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 $\geq$ 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.

[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].

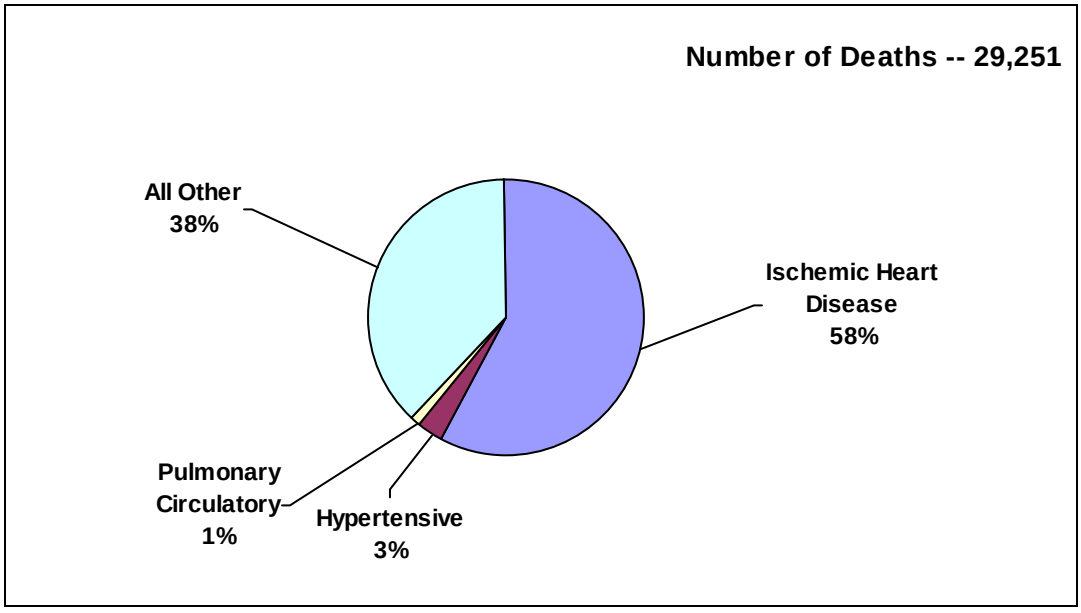
Ischemic heart disease (IHD), the leading type of heart disease death, describes any condition in which the heart muscle works inefficiently or is damaged because of a deficiency or absence of its blood supply. Impairment of the blood's circulation to the heart results most often from atherosclerotic 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

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]	Change since 1989-91[5]	Age-Adjusted Premature Mortality Rates to Age 75[3] YPLL[4]	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]

[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

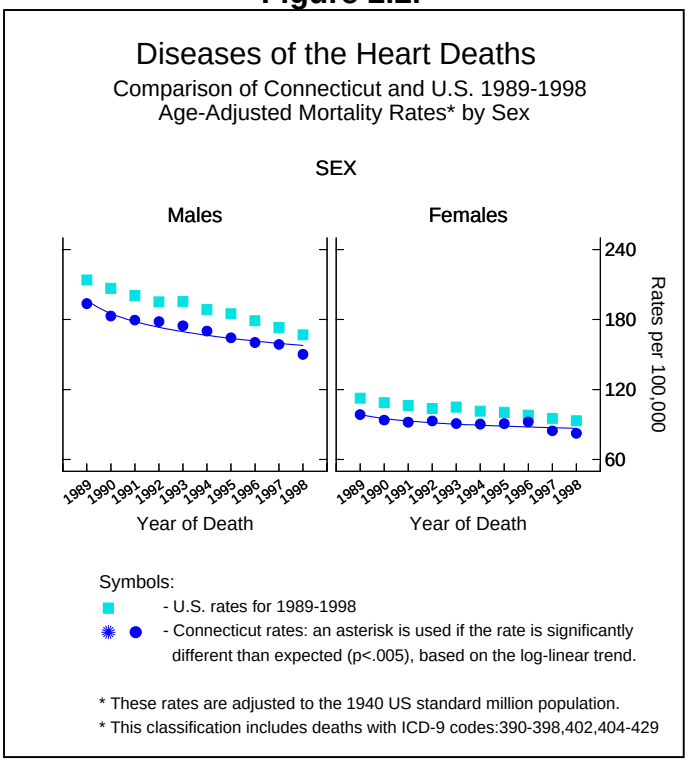
Increasing age is the key risk factor for heart disease. In 1996-1998, those 65 years of age and older accounted for 86% of all heart disease deaths among Connecticut residents. For men, major increases in heart disease mortality rates begin in the 35-to-39-year-old age group; for women, major increases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

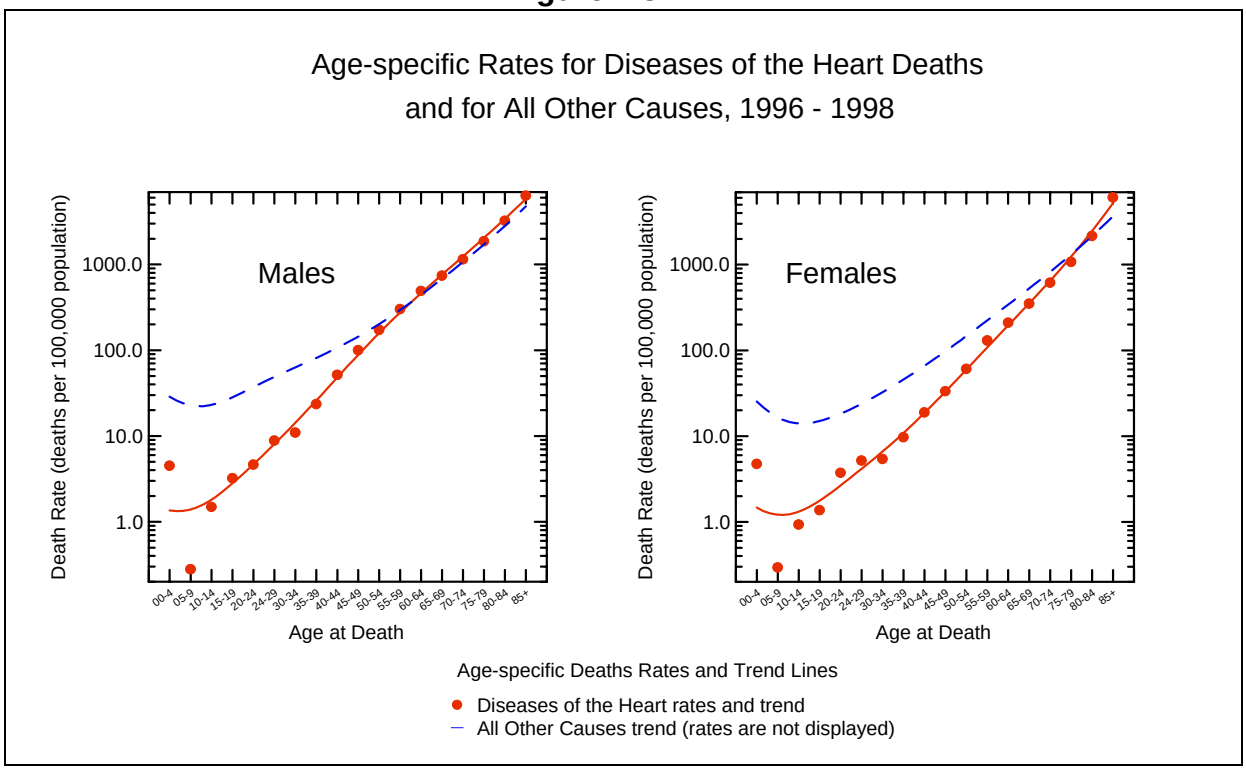
Figure 2.2.



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 2.3.



