SECTION IV. C.

INJURY MORTALITY

CONTENTS

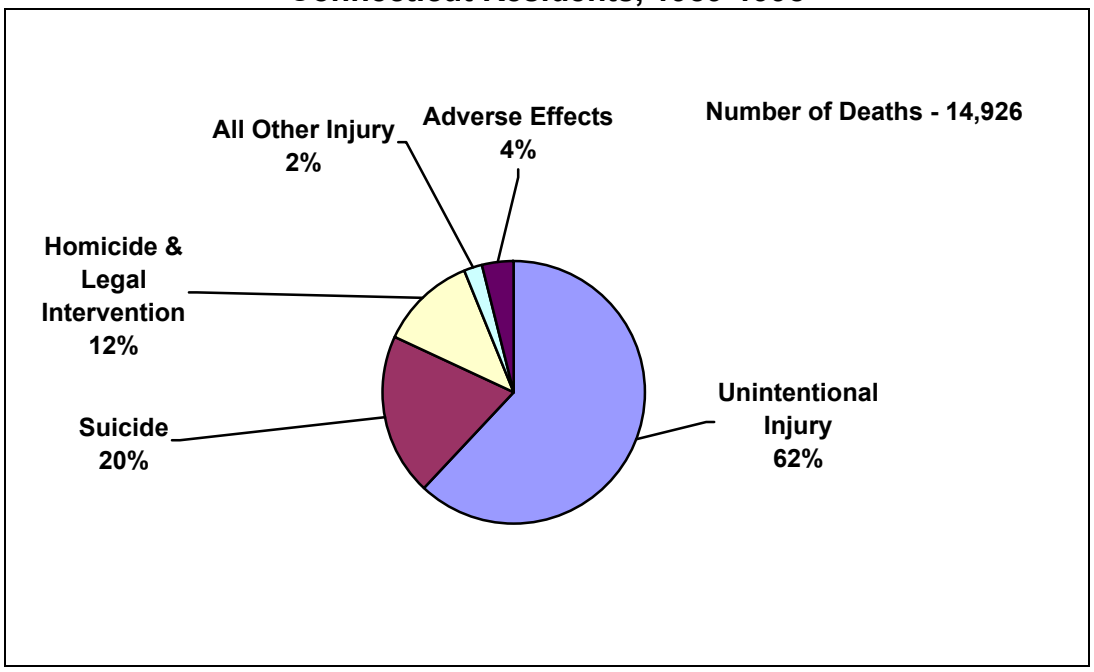
Injury and Other External Causes of Death
(ICD-9 codes E800-E999)

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

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

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

Figure 13.1. Injury and Other External Causes of Death, Percent by Subgroup Connecticut Residents, 1989-1998



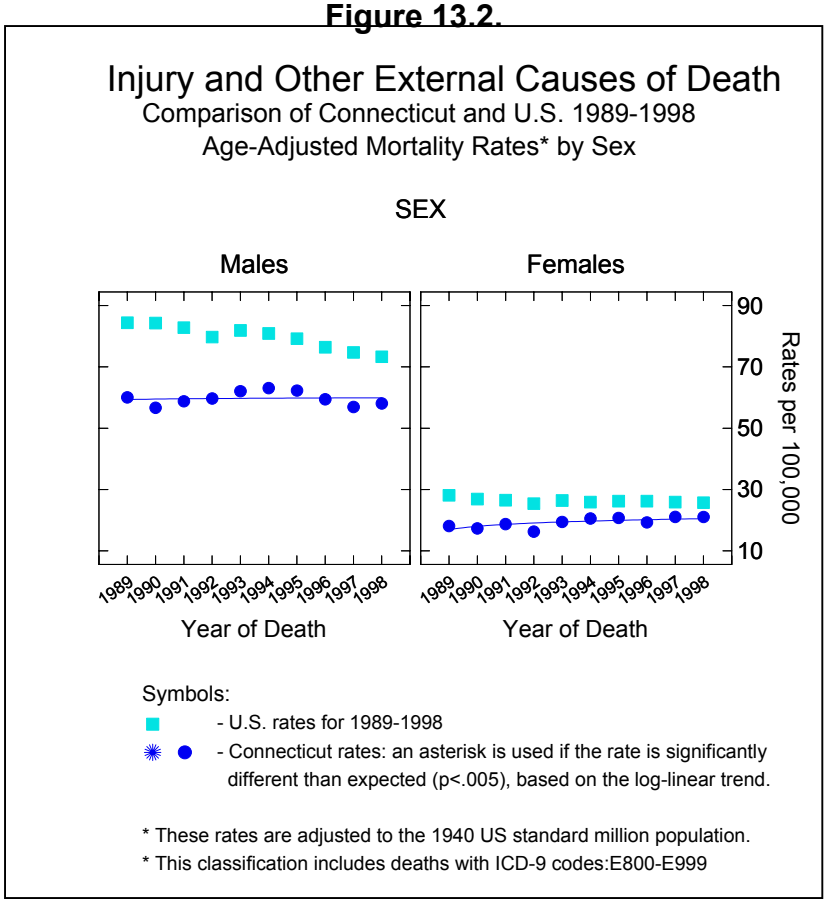
playground, workplace, and on the road.

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

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

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

Figure 13.2.



Unintentional Injury
(ICD-9 codes E800-E949)

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

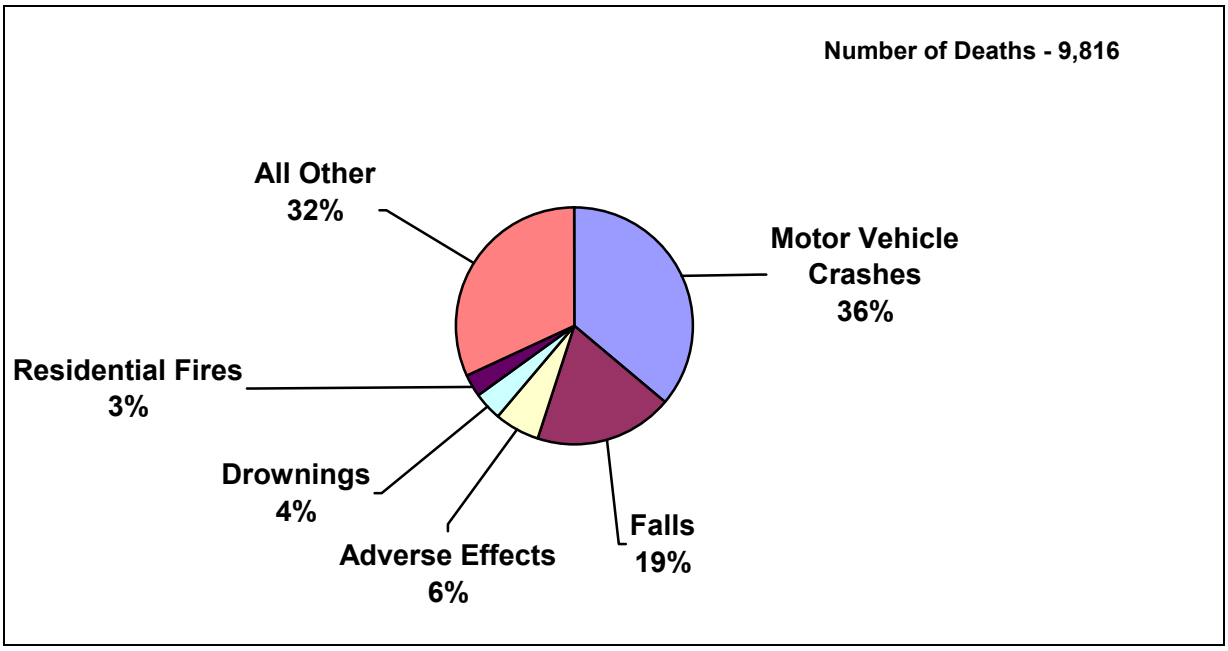
Unintentional injury is the leading cause of death for Connecticut residents under age 45 and the sixth leading cause of death for all Connecticut residents (Appendix V). It is the third leading cause of premature mortality to age 75.

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

1996-1998 Unintentional Injury Deaths, Connecticut Residents

- Sixth leading cause of death for all residents
- The leading cause of death for residents aged 0 – 44
- Third leading cause of premature mortality to age 75
- 3.6% of all deaths
- 2.5% of all deaths among females
- 4.8 % of all deaths among males
- 6.2% of all deaths under 75 years of age
- Significant increase in mortality compared with the 1992-94 period

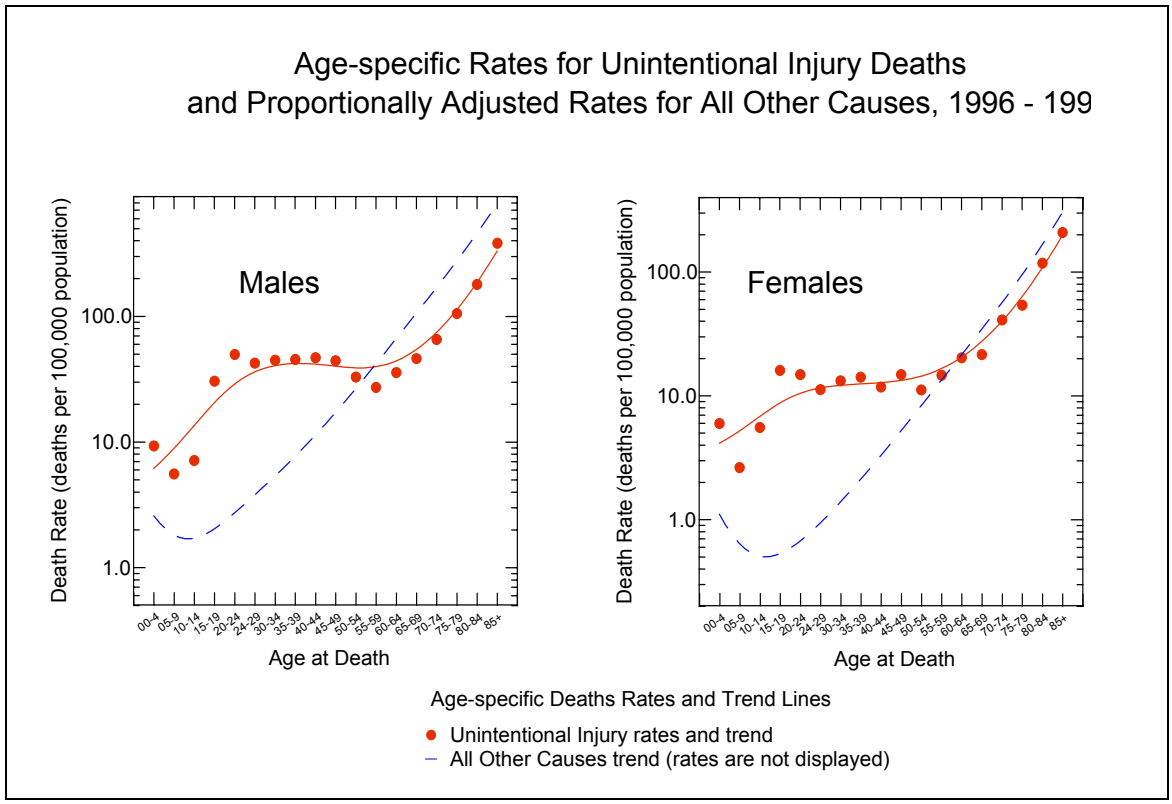
Figure 14.1. Unintentional Injury Deaths, Percent by Type Connecticut Residents, 1989-1998



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

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

Figure 14.2.



