

connecticut **women's** health



CONNECTICUT DEPARTMENT OF PUBLIC HEALTH
2001

STATE OF CONNECTICUT

DEPARTMENT OF PUBLIC HEALTH

Joxel Garcia, M.D., M.B.A.
Commissioner



John G. Rowland
Governor

October, 2001

Dear Colleague:

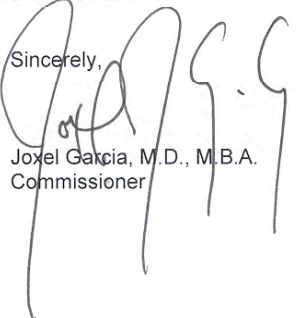
I am pleased to present the Department of Public Health's *Connecticut Women's Health* report developed in response to the increasing need to address the health concerns of over half of our state's population.

In the past, studies of women's health concerned mainly reproductive issues. Because the reproductive years now constitute less than half of a woman's life expectancy, the definition of women's health has expanded to include chronic and infectious diseases, injury and violence, and other issues beyond reproductive health.

Connecticut Women's Health presents the social context of women's health, demographic and socioeconomic characteristics, access issues, and an assessment of more than 22 selected health conditions related to reproductive health, cardiovascular disease, cancer, respiratory disease, other chronic conditions, infectious diseases, injury, and violence.

This report is the second in a series of topical reports on the health of Connecticut residents intended to be an educational and decision-supporting resource for public health policymakers, providers, and advocates. Together, I hope we can promote healthy lifestyles and reduce the health disparities experienced by women in Connecticut.

Sincerely,


Joxel Garcia, M.D., M.B.A.
Commissioner



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CONNECTICUT DEPARTMENT OF PUBLIC HEALTH
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Connecticut Department of Public Health
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Permission is granted for reproduction of this publication with appropriate reference to the source. Suggested citation:
Hofmann, M. and M. A. Hooper, eds. 2001. *Connecticut Women's Health*.
Hartford, CT: Connecticut Department of Public Health.

ACKNOWLEDGMENTS

SPECIAL ACKNOWLEDGMENT

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We gratefully acknowledge the following staff at the Connecticut Department of Public Health who provided data, technical assistance, and critical reviews of this report.

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*Original cover design courtesy of Women's Health Interactive® (www.womens-health.com).
Adapted for this report by Carol E. Bower.*

This report was printed with funds from the: Office of Policy, Planning, and Evaluation; Multicultural Health Program; Tumor Registry and the National Cancer Institute (contract N01-67005); Centers for Disease Control and Prevention WISEWOMAN Program; the HIV Prevention Program; and the Radon Program.

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INTRODUCTION

During the last century, factors such as improvements in medical technology, environmental controls, social legislation, and personal lifestyle changes have increased a woman's life expectancy dramatically—from 48 years for a woman born in 1900 to 80 years for a woman born in 1998 (National Center for Health Statistics, 2000). Despite these successes, women are now at greater risk for disabling and chronic diseases. More than 1 million female adults in Connecticut (90 percent) have at least 1 major risk factor for a serious chronic disease, such as heart disease or cancer. Fourteen percent (approximately 180,000) consider their general health status to be fair or poor and 10 percent of those under age 65 (140,000) have no health care coverage (Centers for Disease Control and Prevention, 2001).

In the past, studies of women's health concerned mainly reproductive issues. Because the reproductive years now constitute less than half of a woman's life expectancy, the definition of women's health has broadened beyond reproductive health to consider social issues, chronic conditions, infectious diseases, and injury and violence that affect women throughout their lives.

Connecticut Women's Health is intended as an assessment of women's health and a decision-making resource for policymakers, health care providers, academics, and advocates who are addressing the health problems and disparities experienced by women. Chapters 1, 2, and 3 of this report present the social context of women's health, demographic and socioeconomic characteristics of Connecticut's women, and access to health care issues. The remaining chapters are divided into seven sections: reproductive health; cardiovascular disease,

cancer, respiratory disease, other chronic conditions; infectious diseases; and injury and violence.

The health conditions presented in *Connecticut Women's Health* were selected according to three criteria: 1) the leading causes of hospitalization and death (e.g., heart disease, stroke, and cancer); 2) health issues and conditions unique to females (e.g., gynecologic cancers, pregnancy and childbirth); and 3) those that are more prevalent in females than males (e.g., sexual and domestic violence, autoimmune diseases, certain sexually transmitted diseases). Each chapter presents the health condition in the context of other relevant perspectives, such as age, race, or ethnic group; by socioeconomic status; and by behavioral risk factor. The Appendices include technical notes with a glossary of terms and summary tables of mortality, hospitalization, and risk factor surveillance data presented in this report.

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- Centers for Disease Control and Prevention. 2001. *Behavioral Risk Factor Surveillance System: Connecticut Statewide Survey Data - Weighted*. Atlanta: Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Behavioral Surveillance Branch.
- National Center for Health Statistics, 2000. *Health, United States, 2000*. Hyattsville, MD: U.S. Government Printing Office.

1. SOCIAL CONTEXT OF WOMEN'S HEALTH

During the past decade, women's health concerns have evolved into key policy issues in the federal government, in the public's awareness, and in the popular press. The development of the women's health agenda in the United States was preceded by decades of the women's health movement, which raised women's awareness of their health and the importance of having greater control over decisions related to their bodies. The Boston Women's Health Book Collective, which published the self-help book *Our Bodies, Ourselves* (1971; 1998), the National Women's Health Network, and the National Black Women's Health Project (Avery, 1996) are a few groups that have exemplified the women's advocacy and self-help movements since the 1970s. Political action by women's groups has increased the public's attention about the lack of funding for women's diseases such as breast cancer, and the need to change the definition of HIV/AIDS to be inclusive of the unique symptoms and conditions that present themselves in women (Auerbach and Figert, 1995).

Until very recently, a large portion of the cardiovascular disease research findings in the U.S. was based on studies of men. Women were excluded from some major clinical trials such as the Multiple Risk Factor Intervention Trial (MRFIT) of 15,000 men, which studied risk factors for coronary heart disease (Multiple Risk Factor Intervention Trial Research Group, 1985) and the 1988 Physicians Health Study, involving 22,071 men, which demonstrated aspirin's effect on reducing cardiovascular disease risk (Manson et al., 1991). The Baltimore Longitudinal Study on Aging did not include women from 1958 to 1978, but issued a 1984 report based on these data entitled "Normal Human Aging" (U.S. Public Health Service, Gerontology Research Center, and National Institutes of Health, 1984).

There have been a variety of reasons given for excluding women from clinical trials, such as researchers' concerns about possible teratogenic effects (damage to a pregnant woman's fetus)

and beliefs that variability among women due to menstrual cycle, pregnancy, and menopause might complicate study designs. Researchers very often made assumptions that there were no important gender differences influencing treatment and tended to focus on groups, such as white middle-aged men, believed to be at high risk for heart disease (Auerbach and Figert, 1995).

Medical researchers commonly reported results from male-only studies, but implied or stated that the findings were applicable to both males and females. Thus, in major studies of chronic disease, the male body and experience became the norm for medical treatment for everyone. The exclusion of women from major clinical studies made the assessment of potential gender differences impossible and, consequently, left wide gaps in knowledge and treatment of chronic disease in women (Auerbach and Figert, 1995). This fact is of particular concern because older women, the group most affected by chronic conditions, are the fastest growing segment of the U.S. population (Matthews et al., 1997).

There is also evidence that physicians commonly failed to recognize symptoms, such as chest pain, in female patients in the mistaken assumption that women do not die from heart disease (American Medical Association, 1991; Kuhn and Rackley, 1993; McKinlay, 1996). Studies have documented physician bias in referring male and female patients for coronary bypass surgery. One study found that, among a group of patients showing evidence of heart disease, 40 percent of the men but only 4 percent of the women were referred for further tests to determine if bypass surgery was needed (Tobin et al., 1987). A second study found that women tended to be referred for coronary bypass surgery at older ages and when they were sicker than men with comparable symptoms. Consequently, women were nearly twice as likely as men to die from this procedure (Khan et al., 1990).

Beginning in 1989, several initiatives placed women's health research on the national agenda thanks to the efforts of congressional legislators, scientists, and advocates. The importance of integrating women's health concerns into the national biomedical health research agenda was recognized by the National Institutes of Health (NIH) when it established an Office of Research on Women's Health (ORWH) in 1990. ORWH has three key mandates: 1) to develop, enhance, and increase research into diseases and health conditions that are unique to, or more common or serious in women, or for which there may be different risk factors for women; 2) to ensure that women are well represented in federally-funded health research, particularly clinical trials; and 3) to lead initiatives for increasing the numbers of women in biomedical careers (Kirschstein, 1991).

In 1991, NIH established the Women's Health Initiative (WHI), a nationwide, long-term study involving 164,500 women ages 50 to 79 which focuses on strategies to prevent chronic conditions of heart disease, breast and colorectal cancer, and osteoporosis in postmenopausal women. This is one of the largest and far-reaching research studies of women's health in the United States. The WHI will assess the risks and benefits of hormone replacement therapy, vitamin and calcium supplements, and exercise and low-fat diet on cardiovascular disease, osteoporosis, and cancer among women at mid-life (Kirschstein, 1991; Matthews et al., 1997; McGowan and Pottern, 2000).

The Nurses' Health Study, started in 1976, is the other major long-term study of chronic disease in American women. It is examining the health habits and lifestyle of over 225,000 women and is producing information regarding the development and treatment of heart disease, diabetes, and breast and ovarian cancer (Colditz et al., 1997; Hankinson et al., 2001). Emerging information from these and other long-term studies will provide important insights and new knowledge regarding risk factors for, and effective treatment of, major chronic conditions affecting American women of diverse ethnicities, socioeconomic backgrounds, and sexual orientations.

The Institute of Medicine has recently formed a scientific committee to review the current understanding of the differing biological influences on women's and men's health and to make recommendations for research in this area. Recent findings in molecular biology, endocrinology, and genetics may provide new insights regarding sex-based differences in physiological functioning, which can lead to the development of new treatment regimens that consider women's particular physiology and neuroendocrine functioning (Institute of Medicine, 2001).

In addition to underlying biological mechanisms, societal factors contribute to the differential health outcomes of women and men. The social sciences have yielded important insights into the ways in which gender, the socially determined roles and societal attitudes regarding males and females, influences health (Lorber, 1997; Bird and Rieker, 1999). Decades of research have helped explain how patterns of disease are influenced by the historical, geographical, cultural, and social context in which people live (Dubos, 1959; Zola, 1966; Ehrenreich and English, 1978; Mechanic, 1982; Sontag, 1989; Evans et al., 1994).

Medical sociologists have documented how women's and men's social roles differentially affect their health. A longstanding observation of researchers has been that, although men in industrialized societies die at younger ages than do women, women experience more illness (Verbrugge, 1976, 1989; Nathanson, 1984; Verbrugge and Wingard, 1987; Lorber, 1997). Systematic studies have shown that a large portion of excess male mortality is attributable to greater rates of smoking and other risk-taking behaviors, such as alcohol, drug, and gun use, by men relative to women (Waldron, 1986; Waldron, 1995). Males, more than females in American society, are encouraged to express personal hostility in a physically aggressive manner, and male images in the popular media reinforce the social acceptability of violent and risk-taking behaviors. Traditional gender roles that reinforce male dominance and aggression contribute to males' premature mortality (Stillion, 1995).

Women's excess morbidity is closely linked to social factors like lesser employment; greater feelings of stress, unhappiness, and vulnerability to illness; and less physically strenuous leisure activities compared to men (Verbrugge, 1989). Women's role as the primary caregivers within the family and in the larger society influences the extent to which they seek health care for both themselves and others (Abel and Nelson, 1990). Women are the likely ones to encourage their family members to get medical care, to make the appointments, and to make sure the appropriate medications are available in the household. They are also more likely than men to take off from work when a child is sick or to care for an elderly family member (Smyke, 1991). Gender inequalities in the job market reinforce the idea that lower wage earning females, rather than higher wage earning men, should miss work time due to family illness (Auerbach, 1988). As caregivers, women are exposed to acute infections and stress, which can contribute to immune system dysfunction (Thoits, 1986).

Other studies suggest that women's longer life expectancy does not necessarily lead to a better quality of life, and that the physical, psychological, and social needs of aging females have not been adequately addressed (Ory and Warner, 1990; Worobey and Angel, 1990). Elderly women are more likely than their male counterparts to be impoverished and living in nursing homes (Longino, 1988). Thus, a longer life may not be physically, emotionally, or materially rewarding for many women. Although women as a group live longer than men, they experience greater poverty, chronic illness, and depression through old age (Auerbach and Figert, 1995).

An understanding of women's health, therefore, requires that we consider how a woman's day-to-day life and multiple social roles profoundly influence her experience of illness. For example, a women's lesser economic and social power relative to men in our society, her race and ethnicity, educational background, marital status, economic circumstances, and presence of dependent family members have varying effects on how her medical condition is treated and how it progresses. These social factors interact to produce different behaviors

when sick and can bring about differing responses by health care providers. A single mother in a low-wage job, for example, has much less time, energy, and educational resources to practice good nutrition; to pursue healthy leisure time activities; to schedule medical appointments when sick; and to have access to quality medical care, compared to a married man who is a well-paid manager in a company with good benefits. Health care providers do not always carefully consider how the social circumstances of a low-income single mother's life keep her from staying healthy. Indifference or judgmental responses by health care providers to a woman's social circumstances can influence the type and effectiveness of medical treatment, as well as the patient's motivation to continue treatment (Lorber, 1997).

A Task Force of the NIH Office of Research on Women's Health has recently issued its *Agenda for Research on Women's Health for the 21st Century*, which includes reports and recommendations related to research on women's health across the life span. Such cutting-edge research on women's health will continue to shed new light on conditions of particular relevance to women that, until very recently, have been neglected. Information emerging from the various research streams should provide valuable evidence for improvement in the health and well-being of all American women for many years to come (U.S. Department of Health and Human Services, 2001).

The practical consequences of women's health research and activism for the physical, psychological, and social conditions of American women have been enormous. For example, physicians are now better informed regarding the early warning signs and treatment of heart disease in women. Medical school training now prepares physicians to communicate better in explaining treatment options and in developing a treatment plan with patients. Cultural competency training programs for health care providers are being widely implemented in professional schools and in continuing education programs throughout the United States. Women of different cultural backgrounds have different health and nutritional practices and may have differing interpretations of medical advice.

Cultural competency training can sensitize health providers to these differences among patients.

American women have benefited greatly from the self-help movement and education regarding everyday health concerns. Principles of balanced eating and exercise and common health practices like breast self-exam are widely disseminated in the popular media. Good sexual and reproductive health requires the regular health practices of women (and men) who are well-educated in this area. Although informational resources for optimal sexual and reproductive health are sorely lacking in some localities, American women today have a broader array of options than previous generations had.

Some workplaces are adopting “family-friendly” policies that allow workers flexibility during the week for child care and other family needs. Workers nationwide have the option of taking family sick leave to care for family members who are seriously ill. Women, as the traditional caretakers of the family, are most likely to benefit from such policies. Although more flexible workplace arrangements are still needed, the net effect of these multiple social changes on the health of American women will be felt for generations to come.

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2. POPULATION DEMOGRAPHICS

A woman's health is the product of her biology and behavior, together with all the social, cultural, economic, and environmental forces that affect the quality and duration of her life. Connecticut's 1.7 million female residents represent 52 percent of the total population, and comprise white, black, Asian and Pacific Islander, and Native American racial groups; over 60 ethnic and ancestry groups including Latin American, West Indian, and Vietnamese; diverse sexual orientations; various education levels; and socioeconomic groups from the impoverished to the wealthy. This chapter presents the diversity of Connecticut women and its effect on their health status.

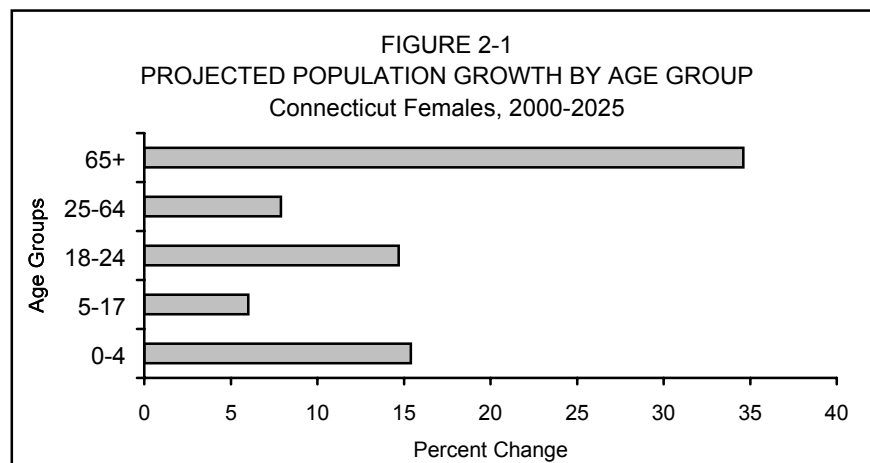
AGE GROUPS

Age and health are profoundly related, because health declines as we age. While some health conditions can occur throughout the course of a woman's life, others are specific to certain age groups. For some health conditions, a woman's behavior early in life can strongly influence the quality of her health in later years. For example, calcium consumption in adolescence can reduce a woman's risk of osteoporosis in later years. Risky sexual behavior is a key health issue of adolescence and

early adulthood that contributes to unplanned pregnancies and sexually transmitted diseases, including HIV/AIDS, where the consequences affect a woman throughout her life. By the time women reach age 40, more than 80 percent have had at least one child (National Center for Health Statistics, 2000).

Of the 1,690,000 females in 1999, 20 percent were under 15 years of age, 41 percent were of childbearing age, 22 percent were 45-64 years of age, and 17 percent were 65 and older. The largest proportional population increase is projected to be for women 65 years of age and older. This group is expected to increase by one-third in number during the next 25 years (Fig. 2-1).

The aging of the female population has important implications for current and future health care needs and disease prevention strategies, as discussed throughout this report. Certain diseases are more prevalent among elderly women, particularly stroke, pneumonia and influenza, chronic obstructive pulmonary disease, arthritis, osteoporosis, and other disabling conditions (U.S. Department of Health and Human Services, 2000).



Source: U.S. Census Bureau, 2000.

RACE AND ETHNICITY

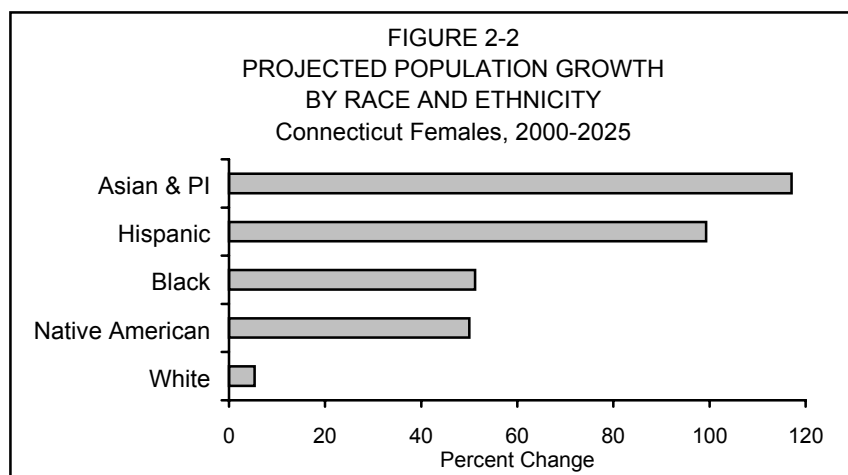
Race and ethnicity are strongly associated with health outcomes. The life expectancy at birth for white females in the U.S. is 5.2 years longer than that for black females. Women of color have higher mortality rates than white women for certain cancers, cardiovascular disease, diabetes, chronic obstructive pulmonary disease, homicide, and HIV/AIDS (Hynes et al., 1999). And health disparities among minority women are more often the consequences of social prejudice and socioeconomic status than of behavior or genetics (Auerbach et al., 2000; Polednak, 1997).

Between 1980 and 1999, estimated numbers of Asians and Pacific Islanders among Connecticut females quadrupled, Native

During the next 25 years, the numbers of Asian and Pacific Islander females are projected to increase by nearly 120 percent and Hispanic females are expected to double. Black and Native American females are projected to increase by 50 percent, compared to a small increase of 5 percent for white females (Fig. 2-2).

FAMILY STRUCTURE

Marital status, the presence of a husband, and children living in a household are all measures of family structure that have an effect on a woman's health status and her quality of life. In the U.S., married women have the lowest age-adjusted death rates, whereas widowed and never-married women have the highest. In 1998, the age-adjusted death rates of widowed and never-married women were about 2.3 times



Source: U.S. Census Bureau, 2000.

American and Alaskan Natives increased by two-thirds, females of Hispanic ethnicity more than doubled, and blacks increased by more than one-third. In contrast, females of white race increased by only 1.8 percent (U.S. Census Bureau, 1999). In 1999, an estimated 87.6 percent of Connecticut females were white, 9.6 percent were black, 2.6 percent were Asian or Pacific Islanders, and 0.2 percent were Native Americans and Alaska Natives; of these, 8 percent were of Hispanic ethnicity.

greater than those of married women (Murphy, 2000). Research indicates that marriage has beneficial health effects for unemployed women, but has insignificant health benefits for employed women (Waldron et al., 1996). This implies that marriage, like employment, can provide personal and financial security to enhance a person's quality of life.

The presence of children also affects a woman's health. While children can provide an emotional benefit to anyone's life, their presence

increases a mother's acute health problems due to the increased exposure to infections and injury (Verbrugge and Madans, 1985). Research also points to the reduced amount of recuperative time and medical care for women with children. These limitations are influenced by the parental demands from children in a household and do not imply that the need for medical care or recuperative time is less for women with children (Verbrugge and Madans, 1985).

Family structure has changed dramatically with an increase in non-traditional households. From 1970 to 2000, both the numbers and proportions of Connecticut families headed by a single female increased substantially. The number doubled from 79,400 to 157,400 and the proportion increased from 10 percent to 18 percent of all families. Nearly 60 percent of these female-headed families included children under 18 years of age. The proportion of female-headed families differs among racial and ethnic groups. In 1990, approximately 40 percent of all black and Hispanic families were female-headed households with no husband present, compared with 12 percent of white families (U.S. Census Bureau, 1990).

Not only are women the primary caregivers for children, but also for the elderly. Research suggests that the prolonged stress and physical demands of caregiving may compromise the physical and mental health of the caregiver (Schulz and Beach, 1999). Studies show that caregivers are less likely to engage in preventive health behavior and have decreased immunity measures (Schulz and Beach, 1999). The majority of women provide care to aging relatives at some time in their lives, with women of color being more likely than white women to have this responsibility (Permanent Commission on the Status of Women et al., 2000).

SOCIOECONOMIC STATUS

Socioeconomic status is a composite measure of income, employment, and education that affect health either directly or indirectly through their influences on people's behaviors. The complicated relationship between socioeconomic status and health is well

documented. Adverse health events have economic consequences; conversely, education, type of employment, and income affect health, but their causes and effects are not clear. It is uncertain, for example, whether more income and wealth cause people to be healthier because of greater access to health care services and less household and occupational stress, or whether poor health results in lower income and wealth, because of higher health care expenditures and less work (National Institute on Aging, 1999).

Environmental health risks, for example, are more common in impoverished communities. Such communities tend to be situated in close proximity to industrial facilities and waste disposal sites, the siting of which may be partly related to the efficacy of land use or discrimination based on class, race, or ethnicity. As a consequence, exposure to environmental toxins and the health consequences of such exposures are often greater among persons in minority groups or with incomes below the federal poverty level than in whites or those with higher incomes (Hynes et al., 1999).

Higher socioeconomic status enables women to obtain safe and sanitary housing, healthy food, safer work conditions, better employee benefits, and appropriate health care (Hynes et al., 1999), whereas lower socioeconomic status contributes to premature morbidity and mortality (Haan et al., 1987; Lantz et al., 1998; Auerbach et al., 2000).

Educational Attainment

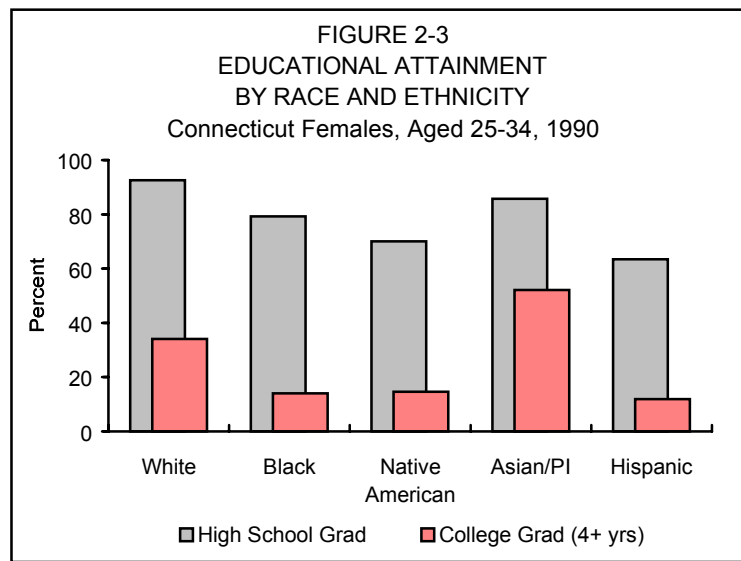
Level of education is linked closely to health, morbidity, and mortality; persons with more education have better health and lower mortality rates than those with less education (Pappas et al., 1993). Education also influences how people make health-related decisions. It was found, for example, that after experiencing a heart attack, nearly 90 percent of college graduates, but only 10 percent of those with less than a high school education, quit smoking (Wray et al., 1998).

In 2000, 10 percent of Connecticut females aged 18 years and older reported they had not graduated from high school, whereas 89 percent were high school graduates and 35 percent were

college graduates (Centers for Disease Control and Prevention, 2001).

The proportion of Connecticut women 25 years of age and older who were high school graduates increased from 57 percent in 1970 to 79 percent in 1990, and the proportion of college graduates rose from 10 percent in 1970 to 24 percent in 1990 (U.S. Census Bureau, 1993). Except for Asians and Pacific Islanders, the educational attainment of minority women in 1990 was lower than that of white women (Fig. 2-3).

There has been a steady increase in the proportion of women in the Connecticut labor force during the past 30 years. Despite this increase, occupational segregation still exists between the sexes. Although women represented 47 percent of the 1990 employed labor force, they held 78 percent of the administrative support positions and 73 percent of elementary and secondary school teaching jobs, but only 41 percent of all executive, administrative, and managerial positions (U.S. Census Bureau, 1993). Disparity in employment and occupation is also evident among Connecticut women of



Employment

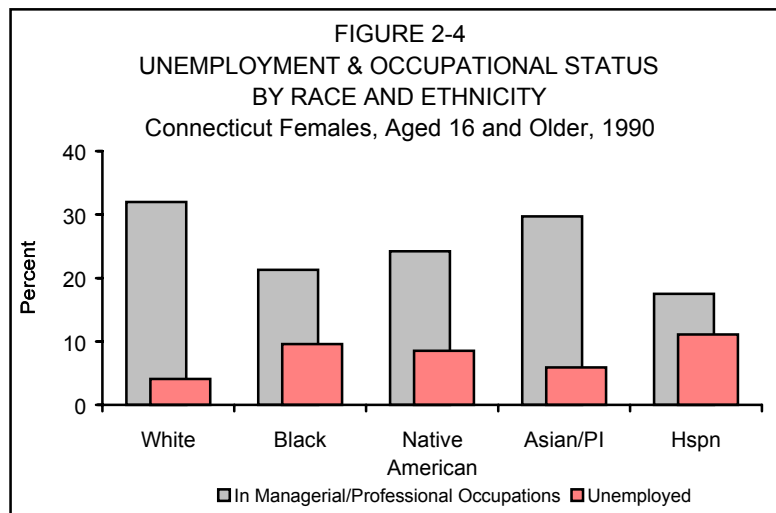
A strong relationship also exists between employment and health, not only through the effects of the resulting income but also because of the contributions of the work environment to health and health behaviors (work-related stress, social support networks, etc.). Women in clerical positions, for example, have a greater risk than administrators for developing coronary heart disease (Marmot et al., 1997), and women office workers in low job classes are more likely than those in higher job classes to be absent from work because of back pain (Hemingway et al., 1997).

different racial and ethnic groups (Fig. 2-4). In 1990, Hispanic women were the most likely to be unemployed, and the least likely to hold professional or managerial positions.

In 2000, 59 percent of Connecticut women aged 18 years and older reported they were employed, compared to 76 percent of men; while 14 percent of the women were students or homemakers, 23 percent were retired or unable to work, and 4 percent were unemployed. Of those who were not retired or unable to work, 77 percent were working (Centers for Disease Control and Prevention, 2001).

The majority of Connecticut women with children participate in the labor force, and the older their children, the more likely mothers are to work. In 1990, 59 percent of women with children under 6 years of age were working, a jump from 41 percent in 1980. Of women with children 6 to 17 years of age, nearly 80 percent were working in 1990, compared to 67 percent in 1980 (U.S. Census Bureau, 1993).

Wealth is measured as household net worth—the value of assets (real estate, stocks, motor vehicles, etc.) minus debts. In 1995 in the U.S., the median net worth of female householders was 9 percent lower than that of male householders and 77 percent lower than that of married-couple householders (\$14,949, \$16,346, and \$64,694, respectively). Among female householders, those less than 35 years of age had the least net worth (\$2,580), and those 65



Income, Wealth, and Poverty

People with higher incomes and more wealth tend to be healthier and live longer than those with lower incomes and those living in poverty, especially at older ages. Conversely, those who live in poverty have shorter lifespans and higher rates of illness than those with incomes above the poverty level (Lyons et al., 1996). It is well known that women earn less income than men, have a lower net worth, and represent a greater proportion of persons living in poverty, which can translate into poor health outcomes.

Working women traditionally have earned less than men. In the U.S. from 1995 to 1999, real wages of young college graduates rose only 9 percent for females, compared to 15 percent for males (Mishel et al., 2001).

years of age and over had the greatest (\$61,549) (Davern and Fisher, 2001).

In Connecticut, the 1989 median income of women 15 years of age and older was \$14,028, or half that of men (\$27,127). Women thus earned an estimated 52 cents for every dollar earned by a man in Connecticut—an improvement over the 42 cents for every dollar earned in 1979. The 1989 median family income in Connecticut was \$49,199 for all families, but only \$25,739 for female-headed families. Females headed two-thirds of Connecticut families living in poverty in 1989.

The number of Connecticut families with incomes below the poverty level¹ dropped by 13 percent from 49,680 in 1979 to 43,965 in 1989, while the number of female-headed families living in poverty decreased by only 2 percent from 30,227 to 29,634. The number of families below the poverty level according to the 2000 Census have not been released, but more than twice the proportion of Connecticut women as men reported household incomes below \$15,000 (7.3 percent and 3.6 percent, respectively) (Centers for Disease Control and Prevention, 2001).

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Appendix B includes a sociodemographic profile of Connecticut females detailing the information presented in this chapter.

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¹ The 1979 and 1989 average poverty thresholds for a family of four were \$8,414 and \$12,674, respectively. The 1999 average poverty threshold for a family of four was \$17,029 (U.S. Census Bureau, 2001).

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3. ACCESS TO HEALTH CARE SERVICES

Access to quality health care is key to a woman's health status. Access encompasses both the ease and timeliness with which health services can be obtained (Office of Health Care Access, 1999; Millman, 1993). Predictors of access to health services include having health insurance, adequate income, and a regular primary care provider or other regular source of care (U.S. Department of Health and Human Services, 2000). Utilization of certain clinical preventive services, such as early prenatal care, mammography, and Pap tests, can also indicate better access to services.

Access to health services can be impeded by economic barriers (no insurance, poverty), supply and distribution barriers (inadequate or inappropriate services or providers, geographic unavailability of services, lack of transportation), and language and cultural barriers.

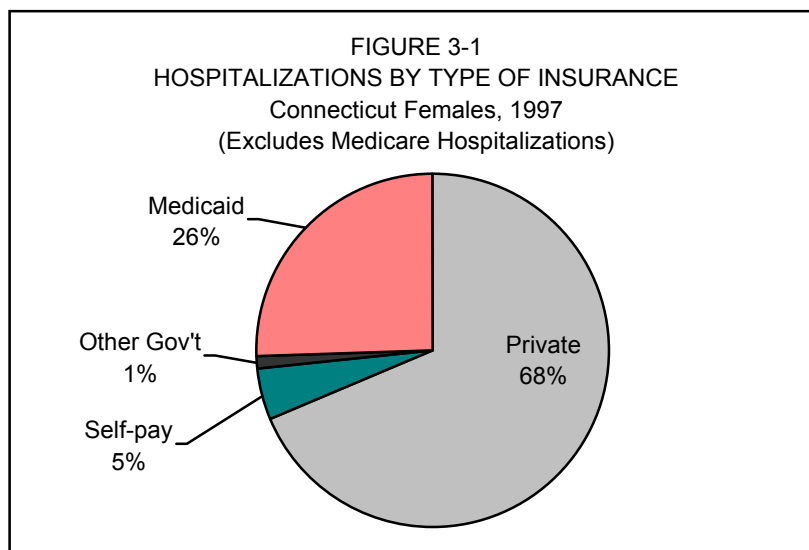
HEALTH INSURANCE COVERAGE

During the past decade, the proportion of Connecticut women without health insurance has varied from 6 percent to 9 percent (Centers for

Disease Control and Prevention, 1991-2000). According to the Connecticut Behavioral Risk Factor Surveillance Survey, Connecticut had the sixth lowest percentage of women without health insurance in the nation in 1999 (Centers for Disease Control and Prevention, 2000).

Of Connecticut women aged 18 to 64 years, 10 percent reported they did not have any kind of health care coverage, including health insurance, prepaid or government plans such as HMOs or Medicare; in line with the national median of 10 percent uninsured. Women aged 65 years and older were the least likely to be uninsured (1 percent), due to Medicare coverage, and women 18-24 years of age were the most likely to be uninsured (20 percent) (Centers for Disease Control and Prevention, 2000).

State-level hospitalization data also are useful for estimating health insurance coverage (Office of Health Care Access, 2001; Turner and Campbell, 1999). In Connecticut in 1997, "self pay" and "no charge," (i.e., not having health insurance coverage) was listed as expected payer on 4,154 hospitalization records of resident females (Fig. 3-1).



Source: Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001.

AFFORDABILITY OF HEALTH CARE

To access quality health care, a woman must have enough money to pay for the services she needs. Although most types of health insurance cover common treatment services and screening and diagnostic tests, many preventive services and interventions are not covered. While most health insurers will pay to treat emphysema, lung cancer, and other tobacco-related diseases, for example, few will reimburse for smoking cessation programs or medications.

In 1999, nearly one out of ten Connecticut women reported that cost was a barrier to getting medical care on at least one occasion during the prior year. Cost was more likely to affect persons of Hispanic ethnicity (26 percent) and black race (11.8 percent) than white non-Hispanics (7 percent), and to affect unmarried persons more than married persons (11 percent and 7 percent, respectively). Those who did not graduate from high school were four times more likely than college graduates to experience cost barriers to health care, and people with incomes under \$25,000 were about five times more likely than those earning more than \$75,000 to be affected by cost (Centers for Disease Control and Prevention, 2000).

AVAILABILITY OF HEALTH CARE

Women who live in geographic areas that lack health care providers are often unable to obtain health care services. In 1998, 14 percent of Connecticut women 18 years of age and older reported that they had no regular source of health care when they were sick or needed advice. Uninsured persons, those with incomes under \$20,000, residents of cities, and people of Hispanic ethnicity were also more likely to have no regular source of care, when compared with insured persons, those with higher incomes, residents of rural areas, and non-Hispanic persons (Centers for Disease Control and Prevention, 1999).

Compared to the national ratio of physicians to population (198 per 100,000) the Connecticut ratio is high (Bureau of Health Professions, 2000). Even so, some regions, population groups, and facilities in Connecticut are federally designated as having health professional shortages with respect to primary care physicians.

A shortage of primary care physicians occurs in a geographic area (county, community, neighborhood) where the physician-to-population ratio falls below certain thresholds. The ordinary threshold is 1 full-time primary care physician per 3,500 population (29 per 100,000). For areas with “unusually high needs” for primary care services or “insufficient capacity of existing providers,” however, the threshold ratio is 1 physician to 3,000 population or 33 per 100,000. “Unusually high needs” means that an area has a high birth rate or infant death rate, or a high percentage of low-income households. “Insufficient capacity of existing providers” means excessive numbers of outpatient visits per doctor, unusually long waits for routine appointments, excessive waiting time at doctors’ offices, excessive emergency room use for routine primary care, low utilization of health services, and large proportions of physicians who do not accept new patients (Bureau of Primary Health Care, 1989).

A population group within a given geographic area may be designated as having a shortage of primary medical professionals when the ratio of physicians to persons in the population group is less than 1:3,000 (33 per 100,000 population), and when barriers to accessing primary care professionals exist. Access barriers can be economic, linguistic, cultural, or architectural barriers, or refusal of providers to accept certain types of patients or Medicaid reimbursement (Bureau of Primary Health Care, 1989).

As of March 31, 2001, 45 service areas, 50 population groups, and 7 facilities, representing 1,324,501 persons or nearly 40 percent of Connecticut residents, were federally designated as health professional shortage areas for primary medical care (Bureau of Primary Health Care,

2001). Shortages occurred in geographic areas or in populations of 16 Connecticut towns, including the state's six largest towns, and among low-income population groups in several smaller towns (Table 3-1), indicating that some women residing in these towns may have difficulty obtaining primary medical care.

As of June, 2001, 9,700 physicians (285 per 100,000 population or one for every 350 state residents) were practicing medicine in Connecticut (Connecticut Department of Public Health, Bureau of Regulatory Services, 2001).

Just under one-third of these (3,022 or 89 per 100,000 population) were primary care physicians (i.e., general or family practice, internal medicine, obstetrics/gynecology, and pediatric specialties, all without sub-specialties) (Table 3-2).

MEASURES OF ACCESS

Utilization of clinical services for the prevention and early detection of health problems is a common measure of access to care

TABLE 3-1
PRIMARY CARE HEALTH PROFESSIONAL SHORTAGE AREAS OR POPULATIONS^a
Connecticut, 2000

| Geographic Areas | Populations ^b |
|------------------|--------------------------|
| Bridgeport | Danbury |
| East Lyme | Meriden |
| Groton | New Britain |
| Hartford | New Haven |
| New Haven | New London |
| Norwalk | Norwich |
| Stamford | Stratford |
| Waterbury | Vernon |
| | Waterbury |
| | West Haven |

Source: U.S. Department of Health and Human Services, 2000.

^a Geographic areas and populations do not necessarily include all census tracts within the named town.

^b All Health Professional Shortage Populations qualify as low-income.

TABLE 3-2
PRACTICING PHYSICIANS AND SURGEONS IN SELECTED SPECIALTIES
Connecticut, 2001

| Specialty | Number | Ratio ^b |
|---------------------------|--------------------|--------------------|
| All | 9,709 | 285 |
| Primary Care: | 3,022 ^a | 89 |
| Family Practice | 455 ^a | 13 |
| General Practice | 11 ^a | <1 |
| General Internal Medicine | 1,350 ^a | 40 |
| Pediatrics | 698 ^a | 165 |
| Obstetrics and Gynecology | 508 ^a | 36 |
| Preventive Medicine | 59 | 2 |
| Surgery (all specialties) | 1,007 | 30 |

Source: Connecticut Department of Public Health, Bureau of Regulatory Services, 2001.

^a Counts are for those without sub-specialties.

^b Ratios are expressed per 100,000 total population for all specialties, family and general practices, general internal medicine, preventive medicine, and surgery. Ratios for pediatrics are expressed per 100,000 children under age 19, and ratios for obstetrics and gynecology are expressed per 100,000 females aged 12 years and older.

and can signal the presence of access barriers. One example is cancer screening. The diagnosis of breast and cervical cancers at later developmental stages is less likely to have a favorable outcome. Mammography utilization is discussed in Chapter 9, *Breast Cancer*; and screenings for cervical and colorectal cancers are discussed in Chapter 11, *Cervical Cancer* and Chapter 10, *Colorectal Cancer*, respectively.

Prompt and effective primary care in a doctor's office or other outpatient setting, followed by proper management, can reduce the need for hospitalization for many medical conditions, such as asthma, dehydration, urinary tract infections, and perforated or bleeding ulcers (Foland, 2000; Office of Health Care Access, 2000). These conditions are referred to as "ambulatory care sensitive" hospital admissions. When early care is delayed or foregone, the result is often "avoidable" or "preventable" hospitalizations which can indicate problems with access to primary health care services or inadequate outpatient management and follow-up. Three out of four "avoidable" hospital admissions occur through emergency rooms (Foland, 2000).

