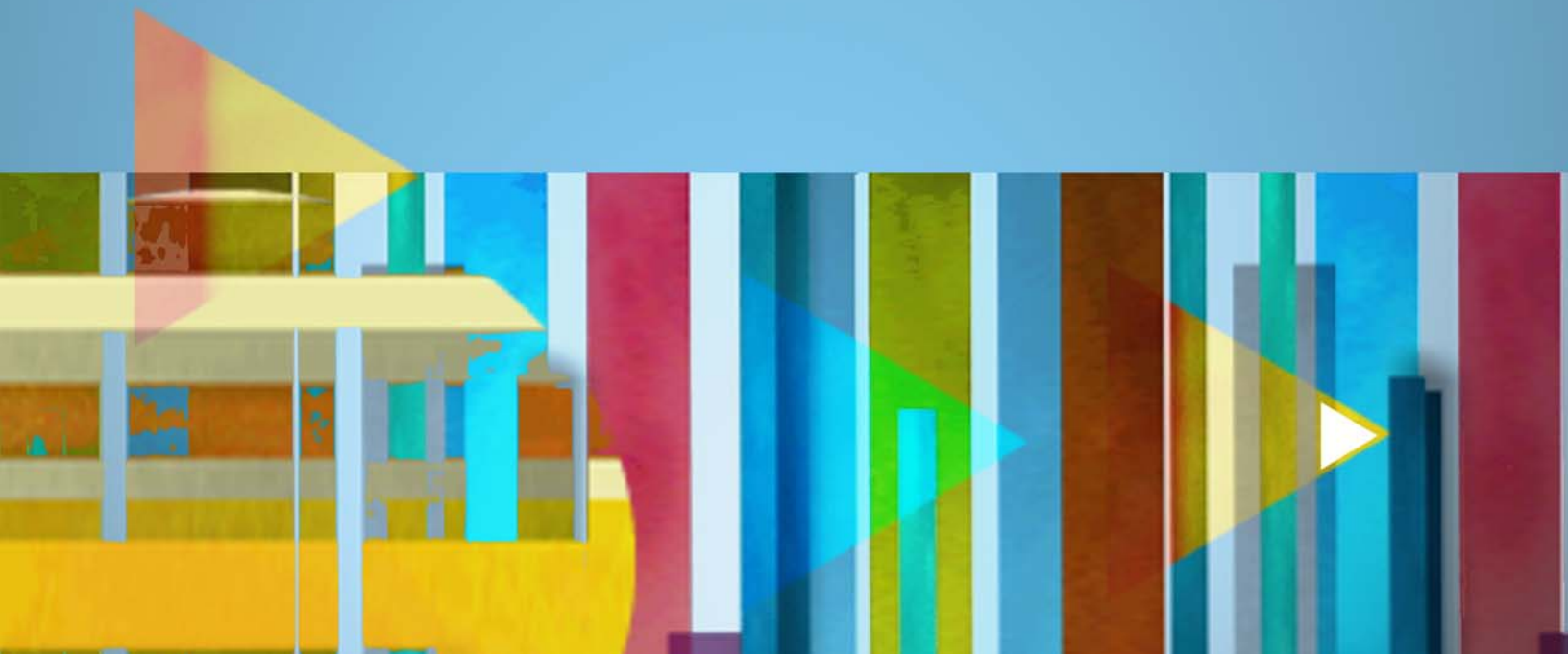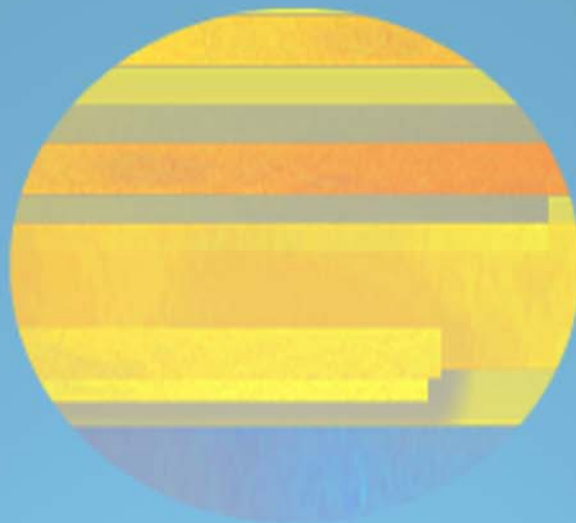Connecticut Department of Public Health

MORTALITY AND ITS RISK FACTORS IN CONNECTICUT

1989 - 1998

Acknowledgments

Mortality and Its Risk Factors in Connecticut, 1989 to 1998

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EXECUTIVE SUMMARY

Mortality and Its Risk Factors in Connecticut, 1989 to 1998 examines trends in the leading causes of death among Connecticut residents by gender, race/ethnicity, and age. Each chapter discusses risk factors for mortality and disease prevention and presents summary tables of age-adjusted mortality (AAMR) and premature mortality (years of potential life lost or YPLL) by gender and race/ethnicity. Statistical tests of significance were performed to assess mortality differences between population subgroups and to assess changes within population subgroups over time. Statistically significant changes of $p < .05$ or better are referred to as "significant." This report also charts Connecticut's progress over time in relation to U.S. mortality and the *Healthy People 2000* and *Healthy Connecticut* target objectives. The study period ends in 1998 since that marks the end of the twenty-year ICD-9 classification era, and statistics based on the ICD-10 are not exactly comparable to earlier figures based on the ICD-9.

All Causes of Death, 1989-1998

- Age-adjusted mortality rates for all causes of death were consistently lower for both Connecticut male and female residents compared with their counterparts nationwide. About 70% of all deaths among Connecticut residents for the ten-year period were due to cardiovascular diseases or cancer.

- Male all-cause mortality decreased significantly by about 1.4% per year from 1989 to 1998. This decrease is accounted for by significant decreases in mortality within the white and black male Connecticut resident populations. Female all-cause mortality did not change significantly in the ten-year period. The male all-cause mortality rate was about 1.5 times higher than the respective female rate (1996-98 period).

- Premature mortality (to age 75) for all causes of death, as measured by age-adjusted rates of years of potential life lost (YPLL), decreased significantly for both Connecticut males and females between 1989-91 and 1996-98. Male premature mortality (YPLL) was 1.7 times higher than that of females (1996-98 period).

- Male all-cause mortality (1996-98 period) was highest among black residents, followed by white, Native American, Hispanic, and Asian and Pacific Islander residents. The all-cause mortality rate of black males was significantly higher than that of white males. All-cause mortality rates for Hispanic and Asian and Pacific Islander males were significantly lower than the white male rate. The all-cause mortality rate of Native American males was not significantly different from the respective white male rate.

- Female all-cause mortality (1996-98 period) was highest among black, followed by white, Hispanic, Native American, and Asian and Pacific Islander female residents. Black females had significantly higher, whereas Hispanic, Native American, and Asian and Pacific Islander females had significantly lower, mortality rates compared with white females.

Chronic Disease Deaths, 1989-1998

- Heart disease is the leading cause of death for both males and females in Connecticut and the second leading cause of premature mortality to age 75. There was a significant decrease in the age-adjusted heart disease death and premature mortality rates for all Connecticut residents from 1989-91 to 1996-98.

- Cerebrovascular disease (stroke) is the third leading cause of death for all Connecticut residents. There was a significant decrease in black male mortality from the 1989-91 to 1996-98 periods and the stroke mortality disparity between black and white males in 1989-91 was reduced by 1996-98. There was a significantly higher stroke mortality rate for black compared

with white females. Premature stroke mortality rates for black males and females were significantly higher than those for white males and females.

- Hypertension-related mortality for all Connecticut residents increased significantly from the 1989-91 to 1996-98 period, which is largely accounted for by increased mortality in the white population. Black males and females had the highest age-adjusted death and premature mortality rates for hypertension-related conditions of all racial/ethnic subgroups. Hypertension-related death and premature mortality rates for black males and females were significantly higher than comparable rates for white males and females in both time periods.

- Diabetes is the seventh leading cause of death for all Connecticut residents. Age-adjusted diabetes death and premature mortality rates increased significantly from 1989-91 to 1996-98, a trend largely accounted for by increased diabetes mortality in the white population. Black males and females had the highest age-adjusted diabetes death and premature mortality rates of all Connecticut subpopulation groups.

- Cancer is the second leading cause of death and the leading cause of premature mortality to age 75 for all Connecticut residents. There was a significant decrease in male age-adjusted cancer death and premature mortality rates and a significant decrease in female premature mortality from 1989-91 to 1996-98.

- Lung cancer is the leading cause of cancer death. Connecticut males had significantly higher lung cancer death and premature mortality rates than females, while black males had the highest death and premature mortality rates of all subpopulation groups in the ten-year period. Female lung cancer mortality increased significantly while male lung cancer mortality decreased significantly from 1989-91 to 1996-98.

- Colorectal cancer is the second leading cause of cancer death among males and the third leading cause of cancer death among females in Connecticut. The age-adjusted colorectal cancer death rate decreased significantly from 1989-91 to 1996-98, a trend largely accounted for by decreased mortality in the white population. Males had significantly higher death and premature mortality rates due to colorectal cancer than females in Connecticut.

- Breast cancer is the second leading cause of cancer deaths among women. Black females had the highest breast cancer death and premature mortality rates (1996-98 period) of all subpopulation groups in Connecticut. There was a significant decrease in breast cancer death and premature mortality rates for Connecticut females from 1989-91 to 1996-98, a trend accounted for by decreased mortality in the white female population.

- There were significant decreases in prostate cancer death and premature mortality rates from 1989-91 to 1996-98 for Connecticut males, a trend accounted for by decreased mortality in the white male population. Black males had the highest prostate cancer death and premature mortality rates of all subpopulation groups.

- Chronic obstructive pulmonary disease (COPD) and allied conditions was the fifth leading cause of death in 1989-91 and the fourth leading cause of death in 1996-98. There was a significant increase in white female COPD mortality from 1989-91 to 1996-98. White Connecticut residents had significantly higher age-adjusted COPD mortality than did black and Hispanic residents.

- Chronic liver disease and cirrhosis was the ninth leading cause of death in 1989-91 and the tenth leading cause of death in 1996-98 for all Connecticut residents. Males accounted for 62% of all deaths. Hispanic males had the highest chronic liver disease and cirrhosis death and premature mortality rates of all subpopulation groups in 1996-98.

Injury Deaths, 1989-1998

- Unintentional injury was the sixth leading cause of death for Connecticut residents in 1996-98 and the leading cause of death for those aged 44 and under. There was a significant increase in unintentional injury mortality from 1992-94 to 1996-98.

- Motor vehicle crashes are the leading cause of injury death, accounting for 36% of all unintentional injury deaths from 1989 to 1998 in Connecticut. There was a significant decrease in motor vehicle crash mortality from 1989-91 to 1996-98. Males accounted for 68% of all motor vehicle crash deaths in the ten-year period.

- Fall and fall-related injuries are the second leading cause of unintentional injury death. White Connecticut residents accounted for 97% of all fall and fall-related injuries, and residents aged 65 and older accounted for 85% of these deaths in 1996-98.

- Suicide was the tenth leading cause of death for all Connecticut residents in 1989-91 and the eleventh leading cause of death in 1996-98. It was the third leading cause of death for those aged 15 to 24 in both periods. Males accounted for 79% of all suicides, and 42% of all suicides were by firearm during the ten-year period.

- Homicide and legal intervention was the fifteenth leading cause of death for Connecticut residents and the second leading cause of death for those aged 15 to 24. Males accounted for 77% of all homicide and legal intervention deaths from 1989 to 1998; 68% of all homicide and legal intervention deaths were by firearm.

- Drug-induced mortality increased significantly from 1992-94 to 1996-98 in Connecticut, a trend accounted for by increased mortality in the white population. Opiates & related narcotics and cocaine were the main subcategories of drug-induced deaths. Males aged 20 to 49 years accounted for 64% of all drug-induced deaths.

Infectious Disease Deaths, 1989-1998

- Pneumonia and influenza (P & I) was the fourth leading cause of death in 1989-91 and the fifth leading cause of death in 1996-98 for all Connecticut residents. There was a significant decrease in P&I mortality from 1989-91 to 1996-1998, a trend accounted for by decreased mortality in the white female population. Black males and females had significantly higher premature mortality rates due to P & I compared with white males and females in 1996-98.

- Septicemia was the eighth leading cause of death for all Connecticut residents. Black males and females had significantly higher septicemia death and premature mortality rates compared with white males and females. There was a significant decrease in all Connecticut resident septicemia mortality from 1989-1991 to 1996-98, a trend largely accounted for by decreased mortality in the white female population.

- HIV infection was the thirteenth leading cause of death for all Connecticut residents in 1996-98 but the third leading cause of death for black residents, and the fourth leading cause of death for Hispanic residents. HIV infection was the fourth leading cause of premature mortality to age 75 in 1989-91 and 1996-98. The HIV mortality rate for all Connecticut residents showed an increasing trend from 1989 to 1995; from 1995 through 1998, HIV mortality appeared to decline. These data parallel national trend data.

SECTION I.

INTRODUCTION

This report examines trends in the leading causes of death among Connecticut residents for the ten-year period 1989-1998. Mortality data are some of the best sources of information about the health of living communities. They provide a snapshot of current health problems, suggest persistent patterns of risk in specific communities, and show trends in specific causes of death over time. Many causes of death are preventable or treatable and, therefore, warrant the attention of public health prevention efforts. Furthermore, because mortality data allow us to identify leading causes of premature death, they provide a valuable benchmark for evaluating progress in increasing years of healthy life for Connecticut residents. As such, they are important indicators of where federal, state, and local prevention efforts should be placed in building healthy communities.

The U.S. Department of Health and Human Services' initiative *Healthy People*, first launched in 1979, provides a framework for analyzing mortality and its risk factors. The national goals and objectives for *Healthy People 2000*, released in 1990, identified health priority areas for the nation (U.S. Department of Health and Human Services 1990). Its goals were to increase the healthy life years of Americans, to reduce health disparities among subgroups within the population, and to achieve universal access to appropriate health services. Overall, *Healthy People 2000* includes about 38 objectives that rely on mortality figures.

In accordance with the national objectives, the Connecticut Department of Public Health developed a framework to assess the health of Connecticut residents by setting objectives by which to measure progress to the year 2000. That framework, known as *Healthy Connecticut 2000,* was outlined in a series of reports. The *Healthy Connecticut 2000* baseline assessment (Connecticut Department of Public Health 1997) set target objectives for health priority areas, and *Looking Toward 2000* (Connecticut Department of Public Health 1999) assessed health status and health services in Connecticut. This report uses the *Healthy People 2000* and *Healthy Connecticut* objectives to measure progress in reducing mortality among Connecticut residents. It also provides a yardstick for Connecticut residents as we measure progress in relation to the *Healthy People 2010* objectives (U.S. Department of Health and Human Services 2000).

While providing a retrospective look at mortality trends for the past decade, findings presented in this report also enable the state of Connecticut to assess its current health status with a view toward setting new goals and objectives for the next decade and millennium. This report comes at an important time for a few reasons:

- Recently there has been a change in the disease classification system used to report mortality statistics throughout the United States. This report covers the period 1989-1998 and uses mortality classifications based on the International Classification of Diseases – 9 (ICD-9) coding standard (World Health Organization 1977). Mortality data from 1999 and later will be classified using the ICD-10 codes. This report includes information from 1989 to 1998, the end of the twenty-year ICD-9 classification era. Statistics based on the ICD-10 will not be exactly comparable to earlier figures based on the ICD-9; consequently, 1998 is a logical endpoint for examining recent mortality trends.

- For more than fifty years, the National Center for Health Statistics has reported mortality data standardized to the age, gender, and racial/ethnic composition of the 1940 population of the United States. Beginning in 2000, the age-adjustment formula used to report mortality data nationwide has been changed to reflect the older age composition of the U.S. population in the year 2000. This shift in the use of the standard population will result in numerous differences among population subgroups. In this report, we provide mortality rates that are age-adjusted to both the 1940 and 2000 standard populations. We use the 1940 standardization when comparing Connecticut resident death rates to U.S. rates from 1989-1998. We use the 2000 standardization to compare gender, racial, and ethnic groups within Connecticut during this same period and when examining changes over time. We discuss the effect of these changes at length in the Methodology and Discussion Sections of this report.

In this report, we describe mortality trends from 1989 to 1998 within and among major population subgroups in Connecticut: males and females, and racial and ethnic sub-populations (whites, blacks/African Americans, Hispanics, Asian and Pacific Islanders, and Native Americans). With this information, we are able to chart Connecticut's progress over time in relation to U.S. mortality rates, the *Healthy People* and *Healthy Connecticut* target objectives, as well as Connecticut's progress toward eliminating health disparities among racial and ethnic sub-populations. We are also able to identify meaningful changes in the health of Connecticut residents during the past decade as well as the problematic areas requiring our attention as we begin to formulate the *Healthy Connecticut* goals for 2010.

SECTION II.

RISK GROUPS AND RISK FACTORS FOR MORTALITY

Social Context

During the twentieth century, Americans experienced a remarkable decline in deaths due to infectious and communicable diseases and a parallel increase in average life expectancy. This decrease in infectious disease mortality has been attributed to a variety of public health measures, such as better nutrition, improved sanitation, better housing conditions, and clean water (McKeown 1975; McKinlay and McKinlay 1977). Advances in medical technology and health care during this period have also enabled people with serious disability and chronic illnesses to live longer. These historic changes in the health of the nation were addressed in a federal initiative that began in the Carter Administration known as *Healthy People* (U.S. Department of Health, Education and Welfare 1979). *Healthy People* emphasized the importance of risk factors in disease prevention.

The term "risk factor" refers to an inherited characteristic of an individual, an environmental exposure, or some aspect of personal behavior that, based on epidemiologic evidence, is known to be associated with some disease condition or health outcome considered preventable (Last 1988). Aspects of social status, such as age, gender, race/ethnicity, marital status, low socioeconomic status or poverty are often viewed as risk factors because they can serve as powerful predictors or markers of disease risk in a given population. Age, gender, race/ethnicity, low socioeconomic status, and poverty may also be viewed as "risk groups," that is, the main strata by which mortality and morbidity differentials are analyzed.

We use age, gender, race/ethnicity, and year of death to identify subgroups of the Connecticut resident mortality data that are analyzed in this study. We do not analyze other social, environmental, or behavioral risk factors for Connecticut resident mortality, since the available mortality data do not include measurements of these factors. Our interpretation of trends, however, requires that we view these mortality data within the context of knowledge gained from studies linking specific risk factors, whether social, environmental, or behavioral, with certain causes of death. For this reason, we have incorporated evidence from various national and statewide sources in our discussion of cause-specific mortality when appropriate.

The *Healthy People* Initiative

For two decades, the *Healthy People* Initiative has provided us with a framework for understanding the underlying factors that put Americans at risk for premature death with an emphasis on prevention of disease and promotion of health. *Healthy People – The Surgeon General's Report on Health Promotion and Disease Prevention*, released in 1979, heralded a national effort aimed at preventing premature illness, disability, and death. The report *Healthy People* noted that chronic diseases like heart disease, stroke, and cancer were the major causes of death and disability for Americans while injuries were the leading cause of death for those under the age of forty (U.S. Department of Health, Education, and Welfare 1979). Two more reports followed: in 1980,

Promoting Health/Preventing Disease: Objectives for the Nation identified over 200 health objectives for the next ten-year period; and in 1990, *Healthy People 2000: National Health Promotion and Disease Prevention Objectives*, identified health goals and objectives for the year 2000 (U.S. Department of Health and Human Services 1980a; 1990).

Since the 1960's, research studies have documented the relationship between mortality risk and health behaviors, such as eating and sleep patterns, physical activity, obesity, tobacco use, and alcohol consumption (Kannel 1967; Belloc and Breslow 1972; Berkman and Breslow 1983; Leon and Connett 1991; Manson, Wilett, Stampfer, et al. 1995; Goldberg, Larson, and Levy 1996; Kant, Schatzkin, Graubard, et al. 2000). It was estimated that, in 1990, the most important preventable contributors to mortality in the United States were tobacco use, diet and activity patterns, microbial agents, toxic agents, firearm use, sexual behavior, motor vehicle use, and illicit use of drugs (McGinnis and Foege 1993). With the knowledge gained from these studies, health care professionals have been able to formulate sound recommendations for the improved health of Americans (U.S. Department of Health, Education, and Welfare 1979; U.S. Department of Health and Human Services 1986; 1990; 2000).

In January of 2000, the Department of Health and Human Services announced the new national objectives for the year 2010. Referred to as "Healthy People in Healthy Communities," the *Healthy People 2010* goals are to continue the advancement in the quality of life for Americans and to ensure that all Americans benefit from these advancements regardless of their race, ethnicity, gender, disability status, income, or educational level (U.S. Department of Health and Human Services 2000). The goals of *Healthy People 2010* are informed by decades of research showing that individual health and behavior are inextricably linked to the health of the community and environment in which individuals live. An underlying premise of *Healthy People 2010* is that an individual's health must be viewed in the context of his or her community.

The overarching goal of *Healthy People 2010* is to increase quality and years of healthy life. During the twentieth century, life expectancy for persons in every age group has increased. Continued differences, however, in life expectancy between certain population groups—male vs. female, black vs. white, Hispanic vs. non-Hispanic, poor vs. non-poor—underscore the importance of examining their mortality differentials. A key objective of *Healthy People 2010* is the elimination of health disparities among racial and ethnic sub-populations. It is also noted in *Healthy People 2010* that inequalities in income and education underlie many health disparities in the United States.

The objectives of *Healthy People 2010* are focused on the combined effects of several determinants of health: the individual, physical, and social environments, as well as the policies and interventions that promote health, prevent disease, and ensure access to health care. A large body of research has established the relationship of social networks, income and educational level, and neighborhood of residence to mortality risk. Research studies have found that individuals with fewer personal and group relationships (Berkman and Breslow 1983; House, Landis, and Umberson 1981), lower levels of income and education (Syme and Berkman 1976; Lantz, House, Lepkowski, et al. 1998), and those living in poorer neighborhoods (Haan, Kaplan, and Camacho 1987; McCord and Freeman 1990) are at increased risk of death from a variety of causes. More recently, researchers

have examined the association of residential segregation and adult mortality. Polednak (1993) found that residential segregation was positively related to increased mortality due to all causes for black residents but inversely related for whites of 38 major U.S. metropolitan areas. Collins and Williams (1999) found that black social isolation tended to predict higher rates of mortality for African Americans in 107 major U.S. cities. All of these social factors should be considered in efforts to meet the *Healthy People* goal of eliminating health disparities by 2010.

Age-Adjusted Mortality among Connecticut Residents, 1989-1998

This report provides a ten-year retrospective look at mortality among Connecticut residents. It reports differences among various age, gender, and racial/ethnic groups because these social characteristics are so closely linked to mortality risk.

Age-group

Age is the overarching risk factor for mortality. With increasing age, come physiological and social changes in humans that are seen across sub-population groups. Different disease outcomes typically follow age-related patterns and the identification of age risk groups is the first step in designing useful interventions (Kaplan, Haan, and Wallace 1999). Furthermore, it is often difficult to see more subtle patterns in chronic disease progression without accounting for the overpowering effect of age on risk of death. In Connecticut, as in the U.S., the leading cause of death varies by age group. For example, between 1996 to 1998 unintentional injury was the leading cause of death for Connecticut residents under the age of 45, cancer the leading cause for those aged 45 to 74, and heart disease the leading cause for persons 75 years and older. This report classifies Connecticut residents into seven age groups (0-14; 15-24; 25-44; 45-64; 65-74; 75-84; and 85 and older) for the Leading Cause of Death Tables (Appendix V) and 18 five-year age groups (0-4; 5-9; 10-14; 15-19; 20-24; 25-29; 30-34; 35-39; 40-44; 45-49; 50-54; 55-59; 60-64; 65-69; 70-74; 75-79; 80-84; and 85 and older) for age-specific and regression analyses.

Gender

Gender is a key determinant of health status, life expectancy, and mortality risk. In Connecticut, as in the U.S. and all major industrialized nations, females have lower age-adjusted mortality for most causes compared with males. [In many developing countries, women's life span is shorter than men's due to maternal mortality and pregnancy-related risks (Santow 1995).] While these gender differences in mortality may be due, in part, to biological factors, there are a variety of important social factors that underlie these differences. For example, research suggests that American women engage in more help-seeking and preventive health behaviors linked to mortality risk compared with men (Verbrugge 1985).

The factors underlying gender differences in chronic disease mortality due to heart disease, stroke, diabetes, and cancer may be an interaction of both social and biological determinants. For example, biological differences clearly play a role in the development of certain types of cancers, such as prostate and breast cancer. Social factors, such as willingness to seek periodic preventive screening and to take appropriate measures once diagnosed, like dietary change and therapeutic treatments, are also important determinants of mortality risk.

Major gender differences exist in injury mortality as well. For example, Connecticut males are more than three times as likely to die from homicide and more than twice as likely to die from motor vehicle crashes and drug-induced causes as are females. These gender differences in mortality are directly linked to social behaviors such as risk-taking, interpersonal violence, and alcohol and drug use.

Race, Ethnicity, Income, and Education

Race, ethnicity, income, and education are overlapping characteristics. Higher income, for example, is strongly associated with higher education and both are strongly associated with better health status and lower mortality risk. Likewise, black race and Hispanic ethnicity tend to be associated with lower income, lower education, and poorer health outcomes. The independent contributions of lower income, lower education, and minority race/ethnicity to higher mortality risk are not easily disentangled. A vast body of research suggests that a complex interaction of factors including socioeconomic conditions, culture and acculturation, specific behaviors, and environmental conditions account for broad racial and ethnic disparities in health status and mortality (U.S. Department of Health and Human Services 1998a; Freeman 1993; Adler, Boyce, Chesney, et al. 1994).

Although genetic factors underlie observed differences among groups of people, research has pointed out that there is greater genetic variation within racial groups than there is among racial groups (Williams, Lavizzo-Mourey, and Warren 1994). Furthermore, racial and ethnic classifications used in the U.S. are socially, not biologically, constructed categories that have changed historically. Scientific evidence points out that racial and ethnic classifications do not capture biological distinctiveness (Fullilove 1998). Rather, the social construct of race embodies the perceptions and values of, and behavior toward, one group by another (Freeman 1998).

Social class, as measured by income, education, and occupation, is known to be another important determinant of mortality risk (Antonovsky 1967; Syme and Berkman 1976; van Rossum, Shipley, van de Mheen et al. 2000). Higher education tends to lead to higher status occupations, which tend to offer higher personal income. Higher personal income allows for greater leisure time, better access to medical care and technology, better housing in safer neighborhoods, and improved opportunities for education and information regarding healthy behaviors. Area-based measures of socioeconomic status, such as income-level in census tract of residence, have been shown to be associated with all-cause mortality, independent of individual income (Anderson, Sorlie, Backlund, et al. 1997). This report does not describe mortality differences among Connecticut residents by

social class because information on decedent's social class, as measured by income, education, occupation, or residential area, is not currently available for Connecticut resident deaths.

Race and ethnicity are strata by which we analyze Connecticut resident mortality in this report. Although we do not include Connecticut resident income, education, or occupation as variables in our analysis, we have incorporated contextual information on the relationship between socioeconomic factors and cause-specific mortality from national data in the narrative.

SECTION III.

METHODOLOGY

Origin of the Mortality Data

Mortality data in this report are taken from the Connecticut Death Registry. Virtually all deaths occurring to Connecticut residents in the United States and Canada are included in this data base. Mortality data are derived from the cause of death information reported on Connecticut death certificates that are completed by funeral directors, attending physicians, medical examiners, or coroners. Sociodemographic information on death certificates is often based on report by next of kin. Original records are filed in the state registration offices.

Several causes of death may be entered on the death certificate. Causes include all diseases, conditions, or injuries that may have resulted in or contributed to death as well as the circumstances or the event that produced any injuries. Tabulations of cause-of-death statistics in this report are based solely on the "underlying" cause of death unless otherwise stated. The underlying cause of death is the disease or injury that initiated the series of events leading directly to death or the circumstances of the event that resulted in the fatal injury. Examination of the combination of causes can shed additional light on factors related to mortality. Therefore, for selected diseases both underlying and contributing causes of death are used as the basis for defining categories in this report. For example, diabetes is the underlying cause of death for approximately 500 to 600 Connecticut residents per year. "Diabetes-related deaths," those deaths for which diabetes may be an underlying and/or a contributing cause, account for almost five times as many Connecticut resident deaths annually as does diabetes alone. Appendix I contains the cause of death categories included in this report with their respective ICD-9 codes.

Classification of Diseases

Classification of cause of death data is based on the Ninth Revision of the International Classification of Diseases (ICD-9), the internationally accepted coding system for determining the underlying and contributing causes of death (World Health Organization 1977). The ICD is revised periodically to take into account the discovery of new diseases and advances in medical diagnoses. This report uses the ninth revision of the International Classification of Diseases, or ICD-9. ICD-9, used since 1979, is replaced by the tenth revision (ICD-10) beginning with 1999 deaths. Changes adopted with the ICD-10 coding will affect how the leading causes of death are determined and may have an impact on the rankings of these causes (Anderson and Rosenberg 1998).

Selection of Cause of Death Categories

The 28 causes of death discussed in this report are a subset of 62 leading causes of death among Connecticut residents for which this analysis was performed. Summary tables of age-adjusted mortality rates (AAMR) and years of potential life lost rates (YPLL) by gender, race, and ethnicity and detailed analyses for the 62 causes of death are located in Appendix VII of this report.

The 62 selected causes of death were derived from the National Center of Health Statistics (NCHS) List of 72 Selected Causes of Death and HIV Infection (National Center for Health Statistics 1994a); the *Healthy People 2000* and *Healthy People 2010* target area categories (U.S. Department of Health and Human Services 1990; U.S. Department of Health and Human Services 2000); and the Council of State and Territorial Epidemiologists (CSTE) Indicators for Chronic Disease Surveillance (Lengerich 1999; Lengerich 2000). Selection of multiple-cause-of-death categories was based partly on an analysis of the ratio of multiple to underlying causes listed on 1997 Connecticut resident death certificates for 33 leading causes of death. Higher ratios identify conditions that are more likely to be listed as contributing rather than underlying causes of death. Such conditions may be important, but frequently overlooked, contributors to mortality. Based on this analysis, contributing causes of death selected for inclusion into this study are diabetes-related, septicemia-related, COPD-related, atherosclerosis-related, nephritis-related, and hypertension-related. Also, despite a relatively low ratio, the category "heart-disease-related deaths" was included due to the large number of additional deaths identified by considering contributing cause of death information. We determined the final list of cause-of-death groups in consultation with local health directors and with Department of Public Health (DPH) staff involved with health programs and surveillance.

Categories included in this report are ranked according to the NCHS leading causes of death. In addition to "all causes of death," there are a total of 14 broad cause-of-death category titles and 13 related or sub-categories (Appendix I). The 14 broad cause-of-death groups account for 82 percent of the total number of deaths (288,034) that occurred in the State during the 1989 to 1998 period.

Statistical Measures Used in This Report

This report provides an overall evaluation of age-specific mortality rates, age-adjusted mortality rates (AAMRs), and years of potential life lost rates (YPLL) for the state of Connecticut (CT) for the period 1989-1998. AAMRs and YPLLs for the period 1989-1991 (or 1992-1994 for select causes) are compared to those for the time period of 1996-1998 for any statistically significant differences. Subgroup analyses by gender, race, and ethnicity are provided for most causes of death.

Several methodological enhancements have been introduced in this report to provide a systematic and comprehensive assessment of the mortality patterns evaluated. These enhancements include the following:

- The margin of error for AAMRs and premature mortality (YPLL) statistics were calculated, thus allowing for a critical evaluation of changes over time and between-group differences;
- Trends over the study period (1989-1998) were assessed using logistic regression models in addition to the basic assessment of changes between 1989-1991 (or 1992-1994) and 1996-1998 found throughout this report. The development of these models allowed us: to estimate the 1989-1998 (or 1992-1998) trend slope, that is the average annual percent change in AAMR, and also to evaluate the statistical significance of these trends; to identify single year rates that do not lie on the trend line; to identify age groups whose trend line differs from the overall trend; and to make these assessments while adjusting for age and adopting appropriate significance thresholds for multiple comparisons.
- Disparities in AAMRs by race and ethnicity were assessed using logistic regression models in addition to making group comparisons for 1996-1998 AAMRs. The development of these models allowed us to identify age groups in which the disparity differed from the overall black/white or Hispanic/white difference in AAMRs and to identify age-specific disparities when no overall disparity was identified.

In addition, Connecticut AAMRs are compared to U.S. AAMRs, and to the Year 2000 Objectives for the state of Connecticut (Connecticut Department of Public Health 1999) and the United States (National Center for Health Statistics 1994b) for most cause of death groups. The leading causes of death among Connecticut residents are ranked by age, gender, and race and ethnicity (Appendix V). The population bases for computing rates were obtained from U.S. Census figures. Additional information on these statistical measures and methods, population denominators, and the definition of terms is presented in Appendix II. Detailed explanations of statistical models used and examples of the use of age-adjustment in calculating mortality and years of potential life lost rates are described in Appendix III and Appendix IV, respectively. Some of the data presented in this report are based on unpublished tables. These and other mortality data are available on the Connecticut Department of Public Health web page http://www.dph.state.ct.us.

Age Standardization of Death Rates: The 1940 and 2000 Standard Populations

Age-adjusted mortality rates (AAMR) and years of potential life lost rates (YPLL) are calculated per 100,000 population using the 1940 and 2000 U.S. standard million population. This adjustment of the mortality rates shows what the rates would be if the Connecticut population had the same age distribution as the U.S. population in 1940 and 2000. The AAMR and YPLL rates were calculated using the 1940 U.S. standard million in order to compare Connecticut's rates with the Connecticut and U.S. Year 2000 Objectives and U.S. AAMRs, which are based on the 1940 U.S. standard

million. AAMRs and YPLLs based on the 2000 standard population are used to compare subgroup populations by sex (male, female) and by race/ethnicity (white, black, Hispanic, Asian Pacific Islander, Native American) in Connecticut. Use of the 2000 standard has two major advantages. First, it more closely approximates the age distribution of Connecticut's population during the period 1989-1998; and secondly, it conforms to the new national standard. Beginning with 1999 deaths, the National Center for Health Statistics is reporting mortality rates adjusted by the 2000 standard million population. The impact of the shift in age standardization from 1940 to 2000 standard population is discussed in detail in Appendix IV-C.

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SECTION IV.

CAUSE OF DEATH GROUPINGS

CONTENTS

All Cause Mortality
(ICD-9 codes 001-E999)

During the 1989 to 1998 period in Connecticut, age-adjusted mortality rates due to all causes of death were consistently lower for both Connecticut male and female residents compared with the respective national figures (Figure 1). Although Connecticut does fare well in comparison with the nation, the age-adjusted all-cause mortality rate of Connecticut residents was significantly higher than the *Healthy Connecticut* target objective during this period (Table 1).

Almost 70% of all deaths among Connecticut residents for the ten-year period were due to cardiovascular diseases or cancer. Other major categories of death included injury, which accounted for about 5%, and pneumonia and influenza, and chronic obstructive pulmonary disease (COPD), which each accounted for about 4% of deaths (Figure 2).

Between the 1989-1991 and 1996-1998 periods, age-adjusted mortality rates for all causes decreased significantly for male but remained about the same for female residents of Connecticut. This trend is consistent with national data. The average annual decrease in male mortality was approximately 1.4% (p< .001) from 1989 to 1998. This

Figure 1.

All-Cause Mortality
Comparison of Connecticut and U.S. 1989-1998
Age-Adjusted Mortality Rates* by Sex

SEX

Symbols:
■ - U.S. rates for 1989-1998
✳ ● - Connecticut rates: an asterisk is used if the rate is significantly different than expected (p<.005), based on the log-linear trend.

* These rates are adjusted to the 1940 US standard million population.
* This classification includes deaths with ICD-9 codes:1-E999

decrease is accounted for by significant decreases in mortality within the white and black male resident populations. Age-adjusted rates of years of potential life lost (YPLL), a measure of premature mortality, decreased significantly for both men and women between the two time periods. The decrease in premature mortality among males is accounted for by significant decreases within the white and black male resident populations (Table 2).
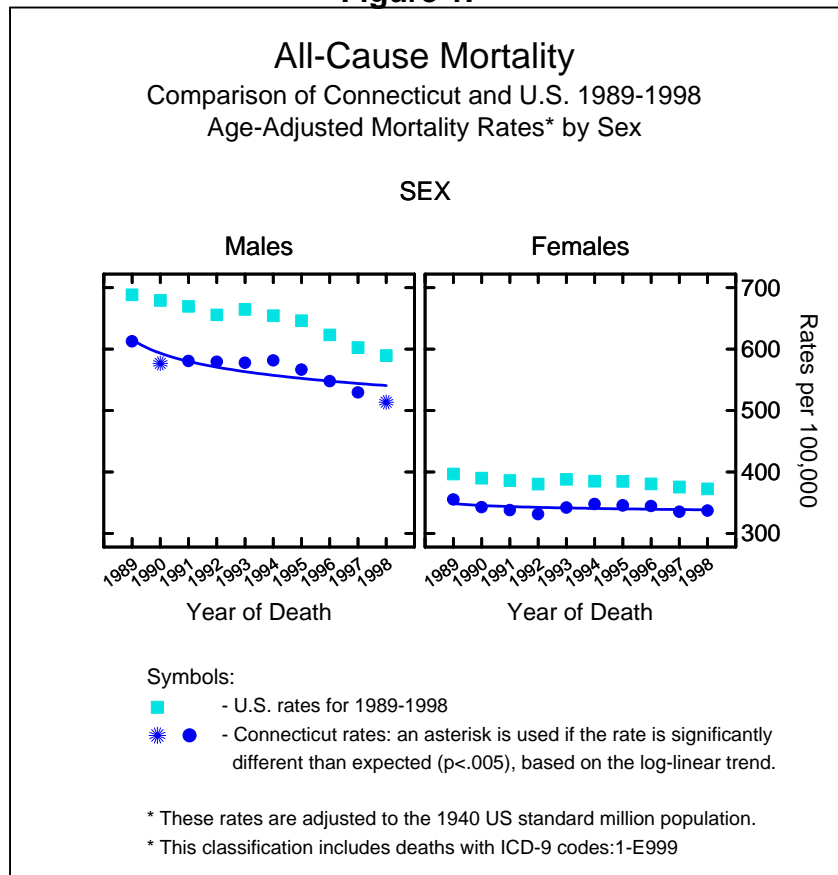
**Table 1. All-Cause Age-Adjusted Death Rates,
Comparison of CT with US - 1989 and 1998**

	1989	1998	1998 CT AAMR Comparison
CT AAMR*	468.2	416.9	
US AAMR*	527.3	471.7	CT AAMR < US AAMR
Healthy CT 2000	328.4	328.4	CT AAMR > *HCT2000* rate

* age-adjusted mortality rates are per 100,000 population, U.S. 1940 standard million population.

**Figure 2. All Mortality, Percent by Cause
Connecticut Residents, 1989-1998**

Number of Deaths - 288,034

All Other Causes 19%
COPD 4%
Pneumonia / Influenza 4%
Injury 5%
Cancer 24%
Cardiovascular 44%
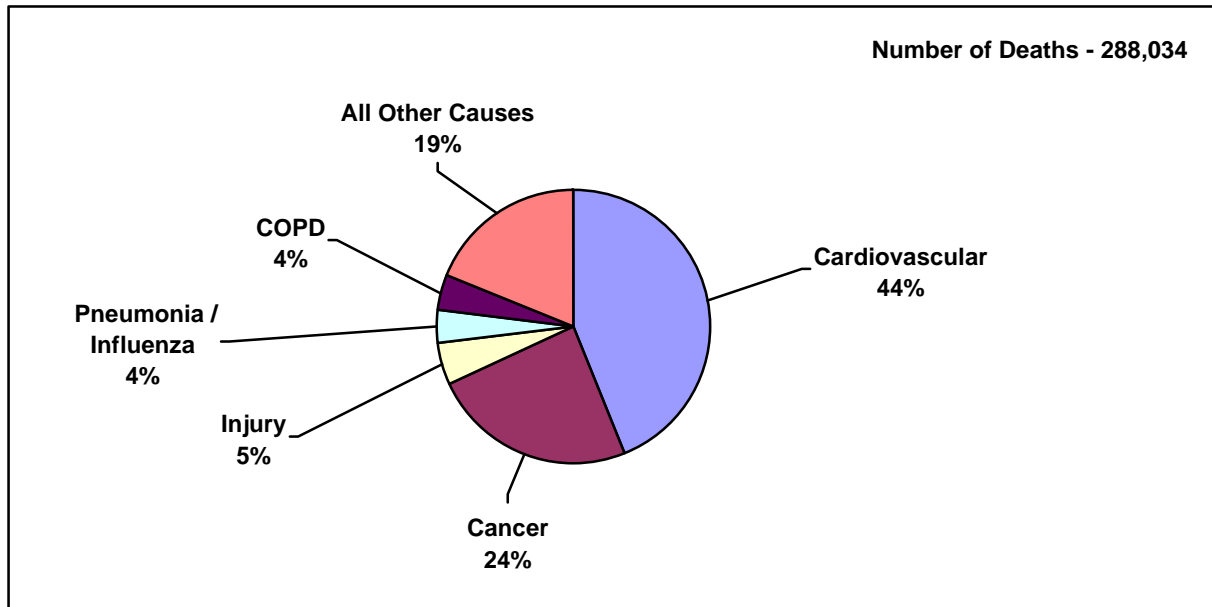
Gender differences in age-adjusted, all-cause mortality rates were consistently large during this time period. Male all-cause mortality rates were about 1.6 times higher than female rates in the 1989-1991 period and 1.5 times higher in the 1996-1998 period (differences were statistically significant at p < .001 during both periods). During the latter period, premature mortality (YPLL) among males was about 1.7 times higher than that of females (p < .001) [Table 2]. The male-female differential in mortality is consistent with comparable national figures and data from other industrialized countries (Wingard 1982, 1984; Waldron 1986, 1995a, 1995b; Nathanson 1990, 1995;

Hemstrom 1998; Nikiforov and Mamaev 1998). The Summary Section of this report contains a detailed discussion of gender differences in mortality.

Age-adjusted, all-cause mortality rates differed among racial and ethnic subgroups in the state (Table 2). For the period 1996-1998, male all-cause, age-adjusted mortality was highest among black residents, followed by white, Native American, Hispanic, and Asian and Pacific Islander residents. The all-cause mortality rate of black males was significantly higher than that of white males. All-cause mortality rates for Hispanic and Asian and Pacific Islander males were significantly lower than the white male rate. The all-cause mortality rate of Native American males was not significantly different from the respective white male rate. Female all-cause mortality was highest among black, followed by white, Hispanic, Native American, and Asian and Pacific Islander female residents for the period 1996-1998. During this period, black females had significantly higher, whereas Hispanic, Native American, and Asian and Pacific Islander females had significantly lower, mortality rates compared with white females (Table 2).

The disparity in black-white and Hispanic-white male all-cause mortality (1996-1998 period) differed by age group ($p < .0014$). For black compared with white males under age 65, the relative risk of death was consistent at 2.3 ($p < .001$), while the disparity for black compared with white males aged 65-84 lessened. Among males aged 85 and over, the all-cause mortality rate was significantly lower for blacks compared with whites. For Hispanic compared with white males under age 60, the relative risk of all-cause mortality was consistent at 1.5 ($p < .001$). There were no significant differences in all-cause mortality for Hispanic and white males aged 60-74. The disparity reversed for those aged 75 and over, with Hispanic males at significantly less risk of death due to all causes compared with white males.

The disparity in black-white and Hispanic-white female all-cause mortality (1996-1998 period) differed by age group ($p < .0014$). For black compared with white females under age 65, the relative risk of death was consistent at 2.0 ($p < .001$), while the disparity for black compared with white females aged 65-84 lessened. Among females aged 85 and over, the all-cause mortality rate was significantly lower for blacks compared with whites. Hispanic females have significantly lower mortality than white females (RR=0.7, $p < .001$ for all ages) and this difference is fairly consistent across age groups with a few exceptions. Hispanic females ages 40-44 have a significantly greater all-cause mortality rate than white females and Hispanic females aged 80 and over have a substantially lower mortality rate than their white counterparts.

Premature mortality (YPLL) rates present a slightly different pattern by race and ethnicity. Age-adjusted, all cause YPLL rates were significantly higher for black and Hispanic male residents but lower for Asian and Pacific Islander males compared with white male resident rates during the 1996-1998 period. Although Hispanic males had significantly lower age-adjusted all-cause mortality rates compared with whites, they had significantly higher *premature* mortality rates. The age-adjusted YPLL rate of Native American males was not significantly different from the respective white male rate. Between the period 1989-1991 and 1996-1998, all-cause mortality rates and premature mortality rates decreased significantly for both white and black males. Rates for the other population subgroups during this time period remained about the same (Table 2).

Table 2. All Causes of Death[1], Connecticut Residents by Gender, Race and Ethnicity[2], 1996-1998

Group	Number of Deaths	Age-Adjusted Mortality Rates[3]		Age-Adjusted Premature Mortality Rates to Age 75[3]	
		AAMR[4]	Change since 1989-91[5]	YPLL[4]	Change since 1989-91[5]
All Residents	88,566	815.1	↓↓↓	6,648.1	↓↓↓
All males	42,173	999.0	↓↓↓	8,386.5	↓↓↓
White	38,650	978.0	↓↓↓	7,555.8	↓↓↓
Black	3,118	1,270.9***	↓↓↓	17,041.0***	↓↓↓
Asian PI	171	374.2***	ns	3,072.6***	ns
Native American	56	761.8	ns	10,907.3	ns
Hispanic	1,351	729.3***	ns	10,202.2***	ns
All females	46,393	684.3	ns	4,972.6	↓
White	43,320	671.4	ns	4,524.0	ns
Black	2,780	846.9***	ns	9,236.4***	ns
Asian PI	141	259.1***	ns	2,149.7***	ns
Native American	34	356.1***	ns	4,895.8	ns
Hispanic	934	434.7***	ns	4,910.9	ns

Notes:

1. This cause of death category includes ICD-9 codes 001-E999.

2. Racial groupings (White, Black, Asian & Pacific Islander, Native American) include persons of Hispanic ethnicity.

3. Age-adjusted Mortality Rates (AAMR) and Years of Potential Life Lost (YPLL) rates are per 100,000 based on race and ethnicity specific population estimates. Age-adjusted rates were calculated by the direct method using the 2000 U.S. standard population. Rates were not calculated for fewer than 15 events.

4. Statistical tests were conducted to evaluate differences in rates between race/ethnic groups. The white population serves as the reference group in each comparison (black vs. white, Asian & PI vs. white, Native American vs. white, Hispanic vs. white). Following are explanations of the notations:

    *** Significantly different than the respective white resident rate at $p < .001$.

5. Statistical tests were conducted to evaluate changes in rates over time. Comparisons of 1996-98 vs. 1989-91 rates are made within each race/ethnicity group. Following are explanations of the notations:

    ↓   1996-98 rate is significantly lower than the 1989-91 rate at $p < .05$.
    ↓↓↓ 1996-98 rate is significantly lower than the 1989-91 rate at $p < .001$.
    ns   Indicates the change from 1989-91 to 1996-98 is not statistically significant.

Premature mortality was significantly higher among black and significantly lower among Asian and Pacific Islander females compared with white females during the 1996-1998 period. There were no significant differences in premature mortality of Hispanic and Native American females compared with white females during this period. Age-adjusted death and premature mortality rates within these female subgroups did not change significantly between the two time periods (Table 2). A comprehensive discussion of racial and ethnic differences in mortality appears in the Summary Section of this report.

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SECTION IV. B.

CHRONIC DISEASE
MORTALITY

CONTENTS

Cardiovascular Disease
(ICD-9 codes 390-459)

Cardiovascular diseases account for more than 40% of all deaths in the United States and Connecticut. They encompass a broad range of heart and blood vessel diseases including ischemic heart disease, hypertension, cerebrovascular diseases (stroke), pulmonary circulatory diseases, and rheumatic heart disease. In the following section, we examine all heart disease, cerebrovascular disease, and hypertension-related disease deaths of Connecticut residents. Summary mortality tables for these as well as other cardiovascular disease categories (heart disease-related, coronary heart disease, congestive heart failure, atherosclerosis, atherosclerosis-related, aortic aneurysm, all cardiovascular diseases) can be found in Appendix VII B of this report.

Atherosclerosis, the disease process underlying most forms of cardiovascular (CVD) disease, is a condition in which the arterial walls become thick and rigid from the deposit of fat and cholesterol. Over many years, atherosclerosis leads to eventual narrowing of the arteries, decreased blood flow, and the build-up of arterial plaque, which can result in an embolism or blood clot.   Although CVD tends to manifest itself clinically in or after middle-age, the process of atherosclerosis begins in childhood (Newschaffer, Brownson, and Dusenbury 1998).

Morbidity and mortality rates from cardiovascular diseases in the United States have declined sharply since 1965. During this time, significant progress has been made in the prevention, diagnosis, and treatment of CVD. Research studies conducted over several decades have identified a complex set of modifiable risk factors that include high blood pressure, high cholesterol, cigarette smoking, physical inactivity, diabetes, and obesity (Newschaffer, Brownson, and Dusenbury 1998). The decline in CVD morbidity has been at least partially attributed to Americans' better understanding of these risk factors and subsequent changes in related behaviors (National Heart, Lung, and Blood Institute 1995).   Improvements in the diagnosis and medical treatment of cardiovascular diseases have also contributed to the decline in CVD mortality (National Heart, Lung and Blood Institute 1998; Braunwald 2001; Rosamond, Folsom, Chambless, et al 2001; Fonarow, Gawlinski, Moughrabi, et al. 2001).

The decrease in CVD mortality nationwide and, likewise, the improvements in CVD health have occurred mostly among better-educated, wealthier Americans. Consequently, the gap in CVD health between lower and higher socioeconomic status groups appears to have widened in the past three decades. Socioeconomic status (SES) is intertwined with the activities of daily life, including family and community life, work experiences, stressors, and access to and use of medical care, including preventive, diagnostic, and therapeutic health care. Such factors mediate the relationship of SES and cardiovascular health (National Heart, Lung, and Blood Institute 1995).   Social risk factors for cardiovascular diseases, including socioeconomic status, race/ethnicity, and gender, are discussed in the heart disease and cerebrovascular disease sections of this report.

CVD Risk Factor Surveillance

The modifiable risk factors for ischemic heart disease and stroke, the major forms of CVD, are the same. These risk factors, however, have differing magnitudes of effect on the two diseases. For example, stroke is strongly associated with high blood pressure and less influenced by cholesterol, whereas ischemic heart disease is most closely associated with high cholesterol (Newschaffer, Brownson, and Dusenbury 1998). Researchers have developed estimates of heart disease and stroke risk attributable to various behavioral factors, which we report in the respective sections. In the following paragraphs we report Connecticut resident risk factor prevalence data associated with CVD.

Risk factors, such as hypertension and smoking, may increase the risk of cardiovascular diseases independently or in combination with each other. It is quite common for people to have more than one risk factor, and combinations of risk factors greatly increase the probability of CVD (Yusuf, Giles, Croft, et al. 1998). An analysis of data from the National Health and Nutrition Examination Survey found that the relative risk for ischemic heart disease associated with having 1, 2, 3, and 4 risk factors (including current smoking, overweight, hypertension, high cholesterol, and diabetes) increased by 1.6, 2.2, 3.1, and 5.0, respectively. Similarly, relative risks for stroke associated with 1, 2, 3, and 4 risk factors were 1.4, 1.9, 2.3, and 4.3, respectively (Yusuf, Giles, Croft, et al. 1998). Screening programs targeting people at high risk for CVD can be most efficient when multiple risk factors are included (Chang, Hahn, Teutsch, et al. 2001). In 1998-1999, almost 28% of Connecticut adults (689,000) reported having 3 or more and 36% reported having 2 or more modifiable risk factors for CVD (Adams 2002).

Connecticut Behavioral Risk Factor Surveillance System (BRFSS) survey findings indicate that between 1990 and 1999, hypertension prevalence remained relatively stable with about 22% of Connecticut adults reporting high blood pressure (Adams 2002). About 25% of U.S. adults reported high blood pressure in 1999 (Ayala, Greenlund, Croft, et al 2002). While substantial progress in hypertension control was made in the 1970s and 1980s nationwide, awareness, treatment, and control of the disease appeared to plateau around 1990 (Cooper, Cutler, Desvigne-Nickens, et al. 2000).

About 22% of Connecticut adults are current cigarette smokers compared with 23% of adults nationwide. This percentage has remained relatively stable in both Connecticut and the United States since 1990 (Centers for Disease Control and Prevention 2001a; Adams 2002). Approximately 58,400 Connecticut middle and high school students currently smoke cigarettes, representing about 10% of all middle and 26% of all high school students (Lowery St. John and Jarvis 2001).

Approximately 27% of Connecticut adults tested have been told by a doctor that they have high cholesterol (1998-1999 period). There appeared to be a slight increase in prevalence of high cholesterol in Connecticut residents between 1994 and 1999 (Adams 2002). Nationwide, 30% of adults reported in 1999 that they had been told that they have high cholesterol (Centers for Disease Control and Prevention 2001a).

Between 1991 and 1998-1999, obesity rates (defined as a body mass index ≥ 30 kg/m^2) increased more than 33% in Connecticut. In 1998-1999, about 15% of Connecticut adults were estimated to be

obese (Adams 2002). From 1991 to 1998, obesity rates nationwide rose 50% from about 12% to 18% (Mokdad, Serdula, Dietz, et al. 1999). About 4% of Connecticut adults are estimated to have diabetes (Adams 2000), which is similar to the national prevalence estimate of almost 6% (Newschaffer, Brownson, and Dusenbury 1998; Centers for Disease Control and Prevention 2001a).

Approximately 27% of Connecticut and U.S. residents did not engage in leisure time physical activity in 1998 and this trend appears to have been stable since 1990. It is further estimated that nearly two million Connecticut adults do not get regular exercise (30 minutes of moderate intensity physical activity at least 5 days per week) as recently recommended by experts[1] (Adams, 2002).

Costs and Prevention

The total cost of all cardiovascular diseases in the United States was estimated at $351.8 billion in 2003. This estimate includes the direct costs of medical expenditures and indirect costs such as lost productivity due to morbidity and mortality. The total cost of heart disease is estimated at $229.9 billion, stroke is estimated at $51.2 billion, and hypertensive disease is estimated at $50.3 billion (American Heart Association 2003). It has been estimated that the overall cost of CVD in Connecticut is approximately $3.6 billion (Adams 2002).

National and international CVD prevention efforts have focused on reducing the prevalence of the major modifiable risk factors in communities. Early studies included the Stanford (U.S.A.) Three-Community Study (1972-1975) and the North Karelia (Finland) Project (1972-1982, and extended to the present). The National Heart, Lung, & Blood Institute (NHLBI) funded three important community-based CVD demonstration projects aimed at risk factor reduction using a multi-tiered approach at the individual, institutional / organizational, and the social environmental levels. The Stanford Five-City Project (1980-1986), the Minnesota Heart Health Project (1981-1988), and the Pawtucket Heart Health Program (1984-1991) used a variety of media, including radio, television, newspaper, community organization, and direct education, to examine CVD risk factors and mortality in treatment and reference cities. Although the treatment effects of the interventions were less than originally expected, these studies established the viability of community-based interventions for modifying CVD risk factors. They have been used as models for community-based interventions and have laid the groundwork for later CVD risk factor control programs (Schooler, Farquhar, Fortmann, et al. 1997).

Findings from the Stanford Five-City Project indicate that the morbidity and mortality event rate declined in both the treatment and control cities at the end of the 14-year study period. The study authors concluded that some other influences on all five cities (such as improved cardiac care technologies), rather than the intervention, account for the observed changes in morbidity and mortality (Fortmann and Varady 2000). The Minnesota Heart Health Program also examined morbidity and mortality outcomes among its participants and did not find clear evidence of an intervention effect (Luepker, Rastam, Hannan, et al. 1996). To date, the Pawtucket Heart Health Program has not reported morbidity and mortality outcomes relative to the study intervention.

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[1] In 2002, the Institute of Medicine issued a new report on healthy eating and exercise, which includes the recommendation that adults and children spend at least a total of one hour daily in moderately intense physical activity. This is double the daily goal set by the Surgeon General in 1996 (Institute of Medicine 2002).

Heart Disease
(ICD-9 codes 390-398, 402, 404-429)

Heart disease is the leading cause of death in Connecticut and the United States, accounting for 34% (97,092) of all deaths from 1989 to 1998. It is the second leading cause of premature mortality in Connecticut, with an estimated 381,784 years of potential life lost to age 75 during the ten-year period.

Heart disease mortality encompasses several subcategories with varying etiologies, including ischemic heart disease (57%); hypertensive heart and hypertensive heart and renal disease (3%); pulmonary circulatory diseases (1%); rheumatic fever and rheumatic heart disease (less than 1%); and "other forms of heart disease" (38%), which includes cardiac arrest (11%), congestive heart failure (6%), cardiomyopathy (4%), and aortic valve disease (2%) [Figure 2.1].

Ischemic heart disease (IHD), the leading type of heart disease death, describes any condition in which the heart muscle works inefficiently or is damaged because of a deficiency or absence of its blood supply. Impairment of the blood's circulation to the heart results most often from atherosclerotic coronary artery disease (CAD), the narrowing of the coronary arteries by atherosclerosis (U.S. Preventive Services Task Force 1996; Newschaffer, Brownson, and Dusenbury 1998). Acute myocardial infarction and coronary atherosclerosis are the most common forms of IHD mortality comprising 35% and 33%, respectively, of all IHD deaths to Connecticut residents in 1996-1998. Ischemic heart disease is also referred to as coronary heart disease (CHD), although CHD death classifications do include the additional subcategories of hypertensive heart disease and unspecified cardiovascular disease as a complication of heart disease (World Health Organization 1977; U.S. Department of Health and Human Services 1990).

Males in both Connecticut and the United States have significantly higher overall heart disease death and premature mortality rates than females. In the 1996-1998 period, Connecticut males had 1.5 times the rate of heart disease deaths ($p < .001$) and 2.3 times the premature mortality rate ($p < .001$) of females (Table 2.1).

1996-1998 Heart Disease Deaths, Connecticut Residents

- The leading cause of death for both males and females

- Second leading cause of premature mortality to age 75

- The leading cause of death for residents aged 75 and older

- Significant decrease in age-adjusted death and premature mortality rates for all residents since the 1989-1991 period

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Figure 2.1.
Heart Disease Deaths, Percent by Subtype
Connecticut Residents, 1996-1998
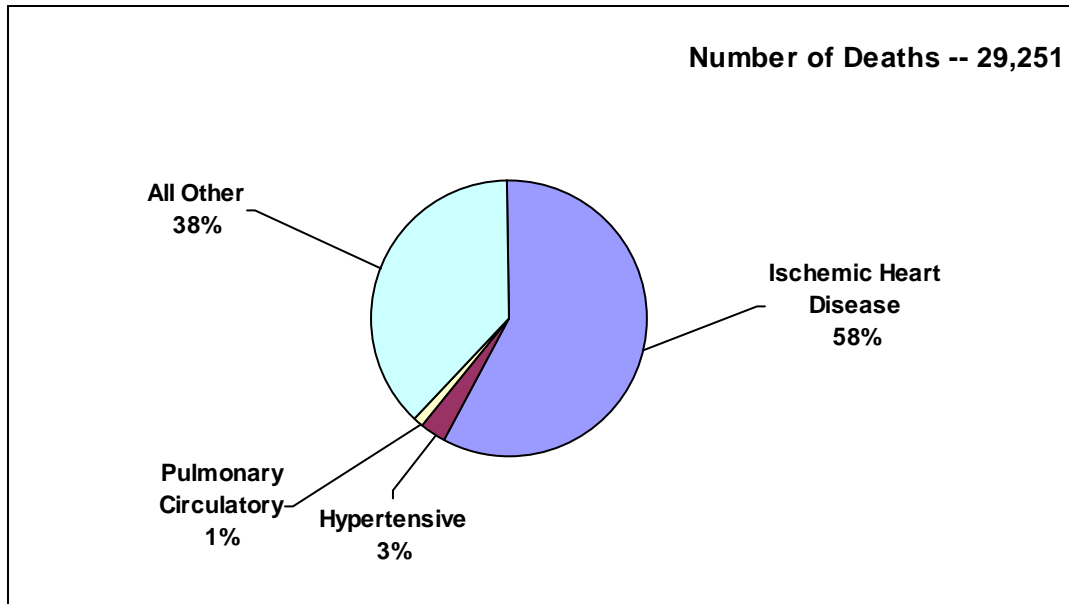
Among racial/ethnic and gender subgroups, white and black males had similar age-adjusted mortality rates from heart disease in the 1996-1998 period (Table 2.1). Although heart disease death rates of black males were not significantly different from those of white males, black males had premature mortality rates about 1.7 times higher than white males during this period (p < .001). White males had significantly higher heart disease death and premature mortality rates than Asian and Pacific Islander males and a significantly higher death rate than Hispanic males. Native American males' heart disease death and premature mortality rates were not significantly different from those of white males (Table 2.1).

Among females, blacks had the highest death and premature mortality rates from heart disease in the 1996-1998 period. Black females had 1.3 times the heart disease death rate (p < .001) and 2.8 times the premature mortality rate (p < .001) of white females. White females had significantly higher heart disease death and premature mortality rates than Asian and Pacific Islander females and a significantly higher heart disease death rate compared with Hispanic females. There were too few heart disease deaths of Native American females to calculate reliable rates (Table 2.1).

Logistic regression analyses of black-white and Hispanic-white female heart disease mortality by age group for the 1996-1998 period showed that the observed disparities differed by age group (p < .0014). For black compared with white females aged 0-64 and 70-74, the relative risk of death was consistent at 2.7 (p < .001), while the disparity for black compared with white females aged 65-69 and 75-79 lessened (RR ages 65-69= 1.5, p < .05; RR ages 75-79=1.4, p < .05). There were no significant differences in heart disease mortality between black and white females aged 80 and over. For Hispanic compared with white females aged 0 to 84, the relative risk of heart disease death was not significantly different; however, for females aged 85 and over, the difference was significant (RR= 0.4, p < .001) favoring Hispanic females.

Table 2.1. Heart Disease Deaths[1]
Connecticut Residents by Gender, Race and Ethnicity[2], 1996-98

Group	Number of Deaths	Age-Adjusted Mortality Rates[3] AAMR[4]	Age-Adjusted Mortality Rates[3] Change since 1989-91[5]	Age-Adjusted Premature Mortality Rates to Age 75[3] YPLL[4]	Age-Adjusted Premature Mortality Rates to Age 75[3] Change since 1989-91[5]
All Residents	29,251	265.4	↓↓↓	1,159.4	↓↓↓
All males	13,582	328.5	↓↓↓	1,647.7	↓↓↓
White	12,772	327.6	↓↓↓	1,576.2	↓↓
Black	748	356.5	↓	2,738.8***	↓
Asian PI	39	90.2***	ns	574.3***	ns
Native American	19	277.7	na	3,052.9	na
Hispanic	251	184.8***	ns	1,333.2	ns
All females	15,669	217.8	↓↓↓	707.0	ns
White	14,764	213.8	↓↓↓	620.9	ns
Black	864	282.5***	ns	1,723.8***	ns
Asian PI	33	68.8***	na	315.7**	na
Native American	4	—		—	
Hispanic	248	136.9***	ns	756.4	ns

Notes:

1. This cause of death category includes ICD-9 codes 390-398,402,404-429.

2. Racial groupings (White, Black, Asian & Pacific Islander, Native American) include persons of Hispanic ethnicity.

3. Age-adjusted Mortality Rates (AAMR) and Years of Potential Life Lost (YPLL) rates are per 100,000 based on race and ethnicity specific population estimates. Age-adjusted rates were calculated by the direct method using the 2000 U.S. standard population. Rates were not calculated for fewer than 15 events.

4. Statistical tests were conducted to evaluate differences in rates between race/ethnic groups. The white population serves as the reference group in each comparison (black vs. white, Asian & PI vs. white, Native American vs. white, Hispanic vs. white). Following are explanations of the notations:

   ** Significantly different from the respective white resident rate at $p < .01$.
   *** Significantly different from the respective white resident rate at $p < .001$.
   — Rate was not calculated due to small numbers.

5. Statistical tests were conducted to evaluate changes in rates over time. Comparisons of 1996-98 vs. 1989-91 rates are made within each race/ethnicity group. Following are explanations of the notations:

   ↓ 1996-98 rate is significantly lower than the 1989-91 rate at $p < .05$.
   ↓↓ 1996-98 rate is significantly lower than the 1989-91 rate at $p < .01$.
   ↓↓↓ 1996-98 rate is significantly lower than the 1989-91 rate at $p < .001$.
   ns Indicates the change from 1989-91 to 1996-98 is not statistically significant.
   na 1989-91 rate was not calculated due to small numbers and so no comparison with 1996-98 rate is available.

Among males nationwide, the highest age-adjusted rates of heart disease mortality occur among blacks, followed by whites, Native Americans, Hispanics, and Asian and Pacific Islanders. In 1995, black males had a heart disease death rate that was 29% higher than the rate for white males, and 90%, 97%, and 126% higher than the rates for Native American, Hispanic, and Asian and Pacific Islander males, respectively (Barnett, Casper, Halverson, et al. 2001). Among females nationwide, the highest age-adjusted rates of heart disease mortality are found among blacks, followed by whites, Native Americans, and Asian and Pacific Islanders. In 1995, black females had a heart disease death rate that was 1.4 times higher than the rate for white females, 2.1 times higher than the rates for Native American and Hispanic females, and 2.6 times higher than the rate for Asian and Pacific Islander females (Casper, Barnett, Halverson, et al. 2000).