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

[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

[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

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

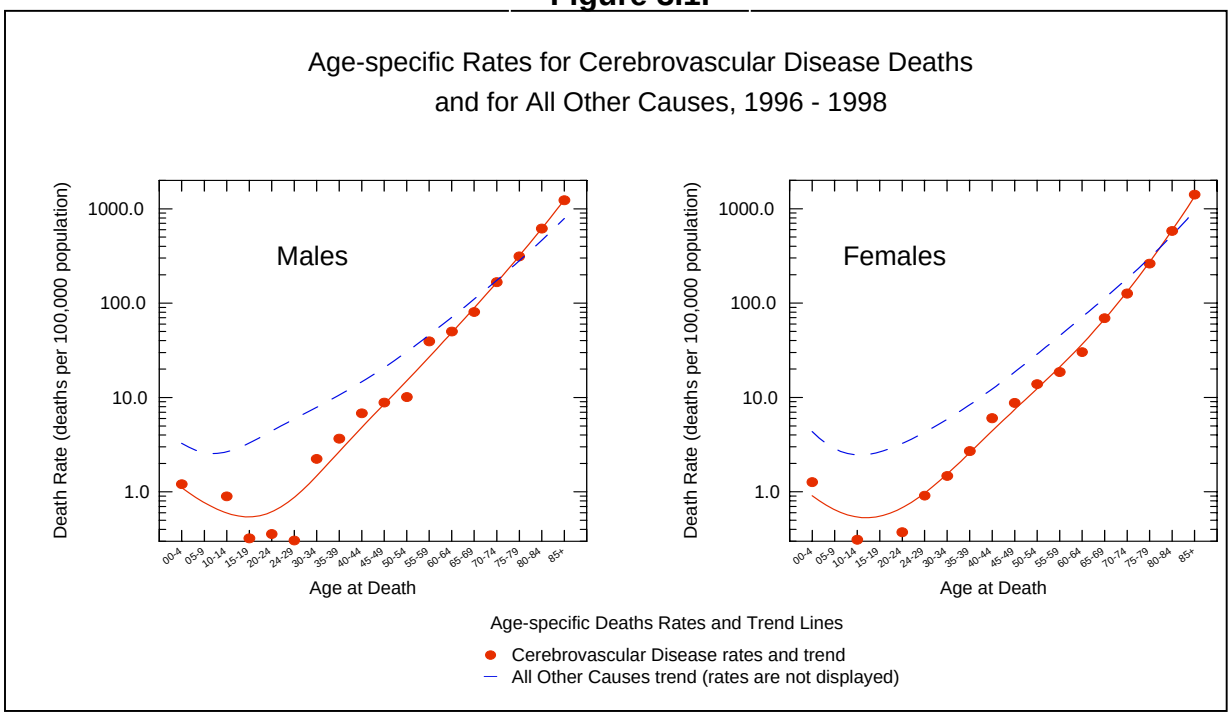
55. Ninety-one percent of all stroke deaths in Connecticut for 1996-1998 occurred among persons 65 and over.

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.



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).

[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] AAMR[4]	Change since 1989-91[5]	Age-Adjusted Premature Mortality Rates to Age 75[3] 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.

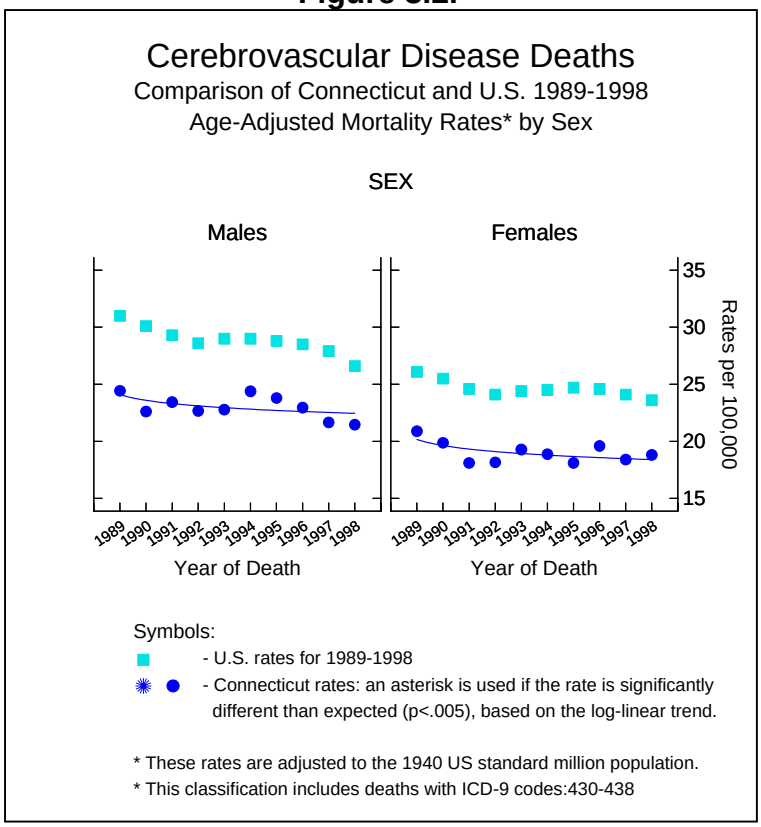


Table 3.2. Cerebrovascular Disease Age-Adjusted Death Rates, Comparison of CT with US - 1989 and 1998

	1989	1998	1998 CT AAMR Comparison
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 ≥ 140 mm Hg)	Strong	20 – 50[2]
Cigarette smoking	Moderate	11 – 25[2]
Diabetes	Moderate	14 – 58[3]
Elevated cholesterol (≥ 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 (≥ 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 ≥ 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

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] AAMR[4]	Change since 1989-91[5]	Age-Adjusted Premature Mortality Rates to Age 75[3] 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.

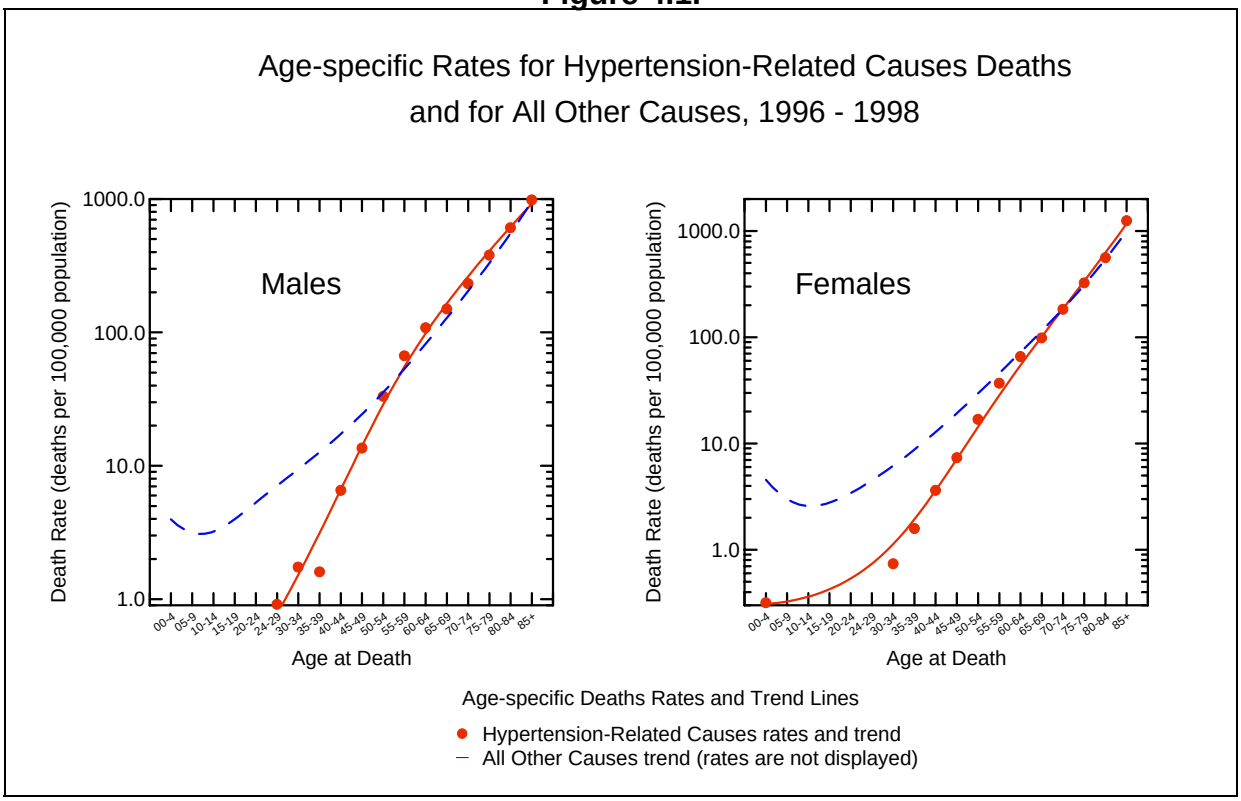
relative risk of 2.0 ($p < .005$), and no significant differences in mortality between black and white women aged 80 and over. There were insufficient numbers of hypertension-related deaths among Hispanic residents within various age groups to detect any age group differences in the Hispanic-white mortality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



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).

