3.0%	2.2%	1.1%
Prevalence, AP, 2006†	9.8 M (4.4%)	5.5 M (4.5%)	4.3%	6.7%	4.5%
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, 2005 CHD§	445.7 K	213.6 K	186.5 K	23.1 K	N/A
Mortality, 2005 MI§	151.0 K	70.9 K	61.6 K	8.0 K	N/A
Stroke					
Prevalence, 2006†	6.5 M (2.9%)	3.9 M (3.2%)	3.2%	4.1%	3.8%
New and recurrent strokes§	795.0 K	425.0 K	365.0 K	60.0 K	N/A
Mortality, 2005§	143.6 K	87.0 K	74.7 K	10.0 K	N/A
HBP					
Prevalence, 2006†	73.6 M (33.3%)	38.3 M (32.1%)	30.3%	43.9%	30.4%
Mortality, 2004§	57.4 K	33.3 K	25.8 K	6.7 K	N/A
HF					
Prevalence, 2006†	5.7 M (2.5%)	2.5 M (2.0%)	1.8%	4.2%	1.4%
Mortality, 2005§††	292.2 K	166.1 K	148.6 K	14.9 K	N/A
Tobacco					
Prevalence, 2006‡	47.1 M (20.8%)	20.9 M (18.1%)	18.8%	18.5%	10.1%#
Blood cholesterol					
Prevalence, 2006					
Total cholesterol ≥200 mg/dL†	98.6 M (45.1%)	53.6 M (47.1%)	47.7%	41.4%	48.0%
Total cholesterol ≥240 mg/dL†	34.4 M (15.7%)	19.8 M (17.3%)	18.1%	13.4%	14.5%
LDL cholesterol ≥130 mg/dL†	71.8 M (32.8%)	36.0 M (31.7%)	33.7%	27.4%	30.3%
HDL cholesterol <40 mg/dL†	33.9 M (15.5%)	7.5 M (6.7%)	6.5%	6.1%	10.5%
PA**					
Prevalence, 2007‡	30.8%	28.9%	N/A	N/A	N/A
Overweight and obesity					
Prevalence, 2005					
Overweight and obesity, BMI ≥25.0†	145.0 M (66.7%)	68.1 M (60.5%)	57.5%	77.7%	73.0%
Obesity, BMI ≥30.0†	74.1 M (33.9%)	39.4 M (35.0%)	32.7%	52.9%	41.9%
Diabetes mellitus					
Prevalence, 2006					
Physician-diagnosed diabetes†	17.0 M (7.7%)	9.5 M (8.0%)	6.1%	13.1%	14.2%
Undiagnosed diabetes†	6.4 M (2.9%)	2.5 M (2.1%)	2.2%	3.1%	1.9%
Prediabetes†	57.0 M (25.9%)	23.0 M (19.9%)	18.7%	19.0%	23.6%
Incidence, diagnosed diabetes†	1.6 M	N/A	N/A	N/A	N/A
Mortality, 2005§	75.1 K	38.6 K	30.1 K	7.2 K	N/A

AP indicates angina pectoris (chest pain); BMI, body mass index; CHD, coronary heart disease (includes heart attack, angina pectoris [chest pain], or both); CVD, cardiovascular disease; K, thousands; M, millions; MI, myocardial infarction (heart attack); mg/dL, milligrams per deciliter; and N/A, data not available.

*New and recurrent MI and fatal CHD.

†Age ≥20 years.

‡Age ≥18 years.

§All ages.

‖Age ≥45 years.

¶Age ≥35 years.

#Hispanic.

**Regular leisure-time physical activity.

††Total mentions.