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

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

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

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

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

Notes:

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

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

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

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

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

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

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

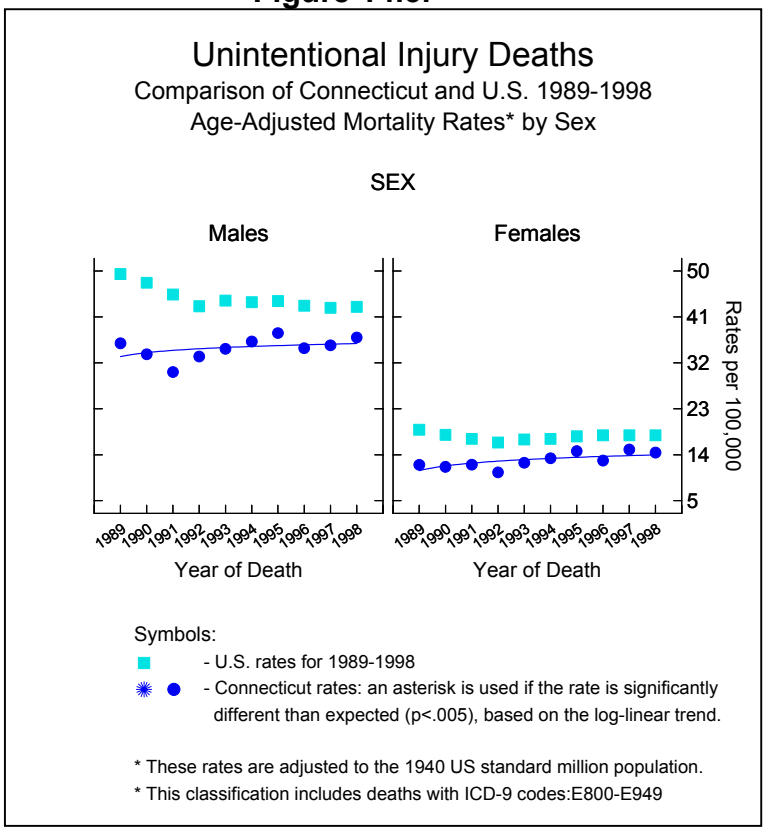
Risk reduction efforts targeting unintentional injuries should be tailored to both the type of injury (motor vehicle, falls, drowning, etc.), the high-risk groups in the population (subgroups of males), and groups that have shown a significant increase over time (white females). Specific categories of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

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

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

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

Figure 14.3.



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

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

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

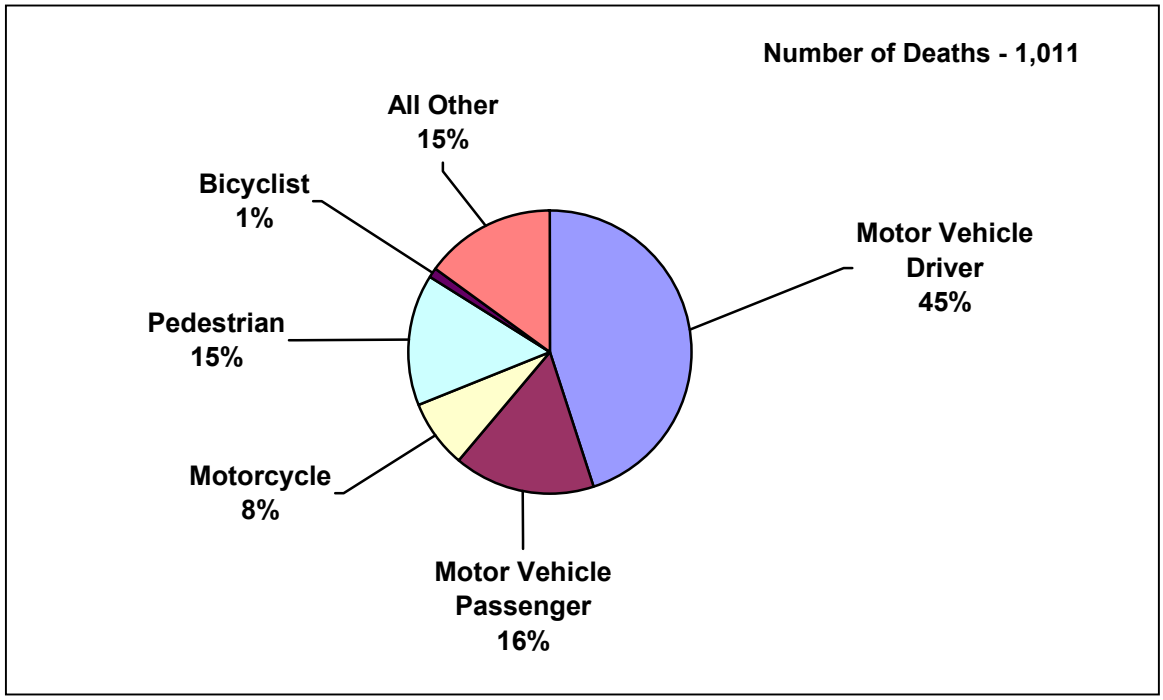
Connecticut's motor vehicle death rate has been consistently lower than the national rate (Figure 15.2). Connecticut ranks among the five lowest states for motor vehicle fatalities (National Highway Traffic Safety Administration 1998). During the ten-year period, the motor vehicle death rate statewide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

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

1996-1998 Motor Vehicle Crash Deaths, Connecticut Residents

- The leading cause of injury death for all CT residents
- A leading cause of premature mortality for all CT residents
- 32% of all unintentional injury deaths
- 68% were male
- High risk age groups: 15 to 34 years · 65 years and over · 4 to 8 years
- Significant decrease in mortality compared with the 1989-91 period

Figure 15.1.
Motor Vehicle Crash Deaths, Percent by Subtype
Connecticut Residents, 1996-1998



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

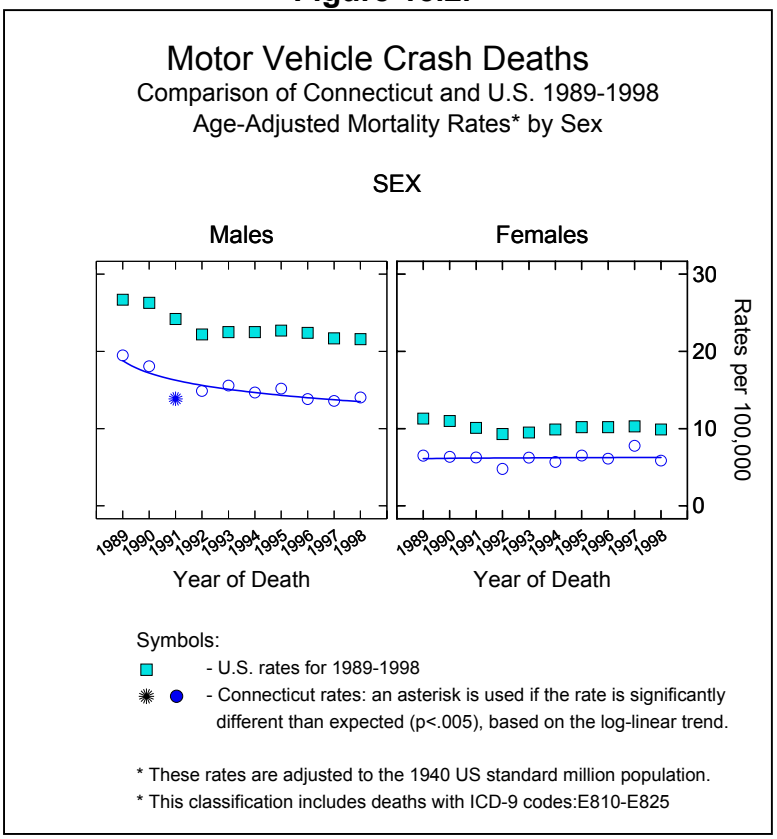
Premature mortality declined significantly among white and black males but not other racial/ethnic and gender subgroups. There were insufficient numbers of motor vehicle crash deaths among Asian and Pacific Islander and Native Americans in the 1996-1998 period to calculate reliable rates (Table 15.2).

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

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

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

Figure 15.2.



Risk Factors

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

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

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

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

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

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

Costs and Prevention

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

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

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

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

Notes:

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

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

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

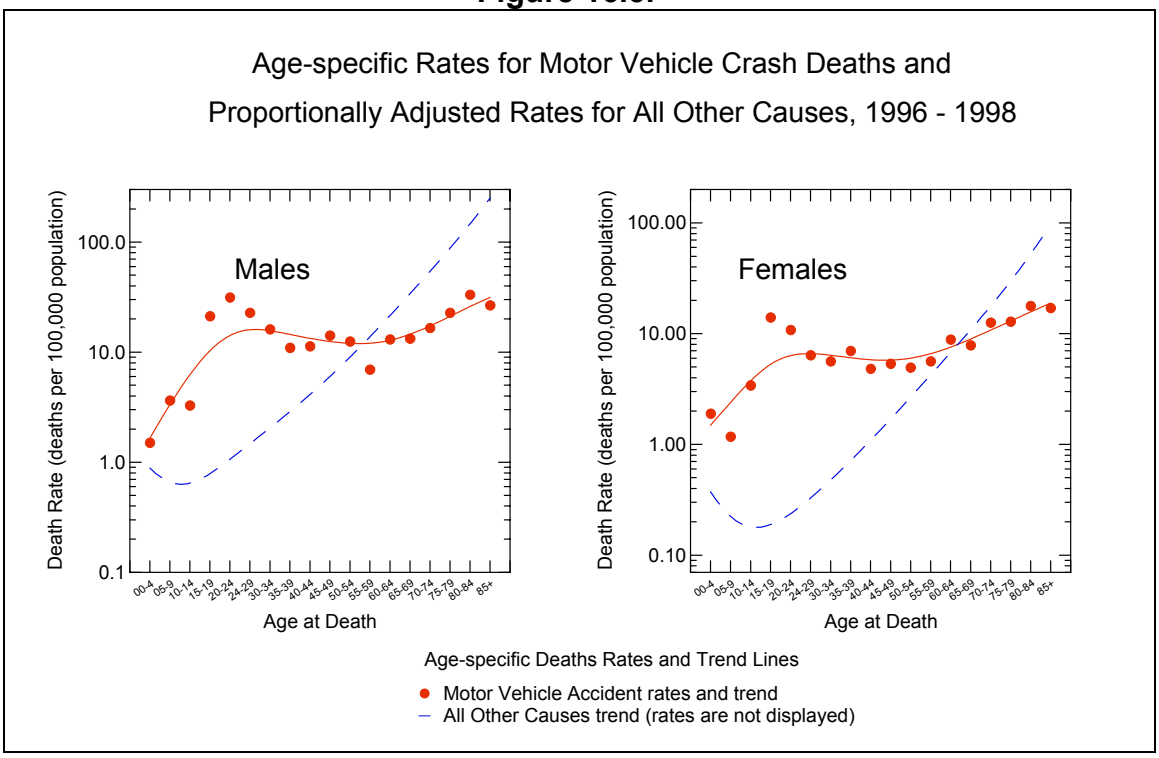
4. Statistical tests were conducted to evaluate differences in rates between race/ethnic groups. The white population serves as the reference group in each comparison (black vs. white, Asian & PI vs. white, Native American vs. white, Hispanic vs. white). Following are explanations of the notations:

   — Rate was not calculated due to small numbers.