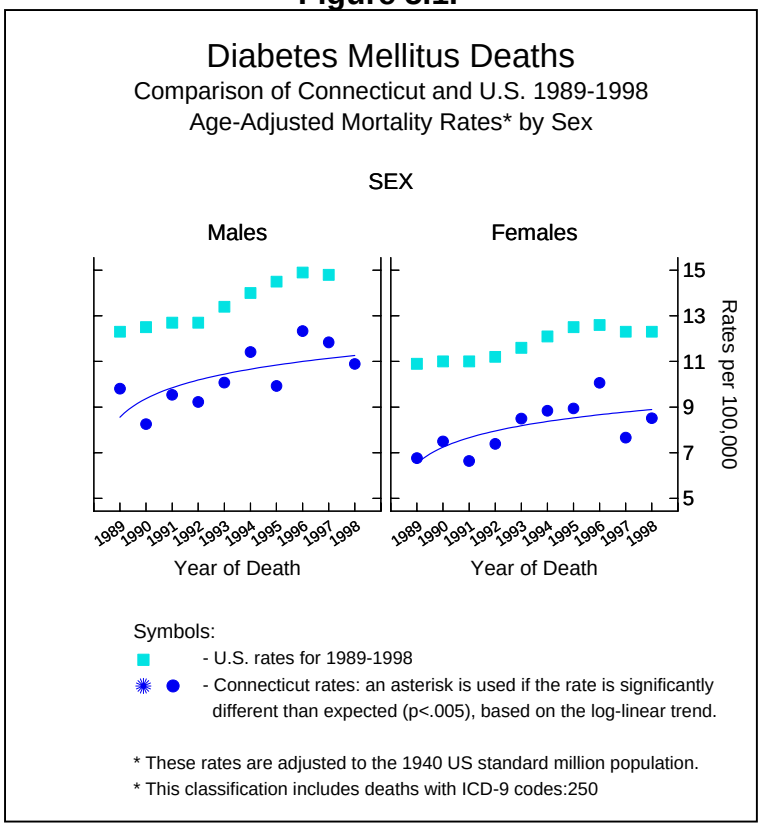
Although age-adjusted diabetes death rates for Connecticut residents were consistently lower than comparable national rates from 1989 to 1998 (Figure 5.1), the increasing death rates for Connecticut residents during this period are notable. Of all the chronic diseases considered in this report, diabetes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

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.

Figure 5.1.



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 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] AAMR[4]	Change since 1989-91[5]	Age-Adjusted Premature Mortality Rates to Age 75[3] 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] AAMR[4]	Change since 1989-91[5]	Age-Adjusted Premature Mortality Rates to Age 75[3] 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.

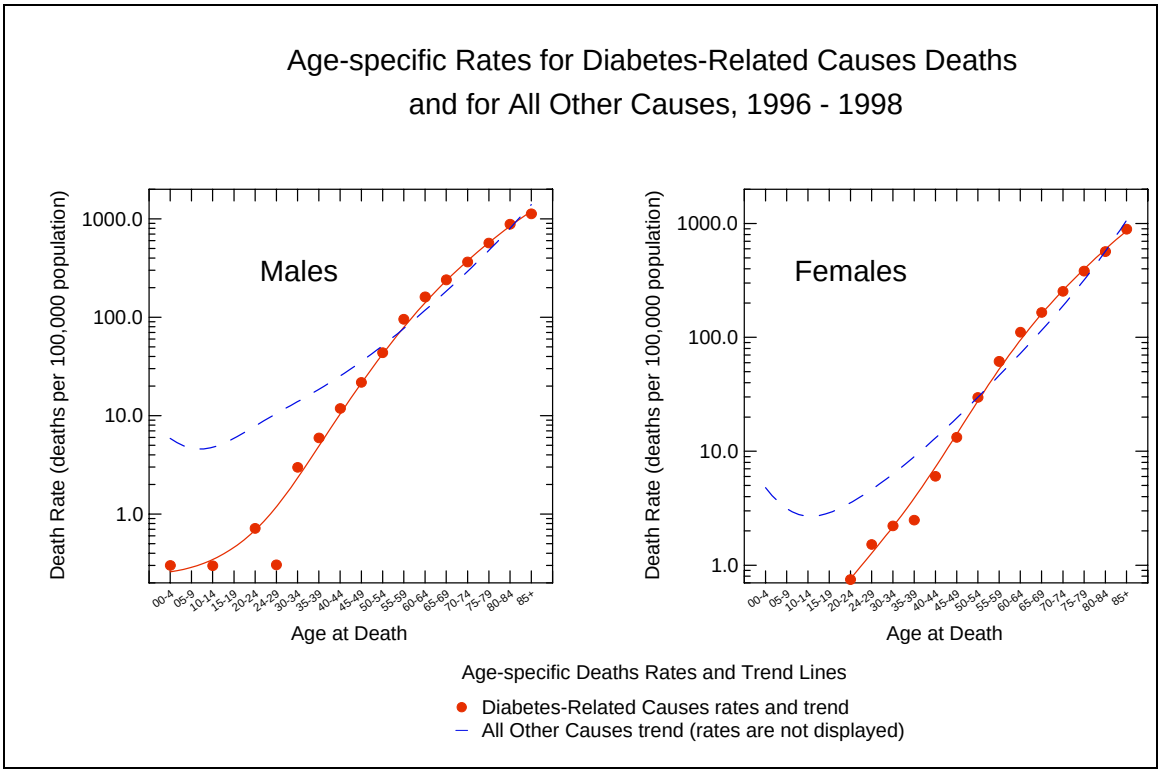
The diabetes mortality rates of Hispanic males and females were not significantly different from the respective white rates in 1996-1998, although Hispanic males did have significantly higher premature mortality due to diabetes compared with white males (Table 5.1). Hispanic females had higher death 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.



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 (≥ 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

[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).

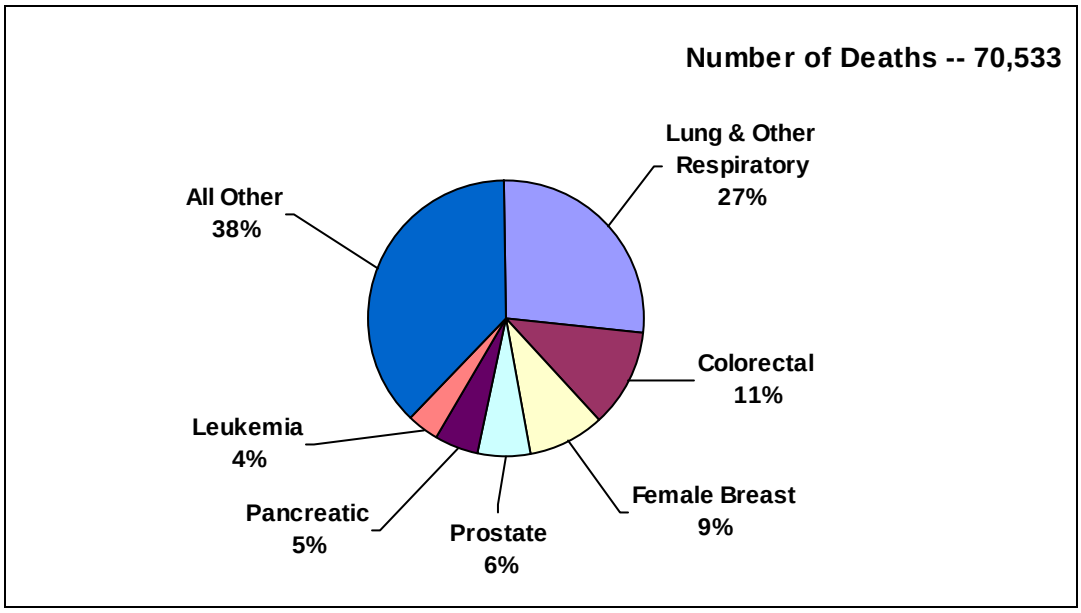
More than half of all cancer deaths in Connecticut and the U.S. are due to lung and other respiratory, colorectal, female breast, and prostate cancers (Figure 6.1) (U.S. Department of Health and Human Services 1990). Mortality from each of these causes is discussed in subsequent sections of this report.

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

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 ($p \leq .05$). 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] AAMR[4]	Change since 1989-91[5]	Age-Adjusted Premature Mortality Rates to Age 75[3] 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.