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]

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

		Whites		Blacks		Mexican Americans		Hispanics/Latinos	
Diseases and Risk Factors	Both Sexes	Males	Females	Males	Females	Males	Females	Males	Females
Total CVD									
Prevalence, 2006†	80.0 M (36.3%)	37.8%	33.3%	45.9%	45.9%	26.1%	32.5%	N/A	N/A
Mortality, 2005§	864.5 K	329.6 K	372.2 K	47.4K	52.4 K	N/A	N/A	N/A	N/A
CHD									
Prevalence, CHD, 2006†	16.8 M (7.6%)	8.8%	6.6%	9.6%	9.0%	5.4%	6.3%	5.7%‡	
Prevalence, MI, 2006†	7.9 M (3.6%)	4.9%	3.0%	5.1%	2.2%	2.5%	1.1%	N/A	N/A
Prevalence, AP, 2006†	9.8 M (4.4%)	4.1%	4.3%	4.4%	6.7%	3.5%	4.5%	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
Mortality, CHD, 2005§	445.7 K	203.9 K	186.5 K	22.9 K	23.1 K	N/A	N/A	N/A	N/A
Mortality, MI, 2005§	151.0 K	70.8 K	61.6 K	7.5 K	8.0 K	N/A	N/A	N/A	N/A
Stroke									
Prevalence, 2006†	6.5 M (2.9%)	2.3%	3.2%	3.9%	4.1%	2.1%	3.8%	2.5%‡	
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
Mortality, 2005§	143.6 K	47.2 K	74.7 K	7.5 K	10.0 K	N/A	N/A	N/A	N/A
HBP									
Prevalence, 2006†	73.6 M (33.3%)	34.1%	30.3%	44.4%	43.9%	23.1%	30.4%	20.6%‡	
Mortality, 2005§	57.4 K	17.3 K	25.8 K	6.0 K	6.7 K	N/A	N/A	N/A	N/A
HF									
Prevalence, 2006†	5.7 M (2.5%)	3.1%	1.8%	4.2%	4.2%	2.1%	1.4%	N/A	N/A
Mortality, 2005§**	292.2 K	112.6 K	148.6 K	11.3 K	14.9 K	N/A	N/A	N/A	N/A
Tobacco									
Prevalence, 2006‡	47.1 M (20.8%)	23.5%	18.8%	26.1%	18.5%	N/A	N/A	20.1%	10.1%
Blood cholesterol									
Prevalence, 2006									
Total cholesterol 200 ≥mg/dL†	98.6 M (45.1%)	42.1%	47.7%	35.6%	41.4%	52.1%	48.0%	N/A	N/A
Total cholesterol ≥240 mg/dL†	34.4 M (15.7%)	14.3%	18.1%	7.9%	13.4%	17.5%	14.5%	29.9%¶	
LDL cholesterol ≥130 mg/dL†	71.8 M (32.8%)	31.0%	33.7%	36.2%	27.4%	45.0%	30.3%	N/A	N/A
HDL cholesterol <40 mg/dL†	33.9 M (15.5%)	24.9%	6.5%	13.5%	6.1%	30.6%	10.5%	N/A	N/A
PA#									
Prevalence, 2007‡	30.8%	33.9%		22.9%		N/A	N/A	23.8%	
Overweight and obesity									
Prevalence 2006									
Overweight and obesity, BMI ≥25.0†	145.0 M (66.7%)	72.4%	57.5%	73.7%	77.7%	74.8%	73.0%	67.8%	
Obesity, BMI ≥30.0†	74.1 M (33.9%)	32.3%	32.7%	36.8%	52.9%	26.8%	41.9%	27.5%	
Diabetes mellitus									
Prevalence, 2006									
Physician-diagnosed diabetes†	17.0 M (7.7%)	5.8%	6.1%	14.9%	13.1%	11.3%	14.2%	11.1%	
Undiagnosed diabetes†	6.4 M (2.9%)	3.6%	2.2%	4.7%	3.1%	6.0%	1.9%	N/A	N/A
Prediabetes†	57.0 M (25.9%)	32.0%	18.7%	22.9%	19.0%	28.5%	23.6%	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
Mortality, 2005§	75.1 K	29.6 K	30.1 K	5.7 K	7.2 K	N/A	N/A	N/A	N/A

AP indicates angina pectoris (chest pain); BMI, body mass index; CHD, coronary heart disease (includes heart attack, angina pectoris [chest pain], or both); CVD, cardiovascular disease; K, thousands; M, millions; MI, myocardial infarction (heart attack); mg/dL, milligrams per deciliter; and N/A, data not available.