Age-adjusted death rates due to heart disease decreased significantly for Connecticut residents from the 1989-1991 to the 1996-1998 period. Most of the decrease in heart disease mortality for Connecticut is due to the decline in ischemic heart disease. In 1989-1991, ischemic heart disease accounted for 61% of all heart disease deaths compared with 57% in 1996-1998. Significant declines in heart disease death and premature mortality occurred for both white and black males (Table 2.1). Heart disease mortality rates for males decreased by 2.5% per year from 1989 to 1998 (p < .001). Between 1989-1991 and 1996-1998, age-adjusted heart disease mortality rates for females decreased significantly, although the premature mortality rate did not change significantly. The decrease in mortality was statistically significant for white females only (Table 2.1). On average, heart disease mortality rates for females decreased by 1.2% per year from 1989 to 1998 (p<.001).

Nationwide, heart disease mortality declined through the 1970s and 1980s, but the rate of decline slowed during the 1990s (Casper, Barnett, Halverson, et al. 2000; Barnett, Casper, Halverson, et al. 2001). Declining mortality from ischemic heart disease accounted for most of the decline in heart disease mortality in the United States since 1979 (National Center for Health Statistics 1996). For the period 1991 to 1995, heart disease death rates dropped 1.9% per year for males and 1.3% for females (Casper, Barnett, Halverson, et al. 2000; Barnett, Casper, Halverson, et al. 2001). Among males, Hispanics and Native Americans experienced greater declines (2.3% and 2.6% per year, respectively) than black (1.7%) and white (1.9%) males. Asian and Pacific Islanders experienced the least decline of 1.1% per year (Barnett, Casper, Halverson, et al. 2001). Among females nationwide, Hispanics and Asian and Pacific Islanders experienced somewhat greater declines (1.5% per year each) than black and white females (1.2% decline each per year). Native American females experienced the least decline of 0.5% per year (Casper, Barnett, Halverson, et al. 2000).

Connecticut heart disease mortality rates for both males and females were significantly lower than comparable U.S. rates from 1989 to 1998 (Table 2.2 and Figure 2.2). There are no *Healthy People 2000* and *Healthy Connecticut* targets for heart disease mortality.[2]

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[2] There are *Healthy People 2000* and *Healthy Connecticut* targets for coronary heart disease (CHD) (ICD-9 codes 402, 410-414, 429.2). The 1998 Connecticut resident CHD AAMR (80.7 per 100,000 population, U.S. 1940 standard million population) was significantly lower than both the *Healthy People 2000* and *Healthy Connecticut* targets of 100,000 and 84.0 per 100,000 population, respectively (p < .05).

Age-specific death rates of Connecticut males and females for 1996-1998 are displayed in Figure 2.3. Age-adjusted heart disease mortality rates for males and females, contrasted with proportionally adjusted rates for all other causes of death, show a pattern of lower rates in the younger age groups, higher rates for males beginning in middle-age (ages 55-59) and extending through the oldest age groups, and higher rates for females in the oldest age groups (80 and older).

Heart disease mortality rates increase with increasing age, with the highest rates found in the 85 and older age group. Seventy percent of all heart disease deaths occurred among Connecticut residents aged 75 and older during the 1996-1998 period. Time trend analyses by age group indicate that the heart disease death rate for females aged 75-79 decreased significantly from 1989 to 1998 while it did not change significantly for other gender/age groups in the population.

**Table 2.2. Heart Disease Age-Adjusted Death Rates,
Comparison of CT with US - 1989 and 1998**

	1989	1998	1998 CT AAMR Comparison
CT AAMR*	139.6	113.3	
US AAMR*	157.5	126.7	CT rate < US rate

* age-adjusted mortality rates are per 100,000 population, U.S. 1940 standard million population.

Risk Factors

Increasing age is the key risk factor for heart disease. In 1996-1998, those 65 years of age and older accounted for 86% of all heart disease deaths among Connecticut residents. For men, major increases in heart disease mortality rates begin in the 35-to-39-year-old age group; for women, major increases in mortality begin at ages 55 to 59.

A family history of heart disease increases one's risk of developing the disease. A combination of inherited characteristics and behavioral patterns (similar dietary, smoking, and activity habits, for example) are thought to explain increased risk within families (Newschaffer, Brownson, and Dusenbury 1998).

Men have higher heart disease mortality rates than do women at all ages, except the youngest (0 to 9 years) age groups. As such, males are considered a high-risk group. Clinically significant coronary artery disease (CAD) is most common in men 40 and over (U.S. Preventive Services Task Force 1996). Heart disease is the leading cause of death among women outnumbering all cancer deaths, the second-leading cause of death among women, by fifty percent (1996-1998 period). CAD and heart disease morbidity in women increases sharply after menopause (U.S. Preventive Services Task Force 1996; Newschaffer, Brownson, and Dusenbury 1998).

Among racial/ethnic groups, black Americans are considered a high-risk group for heart disease with a tendency toward higher mortality rates than whites at all ages except the oldest age group (85 and older). The black-white disparity in age-adjusted heart disease mortality is largely accounted for by the discrepancy between black and white females, with black males tending to have similar or only slightly higher age-adjusted heart disease mortality rates than white males (Newschaffer, Brownson, and Dusenbury 1998).

Lower socioeconomic status (SES) is also considered a key risk factor for heart disease. Persons of lower SES have higher ischemic heart disease (IHD) morbidity and mortality than do middle- or upper-income persons. Over time, the greatest declines in IHD mortality among white Americans have occurred in the upper-income groups. Risk factors for IHD, such as smoking, hypertension, and obesity, are more prevalent in lower SES persons and may explain some of the observed disparity (National Heart, Lung, and Blood Institute 1995). Behavioral risk factors, however, account for only a small proportion of difference in heart disease mortality between upper and lower SES groups, and so other factors contributing to the SES – CVD relationship need to be examined (Lynch, Kaplan, Cohen, et al. 1996; Lantz, House, Lepkowski et al. 1998). Research suggests that neighborhood socioeconomic status may also have effects on individuals apart from their own socioeconomic status, and/or may interact with individual risk factors to increase or decrease individual risk (Diez-Roux, Nieto, Muntaner, et al. 1997; Newschaffer, Brownson, and Dusenbury 1998).

The contribution of SES to excess cardiovascular disease mortality involves several pathways including multiple risk factors, access to and use of risk factor screening, preventive health measures, and long-term management of the disease (National Heart, Lung, and Blood Institute 1995; Howard, Anderson, Russell, et al. 2000). Analysis of data from the National Longitudinal Mortality Study from 1979 to 1989 found that SES (as measured by family income and educational attainment) accounted for more than half of the excess IHD mortality observed for black compared with white women aged 35 to 54. Among men aged 55 to 74, whites were slightly more likely than blacks to die of IHD, and adjustment for SES dramatically increased this disparity, making the white excess mortality higher (Howard, Anderson, Russell, et al. 2000).

Figure 2.2.

Diseases of the Heart Deaths
Comparison of Connecticut and U.S. 1989-1998
Age-Adjusted Mortality Rates* by Sex

Symbols:
■ - U.S. rates for 1989-1998
✳ ● - Connecticut rates: an asterisk is used if the rate is significantly different than expected (p<.005), based on the log-linear trend.

* These rates are adjusted to the 1940 US standard million population.
* This classification includes deaths with ICD-9 codes:390-398,402,404-429

Figure 2.3.

Age-specific Rates for Diseases of the Heart Deaths
and for All Other Causes, 1996 - 1998

Major modifiable risk factors for ischemic heart disease include high blood pressure, elevated blood cholesterol, cigarette smoking, diabetes, obesity, insufficient physical activity, and environment tobacco smoke (Newschaffer, Brownson, and Dusenbury 1998; Centers for Disease Control and Prevention 1999a; Chang, Hahn, Teutsch et al. 2001) [Table 2.3].

Hypertension is an independent risk factor for morbidity and mortality from ischemic heart disease (IHD). People with elevated blood pressure ($\geq$ 140mm Hg systolic / 90 mm Hg diastolic) are 2 to 4 times more likely to develop IHD as are people with normal blood pressure.[3] IHD risk increases with increasing levels of systolic or diastolic blood pressure, and even persons with normal levels of blood pressure can decrease IHD risk by reducing blood pressure. Hypertensive drug therapy has demonstrated effectiveness in reducing risk of cardiovascular disease. Clinical studies of drug treatment for hypertension, however, have not demonstrated a reduction in IHD deaths, possibly because of unfavorable effects of hypertensive drug therapy on other risk factors (Newschaffer, Brownson, and Dusenbury 1998).

Cigarette smoking increases the risk of heart attack two-fold. Smokers have higher IHD mortality rates than non-smokers and their risk of death increases with greater number of cigarettes smoked. Research shows that people who stop smoking have a sizable reduction in IHD mortality

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[3] New federal guidelines classify normal blood pressure as below 120/80 mm Hg and readings from 120/80 mm Hg up to 140/90 mm Hg as prehypertensive. Studies show that heart disease risk begins to rise when blood pressure rises above 115/75 mm Hg (Chobanian, Bakris, Black et al. 2003).

(Newschaffer, Brownson, and Dusenbury 1998). Some research indicates that exposure to environmental tobacco smoke increases the risk of IHD (Garland, Barrett-Connor, Suarez, et al. 1985). A national study of non-smoking women showed that those with regular exposure to second-hand smoke at home were at increased risk of developing IHD compared with those who were not exposed at home (Kawachi, Colditz, Speizer, et al. 1997).

Blood cholesterol levels above 200 mg/dL confer higher risk in middle-aged adults for ischemic heart disease. Risk doubles at levels of 240 mg/dL and over (Newschaffer, Brownson, and Dusenbury 1998). High cholesterol (≥ 240 mg/dL) is a risk factor in middle-aged women, although women's risk is about half that of men's at the same levels of cholesterol (U.S. Preventive Services Task Force 2003). High-density lipoproteins (HDL) and low-density lipoproteins (LDL) move cholesterol in the blood system. High levels of LDL are responsible for the development of atherosclerosis, and later, IHD. An inverse relationship exists between HDL levels and IHD, with higher levels of HDL associated with lower risk for IHD (Newschaffer, Brownson, and Dusenbury 1998).

Diabetes mellitus is considered a major risk factor for ischemic heart disease, with diabetic persons two to four times more likely to develop IHD than the rest of the population. IHD is the most common cause of morbidity and mortality among persons with diabetes. Diabetic women are at higher risk for heart disease than are diabetic men partly because of greater obesity among women (Newschaffer, Brownson, and Dusenbury 1998).

Table 2.3. Modifiable Risk Factors for Ischemic Heart Disease

Factor	Magnitude of Association[1]	Estimated Range of Population Attributable Risk (%)
High blood pressure (≥ 140/90 mm Hg)	Moderate	20 – 29
Elevated cholesterol (≥ 200 mg/dL)	Moderate	39 – 47
Cigarette smoking	Moderate	17 – 25
Diabetes (fasting glucose ≥140 mg/dL)	Moderate	1 – 15
Overweight / Obesity (body mass index > 27.8 kg/m – men; body mass index >27.3 kg/m – women)	Weak	7 – 32
Physical inactivity	Weak	23 – 46
Environmental tobacco smoke exposure	Weak	8 – 23
Heavy alcohol consumption[2]	Possible	–
Elevated plasma homocysteine	Possible	–
Infectious agents	Possible	–
Psychological factors	Possible	–

Source*:* Adapted from Newschaffer, Brownson, and Dusenbury 1998.

1. *Moderate magnitude* indicates a relative risk of between 2 and 4 for those persons with the risk factor compared with those not having the risk factor. *Weak magnitude* indicates a relative risk of less than 2 for those persons with the risk factor compared with those not having the risk factor. *Possible association* indicates that some, but not definitive, evidence exists to support these as risk factors for ischemic heart disease.

2. Light alcohol consumption may reduce risk.

Obesity, defined as a body weight of 120% or greater than desirable weight for height, is another risk factor for IHD. Body mass index (BMI), the ratio of weight to height (kg/m^2), is a common measure of overweight. Persons with BMI greater than or equal to 30 are considered obese. Among persons under 50, those weighing 130% of their desired weight have double the risk of developing IHD.   There is evidence that abdominal fat (as opposed to lower body fat) appears to increase IHD risk across all levels of BMI. Obesity is also associated with other IHD risk factors such as hypertension, diabetes, higher levels of LDL, and lower levels of HDL (Newschaffer, Brownson, and Dusenbury 1998). The long-term effects of weight loss on health are somewhat unclear and appear to depend on several factors including health status, co-morbidities, and intentionality of weight loss (Williamson 1997). Observational studies suggest that intentional weight loss in overweight, middle-aged adults does not reduce the risk of CVD mortality, but may reduce the risk of mortality from diabetes (Williamson, Pamuk, Thun et al. 1999; Williamson, Pamuk, Thun et al. 1995).   Further research, particularly randomized controlled trials, may elucidate the relationship between intentional weight loss and CVD mortality in healthy populations (Williamson, Pamuk, Thun et al. 1999).

Lack of physical activity, another major risk factor (Powell, Thompson, Caspersen, et al 1987; Berlin and Colditz 1990; U.S. Department of Health and Human Services 1996), is associated with a 1.5- to 2.4-fold increased risk of IHD (American Heart Association 2001). Biological processes through which physical activity acts to prevent IHD include the decrease in body weight and blood pressure, improved insulin sensitivity, improved coronary artery blood flow, and increased high-density lipoprotein levels. Research suggests that even low levels of physical activity can have a substantial effect on heart disease mortality (Centers for Disease Control and Prevention 1993a).

Heavy alcohol consumption raises blood pressure levels and increases risk for IHD mortality, whereas moderate alcohol use is associated with lower IHD risk (Garg. Wagener, Madans 1993). The protective effect of alcohol appears to be mediated through increases in HDL cholesterol (Newschaffer, Brownson, and Dusenbury 1998).

The probability of developing IHD increases dramatically when more than one risk factor is present. At minimum, there is an additive effect for IHD when the risk factors of high blood pressure, cigarette smoking, and high cholesterol are present (Newschaffer, Brownson, and Dusenbury 1998). Each individual risk factor in isolation accounts for relatively little IHD mortality, and a large proportion of IHD mortality is attributable to a combination of individual risk factors. In a study of risk factors for IHD in the U.S. population from 1971 to 1992, the population attributable risk (PAR) of several factors combined was estimated to be 41.2% for white men (i.e., current smoking, diabetes, hypertension, and elevated serum cholesterol), 60.5% for white women (i.e., current smoking, diabetes, hypertension, physical inactivity, and nonuse of replacement hormones[4]), 49.2% for black men (i.e., current smoking, diabetes, hypertension, and elevated serum cholesterol), and 71.2% for black women (i.e. current smoking, diabetes, and hypertension). In general, the PAR of single risk factors alone tended to be small, with the exception of hypertension among black women. Hypertension alone accounted for 41% of IHD mortality among black women, compared

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[4] Recent randomized controlled studies indicate that hormone replacement therapy may have little or even a negative influence on IHD in women (Herrington, Reboussion, Brosnihan et al. 2000).

with 7.5% among white men, 6% among black men, and less than 1% among white women (Chang, Hahn, Teutsch, et al. 2001).

There is evidence that risk factors for cardiovascular disease (CVD) differ by both ethnicity and socioeconomic group. Findings from the National Health and Nutrition Examination Survey (1988-1994) indicate that among women of the same socioeconomic status, both black and Mexican Americans have a higher prevalence of CVD risk factors compared with white women (Winkleby, Kraemer, Ahn, et al. 1998). These findings suggest that prevention efforts such as screening and early detection should be targeted to both low SES and certain minority populations.

Prevention

Primary prevention of ischemic heart disease has emphasized changing one or more modifiable risk factors for the disease—hypertension, high blood cholesterol, tobacco use, diet, physical inactivity, and diabetes management. Multiple intervention strategies offer the most promise in reducing the prevalence of risk factors. Early and comprehensive education regarding the importance of behavioral lifestyle factors is a basis for prevention efforts. Other preventive efforts include policy-related or environmental measures such as provision of smoke-free environments, elimination of cigarette vending machines, enforcement of laws prohibiting cigarette sales to minors, availability of public spaces for recreation, improved food choices in schools and workplace, and improved food labeling in grocery stores (Newschaffer, Brownson, and Dusenbury 1998). Evidence suggests that the economic and social environment may also influence IHD disease risk (Diez-Roux, Nieto, Muntaner, et al. 1997); therefore, in addition to specific public health policies, broader economic and social development policies that address neighborhood conditions may be beneficial.

Screening measures can identify persons at risk for heart disease. Screening for high blood pressure and elevated cholesterol levels and assessment of tobacco use and physical activity level are appropriate for the general population (Newschaffer, Brownson, and Dusenbury 1998). The U.S. Preventive Services Task Force (USPSTF) screening and counseling recommendations for health practitioners regarding primary prevention of coronary artery disease are outlined in Table 2.4. USPSTF and the American College of Physicians do not recommend the routine use of resting electrocardiogram (EKG) in asymptomatic adults. The American Academy of Family Physicians (AAFP), however, recommends an EKG for sedentary men about to begin an exercise program and for men 40 and older with two or more risk factors for IHD. A task force of the American College of Cardiology and the American Heart Association recommends baseline EKG testing for all persons over 40 years of age and for persons undergoing exercise stress testing (U.S. Preventive Services Task Force 1996).

Two primary prevention strategies have been used to reduce cardiovascular disease morbidity and mortality —identification of high-risk individuals through mass screening and appropriate referral and population-based interventions that target the entire population through a variety of community-wide strategies emphasizing behavioral and policy change. Community participation in the planning and implementation of interventions is a key aspect of this approach. Successful strategies include mass media broadcasts, legislative initiatives (e.g. smoking restrictions in public

Table 2.4.   U.S. Preventive Services Task Force Recommendations for Primary Prevention of Coronary Disease for All Patients

Factor	Recommended Screening / Counseling
Hypertension	• Screening is recommended for all children and adults.
Tobacco use	• Cessation; prescription of nicotine patches or gum for selected patients.
High cholesterol (Lipid disorders)	• Routine screening for all men 35 and older and women 45 and older. • Routine screening of younger adults (men 20-35 and women 20-45) for lipid disorders if they have other risk factors for coronary heart disease. • Treat abnormal lipids in persons at increased risk of coronary heart disease.
Aspirin use	• Recommended that clinicians discuss aspirin chemoprevention with adults at increased risk for coronary heart disease. Discussion should address the potential benefits and harms of aspirin therapy.
Healthy diet	• There is insufficient evidence to recommend for or against routine behavioral counseling to promote a healthy diet in unselected patients in primary care settings. • Intensive behavioral dietary counseling for adults with hyperlipidemia and other known risk factors for cardiovascular and diet-related chronic disease.
Physical activity	• Incorporate regular physical activity into daily routine.

Source: U.S. Preventive Services Task Force 1996; 2002. http://www.ahcpr.gov/clinic/uspstfix.htm

places), and community events like health fairs. Health interventions target people at multiple levels—individual, organizational, and policy—using institutions such as schools, work sites, health care, and community sites (Newschaffer, Brownson, and Dusenbury 1998).

One example of the high-risk individual approach is the Multiple Risk Factor Intervention Trial (MR FIT) of 15,000 men with multiple risk factors for heart disease. MR FIT was a randomized, primary prevention trial from 1973 to 1982 that tested whether lowering elevated serum cholesterol and diastolic blood pressure and stopping smoking would reduce coronary heart disease mortality (Multiple Risk Factor Intervention Trial Research Group 1982). Results support a long-term, continuing mortality benefit from the program (Multiple Risk Factor Intervention Trial Research Group 1996).

Community-wide approaches include the North Karelia (Finland) Project (1972-1982 and extended to the present), the Stanford Three Community Study (1972-1975), the Minnesota Heart Health Program (1981-1988), and the Pawtucket Heart Health Program (1984-1991). The North Karelia Project demonstrated the effectiveness of smoking cessation programs, nutrition education, and interventions aimed at food producers and distributors and the media in reducing heart disease morbidity and mortality. The Stanford Study showed that use of the media and face-to-face intervention influenced behavior changes and that site-specific (e.g. school, work place) interventions

were particularly effective in changing behavior. The Minnesota and Pawtucket Heart Health Programs used mass media, community organization, and direct education interventions with success in some risk factor reduction. A review of specific components of these several studies indicates that they have provided useful information on effective mechanisms for population-based cardiovascular disease prevention (Schooler, Farquhar, Fortmann, et al. 1997). These interventions have not tended to show a strong overall effect at the community-level, at least partly because of an unanticipated trend toward reduction of cardiovascular disease risk factors in the general population that likely influenced the study comparison groups (Newschaffer, Brownson, and Dusenbury 1998).

Cerebrovascular Disease
(ICD-9 codes 430-438)

Cerebrovascular disease is the third leading cause of death in Connecticut and the U.S., accounting for 6.5% of all Connecticut resident deaths (18,205) from 1989 to 1998. Stroke is the most severe clinical manifestation of cerebrovascular disease, and in this report the two terms are used interchangeably. While the majority of stroke deaths (58%) in Connecticut are classified as "acute but ill-defined cerebrovascular disease," stroke deaths also include those due to cerebral thrombosis and cerebral embolism (referred to as ischemic stroke), intracerebral hemorrhage, subarachnoid hemorrhage, and late effects of cerebrovascular disease (World Health Organization 1977).

Stroke mortality in the United States has decreased by over 65% since 1950. This decline has occurred for both males and females across all race and ethnic groups. Between 1979 and 1995, stroke mortality declined by 20%. Beginning in the 1980s, the rate of decline slowed and then appeared to stabilize in the 1990s. Although reasons for the long-term decline in stroke mortality are not fully understood, possible explanatory factors include the widespread control of hypertension; a decline in cigarette smoking; changes in the American diet, such as decreased intake of saturated fat and decreases in blood cholesterol; and improved medical care and treatment of cerebrovascular disease leading to increased survival times and lower case-fatality rates (Centers for Disease Control and Prevention 1999a).

Age is considered the overarching risk factor for stroke. The effects of aging on the cardiovascular system and the progression of other risk factors over time greatly increase the risk of stroke (Goldstein, Adams, Becker, et al. 2001). Stroke risk doubles in every decade following age

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1996-1998 Cerebrovascular Disease Deaths, Connecticut Residents

- Third leading cause of death for all residents

- Seventh leading cause of premature mortality (to age 75)

- Third leading cause of death for residents aged 75 and older

- Significant decrease in black male mortality since the 1989-1991 period

- 1989-91 disparity between black and white male death rate eliminated

- Significantly higher death rates for black compared with white females

- Significantly higher premature mortality rates for black males and females compared with white males and females

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55. Ninety-one percent of all stroke deaths in Connecticut for 1996-1998 occurred among persons 65 and over.

Age-specific stroke death rates of Connecticut males and females for 1996-1998 are displayed in Figure 3.1. Stroke mortality rates for males and females, contrasted with proportionally adjusted rates for all other causes of death, show a pattern of lower rates in the younger age groups and higher rates beginning in the oldest age groups (ages 75-79 for males and 80-84 for females). Time trend analyses by age group indicate that the stroke death rate did not change significantly for any age group in the Connecticut population from 1989 to 1998.

Stroke incidence is more common in men than in women, and so males are considered at greater risk. Women, however, have higher stroke-related case-fatality rates than men and account for more than half of stroke mortality. In 1997, for example, women accounted for 61% of stroke fatalities in the U.S. (Goldstein, Adams, Becker, et al. 2001) and 63% in Connecticut.

Although males in both Connecticut and the United States have tended to have higher age-adjusted stroke mortality rates than females, in recent years the disparity has diminished. In the 1996-1998 period, there were no significant differences in age-adjusted death rates due to stroke between Connecticut males and females, although males had about 1.3 times the premature mortality rate ($p < .05$) of females (Table 3.1). For persons under age 65, the male stroke mortality rate tends to be greater than the female rate both in Connecticut and nationwide.

Figure 3.1.

Age-specific Rates for Cerebrovascular Disease Deaths and for All Other Causes, 1996 - 1998
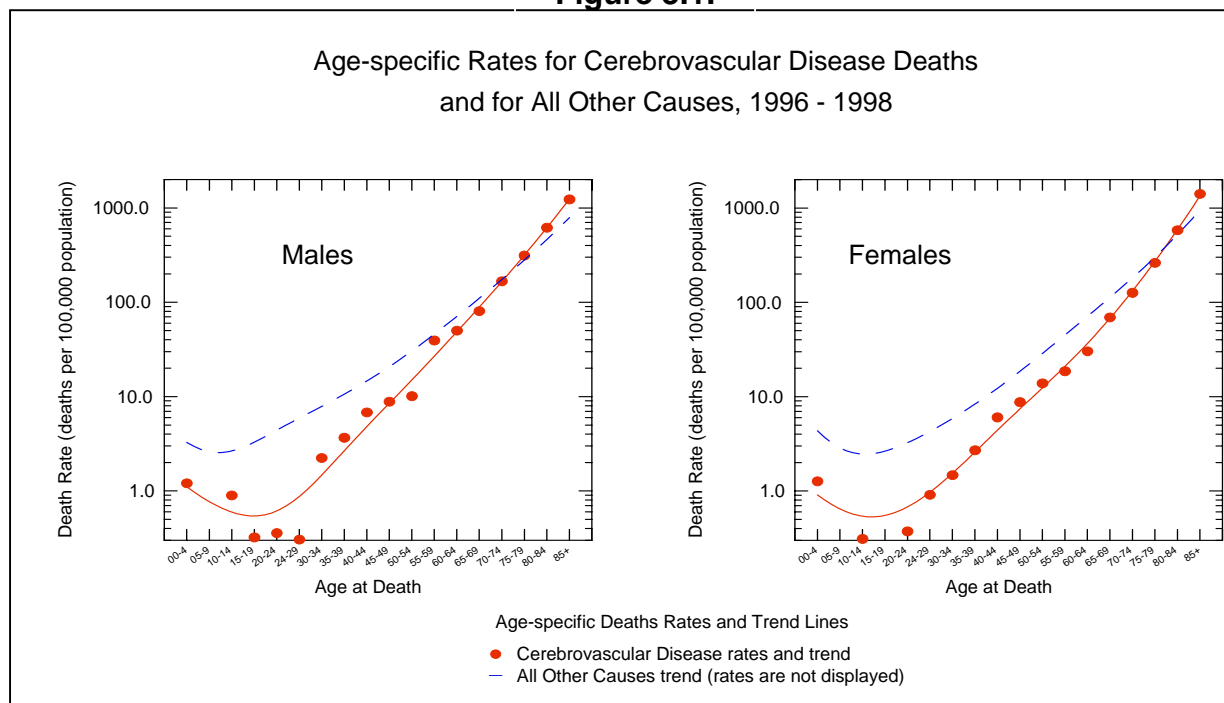
Among racial/ethnic and gender subgroups in Connecticut, black females had the highest age-adjusted stroke death rate and black males had the highest rate of premature mortality due to stroke in the 1996-1998 period. Although age-adjusted stroke death rates of black and Hispanic males were not significantly different from those of white males, black and Hispanic males had premature mortality rates about 2.5 times ($p < .001$) and 1.8 times higher ($p < .05$), respectively, than white males during this period. There were too few stroke deaths among Asian and Pacific Islander males and Native American males to report reliable rates (Table 3.1). There were too few black and Hispanic stroke deaths within specific five-year age groups to reliably identify black-white and Hispanic-white male mortality disparities by age group.[5]

Black females had 1.3 times the death rate ($p < .05$) and 2.1 times the premature mortality rate ($p < .01$) of white females during the 1996-1998 period. Hispanic females had a significantly lower rate of stroke death compared with white females, while there were no significant differences in stroke mortality between white and Asian and Pacific Islander females. There were too few stroke deaths among Native American females to report reliable rates (Table 3.1).

The black-white female mortality disparity varies by age group ($p < .0014$) with larger disparities in younger age groups (for all ages under 85, the black-white relative risk was about 1.7, while the black-white relative risk for ages 85 and over was 0.7). Logistic regression analyses indicate that there is not consistency in the Hispanic-white female relative risk (HW RR=0.7, $p < .01$) across all age groups; however, there were too few Hispanic deaths within specific five-year age groups in 1996-1998 to report reliable rates.

Although the age-adjusted death and premature mortality rates due to stroke did not appear to change significantly for Connecticut male residents from the 1989-1991 to 1996-1998 period (Table 3.1), an examination of annual change from 1989 to 1998 shows that male stroke mortality rates decreased by 1.2% per year from 1989 to 1998 ($p \leq .002$). Among racial and ethnic subgroups, stroke mortality rates among black males decreased significantly by about 40% (from 82.9 to 50.1 per 100,000, $p < .05$) between 1989-1991 and 1996-1998. Female death and premature mortality rates from stroke did not change significantly during this period (Table 3.1).

Connecticut male and female age-adjusted stroke mortality rates were significantly lower than the comparable U.S. rates from 1989 to 1998 (Figure 3.2). In 1998, the Connecticut resident stroke mortality rate was lower than the *Healthy People 2000* target but higher than the *Healthy Connecticut* target (Table 3.2).

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[5] Logistic regression analyses of male stroke mortality by age group indicate that the black-white disparity is not consistent across all age groups. While there were too few black male deaths within certain five-year age groups in 1996-1998 to report reliable rates, there was a clear trend toward a black-white disparity within ages 40-74. The Hispanic-white stroke mortality rate does not appear to be consistent across 5-year age groups; however, there were too few Hispanic male deaths within certain five-year age groups in 1996-1998 to report reliable rates.

Table 3.1. Cerebrovascular Disease Deaths[1]
Connecticut Residents by Gender, Race and Ethnicity[2], 1996-98

Group	Number of Deaths	Age-Adjusted Mortality Rates[3]		Age-Adjusted Premature Mortality Rates to Age 75[3]	
		AAMR[4]	Change since 1989-91[5]	YPLL[4]	Change since 1989-91[5]
All Residents	5,786	51.9	ns	163.8	ns
All males	2,132	53.0	ns	184.3	ns
White	2,012	52.9	ns	166.1	ns
Black	105	50.1	↓	422.2***	ns
Asian PI	13	—		—	
Native American	2	—		—	
Hispanic	55	39.7	ns	305.3*	ns
All females	3,654	50.3	ns	144.8	ns
White	3,451	49.5	ns	133.9	ns
Black	182	62.1*	ns	282.0**	ns
Asian PI	17	42.2	na	103.6	na
Native American	3	—		—	
Hispanic	59	32.9**	ns	183.1	ns

Notes:

1. This cause of death category includes ICD-9 codes 430-438. *Healthy People 2000* refers to these ICD-9 identifying codes as "stroke."

2. Racial groupings (White, Black, Asian & Pacific Islander, Native American) include persons of Hispanic ethnicity.

3. Age-adjusted Mortality Rates (AAMR) and Years of Potential Life Lost (YPLL) rates are per 100,000 based on race and ethnicity specific population estimates. Age-adjusted rates were calculated by the direct method using the 2000 U.S. standard population. Rates were not calculated for fewer than 15 events.

4. Statistical tests were conducted to evaluate differences in rates between race/ethnic groups. The white population serves as the reference group in each comparison (black vs. white, Asian & PI vs. white, Native American vs. white, Hispanic vs. white). Following are explanations of the notations:

> *   Significantly different from the respective white resident rate at $p < .05$.
> **   Significantly different from the respective white resident rate at $p < .01$.
> *** Significantly different than the respective white resident rate at $p < .001$.
> —   Rate was not calculated due to small numbers.

5. Statistical tests were conducted to evaluate changes in rates over time. Comparisons of 1996-98 vs. 1989-91 rates are made within each race/ethnicity group. Following are explanations of the notations:

> ↓   1996-98 rate is significantly lower than the 1989-91 rate at $p < .05$.
> ns   Indicates the change from 1989-91 to 1996-98 is not statistically significant.
> na   1989-91 rate was not calculated due to small numbers and so no comparison with 1996-98 rate is available.

Nationwide, black Americans have age-adjusted stroke mortality rates that are about 80% higher than those of whites. The black-white disparity is greatest for men under 65 and women under 45 years of age with black mortality rates that are over three times that of whites (Newschaffer, Brownson, and Dusenbury, 1998). Black Americans have both higher stroke incidence and mortality, which may be accounted for by several factors, including a higher prevalence of hypertension, obesity, and diabetes mellitus, and lower socioeconomic status (Howard, Russell, Anderson et al. 1995; Gillum 1999; Goldstein, Adams, Becker, et al. 2001). Other evidence suggests that risk factor prevalence for stroke may differ between black men and women with men reporting higher drinking and smoking rates and women reporting higher rates of hypertension, diabetes, and no leisure exercise (Worrall, Johnston, Kongable, et al. 2001).

Figure 3.2.

Cerebrovascular Disease Deaths
Comparison of Connecticut and U.S. 1989-1998
Age-Adjusted Mortality Rates* by Sex
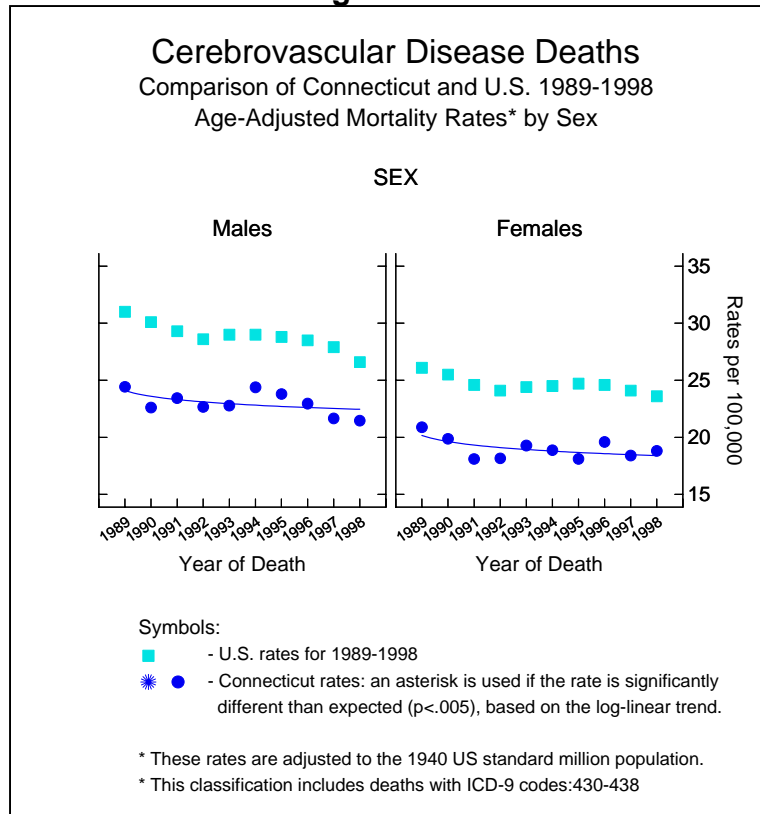
Table 3.2. Cerebrovascular Disease Age-Adjusted Death Rates, Comparison of CT with US - 1989 and 1998			
	<u>1989</u>	<u>1998</u>	<u>1998 CT AAMR Comparison</u>
CT AAMR*	22.4	20.1	
US AAMR*	28.1	25.1	CT rate < US rate
Healthy People 2000*	20.0	20.0	achieved *HP* target
Healthy CT 2000*	16.8	16.8	CT rate > *Healthy CT* target

* age-adjusted mortality rates for cancer are per 100,000 population, U.S. 1940 standard million population.

Among Hispanics, stroke mortality rates are about 10% lower among males and 5% lower among females compared with white Americans. Asian and Pacific Islander and Native Americans tend to have comparable or slightly lower rates than their white counterparts (Newschaffer, Brownson, and Dusenbury, 1998).

Risk Factors

Family (maternal and paternal) history of stroke appears to be associated with increased risk. Possible mechanisms for increased stroke risk include genetic inheritance of risk factors; familial cultural, environmental and lifestyle factors; and the interaction of genetic and environmental factors (Goldstein, Adams, Becker, et al. 2001).

Low-socioeconomic status (SES) has also been identified as an important risk factor for stroke and other cardiovascular diseases (National Heart, Lung and Blood Institute 1994; Gillum 1999; Centers for Disease Control and Prevention 1999a; Howard, Anderson, Russell et al. 2000; Hart, Hole, and Davey Smith 2000; Howard, Howard, and Reasons for Geographic And Racial Differences in Stroke (REGARDS) Investigators 2001). SES differentials may be largely explained by risk factors that are influenced by exposures over the life course, such as high blood pressure and smoking (Hart, Hole, and Davey Smith 2000). Analysis of data from the National Longitudinal Mortality Study showed that low-socioeconomic status accounted for much of the excess stroke mortality risk for black males but not females compared with their white counterparts (Howard, Russell, Anderson et al. 1995; Howard, Anderson, Russell et al. 2000). Persons of lower SES are more likely to have risk factors for stroke, including hypertension, overweight, excessive alcohol consumption, and cigarette smoking. It is also likely that persons of lower SES have less access to, and/or less effectively use preventive health services that are important to the early detection and treatment of hypertension (Kunst, del Rios, Groenhof, et el. 1998). In addition to such individual-level factors, low-income neighborhood or community environments may contribute to increased stroke risk and help to produce poorer stroke outcomes (Boden-Albala and Sacco 2002).

Modifiable risk factors for stroke include hypertension, cigarette smoking, obesity, total cholesterol, physical inactivity, and alcohol consumption (Table 3.3). Hypertension, the major risk factor, is related to all types of stroke. Both isolated systolic hypertension and diastolic blood pressure elevations increase the risk of stroke.

More than 30 years of research evidence has demonstrated that control of high blood pressure decreases the risk of stroke. Population-based studies indicate that small decreases in elevated diastolic blood pressure are associated with 40% to 45% reductions in fatal and nonfatal stroke incidence rates. Research evidence also suggests that among persons with isolated systolic hypertension, reduction in mean systolic pressure is associated with a decrease in stroke incidence (Newschaffer, Brownson, and Dusenbury 1998). Stroke mortality attributable to hypertension is estimated at 26%, although risk varies greatly by age group with risk ranging from 40% for persons aged 50, 20% for persons aged 80 years, and 0% for those over 80 (Newschaffer, Brownson, and Dusenbury 1998; Goldstein, Adams, Becker, et al. 2001).

Both β-blocker therapy and high-dose diuretics were shown to be effective in preventing stroke in a meta-analysis of 18 long-term randomized trials. Clinical trials of stroke in the elderly have also

demonstrated the importance of controlling isolated systolic hypertension to prevent stroke. The Systolic Hypertension in the Elderly Program trial showed a 36% reduction in total stroke incidence with the antihypertensive treatments chlorthalidone or atenolol (Goldstein, Adams, Becker, et al. 2001).

Although hypertension detection, education, and treatment efforts have been widespread since the 1960s, a large proportion of the U.S. population still has undiagnosed or inadequately treated hypertension. This is particularly true for higher-risk populations, especially black Americans (Burt, Cutler, Higgins, et al. 1995; Friday 1999). Hypertension prevalence is estimated to be about 26% in adult males and 22% in adult females in the U.S. It is also estimated that only about 1 out of 4 hypertensive persons are both treated and under control (defined as systolic blood pressure < 140 mmHg and diastolic blood pressure < 90 mmHg) (Cooper, Cutler, Desvigne-Nickens, et al. 2000). While substantial progress in hypertension control was made in the 1970s and 1980s nationwide, awareness, treatment, and control of the disease appeared to plateau around 1990 (Cooper, Cutler, Desvigne-Nickens, et al. 2000). National survey data from 1999-2000 indicate that the prevalence of hypertension is increasing in the United States, especially among women, black Americans, and older persons (Hajjar and Kotchen 2003).

Black Americans have some of the highest rates of hypertension worldwide. They tend to develop high blood pressure at younger ages, and their average pressures are much higher than whites. Among adults aged 20 and older, the prevalence of high blood pressure is 36.7% for black

Table 3.3. Modifiable Risk Factors for Stroke

Factor	Magnitude of Association[1]	Estimated Range of Population Attributable Risk (%)
High blood pressure (systolic $\geq$ 140 mm Hg)	Strong	$20 - 50^2$
Cigarette smoking	Moderate	$11 - 25^2$
Diabetes	Moderate	$14 - 58^3$
Elevated cholesterol ($\geq$ 220 mg/dL)	Moderate	$0 - 20^2$
Obesity (body mass index >27.8 kg/m – men body mass index >27.3 kg/m – women)	Moderate	$12 - 20^3$
Physical inactivity	Possible	30^3
Heavy alcohol use ($\geq$ 5 drinks per day)	Possible	$1.2 - 3.0^3$
Very low cholesterol (<160 mg/dl)	Possible	–
Drug abuse	Possible	–

Source*:* Adapted from Newschaffer, Brownson, and Dusenbury 1998.

1. *Strong magnitude* indicates a relative risk of greater than 4 for those persons with the risk factor compared with those not having the risk factor. *Moderate magnitude* indicates a relative risk of between 2 and 4 for those persons with the risk factor compared with those not having the risk factor. *Weak magnitude* indicates a relative risk of less than 2 for those persons with the risk factor compared with those not having the risk factor. *Possible association* indicates that some, but not definitive, evidence exists to support these as risk factors for stroke.

2. Newschaffer, Brownson, and Dusenbury 1998.

3. Goldstein, Adams, Becker, et al. 2001.

males compared with 25.2% for white males and 36.6% for black females compared with 20.5% for white females (American Heart Association 2002). Some researchers hypothesize that social stress, especially perceived racism, may at least partially explain the elevated rates of hypertension among African Americans (Willams and Neighbors 2001; Bondolo, Rieppi, Kelly et al. 2003). Laboratory evidence suggests that racial stressors are linked to increased cardiovascular reactivity, which is associated with higher cardiovascular risk, but the few population-based studies of racial discrimination and hypertension have not yielded consistent findings. Well-designed population-based studies are needed to further evaluate this theory (Willams and Neighbors 2001; Bondolo, Rieppi, Kelly et al. 2003).

Smoking is another major risk factor for stroke with current smokers having more than twice the risk of stroke as never smokers. The risk associated with smoking differs by age group with smokers under age 55 at greatest risk and those older than 75 with little or no risk compared with non-smokers (Newschaffer, Brownson, and Dusenbury 1998). Smoking causes reduced blood vessel elasticity by increasing arterial wall stiffness. Approximately 18% of strokes are attributable to current cigarette smoking (Goldstein, Adams, Becker, et al. 2001).

The relationship between stroke risk and serum cholesterol levels is not fully understood; however, studies have found that stroke risk can be reduced with cholesterol-lowering medication. Clinical trials have shown that β-hydroxy-β-methylglutaryl-CoA reductase inhibitors, or "statins," can reduce low-density lipoprotein (LDL) levels. These agents have consistently been shown to reduce stroke risk among persons with mild to borderline elevations in cholesterol levels and those with coronary artery disease and elevated cholesterol levels (Goldstein, Adams, Becker, et al. 2001).

National prospective data suggest that fruit and vegetable consumption three or more times per day is associated with 27% lower stroke incidence rate and 42% lower stroke mortality (Bazzano, He, Ogden, et al. 2002). Citrus fruits and cruciferous and green leafy vegetables appear to be the most beneficial foods with greatest effects at five servings per day. Potassium, flavonoids, cereal fiber, and folate appear to be the micronutrients responsible for the protective effects of fruits and vegetables (Renaud 2001).

Obesity (body mass index [BMI] of $\geq$ 30 Kg/m^2) is associated with an increased risk of ischemic stroke ranging from 1.7 to 2.4 for women at increasing levels of BMI. Among men, studies indicate that abdominal obesity, rather than BMI, is closely related to stroke risk. One study found a two-fold risk of stroke for men in the highest quintiles of waist-hip ratio (Goldstein, Adams, Becker, et al. 2001).

Physical activity appears to be beneficial in reducing the risk of stroke. Its protective effects may be partly mediated through the control of known risk factors such as hypertension, diabetes, cardiovascular disease, and body weight. Other relevant biological processes associated with increased physical activity include reductions in plasma fibrinogen and platelet activity, and elevations in plasma tissue plasminogen activator activity and HDL concentrations (Goldstein, Adams, Becker, et al. 2001). Dose-response relationships of physical activity and stroke incidence are not clearly established; however, studies indicate that moderate physical activity, such as walking, significantly reduces the risk of stroke (Wannamethee and Shaper 1992; Kiely, Wolf, Cupples, et al. 1994).

Diabetes has been shown to have an independent effect on ischemic stroke with an estimated relative risk of stroke ranging from 1.8 to 6 times for persons with diabetes. An estimated 40% to 60% of persons with type 2 diabetes have high blood pressure. The co-occurrence of hypertension and hyperglycemia is thought to increase the frequency of stroke and other diabetic complications. Recent research indicates that hypertension control and treatment of high-risk diabetic patients with the ACE inhibitor ramipril may prevent stroke in persons with diabetes. (Goldstein, Adams, Becker, et al. 2001).

The relationship between alcohol consumption and ischemic stroke is not entirely clear. Stroke risk appears to increase among heavy- or binge-drinkers. Several studies have shown no negative effect after controlling for other risk factors and a protective effect for moderate alcohol consumption (Goldstein, Adams, Becker, et al. 2001; Renaud 2001). While data on the relationship between drug use and stroke are limited, some studies have found an increased risk of both ischemic and hemorrhagic stroke from drug abuse (Goldstein, Adams, Becker, et al. 2001).

Although substantial progress has been achieved in the treatment and control of hypertension, the recent increasing prevalence of heart disease, diabetes, and obesity in the United States has increased the likelihood for stroke, particularly among black Americans (Centers for Disease Control and Prevention 2000a). Recent national data suggest that stroke-related mortality has plateaued and that risk factor prevalence appears to be increasing, two facts that would indicate stroke and other cardiovascular diseases may increase in the near future (Goldstein, Adams, Becker, et al. 2001).

Prevention

Primary prevention is the best approach for reducing the burden of stroke. High-risk profile persons can be identified and targeted for intervention. American Stroke Association (ASA) consensus guidelines for the primary prevention of stroke are displayed in a modified format in Table 3.4. ASA provides detailed goals and recommendations for managing other risk factors for stroke.

Stroke mortality can be reduced or delayed by risk factor prevention and by removing the barriers to treatment. Public education regarding the early warning signs of stroke, especially among those considered at high risk, may increase early detection and timely treatment for stroke (Ayala, Croft, Greenlund, et al. 2002).

Population-based approaches to reduce cardiovascular risk factors target the entire population through community-wide strategies emphasizing policy and behavioral change. Studies such as the Stanford Three Community Study, the Minnesota Heart Health Program, and the Pawtucket Heart Health Program have provided effective models for population-based cardiovascular disease prevention (Schooler, Farquhar, Fortmann, et al. 1997). In general, these interventions have not been shown to have large effects on cardiovascular disease risk at the community level, partly because of a downward trend in the prevalence of cardiovascular disease risk factors in the general population (Susser 1995). The intervention models, however, have been successfully replicated in communities throughout the United States.

In 1972, the National Heart, Lung, and Blood Institute (NHLBI) began the National High Blood Pressure Education Program (NHBEP), which has been active in health education with support from

community coalitions, governmental and non-governmental agencies, and professional associations. While this program has not been evaluated, it is thought that this longtime effort has contributed to increased control of hypertension in the past 30 years (Schooler, Farquhar, Fortmenn, et al 1997)   In 1991 and 1994, NHLBI funded community-based stroke prevention projects, known as the Stroke Belt Initiative, through state health departments, in 11 southern states known to be at high risk for

Table 3.4. ASA Consensus Statement for Primary Prevention of Stroke

Factor	Goal
Hypertension	Systolic BP < 140 mm Hg; Diastolic BP < 90 mm Hg
Cigarette smoking	Cessation
Diabetes	Improved glucose control; Hypertension treatment
Poor nutrition	A diet with ≥ 5 servings of fruit and vegetables daily
Physical inactivity	≥ 30 min of moderate-intensity activity daily
Alcohol use	Moderation (≤ 2 drinks/day for men and 1 drink/day for women)
Drug abuse	Cessation

Source*:* Adapted from Goldstein, Adams, Becker, et al. 2001.

stroke. Projects lasted 2 to 3 years and included community education and interventions; public education using mass media; church-based risk factor interventions; and health department clinic and outreach services. These projects demonstrated state health departments' capacity to plan and carry out community-based intervention efforts to prevent risk factors for cardiovascular diseases (National Heart, Lung, and Blood Institute 1996). Long-term effects of the interventions have not been assessed.

Research has documented the effect of social conditions on stroke mortality, suggesting that the role of community factors like poor neighborhoods, social isolation, and social stressors should be examined more closely (Kapral, Wang, Mamdani, et al. 2001). It is possible that, in addition to specific public health interventions, social and economic development programs aimed at low-income neighborhoods may be beneficial in reducing socioeconomic differences in stroke risk and mortality.

Hypertension-Related
(ICD-9 codes 401, 403)

Hypertension-related mortality includes all deaths for which hypertension is listed as an underlying and / or contributing cause. An examination of hypertension-related mortality can indicate the extent to which hypertension as a contributing factor to cardiovascular disease affects age, gender, and racial/ethnic groups differentially. Hypertensive disease itself is not a distinct underlying (primary) cause of death, and its selection as the underlying cause of death may indicate a lack of accurate diagnostic information at death (National Heart, Lung, and Blood Institute 2000).

For the 1996-1998 period, black male and female residents of Connecticut had the highest age-adjusted mortality rates due to hypertension-related causes followed by white, and then Hispanic males and females (Table 4.1). Black males and females had approximately twice the risk of death from hypertension-related causes compared with white males and females ($p < .001$ for both comparisons). Hispanic males had a 30% lesser risk and Hispanic females had a 28% lesser risk compared with their white counterparts ($p < .05$ for both comparisons). There were too few hypertension-related deaths among Asian and Pacific Islanders and Native Americans to calculate reliable rates. Age-adjusted premature mortality rates due to hypertension-related causes were also highest among black males and females with black males having 3.6 times and black females having 5.3 times the premature mortality rate of white males and females, respectively. Premature mortality rates of Hispanic males and females were not significantly different from those of white males and females.

Logistic regression analyses of the black-white hypertension-related mortality disparity by gender and age group indicate that significant differences exist across age groups ($p < .0014$) for both males and females. For males aged 0-54 and 65 and older the black-white (BW) disparity was consistent, with a relative risk (RR) of 1.9 ($p < .001$), whereas the disparity was significantly greater for males aged 55-59 (BW RR=5.1, $p < .001$) and males aged 60-64 (BW RR=3.9, $p < .001$). For females aged 0-74 the black-white disparity was consistent, with a relative risk of 4.2 ($p < .001$). The black-white female disparity lessened with increasing age, with blacks aged 75-79 experiencing a

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1996-1998 Hypertension-Related Deaths, Connecticut Residents

- Black males and females have the highest age-adjusted mortality rates for hypertension of all racial/ethnic subgroups

- Hypertension-related mortality increased significantly since the 1989-91 period

- Hypertension-related death and premature mortality rates for black males and females were significantly higher than comparable rates for white males and females

Table 4.1. Hypertension-Related Deaths[1]
Connecticut Residents by Gender, Race and Ethnicity[2], 1996-98

Group	Number of Deaths	Age-Adjusted Mortality Rates[3]		Age-Adjusted Premature Mortality Rates to Age 75[3]	
		AAMR[4]	Change since 1989-91[5]	YPLL[4]	Change since 1989-91[5]
All Residents	6,314	57.4	↑↑↑	226.1	ns
All males	2,525	60.2	↑↑↑	287.1	ns
White	2,269	57.4	↑↑↑	246.5	ns
Black	243	113.1***	ns	877.5***	ns
Asian PI	11	—		—	
Native American	2	—		—	
Hispanic	51	40.8*	ns	241.2	ns
All females	3,789	53.7	↑↑↑	169.9	ns
White	3,442	50.5	↑↑	127.2	ns
Black	331	106.0***	ns	679.6***	ns
Asian PI	13	—		—	
Native American	3	—		—	
Hispanic	62	36.5*	ns	142.5	ns

Notes:

1. This cause of death category includes ICD-9 codes 401,403. "Hypertension-related" deaths include those for which hypertension is the underlying and/or a contributing cause listed on the death certificate.

2. Racial groupings (White, Black, Asian & Pacific Islander, Native American) include persons of Hispanic ethnicity.

3. Age-adjusted Mortality Rates (AAMR) and Years of Potential Life Lost (YPLL) rates are per 100,000 based on race and ethnicity specific population estimates. Age-adjusted rates were calculated by the direct method using the 2000 U.S. standard population. Rates were not calculated for fewer than 15 events.

4. Statistical tests were conducted to evaluate differences in rates between race/ethnic groups. The white population serves as the reference group in each comparison (black vs. white, Asian & PI vs. white, Native American vs. white, Hispanic vs. white). Following are explanations of the notations:

    *   Significantly different from the respective white resident rate at $p < .05$.
    *** Significantly different from the respective white resident rate at $p < .001$.
    —   Rate was not calculated due to small numbers.

5. Statistical tests were conducted to evaluate changes in rates over time. Comparisons of 1996-98 vs. 1989-91 rates are made within each race/ethnicity group. Following are explanations of the notations:

    ↑↑ 1996-98 rate is significantly higher than the 1989-91 rate at $p < .01$.
    ↑↑↑ 1996-98 rate is significantly higher than the 1989-91 rate at $p < .001$.

    ns   Indicates the change from 1989-91 to 1996-98 is not statistically significant.

relative risk of 2.0 (p < .005), and no significant differences in mortality between black and white women aged 80 and over. There were insufficient numbers of hypertension-related deaths among Hispanic residents within various age groups to detect any age group differences in the Hispanic-white mortality disparity.

Males had significantly higher age-adjusted rates of hypertension-related deaths compared with females (RR=1.1, p < .01) [Table 4.1]. Higher male rates appeared to be consistent across all age groups for which rates could be calculated except the 85 and older age group in which females had 1.3 times the hypertension-related mortality rate of males (p < .001).

There appeared to be a significant increase in age-adjusted mortality rates from hypertension-related causes from the 1989-1991 to 1996-1998 period for Connecticut residents (Table 4.1). National data show a similar increasing trend (personal communication with National Center for Health Statistics, July, 2002). This apparent trend is not easily interpretable because hypertension prevalence rates both in Connecticut and nationwide appeared to be relatively stable through the 1990s. It is possible that this increase may be an artifact of improved reporting of hypertension as a cause of death on the death certificate.

Age-specific hypertension-related death rates of Connecticut males and females (1996-1998 period) are displayed in Figure 4.1. Among males, hypertension-related mortality rates, contrasted

Figure 4.1.
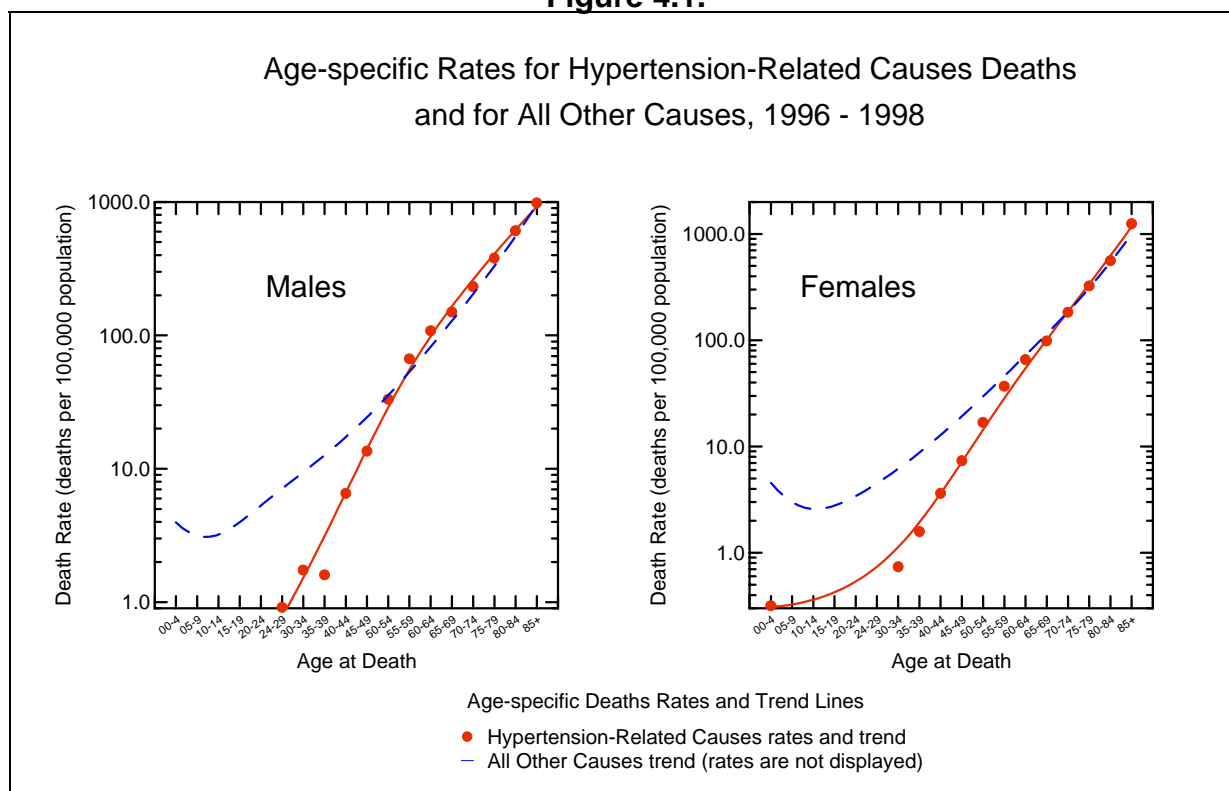
Age-specific Rates for Hypertension-Related Causes Deaths and for All Other Causes, 1996 - 1998

Age-specific Deaths Rates and Trend Lines
● Hypertension-Related Causes rates and trend
– All Other Causes trend (rates are not displayed)

with proportionally adjusted rates for all other causes of death, show a pattern of lower rates in the younger age groups, higher rates beginning in middle-age groups (ages 55-84), and decreasing rates for ages 85 and older.   Hypertension-related mortality rates for females, contrasted with proportionally adjusted rates for all other causes of death, show a pattern of lower rates in the younger age groups and higher rates beginning in the oldest (70 and older) age groups. Time trend analyses by age group indicate that the hypertension-related death rate did not change significantly for any age group in the Connecticut population from 1989 to 1998.

Risk factors for and prevention programs addressing hypertension-related diseases are discussed in the heart disease and stroke mortality sections of this report.

Diabetes Mellitus
(ICD-9 code 250)

Diabetes has become increasingly common in the United States. Between 1990 and 1998, the prevalence of diagnosed diabetes among American adults increased by 33%. Increases occurred in both the male and female populations, in all age and ethnic groups, and all educational levels. The greatest increase (76%) was observed in the 30 to 39 year old age group (Mokdad, Ford, Bowman, et. al. 2000). Almost 16 million Americans are currently estimated to have the disease (Centers for Disease Control and Prevention 2001b). In 1997, 117,200 Connecticut residents were estimated to have diagnosed diabetes with another 58,600 estimated to have an undiagnosed form of the disease (Connecticut Department of Public Health 2000).

Diabetes was the seventh leading cause of death in Connecticut in both the 1989-1991 and 1996-1998 periods. Because most people with diabetes die from its complications rather than from the disease itself, examination of diabetes as the underlying cause of death alone does not accurately represent its extensive contribution to overall mortality. While diabetes was the underlying cause of 659 resident deaths in 1998, it was listed as an underlying or contributing cause of death for 2,576 Connecticut residents. National data also suggest that diabetes is underreported on death certificates. Among persons with diabetes, only 10% have diabetes listed as an underlying cause of death and only 40% have diabetes listed as any cause of death (Centers for Disease Control and Prevention, 2001b).

Age-adjusted death and premature mortality rates due to diabetes increased significantly in Connecticut between the 1989-1991 and 1996-1998 periods (Table 5.1). This increase mirrors a similar trend nationwide. Diabetes death rates nationwide increased through the 1990s (Centers for Disease Control and Prevention 2001c).

Although age-adjusted diabetes death rates for Connecticut residents were consistently lower than comparable national rates from 1989 to 1998 (Figure 5.1), the increasing death rates for Connecticut residents during this period are notable. Of all the chronic diseases considered in this report, diabetes death rates showed the greatest increase among males, and the second greatest increase, after COPD, among females.

1996-1998 Diabetes Deaths, Connecticut Residents

- Significant increase in age-adjusted mortality since the 1989-91 period

- Significant increase in premature mortality since the 1989-91 period

- Seventh leading cause of death for all Connecticut residents

- Fifth leading cause of death for age groups 45 to 74

- Black males and females had the highest death and premature mortality rates

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The average annual percent increase in diabetes mortality for the period 1989 to 1998 was 4.0% for Connecticut male residents and 2.5% for female residents (p < .001 for both groups). Analyses of linear trends by age group for the same period indicate that the diabetes death rate for males aged 85 and over increased significantly while diabetes death rates of other gender/age groups in the population did not.

Racial/ethnic subgroups exhibited differential mortality due to diabetes (Table 5.1) and diabetes-related causes (Table 5.2). While all racial/ethnic groups showed evidence of increasing death rates due to diabetes, the increase reached

Figure 5.1.

Diabetes Mellitus Deaths
Comparison of Connecticut and U.S. 1989-1998
Age-Adjusted Mortality Rates* by Sex

Symbols:

- U.S. rates for 1989-1998

- Connecticut rates: an asterisk is used if the rate is significantly different than expected (p<.005), based on the log-linear trend.

* These rates are adjusted to the 1940 US standard million population.
* This classification includes deaths with ICD-9 codes:250

statistical significance only in the white population. Death rates for both white male and white female residents increased significantly from the 1989-1991 and 1996-1998 periods. There were no significant changes in diabetes-related mortality of Connecticut resident racial and ethnic subpopulations during the decade (Table 5.2).

Of all population groups, black males and females had the highest death rates due to diabetes and diabetes-related causes and significantly higher mortality than the respective white populations during the 1996-1998 period. Black males had 2.3 times the diabetes and 1.6 times the diabetes-related death rates of white males, while black females had 2.4 times the diabetes and diabetes-related mortality rates of white females. Premature mortality (to age 75) due to diabetes and diabetes-related causes was also significantly higher among black compared with white males and females, respectively. Black males had three times the rate of premature deaths due to diabetes and 2.3 times the rate of premature deaths due to diabetes-related mortality compared with white males. Black females had 2.7 times and 3.2 times the rate of premature death due to diabetes and diabetes-related mortality, respectively, of white females (Tables 5.1 and 5.2). These findings are consistent with national figures showing that blacks had more than twice the mortality from diabetes compared with whites during the 1989-1998 period (Centers for Disease Control and Prevention 2001d).
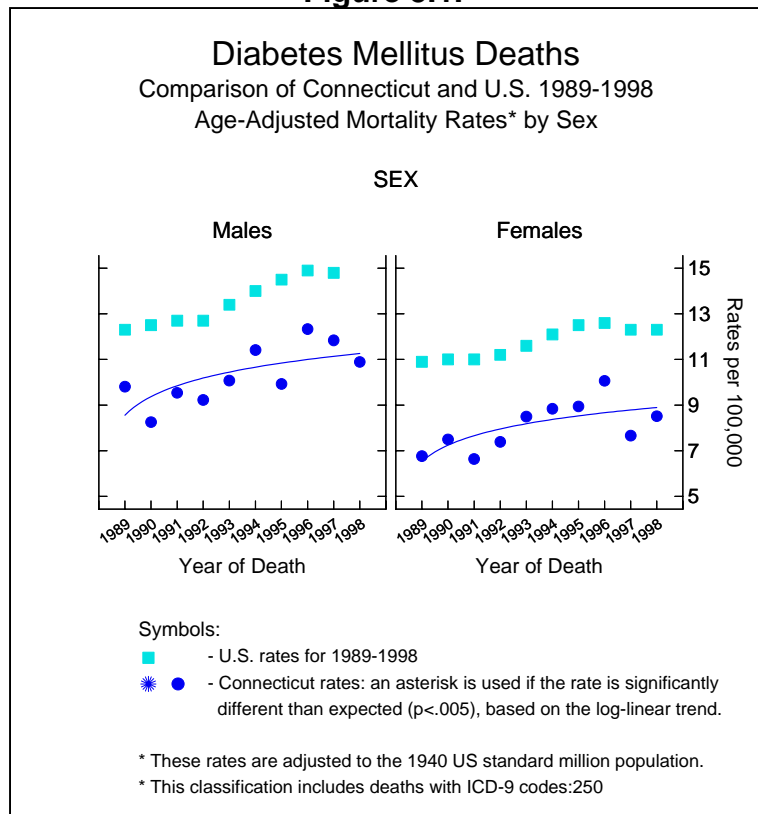
Table 5.1. Diabetes Mellitus Deaths[1]
Connecticut Residents by Gender, Race and Ethnicity[2], 1996-98

Group	Number of Deaths	Age-Adjusted Mortality Rates[3]		Age-Adjusted Premature Mortality Rates to Age 75[3]	
		AAMR[4]	Change since 1989-91[5]	YPLL[4]	Change since 1989-91[5]
All Residents	1,991	18.4	↑↑↑	117.6	↑
All males	933	21.8	↑↑↑	134.4	ns
White	825	20.5	↑↑↑	116.7	ns
Black	100	48.0***	ns	346.6***	ns
Asian PI	5	—		—	
Native American	1	—		—	
Hispanic	42	30.3	na	227.8*	na
All females	1,058	16.2	↑↑	102.0	↑
White	941	15.1	↑↑	92.0	↑
Black	115	36.3***	ns	244.5***	ns
Asian PI	1	—		—	
Native American	1	—		—	
Hispanic	33	17.8	ns	120.6	ns

Notes:

1. This cause of death category includes ICD-9 codes 250.

2. Racial groupings (White, Black, Asian & Pacific Islander, Native American) include persons of Hispanic ethnicity.

3. Age-adjusted Mortality Rates (AAMR) and Years of Potential Life Lost (YPLL) rates are per 100,000 based on race and ethnicity specific population estimates. Age-adjusted rates were calculated by the direct method using the 2000 U.S. standard population. Rates were not calculated for fewer than 15 events.