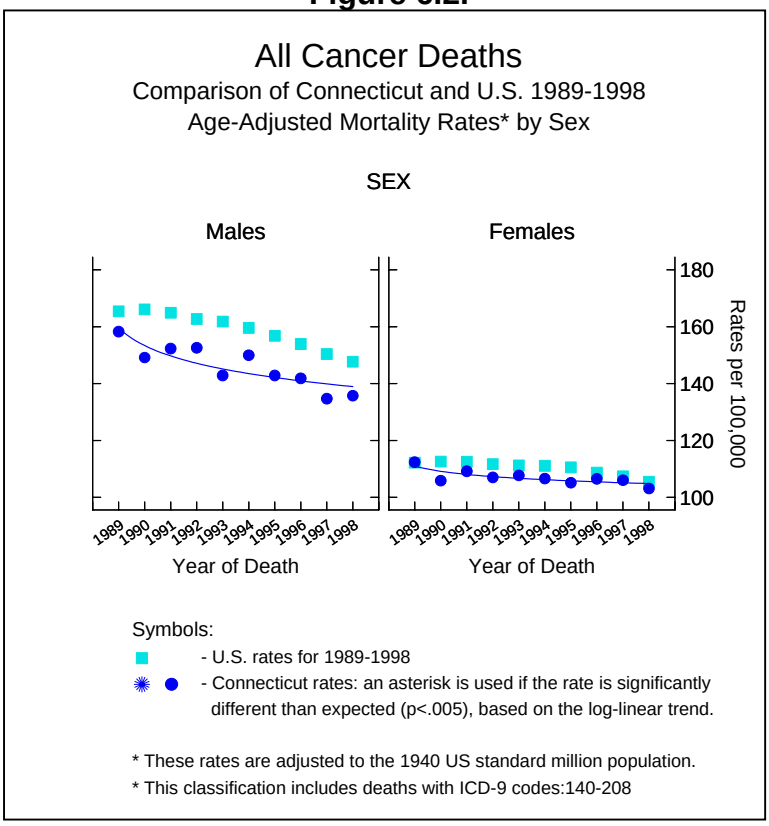
4. Statistical tests were conducted to evaluate differences in rates between race/ethnic groups. The white population serves as the reference group in each comparison (black vs. white, Asian & PI vs. white, Native American vs. white, Hispanic vs. white). Following are explanations of the notations:

   * 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

Figure 6.2.



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 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).

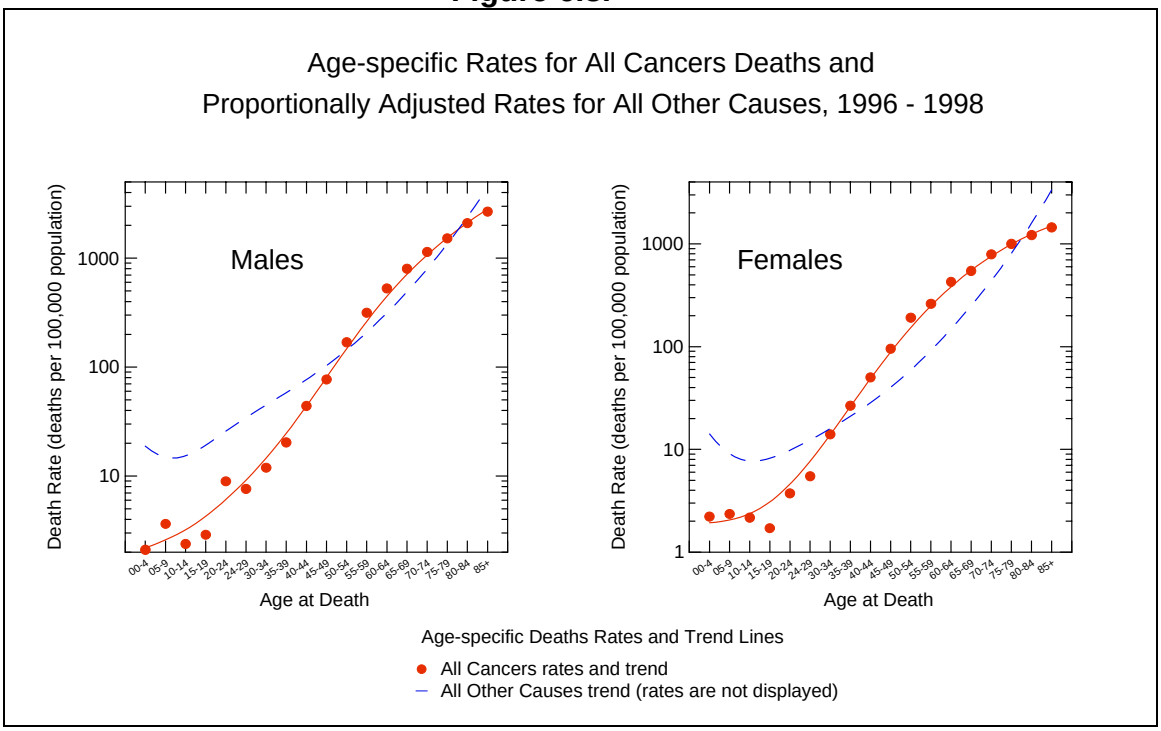
Table 6.2. All Cancer Age-Adjusted Death Rates, Comparison of CT with US - 1989 and 1998

	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.



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).

[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

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

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] AAMR[4]	Change since 1989-91[5]	Age-Adjusted Premature Mortality Rates to Age 75[3] 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

Table 7.2.

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

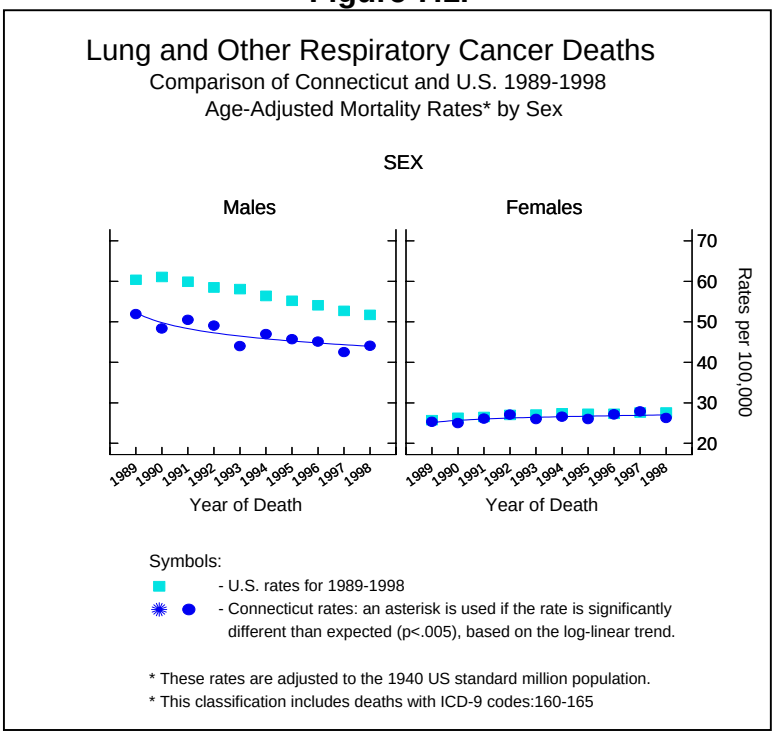
	1989	1998	1998 CT AAMR Comparison
CT AAMR*	36.8	34.1	
US AAMR*	40.7	38.3	CT rate < US rate

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.

Figure 7.1.