Compared to all hospitalizations, uninsured women and those with Medicaid coverage had higher proportions of avoidable hospitalizations when compared with those covered by private insurance (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001).

Avoidable hospitalizations are higher among residents of urban areas. In 1997, female residents of Connecticut's seven largest towns accounted for 36 percent of all avoidable hospitalizations of females, but only 24 percent of Connecticut's female population (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001). Residents of low-income areas are also more likely to be hospitalized for conditions that could have been prevented by routine outpatient care (Center for

Health Economics Research, 1993). Hospitalizations of children and adults for asthma, for example, were four to five times higher in low-income than in high-income neighborhoods (Center for Health Economics Research, 1993).

PERSONAL BARRIERS TO ACCESS

Personal factors such as language, culture, and sexual orientation can profoundly affect both a woman's ability to access appropriate health care services. Such factors can create barriers to access, particularly for members of culturally diverse groups.

Language

Language can be an obstacle to health care access for people who do not speak English and for the deaf and hearing impaired. These groups constitute a sizeable proportion of Connecticut residents. According to the 1990 U.S. Census, about 9 percent of Connecticut's population (279,000 persons) were foreign born, and 466,000 persons or 15 percent of those 5 years of age and older spoke a language other than English at home. Of this group, 184,018 persons (39 percent) did not speak English "very well" (U.S. Census Bureau, 2001). Additionally, an estimated 6 percent of Connecticut's population or about 204,300 persons are hard of hearing, and 25,500 residents are considered profoundly deaf (Connecticut Commission on the Deaf and Hearing Impaired, 2001).

The ability of Connecticut's health care providers to communicate with non-English speaking people and is very limited. In 2001, 3,434 physicians and surgeons practicing medicine in Connecticut (35 percent of total) indicated that a language other than English was spoken at their practice location (Connecticut Department of Public Health, Bureau of Regulatory Services, 2001). Spanish was the most frequently spoken language (Table 3-3).

TABLE 3-3
 FOREIGN LANGUAGES MOST FREQUENTLY SPOKEN
 AT PRACTICE LOCATIONS OF PHYSICIANS AND SURGEONS
 Connecticut, 2001

| Language | No. Locations | Language | No. Locations |
|------------|---------------|----------|---------------|
| Spanish | 2,867 | Chinese | 123 |
| French | 732 | Arabic | 107 |
| Italian | 536 | Greek | 103 |
| German | 275 | Urdu | 87 |
| Hindi | 264 | Hebrew | 84 |
| Polish | 263 | Korean | 62 |
| Portuguese | 216 | Filipino | 52 |
| Russian | 155 | Gujarati | 42 |

Source: Connecticut Department of Public Health, Bureau of Regulatory Services, 2001.

Culture

Culture refers to the system of behaviors and beliefs that enable a group to assign meaning to and make sense of the world in which they live (Bayne-Smith, 1996). Cultural differences also influence health-related behaviors and can create access barriers. The health belief systems of many cultural groups differ greatly from those of Western-trained physicians (Bayne-Smith, 1996). In the health systems of some groups, there is no separation between physical, emotional, mental, and spiritual pain (Bayne-Smith, 1996). Moreover, many non-Western women do not go directly to a physician when they are ill. Instead, they first attempt to treat themselves, and if that fails, they follow the recommendations of friends, family, and in some cases, alternative or folk healers (Bayne-Smith, 1996).

Additional cultural factors affecting health-related behaviors of women in certain minority groups are lack of knowledge about Western medicine, fear of public institutions (based on experiences with discrimination), modesty about their bodies, and the belief that their own needs are secondary to those of their husbands and children (True and Guillermo, 1996). Many health problems of minority women thus go unreported and unrecognized, in part because the women do not communicate the problems, but also because providers cannot relate to the women's cultural norms (Bayne-Smith, 1996).

Sexual Orientation

Sexual orientation is known to have an independent effect on health behaviors and receipt of care (Diamant et al., 2000). Although the health needs of lesbians are much the same as those of other women, many of their health-related behaviors and practices are different. Lesbians are less likely than heterosexual women to seek health care and more likely to encounter barriers in access to care and preventive services. For example, many women who have sex only with women believe they do not need Pap tests, and confusion even exists in clinical practices about whether lesbians should be offered cervical smears routinely (Bailey et al., 2000).

The U.S. health care system itself presents access barriers to lesbians. Health insurance and public entitlements routinely do not cover same-sex partners or provide reimbursement for fertility services to lesbians (Dean et al., 2000).

Fear of discrimination and stigma, homophobic attitudes of, and actual negative experiences with, health care professionals all prevent many lesbians from seeking care for themselves or their families, or keep them from disclosing relevant personal information once they enter care. Moreover, the medical educational system does not educate physicians and other providers about the unique aspects of lesbian health (Dean et al., 2000). Care that is

hostile or insensitive may result in inappropriate interventions and can increase alienation and mistrust of public health recommendations (Clark et al., 2001).

Cultural Competence

The U.S. Department of Health and Human Services, Office of Minority Health, has recommended national standards for *cultural competence*, that is, linguistically and culturally appropriate services in health care to ensure equal access to quality health care by diverse populations. The standards include guidelines for providers, policymakers, accreditation and credentialing agencies, purchasers, patients, advocates, and educators (Office of Minority Health, 2000). Cultural competence has been incorporated into various health education and intervention programs at the Connecticut Department of Public Health. Also, the Department's Office of Public Health Workforce Development has an objective to establish a program to recruit and retain a diversified public health work force in sufficient numbers to serve Connecticut's residents.

Many patients lack the reading and comprehension skills helpful for maintaining a healthy lifestyle and to function in the U.S. health care system. These deficits result not only from poverty and low educational attainment but also from differences in language and culture. Because of the inability of patients to read and understand health-related information, infants are being born with birth defects, diseases are being diagnosed at advanced stages, and medications are being taken improperly.

The issue of *health literacy* is considered a priority in Connecticut. By incorporating health literacy into health education programs and interventions, efforts are being made to educate practitioners about how to communicate effectively with their patients, and to identify and educate patients who are at risk of developing health problems because of poor literacy skills.

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4. FEMALE SEXUAL DEVELOPMENT

INTRODUCTION

The process of sexual development begins shortly after conception, and by the fifth week of pregnancy the fetus' nerves, blood vessels, and tissue bundles have formed in patterns that distinguish males and females. These biological changes continue throughout a woman's lifetime. However, a detailed discussion of female sexual development would be beyond the scope of this report. We have chosen to discuss three key development signposts: menarche, fetal growth status at birth (which is an indicator of reproductive health), and menopause. These three measures span about half of a woman's lifetime, and they reflect biologically and socially meaningful events. All three measures provide a consistent pattern across population subgroups. This suggests that the timing of menarche and the growth characteristics of newborns reflect underlying developmental processes that are shared by all women, regardless of race or ethnic background. The consistency of these developmental processes is emphasized since they are the biological stimulus for many common experiences shared by women over the course of their lifetimes.

Finally, we discuss the central role of women's sexual development and reproductive health within the larger context of women's health.

MENARCHE

Menarche refers to a woman's first menstrual period and signals the beginning of her reproductive life. At regular intervals, rising and falling hormone levels encourage the body to release an egg, ready the lining of the uterus for pregnancy, and shed the uterine lining if no pregnancy occurs. Menstruation or menses is the final phase of this monthly three-phase process.

The median age of menarche is 12.6 years, with little difference by race and ethnicity (National Center for Health Statistics, 1997) (Table 4-1). This snapshot of early female sexual development provides a picture of great consistency among various subsets of the female population.

MEDIAN WEIGHT AT BIRTH, BY GESTATIONAL AGE

For a mother, her infant's growth status at birth provides an indirect measure of her own reproductive health. Birth weight is a strong predictor of perinatal mortality and morbidity (Wilcox et al., 1995). However using birth weight alone to measure growth will fail to distinguish infants who are growing slowly and are small for their gestational age (SGA) from those that are growing at a normal rate but are born too early. Charts of birth weight percentiles

TABLE 4-1
FIRST MENSTRUAL PERIOD BY AGE, RACE, AND ETHNICITY
U.S. Females, Aged 15-44, 1995

| | Percent Distribution by Age | | | | | | | Mean |
|---------------------|-----------------------------|-----|------|------|------|------|------|------|
| | <10 | 10 | 11 | 12 | 13 | 14 | 15+ | |
| All races | 2.8 | 4.5 | 13.8 | 27.2 | 27.9 | 12.6 | 11.1 | 12.6 |
| White, non-Hispanic | 2.5 | 3.8 | 13.2 | 27.3 | 29.7 | 12.9 | 10.6 | 12.7 |
| Black, non-Hispanic | 3.9 | 6.4 | 15.5 | 26.0 | 24.6 | 10.2 | 13.5 | 12.5 |
| Hispanic | 4.2 | 6.3 | 16.5 | 28.6 | 21.8 | 12.6 | 10.0 | 12.4 |

Source: National Center for Health Statistics, 1997.

by gestational age can be used to make this distinction. Such charts are used by clinicians to identify high-risk infants who may need special care, and are used in epidemiological studies to identify infants whose birth weight or gestational age is extreme.

In a recent study of 231,706 births to Connecticut women (Roberts et al., 1996), charts of birth weight percentiles by gestational age were developed to make "appropriate population norms available for Connecticut clinicians and researchers." Rather than taking a "problem-focused" perspective by focusing on the extreme SGA babies, the midpoint, or median birth weight values for each gestational age are presented in Figure 4-1. This figure includes only female infants to simplify the illustration of these trends. The close tracking of median birthweight values for black and white mothers reflect the reliability of the underlying biological processes that regulates fetal growth.

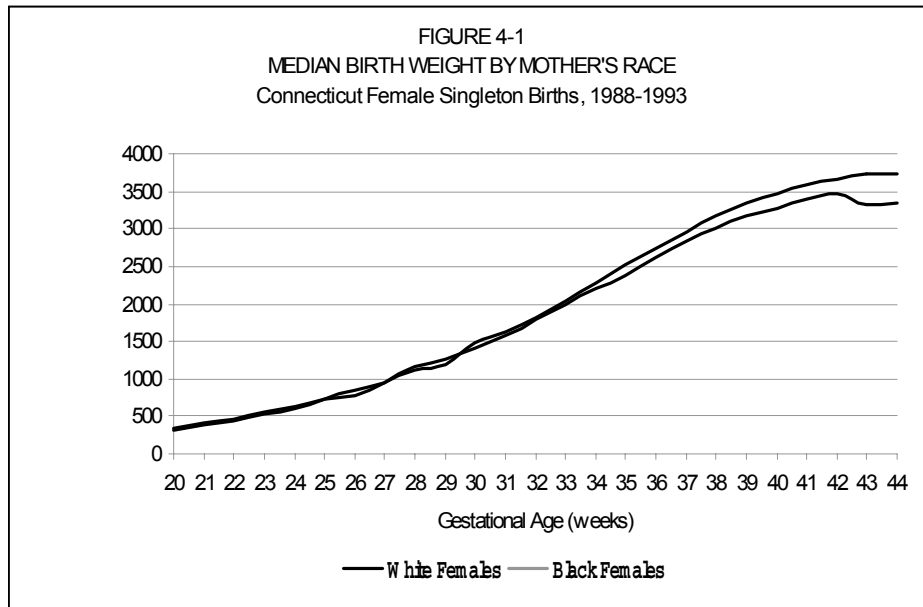
MENOPAUSE

Menopause is the normal biological event in a woman's reproductive cycle marking the end of her natural childbearing years. Many people think of menopause as the long process of

hormonal changes that occurs as a woman's body ages. Medically, the term refers to one specific date - a woman's last menstrual period.

The median age of natural menopause in the United States is 51 years, and the majority of women will enter menopause sometime between the ages of 40 and 55 years. Menopause before age 40 is called premature menopause and occurs in less than 1 percent of women (Endocrinology Society, 2001). Very few women experience menopause after age 60. Induced menopause refers to early menopause caused by a medical intervention that damages the ovaries, such as radiation, certain medications, chemotherapy, or surgery. The abrupt loss of estrogen following surgically induced menopause is likely to cause sudden and severe symptoms that require hormone treatment.

In a recent U.S. study of factors associated with menopause the median age at natural menopause was found to be 51.4 years (Gold et al., 2001). The study sample was described as "one of the largest and most diverse ever studied" to assess factors related to menopause. Statistically significant differences in the median age at menopause were reported for a variety of demographic, lifestyle, and health-related factors. However the magnitude of differences in median



Source: Roberts et al., 1996

age attributable to any single factor were very small. For example, differences in use of oral contraceptives, level of educational attainment, race/ethnicity and smoking were associated with median ages differences no larger than 0.6, 0.7, 0.8, and 0.9 years, respectively. The consistency of median age at menopause for these diverse groups mirrors the consistency reported previously for age at menarche and fetal growth patterns.

At the turn of the 20th century, the average life expectancy of females in the United States was 48 years (American Medical Association, 1997). Menopause and its effect on women's health were not major issues, because the average age at menopause and the average age of death were the same. Today, the life expectancy of women is 79.7 and most women live from one-third to one-half of their lives in postmenopause (North American Menopause Society, 1997). The loss of estrogen's protective qualities after menopause dramatically increases a woman's long-term risk for two serious health problems--coronary heart disease (CHD) and osteoporosis (see Chapters 6 and 17).

Menarche, Pregnancy, Menopause and Risk of Obesity

Recent studies identified three specific high-risk periods during a woman's life that potentially can increase weight gain and the risk of future obesity (North American Association for the Study of Obesity, 2000). They include puberty, especially early menarche, after pregnancy, and after menopause. A study by researchers at Tufts University School of Medicine in Boston suggests that early menarche may be linked to obesity in later life. Because obesity is recognized as a key risk factor for heart disease, diabetes, hypertension, and cancer in women, these studies are especially significant. During the last 20 years, obesity prevalence has nearly doubled among American women (North American Association for the Study of Obesity, 2000). Postmenarchial females were significantly more likely than premenarchial students the same age to have increased their body mass index (Adair and Gordon-Larsen, 1991). Weight retention

following pregnancy and menopause is a health risk factor that can be reduced or eliminated through diet modification and increased physical activity.

FEMALE REPRODUCTIVE SYSTEM AND WOMEN'S HEALTH

The female reproductive system plays a central role in women's health, but it is not the only important dimension. A woman's contact with health care providers is much more likely to be related to her reproductive function and reproductive system than are a man's health care contacts. A recent international study by the World Health Organization (WHO) made a quantitative assessment of the global burden of many diseases (Murray and Lopez, 1996). The study used a measure called "disability adjusted life years (DALYs) lost", and estimated the disease burden resulting from several diseases or injuries. Among women of reproductive age (15-44 years), reproductive conditions account for three of the top ten leading causes of DALYs worldwide. These three conditions were related directly to maternity experiences or to acquired sexually transmitted diseases (STDs). In developed regions like the U.S., disability is not as closely linked to the female reproductive system as it is in developing countries. Nevertheless, having direct access to an obstetrician-gynecologist (ob-gyn) for preventive health care services is important to many women in the U.S.

Obstetrician-gynecologists function as important providers of primary care services for American women. About 41 percent of U.S. women age 18-64 split their care between an ob-gyn specialist and a family practitioner (Kaiser Family Foundation, 1999). Ob-gyns are more likely than other health care providers to provide recommended preventive gynecological services (Table 4-2) like a pelvic exam and Papanicolaou smear and they offer more extensive family planning and STD counseling (Gallop Organization, 1993; U.S. Preventive Services Task Force, 1996). For these reasons, direct access to an ob-gyn for preventive health care services is important.

TABLE 4-2
WOMEN'S REPRODUCTIVE SYSTEM AND
PREVENTIVE HEALTH SERVICES RECOMMENDATIONS

Breast Cancer Screening

All women 19-64 should get a clinical breast exam (CBE) at their annual gyn exam. Routine screening for breast cancer every 1-2 years, with mammography alone or mammography and annual CBE, is recommended for women 50-69.

Cervical Cancer Screening

Papanicolaou (Pap) smears should begin with the onset of sexual activity and be repeated at least every 3 years, following normal Pap tests 3 years in a row.

Contraceptive Counseling

Periodic counseling about effective contraceptive methods is recommended for those at risk for unintended pregnancy

STDs/HIV

All adolescent and adult patients should be advised about risk factors for human immunodeficiency virus (HIV) infection and other sexually transmitted diseases (STDs), and counseled appropriately about effective measures to reduce the risk of infection.

Source: U.S. Preventive Services Task Force, 1996.

In the mid-1990s many managed care plans limited women's access to ob-gyns. In 1994, Maryland was the first state to adopt a policy that regulated access to ob-gyns. By 2000 Connecticut, 37 other states, and the District of Columbia had adopted similar policies to eliminate the requirement for women to obtain a referral before contacting their ob-gyn (Kaiser Family Foundation, 2000). Connecticut women enrolled in HMOs (or covered by other insurance) now have direct access to ob-gyns as a result of state legislation (C.G.S. 38a-530b). This legislation was informed by knowledge of the importance of reproductive health concerns to women in selecting an appropriate health care provider.

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5. PREGNANCY AND BIRTH

INTRODUCTION

Each year, there are about 45,000 births in Connecticut. We expect that most babies will be born healthy, that their mothers will recover quickly after birth, and that they will be able to leave the hospital in about one day. Only two or three generations ago Connecticut residents did not share this common expectation. One century ago, the infant mortality rate was 133 per thousand, a rate almost twenty times higher than the current rate and many Connecticut infants died before their first birthday. Since 1900, declines in the maternal mortality rates have been even more dramatic, dropping by a factor of over 100 (Hoyert, 2000). Numerous social and technical advances, such as improvements in nutrition, clinical medicine, greater access to health care services and in our general quality of life have contributed to this tremendous decline in death rates. Nevertheless, important reproductive health challenges still remain for Connecticut women.

No single indicator exists that adequately describes women's reproductive health. Consequently, this chapter will present several topics spanning women's reproductive health: family planning, infertility, pelvic inflammatory disease (PID), fertility, folic acid intake, adequate prenatal care, alcohol and tobacco use during pregnancy; and prematurity and low birth weight related to multiple births.

PREGNANCY PLANNING

Over a nine-month period within the mother's womb, the fetus experiences a phenomenal amount of growth, comparable to the biological changes that span infancy through middle age. The actions necessary to assure that a woman can provide a healthy environment for an infant's development begin before conception and continue through the final weeks of pregnancy. Planning for a pregnancy can be beneficial in several ways. It can allow the

parents to determine the timing of the pregnancy, to assure that the mother is fit prior to conception, and that both parents recognize the unhealthful behaviors that can pose an unnecessary risk to the fetus or the mother.

The Centers for Disease Control and Prevention has listed family planning as one of the top 10 public health achievements of the twentieth century (Centers for Disease Control and Prevention, 1999a). Improvements in a family's ability to achieve the desired birth spacing and family size has provided substantial economic, social, and health benefits (Centers for Disease Control and Prevention, 1999b). The identification of new scientific information and the distribution of this health information to the public are among the family planning milestones noted by CDC (Centers for Disease Control and Prevention, 1999b). For example, important information such as the actual timing of female ovulation was not known until 1928. Since 1900 average family size in the United States has declined from 3.5 children per family to about 1.7 in 2000.

We live in an era when medicine and public health have made it possible both to prevent unintended pregnancies and to enhance fertility in couples having difficulty conceiving. Preventing unintended pregnancies and enhancing fertility through the use of assisted reproductive technologies (ARTs) are both important avenues for improving reproductive health outcomes.

However, although couples have the opportunity to exercise control over their fertility, yet each year about half of pregnancies in the United States are unintended (National Center for Health Statistics, 1997). These include "miss-timed" and "unwanted" births.

Unintended pregnancies are often associated with poor birth outcomes. Mothers are more likely to obtain prenatal care late or not at all. They are more likely to smoke tobacco or consume alcohol during pregnancy and they are less likely to breast-feed after giving birth

(Brown and Eisenberg, 1995). These consequences are not limited to young teenage women or unmarried mothers. However women in each of these groups are at higher risk for having an unintended pregnancy. The risk associated with unintended pregnancy for teenage mothers also includes the risk that the mother will not complete high school or get married, and their babies are more likely to suffer from low birth weight, neonatal death, or child abuse (Guttmacher, 1994).

The prevention of second or subsequent unintended births to teenage mothers is also important since it places mother and child at increased risk for mental and physical health problems (Klerman, 1998). An analysis of Connecticut 1988-1998 birth records identified 23 percent of mothers under age 20 who had a second or higher order birth.

Concerns about the need to reduce unplanned pregnancies are emphasized in a recent report from the Institute of Medicine (Brown and Eisenberg, 1995), and they are reflected in several of the national *Healthy People 2010* family planning objectives. Strategies focused on reducing unintended pregnancies include encouraging sexual abstinence, especially among adolescents, and the use of birth control (U.S. Department of Health and Human Services, 1999).

Encouraging women to space their pregnancies adequately can also lower their risk of poor pregnancy outcomes. One recent study indicates that females who waited about two years after delivery before conceiving again lowered their risk of adverse outcomes, including low birth weight and premature delivery (Zhu et al., 1999). *Healthy People 2010* Objective 9.2 calls for reducing the proportion of births occurring within 24 months of a previous birth to 6 percent by 2010 (U.S. Department of Health and Human Services, 1999). An analysis of Connecticut 1988-1998 birth data found annual percentages in the range of 9.9 to 10.5 percent, consistent with the 1995 national figure of 11 percent. Achieving this objective by 2010 may be a challenge for family planning efforts within the state since the percentage of Connecticut

women with short interpregnancy intervals has not increased over the last ten years.

IMPAIRED ABILITY TO CONCEIVE

Some couples either have difficulty conceiving or carrying a pregnancy to term. This is called "impaired fecundity," and may be due to medical problems in either the man or the woman (U.S. DHHS 2000). In 1995, 13 percent of married couples with women aged 15-44 years had impaired ability to conceive or maintain a pregnancy. While infertility is usually not a dangerous medical condition, it has significant personal, social, and economic effects. Sexually transmitted diseases (STDs) are a common cause of infertility and they are preventable. Unfortunately, no state-level information is currently available to monitor impaired fecundity in Connecticut. However, some information is available for a condition associated with both infertility in women and STDs, pelvic inflammatory disease.

Pelvic Inflammatory Disease

Pelvic inflammatory disease (PID) is a serious condition that affects the upper female genital tract. The majority of PID cases are secondary to chlamydia or gonorrhea infections, which disrupt natural defenses and allow other organisms to invade the genital tract. PID usually results in chronic pelvic pain, scarring, inflammation of the fallopian tubes, and possible infertility.

The extent of PID is underestimated. Some women may have severe symptoms which require hospitalization, while others have less severe symptoms. In either case, the disease may seriously damage a woman's reproductive organs and result in infertility. The Connecticut PID hospitalization rate for 1993-1997 was 55 per 100,000 women aged 15-44 years, well below the *Healthy People 2000* target of 100. Higher target values were established in 1990 for two high-risk subpopulations: women 15 to 19 years old (110 per 100,000) and black women (150 per 100,000). The Connecticut rate among adolescent women for the same period was 71, also below the year 2000 target. However the

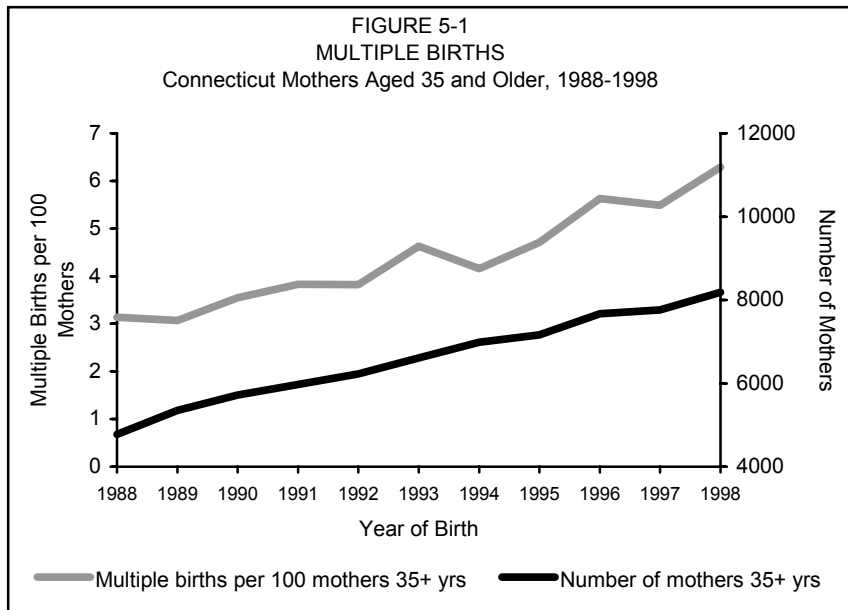
rate for black women, 154.6, was higher than the target value. This finding is consistent with other reports of higher rates of sexually transmitted diseases in Connecticut's black population. As noted in Chapter 20 of this report, black women have higher rates of gonorrhea and chlamydia incidence when compared to the white population.

Assisted Reproductive Technology

New medical treatment options for infertile couples have been successful (Centers for Disease Control and Prevention, 2000). The number of women in Connecticut receiving or making use of ovulation-enhancing fertility treatment, or Assisted Reproductive Technology (ART), such as in vitro fertilization, is unknown.

following New Jersey and Massachusetts, which also had approximately 4 percent.

Multiple gestation births have much higher rates of low birth weight and prematurity than do single deliveries. Based on an analysis of the multiple birth trends in Massachusetts, that state's Department of Public Health concluded that, "the proportion of multiple births is directly responsible for the increase in crude low birth weight rates in Massachusetts from 1989 to 1996 (Cohen et al., 1999). This is also true for Connecticut. Between 1988 and 1998, the risk of having a low birth weight baby increased by 15 percent in Connecticut. About 90 percent of this increase was due to the increase in multiple birth deliveries over the same period (Mueller, 2001a).



Source: Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001.

Nevertheless indirect evidence, based on increasing numbers of multiple births, suggests that this type of treatment is being used extensively.

The overall percentage of Connecticut births that involved twins or higher order multiple deliveries is among the highest in the nation. In 1997 and 1998, 4 percent of all births were multiple gestation deliveries, which placed Connecticut as the third-ranked state, barely

Significant increases in multiple births over the last decade are due to two overlapping trends. More women are delaying childbearing, and they are more likely to have multiple births due to their age (See Figure 5-1). Second, there is an increased use of fertility treatment, which increases the risk of multiple births. For triplets and higher order births, about 20 percent are attributable to older maternal age, 40 percent are attributable to *in vitro* fertilization, and the 40

percent balance may result from infertility treatments designed to induce ovulation (Centers for Disease Control and Prevention, 2000).

There is a general consensus that triplets and higher order multiple births are not a desirable outcome of fertility therapy (Elster, 2000; Faber, 1997). Multiple births are at least three times more likely to be admitted to a neonatal intensive care unit than are singleton births (Callahan et al., 1994) and neonatal deaths are seven times higher in multiple gestation pregnancies than for singleton deliveries (Guyer et al., 1997). The medical challenge that remains is to improve the reliability and success of fertility treatments while minimizing the risks associated with their use.

The Importance of Folic Acid

In 1992, the U.S. Public Health Service published a recommendation that all women of childbearing age should consume 400 micrograms of folic acid daily to prevent two common and serious birth defects, spina bifida and anencephaly. Folic acid is a B-vitamin that can be found in enriched foods and vitamin pills. The incomplete closing of the spine and skull causes these defects. All infants with anencephaly die a short time after birth, while babies born with spina bifida grow to adulthood with varying degrees of disability. CDC has estimated that 50 to 70 percent of these defects could be prevented if women followed the folic acid nutritional recommendations. Since these birth defects occur very early in pregnancy (three to four weeks after conception) folic acid must be consumed *before* a woman knows she is pregnant. Furthermore, because unplanned or unintended, routine consumption of folic acid is needed to provide the benefit.

Unfortunately, many women are still unaware of the importance of folic acid. In 1999, only 40 percent of Connecticut women 18 to 44 years old knew that the reason health experts recommend that women take folic acid was to prevent birth defects. This is slightly better than the 30 percent figure reported by CDC for the U.S. in 1998 (Reeves, 2001).

Overall, 46 percent of Connecticut women reported taking a multivitamin or a folic acid supplement daily. Women 18-24 years old had less understanding of why folic acid is recommended than women 25-44 years old (26 versus 44 percent, $p < 0.05$). They also reported lower daily folic acid consumption (33 versus 50 percent, $p < 0.05$) (Mueller, 2001b).

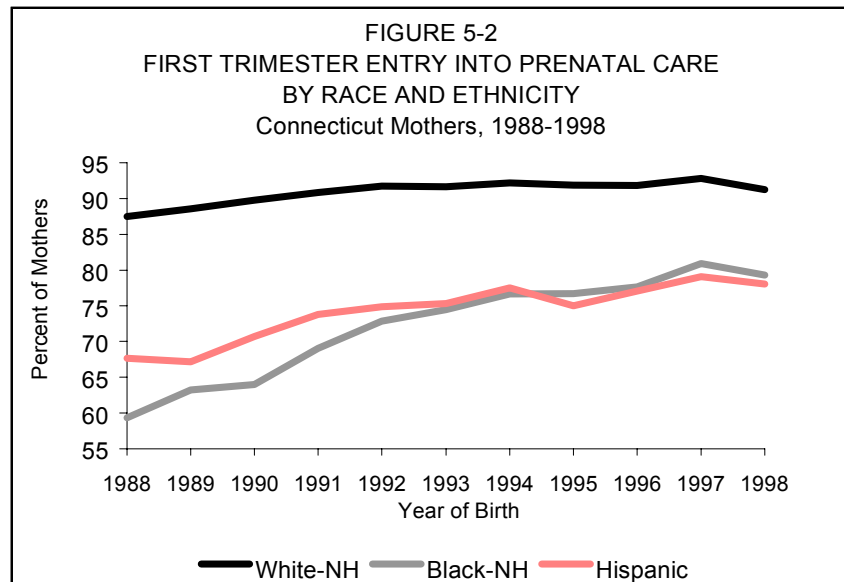
PREGNANCY

The American College of Obstetricians and Gynecologists (ACOG) recommends that pregnant women begin receiving prenatal care during the first trimester of pregnancy. Comprehensive prenatal care is intended to address a variety of modifiable risk factors, including maternal undernutrition, genital tract infections, excessive physical exertion or stress, psychological stress, and adverse health habits such as smoking, alcohol consumption, or use of illicit drugs. Specific studies have documented the effectiveness of intervention in each of these areas. For example, when food supplementation and nutritional education are provided in the course of prenatal care, this can help to increase maternal weight gain and decrease the incidence of low birth weight. Food supplementation and nutritional counseling have been found to increase infant birth weight by 300 grams in two studies (Villar, 1988; Bruce, 1989). Treating pregnant women with documented lower genital tract infections reduces preterm and low birth weight deliveries (Goldenberg, 1996a; Goldenberg, 1996b; Hauth, 1995). Teaching women to recognize and avoid physically strenuous activities can minimize uterine contractions that initiate preterm labor. (Teitleman, 1990; Brett, 1997). Women taught to cope effectively with stressful life situations reduced their participation in adverse health behaviors such as drug, alcohol, or tobacco use. These behavioral changes were associated with reductions in low birth weight (Mamelle, 1997; Rothberg, 1991). In addition, there is evidence that prenatal care services are cost-effective. The Office of Technology Assessment estimated that for every dollar spent on prenatal care at least two dollars are saved in reduced health care costs

for the mother and newborn (Office of Technology Assessment, 1988).

A common strategy for monitoring access to prenatal care is to examine utilization as reported on birth records. The percentage of Connecticut females receiving timely care increased by five percentage points between 1988 and 1998, from 83 percent to 88 percent. Improvements for black non-Hispanic and Hispanic mothers were considerably larger (See Figure 5-2). The annual rates of increase for the white non-Hispanic, black non-Hispanic and Hispanic subgroups were 5 percent, 11 percent, and 6 percent, respectively. Each trend was statistically significant. The rate of increase for the black non-Hispanic population was significantly higher than the corresponding rates for Hispanic and white non-Hispanic mothers (Mueller, 2001a).

Two key behavioral risk factors addressed during prenatal care are the use of alcohol and tobacco during the pregnancy. Maternal smoking is one of the most important preventable causes of low birth weight and other adverse health effects. A recent national study of over three million births documented higher rates of poor pregnancy outcomes (low birth weight, premature delivery, infant mortality) among the infants of mothers who smoked during their pregnancies (Pollack, 2000). The rates of adverse outcomes for smokers ranged from 40 to 120 percent higher than for their non-smoking counterparts. Approximately 11 percent of singleton low birth weight deliveries are attributable to maternal smoking (Pollack, 2000). In addition, increased risks associated with smoking are much higher for twins than for singleton births.



Source: Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001.

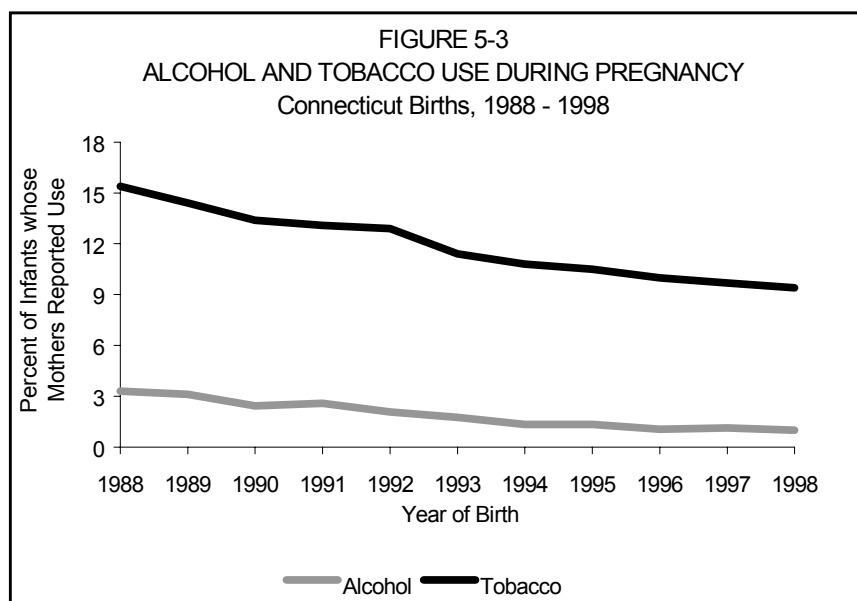
For mothers with singleton births, the percentage receiving early care improved through 1994, when it reached a plateau at 88 percent, just below the *Healthy People 2000* target (90 percent). Fortunately, prenatal care utilization is slightly higher for women with multiple gestation pregnancies, since there is a substantially increased risk associated with these births.

Fortunately, programs aimed at reducing maternal smoking can be cost-effective. Smoking cessation before the end of the first trimester can significantly reduce the risk of delivering a low birth weight or preterm infant (Lightwood et al., 1999). Even cessation programs with modest quit rates of only 15 percent can save between \$3.31 and \$6.58 per dollar spent through reduced utilization of costly

neonatal intensive care and other direct medical care services. (Marks, 1990).

Between 1988 and 1998 the reported rates of tobacco consumption among Connecticut mothers decreased by 39 percent, dropping from 15.4 to 9.4 per 100 (Fig. 5-3). This decline in maternal smoking is significant. While self-reported smoking behavior is thought to be underreported on birth records throughout the United States (Ventura, 2000), reported smoking trends are believed to provide an accurate indication of the true underlying trends.

percent, dropping from 3.3 to 1.0 per 100 births, a statistically significant decline (Fig. 5-3). Caution is warranted in interpreting this decline since alcohol consumption is consistently underreported on birth records (Ventura, 2000). Nevertheless the importance of abstaining from alcohol consumption during pregnancy is clear, and this information needs to be effectively communicated to pregnant women and to women planning to give birth.



Source: Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001.

Heavy and chronic maternal alcohol consumption is an established risk factor for fetal growth retardation (Shu et al., 1995; Mills et al., 1984) and fetal alcohol syndrome (Egeland et al., 1998). Cultural customs and lack of information may lead some women to drink socially during pregnancy, yet even light to moderate drinking presents an unnecessary risk to the fetus. For example, Lundsberg et al. (1997) reported that light to moderate maternal drinking as late as the seventh month of pregnancy is associated with almost a three-fold increase in the risk of delivering a preterm infant. Between 1988 and 1998, the reported rates of maternal alcohol consumption in Connecticut decreased by 70

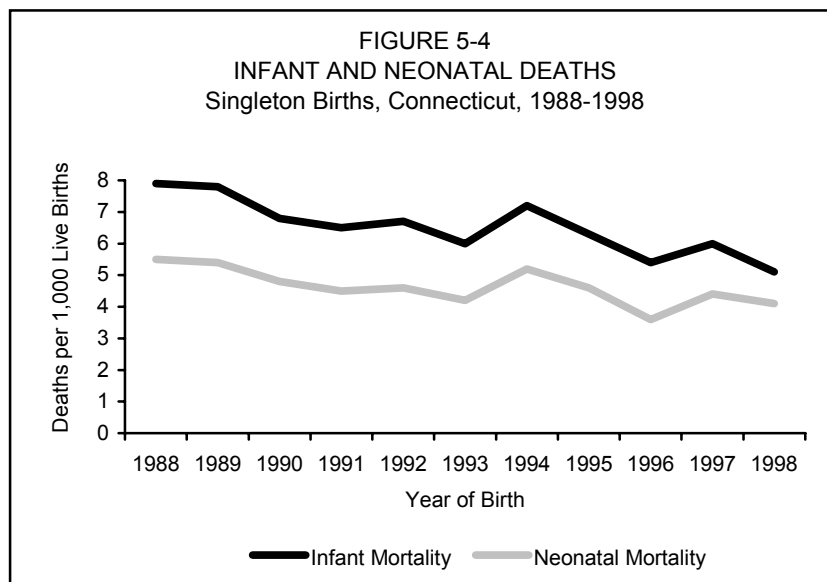
PERINATAL PERIOD

Overall, babies born to Connecticut mothers today are healthier than they have ever been. During the ten-year period from 1988 to 1998, improvements in some pregnancy outcomes have been achieved, though not in all. Declines in infant death rates and perinatal HIV transmission from infected mothers to infants were substantial. In contrast, low birth weight and preterm deliveries have not declined.

Infant Mortality

Improvements in medical care and public health interventions were the driving forces for the infant mortality declines during the second half of this century. Between 1988 and 1998, the Connecticut infant mortality rate declined by 35 percent or 2.8 deaths per 1,000 births (down from 7.9 in 1988 to 5.1 per 1,000 in 1998) (Fig. 5-4). This decline was split evenly between early neonatal deaths (deaths occurring in the first 28 days of life) and later postneonatal deaths (deaths occurring in infants aged 28 to 365 days). Since 1988, the U.S. and Connecticut infant mortality reductions can be attributed to several factors:

- ◆ Between 1989 and 1991, declines were probably due to the use of artificial pulmonary surfactant to prevent and treat respiratory distress syndrome in premature infants (Schoendorf and Kiely, 1997).
- ◆ Between 1991-1997, declines primarily were due to decreases in sudden infant death syndrome (SIDS) and other causes. U.S. sudden infant death syndrome
- ◆ During 1991-1997, U.S. sudden infant death syndrome (SIDS) rates declined by greater than 50 percent, attributed to a national education campaign that recommended that infants be placed to sleep on their backs (Willinger et al., 1998).
- ◆ Reductions in vaccine-preventable diseases (e.g., diphtheria, tetanus, measles, poliomyelitis, and Haemophilus influenzae type b meningitis) had a modest effect. (CDC, 1999c).
- ◆ Advances in prenatal diagnosis of severe central nervous system defects and improved surgical treatment of other structural anomalies (e.g. cardiovascular system anomalies) have helped reduce mortality attributed to these birth defects (CDC, 1998; Montana et al., 1996).



Source: Linked birth infant death file for Connecticut births 1988-1998. Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001.
Notes: Infant deaths include deaths to children less than one year old (365 days). Neonatal deaths include deaths to infants less than 28 days old.

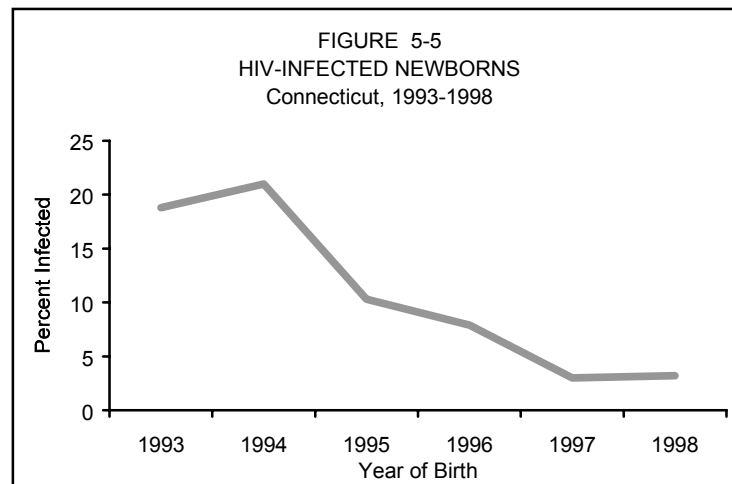
Perinatal transmission of HIV

In August 1994, the USPHS issued guidelines regarding the use of Zidovudine (ZDV) to prevent perinatal HIV transmission (CDC, 1994). Prior to the use of ZDV, an estimated 1,000 to 2,000 HIV infected children were born in the US each year. Vertical transmission of HIV has been reduced from 25 to 8 percent as a result of the successful implementation of these guidelines. In Connecticut, from 1993 to 1998, 503 babies were born to women infected with HIV. The rate of perinatally acquired HIV infection in infants dropped from 19 percent in 1993 to 3 percent in 1998 (Fig. 5-5).