4. Statistical tests were conducted to evaluate differences in rates between race/ethnic groups. The white population serves as the reference group in each comparison (black vs. white, Asian & PI vs. white, Native American vs. white, Hispanic vs. white). Following are explanations of the notations:

> *   Significantly different than the respective white resident rate at $p < .05$.
> *** Significantly different than the respective white resident rate at $p < .001$.
> —   Rate was not calculated due to small numbers.

5. Statistical tests were conducted to evaluate changes in rates over time. Comparisons of 1996-98 vs. 1989-91 rates are made within each race/ethnicity group. Following are explanations of the notations:

> ↑   1996-98 rate is significantly higher than the 1989-91 rate at $p < .05$.
> ↑↑   1996-98 rate is significantly higher than the 1989-91 rate at $p < .01$.
> ↑↑↑ 1996-98 rate is significantly higher than the 1989-91 rate at $p < .001$.
> ns Indicates the change from 1989-91 to 1996-98 is not statistically significant.
> na 1989-91 rate was not calculated due to small numbers and so no comparison with 1996-98 rate is available

Table 5.2. Diabetes-Related Deaths[1]
Connecticut Residents by Gender, Race and Ethnicity[2], 1996-98

| Group | Number of Deaths | Age-Adjusted Mortality Rates[3] | | Age-Adjusted Premature Mortality Rates to Age 75[3] | |
		AAMR[4]	Change since 1989-91[5]	YPLL[4]	Change since 1989-91[5]
All Residents	7,600	69.8	ns	366.2	ns
All males	3,651	85.4	ns	446.3	ns
White	3,343	83.0	ns	405.5	ns
Black	283	131.3***	ns	989.9***	ns
Asian PI	19	44.0**	na	248.1	na
Native American	4	—		—	
Hispanic	133	98.1	ns	729.2***	ns
All females	3,949	59.1	ns	292.4	ns
White	3,516	55.1	ns	251.8	ns
Black	416	134.2***	ns	799.6***	ns
Asian PI	11	—		—	
Native American	6	—		—	
Hispanic	133	73.3*	ns	444.6**	ns

Notes:

1. This cause of death category includes ICD-9 codes 250. "Diabetes-related" deaths include those for which diabetes is the underlying and/or a contributing cause listed on the death certificate.

2. Racial groupings (White, Black, Asian & Pacific Islander, Native American) include persons of Hispanic ethnicity.

3. Age-adjusted Mortality Rates (AAMR) and Years of Potential Life Lost (YPLL) rates are per 100,000 based on race and ethnicity specific population estimates. Age-adjusted rates were calculated by the direct method using the 2000 U.S. standard population. Rates were not calculated for fewer than 15 events.

4. Statistical tests were conducted to evaluate differences in rates between race/ethnic groups. The white population serves as the reference group in each comparison (black vs. white, Asian & PI vs. white, Native American vs. white, Hispanic vs. white). Following are explanations of the notations:

    *   Significantly different than the respective white resident rate at p < .05.
    ** Significantly different than the respective white resident rate at p < .01.
    *** Significantly different than the respective white resident rate at p < .001.
    —   Rate was not calculated due to small numbers.

5. Statistical tests were conducted to evaluate changes in rates over time. Comparisons of 1996-98 vs. 1989-91 rates are made within each race/ethnicity group. Following are explanations of the notations:

    ns   Indicates the change from 1989-91 to 1996-98 is not statistically significant.
    na   1989-91 rate was not calculated due to small numbers and so no comparison with 1996-98 rate is available

Logistic regression analyses indicate that there is consistency of the Connecticut black-white male and black-white female disparity in the diabetes death rate across all 5-year age groups. Similar analyses of the diabetes-related death rates indicate that there is not a consistent disparity across all age groups. Although there were too few black male deaths within specific age groups in 1996-1998 to pinpoint age-specific differences, there was a clear trend toward a decreasing black-white disparity beginning at ages 65-70. There is consistency in the black-white female disparity in diabetes-related mortality across 5-year age groups.

The diabetes mortality rates of Hispanic males and females were not significantly different from the respective white rates in 1996-1998, although Hispanic males did have significantly higher premature mortality due to diabetes compared with white males (Table 5.1). Hispanic females had higher death and premature mortality rates from diabetes-related causes than did white females. Hispanic males did not have significantly different diabetes-related death rates compared with white males, although they did have significantly higher premature mortality rates due to diabetes-related causes (Table 5.2). Logistic regression analyses indicate that there is consistency across 5-year age groups of the Hispanic-white female disparity in the diabetes-related death rate. There does not appear to be consistency in the Hispanic-white male disparity across 5-year age groups. While there were too few deaths within specific 5-year age groups to identify differences, there was a clear trend toward a decreasing Hispanic-white disparity beginning at ages 50-55. Nationwide, Hispanics have tended to have slightly higher diabetes mortality rates compared with non-Hispanic whites (Sorlie, Backlund, Johnson et al. 1993; Swenson, Trepka, Rewers, et al. 2002).

There were insufficient numbers of diabetes deaths among Native American and Asian and Pacific Islander males and females to calculate reliable rates (Table 5.1). The diabetes-related death rate of Asian and Pacific Islander males was significantly lower than that of white males, but there was no significant difference in their premature mortality rates due to diabetes-related causes. Diabetes-related death rates of Native American males and females and Asian and Pacific Islander females were not calculated due to insufficient numbers of deaths (Tables 2). Nationally, Native Americans have higher diabetes mortality rates than do whites, while Asian and Pacific Islanders have been reported to have lower diabetes mortality rates than whites (Carter, Pugh, and Monterrosa 1996).

In the 1996-1998 period, Connecticut male residents had significantly higher mortality from both diabetes ($p < .001$) and diabetes-related causes ($p < .001$) compared with Connecticut females (Table 5.1 and Table 5.2). These findings are consistent with national data from 1989-1998 showing slightly higher diabetes mortality among males.

Figure 5.2 depicts age-specific rates for diabetes deaths relative to all other causes of death (1996-1998) for males and females. Diabetes death rates tend to be lower compared with all other causes of death up to about age 49 for males and age 44 for females after which they tend to be approximately the same as for all other causes of death.

Figure 5.2.

Age-specific Rates for Diabetes-Related Causes Deaths
and for All Other Causes, 1996 - 1998

Age-specific Deaths Rates and Trend Lines

● Diabetes-Related Causes rates and trend
– All Other Causes trend (rates are not displayed)
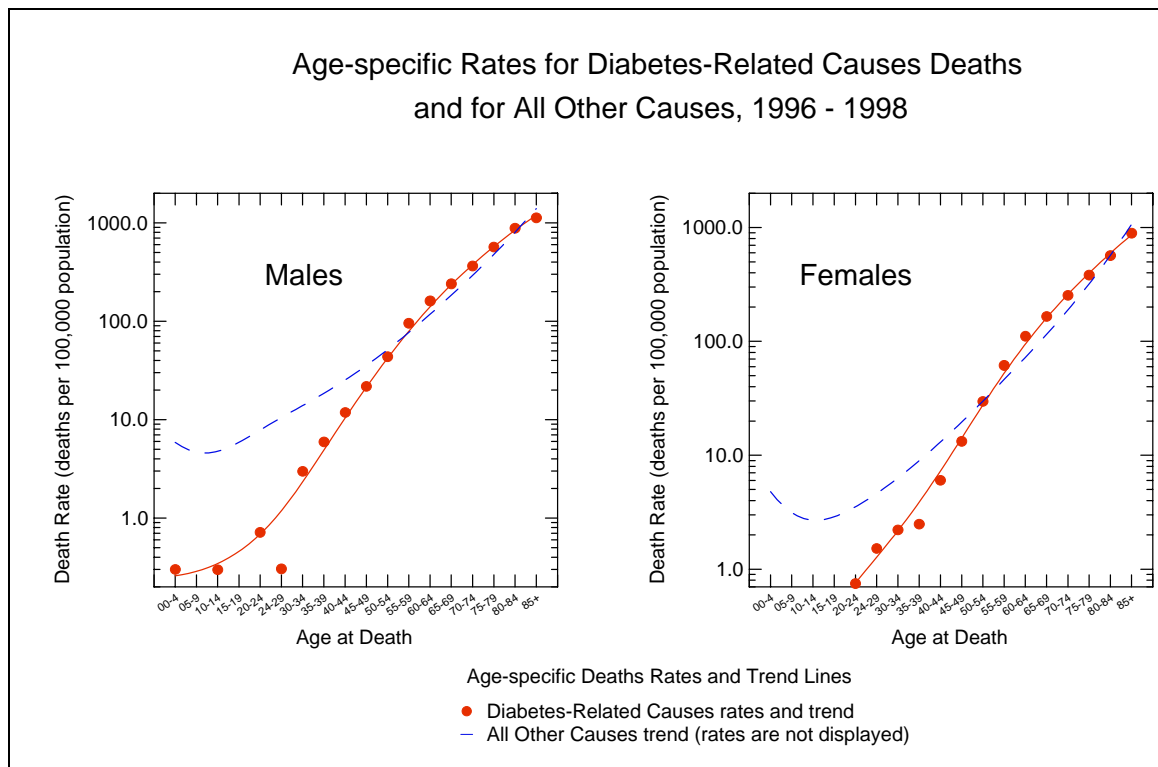
Risk Factors

Type 2 diabetes (formerly called Non-Insulin Dependent Diabetes), the most common form of the disease, affects 90% to 95% of all people with diabetes and most often occurs in adults over age 40. Non-modifiable individual level risk factors for type 2 diabetes include a family history of type 2 diabetes, a history of gestational (pregnancy-related) diabetes, and age over 40 (Bishop, Zimmerman, and Roesler 1998). Type 1 diabetes (formerly called Insulin Dependent Diabetes) is believed to be related to environmental causes such as viral exposures and dietary practices (Bishop, Zimmerman, and Roesler 1998; Disdier-Flores, Rodriguez-Lougo, Perez-Perdomo et al. 2001).

Connecticut Behavioral Risk Factor Surveillance data for 1990-1996 indicate that diabetes prevalence increased dramatically by age group among adults. Connecticut residents 65 and older were almost twice as likely as those aged 45 to 64, and six times more likely than those aged 18 to 44, to report diagnosed diabetes. About half of all Connecticut adults with diabetes are aged 65 and older (Frost 2000).

Males and females are equally at risk for diabetes. About 4.9% of Connecticut females and 4.3% of males reported a diagnosis of diabetes in 1996 (Frost 2000). Nationally, 8.2% of both men and women aged 20 and older have diabetes (U.S. Department of Health and Human Services 1998b).

Type 2 diabetes tends to be more prevalent among black, Hispanic and Native American compared with white persons, while type 1 diabetes is more prevalent among white Americans (Carter, Pugh, Monterrosa 1996; Bishop, Zimmerman, and Roesler 1998; Disdier-Flores, Rodriguez-

Lougo, Perez-Perdomo et al. 2001). Prevalence data for Asian and Pacific Islanders are limited, although some subpopulations may have higher prevalence rates than the white population (U.S. Department of Health and Human Services 1998b). Racial and ethnic minority groups are also more likely to be at higher risk for the complications of diabetes (Centers for Disease Control and Prevention 2001b).

Modifiable risk factors for type 2 diabetes include obesity and physical inactivity, which are conditions associated with increased insulin resistance (Bishop, Zimmerman, and Roesler 1998). Recent findings from a national prospective study suggest that cigarette smoking is associated with an increased risk for type 2 diabetes independent of other risk factors (Will, Galuska, Ford, et al. 2001). Research also suggests that consumption of whole grains may reduce the risk for diabetes by favorably influencing metabolism (McKeown, Meigs, Liu, et al. 2002) [Table 5.3]. No known modifiable risk factors have been established for type 1 diabetes (Disdier-Flores, Rodriguez-Lougo, Perez-Perdomo et al. 2001).

Table 5.3. Modifiable Risk Factors for Diabetes

Risk Factor	Magnitude of Association[1]
Obesity ($\geq$ 20% over desired weight)	Strong
Physical inactivity	Weak
Cigarette smoking	Possible
High fat / low fiber diet	Possible

Source*: Adapted from Bishop, Zimmerman, and Roesler 1998.

1. Strong magnitude indicates a relative risk greater than 4 for those persons with the risk factor compared with those not having the risk factor. Moderate magnitude indicates a relative risk of between 2 and 4 for those persons with the risk factor compared with those not having the risk factor. Weak magnitude indicates a relative risk of less than 2 for those persons with the risk factor compared with those not having the risk factor. Possible association indicates that some, but not definitive, evidence exists to support these as risk factors for diabetes.

Connecticut Behavioral Risk Factor Surveillance Data for 1990-1996 indicate that more than half of Connecticut adults without diagnosed diabetes had one or more modifiable risk factors for the disease. Fifty-one percent of respondents reported insufficient physical activity, 26% reported being overweight, and 22% were current smokers (Frost 2000).

For persons with diagnosed diabetes, modifiable risk factors like obesity, insufficient physical activity, hypertension, high cholesterol, and smoking can interact to increase the risk for and severity of complications from the disease. Cigarette smoking also contributes to a greater likelihood of diabetes complications among persons with the disease (Beckles and Thompson-Reid 2001). Fifty-eight percent of Connecticut adults with diabetes reported insufficient physical activity, 51% reported being overweight, 14% were current smokers, 53% reported high blood pressure, and 33% had elevated cholesterol levels (Frost 2000).

Low socioeconomic status (SES) has been linked to higher prevalence of type 2 diabetes (Brancati, Whelton, Kuller, et al. 1996; Robbins, Vaccarino, Zhang, et al. 2000; Connolly, Unwin, Sherriff et al. 2000; Robbins, Vaccarino, Zhang, et al 2001; Beckles and Thompson-Reid 2002; Everson, Maty, Lynch, et al. 2002). Low-income persons are less likely than higher-income persons to have an adequate diet, sufficient physical activity, and access to medical care, factors known to affect progression of the disease.

Findings from the Third National Health and Nutrition Examination Survey (1988-1994) suggest that for black and non-Hispanic white females the relationship between diabetes prevalence and low SES[6] is independent of obesity; however, among males this relationship is not consistent. Socioeconomic disadvantage is strongly associated with type 2 diabetes prevalence among black females, white females, and white males, but not among black males (Robbins, Vaccarino, Zhang, et al. 2001). One study of black and white adults aged 35 to 54 in three U.S. communities found that black adults had a significantly higher prevalence of diabetes even after controlling for black-white differences in obesity and SES (Brancati, Whelton, Kuller, et al. 1996). This study did not report gender-specific analyses and did not report income adjusted by family size as a measure of SES. Although additional research is needed to more fully understand the interrelationships between SES, minority ethnicity, and diabetes, economic disadvantage appears to be a key explanatory variable in the increased prevalence of the disease (Robbins, Vaccarino, Zhang, et al. 2000; 2001).

Costs and Prevention

Direct and indirect costs of treating diabetes in the U.S. were estimated at $132 billion in 2001. Direct medical expenditures, which include diabetes care, chronic complications of diabetes, and excess prevalence of general medical conditions, totaled $91.8 billion nationwide. More than half of direct medical expenditures were by people over 65. Indirect expenditures, which include costs of lost workdays, restricted activity days, mortality, and permanent disability, totaled $39.8 billion nationwide. After adjustment for differences in the populations with and without diabetes (by age, gender, race/ethnicity), people with diabetes are estimated to have medical expenditures that are about 2.4 times higher than expenditures for people without diabetes (American Diabetes Association 2003). It is estimated that the direct costs of inactivity and obesity account for approximately 9.4% of all health care expenditures in the United States (Colditz 1999). Direct and indirect costs of treating diabetes in Connecticut were estimated at $1.2 billion in 1997 (Connecticut Department of Public Health 2000).

Primary prevention of diabetes may be directed toward entire communities emphasizing risk factor reduction and environmental change or it may target individuals at high-risk for the disease. Community initiatives might focus on improvement in the food supply and distribution, increasing opportunities for exercise, and other related issues of economic disadvantage, such as access to medical care.

Recent findings from two major clinical trials indicate that type 2 diabetes can be prevented through changes in dietary habits, weight loss, and exercise. Researchers in Finland found that

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[6] As measured by categories of the Poverty Income Ratio (PIR), that is annual family income divided by the federal poverty line. This line is adjusted yearly for inflation and varies with household size.

lifestyle changes in a population at high risk for diabetes reduced the incidence of type 2 diabetes by 58% over a four-year period (Tuomilehto, Lindstrom, Eriksson, et al. 2001). The Diabetes Prevention Program study in the U.S. found that high-risk individuals in a lifestyle intervention of dietary change and exercise reduced their risk of type 2 diabetes by 58%. The intervention group, on average, had 30 minutes of moderate intensity exercise daily and lost 5% to 7% of their body weight. (A second intervention group of individuals treated with the oral diabetes drug metformin reduced their risk by 31%). This is the first U.S. nationwide trial to show that diabetes incidence can be delayed effectively through a program of diet and exercise (National Institute of Diabetes & Digestive & Kidney Diseases 2001a; Diabetes Prevention Program Research Group 1999).

The U.S. Preventive Services Task Force (USPSTF) states that there is insufficient evidence to recommend for or against routine screening of asymptomatic adults for type 2 diabetes, impaired glucose tolerance, or impaired fasting glucose. USPSTF does recommend screening for Type 2 diabetes in adults with hypertension or hyperlipidemia (U.S. Preventive Services Task Force 2002).

For persons with diabetes, control of blood pressure and blood cholesterol levels are two important strategies in the management of the disease (National Institute of Diabetes & Digestive & Kidney Diseases 2001b; Snow, Weiss, Mottur-Pilson 2003; Vijan and Hayward 2003). In 2003, the American College of Physicians established new guidelines for treating hypertension in type 2 diabetes based on evidence that tight blood pressure control decreases heart disease, stroke, and early death in patients with type 2 diabetes. Blood pressure levels less than 135/80 are recommended for patients with type 2 diabetes (Vijan and Hayward 2003). Research has shown that the progression of type 2 diabetes can be delayed through improved nutrition, exercise, control of blood glucose levels (Padgett, Mumford, Carter et al. 1988; Clement 1995), and appropriate medical care (Centers for Disease Control and Prevention 2001b). Such self-management measures can reduce long-term complications of the disease, such as heart disease, stroke, blindness, amputation, and kidney disease. Observational studies have suggested that intentional weight loss may reduce diabetes mortality in men and women (Williamson, Pamuk, Thun, et al. 1995; Williamson, Pamuk, Thun, et al. 1999).

Obesity reached epidemic proportions in the United States during the 1990s (Mokdad, Serdula, Dietz, et al. 1999). This fact would suggest that individually-based approaches to preventing overweight and obesity have not been successful (Nestle and Jacobson 2000). Population-based approaches to obesity prevention include educational, legislative, and policy initiatives. Specific suggestions include: enactment of federal policies related to improved food labeling and advertising; changes in food assistance programs to encourage more nutritious choices; emphasis on nutritional and physical activity educational programs in schools and local communities; emphasis on educational curricula for health care providers related to nutrition, obesity, and physical activity; creating a variety of tax incentives at the local, state or federal level for weight management and fitness programs; taxes on low nutrient foods like soft drinks, government subsidies for nutritious foods, and subsidies for recreational areas in communities; and national policy development related to physical fitness, nutrition, and obesity prevention (Nestle and Jacobson 2000).

All Cancer
(ICD-9 codes 140.0-208.9)

Cancer is the second leading cause of death in Connecticut and the United States, accounting for 70,533 Connecticut resident deaths (24% of all deaths) from 1989 to 1998. It is the leading cause of premature mortality in Connecticut, with an estimated 504,585 years of potential life lost to age 75 during the ten-year period. The costs of cancer in Connecticut including costs for direct medical expenses, lost productivity, and mortality have been estimated at $1.3 billion per year or more than $400 per person (Adams 2000). A large proportion of cancer morbidity and mortality is considered preventable through modification of known risk factors, such as tobacco use, dietary factors like high fat and low fiber, and occupational exposures (Greenwald and Sondik 1986).

More than half of all cancer deaths in Connecticut and the U.S. are due to lung and other respiratory, colorectal, female breast, and prostate cancers (Figure 6.1) (U.S. Department of Health and Human Services 1990). Mortality from each of these causes is discussed in subsequent sections of this report.

Males in both Connecticut and the U.S. have significantly higher overall cancer mortality than females. In the 1996-1998 period, Connecticut males had 1.4 times the rate of cancer deaths (p < .001) and 1.1 times the premature mortality rate (p < .05) of females. Among racial/ethnic and gender subgroups, black males had the highest death and premature mortality rates from cancer. Black males had cancer death rates about 1.4 times higher and premature mortality rates about 1.6 times higher than white males during the 1996-1998 period (p < .001). White males had significantly higher cancer death and premature mortality rates than Hispanic and Asian and Pacific Islander males. Among females, there were no significant differences in cancer death rates between white and black females, although black females did have significantly higher premature mortality due to cancer for the 1996-1998 period. White females had significantly higher cancer death and premature mortality rates than Hispanic and Asian and Pacific Islander females (Table 6.1). Nationwide, black males had the highest cancer mortality rates for the 1992-1998 period followed by white males; among females nationwide, black females had the highest all-cancer mortality followed by white females (Howe, Wingo, Thun, et al. 2001).

1996-1998 Cancer Deaths, Connecticut Residents

- The second leading cause of death

- The leading cause of premature mortality (to age 75)

- The leading cause of death for residents aged 45 to 74

- Significant decrease in female premature mortality since the 1989-1991 period

- Significant decrease in male age-adjusted death and premature mortality rates since the 1989-1991 period

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Figure 6.1.
Cancer Deaths, Percent by Subtype
Connecticut Residents, 1989-1998
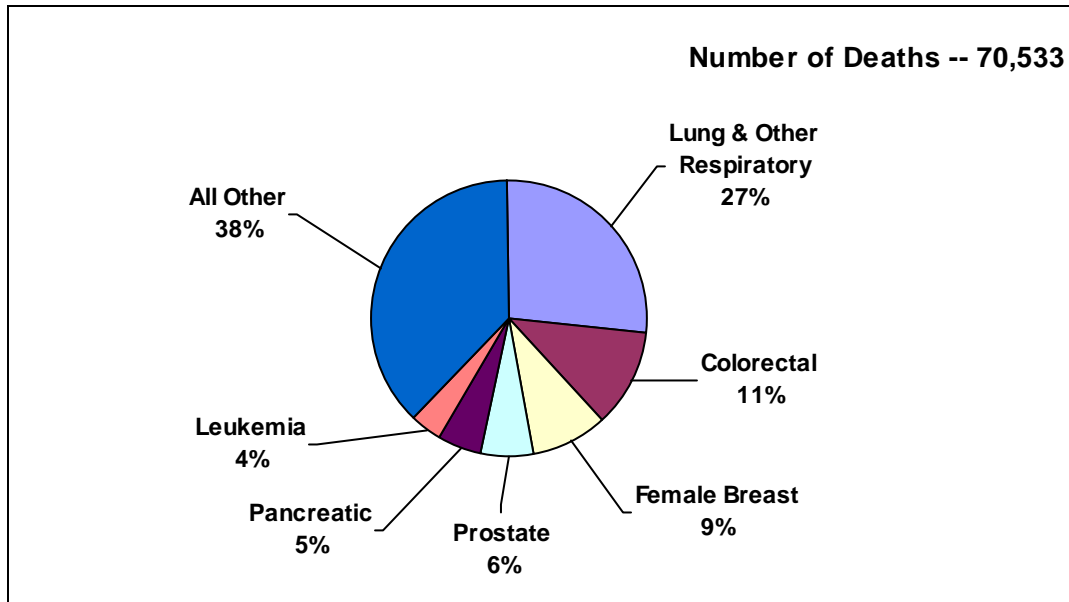
Logistic regression analyses of the black-white male and black-white female cancer mortality disparity and the Hispanic-white male and Hispanic-white female cancer mortality disparity indicate that there is not a consistent disparity across all 5-year age groups; however, no clear patterns in the disparities by 5-year age group are evident.

Age-adjusted death and premature mortality rates due to all cancers decreased significantly for Connecticut male residents from the 1989-1991 to 1996-1998 period. Premature mortality rates (to age 75) for females in Connecticut decreased significantly, although the female age-adjusted death rate did not change significantly between these two periods (Table 6.1). Male cancer mortality rates decreased significantly by 1.4% per year from 1989 to 1998 (p ≤ .05). Female cancer mortality rates did not decrease significantly during this period. U.S. trends for 1992 though 1998 show that all-cancer mortality declined by 2.7% per year in males and 0.3% in females (p < .05 for both groups) (Howe, Wingo, Thun, et al. 2001).

The Connecticut male all-cancer mortality rate was significantly lower than the comparable U.S. rate from 1989 to 1998, whereas the Connecticut female all-cancer mortality rate was significantly lower than the comparable U.S. rate for a few (1990, 1992, 1994, 1995) but not the most recent years of 1996 to 1998 (Figure 6.2). Connecticut resident all-cancer mortality rates were lower than the *Healthy People 2000* target from 1989 to 1998 and lower than the *Healthy Connecticut* target for 1998 only (Table 6.2).

Table 6.1. All Cancer Deaths[1]
Connecticut Residents by Gender, Race and Ethnicity[2], 1996-1998

Group	Number of Deaths	Age-Adjusted Mortality Rates[3]		Age-Adjusted Premature Mortality Rates to Age 75[3]	
		AAMR[4]	Change since 1989-91[5]	YPLL[4]	Change since 1989-91[5]
All Residents	21,300	199.0	↓↓↓	1,566.2	↓↓↓
All males	10,602	243.8	↓↓↓	1,632.6	↓↓↓
White	9,828	240.3	↓↓↓	1,577.9	↓↓
Black	715	332.8***	ns	2,588.9***	↓
Asian PI	46	102.1***	ns	686.5***	ns
Native American	13	—		—	
Hispanic	208	148.3***	ns	1,186.9**	ns
All females	10,698	171.9	ns	1,510.5	↓
White	10,027	171.8	ns	1,494.4	ns
Black	614	187.3	ns	1,799.1*	ns
Asian PI	44	71.7***	na	779.6***	na
Native American	10	—		—	
Hispanic	181	86.2***	ns	959.3***	ns

Notes:

1. This cause of death category includes ICD-9 codes 140-208.

2. Racial groupings (White, Black, Asian & Pacific Islander, Native American) include persons of Hispanic ethnicity.

3. Age-adjusted Mortality Rates (AAMR) and Years of Potential Life Lost (YPLL) rates are per 100,000 based on race and ethnicity specific population estimates. Age-adjusted rates were calculated by the direct method using the 2000 U.S. standard population. Rates were not calculated for fewer than 15 events.

4. Statistical tests were conducted to evaluate differences in rates between race/ethnic groups. The white population serves as the reference group in each comparison (black vs. white, Asian & PI vs. white, Native American vs. white, Hispanic vs. white). Following are explanations of the notations:

   * Significantly different from the respective white resident rate at $p < .05$.
   ** Significantly different from the respective white resident rate at $p < .01$.
   *** Significantly different from the respective white resident rate at $p < .001$.
   — Rate was not calculated due to small numbers.

5. Statistical tests were conducted to evaluate changes in rates over time. Comparisons of 1996-98 vs. 1989-91 rates are made within each race/ethnicity group. Following are explanations of the notations:

   ↓ 1996-98 rate is significantly lower than the 1989-91 rate at $p < .05$.
   ↓↓ 1996-98 rate is significantly lower than the 1989-91 rate at $p < .01$.
   ↓↓↓ 1996-98 rate is significantly lower than the 1989-91 rate at $p < .001$.
   ns Indicates the change from 1989-91 to 1996-98 is not statistically significant.
   na 1989-91 rate was not calculated due to small numbers and so no comparison with 1996-98 rate is available

The decrease in age-adjusted all-cancer mortality was statistically significant for white males but not other racial/ethnic male or female subgroups from 1989-1991 to 1996-1998. Decreases in premature mortality (before age 75) were statistically significant for white and black males but not for other racial/ethnic male subgroups. Cancer mortality decreased significantly for Connecticut females as a whole, but not for any female racial/ethnic subgroup. There were too few cancer deaths among Native American males and females to calculate reliable rates (Table 6.1). National trends for 1992 through 1998 show

Figure 6.2.

All Cancer Deaths
Comparison of Connecticut and U.S. 1989-1998
Age-Adjusted Mortality Rates* by Sex

SEX

Males   Females

Year of Death   Year of Death

Rates per 100,000

Symbols:
- U.S. rates for 1989-1998
- Connecticut rates: an asterisk is used if the rate is significantly different than expected (p<.005), based on the log-linear trend.

* These rates are adjusted to the 1940 US standard million population.
* This classification includes deaths with ICD-9 codes:140-208

that cancer mortality rates decreased significantly for white and black males and white and black females with the greatest decrease in all-cancer mortality, about 2% per year, occurring among black males (Howe, Wingo, Thun, et al. 2001).
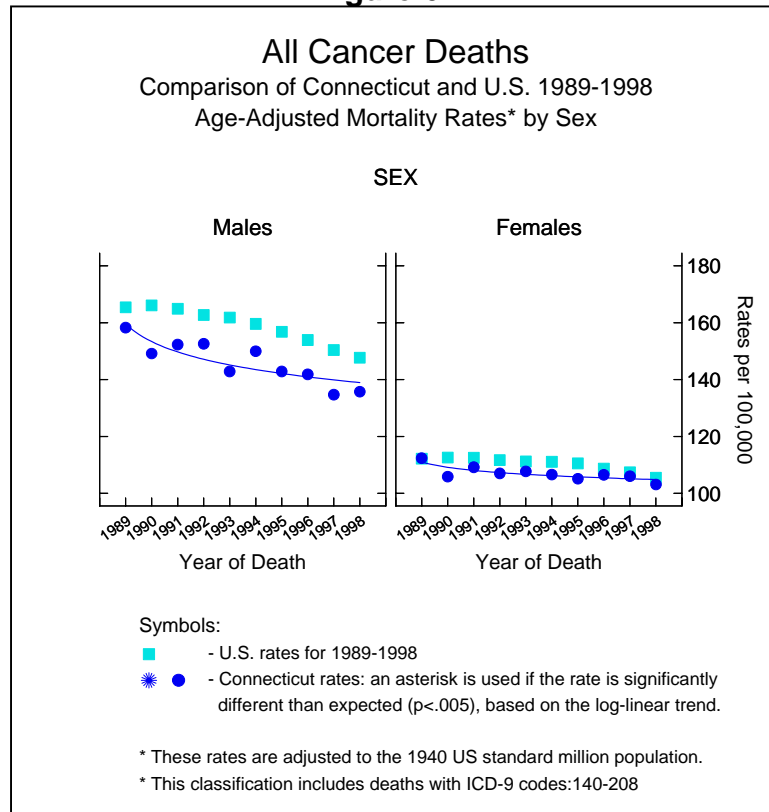
**Table 6.2. All Cancer Age-Adjusted Death Rates,
Comparison of CT with US - 1989 and 1998**

	1989	1998	1998 CT AAMR Comparison
CT AAMR*	131.2	116.8	
US AAMR*	134.3	123.5	CT rate < US rate
Healthy People 2000*	130.0	130.0	achieved *HP* target
Healthy CT 2000*	120.0	120.0	achieved *Healthy CT* target

* age-adjusted mortality rates for cancer are per 100,000 population, U.S. 1940 standard million population.

Age-specific cancer death rates of Connecticut males and females for the period 1996-1998 are displayed in Figure 6.3. Cancer mortality rates for males and females, contrasted with proportionally adjusted rates for all other causes of death, show a pattern of lower rates in the younger age groups, higher rates beginning in middle-age (ages 50-54 for males and 35-39 for females), and slightly lower rates in the oldest age groups (80 and older). Cancer mortality rates increase with increasing age, with the highest rates found in the 85 and older age group. Fifty-five percent of all cancer deaths occurred among Connecticut residents aged 80 and older during the 1996-1998 period.

Figure 6.3.

Age-specific Rates for All Cancers Deaths and
Proportionally Adjusted Rates for All Other Causes, 1996 - 1998
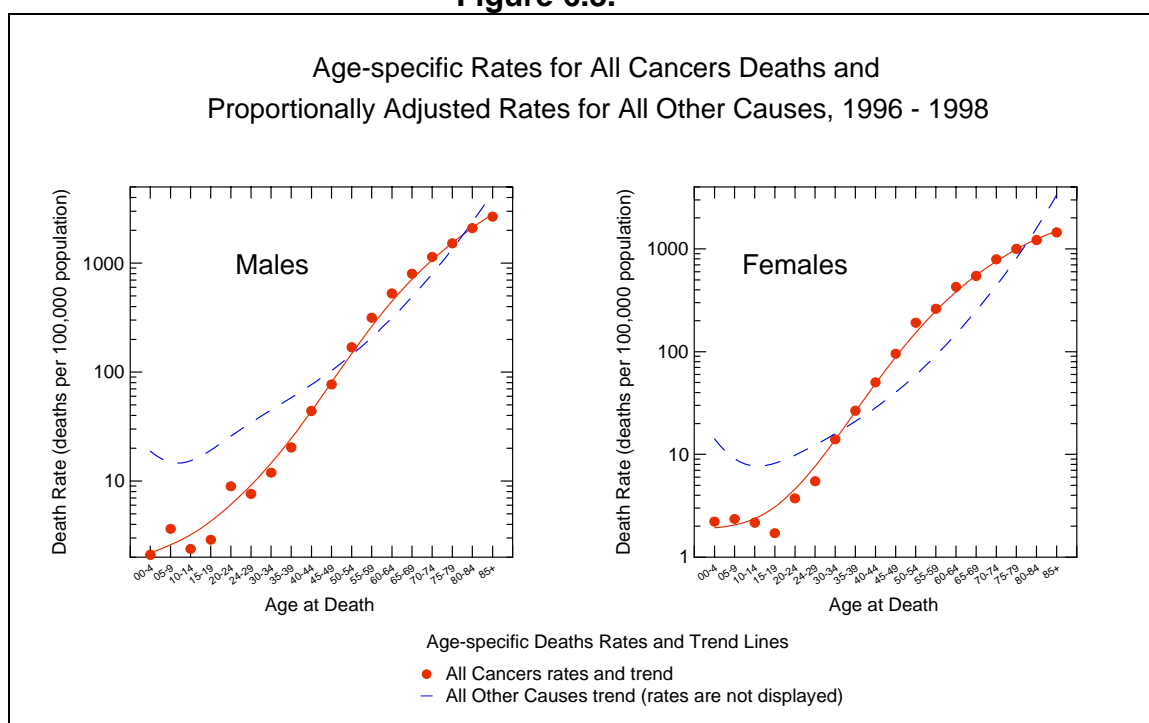
Cancer Incidence

The age-standardized incidence rate for all invasive cancers from the 1990-1994 to 1995-1998 period showed little change for Connecticut males, but increased slightly in Connecticut females due primarily to an increase in lung and a slight increase in breast cancers. For the 1995-1998 period, male residents had 1.3 times the incidence rate for all invasive cancers compared with female residents (Polednak 2001a). From 1990-1995, black male residents had 1.2 times, and Hispanic males 0.9 times the age-standardized incidence rate for all invasive cancers compared with white male residents. Black and Hispanic females had age-standardized incidence rates for all invasive cancers that were each 20% lower than that of white female residents of Connecticut (Polednak 1999a).

Risk Factors

Estimates of the percentage of cancer deaths due to various causes have been developed by Doll and Peto (1981), Miller (1992), and the Harvard Center for Cancer Prevention (1996) [Table 6.3]. These estimates suggest that prevention efforts should target the major behavioral risk factors, emphasizing the elimination of tobacco use, and encouraging physical activity and diets low in fat and high in fresh fruits and vegetables. Cancers associated with these behaviors include lung and other respiratory, colorectal, and breast cancers.

Some researchers have suggested that such estimates tend to underemphasize environmental and occupational exposures to carcinogens (Epstein 1990; Clapp 1998). One study of occupationally-related diseases in Connecticut estimated that between 6% and 10% of all cancer deaths are occupationally-related (Morse and Storey 1999). Still other researchers have identified socioeconomic position, which is not well accounted for in these estimates, as an important determinant of cancer occurrence (Kogevinas, Pearch, Susser, et al. 1997; Krieger, Quesenberry, Peng, et al. 1999).

Table 6.3. Percentage of Cancer Deaths Attributed to Various Factors

Factor	Doll & Peto Estimate	Miller Estimate	Harvard Estimate
Tobacco	30	29	30
Diet	35	20	30
Infections	10	--	5
Occupation	4	9	5
Family history	--	8	5
Reproductive & Sexual History	7	7	3
Sedentary lifestyle	--	--	5
Perinatal factors/growth	--	--	5
Geophysical	3	1	2
Alcohol	3	6	3
Socioeconomic status	--	--	3
Pollution	2	--	2
Medication & medical procedures	1	2	1
Industrial & consumer products	1	--	
Salt/ Other food additives/ Contaminants	--	--	1

Source*: Brownson, Reif, Alavanja, et al. 1998.

Note: Data were compiled from Doll and Peto (1981), Miller (1992), and the Harvard Report on Cancer Prevention (1996).

International cancer incidence and mortality data indicate that for men, in general, excess risk due to respiratory, oral, pharangeal, esophageal, and stomach cancers, are found in the lower socioeconomic (SES) strata, while excess risk due to colon, brain, and skin cancers are found in the higher SES strata. Among women, excess risk due to cervical, stomach, and esophageal cancers are found in lower SES groups, whereas risk due to colon, breast, ovarian, and skin cancer are found in higher SES groups (Faggiano, Partanen, Kogevinas, et al. 1997). Krieger and colleagues found that cancer incidence rates in the San Francisco Bay Area varied equally if not more so by socioeconomic status than by race/ethnicity (Krieger, Quesenberry, Peng, et al. 1999).

The Behavioral Risk Factor Surveillance System (BRFSS), a telephone survey of adults, and the Youth Risk Behavior Survey (YRBS), a school-based self-administered survey, provide prevalence estimates of behavioral risk factors linked to various cancers. Smoking prevalence rates among Connecticut adults declined from about 27% in the late 1980s to 21% between 1994 and 1997. The number of adults who stop smoking annually is approximately the same as the number of young people who initiate smoking each year. Thus, smoking rates have remained stable in recent years (Adams 2000).

Epidemiologic studies show a consistent association between decreased fruit and vegetable intake and increased cancer risk, especially cancers of the lung, esophagus, oral cavity and pharynx, larynx, rectum, stomach, bladder, cervix, and endometrium. Studies of colon cancer indicate a protective effect of vegetable and dietary fiber intake (Brownson, Reif, Alavanja, et al. 1998). The American Cancer Society (ACS) dietary guidelines for reducing cancer risk include limiting the intake of high fat foods and increasing the consumption of foods from plant sources (five servings of fruit and vegetables per day are recommended). Approximately 33% of students and 30% of adults in Connecticut reported consuming five or more servings of fruit and vegetables per day. Male students and young adults under 35 reported the highest levels of high fat food (Adams 2000).

Regular physical activity is linked to lower incidence of colon cancer. Research data do not support an association between physical activity and rectal cancer. Data are too limited or inconsistent to support a conclusive link between physical activity and breast or prostate cancers (U.S. Department of Health and Human Services 1996). The Centers for Disease Control and Prevention and the American Cancer Society recommend thirty minutes of moderate-intensity physical activity each day for beneficial health effects.[7] Only 21% of Connecticut students and adults reported getting the recommended amount of exercise five days a week. More than one-quarter of adults reported no leisure time physical activity (Adams 2000).

Alcohol abuse is linked to an increased risk of cancer of the esophagus, nasopharynx, larynx and liver (Dufour 1998). The American Cancer Society's recommendation for alcohol consumption is 30 or fewer drinks per month for females and 60 or fewer drinks per month for males. About 4% of Connecticut adults reported exceeding the recommended monthly amount of alcohol (Adams 2000).

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[7] The Institute of Medicine recently issued a report on healthy eating and exercise, which recommends that adults and children spend one hour per day in moderate intensity physical activity (Institute of Medicine 2002).

Table 6.4. USPSTF Recommendations for Cancer Screening

Type of cancer	Screening recommendation
Lung	• Routine screening for lung cancer with chest radiography or sputum cytology in asymptomatic persons is *not* recommended.
Prostate	• There is insufficient evidence to recommend for or against routine screening for prostate cancer using prostate specific antigen (PSA) testing or digital rectal examination (DRE).
Colorectal	Recommendation for persons aged 50 and older: • Periodic fecal occult blood testing • Sigmoidoscopy • Or both in combination • Persons considered at high-risk because of family history should consider initiating screening at an earlier age. • There is insufficient evidence to recommend for or against routine screening by double-contrast barium enema, colonoscopy, or newer screening technologies (e.g. computed tomographic colography).
Breast	Recommendation for women aged 40 and older: • Screening for breast cancer ever 1-2 years with mammography alone or mammography and annual clinical breast exam (CBE). • There is insufficient evidence to recommend for or against the use of CBE screening alone for breast cancer. • There is insufficient evidence to recommend for or against teaching or performing routine breast self-examination.

Source*: Adapted from U.S. Preventive Services Task Force *Guide to Clinical Preventive Services, 2nd Edition (1996) and 3rd Edition* (2002) http://www.ahcpr.gov/clinic/uspstfix.htm .

Costs and Prevention

Cancer is the leading cause of premature mortality (before age 75) in the United States and Connecticut (Centers for Disease Control and Prevention 2003). The overall cost of cancer in the United States in 2001 was estimated at $156.7 billion, including $56 billion for direct medical costs and $100.3 billion for indirect costs, such as lost productivity due to illness and premature death (American Cancer Society 2002a). It has been estimated that the overall cost of cancer in Connecticut is approximately $1.3 billion, or more than $400 per person (Adams 2000).

Appropriate screening is a key strategy for cancer prevention. Expert panels in various national organizations, such as the American Cancer Society, the National Cancer Institute, and the American College of Physicians, have issued recommendations for selective cancer screening. Recommendations by these groups vary somewhat and are based on differing interpretations of the

research regarding the effectiveness of screening. In this report, we discuss specific screening recommendations in each of the various cancer sections.

The U.S. Preventive Services Task Force (USPSTF) is an independent panel of experts in primary care and prevention convened by the U.S. Public Health Service, which reviews evidence of effectiveness and develops recommendations for clinical preventive services. USPSTF recommendations for lung, prostate, breast, and colorectal cancer screening are outlined in Table 6. 4.

The following chapters describe the mortality, risk factors, and recommended preventive measures for colorectal cancer, female breast, and prostate cancers.

Lung & Other Respiratory Cancer
(ICD-9 codes 160.0-165.9)

Lung and other respiratory cancer (hereafter referred to as lung cancer) is the leading cause of cancer deaths in Connecticut and the U.S. accounting for 27% of all cancer deaths among Connecticut residents between 1989 and 1998.

Death and premature mortality rates from lung cancer are significantly higher in men than in women. In 1996-1998, males had 1.7 times the death and 1.4 times the premature mortality rates compared with females in Connecticut. Among racial/ethnic and gender subgroups, black males had the highest death rates due to lung cancer. Black males had 1.4 times the death and 1.7 times the premature mortality rate compared with white males (p < .001 for both comparisons). White males had 2.5 times the death and 2.1 times the premature mortality rate of Hispanic males (p < .001 for both comparisons). There were no significant differences between the death and premature mortality rates of black compared with white females; however, white females had 3.1 times the death and 3.8 times the premature mortality rate of Hispanic females during this period (p < .001 for both comparisons). There were insufficient deaths among Asian and Pacific Islanders and Native American males and females during this period to calculate reliable rates (Table 7.1).

Trends over time indicate that male death and premature mortality rates from lung cancer decreased significantly while the female death rate increased significantly from 1989-1991 to 1996-1998 (Table 7.1). Male mortality decreased by 1.5% per year (p < .001), while female mortality increased 1.6% per year (p < .001) from 1989 to 1998. The decline in the male death rate is largely accounted for by a decrease in the white male death rate, while the decline in premature mortality is accounted for by decreases for both white and black males from 1989-1991 to 1996-1998. The increase in the female death rate is accounted for by an increase in the white female death rate (Table 7.1). The decline in male death rates is probably due to decreased smoking prevalence among males

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1996-1998 Lung Cancer Deaths, Connecticut Residents

- Males had significantly higher death and premature mortality rates than females

- Ratio of male to female mortality - 1.7 : 1.0

- Ratio of male to female premature mortality - 1.4 : 1.0

- Black males had the highest death and premature mortality rates

- Male mortality decreased significantly since the 1989-1991 period

- Female mortality increased significantly since the 1989-1991 period

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Table 7.1. Lung and Other Respiratory Cancer Deaths[1]
Connecticut Residents by Gender, Race and Ethnicity[2], 1996-1998

| Group | Number of Deaths | Age-Adjusted Mortality Rates[3] | | Age-Adjusted Premature Mortality Rates to Age 75[3] | |
		AAMR[4]	Change since 1989-91[5]	YPLL[4]	Change since 1989-91[5]
All Residents	5,787	54.4	ns	415.0	↓↓
All males	3,233	72.7	↓↓	493.4	↓↓↓
White	2,998	71.7	↓↓	476.9	↓↓
Black	221	100.7***	ns	790.6***	↓
Asian PI	11	—		—	
Native American	3	—		—	
Hispanic	40	28.2***	ns	229.4***	ns
All females	2,554	42.0	↑↑	344.6	ns
White	2,420	42.6	↑↑	346.1	ns
Black	126	37.9	ns	367.0	ns
Asian PI	7	—		—	
Native American	1	—		—	
Hispanic	25	13.6***	ns	91.0***	ns

Notes:

1. This cause of death category includes ICD-9 codes 160-165.

2. Racial groupings (White, Black, Asian & Pacific Islander, Native American) include persons of Hispanic ethnicity.

3. Age-adjusted Mortality Rates (AAMR) and Years of Potential Life Lost (YPLL) rates are per 100,000 based on race and ethnicity specific population estimates. Age-adjusted rates were calculated by the direct method using the 2000 U.S. standard population. Rates were not calculated for fewer than 15 events.

4. Statistical tests were conducted to evaluate differences in rates between race/ethnic groups. The white population serves as the reference group in each comparison (black vs. white, Asian & PI vs. white, Native American vs. white, Hispanic vs. white). Following are explanations of the notations:

     *** Significantly different from the respective white resident rate at $p < .001$.
     — Rate was not calculated due to small numbers.

5. Statistical tests were conducted to evaluate changes in rates over time. Comparisons of 1996-98 vs. 1989-91 rates are made within each race/ethnicity group. Following are explanations of the notations:

     ↑↑ 1996-98 rate is significantly higher than the 1989-91 rate at $p < .01$.
     ↓   1996-98 rate is significantly lower than the 1989-91 rate at $p < .05$.
     ↓↓ 1996-98 rate is significantly lower than the 1989-91 rate at $p < .01$.
     ↓↓↓ 1996-98 rate is significantly lower than the 1989-91 rate at $p < .001$.
     ns Indicates the change from 1989-91 to 1996-98 is not statistically significant.

since 1965. Since 1965, female smoking rates nationwide have declined but not as dramatically as among males. As a consequence, female lung cancer mortality is not expected to decline soon (U.S. Department of Health and Human Services 2001a; Wingo, Ries, Rosenberg, et al. 1998).

The lung cancer mortality rate nationwide increased sevenfold from 1940 to 1989. Beginning in the 1980s, the rate of increase for all Americans slowed, and then started to decline (Brownson, Reif, Alavanja, et al. 1998). Between 1990 to 1998, the age-adjusted lung cancer death rate nationwide declined by 7% (Keppel, Pearcy, and Wagener 2003), which is accounted for by the declining rate among males. Beginning in the 1960s, female lung cancer mortality increased sharply and still continues to increase. Female mortality rates are not expected to decline until the year 2010 (Brownson, Reif, Alavanja, et al. 1998).

From 1989 to 1998, Connecticut male lung cancer death rates were significantly lower than comparable U.S. rates, while Connecticut female lung cancer death rates were not significantly different from comparable U.S. rates (Figure 7.1 and Table 7.2). *Healthy People 2000* and *Healthy Connecticut 2000* set objectives for lung and bronchus cancer, which constitute about 96% of all

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Table 7.2.

Lung Cancer & Other Respiratory Cancer Age-Adjusted Death Rates[1], Comparison of CT with US - 1989 and 1998

	1989	1998	1998 CT AAMR Comparison
CT AAMR*	36.8	34.1	
US AAMR*	40.7	38.3	CT rate < US rate

Lung & Bronchus Cancer Age-Adjusted Death Rates[2], Comparison of CT with US - 1989 and 1998

	1989	1998	1998 CT AAMR Comparison
CT AAMR*	35.7	32.8	
US AAMR*	39.3	36.9	CT rate < US rate
Healthy People 2000*	42.0	42.0	achieved *HP* target
Healthy CT 2000*	42.0	42.0	achieved *Healthy CT* target

* age-adjusted mortality rates are per 100,000 population, U.S. 1940 standard million population.
[1] Includes ICD-9 codes 160.0-165.9.
[2] Includes ICD-9 codes 162.2-162.9.

Connecticut resident lung and other respiratory cancer deaths. The Connecticut lung cancer mortality rate for the period 1989 through 1998 was significantly lower than the *Healthy People 2000* and *Healthy Connecticut 2000* target objectives for this period (Table 7.2).

Racial and ethnic differences in Connecticut's lung and bronchus cancer mortality rates parallel 1998 national figures which show that mortality rates were higher for blacks and whites than for other racial/ethnic groups (Centers for Disease Control and Prevention 2002).

Figure 7.1.

Hispanics had the lowest age-adjusted mortality rates of all racial/ethnic groups nationwide, followed by Asian and Pacific Islanders, and Native Americans, in both 1990 and 1998 (Keppel, Pearcy, and Wagener 2002).

Age-specific lung cancer death rates for Connecticut males and females for the period 1996-1998 are displayed in Figure 7.2. Mortality rates for males and females, contrasted with proportionally adjusted rates for all other causes of death, show a pattern of lower rates in the younger age groups, higher rates beginning in middle-age (ages 50-54 for males and 40-44 for females), and slightly lower rates in the oldest age groups (80 and older). Lung cancer mortality rates tend to increase with age, with the highest rates found in the 80-84 year old age group. Seventy- three percent of lung cancer deaths occurred among Connecticut residents aged 65 and older during the 1996-1998 period. Time trend analyses by age group indicate that the lung cancer death rate for females aged 80-84 increased significantly from 1989 to 1998 while it did not change significantly for other gender/age groups in the population.

Figure 7.2.

Age-specific Rates for Lung and Other Respiratory Cancer Deaths
and Proportionally Adjusted Rates for All Other Causes, 1996 - 1998
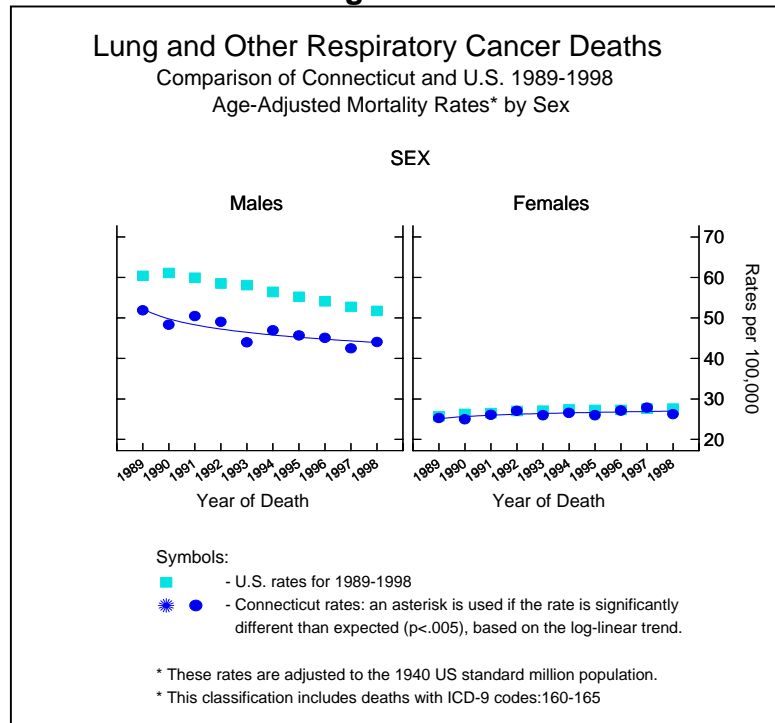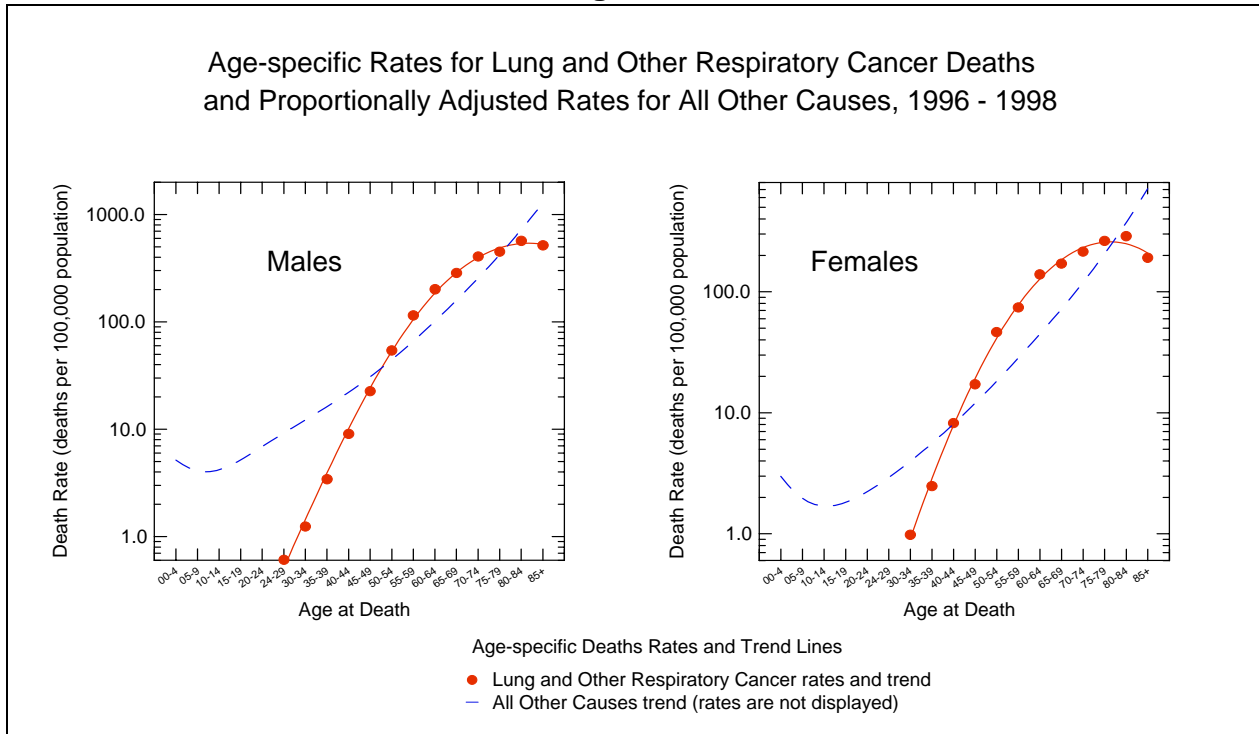
Incidence

Trend data from the Connecticut Tumor Registry for 1980-1984 to 1990-1994 indicate that lung cancer incidence increased for Connecticut females but declined for Connecticut male residents, reflecting trends in smoking (Polednak 1994). For the period 1990-1995, lung cancer incidence rates were highest among black males followed by white and Hispanic male residents. Black and Hispanic males had 1.4 and 0.7 times the incidence rate of white males in Connecticut. Age-standardized lung cancer incidence rates for black and Hispanic females were 20% and 60% lower, respectively, than those of white female residents (Polednak 1999a). These figures mirror national trends. From 1988-1992, age-adjusted lung cancer incidence rates for males were highest among blacks followed by whites, Hispanics and Native Americans. Vietnamese, Alaska Native, and Hawaiian males had rates similar to white, whereas Japanese, Chinese, Filipino, and Korean males had rates similar to Hispanic males. (Rates were approximately two to three times higher for males in each racial/ethnic group compared with females). Among females, Alaska Natives had the highest rates, followed by white, black, Hawaiian, and Vietnamese who all had comparable rates, followed by Hispanic, Korean, Filipino, and Chinese females, all with comparable rates (Miller, Kolonel, and Bernstein, et al. 1996).

Risk Factors

Modifiable risk factors for lung cancer are identified in Table 7.3. Lung cancer is strongly associated with cigarette smoking, and lung cancer mortality patterns closely follow smoking trends with an approximately 30-year latency period (Weiss 1997). The causal relationship between smoking and lung cancer has been well established by research studies, which are summarized in the U.S. Surgeon General's Reports (U.S. Department of Health, Education, and Welfare 1964; U.S. Department of Health and Human Services 1980b; 1982; 1985; 2001a). Male smokers have 10 times, and female smokers 5 times, the relative risk of developing lung cancer compared with non-smokers. It is estimated that 84% to 90% of all lung cancers are attributable to cigarette smoking and that an additional 1% to 6% are attributable to environmental tobacco smoke exposure (Brownson, Reif, Alavanja, et al. 1998).

Connecticut BRFSS and YRBSS provide prevalence estimates of behavioral risk factors linked to various cancers. Smoking prevalence rates among Connecticut adults declined about 6 percent, from 27% to 21% between 1994 and 1997. The number of adults who stop smoking annually is approximately the same as the number of young people who initiate smoking each year. Thus, smoking rates have remained stable in recent years (Adams 2000). Approximately 26% of all Connecticut high school students smoke with equivalent rates for male and female students (Lowery St. John and Jarvis 2001).

Occupational exposures also increase lung cancer risk, with an estimated 10% to 20% of all lung cancers attributable to occupational exposures. Asbestos exposure may occur in shipbuilding, cement work, railroad repair, plumbing, firefighting, and pipe fitting jobs. In 1997, an estimated 19,919 employed workers in Connecticut were potentially exposed to asbestos (Webb, Heyman, Estrada, et al. 2000). Asbestos exposure among nonsmokers increases the risk of developing lung cancer five-fold. Combined with smoking, asbestos exposure increases the risk 50-fold. A 1994 estimate suggests that 1 to 20 Connecticut resident lung cancer deaths may have resulted from asbestos exposure (Siniscalchi, Tibbetts, Mahmood, et al. 1995).

Table 7.3. Modifiable Risk Factors for Lung Cancer

Factor	Magnitude of Association[1]	Estimated Range of Population Attributable Risk (%)
Cigarette smoking	Strong	84 – 90
Occupational exposures	Strong	10 – 20
Residential radon exposure	Weak	7 – 25
Environmental tobacco smoke exposure	Weak	1 – 6
High-fat diet	Possible	–
Urban air pollution	Possible	–

Source*: Adapted from Brownson, Reif, Alavanja, et al. 1998.

1. *Strong magnitude* indicates a relative risk of greater than 4 for those persons with the risk factor compared with those not having the risk factor. *Weak magnitude* indicates a relative risk of less than 2 for those persons with the risk factor compared with those not having the risk factor. *Possible association* indicates that some, but not definitive, evidence exists to support these as risk factors for lung cancer.

Epidemiologic studies of miners established the causal relationship between radon gas and lung cancer (National Research Council 1999). Occupational radon exposure increases lung cancer risk 20-fold and can also interact with smoking to increase risk. Numerous other occupational exposures have been found to increase lung cancer risk (Brownson, Reif, Alavanja, et al. 1998).

Environmental agents found in the home, including radon, asbestos, and second-hand smoke have also been shown to increase lung cancer risk (Samet 1993; U.S. Department of Health and Human Services 2001a). Radon is estimated to contribute to about 7% to 25% of lung cancers (Brownson, Reif, Alavanja, et al. 1998). While radon exposure in the home tends to occur at lower levels than in mines, the Environmental Protection Agency (EPA) has set acceptable radon exposure levels in the home. A Connecticut home radon survey (1987-1988) found that one out of five Connecticut homes tested had radon levels above the Environmental Protection Agency (EPA) action level of 4 picocuries of radon per liter of air (4pCi/L) (Siniscalchi, Tibbetts, Beakes, et al. 1996). An estimated one-third of radon-attributable deaths could be avoided by reducing radon in the home to below the EPA action level (National Research Council 1999).

From 1991 to 1996, the Connecticut Department of Public Health conducted a school test program for radon. An initial investigation found that most of the 217 schools tested had at least one room with radon concentrations above the U.S. EPA guideline (Siniscalchi, Tibbetts, Soto, et al. 1996). Researchers also found that there were seasonal variations in radon concentrations in both air and water samples, suggesting that such fluctuations should be monitored so that neither undetected exposure to high levels of radon nor unnecessary expenses for radon abatement be made (Siniscalchi, Tibbetts, Soto, et al. 1996). Since 1996, the Connecticut Department of Public Health has offered radon testing devices and technical assistance to municipalities wishing to test their schools. Researchers estimated that annual lung cancer mortality due to long-term radon exposure may account for 80 to 143 deaths in Connecticut per year (Siniscalchi, Tibbetts, Beakes, et al. 1996).

Recent findings from a national prospective study from 1982 through 1998 suggest that long-term exposures to combustion-related fine particulate air pollution (particulate matter-2.5 or $PM_{2.5}$) and sulfur oxide pollution (sulfate particles and/or sulfur dioxide) increase the risk of lung cancer mortality. Researchers found that each unit elevation in fine particulate air pollution was associated with an 8% increased risk of lung cancer mortality. Weaker, less consistent associations were found for air pollution for PM_{10} (particulate matter-10) and lung cancer mortality (Pope, Burnett, Thun, et al. 2002). The Environmental Protection Agency identifies areas of the U.S. as "non-attainment areas" for major pollutants such as sulfur dioxide and PM_{10}. Parts of New Haven County are identified as a "moderate non-attainment area" for PM_{10}. No other areas of Connecticut are currently identified as non-attainment areas for either PM_{10} or sulfur dioxide (U.S. Environmental Protection Agency 2002).

Studies suggest that a diet low in fruits and vegetables increases lung cancer risk. Some studies had suggested that the protective effects of fruits and vegetables on lung cancer risk were due to beta-carotene; however, these observations have not been borne out in a clinical trial of beta-carotene supplementation and lung cancer mortality (Omenn, Goodman, Thronquist, et al. 1996). In addition

to increased fruit and vegetable consumption, decreased consumption of animal fats may also decrease lung cancer risk (Brownson, Reif, Alavanja, et al. 1998).

Epidemiological evidence from industrialized countries suggests that lung cancer mortality is inversely related to socioeconomic status. Social class differences in smoking patterns (smoking behavior is more common among persons of lower social class) probably explain some of the social class differences in lung cancer mortality. There are, however, many other risks, such as hazardous occupations, home exposures to cancer-causing agents, poor diet, and limited access to health care, which may account for some of the lung cancer mortality differential between social classes (Stellman and Resnicow 1997).

Prevention

Lung cancer prevention efforts focus on smoking cessation for those who smoke, avoiding second-hand smoke for non-smokers, and discouraging young Americans from adopting the smoking habit. The 2000 Surgeon General's report, *Reducing Tobacco Use*, provides evidence that certain kinds of interventions—educational, clinical, regulatory, economic, and comprehensive—can significantly reduce tobacco use. It furthermore suggests that tobacco use rates could decrease by 50% if its recommendations were implemented (Centers for Disease Control and Prevention 2000b). Statewide tobacco prevention programs in California, Massachusetts, and Florida have demonstrated that comprehensive education efforts can reduce tobacco use. Key components of successful comprehensive programs include public education efforts, community and school-based programs, smoking cessation efforts, and strict enforcement of laws restricting youth access to tobacco and establishing smoke-free areas (Campaign for Tobacco-Free Kids, American Cancer Society, American Heart Association, and American Lung Association 2002).

In 1998, Connecticut and 45 other states reached $246 billion in legal settlements with the tobacco industry for recovery of states' tobacco-related Medicaid health care costs. This settlement provided Connecticut and other states with an unprecedented opportunity to reduce the burden of tobacco smoking on their local communities.

Connecticut currently ranks 45 out of 51 (50 states and Washington, D.C.) in funding tobacco prevention. Connecticut received $260.4 million in tobacco settlement payments through December, 2001. Its current annual funding for tobacco prevention is about $2.5 million ($580,000 in state tobacco settlement and $2,000,000 in other federal and state funds) (Connecticut Department of Public Health, Bureau of Community Health, personal communication 2002). This is well below the Centers for Disease Control and Prevention's (CDC) recommendation for Connecticut of $21.2 to $53.9 million in annual spending for a comprehensive tobacco prevention program (Campaign for Tobacco-Free Kids, American Cancer Society, American Heart Association, and American Lung Asociation 2002; Centers for Disease Control and Prevention 2001e).

Colorectal Cancer
(ICD-9 codes 153.0-154.3, 154.8, 159.0)

Colorectal cancer was the second leading cause of cancer death for males and the third leading cause of cancer death for females in Connecticut between 1989 and 1998. It accounted for 11% of all cancer deaths among Connecticut residents during this time period. Nationwide, it accounts for about 10% of all cancer deaths (Brownson, Reif, Alavanja, et al. 1998).

Death and premature mortality rates from colorectal cancer are significantly higher in men than in women in Connecticut. In 1996-1998, Connecticut males had 1.4 times the age-adjusted colorectal death and premature mortality rates compared with females. This parallels 1998 national figures, which show that males also had about 1.4 times the age-adjusted colorectal cancer death rate of females. There were no significant differences in colorectal cancer mortality rates among racial/ethnic and gender subgroups, although black males and females had significantly higher premature mortality rates compared with white males and females, respectively, in Connecticut. There were insufficient deaths among Asian and Pacific Islanders and Native American males and females and Hispanic females during this period to calculate reliable rates (Table 8.1).