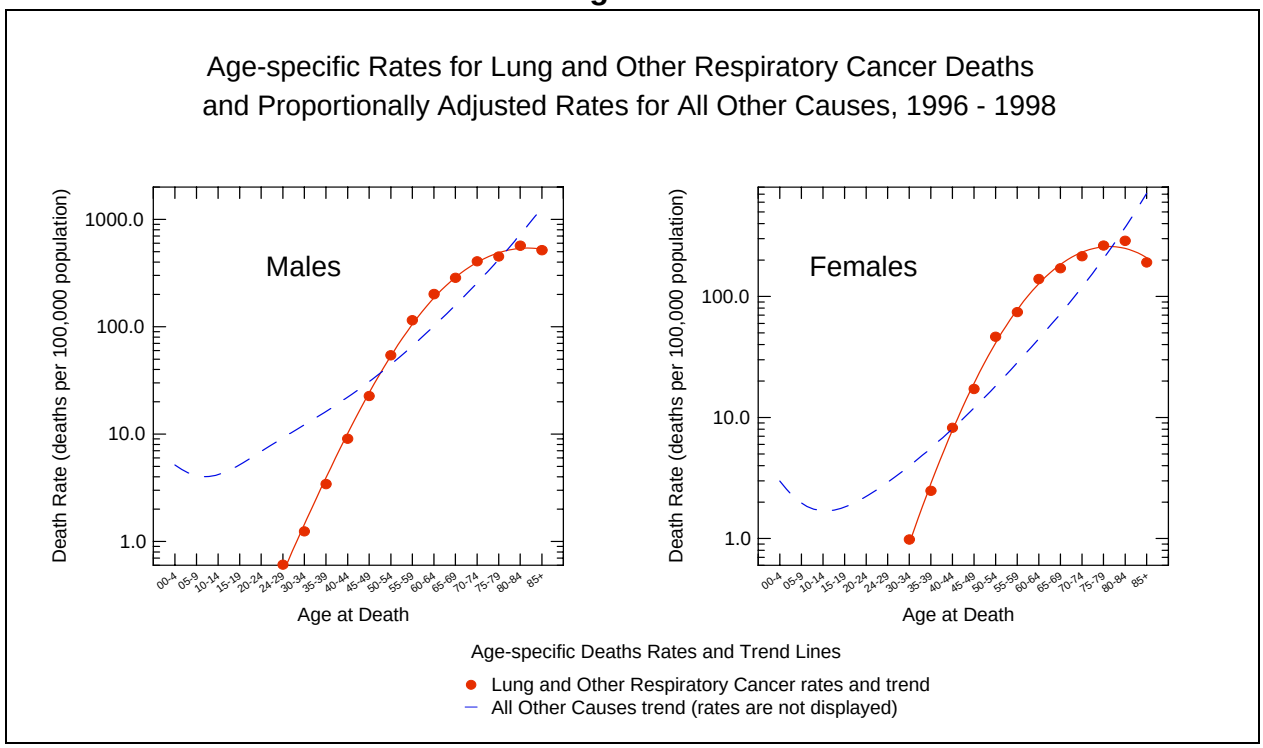


Connecticut resident lung and other respiratory cancer deaths. The Connecticut lung cancer mortality rate for the period 1989 through 1998 was significantly lower than the *Healthy People 2000* and *Healthy Connecticut 2000* target objectives for this period (Table 7.2).

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). 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.



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

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] AAMR[4]	Change since 1989-91[5]	Age-Adjusted Premature Mortality Rates to Age 75[3] 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.

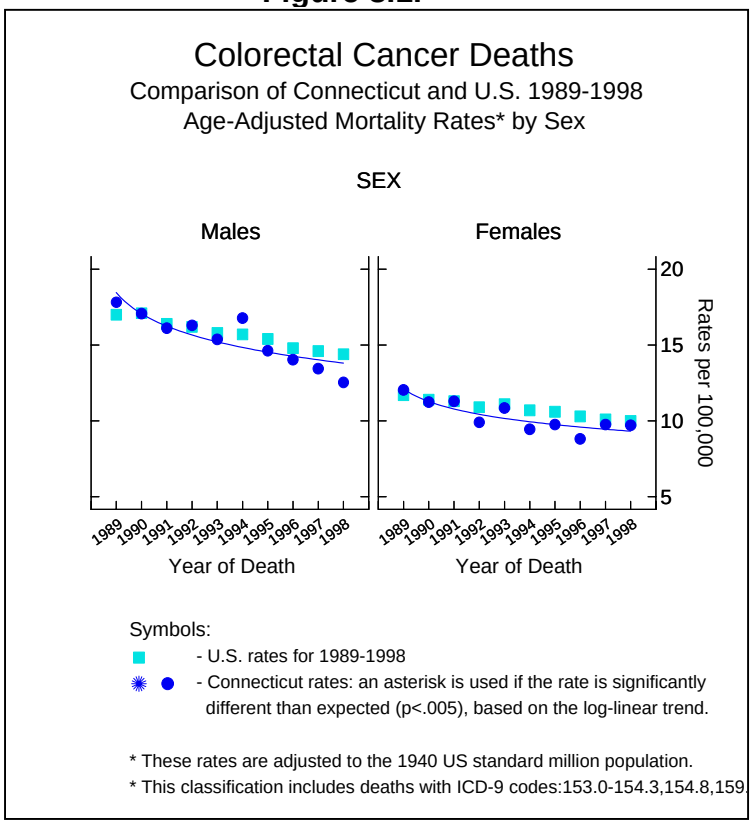
Age-specific colorectal cancer death rates for Connecticut males and females for the period 1996-1998 are displayed in Figure 8.2. Mortality rates for males and females, contrasted with proportionally adjusted rates for all other causes of death, show a pattern of lower rates in the younger age groups, 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).

Figure 8.1.



Risk Factors

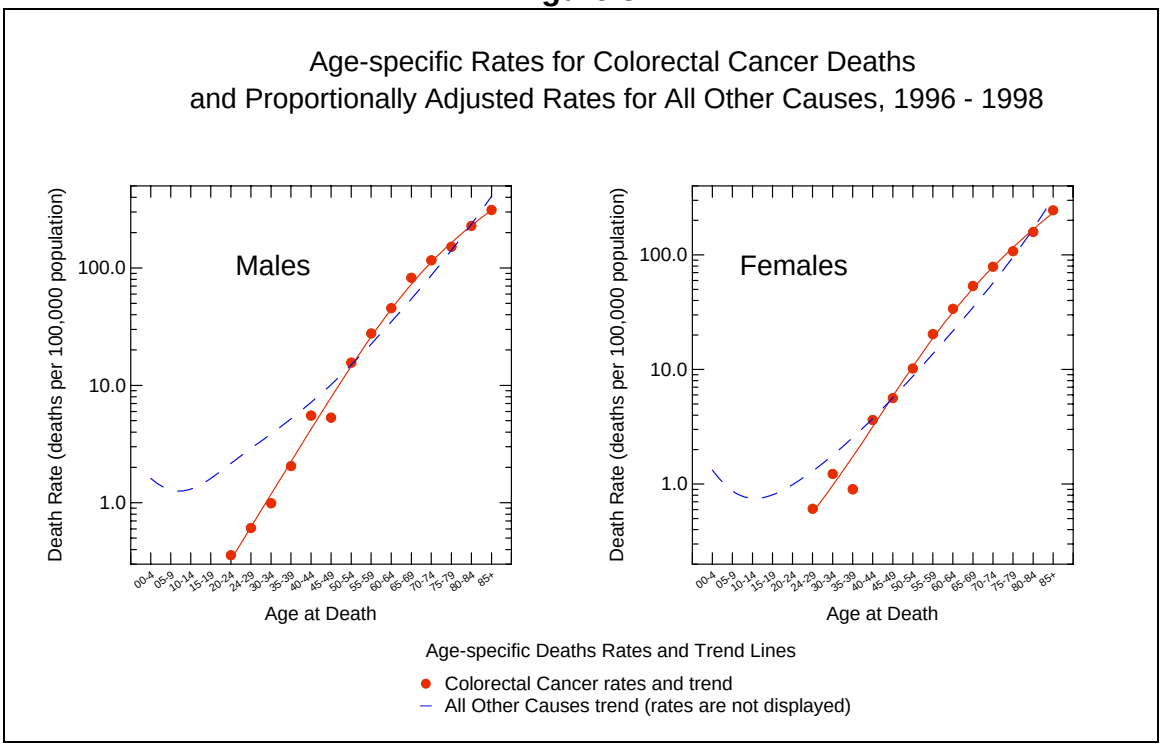
While the etiology of the disease is not well understood, some risk factors for colorectal cancer have been identified. Colorectal cancer incidence increases dramatically after age 50. Familial characteristics associated with increased risk for colorectal cancer include a first-degree family relative 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).

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.



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

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] AAMR[4]	Change since 1989-91[5]	Age-Adjusted Premature Mortality Rates to Age 75[3] 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.