Perinatal HIV infection in Connecticut has been steadily declining since 1994, after the treatment recommendations were first issued. In

poor outcomes. As reported above, however, the increase in poor outcomes is primarily due to the increase in multiple births over this period. Among singleton deliveries, the 1988-1998 rates of low birth weight and prematurely remained stable.

Multiple births account for about 4 percent of all births, but 28 and 21 percent of low birth weight and premature deliveries, respectively. In 1998, Connecticut ranked 27th on percent of low birth weight infants relative to the other states. Even the state ranked first with the lowest rate of low birth weight (Oregon, 5.4 percent low birth weight) did not meet the year 2000 target of 5 percent for this key health indicator. Connecticut and every other state in the nation have yet to achieve this goal.



Source: Connecticut Department of Public Health, Perinatal Infectious Disease Epidemiology, 2001.

Notes: The percent of HIV infected newborns is calculated as a percentage of births to HIV infected mothers.

1995, combination therapy (AZT+ 3TC) and protease inhibitors became available, leading to additional declines in 1997 and 1998.

Prematurity and Low Birth weight

Prematurity, defined as infants less than 37 weeks gestation at delivery, and low birth weight (<2,500 grams) are two key indicators used to identify and track poor birth outcomes. The percent of premature deliveries and the percent low birth weight increased between 1988 and 1998, giving the impression of an increase in

Breastfeeding Benefits

Breastfeeding provides several maternal health benefits, in addition to numerous health benefits for the infant. Mothers who breastfed experience less postpartum bleeding (Chua et al., 1994), earlier return to pre-pregnancy weight, a reduced risk of pregnancy-induced, long-term obesity (Dewey et al., 1993), and a reduced risk of ovarian (Rosenblatt et al., 1993) and premenopausal breast cancer (Newcomb, et al., 1999; Enger et al., 1998). Some studies also show a lower incidence of osteoporosis and hip

fracture after menopause (Melton et al., 1993; Cumming et al., 1993). Women who breastfeed may also experience psychological benefits, such as increased self-confidence and better bonding with their infants (Kuzela et al., 1990; Virden, 1988). Infants who are breastfed experience lower rates of infectious and noninfectious diseases, such as respiratory tract infections, diarrhea and ear infections (Beaudry et al., 1995; Howie et al., 1990; Scariati et al., 1997; Duncan et al., 1993). This is due in part to the immunological agents present in human milk that act against viruses, bacteria, and parasites (Goldman, 1993; Goldman et al., 1990).

In spite of the well-recognized benefits of breastfeeding, the *Healthy People 2000* targets were not met. The objectives were to increase to 75 percent the proportion of mothers who breastfeed their babies in the early postpartum period, and to increase to 50 percent the proportion of mothers who breastfeed until their infant is 5 to 6 months of age. Nationally, in 1998, 64 percent of all mothers breastfed in the early postpartum period and only 29 percent breastfed at 6 months postpartum. Further, disparities exist across racial and ethnic groups, despite significant increases in breastfeeding rates. In particular, only 45 percent of African American mothers breastfed initially, compared with 66 percent of Hispanic mothers and 68 percent of white mothers. In Connecticut, the breastfeeding “initiation rate” during 1998 was 64percent, with only 26 percent of infants breastfeeding until 6 months of age (Mothers Survey, 1998). Among the low income mothers in the state, the 1998 rates were 43 percent and 22 percent respectively, with the lowest rates among black mothers (CDC, 1998).

Healthy People 2010 incorporates the same two objectives and target values for breastfeeding as *Healthy People 2000*. It also includes an objective calling for 25 percent of mothers to breastfeed their babies up to one year of age. Connecticut data are unavailable to evaluate this objective.

The *HHS Blueprint for Action on Breastfeeding*, which was released by the US Surgeon General in October, 2000, calls for the nation to “address these low breastfeeding rates

as a public health challenge and put in place national, culturally appropriate strategies to promote breastfeeding” (USDHHS, 2000). The *Blueprint for Action* outlines specific steps that should be taken by the health care system, the workplace, the family and the community, and identifies several areas for research. If implemented, this plan could substantially increase Connecticut breastfeeding rates and help to reduce the wide racial and ethnic gaps in breastfeeding.

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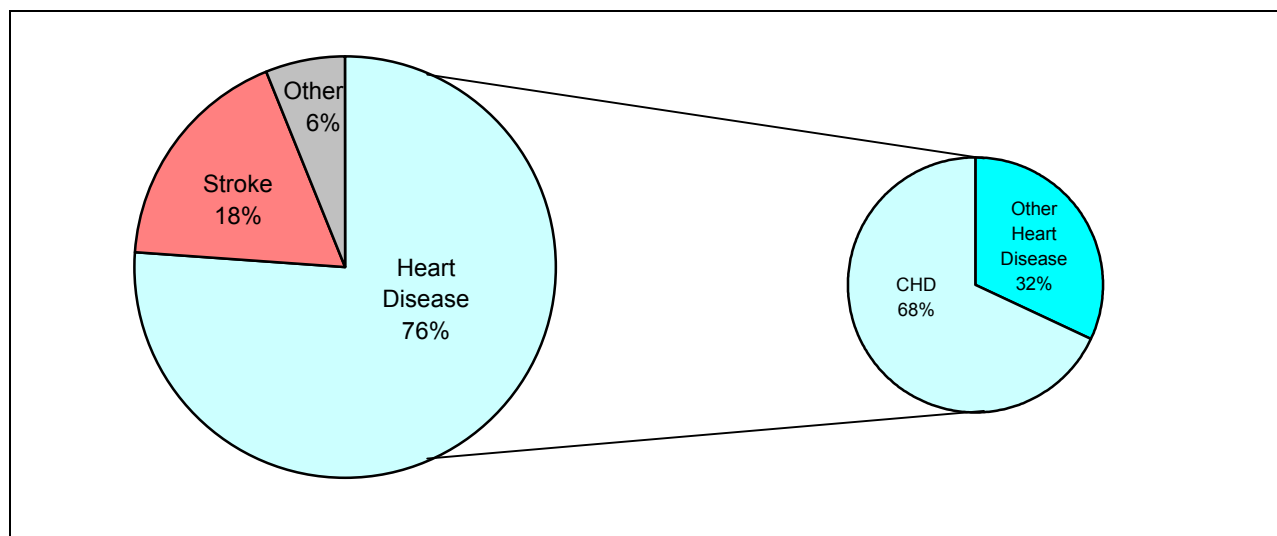
6. CORONARY HEART DISEASE

INTRODUCTION

Cardiovascular disease (CVD) is the leading cause of death and hospitalization for women in Connecticut, accounting for 44 percent of all female deaths in the state. Contrary to the belief that it is a man's disease, approximately 1,000 more women than men die from CVD each year in Connecticut. Despite women's concerns about cancer, nearly twice as many Connecticut women die from CVD than from all cancers combined (21,000 vs. 11,000 for years 1996-1998) (Mueller et al., in preparation). It is estimated that 1 in 2 women will eventually die from CVD compared with 1 in 25 who will eventually die from breast cancer (American Heart Association, 1996).

Cardiovascular disease encompasses heart disease, cerebrovascular disease (stroke) (*Chapter 7*), and other circulatory system disorders (Fig. 6-1). Heart disease accounts for 76 percent of the CVD female deaths and 69 percent of the CVD female hospitalizations in Connecticut. Stroke accounts for 18 percent of CVD female deaths and 18 percent of the CVD female hospitalizations in Connecticut (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001). Heart disease includes congenital and valvular disorders, cardiomyopathies, congestive heart failure, and coronary heart disease (CHD), the latter being the most common form of heart disease.

FIGURE 6-1
CARDIOVASCULAR DISEASE DEATHS
Connecticut Females, 1996-1998



Source: Mueller et al., in preparation.

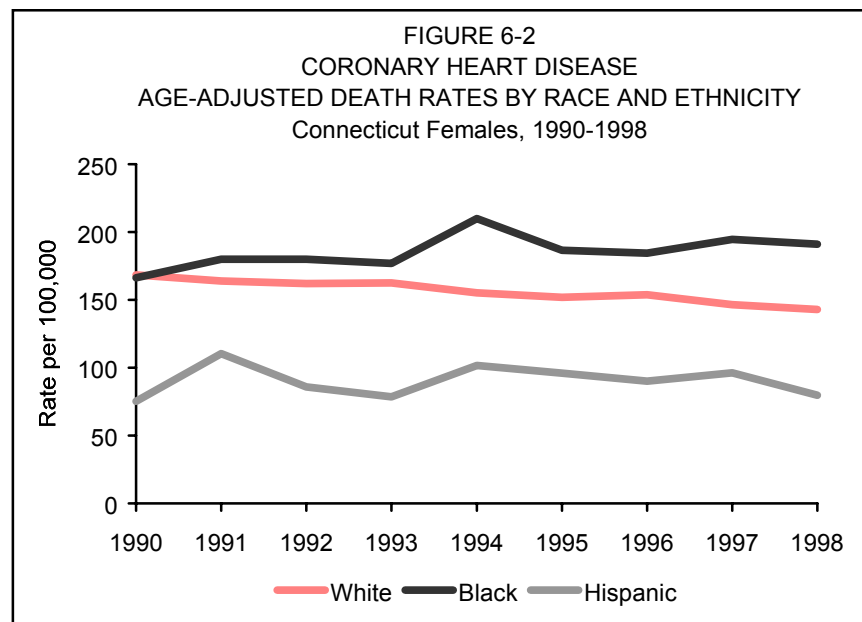
SCOPE OF THE PROBLEM

Coronary heart disease includes acute myocardial infarction (heart attack), angina pectoris (chest pains), and other forms of ischemic heart disease that occur when the flow of blood to the heart is restricted. It is the leading cause of CVD death and hospitalization in Connecticut women. During the three-year period from 1996 to 1998, 10,826 women died from CHD. During 1997, there were 9,813 hospitalizations due to CHD for women, which translates to an age-adjusted rate of 481 per 100,000 females. Hospitalization charges during 1997 exceeded \$172 million (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001).

From 1989 to 1998, there has been a significant downward trend in Connecticut female CHD mortality rates (Fig. 6-2), accounted for by the decrease in CHD deaths among white women. At the same time, the

CHD death rate among black women has remained relatively unchanged. This results in a greater disparity in the mortality rate between black and white women. (Fig. 6-2, Table 6-1). The CHD death rates of Asian and Pacific Islander and Hispanic women were significantly lower than that of white women from 1996 to 1998.

Although the low death rates among Asian and Pacific Islander and Hispanic women may be partly due to under-reporting of these groups on death certificates (Rosenberg, 1999), low Hispanic mortality has been consistently seen in national surveys, and is still largely unexplained (Abraido-Lanza, et al., 1999). Contrary to the disparate black and Hispanic CHD mortality rates, the CHD age-adjusted hospitalization rates from 1993 to 1997 for black, non-Hispanic women and Hispanic women (respectively 557 and 520 per 100,000) were both significantly higher than for white, non-Hispanic women (479 per 100,000).



Source: Mueller et al., in preparation.

Notes: U.S. Census Bureau population estimates used for rate calculations. Rates adjusted to the 2000 U.S. standard million population.

TABLE 6-1
CORONARY HEART DISEASE DEATHS BY RACE AND ETHNICITY
Connecticut Females, 1989-1991 and 1996-1998

| Race/Ethnicity | 1989-1991 | | 1996-1998 | |
|------------------------|------------------|---------------------------------------|------------------|---------------------------------------|
| | Number of Deaths | Age Adjusted Death Rate (per 100,000) | Number of Deaths | Age Adjusted Death Rate (per 100,000) |
| All races | 10,857 | 172.1 | 10,826 | 150.0 * |
| White | 10,318 | 170.1 | 10,224 | 147.5 * |
| African American/Black | 454 | 179.7 | 575 | 189.9 [‡] |
| Asian/Pacific Islander | 10 | † | 22 | 46.8 [‡] |
| Native American | 13 | † | 2 | † |
| Hispanic/Latina | 118 | 97.6 | 157 | 88.2 [‡] |

Source: Mueller et al., in preparation.

* Change in rates from 1989-91 to 1996-98 period is statistically significant ($p < .05$).

† Statistics not calculated for fewer than 15 events.

‡ Rate significantly different from that of whites ($p < .05$)

Notes: U.S. Census Bureau population estimates used for rate calculations. Rates adjusted to the 2000 U.S. standard million population.

RISK FACTORS

Factors that increase the risk of developing CHD, but which cannot be changed, include advancing age and having a family history of heart disease. Other factors associated with an increased CHD risk, but which may be modified by life-style changes and pharmacologic therapy, include hypertension (high blood pressure), hypercholesterolemia (elevated cholesterol), diabetes mellitus, smoking, physical inactivity, poor diet, and obesity. Table 6-2 shows the relative impact of CHD risk factors for women, as well as the percentage of Connecticut women who responded positively to risk factor survey questions from the 1999 Behavioral Risk Factor Surveillance System (BRFSS) survey (Centers for Disease Control and Prevention, 2000).

Lifestyle Factors

Cigarette smoking is a leading cause of CHD in women. Smoking damages the inner arterial wall, raises blood pressure, and promotes intravascular clotting (thrombosis). It is estimated that more than 50 percent of heart attacks among middle-aged women are attributable to cigarette use (Willett et al., 1987). Female smokers have an 80 percent increased risk of developing CHD as compared with female

nonsmokers (Table 6-2). The prevalence of cigarette smoking in Connecticut women was 21 percent in the 1999 BRFSS survey

Dietary factors affect CHD risk. Trans fatty acids have been linked to adverse lipid profiles and increased risk of CHD (Willett et al., 1993). Diets low in saturated fat and high in fruits, vegetables, whole grains, and fiber are associated with a reduced risk of CHD (Willett, 1996). CHD risk is lower in those who drink alcohol moderately (an average of one drink for women per day) than in nondrinkers, but it may raise blood pressure (Fuchs et al., 1995).

Diets high in saturated fat combined with physical inactivity can lead to excess weight and obesity. In addition to being a risk factor for CHD, obesity increases the incidence of other risk factors such as elevated cholesterol levels, high triglyceride levels, and increased likelihood of hypertension and diabetes mellitus. Overweight women are 40 percent more likely to develop CHD than those who are not (Table 6-2). Based upon the 1999 BRFSS data, 27 percent of Connecticut women were considered to be overweight (defined as a body mass index > 27.3).¹

¹ Body mass index is calculated as weight in kilograms divided by height in meters, squared.

Psychosocial factors such as work stress, depression, or loneliness may cause individuals to be at a higher risk of developing heart disease, but their role is not yet clearly defined. That is, psychosocial factors may be interrelated to other health-related behaviors such as smoking, diet, or physical activity, which in turn may increase the risk of coronary heart disease. On the other hand, these factors may be independent and cause direct physiological changes (Hemingway et al., 1999).

Contributing Factors

Hypertension, or high blood pressure, is associated with a 50 percent increase in CHD risk over women without the condition (Table 6-2). Studies indicate a strong association between high levels of both systolic and diastolic blood pressure and risk of CHD in women (Whelton et al., 1996). Of particular concern for women is isolated systolic hypertension which has been associated with a sixfold to tenfold risk of CHD death over 15 years (Bittner, 2000). According to the 1999 BRFSS data, 21 percent of Connecticut women reported ever having been told that they had high blood pressure (defined as >140mm Hg systolic and as >90mm Hg diastolic).

the liver for excretion. A high total cholesterol level (> 240 mg/dL) is associated with a 10 percent increase in CHD risk over women without the condition (Table 6.2), but is a less informative measure since it includes both LDLc and HDLc, which have opposite effects. During 1999, 82 percent of Connecticut female BRFSS respondents reported ever having their cholesterol tested. Of these, 27 percent indicated that they had been told that their cholesterol level was high. Triglycerides are fatty compounds found in LDL and HDL. Their role in CHD risk is unclear, but observational studies suggest they may be a particularly important risk factor in women.

Diabetes mellitus is one of the most important sex-specific risk factors for CHD. It significantly increases CHD risk among women by amplifying the effect of other risk factors. It is associated with a threefold to sevenfold elevation in CHD risk among women compared to nondiabetic women, whereas diabetic men have only a twofold to threefold elevation in risk compared to nondiabetic men. This sex-based difference may be due in part to a more deleterious effect of diabetes on lipids and blood pressure in women (Manson, 1996). Diabetes seems to increase atherosclerosis by changing the

TABLE 6-2
CORONARY HEART DISEASE RISK FACTORS

| Risk Factor | Relative Risk (95% CI) | % CT Women Reporting Presence of Risk Factor |
|------------------------|---------------------------|--|
| Diabetes mellitus | 2.4 (1.9, 3.0) | 4 |
| Smoking | 1.8 (1.5, 2.1) | 21 |
| Hypertension | 1.5 (1.3, 1.8) | 21 |
| Overweight | 1.4 (1.2, 1.6) | 27 |
| High cholesterol level | 1.1 (0.9, 1.2) | 27 |

Sources: Bittner, 1994; Centers for Disease Control and Prevention, 2000.
Note: CI = Confidence interval

Elevated low density lipoprotein (LDL) cholesterol and low high-density lipoprotein (HDL) are risk factors for CHD in women. LDL is the form in which cholesterol is transported to cells for membrane and steroid synthesis. High-density lipoprotein (HDL) helps remove cholesterol from body tissues and transports it to

chemical makeup of some of the substances found in the blood (see Chapter 16, *Diabetes*).

PREVENTION

Prevention of CHD includes preventing the development of CHD, screening for existing risk factors, lifestyle modifications, or pharmacologic intervention. Recommendations include smoking cessation, weight management, increasing physical activity, incorporating a diet low in fat, reducing stress, and diagnosing and treating high blood pressure and high cholesterol levels. Adherence to a healthy lifestyle has been shown to reduce the risk of CHD in women by more than 80 percent (Stampfer, 2000).

Although elimination of smoking may not lead to reversal of atherosclerosis, it has been shown to reduce CHD risk close to that of women who have never smoked (Rich-Edwards et al., 1995).

A healthy diet that is low in fat and rich in fruits and vegetables has been shown to reduce the risk of heart disease by 7 percent to 9 percent. Such a diet works by reducing blood levels of homocysteine, an amino acid that has been linked to an increased risk of CHD (Appel et al., 2000). Dietary interventions, including salt-intake reduction, also play important roles in the reduction of hypertension.

Increased physical activity and loss of excess weight reduce CHD risk directly as well as indirectly. They are important in the control of other CHD risk factors such as hypertension, dyslipidemia (high LDLc, low HDLc), and diabetes. Hypertension and dyslipidemia can also be treated pharmacologically, thereby reducing CHD risk. Clinical trials indicate a 2 percent reduction in CHD rates for each 1 percent decrease in LDL cholesterol (Oberman, 2000).

Because of the increase in heart disease rates for postmenopausal women compared to premenopausal women, loss of estrogen as women age is thought to contribute to a higher risk of heart disease. Epidemiological studies have suggested that hormone replacement therapy (HRT) in postmenopausal women may afford protection from CHD. However, such effect may be confounded due to a "healthy woman selection bias" as documented by the Healthy Women's Study (Matthews et al., 1996). This study suggests that women who are

wealthier and better educated (cardioprotective factors) are more likely to be prescribed HRT and to actually comply with the therapy, which may explain the protection attributed to HRT. Major clinical trials, such as the Women's Health Initiative, are currently underway to quantify the cardioprotective effect of HRT, but final results are not expected until 2006. The American Heart Association's most recent recommendation is *not* to initiate HRT for the sole purpose of primary coronary heart disease prevention, but to take into consideration the treatment's potential benefits and risks related to other diseases and conditions (Mosca et al., 2001).

While the use of HRT in primary prevention is still under investigation, the results of the Heart and Estrogen-progestin Replacement Study (HERS) do *not* support hormone replacement therapy for secondary prevention of CHD in postmenopausal women. Although the study showed an alteration in the lipid profile of postmenopausal women using HRT, there was no significant reduction in death or morbidity among women with established CHD. Therefore, women with existing CHD should not begin HRT, but those already receiving HRT may continue (Hully et al., 1998). The American Heart Association likewise advises against the prescription of HRT for the sole purpose of preventing heart attacks in women who already have CHD (Mosca et al., 2001).

Because of the long-term side effects of HRT, such as an increased risk of endometrial cancer and possibly breast cancer, pharmaceutical companies are developing selective estrogen receptor modulators (SERMs), often referred to as "designer estrogens," as an alternative. Without the negative effects to the breast and uterine tissues, these new drugs may help lower cholesterol and consequently the risk for CHD in postmenopausal women. With respect to CHD, SERMs have been shown to reduce levels of LDL cholesterol and the clotting protein fibrinogen, but to a lesser degree than HRT (Love et al., 1991; Walsh et al., 1998). Ongoing trials will determine the clinical impact of SERMs in women at risk for CHD (Barrett-Connor et al., 1998).

Secondary prevention refers to therapy to reduce mortality and recurrent CHD events in patients with established CHD. It is aimed at both control of risk factors and direct protection of coronary arteries from plaque development and eruption. Thus it involves many of the same factors as primary prevention.

In secondary prevention, aspirin therapy benefits women in both the acute and chronic phases of disease. Yet women are less likely than men to receive aspirin in the secondary prevention setting. Even after adjustment for age and other factors, there appears to be a systematic bias towards men receiving aspirin therapy more often than women. In addition to gender bias, women arriving at the emergency department with acute myocardial infarction may be at greater risk for delayed aspirin therapy because they tend to present with more atypical cardiac symptoms than men, such as neck, back, and jaw pain, nausea, and dizziness (Thoma et al., 2001).

Although beta-blocker therapy after myocardial infarction (heart attack) has been shown to decrease nonfatal myocardial infarction and sudden death by 20 to 30 percent regardless of the patient's sex, it tends to be underused (Yusuf et al., 1985). Clinical trials indicate that thrombolysis after acute myocardial infarction also reduces mortality rates among women and men, but women tend to have higher reinfarction rates than men (Bittner, 2000).

Use of diagnostic and therapeutic revascularization modalities may be related to sex differences in morbidity and mortality. Data from the Myocardial Infarction Triage and Intervention registry suggest that the gap in mortality between the sexes due to acute myocardial infarction is associated with a lower likelihood of women receiving CHD interventions. Women had twice the in-hospital mortality of men and were half as likely to undergo acute catheterization, angioplasty, or coronary bypass surgery (Kudenchuk et al., 1996). However, another study suggested that women were referred for bypass surgery at least as often as men when surgery offered the greatest survival benefits, while men were referred more often for surgery when it offered the least benefit relative to medical treatment (Bickell, 1992).

From 1993 to 1997, a significantly higher proportion of Connecticut women hospitalized with AMI died than Connecticut men hospitalized with AMI (12% vs. 7%, respectively), and women were also significantly less likely to undergo percutaneous transluminal coronary angioplasty (15% vs. 21%) or coronary artery bypass graft operations (8% vs. 11%). Because the onset of CHD tends to occur later in women than in men, the older age of women may be a reason why fewer are referred for angioplasty or coronary artery bypass graft surgery. Results of surgery are usually poorer in older persons. Differences in procedure rates could also be due to differences in severity of coronary occlusion.

Cardiac rehabilitation after myocardial infarction reduces CHD risk. It includes exercise training, risk factor modification, and psychosocial and vocational counseling. Despite its benefits, women are less likely to enroll in these programs than men (7 percent vs. 13 percent) (Thomas et al., 1996).

In summary, there is compelling evidence that comprehensive risk factor interventions in women with CHD extend overall survival, improve quality of life, decrease or postpone the need for more invasive interventional procedures, and reduce the incidence of subsequent heart attacks. Therefore, women need to become more aware of heart disease, its risk factors, and its symptoms so that they can actively participate in risk reduction and disease prevention.

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7. CEREBROVASCULAR DISEASE

SCOPE OF THE PROBLEM

Cerebrovascular disease, more commonly known as stroke, is the third leading cause of death in Connecticut women, accounting for 1 of every 13 deaths during 1996-1998. It is also among the ten leading causes of hospitalization. During 1997 there were 5,343 Connecticut female stroke hospitalizations, which translates to an age-adjusted rate of 247 per 100,000 females, with charges approaching \$70 million (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001).

A stroke occurs when blood flow to the brain is interrupted either by a clogged artery (ischemic stroke) or a blood vessel rupture (hemorrhagic stroke). Approximately 85 percent of all strokes are ischemic. Ischemic strokes are most commonly caused by a buildup of plaque inside the arteries, whereas the most common cause of hemorrhagic stroke is high blood pressure.

Demographics and Trends

More women die from stroke than men (Table 7-1). The main reason for this is simply that, on average, women live longer than men. Half of all female stroke deaths occur in women

over 84 years of age, whereas less than a quarter of all male stroke deaths occur in this age group. Hospitalization and death rates for stroke increase with age (Figure 7-1). The number of deaths in Connecticut females doubles in each successive 5-year age group beginning at ages 65-69.

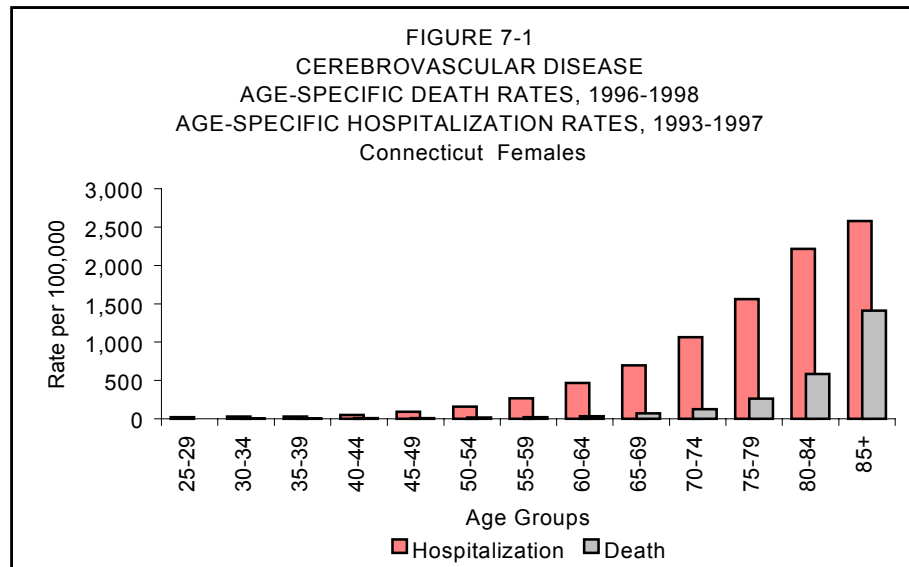
There has not been a significant change in mortality rates over the last decade for cerebrovascular disease in Connecticut females. The mortality rate from 1989 to 1991 was 51 per 100,000 females and was only slightly lower at 50 per 100,000 from 1996 to 1998. However, over the five-year period from 1993 to 1997, the hospitalization rate due to cerebrovascular disease for Connecticut females has significantly increased by 2.6 percent annually from 224 per 100,000 in 1993 to 247 per 100,000 in 1997.

Although mortality rates have not changed over time, rates were significantly higher for black women than for white women (62 vs. 50 per 100,000) (Table 7-2). The 1993 to 1997 age-adjusted hospitalization rate for black, non-Hispanic women was significantly higher than for white, non-Hispanic women (396 vs. 226 per 100,000), whereas the Hispanic rate of 165 per 100,000 was significantly lower than for white, non-Hispanic women.

TABLE 7-1
CEREBROVASCULAR DISEASE DEATHS
Connecticut, 1996-1998

| Age | Number of Deaths | |
|-------------|------------------|---------|
| | Males | Females |
| Under 65 | 3,239 | 1,510 |
| 65-84 | 9,571 | 8,835 |
| 85 and over | 3,937 | 10,241 |
| All ages | 16,747 | 20,586 |

Source: Mueller et al., in preparation.



Source: Mueller et al., in preparation; Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001.

TABLE 7-2
CEREBROVASCULAR DISEASE DEATHS BY RACE AND ETHNICITY
Connecticut Females, 1989-1991 and 1996-1998

| Race/Ethnicity | 1989-1991 | | 1996-1998 | |
|------------------------|------------------|---------------------------------------|------------------|---------------------------------------|
| | Number of Deaths | Age Adjusted Death Rate (per 100,000) | Number of Deaths | Age Adjusted Death Rate (per 100,000) |
| All races | 3,219 | 51.3 | 3,654 | 50.3 |
| White | 3,060 | 50.7 | 3,451 | 49.5 |
| African American/Black | 140 | 57.4 | 182 | 62.1 [†] |
| Asian/Pacific Islander | 5 | † | 17 | 42.2 |
| Native American | 3 | † | 3 | † |
| Hispanic/Latina | 48 | 39.0 | 59 | 32.9 |

Source: Mueller et al., in preparation.

[†] Statistics not calculated for fewer than 15 events.

[‡] Rate significantly different from that of whites ($p < .05$)

Notes: U.S. Census Bureau population estimates used for rate calculations. Rates adjusted to the 2000 U.S. standard million population.

RISK FACTORS

The established modifiable cardiovascular risk factors of smoking, obesity, sedentary lifestyle, high blood pressure, and dyslipidemia also convey risk for stroke (Stamler, 1998; Yusuf, 1998). However, age is the most important factor associated with an increased risk of stroke. The risk of stroke doubles in each successive decade after age 55 (Goldstein, 2001).

Besides age, other nonmodifiable factors that have been identified as markers for risk of stroke include race, ethnicity, and family history. An increased incidence of stroke in families has long been identified. Possible reasons include genetic heritability of stroke risk factors, or a familial exposure to common environmental or lifestyle risks.

Modifiable Risk Factors

Hypertension is the greatest modifiable risk factor for stroke. It is a major risk factor for both ischemic and hemorrhagic stroke. The risk of stroke increases in proportion to both systolic and diastolic blood pressures. For every 7.5 mm Hg increase in diastolic blood pressure, there is a 46 percent increase in stroke risk (MacMahon, 1990). Going from mild hypertension to high blood pressure, the risk may increase 10-fold (Sacco et al., 1997). Twenty-one percent of Connecticut women surveyed in 1999 reported ever having been told that they had high blood pressure.

Active cigarette smoking is also related to stroke risk. In the Nurses' Health Study, smoking increased the relative risk for ischemic stroke in women by a factor of 2.5 and for subarachnoid hemorrhage by 4.9 (Kawachi et al., 1993). Exposure to environmental tobacco smoke may also lead to an increased risk for stroke as a result of increased atherosclerosis. One study found almost a two-fold increase in the risk of stroke among nonsmokers and long-term ex-smokers exposed to environmental tobacco smoke. The risk was significant in both men and women (Bonita, 1999). As previously noted, the prevalence of cigarette smoking in Connecticut women was 21 percent, or one in five of adult women, according to the 1999 BRFSS survey.

Although insulin-dependent diabetics have both an increased susceptibility to atherosclerosis and an increased prevalence of risk factors associated with atherosclerosis, epidemiological studies have confirmed an independent effect of diabetes on ischemic stroke, with an increased relative risk ranging from 1.8 to 6 (Goldstein et al., 2001).

Pre-existing heart disease also increases stroke risk. One notable cardiac abnormality associated with increased stroke risk is atrial fibrillation, an irregular heartbeat whereby blood flow is altered and clots can form that can travel to the brain, block circulation, and cause a stroke. Results from the Framingham Heart Study indicate that the risk of stroke associated with atrial fibrillation in women is 3.2 versus 1.8 in

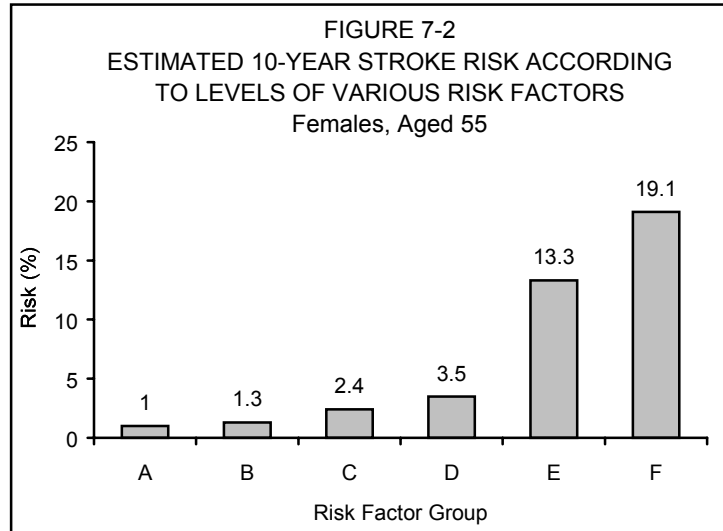
men (Wolf et al., 1991). Other types of cardiac disease that increase the risk of stroke are heart attack (also associated with atrial fibrillation), congestive heart failure, valvular heart disease, and intracardiac congenital defects (Benjamin et al., 1998). Based on the Framingham Heart Study, 11 percent of women will have a stroke within 6 years after a heart attack.

Transient ischemic attacks (TIAs) are a significant independent risk factor for subsequent stroke. A TIA is a temporary interruption of blood flow to part of the brain, sometimes called a mini-stroke. The risk of stroke for women with TIAs is more than fivefold that of those without TIAs (Whisnant, 1996).

While dyslipidemia has traditionally been regarded as a risk factor for coronary heart disease, the link to stroke has been uncertain. Recent studies, however, have shown that the risk of stroke and amount of atherosclerosis of the carotid arteries, which are the principal blood supply to the head and neck, can be reduced with cholesterol-lowering medications (Goldstein et al., 2001).

The epidemiological evidence has been less clear regarding the association of various lifestyle factors, such as obesity, poor diet, and stress, with increased stroke risk. Their effect may be more indirect; for instance, they can increase the risk of high blood pressure, which in turn increases the risk of stroke.

Although individual risk factors increase the probability of stroke, multiple risk factors progressively increase stroke risk (Figure 7-2). Age and sex specific risk profiles have been developed to provide a quantitative determination of the probability of stroke in a person with a combination of risk factors. The risk factor groups, labeled A to F in the table and figure below, represent increasing numbers of risk factors. Figure 7-2 shows that 10-year stroke risk increases from 1 percent to 19 percent as the number of risk factors increase from 0 to 5 for a 55-year old female. For example, a 55-year-old female with diabetes and systolic blood pressure of 115-124 mm Hg (Group C below) has a 2.4 percent probability of having a stroke within the next ten years.



| Risk Factor | Risk Factor Groups | | | | | |
|---------------------------|--------------------|---------|---------|---------|---------|---------|
| | A | B | C | D | E | F |
| Systolic BP | 95-104 | 115-124 | 115-124 | 115-124 | 115-124 | 115-124 |
| Diabetes | No | No | Yes | Yes | Yes | Yes |
| Cigarettes | No | No | No | Yes | Yes | Yes |
| Prior Atrial Fibrillation | No | No | No | No | Yes | Yes |
| Prior CVD | No | No | No | No | No | Yes |

Source: Wolf et al., 1991.

PREVENTION

Primary stroke prevention focuses on the modification of risk factors that can predispose a patient to having a stroke. It is well established that the control of high blood pressure helps to prevent stroke. Clinical trials indicate that hypertension treatment may reduce stroke by 38 percent and fatal strokes by 40 percent (Sacco, 1997). Anti-hypertensives have also been highly effective in preventing stroke in elderly women with isolated systolic hypertension, the most prevalent form of hypertension in persons over 65. A recent women's study indicates that physical activity is associated with a substantial reduction in stroke risk by lowering blood pressure, increasing high-density lipoprotein cholesterol, and by controlling weight (Hu et al., 2000). Consequently, priority should be given to identifying women with hypertension and instituting appropriate therapy such as weight control, exercise, or pharmacologic interventions.

Smoking cessation is associated with a considerable and rapid reduction of the risk of stroke. The Nurses' Health Study indicates that stroke risk in women who have quit smoking dropped to the level of female never-smokers within four years after smoking cessation, and that the benefits of cessation were independent of the age of initiation and the number of cigarettes smoked per day (Kawachi et al., 1993). Avoidance of exposure to environmental tobacco smoke may also play a role in the primary prevention of stroke.

New studies indicate that control of hypertension in diabetics and treatment of high-risk diabetic patients with the angiotensin-converting enzyme (ACE) inhibitor ramipril prevent stroke. A clinical trial of diabetic patients using ramipril showed a 33 percent stroke reduction (Heart Outcomes Prevention Evaluation Study Investigators, 2000).

In patients with nonvalvular atrial fibrillation (AF), judicious use of antithrombotic therapy should be considered to reduce stroke risk. In clinical trials, adjusted-dose warfarin has been shown to reduce stroke by 62 percent whereas aspirin is efficacious, but to a lesser degree, reducing stroke by only 22 percent (Hart et al., 1999). Warfarin is most effective in patients who are at the highest risk for stroke, namely persons older than 75 years of age or persons of any age with multiple risk factors. Warfarin, however increases the likelihood of bleeding, so its use is limited.

After the underlying cause of a TIA is determined, medication, surgery, or lifestyle changes may reduce the risk of having a fatal or disabling stroke. Antiplatelet therapy substantially reduces the risk for stroke in patients with TIAs.

Women who have had a TIA or stroke are at risk for another stroke. Aggressive medical evaluation to determine the specific vascular anatomy and etiology that caused the event should be performed. Subsequent treatment may include the use of antiplatelet agents, such as aspirin, or anticoagulant medication, such as warfarin.

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8. LUNG CANCER

SCOPE OF THE PROBLEM

Lung cancer is the second most common cancer diagnosed in Connecticut women, accounting for 13 percent of all new cases of invasive cancers in 1997. It is the leading cause of cancer deaths in Connecticut women, accounting for nearly one in four cancer deaths in 1998, and is the second leading cause cancer deaths before age 75.

Stage at Diagnosis and Relative Survival Rate

References to national cancer incidence and survival data are to the Surveillance, Epidemiology, and End Results Program (SEER) database (see *Appendix A*). National data, and Connecticut when reported by SEER, are for lung and bronchus cases, which are close to numbers and rates for lung only. For example, the Connecticut 1996 through 1998 annual, age-adjusted (2000 US standard) female cancer mortality rates per 100,000 were 41 for lung and 42 for lung and bronchus.

The relative survival rate for persons with cancer is strongly affected by the extent or “stage” of the disease at the time it is diagnosed. Invasive cancers may be:

1. *local* (confined entirely to the organ of origin),
2. *regional* (extend beyond the limits of the organ of origin directly into surrounding tissues or organs, or into lymph nodes in the region), or
3. *distant* (spread to parts of the body remote from the primary tumor, either by direct extension or by metastasis through the blood or lymphatic systems).

Nationally between 1992 and 1997, 47 percent of lung cancers in women were

diagnosed at the distant stage, where the 5-year relative survival rate was 3 percent; in contrast, only 16 percent were diagnosed at the localized stage, where the 5-year relative survival rate was 52 percent, 23 percent were diagnosed at the regional stage, where the relative survival rate was 23 percent, and 14 percent were unstaged (Ries et al., 2001). Lung cancer survival rates in Connecticut are similar to national rates. Between 1986 and 1993 (the last years for which comparisons were published) the five-year relative survival rates among women were 16 percent nationally and in Connecticut (Ries et al., 1997). Between 1992 and 1997 the U.S. five-year relative survival rate was 16.5 percent.