Age-adjusted colorectal death rates decreased significantly from 1989-1991 to 1996-1998, which is accounted for by the decreases in white male and female death rates. Premature mortality due to colorectal cancer decreased during this period as well, a change accounted for by the decrease in premature mortality among white female residents.

Connecticut resident death rates for colorectal cancer did not differ significantly from U.S. rates during most of the decade, although the 1998 Connecticut resident death rate was significantly lower than the U.S. rate. While the 1998 Connecticut male death rate was significantly lower than the comparable U.S. rate, the Connecticut female death rate was not significantly different from the comparable U.S. rate for 1998. Connecticut resident death rates for 1996 and 1998 were significantly lower than the *Healthy People 2000* target objective. There is no *Healthy Connecticut 2000* objective for colorectal cancer (Table 8.2, Figure 8.1).

1996-1998 Colorectal Cancer Deaths, Connecticut Residents

- Males had significantly higher death and premature mortality rates than females

- Ratio of male to female mortality - 1.4 : 1.0

- Ratio of male to female premature mortality - 1.4 : 1.0

- Hispanics had significantly lower death and premature mortality rates compared with whites

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Table 8.1. Colorectal Cancer Deaths[1]
Connecticut Residents by Gender, Race and Ethnicity[2], 1996-1998

Group	Number of Deaths	Age-Adjusted Mortality Rates[3]		Age-Adjusted Premature Mortality Rates to Age 75[3]	
		AAMR[4]	Change since 1989-91[5]	YPLL[4]	Change since 1989-91[5]
All Residents	2,237	20.6	↓↓↓	123.2	↓
All males	1,068	24.8	↓↓↓	144.7	ns
White	999	24.6	↓↓↓	138.5	ns
Black	64	27.3	ns	255.5*	ns
Asian PI	5	—		—	
Native American	0				
Hispanic	25	17.4	na	142.2	na
All females	1,169	17.8	↓↓↓	103.7	ns
White	1,097	17.5	↓↓↓	96.1	↓
Black	65	19.9	ns	185.5*	ns
Asian PI	7	—		—	
Native American	0				
Hispanic	15	—		—	

Notes:

1. This cause of death category includes ICD-9 codes 153.0-154.3,154.8,159.0.

2. Racial groupings (White, Black, Asian & Pacific Islander, Native American) include persons of Hispanic ethnicity.

3. Age-adjusted Mortality Rates (AAMR) and Years of Potential Life Lost (YPLL) rates are per 100,000 based on race and ethnicity specific population estimates. Age-adjusted rates were calculated by the direct method using the 2000 U.S. standard population. Rates were not calculated for fewer than 15 events.

4. Statistical tests were conducted to evaluate differences in rates between race/ethnic groups. The white population serves as the reference group in each comparison (black vs. white, Asian & PI vs. white, Native American vs. white, Hispanic vs. white). Following are explanations of the notations:

    *   Significantly different from the respective white resident rate at $p < .05$.
    — Rate was not calculated due to small numbers.

5. Statistical tests were conducted to evaluate changes in rates over time. Comparisons of 1996-98 vs. 1989-91 rates are made within each race/ethnicity group. Following are explanations of the notations:

    ↓   1996-98 rate is significantly lower than the 1989-91 rate at $p < .05$.
    ↓↓↓ 1996-98 rate is significantly lower than the 1989-91 rate at $p < .001$.
    ns Indicates the change from 1989-91 to 1996-98 is not statistically significant.
    na 1989-91 rate was not calculated due to small numbers and so no comparison with 1996-98 rate is available

**Table 8.2. Colorectal Cancer Age-Adjusted Death Rates,
Comparison of CT with US - 1989 and 1998**

	1989	1998	1998 Comparison
CT AAMR*	14.4	10.9	
US AAMR*	14.0	12.0	CT rate < US rate
Healthy People 2000*	13.2	13.2	CT rate < *HP* target

* age-adjusted mortality rates are per 100,000 population, U.S. 1940 standard million population.

Age-specific colorectal cancer death rates for Connecticut males and females for the period 1996-1998 are displayed in Figure 8.2. Mortality rates for males and females, contrasted with proportionally adjusted rates for all other causes of death, show a pattern of lower rates in the younger age groups, higher rates beginning in middle-age (ages 50-54 for males and females), and slightly lower rates in the oldest age group (85 and older). Colorectal cancer mortality rates tend to increase with increasing age, with the highest rates found in the 85 and older age group. Eighty-one percent of colorectal cancer deaths occurred among Connecticut residents aged 65 and older during the 1996-1998 period. Time trend analyses did not show any significant changes in the colorectal cancer death rate by age group for males or females.

Racial and ethnic differences in Connecticut's colorectal cancer mortality rates parallel 1998 national figures which show that mortality rates were highest for black, followed by white residents (Centers for Disease Control and Prevention 2002).

From 1973 to 1990, colorectal cancer mortality nationwide decreased among white Americans but increased among black Americans (Brownson, Reif, Alavanja, et al. 1998). Between 1990 and 1998, colorectal cancer death rates declined for all Americans; however, the rate of decrease was about 50% less for black than for white Americans (Centers for Disease Control and Prevention 2002). The decline in colorectal cancer mortality rates over the past 30 years is probably accounted for by decreased incidence and increased survival rates (American Cancer Society 1999), and may be related to the increased use of screening tests for the disease (Brownson, Reif, Alavanja, et al. 1998).

Other factors that may have contributed to the decrease in colorectal cancer incidence include dietary changes (such as increased intake of vegetables and other food fibers and decreased saturated fat and alcohol intake), increased physical activity, and increased consumption of nonsteroidal anti-inflammatory drugs (NAID) such as aspirin (Ries, Wingo, Miller, et al 2000).

Risk Factors

While the etiology of the disease is not well understood, some risk factors for colorectal cancer have been identified. Colorectal cancer incidence increases dramatically after age 50. Familial characteristics associated with increased risk for colorectal cancer include a first-degree family relative with the disease, a family history of multiple adenomatous polyps, which can elevate risk at younger ages, as well as a familial gene that has been recently identified for colorectal cancer. Persons with inflammatory bowel disease are considered at risk (Brownson, Reif, Alavanja, et al. 1998).

Figure 8.1.

Colorectal Cancer Deaths
Comparison of Connecticut and U.S. 1989-1998
Age-Adjusted Mortality Rates* by Sex

Symbols:
- U.S. rates for 1989-1998
- Connecticut rates: an asterisk is used if the rate is significantly different than expected (p<.005), based on the log-linear trend.

* These rates are adjusted to the 1940 US standard million population.
* This classification includes deaths with ICD-9 codes:153.0-154.3,154.8,159.

Epidemiological studies support the association between a diet high in saturated fat, low in vegetables, and low in high-fiber grains and increased risk of colorectal cancer. Evidence is also growing for lack of physical activity as a risk factor for colorectal cancer. It is estimated that up to 25% of colorectal cancer is attributable to a diet high in saturated fat, as much as 35% to a diet low in fruits and vegetables, and about 32% to physical inactivity (Table 8.3) (Brownson, Reif, Alavanja, et al. 1998). It has been estimated that the colorectal cancer incidence rate could be reduced by as much as 75% with changes in diet and lifestyle in the population (World Cancer Research Fund, American Institute for Cancer Research 1997).

Incidence

The age-standardized incidence rates of colon and rectal cancer for all Connecticut residents declined after 1980-1984 (Polednak 2001a). In the 1995-1998 period, males had 1.5 times the age-standardized incidence rate of rectal cancer and 1.3 times the colon cancer incidence rate compared with females. Among males, incidence rates of colon cancer were highest for black, followed by white, and Hispanic males (1990-1995 period). Blacks had 1.2 times, and Hispanics 0.7 times the colon cancer incidence rate of white males. White males had the highest incidence rate of rectal cancer, followed by blacks and Hispanics, with rates that were 30% lower than those of whites. Among Connecticut females, colon cancer incidence rates were highest among black, followed by white, and Hispanic females. Blacks had 1.2 times and Hispanics 0.8 times the colon cancer

Figure 8.2.

Age-specific Rates for Colorectal Cancer Deaths
and Proportionally Adjusted Rates for All Other Causes, 1996 - 1998
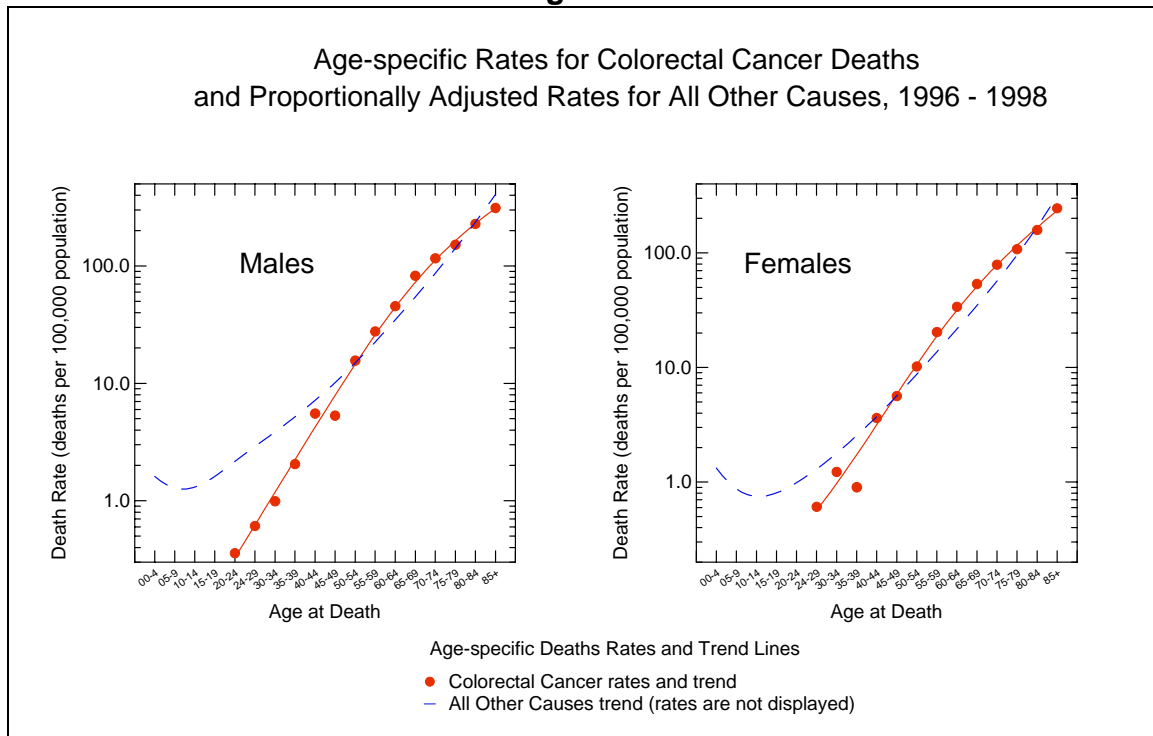
incidence rate of white females. White, black, and Hispanic females had equivalent incidence rates for rectal cancer (Polednak 1999a). Nationwide colorectal cancer incidence rates decreased from 1985 to 1995, and remained unchanged (or increased slightly in women, possibly due to increased screening) through 1998. Among racial and ethnic subgroups, incidence rates were highest in blacks, followed by white, Asian and Pacific Islanders, and Native Americans and Hispanics in the 1992-1998 period (American Cancer Society 2002a).

Evidence from a national prospective study suggests that obesity may increase the risk of colon cancer death, and that the strength of this relationship is greater for men than for women (Murphy, Calle, Rodriguez, et al. 2000). Aspirin and other non-steroidal anti-inflammatory drugs (Thun, M.J., M. Namboodiri, and C.W. Health. 1991; Saha, Roman, and Beauchamp 2002; Gwyn and Sinicrope 2002; Jolly, Cheng, and Langman 2002) and the removal of adenomatous polyps detected through colorectal screening may reduce the incidence of colorectal cancer (Ries, Wingo, Miller, et al. 2000).

Colorectal cancer incidence has not been consistently linked to measures of socioeconomic status (Krieger, Quesenberry, Peng, et al. 1999). Certain occupational exposures, however, may be linked to colorectal cancers (Spiegelman and Wegman 1985). Evidence from a retrospective study in Sweden suggests that occupation may play a small role in colon cancer, with the primary causal factor being physical inactivity of the job (Chow, Malker, Hsing, et al. 1994).

Table 8.3. Modifiable Risk Factors for Colorectal Cancer

Factor	Magnitude of Association[1]	Estimated Range of Population Attributable Risk (%)
High-fat diet	Weak	15 – 25
Low-vegetable diet	Weak	25 – 35
Physical inactivity	Weak	32
Alcohol consumption	Possible	–
Occupation	Possible	–
Aspirin use	Possible	–
Obesity	Possible	–

Source*:* Adapted from Brownson, Reif, Alavanja, et al. 1998.

1. *Weak magnitude* indicates a relative risk of less than 2 for those persons with the risk factor compared with those not having the risk factor. *Possible association* indicates that some, but not definitive, evidence exists to support these as risk factors for colorectal cancer.

Socioeconomic factors, other than occupation, may play a role in colorectal cancer survival. There is some evidence that health insurance status and type of coverage may influence cancer outcomes. One study found that uninsured persons were significantly more likely to be diagnosed at a late stage for colorectal cancer compared with commercial indemnity insured persons (Roetzheim, Pal, Tennant, et al. 1999). Another study found that among non-Medicare patients, uninsured, Medicaid, and commercial HMO patients all had higher adjusted risks of colorectal cancer death relative to those with commercial fee-for-service insurance (Roetzheim, Pal, Gonzalez, et al. 2000). A study of Connecticut residents found reduced colorectal cancer survival rates in persons living in higher poverty areas, independent of age, stage at diagnosis, and comorbidity (Polednak 2001b). Additional study of such factors and their impact on colorectal cancer survival is warranted.

Prevention

Early detection and treatment of colorectal cancer in its earliest stages and precancerous polyps, which can be present for years before invasive cancer develops, are possible through colorectal cancer screening. Although different screening methods are readily available, colorectal cancer screening is not used widely (American Cancer Society 2002b).

Screening recommendations of the U.S. Preventive Services Task Force (USPSTF) for persons aged 50 and older include a fecal occult blood testing periodically or sigmoidoscopy, or both in combination. Good evidence exists that both reduce colorectal cancer mortality. USPSTF also recommends that persons considered at high-risk because of family history (e.g. a first degree relative diagnosed with colorectal cancer before age 60) consider beginning screening at a younger age. The USPSTF states that there is no direct evidence that routine screening by double-contrast barium enema or colonoscopy reduces colorectal cancer mortality (U.S. Preventive Services Task Force 2002).

American Cancer Society guidelines (ACS) differ somewhat, recommending that persons aged 50 and over have a fecal occult blood test every year and a flexible sigmoidoscopy and digital rectal

exam simultaneously every five years. In 1997, only 24% of Connecticut adults aged 50 and older reported having a blood stool test within the past year, and 35% reported having a sigmoidoscopy within the past five years (Adams 2000).

The American Cancer Society's dietary guidelines recommend consuming foods from plant sources (5 servings per day of fruits and vegetables) and limiting the intake of high fat foods. Over two-thirds of Connecticut adults (1996-1997) and students (1997) reported consuming less than five servings of fruits and vegetables per day. Eighty-three percent of adults and about 67% of students reported consuming two or less servings of high fat foods per day (Adams 2000).

Female Breast Cancer
(ICD-9 code 174)

Breast cancer was the second leading cause of cancer death for females in Connecticut between 1989 and 1998, and the leading cause of premature mortality due to cancer. It accounted for 9% of cancer deaths among all Connecticut residents during this time period. Black females had the highest death and premature mortality rates due to breast cancer, followed by white and Hispanic females. Black females had significantly higher death and premature mortality rates, and Hispanic females had significantly lower breast cancer death rates compared with white females for the 1996-1998 period. There were insufficient numbers of deaths among Asian and Pacific Islander and Native American females during this period to calculate reliable rates.

Female breast cancer mortality decreased by 1.4% per year (p < .01) from 1989 to 1998. There was a significant decrease in age-adjusted death and premature mortality rates for breast cancer between 1989-1991 and 1996-1998, which is accounted for by the decreasing rates among white females in Connecticut (Table 9.1).   These declines may be attributable to earlier detection through breast cancer screening and improvements in treatment modalities (Polednak 1999b).

Connecticut female breast cancer death rates were not significantly different from comparable U.S. rates from 1996 to 1998 (Figure 9.1). The Connecticut death rate was significantly lower than the *Healthy Connecticut 2000* objective but not significantly different from the U.S. *Healthy People 2000* objective for breast cancer (Table 9.2).

Age-specific breast cancer death rates for Connecticut females for the period 1996-1998 are displayed in Figure 9.2. Breast cancer mortality rates, contrasted with proportionally adjusted rates for all other causes of death, show a pattern of lower rates in the younger age groups, higher rates beginning in middle-age (ages 35-39), and lower rates in the oldest age groups (75 and older). Breast cancer mortality rates tend to increase with increasing age, with the highest rates found in the 85-and-over age group. Sixty-three percent of deaths from breast cancer occurred among Connecticut residents aged 65 and older during this period. Time trend analyses indicate that the breast cancer death rate did not change significantly within any age group in the Connecticut female population.

1996-1998 Female Breast Cancer Deaths, Connecticut Residents

- The second leading cause of cancer deaths among women

- Black females had the highest death and premature mortality rates

- Ratio of black to white mortality – 1.3 : 1.0

- Ratio of black to white premature mortality – 1.4 : 1.0

- Significant decrease in death and premature mortality rates since the 1989-91 period

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Table 9.1. Female Breast Cancer Deaths[1]
Connecticut Residents by Gender, Race and Ethnicity[2], 1996-98

| Group | Number of Deaths | Age-Adjusted Mortality Rates[3] | | Age-Adjusted Premature Mortality Rates to Age 75[3] | |
		AAMR[4]	Change since 1989-91[5]	YPLL[4]	Change since 1989-91[5]
All Residents	1,796				
All females	1,796	30.0	↓	353.4	↓
White	1,653	29.7	↓	344.0	↓
Black	135	39.5*	ns	496.2*	ns
Asian PI	5	—		—	
Native American	1	—		—	
Hispanic	33	13.2***	ns	252.9	ns

Notes:

1. This cause of death category includes ICD-9 codes 174.

2. Racial groupings (White, Black, Asian & Pacific Islander, Native American) include persons of Hispanic ethnicity.

3. Age-adjusted Mortality Rates (AAMR) and Years of Potential Life Lost (YPLL) rates are per 100,000 based on race and ethnicity specific population estimates. Age-adjusted rates were calculated by the direct method using the 2000 U.S. standard population. Rates were not calculated for fewer than 15 events.

4. Statistical tests were conducted to evaluate differences in rates between race/ethnic groups. The white population serves as the reference group in each comparison (black vs. white, Asian & PI vs. white, Native American vs. white, Hispanic vs. white). Following are explanations of the notations:

> *   Significantly different from the respective white resident rate at $p < .05$.
> *** Significantly different from the respective white resident rate at $p < .001$.
> —   Rate was not calculated due to small numbers.

5. Statistical tests were conducted to evaluate changes in rates over time. Comparisons of 1996-98 vs. 1989-91 rates are made within each race/ethnicity group. Following are explanations of the notations:

> ↓   1996-98 rate is significantly lower than the 1989-91 rate at $p < .05$.
> ns   Indicates the change from 1989-91 to 1996-98 is not statistically significant.

Between 1973 and 1990, breast cancer mortality rate nationwide increased 2% among females overall and 21% among black females. Breast cancer incidence rates also increased between 1973 and 1990, a trend that is attributable, in part, to increased use of mammography screening (Brownson, Reif, Alavanja, et al. 1998). Between 1990 and 1998, the age-adjusted breast cancer mortality rate nationwide declined by 18% for all females, 19% for white women, 14% for Hispanic women, and 4% for black women. Rates for Asian and Pacific Islander and Native American females did not change significantly (Keppel, Pearcy, and Wagener 2002).

**Table 9.2. Female Breast Cancer Age-Adjusted Death Rates,
Comparison of CT with US - 1989 and 1998**

	1989	1998	1998 CT AAMR Comparison
CT AAMR*	24.0	19.7	
US AAMR*	23.1	18.8	not significantly different
Healthy People 2000*	20.6	20.6	not significantly different
Healthy CT 2000*	23.1	23.1	achieved *Healthy CT* target

* age-adjusted mortality rates are per 100,000 population, U.S. 1940 standard million population.

Incidence

Between 1992 and 1998, age-adjusted breast cancer incidence and mortality rates of Hispanic, Asian and Pacific Islander, and Native American women nationwide were similar to each other but lower than those of black and white women. Black women tend to be diagnosed with breast cancer at a later stage of the disease when five-year survival is less likely. They also have lower survival rates for the same stage as white women (Ries, Eisner, Kosary et al. 2001). There is evidence of progress, however, in the black-white disparity in breast cancer diagnosis and survival. A study of trends in late-stage diagnosis for breast cancer for black and white women from 1988 to 1995 in the National Cancer Institute's Surveillance, Epidemiology and End Results (SEER) Program revealed that proportions of breast cancers diagnosed at the late stage declined for both groups, and that the black-white disparity in late-stage diagnosis declined (Polednak 2000).

From 1990-1994 to 1995-1998, the age-standardized female breast cancer incidence rates in Connecticut increased slightly, which may be largely attributable to increased screening (Polednak 2001a). Breast cancer incidence rates were higher for white, than for black and Hispanic females for the period 1990-1995 (Polednak 1999b).

Risk Factors

Non-modifiable risk factors for female breast cancer include increasing age, early age of menarche (< 12 years), later age at menopause (≥ 55), family history of a first- or second-degree relative with breast cancer, and the presence of a susceptibility gene, such as BRCA1 or BRCA2. Women who possess mutations in either of these genes are estimated to have a 60% to 85% lifetime risk of developing breast cancer (Armstrong, Eisen, and Weber 2000). An estimated 5% to 10% of all breast cancers are attributable to inherited mutations in susceptibility genes like BRCA1 and BRCA2 (American Cancer Society 2002c).

International data suggest that, in general, female breast cancer incidence and mortality rates tend to increase on a social class gradient (Faggiano, Partanen, Kogevinas, et al. 1997). The U.S. Longitudinal Mortality Study demonstrated that among the white female population, breast cancer mortality rates increased with increasing level of education (Rogot, Sorlie, Johnson, et al. 1992). Lack of health insurance, however, is associated with decreased survival among women breast cancer, and low-income breast cancer patients have a lower 5-year survival rate than higher-income patients (American Cancer Society 2002c).

Epidemiological studies have identified several modifiable risk factors for breast cancer (Table 9.3). Few of these risk factors, however, are strongly associated with development of the disease, and each one accounts for a relatively small proportion of overall breast cancer incidence. Reproductive risk factors—nulliparity, older age at first birth, never having breast-fed a child, early menarche, and late menopause—are related to hormonal exposures.

Moderate or heavy alcohol consumption, use of oral contraceptives, and use of estrogen replacement during menopause may be associated with increased risk for the disease (Brownson, Reif, Alavanja, et al. 1998). Alcohol is the one dietary factor associated with increased risk of breast cancer. It is estimated that two alcoholic drinks per day may increase breast cancer risk by 25% (American Cancer Society 2002c). Recent use of oral contraceptives may increase breast cancer risk

Figure 9.1.

Female Breast Cancer Deaths
Comparison of Connecticut and U.S. 1989-1998
Age-Adjusted Mortality Rates*, Females Only

Symbols:
■ - U.S. rates for 1989-1998
✳ ● - Connecticut rates: an asterisk is used if the rate is significantly different than expected (p<.005), based on the log-linear trend

* These rates are adjusted to the 1940 US standard million population.
* This classification includes deaths with ICD-9 codes:174

slightly; but those who stopped use for ten years or more have equivalent risk as those women who have never used oral contraceptives. Recent use of estrogen replacement therapy for more than five years is also associated with increased breast cancer risk. This effect apparently disappears five to ten years after therapy is stopped (American Cancer Society 2002c). Evidence suggests that smoking is a weak risk factor for breast cancer, and that risk is higher in the premenopausal period and in those who started smoking at an early age (Khuder, Mutgi, and Nugent 2001). Evidence for an association between pesticides, such as DDT, DDE, and dieldrin, and breast cancer risk has been mixed, and further exploration of these associations is warranted (Snedeker 2001). Use of the synthetic estrogen DES by women during the 1940's through 1960s has been associated with increased breast cancer risk, independent of family history, use of oral contraceptives or hormone replacement therapy (Titus-Ernstoff, Hatch, Hoover, et al. 2001).

Figure 9.2.

Age-specific Rates for Female Breast Cancer Deaths and
Proportionally Adjusted Rates for All Other Causes, 1996 - 1998
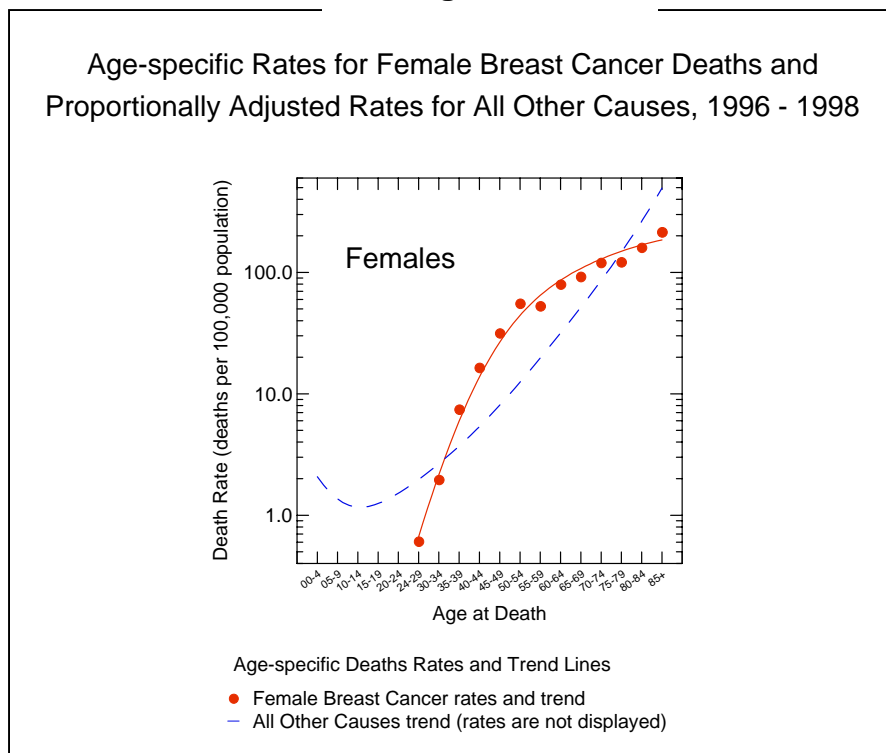
Prevention

Breast cancer prevention efforts focus on early detection of the disease. Clinical breast examination by a physician or nurse, mammography screening, and breast self-examination are the preventive measures commonly practiced. There has been some disagreement among scientific experts regarding the risks and benefits of mammography screening women in their forties, but there is evidence of effectiveness of mammography for women aged 50 to 69. Routine mammography screening for women 50 and over is estimated to reduce breast cancer mortality by about one-third (Armstrong, Eisen, and Weber 2000).

Table 9.3. Modifiable Risk Factors for Breast Cancer

Factor	Magnitude of Association[1]	Estimated Range of Population Attributable Risk (%)
Large doses of chest radiation	Moderate	1 – 3
Never having children	Weak	1– 9
First full-term pregnancy after age 30	Weak	1– 13
Oophorectomy	Weak	–
Obesity after menopause	Weak	8 – 16
Alcohol consumption	Weak	–
Physical inactivity	Possible	–
Cigarette smoking	Possible	–
Pesticide exposure	Possible	–
Lack of breast-feeding	Possible	–
Use of diethylstilbestrol	Possible	–
Recent use of oral contraceptives or Estrogen replacements	Possible	–

Source*: adapted from Brownson, Reif, Alavanja, et al. 1998.

1. Moderate magnitude indicates a relative risk of between 2 and 4 for those persons with the risk factor compared with those not having the risk factor. *Weak magnitude* indicates a relative risk of less than 2 for those persons with the risk factor compared with those not having the risk factor. *Possible association* indicates that some, but not definitive, evidence exists to support these as risk factors for breast cancer.

The U.S. Preventive Services Task Force (USPSTF) recommends that women aged 40 and older have breast cancer screening every 1-2 years with mammography alone or mammography with a clinical breast exam. The American Cancer Society recommends that women 40 and older should have an annual mammogram, and an annual clinical breast exam by a health care professional. The USPSTF states that there is insufficient evidence to recommend for or against breast self-exam or clinical breast examination, although the American Cancer Society recommends monthly breast self-exam for women aged 20 and older (U.S. Preventive Services Task Force 2002; American Cancer Society 2002c). Connecticut BRFSS estimates for 1996-1997 indicate that 81.5% of Connecticut women aged 40 and older had had a mammogram and clinical breast exam within the past year (Adams 2000).

Besides regular mammography, the American Cancer Society's recommendations for reducing risk factors for breast cancer are to increase the level of physical activity, minimize alcohol intake, and avoid obesity. The role of the various components of physical activity—that is the type of activity and its frequency, duration, and intensity—in breast cancer risk reduction are not well understood and need further investigation.   Obesity is associated with increased breast cancer risk in post-menopausal women, and maintenance of a healthy body weight at any age is important for overall health and to reduce breast cancer risk (American Cancer Society 2002c). The effect of alcohol consumption on breast cancer should be viewed in the context of its beneficial effects on cardiovascular disease.

Tamoxifen is a drug that has been commonly used as a treatment for certain breast cancers. Evidence from a clinical trial suggests that tamoxifen can reduce breast cancer risk in women who are at higher risk for developing the disease. Side effects of tomoxifen use are associated with an increase of endometrial cancer and thromboembolic events, however (Fisher, Costantino, Wickerham, et al. 1998). The U.S. Preventive Services Task Force (USPSTF) recommends against routine use of tamoxifen (and raloxifene) for primary prevention in women at average or low risk for breast cancer. Although some evidence exists that tamoxifen and raloxifene may prevent breast cancers in women, USPSTF determined that the potential harms (e.g. stroke, pulmonary embolism, and deep venous thrombosis) outweigh the benefits in women who are not at high risk for the disease. USPSTF suggests that clinicians discuss the potential benefits and harms of the therapy with women considered at high risk of breast cancer and low risk of adverse effects of chemoprevention (U.S. Preventive Services Task Force 2002).

Prostate Cancer
(ICD-9 codes 185.0-185.9)

Prostate cancer was the third leading cause of cancer death for males in Connecticut between 1989 and 1998. It accounted for 6% of cancer deaths among all Connecticut residents during this time period. Death and premature mortality rates from prostate cancer are significantly higher in black than in white male Connecticut residents. In 1996-1998, black males had 2.4 times the prostate cancer death rate and 3.3 times the premature mortality rate compared with white males in Connecticut.   There were insufficient prostate cancer deaths among Hispanic, Asian and Pacific Islanders, and Native American males during this period to calculate reliable rates (Table 10.1).

Age-adjusted mortality for prostate cancer decreased by 1.5% per year (p < .001) from 1989 to 1998 for Connecticut males. Both death and premature mortality rates decreased significantly from 1989-1991 to 1996-1998, a trend which is accounted for by decreases in the white male death and premature mortality rates (Table 10.1).

Connecticut male death rates were significantly lower than comparable U.S. rates in 1997 and 1998 (Figure 10.1 and Table 10.2). There are no *Healthy People 2000* and *Healthy Connecticut 2000* objectives set for prostate cancer.

Age-specific prostate cancer death rates for Connecticut males for the period 1996-1998 are displayed in Figure 10.3. Prostate cancer mortality rates, contrasted with proportionally adjusted rates for all other causes of death, show a pattern of lower rates in the younger age groups, with higher rates beginning in the 75-79 age group and the highest rate in the oldest age group (85 and older). Prostate cancer mortality rates tend to increase with increasing age, with the highest rates found in the 85-and-over age group. Ninety-three percent of deaths occurred among Connecticut residents aged 65 and older during the 1996-98 period. Time trend analyses indicate that the prostate cancer death rate did not change significantly for any age group in the male Connecticut resident population from 1989 to 1998.

Racial and ethnic differences in Connecticut's prostate cancer mortality rates parallel 1998 national figures, which show that age-adjusted mortality rates were highest for black males, followed by white, Hispanic, Native American, and Asian and Pacific Islander (API) males. Black males had

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1996-1998 Prostate Cancer Deaths, Connecticut Residents

- Black males had the highest death and premature mortality rates

- Ratio of black to white mortality – 2.4: 1.0

- Ratio of black to white premature mortality – 3.3: 1.0

- Significant decreases in the death and premature mortality rates since the 1989-91 period

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Table 10.1. Prostate Cancer Deaths[1]
Connecticut Residents by Gender, Race and Ethnicity[2], 1996-98

| Group | Number of Deaths | Age-Adjusted Mortality Rates[3] | | Age-Adjusted Premature Mortality Rates to Age 75[3] | |
		AAMR[4]	Change since 1989-91[5]	YPLL[4]	Change since 1989-91[5]
All Residents	1,253				
All males	1,253	31.0	↓	56.1	↓
White	1,132	29.5	↓	49.4	↓↓
Black	116	71.2***	ns	164.9***	ns
Asian PI	4	—		—	
Native American	1	—		—	
Hispanic	15	—		—	

Notes:

1. This cause of death category includes ICD-9 codes 185.

2. Racial groupings (White, Black, Asian & Pacific Islander, Native American) include persons of Hispanic ethnicity.

3. Age-adjusted Mortality Rates (AAMR) and Years of Potential Life Lost (YPLL) rates are per 100,000 based on race and ethnicity specific population estimates. Age-adjusted rates were calculated by the direct method using the 2000 U.S. standard population. Rates were not calculated for fewer than 15 events.

4. Statistical tests were conducted to evaluate differences in rates between race/ethnic groups. The white population serves as the reference group in each comparison (black vs. white, Asian & PI vs. white, Native American vs. white, Hispanic vs. white). Following are explanations of the notations:

     *** Significantly different from the respective white resident rate at $p < .001$.
     — Rate was not calculated due to small numbers.

5. Statistical tests were conducted to evaluate changes in rates over time. Comparisons of 1996-98 vs. 1989-91 rates are made within each race/ethnicity group. Following are explanations of the notations:

     ↓   1996-98 rate is significantly lower than the 1989-91 rate at $p < .05$.
     ↓↓ 1996-98 rate is significantly lower than the 1989-91 rate at $p < .01$.
     ns Indicates the change from 1989-91 to 1996-98 is not statistically significant.

2.5 times the death rate of white males nationwide. Between 1990-1998, prostate cancer death rates nationwide declined for all racial/ethnic subgroups except Native Americans. Declines for whites and APIs were about twice the decline for blacks, Hispanics and Native Americans (Centers for Disease Control and Prevention 2002).

Incidence

Trend data from the Connecticut Tumor Registry indicate that the incidence of prostate cancer increased during the late 1980s through early 1990s. The increase coincided with the increased use of the prostate specific antigen (PSA) screening test. After 1992, rates in Connecticut began to decline for most age groups (Polednak 2001a).

Figure 10.1.

Prostate Cancer Deaths
Comparison of Connecticut and U.S. 1989-1998
Age-Adjusted Mortality Rates*, Males Only

Symbols:

■ - U.S. rates for 1989-1998

✳ ● - Connecticut rates: an asterisk is used if the rate is significantly different than expected (p<.005), based on the log-linear trend.

* These rates are adjusted to the 1940 US standard million population.
* This classification includes deaths with ICD-9 codes:185

Prostate cancer incidence rates are highest for black, followed by white and Hispanic residents of Connecticut. From 1990-1995, black males had 1.6 times, and Hispanic males had 0.9 times the age-standardized incidence rate of white males in Connecticut (Polednak 1999a). Prostate cancer incidence rates are highest among African American men worldwide (American Cancer Society 1999). Nationwide, Hispanic males have lower prostate cancer incidence rates compared with non-Hispanics. In 1988-1991, Hispanic males had rates approximately 20% lower than non-Hispanics (American Cancer Society 2001).

Risk Factors

The etiology of prostate cancer is unknown. Environmental and family factors are believed to contribute to an increased risk for the disease. Increasing age is strongly associated with increased risk for the disease. Over 70% of prostate cancers are diagnosed in men over age 65. Prostate cancer in a first-degree relative may double one's risk. It is estimated that familial genetic factors may account for 5% to 10% of prostate cancers (American Cancer Society 2002a). Some epidemiologic evidence supports a link between a diet high in animal fat and increased risk for prostate cancer (Michaud, Augustsson, Rimm et al. 2001; Moyad 2002). Occupational exposures to cadmium, and
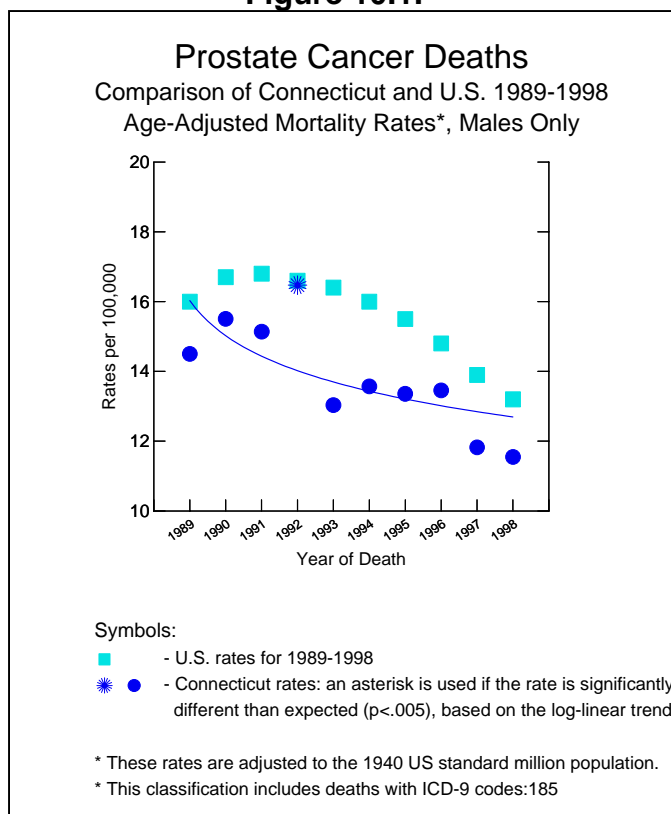
work in certain industries, including farming and rubber manufacturing are also linked to an increased risk for the disease (Brownson, Reif, Alavanja, et al. 1998).

**Table 10.2. Prostate Age-Adjusted Death Rates,
Comparison of CT with US - 1989 and 1998**

	1989	1998	1998 Comparison
CT AAMR*	14.5	11.6	
US AAMR*	16.0	13.2	CT rate < US rate

* age-adjusted mortality rates are per 100,000 population , U.S. 1940 standard million population.

Figure 10.2.

Age-specific Rates for Prostate Cancer Deaths and
Proportionally Adjusted Rates for All Other Causes, 1996 - 1998

Age-specific Deaths Rates and Trend Lines
- Prostate Cancer rates and trend
- All Other Causes trend (rates are not displayed)
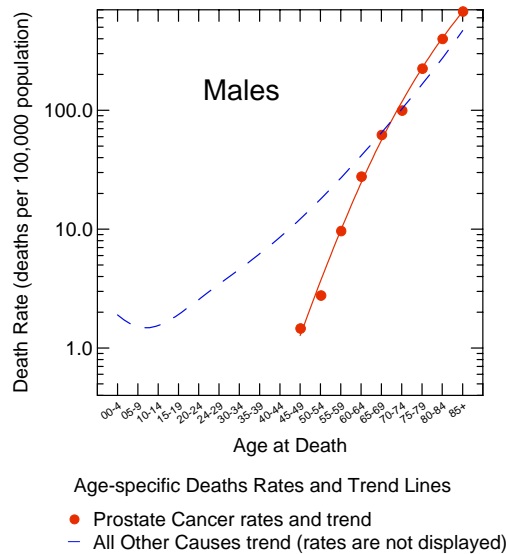
International data do not support a strong or consistent association of socioeconomic status and prostate cancer incidence or mortality (Faggiano, Partanen, Kogevinas, et al. 1997). Stage at diagnosis of prostate cancer is a key determinant of survival. Black Americans tend to be diagnosed at a later stage than whites and these differences need to be further investigated with attention to other possible intervening factors, like socioeconomic status and racial discrimination, on stage of diagnosis and treatment (Polednak 1998).

Prevention

There are no recommended primary preventive measures for prostate cancer because the causal sequence leading to the disease is not well understood. Recommendations for prostate cancer screening vary across expert groups. The American Cancer Society recommends an annual prostate-specific antigen (PSA) test and digital rectal examination beginning at age 50 for men with a life expectancy of ten years or more and at age 45 for men considered at high risk, including African Americans and men with a first degree relative diagnosed at a young age (American Cancer Society 2002a). The U.S. Preventive Services Task Force (USPSTF) uses strict criteria to judge the merits of experimental trials designed to show benefit in screening for cancer. USPSTF does not recommend for or against routine screening for prostate cancer using PSA testing or digital rectal exam. Although there is good evidence that PSA screening can detect early-stage prostate cancer, there is inconclusive evidence that early detection improves health outcomes. USPSTF concludes that there is insufficient evidence to indicate whether the benefits of screening outweigh its harms, which may include false-positive test results, anxiety, and complications of treatment (U.S. Preventive Services Task Force 2002).

Chronic Obstructive Pulmonary Disease (COPD)
And Allied Conditions
(ICD-9 codes 490-496)

Chronic obstructive pulmonary disease (COPD) is a condition characterized by progressive airflow obstruction, which is not completely reversible, due to chronic bronchitis or emphysema (American Thoracic Society 1995; Barnes 2000). Asthma, a clinically distinct condition that is associated with reversible airflow obstruction, is also included in COPD surveillance. *COPD and Allied Conditions* (ICD-9 codes 490-496, hereafter referred to as COPD), reported by the National Center for Health Statistics and the Connecticut Department of Public Health, includes chronic bronchitis, emphysema, and asthma as well as chronic airways obstruction and other less common pulmonary conditions.

In the 1996 to 1998 period, COPD was the fourth leading cause of death among Connecticut residents of all ages and the third leading cause for residents aged 65 to 74. Most COPD deaths were categorized as chronic airways obstruction (2,854 deaths), followed by emphysema (545 deaths), asthma (166 deaths), and bronchitis (97 deaths) [Figure 11.1].

Death in COPD patients often results from some other medical condition or complication. For this reason, and because its contribution to other major causes of death is not always considered, the importance of COPD as a cause of death is probably underestimated. From 1996 to 1998, COPD was the primary cause of death for 3,700 and a contributing cause in the deaths of 8,538 Connecticut residents.

Death rates due to chronic obstructive pulmonary disease increase dramatically with age beginning at ages 45 through 54 (Goldring, James, and Anderson 1998). Connecticut residents 65 years of age and older, about 17% of Connecticut's total population, accounted for 90% of COPD deaths in the 1996 to 1998 period. Age-specific COPD death rates of Connecticut males and females (1996-1998) are depicted in Figure 11.2. Age-specific death rates for COPD are lower compared with all other causes of death up to about age 60 for females and 65 for males at which point they exceed death rates for all other causes. COPD death rates for females 85 years and older decline slightly in relation to all other causes. COPD-related mortality, deaths for which COPD is a

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1996-1998 COPD & Allied Conditions Deaths, Connecticut Residents

- Fourth leading cause of death for all Connecticut residents
- Third leading cause of death for age groups 65 to 74
- White males and females had the highest mortality rates
- Significant increase in white female age-adjusted mortality since 1989-91

Figure 11.1.
COPD and Allied Conditions Deaths
Connecticut Residents. 1996-1998
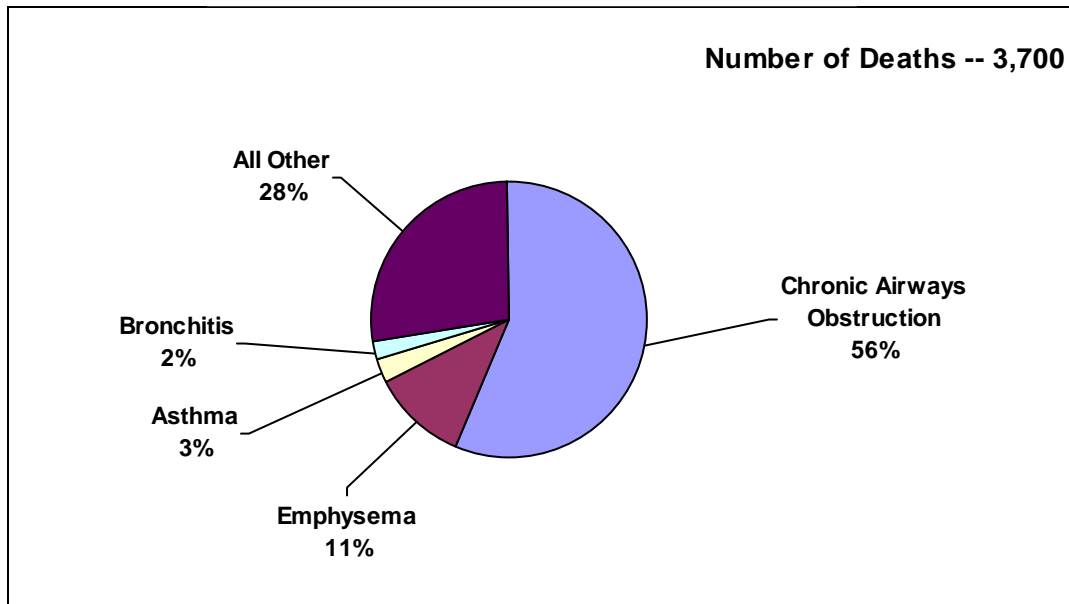
contributing cause, follows a similar pattern.

COPD mortality rates both nationally and in Connecticut, have tended to be higher in men than in women. Earlier initiation and higher rates of smoking among men historically are thought to explain these gender differences (Goldring, James, and Anderson 1998). However, national data (1979 to 1985) indicate that COPD mortality nationwide has increased more rapidly for women than for men (Centers for Disease Control and Prevention 2001f). From 1989 to 1998 in Connecticut, age-adjusted death rates for COPD and COPD-related causes decreased significantly for males by about 1% each per year but increased significantly for females by an average of 4% for COPD and 3% for COPD-related deaths per year. COPD death rates among females showed the greatest increase of all chronic diseases included in this report.

COPD and COPD-related mortality for all Connecticut residents increased significantly between the periods 1989-1991 and 1996-1998. This change is accounted for by an increase in the death rate among white females (Tables 11.1 and 11.2). Although national evidence suggests that there has been a real increase in COPD mortality over time, part of this increase may be artificial and reflect changes in reporting practices over time (Centers for Disease Control and Prevention 2001g). Connecticut male and female mortality rates for COPD have been consistently lower than the respective U.S. rates for the period 1989 to 1998 (Figure 11.3).

COPD and COPD-related mortality rates were significantly higher in white males and females compared with black and Hispanic males and females, respectively, in both the 1989-1991 and 1996-1998 periods. White males and females had about twice the risk of death compared with black and Hispanic males and females, respectively, for COPD (Tables 11.1 and 11.2). There were too few COPD and COPD-related deaths among Asian and Pacific Islander and Native American males and females to report reliable rates.

Figure 11.2.

Age-specific Rates for Chronic Obstructive Pulmonary Disease Deaths
and for All Other Causes, 1996 - 1998

Age-specific Deaths Rates and Trend Lines
● Chronic Obstructive Pulmonary Disease rates and trend
— All Other Causes trend (rates are not displayed)
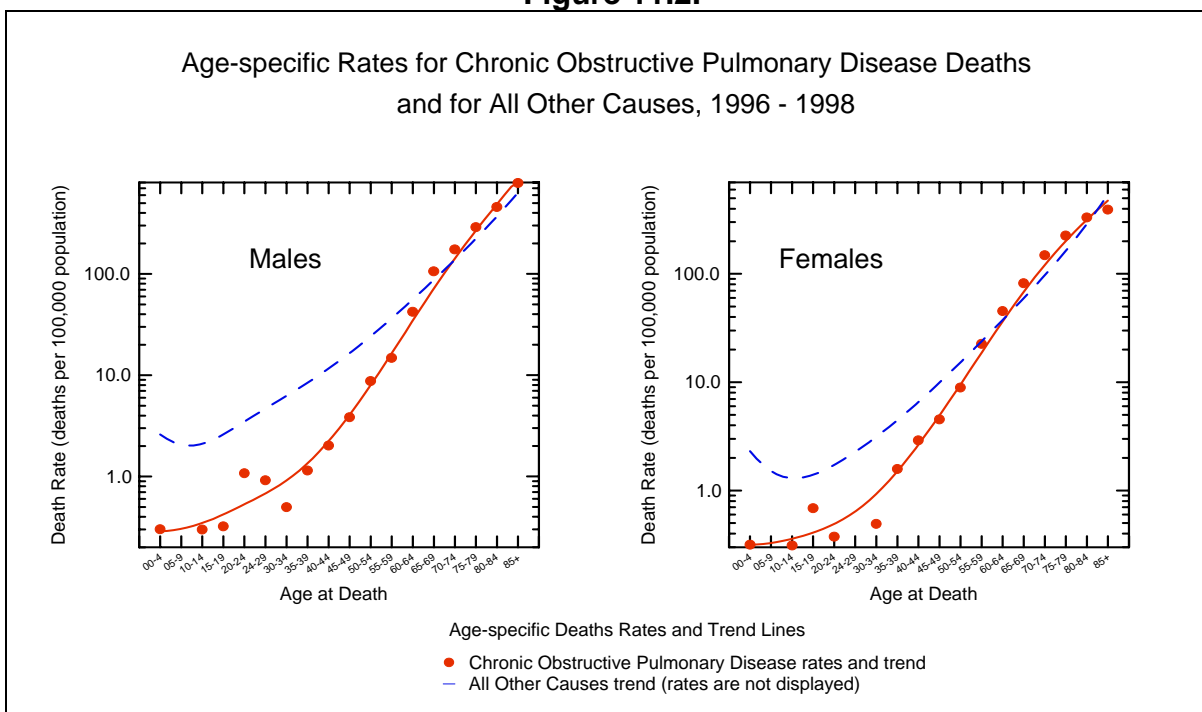
Racial and ethnic differences in mortality reflect differences in the subcategories of COPD deaths by race and ethnicity (Figure 11.4). During the 1996-1998 period, 78% of white resident COPD deaths were due to chronic airways obstruction compared with 60% of black and 49% of Hispanic resident COPD deaths; 15% of white, 12% of black, and 6% of Hispanic resident COPD deaths were due to emphysema. Only 4% of white, compared with 25% of black and 38% of Hispanic COPD deaths, were due to asthma.

This is consistent with national data which show that blacks have lower COPD but higher asthma mortality compared with whites (Gillum 1990). Limited data are available on Hispanics nationwide. One study of 15 states (1979-1981) found that Hispanics have lower COPD mortality compared with non-Hispanics (Goldring, James, and Anderson 1998). A second study indicates that asthma mortality of Hispanic subpopulations (1990-1995) differs by ethnicity nationwide, with Puerto Ricans having the highest rates, followed by non-Hispanic blacks, non-Hispanic whites, Cuban-Americans, and Mexican- Americans (Homa, Mannino, and Lara 2000). Similar national comparisons of COPD mortality in Asian and Pacific Islanders and Native Americans are not available.

Age-adjusted COPD mortality nationwide was higher in whites compared with blacks in 1992; however, time trends (1980 to 1992) indicate a similar sharp increase in age-adjusted COPD mortality for both white (75%) and black females (78%). In contrast, age-adjusted death rates increased by less than 1% for white males and 19% for black males (Centers for Disease Control and Prevention 2001f).

Although no single explanation exists for the significant increase in COPD mortality among Connecticut white female residents during the 1989 to 1998 period, there are several factors which, taken together, shed some light on the observed patterns. Increased tobacco smoking among females, a key factor in explaining rising COPD mortality, is discussed later as a risk factor for COPD. National data suggest that the observed increase in COPD mortality may partially be attributable to a decrease

Figure 11.3.

Chronic Obstructive Pulmonary Disease (COPD) Deaths
Comparison of Connecticut and U.S. 1989-1998
Age-Adjusted Mortality Rates* by Sex

Symbols:
- ■ - U.S. rates for 1989-1998
- ✳ ● - Connecticut rates: an asterisk is used if the rate is significantly different than expected (p<.005), based on the log-linear trend.

* These rates are adjusted to the 1940 US standard million population.
* This classification includes deaths with ICD-9 codes:490-496

in other causes of deaths (Barnes 2000). Older populations have a high likelihood of co-morbidities, that is, multiple risks and disease conditions for mortality. Such conditions are often referred to as "competing risks for" or "competing causes of" death (Kaplan, Haan, and Wallace 1999). The five leading causes of death for Connecticut residents aged 65 & over in the 1989-1991 and 1996-1998 periods included heart disease, cancer, cerebrovascular disease, pneumonia & influenza, and COPD. COPD was the fifth leading cause of death in the 1989-1991 period and the fourth leading cause of death in the 1996-1998 period. Between the two time periods, mortality among white females decreased significantly for heart disease and pneumonia & influenza, but increased significantly for COPD. Decreases in mortality due to heart disease and pneumonia & influenza increase the likelihood that other co-morbid conditions, such as COPD, will become the primary cause of death.

Excess COPD mortality among white compared with black and Hispanic females in Connecticut is at least partially explained by the different age structure of the white and minority populations. As noted earlier, COPD mortality increases with age with the highest rates in persons aged 65 and over. Women aged 65 and over comprise a larger percentage of the white female population relative to other subpopulation groups. About 18% of white Connecticut females are aged 65 or older, compared with 7% of black and 5% of Hispanic females.

Table 11.1. Chronic Obstructive Pulmonary Disease (COPD) Deaths[1]
Connecticut Residents by Gender, Race and Ethnicity[2], 1996-98

Group	Number of Deaths	Age-Adjusted Mortality Rates[3]		Age-Adjusted Premature Mortality Rates to Age 75[3]	
		AAMR[4]	Change since 1989-91[5]	YPLL[4]	Change since 1989-91[5]
All Residents	3,700	33.5	↑↑↑	123.2	ns
All males	1,695	41.0	ns	122.5	ns
White	1,645	42.0	ns	121.0	ns
Black	45	22.0***	↓	150.4	ns
Asian PI	3	—		—	
Native American	2	—		—	
Hispanic	32	21.7***	na	197.5	na
All females	2,005	29.9	↑↑↑	124.3	ns
White	1,955	30.7	↑↑↑	119.5	ns
Black	50	15.1***	ns	173.2	ns
Asian PI	0				
Native American	0				
Hispanic	31	16.2***	ns	126.5	ns

Notes:

1. This cause of death category includes ICD-9 codes 490-496.

2. Racial groupings (White, Black, Asian & Pacific Islander, Native American) include persons of Hispanic ethnicity.

3. Age-adjusted Mortality Rates (AAMR) and Years of Potential Life Lost (YPLL) rates are per 100,000 based on race and ethnicity specific population estimates. Age-adjusted rates were calculated by the direct method using the 2000 U.S. standard population. Rates were not calculated for fewer than 15 events.

4. Statistical tests were conducted to evaluate differences in rates between race/ethnic groups. The white population serves as the reference group in each comparison (black vs. white, Asian & PI vs. white, Native American vs. white, Hispanic vs. white). Following are explanations of the notations:

     *** Significantly different than the respective white resident rate at p < .001.
     — Rate was not calculated due to small numbers.

5. Statistical tests were conducted to evaluate changes in rates over time. Comparisons of 1996-98 vs. 1989-91 rates are made within each race/ethnicity group. Following are explanations of the notations:

     ↑↑↑ 1996-98 rate is significantly higher than the 1989-91 rate at p < .001.
     ↓   1996-98 rate is significantly lower than the 1989-91 rate at p < .05.
     ns Indicates the change from 1989-91 to 1996-98 is not statistically significant.
     na 1989-91 rate was not calculated due to small numbers and so no comparison with 1996-98 rate is available

Table 11.2. COPD-Related Deaths[1]
Connecticut Residents by Gender, Race and Ethnicity[2], 1996-98

Group	Number of Deaths	Age-Adjusted Mortality Rates[3]		Age-Adjusted Premature Mortality Rates to Age 75[3]	
		AAMR[4]	Change since 1989-91[5]	YPLL[4]	Change since 1989-91[5]
All Residents	8,538	77.4	↑↑↑	278.2	ns
All males	4,202	101.4	↓	301.5	ns
White	4,035	102.6	ns	297.8	ns
Black	155	83.4*	ns	400.6	ns
Asian PI	7	—		—	
Native American	5	—		—	
Hispanic	79	66.5***	ns	327.6	ns
All females	4,336	64.1	↑↑↑	258.9	↑
White	4,202	65.4	↑↑↑	252.9	↑
Black	129	41.5***	ns	332.4	ns
Asian PI	5	—		—	
Native American	0				
Hispanic	65	34.6***	ns	244.2	ns

Notes:

1. This cause of death category includes ICD-9 codes 490-496. "COPD-related" deaths include those for which COPD is the underlying and/or a contributing cause listed on the death certificate.

2. Racial groupings (White, Black, Asian & Pacific Islander, Native American) include persons of Hispanic ethnicity.

3. Age-adjusted Mortality Rates (AAMR) and Years of Potential Life Lost (YPLL) rates are per 100,000 based on race and ethnicity specific population estimates. Age-adjusted rates were calculated by the direct method using the 2000 U.S. standard population. Rates were not calculated for fewer than 15 events.

4. Statistical tests were conducted to evaluate differences in rates between race/ethnic groups. The white population serves as the reference group in each comparison (black vs. white, Asian & PI vs. white, Native American vs. white, Hispanic vs. white). Following are explanations of the notations:

> *   Significantly different than the respective white resident rate at $p < .05$.
> *** Significantly different than the respective white resident rate at $p < .001$.
> —   Rate was not calculated due to small numbers.

5. Statistical tests were conducted to evaluate changes in rates over time. Comparisons of 1996-98 vs. 1989-91 rates are made within each race/ethnicity group. Following are explanations of the notations:

> ↑   1996-98 rate is significantly higher than the 1989-91 rate at $p < .05$.
> ↑↑↑ 1996-98 rate is significantly higher than the 1989-91 rate at $p < .001$.
> ↓   1996-98 rate is significantly lower than the 1989-91 rate at $p < .05$.
> ns   Indicates the change from 1989-91 to 1996-98 is not statistically significant.

Risk Factors

Modifiable risk factors for COPD are listed in Table 11.3. Cigarette smoking has been identified as the primary causal factor for COPD, with risk increasing by amount smoked and duration of smoking. Smokers have ten times the risk of developing COPD compared with non-smokers (Goldring, James, and Anderson 1998). Approximately 80% to 90% of COPD mortality among both American men and women has been attributed to cigarette smoking (Centers for Disease Control and Prevention 2001f; U.S. Department of Health and Human Services 2000). Secondhand tobacco smoke contributes to the exacerbation of emphysema, bronchitis, and asthma (Goldring, James, and Anderson 1998).

Figure 11.4.

**Subcategories of COPD Deaths
CT Residents by Race & Ethnicity, 1996-1998**

Increasing COPD mortality rates among women nationwide in the last 30 years are most likely linked to the widespread adoption of smoking by American women in the post-World War II era (Centers for Disease Control and Prevention 2001f). Since 1990, smoking rates for both female and male Connecticut residents have been relatively stable. About 20% of male and 19% of female Connecticut residents aged 18 and older (approximately 500,000 people) are current smokers (Centers for Disease Control and Prevention 2001a). Approximately 58,400 Connecticut middle and high school students currently smoke cigarettes, representing about 10% of all middle and 26% of all high school students (Lowery St. John and Jarvis 2001). Survey estimates suggest that about 186,000 children in Connecticut are exposed to secondhand smoke at home (Campaign for Tobacco-Free Kids, American Cancer Society, American Heart Association, et al. 2002; Centers for Disease Control and Prevention 1997).

In addition to cigarette smoking, ozone and particulates are other air pollutants that can exacerbate COPD symptoms (Goldring, James, and Anderson 1998). Research has established that workplace exposures, such as coal mine dust, cotton dust, silica, and grain dust, can cause COPD. Workers in the agricultural, transportation, textile, wood, paper, construction, and mining industries are considered at increased risk for the development of the disease (Viegi, Scognamiglio, Baldacci et al 2001). Over 200 agents are implicated in causing occupational asthma (Chan-Yeung and Malo 1994; Leigh, Romano, Schenker et al. 2002).
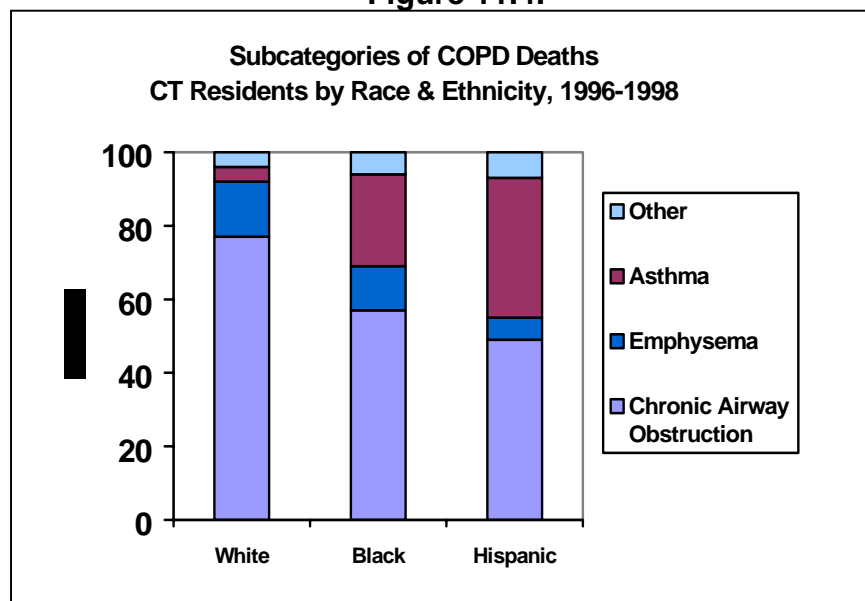
Table 11.3. Modifiable Risk Factors for COPD

Factor	Magnitude of Association[1]	Estimated Range of Population Attributable Risk (%)
Cigarette smoking	Strong	90%
Some occupational exposures	Weak	15%
Air pollution	Weak	–

Source*:* USDHHS 2000; Viegi, Scognamiglio, Baldacci et al 2001.

1. *Strong magnitude* indicates a relative risk of greater than 4 for those persons with the risk factor compared with those not having the risk factor. *Weak magnitude* indicates a relative risk of less than 2 for those persons with the risk factor compared with those not having the risk factor for COPD.

The National Institute for Occupational Safety and Health (NIOSH) estimates that more than 20 million workers in the United States may be exposed to occupational agents that can cause airway obstruction diseases (National Institute for Occupational Safety and Health 1999). The American Thoracic Society estimates occupationally-induced COPD (emphysema and chronic bronchitis) and asthma to account for 15% of all COPD. Occupational exposures may trigger asthma episodes for 5% to 30% of adults with the disease (Viegi, Scognamiglio, Baldacci et al 2001; Leigh, Romano, Schenker et al. 2002).

Low socioeconomic status (SES) is considered a risk factor for emphysema, chronic bronchitis (Viegi, Scognamiglio, Baldacci et al 2001) and asthma (U.S. Department of Health and Human Services 2000). The U.S. National Longitudinal Mortality Study found that among white men and women, lower socioeconomic status was associated with higher COPD mortality (Rogot, Sorlie, Johnson, et al. 1992). Low SES is also associated with other risks for COPD, such as higher smoking rates, greater exposure to environmental tobacco smoke and workplace pollutants, residence in housing and neighborhoods with more environmental pollutants, and reduced access to health care (National Center for Health Statistics 1998). The interrelationship of such factors with COPD is not well understood. Some research has shown, however, that low SES in early life increases adult risk for COPD independent of smoking behavior (Prescott, Lange, and Vestbo 1999).

Familial factors can also play a role in COPD.   Low birth weight appears to increase the risk of COPD, possibly because poor fetal nutrition results in small lungs and poorer lung function. The deficiency of a protein, alpha-1-antitrypsin, may be associated with early-onset emphysema among smokers (Barnes 2000; Viegi, Scognamiglio, Baldacci et al 2001). The effects of multiple risk factors for COPD seem to be additive, so it is important to identify persons with more than one risk factor (Goldring, James, and Anderson 1998).

Costs and Prevention

The economic burden of COPD has not been widely appreciated and is expected to increase substantially in the next thirty years. The estimated cost of emphysema and chronic bronchitis in the United States was $23.9 billion in 1993. This includes direct costs of medical care services (61%) and indirect costs of morbidity and premature mortality (39%) [Sullivan, Ramsey, and Lee 2000].

Assuming that per person expenditures in Connecticut are similar to those nationwide, a conservative estimate of the total costs of emphysema and chronic bronchitis in Connecticut is about $227 million per year. The estimated total cost (medical expenditures and indirect economic losses including premature mortality) of asthma in the United States was $10.7 billion in 1994 (Weiss, Sullivan, and Lyttle 2000). Asthma-related medical care accounts for approximately 57% of these costs. Indirect costs, 43% of the total, include school or workdays lost and premature mortality due to asthma (Asthma and Allergy Foundation of America 2000). Assuming the same per person costs in Connecticut, the total cost of asthma in the state is estimated at $134 to $201 million per year.