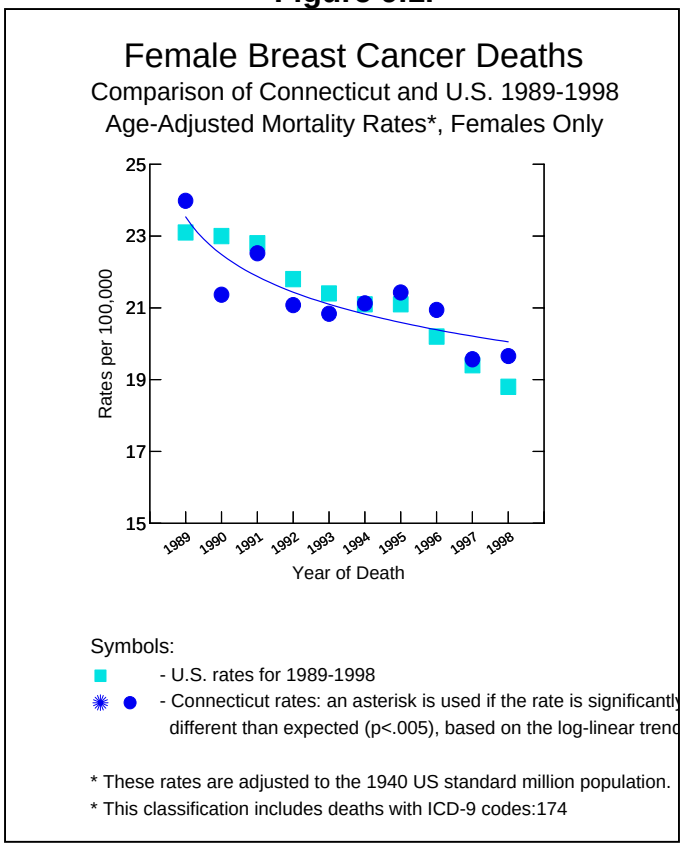
Incidence

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

Figure 9.1.



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).

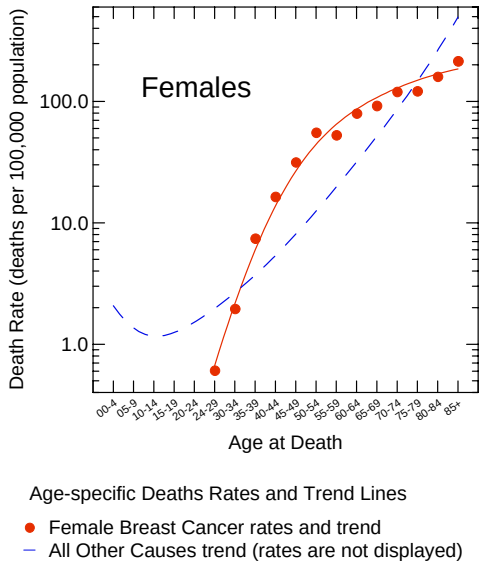
Epidemiological studies have identified several modifiable risk factors for breast cancer (Table 9.3). Few of these risk factors, however, are strongly associated with development of the disease, and each one accounts for a relatively small proportion of overall breast cancer incidence. Reproductive 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

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

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

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] AAMR[4]	Change since 1989-91[5]	Age-Adjusted Premature Mortality Rates to Age 75[3] 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:

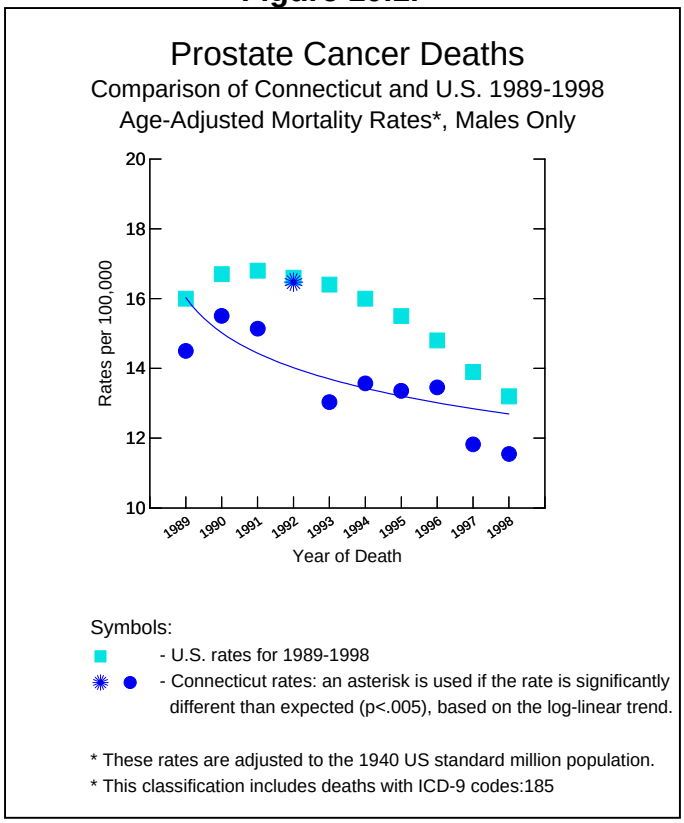
   *** Significantly different from the respective white resident rate at $p < .001$.
   — Rate was not calculated due to small numbers.

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).

Figure 10.1.



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).

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

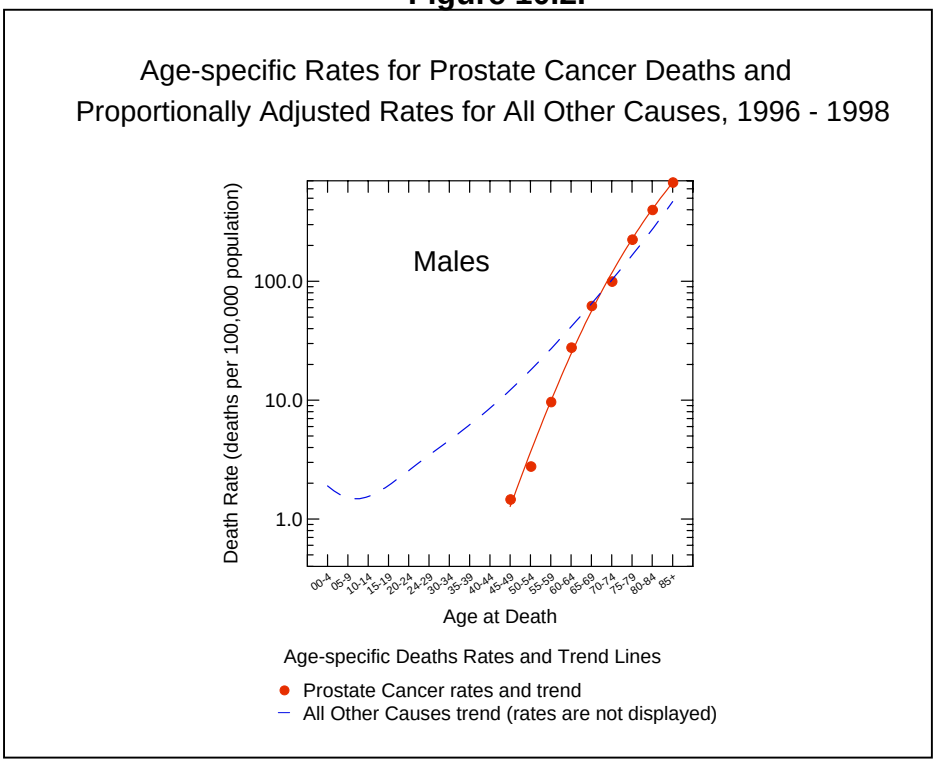
work in certain industries, including farming and rubber manufacturing are also linked to an increased risk for the disease (Brownson, Reif, Alavanja, et al. 1998).

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.



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].