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

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

Figure 16.3.



children ages 1 to 4 (Kahane 1986; National Highway Traffic Safety Administration 1996). They can also reduce hospitalization for children ages 4 and under by almost 70% (National Highway Traffic Safety Administration 1998b).

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

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

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

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

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

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

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

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

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

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

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

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

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

- Second leading cause of unintentional injury death
- 85% occurred among CT residents aged 65 years and older
- 97% occurred among white CT residents
- Ratio of male to female mortality - 2:1

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

Group	Number of Deaths	Age-Adjusted Mortality Rates[3] 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	619	5.6	ns	36.4	ns
All males	326	8.0	ns	58.9	ns
White	314	8.2	ns	62.5	ns
Black	12	—		—	
Asian PI					
Native American					
Hispanic	8	—		—	
All females	293	4.0	ns	14.5	ns
White	287	4.1	ns	14.0	ns
Black	3	—		—	
Asian PI	1	—		—	
Native American	2	—		—	
Hispanic	3	—		—	

Notes:

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

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

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

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

   — Rate was not calculated due to small numbers.

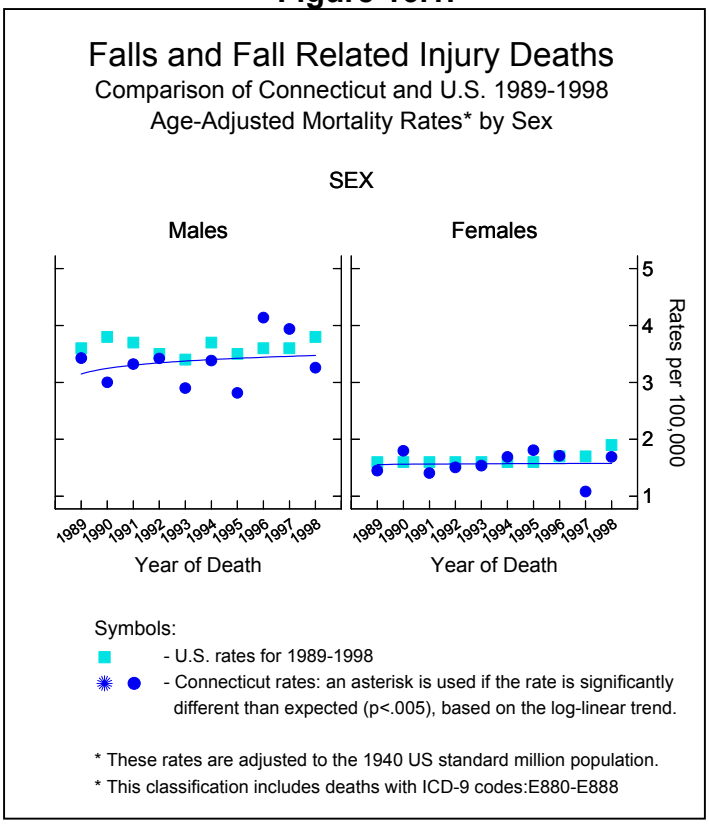
5. Statistical tests were conducted to evaluate changes in rates over time. Comparisons of 1996-98 vs. 1989-91 rates are made within each race/ethnicity group. Following are explanations of the notations:

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

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

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

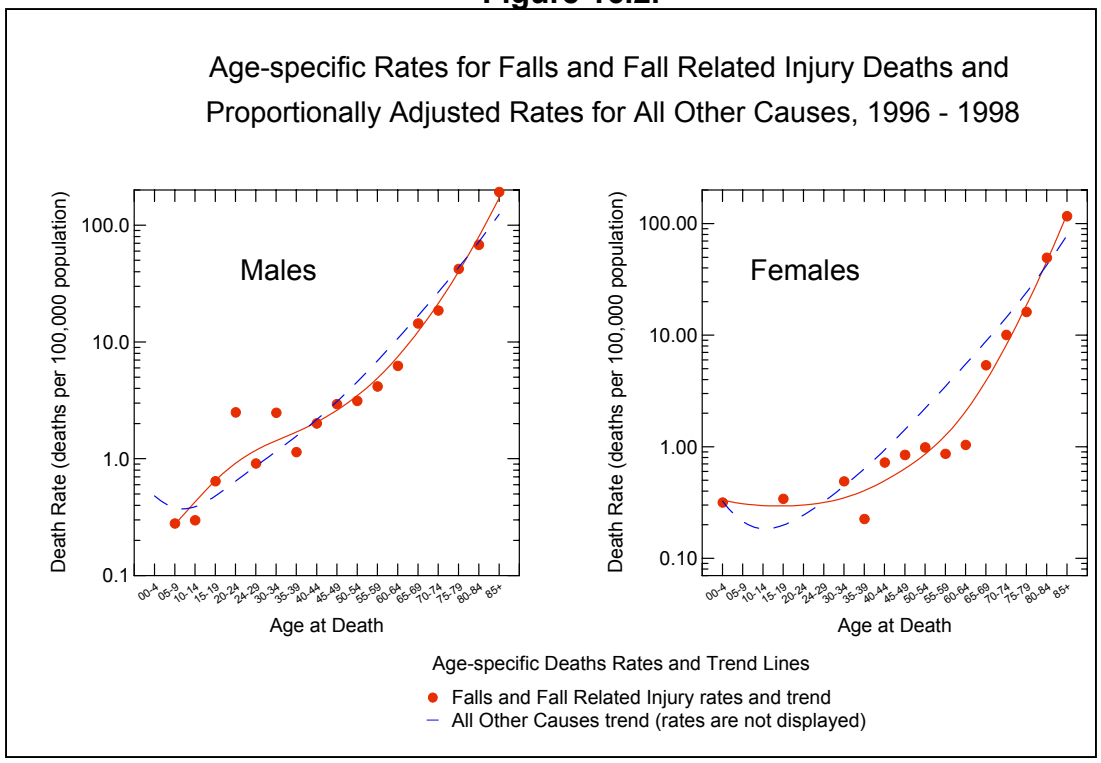
Figure 16.1.



Risk Factors

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

Figure 16.2.



Costs and Prevention

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

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

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

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

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

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

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

Table 16.4. Risk Factors for Falls

- Increasing age
- Muscle weakness
- Lack of physical activity
- Difficulties in gait and balance
- Visual impairment
- Osteoporosis
- Low body mass index
- Foot problems
- Inappropriate footwear
- Use of psychoactive medications
- Some combinations of medications
- Alcohol use
- A history of falls
- Home hazards (i.e. rugs, floor clutter)

Source: Sattin 1992; Stevens and Olson 2000.

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

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

Suicide
(ICD-9 codes E950-E959)

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

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

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

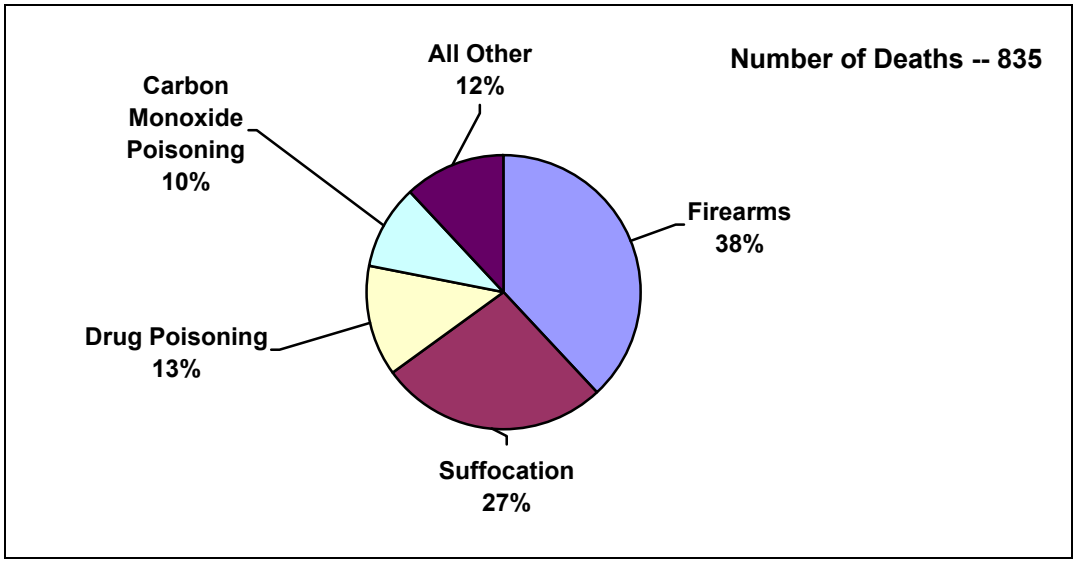
Figure 17.2 depicts age-specific suicide death rates for males and females (1996-1998) in relation to all other causes of death. Suicide death rates in both males and females were higher compared with proportionally adjusted rates for all other causes up to about age 59, at which point suicide death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

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

1996-1998 Suicide Deaths, Connecticut Residents

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

Figure 17.1. Suicide Deaths by Method Used



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

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

Risk Factors

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

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

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

Notes:

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

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

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

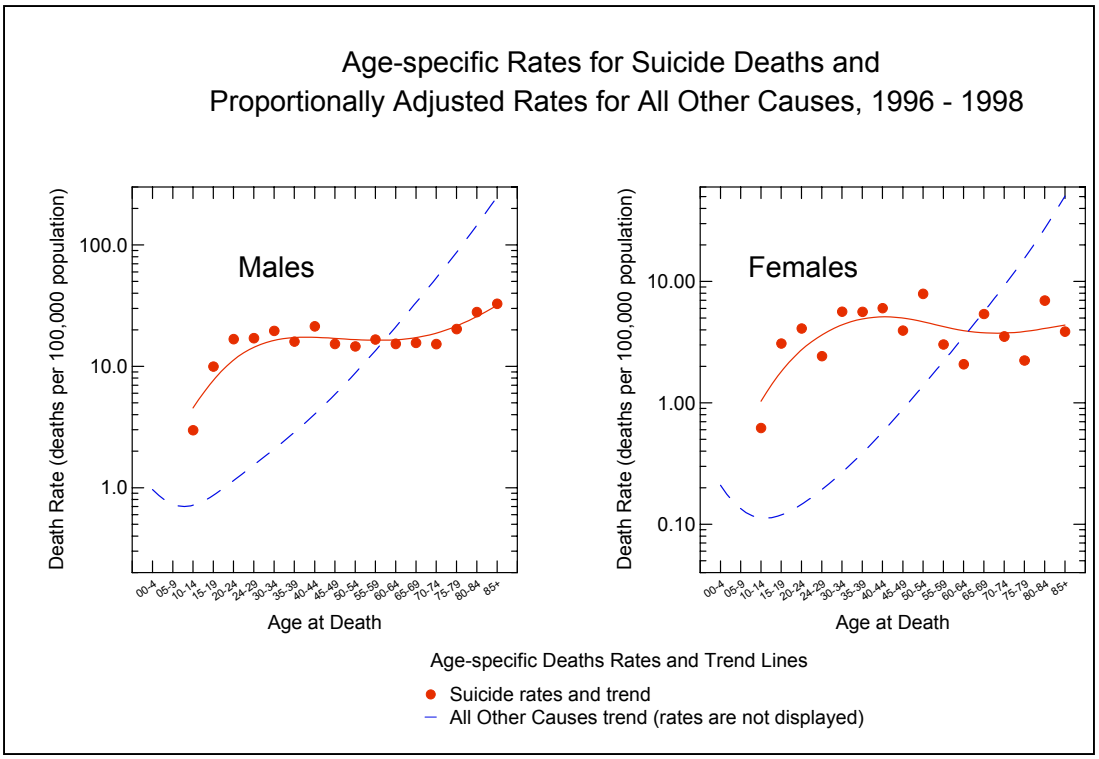
4. Statistical tests were conducted to evaluate differences in rates between race/ethnic groups. The white population serves as the reference group in each comparison (black vs. white, Asian & PI vs. white, Native American vs. white, Hispanic vs. white). Following are explanations of the notations:

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

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

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

Figure 17.2.