COPD prevention efforts focus on smoking cessation for those who smoke, avoiding second-hand smoke for non-smokers, and discouraging young Americans from adopting the smoking habit. The 2000 Surgeon General's report, *Reducing Tobacco Use*, provides evidence that certain kinds of interventions—educational, clinical, regulatory, economic, and comprehensive—can significantly reduce tobacco use. It furthermore suggests that tobacco use rates could decrease by 50% if its recommendations were implemented (Centers for Disease Control and Prevention 2000). Statewide tobacco prevention programs in California, Massachusetts, and Florida have demonstrated that comprehensive education efforts can reduce tobacco use. Key components of successful comprehensive programs include public education efforts, community and school-based programs, smoking cessation efforts, and strict enforcement of laws restricting youth access to tobacco and establishing smoke-free areas (Campaign for Tobacco-Free Kids, American Cancer Society, American Heart Association, et al. 2002).

Smoking cessation is the key intervention in the management of COPD and is likely to be most beneficial when begun at younger ages (Mannino, Gagnon, Petty et al. 2000). Although limited data are available regarding the effects of smoking cessation on COPD mortality, two studies found that COPD mortality was lower in former compared with current female smokers. Furthermore, COPD mortality rates were higher for female former smokers compared with those who had never smoked (Centers for Disease Control and Prevention 2001f). Results from a 40-year study of British male physicians indicate that those who quit smoking at earlier ages tended to decrease their risk of dying from COPD relative to smokers (Viegi, Scognamiglio, Baldacci et al 2001).

Other important prevention measures, such as minimizing occupational and environmental air pollutants, are best achieved through environmental and workplace regulations. The federal government is charged with regulation of workplace hazards and outdoor air pollution. Regulation of indoor air pollutants like cigarette smoke and pesticides takes place through state and local governments. In 2003, the Connecticut legislature passed tighter restrictions on smoking in workplaces and public buildings by banning smoking in workplaces with more than 10 employees; restaurants, cafes, and taverns; state and municipal buildings; and health care institutions, except in rooms designated for smoking (Connecticut General Assemby 2003).

Information gained from Connecticut's Occupational Disease Surveillance System (ODSS) is used to guide follow-up prevention and intervention activities aimed at the industry, workplace and individual levels. ODSS data are shared with health care providers, local health departments, and occupational safety and health-oriented agencies and professional organizations. These data are also

accessible to the public through a series of publications available on the Connecticut Department of Health web site www.dph.state.ct.us (Webb, Heyman, Estrada, et al. 2000).

Regulation of outdoor air pollution in the U.S. began with the Air Pollution Control Act (1955) and Clean Air Act (1963) and its amendments (1970, 1977, and 1990) (Goldring, James, and Anderson 1998). The Clean Air Act requires the U.S. Environmental Protection Agency (EPA) to set National Ambient Air Quality Standards for several pollutants, including ozone, carbon monoxide, nitrogen dioxide, sulfur dioxide, particulate matter, and lead, considered harmful to public health and the environment. As of February 6, 2003, all counties (Fairfield, Hartford, Litchfield, Middlesex, New Haven, New London, Tolland, and Windham Counties) in Connecticut are designated as "nonattainment areas" (areas that persistently exceed the national ambient air quality standards) for criteria pollutants. These counties also exceed national standards for ozone (U.S. Environmental Protection Agency 2003). The Connecticut Department of Environmental Protection (http://dep.state.ct.us) issues air pollution alerts when air pollution levels exceed standards and informs persons with COPD and other disease conditions to limit unnecessary activities.

COPD has been widely underdiagnosed in primary care settings (Voelkel 2000), particularly among women (Chapman, Tashkin, and Pye 2001). Signs and symptoms alone are not adequate for a diagnosis of COPD. Early detection of COPD can be achieved in a primary care setting by means of spirometry (lung airflow measurement). The National Lung Health Education Program recommends widespread use of office spirometry by primary care providers for at-risk patients, that is, current smokers 45 years or older (Ferguson, Enright, Buist, et al 2000). If individuals are identified in the early and asymptomatic stages of COPD, interventions such as smoking cessation can most likely prevent further disease progression (Barnes 2000; Mannino, Gagnon, Petty, et al 2000; Centers for Disease Control and Prevention 2001f; Campaign for Tobacco-Free Kids, American Cancer Society, American Heart Association, et al. 2002; Viegi, Scognamiglio, Baldacci et al 2001; Centers for Disease Control and Prevention 2000).

Since 1980, most of the increase in COPD mortality has occurred in people over age 65. With the aging of the U.S. and Connecticut populations in the next century, as well as with the improved management of other chronic diseases, it is likely that there will be a corresponding increase in persons with COPD unless major risk factors, particularly tobacco smoking, are reduced or eliminated (U.S. Department of Health and Human Services 2000).

Chronic Liver Disease & Cirrhosis
(ICD-9 code 571)

Chronic liver disease and cirrhosis can result from a variety of causal factors that include alcohol consumption, exposures to various drugs and toxic chemicals, viral hepatitis, and other viral and infectious diseases (Saadatmand, Stinson, Grant, et al. 2000). Between 1996 and 1998, 882 Connecticut residents died of chronic liver disease and cirrhosis. It was the tenth leading cause of death for all Connecticut residents and the eighth leading cause of premature mortality to age 75 during this period. This represents a slight change from the 1989-1991 period when chronic liver disease and cirrhosis was the ninth leading cause of death for Connecticut residents.

One-third of these chronic liver disease and cirrhosis deaths were associated with alcohol use (alcoholic fatty liver, acute alcoholic hepatitis, alcoholic cirrhosis of liver, alcoholic liver damage, unspecified). Two-thirds were deaths due to cirrhosis of the liver with no mention of alcohol, chronic hepatitis, biliary cirrhosis, or unspecified chronic liver disease without mention of alcohol (Figure 12.1).

Connecticut male residents had significantly higher age-adjusted death and premature mortality rates due to chronic liver disease and cirrhosis compared with female residents during both the 1989-1991 and 1996-1998 periods. Males had twice the mortality and 2.6 times the premature mortality to age 75 of females (Table 12.1). This disparity is consistent with national data, which show that historically males have had two or more times the chronic liver disease and cirrhosis mortality rate as females (Singh and Hoyert 2000).

Hispanic males had the highest age-adjusted death and premature mortality rates due to chronic liver disease and cirrhosis of all Connecticut gender/ethnic subgroups during the 1996-1998 period. They had about twice the death rate and 2.6 times the premature mortality rate of white males. There were no statistically significant differences in the death and premature mortality rates of black and white males or of black and Hispanic females compared with white females. There were too few deaths among Asian and Pacific Islander and Native American males and females to calculate reliable death and premature mortality rates (Table 12.1).

1996-1998 Chronic Liver Disease & Cirrhosis Deaths, Connecticut Residents

- Tenth leading cause of death for all Connecticut residents

- Eighth leading cause of premature mortality to age 75

- Sixth leading cause of death for age groups 45 to 64

- Males accounted for 62% of all deaths

- Hispanic males had the highest death and premature mortality rates

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Figure 12.1.
Chronic Liver Disease and Cirrhosis Deaths, Percent by Type
Connecticut Residents, 1996-1998
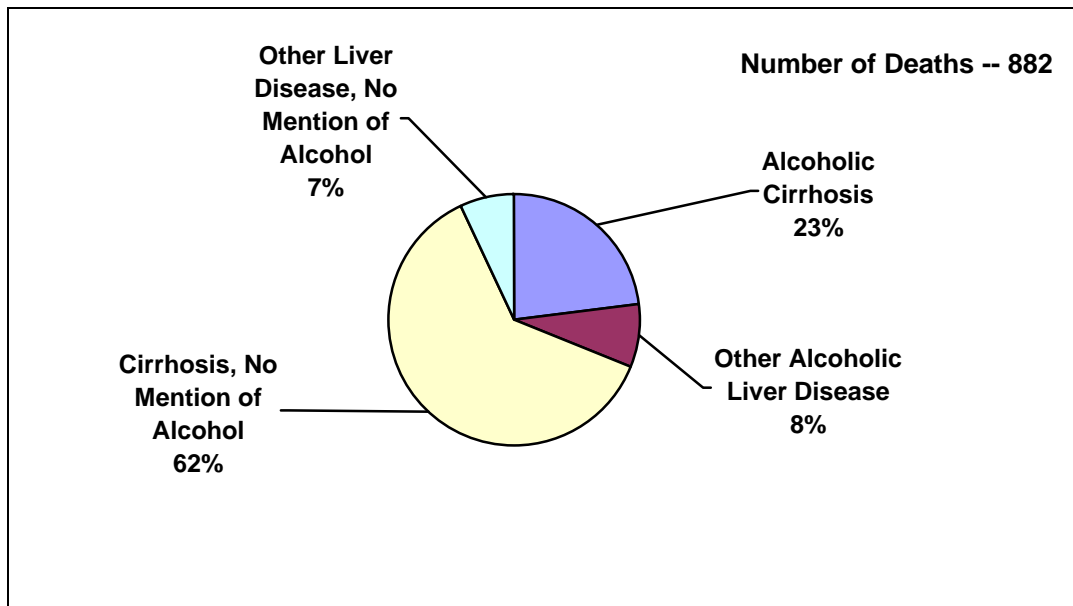
Figure 12.2 depicts age-specific death rates for chronic liver disease and cirrhosis relative to all other causes of death for males and females during this period. Chronic liver disease and cirrhosis death rates were higher compared with all other causes of death for males aged 30 to 69 and for females aged 24 through 79.   In the 1996-1998 period, chronic disease and cirrhosis was the sixth-ranked leading cause of death for females aged 24 through 79.

There were no significant changes in chronic liver disease and cirrhosis age-adjusted death and premature mortality rates between the 1989-1991 and 1996-1998 periods (Table 12.1). Connecticut mortality rates tended to be lower than U.S. rates during the 1989 to 1998 period (Figure 12.3). In 1989, Connecticut mortality was significantly higher than the U.S. *Healthy People 2000* target but by 1996 Connecticut mortality was not significantly different from the U.S. target (Table 12.2).

National data from 1979 to 1989 show that Hispanic and Native American males and black females had excess mortality due to chronic liver disease and cirrhosis compared with their white counterparts. Higher rates of cirrhosis mortality have long been associated with lower socioeconomic status, and Hispanic, black, and Native American persons have a lower socioeconomic profile than do white Americans. This excess mortality risk for Hispanic and Native American males and black females, however, was found to be independent of socioeconomic status (Singh and Hoyert 2000).

National data indicate that excess cirrhosis mortality for Native Americans may be partly attributable to higher alcohol consumption rates. Black and Hispanic Americans, however, have lower alcohol consumption and higher abstention rates than do white Americans. Although there is no obvious explanation for the observed mortality disparity of black and Hispanic Americans nationwide, researchers suggest that less access to alcohol education and other preventive services,

Table 12.1. Chronic Liver Disease and Cirrhosis Deaths[1]
Connecticut Residents by Gender, Race and Ethnicity[2], 1996-98

Group	Number of Deaths	Age-Adjusted Mortality Rates[3]		Age-Adjusted Premature Mortality Rates to Age 75[3]	
		AAMR[4]	Change since 1989-91[5]	YPLL[4]	Change since 1989-91[5]
All Residents	882	8.6	ns	133.8	ns
All males	548	11.9	ns	196.6	ns
White	509	11.9	ns	198.3	ns
Black	36	12.6	ns	224.0	ns
Asian PI	2	—		—	
Native American	1	—		—	
Hispanic	55	25.8**	ns	511.8***	ns
All females	334	5.7	ns	74.4	ns
White	311	5.7	ns	73.4	ns
Black	22	5.5	ns	100.3	ns
Asian PI	1	—		—	
Native American	0				
Hispanic	22	10.3	na	117.6	na

Notes:

1. This cause of death category includes ICD-9 codes 571. *Healthy People 2000* refers to these ICD-9 identifying codes as "cirrhosis."

2. Racial groupings (White, Black, Asian & Pacific Islander, Native American) include persons of Hispanic ethnicity.

3. Age-adjusted Mortality Rates (AAMR) and Years of Potential Life Lost (YPLL) rates are per 100,000 based on race and ethnicity specific population estimates. Age-adjusted rates were calculated by the direct method using the 2000 U.S. standard population. Rates were not calculated for fewer than 15 events.

4. Statistical tests were conducted to evaluate differences in rates between race/ethnic groups. The white population serves as the reference group in each comparison (black vs. white, Asian & PI vs. white, Native American vs. white, Hispanic vs. white). Following are explanations of the notations:

    **   Significantly different than the respective white resident rate at $p < .01$.
    ***   Significantly different than the respective white resident rate at $p < .001$.
    —   Rate was not calculated due to small numbers.

5. Statistical tests were conducted to evaluate changes in rates over time. Comparisons of 1996-98 vs. 1989-91 rates are made within each race/ethnicity group. Following are explanations of the notations:

    ns   Indicates the change from 1989-91 to 1996-98 is not statistically significant.
    na   1989-91 rate was not calculated due to small numbers and so no comparison with
          1996-98 rate is available.

Figure 12.2.

Age-specific Rates for Chronic Liver Disease and Cirrhosis Deaths
and for All Other Causes, 1996 - 1998

alcoholism treatment, and medical services compared with white Americans may be a partial explanation (Singh and Hoyert 2000).

National trends indicate that chronic liver disease and cirrhosis mortality has shown distinctive patterns during the twentieth century. It was high before the Prohibition era, decreased during Prohibition (1920-1933), increased again during the Great Depression (1929-1939) and continued to rise during World War II, reaching a peak in the 1970s.

From the early 1970s through 1997, mortality from chronic liver disease and cirrhosis decreased uniformly for the entire U.S. population and the major ethnic and gender subpopulations. Researchers have suggested that the consistent decline in cirrhosis mortality for almost thirty years is most likely associated with decreased alcohol consumption and, particularly, in decreased consumption of hard liquors by Americans (Singh and Hoyert 2000). Other researchers have called for a closer examination of decreasing trends in chronic liver disease deaths not identified as alcohol-related, noting that these account for a larger proportion of the mortality decline (Hurwitz, Holman, Strine et al. 1995).
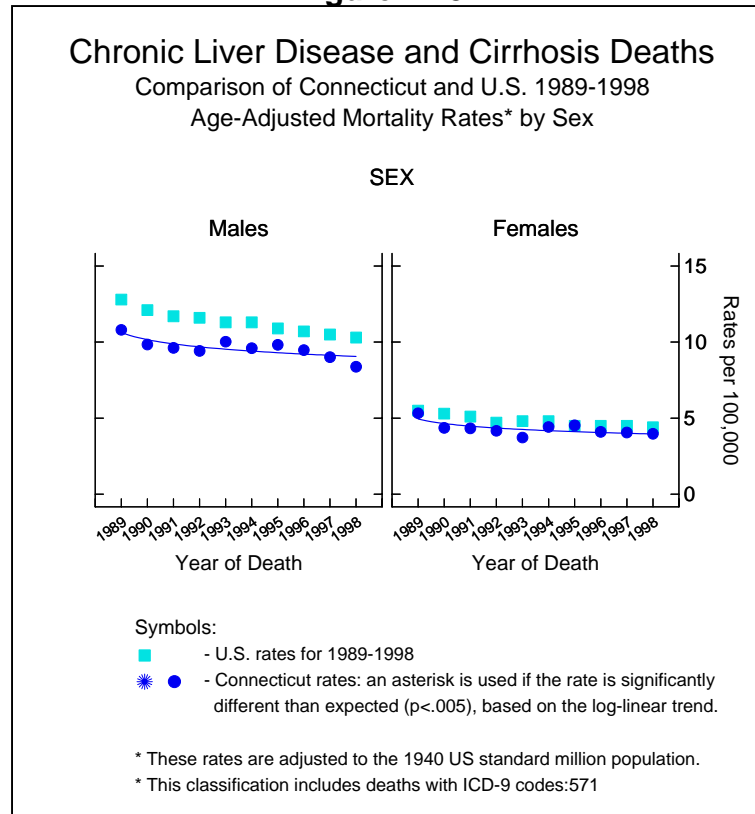
Risk Factors

In the United States, alcohol use is considered the most important risk factor for chronic liver disease followed by hepatitis B and C. There is evidence that alcohol-related diagnoses may be underreported on the death certificate. It is believed that alcohol use may be the causal factor in as

many as 50% of deaths coded as chronic liver disease unspecified or with no mention of alcohol (Centers for Disease Control and Prevention 1993b; Hurwitz, Holman, Strine, et al. 1995).

Various measures of socioeconomic status, such as low educational attainment, low income, blue-collar job, and unemployment, are social risks associated with cirrhosis mortality (Klatsky and Armstrong 1992; Harford and Brooks 1992; Rosenberg, Burnett, Maurer et al. 1993; Smith, Neaton, Wentworth et al. 1996; Singh and Hoyert 2000). Males are twice or more likely to die of the disease as are females. Subgroups at higher

Figure 12.3.

Chronic Liver Disease and Cirrhosis Deaths
Comparison of Connecticut and U.S. 1989-1998
Age-Adjusted Mortality Rates* by Sex

Symbols:
- U.S. rates for 1989-1998
- Connecticut rates: an asterisk is used if the rate is significantly different than expected (p<.005), based on the log-linear trend.

* These rates are adjusted to the 1940 US standard million population.
* This classification includes deaths with ICD-9 codes:571

risk of cirrhosis mortality include the older-aged, U.S.-born male, Hispanic and Native American, and urban resident populations (Singh and Hoyert 2000).

National data indicate that more than half of persons diagnosed with hepatitis B and C report that their risk factors are unknown. The most commonly reported risk factor for both hepatitis B and C is injection drug use, followed by sexual contact with hepatitis-infected persons. Other high-risk categories include blood transfusions, hemodialysis, employment in a health care setting, and household contact with an infected person (Centers for Disease Control and Prevention 2000c). Excessive alcohol use by persons infected with hepatitis B and/or C can increase their risk for chronic liver disease (Frieden, Ozick, McCord, et al. 1999).

Costs and Prevention

Hepatitis C (HCV) poses a serious concern because it is currently the most common bloodborne infection in the U.S. with an estimated 3.9 million Americans infected. Many of these persons may not be aware that they are infected because they are not clinically ill, and for this reason, are at increased risk of transmitting the disease to others. Evidence suggests that 40% of chronic liver disease cases in the U.S. are linked to HCV infection. The majority of HCV-infected persons are 30 to 49 years of age. The number of HCV-related chronic liver disease deaths is likely to increase

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Table 12.2. Comparison of CT with US, 1989 and 1998

	1989	1998	1998 Comparison
CT AAMR*	7.9	6.1	---
US AAMR*	9.2	7.2	CT < US AAMR
*Healthy People 2000**	6.0	6.0	CT not significantly different than *HP2000* target

* age-adjusted mortality rates are per 100,000 population, U.S. 1940 standard million population.

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during the next two decades as this cohort reaches the age at which chronic liver disease complications occur (Centers for Disease Control and Prevention 1998).   The future economic burden posed by premature mortality (under age 65) due to HCV-related cirrhosis mortality has been estimated at $54.2 billion nationwide between the years 2010 and 2019. Morbidity due to disability from HCV-related cirrhosis and liver cancer during this period is estimated to reach $21.3 billion (Wong, McQuillan, McHutchison et al. 2000).

Most subtypes of chronic liver disease are considered preventable.   Both national and Connecticut Behavioral Risk Factor Surveillance System survey data indicate that males comprise a greater proportion of chronic and binge drinkers than do females (Centers for Disease Control and Prevention 2001h).   Societal approaches to reducing alcohol consumption and thus, cirrhosis mortality, include measures such as increased taxes on alcohol, controlling the physical availability and legal accessibility of alcohol, health warning labels, and health information messages and education (Horgan, Skwara, Strickler, et al. 2001).

Strategies for preventing hepatitis B and C focus on the common modes of transmission and high-risk groups.   Hepatitis B (HBV) is transmitted both by percutaneous blood and mucosal exposures while hepatitis C (HC/v) is transmitted mostly by percutaneous blood exposure (Mast, Alter, and Margolis 1999).

Prevention strategies for hepatitis B transmission have been outlined by the national Advisory Committee on Immunization Practices. Primary prevention includes vaccination of all children 18 years and younger during routine medical visits; prevention of perinatal transmission by identifying and providing treatment to infants of mothers testing positive for HBV; and universal hepatitis B vaccination of infants. The majority of adult HBV infections are found among persons with defined risk factors. Primary prevention includes the identification of settings where persons at high risk can be vaccinated such as family planning, sexually transmitted disease, and drug treatment clinics; HIV prevention sites in the community; and correctional facilities (Centers for Disease Control and Prevention 1999b). Other measures include prevention of nosocomial transmission through exposure

to contaminated blood and eliminating practices of unclean needle sharing among injection drug users. The Centers for Disease Control and Prevention recommends that health care institutions educate health care workers regarding risk for and prevention of bloodborne infections and the importance of vaccination, and implement barrier precautions and workplace design features to prevent exposure to blood. Protocols for the reporting and follow-up of blood or body fluid exposures should be in place and adhered to (Centers for Disease Control and Prevention 1998). The Occupational Health and Safety Administration (OSHA) Bloodborne Pathogen Standard of 1992 set workplace regulations for minimizing occupational exposure to bloodborne pathogens (Udasin and Gochfeld 1994).

While there is currently no vaccine developed for hepatitis C, primary prevention efforts focus on the prevention of nosocomial exposures and risky practices such as sharing of contaminated needles and other drug equipment (Mast, Alter, and Margolis 1999). It is estimated that as many as 90% of injection drug users are infected with HCV and they risk transmitting the infection to others. Prevention efforts in this population focus on substance abuse treatment, safer injection practices, and information about preventing bloodborne diseases (Alter and Moyer 1998). Prevention of chronic liver disease in persons with HBV and/or HCV infection includes reducing alcohol consumption and avoiding needle sharing (Frieden, Ozick, McCord, et al. 1999).

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SECTION IV. C.

INJURY MORTALITY

CONTENTS

Injury and Other External Causes of Death
(ICD-9 codes E800-E999)

Injury is a leading cause of premature death both in Connecticut and the nation. Injury deaths together with those due to adverse effects of medical care and therapeutic drugs comprise the category "all external causes of death." During the period 1989-1998, 14,926 Connecticut residents died as a result of injury and other external causes. Injury deaths, by definition, are preventable and include intentional types such as homicides and suicides as well as unintentional types such as motor vehicle crashes and falls. The largest numbers of externally-caused deaths are attributable to unintentional injuries, suicide, and homicide and legal intervention (Figure 13.1). Other broad categories of injury mortality, which may be intentional or unintentional, include firearm mortality, about 17% of all injury deaths, and poisoning mortality, about 16% of all injury deaths.

Certain subgroups of the population are at higher risk for injury and other external causes of deaths than are others. During the ten-year period, 69% of all externally-caused deaths (10,332) occurred among males. Connecticut residents aged 65 years and over accounted for about 30% (4,448) and residents aged 15 to 34 years accounted for about 34% (5,050) of all externally caused deaths. External causes accounted for about 54% of all deaths to Connecticut residents aged 15 to 34 years.

Reduction of injury risk requires an understanding of how injuries vary across different physical and social environments. Effective prevention strategies can be developed through an understanding of the patterns of injury across the many settings in which people spend time—home, school,

Figure 13.1. Injury and Other External Causes of Death, Percent by Subgroup Connecticut Residents, 1989-1998
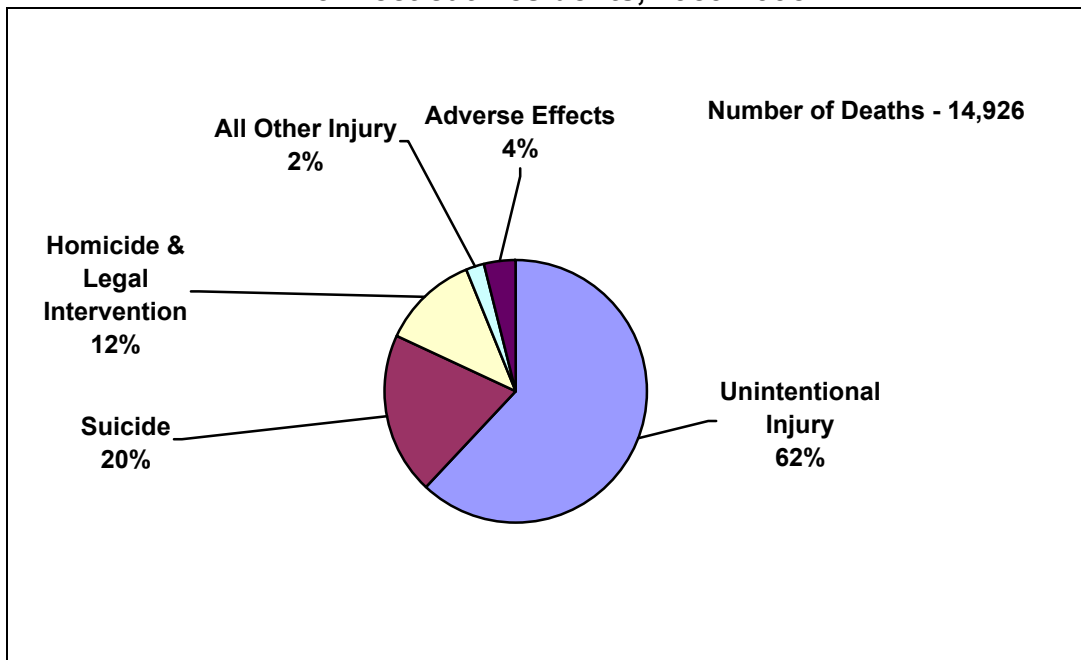
playground, workplace, and on the road.

Death rates for injury and other external causes have remained about the same for both male and female Connecticut residents over the ten-year time period. Connecticut death rates have been consistently lower than comparable national rates (Figure 13.2).

The following sections examine the patterns of injury mortality in motor vehicle crashes and falls and fall-related injury, the two leading causes of unintentional injury deaths; homicide and legal intervention; suicide; and in related categories of injury deaths—poisoning, drug-induced, and alcohol-induced mortality.

Changes in the coding of certain categories of deaths beginning in 1991 may result in an artificial increase in various subcategories of injury deaths when examining trends over time. Beginning with the 1992 mortality file, the Connecticut Department of Public Health (DPH) was able to significantly reduce the number of deaths identified as "pending further investigation" by the Connecticut Medical Examiner's (ME) Office through an improved system of communication. Speedier processing of findings from the ME investigations has resulted in more complete and more accurate cause-of-death classification being entered into the death records. Many drug-induced deaths and categories that overlap with it (injury and other external causes, unintentional injuries, suicide, poisoning) tend to be included in the category of "pending." For this reason, trend analyses reported for these categories of deaths (injury and other external causes, unintentional injuries, suicide, poisoning, and drug-induced) exclude the years 1989 through 1991 and instead compare the periods 1992-1994 and 1996-1998.

Figure 13.2.

Injury and Other External Causes of Death
Comparison of Connecticut and U.S. 1989-1998
Age-Adjusted Mortality Rates* by Sex

SEX

Males   Females

Year of Death   Year of Death

Symbols:
■ - U.S. rates for 1989-1998
✳ ● - Connecticut rates: an asterisk is used if the rate is significantly different than expected (p<.005), based on the log-linear trend.

* These rates are adjusted to the 1940 US standard million population.
* This classification includes deaths with ICD-9 codes:E800-E999

Unintentional Injury
(ICD-9 codes E800-E949)

From 1989 to 1998, 9,816 Connecticut residents died as a result of unintentional injuries. Major categories of unintentional injury deaths include those due to motor vehicle crashes, falls, drowning, and residential fires. Also included in the unintentional injury category are deaths due to adverse effects of surgical and medical care; abnormal reactions or later complications of surgical and medical procedures; and medicinal drugs and biological substances causing adverse effects when used therapeutically (Figure 14.1). Other categories of unintentional injury deaths include those caused by poisoning; cutting and piercing instruments or objects; machinery or transportation vehicles; suffocation; excessive cold or heat; and overexertion.

Unintentional injury is the leading cause of death for Connecticut residents under age 45 and the sixth leading cause of death for all Connecticut residents (Appendix V). It is the third leading cause of premature mortality to age 75.

Age-specific death rates of Connecticut males and females for the period 1996-1998 are displayed in Figure 14.2. Unintentional injury mortality rates for males and females, contrasted with proportionally adjusted rates for all other causes of death, show a distinctive pattern of higher rates in the younger age groups and slightly lower but parallel rates in the older age groups. The largest percentage of unintentional injury deaths occurs in the oldest age group, a pattern that is consistent with other causes of death. During the 1996-1998 period, 40% of all unintentional injury deaths occurred among Connecticut residents 65 and older; 29% among those aged 25 to 44; 17% among those 45 to 64; 10% among 15 to 24 year-olds; and 4% among residents aged under age one to 14 years.

1996-1998 Unintentional Injury Deaths, Connecticut Residents

- Sixth leading cause of death for all residents

- The leading cause of death for residents aged 0 – 44

- Third leading cause of premature mortality to age 75

- 3.6% of all deaths

- 2.5% of all deaths among females

- 4.8 % of all deaths among males

- 6.2% of all deaths under 75 years of age

- Significant increase in mortality compared with the 1992-94 period

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**Figure 14.1. Unintentional Injury Deaths, Percent by Type
Connecticut Residents, 1989-1998**
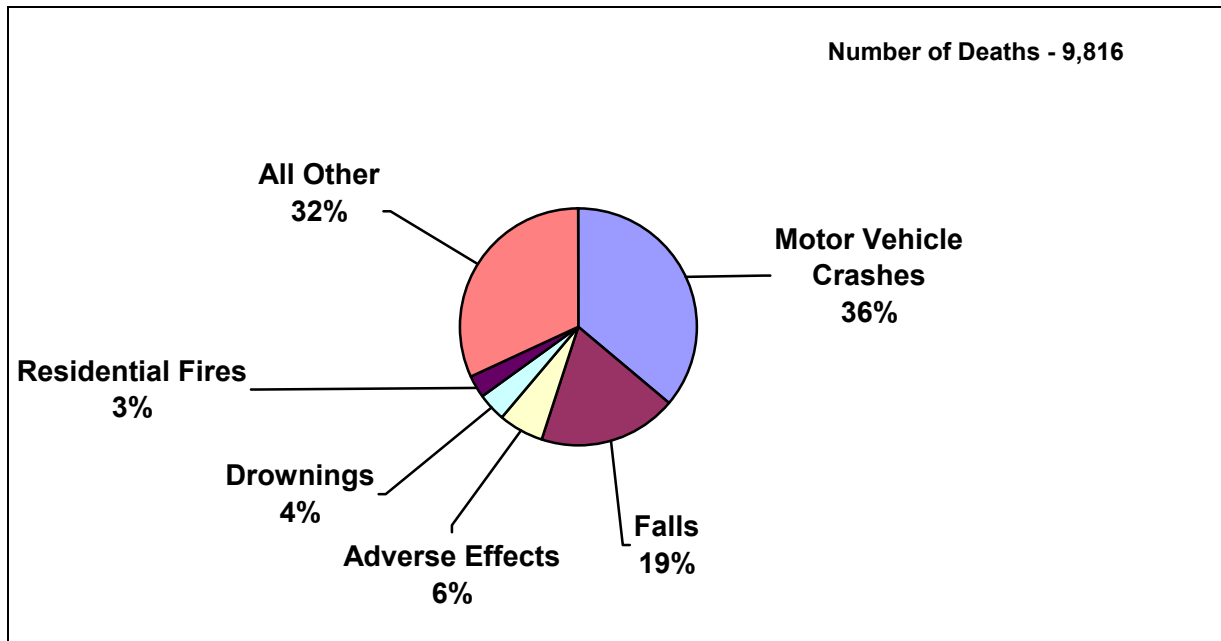
Males accounted for about 65% of all unintentional injury deaths and 75% of premature mortality to age 75 during the ten-year period. During the 1996-1998 period, black males were at highest risk of death due to unintentional injuries, followed by Hispanic, white, and Asian and Pacific Islander males. Black males were significantly more likely and Asian and Pacific Islander males were significantly less likely than white males to die from unintentional injuries. Black males had 1.3 times the rate of unintentional injury deaths as did white males (Table 14.1). Logistic regression analyses of the black-white male unintentional injury mortality (1996-1998) showed that the observed disparity differed significantly by age group with black males aged 45 to 59 years having a three-fold risk of death compared with white males. There were no significant differences in unintentional injury mortality by age group for Hispanic compared with white males during this period. Among females, the black-white disparity in unintentional injury mortality appeared to differ by age group (p < .001), but relative risk estimates were not calculated due to the small number of black female deaths within each age group. There were no significant differences between Hispanic and white females in unintentional injury mortality by age group during this period.

Between the periods 1992-1994 and 1996-1998, unintentional injury mortality rates for all Connecticut residents increased significantly. Analysis of unintentional injury mortality trends for population subgroups present a more detailed picture of changes over time. While mortality rates for white males, black males and females, and Hispanic males and females remained about the same, they increased significantly for white females between the two time periods. There were insufficient numbers of unintentional injury deaths among Asian and Pacific Islanders and Native American males and females during the two time periods to evaluate changes over time (Table 14.1).

Figure 14.2.

Age-specific Rates for Unintentional Injury Deaths
and Proportionally Adjusted Rates for All Other Causes, 1996 - 199
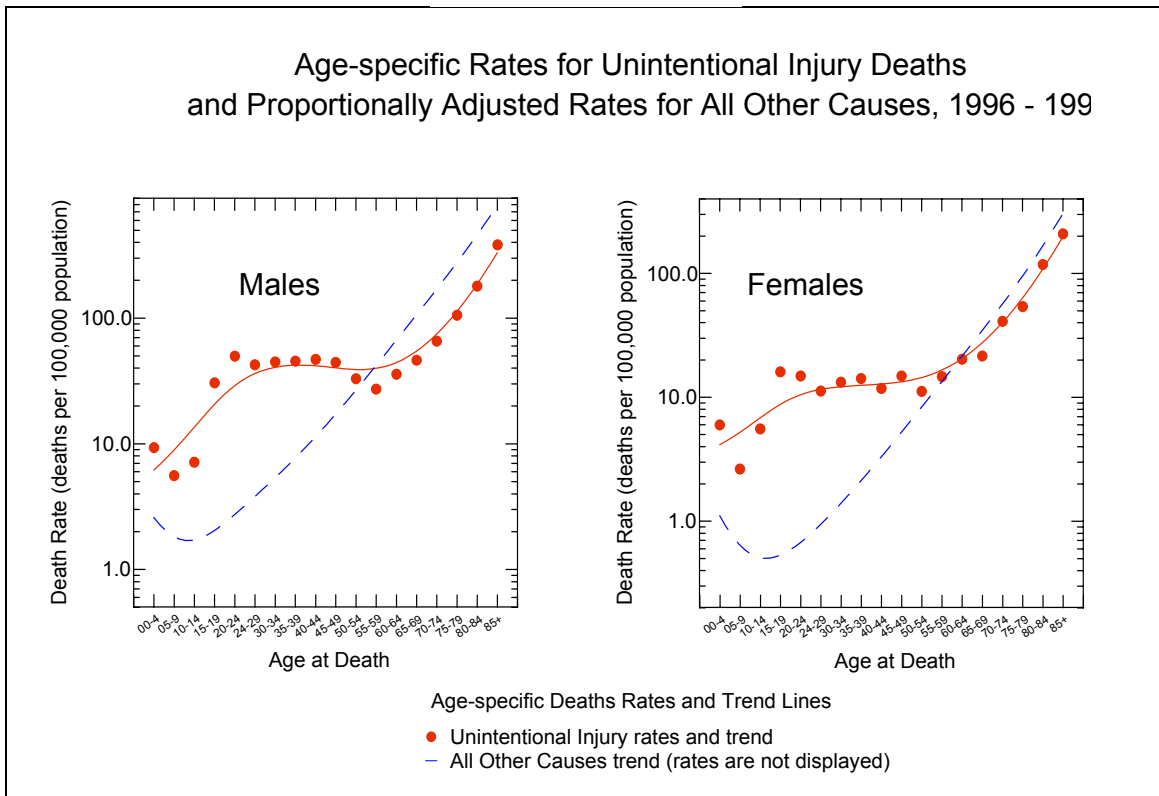
Between 1992 and 1998, the average annual increase in unintentional injury deaths for white females was 4.3% (p < .001). Results of logistic regression analyses of changes in age groups over time show that there was an average annual decrease in unintentional injury deaths of 6.2% for males aged 15-19 between 1989 and 1998 (p<.005). Most of this observed change is accounted for by decreases in motor vehicle crash death rates in this age group.

Premature mortality due to unintentional injuries was significantly higher among black and Hispanic males but significantly lower among Asian and Pacific Islander males compared with white males during the 1996–1998 period. There were no significant changes in premature mortality for male racial and ethnic subgroups between the 1992-1994 and 1996-1998 periods. Premature mortality among female racial and ethnic subgroups did not differ significantly during the 1996-1998 period. However, there was a statistically significant increase in premature mortality due to unintentional injuries among white females from the 1992-1994 to the 1996-1998 period (Table 14.1).

Age-adjusted mortality rates for Connecticut residents were consistently lower than the U.S. rates (Figure 14.3) as well as the national *Healthy People 2000* target (Table 14.2). There is no Connecticut target for unintentional injuries.

Table 14.1. Unintentional Injury Deaths[1], Connecticut Residents by Gender, Race and Ethnicity[2], 1996-1998

Group	Number of Deaths	Age-Adjusted Mortality Rates[3]		Age-Adjusted Premature Mortality Rates to Age 75[3]	
		AAMR[4]	Change since 1992-94[5]	YPLL[4]	Change since 1992-94[5]
All Residents	3,158	30.7	↑	822.5	ns
All males	2,016	44.3	ns	1,205.2	ns
White	1,789	43.5	ns	1,173.4	ns
Black	201	57.2*	ns	1,714.5**	ns
Asian PI	19	23.7*	na	640.0**	na
Native American	4	—		—	
Hispanic	164	50.2	ns	1,643.7**	ns
All females	1,142	19.1	ns	441.1	ns
White	1,047	19.1	↑	439.6	↑
Black	80	19.3	ns	513.6	ns
Asian PI	8	—		—	
Native American	5	—		—	
Hispanic	48	13.9	ns	446.6	ns

Notes:

1. This cause of death category includes ICD-9 codes E800-E949. (NCHS cause of death classification refers to codes E800-E949 as "Accidents and Adverse Effects").

2. Racial groupings (White, Black, Asian & Pacific Islander, Native American) include persons of Hispanic ethnicity.

3. Age-adjusted Mortality Rates (AAMR) and Years of Potential Life Lost (YPLL) rates are per 100,000 based on race and ethnicity specific population estimates. Age-adjusted rates were calculated by the direct method using the 2000 U.S. standard population. Rates were not calculated for fewer than 15 events.

4. Statistical tests were conducted to evaluate differences in rates between race/ethnic groups. The white population serves as the reference group in each comparison (black vs. white, Asian & PI vs. white, Native American vs. white, Hispanic vs. white). Following are explanations of the notations:

    * Significantly different than the respective white resident rate at $p < .05$.
    ** Significantly different than the respective white resident rate at $p < .01$.
    — Rate was not calculated due to small numbers.

5. Statistical tests were conducted to evaluate changes in rates over time. Comparisons of 1996-98 vs. 1992-94 rates are made within each race/ethnicity group. Following are explanations of the notations:

    ↑ 1996-98 rate is significantly higher than the 1992-94 rate at $p < .05$.
    ns Indicates the change from 1992-94 to 1996-98 is not statistically significant.
    na 1992-94 rate was not calculated due to small numbers and so no comparison with 1996-98 rate is available

Risk reduction efforts targeting unintentional injuries should be tailored to both the type of injury (motor vehicle, falls, drowning, etc.), the high-risk groups in the population (subgroups of males), and groups that have shown a significant increase over time (white females). Specific categories of unintentional injury deaths—motor vehicle crashes and falls—their risk factors, and risk reduction measures are discussed in the following pages.

Table 14.2. Unintentional Injury Age-Adjusted Death Rates, Comparison of CT with US - 1992 and 1998

	1992	1998	1998 CT AAMR Comparison
CT AAMR*	21.7	25.4	
US AAMR*	29.5	30.1	CT AAMR < US AAMR
*Healthy People 2000**	29.3	29.3	CT AAMR < *HP2000* rate

* age-adjusted mortality rates for unintentional injury are per 100,000 population, U.S. 1940 standard million population.

Figure 14.3.

Unintentional Injury Deaths
Comparison of Connecticut and U.S. 1989-1998
Age-Adjusted Mortality Rates* by Sex

SEX

Symbols:
■ - U.S. rates for 1989-1998
✳ ● - Connecticut rates: an asterisk is used if the rate is significantly different than expected (p<.005), based on the log-linear trend.

* These rates are adjusted to the 1940 US standard million population.
* This classification includes deaths with ICD-9 codes:E800-E949

Motor Vehicle Crashes
(ICD-9 codes E810-E825)

Motor vehicle crashes are the leading cause of injury death both in Connecticut and the nation, comprising more than one-third of all unintentional injuries among Connecticut residents during the period 1989-1998. They are a leading cause of premature mortality in the state, averaging 12,558 years of potential life lost per year before age 75 during this period.

Motor vehicle crashes include deaths from motor vehicles that occur in traffic on public highways as well as deaths resulting from motor vehicles being used in recreational or sporting activities off the highway. Such deaths may occur to drivers, passengers, cyclists, or pedestrians. Between 1996 and 1998, almost 70% of all Connecticut resident motor vehicle crash deaths occurred among drivers and passengers of cars, motorcycles or other vehicles, 15% to pedestrians, and 1% to bicyclists (Figure 15.1).

Connecticut's motor vehicle death rate has been consistently lower than the national rate (Figure 15.2). Connecticut ranks among the five lowest states for motor vehicle fatalities (National Highway Traffic Safety Administration 1998). During the ten-year period, the motor vehicle death rate statewide decreased significantly on an average of 1.4% per year (p < .05). By the 1996-1998 period it had reached the *Healthy Connecticut* target (Table 15.1). This trend is consistent with national statistics that showed a decrease in the death rate from 1979-1995 by almost one-third (National Highway Traffic Safety Administration 1997).

Sixty-eight percent of motor vehicle crash fatalities occurred among men in the 1996-1998 period. Male Connecticut residents were about twice as likely as females to die from motor vehicle crashes, with the largest male to female ratio found in the 15-34 year age group (Appendix VII A). There were no significant differences in motor vehicle crash death or premature mortality rates among racial and ethnic subgroups (Table 15.2).

1996-1998 Motor Vehicle Crash Deaths, Connecticut Residents

- The leading cause of injury death for all CT residents

- A leading cause of premature mortality for all CT residents

- 32% of all unintentional injury deaths

- 68% were male

- High risk age groups: 15 to 34 years · 65 years and over · 4 to 8 years

- Significant decrease in mortality compared with the 1989-91 period

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Figure 15.1.
Motor Vehicle Crash Deaths, Percent by Subtype
Connecticut Residents, 1996-1998
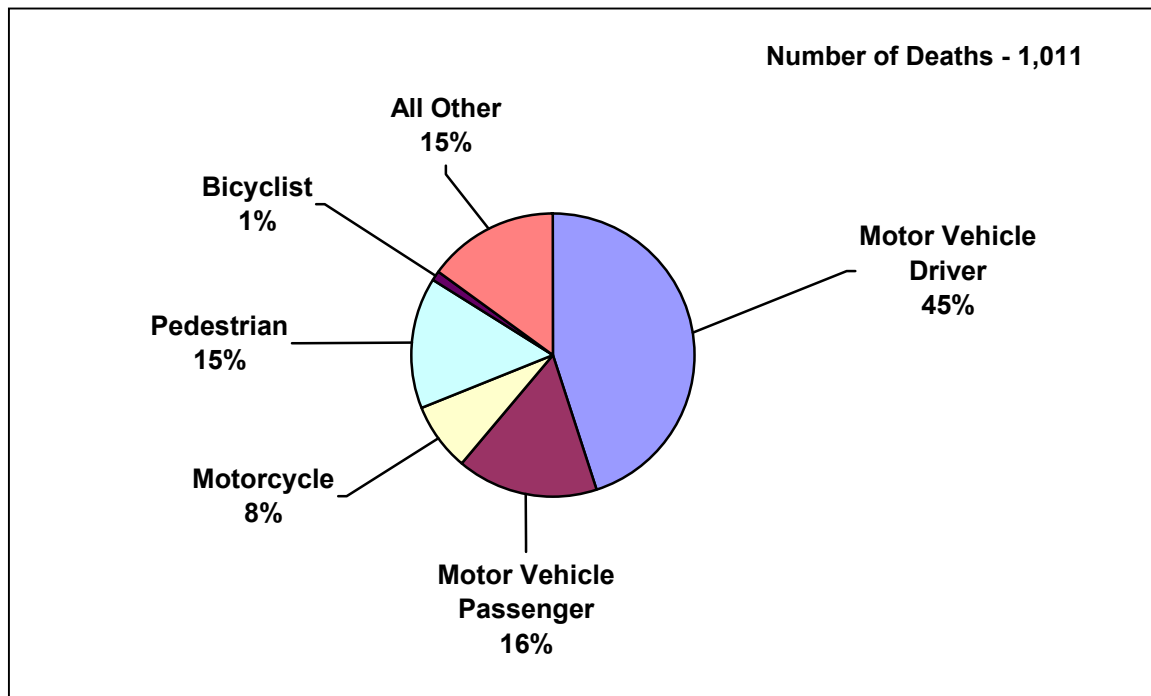
Between the periods 1989-1991 and 1996-1998, motor vehicle crash mortality decreased significantly among males (a change accounted for by a decrease in mortality among white males) but remained about the same for females (Table 15.2). Results from linear trend analysis for 1989 to 1998 show that male mortality decreased by about 3% per year (p < .005) [the 1991 rate was excluded from the analysis because it lay below the trend line].

Premature mortality declined significantly among white and black males but not other racial/ethnic and gender subgroups. There were insufficient numbers of motor vehicle crash deaths among Asian and Pacific Islander and Native Americans in the 1996-1998 period to calculate reliable rates (Table 15.2).

Motor vehicle fatalities are more likely to occur among certain age groups in the population. Connecticut residents aged 15-34 and those aged 65 and over were at highest risk of death due to motor vehicle crashes during the 1996-1998 period. Age-specific motor vehicle crash death rates for males and females are depicted in Figure 15.3. Motor vehicle crash death rates for males aged 5-54 and females 0-64 exceeded proportionally adjusted rates for all other causes of death.

Logistic regression analyses show that there was an average annual decrease in unintentional injury deaths of 6.2% for males aged 15-19 between 1989 and 1998 (p < .005). Most of this observed change is accounted for by decreases in motor vehicle crash death rates in this age group.

This decrease in mortality is quite likely linked to a change in drinking and driving patterns among Connecticut youth. Between 1985 and 1998, driving while intoxicated arrests of 16 to 18 year olds in Connecticut decreased by 66%, a trend that may be partially attributed to a change in the legal drinking age to 21 (Connecticut General Assembly 1999). National data indicate that teenaged motor vehicle mortality in the U.S. declined by 15% as a result of raising the legal drinking age to 21 years (Wagenaar 1993). Since 1982, youth driving and drinking has decreased dramatically, as measured by self-reported behavior and drinking drivers in fatal car crashes (Hedlund, Ulmer, and Preusser 2001).

Figure 15.2.

Motor Vehicle Crash Deaths
Comparison of Connecticut and U.S. 1989-1998
Age-Adjusted Mortality Rates* by Sex

SEX

Males   Females

Rates per 100,000

Year of Death   Year of Death

Symbols:

☐ - U.S. rates for 1989-1998

✳ ● - Connecticut rates: an asterisk is used if the rate is significantly different than expected (p<.005), based on the log-linear trend.

* These rates are adjusted to the 1940 US standard million population.
* This classification includes deaths with ICD-9 codes:E810-E825

Risk Factors

Key factors related to the likelihood of a motor vehicle crash include speed, vehicle instability and braking deficiencies, inadequate road design, and alcohol intoxication (Rice, MacKenzie, and Associates 1989). Driving under the influence of alcohol is a major risk factor for motor vehicle crash mortality. In 1998, approximately 43% of traffic fatalities in Connecticut involved alcohol and about 34% involved persons with blood alcohol content above the legal limit of 0.10 g/dl (National Highway Traffic Safety Administration 1998a). The major determinants of injury severity following a motor vehicle crash include the speed at impact, vehicle crashworthiness, as well as the use of airbags, safety belts, and motorcycle helmets (Rice, MacKenzie, and Associates 1989).

Risk factors for crashes differ by age group. Younger drivers are more likely to have crashes related to risk-taking and lack of skill such as driving at high speeds, on curved roads, during adverse weather, and when fatigued. Older drivers are more likely to have crashes involving perceptual problems (such as vision or judging the speed of an oncoming vehicle) that create risks when negotiating traffic intersections, while turning, and changing lanes (McGwin and Brown 1999).

Table 15.1. Motor Vehicle Crash Age-Adjusted Death Rates, Comparison of CT with US - 1989 and 1998

	<u>1989</u>	<u>1998</u>	<u>1998 CT AAMR Comparison</u>
CT AAMR*	13.0	10.0	
US AAMR*	18.8	15.6	CT AAMR < US AAMR
Healthy CT 2000*	10.8	10.8	achieved *Healthy CT* target

* age-adjusted mortality rates for motor vehicle crashes are per 100,000 population, U.S. 1940 standard million population.

Nonuse of safety restraints is a key risk factor in motor vehicle injuries. In 1998, 59% of passenger car occupants killed in Connecticut were not wearing safety restraints (National Highway Traffic Safety Administration 1998a). Children between the ages of 4 and 8 are a population at high risk for injury. While children up to age four are typically placed in a child safety seat, children between 4 and 8 may outgrow safety seats and often are seated unrestrained or put inappropriately into adult safety systems. National data for 1994-1998 indicate that almost two-thirds of fatally injured children aged 4-8 were not restrained at the time of the crash and only about 5% of children in this age group were seated in appropriate booster seats when riding in motor vehicles (Centers for Disease Control and Prevention, National Center for Injury Prevention and Control 2000a).

Costs and Prevention

The total cost of motor vehicle crashes in the United States was estimated at $150.5 billion in 1994, or $580 per person. This represents the costs of emergency services, medical care and rehabilitation, property damage, productivity losses, costs to employers, insurance, legal and court costs, and premature funeral costs. Estimated total costs of motor vehicle crashes in Connecticut in 1994 were $2.1 billion or $646 per person (Blincoe 1994). Costs of speeding-related crashes in Connecticut for 1998 were estimated at $371 million (National Highway Traffic Safety Administration 1998a).

Several prevention efforts have been highly effective in reducing injuries and deaths related to motor vehicle crashes. Connecticut has a mandatory seat-belt law requiring any person in the front seat of a vehicle to wear a seat belt. State law also requires children to be in a proper child safety restraint system while riding in a vehicle (State of Connecticut 2001). When correctly installed and used, child safety seats reduce the risk of death by about 70% for infants and by about 50% for

Table 15.2. Motor Vehicle Crash Deaths[1], Connecticut Residents by Gender, Race and Ethnicity[2], 1996-1998

| Group | Number of Deaths | Age-Adjusted Mortality Rates[3] | | Age-Adjusted Premature Mortality Rates to Age 75[3] | |
		AAMR[4]	Change since 1989-91[5]	YPLL[4]	Change since 1989-91[5]
All Residents	1,011	10.3	ns	379.2	↓↓
All males	652	13.9	↓	514.7	↓↓
White	561	13.5	↓	493.8	↓↓↓
Black	74	18.8	ns	689.4	Ns
Asian PI	12	—		—	
Native American	2	—		—	
Hispanic	55	16.6	ns	571.0	Ns
All females	359	6.9	ns	242.4	Ns
White	325	7.1	ns	255.1	Ns
Black	26	5.8	ns	183.7	Ns
Asian PI	6	—		—	
Native American	0				
Hispanic	21	5.5	na	214.0	Na

Notes:

1. This cause of death category includes ICD-9 codes E810-E825. The National Center for Health Statistics refers to these ICD-9 identifying codes as "motor vehicle accidents."

2. Racial groupings (White, Black, Asian & Pacific Islander, Native American) include persons of Hispanic ethnicity.

3. Age-adjusted Mortality Rates (AAMR) and Years of Potential Life Lost (YPLL) rates are per 100,000 based on race and ethnicity specific population estimates. Age-adjusted rates were calculated by the direct method using the 2000 U.S. standard population. Rates were not calculated for fewer than 15 events.

4. Statistical tests were conducted to evaluate differences in rates between race/ethnic groups. The white population serves as the reference group in each comparison (black vs. white, Asian & PI vs. white, Native American vs. white, Hispanic vs. white). Following are explanations of the notations:

    — Rate was not calculated due to small numbers.

5. Statistical tests were conducted to evaluate changes in rates over time. Comparisons of 1996-98 vs. 1989-91 rates are made within each race/ethnicity group. Following are explanations of the notations:

    ↓   1996-98 rate is significantly lower than the 1989-91 rate at $p < .05$.
    ↓↓   1996-98 rate is significantly lower than the 1989-91 rate at $p < .01$.
    ↓↓↓ 1996-98 rate is significantly lower than the 1989-91 rate at $p < .001$.
    ns   Indicates the change from 1989-91 to 1996-98 is not statistically significant.
    na   1989-91 rate was not calculated due to small numbers, so no comparison with 1996-98 rate is available.

Figure 16.3.

Age-specific Rates for Motor Vehicle Crash Deaths and

Proportionally Adjusted Rates for All Other Causes, 1996 - 1998
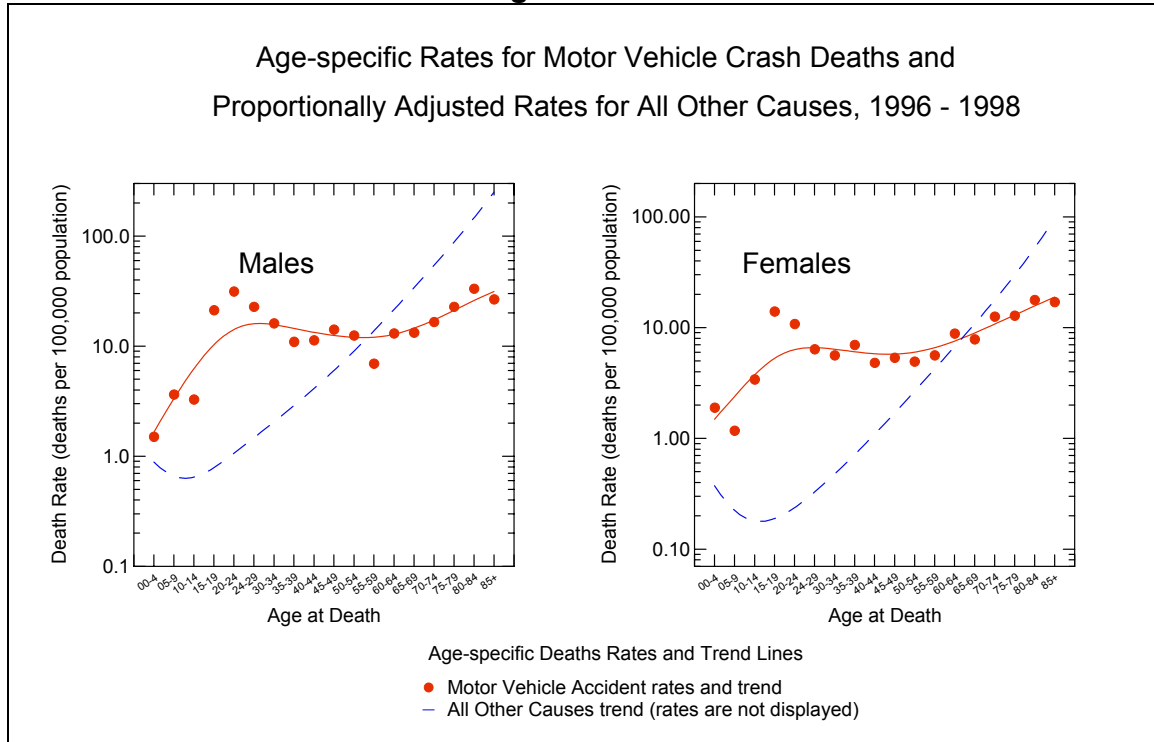
children ages 1 to 4 (Kahane 1986; National Highway Traffic Safety Administration 1996). They can also reduce hospitalization for children ages 4 and under by almost 70% (National Highway Traffic Safety Administration 1998b).

A study of the effectiveness of safety restraints in reducing mortality from car collisions nationwide (1992-1997) found that lap-shoulder seat belt and air bag use reduced mortality by 72% and 63%, respectively. The combination of air bag and seat belt use reduced mortality by over 80% (Crandall, Olson, and Sklar 2001).

Several policies have been shown to be effective in reducing alcohol-related crashes among drivers (Wagenaar, Zobeck, Williams, et al. 1995). Legislation prohibiting youth drinking and driving is currently in effect in all 50 states, although publicity and enforcement of the new laws have been limited. Public awareness campaigns to improve knowledge regarding, and enforcement of, this law could increase its effectiveness (Wagenaar, O'Malley, and LaFond 2001). A 1999 Connecticut law imposes additional restrictions on drivers under 21 years of age. This "zero tolerance" law imposes penalties for driving with any measurable amount of alcohol in the blood, defined as 0.02% blood alcohol content or more (State of Connecticut 2001).

Another strategy, "administrative license revocation" (ALR) or suspension laws, is in effect in Connecticut and about 40 other states. ALR laws allow police to quickly suspend a driver's license based on results of a blood alcohol test, thus providing an immediate deterrent for driving under the influence. National studies suggest that such laws have contributed to the reduction in fatal crashes involving alcohol (Zador, Lund, Fields, et al. 1989; Voas, Tippetts, and Fell 2000).

Studies have also shown that decreases in motorcycle helmet usage are associated with increases in head injury and deaths across states (Sosin, Sacks, and Holmgreen 1990). Connecticut currently does not have a mandatory motorcycle helmet law. In 1998, 66% of Connecticut motorcyclists killed were not helmeted (National Highway Traffic Safety Administration 1998a). Analysis of Connecticut data from 1985-1987 revealed that non-helmeted motorcyclists were more than three times as likely to die from crashes as were helmeted riders. Estimates of savings of a uniform helmet law in Connecticut for 1992 included an average of 10 lives saved and 90 nonfatal injuries prevented at a cost savings of $5.1 million (Braddock, Schwartz, Lapidus et al. 1992).

Bicycle helmets have been shown to be effective in reducing injuries from motor vehicle crashes. Bike helmets are estimated to reduce serious head injury by as much as 85% and brain injury by 88% (Thompson, Rivara, and Thompson 1989).

Most pedestrians killed by motor vehicles in the U.S. are young children, older adults, or intoxicated persons. In 1996, about 25% of pedestrians killed in traffic were under 16 years of age and about 18% were aged 70 or older. More than half of all adult pedestrians killed in nighttime crashes had BACs of 0.10% (considered legally drunk in many localities) or higher. Appropriate and effective interventions differ by age group. Child pedestrian injuries, for example, frequently occur while the child is playing. Research on child pedestrian injury indicates that changes in the physical environment (e.g. play spaces separated from the street) are more effective than education because young children may not easily recognize and react to traffic hazards (Centers for Disease Control and Prevention, National Center for Injury Prevention & Control 2000b).

More than one-third of pedestrian deaths among persons 65 and older take place at traffic intersections (Centers for Disease Control and Prevention, National Center for Injury Prevention & Control 2000b). Examples of community measures aimed at increasing safety for older pedestrians include: the modification of stop light signals at intersections to increase walking time; roadway marks to highlight pedestrian crosswalks and traffic lanes; enlarged speed limit signs; and pedestrian signals on median islands (Centers for Disease Control and Prevention 1989). Other public health strategies used to prevent pedestrian fatalities include the separation of pedestrians from traffic lanes by guard rail or overpasses; public education in locations such as night spots where people may be likely to drink and drive; and increased availability of buses, taxis and other public transportation to discourage drinking and driving (Centers for Disease Control and Prevention 1993). Motor vehicle crash prevention efforts should target high-risk subgroups through both legislative initiatives and public safety education.

The U.S. Preventive Services Task Force recommends that physicians counsel all patients and the parents of young children to use proper safety restraints while riding in cars, to wear proper helmets when riding motorcycles, and to refrain from driving while under the influence of alcohol or other drugs (U.S. Preventive Services Task Force 1996).

Fall and Fall-Related Injuries
(ICD-9 codes E880-E888)

Fall and fall-related injuries are the second leading cause of unintentional injury death among all Connecticut residents and the leading cause of unintentional injury deaths among persons aged 75 and older. Every year, approximately 30% of Americans over age 65 experience a fall (Tinetti, Baker, McAvay, et al. 1994; Sattin 1992). About 20% to 30% in this group (28,000 to 42,000 people in Connecticut) sustain serious injuries that reduce their mobility and independence, and increase their risk for premature death (Alexander, Rivara, and Wolf 1992).

Between 1989 and 1998, 1,872 Connecticut residents died from falls and fall-related injuries. About 86% of all such deaths occurred among Connecticut residents aged 60 and over. Males have significantly higher fall and fall-related injury mortality rates compared with females. Although the highest rates are found among males in the 85 and older age group, 67% of deaths in that age group (1996-1998 period) were female [Appendix VII A].

Ninety-seven percent of fall and fall-related injury deaths in the 1996-1998 period occurred among white residents. Death rates were not calculated for black, Hispanic, Asian American and Pacific Islander, and Native American male and female residents due to insufficient numbers (Table 1). Nationwide data indicate that fall-related death rates for both men and women are higher for whites than for blacks (U.S. Department of Health and Human Services 1999). Researchers have suggested that greater bone mass among black compared with white persons 65 and older may partially explain their lower prevalence of osteoporosis, bone fractures, and fall-related injuries (Snelling, Crespo, Schaeffer et al. 2001; U.S. Department of Health and Human Services 1999). Such hypotheses are not well supported by research evidence, however (Kessinich 2000). Racial and ethnic differences in bone health and osteoporosis prevalence have not been well studied and the National Institutes of Health has identified this as an important area for further investigation (U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health 2001).

1996-1998 Fall and Fall-Related Injury Deaths, Connecticut Residents

- Second leading cause of unintentional injury death

- 85% occurred among CT residents aged 65 years and older

- 97% occurred among white CT residents

- Ratio of male to female mortality - 2:1

Table 16.1. Fall and Fall-Related Injury Deaths[1], Connecticut Residents by Gender, Race and Ethnicity[2], 1996-1998

| Group | Number of Deaths | Age-Adjusted Mortality Rates[3] | | Age-Adjusted Premature Mortality Rates to Age 75[3] | |
		AAMR[4]	Change since 1989-91[5]	YPLL[4]	Change since 1989-91[5]
All Residents	619	5.6	ns	36.4	ns
All males	326	8.0	ns	58.9	ns
White	314	8.2	ns	62.5	ns
Black	12	—		—	
Asian PI					
Native American					
Hispanic	8	—		—	
All females	293	4.0	ns	14.5	ns
White	287	4.1	ns	14.0	ns
Black	3	—		—	
Asian PI	1	—		—	
Native American	2	—		—	
Hispanic	3	—		—	

Notes:

1. This cause of death category includes ICD-9 codes E880-E888. (*Healthy People 2000* cause of death classification refers to codes E880-E888 as "Falls and Fall-Related Injuries").

2. Racial groupings (White, Black, Asian & Pacific Islander, Native American) include persons of Hispanic ethnicity.

3. Age-adjusted Mortality Rates (AAMR) and Years of Potential Life Lost (YPLL) rates are per 100,000 based on race and ethnicity specific population estimates. Age-adjusted rates were calculated by the direct method using the 2000 U.S. standard population. Rates were not calculated for fewer than 15 events.

4. Statistical tests were conducted to evaluate differences in rates between race/ethnic groups. The white population serves as the reference group in each comparison (black vs. white, Asian & PI vs. white, Native American vs. white, Hispanic vs. white). Following are explanations of the notations:

    — Rate was not calculated due to small numbers.

5. Statistical tests were conducted to evaluate changes in rates over time. Comparisons of 1996-98 vs. 1989-91 rates are made within each race/ethnicity group. Following are explanations of the notations:

ns Indicates the change from 1989-91 to 1996-98 is not statistically significant.

Fall and fall-related injury mortality rates have not changed significantly during the ten-year period for Connecticut residents as a whole or for any subpopulation in the state. During the ten-year period, age-adjusted mortality rates in Connecticut have been similar to those nationwide (Figure 16.1) [Centers for Disease Control and Prevention 1999] and have tended to be about the same as the *Healthy People* and *Healthy Connecticut* target objectives of 2.3 per 100,000 population (Table 16.3).

The risk of falling and fall-related mortality increases with age. Age-specific death rates for fall and fall-related mortality are depicted in Figure 16.2. Age-specific fall and fall-related death rates tend to be higher for males aged 20-34 and 85 and older and for females aged 80 and older relative to proportionally adjusted rates for all other causes of death.

Risk Factors

Risk factors for falling among the elderly include health problems that limit the performance of daily activities like dressing and bathing, vision problems, muscle weakness, problems with balance, and osteoporosis. The presence of chronic conditions such as cardiovascular, cerebrovascular, and neurologic disorders may also increase risk for falls (Sattin 1992). Behavioral factors associated with increased risk for falls include alcohol use, use of multiple medications or psychoactive drugs, a sedentary lifestyle, and a history of falls. Environmental risk factors include home hazards like floor clutter, slippery surfaces, and poor lighting (Stevens and Olson 2000) [Table 16.4]. The likelihood of falling has been shown to increase with the number of risk factors present (Tinetti, Speechley, and Ginter 1988; Nevitt, Cummings, Kidd, et al. 1989).

Figure 16.1.

Falls and Fall Related Injury Deaths
Comparison of Connecticut and U.S. 1989-1998
Age-Adjusted Mortality Rates* by Sex

Symbols:
- U.S. rates for 1989-1998
- Connecticut rates: an asterisk is used if the rate is significantly different than expected (p<.005), based on the log-linear trend.

* These rates are adjusted to the 1940 US standard million population.
* This classification includes deaths with ICD-9 codes:E880-E888

Figure 16.2.