Incidence, Hospitalizations, and Deaths

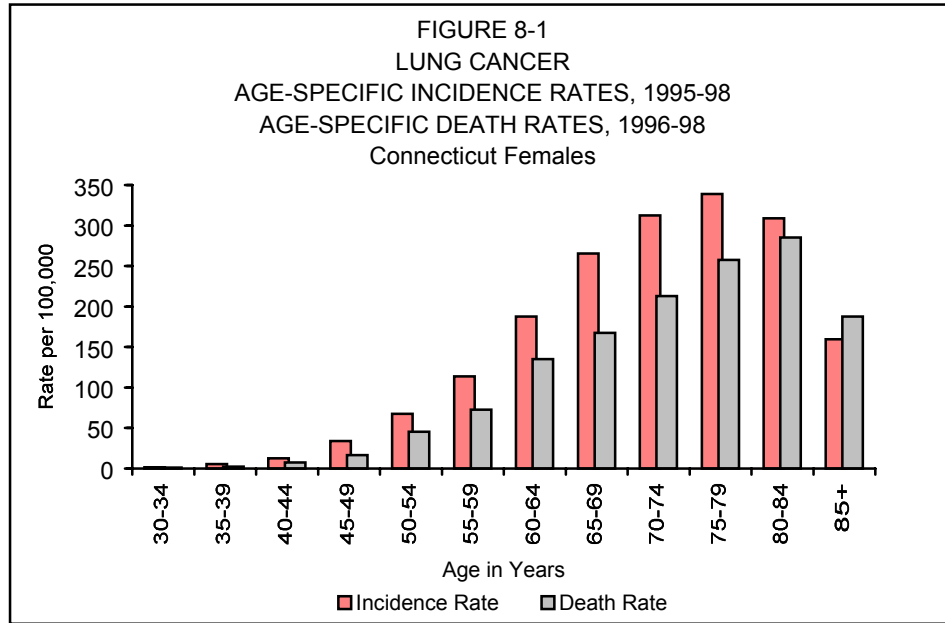
Between 1995 and 1998, 4,528 new cases of lung cancer were diagnosed in Connecticut women, and the annual, age-adjusted incidence rate was 49 cases per 100,000 women (Connecticut Tumor Registry, 2001).

In 1997 there were 979 hospital admissions of Connecticut women for lung cancer, resulting in hospital charges of \$19.6 million. The age-adjusted hospitalization rate was 50 hospitalizations per 100,000 women (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001).

Between 1996 and 1998, 2,503 Connecticut women died of invasive lung cancer, for an annual, age-adjusted rate of 41 deaths per 100,000 (Mueller et al., in preparation).

Age

Rates of both diagnosed cases and deaths from lung cancer in women rise with age (Fig. 8-1). The declines in oldest age may reflect lower initiation rates of female smoking at the time these women were young.



Sources: Connecticut Tumor Registry, 2001; Mueller et al., in preparation.

Race and Ethnicity

Nationally, the lung cancer incidence rate was slightly higher among black women than white women between 1992 and 1998, while rates in Hispanic, American Indian or Alaskan native, Asian or Pacific Islander women were about half the rates in black or white women. The lung cancer mortality rate was slightly higher in white women than black women and much lower in the other ethnic and racial groups (Ries et al., 2001).

Between 1995 and 1998, the annual, age-adjusted lung cancer incidence rates for white, black, and Hispanic females in Connecticut were 60, 46, and 30 per 100,000, respectively. The rates in black and Hispanic women were significantly lower than the rate in white women (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001).

Between 1993 and 1997 the annual, age-adjusted hospital discharge rates for lung cancer for white, black, and Hispanic females were 46,

49, and 18 per 100,000, respectively. The hospital discharge rate in Hispanic women was significantly lower than in white women (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001).

Compared to the rate for white women, the annual, age-adjusted death rate between 1996 and 1998 for black women was 14 percent lower (not statistically significant), and the rate for Hispanic women was 67 percent lower, a significant difference (Table 8-1). Lung cancer was the leading cause of years of potential life lost (i.e. years lost by death prior to age 75) due to cancer among Connecticut white women, but ranked behind breast cancer among black and Hispanic women (Mueller et al., in preparation). These differences partly reflect historically lower smoking rates among black women and Latinas (Anderson and Burns, 2000). Also, incidence and mortality rates for Hispanics are potentially underestimated because ethnicity is not self-reported in the data sources for cancers and deaths.

TABLE 8-1
LUNG CANCER DEATHS BY RACE AND ETHNICITY
Connecticut Females, 1989-1991 and 1996-1998

| Race/Ethnicity | 1989-1991 | | 1996-1998 | |
|------------------------|------------------|---------------------------------------|------------------|---------------------------------------|
| | Number of Deaths | Age Adjusted Death Rate (per 100,000) | Number of Deaths | Age Adjusted Death Rate (per 100,000) |
| All races | 2,109 | 36.4 | 2,503 | 41.2* |
| White | 2,004 | 36.6 | 2,378 | 41.8* |
| African American/Black | 88 | 29.9 | 119 | 35.8 |
| Asian/Pacific Islander | 2 | † | 5 | † |
| Native American | 1 | † | 1 | † |
| Hispanic/Latina | 19 | 13.4‡ | 25 | 13.6‡ |

Source: Mueller et al., in preparation.

* Change in rates from 1989-91 to 1996-98 period is statistically significant ($p < .05$).

† Statistics not calculated for fewer than 15 events.

‡ Rate significantly different from that of whites ($p < .05$).

Notes: U.S. Census Bureau population estimates used for rate calculations. Rates adjusted to the 2000 U.S. standard million population.

Socioeconomic Status

Overall, lung cancer in America is associated with low socioeconomic status (Krieger et al., 1999), which reflects the pattern of smoking. However, smoking rates among Latinas in the United States increase with income, because their smoking is influenced by acculturation, which also increases with income among Latinas.

Geographic Region

Between 1994 and 1998, Connecticut ranked 27th highest among the states and District of Columbia for average annual age-adjusted lung cancer mortality in females (Ries et al., 2001). The ranks of neighboring states were: New Jersey 25, New York 37, Rhode Island 11, Massachusetts 18, Vermont 22, New Hampshire 6, and Maine 5.

TRENDS OVER TIME

In the United States from 1973 to 1998, the incidence rate for lung cancer in women more than doubled, while falling 4.8 percent in men (Ries et al., 2001). These differing patterns, by which lung cancer rates in women approached the higher rates in men, largely reflect the increases in women's smoking rates that

occurred prior to the release of the Surgeon General's report on smoking in 1964, and the smaller decreases in smoking since 1965 among women, compared to men (Holford et al., 1996). Both male and female lung cancer incidence rates declined between 1988 and 1998 in Americans less than 65 years old. From 1991 to 1998, for all ages, the incidence rate declined at an annual rate of 2.4 percent in men but increased 0.2 percent annually in women (Ries et al., 2001), a reflection of the continuing rise in lung cancer among women older than 65 years.

In 1965, age-adjusted female lung cancer mortality rate was lower than colorectal or ovarian cancer mortality, and was one-fourth the rate for breast cancer, but began to rise rapidly. In 1987, female lung cancer mortality passed breast cancer to become the leading cause of cancer deaths among women (Brownson et al., 1998). While the rate of increase slowed after the 1970's, from 1991 to 1998 the 0.9 percent annual increase was significant (Ries et al., 2001). Female lung cancer mortality rates in the United States are not projected to start to decline until 2010.

Lung cancer five-year relative survival rates have changed little in two decades, nor has there been much change in the stage at which the cancer is detected (Brownson et al., 1998; Ries et

al., 2001). This is because there is no practical screening tool for lung cancer.

Connecticut lung cancer rate changes are consistent with the national pattern, including the approximately 20-year lag for females to males in smoking rates (Polednak, 1994). Between 1980 through 1984, and 1995 through 1998, the annual, age-adjusted incidence rate for lung cancer in Connecticut women increased by 48 percent, from 33 to 49 cases per 100,000. During the same time period, the rates in males declined by 11 percent from 85 to 76 per 100,000 (Connecticut Tumor Registry, 2001). The 8 percent increased annual incidence rate in females between 1990 through 1994 and 1995 through 1998 was also significant. Age-adjusted hospitalization rates increased by 15 percent from 1993 to 1997 and age-adjusted death rates rose, significantly, by 1.7 percent per year between 1989 and 1998 (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001).

RISK FACTORS

The principal non-modifiable risk factors for lung cancer are age and genetic factors, governing, for example, the body's ability to break down or excrete carcinogenic chemicals. A great deal of work with biomarkers for disease susceptibility is underway (Au et al., 2001; Forgacs et al., 2001). Modifiable risk factors are discussed below.

Cigarette Smoking

Cigarette smoking directly accounts for about 87 percent of lung cancers, and increases the risk of developing the disease by 5 to 10 times relative to non-smokers (Brownson et al., 1998). Between 1 percent and 6 percent of lung cancers beyond the direct effects are attributed to second-hand smoke.

The prevalence of current adult female smokers was almost unchanged at 21 percent between 1990 and 1999 (Adams, 2000; Centers for Disease Control and Prevention, 2000).

Between 1995 and 1998, however, the mean number of cigarettes smoked per day by regular smokers increased by 14 percent for women (Centers for Disease Control and Prevention, 1999). About one in three female students in Connecticut, grades 9-12, reported current cigarette smoking in 1999, and 13.9 percent said they smoked on 20 or more of the preceding 30 days (Kann et al., 2000).

The percentage of adult females who said in 2000 that they currently smoked or had ever smoked are shown in Table 8-2 for Connecticut and neighboring states. Connecticut was the 6th highest of the 8 states listed for the prevalence of ever smoking, and 7th for current smoking among women. The median prevalence of females that ever smoked and current smokers among 50 states, the District of Columbia, and Puerto Rico were 43 percent and 21 percent, respectively.

Radon Exposure

Radon is an odorless, colorless gas that arises naturally within the ground from the decay of radioactive elements. Decay products of radon can cause DNA mutations, leading to cancer. The relationship between radon and lung cancer is well established in studies of underground miners, but most radon exposure occurs indoors at lower doses. The highest radon levels are found with granite or permeable soils (Melloni et al., 2000). While the exact number of women exposed to harmful levels of radon is not known, in 1994 an estimated 125,000 homes in Connecticut had radon levels at or above the Environmental Protection Agency "action level" of 4 picocuries per liter of air (Bower, 1995).

Radon is estimated to contribute to 10 percent (range 7-25) of lung cancers. It has a synergistic effect on cancer risk when combined with smoking. Perhaps one-third of radon-attributed lung cancer deaths in the United States would be avoided if all homes had radon concentrations below the EPA action level; of these, about 87 percent would be in people who have ever smoked (National Research Council, 1999).

TABLE 8-2
SMOKING RATES
Northeastern States, Females, 2000

| State | Ever Smoked* | | Current Smoker** | |
|---------------|--------------|-------------------------|------------------|-------------------------|
| | % Prevalence | 95% Confidence Interval | % Prevalence | 95% Confidence Interval |
| Connecticut | 45.7 | 43.4, 47.9 | 19.4 | 17.6, 21.2 |
| Maine | 48.9 | 45.7, 52.0 | 23.1 | 20.4, 25.7 |
| Massachusetts | 46.6 | 44.9, 48.3 | 19.7 | 18.4, 21.1 |
| New Hampshire | 51.8 | 48.5, 55.1 | 23.7 | 20.9, 26.6 |
| New Jersey | 39.6 | 37.3, 41.9 | 18.6 | 16.8, 20.3 |
| New York | 44.2 | 41.7, 46.7 | 20.7 | 18.7, 22.6 |
| Rhode Island | 47.5 | 45.0, 49.9 | 23.0 | 21.0, 25.1 |
| Vermont | 46.9 | 44.5, 49.3 | 21.2 | 19.2, 23.2 |

Source: Centers for Disease Control and Prevention, 2001.

*Smoked at least 100 cigarettes in lifetime **Smokes every day or some days

Occupation

Exposure to polycyclic hydrocarbons and inorganic arsenic have been associated with lung cancer in various industries (gas, coal, steel, asphalt, smelters, pesticides). While an estimated 13 percent of all lung cancers have been attributed to these occupational exposures, the overwhelming majority of people at risk have been men.

Diet

Fruits and vegetables are plentiful sources of anti-oxidants, substances that prevent the damage of DNA. The anti-oxidant beta-carotene has been associated with decreased risk of a variety of cancers in observational studies and in animal studies, but has been associated with increased lung cancer risk in a clinical trial with humans. Other promising retinoids, including vitamin A, have likewise failed in clinical trials (Tockman, 2001). It is however, conceivable that fruits and vegetables provide benefits against cancer and that scientists have simply not isolated the particular biochemical sources of benefit. Up to 5 percent of lung cancers may be attributable to high fat diet, low beta-carotene, and other dietary factors (Brownson et al., 1998).

PREVENTION AND RISK REDUCTION

The attributable risk estimates cited above indicate how much lung cancer may be prevented by various strategies. Thus about nine out of ten lung cancers may be preventable through abstinence from tobacco, with the remaining cases being related mainly to occupational exposures and radon.

Reduction of population smoking rates requires awareness of, and response to, the advertising strategies of tobacco manufacturers (Satcher, 2001). Anti-tobacco advertisements can be quite expensive and it is important to document evidence linking them to reduced tobacco use. Using money from the settlement with the tobacco industry, Florida's "truth" media campaign lowered the rate of youth smoking initiation (Sly et al., 2001).

Mitigation of radon involves pipes and fans to transport soil gases from under the foundation of a building and exhaust them above the roof eave. The Connecticut Department of Public Health (DPH) Radon Program distributes federal Environmental Protection Agency publications on designs for buildings to resist radon. The DPH School Testing Program has tested for

radon in 248 Connecticut schools and will test in additional schools.

Increased consumption of fruits and vegetables, along with decreased consumption of fats, may also decrease lung cancer risk.

Screening and Early Detection

Because symptoms of lung cancer typically do not occur until the disease is in advanced stages, early detection is difficult. It has been suggested that C-T (computerized tomography) scans may detect early stage lung tumors, but randomized trials are needed to assess efficiency in reducing mortality. Chest x-ray, sputum analysis, and fiberoptic bronchial examination are commonly used methods of diagnosis, but they are not useful for screening purposes.

TREATMENT

For a general discussion of cancer treatment, see the chapter on colorectal cancer.

Lung resection is the preferred treatment. Less effective treatments are used when definitive surgery is not possible. Radiation and chemotherapy often are used in combination with surgery. Patients with inoperable lung cancer may be treated with irradiation (Humphrey et al., 1995). Regardless of stage, the current prognosis for patients with small cell lung cancer is unsatisfactory; therefore all patients with this type of cancer may be considered for inclusion in clinical trials at the time of diagnosis (National Cancer Institute, 2001).

The National Cancer Institute provides information on types of lung cancer, risk factors, prevention, testing, diagnosis, coping, support, and treatment, for both patients and health care providers (National Cancer Institute, 2001).

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9. BREAST CANCER

SCOPE OF THE PROBLEM

Breast cancer is the most common cancer diagnosed in Connecticut women, accounting for three out of every ten incident cancers. It is second only to lung cancer as a cause of cancer deaths, and is first among cancers for causing premature death. About one out of eight American women (13 percent) will develop breast cancer in their lifetime, and the lifetime risk of dying from breast cancer is about 3 percent, or one out of 31 women (Ries et al., 2001).

Stage at Diagnosis and Relative Survival Rate

Microscopically, malignant breast tumors fall into two general categories: non-invasive (*in situ*), which are confined within the superficial layers or epithelium; and invasive, which have invaded deeper layers and can spread to other tissues or organs. In simple staging systems, there are three stages of invasive breast cancer:

1. *localized* (confined entirely to the breast);
2. *regional* (extending beyond the breast to nearby tissues or lymph nodes); and
3. *distant* (spread to parts of the body distant or remote from the breast).

The prognosis of breast cancer depends on the extent or stage of disease at the time of diagnosis. National cancer incidence and survival rates are reported through the SEER database (see Appendix A). Nationally from 1992 to 1997, 63 percent of invasive breast cancers were diagnosed at the localized stage, where five-year relative survival was 96.4 percent; 28 percent were diagnosed at the regional stage, where survival was 77.7 percent; 6 percent were diagnosed at the distant stage, where survival was 21.1 percent, and 3 percent were unstaged (Ries et al., 2001). Breast cancer survival rates in Connecticut are similar to

national rates. Between 1986 and 1993 (the last years for which comparisons were published), the five-year female breast cancer relative survival rates were 84 percent both nationally and in Connecticut (Ries et al., 1997). Between 1992 and 1997 the national five-year survival rate was 85.5 percent. Unpublished analyses show that the Connecticut survival rate was similar (Connecticut Tumor Registry, 2001b).

In Connecticut in 1997, 19 percent of diagnosed breast cancers were *in situ* (non-invasive), 55 percent were localized, 19 percent were regional, 4 percent were distant, and the stage was unknown for 4 percent (Connecticut Tumor Registry, 1999b). Excluding the *in situ* cases, 68 percent of the remainder were localized, 23 percent regional, 5 percent distant, and 5 percent unstaged.

Incidence, Hospitalizations, and Deaths

Between 1995 and 1998, 10,762 new cases of invasive breast cancer were diagnosed in Connecticut women (annual age-adjusted incidence rate 122 per 100,000).

In 1997 there were 1,588 hospitalizations (age-adjusted hospitalization rate 86.0 per 100,000), resulting in total in-patient hospital charges of \$14.1 million (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001).

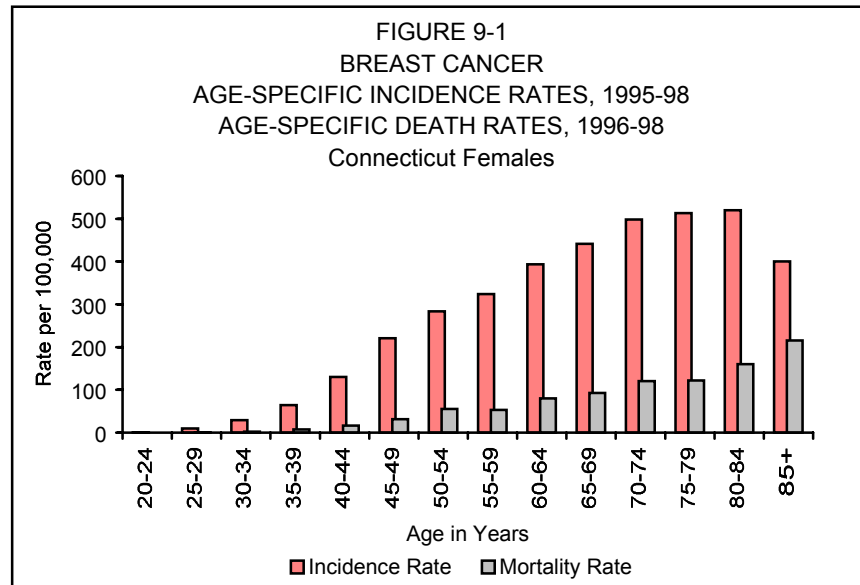
Between 1996 and 1998, 1,796 Connecticut women died of breast cancer (age-adjusted mortality rate 30.0 per 100,000 women per year).

Age

Both the incidence and mortality rates for breast cancer increase with age (Figure 9-1); numbers of hospitalizations also increase with age. Seventy-nine percent of new cases in 1997 and 88 percent of deaths between 1996 and 1998 occurred among women 50 years of age and older. Still, breast cancer is the leading cause of premature cancer deaths among Connecticut females (Connecticut Department of Public

Health, Division of Policy, Planning, and Analysis, 2001), because it is found at a younger average age than lung cancer or coronary heart disease.

rate in whites was significantly higher than in blacks or Hispanics. Hospital discharge rates during 1993 through 1997 for breast cancer were significantly lower for Hispanic, Asian or Pacific



Source: Connecticut Tumor Registry 2001a; Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001.

Notes: U.S. Census Bureau population estimates used for rate calculations. Death rates adjusted to the 2000 U.S. standard million population. Incidence rates adjusted to 1970 U.S. standard million population.

Race and Ethnicity

In Connecticut and nationally, breast cancer incidence rates are lower in black and Hispanic women than in white women. Mortality rates are also lower in Hispanics than whites both in Connecticut and nationally, but higher in blacks than whites. The national breast cancer incidence rates and national breast cancer mortality rates between 1992 and 1998 in Asian/Pacific Islander and American Indian/Alaskan native women were close to Hispanics and lower than white or black women (Ries et al., 2001). However, there are problems estimating rates due to considerable race-ethnicity misclassification for races other than white or black.

Between 1995 and 1998, the annual, age-adjusted breast cancer incidence rates for white, black, and Hispanic females in Connecticut were 147, 119, and 107 per 100,000, respectively; the

Island women compared to white women (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001).

Between 1990 and 1994 in Connecticut, 57 percent of breast cancers were diagnosed at the local stage and 23 percent at the regional stage in white women, compared with 47 percent local and 34 percent regional in black women. *In situ*, distant, and unknown stage cancers comprised similar proportions in blacks and whites (Polednak, 1999). Black women were also diagnosed with breast cancer at higher stage (where five-year survival is lower) nationally, and had lower survival for the same stage as white women (Ries et al., 2001).

Obesity interferes with the ability of mammography to distinguish normal tissue from malignancy. A study in Connecticut found that obesity was associated with later stage at breast cancer diagnosis and with black race. Severe

obesity accounted for 33 percent of the excess risk of late stage at diagnosis in blacks relative to whites (Jones et al., 1997).

Breast cancer results in more years of potential life lost (i.e. years of life lost by death prior to age 75) than any other cancer among black and Hispanic women in Connecticut, and is exceeded only by lung cancer among white women. Connecticut age-adjusted death rates from 1996 to 1998 are significantly higher for black women than for white women (Table 9-1). Age-adjusted death rates for Hispanic females are significantly lower than rates for whites, as are incidence rates. Lower breast cancer incidence and mortality rates among Hispanics than whites in Connecticut are thought to reflect differences in socioeconomic status, age at first pregnancy, and total number of pregnancies (Polednak, 1999).

Socioeconomic Status

Breast cancer is associated in the United States with higher individual social class. The higher incidence rates among women of higher income and social class can be explained, in part, by established risk factors such as reproductive history (see below).

However, when breast cancer incidence was compared with the socioeconomic level of the neighborhood in the San Francisco Bay area, there was a positive relationship only among Latinas (Krieger et al., 1999).

Geographic Region

Death rates from breast cancer in the U.S. are higher in the Northeast and lower in the Southwest. Between 1994 and 1998, Connecticut ranked 12th highest among the states and District of Columbia for annual age-adjusted female breast cancer mortality rate. The ranks of neighboring states were: New Jersey 2, New York 4, Rhode Island 6, Massachusetts 9, Vermont 25, New Hampshire 16, and Maine 21 (Ries et al., 2001).

In a study of six Connecticut cities between 1986 and 1995, federally-defined Medically Underserved Areas were associated with higher percentages of late stage breast cancers at diagnosis (Polednak, 2000a).

TABLE 9-1
BREAST CANCER DEATHS BY RACE AND ETHNICITY
Connecticut Females, 1989-1991 and 1996-1998

| Race/Ethnicity | 1989-91 | | 1996-98 | |
|------------------------|------------------|---------------------------------------|------------------|---------------------------------------|
| | Number of Deaths | Age Adjusted Death Rate (per 100,000) | Number of Deaths | Age Adjusted Death Rate (per 100,000) |
| All races | 1,885 | 33.3 | 1,796 | 30.0* |
| White | 1,777 | 33.3 | 1,653 | 29.7* |
| African American/Black | 92 | 30.1 | 135 | 39.5 [‡] |
| Asian/Pacific Islander | 1 | † | 5 | † |
| Native American | 1 | † | 1 | † |
| Hispanic/Latina | 16 | 9.3 | 33 | 13.2 [‡] |

Source: Mueller et al., in preparation.

* Change in rates from 1989-91 to 1996-98 period is statistically significant (p< .05).

† Statistics not calculated for fewer than 15 events.

‡ Rate significantly different from that of whites (p< .05)

Notes: U.S. Census Bureau population estimates used for rate calculations. Rates adjusted to the 2000 U.S. standard million population.

TRENDS OVER TIME

Rates of cancer incidence and deaths change over time for many reasons, including changes in risk factor levels, changes in screening rates, and treatment improvements. Age at menarche has decreased in the United States and worldwide over the past 100 years, leading to a longer lifetime period of exposure to hormones produced during the menstrual cycle (see the section on risk factors, below).

Female breast cancer incidence in the United States was characterized by an annual 0.7 percent decrease between 1973 and 1980, a significant 3.8 percent annual increase between 1980 and 1987 (a period during which breast cancer screening became more common and consequently more cases were detected), and a 0.5 percent annual increase between 1987 and 1998. From 1992 to 1998 breast cancer incidence increased slightly, largely because of increases among the older age groups, possibly as a result of increased early detection (Howe et al., 2001).

Nationally between 1983 and 1998 the incidence rate of *in situ* breast cancers increased by more than 350 percent and stage 1 cancers by more than 100 percent. The stage 3 and 4 (late stage) breast cancer incidence rate decreased by more than 10 percent, and the incidence rate for all breast cancers increased by more than 46 percent among women over age 50. Similar patterns of change were observed in women under age 50, and in white and black women (Ries et al., 2001). For the period between 1988 and 1995 there was a narrowing nationally of black-white disparities in the proportion of breast cancers detected at late stage, especially in women aged over 65 years (Polednak, 2000b).

In Connecticut the rate of *in situ* breast cancer increased from 3.5 per 100,000 during 1973 through 1975, to 17.5 per 100,000 during 1991 through 1992, with most of the rise coming since 1980. The pattern was seen in both black and white women (Zheng et al., 1997). The Connecticut and national data suggest that increased screening led to the detection of

tumors at earlier stages, and was largely responsible for the rise in breast cancer incidence during the 1980's.

The Connecticut annual, age-adjusted incidence rates for invasive breast cancer increased significantly by 1 percent annually from 1990 through 1998 (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001). In contrast, age-adjusted death rates for breast cancer decreased significantly from 1989 to 1998, with an average annual decline of 1.4 percent.

RISK FACTORS

The principal non-modifiable risk factors for breast cancer are female sex (the incidence is about 100 times as high in women as in men), age, and possession of susceptibility genes, which include, but are not limited to BRCA1 and BRCA2.

BRCA1 exists in several hundred variants and in a variety of populations worldwide, and confers susceptibility to both breast and ovarian cancer. The BRCA1 gene confers 80-90 percent risk of developing breast cancer during one's lifetime, and accounts for 5-7 percent of all breast cancers. Women of Ashkenazic Jewish ancestry have about a 1 percent prevalence of the BRCA1 gene. The BRCA2 gene also shows many variants. It is thought to underlie 14 percent of male breast cancers, but less than 2 percent of female breast cancers. Even in women with BRCA1 or BRCA2 genes, development of cancer is associated with somatic changes after conception such as the loss of the remaining wild-type allele and amplification of oncogenes (Newman et al., 1997).

For a discussion of genetic counseling and testing, see the chapter on ovarian cancer.

Family History

A family history of breast cancer reflects the contribution of both genetic and non-genetic risks factors. A woman's risk of breast cancer is increased by any maternal or paternal family history, and is greater if a first-degree relative

(mother, sister, daughter) has the disease. About 9 percent of breast cancers are attributable to family history, but only a small proportion of these are “hereditary,” i.e., associated with inherited mutations in genes that govern breast cancer susceptibility. Most are related to somatic mutations induced by environmental factors or inadequate repair of DNA.

Medical History

Women who have been diagnosed and treated for breast cancer have a higher risk of developing the disease in the remaining breast tissue. Those with certain non-cancerous changes characterized by epithelial proliferation in their breast tissue are also at greater risk of developing breast cancer.

Reproductive and Hormonal Factors

Reproductive hormones influence breast cancer risk through their effects on cell growth and female breast development; increases in and prolonged exposure to estrogen increase a woman’s lifetime risk of developing breast cancer. Estrogen levels are partly modifiable and partly unmodifiable. The estrogen-related factors that increase risk are: early age at first menstrual period; no children; first pregnancy late in life; late age at menopause; and prolonged hormone replacement therapy.

The initiation of ovulation and menstruation requires adequate childhood growth. Improved nutrition and the control of infectious diseases lower the age of menarche (Henderson et al., 1996).

Pregnancy causes large increases in estrogens, but lowers the baseline levels of several hormones for the rest of life. The first pregnancy also results in breast maturation that lowers long-term breast cancer risk. Thus pregnancy increases breast cancer risk during up to 15 years after delivery, but lowers risk beyond 15 years after delivery, when absolute risk is much higher because women are older (Lambe et al., 1994). The later the first pregnancy, the larger the ratio of the early increased risk period to the later decreased risk period. About 12 percent (range 2-22 percent) of breast cancers

may be attributed to first pregnancy after 30 years of age and negative history of a full-term pregnancy (Brownson et al., 1998).

Progestin when added to estrogen cancels the increased risk of endometrial cancer due to unopposed estrogen, but there is evidence that it adds to the breast cancer risk of estrogen (Schairer et al., 2000). However, cancer risk is not a concern with short term (2-3 year) use of hormones to relieve menopausal symptoms (Willett et al., 2000).

All of the risk factors discussed below are thought to modify breast cancer risk through changes in estrogen levels.

Overweight and Sedentary Lifestyle

The body produces a small quantity of estrogens from the conversion of stored adipose tissue. Before menopause, the contribution of this source of estrogen is negligible in comparison to the ovulatory cycle. After menopause, circulating estrogen level is directly related to fat reserves (Henderson et al., 1996). Between 8 and 16 percent of breast cancer in the United States is attributed to obesity after menopause (Brownson et al., 1998).

Sedentary lifestyle is associated with being overweight. For the prevalences of being overweight and of lack of physical activity among Connecticut women, see the chapters on endometrial and colorectal cancer.

When severe obesity is associated with amenorrhea, or when strenuous physical activity disrupts ovulation, the resulting decreased estrogen exposure lowers breast cancer risk (Henderson et al., 1996).

Alcohol and Tobacco

Daily consumption of two or more alcoholic drinks may be associated with a slight increase in breast cancer risk, presumably through modification of estrogen levels (Henderson et al., 1996). In 1997, less than 2 percent of adult Connecticut women reported having 60 or more drinks in the last month (Centers for Disease Control and Prevention, 1998). Smoking may increase the risk of breast cancer in some women

who are genetically susceptible, but may decrease risk in general through an anti-estrogenic effect (Henderson et al., 1996). For smoking prevalence, see the chapter on lung cancer.

Radiation

Ionizing radiation, including medical x-rays, carries a finite increased risk of cancer. Radiation to the chest is thought to cause approximately 2 percent of breast cancers in America (Brownson et al., 1998).

PREVENTION AND RISK REDUCTION

The ability to prevent breast cancer is presently limited, in part because the deleterious effects of estrogen for breast and uterine cancer are weaker on a population level than its beneficial effects on a number of other conditions, such as cardiovascular disease and osteoporosis. In an individual with genetic risk factors, the risk for breast cancer may be more important than these other diseases.

Risk may be reduced by maintaining proper body weight, limiting alcohol consumption, and exercising regularly. The drug tamoxifen may reduce the risk of breast cancer by 45 percent by competing for the sites on breast cells at which estrogen binds and becomes active (Fisher et al., 1998). Tamoxifen is used to reduce the risk of breast cancer recurrence and in other women with a high risk of breast cancer. However, tamoxifen is associated with increased risk of endometrial cancer and deep vein thrombosis, so the choice to use it should be an individual one and not a general public health recommendation for women at average risk (Rockhill, 2001). Raloxifene (Evista), a drug used to treat osteoporosis in postmenopausal women, dramatically reduced the risk of invasive breast cancer in a recent study (Cummings et al., 1999). The immunological link between pregnancy and breast cancer is being used in research toward a breast cancer vaccine (Janerich, 1994).

Screening and Early Detection

Regular breast self-examination (BSE) and

professional screening (mammography, clinical breast examination) can detect breast cancer at early stages and before physical symptoms develop, when it is most treatable. Monthly BSE beginning at age 20 years is recommended by the American Cancer Society, but its effectiveness has not been proven. However, patients who perform BSE present with smaller lesions (Henderson, 1995).

Annual screening for breast cancer for women age 50 and older has been widely recommended. The American Cancer Society also recommends screening by mammography every one to two years for women age 40 to 49 years. The NIH Consensus Conference on Breast Cancer Screening (January 1997) and the National Cancer Institute did not agree, but the National Cancer Institute changed its guidelines during the ensuing highly publicized controversy (Lawson et al., 2000).

Screening mammography lowers the stage at which breast cancer is detected (Parker et al., 1999). Elderly women and women with low-income are the least likely groups to have mammograms performed. The major barriers to screening appear to be health care providers not recommending it and women not regarding it as necessary (Fink and Mettlin, 1996). When older women undergo regular mammography, they are diagnosed with an earlier stage of breast cancer and they are less likely to die from the disease than women who did not receive mammography (McCarthy et al., 2000).

Authorities do not agree whether screening mammography should be recommended beyond a certain age. Because deaths from other causes rise steeply with age, screening for breast cancer in older women will result in increased diagnosis, but may not affect mortality (National Cancer Institute, 2001). The US Preventive Services Task Force recommends that screening cease at age 70 years, the American College of Physicians discourages screening after age 75, the American Geriatrics Society recommends possible discontinuation at age 85, and the American Cancer Society puts no upper age limit on its screening recommendations. The benefits of screening should be weighed against (1)

complications from additional diagnostic procedures due to inaccurate test results, (2) identification and treatment of clinically unimportant cancers, and (3) psychological distress from screening (Walter and Covinsky, 2001). These concerns are also present for other cancer screening, such as cervical and colorectal cancer.

If all women ages 50-74 followed breast cancer screening guidelines, breast cancer death rates could drop by more than 30 percent (Henderson, 1995). Indeed, the efficacy of screening could be larger than what randomized trials have shown. Data from Sweden covering a 29 year period indicated a 63 percent decline in breast cancer mortality and 50 percent decline in all-cause mortality in women age 40-69 years who were screened, with no change in mortality among women who did not undergo screening (Tabar et al., 2001).

In 1999, 86 percent of Connecticut women age 50 and over reported having a mammogram in the previous 2 years and 83 percent said they had a breast exam in the previous 2 years (Centers for Disease Control and Prevention, 2000). During 1996 through 1997, Connecticut was slightly better than the median for all states in mammography screening (Adams, 2000). Connecticut women of black race, Hispanic ethnicity, lower income, or age 65 and older were less likely to receive screening mammography.

The National Breast and Cervical Cancer Early Detection Program (NBCCEDP) aims to increase breast and cervical cancer screening among uninsured, low-income women. NBCCEDP reaches 12-15 percent of uninsured women aged 50-64 who are eligible for screening services. In 1991, NBCCEDP data indicated that only 57 percent of their mammograms were provided to women age 50 years or more. The 1998 NBCCEDP policy is to provide at least 75 percent of mammograms to women aged 50 years or more who are not eligible to receive Medicare Part B benefits or are unable to pay the premium to enroll in Medicare Part B, and this goal was met during 1997 through 1998. From 1991 to 1998, 92 percent of NBCCEDP clients in whom

breast cancer was diagnosed initiated treatment. The remainder refused, were lost to follow-up, or had an outcome pending (Lawson et al., 2000).

The Connecticut Breast and Cervical Cancer Early Detection Program (CBCCEDP) began screening women in 1995, funded by the Centers for Disease Control and Prevention and the state. Eligible women were age 40 and older, at or below 200 percent of the federal poverty level and who had no health insurance, or age 35-39 with established risk factors as defined by the American Cancer Society. Between 1995 and 2000, 15,502 women, or an estimated 40% of eligible women, had been enrolled. Sixty-five percent of enrolled women were between ages 40 and 59. Follow-up of 2,023 abnormal mammograms and 1,451 abnormal CBEs resulted in the diagnosis of 68 cases of invasive breast cancer, plus 34 cancers *in situ* (Mitchell, 2001). Cancers other than breast or cervical were also detected through the program. Future efforts will include the re-screening of enrolled women (McCooley et al., 1999).

In a review of strategies for increasing women's participation in screening, the following had evidence for effectiveness: letter of invitation, mailed educational material, letter of invitation plus phone call, phone call, and training activities plus direct reminders for the women (Bonfill et al., 2001). The CBCCEDP was successful with a 12-week mass media (television, radio, newspapers) campaign, although the most frequently cited source of referral was medical personnel.

Table 9-2 shows the percentage of women in Connecticut and neighboring states in 2000 who said they never had a mammogram. Among the eight states listed, Connecticut ranked 8th highest for women age 40 and over who never had mammography, and 7th for women 50 and over. The median prevalences of never having a mammogram among 49 states, the District of Columbia, and Puerto Rico were 12 percent and 21 percent, respectively, for women 40 and older, and 50 and older.

TREATMENT

For a general discussion of cancer treatment, see the chapter on colorectal cancer. The type of treatment recommended depends on the stage, type, and size of the breast tumor and

performed on older women, women who lived in higher income communities, and who were uninsured (Ferrante et al., 2000). Same-day discharge patients have reported faster recovery and better psychological adjustment (Margoese and Lasry, 2000). A Canadian model of breast

TABLE 9-2
NEVER HAD MAMMOGRAM
Northeastern States, Females, 2000

| State | Age 40 and Over | | Age 50 and Over | |
|---------------|-----------------|-------------------------|-----------------|-------------------------|
| | % Prevalence | 95% Confidence Interval | % Prevalence | 95% Confidence Interval |
| Connecticut | 7.3 | 5.7, 8.8 | 15.4 | 12.8, 18.0 |
| Maine | 9.3 | 7.2, 11.4 | 17.1 | 13.7, 20.5 |
| Massachusetts | 7.9 | 6.8, 9.0 | 14.0 | 12.2, 15.8 |
| New Hampshire | 9.0 | 6.9, 11.2 | 15.9 | 12.3, 19.5 |
| New Jersey | 14.0 | 11.8, 16.2 | 22.7 | 19.5, 25.8 |
| New York | 9.8 | 7.7, 11.9 | 18.4 | 14.9, 21.8 |
| Rhode Island | 8.1 | 6.4, 9.7 | 13.3 | 11.0, 15.7 |
| Vermont | 12.6 | 10.7, 14.6 | 19.9 | 17.1, 22.8 |

Source: Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion. Behavioral Risk Factor Surveillance System Survey, 2000.

the patient's medical history. Types of treatment, given alone or in combination, include surgery (i.e., either lumpectomy or mastectomy), radiation therapy, chemotherapy with one or more drugs, and hormone therapy (i.e., tamoxifen to block the effects of estrogen on breast cancer cell growth), and monoclonal antibody therapy (i.e., use of Herceptin to block the effect of proteins that promote breast cancer cell growth). Survival of patients treated with lumpectomy plus radiotherapy is equivalent to mastectomy or mastectomy plus adjuvant therapy (Henderson, 1995).

Some mastectomies for early stage breast cancers are performed on an outpatient basis. The procedure is safe and effective without lymph node excision when axillary lymph nodes are not palpable (Galante et al., 1994). A French study found no difference between inpatients and outpatients in postoperative morbidity, except for higher rate of axillary seroma after axillary lymph node dissection among outpatients (Dravet et al., 2000). In a Florida study, outpatient mastectomies were more likely to be

cancer care estimated that 53 percent of costs for the initial treatment of breast cancer in stages I and II were surgical. Under an outpatient/early discharge strategy, surgical costs would comprise only 21 percent of total care cost (Evans et al., 2000). Another study found that in early stage breast cancer, total costs including subsequent medical care, for breast conserving therapy, compared with mastectomy, were higher at six months post-diagnosis, but lower at five years post-diagnosis (Barlow et al., 2001).

The National Cancer Institute provides information on types of breast cancer, risk factors, prevention, testing, diagnosis, coping, support, and treatment, for both patients and health care providers (National Cancer Institute, 2001).

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10. COLORECTAL CANCER

SCOPE OF THE PROBLEM

In Connecticut in 1997, invasive cancer of the colon and rectum (colorectal cancer) was the third leading cause of cancer deaths among women of all ages, behind lung and breast cancers, and the leading cause of cancer deaths among women 85 or more years of age. It was the second most common cancer in Connecticut women in 1997, accounting for 13 percent of the cancers diagnosed.

Stage at Diagnosis and Relative Survival Rates

National cancer incidence and survival rates are reported through SEER. See Appendix A for information about SEER. The relative survival rate from colorectal cancer in Connecticut is similar to the national survival rate. Between 1986 and 1993, the most recent period for which comparisons were published, the 5-year relative survival rate was 61 percent both nationally and in Connecticut (Ries et al., 1997).

Table 10-1 shows the proportion of colon and rectum cancers according to stage at diagnosis, and the relative five-year survival rates nationally between 1992 and 1997. A higher proportion of rectal cancers than colon

cancers were diagnosed at the local stage, reflecting the fact that the entire rectum but only the descending portion of the colon (which adjoins the rectum) are accessible to sigmoidoscopy. Prognosis was strongly related to the stage at diagnosis, with better survival when cancers were detected at the local stage. The staging pattern in Connecticut was very similar (Connecticut Tumor Registry, 1999a).

Incidence, Hospitalizations, and Deaths

Between 1995 and 1998, 4,437 cases of colorectal cancer (annual age-adjusted rate of 42.0 per 100,000) were reported in women (Connecticut Tumor Registry, 2001).

There were 1,100 hospitalizations of Connecticut women for colorectal cancer in 1997, resulting in hospital charges of \$24.4 million. The age-adjusted hospitalization rate was 53.0 per 100,000 females (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001). Between 1992 and 1996, 20 percent of first hospital admissions for colorectal cancer (both genders) were through the emergency room (ER). Admission through the ER was associated with older age, and distant stage at diagnosis (Polednak, 2000).