*New and recurrent MI and fatal CHD.

†Age ≥20 years.

‡Age ≥18 years.

§All ages.

‖Age ≥35 years.

¶BRFSS.[3]

#Regular leisure-time physical activity.

**Total mention.

Sources: See summary tables for each chapter in this update. For data on other ethnic groups, see other chapters and Statistical Fact Sheets.[4]

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, 2005†	3.6 K	1.9 K	1.7 K	1.6 K	1.3 K	0.3 K	0.3 K	N/A	N/A
Mortality, 2005 (age <15 years)	2.0 K	1.1 K	0.9 K	0.8 K	0.7 K	0.2 K	0.2 K	N/A	N/A
Tobacco									
Prevalence, ages 12 to 17 years									
Cigarette use in the past month, 2006	10.4%	10.0%	10.7%	11.8%	13.0%	5.9%	6.2%	8.6%*	7.7%*
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 years	165.8	165.4	166.3	166.5	165.9	166.5	165.1	162.3	160.8
Ages 12 to 19 years	160.4	156.8	164.2	154.5	165.0	161.7	162.8	158.2	163.1
Mean HDL cholesterol, mg/dL									
Ages 4 to 11 years	56.3	57.4	55.3	57.5	54.9	62.2	59.2	54.5	51.9
Ages 12 to 19 years	52.2	49.8	54.7	48.2	53.8	55.3	57.7	49.8	53.8
Mean LDL cholesterol, mg/dL	87.9	85.4	91.2	84.0	91.2	90.2	91.4	87.6	91.2
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 years	23.4 M (31.9%)	12.3 M (32.7%)	11.1 M (31.0%)	31.9%	29.5%	30.8%	39.2%	40.8%	35.0%
Students in grades 9 to 12§	15.8%	15.1%	9.6%	15.7%	12.8%	16.6%	21.4%	18.3%*	17.9%*

K indicates thousands; M, millions; mg/dL, milligrams per deciliter; overweight, body mass index in the 95th percentile of the CDC 2000 growth chart; and N/A, data not available.

*Hispanic.

†All ages.

‡Regular leisure-time physical activity.

§CDC.[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]

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 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 mortality.
- *Agency for Healthcare Research and Quality (AHRQ)*—A part of the US Department of Health and Human Services, this is the lead agency charged with supporting research designed to improve the quality of health care, to reduce the cost of health care, to improve patient safety, to decrease the number medical errors, and to 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 (USDHHS). 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 (ongoing)
  - — 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)
- *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 mortality by the population gives a crude death rate. It is restricted because it does not reflect a population's composition with regard to such characteristics as age, sex, race, or ethnicity. Thus, 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." Mortality data for states can be obtained from the NCHS Web site (http://cdc.gov/nchs/), by direct communication with the CDC/NCHS, or from our National Center Biostatistics Program Coordinator on request. (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 our data are for Mexican Americans or Mexicans, as reported by government agencies or specific studies. In many cases, data for all Hispanics are more difficult to obtain.
- *Hospital discharges*—The number of inpatients 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 about 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, 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*—The total number of deaths from a given disease in a population during a specific interval of time, usually a year. 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 about 2 years. Mortality is "hard" data, so it is possible to do time-trend analysis and compute percentage changes over time.
- *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:
  - — 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 report 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 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);

*According to criteria established by the American Heart Association/National Heart, Lung, and Blood Institute, in "Diagnosis and Management of the Metabolic Syndrome: An American Heart Association/National Heart, Lung, and Blood Institute Scientific Statement," published in *Circulation* (*Circulation*. 2005;112:2735–2752).

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 or contributing cause of death*—These terms are used by the NCHS when defining mortality. Underlying mortality 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 mortality would be any other disease or condition that the decedent may also have had.