Age-specific Rates for Falls and Fall Related Injury Deaths and
Proportionally Adjusted Rates for All Other Causes, 1996 - 1998

Age-specific Deaths Rates and Trend Lines
● Falls and Fall Related Injury rates and trend
– All Other Causes trend (rates are not displayed)

Costs and Prevention

Fall-related injury costs are usually expressed in terms of the direct costs of all medical and rehabilitation care, prescription drugs, home modifications, and insurance administration. Direct costs do not include the long-term costs of these injuries, including disability and decreased productivity and reduced quality of life due to disability (Centers for Disease Control and Prevention 2003). The cost of all fall injuries for persons aged 65 and older nationwide was $20.2 billion in 1994. By 2020, the cost of fall injuries in the United States is projected to reach $32.4 billion before adjustment for inflation (Centers for Disease Control and Prevention 2003).

Programs that target several of the modifiable risk factors offer promise in reducing fall incidence (Tinetti, Baker, McAvay, et al. 1994.) Combination strategies include regular exercise programs that improve strength, balance, mobility and flexibility (Judge, Lindsey, Underwood, et al. 1993; Lord, Caplan, and Ward 1993; Lord, Ward, and Williams 1996). Early detection and treatment of common vision conditions such as cataracts and glaucoma might reduce falls. Prevention and appropriate treatment of chronic illnesses such as cardiovascular disease can also decrease the number of falls and related injuries (Sattin 1992). Other strategies include the review and adjustment of medications by a health care professional to minimize side effects such as dizziness, drowsiness, or disorientation; and education about fall prevention through written materials and home visits and assessments (Stevens and Olson 2000).

Table 16.3. Fall and Fall-Related Injury Age-Adjusted Death Rates, Comparison of CT with US - 1989 and 1998

	1989	1998	1998 CT AAMR Comparison
CT AAMR*	2.4	2.4	
US AAMR*	2.7	2.8	not significantly different
*Healthy People 2000**	2.3	2.3	not significantly different
*Healthy CT 2000**	2.3	2.3	not significantly different

- age-adjusted mortality rates for fall and fall-related injuries are per 100,000 population, U.S. 1940 standard million population.

Educational strategies are most effective when accompanied by environmental modifications such as installation of appropriate safety devices. Sixty percent of fatal falls among older Americans occur in the home (Sorock 1988), so identification of household hazards is particularly important. Modifications such as increasing lighting, installing rails on both sides of stairs, installing grab bars, removing tripping hazards such as loose rugs, objects on floors, and unstable furniture, and correcting uneven or slippery floors are particularly important (Stevens and Olson 2000; Centers for Disease Control and Prevention, National Center for Injury Prevention and Control 1999).

Basic clinical screening tests can accurately identify seniors who are at high risk for falls (Centers for Disease Control and Prevention, National Center for Injury Prevention and Control 2000a). Health care providers, however, do not routinely screen high-risk patients for falls. The goal of a current initiative, the Connecticut Collaborative for Fall Prevention, is

Table 16.4. Risk Factors for Falls

- Increasing age
- Muscle weakness
- Lack of physical activity
- Difficulties in gait and balance
- Visual impairment
- Osteoporosis
- Low body mass index

- Foot problems
- Inappropriate footwear
- Use of psychoactive medications
- Some combinations of medications
- Alcohol use
- A history of falls
- Home hazards (i.e. rugs, floor clutter)

Source: Sattin 1992; Stevens and Olson 2000.

to institutionalize fall prevention into the health care system for older adults. Other promising prevention strategies include identifying footwear that promotes stability and balance, developing more effective home lighting systems, and designing undergarments with energy absorbing hip pads (Stevens and Olson 2000). Such multifactorial approaches have been shown to be cost-effective (Rizzo, Baker, McAvay et al. 1996; Centers for Disease Control and Prevention, National Center for Injury Prevention and Control 1999).

The U.S. Preventive Services Task Force recommends that physicians counsel elderly patients on specific measures to prevent falls. The Task Force recommends individualized multifactorial intervention for high-risk elderly patients in settings that have the resources to deliver such services (U.S. Preventive Services Task Force 1996).

Suicide
(ICD-9 codes E950-E959)

During the period 1989-1998, 3,003 Connecticut residents committed suicide. This accounts for about 63% of all intentional injury deaths. Firearms are the most common weapons used in completed suicides. Other common methods of completed suicides are suffocation by hanging and other means, drug overdose, and carbon monoxide poisoning (Figure 17.1).

As a group, males are at considerably higher risk for suicide than are females (Meehan, Saltzman, and Sattin 1991; Kachur, Potter, James, et al. 1995; Lewinsohn, Rohde, and Seeley 1996; Gould, Fisher, Parides et al. 1996; Moscicki 1997; Gould, King, Greenwald, et al. 1998). Men are about four times more likely to commit suicide than are females both in Connecticut and the U.S. (U.S. Department of Health and Human Services 1999). About 77% of all suicide deaths in Connecticut during the 1996-1998 period were male, and males accounted for about 78% of all premature mortality due to suicide (Table 17.1).

Certain age groups in the population are at higher risk for suicide death (Sorensen 1991; Kachur, Potter, James, et al. 1995; Moscicki 1997). While suicide was the eleventh leading cause of death in Connecticut during the 1996-1998 period, it was the third leading cause for residents aged 15 to 24. Although suicide death rates are highest among elderly males in Connecticut, 50% of all suicides were committed by males aged 15 to 49 (Appendix VII A). Suicide was the fifth leading cause of premature death before age 75 among Connecticut residents in 1996-1998 (Appendix V).

Figure 17.2 depicts age-specific suicide death rates for males and females (1996-1998) in relation to all other causes of death. Suicide death rates in both males and females were higher compared with proportionally adjusted rates for all other causes up to about age 59, at which point suicide death rates decrease relative to all other causes.

Suicide death and premature mortality rates for all Connecticut residents and for gender and racial/ethnic subgroups remained unchanged from the 1992-1994 to 1996-1998 period. Results of logistic regression analyses for the period 1992-1998, however, indicate that suicide death rates for males showed a significant decrease of about 3.2% per year (p < .01).

1996-1998 Suicide Deaths, Connecticut Residents

- Eleventh leading cause of death for all CT residents
- Third leading cause of death for age groups 15 to 24
- Fifth leading cause of premature death for CT residents
- 77% were male
- 38% were firearm suicides

**Figure 17.1. Suicide Deaths
by Method Used**
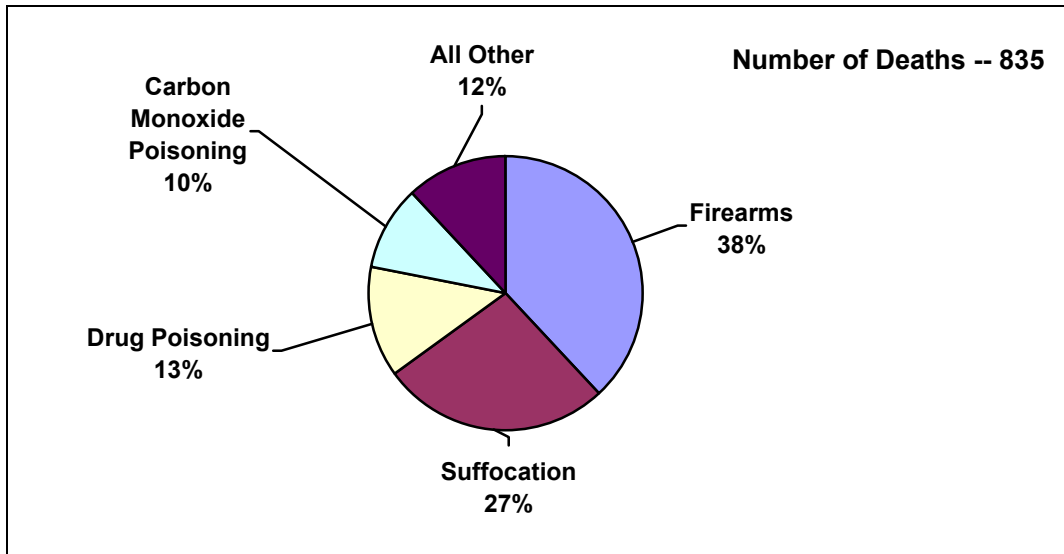
During the 1996-1998 period, suicide death rates of white and black males were not significantly different, while Hispanic males had a significantly lower suicide death rate than did white males. About 92% of all female suicides completed during 1996-1998 period were by white females. There were too few suicide deaths among black and Hispanic females, and Asian and Pacific Islander and Native American males and females to calculate reliable rates (Table 17.1).

Connecticut's suicide rate has been consistently lower than the national rate (Figure 17.3) as well as the national *Healthy People 2000* target; however, it was significantly higher than the Healthy Connecticut 2000 target from 1992 through 1996. By 1997 and 1998, the suicide rate for all Connecticut residents was not significantly different from the Healthy Connecticut target (Table 17.2).

Risk Factors

Suicide risk differs dramatically by gender and age group (Meehan, Saltzman, and Sattin 1991; Lewinsohn, Rohde, and Seeley 1996; Gould, Fisher, Parides, et al 1996; Moscicki 1997; Gould, King, Greenwald, et al. 1998). For this reason, diagnostic risk profiles for suicide should include appropriate gender and age-specific characteristics (Gould, King, Greenwald, et al. 1998). Suicide prevention requires a multifaceted approach that addresses both risk groups and demonstrated risk factors. Research has consistently shown that the causes of suicide are multifactorial. Mental disorders, most notably mood, personality, and substance abuse, are underlying conditions of most attempted and completed suicides (Moscicki 1997). These conditions have been shown to be risk factors independent of sociodemographic characteristics like age, gender, race, and socioeconomic status (Gould, King, Greenwald, et al. 1998).

**Table 17.1. Suicide Deaths[1], Connecticut Residents
by Gender, Race and Ethnicity[2], 1996-1998**

| Group | Number of Deaths | Age-Adjusted Mortality Rates[3] | | Age-Adjusted Premature Mortality Rates to Age 75[3] | |
		AAMR[4]	Change since 1992-94[5]	YPLL[4]	Change since 1992-94[5]
All Residents	835	8.3	ns	276.5	ns
All males	647	13.6	ns	434.9	ns
White	586	13.7	ns	426.8	ns
Black	54	12.5	ns	514.8	ns
Asian PI	4	—		—	
Native American	2	—		—	
Hispanic	34	8.9*	ns	346.1	ns
All females	188	3.6	ns	119.2	ns
White	173	3.7	ns	123.3	ns
Black	12	—		—	
Asian PI	2	—		—	
Native American	1	—		—	
Hispanic	5	—		—	

Notes:

1. This cause of death category includes ICD-9 codes E950-E959.

2. Racial groupings (White, Black, Asian & Pacific Islander, Native American) include persons of Hispanic ethnicity.

3. Age-adjusted Mortality Rates (AAMR) and Years of Potential Life Lost (YPLL) rates are per 100,000 based on race and ethnicity specific population estimates. Age-adjusted rates were calculated by the direct method using the 2000 U.S. standard population. Rates were not calculated for fewer than 15 events.

4. Statistical tests were conducted to evaluate differences in rates between race/ethnic groups. The white population serves as the reference group in each comparison (black vs. white, Asian & PI vs. white, Native American vs. white, Hispanic vs. white). Following are explanations of the notations:

    *   Significantly different from the respective white resident rate at $p < .05$.
    —   Rate was not calculated due to small numbers.

5. Statistical tests were conducted to evaluate changes in rates over time. Comparisons of 1996-98 vs. 1992-94 rates are made within each race/ethnicity group. Following are explanations of the notations:

ns   Indicates the change from 1992-94 to 1996-98 is not statistically significant.

Figure 17.2.

Age-specific Rates for Suicide Deaths and
Proportionally Adjusted Rates for All Other Causes, 1996 - 1998

Age-specific Deaths Rates and Trend Lines

● Suicide rates and trend
– All Other Causes trend (rates are not displayed)

Prevention

Suicide prevention efforts should be based on risk factors that have been identified in clinical research (Table 17.3) [U.S. Department of Health and Human Services 1999]. Recommended strategies include a long-term integrated approach that treats individuals and their underlying conditions in the context of their physical and social environments. Recommended community-level strategies include restriction of access to firearms and prescriptive medications, two commonly used methods of suicide (Moscicki 1997).

A public health approach to suicide prevention has been outlined in the *National Strategy for Suicide Prevention: Goals and Objectives for Action* a collaboration of federal government agencies. The strategy aims to promote efforts to modify the social infrastructure that will influence public attitudes about suicide and that will modify judicial, educational, social service, and health care systems. It also emphasizes the coordination of resources and culturally appropriate services at all levels of government and with the private sector (Table 17.4). Some goals of this national strategy include developing broad-based support for suicide prevention; implementing strategies to reduce the stigma of being a user of mental health services; implementing suicide prevention programs; reducing access to lethal means and methods of self-harm; improving access to and community linkages with mental health and substance abuse services; improving the portrayals of suicidal behavior, mental illness, and substance abuse in the media; and improving and expanding surveillance systems (U.S. Department of Health and Human Services 2001a).

The U.S. Preventive Services Task Force does not recommend that primary care clinicians routinely screen asymptomatic patients for suicide. The Task Force, however, does recommend that clinicians routinely ask patients about drug and alcohol use; that they be alert to signs and symptoms of depression; and that they recognize the signs of suicidal ideation in persons with established risk factors (U.S. Preventive Services Task Force 1996).

Figure 17.3.

Suicide Deaths
Comparison of Connecticut and U.S. 1989-1998
Age-Adjusted Mortality Rates* by Sex

Symbols:
■ - U.S. rates for 1989-1998
✳ ● - Connecticut rates: an asterisk is used if the rate is significantly different than expected (p<.005), based on the log-linear trend.

* These rates are adjusted to the 1940 US standard million population.
* This classification includes deaths with ICD-9 codes:E950-E959

**Table 17.2. Suicide Age-Adjusted Death Rates,
Comparison of CT with US - 1992 and 1998**

	1992	1998	1998 CT AAMR Comparison
CT AAMR*	8.5	7.5	
US AAMR*	11.2	10.4	CT AAMR < US AAMR
*Healthy People 2000**	10.5	10.5	CT AAMR < HP 2000 rate
*Healthy CT 2000**	6.7	6.7	not significantly different

* age-adjusted mortality rates for suicide are per 100,000 population, U.S. 1940 standard million population.

Table 17.3. Risk Factors for Suicide

- Previous suicide attempt
- Mental disorders
- Co-occurring mental and alcohol and substance abuse disorders
- Family history of suicide
- Feelings of hopelessness
- Impulsive and /or aggressive tendencies
- Barriers to accessing mental health treatment
- Relational, social, work, or financial loss

- Physical illness
- Easy access to lethal methods, i.e. guns
- Unwillingness to seek help because of stigma attached to mental and substance abuse disorders and/or suicidal thoughts
- Influence of significant people who have died by suicide
- Cultural and religious beliefs
- Isolation from other people
- Local epidemics of suicide

Source*:* U.S. Department of Health and Human Services 1999.

Table 17.4. Aims of the National Strategy

- Prevent premature deaths due to suicide across the life span

- Reduce the rates of other suicidal behaviors

- Reduce the harmful after-effects associated with suicidal behaviors and the traumatic impact of suicide on family and friends

- Promote opportunities and settings to enhance resiliency, resourcefulness, respect, and interconnectedness for individuals, families, and communities

Source*:* U.S. Department of Health and Human Services 2001a.

Homicide and Legal Intervention
(ICD-9 codes E960-E978)

Homicide deaths include those inflicted by another person with the intention to injure or kill while legal intervention deaths encompass those inflicted by police or other law enforcement officials in the course of a legal action as well as legal executions. During the 1989-1998 period, 1,763 Connecticut residents died as a result of homicide (99%) or legal intervention (1%). During the ten-year period, Connecticut rates paralleled, but were consistently lower than, national rates for this time period (Figure 18.1, Table 18.1).

Certain population groups are at higher risk for homicide and legal intervention death. Males were more than three times as likely as females to die from homicide or legal intervention during the 1996-1998 period. Males accounted for 77% of all deaths and 78% of all premature mortality to age 75 (Table 18.2). Young men between the ages of 15-34 accounted for about 54% of all homicide and legal intervention victims in Connecticut during this time period. Figure 18.2 depicts the relationship of age-specific homicide and legal intervention death rates with proportionally adjusted rates for all other causes of death. Males up to age 54 and females up to age 64 had higher rates of death from homicide and legal intervention relative to proportionally adjusted rates from all other causes of death.

The concentration of homicide deaths among younger people is also reflected in summary measures of premature mortality to age 75. While homicide and legal intervention ranks thirteenth as a leading cause of death among Connecticut male residents, it is the sixth-ranked leading cause of death by years of potential life lost to age 75. Homicide and legal intervention ranks fifteenth as a leading cause of death among Connecticut female residents, but it is the ninth-ranked leading cause of death by years of potential life lost to age 75 (Appendix V-A and V-D).

1996-1998 Homicide & Legal Intervention Deaths, Connecticut Residents

- Fifteenth leading cause of death for all CT residents

- Second leading cause of death for age groups 15 to 24

- Sixth leading cause of premature death (at age 75) for all CT residents

- 77% were male

- 65% were firearm homicides

Figure 18.1.

Black and Hispanic males and black females were at highest risk for homicide and legal intervention deaths during the decade. Although black residents comprised about 8% of the population, they accounted for about 45% of all homicide deaths in Connecticut. While Hispanic residents were about 7% of the total population, they accounted for about 19% of all homicide deaths. More than 80% of homicide deaths in the black and Hispanic population occurred among men, compared with about 70% among whites. Young black men were at particularly high risk for death. While homicide and legal intervention accounted for less than 1% of all deaths among Connecticut residents from 1989-1998, it accounted for about 38% of all deaths among black males aged 15 to 34.
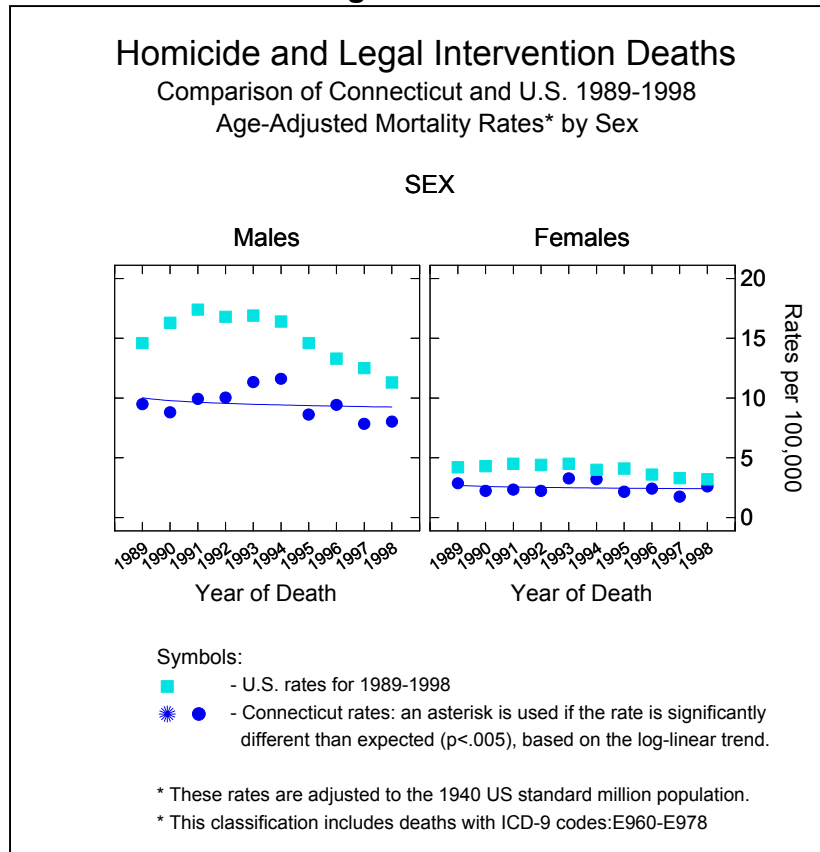
Homicide and Legal Intervention Deaths
Comparison of Connecticut and U.S. 1989-1998
Age-Adjusted Mortality Rates* by Sex

Symbols:

■ - U.S. rates for 1989-1998

✳ ● - Connecticut rates: an asterisk is used if the rate is significantly different than expected (p<.005), based on the log-linear trend.

* These rates are adjusted to the 1940 US standard million population.
* This classification includes deaths with ICD-9 codes:E960-E978

During the period 1996-1998, black males had about five times and Hispanic males almost twice the mortality from homicide and legal intervention as did white males. Black females had about four times the mortality relative to white females (Table 18.1). Logistic regression analyses of the black-white male, Hispanic-white male, and black-white female homicide mortality show that the disparities were consistent across age groups. Premature mortality rates for black and Hispanic males and black females were significantly higher than those of white males and females, respectively (Table 18.2). There were too few homicide and legal intervention deaths among Hispanic females and Asian and Pacific Islander and Native American males and females to calculate reliable mortality rates (Table 18.2).

During the decade, homicide and legal intervention death rates for males did not follow a linear trend, showing a tendency to increase from 1989 until 1994 followed by a decreasing trend from 1994 to 1998. Homicide and legal intervention death rates did not change significantly for females during the decade (Table 18.2).

**Table 18.1. Homicide and Legal Intervention Age-Adjusted Death Rates,
Comparison of CT with US - 1989 and 1998**

	1989	1998	1998 CT AAMR Comparison
CT AAMR*	6.2	5.3	
US AAMR*	9.4	7.3	CT AAMR < US AAMR

* age-adjusted mortality rates for homicide and legal intervention are per 100,000 population, U.S.
1940 standard million population.

About 68% of all homicide and legal intervention deaths for 1989-1998 (65% for the 1996-1998 period) were by firearms. Trends in firearm-related homicides among Connecticut residents were parallel to, but consistently lower than, U.S. rates. Firearm-related homicides increased steadily in the 1980s and early 1990s. Since 1993, there has been a steady decrease in firearm-related homicides both nationally and in Connecticut. This trend has been attributed to successful interventions like focused policing efforts and handgun control legislation, more restrictive licensure requirements for firearms dealers, comprehensive tracing of firearms used in a crime, and background checks on handgun purchasers (Wintemute 1999).

Risk Factors

Risk factors for homicide are best viewed in the context of interpersonal and community relationships. Research points to several individual-level factors that increase the probability of violence during adolescence and young adulthood: exposure to violence; social problem-solving skill deficits; power differentials in interpersonal relationships; negative peer influences; and access to firearms (Dahlberg 1998). Interpersonal violence exists within a community subculture (Parker and Toth 1990). Research has identified residence in neighborhoods with high poverty, transiency, family disruption, and social isolation as particularly influential risk factors for increasing the probability of youth violence (Dahlberg 1998).

The 2001 report *Youth Violence: A Report of the Surgeon General* groups risk factors for youth violence into five domains: individual, family, peer group, school, and community. Research suggests that there are two developmental periods when violent behaviors emerge—before and after puberty--each with distinct risk factors, which should be addressed in prevention programs (U.S. Department of Health and Human Services 2001b).

Table 18.2. Homicide and Legal Intervention Deaths[1], Connecticut Residents by Gender, Race and Ethnicity[2], 1996-1998

| Group | Number of Deaths | Age-Adjusted Mortality Rates[3] | | Age-Adjusted Premature Mortality Rates to Age 75[3] | |
		AAMR[4]	Change since 1989-91[5]	YPLL[4]	Change since 1989-91[5]
All Residents	452	4.8	ns	223.7	ns
All males	346	7.4	ns	348.5	ns
White	158	3.8	ns	173.7	ns
Black	179	38.4***	ns	1,783.8***	ns
Asian PI	4	—		—	
Native American	1	—		—	
Hispanic	60	14.6***	ns	633.5***	ns
All females	106	2.1	ns	95.5	ns
White	61	1.4	ns	57.9	ns
Black	42	8.3***	ns	395.7***	ns
Asian PI	1	—		—	
Native American	2	—		—	
Hispanic	9	—		—	

Notes:

1. This cause of death category includes ICD-9 codes E960-E978.

2. Racial groupings (White, Black, Asian & Pacific Islander, Native American) include persons of Hispanic ethnicity.

3. Age-adjusted Mortality Rates (AAMR) and Years of Potential Life Lost (YPLL) rates are per 100,000 based on race and ethnicity specific population estimates. Age-adjusted rates were calculated by the direct method using the 2000 U.S. standard population. Rates were not calculated for fewer than 15 events.

4. Statistical tests were conducted to evaluate differences in rates between race/ethnic groups. The white population serves as the reference group in each comparison (black vs. white, Asian & PI vs. white, Native American vs. white, Hispanic vs. white). Following are explanations of the notations:

    *** Significantly different than the respective white resident rate at p < .001.
    —   Rate was not calculated due to small numbers.

5. Statistical tests were conducted to evaluate changes in rates over time. Comparisons of 1996-98 vs. 1989-91 rates are made within each race/ethnicity group. Following are explanations of the notations:

    ns   Indicates the change from 1989-91 to 1996-98 is not statistically significant.

Figure 18.2.

Age-specific Rates for Homicide and Legal Intervention Deaths and
Proportionally Adjusted Rates for All Other Causes, 1996 - 1998

Prevention

Violence has been identified as a public health priority issue by the Centers for Disease Control and Prevention and the research community because it exacts an enormous toll in both mortality and morbidity (Rosenberg 1988; Whitman 1988; Edelman and Satcher 1993; Moore 1993; Prothrow-Stith 1995; Rosenberg 1995; Dahlberg 1998; Asencio 1999; U.S. Department of Health and Human Services 2001b).

Violence disrupts the quality of life in, and cohesion of, communities. A public health approach to reducing violence is one that first examines patterns, risk factors, and causes; designs appropriate interventions and evaluations; and creates effective programs (Edelman and Satcher 1993; U.S. Department of Health and Human Services 2001b).

Violence prevention efforts should include interventions at multiple levels-- individual, family, community, and society. Effective programs focus on young children and their parents emphasizing changes in knowledge, skills, and attitudes. Community-based efforts emphasize changing the social environment, such as increasing opportunities for adequate housing, job training or employment, and academic achievement (Edelman and Satcher 1993; Rosenberg 1995; U.S. Department of Health and Human Services 2001b). Local interventions that identify and target sources of firearm violence such as youth gangs and focus on modifying multiple risk factors have also demonstrated success (Wintemute 1999). Although programs that address the primary risk factors for violence exist

throughout the United States, evaluations to assess program effectiveness are still needed (Rosenberg 1995; Powell and Hawkins 1996; U.S. Department of Health and Human Services 2001b).

Poisoning
(ICD-9 codes E850-E869, E950-E952, E962, E972, E980-E982)

Poisoning deaths may result from drug use, inhalation or ingestion of toxic fumes or substances, or alcohol binge drinking. While most poisoning deaths are unintentional, some are suicides, homicides, or of undetermined intent. Not all deaths in which toxic substances play a part are counted as poisonings based on the National Center for Health Statistics (NCHS) definition used in this report. For example, deaths due to falls or suicide by hanging in which narcotics use was involved are not considered poisonings; rather, they are classified as falls and suicides, respectively. Thus, poisoning mortality rates understate the extent to which toxic substances contribute to mortality. The E-codes for most poisoning deaths specify both the intent and causative agent of the poisoning. Code E860.0, for example, refers to accidental poisoning by alcoholic beverage (Fingerhut and Cox 1998).

During the period 1989-1998, 2,371 Connecticut residents died as a result of poisoning. Poisoning was the fourth-ranked leading cause of injury mortality for Connecticut residents, behind motor vehicle accidents, suicide, and firearms. The most common types of poisoning deaths for Connecticut residents (1996-1998) were accidental deaths from opiates (heroin) and other narcotics, cocaine, and other drugs (63% of all poisoning deaths); and suicides by drugs and carbon monoxide and other gases (22%) [Figure 19.1].

Poisoning mortality rates appeared to increase during the ten-year period for both men and women; however, some of the apparent increase is due to improved classification of drug-related causes of death beginning in 1992. For this reason, the trend analysis presented here covers the period from 1992 to 1998.

For the period 1996-1998, Connecticut males were almost three times more likely than females to die as a result of poisoning (Table 19.1). Sixty-four percent of all poisoning deaths were among males ages 20 to 54. Death rates peaked in the 35-44 age group (Appendix VII A). Poisoning was the leading cause of injury mortality in this age group surpassing motor vehicle crashes. Figure 19.2 depicts male and female age-specific death rates for poisoning and all other remaining causes of

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1996-1998 Poisoning Deaths, Connecticut Residents

- Leading cause of injury mortality in Connecticut

- 63% were accidental drug deaths

- Ratio of male to female mortality — 3: 1

- 64% were males aged 20 to 54

- Connecticut mortality was significantly higher than U.S. mortality

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**Figure 19.1. Poisoning Deaths, Percent by Type
Connecticut Residents, 1996-1998**
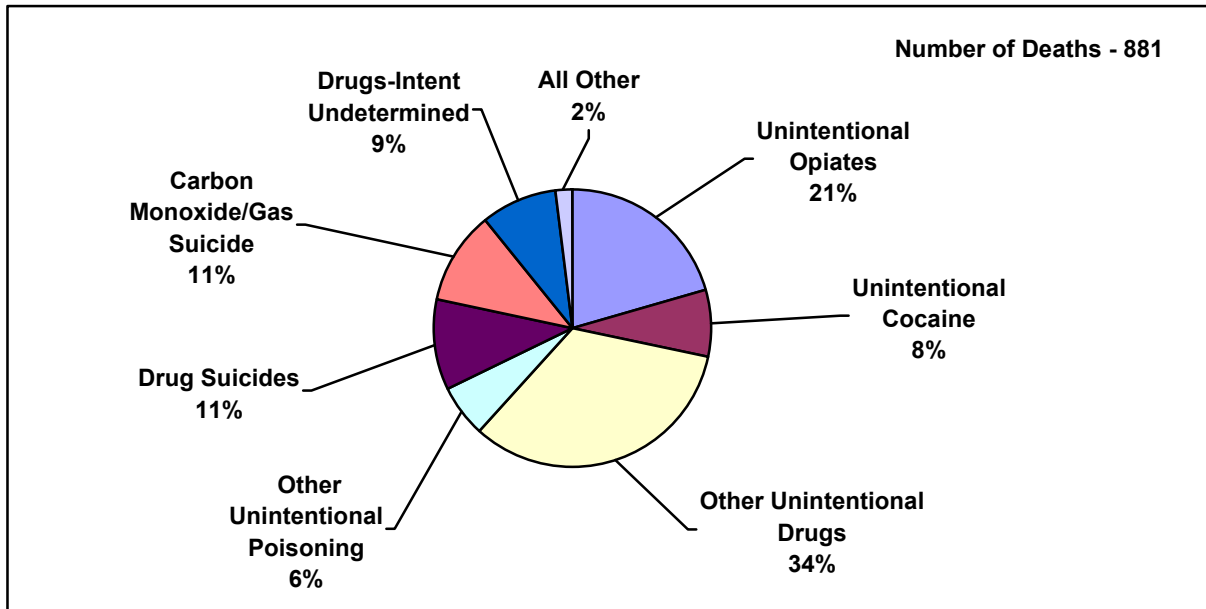
death. Poisoning death rates exceed proportionally adjusted rates due to all other causes of death for males ages 5 to 54 and for females up to age 59, after which point they decrease relative to all remaining causes of death.

For the period 1996-1998, the highest age-adjusted rates of poisoning deaths occurred among Hispanic and black males, with rates about 1.5 times higher than comparable rates for white males (p < .05). The black-white and Hispanic-white disparity was consistent across five-year age groups. There were no significant differences in mortality rates of white and black females for this same period. There were too few poisoning deaths among Hispanic females and Asian and Pacific Islander and Native American males and females to calculate reliable rates for this period (Table 19.1).

Between the periods 1992-1994 and 1996-1998, poisoning death rates did not increase significantly for all Connecticut residents or racial/ethnic subpopulations. Poisoning mortality did increase significantly for female residents, however, and this change is accounted for by a significant increase in poisoning mortality within the white female population (Table 19.1). Although there were no apparent statistically significant increases in any of the major subcategories of poisoning deaths for Connecticut females, some of the increase in the poisoning death rate may be attributable to increases in opiate and related narcotics deaths. This category includes deaths due to heroin, methadone, morphine, codeine, opium, and pethidine. Opiate and related narcotic deaths accounted for about 9% of all poisoning deaths among Connecticut female residents in 1992, compared with

Table 19.1. All Poisoning Deaths[1], Connecticut Residents by Gender, Race and Ethnicity[2], 1996-1998

Group	Number of Deaths	Age-Adjusted Mortality Rates[3] AAMR[4]	Change since 1992-94[5]	Age-Adjusted Premature Mortality Rates to Age 75[3] YPLL[4]	Change since 1992-94[5]
All Residents	881	8.7	ns	312.5	ns
All males	639	12.9	ns	465.9	ns
White	566	12.8	ns	471.8	ns
Black	71	18.6*	ns	619.8	ns
Asian PI	1	—		—	
Native American	1	—		—	
Hispanic	73	19.5*	ns	749.2*	ns
All females	242	4.6	↑	161.4	↑
White	216	4.7	↑	162.6	↑
Black	24	5.1	ns	185.2	ns
Asian PI	1	—		—	
Native American	1	—		—	
Hispanic	15	—		—	

Notes:

1. This cause of death category includes ICD-9 codes E850-E869, E950-E952,E962,E972,E980-E982.

2. Racial groupings (White, Black, Asian & Pacific Islander, Native American) include persons of Hispanic ethnicity.

3. Age-adjusted Mortality Rates (AAMR) and Years of Potential Life Lost (YPLL) rates are per 100,000 based on race and ethnicity specific population estimates. Age-adjusted rates were calculated by the direct method using the 2000 U.S. standard population. Rates were not calculated for fewer than 15 events.

4. Statistical tests were conducted to evaluate differences in rates between race/ethnic groups. The white population serves as the reference group in each comparison (black vs. white, Asian & PI vs. white, Native American vs. white, Hispanic vs. white). Following are explanations of the notations:

    * Significantly different than the respective white resident rate at $p < .05$.
    — Rate was not calculated due to small numbers.

5. Statistical tests were conducted to evaluate changes in rates over time. Comparisons of 1996-98 vs. 1992-94 rates are made within each race/ethnicity group. Following are explanations of the notations:

    ↑ 1996-98 rate is significantly higher than the 1992-94 rate at $p < .05$.

    ns Indicates the change from 1992-94 to 1996-98 is not statistically significant.

Figure 19.2.

Age-specific Rates for All Poisoning Deaths and
Proportionally Adjusted Rates for All Other Causes, 1996 - 1998

about 13% in 1998. The shift in type of poisoning death follows a similar (but not statistically significant) pattern to that of males. Opiate and related narcotic deaths accounted for about 16% of all poisoning deaths among Connecticut male residents in 1992, compared with 27% in 1998.
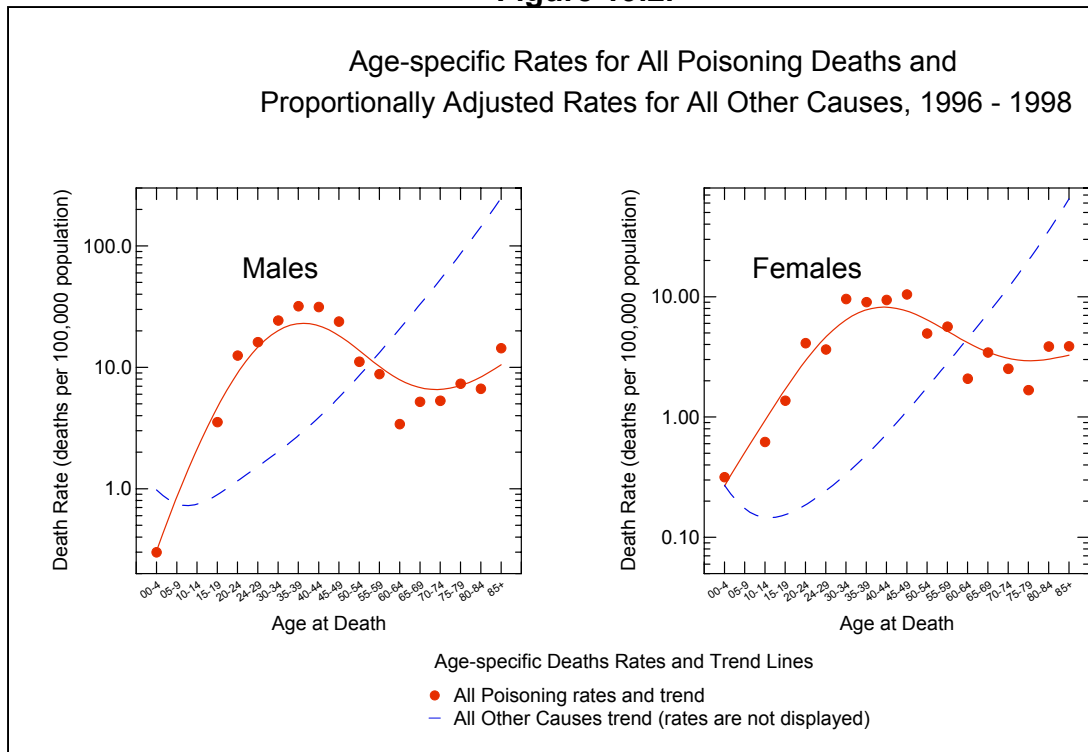
Since 1992, Connecticut poisoning mortality rates have consistently exceeded U.S. rates (Table 19.2). Some of the disparity in these rates may be attributable to differences in opiate and related narcotic death rates. In 1997, for example, opiate and related narcotics accounted for 13% of all poisoning deaths nationwide (Centers for Disease Control and Prevention, 2000) compared with 23% in Connecticut. The recent increase in opiate production and use and its relationship to mortality is discussed in the drug-induced mortality section. Male and female mortality rates for U.S. and Connecticut residents (1989-1998) are depicted in Figure 19.3.

Risk Factors

Risk factors for poisoning mortality overlap with those for alcohol-induced and drug-induced mortality. They are discussed in the alcohol-induced and drug-induced mortality sections in the following pages.

Prevention

Prevention efforts aimed at reducing risk for poisoning mortality should first, be tailored to the mechanism (drugs, alcohol, or carbon monoxide) and intent (unintentional or suicide). Secondly, efforts should address high-risk groups in the population (subgroups of males between the ages of 20 and 54 and females aged 30 to 49) and any groups that have shown a significant increase over time (white females in Connecticut). Prevention efforts for poisoning overlap with those for alcohol and drug abuse and so are discussed in the alcohol-induced and drug-induced mortality sections of this report.

Figure 19.3.

All Poisoning Deaths
Comparison of Connecticut and U.S. 1989-1998
Age-Adjusted Mortality Rates* by Sex

SEX

Males

Females

Year of Death

Year of Death

Rates per 100,000

Symbols:

■ - U.S. rates for 1989-1998

✸ ● - Connecticut rates: an asterisk is used if the rate is significantly different than expected (p<.005), based on the log-linear trend.

* These rates are adjusted to the 1940 US standard million population.

* This classification includes deaths with ICD-9 codes:E850-E869,E950-E952, E962,E972,E980-E982

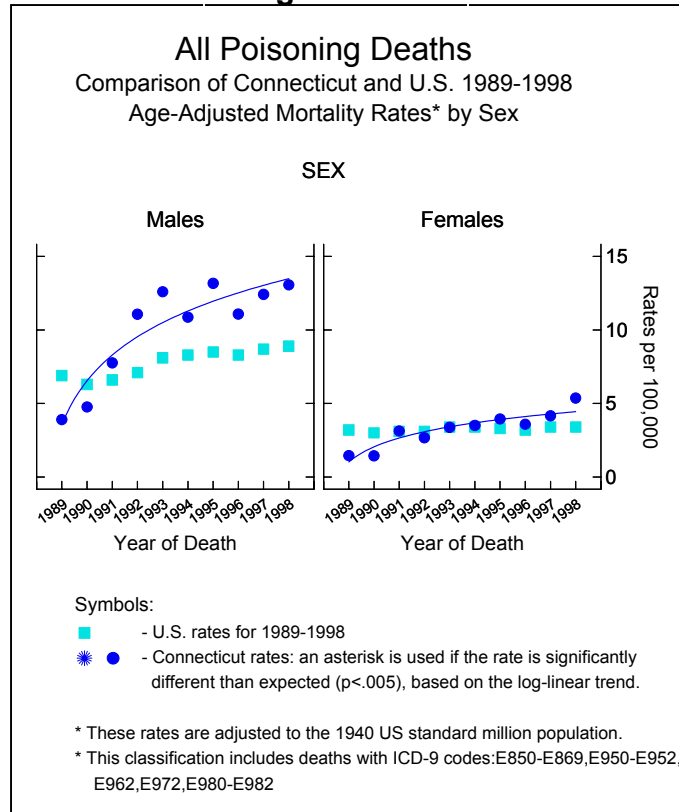**Table 19.2. Poisoning Age-Adjusted Death Rates, Comparison of CT with US - 1992 and 1998**

	1992	1998	1998 CT AAMR Comparison
CT AAMR*	6.8	9.1	
US AAMR*	5.3	6.3	CT AAMR > US AAMR

* age-adjusted mortality rates for poisoning are per 100,000 population, U.S. 1940 standard million population.

Alcohol-Induced
(ICD-9 codes 291, 303, 305.0, 357.5, 425.5, 535.3, 571.0-.3, 790.3, E860)

During the 1989-1998 period, 1,762 Connecticut residents died from alcohol-induced causes. Alcohol-induced mortality includes deaths due to alcohol dependence syndrome; nondependent abuse of alcohol; chronic liver disease and cirrhosis due to alcohol; alcoholic psychoses; and alcoholic polyneuropathy, cardiomyopathy and gastritis. It excludes accidents, homicides, other causes indirectly related to alcohol use, and deaths due to fetal alcohol syndrome. As such, it understates the extent to which alcohol contributes to mortality (Murphy 2000). Major subcategories of alcohol-induced deaths among Connecticut residents for the 1996-1998 period include chronic liver disease and cirrhosis, alcohol dependence syndrome, accidental poisoning, and nondependent abuse of alcohol (Figure 20.1).

Alcohol-induced mortality differs dramatically by gender and age group. During the period 1996-1998 males were about three times more likely to die of alcohol-induced causes than were females (Table 20.1). Males in the 45-69 age group accounted for about 44% of all alcohol-induced Connecticut resident deaths (Appendix VII A).

Figure 20.2 depicts age-specific alcohol-induced death rates for males and females compared with proportionally adjusted rates for all other causes. Alcohol-induced death rates for males and females aged 30 to 64 were higher than the respective death rates for all remaining causes.

Hispanic male residents of Connecticut had the highest mortality and premature mortality rates of all subpopulation groups (Table 20.1). They were almost twice as likely as white males to die of alcohol-induced causes and suffered significantly higher premature mortality to age 75 compared with white males. The Hispanic-white male mortality disparity was consistent across five-year age groups. Age-adjusted and premature mortality rates for black males and females were not significantly different from those for white males and females, respectively. There were too few alcohol-induced deaths among Hispanic females and Asian and Pacific Islander and Native American males and females to calculate reliable rates (Table 20.1).

1996-1998 Alcohol-Induced Deaths, Connecticut Residents

- 57% were from chronic liver disease & cirrhosis

- Ratio of male to female mortality — 3 : 1

- 44% were males aged 45 to 69 years

- Hispanic males had highest death and premature mortality rates

- Connecticut mortality was significantly lower than U.S. mortality

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**Figure 20.1. Alcohol-Induced Deaths, Percent by Type
Connecticut Residents, 1996-1998**
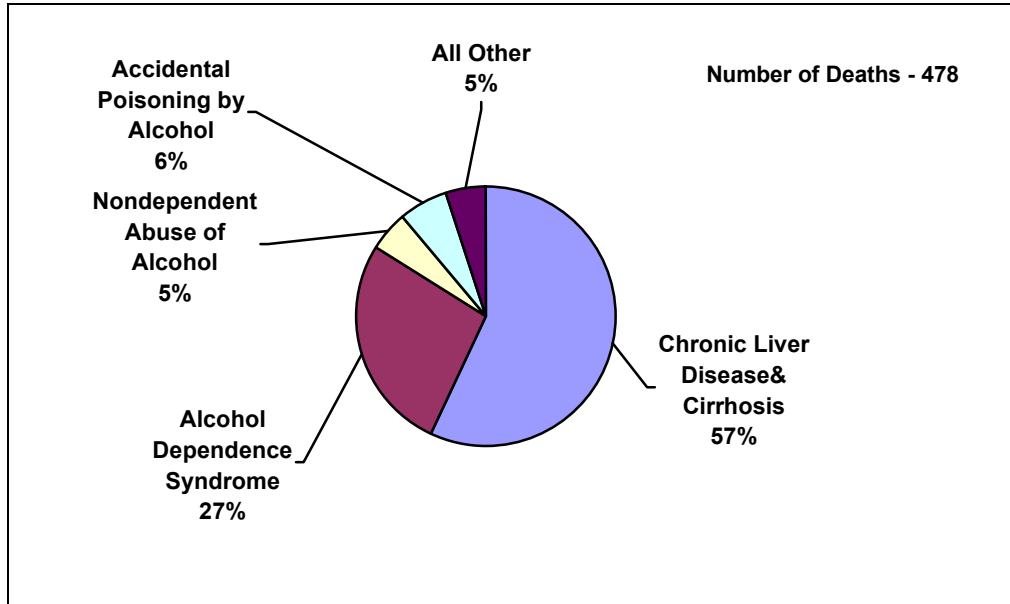
Age-adjusted and premature mortality rates for all Connecticut residents and gender and racial/ethnic subpopulations did not change significantly from the period 1989-1991 to 1996-1998 (Table 20.1).   Since 1989, alcohol-induced mortality among Connecticut residents has been significantly lower than comparable mortality nationwide.   Connecticut male residents had consistently lower alcohol-induced age-adjusted death rates than males nationwide. Connecticut female residents had significantly lower rates than females nationwide in five of the ten years displayed. There are no *Healthy People 2000* or *Healthy Connecticut* targets for alcohol-induced mortality (Figure 20.3, Table 20.2).

Risk Factors

Nondependent heavy drinkers account for most alcohol-related morbidity and mortality (U.S. Preventive Services Task Force 1996).   Risk factors for drinking during adolescence are considered important because experimentation with drinking increases dramatically during this period (Simons-Morton, Haynie, Crump et al. 2001).   Alcohol is the most commonly used drug among young Americans. Recent survey estimates indicate that 50% of high school students reported drinking and 32% reported being drunk in the past month. Binge drinking among college students is widespread and considered to be the most serious public health problem on campuses (Horgan, Skwara, Strickler, et al. 2001). A 2002 national study found that college student drinking (ages 18 to 24) contributes to

**Table 20.1.   Alcohol-Induced[1] Deaths, Connecticut Residents
by Gender, Race and Ethnicity[2], 1996-1998**

| Group | Number of Deaths | Age-Adjusted Mortality Rates[3] | | Age-Adjusted Premature Mortality Rates to Age 75[3] | |
		AAMR[4]	Change since 1989-91[5]	YPLL[4]	Change since 1989-91[5]
All Residents	478	4.7	ns	101.4	ns
All males	347	7.4	ns	155.8	ns
White	311	7.2	ns	153.2	ns
Black	36	11.7	ns	244.4	ns
Asian PI					
Native American					
Hispanic	34	14.1*	ns	345.9**	ns
All females	131	2.4	ns	49.4	ns
White	117	2.4	ns	47.7	ns
Black	13	—		—	
Asian PI					
Native American					
Hispanic	10	—		—	

Notes:

1. This cause of death category includes ICD-9 codes 291,303,305.0,357.5,425.5,535.3, 571.0-.3,790.3,E860.

2. Racial groupings (White, Black, Asian & Pacific Islander, Native American) include persons of Hispanic ethnicity.

3. Age-adjusted Mortality Rates (AAMR) and Years of Potential Life Lost (YPLL) rates are per 100,000 based on race and ethnicity specific population estimates. Age-adjusted rates were calculated by the direct method using the 2000 U.S. standard population. Rates were not calculated for fewer than 15 events.

4. Statistical tests were conducted to evaluate differences in rates between race/ethnic groups. The white population serves as the reference group in each comparison (black vs. white, Asian & PI vs. white, Native American vs. white, Hispanic vs. white). Following are explanations of the notations:

    *   Significantly different than the respective white resident rate at $p < .05$.
    ** Significantly different than the respective white resident rate at $p < .01$.
    —   Rate was not calculated due to small numbers.

5. Statistical tests were conducted to evaluate changes in rates over time. Comparisons of 1996-98 vs. 1989-91 rates are made within each race/ethnicity group. Following are explanations of the notations:

    ↓   1996-98 rate is significantly lower than the 1989-91 rate at $p < .05$.
    ns Indicates the change from 1989-91 to 1996-98 is not statistically significant.

Figure 20.2.

Age-specific Rates for Alcohol-Induced Deaths and
Proportionally Adjusted Rates for All Other Causes, 1996 - 1998

Age-specific Deaths Rates and Trend Lines
● Alcohol-Induced rates and trend
– All Other Causes trend (rates are not displayed)

**Table 20.2. Alcohol-Induced Age-Adjusted Death Rates,
Comparison of CT with US - 1989 and 1998**

	1989	1998	1998 CT AAMR Comparison
CT AAMR*	5.4	4.1	
US AAMR*	7.4	6.1	CT AAMR < US AAMR

* age-adjusted mortality rates for alcohol-induced causes are per 100,000 population, U.S. 1940 standard million population.

an estimated 1,400 student deaths per year (National Institute on Alcohol Abuse and Alcoholism 2002). National and Connecticut Behavioral Risk Factor Surveillance System survey data show that males comprise a greater proportion of chronic and binge drinkers than do females (Centers for Disease Control and Prevention 2001).

Risk factors for alcohol and drug problems fall into two broad categories: societal and cultural factors that provide the legal and normative expectations for behavior; and individuals and their interpersonal environments, primarily families, schools, and peer groups. Specific risk factors are outlned in Table 20.3.

Costs and Prevention

The social and economic costs of alcohol abuse and alcoholism are staggering. Economic costs in the United States, estimated at $148 billion in 1992, include health care expenditures for problems attributed to alcohol, such as alcohol-related illnesses and trauma; impaired productivity accrued in the form of lost household productivity; and lost earnings due to work not performed. Also included are costs of motor vehicle crashes; crime, such as driving under the influence and interpersonal violence; social welfare costs of alcohol-related impairment; and premature death due to alcohol abuse or excessive long-term consumption (Harwood, Fountain, and Livermore 1998). The social toll of alcoholism, which extends to family members, friends,

Figure 20.3.

Alcohol-Induced Deaths
Comparison of Connecticut and U.S. 1989-1998
Age-Adjusted Mortality Rates* by Sex

Symbols:
■ - U.S. rates for 1989-1998
✱ ● - Connecticut rates: an asterisk is used if the rate is significantly different than expected (p<.005), based on the log-linear trend.

* These rates are adjusted to the 1940 US standard million population.
* This classification includes deaths with ICD-9 codes:291,303,305.0,357.5, 425.5,535.3,571.0-.3,790.3,E860

and co-workers, includes family disruption and violence; the neglect or mistreatment of children; and the loss of loved ones through premature death. Alcoholism also damages social relationships in the workplace and in social networks (Horgan, Skwara, Strickler, et al. 2001).
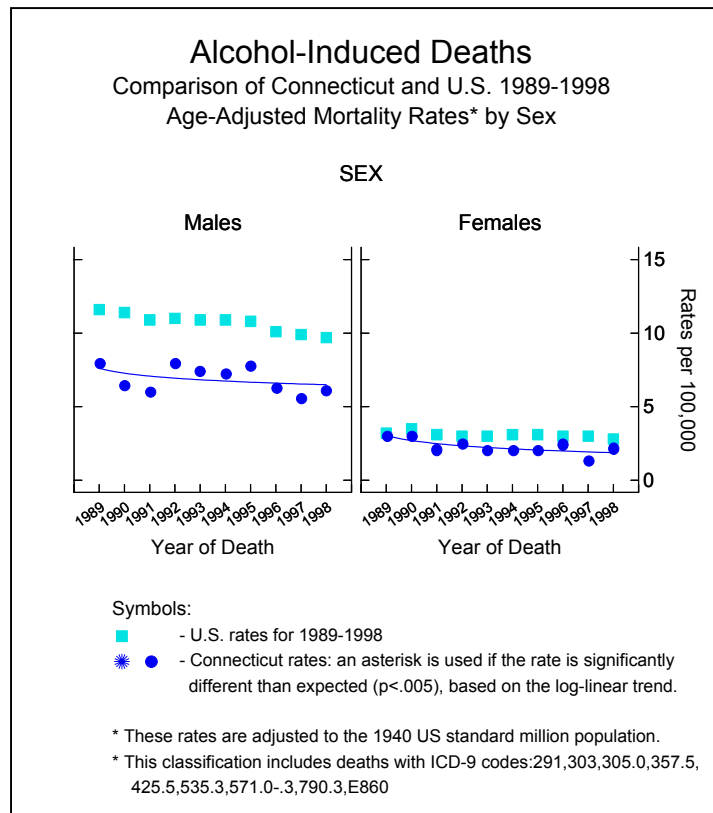
Alcohol prevention efforts tend to target excessive drinking among non-alcoholics, which is much more common than alcoholism. State and community approaches to reducing alcohol consumption include measures such as increased taxes on alcohol, controlling the physical availability and legal accessibility of alcohol, health warning labels, and health information messages and education (Horgan, Skwara, Strickler, et al. 2001). Connecticut Governor Rowland convened a Blue Ribbon Task Force on Substance Abuse in 1995 to identify effective prevention and treatment methods for alcohol and drug addiction in the state. The Connecticut Alcohol and Drug Policy Council (ADPC), an outgrowth of this task force, identified strategies for a comprehensive statewide plan for substance abuse prevention, treatment and enforcement in its 1997 report (Connecticut Alcohol and Drug Policy Council 2001).

The Council submitted a three-year plan in 1999 with a revised plan scheduled for 2002, both of which identify three focus areas--prevention, treatment, and criminal justice. The prevention focus area of the 2002 plan includes three goals: 1) to increase the capacity and improve the effectiveness of the prevention system using empirically grounded approaches and trained staff; 2) to increase successful programming by using age-appropriate strategies and gender-specific models; and 3) to expand the coordination of prevention programs in the state (in the areas of violence, mental illness

Table 20.3. Risk Factors for Alcohol Abuse

Societal / Cultural	*Individuals and Interpersonal Environments*
• Laws (liquor tax, how and to whom liquor is sold)	• Physiological factors (biochemical and genetic susceptibility)
• Cultural norms (exposure to advertising promoting drinking; ethnic and other community norms)	• Family behavior (peer and sibling alcoholism, perceived parent permissiveness toward alcohol use)
• Alcohol availabilility	• Early initiation of alcohol use
• Extreme economic deprivation (in conjunction with childhood behavior problems)	• Early and persistent behavior problems (aggressive behavior that continues into early adolescence; school misconduct)

Source: Hawkins, Catalano, and Miller 1992.

and substance abuse programs, child abuse and neglect, delinquency, school dropouts, teen pregnancy, HIV, and family and youth self-sufficiency). An important related initiative, the Governor's Prevention Initiative for Youth, targets the reduction of alcohol, tobacco, and other drug use among young people ages 12 to 17. A uniform evaluation system was initiated in 2000 to assess all current state-supported substance abuse prevention programs (Connecticut Alcohol and Drug Policy Council 2001).

The U.S. Preventive Services Task Force recommends that clinicians screen for problem drinking in all adult and adolescent patients by taking a detailed history of alcohol use (U.S. Preventive Services Task Force 1996). Studies have shown that primary care clinicians can assist in reducing alcohol consumption levels in problem drinkers who are not alcohol dependent (U.S. Department of Health and Human Services 2003).

Drug-Induced
(ICD-9 codes 292, 304, 305.2-305.9, E850-E858, E950.0-E950.5, E962.0, E980.0-E980.5)

During the period 1989 – 1998, 1,921 Connecticut residents died from drug-induced causes. The number of drug-induced deaths climbed steadily during the decade with more deaths occurring in 1998 than in any previous year. Improved reporting of cause of death beginning in 1991 is a partial explanation for the apparent increase in drug-induced mortality between 1989 and 1992. Beginning with the 1992 mortality data, the Connecticut Department of Public Health (DPH) was able to significantly reduce the number of deaths identified as "pending further investigation" by the Connecticut Medical Examiner's (ME) Office through an improved system of communication. Speedier processing of findings from the ME investigations has resulted in more complete and more accurate cause-of-death classification being entered into the death records. Many drug-induced deaths tend to be included in the category of "pending." For this reason, time trends discussed here begin with the period 1992.

Drug-induced and poisoning mortality include overlapping categories of deaths, specifically, those poisoning deaths that are related to drug use. Drug-induced deaths include those due to drug abuse (excluding tobacco or alcohol), drug dependence, and drug psychoses, as well as unintentional, suicide, and homicide poisonings by use of either legally prescribed or illicit drugs. This definition, which is consistent with that used by the Centers for Disease Control and Prevention (CDC) and the National Center for Health Statistics (NCHS), has some limitations. Reported deaths are based on the underlying (primary) cause of death only. These figures greatly underestimate the mortality related to illicit drug use because they exclude deaths from causes such as AIDS among injecting drug users, hepatitis and tuberculosis, as well as deaths from homicides, and newborn deaths associated with mother's drug use (Murphy 2000).

In the 1996-1998 period, the main subcategories of drug-induced deaths among Connecticut residents included opiates and related narcotics (that is, heroin, methadone, morphine, codeine,

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1996-1998 Drug-Induced Deaths, Connecticut Residents

- Main subcategories: opiates & related narcotics and cocaine

- Ratio of male to female mortality — 2.6: 1

- Males aged 20 to 49 years accounted for 64% of these deaths

- Connecticut mortality was significantly higher than U.S. mortality

- Significant increase in mortality compared with the 1992-94 period

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opium, and pethidine); cocaine; and all other drug overdoses that were unintentional, suicides, or of undetermined intent (Figure 21.1). Males were almost three times more likely than females to die from drug-induced causes (Table 21.1). Deaths were highest in the 20 to 49 year old age groups. Males in these age groups accounted for about 64% of all drug-induced deaths but only 6% of deaths due to other causes (Figure 21.2 and Appendix VII A).

Heroin and related narcotics deaths in Connecticut appeared to increase relative to other drug-induced deaths during the 1992-1998 period, accounting for 21% of all such deaths in 1992 and 26% in 1998. Further analyses of the male and female heroin death rate from the 1992-1994 to 1996-1998 periods, however, do not reveal statistically significant changes. Cocaine deaths appeared to decline relative to other drug induced deaths from 1992 to 1998. Cocaine accounted for about 15% of all drug-induced deaths in 1992 compared with 9% in 1998, while the percentage of deaths from all other drug-induced causes remained about the same in 1992 and 1998. Analyses of the cocaine death rate from the 1992-1994 to 1996-1998 periods, however, did not show statistically significant changes in the Connecticut male or female populations

Changes in heroin use may be linked to increases in the production of heroin worldwide, beginning in 1989 (Hamid, Curtis, McCoy, et al. 1997). Since 1992, there has been a documented increase in heroin use both nationally (Horgan, Skwara, Strickler, et al. 2001) and regionally (Hamid, Curtis, McCoy, et al. 1997). The increase in heroin initiation may be partly explained by the increased profitability in heroin sales, increased purity of the drug, and new modes of use like smoking, snorting, and sniffing rather than intravenous needle injection (Horgan, Skwara, Strickler, et al. 2001; Connecticut Alcohol and Drug Policy Council 2001).

**Figure 21.1. Drug-Induced Deaths, Percent by Type
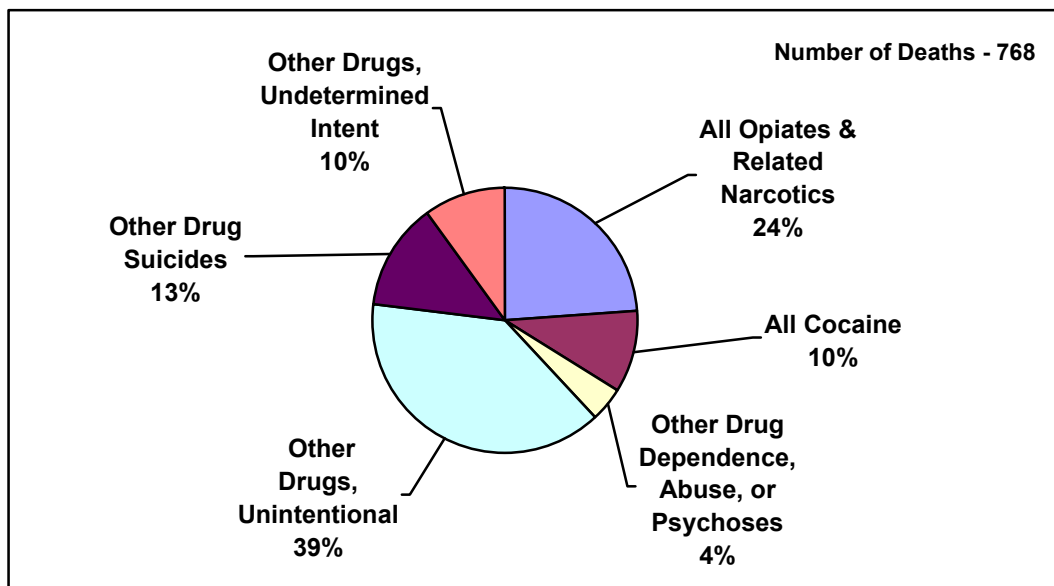Connecticut Residents, 1996-1998**

Number of Deaths - 768

Other Drugs, Undetermined Intent 10%

Other Drug Suicides 13%

All Opiates & Related Narcotics 24%

All Cocaine 10%

Other Drug Dependence, Abuse, or Psychoses 4%

Other Drugs, Unintentional 39%

Table 21.1. Drug-Induced Deaths[1] , Connecticut Residents
by Gender, Race and Ethnicity[2], 1996-1998

Group	Number of Deaths	Age-Adjusted Mortality Rates[3]		Age-Adjusted Premature Mortality Rates to Age 75[3]	
		AAMR[4]	Change since 1992-94[5]	YPLL[4]	Change since 1992-94[5]
All Residents	768	7.5	↑	283.5	↑
All males	547	10.9	ns	418.9	ns
White	479	10.8	↑	421.2	↑
Black	67	17.3*	ns	592.9	ns
Asian PI	1	—		—	
Native American					
Hispanic	72	19.4**	ns	738.5**	ns
All females	221	4.2	↑	149.7	↑
White	198	4.3	↑↑	152.3	↑↑
Black	21	4.5	ns	161.8	ns
Asian PI	1	—		—	
Native American	1	—		—	
Hispanic	15	—		—	

Notes:

1. This cause of death category includes ICD-9 codes 292,304,305.2-.9,E850-E858,E950.0-.5, E962.0,E980.0-.5.
   (*Healthy People 2000* cause of death classification refers to codes 292,304,305.2-.9, E850-E858,E950.0-
   .5,E962.0,E980.0-.5 as "Drug-Related Deaths).

2. Racial groupings (White, Black, Asian & Pacific Islander, Native American) include persons of Hispanic
   ethnicity.

3. Age-adjusted Mortality Rates (AAMR) and Years of Potential Life Lost (YPLL) rates are per 100,000 based on
   race and ethnicity specific population estimates. Age-adjusted rates were calculated by the direct method using
   the 2000 U.S. standard population. Rates were not calculated for fewer than 15 events.

4. Statistical tests were conducted to evaluate differences in rates between race/ethnic groups. The white
   population serves as the reference group in each comparison (black vs. white, Asian & PI vs. white, Native
   American vs. white, Hispanic vs. white). Following are explanations of the notations:

   *   Significantly different than the respective white resident rate at $p < .05$.
   **   Significantly different than the respective white resident rate at $p < .01$.
   —   Rate was not calculated due to small numbers.

5. Statistical tests were conducted to evaluate changes in rates over time. Comparisons of 1996-98 vs. 1992-94
   rates are made within each race/ethnicity group. Following are explanations of the notations:

   ↑   1996-98 rate is significantly higher than the 1992-94 rate at $p < .05$.
   ↑↑   1996-98 rate is significantly higher than the 1992-94 rate at $p < .01$.
   ns   Indicates the change from 1992-94 to 1996-98 is not statistically significant.

Age-specific drug-induced death rates for males and females compared with proportionally adjusted rates for all other causes of death (1996-1998) are depicted in Figure 21.2. Drug-induced death rates for males aged 15-49 and females aged 15-59 exceeded proportionally adjusted rates for all other causes of death, after which point they decrease relative to all other causes of death.

Assessment of the annual percent change in drug-induced mortality for the years 1992-1998 indicates a significant increase of 10.7% for females (p < .001) and 3.9% for males (p < .01). An examination of racial and ethnic subgroup detail provides some additional insight into changes over time.

In the period 1996-1998, black and Hispanic males were 1.6 and 1.8 times, respectively, more likely than white males to die from drug-induced causes (Table 21.1). These disparities were consistent across the five-year age groups. There were no significant differences in the drug-induced death rates of black and white females. Between the periods 1992-1994 and 1996-1998, drug-induced death and premature mortality rates increased for white males and females while they remained about the same for black and Hispanic males and black females. There were insufficient numbers of drug-induced deaths among Hispanic females and Asian and Pacific Islander and Native American males and females to calculate reliable rates for these time periods (Table 21.1).

Since 1992, drug-induced mortality rates tended to be higher among Connecticut male residents than comparable rates nationwide. In 1998, the drug-induced mortality rate was higher among Connecticut female residents than the comparable national rate. Connecticut resident male and female rates exceeded the *Healthy People 2000* target. There is no *Healthy Connecticut* target for drug-induced mortality (Figure 21.3, Table 21.2).