TABLE 10-1
INVASIVE COLON AND RECTAL CANCERS
PROPORTIONS DIAGNOSED AT EACH STAGE AND SEER RELATIVE SURVIVAL RATES
U.S. Females, 1992-1997

| Staging | Colon Cancer | | Rectum Cancer | |
|----------|--------------|----------|---------------|----------|
| | % of Total | 5-Yr RSR | % of Total | 5-Yr RSR |
| Local | 34 | 90.2 | 44 | 87.6 |
| Regional | 40 | 66.7 | 34 | 57.6 |
| Distant | 21 | 8.9 | 15 | 7.6 |
| Unknown | 6 | 26.1 | 8 | 35.5 |
| Overall | 100 | 60.5 | 100 | 61.5 |

Source: Ries et al., 2001. RSR=relative survival rate.

Between 1996 and 1998 1,169 Connecticut women died of colorectal cancer, and the annual age-adjusted rate was 17.8 deaths per 100,000 population (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001)

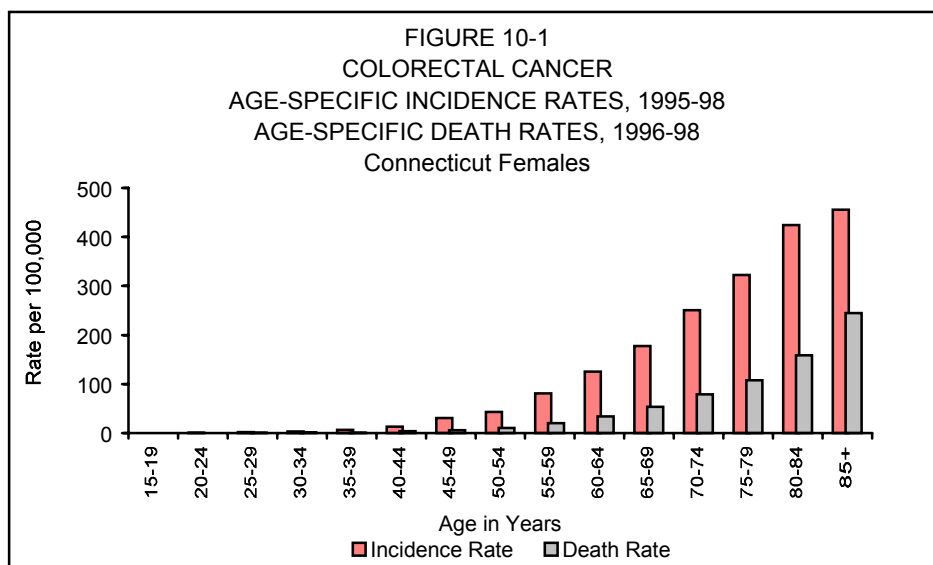
Age

Colorectal cancer incidence and death rates continue to increase through age 85 and older (Fig. 10-1) (Connecticut Tumor Registry, 2001; Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001).

guidelines is similar overall (CDC, 1999; Adams, 2000).

Socioeconomic Status

Colorectal cancer incidence has not been consistently associated with personal or neighborhood measures of affluence (Krieger et al., 1999). Persons with Medicaid or no insurance were diagnosed with colorectal cancer, breast cancer, or melanoma at later stage than those with commercial insurance, in one study (Roetzheim et al., 1999). Colorectal cancer mortality rates were higher among patients with Medicaid, commercial HMO insurance, or no



Sources: Connecticut Tumor Registry, 2001; Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001.

Sex

Between 1994 and 1998, the national mortality rate from colorectal cancer was 31 percent lower for women than for men, while the incidence rate was 28 percent lower (Ries et al., 2001). Between 1996 and 1998 the age-adjusted death rate for Connecticut women was 28 percent lower than that for men (17.8 and 24.8 deaths per 100,000 population, respectively) (Mueller et al., in preparation). Despite this, cancers of the proximal colon, which cannot be found by sigmoidoscopy, and which present at more advanced stage, are more common in females (Gonzalez et al., 2001). Adherence to screening

insurance, compared to commercial fee-for-service (Roetzheim et al., 2000). Persons living in low-income areas were less likely to receive post-treatment colon screening and metastatic disease testing (Elston et al., 2001).

Race and Ethnicity

Nationally between 1992 and 1998, both the incidence and mortality rates of colorectal cancer were higher in blacks and lower in Hispanics, Asians or Pacific Islanders, and American Indians, relative to the white population. (However, there is substantial misclassification in reporting of race and ethnicity other than white or black.) The rectal cancer incidence rate

was slightly higher in white non-Hispanic women than black women (Ries et al., 2001). Black-white differences have been related to the patterns of cancer appearance, discussed here, screening (see the screening section below), and socioeconomic status. Differences in tumor characteristics and socioeconomic factors are each estimated to account for one third of the excess colorectal mortality risk among blacks relative to whites (Marcella and Miller, 2001).

Polyps and cancer appear more often in the right (ascending) colon in blacks than whites (Rex et al., 2000). These are not accessible to

In Connecticut, the 1989 through 1991, and 1996 through 1998 age-adjusted death rates for black women were not significantly different from those for white women, whereas rates for Latina women were significantly lower during both periods (Table 10-2). Between 1995 and 1998, the annual age-adjusted colorectal cancer incidence rates for white, black, and Hispanic females in Connecticut were 53, 59, and 41 per 100,000, respectively. The incidence rate for Latinas was significantly lower than for white women; the same pattern was seen for hospital discharge rates from 1993 to 1997 (Connecticut

TABLE 10-2
 COLORECTAL CANCER DEATHS BY RACE AND ETHNICITY
 Connecticut Females, 1989-1991 and 1996-1998

| Race/Ethnicity | 1989-1991 | | 1996-1998 | |
|------------------------|------------------|---------------------------------------|------------------|---------------------------------------|
| | Number of Deaths | Age Adjusted Death Rate (per 100,000) | Number of Deaths | Age Adjusted Death Rate (per 100,000) |
| All races | 1,325 | 21.8 | 1,169 | 17.8* |
| White | 1,265 | 21.9 | 1,097 | 17.5* |
| African American/Black | 52 | 20.0 | 65 | 19.9 |
| Asian/Pacific Islander | 1 | † | 7 | † |
| Native American | 0 | - | 0 | - |
| Hispanic/Latina | 16 | 13.1‡ | 15 | 7.2‡ |

Source: Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001.
 * Change in rates from 1989-91 to 1996-98 period is statistically significant (p < .05).
 † Statistics not calculated for fewer than 15 events.
 ‡ Rate significantly different from that of whites (p < .05)
 Notes: U.S. Census Bureau population estimates used for rate calculations. Rates adjusted to the 2000 U.S. standard million population.

sigmoidoscopy, and only partly accessible to colonoscopy. In colorectal adenocarcinomas, the MUC1 antigen was expressed equally in whites and blacks, but was associated with a grave prognosis only in whites (Manne et al., 2000). Nationally between 1992 and 1997, a higher percentage of colorectal cancers among black women were diagnosed at distant stage than among white women (24 percent vs. 19 percent), and 5 year relative survival was worse for black women overall, and at each stage except distant (Ries et al., 2001). In Connecticut between 1994 and 1998, the colorectal mortality rate was 15 percent higher among black women than white women, while the incidence rate was 11 percent higher.

Department of Public Health, Division of Policy, Planning, and Analysis, 2001). Low Latina colorectal cancer incidence and mortality rates were also seen in national data. Dietary patterns may underlie these observations.

In a study among patients enrolled in a managed care organization at colorectal cancer diagnosis, who had received treatment, and presumably with equal access to care, whites were more likely than minorities to receive surveillance care after treatment, which is recommended in most guidelines (Elston et al., 2001). In another study, racial differences in stage at diagnosis were not explained by insurance coverage or socioeconomic status (Roetzheim et al., 1999).

Geographic Region

Between 1994 and 1998, Connecticut ranked 34th highest among the states and District of Columbia for average annual age-adjusted colorectal cancer mortality in females (Ries et al., 2001). The ranks of neighboring states were: New Jersey 5, New York 15, Rhode Island 20, Massachusetts 14, Vermont 4, New Hampshire 3, and Maine 6.

TRENDS OVER TIME

The decline in colorectal cancer incidence nationally since 1985 is consistent with the increased use of screening, detection of pre-cancerous polyps, and their removal. The decreases have been most evident for the rectum and the sigmoid colon, that is, the part of the colon accessible to sigmoidoscopy (Cress et al., 2000; Inciardi et al., 2000). During the same time interval, the prevalence of many risk factors (diet, physical activity, weight) either did not change, or changed in the wrong direction (Nelson et al., 1999). Mortality changes reflect changes in incidence some years earlier. Some authorities recommend not separating colon and rectum in studies of trends, as there is substantial misclassification of sigmoid colon versus rectum on death certificates (Schottenfeld and Winawer, 1995).

Between 1992 and 1998, national colorectal cancer incidence declined among all females except American Indian or Alaska natives (Ries et al., 2001). Among all females, colon cancer incidence rates decreased 0.5 percent annually while rectal cancer increased 0.2 percent annually. Between 1980 through 1984, and 1990 through 1994, the average annual age-adjusted incidence rate for colorectal cancer in Connecticut women decreased by 16 percent, but between 1990 through 1994, and 1995 through 1998, there was no further decline (Connecticut Tumor Registry, 2001).

Nationally, between 1992 and 1998, colon cancer mortality rates declined 0.3 percent

annually while rectal cancer mortality rates declined 1.7 percent annually. In Connecticut between 1989-91 and 1996-98, the age-adjusted death rate for colorectal cancer decreased by about 18 percent overall, or 2.8 percent annually, which was significant (Mueller et al., in preparation).

RISK FACTORS

The established non-modifiable risk factors for colorectal cancer are age, a personal or family history of colorectal cancer or polyps, and a medical history of inflammatory bowel disease, such as ulcerative colitis or Crohn's disease (Brownson et al., 1998). Inherited conditions may account for 10-15 percent of colorectal cancers (Schottenfeld and Winawer, 1996).

Diet

A diet high in saturated fat (especially via meat consumption) and low in vegetables and fiber appears to increase the risk of colorectal cancer. An estimated 25-35 percent of colorectal cancer cases may be attributable to inadequate consumption of fruits and vegetables, and an additional 15-25 percent to high fat intake (Brownson et al., 1998).

Table 10-3 shows the percentage of adult females who did not eat five or more servings of fruit and vegetables daily, for Connecticut and neighboring states. Connecticut ranked 5th highest of the eight states listed. The median percentage of women who said that they did not meet this health objective was 73 percent in the fifty states, the District of Columbia, and Puerto Rico (Centers for Disease Control, 2000).

During 1996 and 1997, less than four in ten Connecticut women 18 years of age and older, and only three out of ten Connecticut girls in grades 9-12 reported consuming five or more servings of fruits and vegetables daily (including fruit juice), with blacks and younger adults reporting the lowest consumption (Adams, 2000).

TABLE 10-3
DID NOT EAT 5 SERVINGS OF FRUIT AND VEGETABLES DAILY
Northeastern States, Females, 2000

| State | % Prevalence | 95% Confidence Interval |
|---------------|--------------|-------------------------|
| Connecticut | 67.5 | 65.4, 69.6 |
| Maine | 68.8 | 65.9, 69.7 |
| Massachusetts | 64.6 | 62.9, 66.2 |
| New Hampshire | 68.9 | 65.9, 71.9 |
| New Jersey | 69.4 | 67.2, 71.6 |
| New York | 67.3 | 64.9, 69.7 |
| Rhode Island | 65.1 | 62.7, 67.5 |
| Vermont | 64.9 | 62.6, 67.1 |

Source: Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion. Behavioral Risk Factor Surveillance System Survey, 2000.

Fat intake was calculated from responses to 13 questions on consumption of high fat foods, including hamburger, hot dog, sausage, French fries, potato chips, cookies, doughnuts, pie, and cake. Younger adults (18-24 years of age) were more than four times more likely than those 65 years of age and older to eat more than two servings of fat daily (40.4 percent and 9.9 percent, respectively), but fat consumption was not associated with race or ethnicity (Adams, 2000).

Sedentary Lifestyle

A lack of either recreational or occupational physical activity may slightly increase the risk of colorectal cancer, as may obesity (Murphy et al., 2000). An estimated 32 percent of colorectal cancers in America are caused by sedentary lifestyle (Brownson et al., 1998).

Between 1996 and 1997, 27 percent of Connecticut adult women reported they did not engage in any leisure time physical activity (Adams, 2000). The lack of physical activity was greater in older adults, lower income persons, blacks, and Hispanics.

Table 10-4 shows physical activity among females. Of the eight states listed, Connecticut ranked 5th highest in the prevalence of no leisure time physical activity and in the prevalence of no

regular or sustained physical activity. The median prevalences of these survey responses in 50 states, the District of Columbia, and Puerto Rico were 29 percent and 79 percent, respectively.

PREVENTION AND RISK REDUCTION

Changes in diet and activity may reduce colorectal cancer deaths by more than 80 percent. These changes include increasing consumption of fiber from fruits and vegetables, and decreasing consumption of high-fat foods (Brownson et al., 1998), and increasing physical activity (Pace and Glass, 2000). Supplemental calcium or low-fat dairy foods also appear to be protective (Holt, 1999). Although fruit and vegetable consumption has been associated with reduced risk in many observational studies, these findings are equivocal (Michels et al., 2000).

A variety of dietary interventions can have a positive impact on dietary behaviors (fruit and vegetable intake, dietary fat) associated with cancer risk. These factors include social support, goal setting, small groups, food-related activities, and the incorporation of family elements. Interactions with food, such as cooking and taste testing seemed particularly promising (Efficacy of Interventions to Modify Dietary Behavior Related to Cancer Risk, 2000).

TABLE 10-4
PHYSICAL ACTIVITY
Northeastern States, Females, 2000

| State | No Leisure Time Physical Activity | | No Regular and Sustained Physical Activity* | |
|---------------|-----------------------------------|-------------------------|---|-------------------------|
| | % Prevalence | 95% Confidence Interval | % Prevalence | 95% Confidence Interval |
| Connecticut | 27.7 | 25.6, 29.7 | 77.5 | 75.7, 79.4 |
| Maine | 27.9 | 25.1, 30.8 | 77.7 | 75.0, 80.3 |
| Massachusetts | 26.0 | 24.5, 27.5 | 77.1 | 75.7, 78.5 |
| New Hampshire | 27.7 | 24.5, 30.5 | 77.4 | 74.7, 80.2 |
| New Jersey | 30.6 | 28.4, 32.7 | 79.9 | 77.9, 81.8 |
| New York | 31.6 | 29.2, 34.0 | 80.1 | 78.1, 82.1 |
| Rhode Island | 30.6 | 28.3, 32.9 | 76.1 | 74.1, 78.2 |
| Vermont | 23.3 | 21.3, 25.3 | 74.6 | 72.4, 76.7 |

Source: Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion. Behavioral Risk Factor Surveillance System Survey, 2000.

*Five or more times per week, 30 or more minutes per session

Screening and Early Detection

Screenings for colorectal cancer can reduce deaths by detecting blood, pre-cancerous polyps, or other signs of disease early, when treatment is most effective.

Beginning at age 50, men and women should have one of the following: fecal occult blood testing (FOBT, blood stool testing) and flexible sigmoidoscopy or colonoscopy; or double contrast barium enema. If normal, FOBT should be repeated yearly, sigmoidoscopy every 5 years, colonoscopy every 10 years, and barium enema every 5-10 years. Digital rectal examination should be performed at every examination. Examinations should begin before age 50 in persons with a personal or family history (American Cancer Society, 2000).

Each of the tests has its advocates. FOBT is the only test shown in randomized trials to decrease colorectal mortality. Sigmoidoscopy is supported by retrospective data, and randomized trials are underway. Whole bowel tests are supported by logic, and colonoscopy detects more proximal disease than sigmoidoscopy. Barium enema is less accurate than colonoscopy. The question is not whether colonoscopy detects more neoplasms, but whether the benefit to a few justifies screening the many who will never get cancer even without screening. Unfortunately,

reliable data about complications of colonoscopy are lacking. Allowing people to select the test they prefer may be better than having only one test available (Woolf, 2000).

Retrospective chart review showed that resident physicians at a university medical center adhered poorly to colorectal screening recommendations, with no differences between white and black patients (Borum, 1999). *Put Prevention into Practice* is a national campaign to improve the delivery of clinical preventive services such as screening tests, immunizations, and counseling for behavior change. *The Clinician's Handbook of Preventive Services* provides practical instructions for incorporating prevention into office and clinic routines (10 Steps: Implementation Guide, 1998).

The Medicare program pays for preventive care, including breast, cervical, and colorectal cancer screening. Information about procedures and the frequency of screening which is covered are available at a website (Medicare, 2001). In a study from Michigan among Medicare beneficiaries, sigmoidoscopy screening rates declined dramatically with increasing age. Urban areas and communities with higher education levels had higher screening rates. Among procedures to examine the entire colon, isolated barium enema was used more frequently in African Americans, the elderly, and females.

The combination of barium enema and sigmoidoscopy was used more frequently among females and the newest technology, colonoscopy, among males (McMahon et al., 1999).

Between 1996 and 1997, 26 percent of Connecticut women 50 years of age and older reported having a blood stool test in the last year, and 33 percent had a sigmoidoscopy in the last 5 years; only 13 percent, however, had both tests performed in accordance with guidelines of the American Cancer Society (Adams, 2000). Non-whites were less likely than whites to have ever had either type of test (Adams, 2000). As noted above, more than only these two tests are available, making it difficult to determine actual levels of colorectal screening. Elsewhere, African American males have reported higher colorectal screening prevalence than white males (Baquet and Commiskey, 1999).

Table 10-5 shows colorectal screening among females age 50 and older for Connecticut and neighboring states in 1999. Among the eight listed, Connecticut ranked 7th (1 is worst) for never having sigmoidoscopy. Among 50 states,

the District of Columbia and Puerto Rico, the median was 50.9 percent of women 50 and older never had sigmoidoscopy. Connecticut ranked 6th among the eight states listed for not having used a home blood stool kit in the previous two years. Among 50 states, the District of Columbia and Puerto Rico, the median was 76.7 percent of women 50 and older had not used a home blood stool kit in the past 2 years (Centers for Disease Control, 1999).

TREATMENT

In general, to plan effective cancer treatment, histologic diagnosis must be established by biopsy prior to resection or therapy. To treat effectively, the extent of tumor spread must be established, based upon size, involvement of lymph nodes, and evidence of metastasis. In some cases of lung or colon cancer, the diagnosis and stage are established at the time of definitive resection (Fleming et al., 1995).

TABLE 10-5
 COLORECTAL CANCER SCREENING
 Northeastern States, Females Aged 50 and Older, 1999

| State | Never had Sigmoidoscopy | | Did Not use Home Blood Stool Kit within past 2 Years | |
|---------------|-------------------------|----------------------------|---|----------------------------|
| | % Prevalence | 95% Confidence Interval | % Prevalence | 95% Confidence Interval |
| Connecticut | 49.5 | 42.9, 56.0 | 69.2 | 65.5, 73.0 |
| Maine | 50.9 | 43.7, 58.0 | 64.7 | 60.2, 69.3 |
| Massachusetts | 53.6 | 48.5, 58.6 | 72.1 | 69.3, 74.8 |
| New Hampshire | 50.7 | 41.3, 60.2 | 71.1 | 66.1, 76.1 |
| New Jersey | 55.0 | 49.1, 60.9 | 71.5 | 67.9, 75.1 |
| New York | 52.2 | 45.8, 58.6 | 77.3 | 74.0, 80.5 |
| Rhode Island | 46.2 | 41.3, 51.2 | 73.7 | 70.9, 76.5 |
| Vermont | 50.7 | 45.5, 55.9 | 68.4 | 65.4, 71.4 |

Source: Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion. Behavioral Risk Factor Surveillance System Survey, 1999.

Ninety percent of solid malignant tumors are cured by surgical resection either alone or combined with radiation and chemotherapy. The malignancy is removed along with a margin of normal tissue. In recent years the trend is toward complete but less radical resection in combination with radiation or chemotherapy or both.

The most common treatment for colorectal cancer is surgery, which is especially effective when the cancer is localized. Intraoperative ultrasound is used to look for adjacent or distal spread (Steele, 1995). Radiation, chemotherapy, or both are commonly given after surgery when the cancer has spread beyond the bowel or into lymph nodes. Aggressive chemotherapy or radiotherapy requires support to manage complications of malnutrition, neutropenia, and infection.

Radiation destroys cellular DNA. The therapeutic dose is determined by the ratio of tumor control to complications in the surrounding normal tissue. In general, radiation oncologists attempt to limit serious complications to less than 5 percent. The dose producing this varies with the target organ. As cell kill is a random process, there is no dose of radiation that will guarantee a cure.

Chemotherapeutic drugs destroy cancer cells by interfering with DNA synthesis or function. Side effects may include nausea, vomiting, hair loss, and hematologic suppression. Chemotherapy is sometimes used to shrink tumors prior to resection. Residual micrometastatic tumors following definitive local therapy should be more susceptible to chemotherapy.

Home chemotherapy for colorectal cancer was associated with higher patient satisfaction than outpatient therapy in one study, with no significant difference in the use of health services. Treatment-related toxicity was similar in both study groups, but there were more voluntary withdrawals from treatment (14 percent vs. 2 percent) in the outpatient group (Borras et al., 2001).

Cancer pain is often managed on the lowest level with non-steroidal anti-inflammatory drugs, with the addition of a weak opioid if needed, and the substitution of a strong opioid for the worst pain. The “three step” management of cancer pain has documented efficacy, but randomized controlled trials are sparse. Opioid rotation can improve pain management with decrease in toxic effects. The clinician’s fear of shortening life by increasing opioid dose is usually unfounded, because patients become opioid tolerant during long-term therapy (National Cancer Institute, 2001).

The literature concerning the relative analgesic efficacy of pharmacological and nonpharmacologic therapy, radiation or radionucleotide therapy, physical or psychological treatments, acupuncture, nerve blocks, and neurablation, is difficult to synthesize due to lack of controls and other deficiencies in studies. Leading investigators have called for improving the quality of clinical trials (Management of Cancer Pain, 2001).

The National Cancer Institute provides information on types of colorectal cancer, risk factors, prevention, testing, diagnosis, coping, support, and treatment, for both patients and health care providers (National Cancer Institute, 2001).

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11. CERVICAL CANCER

SCOPE OF THE PROBLEM

Cancer of the uterine cervix (cervical cancer) accounts for about 2 percent of invasive cancers found in Connecticut women each year, and about 1 percent of cancer deaths. It is the second most common cancer among women worldwide, but is the 16th most common cancer in the U.S. (Brownson et al., 1998) and the 13th most common in Connecticut. Although it once was one of the most common causes of death among American women, it now ranks ninth among all cancer deaths (Connecticut Department of Public Health, Breast and Cervical Cancer Early Detection Program, 1999), with improvements due largely to screening, early detection, and treatment.

Stage at Diagnosis and Relative Survival Rate

In Connecticut in 1995, the last year for which data on *in situ* cervical cancers were collected, 87 percent of cervical cancers were *non-invasive*, where the 5-year relative survival rate is nearly 100 percent. Of invasive cervical cancers diagnosed in Connecticut women in 1997, 54 percent were localized, where the 5-year relative survival rate is 92 percent; 28 percent were regional, where the 5-year relative survival rate is 49 percent; and 5 percent were distant, where survival drops to 15 percent (Ries et al., 2001).

Cancer incidence and survival in the United States are reported through the SEER program (described in Appendix A). Between 1986 and 1993, the last years for which comparisons were published, the 5-year relative survival rate for cervical cancer was 69 percent nationally and 67 percent in Connecticut (Ries et al., 1997).

Incidence, Hospitalizations, and Deaths

Between 1995 and 1998, 632 new cases of invasive cervical cancer were diagnosed in Connecticut women, for an annual, age-standardized annual incidence rate of 7.5 cases per 100,000 females (Connecticut Tumor Registry, 2001),

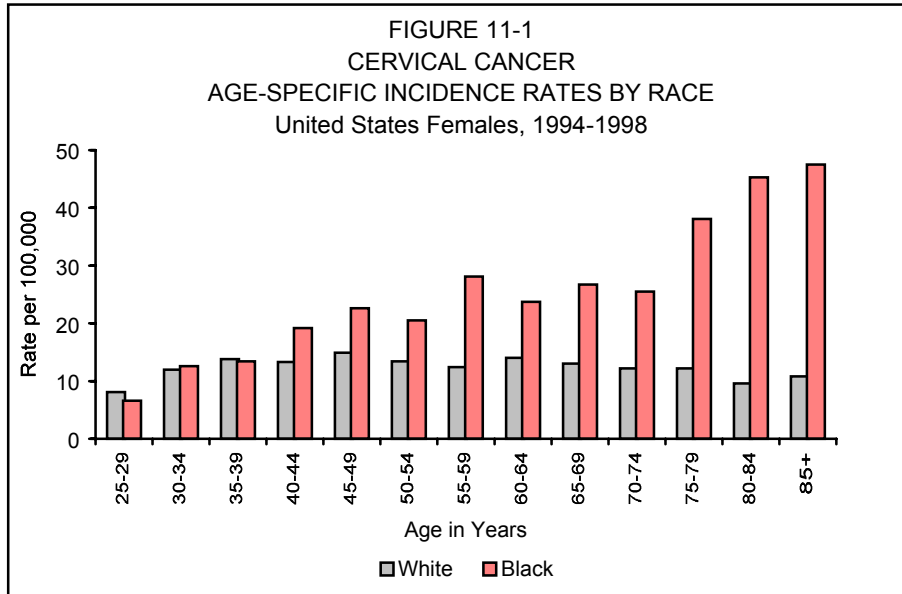
There were 188 hospitalizations of Connecticut women for cervical cancer during 1997, resulting in hospital charges of more than \$2 million. The age-adjusted hospitalization rate was 10.2 hospitalizations per 100,000 females (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001).

Between 1996 and 1998, 127 Connecticut women died of invasive cervical cancer, for an annual, age-adjusted rate of 2.2 deaths per 100,000 females (Mueller et al., in preparation).

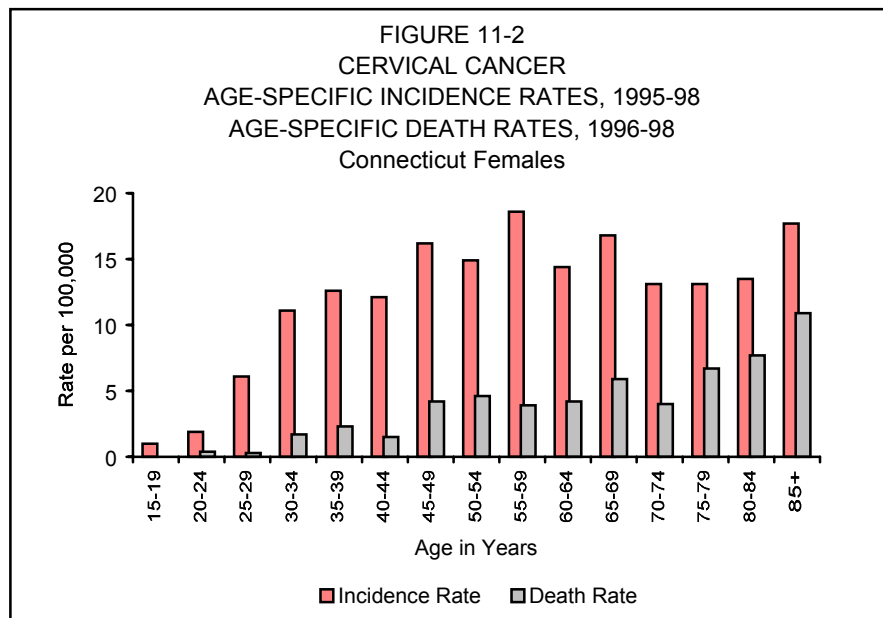
Age

Rates for *in situ* cervical cancer (not shown) peak for both blacks and whites between 20 and 30 years of age (National Cancer Institute, 2001). Incidence rates for invasive cervical cancer rise similarly in blacks and whites until 45-49 years of age, with further increase among blacks but not whites, after age 50 (Ries et al., 2001) (Fig. 11-1).

Nationally, incidence and mortality rates increase with age in both white and black women. Data for Connecticut are shown below in Figure 11-2.



Source: Ries et al., 2001.



Sources: Connecticut Tumor Registry, 2001; Mueller et al., in preparation.

Race and Ethnicity

Both incidence and death rates for cervical cancer are higher among black and Hispanic women than among white women in the Connecticut and national reporting systems. Between 1995 and 1998, the annual, age-adjusted cervical cancer incidence rates for

white, black, and Hispanic females in Connecticut were 8, 15, and 18 per 100,000, respectively. Both black and Hispanic women had significantly higher rates than white women. Black and Hispanic women also had significantly higher hospital discharge rates for cervical cancer between 1993 and 1997, compared with white women (Connecticut Department of Public

Health, Division of Policy, Planning, and Analysis, 2001).

Cervical cancer death rates for black women were significantly higher than those for white women in both the 1989 through 1991, and 1996 through 1998 periods. Between 1996 and 1998 the age-adjusted death rate for black women in Connecticut was 3 times as great as that for white women (Table 11-1) (Mueller et al., in preparation). There were insufficient numbers of cervical cancer deaths among Hispanic, Asian and Pacific Islander, and Native American women to calculate reliable rates among them.

Socioeconomic Status

Low socioeconomic status is associated with a high risk of cervical cancer (Brownson et al., 1998), most likely through patterns of sexual behavior. This has also been seen when using a neighborhood measure of affluence (Krieger et al., 1999). According to the 1990 U.S. Census, Connecticut residents age 18 and over living below the poverty level, by race and ethnicity, were: white (4 percent), Asian (9 percent), black (16 percent), and Hispanic (21 percent). Figures for Native Americans in Connecticut were not

TABLE 11-1
CERVICAL CANCER DEATHS BY RACE AND ETHNICITY
Connecticut Females, 1989-1991 and 1996-1998

| Race/Ethnicity | 1989-91 | | 1996-98 | |
|------------------------|------------------|---------------------------------------|------------------|---------------------------------------|
| | Number of Deaths | Age Adjusted Death Rate (per 100,000) | Number of Deaths | Age Adjusted Death Rate (per 100,000) |
| All races | 150 | 2.8 | 127 | 2.2 |
| White | 126 | 2.5 | 101 | 1.9 |
| African American/Black | 22 | 6.6 | 22 | 5.8 [‡] |
| Asian/Pacific Islander | 1 | † | 2 | † |
| Native American | 0 | † | 2 | † |
| Hispanic/Latina | 7 | † | 11 | † |

Source: Mueller et al., in preparation.

† Statistics not calculated for fewer than 15 events.

‡ Rate significantly different from that of whites (p < .05)

Notes: U.S. Census Bureau population estimates used for rate calculations. Rates adjusted to the 2000 U.S. standard million population.

Except for the black and white races, there is considerable race and ethnicity misclassification in the reporting of cancer incidence and mortality. In SEER national data, which do not include statistical tests of race-ethnicity differences, the annual cervical cancer rates were 10.3 (incidence) and 2.7 (mortality) per 100,000 in American women from Asia and Pacific Islands, compared with 6.9 and 2.7 in white non-Hispanic Americans for 1992-98. Incidence and mortality rates were 6.4 and 2.9 in American Indian/Alaska native women (Ries et al., 2001).

reported (Miller et al., 1996).

In a study of six Connecticut cities between 1986 and 1995, federally-defined Medically Underserved Areas (MUAs) were associated with higher percentages of late stage cervical cancers at diagnosis (Polednak, 2000a). MUAs were defined by the ratio of primary care physicians to the population, the infant mortality rate, and the proportion of the population that is elderly and poor (Wright et al., 1996).

Geographic Region

Death rates for invasive cervical cancer in the U.S. generally are higher in the Southeast and lower in the West and Midwest (Brownson et al., 1998). Between 1994 and 1998, Connecticut

ranked 45th highest among the states and District of Columbia for annual age-adjusted cervical cancer mortality rate (Ries et al., 2001). The ranks of neighboring states were: New Jersey 23, New York 24, Rhode Island 31, Massachusetts 42, Vermont 9, New Hampshire 35, and Maine 12.

TRENDS OVER TIME

Between 1973 and 1998 cervical cancer incidence and mortality rates have both declined nationally, in both white and black, and younger and older women. The decline in mortality rates were slower since 1980 in each of the groups, compared to the 1970's. Between 1992 and 1998 the annual decrease in the cervical cancer incidence rate was 2.1 percent overall and the decline in mortality rate 2.3 percent, with larger survival improvements among blacks, Hispanics, and American Indians (Ries et al., 2001).

In 1935, cervical cancer incidence rates in Connecticut stood at about three times their present level, then declined sharply for the next fifty years (Polednak, 1994). In the past decade no significant changes have been observed (Connecticut Tumor Registry, 2001; Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001).

Between 1989 through 1991, and 1996 through 1998, the age-adjusted death rate for invasive cervical cancer in Connecticut women decreased from 2.8 to 2.2 per 100,000, which was not a statistically significant change (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001).

Between 1988 and 1995 in the SEER national program, there was a narrowing of black-white disparities in the proportion of cervical cancers detected at late stage, especially in women aged over 65 years (Polednak, 2000b).

RISK FACTORS

Human Papillomavirus

Human papillomavirus (HPV), which is transmitted through sexual contact, is believed to be the leading cause of cervical cancer. The relative risk for developing cervical cancer in women with HPV is greater than 10-fold, while the absolute risk is greater than 30 percent (Schiffman et al., 1996). The population-attributable risk of a history of sexually-transmitted diseases, including HPV, is quite imprecise. Brownson and colleagues (1998) cited an attributable risk of 5 percent, with a range of 1-50 percent. More recently (1999), Walboomers and colleagues, after reanalyzing specimens using three different polymerase chain reaction assays, estimated that HPV is present in 99.7 percent of cervical carcinomas worldwide.

Sexual Behavior

An estimated 38 percent of cases of cervical cancer are attributed to having multiple sex partners, and early age at first intercourse (less than 18 years) accounts for another 25 percent (Brownson et al., 1998). Both of these relate to the risk of and time of HPV infection. The relative risk of cervical cancer is 16 times as high in women who had first intercourse prior to age 16, compared with first intercourse after age 19, while the relative risk associated with having more than four sexual partners is 3.6 (Averette and Nguyen, 1995). The number of sexual partners of a woman's sexual partner is itself also a risk factor.

That the number of sexual partners remains an independent risk factor for cervical cancer after controlling for HPV infection may reflect false negative HPV testing or the independent role of other sexually transmitted diseases. Early age at first intercourse could be a proxy for a longer latency period of carcinogenesis, or it could suggest that the cervix is most vulnerable in adolescence (Schiffman et al., 1996). According to behavioral risk surveys conducted between 1996 and 1997, 4 percent of Connecticut women 18-64 years of age, and 25 percent of girls in grades 9 through 12, reported

having multiple sex partners. Of the students, 42 percent reported they had sex before age 18, and 3 percent said they had sex before age 13 (Adams, 2000).

In 1998, 8 percent of sexually active Connecticut women said they had more than one sex partner in the prior year. Of these, nearly three out of four (74 percent) said they don't always use condoms, and nearly one in three (32 percent) said they never use condoms. Women with multiple sex partners were almost twice as likely as those with only one sex partner to use condoms all the time (27 percent and 15 percent, respectively), however (Connecticut Department of Public Health, AIDS; Counseling and Testing Program, 2000).

HIV and Chlamydia Infections

Unprotected sex can lead to infection with HIV and *Chlamydia*. Women infected with HIV have about 3 times the risk of uninfected women for developing invasive cervical cancer. (See Chapter 21, *HIV/AIDS*).

Chlamydia is the most common sexually transmitted disease among Connecticut women (see Chapter 20, *Sexually Transmitted Diseases*). Exposure to certain types of *Chlamydia trachomatis* bacteria may enhance the progression of HPV infection to invasive cervical cancer, and chlamydia infection may also be an independent risk factor (Anttila et al., 2001; Zenilman, 2001).

Cigarette Smoking

Cigarette smoking is a risk factor for progression of HPV infection to cervical cancer (Zenilman, 2001; Moscicki et al., 2001), and it accounts for about 32 percent of population-attributable risk for cervical cancer in the United States (Brownson et al., 1998). See the chapter on lung cancer for data concerning smoking in Connecticut.

PREVENTION AND RISK REDUCTION

The risk of developing cervical cancer can be reduced by limiting the number of sexual partners, delaying sexual intercourse until a later age, avoiding sexually transmitted diseases, by using barrier contraceptive methods (condom or diaphragms), and by not smoking (Brownson et al., 1998). Cervical cancer vaccines against HPV have been successful in several animal systems, and early phase 1 human trials indicate an enhanced immune response through vaccination, but these results have not yet been confirmed through long-term human clinical trials (Im et al., 2001).

Screening and Early Detection

Cervical cancer is slow in development, making it ideal for early detection and intervention. The Papanicolaou ("Pap") smear test is a rapid, simple, and relatively inexpensive method of detecting cervical cancer. About half of all invasive cervical cancers are diagnosed in women who never had a Pap test, whereas 99 to 100 percent of those diagnosed with *in situ* cervical cancer have had Pap tests (Holmquist, 2000). The American Cancer Society recommends annual Pap tests for all women who are or have been sexually active or who are 18 or more years of age. It is estimated that between 37 percent and 60 percent of cervical cancer deaths could be prevented by full use of the Pap test (Brownson et al., 1998).

For older women who have had a normal Pap smear, the optimum schedule for repeat testing is unknown. There is little or no benefit of routine vaginal screening for women who have had a hysterectomy for benign conditions (National Cancer Institute, 2001).

Nationally, cervical cancer screening rates are lower among certain demographic groups, including Hispanics, lesbians, women with low income, education, or literacy levels, and those living in rural areas (Brownson et al., 1998; Connecticut Tumor Registry, 1999a). During 1996 and 1997, in Connecticut, low-income women between 18 and 24 years of age, and 65 or more years of age were the least likely to have

had a Pap test. Hispanic women were less likely than black women or white, non-Hispanic women to have been tested recently or ever (Adams, 2000).

The Connecticut Breast and Cervical Cancer Early Detection Program (CBCCEDP) is described in the chapter on breast cancer. Between 1995 and 2000, 15,502 women were enrolled and 26,723 Pap test were performed. Follow-up of 518 abnormal Pap tests resulted in the diagnosis of 9 invasive and 94 *in situ* cervical cancers (Mitchell, 2001).

Table 11-2 shows the percentage of women in Connecticut and neighboring states in 2000, among those who were 18 years or older and had an intact cervix, who said they had not had a Pap smear ever or not within three years. Among the eight states listed, Connecticut ranked 3rd highest for not having had a Pap smear ever and for not having one within the past 3 years. The median prevalences of never having had a Pap smear or not within 3 years among 49 states, the District of Columbia, and Puerto Rico were 5 percent and 13 percent, respectively. Self-reports tend to exaggerate use of Pap tests, and patients recall tests as occurring more recently than they actually occurred (Holmquist, 2000).

As noted above, HPV may cause virtually all cases of cervical cancer. Thus, screening for HPV may come into widespread use in the future.

The Woman to Woman Study evaluated a peer-delivered 16-month intervention, through the cooperation of the Service Employees International Union in Boston, and designed to increase breast and cervical cancer screening. The study was intended to reach women in low-income jobs. Although the majority of women reached were in unionized state agencies and health care settings and had household incomes above \$50,000, the intervention resulted in significant increase in Pap smear screening, and modest, non-significant increases in mammography and clinical breast examination rates. Having had a Pap test within the past 3 years increased from 85 percent to 90 percent in the intervention group (+4.7) and from 86 percent to 88 percent in the control group (+1.9). The authors suggested that interventions through churches or housing developments might better reach underserved populations (Allen et al., 2001).

TABLE 11-2
DID NOT HAVE A PAP SMEAR
Northeastern States, Females Aged 18 and Older with Intact Cervix, 2000

| State | Ever | | Within Past 3 Years | |
|---------------|--------------|-------------------------|---------------------|-------------------------|
| | % Prevalence | 95% Confidence Interval | % Prevalence | 95% Confidence Interval |
| Connecticut | 5.9 | 4.6, 7.2 | 12.0 | 10.2, 13.7 |
| Maine | 4.6 | 2.8, 6.4 | 11.0 | 8.6, 13.3 |
| Massachusetts | 5.4 | 4.5, 6.3 | 10.4 | 9.2, 11.6 |
| New Hampshire | 3.1 | 1.5, 4.7 | 9.9 | 7.7, 12.1 |
| New Jersey | 10.0 | 8.2, 11.7 | 18.1 | 16.0, 20.2 |
| New York | 7.6 | 5.9, 9.2 | 14.4 | 12.2, 16.5 |
| Rhode Island | 5.5 | 4.1, 6.9 | 11.5 | 9.7, 13.3 |
| Vermont | 5.8 | 4.2, 7.4 | 11.9 | 10.0, 13.8 |

Source: Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion. Behavioral Risk Factor Surveillance System Survey, 2000.