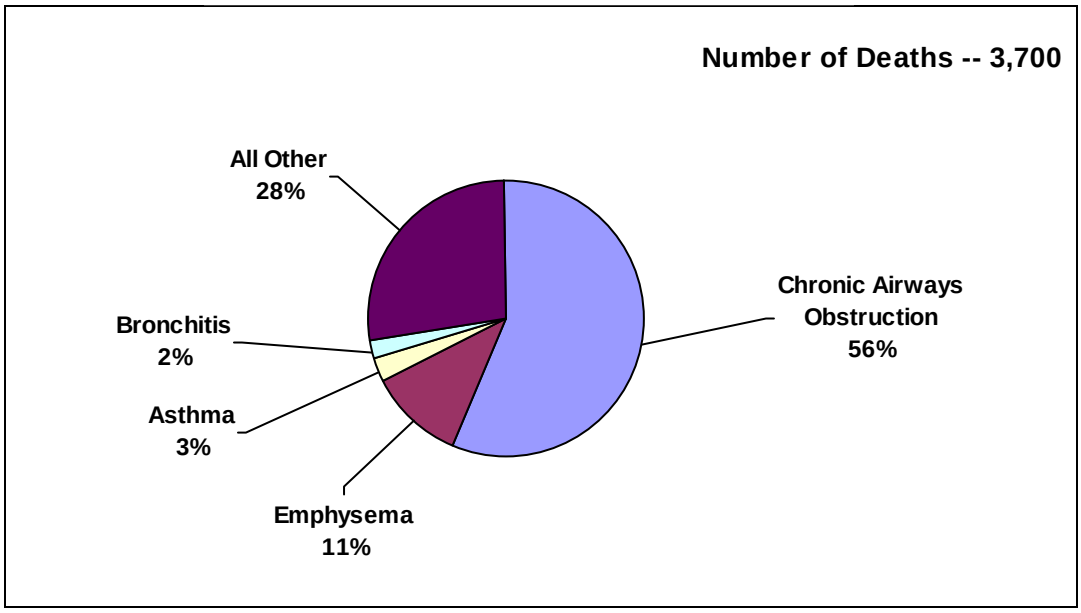
Death in COPD patients often results from some other medical condition or complication. For this reason, and because its contribution to other major causes of death is not always considered, the importance of COPD as a cause of death is probably underestimated. From 1996 to 1998, COPD was the primary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

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



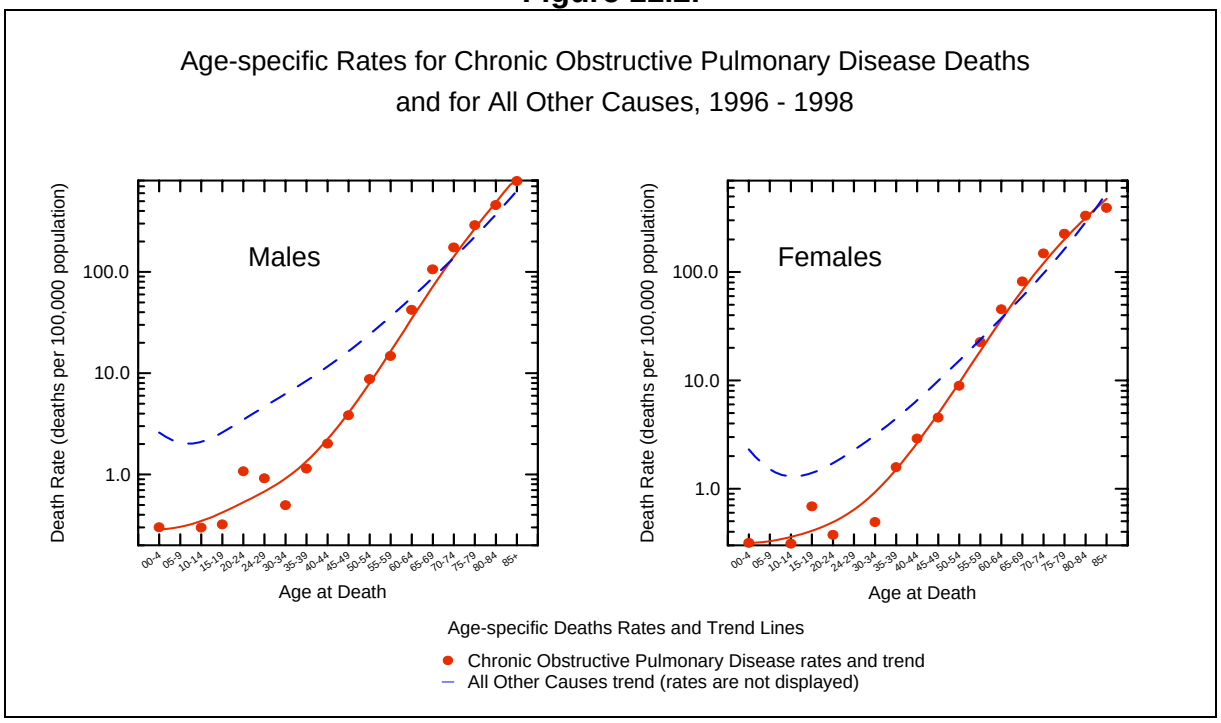
contributing cause, follows a similar pattern.

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.

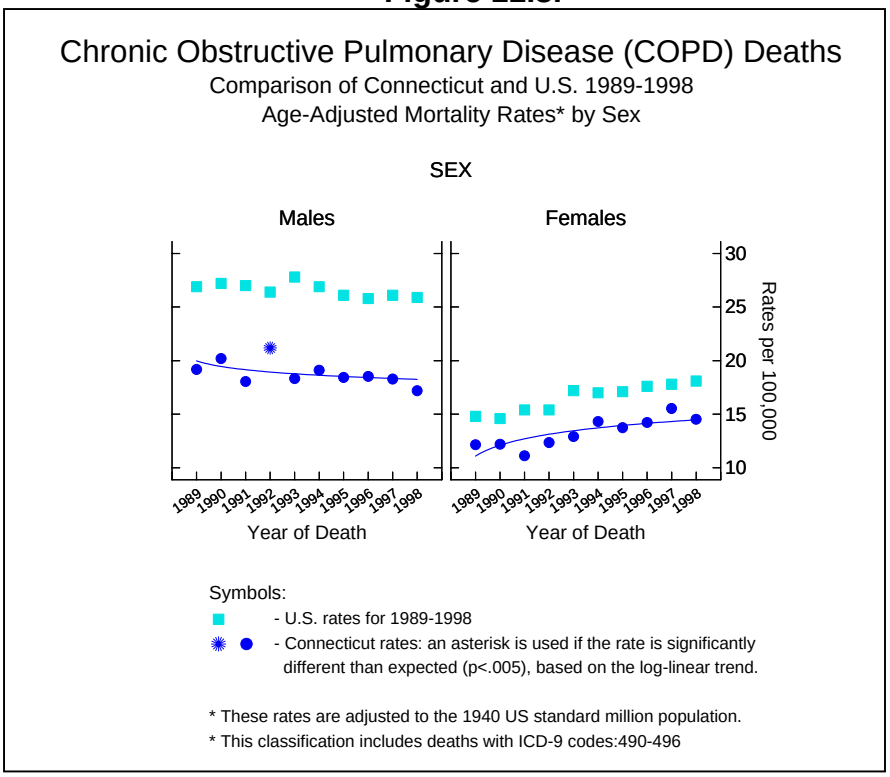


Racial and ethnic differences in mortality reflect differences in the subcategories of COPD deaths by race and ethnicity (Figure 11.4). During the 1996-1998 period, 78% of white resident COPD deaths were due to chronic airways obstruction compared with 60% of black and 49% of Hispanic resident COPD deaths; 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).

Figure 11.3.



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 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] AAMR[4]	Change since 1989-91[5]	Age-Adjusted Premature Mortality Rates to Age 75[3] 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] AAMR[4]	Change since 1989-91[5]	Age-Adjusted Premature Mortality Rates to Age 75[3] 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.

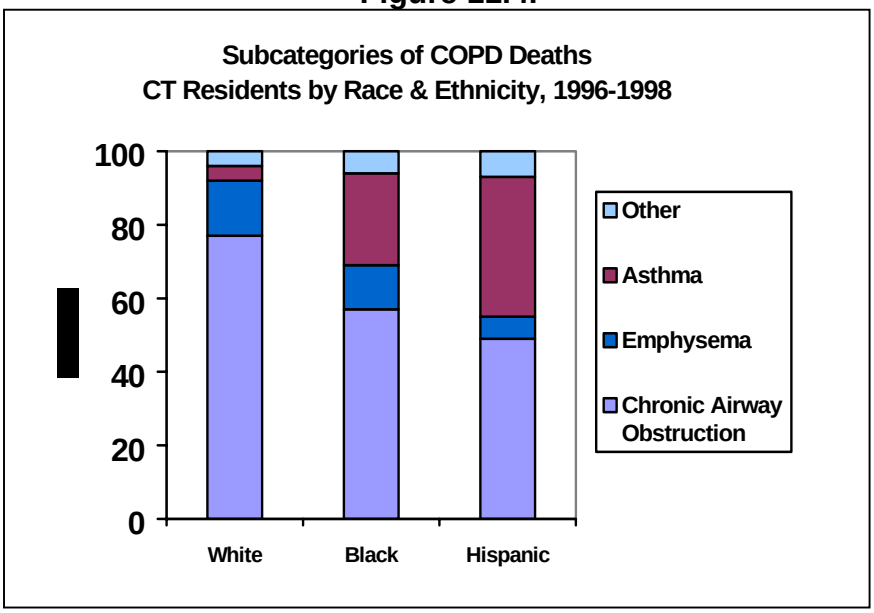
5. Statistical tests were conducted to evaluate changes in rates over time. Comparisons of 1996-98 vs. 1989-91 rates are made within each race/ethnicity group. Following are explanations of the notations:

   ↑ 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.