Figure 21.2.

Age-specific Rates for Drug-Induced Deaths and Proportionally Adjusted Rates for All Other Causes, 1996 - 1998
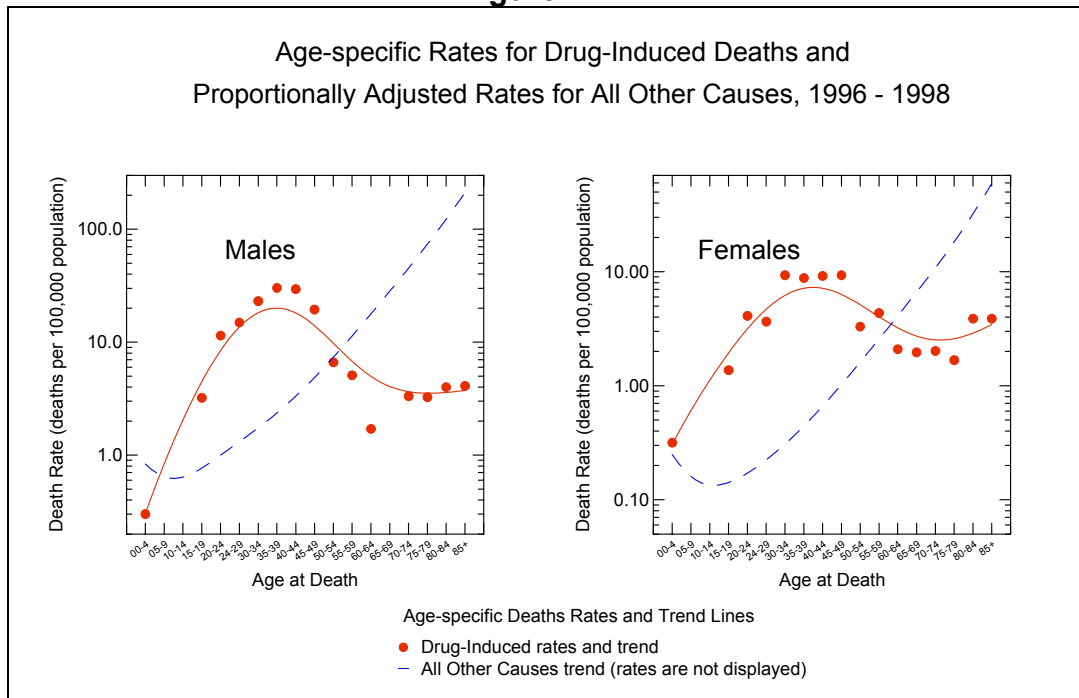
Risk Factors

Research has identified risk factors for drug abuse within two broad categories—societal/cultural norms and personal and interpersonal environments (Hawkins, Catalano, and Miller 1992). Factors include the larger social environment (such as neighborhood norms), early socialization in the family, and social groups of peers and school (Table 21.3). These social settings can also enhance the development of positive social attitudes and behaviors and provide a setting for increased social and self-competency skills, factors which can deter the initiation of drug use (Sloboda and David 1997).

Figure 21.3.

Drug-Induced Deaths
Comparison of Connecticut and U.S. 1989-1998
Age-Adjusted Mortality Rates* by Sex

Symbols:
■ - U.S. rates for 1989-1998
✳ ● - Connecticut rates: an asterisk is used if the rate is significantly different than expected (p<.005), based on the log-linear trend.

* These rates are adjusted to the 1940 US standard million population.
* This classification includes deaths with ICD-9 codes:292,304,305.2-.9, E850-E858,E950.0-.5,E962.0,E980.0-.5

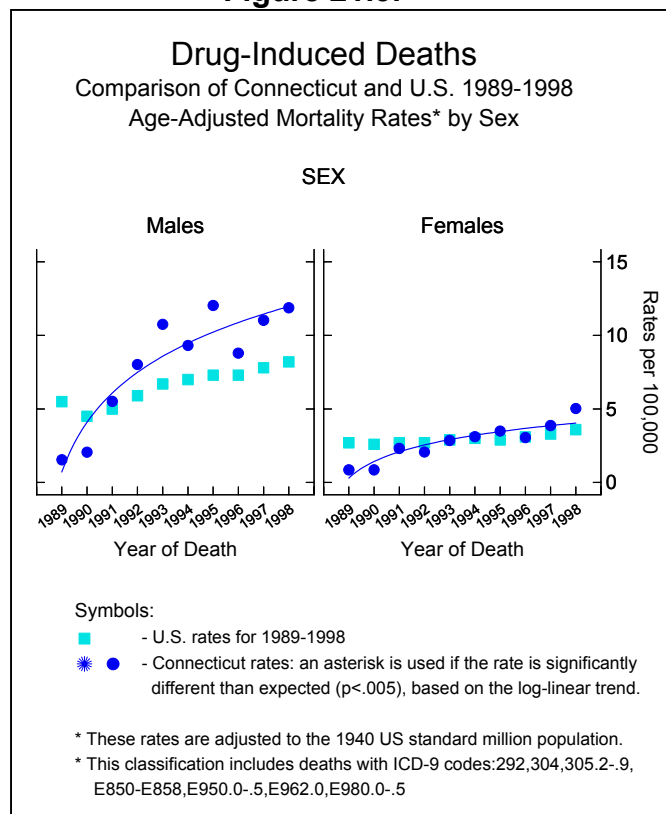**Table 21.2. Drug-Induced Age-Adjusted Death Rates,
Comparison of CT with US - 1992 and 1998**

	1992	1998	1998 CT AAMR Comparison
CT AAMR*	5.0	8.4	
US AAMR*	4.3	5.9	CT AAMR > US AAMR
*Healthy People 2000**	3.0	3.0	CT AAMR > HP 2000 rate

* age-adjusted drug-induced mortality rates are per 100,000 population , U.S. 1940 standard million population.

Table 21.3. Risk Factors for Drug Abuse

Societal / Cultural	*Individuals and Interpersonal Environments*
• Illegal drug availability	• Early and persistent behavior problems (aggressive behavior that continues into early adolescence; school misconduct)
• Extreme poverty (in conjunction with child behavioral problems)	• Academic failure
• Neighborhood disorganization (high population density, high residential mobility, physical deterioration, high adult crime related to high juvenile crime and drug trafficking)	• Ineffective parenting, especially for children with difficult temperaments and conduct disorders
	• Lack of mutual attachment and nurturing
• Perceptions of approval of drug-using behaviors in the school, community, and peer environments	• Family behavior (parental and sibling drug use increase risk of drug use initiation and drug abuse in children); chaotic home environments
	• Affiliations with deviant peers or peers around deviant behaviors

Sources: Hawkins, Catalano, and Miller 1992; Sloboda, Z. and S.L. David 1997.

Costs and Prevention

Total economic costs of drug abuse in the United States, estimated at $98 billion in 1992, include health care expenditures (such as the cost of treatment for health problems attributed to drug abuse, prevention, and rehabilitation); the cost of lost potential productivity due to impairment; the estimated cost of premature death due lifetime earnings lost; crime attributed to illicit drug abuse; and social welfare costs of drug-related impairment (Harwood, Fountain, and Livermore 1998).

The National Institute on Drug Abuse has outlined several important principles for prevention programs gleaned from over two decades of research. They include an emphasis on, and support of, protective factors such as close family communication and parental involvement in children's lives; and secondly, the identification and reduction of known risk factors such as chaotic home environments where the parents may be substance abusers and/or suffer from mental illness (Sloboda and David 1997).

Prevention programs that include general life skills training can increase social competency in areas such as assertiveness, self-efficacy, communication, and peer relationships, thereby enabling youth to resist drugs when offered. Community programs, such as media campaigns and policies that restrict access to drugs, are most effective when complemented by school and family interventions. The school environment serves as an important setting to reach all subpopulations including those specifically at risk for drug abuse, such as children with behavioral problems or with parents who are substance abusers (Sloboda and David 1997). The U.S. Preventive Services Task Force recommends

that clinicians be alert to signs and symptoms of drug abuse in patients and that they refer drug-abusing patients to appropriate specialized treatment (U.S. Preventive Services Task Force 1996).

State efforts to address problems of substance abuse include the Connecticut Alcohol and Drug Policy Council, which identified strategies for a comprehensive statewide plan for substance abuse prevention, treatment, and enforcement in its 1997 report. The Council's 1999 and 2002 plans, identify the three focus areas of prevention, treatment, and criminal justice. The Governor's Prevention Initiative for Youth, targets the reduction of alcohol, tobacco, and other drug use among young people ages 12 to 17. A uniform evaluation system was initiated in 2000 to assess all current state-supported substance abuse prevention programs (Connecticut Alcohol and Drug Policy Council 2001).

Evaluation research findings indicate that prevention programs can be cost-effective (Bukoski, 1997; Bukowski and Evans 1998; Horgan, Skwara, Strickler, et al. 2001; Pentz 1998). It is estimated that for every dollar spent on drug use prevention, communities can save four to five dollars in drug abuse treatment and counseling costs (Pentz 1998).

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INFECTIOUS DISEASE MORTALITY

CONTENTS

Pneumonia and Influenza
## (ICD-9 codes 480-487)

Pneumonia is an inflammation of the lungs caused by bacterial, viral, or fungal infections that affects over three million Americans per year. It may often occur as a complication of influenza, a common viral infection of the respiratory tract (Butler and Schuchat 1999; Sethi 2002). Pneumonia and influenza account for 50,000 to 80,000 deaths per year in the United States (National Coalition for Adult Immunization 1998). In the 1996-1998 period, pneumonia and influenza (P&I) accounted for 3,609 Connecticut resident deaths. Ninety-two percent of these pneumonia and influenza deaths were categorized as "pneumonia, organism unspecified." Those at greatest risk of death from P&I are older persons, persons with compromised immune systems, and persons with pre-existing chronic conditions such as liver, renal, lung, and cardiac diseases (National Coalition for Adult Immunization 1998). Influenza epidemics are associated with increased hospitalizations and mortality from numerous causes in addition to pneumonia, including chronic obstructive pulmonary disease (COPD), congestive heart failure, and septicemia (Thompson, Shay, Weintraub, et al. 2003).

Pneumonia and influenza is the fifth leading cause of death for all Connecticut residents and the fourth leading cause of death for residents aged 85 and over (Appendix V). Connecticut resident death rates due to pneumonia and influenza increase dramatically with age beginning at ages 55 to 59. Connecticut residents 65 years and older, about 17% of Connecticut's total population, accounted for 92% of all P&I deaths in the 1996-1998 period. Age-specific P&I death rates of Connecticut males and females (1996-1998) are depicted in Figure 22.1. P&I age-specific death rates exceed death rates for all other causes combined for males above 79 years of age and for females above 84 years of age.

Pneumonia and influenza mortality rates both nationally and in Connecticut, tend to be higher in men than in women. Age-adjusted P&I death rates were 1.6 times higher for males compared with females during both the 1989-1991 and 1996-1998 periods (p < .001 for both comparisons). Premature mortality was also significantly higher for males compared with females in both time periods. In 1996-1998, males had 1.9 times the premature P&I mortality rate of females (p < .001).

1996-1998 Pneumonia & Influenza Deaths, CT Residents

- Fifth leading cause of death for all Connecticut residents

- Fourth leading cause of death for ages 85 and over

- Significant decrease in white female mortality since the 1989-1991 period

- Significantly higher premature mortality rates for black males and females compared with white males and females

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Age-specific Rates for Pneumonia & Influenza Deaths and for All Other Causes, 1996 - 1998
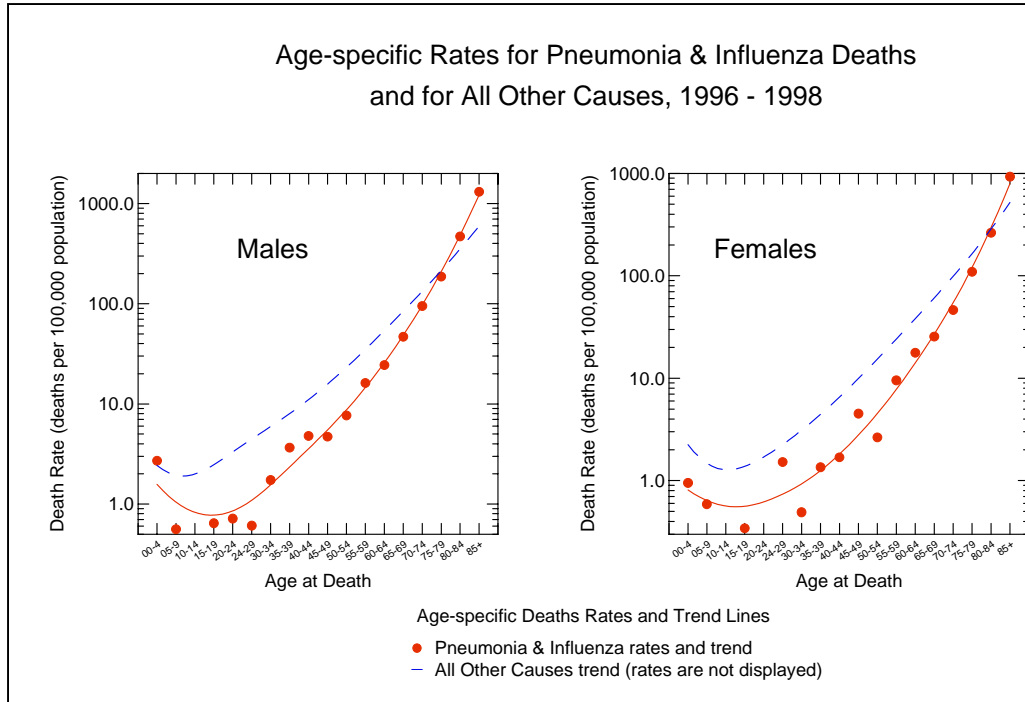
In Connecticut, P&I mortality rates were significantly lower in Hispanic males and females compared with white males and females in both the 1989-1991 and 1996- 1998 periods. There were no significant differences in P&I mortality rates between white and black males and females, respectively, during these same time periods. There were too few P&I deaths among Asian and Pacific Islander and Native American males and females to report reliable rates (Table 22.1). Nationwide, pneumonia and influenza mortality was 69% higher for males than for females and 48% higher for blacks than for whites (Pickle, Mungiole, Jones et al. 1996)

Black males and females had significantly higher premature mortality due to P&I compared with white males and females in Connecticut, respectively, in both the 1989-1991 and 1996-1998 periods. Black males had 3.3 times the premature mortality rate of white males and black females had 2.2 times the premature death rate of white females. There were no significant differences in premature mortality between white and Hispanic males and white and Hispanic females during these periods (Table 22.1).

From 1989 to 1998, age-adjusted death rates for pneumonia and influenza in Connecticut decreased significantly by an average of 1.4% per year (p < .001) for males and by 1.5% for females per year (p < .01). P&I mortality for all Connecticut residents decreased significantly between the periods 1989-1991 and 1996-1998. Among gender and racial/ethnic subpopulations, the decrease was statistically significant for white females only (Table 22.1). Connecticut male and female mortality rates for P&I have tended to be lower than the respective U.S. rates for the period 1989 to 1998 (Figure 22.2, Table 22.2).

Table 22.1. Pneumonia & Influenza Deaths[1]
Connecticut Residents by Gender, Race and Ethnicity[2], 1996-98

Group	Number of Deaths	Age-Adjusted Mortality Rates[3]		Age-Adjusted Premature Mortality Rates to Age 75[3]	
		AAMR[4]	Change since 1989-91[5]	YPLL[4]	Change since 1989-91[5]
All Residents	3,609	32.2	↓↓	96.0	ns
All males	1,621	42.2	ns	125.8	ns
White	1,524	41.9	ns	107.0	ns
Black	87	44.0	ns	352.0***	ns
Asian PI	8	—		—	
Native American	2	—		—	
Hispanic	30	24.9**	ns	129.8	ns
All females	1,988	26.6	↓	67.7	ns
White	1,892	26.4	↓	62.9	ns
Black	91	30.9	ns	136.5*	ns
Asian PI	4	—		—	
Native American	1	—		—	
Hispanic	32	18.4*	ns	73.4	ns

Notes:

1. This cause of death category includes ICD-9 codes 480-487.

2. Racial groupings (White, Black, Asian & Pacific Islander, Native American) include persons of Hispanic ethnicity.

3. Age-adjusted Mortality Rates (AAMR) and Years of Potential Life Lost (YPLL) rates are per 100,000 based on race and ethnicity specific population estimates. Age-adjusted rates were calculated by the direct method using the 2000 U.S. standard population. Rates were not calculated for fewer than 15 events.

4. Statistical tests were conducted to evaluate differences in rates between race/ethnic groups. The white population serves as the reference group in each comparison (black vs. white, Asian & PI vs. white, Native American vs. white, Hispanic vs. white). Following are explanations of the notations:

   * Significantly different than the respective white resident rate at $p < .05$.
   ** Significantly different than the respective white resident rate at $p < .01$.
   *** Significantly different than the respective white resident rate at $p < .001$.
   — Rate was not calculated due to small numbers.

5. Statistical tests were conducted to evaluate changes in rates over time. Comparisons of 1996-98 vs. 1989-91 rates are made within each race/ethnicity group. Following are explanations of the notations:

   ↓ 1996-98 rate is significantly lower than the 1989-91 rate at $p < .05$.
   ↓↓ 1996-98 rate is significantly lower than the 1989-91 rate at $p < .01$.
   ns Indicates the change from 1989-91 to 1996-98 is not statistically significant.

Figure 22.2.

Pneumonia & Influenza Deaths
Comparison of Connecticut and U.S. 1989-1998
Age-Adjusted Mortality Rates* by Sex

Symbols:

■ - U.S. rates for 1989-1998

✸ ● - Connecticut rates: an asterisk is used if the rate is significantly
      different than expected (p<.005), based on the log-linear trend.

* These rates are adjusted to the 1940 US standard million population.
* This classification includes deaths with ICD-9 codes:480-487

Table 22.2. Comparison of CT with US, 1989 and 1998

	1989	1998	1998 Comparison
CT AAMR*	13.0	11.1	---
US AAMR*	13.7	13.2	CT < US AAMR

* age-adjusted mortality rates are per 100,000 population, U.S. 1940 standard million population.

Risk Factors

Elderly persons aged 65 and over, very young children, persons with chronic diseases, and immunocompromised persons are considered at high risk for complications of pneumonia and influenza. Certain other population groups are also considered at risk for complications of P&I because of historically low rates of immunization (Table 22.3).

Among the elderly, those at highest risk of complications from P&I include persons with co-morbid conditions and frequent hospitalizations. The severity of pneumonia in the elderly may not be a result of chronological age per se but rather the presence of underlying illness (Feldman 2001). Respiratory viral infections, especially influenza, oftentimes provoke acute respiratory episodes among persons with chronic underlying conditions, such as COPD and congestive heart failure. This fact underscores the need for effective vaccines and vaccinations for these viruses (Glezen, Greenberg, Atmar, et al. 2000). In 1996-1998, Septicemia was a contributing cause in 10% of Connecticut resident deaths due to P&I.

The influence of low socioeconomic status on increased risk of pneumonia has not been well studied and is not well understood (Butler and Schuchat 1999). The Centers for Disease Control and Prevention (CDC) surveillance data from Greater Atlanta indicate that rates of disease are higher in low-income communities, and higher in blacks than in whites in the lowest income communities; however, in the highest income level communities, there are no differences in pneumonia infection rates of blacks and whites. Recent data examining the

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**Table 22.3. – Risk Groups and Risk Factors
for Complications of Pneumonia & Influenza (Flu)**

Disease / Medical Conditions	**Age**
• immunodeficient conditions	• persons over 65
• diabetes mellitus	• nursing home residents 50 & over
• chronic liver disease	• children, 2-23 months (pneumonia)
• chronic renal disease	• children, 6-23 months (flu)
• chronic lung disease	**Due to Historic Underimmunization**
• chronic cardiac disease	• racial & ethnic minorities
• HIV infection	• low socioeconomic status
• cancer	• inner-city residents
• alcoholism (pneumonia)	• low educational level
	• lacking medical services

Sources: National Coalition for Adult Immunization 1998; U.S. Preventive Services Task Force 1996; Centers for Disease Control and Prevention 2000; File 2000; Bridges, Harper, Fukuda, et al. 2003.

population-level impact of vaccinating infants against pneumococcal disease suggests that more frequent contact with children may be a factor in higher P&I incidence rates among black Americans and that vaccination of children can reduce or eliminate the black-white disparity in the incidence of pneumoccocal disease (Flannery, Schrag, Bennett, et al. 2004).

Case-fatality rates from P&I vary considerably among the developed countries, from a low of 9% in Sweden to 40% in Israel and the United States (National Coalition for Adult Immunization 1998). Such broad cross-national differences suggest that preventive measures can play an important role in reducing mortality from P&I.

Costs and Prevention

Total costs of influenza epidemics in the United States are estimated to exceed $12 billion each year (Nichol, Margolis, Wuorenma, et al. 1994). In 1998, pneumonia and influenza hospitalization charges alone in Connecticut exceeded $170 million with average charges of $14,074 for pneumonia and $13,310 for influenza (Bower 2000).

Recommendations for Vaccination

Vaccination is the primary preventive measure for influenza, pneumonia, and their complications. The U.S. Preventive Services Task Force (USPSTF) recommends annual flu vaccination for persons 65 and older and for persons in high-risk groups. Among the general population under age 65, the efficacy of influenza vaccine in preventing or reducing the severity of the illness is estimated at 70% to 80% (U.S. Preventive Services Task Force 1996). Among older persons the efficacy is somewhat less but still substantial.

The CDC Advisory Committee on Immunization Practices (ACIP) considers October through November to be the optimal time for flu vaccination, although vaccination is still beneficial if received during December and later. ACIP identifies key target groups for immunization as persons 65 and older, infants 6 to 23 months of age and pregnant women, persons 2 years and older with chronic medical conditions, persons aged 50-64 because of their high prevalence of chronic medical conditions, and persons living with or caring for those at high-risk, including health care workers and employees of chronic care facilities (Bridges, Harper, Fukuda, et al. 2003).

USPSTF recommends pneumococcal vaccination of persons 65 and older who are immunocompetent (capable of developing an immune response). USPSTF notes that there is insufficient evidence to recommend for or against pneumococcal vaccination of high-risk immunocompromised individuals, but recommendations for vaccinating may be made for other reasons, such as the high incidence of and case-fatality rates from pneumococcal disease and few adverse effects from the vaccine. Finally, USPSTF states that evidence of the efficacy of vaccination for the general U.S. population is, as yet, not conclusive (U.S. Preventive Services Task Force 1996). ACIP also recommends pneumococcal vaccination for immunocompetent persons 2 years and older with chronic illness or asplenia and for those living in high-risk environments, and for all infants 2 to 23 months of age (Centers for

Disease Control and Prevention 2000). Finally, a recent study suggests that vaccination of young children against pneumococcal disease has resulted in a substantial reduction of pneumococcal disease in all age groups (Whitney, Farley, Hadler, et al. 2003).

Cost-Effectiveness

Flu vaccination of both the elderly and working adults has been shown to improve health and save costs (Riddiough, Sisk, and Bell 1983; Mullooly, Bennett, Hornbrook 1994; Nichol, Margolis, Wuorenma, et al. 1994; Centers for Disease Control and Prevention 1993; Yassi, Kettner, Hammond, et al. 1991; Nichol, Lind, Margolis, et a. 1995; Nichol, Wuorenma, and von Sternberg 1998; Nichol and Goodman 2002; Lee, Matchar, Clements, et al. 2002). A study of the cost-effectiveness of pneumococcal vaccination in the 65 and older population showed that vaccination saved costs both in improved health and reduced medical expenses (Sisk, Moskowitz, Whang, et al. 1997). A second study of the cost-effectiveness of invasive pneumococcal vaccination among 50 to 64 year olds suggests that there were cost savings for both high-risk and low-risk individuals (Sisk, Whang, Butler, et al. 2003).

Factors Associated with and Barriers to Immunization

The national *Healthy People 2000* objective is to increase influenza and pneumococcal vaccination to levels of at least 60% among high-risk persons and those aged 65 and over. In 1997, 67% of Connecticut residents aged 65 and over reported that they had received the flu vaccine in the past year and 43% reported they had ever received the pneumococcal vaccine (Centers for Disease Control and Prevention 1998). The national *Healthy People 2010* objective is to increase influenza and pneumococcal vaccination to levels of at least 90% among persons aged 65 and over. In 2001, 69% of Connecticut residents 65 and over reported that they had received the flu vaccine in the past year and 63% reported they had ever received the pneumococcal vaccine (Centers for Disease Control and Prevention 2002).

National Behavioral Risk Factor Surveillance System (BRFSS) survey data suggest that white Americans, persons with higher education, higher income, health insurance, and a physician visit in the previous year were most likely to have been vaccinated. Persons with poorer perceived health status and co-morbidities were also more likely to have been vaccinated in the past year (Kamal, Madhavan, and Amonkar 2003). Another study of the elderly suggested that the main predictor of pneumococcal vaccination included: a recommendation by a physician's office staff person, a physician's recommendation, and a belief that the vaccine is a wise idea (Zimmerman, Santibanez Fine, et al. 2003).

In 1995, about 60% of white Americans received flu shots, compared with only 50% of Hispanics and 39% of African Americans. Approximately 37% of whites received pneumococcal vaccinations compared with about 24% of Hispanics and 20% of blacks (National Coalition for Adult Immunization 1998). Several factors are associated with lower immunization rates among minority Americans including lack of health insurance, missed opportunities during physician visits, and low physician reimbursement rates (Table 22.4).

Table 22.4. - Barriers to Immunization Among Minority Populations
• Lack of access due to lack of health insurance and/or low socioeconomic status
• Other family factors, such as lack of knowledge about the risks of P&I and the benefits of immunization, language barriers, undocumented status, and lack of stable residence
• Lack of incentive to primary care providers to recommend immunization due to low reimbursement
• Missed opportunities during office visits or hospitalizations to immunize

Source: National Coalition for Adult Immunization 1998.

A recent study points to the importance of organizational characteristics in improving immunization rates. Changes in a given health care organization, including changes in staffing and clinical procedures, have been shown to be the factors most likely to improve rates of appropriate adult immunization according to a meta-analysis of 108 studies of adherence to immunization guidelines. Examples of such organizational features include: changes that identify and make delivery of immunizations part of routine patient care, patient reminders that supplement such organizational changes, and financial incentives to immunize, if not in place already (Stone, Morton, Hulscher, et al. 2002).

Standing orders programs, another example of an organizational change, have been demonstrated to improve vaccination rates in health care institutions. Where allowed by state law, standing orders can authorize nurses and pharmacists to administer vaccinations according to an approved protocol without prior order or examination by a physician. The Advisory Committee on Immunization Practices recommends standing orders programs for use in inpatient and outpatient settings (Centers for Disease Control and Prevention 2003a). Up until recently Connecticut hospitals were required by Connecticut law to obtain an individual physician order for each patient vaccination. Pneumococcal vaccination rates in Connecticut adult acute-care hospitals range from 0% to 94% with a median rate of 41% (Connecticut Department of Public Health 2004a). Effective July 1, 2004, Connecticut hospitals are allowed by law to employ standing orders for pneumonia and flu vaccinations of their patients (Connecticut Department of Public Health 2004b).

With the aging of the Connecticut and U.S. populations, respiratory infectious diseases among the elderly will continue to be a major public health concern. This fact underscores the need for appropriate prevention strategies and health care services (Centers for Disease Control and Prevention 1995).

Septicemia
(ICD-9 code 038)

Septicemia is a bacterial infection of the bloodstream that can be life threatening. It usually occurs when another infection is present in the body, commonly in the abdomen, lungs, skin, or urinary tract. Septicemia was the eighth leading cause of death for all Connecticut residents in both the 1989-1991 and 1996-1998 periods (see Appendix V for leading cause of death tables). Because it is a frequent complication of other fatal conditions, such as pneumonia and influenza, examination of septicemia as the underlying cause of death alone does not accurately represent its extensive contribution to overall mortality. While septicemia was the underlying cause of 3,962 resident deaths from 1989-1998, it was listed as an underlying or contributing cause of death for 14,034 Connecticut residents in the ten-year period.

Nationwide, there was an increasing trend in septicemia mortality from at least the 1930s through the 1980s (Salive, Wallace, Ostfeld, et al. 1993). Increased rates of septicemia have been attributed to a few factors, including: 1) increasing numbers of immunocompromised persons at high risk for infections; 2) increased use of invasive medical procedures and devices that have accompanied high technology medicine; and 3) greater awareness and increased recognition of the disease through diagnostic tests (Salive, Wallace, Ostfeld, et al. 1993; Sands, Bates, Lanken, et al. 1997).

Connecticut resident death rates due to septicemia increase dramatically with age beginning at ages 55 to 59. Connecticut residents 65 years and older, about 17% of Connecticut's total population, accounted for 85% of all septicemia deaths in the 1996-1998 period.

Septicemia mortality rates tend to be higher in men than in women. Age-adjusted septicemia death and premature mortality rates were 1.4 times higher for Connecticut male residents compared with female residents in the 1996-1998 period (p < .001 for both comparisons) [Table 23.1].

1996-1998 Septicemia Deaths, CT Residents

- Eighth leading cause of death for all Connecticut residents

- Significant decrease in all-CT resident and white female mortality since the 1989-1991 period

- Significantly higher death and premature mortality rates for black males and females compared with white males and females

---

Table 23.1   Septicemia Deaths[1]
Connecticut Residents by Gender, Race and Ethnicity[2], 1996-98

Group	Number of Deaths	Age-Adjusted Mortality Rates[3]		Age-Adjusted Premature Mortality Rates to Age 75[3]	
		AAMR[4]	Change since 1989-91[5]	YPLL[4]	Change since 1989-91[5]
All Residents	1,147	10.4	↓↓	55.8	↓
All males	509	12.4	ns	65.6	ns
White	461	11.9	ns	52.7	ns
Black	45	20.0*	ns	211.0***	ns
Asian PI	2	—		—	
Native American	0				
Hispanic	15	—		—	
All females	638	9.1	↓	46.8	ns
White	584	8.7	↓	39.3	ns
Black	52	16.5**	ns	136.0**	ns
Asian PI	0				
Native American	0				
Hispanic	18	9.6	na	66.7	na

Notes:

1. This cause of death category includes ICD-9 codes 38.

2. Racial groupings (White, Black, Asian & Pacific Islander, Native American) include persons of Hispanic ethnicity.

3. Age-adjusted Mortality Rates (AAMR) and Years of Potential Life Lost (YPLL) rates are per 100,000 based on race and ethnicity specific population estimates. Age-adjusted rates were calculated by the direct method using the 2000 U.S. standard population. Rates were not calculated for fewer than 15 events.

4. Statistical tests were conducted to evaluate differences in rates between race/ethnic groups. The white population serves as the reference group in each comparison (black vs. white, Asian & PI vs. white, Native American vs. white, Hispanic vs. white). Following are explanations of the notations:

>   *   Significantly different than the respective white resident rate at p < .05.
>   **   Significantly different than the respective white resident rate at p < .01.
>   *** Significantly different than the respective white resident rate at p < .001.
>   — Rate was not calculated due to small numbers.

5. Statistical tests were conducted to evaluate changes in rates over time. Comparisons of 1996-98 vs. 1989-91 rates are made within each race/ethnicity group. Following are explanations of the notations:

>   ↓   1996-98 rate is significantly lower than the 1989-91 rate at p < .05.
>   ↓↓ 1996-98 rate is significantly lower than the 1989-91 rate at p < .01.
>   ns   Indicates the change from 1989-91 to 1996-98 is not statistically significant.
>   na   1989-91 rate was not calculated due to small numbers and so no comparison with
>   1996-98 rate is available

Septicemia death and premature mortality rates were significantly higher in black males and females compared with white males and females in both the 1989-1991 and 1996-1998 periods. In 1996-1998, black males had 4 times the premature mortality rate of white males and black females had 3.5 times the premature mortality rate of white females (Table 23.1). Septicemia-*related* death and premature mortality rates were also significantly higher in black males and females compared with white males and females in the 1989-1991 and 1996-1998 periods. Black males had 3 times and black females had 2.7 times the premature mortality rate of white males and females, respectively (Table 23.2).

There were no significant differences in septicemia death and premature mortality rates of white and Hispanic females in 1996-1998. There were too few septicemia deaths among Hispanic males in 1996-1998 to report reliable rates (Table 23.1). The septicemia-related death and premature mortality rates of Hispanic and white females during this period were not significantly different (Table 23.2). There were no significant differences in the septicemia-related death rates of Hispanic and white males but Hispanic males had 1.9 times the premature mortality rate of white males. There were too few septicemia and septicemia-related deaths among Asian and Pacific Islander and Native American males and females to report reliable rates (Table 23.1).

Age-specific septicemia death rates of Connecticut males and females (1996-1998) are depicted in Figure 23.1. Age-specific septicemia death rates tend to be lower compared with all other causes of death up to about age 80 for females and up to age 75 for males, at which point they exceed death rates for all other causes.

Figure 23.1
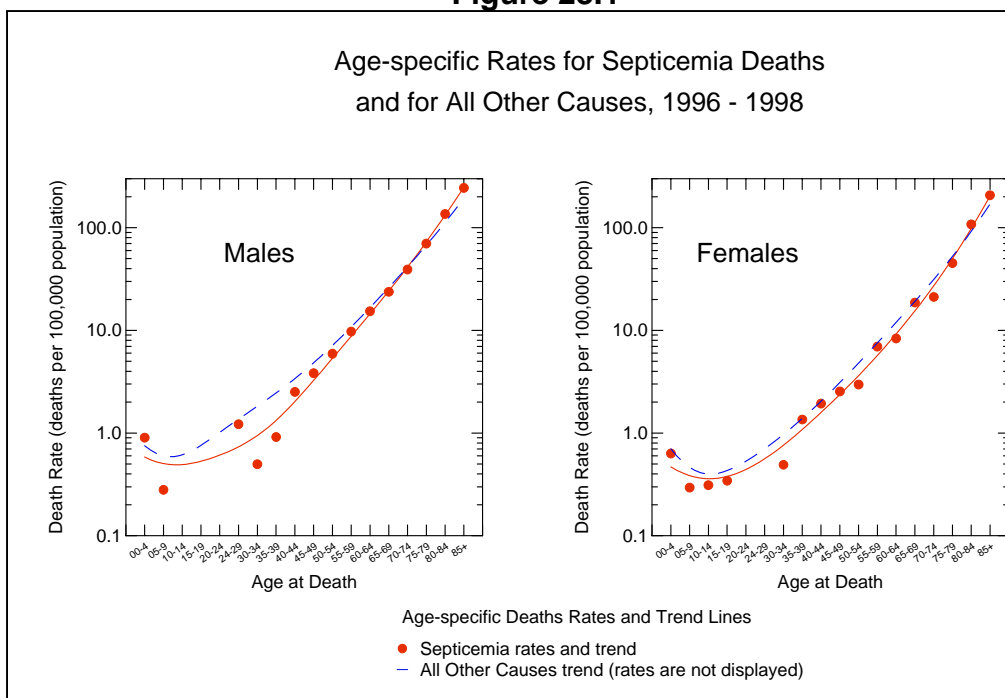
Table 23.2   Septicemia-Related Deaths[1]
Connecticut Residents by Gender, Race and Ethnicity[2], 1996-98

| Group | Number of Deaths | Age-Adjusted Mortality Rates[3] | | Age-Adjusted Premature Mortality Rates to Age 75[3] | |
		AAMR[4]	Change since 1989-91[5]	YPLL[4]	Change since 1989-91[5]
All Residents	4,238	38.8	↓↓↓	254.9	↓↓
All males	1,948	46.6	↓↓	296.8	↓↓
White	1,752	44.7	↓	256.8	ns
Black	188	85.7***	ns	805.6***	↓
Asian PI	6	—		—	
Native American	1	—		—	
Hispanic	74	46.7	ns	491.7**	ns
All females	2,290	33.8	↓	216.0	ns
White	2,090	32.3	↓	188.9	ns
Black	190	58.8***	ns	506.3***	ns
Asian PI	7	—		—	
Native American	1	—		—	
Hispanic	51	26.0	ns	228.5	ns

Notes:

1. This cause of death category includes ICD-9 codes 38. "Septicemia-related" deaths include those for which septicemia is the underlying and/or a contributing cause listed on the death certificate.

2. Racial groupings (White, Black, Asian & Pacific Islander, Native American) include persons of Hispanic ethnicity.

3. Age-adjusted Mortality Rates (AAMR) and Years of Potential Life Lost (YPLL) rates are per 100,000 based on race and ethnicity specific population estimates. Age-adjusted rates were calculated by the direct method using the 2000 U.S. standard population. Rates were not calculated for fewer than 15 events.

4. Statistical tests were conducted to evaluate differences in rates between race/ethnic groups. The white population serves as the reference group in each comparison (black vs. white, Asian & PI vs. white, Native American vs. white, Hispanic vs. white). Following are explanations of the notations:

    ** Significantly different than the respective white resident rate at p < .01.
    *** Significantly different than the respective white resident rate at p < .001.
    — Rate was not calculated due to small numbers.

5. Statistical tests were conducted to evaluate changes in rates over time. Comparisons of 1996-98 vs. 1989-91 rates are made within each race/ethnicity group. Following are explanations of the notations:

    ↓ 1996-98 rate is significantly lower than the 1989-91 rate at p < .05.
    ↓↓ 1996-98 rate is significantly lower than the 1989-91 rate at p < .01.
    ↓↓↓ 1996-98 rate is significantly lower than the 1989-91 rate at p < .001.
    ns Indicates the change from 1989-91 to 1996-98 is not statistically significant.

From 1989 to 1998, age-adjusted death rates for septicemia-related deaths in Connecticut decreased significantly by an average of 1.7% per year (p < .001) for males and by 1.2% for females per year (p < .01). Septicemia and septicemia-related mortality for all Connecticut residents decreased significantly between the periods 1989-1991 and 1996-1998.

Among subpopulation groups, the decrease in septicemia mortality was statistically significant among white females only (Table 23.1), while the decrease in septicemia-related mortality was statistically significant for white males and females only (Table 23.2).

Figure 23.2.

Septicemia Deaths
Comparison of Connecticut and U.S. 1989-1998
Age-Adjusted Mortality Rates* by Sex

SEX

Males   Females

Rates per 100,000

Year of Death   Year of Death

Symbols:

■ - U.S. rates for 1989-1998

✳ ● - Connecticut rates: an asterisk is used if the rate is significantly different than expected (p<.005), based on the log-linear trend.

* These rates are adjusted to the 1940 US standard million population.
* This classification includes deaths with ICD-9 codes:038

Connecticut male and female septicemia mortality rates have tended to be higher than the respective U.S. rates for the period 1989 to 1998 (Figure 23.2, Table 23.3).

Table 23.3 Comparison of CT with US, 1989 and 1998

	1989	1998	1998 Comparison
CT AAMR*	5.5	5.2	---
US AAMR*	4.0	4.4	CT > US AAMR

* age-adjusted mortality rates are per 100,000 population, U.S. 1940 standard million population.

Risk Factors

Septicemia is more likely to occur in persons whose immune systems are weakened due to some illness, such as HIV, or medical treatment, such as chemotherapy. It is commonly associated with the risks of high technology medicine (Pittet, Thievent, Wenzel, et al. 1993; Pittet, Tarara, and Wenzel 1994; Powe, Jaar, Furth, et al. 1999). Very low birth weight infants are also at high risk for septicemia (Fanaroff, Korones, Wright, et al. 1998).

Several research studies have identified risk factors for septicemia in high-risk environments (e.g., hospitals) or populations (e.g., dialysis patients).   One multicenter prospective study of hospital intensive care units found that greater severity of illness, chronic cardiovascular insufficiency, multiple organ failures at the time of sepsis, shock, and a low blood ph predicted mortality from septicemia (Brun-Buisson, Doyon, Carlet, et al. 1995). Other clinical risk factors for septicemia mortality include adequacy of the antibiotics used to treat the condition, the source of and type of infection, and the site of infection. Bacteremia caused by Candida and Enterococcus accounts for 30% to 40% of septicemia mortality.   Intra-abdominal and lower respiratory tract infections are associated with increased mortality (Angus and Wax 2001).

Persons with end-stage renal disease (ESRD) treated by dialysis are at higher risk of septicemia mortality compared with the general population (Sarnak and Jaber 2000). A

Table 22.4. – Risk Groups and Risk Factors for Septicemia-Related Mortality

Disease / Medical Conditions	Other Risk Factors
• immunodeficient conditions	current heavy smoking (possible)
• end-stage renal disease	
• cancer	
• HIV / AIDS	**Risk Groups**
• diabetes mellitus (possible)	• very low birthweight infants
• prior splenectomy	• increasing age (persons aged 65 and over)
• severe physical disability (possible)	• male gender
• severe cognitive impairment (possible)	• black race

<u>Sources</u>: Salive, Wallace, Ostfeld, et al. 1993; Sarnak and Jaber 2000; Angus, Linde-Zwirble, Lidicker et al. 2001; McBean and Rajamani 2001; Martin, Mannino, Eaton, et al. 2004. et al.

longitudinal study of dialysis patients with ESRD found that among this population, older age and diabetes were independent risk factors for septicemia. Among hemodialysis patients, risk factors associated with increased risk of septicemia mortality included temporary catheters used for vascular access as opposed to permanent catheters; low serum albumin, which may be indicative of nutritional deficiency; and dialyzer reuse (Powe, Jaar, Furth, et al. 1999).

There have been few population-based prospective studies of risk factors for septicemia. Risk factors identified in the general population include advancing age, male gender, and African American / black race (Salive, Wallace, Ostfeld, et al. 1993). One study of septicemia-associated mortality among older adults in a community setting identified heavy smoking and diabetes mellitus as the strongest risk factors for septicemia mortality. Severe cognitive and physical impairment were also associated with a greater risk of septicemia mortality (Salive, Wallace, Ostfeld, et al. 1993). Further investigation of the possible causal relationship between each of these risk factors in the general population and septicemia mortality is needed.

Costs and Prevention

Total nationwide costs of hospitalizations for septicemia have been estimated at $16.7 billion. Eighty-three percent of those costs are spent on patients aged 65 and over. The average cost per hospital patient is estimated at $22,100 with the average length of stay being 19.6 days (Angus, Linde-Zwirble, Lidicker, et al. 2001).

Prevention of septicemia involves appropriate treatment of localized infections, and among high-risk groups (such as heart valve patients) appropriate antibiotic treatment prior to surgery. One multicenter randomized controlled study found that adequate antibiotic therapy for sepsis was significantly associated with decreased mortality (MacArthur, Miller, Albertson, et al. 2004). The recent use of minimally invasive surgical techniques and noninvasive monitoring devices in hospital settings can reduce the risk of bloodstream infections associated with invasive devices and procedures that break the skin and mucous membrane barriers (Weinstein 1998). Among diabetic hemodialysis patients, improving nutritional status and minimizing catheter use may decrease the risk of septicemia (Jaar, Hermann, Furth, et al. 2000).

Hospital-acquired septicemia can be prevented and controlled through implementation of new infection-control technology and techniques. Quality assessment that incorporates hospital staff expertise with infection control surveillance, feedback, and protocols has been shown to predict lower infection rates. Other promising strategies include improved handwashing compliance associated with easily accessible soap dispensers as well as other specific modifications in the hospital environment (Wenzel and Edmond 2001).

The Centers for Disease Control and Prevention (CDC) has established screening-based guidelines for the prevention of early onset group B streptococcal (GBS) sepsis in neonatal infants (Schrag, Gorwitz, Fultz-Butts, et al. 2002). CDC recommends the universal prenatal screening of all pregnant women at 35 to 37 weeks' gestation for vaginal and rectal GBS colonization and also administration of intrapartum antibiotic prophylaxis to women identified as GBS carriers and to women who enter premature labor or rupture of membranes at less than 37 weeks gestation. The CDC also suggests that state and local public health agencies together with area hospitals establish surveillance for early-onset GBS disease and promote perinatal GBS disease prevention and education within the appropriate target populations (Schrag, Gorwitz, Fultz-Butts, et al. 2002). One study review and reanalysis of published data found that intrapartum antibiotic prophylaxis reduced early-onset GBS infection rate by 80% (Benitz, Gould, and Druzin 1999).

HIV Infection
(ICD-9 codes 042-044)

The first cases of what we now know as HIV/AIDS were reported in the United States and Connecticut in 1981 (Centers for Disease Control and Prevention 1981; Connecticut Department of Public Health 2001a). Researchers found that the illness "acquired immune deficiency syndrome" (AIDS), initially recognized in homosexual men and hemophiliacs, was caused by the human immunodeficiency virus (HIV). In 1986, HIV was designated as a disease with a disease classification scheme (Gold and Dwyer 1994). Two decades later, the human immunodeficiency virus (HIV) infection has become a major cause of illness, disability, and death worldwide. From 1996 to 1998, HIV/AIDS accounted for about 61,000 deaths in the United States and 755 deaths in Connecticut (Centers for Disease Control and Prevention 2004) [Table 24.1].

HIV infection is the thirteenth leading cause of death for all Connecticut residents and the fourth leading cause of death for residents aged 25 to 44 (Appendix V). Connecticut residents 25 to 44 years accounted for 66% of all HIV deaths in the 1996-1998 period. Age-specific HIV death rates of Connecticut males and females (1996-1998) are depicted in Figure 24.1. Age-specific HIV death rates for males are lower compared with all other causes of death up to about age 24; from ages 25 to 59 HIV death rates are higher compared with all other causes of death; and from ages 60 and older HIV death rates are lower compared with death rates for all other causes. Among females, age-specific HIV death rates tend to be higher compared with all other causes of death up through age 59. Beginning at ages 60 and older, HIV death rates are lower compared with death rates for all other causes.

HIV mortality rates, both nationally and in Connecticut, are higher in men than in women. Age-adjusted HIV death rates were 4.0 times higher for males compared with females in the 1989-1991 period and 2.8 times higher for males compared with females in the 1996-1998 period (p < .001 for both comparisons). In 1998, HIV mortality nationwide was 3.4 times higher for males than for females (Murphy 2000). Premature mortality (to age 75)

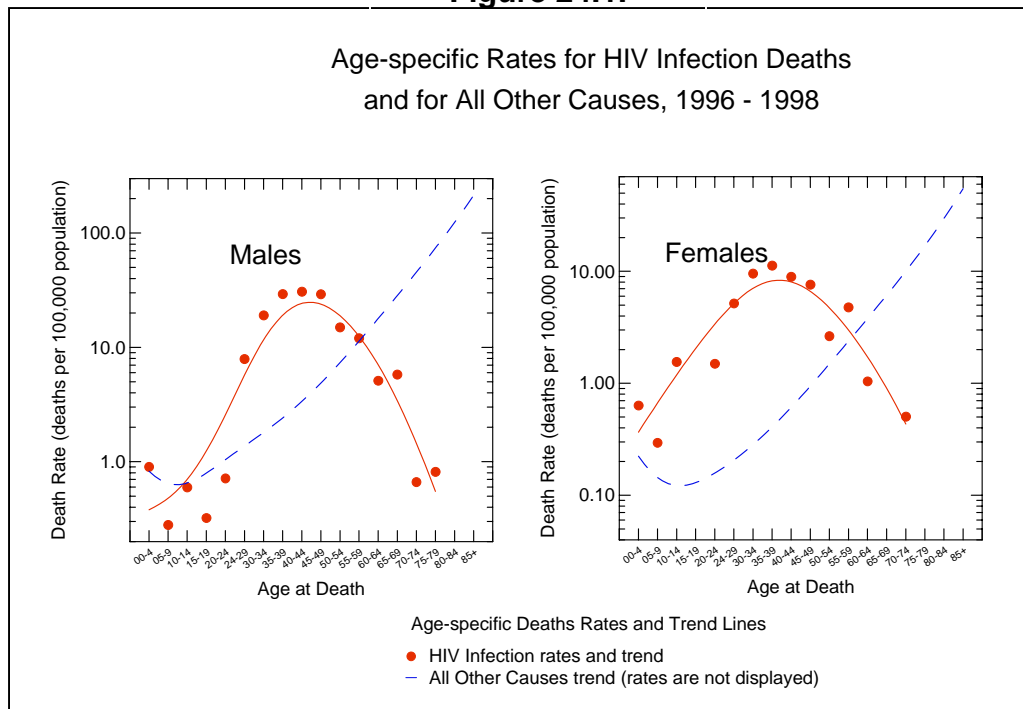
1996-1998 HIV Infections Deaths, CT Residents

- Thirteenth leading cause of death for all Connecticut residents

- Fourth leading cause of death for residents aged 25-44

- Third leading cause of death for black residents

- Fourth leading cause of death for Hispanic residents

- Fourth leading cause of premature mortality

Table 24.1. HIV Infection Deaths[1]
Connecticut Residents by Gender, Race and Ethnicity[2], 1996-98

| Group | Number of Deaths | Age-Adjusted Mortality Rates[3] | | Age-Adjusted Premature Mortality Rates to Age 75[3] | |
		AAMR[4]	Change since 1989-91[5]	YPLL[4]	Change since 1989-91[5]
All Residents	755	7.4	↓↓	265.0	↓↓
All males	551	11.0	↓↓↓	381.4	↓↓↓
White	338	7.5	↓	264.1	↓↓
Black	213	56.1***	ns	1,831.4***	↓
Asian PI	0				
Native American	0				
Hispanic	131	40.6***	ns	1,333.7***	ns
All females	204	3.9	ns	152.4	ns
White	102	2.3	ns	89.7	ns
Black	101	21.1***	ns	763.5***	ns
Asian PI	0				
Native American	0				
Hispanic	51	13.4***	ns	498.0***	ns

Notes:

1. This cause of death category includes ICD-9 codes 042-044.

2. Racial groupings (White, Black, Asian & Pacific Islander, Native American) include persons of Hispanic ethnicity.

3. Age-adjusted Mortality Rates (AAMR) and Years of Potential Life Lost (YPLL) rates are per 100,000 based on race and ethnicity specific population estimates. Age-adjusted rates were calculated by the direct method using the 2000 U.S. standard population. Rates were not calculated for fewer than 15 events.

4. Statistical tests were conducted to evaluate differences in rates between race/ethnic groups. The white population serves as the reference group in each comparison (black vs. white, Asian & PI vs. white, Native American vs. white, Hispanic vs. white). Following are explanations of the notations:

    *** Significantly different than the respective white resident rate at $p < .001$.
    — Rate was not calculated due to small numbers.

5. Statistical tests were conducted to evaluate changes in rates over time. Comparisons of 1996-98 vs. 1989-91 rates are made within each race/ethnicity group. Following are explanations of the notations:

    ↓   1996-98 rate is significantly lower than the 1989-91 rate at $p < .05$.
    ↓↓   1996-98 rate is significantly lower than the 1989-91 rate at $p < .01$.
    ↓↓↓ 1996-98 rate is significantly lower than the 1989-91 rate at $p < .001$.
    ns Indicates the change from 1989-91 to 1996-98 is not statistically significant.

Figure 24.1.

Age-specific Rates for HIV Infection Deaths
and for All Other Causes, 1996 - 1998

was also significantly higher for Connecticut males compared with females. In 1996-1998, males had 2.5 times the premature HIV mortality rate of females (p < .001).

HIV mortality rates were significantly higher in black males and females and Hispanic males and females compared with white males and females, respectively, in both the 1989-1991 and 1996-1998 periods. In the latter period, black males had 7.5 times and Hispanic males 5.4 times the HIV mortality rate of white males in Connecticut, while black females had 9.2 times and Hispanic females had 5.8 times the HIV mortality rate of white females in Connecticut. There were no HIV deaths among Asian and Pacific Islander and Native American males and females in 1996-1998 (Table 24.1).

Black and Hispanic males and females also had significantly higher premature mortality due to HIV compared with white males and females in Connecticut in both the 1989-1991 and 1996-1998 periods. In 1996-1998, black males had 6.9 times and Hispanic males had 5 times the premature mortality rate (to age 75) of white males due to HIV. In the same period, black females had 8.5 times and Hispanic females had 5.6 times the premature mortality rate (to age 75) of white females due to HIV (Table 24.1).

Between the periods 1989-1991 and 1996-1998, the HIV death and premature mortality rates declined significantly for all Connecticut residents. The decline in the HIV death rate was largely accounted for by a significant decrease in deaths in the white male population. The decline in the HIV premature mortality rate was largely accounted for by significant decreases in deaths among both white and black males (Table 24.1).

Mortality analyses of persons with HIV/ AIDS for the 1994-1998 period indicate that age, gender, race/ ethnicity, and infection risk group were not consistently associated with the risk of death. The most important predictor of death from HIV was being aged 50 years or older at the time of diagnosis (p < .05 for the years 1994, 1995, and 1998) [Carley, Roome, and Hadler 1999; Connecticut Department of Public Health 2000].

From 1989 to 1998, age-adjusted death rates for HIV in Connecticut did not exhibit a

Figure 24.2.

HIV Infection Deaths
Comparison of Connecticut and U.S. 1989-1998
Age-Adjusted Mortality Rates* by Sex

SEX

Males   Females

Rates per 100,000

Year of Death   Year of Death

Symbols:
■   - U.S. rates for 1989-1998
✳ ● - Connecticut rates: an asterisk is used if the rate is significantly different than expected (p<.005), based on the log-linear trend.

* These rates are adjusted to the 1940 US standard million population.
* This classification includes deaths with ICD-9 codes:042-044

consistent trend for either males or females (Figure 24.2). The HIV mortality rate for all Connecticut residents showed an increasing trend from 1989 to 1995; from 1995 through 1998 the HIV mortality rate appeared to decrease. This parallels the national trend.

HIV mortality nationwide increased by about 16% per year from 1987 to 1994. In 1994 and 1995 age-adjusted HIV mortality stabilized. By 1996, HIV mortality decreased by about 29%, and in 1997 by about 48% (Centers for Disease Control and Prevention 1999). The dramatic decrease in the HIV death rate in the late 1990s is attributable to newer treatments for HIV, such as highly active anti-retroviral therapy (HAART), that have enabled infected persons to live healthier, longer lives (Centers for Disease Control and Prevention 1999; Connecticut Department of Public Health 2000).

Connecticut male HIV mortality rates were significantly lower than the respective U.S. rates for the years 1989 to 1992, but were not significantly different from the respective U.S. rates in 1997 and 1998. Connecticut female HIV mortality rates were significantly higher than the respective U.S. rates for the years 1989 to 1997. The 1998 Connecticut female HIV mortality rate was not significantly different from the respective U.S. rate (Figure 24.2, Table 24.2).
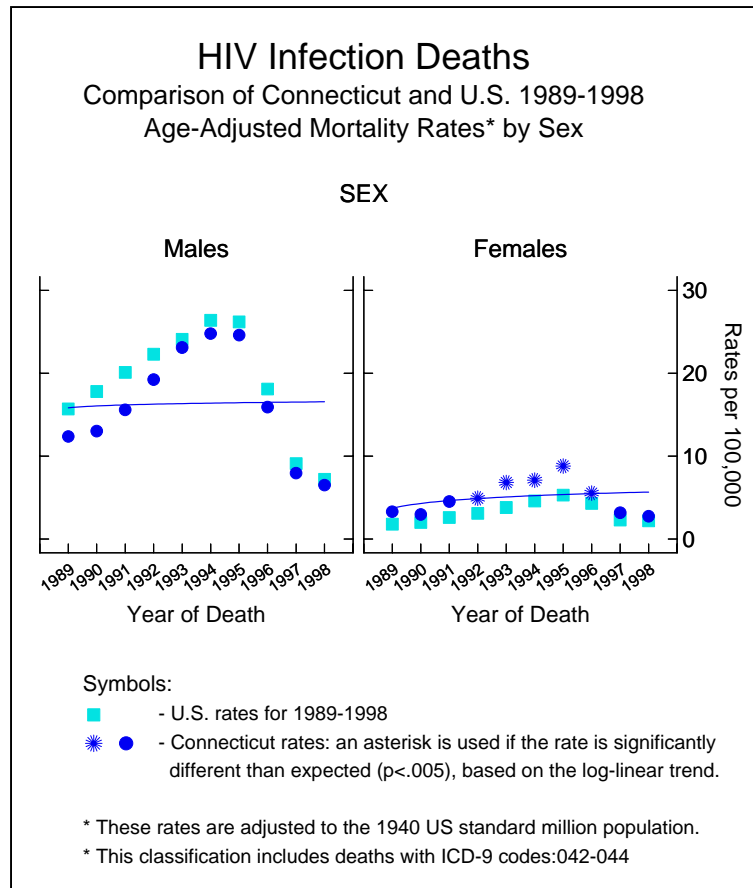
Table 24.2. Comparison of CT with US, 1989 and 1998

	1989	1998	1998 Comparison
CT AAMR*	7.8	4.6	---
US AAMR*	8.7	4.6	not significantly different

* age-adjusted mortality rates are per 100,000 population, U.S. 1940 standard million population.

AIDS Incidence

There were 662 AIDS cases reported to the Connecticut Department of Public Health in 1998, a decrease of 44% from the number reported the previous year (1,193 cases in 1997) and a decrease of 62% from the high of 1,759 cases reported in 1993. From 1998 to 2000, the number of AIDS cases remained relatively stable. An expansion of the AIDS case definition in 1993 accounted for a large part of the observed increase in AIDS cases in that year (Connecticut Department of Public Health 2001a with 2004 updated figures). The introduction of HAART in the mid-1990s appears to be the most important factor in the decreasing number of AIDS cases; however, a decrease in underlying HIV infection in the population may also account for the observed decrease in AIDS cases (Connecticut Department of Public Health 2000).

Specific subpopulation groups in Connecticut are disproportionately represented among persons with AIDS, with 74% of all cases occurring among males, 46% in persons 30 to 39 years of age, 30% occurring among African American/black residents, 24% in Hispanics, 49% in persons with a history of intravenous (IV) drug use, and 23% in men who have had sex with men (MSM) [Connecticut Department of Public Health 2001a]. The percentage of AIDS cases that are female increased from 26% in 1995 to 38% of all those reported in 2000. [Connecticut Department of Public Health 2001b].

The profile of persons with AIDS in Connecticut (1999) and the United States (1998-1999) differs. A higher percentage of persons with AIDS in Connecticut are female (31.3% vs. 23.4%), Hispanic (29.3% vs. 19.7%), and have injection drug as a likely source of infection use (37.3% vs. 22.4%) [Connecticut Department of Public Health 2000].

Risk Factors

Risk factors for HIV/AIDS are associated with the following social and behavioral risk groups: men who have sex with men (MSM); male injection drug users (IDU); female injection drug users; and women who have sex with high-risk men (Connecticut Department of Public Health 2001b). Appropriate prevention strategies require an understanding of the social settings and behaviors that put each of these groups at higher risk for HIV/AIDS.

HIV is transmitted through the exchange of bodily fluids, such as blood and semen. Transmission of HIV in the United States and other Western countries follows a different pattern from that of the developing world, where heterosexual intercourse is the main source of transmission and females comprise about half of those infected with HIV/AIDS. Over the course of the epidemic, the main sources of transmission in the United States (and Connecticut) have been unprotected anal intercourse and shared use of non-sterile needles to inject drugs (Lorber 1997). More recently, unprotected vaginal intercourse has become an increasingly common source of transmission in the United States [Table 24.3]. In Connecticut, heterosexual transmission of HIV increased from 13% of diagnosed cases in 1990 to 26% in 1998 (Connecticut Department of Public Health 2001a).

Several factors may increase a person's risk for HIV infection. The presence of other sexually transmitted diseases (STDs) that cause ulcerations, such as chlamydia, syphilis, gonorrhea, and genital herpes is an important risk factor for HIV infection (Chesson and Pinkerton 2000). Multiple sexual partners or a partner having multiple sexual partners within a relatively short period of time increases the likelihood of infection by both STDs and HIV (Finer, Darroch, and Singh 1999), as does a high prevalence of HIV infection in the peer group. Exchanging sex to support an addiction (i.e. through prostitution or sex work) is another behavior that places people at higher risk for infection (Des Jarlais 1997).

The AIDS epidemic disproportionately affects low-income, urban minority communities (Karon, Fleming, Steketee, et al. 2001). Approximately 49% of all HIV/AIDS cases in Connecticut have been located in the three largest cities of Hartford, New Haven, and Bridgeport, and 89% of all cases in the state are located in the highly urbanized surrounding areas of Hartford, New Haven, and Fairfield counties (Connecticut Department of Public Health 1998).

Table 24.3. – Risk Groups and Risk Factors for HIV / AIDS	
Risk Groups	Risk Factors
• Men who have sex with men (MSM) • Injection drug users (IDU) • Women who have sex with high-risk men • Sex workers	• Use of non-sterile needles or other implements to inject drugs • Unprotected anal intercourse • Unprotected vaginal intercourse • Multiple sexual partners over a short period of time • Concomitant sexually transmitted disease (STD) infections

Sources: Connecticut Department of Public Health (2001b); Des Jarlais (1997); Chesson and Pinkerton (2002); Fife (2002); Finer, Darroch, Singh (1999)

Prevention

Since the beginning of the AIDS epidemic, the CDC has funded primary prevention efforts in local communities that target persons at risk for becoming infected with HIV/AIDS. The main focus of these programs has been to decrease risky sexual and drug-using behaviors, primarily through use of condoms and clean needles, respectively (Centers for Disease Control and Prevention 2001a; Centers for Disease Control and Prevention 2003b). Prevention programs have been tailored to meet the specific needs of subpopulation groups, such as heterosexual adults (Neumann, Johnson, Semann, et al. 2002), sexually active adolescents (Mullen, Ramirez, Strouse, et al. 2002), men who have sex with men (Johnson, Hedges, Ramirez, et al. 2002), and injection drug users (Semaan, Des Jarlais, Sogolow, et al. 2002; Kelly and Kalichman 2002).

In 1996, the CDC initiated the HIV/AIDS Prevention Research Synthesis Project in order to systematically analyze the effectiveness of HIV prevention-intervention studies in reducing sexual and drug risk-taking behaviors (Sogolow, Peersman, Semaan 2002). Key findings from these analyses suggest that:

- Most people at risk will change their behavior to reduce their risk of contracting HIV;

- No single set of intervention programs has successfully eliminated HIV risk-taking behaviors. The "residual" risk behavior that continues after successful interventions have been implemented is of serious concern especially in places with high HIV prevalence, where even low levels of risky behavior may lead to high levels of new infections;

- Reduction of HIV risk behavior is a complex task and no single type of program intervention is effective across the various subpopulation groups; and

- Even with numerous successful prevention efforts, there have been some notable failures (such as the resurgence of HIV in a younger generation of U.S. gay males) that are instructive. Such failures suggest that there may be a decrease in effectiveness of HIV interventions over time, that there may be a need for multiple interventions within a single risk group, and that an effective intervention in one community may not be appropriate for another community. Unsuccessful prevention efforts suggest that a better knowledge base is needed to reduce HIV among high-risk groups (Des Jarlais and Semaan 2002).

Although HIV morbidity and mortality has stabilized since the beginning of the epidemic, there is an increase in the number of persons with HIV and a growing public concern about increasing HIV incidence in certain communities. In 2003, the CDC announced a new initiative "Advancing HIV Prevention: New Strategies for a Changing Epidemic" whose aim is to reduce barriers to early diagnosis of HIV and to increase access to appropriate medical care and prevention services. This initiative emphasizes both primary and secondary prevention strategies and relies on long-standing public health approaches to

reduce the incidence and spread of disease, such as routine screening as appropriate, new case identification, partner notification, and available ongoing treatment and prevention services for infected persons. The initiative is made up of four main strategies: 1) making HIV testing a routine part of medical care; 2) implementing new models for diagnosing HIV infections outside medical settings; 3) preventing new infections by working with persons diagnosed with HIV and their partners; and 4) further decreasing perinatal HIV transmission (Centers for Disease Control and Prevention 2003b).

The Connecticut Department of Public Health provides HIV prevention services, program services for people with HIV/AIDS and those affected by HIV, as well as surveillance of HIV/AIDS in the state. The HIV/AIDS Prevention Program (HAPP) supports HIV antibody testing and counseling, needle exchange, drug treatment advocacy, health education, and street outreach. The HIV/AIDS Planning Program (HAP) supervises public information campaigns and community-based HIV prevention campaigns. The Health Care and Social Services Program (HCSS) supervises services for persons with HIV infection, including case management, payment for medication, and help for persons affected by HIV such as the uninfected children of persons with HIV (Connecticut Department of Public Health 2004b). The HIV/AIDS Surveillance Program provides up-to-date information on HIV case surveillance in Connecticut by gender, race/ethnicity, age, mode of transmission, and town, as well as trends in HIV morbidity and mortality over time (Connecticut Department of Public Health 2004c).

Barriers to HIV Prevention

A major societal barrier to HIV/AIDS prevention is stigma, that is, prejudice and discrimination directed toward people with HIV/AIDS. Fear of persons with HIV/AIDS, homophobia, and prejudice against drug users on the part of health care workers can impede prevention efforts by discouraging people from acknowledging their risk, seeking out HIV testing, and delaying treatment. Stigma experienced on multiple levels—individual (i.e., negative interactions with at-risk persons), community (i.e., anti-gay attitudes, lack of candid discussions regarding sexuality and sexual decisionmaking), and societal structures (i.e. discriminatory laws)—can interfere with the effectiveness of HIV prevention and care programs (Centers for Disease Control and Prevention 2001b). Societal and individual attitudes that reinforce male dominance and female submissiveness regarding safer-sex practices are other important barriers to successful HIV/AIDS prevention (Asencio 2002).

Strategies to combat stigmatization of people with HIV/AIDS and high-risk groups include: 1) continued public education about how HIV is and is not transmitted based on facts, not prejudice; 2) making sure that prevention programs are not stigmatizing to the target population and ensuring that programs are reflective of the needs and preferences of that population group; and 3) ongoing education of health care and other service providers who work with HIV-positive people (Valdiserri 2002). Continued leadership at the local, state, and national levels are needed to address discriminatory laws, societal structures, and negative attitudes that can hamper successful HIV prevention efforts (Centers for Disease Control and Prevention 2001b).