TREATMENT

In situ cervical cancers may be treated by cryotherapy (cell destruction by extreme cold), electrocoagulation (cell destruction by extreme heat), laser surgery, or traditional surgery. Invasive cervical cancers are treated surgically or with radiation, either alone or in combination. Some procedures, such as with a laser, can be performed on an outpatient basis. Surgical treatment can preserve ovarian function, prevent vaginal stenosis in young women, and avoid the bladder and bowel complications of radiotherapy. While radiotherapy shows no survival advantage, it reduces pelvic recurrences of cancer. Stage I and II cervical carcinoma is usually treated by surgery and stage III and IV by radiotherapy (Averette and Nguyen, 1995).

The National Cancer Institute provides information on types of cervical cancer, risk factors, prevention, testing, diagnosis, coping, support, and treatment, for both patients and health care providers (National Cancer Institute, 2001).

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12. OVARIAN CANCER

SCOPE OF THE PROBLEM

Ovarian cancer is the seventh most common malignancy diagnosed in Connecticut women, accounting for 3.5 percent of invasive cancers. It is the leading cause of death from gynecological malignancies, accounting for about 5 percent of cancer deaths among Connecticut women or 200 deaths each year.

Stage at Diagnosis and Relative Survival Rate

Ovarian cancer has the worst prognosis of any gynecological cancer, because it produces no symptoms until it is at an advanced stage. In the United States, cancer incidence and survival rates are reported through the SEER program (see Appendix A). Between 1992 and 1997, 26 percent of ovarian cancers were localized at diagnosis, where the five-year relative survival rate was 95 percent, 10 percent had spread regionally by the time of diagnosis, where survival was 81 percent, 59 percent were at the distant stage at diagnosis, where survival was 29 percent, and 6 percent were unstaged (Table 12-1)(Ries et al., 2001).

Ovarian cancer survival rates in Connecticut are similar to national rates. Between 1986 and 1993, the last year for which comparisons were published, the five-year relative survival rates were 46 percent nationally and in Connecticut (Ries et al., 1997). Between 1992 and 1997 the national five-year relative survival rate was 52 percent.

In Connecticut in 1997, 47 percent of ovarian cancers were diagnosed at the distant stage, 24 percent were local, 17 percent were regional, and 11 percent were unstaged (Connecticut Tumor Registry, 1999a).

Incidence, Hospitalizations, and Deaths

Between 1995 and 1998, 1,261 new cases of invasive ovarian cancer were diagnosed in Connecticut women; the annual age-adjusted incidence rate was 14.7 cases per 100,000 women (Connecticut Tumor Registry, 2001).

In 1997, there were 321 hospital admissions of Connecticut women with a principal diagnosis of ovarian cancer, resulting in hospital charges of \$6.6 million. The age-adjusted hospitalization rate was 17.2 hospitalizations per 100,000 females (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001).

TABLE 12-1
OVARIAN CANCER STAGING AND RELATIVE SURVIVAL RATE
U.S. Females, 1992-1997 and Connecticut Females, 1997

| Staging | United States, 1992-1997 | | Connecticut, 1997 |
|----------|-----------------------------|----------|----------------------|
| | Percent of Total | 5-Yr RSR | Percent of Total |
| Local | 26 | 95.1 | 24 |
| Regional | 10 | 80.5 | 17 |
| Distant | 59 | 29.4 | 47 |
| Unknown | 6 | 27.2 | 11 |
| Total | 100 | 52.1 | 100 |

Sources: Ries et al., 2001; Connecticut Tumor Registry, 1991a. RSR=relative survival rate

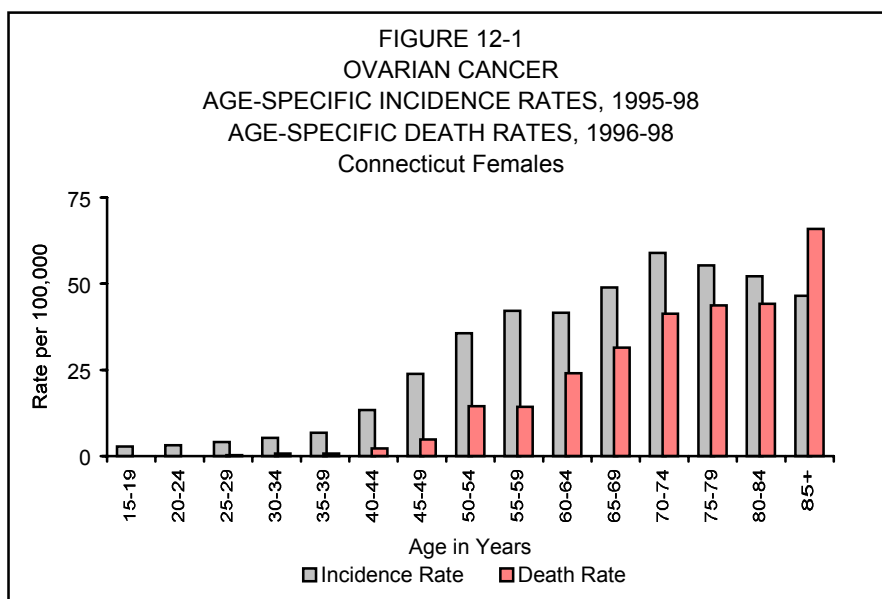
Between 1996 and 1998, 522 Connecticut women died of invasive ovarian cancer, giving an annual, age-adjusted mortality rate of 8.6 deaths per 100,000 females.

Demographic Differences

Ovarian cancer incidence and mortality rise with age (Fig. 12-1). Globally, incidence rates plateau after age 70, which is related to the exhaustion of oocytes at menopause (Weiss et al., 1996).

in the reporting of race-ethnicity other than black or white.

Between 1995 and 1998, the annual, age-adjusted ovarian cancer incidence rates for white, black, and Hispanic females in Connecticut were 17.9, 8.0, and 14.5 per 100,000, respectively; the rate in blacks was significantly lower than that in whites. Hospitalization rates between 1993 and 1997 for ovarian cancer were significantly lower in both black and Hispanic women, compared to white women (Connecticut Department of Public



Source: Connecticut Tumor Registry 2001; Mueller et al., in preparation.
Notes: U.S. Census Bureau population estimates used for rate calculations. Death rates adjusted to the 2000 U.S. standard million population. Incidence rates adjusted to 1970 U.S. standard million population.

Race and Ethnicity

The incidence rate of ovarian cancer is greatest among white and Hawaiian women, intermediate among African-American, Hispanic, and Asian-American women, and lowest among Native American women (Daly and O Abrams, 1998). The mortality rate is greatest among white non-Hispanic women (Ries et al., 2001). Note that rates are affected by misclassification

Health, Division of Policy, Planning, and Analysis, 2001). Similarly, between 1996 and 1998 the age-adjusted mortality rate from ovarian cancer was significantly lower in black women compared to white women (Table 12-2) (Mueller et al., in preparation). These differences partly reflect different hormone levels and child-bearing patterns by race (see the section on risk factors, below).

TABLE 12-2
 OVARIAN CANCER DEATHS BY RACE AND ETHNICITY
 Connecticut Females, 1989-1991 and 1996-1998

| Race/Ethnicity | 1989-1991 | | 1996-1998 | |
|------------------------|------------------|---------------------------------------|------------------|---------------------------------------|
| | Number of Deaths | Age Adjusted Death Rate (per 100,000) | Number of Deaths | Age Adjusted Death Rate (per 100,000) |
| All races | 526 | 9.2 | 522 | 8.6 |
| White | 506 | 9.4 | 505 | 8.9 |
| African American/Black | 17 | 5.9 | 15 | 4.6 [‡] |
| Asian/Pacific Islander | 1 | † | 1 | † |
| Native American | 0 | † | 1 | † |
| Hispanic/Latina | 9 | † | 10 | † |

Source: Mueller et al., in preparation.

† Statistics not calculated for fewer than 15 events.

‡ Rate significantly different from that of whites (p < .05)

Notes: U.S. Census Bureau population estimates used for rate calculations. Rates adjusted to the 2000 U.S. standard million population.

Socioeconomic Status

Ovarian cancer is associated with high socioeconomic status, largely through reproductive behavior.

Geographic Region

Between 1994 and 1998, Connecticut ranked 40th highest among the states and District of Columbia for average annual age-adjusted ovarian cancer mortality (Ries et al., 2001). The ranks of neighboring states were: New Jersey 7, New York 14, Rhode Island 18, Massachusetts 28, Vermont 1, New Hampshire 8, and Maine 6.

TRENDS OVER TIME

In most parts of North America and Europe, ovarian cancer incidence rates have remained nearly constant for decades (Weiss et al., 1996). In the 1980's, ovarian cancer incidence rates and mortality rates in the United States both increased slightly, while between 1991 and 1998, they each declined by about 1.1 percent annually. Both changes were statistically significant (Ries et al., 2001). Connecticut ovarian cancer incidence rates did not change significantly between 1980 and 1998. Mortality rates declined slightly by approximately 0.9 percent annually between 1989-91 and 1996-98, not a significant change.

RISK FACTORS

The principal non-modifiable risk factors for ovarian cancer are age and genetic susceptibility. Genetic predisposition is conferred in part by inherited mutations in BRCA1 and BRCA2 genes (especially BRCA1), which normally act as tumor suppressors. Carriers of these mutations account for about 8 percent of cases of ovarian cancer (Elit, 2001). The BRCA1 mutation conveys a 63 percent risk of developing ovarian cancer during a woman's lifetime, and the BRCA2 mutation a 27 percent risk (Elit, 2001). See the section *Genetic Counseling and Testing*, below.

Reproductive History and Hormone Use

Several lines of evidence point to a positive relationship between a woman's number of ovulatory cycles and the risk for development of ovarian cancer (Schildkraut et al., 1997). Ovulation is inhibited during pregnancy and by birth control pills. Accordingly, never having had a full-term pregnancy and infertility increase risk (Mosgaard et al., 1997). Risk also increases slightly with a first pregnancy before age 20 years (Cooper et al., 1999).

Oral contraceptive use reduces ovarian cancer risk by at least one-half, a benefit that grows with increasing duration of use and continues for 15 or more years after cessation of

use (Hulka, 1997; LaVecchia and Franceschi, 1999). Interestingly, other contraceptives including intrauterine device, barrier methods, tubal ligation, and vasectomy all reduced ovarian cancer risk in multigravid women, but not nulligravid women, after adjustment for other risk factors. The effects were greatest for tubal ligation and oral contraceptives. These results are compatible with a hormonal or ovulatory effect, but suggest that some contraceptives decrease ovarian cancer risk through other mechanisms (Ness et al., 2001). In another study, tubal ligation decreased ovarian cancer risk among women with the BRCA1 mutation, but not among women with the BRCA2 mutation (Narod et al., 2001).

After menopause, estrogen replacement therapy without progesterone may increase the risk of ovarian cancer mortality by more than 100 percent in women who have used ERT for 10 years or more (Rodriguez et al., 2001). This is consistent with the effects of ERT on breast and endometrial cancer risk.

Family History

Family history of breast, colon, or endometrial cancer in a first-degree relative (mother or sister) is considered a risk factor (Averette and Nguyen, 1995) and family history of ovarian cancer is present in 20 percent of ovarian cancers (Elit, 2001). Family history represents a combination of genetic inheritance and shared environment.

Diet

While evidence is inconsistent, in some studies, high consumption of cholesterol (eggs), has been associated with an increased risk of ovarian cancer. A high intake of green leafy vegetables may decrease risk (Kushi et al., 1999).

RISK REDUCTION

Because the causes of ovarian cancer are poorly understood, prevention is difficult. Risk may prove to be modifiable by changes in diet (increasing consumption of green, leafy vegetables). In 1997, less than four in ten adult Connecticut women and only three in ten high school students reported consuming five or more servings of fruits and vegetables daily, with blacks and younger adults reporting the lowest consumption (Adams, 2000). See the chapter on colorectal cancer.

Prophylactic oophorectomy (surgical removal of the ovaries) reduces risk, although not completely, and may be considered by high-risk women who have completed childbearing (Eisen et al., 2000). For women with the BRCA1 mutation, prophylactic oophorectomy would reduce both breast and ovarian cancer risk (Rebbeck, 2000). Its value in reducing mortality requires further study.

Genetic Counseling and Testing

A health care professional should determine which patients are at increased risk of breast and ovarian cancer by eliciting their personal and family histories. A family history should include at least three generations on both maternal and paternal sides. Assessment of risk is based on the number of first and second degree relatives with breast, ovarian, prostate, colon and some other cancers, their ages at diagnoses, and the occurrence of multiple or bilateral cancers. (New York State Department of Health, 1999). It is not currently recommended that widespread genetic screening of any subpopulation be initiated. For women with a history of breast and ovarian cancer in several relatives, alternatives to genetic testing include increased surveillance, participation in clinical research, and use of chemopreventive agents (e.g. tamoxifen).

Screening and Early Detection

Ovarian cancer is rarely diagnosed in its early stages, when successful treatment is possible. One step screening tests are not feasible, except perhaps in some high risk women.

A two-step screening strategy is under consideration. Elevated levels of cancer antigen 125 (CA 125) are found in women with ovarian cancer prior to clinical presentation. However, elevated levels are sometimes also found in other women. Transvaginal ultrasonography (also called endovaginal ultrasound, or EVUS), when used as a second-line test in post-menopausal women with elevated CA 125, appears to distinguish those at normal risk from those at approximately 300-fold elevated risk of ovarian cancer (Rosenthal and Jacobs, 1998; Menon et al., 1999). In a study from Yale, 252 women with a family history of ovarian cancer underwent regular pelvic examinations, screening for CA 125, and EVUS. Two ovarian cancers were detected, and 11 women developed breast cancer. The authors recommended that mammography be performed at the time of ovarian cancer screening (Taylor and Schwartz, 2001).

TREATMENT

For a larger discussion of cancer treatment, see the chapter on colorectal cancer. Ovarian cancers are treated by surgery (hysterectomy and removal of one or both ovaries), often followed by chemotherapy for women with advanced stage cancer or a family or personal history. The aggressiveness of surgery depends upon whether resection is safe (Averette and Nguyen, 1995). Patients with any stage of ovarian cancer are appropriate candidates for clinical trials (National Cancer Institute, 2001).

The National Cancer Institute provides information on types of ovarian cancer, risk factors, prevention, testing, diagnosis, coping, support, and treatment, for both patients and health care providers (National Cancer Institute, 2001).

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13. ENDOMETRIAL CANCER

SCOPE OF THE PROBLEM

Cancer of the body of the uterus (uterine corpus) is the most common cancer of the female reproductive system and is the fourth most common cancer among American women, behind breast, lung, and colorectal cancers. It accounts for more than half of gynecologic cancers and about 6 percent of all invasive cancers found in Connecticut women each year (Connecticut Tumor Registry, 2001).

Stage at Diagnosis and Relative Survival Rate

The vast majority of corpus uteri cancers in white women are endometrial, involving the lining of the uterus, as are also most corpus uteri cancers in black women (Grady and Erstner, 1996). Both endometrial and myometrial cancers, as well as a few cancers of the cervix uteri, have on occasion been reported as uterus, not otherwise specified (NOS). In the 1980's, about half of deaths reported as due to cancer of the uterus, NOS, were actually of the corpus. Over time, fewer uterine cancers are being reported as NOS (Grady and Erstner, 1996). Currently NOS represents 2% of total uterine cases (National Cancer Institute, 2001). In this

chapter we group cancer of the uterine corpus and uterus, NOS together, following the practice of the SEER program. To distinguish the corpus from the uterine cervix, in this chapter we refer to the former as the endometrium, as this is a common practice, although not completely accurate.

National cancer incidence and survival rates are reported using the SEER database (see Appendix A). Between 1992 and 1997, the national five-year relative survival rate for all stages of endometrial cancers was 84 percent (Table 13-1). Seventy-three percent of endometrial cancers were diagnosed at the localized stage, where five year relative survival was 96 percent, 14 percent were diagnosed at the regionalized stage, where survival was 63 percent, 8 percent were diagnosed at the distant stage, where survival was 26 percent, and 5 percent were unstaged (Ries et al., 2001).

Endometrial cancer survival rates in Connecticut are similar to national rates. Between 1986 and 1993, the most recent years for which comparisons were published, the five year relative survival rates were 84 percent nationally and 83 percent in Connecticut (Ries et al., 1997).

TABLE 13-1
ENDOMETRIAL CANCER STAGING AND RELATIVE SURVIVAL RATE
U.S. Females, 1992-1997 and Connecticut Females, 1997

| Staging | United States, 1992-97 | | Connecticut, 1997 | |
|--------------|------------------------|----------|-------------------|----------|
| | Percent of Total | 5-Yr RSR | Percent of Total | 5-Yr RSR |
| Non-invasive | 0 | NA | 3 | |
| Local | 73 | 96.1 | 73 | |
| Regional | 14 | 62.7 | 10 | |
| Distant | 8 | 25.8 | 5 | |
| Unknown | 5 | 49.1 | 9 | |
| Overall | 100 | 84.0 | 100 | 83.2* |

Sources: Ries et al., 1997, 2001; Connecticut Tumor Registry, 2000.
RSR=Relative Survival Rate *1986-1993

In Connecticut in 1997, 73 percent of invasive endometrial cancers were diagnosed at the local stage, 10 percent were regional, and 5 percent were distant (Connecticut Tumor Registry, 2000). The balance of tumors were noninvasive (3 percent) or of unknown stage (9 percent).

Incidence, Hospitalizations, and Deaths

True rates involving endometrial cancer depend on the number of women at risk, that is, the number of women with an intact uterus. By the 1970's, one third of American women over age 50 had received a hysterectomy and so were no longer at risk for endometrial cancer (see the section *Trends over Time*, below). However, endometrial cancer rates, including those reported in this chapter, are almost always calculated from populations that include women who have had hysterectomies, so that the rates are artificially low.

Between 1995 and 1998, 2,121 new cases of invasive endometrial cancer were found in Connecticut women, and the annual, age-

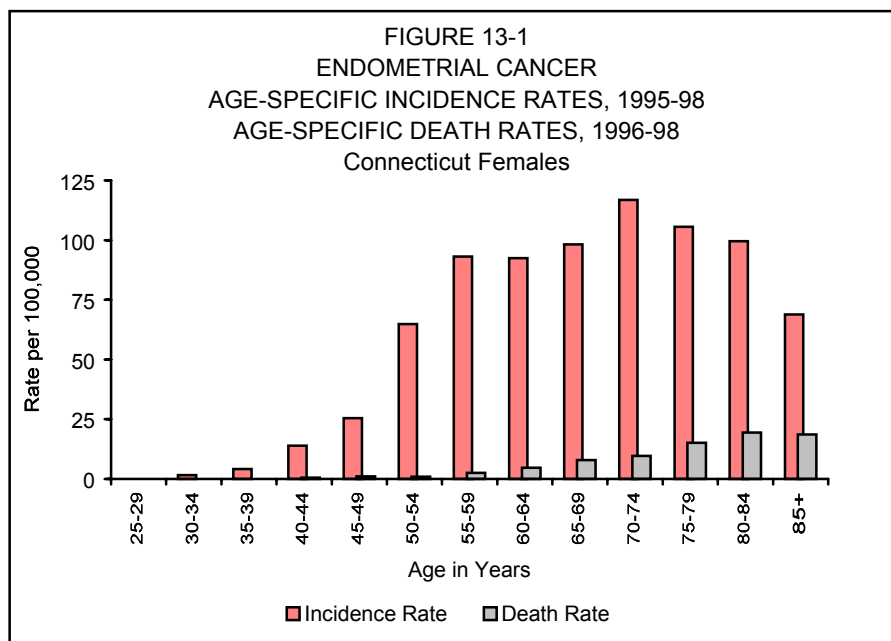
adjusted incidence rate was 25 cases per 100,000 females (Connecticut Tumor Registry, 2001).

Between 1993 and 1997 the annual, age-adjusted hospitalization rate (AAHR) was 27 hospitalizations per 100,000 females. In 1997, there were 494 hospitalizations (AAHR 26 per 100,000 females), resulting in total hospital charges of \$5.9 million dollars (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001).

Between 1996 and 1998, 135 Connecticut women died of invasive endometrial cancer, and the annual, age-adjusted rate was 2.1 deaths per 100,000 females (Mueller et al., in preparation).

Age

Endometrial cancer incidence rates increase with age, and less rapidly after the age of menopause, before falling after age. Mortality rates, however, increase slowly throughout a woman's lifetime 75 (Fig. 13-1).



Source: Connecticut Tumor Registry, 2001; Mueller et al., in preparation.
Notes: U.S. Census Bureau population estimates used for rate calculations. Death rates adjusted to the 2000 U.S. standard million population. Incidence rates adjusted to 1970 U.S. standard million population.

Socioeconomic Status

Endometrial cancer has been inconsistently associated with high socioeconomic status. If present, the association is probably due to estrogen replacement therapy (ERT) and more complete diagnosis in women who use ERT (Grady and Erstner, 1996).

Race and Ethnicity

In the U.S., white, non-Hispanic women have the highest annual incidence rate of endometrial cancer (23 per 100,000 between 1994 and 1998), whereas American Indian/Alaskan native (9 per 100,000), Hispanic (13), Asian/Pacific Islander (15), and black women (15) have much lower incidence rates. However, there is some misclassification of race and ethnicity in the reporting of cancer incidence and mortality. Annual mortality rates among black women (6), however, are 1.8 times greater than those of white women (3) and 2-3 times greater than those of Asian/Pacific Islanders, American Indians, and Hispanics (Ries, et al., 2001). Similarly, the relative survival rates for white women are greater than for black women at every diagnostic stage (Table 13-2). This is most evident among women aged 50 or older (American Cancer Society, 2000; Ries et al., 2001).

types of cancer, or that they have better access to the best treatment (Grady and Erstner, 1996). The latter two possibilities have been intensively studied.

Histologic types do differ by race. Cancers of the myometrium (which are primarily sarcomas) are more common among black women than white women, and these cancers are reported as uterus, NOS. In late life, mortality rates for uterus, NOS, are actually higher than for corpus both in whites and blacks (Grady and Erstner, 1996).

Some studies have suggested that the poor endometrial cancer prognoses in African American women were due to the cumulative number of poor prognostic factors, including tumor characteristics and socioeconomic status, beyond which race had no predictive value (Hicks et al., 1997). Others have found race significant even after controlling for pathologic and socioeconomic factors (Connell et al., 1999). While income generally had no effect on whether treatment was provided, African American women were treated less often by surgery, and had poorer survival than white women even for stage I adenocarcinoma treated surgically (Hicks et al., 1998). One study estimated that after adjusting for age and geographic location, the relative mortality risk in blacks was four times

TABLE 13-2
ENDOMETRIAL CANCER STAGING AND RELATIVE SURVIVAL RATE BY RACE
U.S. Females, 1992-1997

| Staging | White | | Black | |
|----------|------------------|----------|------------------|----------|
| | Percent of Total | 5-Yr RSR | Percent of Total | 5-Yr RSR |
| Local | 75 | 96.9 | 52 | 82.9 |
| Regional | 13 | 65.1 | 22 | 42.7 |
| Distant | 8 | 27.7 | 18 | 13.1 |
| Unstaged | 4 | 47.6 | 9 | 48.9 |
| Total | 100 | 85.8 | 100 | 58.9 |

Source: Ries et al., 2001. RSR=relative survival rate

Better survival for white women compared to black women within each cancer stage suggests that stage may be less advanced for white women within each staging category, that they may develop more favorable histologic

that in whites; 40 percent of the difference was attributed to more advanced stage, 23 percent to tumor characteristics and treatment, and 17 percent to socioeconomic, hormonal, reproductive, comorbidity, and health behavior

characteristics, but leaving 20 percent unexplained (Hill et al., 1996).

The racial disparities in stage at diagnosis seem to be due to more aggressive types of cancer appearing among black women (Barrett et al., 1995). For example, adenocarcinomas were usually diagnosed at early stage while papillary serous and clear cell cancers, which were more common in black than in white women, were diagnosed at later stages. Within each stage, patients were treated similarly regardless of cancer type or race (Matthews et al., 1997).

Recently, black and white differences have been examined at the genetic level. Among patients with stage I endometrial cancer, the mutant p53 tumor suppressor gene was found in 34 percent of blacks and 11 percent of whites, and recurrent disease, which is related to the p53 gene, was seen in more blacks than whites (Clifford et al., 1997). After collecting information about possible confounding variables, p53 alteration was found to be the most important prognostic variable, and only p53 expression and stage entered a multivariate model (Sung et al., 2000). The PTEN mutation was found more often in whites with endometrial cancer (22 percent) than in blacks (5 percent),

and this mutation conferred better survival (Maxwell et al., 2000). Microsatellite instability (MSI) in DNA was associated with the absence of p53 overexpression in endometrial cancer (Maxwell et al., 2001). There is controversy over whether MSI in endometrial cancer patients is associated with white race or better survival (Basil et al., 2000; Maxwell et al., 2000, 2001).

Between 1995 and 1998 in Connecticut, the annual, age-adjusted endometrial cancer incidence rate was 29.3 per 100,000 in white women, which was significantly higher than the rate of 20.8 per 100,000 in black women or 18.7 per 100,000 in Hispanic women, a pattern also seen in hospital discharges between 1993 and 1997 (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001). Between 1996 and 1998, there were too few endometrial cancer deaths among non-white women for racial and ethnic comparisons (Table 13-3). However, between 1994 and 1998, the mortality rate in Connecticut was about 50 percent higher among blacks than whites, as was true nationally (Ries et al., 2001).

TABLE 13-3
ENDOMETRIAL CANCER DEATHS BY RACE AND ETHNICITY
Connecticut Females, 1989-1991 and 1996-1998

| Race/Ethnicity | 1989-91 | | 1996-98 | |
|------------------------|------------------|---------------------------------------|------------------|---------------------------------------|
| | Number of Deaths | Age Adjusted Death Rate (per 100,000) | Number of Deaths | Age Adjusted Death Rate (per 100,000) |
| All races | 133 | 2.2 | 135 | 2.1 |
| White | 127 | 2.2 | 126 | 2.1 |
| African American/Black | 6 | † | 9 | † |
| Asian/Pacific Islander | 0 | † | 0 | † |
| Native American | 0 | † | 0 | † |
| Hispanic/Latina | 0 | † | 2 | † |

Source: Mueller et al., in preparation.

† Statistics not calculated for fewer than 15 events.

Notes: U.S. Census Bureau population estimates used for rate calculations. Rates adjusted to the 2000 U.S. standard million population.

Geographic Region

Endometrial cancer incidence rates for white, black, and Asian women in the United States are higher than for their racial counterparts in Europe and Japan (Grady and Erstner, 1996). Between 1994 and 1998, Connecticut ranked 27th highest among the states and District of Columbia for average annual age-adjusted endometrial cancer mortality. The ranks of neighboring states were: New Jersey 2, New York 4, Rhode Island 9, Massachusetts 20, Vermont 3, New Hampshire 7, and Maine 28 (Ries et al., 2001).

TRENDS OVER TIME

Since the 1950's, endometrial cancer mortality rates have apparently declined by more than 60 percent in the United States, although part of this decline is an artifact of the increased rates of hysterectomies, while including such women in the populations from which rates are calculated. One study estimated that adjustment for hysterectomy raised the age-adjusted endometrial cancer rate by 20% (National Cancer Institute, 2001). During 1996 and 1997, the proportions of Connecticut women who said they had had a hysterectomy, by age, were: 1 percent at ages 18-34 years, 9 percent at ages 35-49, 33 percent at ages 50-64, and 42 percent at ages 65 and older (Adams, 2000).

Hysterectomies are performed for a variety of reasons. In 1998 in North Carolina, cancer was the primary diagnosis for the hysterectomy in only 6 percent of cases; the leading diagnosis (34 percent) was uterine leiomyoma or fibroids (Jones-Vessey, 2000). In 1998, 645,000 hysterectomies were performed in the United States, a rate of 236 per 100,000 women (Popovic and Kozak, 2000), while 4,994 were performed as a primary hospital procedure in Connecticut, a rate of 296 per 100,000 women (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001).

Endometrial cancer incidence rates rose during the 1970's due to the use of high doses of unopposed estrogen by postmenopausal women, before falling to previous levels with the introduction of lower dose, combination hormone replacement therapy (Grady and Erstner, 1996). Mortality rates did not increase during the 1970's, possibly because the estrogen-induced tumors were less aggressive or were detected earlier. Among women of all ages, mortality rates between 1973 and 1998 declined more rapidly in blacks than whites. Mortality rates among blacks aged less than 50 years declined more rapidly (4.6 percent annually) than blacks aged 50 years and over (0.8 percent annually). Between 1988 and 1998, national incidence rates increased by 0.5 percent annually, while national mortality rates decreased between 1989 and 1998 by 0.6 percent annually (Ries et al., 2001); both trends were statistically significant. Between 1980 through 1984 (23.1 per 100,000), and 1995 through 1998 (24.8 per 100,000), and intermediate periods, there were no significant changes in the annual, age-adjusted incidence rate for endometrial cancer in Connecticut (Polednak, 2001). Between 1989 through 1991, and 1996 through 1998, there was no significant change in the annual mortality rate (Table 13-3).

RISK FACTORS

The most important non-modifiable risk factors for endometrial cancer are age and genetic susceptibility. For prognostic factors, see the section *Race and Ethnicity*, above. A personal or family history of breast or ovarian cancer increases the risk for endometrial cancer, but may represent a common effect of reproductive risk factors. Polycystic ovary syndrome may be present in up to 30 percent of endometrial cancers in selected groups of premenopausal women (Grady and Erstner, 1996).

Estrogen is the main risk factor for the most common form of endometrial cancer. Estrogen-related factors include early onset of menstruation, late menopause, a history of failure to ovulate, never having children, use of tamoxifen, and estrogen replacement therapy

(ERT). The relative risk of endometrial cancer from ever using ERT is 2.3, with risk increasing according to estrogen dose and duration of usage (Grady and Ernstner, 1996). When estrogen is taken in lower doses with progesterone (hormone replacement therapy, HRT), however, the risk is actually lower than in women not taking hormones (Grady and Ernstner, 1996). Smoking may reduce the risk of endometrial cancer incidence through an androgenic effect. However, smoking may not reduce endometrial cancer mortality risk, if it prevents only clinically unimportant tumors (Grady and Ernstner, 1996).

The most important modifiable risk factor for endometrial cancer is obesity, which increases risk by 3 to 10 times (Merck & Co., 2000). Body weight, weight gain, and accumulation of central body fat all are positively associated with endometrial cancer (Ballard-Barbash and Swanson, 1996). The body converts adipose tissue to estrogens (see the chapter on breast cancer). Diabetes and gallbladder disease are associated with endometrial cancer (Grady and Ernstner, 1996), probably through their link with obesity.

Table 13-4 shows the prevalence of being overweight or obese among females in Connecticut and neighboring states. (Technically defined, *obesity* is a greater body mass index than *overweight*; BMI is calculated from weight and height; for the formula, see Appendix A.) Of the eight states listed, Connecticut was the 4th highest for overweight or obesity. For not restricting calories or exercise to lose weight among overweight or obese women, Connecticut ranked 7th. The median prevalences for being overweight or obese, and among these, for not attempting to lose weight by eating fewer calories or exercising, were 49 percent and 51 percent, respectively, in 50 states, the District of Columbia, and Puerto Rico. In 1997, the Youth Risk Behavior Surveillance Survey found that one-third of female high school students in Connecticut were overweight, then defined as a BMI of 27.3 or above (Kann et al., 1998).

Table 13-5 shows the prevalence of being overweight or obese in Connecticut by race-ethnicity. Obesity was most common in black women and least common in white women.

TABLE 13-4
OVERWEIGHT OR OBESE
Northeastern States, Females, 2000

| State | Overweight or Obese* | | Not Eating Fewer Calories or Exercising to Lose Weight* | |
|---------------|----------------------|-------------------------|---|-------------------------|
| | % Prevalence | 95% Confidence Interval | % Prevalence | 95% Confidence Interval |
| Connecticut | 45.1 | 42.7, 47.4 | 47.1 | 43.4, 50.8 |
| Maine | 48.6 | 45.3, 51.9 | 53.1 | 48.1, 58.0 |
| Massachusetts | 41.9 | 40.1, 43.6 | 49.5 | 46.7, 52.3 |
| New Hampshire | 42.8 | 39.4, 46.2 | 51.0 | 45.6, 56.3 |
| New Jersey | 47.0 | 44.5, 49.4 | 52.8 | 49.0, 56.6 |
| New York | 49.2 | 46.5, 51.8 | 53.9 | 49.8, 58.0 |
| Rhode Island | 44.6 | 42.1, 47.1 | 51.1 | 46.9, 55.2 |
| Vermont | 44.3 | 41.8, 46.7 | 45.7 | 41.9, 49.5 |

Source: Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion. Behavioral Risk Factor Surveillance System Survey, 2000.

*Body mass index greater than or equal to 25.0

TABLE 13-5
OVERWEIGHT OR OBESE BY RACE AND ETHNICITY
Connecticut Females, 2000

| Race-Ethnicity | Overweight* | | Obese** | |
|---------------------|--------------|-------------------------|--------------|-------------------------|
| | % Prevalence | 95% Confidence Interval | % Prevalence | 95% Confidence Interval |
| White, Non-Hispanic | 28.1 | 26.7, 29.6 | 14.7 | 13.9, 15.4 |
| Black, Non-Hispanic | 33.8 | 27.9, 39.6 | 30.1 | 24.8, 35.3 |
| Hispanic, All Races | 30.0 | 25.7, 34.2 | 24.0 | 20.6, 27.4 |

Sources: Connecticut Department of Public Health, Bureau of Community Health, unpublished data, 2001 (prevalence); Division of Policy, Planning and Analysis, 2001 (95% confidence intervals).

*Body mass index between 25.0 and 29.9 **Body mass index greater than or equal to 30.0

PREVENTION AND RISK REDUCTION

Sequential estrogen and progestin oral contraceptives increased the risk of endometrial cancer, and were removed from the market in the 1970's. Long-term use of combination oral contraceptives substantially reduces the risk of endometrial cancer (Grimes and Economy, 1995; Grady and Ernstner, 1996). Weight control may prevent endometrial cancer, also through hormonal mechanisms (Hulka and Brinton, 1995; Ballard-Barbash and Swanson, 1996).

Tamoxifen's benefits in reducing breast cancer risk outweigh its risks of increased endometrial cancer in women with a history of breast cancer, but in women at low to moderate breast cancer risk, the risks incurred by tamoxifen use may exceed its benefits (Grady and Ernstner, 1996).

Screening and Early Detection

There is no recommended screening procedure for endometrial cancer, although cytological screening is under investigation (Yoshida et al., 2001). Screening may detect clinically unimportant tumors, as the experience in the 1970's with ERT showed (Grady and Ernstner, 1996).

More than 90 percent of women with endometrial cancer have abnormal bleeding, and about one-third of women with postmenopausal bleeding have endometrial cancer (Merck & Co., Inc., 2000). Endometrial biopsy, performed in a

physician's office, is the definitive method of diagnosis and is recommended at menopause and periodically thereafter for high-risk women or when irregular pre- or post-menopausal bleeding occurs (American Cancer Society, 2000).

TREATMENT

Endometrial cancers are usually treated with surgery (hysterectomy plus salpingo-oophorectomy), radiation for those with any risk factors, medroxyprogesterone, and chemotherapy when systemic disease is present (American Cancer Society, 2000).

The National Cancer Institute provides information on types of endometrial cancer, risk factors, prevention, testing, diagnosis, coping, support, and treatment, for both patients and health care providers (National Cancer Institute, 2001).

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14. CHRONIC OBSTRUCTIVE PULMONARY DISEASE

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a term for several lung diseases all characterized by progressive airflow limitation that is not reversible (Barnes, 2000). COPD is one of the most common diseases of adults in industrialized countries, with an estimated 16 million Americans considered to be symptomatic. More than half of these people are probably undiagnosed. Many more Americans have asymptomatic forms of the disease (Petty and Master, 2000). COPD exacts enormous human and economic costs, affecting people during their peak work years. After several years with the disease, most COPD patients are disabled by chronic shortness of breath, coughing, and wheezing, and are no longer able to work productively (Goldring et al., 1998). At a time when the mortality rates for other major conditions are decreasing, COPD death rates are rising among women in the U.S. and Connecticut. Reasons for the increase probably include reduced mortality from other causes as well as an increase in cigarette smoking (Barnes, 2000). Part of this increase may be artificial due to changes in reporting practices (Centers for Disease Control and Prevention, 2001a).

Subgroups of COPD

Chronic obstructive pulmonary disease includes the diseases of emphysema and chronic bronchitis. Asthma, a clinically distinct condition, is also commonly included in COPD surveillance. These three diseases may be present singly or together in any individual person.

Chronic bronchitis, emphysema, and chronic airway obstruction are oftentimes grouped as COPD because, from a clinical standpoint, they may be difficult to distinguish from one another. *Chronic bronchitis* is diagnosed when a patient has a persistent cough caused by excessive airway mucus secretion over a long period of time. Persons with chronic bronchitis may also experience difficulty in breathing due to narrowing of the airways. *Emphysema* is a condition defined as the permanent destruction of the alveoli, the small elastic air sacs of the lung. This loss of elasticity can also cause narrowing or collapse of the bronchioles, or small air passages, which then limits airflow from the lungs. *Chronic airway obstruction* is a term used to identify other obstructed airway conditions that are not classified in any other subcategories (Goldring et al., 1998).

TABLE 14-1
MAIN SUBCATEGORIES OF COPD AND ALLIED CONDITIONS

| Disease (ICD-9 code) | Definition |
|----------------------------------|--|
| Chronic Bronchitis (490-491) | Excessive mucus production associated with narrowing of the bronchial airways and cough |
| Emphysema (492) | Alveolar destruction and associated airspace enlargement |
| Asthma (493) | Airway obstruction with airway inflammation and increased airways responsiveness to a variety of stimuli |
| Chronic Airway Obstruction (496) | Generalized airway obstruction not classifiable as chronic bronchitis or chronic obstructive bronchitis |

Adapted from Goldring et al., 1998.

Asthma is an inflammatory response of the airways to a variety of stimuli that results in a usually temporary airway obstruction. Inadequate treatment of asthma may lead to chronic airway obstruction and asthma patients are considered to have COPD only when their airflow obstruction cannot be reversed by medication (Goldring et al., 1998). Because of its importance as a chronic condition affecting increasing numbers of women, asthma is discussed in depth in Chapter 15 of this report. Table 14-1 summarizes the main subgroups referred to as “COPD and Allied Conditions” in mortality and morbidity surveillance.

SCOPE OF THE PROBLEM

In the 1996 to 1998 period, COPD and allied conditions was the fourth leading cause of death among Connecticut women of all ages. Most COPD deaths were categorized as chronic airway obstruction (1,536 deaths), followed by emphysema (277 deaths), asthma (114 deaths), and bronchitis (50 deaths) (Figure 14-1).

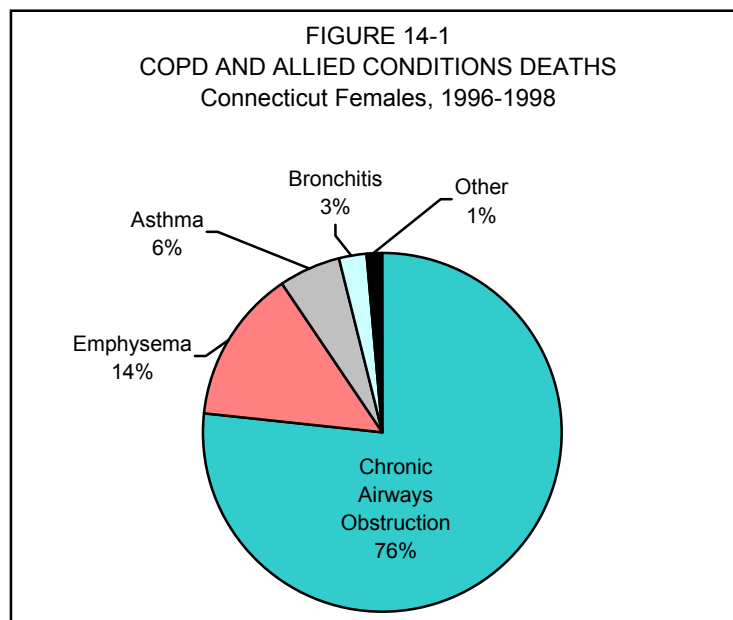
Death in COPD patients, however, often results from some other medical condition or complication. For this reason, and because its

contribution to other major causes of death is not always considered, the importance of COPD as a cause of death is probably underestimated. COPD was the primary cause of death for 2,005 Connecticut women from 1996 to 1998. During the same period, COPD was a contributing cause in the deaths of 4,336 women, more than double the number for COPD alone (Mueller et al., in preparation).