Prevention

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

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

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

Figure 17.3.

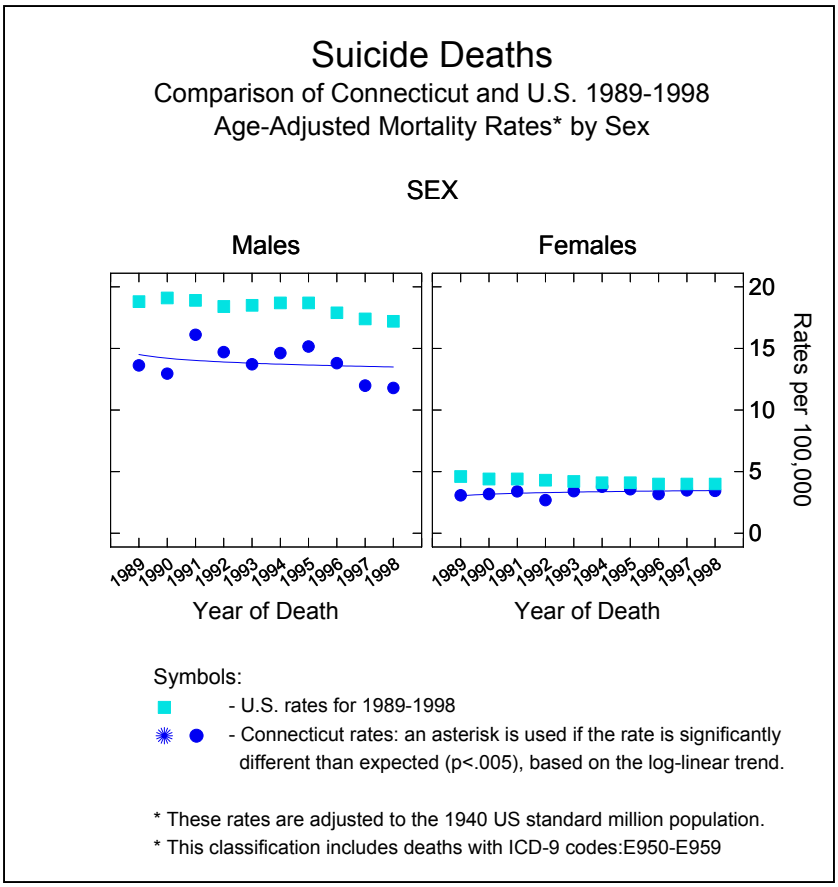


Table 17.2. Suicide Age-Adjusted Death Rates, Comparison of CT with US - 1992 and 1998

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

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

Table 17.3. Risk Factors for Suicide

- Previous suicide attempt
- Mental disorders
- Co-occurring mental and alcohol and substance abuse disorders
- Family history of suicide
- Feelings of hopelessness
- Impulsive and /or aggressive tendencies
- Barriers to accessing mental health treatment
- Relational, social, work, or financial loss
- Physical illness
- Easy access to lethal methods, i.e. guns
- Unwillingness to seek help because of stigma attached to mental and substance abuse disorders and/or suicidal thoughts
- Influence of significant people who have died by suicide
- Cultural and religious beliefs
- Isolation from other people
- Local epidemics of suicide

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

Table 17.4. Aims of the National Strategy

- Prevent premature deaths due to suicide across the life span
- Reduce the rates of other suicidal behaviors
- Reduce the harmful after-effects associated with suicidal behaviors and the traumatic impact of suicide on family and friends
- Promote opportunities and settings to enhance resiliency, resourcefulness, respect, and interconnectedness for individuals, families, and communities

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

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

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

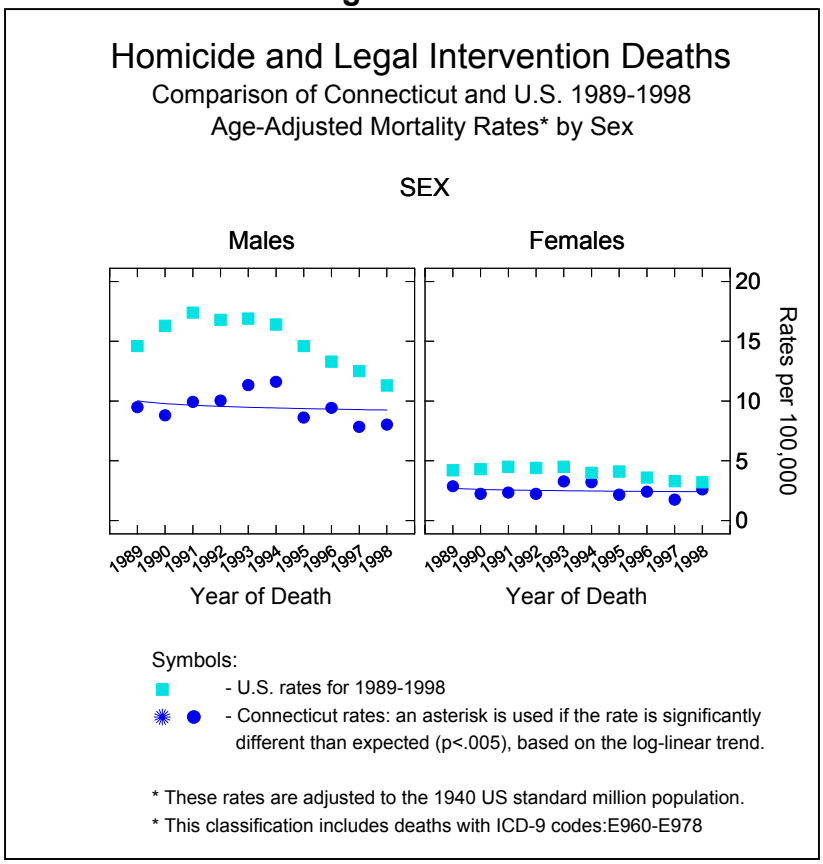
Certain population groups are at higher risk for homicide and legal intervention death. Males were more than three times as likely as females to die from homicide or legal intervention during the 1996-1998 period. Males accounted for 77% of all deaths and 78% of all premature mortality to age 75 (Table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

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

1996-1998 Homicide & Legal Intervention Deaths, Connecticut Residents

- Fifteenth leading cause of death for all CT residents
- Second leading cause of death for age groups 15 to 24
- Sixth leading cause of premature death (at age 75) for all CT residents
- 77% were male
- 65% were firearm homicides

Figure 18.1.



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

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

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

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

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

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

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

Risk Factors

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

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

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

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

Notes:

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

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

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

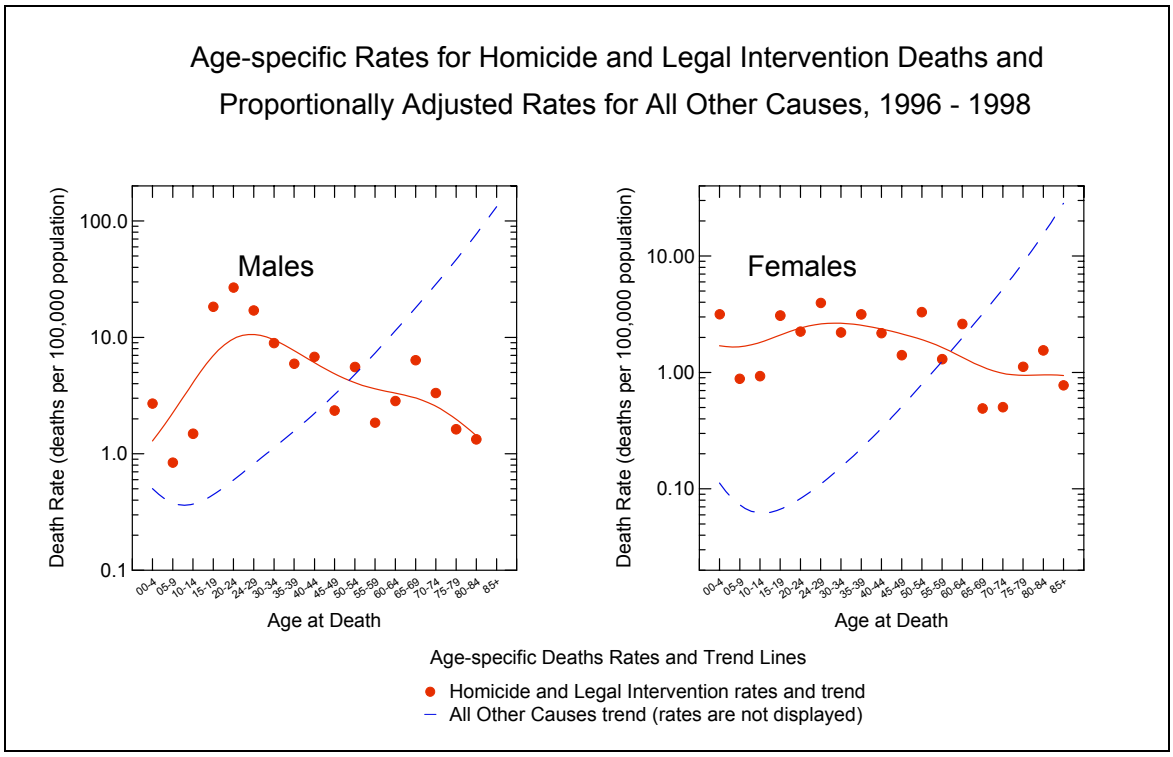
4. Statistical tests were conducted to evaluate differences in rates between race/ethnic groups. The white population serves as the reference group in each comparison (black vs. white, Asian & PI vs. white, Native American vs. white, Hispanic vs. white). Following are explanations of the notations:

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

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

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

Figure 18.2.



Prevention

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

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

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

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

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

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

During the period 1989-1998, 2,371 Connecticut residents died as a result of poisoning. Poisoning was the fourth-ranked leading cause of injury mortality for Connecticut residents, behind motor vehicle accidents, suicide, and firearms. The most common types of poisoning deaths for Connecticut residents (1996-1998) were accidental deaths from opiates (heroin) and other narcotics, cocaine, and other drugs (63% of all poisoning deaths); and suicides by drugs and carbon monoxide and other gases (22%) [Figure 19.1].