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SECTION IV. E.

MORTALITY SUMMARY SECTION

CONTENTS

Summary of Male – Female Mortality

Leading Causes of Death and Premature Mortality, 1989 - 1998

Connecticut male residents

During the 1990s, heart disease was the leading cause of death and premature mortality for Connecticut males, followed by all cancers. Heart disease and cancer, together, accounted for 57% of all deaths and 39% of all premature mortality under age 75 in 1996-98. Cerebrovascular disease was the third leading cause of death and the eighth leading cause of premature mortality under age 75. Other leading causes of death that contributed most to premature mortality among Connecticut males during the decade included unintentional injuries, suicide, HIV infection, and homicide and legal intervention. The leading causes of death and premature mortality by gender for 1996-98 are displayed in Table 25.1.

Between 1989 and 1998, age-adjusted death rates for all causes of death and for heart disease decreased significantly for all Connecticut male residents, as well as for both the white and black male populations. Age-adjusted death rates for all cancers, lung cancer, colorectal cancer, prostate cancer, HIV infection, and motor vehicle crashes decreased significantly for all Connecticut males and for white males, while there were significant decreases in age-adjusted cerebrovascular disease and COPD death rates for black male residents. COPD-related death rates also decreased significantly for all Connecticut males during the 1990s (Table 25.2). There were significant increases in age-adjusted death rates due to diabetes mellitus and hypertension-related causes for all Connecticut males and for white males, while age-adjusted death rates due to drug-induced causes increased among white males only (Table 25.3).

Premature mortality rates (under age 75) for all causes of death decreased significantly during the 1990s. Decreases were significant for both white and black males. Between 1989 and 1998, premature mortality for heart disease, all cancers, lung and other respiratory cancer, and HIV infection declined significantly for both white and black males. Premature mortality due to prostate cancer and motor vehicle crashes declined significantly for all males and for white males only. Premature mortality due to septicemia-related causes declined significantly for all males and for black males only (Table 25.2). The only increasing trend in premature mortality during the decade occurred in drug-induced causes among white males (Table 25.3).

Connecticut female residents

Heart disease was the leading cause of death and the second leading cause of premature mortality for Connecticut females during the 1990s. Cancer was the second leading cause of death and the leading cause of premature mortality. Heart disease and cancer accounted for 57% of all deaths and 46% of all premature mortality under age 75 among Connecticut females for 1996-98 (Table 25.1). Cerebrovascular disease was the third leading cause of death and the fifth leading cause of premature mortality under age 75 in 1996-98 (it was the fourth leading cause of death in 1989-91). Other leading causes of death that contributed most to premature mortality among Connecticut females

Table 25.1   Leading Causes of Death and Premature Mortality[1] by Gender, All Connecticut Residents, 1996-98

Cause of Death	Number of Deaths	Rank – Deaths[2]	Deaths Under Age 75	YPLL[3] Under Age 75	Rank – Premature Mortality
All Residents					
All cancer	21,300	2	11,315	144,516	1
Diseases of the heart	29,251	1	8,916	107,235	2
Unintentional injuries	3,158	6	2,186	74,610	3
HIV infection	755	13	754	25,610	4
Suicide	835	11	755	25,408	5
Homicide and legal intervention	452	15	444	19,523	6
Cerebrovascular disease	5,786	3	1,302	15,271	7
Chronic liver disease and cirrhosis	882	10	682	12,402	8
COPD	3,700	4	1,283	11,537	9
Diabetes mellitus	1,991	7	903	10,918	10
All Males					
Diseases of the heart	13,582	1	5,782	73,125	1
All cancer	10,602	2	5,855	72,322	2
Unintentional injuries	2,016	4	1,564	54,812	3
Suicide	647	8	585	19,883	4
HIV infection	551	9	550	18,186	5
Homicide and legal intervention	346	13	343	15,251	6
Chronic liver disease and cirrhosis	548	10	462	8,817	7
Cerebrovascular disease	2,132	3	684	8,238	8
Diabetes mellitus	933	7	481	5,993	9
Pneumonia & influenza	1,621	6	399	5,686	10
All Females					
All cancer	10,698	2	5,460	72,194	1
Diseases of the heart	15,669	1	3,134	34,110	2
Unintentional injuries	1,142	6	622	19,798	3
HIV infection	204	13	204	7,424	4
Cerebrovascular disease	3,654	3	618	7,033	5
COPD	2,005	4	670	6,077	6
Suicide	188	14	170	5,525	7
Diabetes mellitus	1,058	7	422	4,925	8
Homicide and legal intervention	106	15	101	4,273	9
Chronic liver disease and cirrhosis	334	12	220	3,585	10

Notes:

1. Premature mortality is defined as the total number of years of potential life lost (YPLL) before age 75. See Section V—Appendix II of this report for a discussion of the YPLL measure.
2. These ranks are based on the National Center for Health Statistics (NCHS) leading causes of death list. See Section V—Appendix V for a complete list of leading causes of death in Connecticut.
3. "YPLL before age 75" indicates the total number of potential life years lost before age 75 and is the basis for ranking premature mortality.

Table 25.2   Significant Decreasing Mortality Trends [1]
Connecticut Residents, from 1989-91 to 1996-98

Decreases in Age-Adjusted Mortality Rates

- All causes (all residents, all males, white males, black males)[3]
- Diseases of the heart (all residents, all males, white males, black males, all females, white females)
- Cerebrovascular disease (black males)
- All cancer (all residents, all males, white males)
- Lung & other respiratory cancer (all males, white males)
- Colorectal cancer (all residents, all males, white males, all females, white females)
- Breast cancer (all females, white females)
- Prostate cancer (all males, white males)
- COPD (black males)
- COPD-related (all males)
- Motor vehicle crashes (all males, white males)
- Pneumonia & influenza (all residents, all females, white females)
- Septicemia (all residents, all females, white females)
- Septicemia-related (all residents, all males, white males, all females, white females)
- HIV (all residents, all males, white males)

Decreases in Premature Mortality Rates[2]

- All causes (all residents, white males, black males)[3]
- Diseases of the heart (all residents, all males, white males, black males)

- All cancer (all residents, all males, white males, black males, all females)
- Lung & other respiratory cancer (all residents, all males, white males, black males)
- Colorectal cancer (all residents, white females)
- Breast cancer (all females, white females)
- Prostate cancer (all males, white males)

- Motor vehicle crashes (all residents, all males, white males)
- Pneumonia & influenza (all residents, white females)
- Septicemia (all residents)

- Septicemia-related (all residents, all males, black males)
- HIV (all residents, all males, white males, black males)

Notes:

1. Decreasing trends reflect causes of death that showed statistically significant changes between the 1989-91 and 1996-98 time periods.

2. Premature mortality is defined as the total number of years of potential life lost (YPLL) before age 75. This table reflects decreases in age-adjusted premature mortality, or age-adjusted YPLL under age 75.

3. Black and white Connecticut residents include persons of Hispanic ethnicity.

Table 25.3   Significant Increasing Trends in Mortality[1]
Connecticut Residents, from 1989-91 to 1996-98

Increases in Age-Adjusted Mortality Rates	*Increases in Premature Mortality Rates*[2]
• Hypertension-related (all residents, all males, white males, all females, white females)[3]	
• Diabetes (all residents, all males, white males, all females, white females)	• Diabetes (all residents, all females, white females)
• Lung & other respiratory cancers (all females, white females)	
• COPD (all residents, all females, white females)	• COPD (all females, white females)
• COPD-related (all residents, all females, white females)	• COPD-related (all females, white females)
• Unintentional injuries (all residents, white females)	• Unintentional injuries (white females)
• Poisoning (all females, white females)[4]	• Poisoning (all females, white females)[4]
• Drug-induced (all residents, white males, all females, white females)[4]	• Drug-induced (all residents, white males, all females, white females)[4]

Notes:

1. Increasing trends reflect causes of death that showed statistically significant changes between the 1989-91 and 1996-98 time periods.

2. Premature mortality is defined as the total number of years of potential life lost (YPLL) before age 75. This table reflects increases in age-adjusted premature mortality, or age-adjusted YPLL under age 75.

3. Black and white Connecticut residents include persons of Hispanic ethnicity.

4. Comparison periods are 1992-94 vs. 1996-98.

during the 1990s include unintentional injuries, HIV infection, cerebrovascular disease, and COPD.

Between 1989 and 1998, age-adjusted death rates for heart disease, breast cancer, colorectal cancer, septicemia, septicemia-related, and pneumonia and influenza decreased significantly for all Connecticut female residents and for white females (Table 25.2). There were significant increases in age-adjusted death rates due to diabetes mellitus, hypertension-related causes, lung and other respiratory cancers, COPD, COPD-related causes, poisoning, and drug-induced causes for all Connecticut females and for white females during the 1990s, while unintentional injury death rates increased among white females only (Table 25.3).

Premature mortality rates (under age 75) due to all cancers decreased significantly for Connecticut females during the 1990s. Premature mortality due to breast cancer decreased for all females and for white females, while premature mortality due to colorectal cancer and pneumonia and influenza decreased for white females only (Table 25.2). Premature mortality due to diabetes, COPD, COPD-related causes, poisoning, and drug-induced causes increased in the 1990s for all

females and for white females. Premature mortality due to unintentional injuries increased for white females only (Table 25.3).

Gender differences in mortality

Compared with females, Connecticut male residents experienced significantly higher age-adjusted mortality from all causes of death and from 22 of the 23 non-sex-specific categories of death considered in this report. For the period 1996-98, the Connecticut male resident age-adjusted death rate from all causes was about fifty percent higher than that of females (Table 25.4). Connecticut male excess deaths (the additional deaths that would not have occurred if males had the same mortality rate as females) for all causes are estimated at more than 4,000 per year for the period 1996-98 (Table 25.4). For one cause, cerebrovascular disease, there was not a significant difference in male and female mortality rates for the 1996-98 period. About 35% of all male excess deaths were due to cardiovascular diseases, 24% were due to cancer, and 14% were due to injuries.

Among the leading causes of death, the greatest male-female differences in age-adjusted death rates (1996-1998) were found in suicide, followed by homicide and legal intervention, and HIV infection (Table 25.4). These findings parallel national trends that show male to female risk ratios being highest for suicide, followed by homicide and HIV infection (MMWR 1999). The numbers of estimated excess male deaths were greatest for heart disease, all cancers (of which 78% were attributed to lung cancer), unintentional injuries (of which 29% were motor vehicle crashes), and pneumonia and influenza (Table 25.4).

Time Trend Analyses

The male-female mortality disparity decreased significantly for several causes of death during the 1990s. Between 1989-91 and 1996-98, the age-adjusted all-cause mortality rate decreased significantly for Connecticut males, but remained unchanged for Connecticut females (Figure 25.1). The male-female mortality disparity for heart disease, all cancers, lung and other respiratory cancers, COPD and COPD-related causes also decreased significantly ($p < .05$) during this period (Figure 25.1). There were no increases in the male-female mortality disparity for any causes of death considered in this report between 1989-91 and 1996-98.

Table 25.4   Summary Table of Male-Female Mortality Disparities[1]
Connecticut Residents, 1989-91 and 1996-98

	1989-91		1996-98	
Cause of Death	Male-Female Relative Risk[2,3]	Excess Male Deaths[4]	Male-Female Relative Risk[2,3]	Excess Male Deaths[4]
All Causes	1.6**	5,181	1.5**	4,428
Chronic Diseases				
Heart disease	1.7**	1,852	1.5**	1,526
Cerebrovascular	1.1**	69	1.1	36
Hypertension-related	1.1	44	1.1**	91
Diabetes	1.2*	35	1.3**	80
Diabetes-related	1.4**	250	1.5**	375
All cancer	1.5**	1,237	1.4**	1,042
Lung & other respiratory cancer	2.2**	614	1.7**	815
Colorectal cancer	1.5**	139	1.4**	100
COPD	1.9**	180	1.4**	153
COPD-related	2.1**	679	1.6**	385
Chronic liver disease & cirrhosis	2.1**	102	2.1**	95
Injury				
Unintentional injuries	2.4**	346	2.3**	382
Motor vehicle crashes	2.7**	165	2.1**	109
Fall & fall-related injuries	1.6**	29	2.0**	54
Suicide	4.4**	191	3.7**	159
Homicide & legal intervention	3.5**	103	3.5**	83
All poisoning	2.7**	60	2.8**	137
Alcohol-induced	2.6**	77	3.1**	78
Drug-induced	2.2**	30	2.6**	112
Infectious Diseases				
Pneumonia & influenza	1.6**	171	1.6**	200
Septicemia	1.3**	43	1.4**	45
Septicemia-related	1.4**	195	1.4**	178
HIV infection	4.0**	179	2.8**	119

Notes:

1. Includes all Connecticut male and female residents.

2. Relative risk is the ratio of the male Connecticut resident age-adjusted mortality rate (AAMR) to the female Connecticut resident AAMR (2000 U.S. standard population). Relative risks are not calculated for fewer than 15 deaths in either the male or female population.

3. Statistical tests were conducted to evaluate differences in male and female resident AAMRs. Following are explanations of the notations:
    *   * Significantly different than the female resident rate at p < .05.
    *   ** Significantly different than the female resident rate at p < .01.

4. Excess deaths are those deaths that would not have occurred if the male population had the same rate as the female population, and are presented on an annualized or per year basis.

Figure 25.1. Age-Adjusted Mortality Rates by Gender, Connecticut Residents – causes of death for which the male-female mortality disparity changed significantly from 1989-91 to 1996-98
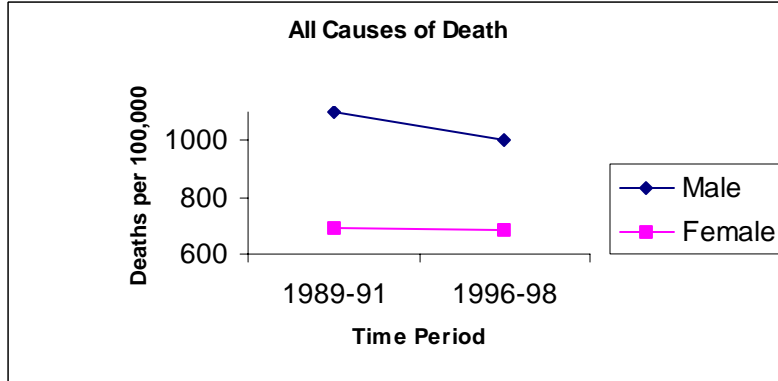
All Causes of Death

The male-female mortality disparity for all causes of death decreased significantly from 409.4 to 314.7 during this time period.

Diseases of the Heart
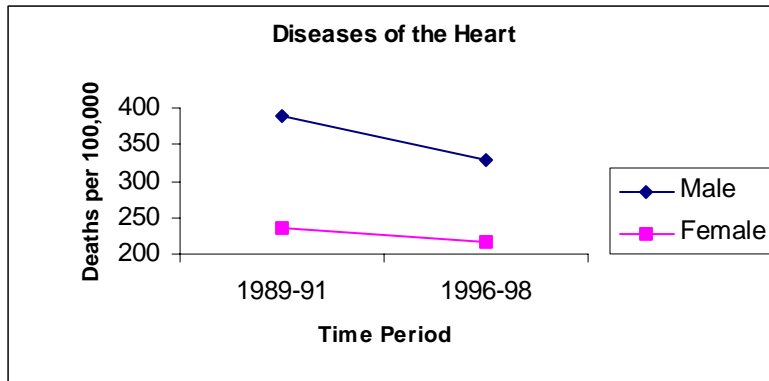
The male-female mortality disparity for heart disease decreased significantly from 152.9 to 110.7 during this time period.
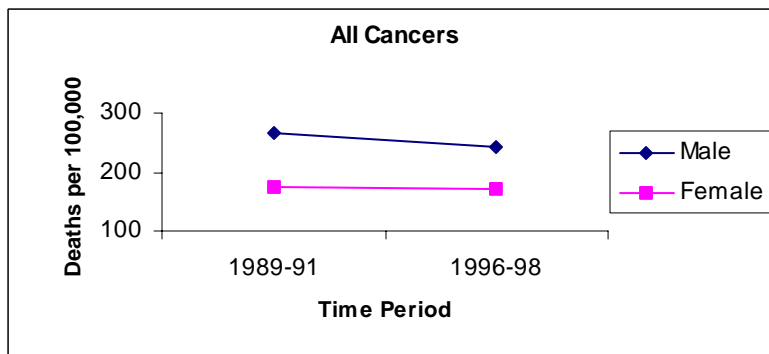
All Cancers

The male-female mortality disparity for all cancers decreased significantly from 92.8 to 71.9 during this time period.

**Figure 25.1. Age-Adjusted Mortality Rates by Gender,
Connecticut Residents – causes of death for which the
male-female mortality disparity changed significantly
from 1989-91 to 1996-98 (continued)**

Lung & Other Respiratory Cancer
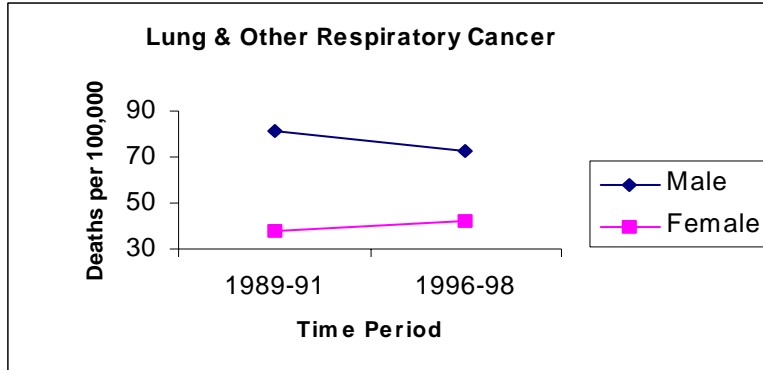
The male-female mortality disparity for lung & other respiratory cancers
decreased significantly from 44.2 to 30.7 during this time period.

Chronic Obstructive Pulmonary Disease (COPD)
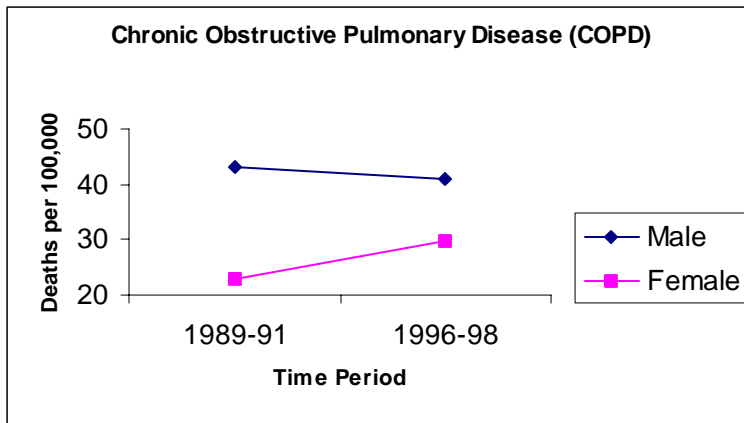
The male-female mortality disparity for COPD decreased significantly
from 20.2 to 11.1 during this period.
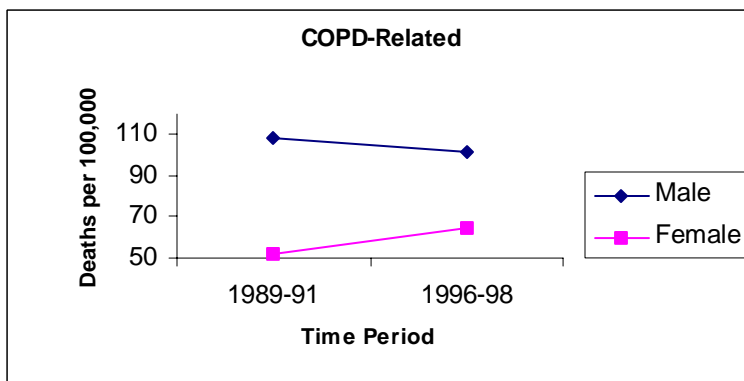
COPD-Related

The male-female mortality disparity for COPD-related causes
decreased significantly from 56.9 to 37.3 during this period.

Discussion

The trend of higher mortality among Connecticut males compared with females follows a similar pattern nationwide and in other industrialized countries (Nathanson 1990, 1995; Wingard 1984; Doyal 1995; Lorber 1997; Waldron 1995a, 1995b; Nikiforov and Mamaev 1998; Hemstrom 1999). Gender differences in mortality may be the result of social and biological factors that interact to produce discernable patterns. Relevant biological factors include the effects of sex differences in reproductive anatomy and physiology, sex hormones, and chromosomally-linked genes. Social factors include cultural influences that produce gender differences in risk-taking behavior and differences in access to societal resources such as nutrition and health care, and environmental exposures (Nathanson 1977, 1990, 1995; Wingard 1982, 1984; Doyal 1995; Lorber 1997; Waldron 1995a, 1995b; Bird and Rieker 1999). Technological and economic changes in the home and workplace have also affected the mortality risks to which males and females are differentially susceptible (Waldron 1995a; Bird and Rieker 1999).

Gender differences in mortality and life expectancy have varied historically and across cultures. Women live longer than men do in contemporary economically developed nations, yet at the beginning of the twentieth century this female advantage was much smaller than it is today (Waldron 1995a). In contrast, males in contemporary developing nations tend to have lower mortality and greater life expectancy than do females (Santow 1995; Waldron 1995a; United Nations 1998).

Research has established that gender differences in behavior are a major cause of gender differences in mortality (Nathanson 1977, 1990, 1995; Wingard 1984; Doyal 1995; Lorber 1997; Waldron 1995a, 1995b, 1996; Bird and Rieker 1999). Men's earlier adoption and higher rate of cigarette smoking compared to women is considered the key factor of the male mortality disadvantage due to chronic diseases of coronary heart disease, lung cancer, chronic obstructive pulmonary disease (USDHHS 1989; USDHHS 1990; Waldron 1986; Waldron 1995a). It is estimated that men's higher smoking-related morality accounts for up to half of all gender differences in mortality in the United States including 30% of gender differences in coronary heart disease and 90% of gender differences in lung cancer (Waldron 1986; Waldron 1995a). Women's adoption of the smoking habit in the past half-century most likely accounts for the observed increases in female lung cancer, COPD, and COPD-related deaths in the 1990s and the relative decreases in the male-female disparity for these causes of death.

Gender differences in behavior influence gendered patterns in mortality. U.S. data indicate that men's diets have higher ratios of saturated to polyunsaturated fat and lower vitamin C intake compared with women's which could contribute to higher coronary heart disease mortality (Waldron 1995a, 1995b). Men are more likely than women to engage in vigorous exercise, which likely contributes to a decreased risk for heart disease mortality (Waldron 1995a, 1995b). Some studies report that men score higher on psychological measures of hostility and mistrust, both of which are psychological characteristics associated with increased risk of coronary heart disease (Waldron 1995a, 1995b). U.S. data show that women visit physicians more often than do men (Nathanson 1977; Verbrugge 1985; Verbrugge and Wingard 1987) and, oftentimes, this fact is advanced as an explanation for women's lower mortality rates. Women, however, tend to delay medical care for

various types of cancer and heart disease compared with men. For most types of cancer and heart attacks, women do not have a better prognosis than men (Waldron 1995a, 1995b). It is therefore unclear, if and how much women's health-care seeking behaviors actually contribute to their lower mortality.

Males' heavier drinking patterns contribute to their higher mortality due to liver disease and cirrhosis, motor vehicle accidents, homicide, and suicide compared with females (Waldron 1986; Waldron 1995a). Men are more likely to use guns and take other physical risks that contribute to higher mortality due to violent and accidental deaths. Men are more likely to inject drugs that contribute to higher HIV and drug-related mortality. Drug use is also associated with higher rates of homicide (USDHHS 1990). Men are also more likely than women to be employed outside the home in more hazardous occupations. Their greater exposure to occupational hazards contributes to their higher rates of accident and lung cancer mortality (Nathanson 1990; Doyal 1995; Lorber 1997; Waldron 1995a, 1995b, 1996; Bird and Rieker 1999).

Biological differences also contribute to gender differences in mortality. Clearly, sex-specific conditions such as prostate, cervical, ovarian, and breast cancer as well as reproductive (i.e. childbearing) capacity, are biological factors directly linked to higher mortality risk. Research suggests that female sex hormones may confer a protective effect on women's risk while men's tendency to accumulate fat in the upper abdomen may increase their risk for coronary heart disease. Female sex hormones are believed to contribute to higher HDL (high-density lipoprotein) and lower LDL (low-density lipoprotein) cholesterol levels in pre-menopausal women. This fact may confer a protective effect on pre-menopausal women's risk for coronary heart disease (Waldron 1995b; Bird and Rieker 1999). Limited evidence suggests that higher levels of HDL cholesterol in females could account for up to half of the sex differences in coronary heart disease mortality. The female mortality advantage due to lower LDL cholesterol levels, however, appears to reverse at older ages (Waldron 1995b).

Although biologically-based explanations for men's greater risk-taking and aggressive behaviors are advanced occasionally in the popular media, such reasoning is not well supported by scientific evidence. Specifically, studies advancing the ideas that males' possession of higher testosterone levels and/or a *y* chromosome might predispose them to higher levels of physical aggression have been methodologically flawed and/or inconclusive (Bleier 1988; Rosoff 1991; Sunday 1991; Fausto-Sterling 1992). Rather, gender differences in risk-taking behavior can be viewed in the context of accepted cultural values that encourage males to behave in more physically aggressive ways and in riskier activities compared with females (Rosoff 1991; Fausto-Sterling 1992; Stillion 1995). Males, more than females, are encouraged to express individual aggression, and male images in the popular media reinforce the social acceptability of violent and risk-taking behaviors. Programs that aim to reduce premature male mortality due to injury should first address the societal costs of what are considered acceptable male behaviors such as physical aggression and gun use (Stillion 1995).

The magnitude of the male-female mortality differential in the U.S. (and other industrialized countries) has varied over time. At the beginning of the twentieth century, the female mortality advantage was small. In the mid-twentieth century female mortality decreased more dramatically than male's due primarily to decreases in deaths affecting women--maternal mortality and uterine

cancer. At the same time, male lung cancer and coronary heart disease mortality, both closely linked to the widespread adoption of smoking behaviors by males, increased (Verbrugge 1980; Wingard 1984; Waldron 1995a).

Beginning in the 1970s, the trend toward increasing gender differentials in mortality in the U.S. reversed (Waldron 1995a). Between 1970 and 1989, male mortality decreased more than female and the corresponding mortality ratio decreased as well. Trend changes in several causes of death have contributed to the changing gender differential. Lung cancer and COPD mortality increased for females but not males, while males showed greater decreases in coronary heart disease and motor vehicle crash mortality than did females. Increasing lung cancer and COPD mortality among women are closely linked to the widespread adoption of smoking behaviors among American women beginning in the post-World War II era (USDHHS 1990).

Summary of Connecticut Black Resident Mortality

Leading Causes of Death and Premature Mortality, 1989 - 1998

Heart disease was the leading cause of death for black males during the 1990s, followed by all cancers and HIV infection. Heart disease was the leading cause of premature mortality, accounting for 13.2% of black male deaths under age 75 in the 1996-98 period, followed by homicide and legal intervention (12.9%) and cancer (12.1%) [Table 26.1].

Heart disease was the leading cause of death for black females during the 1990s, followed by all cancers and cerebrovascular disease. Cancer was the leading cause of premature mortality, accounting for 17.7% of all black female deaths under age 75 in the 1996-98 period, followed by heart disease (16.8%) and HIV infection (9.0%) [Table 26.1].

1996-1998 Deaths, Black Connecticut Residents

- Heart disease was the leading cause of death for black males and females followed by cancer.

- Heart disease was the leading cause of premature mortality under age 75 for black males followed by homicide and legal intervention.

- Cancer was the leading cause of premature mortality under age 75 for black females followed by heart disease.

- Compared with black females, black males had significantly higher mortality rates for all causes of death, heart disease, all cancers, lung and other respiratory cancer, chronic liver disease & cirrhosis, COPD-related causes, HIV infection, septicemia-related causes, unintentional injuries, motor vehicle crashes, homicide and legal intervention, all poisoning, and drug-induced causes.

- Compared with white males, black males had significantly higher mortality due to HIV infection, septicemia, septicemia-related causes, unintentional injuries, homicide and legal intervention, poisoning, drug-induced causes, all cancers, lung and other respiratory cancer, prostate cancer, hypertension-related, diabetes, and diabetes-related causes.

- Compared with white females, black females had significantly higher mortality due to HIV infection, septicemia, septicemia-related causes, homicide and legal intervention, heart disease, cerebrovascular disease, hypertension-related causes, diabetes, diabetes-related causes, and breast cancer.

- Compared with white males and females, black males and females had significantly lower mortality due to COPD and COPD-related causes.

Table 26.1   Leading Causes of Premature Mortality[1] by Gender
Connecticut Black[2] Residents, 1996-98

Cause of Death	Number of Deaths	Rank – Deaths[3]	Deaths Under Age 75	YPLL[4] Before Age 75	Rank
All Black Residents					
Diseases of the heart	1,612	1	971	15,225	1
All cancer	1,329	2	939	14,851	2
HIV infection	314	3	313	10,521	3
Homicide and legal intervention	221	6	220	10,273	4
Unintentional injuries	281	5	260	9,435	5
Suicide	66	11	64	2,710	6
Cerebrovascular disease	287	4	153	2,457	7
Diabetes mellitus	215	7	142	1,980	8
Pneumonia & influenza	178	8	92	1,873	9
Septicemia	97	10	58	1,309	10
All Black Males					
Diseases of the heart	748	1	523	8,539	1
Homicide and legal intervention	179	5	178	8,328	2
All cancer	715	2	518	7,819	3
Unintentional injuries	201	4	192	7,017	4
HIV infection	213	3	212	6,946	5
Suicide	54	9	53	2,338	6
Cerebrovascular disease	105	6	74	1,357	7
Pneumonia & influenza	87	8	55	1,314	8
Diabetes mellitus	100	7	70	1,080	9
Septicemia	45	11	31	755	10
All Black Females					
All cancer	614	2	421	7,032	1
Diseases of the heart	864	1	448	6,686	2
HIV infection	101	5	101	3,575	3
Unintentional injuries	80	7	68	2,418	4
Homicide and legal intervention	42	11	42	1,945	5
Cerebrovascular disease	182	3	79	1,100	6
Diabetes mellitus	115	4	72	900	7
COPD	50	10	33	720	8
Pneumonia & influenza	91	6	37	560	9
Septicemia	52	9	27	555	10

Notes:
1. Premature mortality is defined as the total number of years of potential life lost (YPLL) before age 75. See Section V—Appendix II of this report for a discussion of the YPLL measure.
2. Black Connecticut residents include persons of Hispanic ethnicity.
3. These ranks are based on the National Center for Health Statistics (NCHS) leading causes of death list. See Section V—Appendix V for a complete list of leading causes of death in Connecticut.
4. "YPLL before age 75" indicates the total number of potential life years lost before age 75.

Gender differences in mortality

Compared with black females, black male residents experienced significantly higher age-adjusted mortality from all causes of death. For the period 1996-98, the age-adjusted all-cause death rate for black males was 50% higher than that of black female residents. The largest mortality gaps between black males and females exist in homicide and legal intervention, drug-induced deaths, poisoning, alcohol-induced deaths, unintentional injuries, motor vehicle crashes, and HIV infection (Table 26.2).

Black male excess deaths were calculated in comparison with black female deaths. Excess deaths are those that would not have occurred if the black male population had the same rate as the black female population, and are presented on an annualized or per year basis. It is estimated that black males had 347 excess deaths per year compared with females from 1996 to 1998. The largest numbers of excess deaths among black males were in all cancers, lung and other respiratory cancer, heart disease, homicide and legal intervention, HIV infection, and unintentional injuries. Excess male deaths due to suicide, falls and fall-related injuries, and alcohol-induced deaths were not calculated because of the small number of female deaths in these categories (Table 26.2).

Black vs. White Mortality by Gender

Black vs. white differences in mortality are summarized in Table 26.3. Black residents had significantly higher age-adjusted all-cause mortality compared with white residents of Connecticut with an estimated 438 excess deaths per year for the 1996-1998 period.

In 1996-1998, black male Connecticut residents had significantly higher mortality due to HIV infection, septicemia, septicemia-related causes, unintentional injuries, homicide and legal intervention, poisoning, drug-induced causes, all cancers, lung and other respiratory cancer, prostate cancer, hypertension-related causes, diabetes, and diabetes-related causes compared with white males. There were no statistically significant differences between black and white males in age-adjusted death rates due to heart disease, cerebrovascular disease, colorectal cancer, chronic liver disease and cirrhosis, motor vehicle crashes, suicide, alcohol-induced causes, and pneumonia and influenza deaths (Table 26.3).

Black female Connecticut residents had significantly higher mortality due to HIV infection, septicemia, septicemia-related causes, homicide and legal intervention, heart disease, cerebrovascular disease, hypertension-related causes, diabetes, diabetes-related causes, and breast cancer compared with white females. There were no statistically significant differences between black and white females in age-adjusted death rates due to all cancers, lung and other respiratory cancers, colorectal cancer, chronic liver disease and cirrhosis, unintentional injuries, motor vehicle crashes, poisoning, drug-induced causes, and pneumonia and influenza deaths (Table 26.3).

Black males and females had significantly lower mortality due to COPD and COPD-related causes compared with white males and females in Connecticut.

Table 26.2 Summary Table of Black Male and Female Mortality[1]
Connecticut Residents, 1996-98

Cause of Death	Black Males		Black Females		Male-Female Relative Risk[3,4]	Male Excess Deaths[5]
	Deaths	AAMR[2]	Deaths	AAMR[2]		
All Causes	3,118	1,270.9	2,780	846.9	1.5**	347
Chronic Diseases						
Heart disease	748	356.5	864	282.5	1.3**	52
Cerebrovascular	105	50.1	182	62.1	0.8	(8)
Hypertension-related	243	113.1	331	106.0	1.1	5
Diabetes	100	48.0	115	36.3	1.3	8
Diabetes-related	283	131.3	416	134.2	1.0	(2)
All cancer	715	332.8	614	187.3	1.8**	104
Lung & other respiratory cancer	221	100.7	126	37.9	2.7**	46
Colorectal cancer	64	27.3	65	19.9	1.4	6
COPD	45	22.0	50	15.1	1.5	5
COPD-related	155	83.4	129	41.5	2.0**	16
Chronic liver disease & cirrhosis	36	12.6	22	5.5	2.3*	7
Injury						
Unintentional injuries	201	57.2	80	19.3	3.0**	44
Motor vehicle crashes	74	18.8	26	5.8	3.2**	17
Fall & fall-related injuries	12	—	3	—	—	
Suicide	54	12.5	12	—	—	
Homicide & legal intervention	179	38.4	42	8.3	4.5**	46
All poisoning	71	18.6	24	5.1	3.6**	17
Alcohol-induced	36	11.7	13	—	—	
Drug-induced	67	17.3	21	4.5	3.8**	17
Infectious Diseases						
Pneumonia & influenza	87	44.0	91	30.9	1.4	9
Septicemia	45	20.0	52	16.5	1.2	3
Septicemia-related	188	85.7	190	58.8	1.5*	20
HIV infection	213	56.1	101	21.1	2.7**	44

Notes:
1. Black race includes persons of any ethnicity.
2. Age-adjusted Mortality Rates (AAMR) are per 100,000 based on race and ethnicity specific population estimates. Age-adjusted rates were calculated by the direct method using the 2000 U.S. standard population. Rates were not calculated for fewer than 15 events.
3. Relative risk is the ratio of the Connecticut black male resident age-adjusted mortality rate (AAMR) to the black female resident AAMR (2000 U.S. standard population). Relative risks are not calculated for fewer than 15 deaths in either the black male or female population.
4. Statistical tests were conducted to evaluate differences in black male and female resident AAMRs. Following are explanations of the notations:
    * Significantly different at p < .05.
    ** Significantly different at p < .01.
    — Rate (and relative risk) were not calculated due to small numbers.
5. Excess deaths are those deaths that would not have occurred if the black male population had the same rate as the black female population, and are presented on an annualized or per year basis. Parentheses indicate fewer deaths.

Table 26.3   Summary Table of Black-White Mortality Disparities by Gender[1]
Connecticut Residents, 1996-98

Cause of Death	All		Males		Females	
	Black-White Relative Risk[2,3]	Excess (Fewer) Deaths[4]	Black-White Relative Risk[2,3]	Excess (Fewer) Deaths[4]	Black-White Relative Risk[2]	Excess (Fewer) Deaths[4]
All Causes	1.3**	438	1.3**	240	1.3**	192
Chronic Diseases						
Heart disease	1.2**	90	1.1	20	1.3**	70
Cerebrovascular	1.1	10	0.9	(2)	1.3*	12
Hypertension-related	2.0**	98	2.0**	40	2.1**	58
Diabetes	2.4**	41	2.3**	19	2.4**	22
Diabetes-related	2.0**	117	1.6**	35	2.4**	82
All cancer	1.2**	83	1.4**	66	1.1	17
Lung & other respiratory cancer	1.1	16	1.4**	21	0.9	(5)
Colorectal cancer	1.1	5	1.1	2	1.1	3
Breast cancer (females only)	—	—	—	—	1.3*	11
Prostate cancer (males only)	—	—	2.4**	23	—	—
COPD	0.5**	(31)	0.5**	(14)	0.5**	(17)
COPD-related	0.7**	(37)	0.8*	(12)	0.6**	(25)
Chronic liver disease & cirrhosis	1.0	1	1.1	1	1.0	0
Injury						
Unintentional injuries	1.2*	16	1.3*	16	1.0	0
Motor vehicle crashes	1.1	5	1.4	7	0.8	(2)
Fall & fall-related injuries	0.5**	—	—	—	—	—
Suicide	0.9	(2)	0.9	(2)	—	—
Homicide & legal intervention	8.7**	66	10.1**	54	5.9**	12
All poisoning	1.3	8	1.5*	7	1.1	1
Alcohol-induced	1.5	5	1.6	5	—	—
Drug-induced	1.4*	8	1.6*	8	1.0	0
Infectious Diseases						
Pneumonia & influenza	1.1	5	1.1	1	1.2	4
Septicemia	1.8**	14	1.7*	6	1.9**	8
Septicemia-related	1.9**	59	1.9**	30	1.8**	29
HIV infection	7.5**	92	7.5**	62	9.2**	30

Notes:

1. Black and white race includes persons of Hispanic ethnicity.
2. Relative risk is the ratio of the black Connecticut resident age-adjusted mortality rate (AAMR) to the white Connecticut resident AAMR (2000 U.S. standard population). Relative risks are not calculated for fewer than 15 deaths in either the black or white population.
3. Statistical tests were conducted to evaluate differences in black and white resident AAMRs. Following are explanations of the notations:
     *   Significantly different than the white resident rate at $p < .05$.
     ** Significantly different than the white resident rate at $p < .01$.
     — Not calculated due to small numbers of deaths or no deaths.
4. Excess deaths are those deaths that would not have occurred if the black population had the same rate as the white population, and are presented on an annualized or per year basis. Parentheses indicate fewer events. The total number of excess deaths is the sum of the total excess deaths of males and females.

These figures are generally consistent with national statistics showing that black Americans have higher age-adjusted mortality rates for all causes, all cancer, cardiovascular diseases, diabetes, chronic liver disease and cirrhosis, HIV infection, septicemia, homicide and legal intervention, and unintentional injuries compared with white Americans. Black Americans tend to have lower age-adjusted mortality due to COPD and suicide compared with white Americans (Centers for Disease Control and Prevention 2004).

Black vs. White Mortality by Age Group

There are some important differences in the black-white mortality differential by age group. The disparity in black-white Connecticut male all-cause mortality (1996-1998 period) differed significantly by age group (p < .0014).   For black compared with white males under age 65, the relative risk of death was consistent at 2.3 (p < .001), while the disparity for black compared with white males aged 65-84 lessened. Among males aged 85 and over, the all-cause mortality rate was significantly lower for blacks compared with whites. The disparity in black-white female all-cause mortality (1996-1998 period) also differed by age group (p < .0014). For black compared with white females under age 65, the relative risk of death was consistent at 2.0 (p < .001), while the disparity for black compared with white females aged 65-84 lessened. The all-cause mortality rate was significantly lower for black compared with white females aged 85 and over.   These findings are consistent with other studies showing that racial and socioeconomic differences in mortality tend to be most prominent in the middle-adult (35-64 years) age groups (Geronimus 2001; Adler, Boyce, Chesney, et al. 1993; House, Kessler, Herzog et al. 1990; Elo and Preston 1996).

Black vs. White Premature Mortality

Age-adjusted all-cause *premature* mortality under age 75 was significantly higher for black residents of Connecticut compared with white residents. Black males had 2.3 times the all-cause premature mortality rate of white males (1996-98 period) with heart disease, homicide and legal intervention, all cancers, HIV infection, and unintentional injuries contributing to greater overall rates of premature mortality (Table 26.4). Black females had 2.0 times the all-cause premature mortality rate of white females (1996-98 period) with all cancers, heart disease, HIV infection, homicide and legal intervention, and cerebrovascular disease contributing to greater overall premature mortality under age 75 (Table 26.4).

Poverty and lower socioeconomic status (SES) are the most common explanatory factors for black-white disparities in mortality. Poverty and lower SES have been consistently linked to poor health outcomes and higher mortality, and black Americans have consistently had higher poverty rates and lower SES than white Americans (Williams and Collins 1995; Budrys 2003).   Some research suggests, however, that adjustment for SES reduces but does not eliminate the racial disparity in mortality (Otten, Teutsch, Williamson, et al. 1990).

Table 26.4 Summary Table of Black-White Premature Mortality [1]
Disparities by Gender[2] Connecticut Residents, 1996-98

Cause of Death	All Residents Black-White Relative Risk[3,4]	Males Black-White Relative Risk[3,4]	Females Black-White Relative Risk[3,4]
All Causes	2.1**	2.3**	2.0**
Chronic Diseases			
Heart disease	2.0**	1.7**	2.8**
Cerebrovascular	2.3**	2.5**	2.1**
Hypertension-related	4.1**	3.6**	5.3**
Diabetes	2.8**	3.0**	2.7**
Diabetes-related	2.7**	2.4**	3.2**
All cancer	1.4**	1.6**	1.2*
Lung & other respiratory cancer	1.3**	1.7**	1.1
Colorectal cancer	1.9**	1.8*	1.9*
Breast cancer (females only)			1.4*
Prostate cancer (males only)		3.3**	
COPD	1.3	1.2	1.4
COPD-related	1.3*	1.3	1.3
Chronic liver disease & cirrhosis	1.2	1.1	1.4
Injury			
Unintentional injuries	1.3**	1.5**	1.2
Motor vehicle crashes	1.1	1.4	0.7
Fall & fall-related injuries	0.6	—	—
Suicide	1.1	1.2	—
Homicide & legal intervention	9.2**	10.3**	6.8**
All poisoning	1.2	1.3	1.1
Alcohol-induced	1.5	1.6	—
Drug-induced	1.3	1.4	1.1
Infectious Diseases			
Pneumonia & influenza	2.8**	3.3**	2.2*
Septicemia	3.7**	4.0**	3.5**
Septicemia-related	2.9**	3.1**	2.7**
HIV infection	7.1**	6.9**	8.5**

Notes:

1. Premature mortality is measured by the "years of potential life lost" (YPLL) below a specified age. In this table, the YPLL represents the number of years of potential life lost by each death before age 75.

2. Black and white racial groups include persons of Hispanic ethnicity.

3. Relative risk is the ratio of the black Connecticut resident age-adjusted Years of Potential Life Lost (YPLL) rate to the white Connecticut resident YPLL (2000 U.S. standard population). Relative risks are not calculated for fewer than 15 deaths in either the black or white population.

4. Statistical tests were conducted to evaluate differences in black and white resident YPLLs. Following are explanations of the notations: * Significantly different than the white resident rate at $p < .05$.
   ** Significantly different than the white resident rate at $p < .01$.
   — Rate not calculated due to small numbers.

Recent studies have also examined the impact of place of residence on the black-white mortality disparity (Polednak 1996; Polednak 1997; Geronimus, Bound, and Waidmann 1999; Jackson, Anderson, Johnson, et al. 2000). Polednak found that black residential segregation was associated with all-cause mortality after adjusting for poverty (Polednak 1996). Jackson et al. found that black residential segregation affected mortality independently of individual-level measures of socioeconomic status (Jackson, Anderson, Johnson, et al 2000). Geronimus et al. found that specific conditions in various poor communities (e.g. rural vs. urban poverty) appeared to explain differences in causes and levels of excess black mortality (Geronimus, Bound, and Waidmann 1999). Residential segregation of black Americans is associated with inequities in household and neighborhood living conditions, local services, and medical care, which are all factors that may influence health status, morbidity, and mortality.

Time Trend Analyses

We assessed changes in the mortality rates of black male and female Connecticut residents from 1989-1991 to 1996-1998. For both black and white males, there were significant decreases in age-adjusted mortality rates for all causes of death and heart disease. Cerebrovascular disease and COPD mortality rates decreased significantly for black males only. There were no statistically significant increases among black males in age-adjusted death rates for any other causes of death considered in this report. There were no statistically significant changes in age-adjusted death rates of black females for any causes of death during the 1990s.

Changes in Disparities over Time

We assessed changes in black-white mortality disparities over time and found that the black-white male disparity in alcohol-induced deaths decreased significantly from 1989-91 to 1996-98 (BW RR, 1989-91 = 2.7; BW RR, 1996-98 = 1.6). There were no statistically significant increases in black-white male mortality disparity from 1989-91 to 1996-98. There were no statistically significant changes in the black-white female mortality disparity during the 1990s for any of the causes of death considered in this report.

The Connecticut black resident mortality trends presented here reinforce previous findings that significant disparities exist in the major causes of death for black residents compared with white residents (Hynes, Mueller, Hofmann, et al. 1999). Better understanding of the underlying causes of these disparities requires that we pay attention to the specific conditions in local communities that put black residents at risk for premature mortality. Elimination of these disparities, a goal of *Healthy People 2010*, will require the implementation of broad-based social policy initiatives that improve the living conditions in local Connecticut communities and address racial disparities in health care access and quality.

Summary of Connecticut Hispanic Resident Mortality

Leading Causes of Death and Premature Mortality, 1989 - 1998

Heart disease was the leading cause of death for Hispanic males during the 1990s, followed by all cancers, unintentional injuries, HIV infection, and homicide and legal intervention (Table 27.1). Unintentional injuries were the leading cause of premature mortality accounting for 19% of Hispanic male deaths under age 75, followed by HIV infection (13%), and heart disease (10%) in the 1996-98 period.

Heart disease was the leading cause of death for Hispanic females during the 1990s, followed by all cancers, cerebrovascular disease, HIV infection, and unintentional injuries (Table 27.1). Cancer was the leading cause of premature mortality, accounting for 15% of all Hispanic female deaths under age 75, followed by heart disease (13%), and HIV infection (11%) in the 1996-98 period.

1996-1998 Deaths, Hispanic Connecticut Residents

- Heart disease was the leading cause of death for Hispanic males and females followed by cancer.

- Unintentional injuries were the leading cause of premature mortality under age 75 for Hispanic males followed by HIV infection.

- Heart disease was the leading cause of premature mortality under age 75 for Hispanic females followed by cancer.

- Compared with Hispanic females, Hispanic males had significantly higher mortality from all causes of death.

- Compared with white males, Hispanic males had significantly lower mortality due to heart disease, hypertension-related conditions, all cancers, lung and other respiratory cancer, prostate cancer, COPD and COPD-related conditions, and pneumonia and influenza.

- Compared with white males, Hispanic males had significantly higher mortality due to chronic liver disease and cirrhosis, HIV infection, unintentional injuries, homicide and legal intervention, all poisoning, and alcohol-induced and drug-induced conditions.

- Compared with white females, Hispanic females had significantly lower mortality due to heart disease, cerebrovascular disease, hypertension-related causes, all cancers, lung and other respiratory cancer, colorectal cancer, breast cancer, COPD and COPD-related conditions.

- Compared with white females, Hispanic females had significantly higher mortality due to diabetes-related causes and HIV.

Table 27.1 Leading Causes of Death and Premature Mortality[1] by Gender
Connecticut Hispanic[2] Residents, 1996-98

Cause of Death	Number of Deaths	Rank – Deaths[3]	Deaths Under Age 75	YPLL[4] Under Age 75	Rank - Premature Mortality
All Hispanic Residents					
Unintentional injuries	212	3	204	8,441	1
HIV infection	182	4	182	6,427	2
Diseases of the heart	499	1	290	5,682	3
All cancer	389	2	287	5,540	4
Homicide and legal intervention	69	8	69	3,252	5
Suicide	39	11	39	1,593	6
Chronic liver disease and cirrhosis	77	6	71	1,573	7
Cerebrovascular disease	114	5	65	1,260	8
COPD	63	9	38	1,044	9
Diabetes mellitus	75	7	54	745	10
All Hispanic Males					
Unintentional injuries	164	3	159	6,574	1
HIV infection	131	4	131	4,473	2
Diseases of the heart	251	1	170	3,415	3
All cancer	208	2	149	2,860	4
Homicide and legal intervention	60	5	60	2,805	5
Suicide	34	9	34	1,375	6
Chronic liver disease and cirrhosis	55	6	53	1,233	7
Cerebrovascular disease	55	7	38	772	8
COPD	32	10	20	677	9
Diabetes mellitus	42	8	32	465	10
All Hispanic Females					
All cancer	181	2	138	2,680	1
Diseases of the heart	248	1	120	2,267	2
HIV infection	51	4	51	1,955	3
Unintentional injuries	48	5	45	1,868	4
Cerebrovascular disease	59	3	27	488	5
Homicide and legal intervention	9	12	9	447	6
COPD	31	8	18	367	7
Chronic liver disease and cirrhosis	22	9	18	340	8
Diabetes mellitus	33	6	22	280	9
Pneumonia & Influenza	32	7	10	242	10

Notes:

1. Premature mortality is defined as the total number of years of potential life lost (YPLL) before age 75. See Section V.—Appendix II of this report for a discussion of the YPLL measure.
2. Hispanic ethnicity includes persons of any race.
3. These ranks are based on the National Center for Health Statistics (NCHS) leading causes of death list. See Section V, Appendix V for a complete list of leading causes of death in Connecticut.
4. "YPLL before age 75" indicates the total number of potential life years lost before age 75.

Limitations on time trend analyses

Before the early 1990s, there was substantial missing information on Hispanic ethnicity of deceased persons. Reporting of Hispanic ethnicity on the Connecticut death certificate improved during the decade particularly after 1989 and 1990 when 67% and 23% of all decedents, respectively, were missing information on Hispanic ethnicity. By 1998 and 1999, only 3% of all death records were missing information on Hispanic ethnicity (see Appendix II "Hispanic origin" for a detailed discussion of data quality).

During the 1990s, age-adjusted death rates for all causes of death and the leading causes of death did not appear to change significantly for Connecticut Hispanic residents; however, because there was substantial missing information regarding Hispanic ethnicity until the mid-1990s, we consider these time trend analyses inconclusive.

Gender differences in mortality

Compared with Hispanic females, Hispanic male residents experienced significantly higher age-adjusted mortality from all causes of death. For the period 1996-98, the age-adjusted all-cause death rate for Hispanic males was 68% higher than that of Hispanic female residents. This represents a larger gender gap in mortality compared with all state residents, in which male all-cause mortality is about 50% higher than all-cause female mortality. Connecticut Hispanic males had higher age-adjusted death rates compared with Hispanic females for most causes of death considered in this report. The largest mortality gaps between Hispanic males and females exist in drug-induced, alcohol-induced, and poisoning deaths, unintentional injuries and motor vehicle crashes, homicide and legal intervention, HIV infection, septicemia-related conditions, heart disease, all cancers, colorectal cancer, lung and other respiratory cancer, chronic liver disease and cirrhosis, diabetes, and COPD-related conditions (Table 27.2).

Hispanic male excess deaths were calculated in comparison with Hispanic female deaths. Excess deaths are those that would not have occurred if the Hispanic male population had the same rate as the Hispanic female population, and are presented on an annualized or per year basis. It is estimated that Hispanic males had 185 excess deaths per year from 1996 to 1998. The largest numbers of excess deaths among Hispanic males were in unintentional injuries, HIV infection, and all cancers. Excess male deaths due to suicide, homicide and legal intervention, alcohol-induced, and drug-induced causes were not calculated because of the small number of female deaths in these categories (Table 27.2).

Hispanic vs. White Non-Hispanic Mortality by Gender

Mortality differences between Hispanics and non-Hispanic whites are summarized in Table 27.3. Overall, Hispanics had significantly lower age-adjusted all-cause mortality compared with white residents of Connecticut with an estimated 280 fewer deaths per year. Hispanic males had significantly lower age-adjusted mortality due to heart disease, all cancers, lung and other respiratory

Table 27.2   Summary Table of Hispanic Male and Female Mortality[1]
Connecticut Residents, 1996-98

Cause of Death	Hispanic Males		Hispanic Females		Male-Female Relative Risk[3,4]	Male Excess Deaths[5]
	Deaths	AAMR[2]	Deaths	AAMR[2]		
All Causes	1,351	729.3	934	434.7	1.7**	185
Chronic Diseases						
Heart disease	251	184.8	248	136.9	1.3*	19
Cerebrovascular	55	39.7	59	32.9	1.2	3
Hypertension-related	51	40.8	62	36.5	1.1	2
Diabetes	42	30.3	33	17.8	1.7	6
Diabetes-related	133	98.1	133	73.3	1.3	10
All cancer	208	148.3	181	86.2	1.7**	29
Lung & other respiratory cancer	40	28.2	25	13.6	2.1	7
Colorectal cancer	25	17.4	15	7.2	2.4	5
Breast cancer (females only)			33	13.2		
Prostate cancer (males only)	15	—				
COPD	32	21.7	31	16.2	1.3	2
COPD-related	79	66.5	65	34.6	1.9*	12
Chronic liver disease & cirrhosis	55	25.8	22	10.3	2.5**	11
Injury						
Unintentional injuries	164	50.2	48	13.9	3.6**	39
Motor vehicle crashes	55	16.6	21	5.5	3.0**	12
Fall & fall-related injuries	8	—	3	—	—	
Suicide	34	8.9	5	—	—	
Homicide & legal intervention	60	14.6	9	—	—	
All poisoning	73	19.5	15	3.7	5.3**	18
Alcohol-induced	34	14.1	10	—	—	
Drug-induced	72	19.4	15	—	5.2**	18
Infectious Diseases						
Pneumonia & influenza	30	24.9	32	18.4	1.4	3
Septicemia	15	—	18	9.6	1.1	0
Septicemia-related	74	46.7	51	26.0	1.8*	11
HIV infection	131	40.6	51	13.4	3.0**	29

Notes:
1.   Hispanic ethnicity includes persons of any race.
2.   Age-adjusted Mortality Rates (AAMR) are per 100,000 based on race and ethnicity specific population estimates. Age-adjusted rates were calculated by the direct method using the 2000 U.S. standard population. Rates were not calculated for fewer than 15 events.
3.   Relative risk is the ratio of the Connecticut Hispanic male resident age-adjusted mortality rate (AAMR) to the Hispanic female resident AAMR (2000 U.S. standard population). Relative risks are not calculated for fewer than 15 deaths in either the Hispanic male or female population.
4.   Statistical tests were conducted to evaluate differences in Hispanic male and female resident AAMRs. Following are explanations of the notations:
       *   Significantly different at p < .05.
       ** Significantly different at p < .01.
       — Rate (and relative risk) were not calculated due to small numbers.
5.   Excess deaths are those deaths that would not have occurred if the Hispanic male population had the same rate as the Hispanic female population, and are presented on an annualized or per year basis.

cancer, prostate cancer, COPD, and pneumonia and influenza compared with white males. Hispanic males had significantly higher age-adjusted mortality due to chronic liver disease and cirrhosis, HIV infection, unintentional injuries, homicide and legal intervention, poisoning, alcohol-induced, and drug-induced causes compared with white, non-Hispanic males.

Hispanic females had significantly lower age-adjusted mortality due to heart disease, cerebrovascular disease, hypertension-related causes, all cancer, lung and other respiratory cancer, colorectal cancer, breast cancer, COPD and COPD-related causes compared with white, non-Hispanic females. Hispanic females had significantly higher age-adjusted mortality due to diabetes-related causes and HIV infection compared with white, non-Hispanic females (Table 27.3).

These figures parallel national statistics showing that Hispanics have lower age-adjusted and income-adjusted mortality rates for all causes, cancer, and cardiovascular diseases compared with white non-Hispanics (Sorlie, Blacklund, Johnson, et al. 1993). *Lower* socioeconomic status has long been associated with *higher* mortality across various population groups, and so the phenomenon of lower all-cause mortality among U.S. Hispanics who also tend to have lower socioeconomic status compared with white, non-Hispanics has been referred to as the "Hispanic mortality paradox" (Abraído-Lanza, Dohrenwend, Ng-Mak, et al. 1999). Various explanations have been offered for the observed Hispanic mortality advantage over white, non-Hispanics. Some researchers have suggested that the Hispanic mortality advantage is not real but rather an artifact, which may result from misclassification of ethnicity on the death certificates or return migration to the country of origin in later years, both of which would bias Hispanic mortality downward (Swallen and Guend 2003; Abraído-Lanza, Dohrenwend, Ng-Mak, et al. 1999). Other researchers have suggested that the Hispanic mortality advantage in cardiovascular diseases and cancer may be real and related to more favorable health behaviors practiced by Hispanics relative to non-Hispanics (Abraído-Lanza, Dohrenwend, Ng-Mak, et al. 1999). These patterns in Hispanic mortality warrant further study, particularly because Hispanics in the United States and Connecticut are the fastest growing ethnic subpopulation in terms of overall numbers.

Hispanic vs. White Mortality by Age Group

Our analyses of Connecticut Hispanic vs. white resident all-cause mortality suggest that there are some important differences in the all-cause mortality differential by age group in the 1996-98 period. Hispanic males aged 25 to 49 had significantly higher all-cause mortality rates compared with white males. HIV infection, unintentional injuries, and homicide and legal intervention accounted for 49% of all Hispanic male deaths in these age groups compared with 32% of white male deaths. There were no significant differences between Hispanic and white males aged 50 through 69 in all-cause mortality; while there was significantly lower all-cause mortality for Hispanic compared with white males aged 70 and older. Hispanic females have significantly lower mortality than white females (RR=0.7, p < .001 for all ages) and this difference is fairly consistent across age groups with a few exceptions. Hispanic females ages 40-44 have similar all-cause mortality rates to white females, while Hispanic females aged 80 and over have a substantially lower mortality rate than their white counterparts (ages 80-84, RR=0.5, p < .001; ages 85 and over, RR=0.4, p < .001).

Table 27.3   Summary Table of Hispanic and White, Non-Hispanic Mortality Disparities by Gender[1] Connecticut Residents, 1996-98

Cause of Death	All		Males		Females	
	Hispanic-Non-Hispanic White Relative Risk[2,3]	Excess (Fewer) Deaths[4]	Hispanic-Non-Hispanic White Relative Risk[2,3]	Excess (Fewer) Deaths[4]	Hispanic-Non-Hispanic White Relative Risk[2,3]	Excess (Fewer) Deaths[4]
All Causes	0.7**	(280)	0.8**	(127)	0.7**	(153)
Chronic Diseases						
Heart disease	0.6**	(104)	0.6**	(61)	0.7**	(43)
Cerebrovascular	0.7**	(14)	0.8	(5)	0.7**	(9)
Hypertension-related	0.7**	(15)	0.7*	(7)	0.7*	(8)
Diabetes	1.4*	7	1.6	5	1.2	2
Diabetes-related	1.3**	20	1.2	8	1.4**	12
All cancer	0.6**	(99)	0.6**	(41)	0.5**	(58)
Lung & other respiratory cancer	0.4**	(39)	0.4**	(21)	0.3**	(18)
Colorectal cancer	0.6**	(10)	0.7	(3)	0.4**	(7)
Breast cancer (females only)	—	—	—	—	0.5**	(13)
Prostate cancer (males only)	—	—	0.5**	(5)	—	—
COPD	0.5**	(18)	0.5**	(9)	0.5**	(9)
COPD-related	0.6**	(33)	0.7**	(14)	0.5**	(19)
Chronic liver disease & cirrhosis	2.3**	15	2.4**	11	2.0	4
Injury						
Unintentional injuries	1.2	9	1.3*	13	0.8	(4)
Motor vehicle crashes	1.3	5	1.5	6	0.9	(1)
Fall & fall-related injuries	—	—	—	—	—	—
Suicide	0.6**	(9)	0.7	(6)	—	—
Homicide & legal intervention	4.6**	18	6.1**	17	—	—
All poisoning	1.4*	9	1.7**	10	0.8	(1)
Alcohol-induced	2.0**	7	2.2**	6	—	—
Drug-induced	1.6**	11	2.0**	12	0.9	(1)
Infectious Diseases						
Pneumonia & influenza	0.7**	(10)	0.6**	(6)	0.7	(4)
Septicemia	1.0	1	0.9	0	1.2	1
Septicemia-related	0.9	(2)	1.1	2	0.8	(4)
HIV infection	9.0**	55	8.5**	39	12.2**	16

Notes:

1. Hispanic ethnicity includes persons of any race. White race excludes persons of Hispanic ethnicity.
2. Relative risk is the ratio of the Hispanic Connecticut resident age-adjusted mortality rate (AAMR) to the white, non-Hispanic Connecticut resident AAMR (2000 U.S. standard population). Relative risks are not calculated for fewer than 15 deaths in either the Hispanic or white population.
3. Statistical tests were conducted to evaluate differences in Hispanic and non-Hispanic white resident AAMRs. Following are explanations of the notations:
   *   Significantly different than the white resident rate at $p < .05$.
   ** Significantly different than the white resident rate at $p < .01$.
   — Not calculated due to small numbers of deaths or no deaths.
4. Excess deaths are those deaths that would not have occurred if the Hispanic population had the same rate as the white population, and are presented on an annualized or per year basis. Parentheses indicate fewer deaths. The total number of excess deaths is the sum of the total excess deaths of males and females

Table 27.4 Summary Table of Hispanic – White, Non-Hispanic Premature Mortality[1] Disparities by Gender[2] Connecticut Residents, 1996-98

Cause of Death	All Residents Hispanic - Non-Hispanic White Relative Risk[3,4]	Males Hispanic - Non-Hispanic White Relative Risk[3,4]	Females Hispanic - Non-Hispanic White Relative Risk[3,4]
All Causes	1.4**	1.5**	1.2**
Chronic Diseases			
Heart disease	1.0	0.9	1.3
Cerebrovascular	1.8**	2.2**	1.5
Hypertension-related	1.0	1.0	1.1
Diabetes	1.8*	2.1*	1.4
Diabetes-related	1.9**	1.9**	1.9**
All cancer	0.7**	0.8*	0.7**
Lung & other respiratory cancer	0.4**	0.5**	0.3**
Colorectal cancer	1.0	1.0	0.8
Breast cancer (females only)			0.8
Prostate cancer (males only)		0.7	
COPD	1.5	1.9	1.1
COPD-related	1.1	1.2	1.0
Chronic liver disease & cirrhosis	2.6**	3.0**	1.8
Injury			
Unintentional injuries	1.5**	1.7**	1.2
Motor vehicle crashes	1.2	1.4	1.0
Fall & fall-related injuries	—	—	—
Suicide	0.7	0.8	—
Homicide & legal intervention	5.0**	6.4**	—
All poisoning	1.5*	1.8**	0.9
Alcohol-induced	2.4**	2.5**	—
Drug-induced	1.6**	2.0**	1.0
Infectious Diseases			
Pneumonia & influenza	1.3	1.3	1.2
Septicemia	2.0	1.8	2.1
Septicemia-related	1.7**	2.1**	1.3
HIV infection	8.7**	8.1**	11.7**

Notes:

1. Premature mortality is measured by the "years of potential life lost" (YPLL) below a specified age. In this table, the YPLL represents the number of years of potential life lost by each death before age 75.
2. Hispanic ethnicity includes persons of any race. White racial group excludes persons of Hispanic ethnicity.
3. Relative risk is the ratio of the Hispanic Connecticut resident age-adjusted Years of Potential Life Lost (YPLL) rate to the white, non-Hispanic Connecticut resident YPLL (2000 U.S. standard population). Relative risks are not calculated for fewer than 15 deaths in either the Hispanic or white, non-Hispanic population.
4. Statistical tests were conducted to evaluate differences in Hispanic and white, non-Hispanic resident YPLLs. Following are explanations of the notations:
   * Significantly different than the white, non-Hispanic resident rate at $p < .05$.
   ** Significantly different than the white, non-Hispanic resident rate at $p < .01$.
   — Rate not calculated due to small numbers of Hispanic deaths.

Hispanic vs. White Premature Mortality

Age-adjusted all-cause *premature* mortality under age 75 was significantly higher for Connecticut Hispanics compared with white, non-Hispanic residents. Hispanic males had 1.5 times the all-cause premature mortality rate of white, non-Hispanic males with HIV infection, diabetes, cerebrovascular disease, chronic liver disease and cirrhosis, unintentional injuries, and homicide and legal intervention contributing to greater overall rates of premature mortality. Hispanic males had significantly lower premature mortality due to all cancers and lung and other respiratory cancer compared with white, non-Hispanic males (Table 27.4). Hispanic females had 1.2 times the all-cause premature mortality rate of white, non-Hispanic females (1996-98 period) with HIV infection accounting for most of the difference in premature mortality under age 75. Hispanic females had significantly lower premature mortality due to all cancers and lung and other respiratory cancer compared with white females (Table 27.4).

These Connecticut Hispanic resident mortality trends warrant further study over time, particularly as this population group ages, as its migration patterns change, and as the composition of Hispanic ethnic subpopulation groups in Connecticut diversifies.

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