Chronic obstructive pulmonary disease is a leading cause of hospital admissions among Connecticut women, with about 5,000 per year during the 1993 to 1997 period. In 1997, there were 5,110 hospitalizations, resulting in total hospital charges of \$51.6 million dollars (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001). All COPD hospitalizations are considered “avoidable,” that is, hospitalizations that could be avoided with appropriate and timely primary care (Pappas et al., 1997).

Age

Deaths due to chronic obstructive pulmonary disease increase dramatically with age. Women 65 years of age and older, who represent less than 17 percent of Connecticut’s



Source: Mueller et al., in preparation.

female population, accounted for 90 percent of COPD deaths in the 1996 to 1998 period.

Race and Ethnicity

Age-adjusted death rates for COPD among Connecticut women rose significantly by an average of 3.6 percent per year from 1989 and 1998 and increased significantly between the periods 1989 to 1991 and 1996 and 1998. This change is accounted for by an increase in the death rate among white females (Table 14-2). The annual increase was the greatest noted for any of the selected causes discussed in this report (Mueller et al., in preparation).

compared with 28 percent of black, and 39 percent of Hispanic female COPD deaths.

These findings are consistent with available national data that show blacks have lower COPD, but higher asthma mortality compared with whites (Gillum, 1990). COPD mortality was higher in white compared with black women in 1992, although time trends indicate a similar sharp increase in COPD mortality for both white and black women from 1980 to 1992 (Centers for Disease Control and Prevention, 2001b). Similar national comparisons of COPD mortality in Hispanic, Asian and Pacific Islander, and Native American women are not available. Continued

TABLE 14-2
COPD AND ALLIED CONDITIONS DEATHS
BY RACE & ETHNICITY
Connecticut Females, 1989-1991 and 1996-1998

| Race/Ethnicity | 1989-1991 | | 1996-1998 | |
|------------------------|------------------|---------------------------------------|------------------|---------------------------------------|
| | Number of Deaths | Age Adjusted Death Rate (per 100,000) | Number of Deaths | Age Adjusted Death Rate (per 100,000) |
| All races | 1,412 | 23.0 | 2,005 | 29.9* |
| White | 1,369 | 23.3 | 1,955 | 30.7* |
| African American/Black | 43 | 15.3‡ | 50 | 15.1‡ |
| Asian/Pacific Islander | 0 | † | 0 | † |
| Native American | 0 | † | 0 | † |
| Hispanic/Latina | 19 | 14.7‡ | 31 | 16.2‡ |

Source: Mueller et al., in preparation.

* Change in rates from 1989-91 to 1996-98 period is statistically significant (p< .05).

† Statistics not calculated for fewer than 15 events.

‡ Rate significantly different from that of whites (p< .05).

Notes: U.S. Census Bureau population estimates used for rate calculations. Rates adjusted to the 2000 U.S. standard million population.

COPD death rates were significantly higher in white women compared with black and Hispanic women in both the 1989 to 1991 and 1996 to 1998 periods. There were, however, differences in the racial and ethnic populations by subcategories of COPD deaths. For example, in the 1996 to 1998 period, 77 percent of white female COPD deaths were due chronic airway obstruction compared with 58 percent of black and 45 percent of Hispanic female COPD deaths; 14 percent of white compared with 10 percent of black and Hispanic female COPD deaths were due to emphysema. Only 5 percent of white female COPD deaths were due to asthma,

investigation of racial and ethnic differences in COPD mortality may enhance understanding of its etiology and prevention.

Black and Hispanic women had significantly more hospitalizations due to COPD and allied conditions compared with white women during the 1993 to 1997 period (Table 14.3). Asian and Pacific Islander women had significantly fewer hospitalizations due to COPD compared with white women. There were too few COPD hospitalizations among Native American women to calculate reliable rates. Racial and ethnic subgroups differed in their types of COPD hospitalizations. For example, about 89 percent of all COPD hospitalizations

among Latinas and about 83 percent of those among black women were due to asthma compared with about 40 percent of COPD hospitalizations among white women. Forty-two percent of COPD hospitalizations among white women were due to chronic bronchitis compared with 8 percent of such hospitalizations among Latinas and 12 percent among black women during this period. These data suggest there are barriers to adequate or timely primary care for COPD among Connecticut women and that such barriers are greater for blacks and Latinas compared with white women.

Increases in COPD mortality nationwide are closely linked to the widespread adoption of smoking behaviors among American women beginning in the post-World War II era (U.S. Department of Health and Human Services, 1990). In Connecticut, cigarette smoking rates among women have been relatively stable since 1990 (Centers for Disease Control and Prevention, 2001c). In 1999, about 21 percent of adult females ages 18 and older were current smokers (Adams, 2001). Most adult smokers initiate the habit during adolescence, so high rates of adolescent usage are a serious public

TABLE 14-3
COPD AND ALLIED CONDITIONS HOSPITALIZATIONS
BY RACE & ETHNICITY
Connecticut Females, 1993-1997

| Race/Ethnicity | Number | Age Adjusted Rate (per 100,000) |
|------------------------|--------|------------------------------------|
| All races | 25,463 | 279.4 |
| White | 18,486 | 225.9 |
| African American/Black | 3,278 | 489.1 [‡] |
| Asian/Pacific Islander | 57 | 40.8 [‡] |
| Native American | 13 | † |
| Hispanic/Latina | 3,413 | 664.7 [‡] |

Source: Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001.

† Statistics not calculated for fewer than 25 events.

‡ Rate significantly different from that of whites (p<.05).

Notes: U.S. Census Bureau population estimates used for rate calculations. Rates adjusted to the 2000 U.S. standard million population.

RISK FACTORS

Cigarette Smoking

Cigarette smoking is the key risk factor for the development of COPD. Smokers have ten times the relative risk of non-smokers for COPD occurrence (Goldring et al., 1998). Although not all smokers develop COPD, its prevalence does increase with age. Among current smokers 40 years and older, COPD prevalence is estimated to increase from 17 percent to 43 percent with increasing age (Stang et al., 2000). An estimated 90 percent of all COPD deaths among women are attributable to smoking (Centers for Disease Control and Prevention, 2001b).

health concern. About one in three (32 percent) female students, grades 9-12 in Connecticut reported current cigarette smoking in 1999 and about 14 percent said they smoked on at least 20 of the last 30 days (Kann et al., 2000).

Environmental Pollutants and Other Factors

Outdoor air pollution at high levels (especially ozone, sulfur dioxide, and particulates), exposure to chemical fumes or dust in occupational settings, exposure to second-hand tobacco smoke, and use of solid fuels for heating and cooking in poorly ventilated spaces all can contribute to COPD. Low birth weight appears to increase the risk of COPD, and a deficiency of a protein called alpha-1-antitrypsin may be associated with emphysema (Barnes,

2000). The effects of multiple risk factors for COPD seem to be additive, so it is important to identify persons with more than one risk.

PREVENTION AND TREATMENT

Because cigarette smoking is the key causative factor in the development of chronic obstructive pulmonary disease, smoking cessation for those who smoke and avoiding second-hand smoke for non-smokers are key preventive measures. Minimizing exposure to occupational and environmental air pollutants, another important preventive measure, is best achieved through environmental and workplace safety regulations. Government agencies are charged with regulating worker exposure levels to harmful occupational pollutants. Regulation of outdoor air pollution in the U.S. began with the Air Pollution Control Act (1955) and Clean Air Act (1963) and its amendments (1970, 1977, and 1990). States and municipalities have passed clean indoor acts to prohibit indoor pollutants such as cigarette smoke and pesticides. Many municipalities issue air pollution alerts when air pollution levels exceed standards, so that persons with COPD know to limit unnecessary activities (Goldring et al., 1998).

COPD has been widely underdiagnosed in primary care settings (Voelkel, 2000), particularly among women (Chapman, et al., 2001). Signs and symptoms alone are not adequate for the diagnosis of COPD. Early detection can be performed by spirometry, lung airflow measurement, in a primary care setting. The National Lung Health Education Program recommends widespread use of spirometry by primary care providers for at-risk patients, that is, current smokers 45 years or older (Ferguson et al., 2000). If people are identified in the early and asymptomatic stages of COPD, interventions like smoking cessation can prevent further disease progression (Petty and Master, 2000; Ferguson et al., 2000). Smoking cessation can slow the progression of the disease, but no treatment can reverse the damage that has already occurred. Common medical interventions include inhaled corticosteroid and bronchodilator drugs, antibiotics, and home

oxygen therapy. Pulmonary rehabilitation—a structured program of education, exercise, breathing retraining, and psychosocial support—can improve the exercise capacity and quality of life of COPD patients (Barnes, 2000).

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15. ASTHMA

INTRODUCTION

Asthma is a chronic inflammatory disorder of the airways that become hypersensitive and undergo changes when stimulated by an allergen or other environmental trigger. There are gender differences in asthma development and treatment. Girls are at substantially lower risk than boys of developing asthma during childhood, but after puberty the risk in women increases until it is higher than that in men (deMarco et al., 2000). There is evidence that female high-risk asthma patients are admitted for hospitalization twice as often as male patients and tend to have longer admissions (Trawick et al., 2001). Research suggests that the size of a person's airway, in addition to hormonal factors, could explain the different patterns of asthma incidence in males and females (de Marco et al., 2000).

SCOPE OF THE PROBLEM

Approximately 1 in 16 persons (17 million) in the U.S. and 1 in 12 persons (266,000) in Connecticut reported having asthma in 1998 and 1999 (U.S. Department of Health and Human Services, 2000; Connecticut Department of Public Health, Bureau of Community Health, 2001). In Connecticut, adult women reported current asthma (9 percent) at a significantly higher prevalence rate than adult men (5 percent) (Connecticut Department of Public Health, Bureau of Community Health, 2001).

Asthma is not a leading cause of death, and mortality rates have remained stable over the past decade. However, more Connecticut females died from asthma than males (114 and 52 deaths,

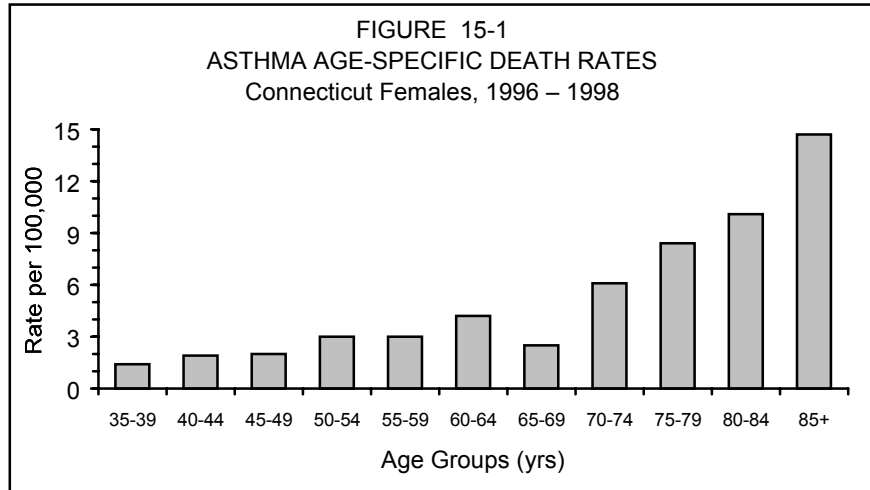
respectively) and the age-adjusted mortality rates from 1996 to 1998 were significantly higher for females than males (2 and 1 deaths per 100,000, respectively) (Mueller et al., in preparation).

Asthma prevalence can be measured according to the number of asthma and asthma-related conditions. Asthma may be secondary and related to a primary diagnosis of pneumonia, COPD, or other respiratory conditions. The age-adjusted hospitalization rate for asthma, as a primary diagnosis, decreased from 166 per 100,000 females in 1993 to 149 per 100,000 in 1997. Conversely, there was a significant increase in the hospitalization rate for all asthma-related diagnoses from 454 per 100,000 females in 1993 to 556 per 100,000 in 1997.

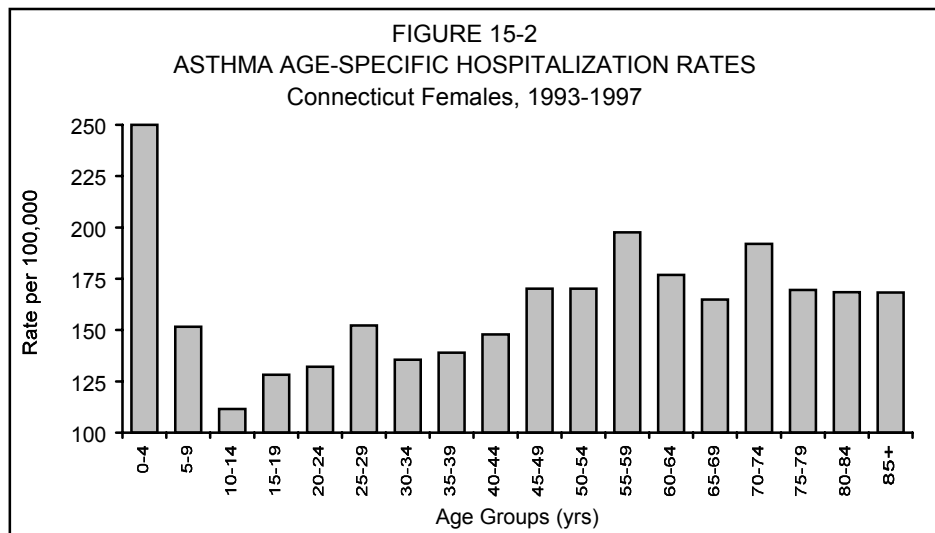
Age

Asthma mortality is low throughout the first seven decades of life, rising steadily thereafter. The highest asthma mortality rate occurs in females aged 85 years and older (Fig. 15-1).

Asthma mortality is highest for elder females, but the highest rate of asthma hospitalization occurs among children. Asthma, as a primary diagnosis, was the eighth leading cause of hospitalization for all females in Connecticut but third for females under 15 years of age (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001). This was due to the large number of hospitalizations of children under age 5. The age-specific hospitalization rate for females 0-4 years of age was higher than for any other age group from 1993 to 1997 (250 hospitalizations per 100,000 females) (Fig. 15-2).



Source: Mueller et al., in preparation.
Notes: U.S. Census Bureau population estimates used for rate calculations. Rates adjusted to the 2000 U.S. standard million population.



Source: Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001.
Notes: U.S. Census Bureau population estimates used for rate calculations. Rates adjusted to the 2000 U.S. standard million population.

Race and Ethnicity

There were significant differences in asthma hospitalization rates by race and ethnicity for Connecticut females. During the 5-year period from 1993 to 1997, the age-adjusted

hospitalization rate for black, non-Hispanic females, and all Hispanic females was significantly higher than the rate for white, non-Hispanic females. (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001) (Table 15-1).

TABLE 15-1
 ASTHMA HOSPITALIZATIONS BY RACE AND ETHNICITY
 Connecticut Females, 1993-1997

| Race/Ethnicity | Number of Discharges | Age Adjusted Hospitalization Rate (per 100,000) |
|-------------------------------|----------------------|---|
| All races | 13,403 | 158.6 |
| White, non-Hispanic | 7,452 | 104.4 |
| Black, non-Hispanic | 2,708 | 380.4 [‡] |
| Asian & PI, non-Hispanic | 48 | 30.5 [‡] |
| Native American, non-Hispanic | 11 | † |
| Hispanic/Latina | 3,043 | 546.4 [‡] |

Source: Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001.
[‡] Rate significantly different from that of whites (p< .05)
[†] Statistics not calculated for fewer than 25 events.
 Notes: U.S. Census Bureau population estimates used for rate calculations. Rates adjusted to the 2000 U.S. standard million population.

Racial and ethnic differences in asthma mortality cannot be reliably measured due to insufficient numbers of asthma deaths among black and Hispanic females between the two periods from 1989 to 1991 and 1996 to 1998 (Table 15-2). There were no asthma deaths among Asian and Pacific Islanders and Native Americans during these time periods.

for both the development of asthma in childhood and exacerbation of existing asthma.

Triggers that worsen asthma symptoms in susceptible persons include dust mites, pet dander, cockroach antigens, perfumes, and fungi (Institute of Medicine, 2000). Ambient air pollutants (e.g., ozone, sulfur dioxide, nitrogen dioxide, acid aerosols, and particulate matter),

TABLE 15-2
 ASTHMA DEATHS BY RACE AND ETHNICITY
 Connecticut Females, 1989-1991 and 1996-1998

| Race/Ethnicity | 1989-1991 | | 1996-1998 | |
|------------------------|------------------|---------------------------------------|------------------|---------------------------------------|
| | Number of Deaths | Age Adjusted Death Rate (per 100,000) | Number of Deaths | Age Adjusted Death Rate (per 100,000) |
| All races | 85 | 1.5 | 114 | 1.9 |
| White | 73 | 1.3 | 100 | 1.8 |
| African American/Black | 12 | † | 14 | † |
| Hispanic/Latina | 7 | † | 12 | † |

Source: Mueller et al, in preparation.
[†] Statistics not calculated for fewer than 15 events.
 Notes: U.S. Census Bureau population estimates used for rate calculations. Rates adjusted to the 2000 U.S. standard million population.

RISK FACTORS

Risk factors for the development of asthma are poorly understood, but likely include a family history of the disease, allergies, and environmental exposures. Environmental tobacco smoke is the leading modifiable agent

airborne allergens, and cold weather also exacerbate the condition. Medications like aspirin and some types of food, such as eggs in young children, can be asthma triggers due to allergic reactions. Stress, exercise, and emotional or psychological problems can stimulate asthma attacks or exacerbate the asthma condition.

Asthma in women is affected by hormonal changes related to menstrual cycles and pregnancy. In a recent study, one-third of emergency department visits for asthma among women occurred during the preovulatory phase of the menstrual cycle (days 5-11) (Zimmerman et al., 2000). Endometriosis also has been associated with allergic and immunological factors and may worsen asthma symptoms in women (Frieri, 1997).

Recent studies identify overweight and obesity as risk factors for asthma, particularly in females. Girls who become overweight or obese between the ages of 6 and 11 years have a sevenfold increased risk of developing new asthma symptoms at age 11 or 13 (Castro-Rodriguez et al., 2001).

ECONOMIC BURDEN

People with asthma can lead normal, productive lives with effective symptom management. However, inadequate control is costly, can have physical consequences, and can lower a person's quality of life. Annual direct health care costs for asthma total nearly \$10 billion in the U.S. and \$75 million in Connecticut (Connecticut Department of Public Health, 2001).

The indirect, nonmedical costs associated with asthma account for approximately 50 percent of total illness costs (U.S. Department of Health and Human Services, 2000). Indirect costs include days missed from work or school, caregiver expenditures, travel and waiting time, early retirement due to disability, and premature mortality. According to a national survey, asthma resulted in nearly half of children missing school, and more than one-quarter of adults missing work (Glaxo Wellcome, 1998).

PREVENTION AND MANAGEMENT

Asthma prevention has not been studied sufficiently, however, reducing early infections and early exposure to allergens or tobacco smoke are considered important interventions (U.S. Department of Health and Human Services,

2000). Asthma management reduces the likelihood of hospitalization, but requires a comprehensive approach that includes the use of asthma medication, reduction of specific environmental factors that trigger attacks, and patient education and self-management (Brownson et al., 1998). The leading medications for controlling asthma are corticosteroids, leukotriene-antagonists, and cromolyn. Short-acting bronchodilators are used to open airways quickly during an attack, whereas long-acting bronchodilators are considered an effective preventive treatment.

Public health interventions in Connecticut include environmental controls established through clean air legislation that reduce air pollution. Restriction of cigarette smoking in public places in Connecticut also reduces exposure to environmental tobacco smoke. Local housing codes do not directly address asthma risk factors, but they do regulate ventilation and moisture control, which are contributing factors to asthma.

Healthy People 2010 objective 24-8 recommends that states establish a surveillance system for tracking asthma mortality and morbidity, the impact of occupational and environmental factors on asthma, access to medical care, and asthma management (U.S. Department of Health and Human Services, 2000). No such comprehensive surveillance system currently exists.

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16. DIABETES

INTRODUCTION

Diabetes mellitus is a metabolic disorder in which the body is unable to produce or use insulin, a hormone it needs to convert food into energy. Diabetes affects approximately 70,000 Connecticut women aged 18 and over (Centers for Disease Control and Prevention, 1998), with another 35,000 women estimated to have, but not to have been diagnosed with the disease (U.S. Department of Health and Human Services, 1998). Between 1990 and 1998, the prevalence of diagnosed diabetes in the U.S. increased by 33 percent. Diabetes prevalence rates increased 70 percent for women aged 30 to 39 (Centers for Disease Control and Prevention, 2001).

Because symptoms develop gradually and severe symptoms may not occur for several years, diabetes is often undiagnosed or not recognized until a later stage. The disease primarily affects the circulatory system and increases the risk for a variety of disabling conditions including stroke, heart disease, arteriosclerosis (hardening of the arteries), kidney failure, blindness, and lower extremity amputations. Diabetes is the leading cause of non-traumatic amputations, blindness among working-aged adults, and end-stage renal disease. These disabling conditions contribute to a severe decrease in a person's quality of life (U.S. Department of Health and Human Services, 2000).

The economic burden of diabetes is enormous. It is a major cause of disability, morbidity, and mortality. Diabetes' direct medical costs and the indirect costs of lost productivity and premature mortality were estimated at \$1.2 billion in 1997 in Connecticut, or approximately \$12,000 per year for every person with diabetes (Connecticut Department of Public Health, Diabetes Control Program, 2000).

TYPES OF DIABETES

There are three types of diabetics. Approximately 5 to 10 percent of all people with diabetes have "type 1" diabetes, a condition that typically begins in childhood or adolescence and requires lifelong insulin treatment. The vast majority of people with diabetes (90 to 95 percent) have "type 2" diabetes, a condition that typically develops in adults over 30 who have a family history of diabetes, are overweight, or are physically inactive. Type 2 diabetes can be controlled through a combination of proper diet, weight loss, and exercise, although oral medications or insulin are often necessary. A third type, "gestational" diabetes, develops during pregnancy and can have harmful effects on both the mother and child because of elevated glucose levels. It is estimated that up to 4 percent of all women develop gestational diabetes during pregnancy. In most cases, blood glucose levels return to normal following the pregnancy. Women with gestational diabetes, however, have up to a 45 percent increased risk of recurrence with the next pregnancy and up to a 63 percent increased risk of developing type 2 diabetes in later life (Centers for Disease Control and Prevention, 2001).

SCOPE OF THE PROBLEM

Mortality due to diabetes and diabetes-related causes among Connecticut resident females is shown in Table 16-1. Because more people with diabetes die from complications of the disease rather than the disease itself, diabetes death rates alone understate the extent to which diabetes contributes to mortality. Between 1996 to 1998, more than 1,000 Connecticut women died as a direct result of diabetes. Almost four times that many women died from diabetes-related causes (Table 16-1).

Between 1993 and 1997, there were more than 1,800 hospitalizations of Connecticut females each year for diabetes as a primary diagnosis. People with diabetes, however, are often hospitalized for the complications of diabetes rather than for the disease itself, so these numbers understate the extent of total hospitalizations for diabetes. During the 1993 to 1997 period, there were 11 times as many hospitalizations for diabetes as a secondary diagnosis (105,620) as there were for diabetes as a primary diagnosis (9,462) among Connecticut females (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001).

Race and Ethnicity

Black women suffer a disproportionate number of deaths from diabetes. While diabetes was the seventh leading cause of death for all

Connecticut women, it was the fourth leading cause among black women for the period 1996 to 1998. During this time, the diabetes death rate of black women was more than twice that of white and Hispanic women (Table 16-1). Similarly, the diabetes-related death rate was highest for black women, more than twice that for white, and almost twice the rate for Hispanic women during this period (Table 16-1). There were too few diabetes and diabetes-related deaths among Asian and Pacific Islander and Native American women in Connecticut during these time periods to calculate reliable rates.

The diabetes death rate for Connecticut females increased significantly between the periods 1989 to 1991 and 1996 to 1998 (Table 16-1). Between 1989 and 1998, diabetes death rates increased by 2.5 percent per year. This change is accounted for by the increase in the death rate among white and black women. There

TABLE 16-1
DIABETES AND DIABETES-RELATED DEATHS
BY RACE AND ETHNICITY
Connecticut Females, 1989-1991 and 1996-1998

| Race/Ethnicity | 1989-1991 | | 1996-1998 | |
|--------------------------------|------------------|---------------------------------------|------------------|---------------------------------------|
| | Number of Deaths | Age Adjusted Death Rate (per 100,000) | Number of Deaths | Age Adjusted Death Rate (per 100,000) |
| DIABETES DEATHS | | | | |
| All races | 822 | 13.5 | 1,058 | 16.2* |
| White | 735 | 12.6 | 941 | 15.1* |
| African American/Black | 83 | 30.7 [‡] | 115 | 36.3 [‡] |
| Asian/Pacific Islander | 1 | † | 1 | † |
| Native American | 0 | † | 1 | † |
| Hispanic/Latina | 25 | 20.6 | 33 | 17.8 |
| DIABETES-RELATED DEATHS | | | | |
| All races | 3,784 | 61.5 | 3,949 | 59.1 |
| White | 3,453 | 58.4 | 3,516 | 55.1 |
| African American/Black | 314 | 117.7 [‡] | 416 | 134.2 [‡] |
| Asian/Pacific Islander | 4 | † | 11 | † |
| Native American | 5 | † | 6 | † |
| Hispanic/Latina | 80 | 60.1 | 133 | 73.3 [‡] |

Source: Mueller et al., in preparation.

* Change in rates from 1989-91 to 1996-98 period is statistically significant ($p < .05$).

† Statistics not calculated for fewer than 15 events.

‡ Rate significantly different from that of whites ($p < .05$)

Notes: U.S. Census Bureau population estimates used for rate calculations. Rates adjusted to the 2000 U.S. standard million population.

were no significant changes in diabetes death rates among Hispanic women during these time periods. Death rates due to diabetes-related causes (Table 16-1) remained unchanged between the 1989 to 1991 and 1996 to 1998 periods.

Hospitalization rates for diabetes for the period from 1993 to 1997 in Connecticut were highest among African American and black women, followed by Hispanic, and white women (Table 16-2). African American and black women had more than four times and Latinas almost twice the rate of hospitalizations for diabetes listed as a primary diagnosis compared with white women. There were too few hospitalizations of Asian and Pacific Islander and Native American women during this period to calculate reliable rates.

diabetes among black and non-Hispanic white women (Robbins et al., 2001). Low-income women are less likely to have adequate diet and physical activity, and appropriate medical care, factors known to affect the development and course of the disease. The relative importance of these and genetic factors in explaining the higher prevalence of type 2 diabetes in minority women is not well understood (Carter et al., 1996).

The risk of developing type 2 diabetes increases with age and usually develops after age 40, occurring when the body's cells become resistant to insulin. The exact cause of type 2 diabetes is unclear, although several factors have been linked to the risk of developing the disease (Table 16-3). Women at risk for developing type 2 diabetes include those who are overweight, have high blood pressure, and/or a sedentary

TABLE 16-2
DIABETES* HOSPITALIZATIONS BY RACE AND ETHNICITY
Connecticut Females, 1993-1997

| Race/Ethnicity | Number | Age Adjusted Rate (per 100,000) |
|------------------------|--------|------------------------------------|
| All races | 9,462 | 103.7 |
| White | 6,723 | 83.8 |
| African American/Black | 1,967 | 337.3 [‡] |
| Asian/Pacific Islander | 14 | † |
| Native American | 4 | † |
| Hispanic/Latina | 646 | 156.6 [‡] |

Source: Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001.

* Diabetes as a primary diagnosis.

† Statistics not calculated for fewer than 25 events.

‡ Rate significantly different from that of whites (p < .05).

Notes: U.S. Census Bureau population estimates used for rate calculations. Rates adjusted to the 2000 U.S. standard million population.

RISK FACTORS

Not everyone in the population is equally at risk for developing diabetes. While the prevalence of type 1 diabetes tends to be higher among white than among minority Americans, the prevalence of type 2 diabetes tends to be higher among black, Hispanic, and Native Americans than among whites (Brownson et al., 1998). Low socioeconomic status is strongly associated with higher prevalence of type 2

lifestyle, and who have a history of gestational diabetes. Findings from the Behavioral Risk Factor Surveillance Survey (1990-1996) indicate that among Connecticut women not diagnosed with diabetes: 25 percent are overweight, 52 percent have insufficient physical activity, and 20 percent have high blood pressure (Frost, 2000).

About 80 percent of the people with type 2 diabetes are obese at the time of diagnosis. Evidence suggests that the number of years of obesity, a high fat diet, and a lack of physical activity are risk factors for development of the

disease. Research findings also suggest that cigarette smoking may be another risk factor for development of the disease. For people who have already developed the disease, factors such as smoking and hypertension interact to increase the likelihood of complications like stroke and heart disease (Brownson et al., 1998).

clinical trial of prevention of type 2 diabetes conducted in Finland confirm this link. Researchers found that lifestyle changes reduced the risk of progression to type 2 diabetes in the intervention group by 58 percent over a four-year period, concluding that type 2 diabetes can be prevented by lifestyle changes in high-risk subjects (Tuomilehto, et al., 2001). Another major study, the diabetes prevention program, is currently underway in the U.S. and will be

TABLE 16-3
RISK FACTORS FOR TYPE 2 DIABETES

| | |
|---|---|
| ◆ | Family history of diabetes among parents or siblings |
| ◆ | History of obesity (≥ 20 percent over desired weight) |
| ◆ | History of hypertension (blood pressure $\geq 140/90$ mmHg) |
| ◆ | History of physical inactivity |
| ◆ | Previous test indicating Impaired Fasting Glucose or Impaired Glucose Tolerance |
| ◆ | History of gestational diabetes mellitus or delivery of babies over 9 pounds |
| ◆ | Age of 40 years or older |

Source: Brownson et al., 1998.

PREVENTION AND TREATMENT

Clinical recommendations for the treatment of diabetes emphasize preventing the complications of the disease. Studies have documented that diabetes can be controlled by maintaining blood glucose at normal levels through diet, exercise, and oral medications or insulin injections (Padgett et al., 1988; Clement, 1995). Such self-management measures can reduce some long-term complications of the disease such as retinopathy and neuropathy (eye and nerve damage). Blood glucose monitoring is an essential part of self-management because it can indicate progress or impending problems related to the disease. Management of diabetes is a life-long process that requires knowledge and active involvement of the woman with diabetes in following diet and related lifestyle practices, and regular assessments by her health care team.

The evidence supporting the feasibility of primary prevention of diabetes (that is, preventing the disease before it develops) is accumulating. Type 2 diabetes has long been associated with overweight, physical inactivity, and dietary habits. Findings from a recent

completed by 2002 (Diabetes Prevention Program Research Group, 1999). Findings from these key prospective studies should provide valuable information for the implementation of programs aimed at the prevention of type 2 diabetes.

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17. OSTEOPOROSIS

INTRODUCTION

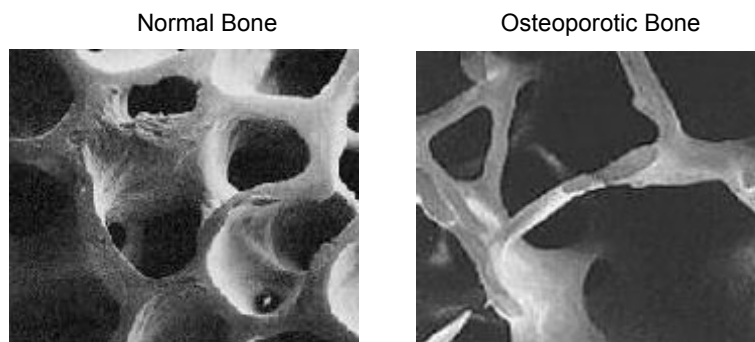
Osteoporosis is a serious, degenerative bone condition affecting the health of approximately 23 million American women. More than 8 million women have the disease, while over 14 million have osteopenia or “low bone mass” placing them at increased risk of developing osteoporosis (National Osteoporosis Foundation, 2001a).

The development of maximum bone strength and density begins in early childhood, and the skeletal bones reach their peak density by about age 30. Throughout life, bone is constantly being removed and replaced with new bone in a process called remodeling. However, as the body ages, too much bone may be removed

causing rapid mineral depletion and thinning of the bones. Bone loss associated with aging averages about 0.5 percent a year, and increases to about 1 to 2 percent a year at menopause. The average women will lose 20 percent of her bone mass between the ages of 40 and 70.

While almost everyone experiences some bone loss with age, osteoporosis is not a normal part of aging and should not be ignored (National Osteoporosis Foundation, 1997). Another cause of osteoporosis, called secondary osteoporosis, may develop following long-time use of oral steroid drug therapy for other diseases and conditions. Figure 17-1 presents micrograph examples of normal and weakened osteoporotic bone.

FIGURE 17-1
OSTEOPOROTIC BONE MICROGRAPHS



Reproduced from *J Bone Miner Res* 1986; 1:16-21 with permission of the American Society For Bone and Mineral Research.

or not enough new bone replaced or both. This leads to bone deterioration, low bone mass, osteoporosis, and an increased risk of bone fracture. The lack of optimal bone development in childhood is an important consideration

Declining hormone levels in women at the time of menopause, inadequate calcium intake during child- and young adulthood, and a lack of weight-bearing exercise all affect the bone remodeling process negatively. The loss of estrogen through natural menopause or surgical removal of the ovaries accelerates this process,

SCOPE OF THE PROBLEM

Osteoporosis is accurately named the “silent disease” because it weakens and thins bones without early warning signs or symptoms. The first sign of the disease is often a sudden fracture of the hip or spine. In advanced osteoporosis, the bones are so fragile that they fracture with only slight exertion, such as picking up a grocery bag, a child, or merely bending over. In the first five to seven years after menopause, bone density

may decline by as much as 20 percent (National Institutes of Health, 2000a). One in two women over 50 will have an osteoporosis-related fracture during her lifetime (National Institutes of Health, 2000a) and almost all fractures in older adults are due in part to low bone density (National Osteoporosis Foundation, 2001a).

Nationally, osteoporosis is responsible for 1.5 million fractures per year or one fracture every 20 seconds (National Osteoporosis Foundation, 2001b). The consequences of osteoporotic fractures can be severe, and cause major lifestyle changes and a diminished quality of life, especially for older women. The collapse of weakened bone structures in the spine or hip may result in physical deformity, lifelong pain, the loss of independence, and the need for long-term care.

Prevalence

Eighty percent of people with osteoporosis are women. Females experience hip fractures at a rate two to three times higher than males. One in five persons dies within a year of sustaining an osteoporotic hip fracture (National Institutes of Health, 2001). A woman's lifetime risk for an osteoporosis-related hip fracture is equal to her risk of breast, uterine, and ovarian cancer combined (National Osteoporosis Foundation, 2001a).

The consequences of an osteoporosis-related hip fracture can be severe. Twenty-four percent or one in every five persons who sustains an osteoporotic hip fracture dies within the first year (National Osteoporosis Foundation, 1999). Fifty percent will be unable to walk alone, while another 25 percent will require long-term nursing care (National Institute of Arthritis and Musculoskeletal and Skin Diseases, 2000).

In 1996, the estimated osteoporosis prevalence rate for Connecticut females age 50 and over was 213 per 1,000 females compared to the U.S. prevalence rate of 209 per 1,000. For females age 50 and over with osteoporosis and

low bone mass, the estimated prevalence rate was 617 per 1,000 females in 1996, compared with the U.S. rate of 611 per 1,000 (National Osteoporosis Foundation, 1997). By 2015, the number of Connecticut females with both osteoporosis and low bone mass is expected to increase by 36 percent, from 316,613 to 429,000 (National Osteoporosis Foundation, 1997).

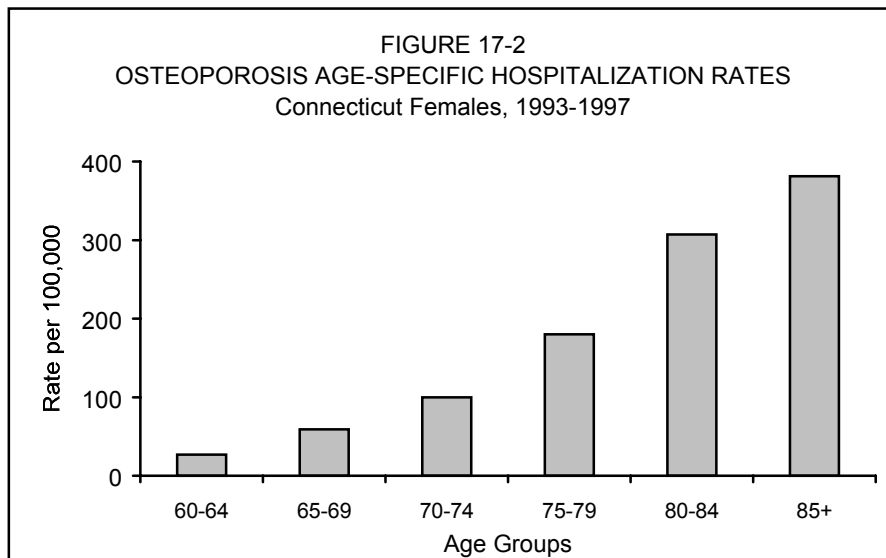
Hospitalization

Between 1993 and 1997, there were 2,827 females discharged from Connecticut acute care hospitals with a primary diagnosis of osteoporosis and 17,046 female discharges that were osteoporosis-related. The age-adjusted hospitalization rates for black, non-Hispanic females (8 per 100,000) and all Hispanic females (7.5 per 100,000) were significantly lower than the rate for white, non-Hispanic females (27 per 100,000) (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis).

As shown in Figure 17-2, the osteoporosis hospitalization rate nearly doubles between each five-year age group beginning at age 60. Females aged 85 and over were hospitalized for osteoporosis at about three times the rate of females aged 70 to 74.

Total Connecticut hospitalization charges for osteoporosis between 1993 and 1997 were a little over \$38 million (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001). When all osteoporosis-related discharges are considered, the total five-year hospitalization charges reached \$234,174,766.

Nationwide, more than 1.5 million fractures occur annually as a result of osteoporosis including 300,000 hip fractures and 700,000 vertebral fractures. Costs associated with osteoporotic fractures in the United States is estimated at \$13.8 billion a year or \$38 million each day (National Institutes of Health, 2000a).



Source: Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001.
 Notes: Osteoporosis as a primary diagnosis.
 U.S. Census Bureau population estimates used for rate calculations. Rates adjusted to the 2000 U.S. standard million population. Based on OA primary diagnoses only.

RISK FACTORS

A woman’s risk for osteoporosis increases with the number of her risk factors. Table 17-1 presents the modifiable and non-modifiable risk factors for osteoporosis.

Persons of all ages can develop the disease, but females are at greater risk and the risk grows with age. Women with a family history of the disease, as well as those who have a small body frame or experience early menopause are at

greatest risk of developing the disease.

PREVENTION

Osteoporosis is highly preventable and treatable, but as of now there is no cure. Prevention efforts are targeted toward two processes in a woman’s life: the development of a greater peak bone mass early in life and slowing the rate of bone loss after menopause.

TABLE 17-1
OSTEOPOROSIS RISK FACTORS

| NON-MODIFIABLE | MODIFIABLE |
|--|---|
| ◆ Female | ◆ Current cigarette smoking |
| ◆ Advanced age | ◆ Low calcium intake (lifelong) |
| ◆ Family history of osteoporosis | ◆ Low vitamin d intake |
| ◆ Small body frame | ◆ Low body weight (under 127 pounds) |
| ◆ Caucasian or Asian race | ◆ Anorexia |
| ◆ Estrogen deficiency | ◆ Lack of weight-bearing exercise |
| ◆ Early menopause (before 45) | ◆ Long-time use of certain medications (steroids, anti- |
| -from surgical removal of both ovaries | convulsants, excessive thyroid hormone, etc.) |
| -abnormal absence of menstrual periods (>1 yr) | |

The critical window of opportunity for building peak bone mass is childhood and young adulthood, the primary bone-building years. From birth through approximately age 30, the skeletal bones increase in size and density. Bones need sufficient calcium, 400 and 800 IU of Vitamin D, and regular weight-bearing exercise on a daily basis to reach peak bone mass. If calcium consumption is too low to meet the body's metabolic needs, calcium will be taken from the bones. Women also need extra calcium when they are pregnant or breastfeeding.

Physical activity is important for building bone strength, agility, and balance, and should include weight-bearing exercise, such as walking, stair climbing, running, cross-country skiing, gardening, dancing, and weight lifting. The NOF also cautions women to limit their intake of caffeine beverages and soft drinks, as caffeine may interfere with calcium absorption (National Osteoporosis Foundation, 1999). Smoking and excessive alcohol consumption negatively affect bone health, but both are modifiable risk factors.

SCREENING AND TREATMENT

Bone mineral density screening exams are relatively quick, painless, and noninvasive tests that can detect, predict, and monitor a person's risk for an osteoporosis fracture. These exams are covered by most insurance plans, including Medicare, which began covering the costs of bone density screening in July, 1998.