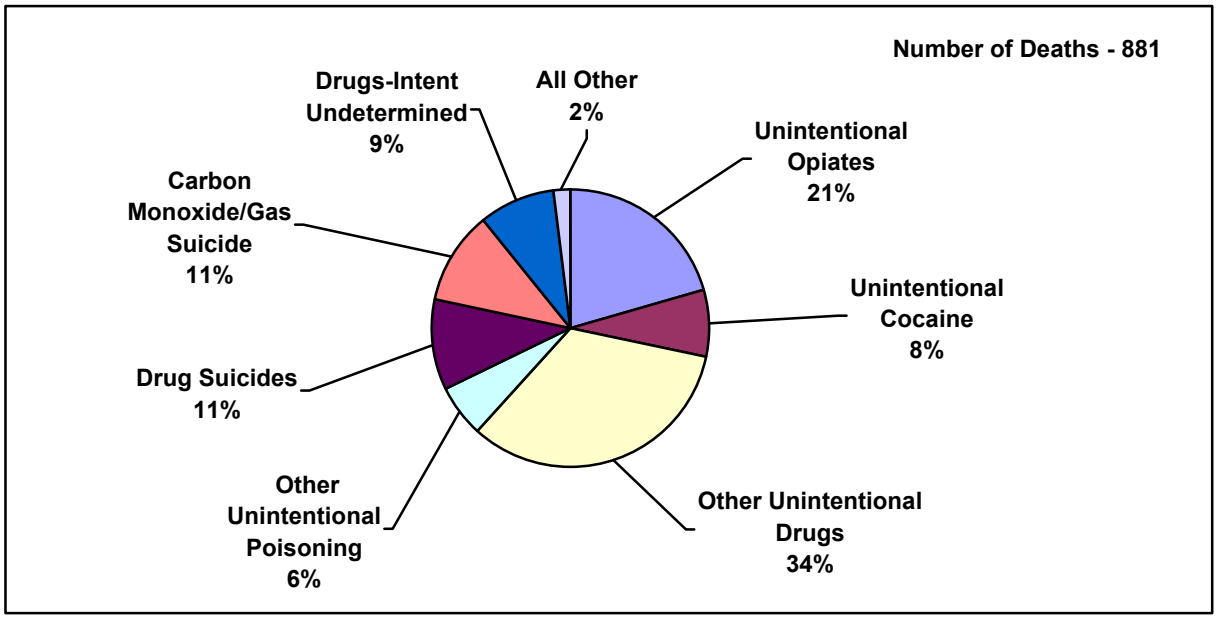
Poisoning mortality rates appeared to increase during the ten-year period for both men and women; however, some of the apparent increase is due to improved classification of drug-related causes of death beginning in 1992. For this reason, the trend analysis presented here covers the period from 1992 to 1998.

For the period 1996-1998, Connecticut males were almost three times more likely than females to die as a result of poisoning (Table 19.1). Sixty-four percent of all poisoning deaths were among males ages 20 to 54. Death rates peaked in the 35-44 age group (Appendix VII A). Poisoning was the leading cause of injury mortality in this age group surpassing motor vehicle crashes. Figure 19.2 depicts male and female age-specific death rates for poisoning and all other remaining causes of

1996-1998 Poisoning Deaths, Connecticut Residents

- Leading cause of injury mortality in Connecticut
- 63% were accidental drug deaths
- Ratio of male to female mortality — 3: 1
- 64% were males aged 20 to 54
- Connecticut mortality was significantly higher than U.S. mortality

Figure 19.1. Poisoning Deaths, Percent by Type
Connecticut Residents, 1996-1998



death. Poisoning death rates exceed proportionally adjusted rates due to all other causes of death for males ages 5 to 54 and for females up to age 59, after which point they decrease relative to all remaining causes of death.

For the period 1996-1998, the highest age-adjusted rates of poisoning deaths occurred among Hispanic and black males, with rates about 1.5 times higher than comparable rates for white males ($p < .05$). The black-white and Hispanic-white disparity was consistent across five-year age groups. There were no significant differences in mortality rates of white and black females for this same period. There were too few poisoning deaths among Hispanic females and Asian and Pacific Islander and Native American males and females to calculate reliable rates for this period (Table 19.1).

Between the periods 1992-1994 and 1996-1998, poisoning death rates did not increase significantly for all Connecticut residents or racial/ethnic subpopulations. Poisoning mortality did increase significantly for female residents, however, and this change is accounted for by a significant increase in poisoning mortality within the white female population (Table 19.1). Although there were no apparent statistically significant increases in any of the major subcategories of poisoning deaths for Connecticut females, some of the increase in the poisoning death rate may be attributable to increases in opiate and related narcotics deaths. This category includes deaths due to heroin, methadone, morphine, codeine, opium, and pethidine. Opiate and related narcotic deaths accounted for about 9% of all poisoning deaths among Connecticut female residents in 1992, compared with

Table 19.1. All Poisoning Deaths[1], Connecticut Residents by Gender, Race and Ethnicity[2], 1996-1998

Group	Number of Deaths	Age-Adjusted Mortality Rates[3] AAMR[4]	Change since 1992-94[5]	Age-Adjusted Premature Mortality Rates to Age 75[3] YPLL[4]	Change since 1992-94[5]
All Residents	881	8.7	ns	312.5	ns
All males	639	12.9	ns	465.9	ns
White	566	12.8	ns	471.8	ns
Black	71	18.6*	ns	619.8	ns
Asian PI	1	—		—	
Native American	1	—		—	
Hispanic	73	19.5*	ns	749.2*	ns
All females	242	4.6	↑	161.4	↑
White	216	4.7	↑	162.6	↑
Black	24	5.1	ns	185.2	ns
Asian PI	1	—		—	
Native American	1	—		—	
Hispanic	15	—		—	

Notes:

1. This cause of death category includes ICD-9 codes E850-E869, E950-E952,E962,E972,E980-E982.

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

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

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

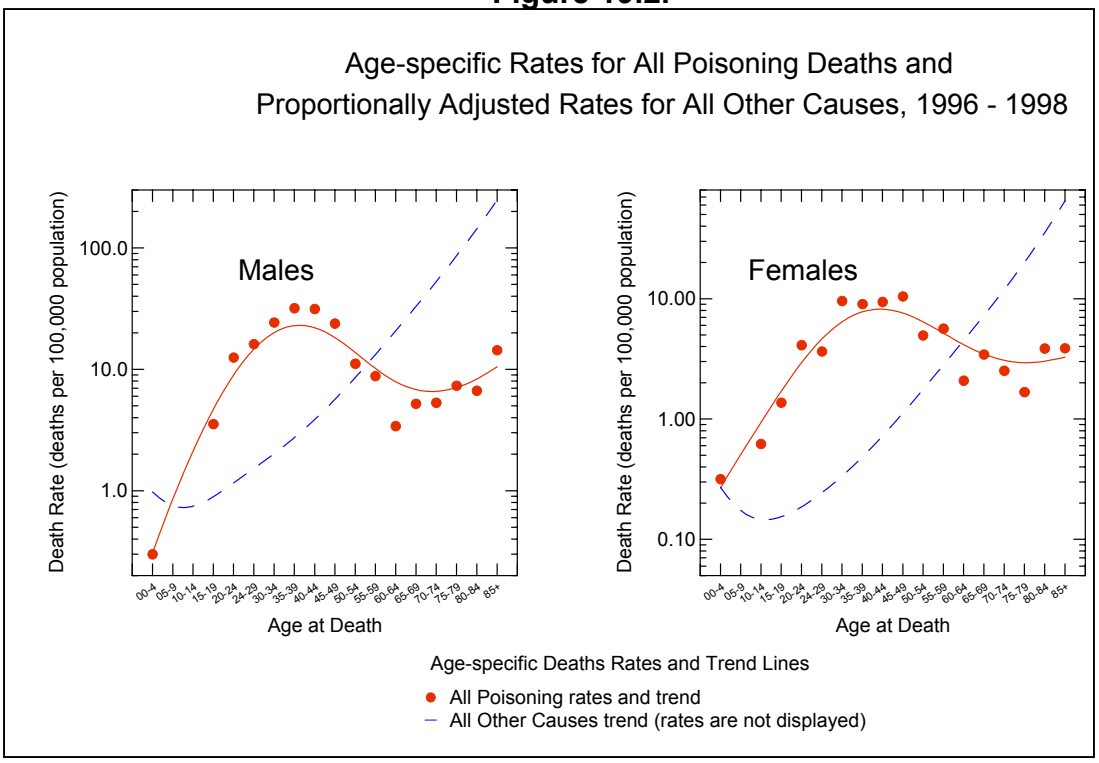
   * Significantly different than the respective white resident rate at $p < .05$.
   — Rate was not calculated due to small numbers.

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

   ↑ 1996-98 rate is significantly higher than the 1992-94 rate at $p < .05$.

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

Figure 19.2.



about 13% in 1998. The shift in type of poisoning death follows a similar (but not statistically significant) pattern to that of males. Opiate and related narcotic deaths accounted for about 16% of all poisoning deaths among Connecticut male residents in 1992, compared with 27% in 1998.

Since 1992, Connecticut poisoning mortality rates have consistently exceeded U.S. rates (Table 19.2). Some of the disparity in these rates may be attributable to differences in opiate and related narcotic death rates. In 1997, for example, opiate and related narcotics accounted for 13% of all poisoning deaths nationwide (Centers for Disease Control and Prevention, 2000) compared with 23% in Connecticut. The recent increase in opiate production and use and its relationship to mortality is discussed in the drug-induced mortality section. Male and female mortality rates for U.S. and Connecticut residents (1989-1998) are depicted in Figure 19.3.

Risk Factors

Risk factors for poisoning mortality overlap with those for alcohol-induced and drug-induced mortality. They are discussed in the alcohol-induced and drug-induced mortality sections in the following pages.

Prevention

Prevention efforts aimed at reducing risk for poisoning mortality should first, be tailored to the mechanism (drugs, alcohol, or carbon monoxide) and intent (unintentional or suicide). Secondly, efforts should address high-risk groups in the population (subgroups of males between the ages of 20 and 54 and females aged 30 to 49) and any groups that have shown a significant increase over time (white females in Connecticut). Prevention efforts for poisoning overlap with those for alcohol and drug abuse and so are discussed in the alcohol-induced and drug-induced mortality sections of this report.

Figure 19.3.