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

Figure 12.1.
Chronic Liver Disease and Cirrhosis Deaths, Percent by Type
Connecticut Residents, 1996-1998

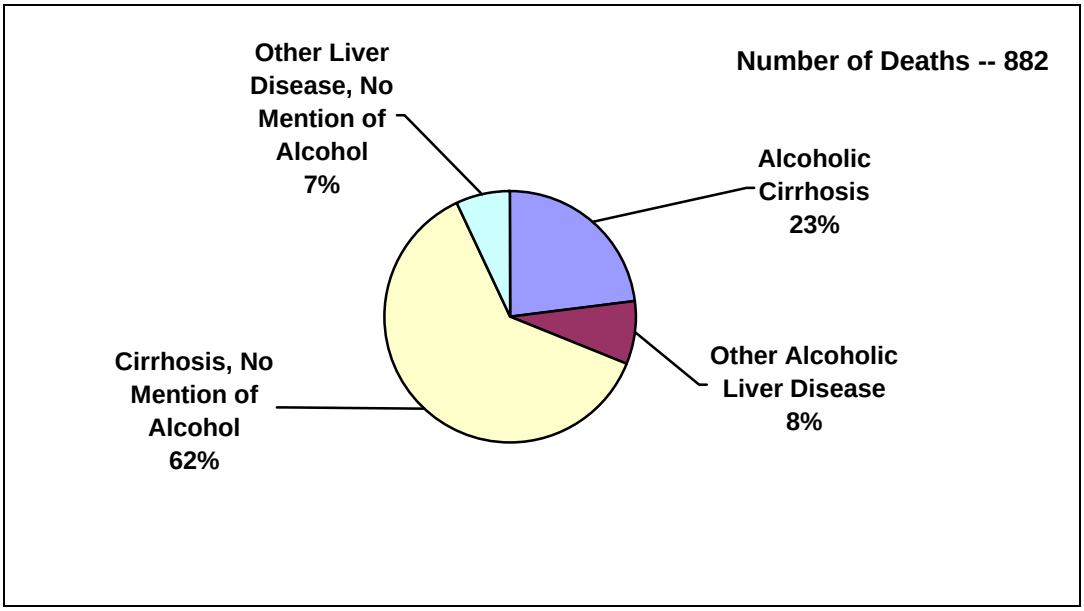


Figure 12.2 depicts age-specific death rates for chronic liver disease and cirrhosis relative to all other causes of death for males and females during this period. Chronic liver disease and cirrhosis death rates were higher compared with all other causes of death for males aged 30 to 69 and for females 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] AAMR[4]	Change since 1989-91[5]	Age-Adjusted Premature Mortality Rates to Age 75[3] 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.

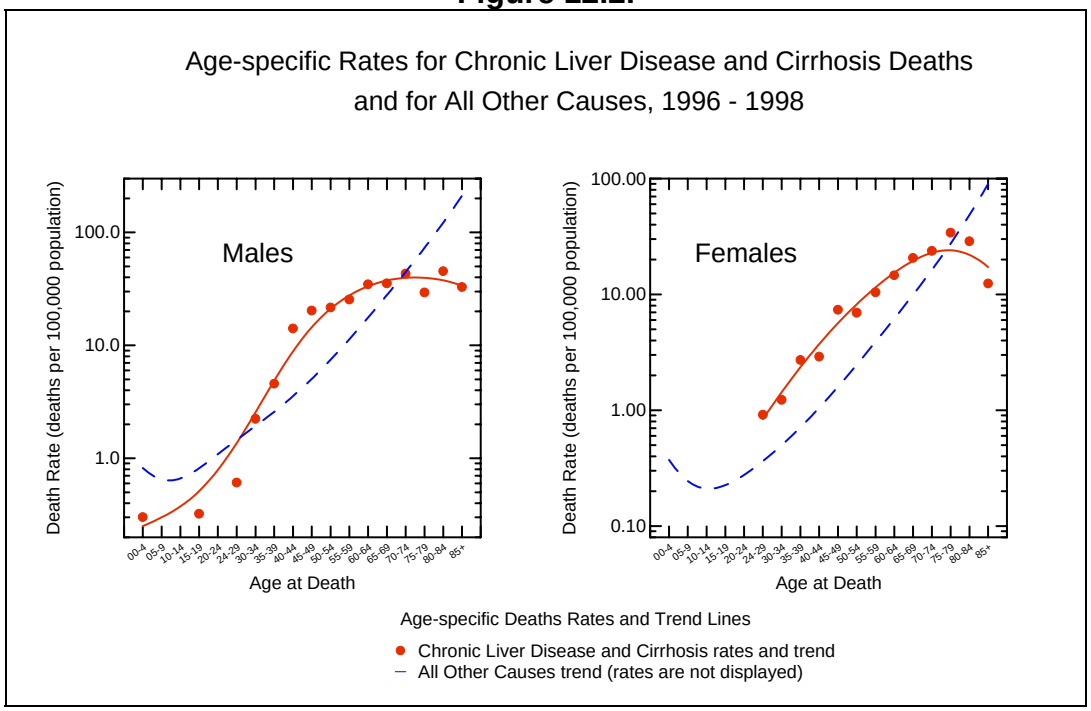
4. Statistical tests were conducted to evaluate differences in rates between race/ethnic groups. The white population serves as the reference group in each comparison (black vs. white, Asian & PI vs. white, Native American vs. white, Hispanic vs. white). Following are explanations of the notations:

   ** 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.



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

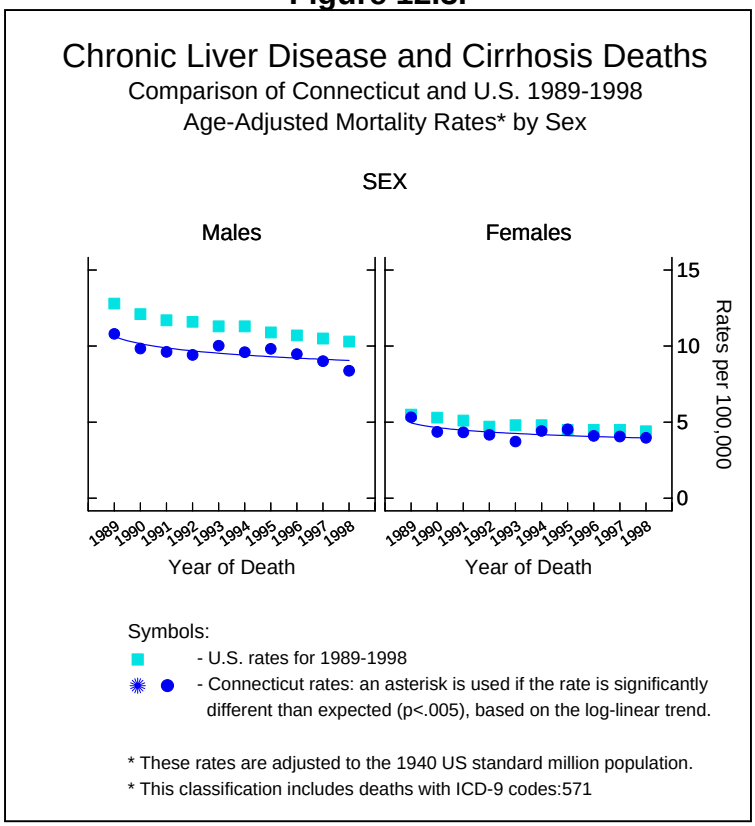
National trends indicate that chronic liver disease and cirrhosis mortality has shown distinctive patterns during the twentieth century. It was high before the Prohibition era, decreased during Prohibition (1920-1933), increased again during the Great Depression (1929-1939) and continued to rise during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

Figure 12.3.



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 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

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.

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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