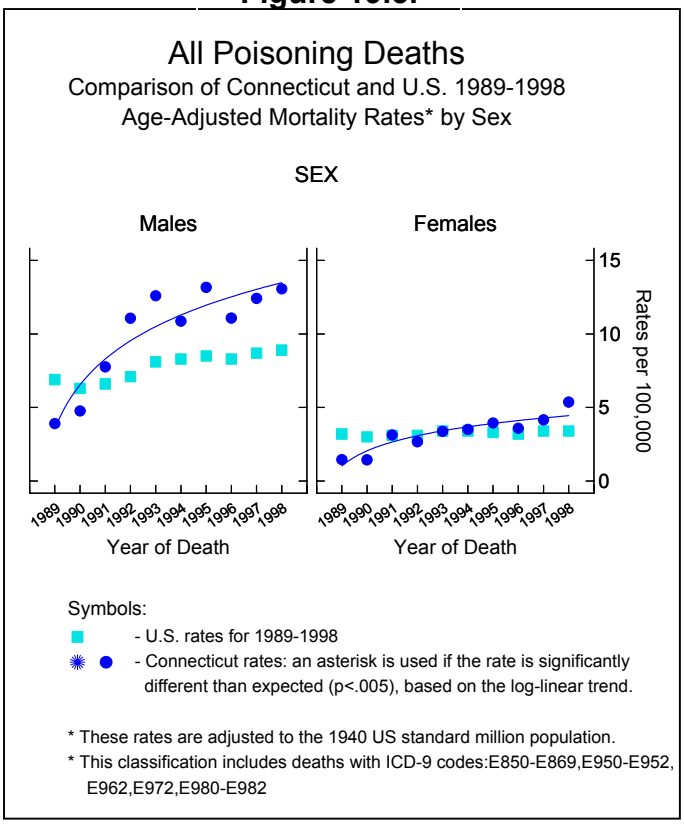


Table 19.2. Poisoning Age-Adjusted Death Rates, Comparison of CT with US - 1992 and 1998

	1992	1998	1998 CT AAMR Comparison
CT AAMR*	6.8	9.1	
US AAMR*	5.3	6.3	CT AAMR > US AAMR

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

Alcohol-Induced
(ICD-9 codes 291, 303, 305.0, 357.5, 425.5, 535.3, 571.0-.3, 790.3, E860)

During the 1989-1998 period, 1,762 Connecticut residents died from alcohol-induced causes. Alcohol-induced mortality includes deaths due to alcohol dependence syndrome; nondependent abuse of alcohol; chronic liver disease and cirrhosis due to alcohol; alcoholic psychoses; and alcoholic polyneuropathy, cardiomyopathy and gastritis. It excludes accidents, homicides, other causes indirectly related to alcohol use, and deaths due to fetal alcohol syndrome. As such, it understates the extent to which alcohol contributes to mortality (Murphy 2000). Major subcategories of alcohol-induced deaths among Connecticut residents for the 1996-1998 period include chronic liver disease and cirrhosis, alcohol dependence syndrome, accidental poisoning, and nondependent abuse of alcohol (Figure 20.1).

Alcohol-induced mortality differs dramatically by gender and age group. During the period 1996-1998 males were about three times more likely to die of alcohol-induced causes than were females (Table 20.1). Males in the 45-69 age group accounted for about 44% of all alcohol-induced Connecticut resident deaths (Appendix VII A).

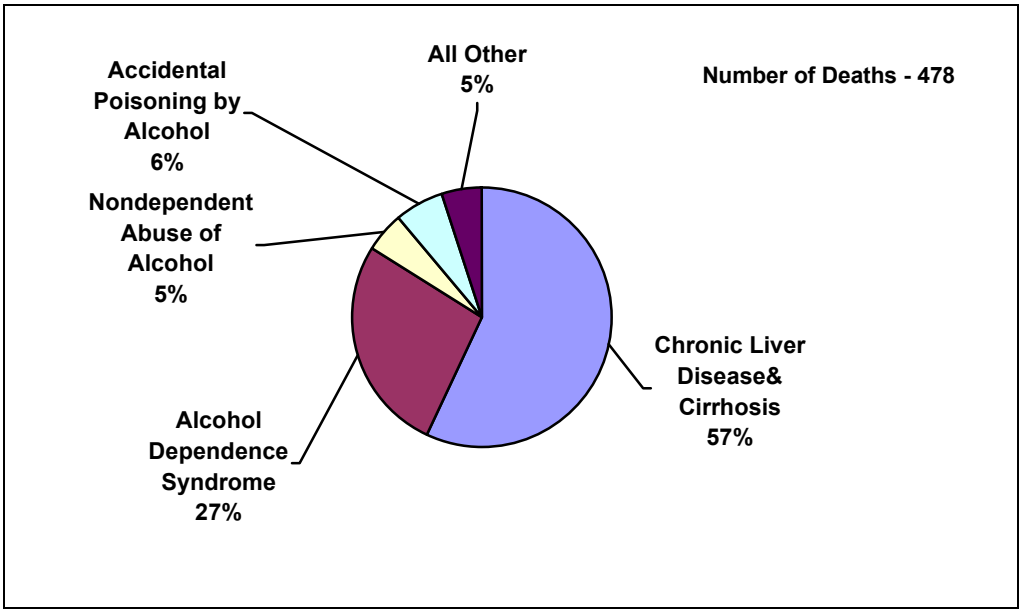
Figure 20.2 depicts age-specific alcohol-induced death rates for males and females compared with proportionally adjusted rates for all other causes. Alcohol-induced death rates for males and females aged 30 to 64 were higher than the respective death rates for all remaining causes.

Hispanic male residents of Connecticut had the highest mortality and premature mortality rates of all subpopulation groups (Table 20.1). They were almost twice as likely as white males to die of alcohol-induced causes and suffered significantly higher premature mortality to age 75 compared with white males. The Hispanic-white male mortality disparity was consistent across five-year age groups. Age-adjusted and premature mortality rates for black males and females were not significantly different from those for white males and females, respectively. There were too few alcohol-induced deaths among Hispanic females and Asian and Pacific Islander and Native American males and females to calculate reliable rates (Table 20.1).

1996-1998 Alcohol-Induced Deaths, Connecticut Residents

- 57% were from chronic liver disease & cirrhosis
- Ratio of male to female mortality — 3 : 1
- 44% were males aged 45 to 69 years
- Hispanic males had highest death and premature mortality rates
- Connecticut mortality was significantly lower than U.S. mortality

Figure 20.1. Alcohol-Induced Deaths, Percent by Type Connecticut Residents, 1996-1998



Age-adjusted and premature mortality rates for all Connecticut residents and gender and racial/ethnic subpopulations did not change significantly from the period 1989-1991 to 1996-1998 (Table 20.1). Since 1989, alcohol-induced mortality among Connecticut residents has been significantly lower than comparable mortality nationwide. Connecticut male residents had consistently lower alcohol-induced age-adjusted death rates than males nationwide. Connecticut female residents had significantly lower rates than females nationwide in five of the ten years displayed. There are no *Healthy People 2000* or *Healthy Connecticut* targets for alcohol-induced mortality (Figure 20.3, Table 20.2).

Risk Factors

Nondependent heavy drinkers account for most alcohol-related morbidity and mortality (U.S. Preventive Services Task Force 1996). Risk factors for drinking during adolescence are considered important because experimentation with drinking increases dramatically during this period (Simons-Morton, Haynie, Crump et al. 2001). Alcohol is the most commonly used drug among young Americans. Recent survey estimates indicate that 50% of high school students reported drinking and 32% reported being drunk in the past month. Binge drinking among college students is widespread and considered to be the most serious public health problem on campuses (Horgan, Skwara, Strickler, et al. 2001). A 2002 national study found that college student drinking (ages 18 to 24) contributes to

Table 20.1. Alcohol-Induced[1] Deaths, Connecticut Residents by Gender, Race and Ethnicity[2], 1996-1998

Group	Number of Deaths	Age-Adjusted Mortality Rates[3] 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	478	4.7	ns	101.4	ns
All males	347	7.4	ns	155.8	ns
White	311	7.2	ns	153.2	ns
Black	36	11.7	ns	244.4	ns
Asian PI					
Native American					
Hispanic	34	14.1*	ns	345.9**	ns
All females	131	2.4	ns	49.4	ns
White	117	2.4	ns	47.7	ns
Black	13	—		—	
Asian PI					
Native American					
Hispanic	10	—		—	

Notes:

1. This cause of death category includes ICD-9 codes 291,303,305.0,357.5,425.5,535.3, 571.0-.3,790.3,E860.

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

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

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

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

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

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

Figure 20.2.

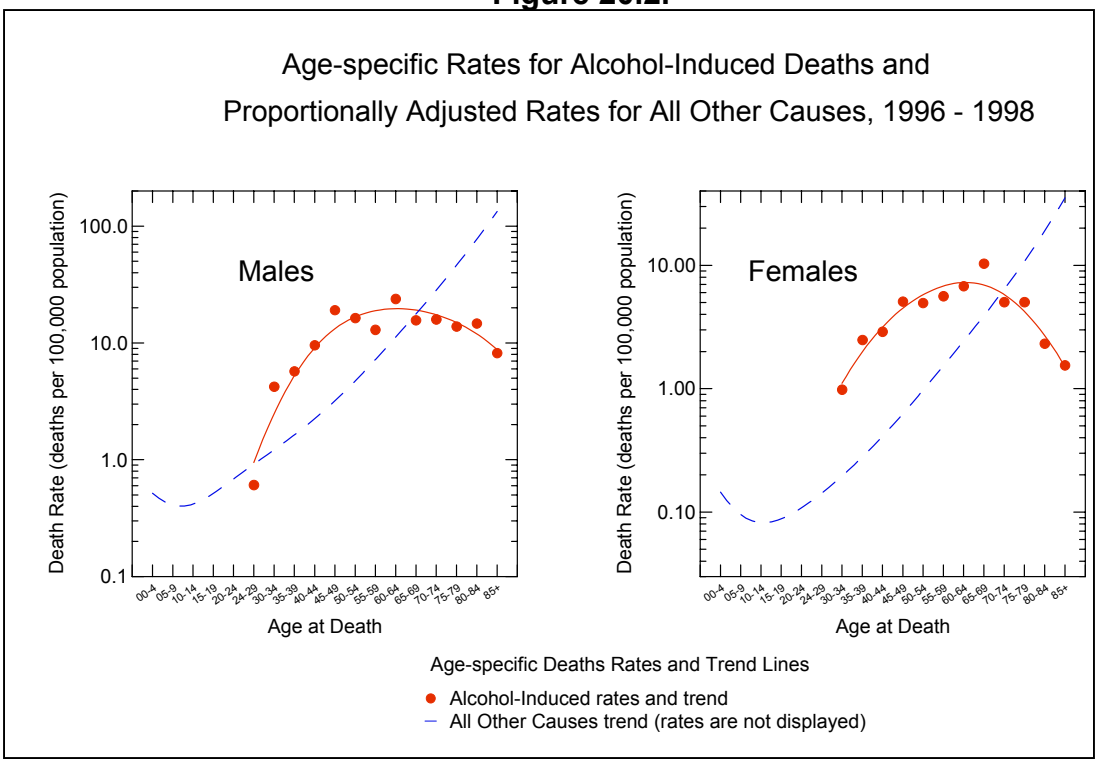


Table 20.2. Alcohol-Induced Age-Adjusted Death Rates, Comparison of CT with US - 1989 and 1998

	1989	1998	1998 CT AAMR Comparison
CT AAMR*	5.4	4.1	
US AAMR*	7.4	6.1	CT AAMR < US AAMR

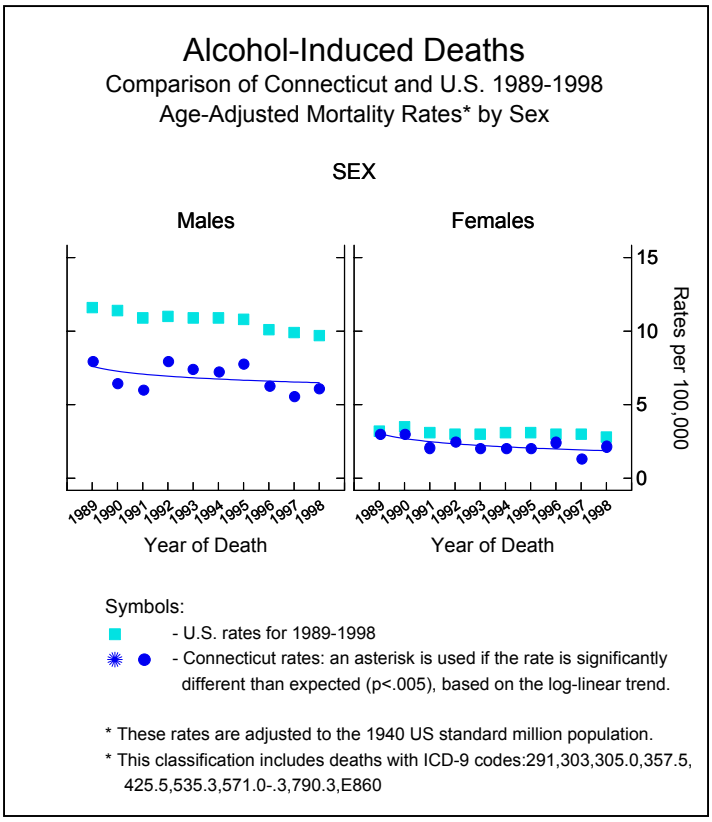
* age-adjusted mortality rates for alcohol-induced causes are per 100,000 population, U.S. 1940 standard million population.

an estimated 1,400 student deaths per year (National Institute on Alcohol Abuse and Alcoholism 2002). National and Connecticut Behavioral Risk Factor Surveillance System survey data show that males comprise a greater proportion of chronic and binge drinkers than do females (Centers for Disease Control and Prevention 2001).

Risk factors for alcohol and drug problems fall into two broad categories: societal and cultural factors that provide the legal and normative expectations for behavior; and individuals and their interpersonal environments, primarily families, schools, and peer groups. Specific risk factors are outlned in Table 20.3.

Costs and Prevention

Figure 20.3.



The social and economic costs of alcohol abuse and alcoholism are staggering. Economic costs in the United States, estimated at $148 billion in 1992, include health care expenditures for problems attributed to alcohol, such as alcohol-related illnesses and trauma; impaired productivity accrued in the form of lost household productivity; and lost earnings due to work not performed. Also included are costs of motor vehicle crashes; crime, such as driving under the influence and interpersonal violence; social welfare costs of alcohol-related impairment; and premature death due to alcohol abuse or excessive long-term consumption (Harwood, Fountain, and Livermore 1998). The social toll of alcoholism, which extends to family members, friends, and co-workers, includes family disruption and violence; the neglect or mistreatment of children; and the loss of loved ones through premature death. Alcoholism also damages social relationships in the workplace and in social networks (Horgan, Skwara, Strickler, et al. 2001).

Alcohol prevention efforts tend to target excessive drinking among non-alcoholics, which is much more common than alcoholism. State and community approaches to reducing alcohol consumption include measures such as increased taxes on alcohol, controlling the physical availability and legal accessibility of alcohol, health warning labels, and health information messages and education (Horgan, Skwara, Strickler, et al. 2001). Connecticut Governor Rowland convened a Blue Ribbon Task Force on Substance Abuse in 1995 to identify effective prevention and treatment methods for alcohol and drug addiction in the state. The Connecticut Alcohol and Drug Policy Council (ADPC), an outgrowth of this task force, identified strategies for a comprehensive statewide plan for substance abuse prevention, treatment and enforcement in its 1997 report (Connecticut Alcohol and Drug Policy Council 2001).

The Council submitted a three-year plan in 1999 with a revised plan scheduled for 2002, both of which identify three focus areas--prevention, treatment, and criminal justice. The prevention focus area of the 2002 plan includes three goals: 1) to increase the capacity and improve the effectiveness of the prevention system using empirically grounded approaches and trained staff; 2) to increase successful programming by using age-appropriate strategies and gender-specific models; and 3) to expand the coordination of prevention programs in the state (in the areas of violence, mental illness

Table 20.3. Risk Factors for Alcohol Abuse

Societal / Cultural	*Individuals and Interpersonal Environments*
• Laws (liquor tax, how and to whom liquor is sold)	• Physiological factors (biochemical and genetic susceptibility)
• Cultural norms (exposure to advertising promoting drinking; ethnic and other community norms)	• Family behavior (peer and sibling alcoholism, perceived parent permissiveness toward alcohol use)
• Alcohol availabilility	• Early initiation of alcohol use
• Extreme economic deprivation (in conjunction with childhood behavior problems)	• Early and persistent behavior problems (aggressive behavior that continues into early adolescence; school misconduct)

Source: Hawkins, Catalano, and Miller 1992.

and substance abuse programs, child abuse and neglect, delinquency, school dropouts, teen pregnancy, HIV, and family and youth self-sufficiency). An important related initiative, the Governor's Prevention Initiative for Youth, targets the reduction of alcohol, tobacco, and other drug use among young people ages 12 to 17. A uniform evaluation system was initiated in 2000 to assess all current state-supported substance abuse prevention programs (Connecticut Alcohol and Drug Policy Council 2001).

The U.S. Preventive Services Task Force recommends that clinicians screen for problem drinking in all adult and adolescent patients by taking a detailed history of alcohol use (U.S. Preventive Services Task Force 1996). Studies have shown that primary care clinicians can assist in reducing alcohol consumption levels in problem drinkers who are not alcohol dependent (U.S. Department of Health and Human Services 2003).

Drug-Induced

(ICD-9 codes 292, 304, 305.2-305.9, E850-E858, E950.0-E950.5, E962.0, E980.0-E980.5)

During the period 1989 – 1998, 1,921 Connecticut residents died from drug-induced causes. The number of drug-induced deaths climbed steadily during the decade with more deaths occurring in 1998 than in any previous year. Improved reporting of cause of death beginning in 1991 is a partial explanation for the apparent increase in drug-induced mortality between 1989 and 1992. Beginning with the 1992 mortality data, the Connecticut Department of Public Health (DPH) was able to significantly reduce the number of deaths identified as "pending further investigation" by the Connecticut Medical Examiner's (ME) Office through an improved system of communication. Speedier processing of findings from the ME investigations has resulted in more complete and more accurate cause-of-death classification being entered into the death records. Many drug-induced deaths tend to be included in the category of "pending." For this reason, time trends discussed here begin with the period 1992.

Drug-induced and poisoning mortality include overlapping categories of deaths, specifically, those poisoning deaths that are related to drug use. Drug-induced deaths include those due to drug abuse (excluding tobacco or alcohol), drug dependence, and drug psychoses, as well as unintentional, suicide, and homicide poisonings by use of either legally prescribed or illicit drugs. This definition, which is consistent with that used by the Centers for Disease Control and Prevention (CDC) and the National Center for Health Statistics (NCHS), has some limitations. Reported deaths are based on the underlying (primary) cause of death only. These figures greatly underestimate the mortality related to illicit drug use because they exclude deaths from causes such as AIDS among injecting drug users, hepatitis and tuberculosis, as well as deaths from homicides, and newborn deaths associated with mother's drug use (Murphy 2000).

In the 1996-1998 period, the main subcategories of drug-induced deaths among Connecticut residents included opiates and related narcotics (that is, heroin, methadone, morphine, codeine,

1996-1998 Drug-Induced Deaths, Connecticut Residents

- Main subcategories: opiates & related narcotics and cocaine
- Ratio of male to female mortality — 2.6: 1
- Males aged 20 to 49 years accounted for 64% of these deaths
- Connecticut mortality was significantly higher than U.S. mortality
- Significant increase in mortality compared with the 1992-94 period

opium, and pethidine); cocaine; and all other drug overdoses that were unintentional, suicides, or of undetermined intent (Figure 21.1). Males were almost three times more likely than females to die from drug-induced causes (Table 21.1). Deaths were highest in the 20 to 49 year old age groups. Males in these age groups accounted for about 64% of all drug-induced deaths but only 6% of deaths due to other causes (Figure 21.2 and Appendix VII A).

Heroin and related narcotics deaths in Connecticut appeared to increase relative to other drug-induced deaths during the 1992-1998 period, accounting for 21% of all such deaths in 1992 and 26% in 1998. Further analyses of the male and female heroin death rate from the 1992-1994 to 1996-1998 periods, however, do not reveal statistically significant changes. Cocaine deaths appeared to decline relative to other drug induced deaths from 1992 to 1998. Cocaine accounted for about 15% of all drug-induced deaths in 1992 compared with 9% in 1998, while the percentage of deaths from all other drug-induced causes remained about the same in 1992 and 1998. Analyses of the cocaine death rate from the 1992-1994 to 1996-1998 periods, however, did not show statistically significant changes in the Connecticut male or female populations

Changes in heroin use may be linked to increases in the production of heroin worldwide, beginning in 1989 (Hamid, Curtis, McCoy, et al. 1997). Since 1992, there has been a documented increase in heroin use both nationally (Horgan, Skwara, Strickler, et al. 2001) and regionally (Hamid, Curtis, McCoy, et al. 1997). The increase in heroin initiation may be partly explained by the increased profitability in heroin sales, increased purity of the drug, and new modes of use like smoking, snorting, and sniffing rather than intravenous needle injection (Horgan, Skwara, Strickler, et al. 2001; Connecticut Alcohol and Drug Policy Council 2001).

Figure 21.1. Drug-Induced Deaths, Percent by Type
Connecticut Residents, 1996-1998

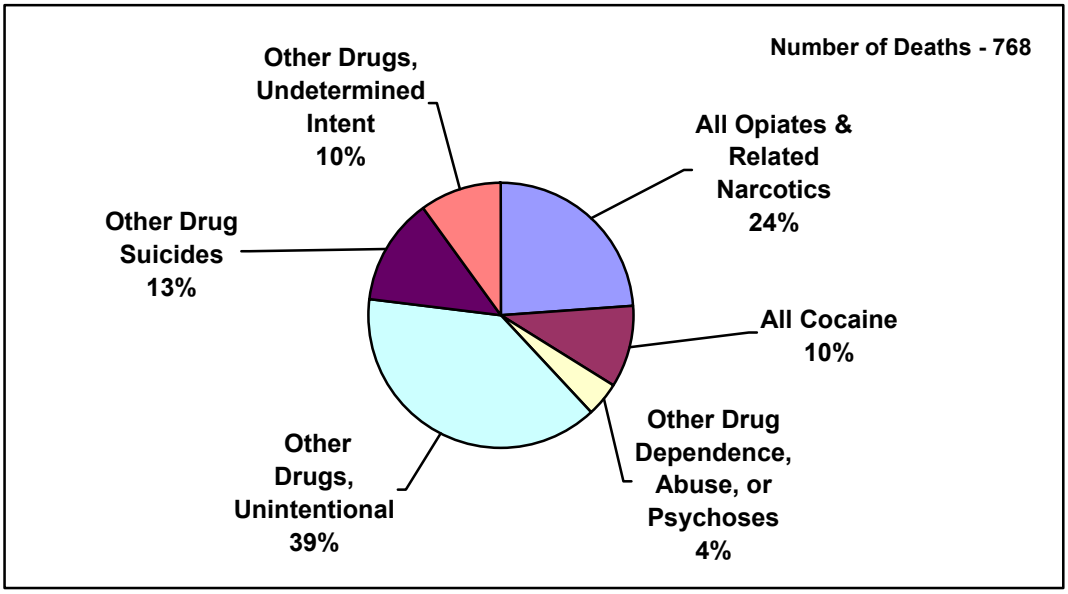


Table 21.1. Drug-Induced Deaths[1], Connecticut Residents by Gender, Race and Ethnicity[2], 1996-1998

Group	Number of Deaths	Age-Adjusted Mortality Rates[3] AAMR[4]	Change since 1992-94[5]	Age-Adjusted Premature Mortality Rates to Age 75[3] YPLL[4]	Change since 1992-94[5]
All Residents	768	7.5	↑	283.5	↑
All males	547	10.9	ns	418.9	ns
White	479	10.8	↑	421.2	↑
Black	67	17.3*	ns	592.9	ns
Asian PI	1	—		—	
Native American					
Hispanic	72	19.4**	ns	738.5**	ns
All females	221	4.2	↑	149.7	↑
White	198	4.3	↑↑	152.3	↑↑
Black	21	4.5	ns	161.8	ns
Asian PI	1	—		—	
Native American	1	—		—	
Hispanic	15	—		—	

Notes:

1. This cause of death category includes ICD-9 codes 292,304,305.2-.9,E850-E858,E950.0-.5, E962.0,E980.0-.5. (*Healthy People 2000* cause of death classification refers to codes 292,304,305.2-.9, E850-E858,E950.0-.5,E962.0,E980.0-.5 as "Drug-Related Deaths).

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

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

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

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

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

   ↑ 1996-98 rate is significantly higher than the 1992-94 rate at $p < .05$.
   ↑↑ 1996-98 rate is significantly higher than the 1992-94 rate at $p < .01$.
   ns Indicates the change from 1992-94 to 1996-98 is not statistically significant.

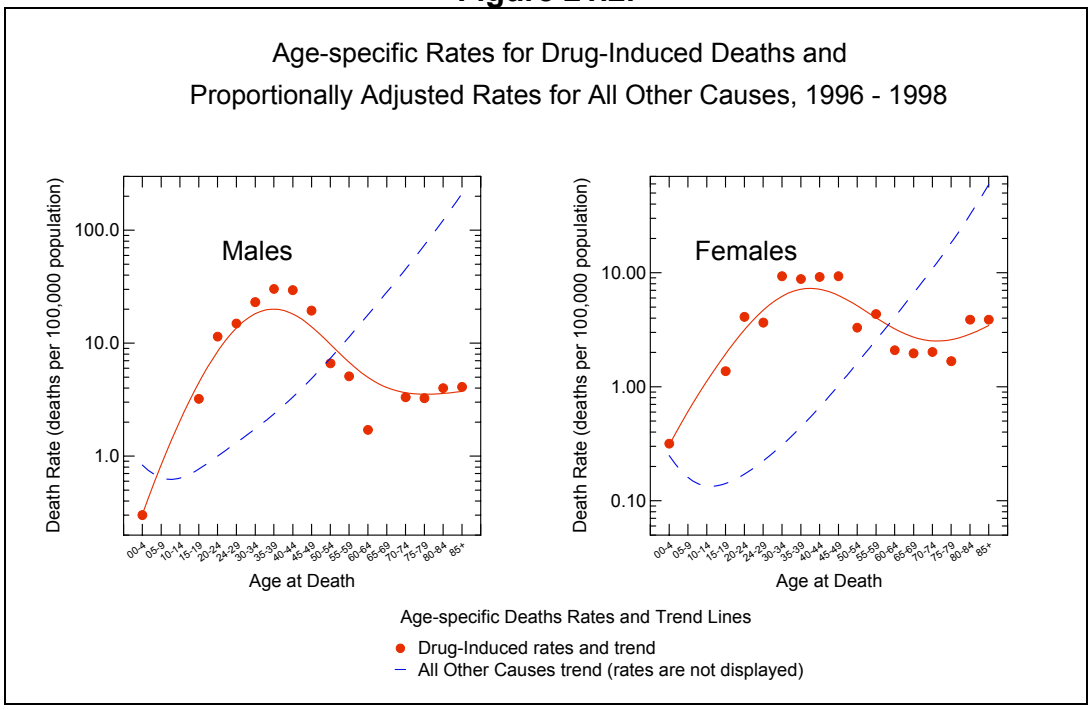
Age-specific drug-induced death rates for males and females compared with proportionally adjusted rates for all other causes of death (1996-1998) are depicted in Figure 21.2. Drug-induced death rates for males aged 15-49 and females aged 15-59 exceeded proportionally adjusted rates for all other causes of death, after which point they decrease relative to all other causes of death.

Assessment of the annual percent change in drug-induced mortality for the years 1992-1998 indicates a significant increase of 10.7% for females ($p < .001$) and 3.9% for males ($p < .01$). An examination of racial and ethnic subgroup detail provides some additional insight into changes over time.

In the period 1996-1998, black and Hispanic males were 1.6 and 1.8 times, respectively, more likely than white males to die from drug-induced causes (Table 21.1). These disparities were consistent across the five-year age groups. There were no significant differences in the drug-induced death rates of black and white females. Between the periods 1992-1994 and 1996-1998, drug-induced death and premature mortality rates increased for white males and females while they remained about the same for black and Hispanic males and black females. There were insufficient numbers of drug-induced deaths among Hispanic females and Asian and Pacific Islander and Native American males and females to calculate reliable rates for these time periods (Table 21.1).

Since 1992, drug-induced mortality rates tended to be higher among Connecticut male residents than comparable rates nationwide. In 1998, the drug-induced mortality rate was higher among Connecticut female residents than the comparable national rate. Connecticut resident male and female rates exceeded the *Healthy People 2000* target. There is no *Healthy Connecticut* target for drug-induced mortality (Figure 21.3, Table 21.2).

Figure 21.2.



Risk Factors

Research has identified risk factors for drug abuse within two broad categories─societal/cultural norms and personal and interpersonal environments (Hawkins, Catalano, and Miller 1992). Factors include the larger social environment (such as neighborhood norms), early socialization in the family, and social groups of peers and school (Table 21.3). These social settings can also enhance the development of positive social attitudes and behaviors and provide a setting for increased social and self-competency skills, factors which can deter the initiation of drug use (Sloboda and David 1997).

Figure 21.3.

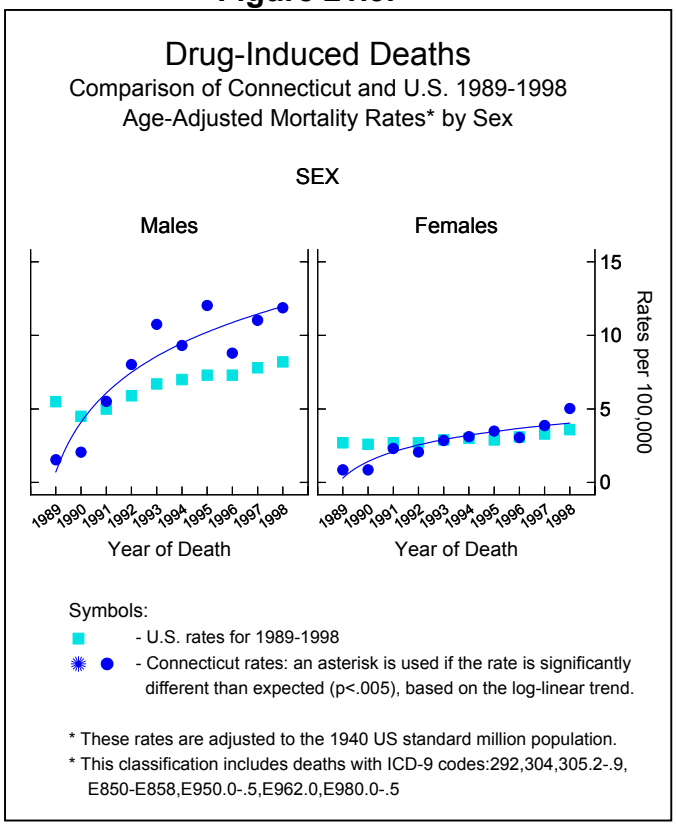


Table 21.2. Drug-Induced Age-Adjusted Death Rates, Comparison of CT with US - 1992 and 1998

	1992	1998	1998 CT AAMR Comparison
CT AAMR*	5.0	8.4	
US AAMR*	4.3	5.9	CT AAMR > US AAMR
*Healthy People 2000**	3.0	3.0	CT AAMR > HP 2000 rate

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

Table 21.3. Risk Factors for Drug Abuse

Societal / Cultural	*Individuals and Interpersonal Environments*
• Illegal drug availability • Extreme poverty (in conjunction with child behavioral problems) • Neighborhood disorganization (high population density, high residential mobility, physical deterioration, high adult crime related to high juvenile crime and drug trafficking) • Perceptions of approval of drug-using behaviors in the school, community, and peer environments	• Early and persistent behavior problems (aggressive behavior that continues into early adolescence; school misconduct) • Academic failure • Ineffective parenting, especially for children with difficult temperaments and conduct disorders • Lack of mutual attachment and nurturing • Family behavior (parental and sibling drug use increase risk of drug use initiation and drug abuse in children); chaotic home environments • Affiliations with deviant peers or peers around deviant behaviors

Sources: Hawkins, Catalano, and Miller 1992; Sloboda, Z. and S.L. David 1997.

Costs and Prevention

Total economic costs of drug abuse in the United States, estimated at $98 billion in 1992, include health care expenditures (such as the cost of treatment for health problems attributed to drug abuse, prevention, and rehabilitation); the cost of lost potential productivity due to impairment; the estimated cost of premature death due lifetime earnings lost; crime attributed to illicit drug abuse; and social welfare costs of drug-related impairment (Harwood, Fountain, and Livermore 1998).

The National Institute on Drug Abuse has outlined several important principles for prevention programs gleaned from over two decades of research. They include an emphasis on, and support of, protective factors such as close family communication and parental involvement in children's lives; and secondly, the identification and reduction of known risk factors such as chaotic home environments where the parents may be substance abusers and/or suffer from mental illness (Sloboda and David 1997).

Prevention programs that include general life skills training can increase social competency in areas such as assertiveness, self-efficacy, communication, and peer relationships, thereby enabling youth to resist drugs when offered. Community programs, such as media campaigns and policies that restrict access to drugs, are most effective when complemented by school and family interventions. The school environment serves as an important setting to reach all subpopulations including those specifically at risk for drug abuse, such as children with behavioral problems or with parents who are substance abusers (Sloboda and David 1997). The U.S. Preventive Services Task Force recommends

that clinicians be alert to signs and symptoms of drug abuse in patients and that they refer drug-abusing patients to appropriate specialized treatment (U.S. Preventive Services Task Force 1996).

State efforts to address problems of substance abuse include the Connecticut Alcohol and Drug Policy Council, which identified strategies for a comprehensive statewide plan for substance abuse prevention, treatment, and enforcement in its 1997 report. The Council's 1999 and 2002 plans, identify the three focus areas of prevention, treatment, and criminal justice. The Governor's Prevention Initiative for Youth, targets the reduction of alcohol, tobacco, and other drug use among young people ages 12 to 17. A uniform evaluation system was initiated in 2000 to assess all current state-supported substance abuse prevention programs (Connecticut Alcohol and Drug Policy Council 2001).

Evaluation research findings indicate that prevention programs can be cost-effective (Bukoski, 1997; Bukowski and Evans 1998; Horgan, Skwara, Strickler, et al. 2001; Pentz 1998). It is estimated that for every dollar spent on drug use prevention, communities can save four to five dollars in drug abuse treatment and counseling costs (Pentz 1998).

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