Although there is no cure for osteoporosis, there are a growing number of Federal Drug Administration (FDA) approved pharmaceuticals for preventing and treating the disease. Hormone replacement therapy (HRT), alendronate, risedronate, and raloxifene, are approved for the prevention and treatment of osteoporosis. Salmon calcitonin is approved only for the treatment of osteoporosis.

HRT is known to increase the risk of breast cancer and thrombosis in some patients (see Chapter 9 *Breast Cancer* and Chapter 6 *Coronary Heart Disease*). Women who are unable or unwilling to receive HRT therapy may

be helped by one of the other FDA-approved prevention or treatment pharmaceutical therapies.

Natural estrogens, especially those that are plant-derived, are popular osteoporosis prevention alternatives for many women, but their effectiveness is still under investigation (National Institutes of Health, 2000b).

Vertebral fractures are more common than hip fractures, but not as disabling. Two new, minimally invasive procedures called kyphoplasty have been developed to treat these types of fractures. In both techniques, bone cement is injected into the fractured vertebra to stabilize the spinal column and relieve pain. Additional studies are needed to determine the long-term effect of the procedure on adjacent vertebrae (National Institutes of Health, 2000b).

Osteoporotic hip fractures may require surgical intervention including total hip replacement. Up to one-fourth of these patients requires an extensive recovery period at home or in a nursing home. Hip fractures are discussed further in Chapter 25 of this report.

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18. OSTEOARTHRITIS

INTRODUCTION

Osteoarthritis (OA) is a chronic, degenerative joint disease and the most commonly occurring form of the more than 100 known types of arthritis and rheumatic diseases and conditions. An estimated 21 million adults (12 percent of Americans aged 25-74) have OA, and approximately 16 million (76 percent) are women (Arthritis Foundation et al., 1999).

While OA can occur in any body joint, it most frequently affects weight-bearing joints: the hips, knees, feet, and hands. OA affects the entire joint structure including the ligaments, muscles, bone, and soft tissue. The disease usually develops slowly and progresses in response to the breakdown of articular cartilage that cushions the ends of joint bones. Without cartilage, the bones directly rub against each other causing pain and inflammation. Cartilage inflammation stimulates the growth of bone spurs in the affected joint and small pieces of bone or cartilage can break off and float in the joint fluid, causing additional pain, inflammation, and damage.

Early in the development of the disease, OA pain is usually associated with movement or weight bearing, and disappears at rest. However, as the disease progresses, joint pain often becomes continuous. In addition to pain, persons with OA may experience morning stiffness, tenderness, swelling, and limited joint function with or without inflammation. Symptoms range from mild to very severe and profoundly affect the quality of everyday life and emotional well being of those with OA, their families, and caregivers. When severe, OA prevents people from exercising, going to school, or earning a living. It isolates its victims and robs them of their independence.

SCOPE OF THE PROBLEM

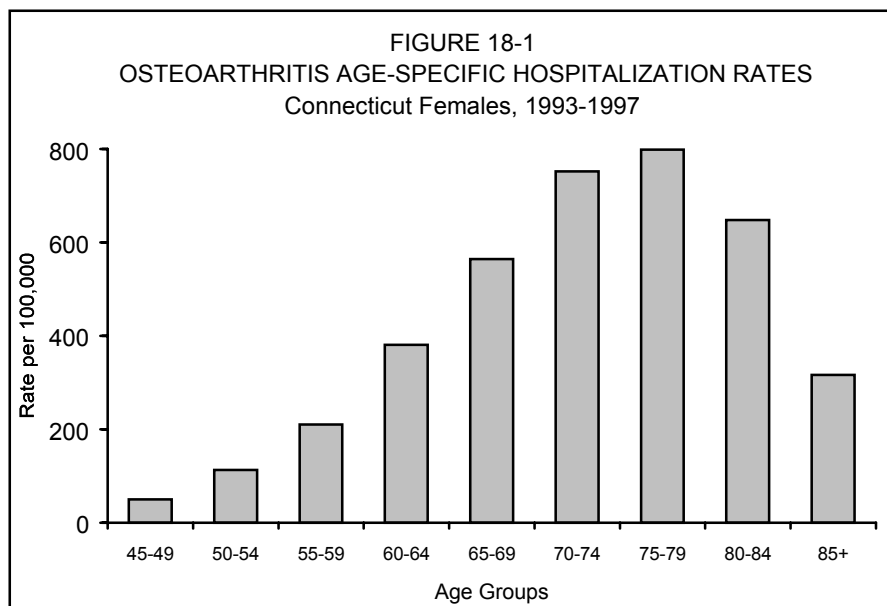
Prevalence

OA most frequently affects persons over 45, and its prevalence increases with age at least through age 74 (Brownson et al., 1998). One in two persons aged 65 or older shows x-ray evidence of OA in at least one joint, though they may be asymptomatic (Felson et al, 2000). OA is debilitating and the second most common reason after chronic heart disease leading to Social Security Disability payments because of long-term absence from work (La Plante, 1988). OA is expected to increase significantly by the year 2030 primarily because of the aging of the population.

Forty-two percent of adult females who responded to the 2000 Connecticut Behavioral Risk Factor Surveillance Survey (BRFSS) indicated that they had aching, stiffness, pain, or swelling in or around a joint during the last 12 months. Of these, 56 percent reported that the symptoms were present on most days for at least a month. Twenty-eight percent of adult women respondents indicated that a doctor had told them they had arthritis and one-third (36%) had been told they had OA. A surprising 42 percent of women responded that they did not know what kind of arthritis they had (Centers for Disease Control, 2000).

Hospitalization

Between 1993 and 1997, a total of 12,026 Connecticut females or a rate of 143 discharges per 100,000 were hospitalized with a primary diagnosis of OA. Eighty-one percent of these discharges were for females aged 74 and under. The frequency of female age-specific OA hospitalizations increased sharply between ages 45 and 79 during this same period (Fig. 18-1).



Source: Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001.
 Notes: U.S. Census Bureau population estimates used for rate calculations. Rates adjusted to the 2000 U.S. standard million population. Based on OA primary diagnoses only.

When all osteoarthritis-related females discharges are considered for the same time period, the total number more than triples to 36,428 or a rate of 342 per 100,000 (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001).

As shown below in Table 18-1, OA hospitalization rates in Connecticut between 1993 and 1997 were highest for black, non-

Hispanic women (124 per 100,000) and white, non-Hispanic women (122 per 100,000). These rates were three times higher than the rate of OA hospitalizations for Hispanic women (45 per 100,000) (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001).

TABLE 18-1
 OSTEOARTHRITIS* HOSPITALIZATIONS BY RACE AND ETHNICITY
 Connecticut Females, 1993-1997

| Race/Ethnicity | Number of Hospitalizations | Age Adjusted Hospitalization Rate (per 100,000) |
|------------------------|----------------------------|---|
| All races | 12,026 | 120.5 |
| White | 11,070 | 121.6 |
| African American/Black | 626 | 123.9 |
| Asian/Pacific Islander | 9 | † |
| Native American | 7 | † |
| Hispanic/Latina | 143 | 45.3‡ |

Source: Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001

Notes:

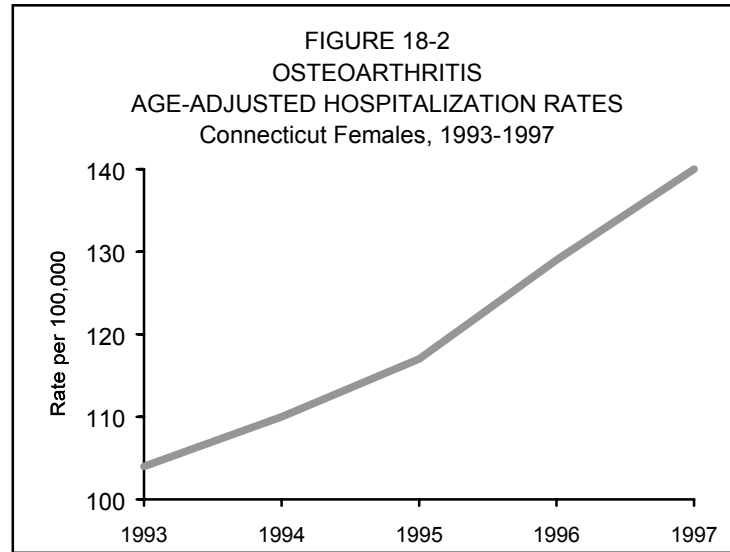
* Osteoarthritis as a primary diagnosis

† Statistics not calculated for fewer than 25 events.

‡ Rate significantly different from that of whites (p< .05)

Osteoarthritis age-adjusted hospitalization rate for females has significantly increased (7.8% annually) from 103.9 per 100,000 women in 1993 to 140.3 in 1997 (Fig. 18-2).

underlying cause for at least 50% of hand and hip OA cases. For example, females with bony node syndrome of the finger joints known as Herberden nodes are known to be at greater risk



Source: Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001.
Notes: U.S. Census Bureau population estimates used for rate calculations. Rates adjusted to the 2000 U.S. standard million population. Based on OA primary diagnoses only.

OA is second only to chronic heart disease as a leading cause of work disability (National Institutes of Health, 1998). The loss of wages and cost of treatments reduce the quality of life for OA patients, their families, and others who care for them. In Connecticut, inpatient hospitalization charges for females with a primary diagnosis of OA averaged \$18,773, and totaled \$244 million over the five-year period from 1993 to 1997. The total 5-year charges for Connecticut OA-related hospitalizations was \$528 million with an average charge of \$14,506 per discharge (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001).

RISK FACTORS

OA's non-modifiable and modifiable risk factors are shown below (Table 18-2). Inherited genetic factors predispose certain population groups to a higher risk for OA disease and are the

of developing OA of the hands (Brownson et al., 1998). Persons with congenital or developmental bone and joint diseases; prior inflammatory joint problems such as gout or rheumatoid arthritis; and those with metabolic diseases such as hyper- and hypo-parathyroidism and chondrocalcinosis are also at increased risk.

Advancing age and being female are two key non-modifiable risk factors leading to OA. Before age 50, men have a higher prevalence of OA in most joints, but after age 50, the trend reverses and more women are affected by OA of the foot, hand, and knee (National Institute of Health, 2000). On average, females have a higher number of OA afflicted joints than men (Brownson et al., 1998). Researchers expect OA to increase significantly by 2030, fueled by increasing longevity, especially of women, and the aging of the baby boomer generation.

TABLE 18-2
OSTEOARTHRITIS RISK FACTORS

| Non-Modifiable | Modifiable |
|---|------------------------|
| ◆ genetic disposition | ◆ joint trauma |
| ◆ female (over 50) | ◆ repetitive joint use |
| ◆ age | ◆ obesity |
| ◆ metabolic disorders and prior inflammatory diseases | |

A history of joint trauma is the strongest modifiable risk factor associated with the development of unilateral hip or knee OA (Brownson et al., 1998). Injuries in young adulthood or middle age are associated with a fivefold greater risk of developing knee OA. Younger people, especially those with a history of performing heavy, repetitive, or continuous physical work, or those with injuries including sports injuries are at increased risk of developing OA in later life (Felson, 2000).

Obesity is recognized as a significant, but modifiable risk factor for OA of the knee. Data from the 1992 Framingham Study showed that being overweight in young adulthood was a strong predictor of future development of OA in the knee. The study also showed that women who had lost an average of 11 pounds prior to the study decreased their risk of developing symptomatic knee OA by 50 percent (Felson et al., 1992).

PREVENTION

Primary prevention strategies for OA are limited, but effective. Reducing repetitive joint movement on the job, such as kneeling, squatting and heavy lifting helps protect joints and greatly reduces the risk for future OA development. At the same time, properly performed exercise and physical activity keeps joints limber and helps alleviate pain. Research suggests that obesity contributes to OA by placing increased mechanical stress on weight-bearing joint cartilage, especially of the knee. With each one pound increase in weight, the overall force across the knee in a single-leg stance increases by 2 to 3 pounds (Felson, 2000) emphasizing the need for reduction of body fat for persons with OA.

Nutrition plays an important part in OA prevention. Studies have shown there is an association between high intakes of vitamin C, less knee pain, and lower x-ray rates of OA (National Institute of Health, 2001). Sufficient levels of Vitamin D are also important and help protect against new and expanding OA disease. Supplements with Vitamin D may be needed during the winter months when there is less sunlight.

TREATMENT

There is no known cure for OA, but the disease is treatable with a number of different nonpharmacologic and pharmacologic options. Non-pharmacologic treatments include heat and cold therapies, braces, canes, and other adaptive devices. Range-of-motion, strengthening, and endurance exercises can help relieve pain and restore joint function. Equally important is rest and joint care to prevent overexercising an affected joint.

The most common pharmaceutical therapies for OA are aspirin, acetaminophen, topical pain-relieving creams, rubs or sprays applied directly to the skin, and non-steroidal anti-inflammatory drugs (NSAIDs) such as ibuprofen. Long term use of aspirin or NSAIDs, particularly by the elderly, is associated with stomach irritation, ulcers, bleeding and perforation. Overuse of NSAIDs can affect kidney function.

Acetaminophen is one of the safest analgesics for OA, but its use has been associated with adverse events in patients with existing liver disease. The Federal Drug Administration has approved two new NSAID drugs known as COX II (Cyclooxygenase) inhibitors for the treatment

of OA. While they cause less gastric irritation than NSIAD (COX I) medications, they can impair renal function and cause sodium and water retention. Intra-articular corticosteroid injections or opioid pain medication may be used when joint symptoms are severe or do not respond to other treatments.

Nontraditional approaches for treating OA include acupuncture, glucosamine, and chondroitin sulfate therapies. Studies on the efficacy of acupuncture for OA have been inconclusive to-date; however, a multi-site clinical trial funded by the National Institute of Health (NIH) is underway and expected to provide additional information in June, 2001. Glucosamine and chondroitin therapies have gained popularity in recent years and have shown positive results primarily in manufacturer-funded studies. An NIH funded, double-blind, placebo-controlled study of patients taking glucosamine alone, chondroitin sulfate alone, glucosamine and chondroitin sulfate together, or a placebo is underway and will be completed in 2004 (Felson, 2000).

Improvement in joint function is often achieved through a combination of rest and physical and occupational therapy. Range of motion, flexibility, muscle conditioning, and aerobic cardiovascular exercise are also important for maintaining or regaining joint function.

Tertiary treatment, in the form of surgery is considered if nonsurgical treatments are unsuccessful or unsatisfactory. There are four categories of surgical treatments currently in use: osteotomy, arthroscopy, arthrodesis and arthroplasty or total joint replacement. In early OA, osteotomy, (cutting and repositioning the affected bones) is sometimes used to alleviate symptoms and slow the progression of the disease. Arthroscopic debridement and lavage of the joint is also used to alleviate symptoms. In this procedure the surgeon visualizes the joint through small incisions and cleans debris out of the joint space. Joint fusion or arthrodesis is often used in the small joints of the spine, hand, foot and wrist, but is not appropriate for the major joints of the lower and upper extremities. Total joint replacement or arthroplasty is

considered the most effective of all medical interventions for OA, and is a cost-effective way to relieve pain and restore joint function in more severe OA cases. Usually, surgery is postponed as long as possible because the implants only remain functional for about 20 years (Felson, 2000).

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19. AUTOIMMUNE DISEASES

INTRODUCTION

The function of a healthy immune system is to produce antibodies that protect the body against “non-self” invaders such as viruses, bacteria, fungi, parasites, or malfunctioning (e.g., cancerous) cells. Autoimmune diseases (ADs) occur when the immune system fails to recognize the body’s own normal tissues, cells, or proteins as “self,” and begins to manufacture autoantibodies to attack them.

SCOPE OF THE PROBLEM

Clinical studies, data, and preventive strategies are limited or non-existent for many ADs (Quill, 1998). However, a recent University of Connecticut study demonstrated that the total 1995 U.S. death count for all ADs combined exceeded that for chronic liver disease and cirrhosis, the 10th leading cause of death, among women under age 65. In that study, multiple sclerosis and rheumatic fever were the two leading autoimmune diseases based on the underlying cause of death among women below age 65, but type 1 diabetes deaths were included only for women younger than 35 years. Systemic lupus erythematosus was the underlying cause of death for more women than any other autoimmune disease between ages 25 and 44, and type 1 diabetes, though only counted for ages 25 to 34, was second (Walsh and Rau, 2000). Collectively, ADs are the fourth major cause of disability among American women (National Women's Health Information Center, 2000).

Prevalence

Nationwide prevalence of autoimmune disorders has been estimated as ranging from at least 10 million (National Institutes of Environmental Health Sciences, 1999) to as many as 50 million people (American Autoimmune Related Diseases Association, 2000b). This discrepancy reflects the current lack of consensus surrounding which diseases meet the definition of autoimmunity. Also, correct diagnosis is not easy and may take place several years after medical care is first sought, and prevalence estimates may include undiagnosed cases. Several of the autoimmune diseases share some of the same symptoms.

AD disproportionately affects women. Seventy-five percent of all cases first appear in women between 15 and 44 years of age. Examples of ADs by body system are listed in Table 19-1.

Hospitalization

In Connecticut, hospitalizations are used to estimate the prevalence of the most severe cases of AD. For the five years between 1993 and 1997, over 2,300 women were hospitalized for a select number of ADs. Table 19-2 shows hospitalizations with an autoimmune disease among any of the first ten diagnoses listed on the hospital discharge abstract.

TABLE 19 - 1
 EXAMPLES OF AUTOIMMUNE DISEASES BY BODY SYSTEM

| | |
|-------------------------|--------------------------------|
| Neuromuscular Diseases | Connective Tissue |
| ◆ Multiple Sclerosis | ◆ Rheumatoid Arthritis |
| ◆ Myasthenia Gravis | ◆ Systemic Lupus Erythematosus |
| ◆ Fibromyalgia | ◆ Sicca/Sjogren Syndrome |
| Blood and Blood Vessels | Endocrine |
| ◆ Pernicious Anemia | ◆ Graves' /Hyperthyroidism |
| ◆ Hemolytic Anemia | ◆ Hashimoto's Thyroiditis |
| ◆ Temporal Arteritis | ◆ Juvenile Diabetes Type I |
| Gastrointestinal System | Skin Diseases |
| ◆ Autoimmune Hepatitis | ◆ Psoriasis |
| ◆ Crohn's Disease | ◆ Vitiligo |
| ◆ Ulcerative Colitis | |

Source: American Autoimmune Related Diseases Association, 2000a.

TABLE 19-2
 AUTOIMMUNE DISEASES
 HOSPITALIZATIONS AND CHARGES
 Connecticut Females, 1993-1997

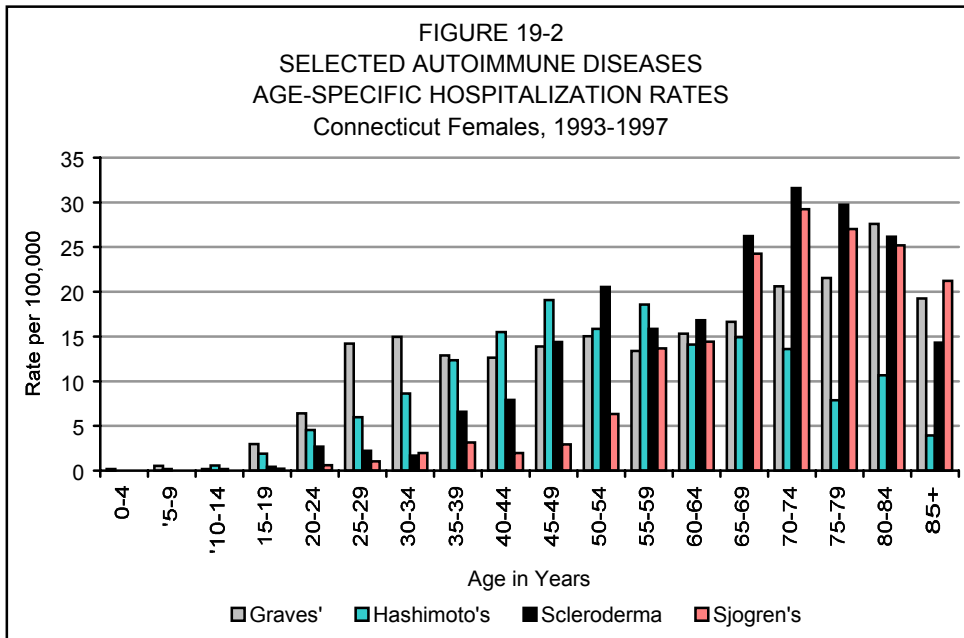
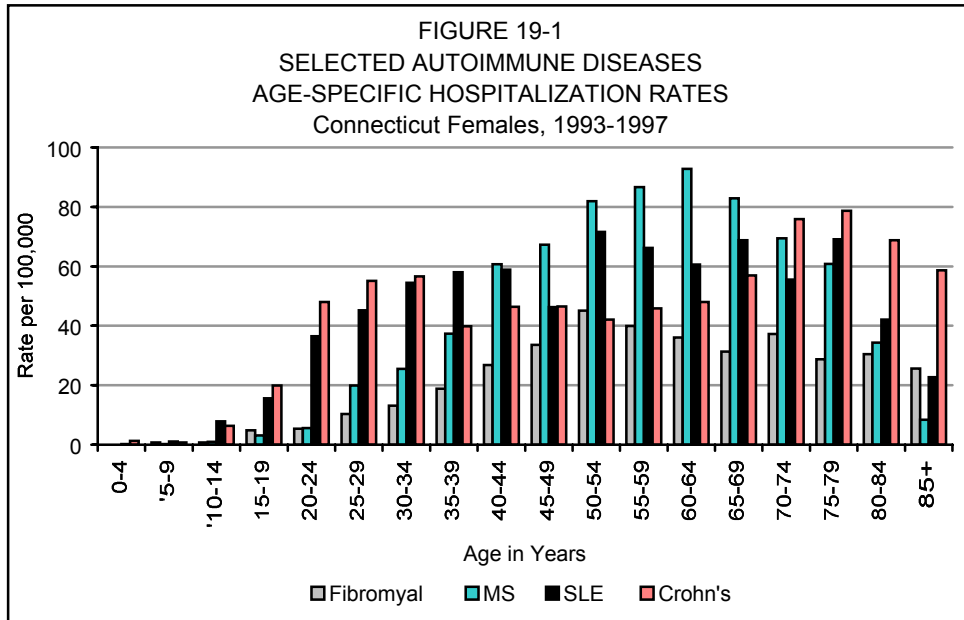
| Autoimmune Condition | Hospital Discharges | Age-Adjusted Rate | Mean Charge, \$ | Total Charges, \$ |
|-------------------------|---------------------|-------------------|-----------------|-------------------|
| Rheumatoid Arthritis | 9,334 | 92.6 | 13,438 | 125,434,357 |
| Lupus Erythematosus | 3,575 | 41.0 | 14,059 | 50,262,256 |
| Crohn's Disease | 3,434 | 38.5 | 13,238 | 45,458,701 |
| Multiple Sclerosis | 3,212 | 37.0 | 13,059 | 41,945,337 |
| Ulcerative Colitis | 1,692 | 18.3 | 15,099 | 25,548,318 |
| Fibromyalgia | 1,632 | 18.7 | 10,256 | 16,738,346 |
| Graves' Disease | 955 | 10.5 | 10,239 | 9,778,654 |
| Scleroderma | 805 | 8.8 | 16,353 | 13,163,964 |
| Hashimoto's Thyroiditis | 775 | 9.0 | 8,638 | 6,694,448 |
| Sjogren's Syndrome | 564 | 5.8 | 12,953 | 7,305,344 |
| Not Otherwise Stated | 88 | 1.0 | 18,144 | 1,596,673 |

Source: Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001.

Note: Diagnoses are not mutually exclusive and discharges rather than people were counted. Rates are per 100,000 women per year, adjusted to the 2000 US population standard.

Figures 19-1 and 19-2 show the hospitalization rates in Connecticut women for selected autoimmune diseases, according to age. Note that the vertical scales differ between the

figures. Declining rates for multiple sclerosis after age 64 may reflect early mortality. Rates for rheumatoid arthritis, which are not shown, increased with each age group.



RISK FACTORS

As indicated in Table 19-3, ADs have been linked to genes, infections, and the environment (Smith and Germolec, 1999). ADs frequently occur in one form or another within a family. The Human Leukocyte Antigen (HLA) regions on chromosomes are associated with several autoimmune diseases. Prenatal exposure to certain immunotoxicants may play a role in compromising autoimmunity in adult life (Holladay, 1999). A hormonal component is also

suspected, because some types of AD worsen during pregnancy (lupus) while others improve (Hashimoto's thyroiditis, Graves' disease, rheumatoid arthritis) only to worsen when the baby is delivered. It should be noted that in a comparison of all relevant studies, silicone breast implants were not associated with connect tissue diseases, including rheumatoid arthritis, systemic lupus erythematosus, scleroderma, and Sjogren's syndrome (Janowsky et al., 2000).

TABLE 19-3
GENDER RATIO AND RISK FACTORS FOR SELECTED AUTOIMMUNE DISEASES

| Disease | Female to Male Ratios | Heritable Pattern | Environmental and Other Risk Factors |
|------------------------------|-----------------------|----------------------------|--|
| Rheumatoid Arthritis | 4:1 | Strong genetic | Infectious arthritis, gout, repeated injuries, obesity, age, occupational exposure to silica dust. |
| Systemic Lupus Erythematosus | 9:1 | Strong genetic, black race | Ultraviolet light, hormonal factors, industrial chemicals; stress may cause a relapse. |
| Multiple Sclerosis | 2:1 | Strong genetic | Exposure to a virus or bacteria, smoking. |
| Crohn's Disease | >1:1 | Genetic, Jewish | Lack of earlier exposure to infections. |
| Ulcerative Colitis | <1:1 | Genetic | Same as Crohn's disease. |
| Fibromyalgia | 6:1 | ? | Injury, trauma, stress, or a virus. |
| Graves' Disease | 7:1 | Strong familial | Physical or emotional stress. Environmental factors may trigger. |
| Scleroderma | 3:1 | Black race | Occupational exposure to silica dust or vinyl chloride. Childbearing years. |
| Hashimoto's Thyroiditis | 50:1 | Strong familial | Iodine intake, lithium, age; subacute form may be caused by a virus. |
| Sjogren's Syndrome (Sicca) | 9:1 | Strong familial | Mid-adult years. |
| Type 1 Diabetes | 1:1 | Strong genetic | Viruses may trigger. |

Sources: Cooper et al., 1999; Autoimmune Related Disease Association, 2000a; National Institute of Environmental Health Sciences, 2000; Dayan and Daniels, 1996; Reveille and Arnett, 1992; Steenland et al., 2001; Karlinger et al., 2000.

PREVENTION AND TREATMENT

At present, there is no known way to prevent autoimmune diseases. However, with proper medication and careful monitoring, many people are able to live fairly normal lives. There are four general immunologic approaches to autoimmune disease treatment: altering thresholds of immune activation, modulating antigen-specific responses, reconstituting the immune system with autologous (one's own) or allogeneic (genetically different) stem cells, and sparing of target organs (Davidson and Diamond, 2001). Non-surgical treatment for the rheumatic ADs (e.g., rheumatoid arthritis, lupus, and scleroderma) includes healthy diet, prompt treatment of infections, immunosuppressants, corticosteroids, and anti-inflammatory medications. Current treatment protocols for the more severe outcomes of ADs include organ transplants and surgery to repair or replace damaged joints.

It is predicted that genetic research will enable earlier and more precise diagnosis of ADs and highly individualized drug treatment (Koopman, 2001; Kimberly, 2001).

SELECTED AUTOIMMUNE DISEASES

The following auto-immune diseases, discussed below, either affect large numbers of people, or are associated with high mortality rates.

- ◆ Rheumatoid Arthritis
- ◆ Systemic Lupus Erythematosus
- ◆ Multiple Sclerosis
- ◆ Irritable Bowel Disease
- ◆ Type 1 Diabetes
- ◆ Psoriasis
- ◆ Fibromyalgia and Chronic Fatigue Syndrome

Rheumatoid Arthritis (RA)

Rheumatoid arthritis causes pain, swelling, warmth, redness, and stiffness in the joints due to immune attack upon the joint capsule. An antibody called rheumatoid factor is often detected in laboratory tests. About a quarter of

patients develop rheumatoid nodules, bumps under the skin, near joints. The same joints on both sides of the body are usually affected, and the proximal (closer to the hand) rather than the distal finger joints. Symptoms are worse upon arising from rest, and vary from day to day. Change in weather can precipitate a painful episode, called a flare. As RA progresses, the cartilage and bone destruction are readily visible on x-rays.

More than two million Americans have RA. Recently the number of cases appears to have decreased slightly. The peak incidence occurs between ages 35 and 45, while the prevalence of RA increases with age. After ten years with the disease, about half of patients are unable to work. Mortality rates are increased two-fold in persons with RA (Koopman, 2001).

As with many diseases, RA results from a combination of genetic and environmental factors. Hormones, bacteria, and viruses may be involved.

Drug treatment of RA is intended to reduce pain and inflammation and improve function. Non-steroidal drugs such as aspirin have long been used. Methotrexate is now considered the first-line drug (Davidson and Diamond, 2001). Gold, antimalarial drugs, penicillamine, and sulfasalazine are sometimes prescribed to alter the course of rheumatoid arthritis. They require monitoring and have side effects. Corticosteroids and immunosuppressants, when used, require close monitoring. Alcohol may interfere with medications. Overuse of injected steroids may accelerate joint destruction.

Biologics are based upon substances naturally occurring in the body, and are used against specific aspects of the inflammatory process. A tumor necrosis factor inhibiting biologic was more effective than the standard RA treatment in a recent trial, but the cost of \$10,000-\$12,000 per patient per year is the chief barrier to entry into general use (Klippel, 2000). Omega-3 fatty acids, which are found in some fish or plant seed oils, may reduce inflammation in RA. However, some people may not tolerate the large quantities of oil needed to reach a benefit (National Institute of Health, 2000).

Rest and exercise are both needed at different times. Moist heat to an arthritic joint before exercise and a cold pack afterwards may facilitate exercise. Swimming is an activity that does not put much weight onto joints. Emotional stress is not thought to bring on rheumatoid arthritis or flares, but relaxation techniques are helpful. Surgery, including tendon reconstruction and joint replacement, may be necessary to relieve pain, correct deformity, and improve function.

Systemic Lupus Erythematosus (SLE)

Lupus erythematosus is found in three major forms. The systemic form is the most common, and is described in more detail below. The discoid form primarily affects the skin in sun-exposed areas, with red, scaly, circular rashes and possible scarring. Lupus may be induced by some prescription medications including procainamide (for heart problems), hydrazine (for hypertension), and dilantin (for seizures). There is no gender difference in the incidence of drug-induced lupus, and this form usually goes away when the causative drug is stopped.

SLE is a multi-organ autoimmune disease characterized by hyperactivity of immune B cells with overproduction of antibodies, and the deposition of immune complexes in organs, resulting in inflammation and damage. Periods of disease “flares” are followed by periods of remission. There is substantial mortality from coronary heart disease and kidney disease. Between ages 35 and 44, coronary artery disease may be 50 times as common among women with SLE than women without it (Karrar et al., 2001). Prior to the advent of dialysis, severe kidney damage from lupus was fatal.

It is possible that SLE represents several diseases with common final pathways (McQuire and Lambert, 1997). Several genes may increase susceptibility to SLE. When one twin has SLE the other twin has a 30 percent chance of also getting it, which shows that both genetic and environmental factors are involved. SLE is diagnosed by the sequential or simultaneous appearance of at least four features from a list of eleven. Prominent features include butterfly rash

across the nose and cheeks, photosensitivity, fever, fatigue, oral ulceration, painful swelling in two or more joints, chest pain on breathing, heart, kidney, neurologic, bleeding, and immunologic disorders, and especially antibodies against DNA or other parts of the person’s own cells. Symptoms may change over time in a person.

It is estimated that 240,000 Americans are living with lupus. The highest incidence rates for SLE are between ages 20 and 40. SLE is ten times more common among women than men during the female reproductive years, and about twice as common in women as men, after the age of menopause. Worldwide, the highest rates of disease have been reported in Afro-Caribbean, Chinese, Asian, and South American Indian women (Snaith and Isenberg, 1996). In the United States, the incidence rate in black women is three to four times that of white women (Hochberg, 1993), and the rates in Hispanics (Kimberly, 2001) and Native Americans (Peschken and Esdaile, 2000) are also higher than white Americans. Both incidence and length of survival continue to increase (Ruiz-Irastorza et al., 2001).

SLE disease activity, SLE disease damage, and poverty were independently associated with mortality in a multiethnic cohort (Alarcon et al., 2001).

Ultraviolet light in the 295-305 nanometer range is toxic to SLE patients, and sunlight can trigger a flare. Lupus patients metabolize estrogen and testosterone abnormally, and it is thought that pregnancy, menses, and contraceptives containing estrogen may alter the course of SLE (Ahmed and Tal, 1993). The role of retroviruses and bacterial infection in SLE are being studied.

Treatment consists largely of nonsteroidal anti-inflammatory drugs (for fever and arthritis), corticosteroids (injection, oral, or cream), immunosuppressants (including anti-malarial drugs), prompt attention to infections, avoidance of direct sunlight, and reduction of stress. These drug treatments have side effects. Hormone and other treatments are under investigation. Patients can improve their quality of life by learning to

recognize warning signs of SLE flares and how to manage their disease with help from doctors, friends, family, and support organizations (National Institutes of Health, 2001).

Multiple Sclerosis (MS)

Multiple sclerosis is the most common chronic neurologic disease of adults between ages 20 and 50. Immune T cells react with both a peptide from the autoantigen myelin basic protein and peptides from the Epstein-Barr virus, influenza type A, and human papillomavirus (Davidson and Diamond, 2001). Plaques or lesions of the myelin sheath surrounding the neuron characterize MS. Symptoms include disturbances of vision, balance, sensation, bowel and bladder function, tremor, and weakness. The diagnosis may be established using magnetic resonance imaging of the brain and cerebrospinal fluid analysis in addition to clinical signs. Common disabilities include difficulty bathing, dressing, and climbing stairs, sexual dysfunction, and inability to drive, but many MS patients continue to work full time (McDonnell and Hawkins, 2001). Depression and suicide are not uncommon. 20 percent of cases are chronic and progressive, the rest are intermittent and relapsing.

Worldwide, the prevalence of MS increases with distance from the equator, in most ethnic groups (Brownson et al., 1998). In the United States there are between 250,000 and 350,000 patients (Noseworthy et al., 2000). If a person has MS, the risk of MS in an identical twin is 31 percent and in a non-identical twin, 5 percent. The absolute risk of MS in a first-degree relative is less than 5 percent, and 85 percent of MS patients do not have an affected relative (Jr and Sriram, 2001); however, this is 20-40 times the risk in the general population.

There is no consensus about the early causes of MS. Although infectious causes of MS have been suggested, and *Chlamydia pneumoniae* in cerebrospinal fluid has been associated with MS, this organism has also been found in other neurological diseases (Gieffers et al., 2001). In the Nurses' Health Study, cigarette smoking was

associated with a 60 percent increase in risk (Hernan et al., 2001).

Corticosteroids are often used to treat MS. Interferon has a proven benefit in established MS and also appears effective as initial therapy (Jacobs et al., 2000).

Irritable Bowel Disease (IBD)

Irritable bowel disease or syndrome is also known as chronic ileitis, regional enteritis, or granulomatous colitis. IBD is a chronic, relapsing and remitting inflammatory disease of the digestive tract. Diarrhea or constipation may be present with abdominal pain. Ulcerative colitis affects the superficial cell layer of the colon and Crohn's disease affects several cell layers, but they are otherwise quite similar. Both begin relatively early in life and while not fatal, are associated with increased gastrointestinal cancer incidence and with two-fold increased mortality rate. Depression or emotional stress are common antecedents (Berkow and Talbot, 1977).

Worldwide the prevalence of IBD has been increasing (Karlinger et al., 2000). It is estimated that 830,000 Americans have IBD, and 45,000 new cases occur each year, with no gender predilection (Blumberg and Strober, 2001).

IBD is a disease of urban areas, and also has a genetic element that leads to abnormal T helper cell function. The immune system normally tolerates certain intestinal bacteria which live in symbiosis with humans and aid the digestive process. In IBD the immune system appears to target these bacteria. One theory is that in urban environments people may not encounter the microbial antigens necessary to proper immune system maturation, due to improved hygiene, vaccination, antimicrobial medication, and the decline in consumption of naturally fermented and dried foods (Isolauri, 2001).

Diagnosis involves, among other things, ruling out food allergies and infection by bacteria other than normal intestinal flora. Treatment may involve steroids (which have side effects), or even removal of part of the colon. In order to

withdraw steroids, or in patients who become refractory to steroid treatment, immunomodulators, tumor necrosis factor-alpha antibody, or budesonide have been used (Lichtenstein, 2001). Stress and diet should be monitored to prevent relapses. Antibody-based treatments have been initiated, based upon animal studies. Future research will involve genetic and molecular studies, possibly for identification of therapies based upon harmful and protective bacteria strains for genetic subsets of patients (Blumberg and Strober, 2001). More simply, "probiotic" diets that have been developed to treat acute diarrhea have the potential to reinforce normal gut function (Isolauri, 2001).

Type 1 Diabetes

Type 1 diabetes is also known as insulin dependent diabetes or juvenile onset diabetes. It is the most common chronic disease of childhood. The coxsackie virus P2-C protein peptide resembles a peptide from the autoantigen glutamic acid decarboxylase (Davidson and Diamond, 2001). The cause of type 1 diabetes is autoimmune attack on the islet cells of the pancreas, which produce the insulin by which blood glucose is regulated. Symptoms include abrupt onset of unexplained thirst, weight loss with excessive urination, protein in the urine, and an absolute need for insulin to sustain life. Diagnosis is confirmed with a blood glucose test.

The prevalence of type 1 diabetes varies tremendously worldwide. In the United States 300,000 to 500,000 people are estimated to have type 1 diabetes, with 30,000 new cases annually. Seven percent of diabetes diagnosed between ages 30 and 74 years is adult onset type 1 diabetes. The incidence rate is higher among whites than blacks, and does not differ by gender (Laporte et al., 1995).

There is seasonal variation in type 1 diabetes incidence, which has been attributed to patterns of infection, nutrition, or hormones as triggers in genetically susceptible persons (Seikikawa and LaPorte, 1998). An international study is testing the hypothesis that geographic differences in diabetes risk reflect variation in

the frequencies of susceptibility genes (Dorman, 1997).

Diabetes affects especially the eyes, kidney, and neurologic system through microvascular disease, and the heart through accelerated atherosclerosis. Acute complications such as diabetic coma are the leading causes of death in early years (<10 years diabetes duration), renal disease during the middle years, and cardiovascular disease in persons with more than 30 years of diabetes duration. The mortality rate is higher among blacks than whites (Portuese and Orchard, 1995). Acute complications accounted for the differences, suggesting that some of the excess mortality may be preventable (Tull and Barinas, 1996; Lipton et al., 1999). Depression is common due to the progressive course of disabilities, and there is considerable stress upon the family of the affected child.

Persons with type 1 diabetes utilize more medical resources than those without diabetes. In the 1989 National Health Interview Survey, 17.4 percent of persons with type 1 diabetes and aged 18-44 reported that they had been hospitalized in the previous year. The risk of hospitalization was associated with the presence of diabetic complications (Aubert et al., 1995). Adolescent girls have somewhat more hospitalizations than boys (Cohn et al., 1997). In an insurance program, inpatient rates for the type 1 diabetic population were 8.3 times higher for established complications (e.g. myocardial infarction, heart failure, and coronary bypass surgery) than the nondiabetes population. Rates of use of physician services were about ten times higher for each of endocrinologists, ophthalmologists, and nephrologists (Laditka et al., 2001).

In Connecticut between 1993 and 1997, there were 2,574 hospital discharges with any diagnosis of type 1 diabetes among females aged less than 30 years. During these ages the annual hospitalization rate was 81 per 100,000. 1,263 of the 2,574 discharges were with a first diagnosis of type 1 diabetes, and the average charge among these patients was \$6,612 (Connecticut

Department of Public Health, Division of Policy, Planning, and Analysis, 2001).

Mortality rates from type 1 diabetes have declined markedly among person diagnosed in 1975-79 compared to 1965-69. This trend is consistent with the introduction of testing for glycosylated hemoglobin (which indicates average glucose levels over several weeks time), home glucose monitoring, and improved blood pressure therapy in the 1980's (Nishimura et al., 2001). The use of specialist care is associated with better glucose control and fewer complications, compared to care by a generalist. Receiving specialist care is associated with income, education, health insurance, and female gender (Zgibor et al., 2000).

The management of diabetes involves control of blood sugar levels through monitoring and insulin administration, frequent medical attention, control of risk factors for heart disease (smoking, hypertension, lipid levels) and early treatment of complications. Pregnancy was formerly discouraged in women with type 1 diabetes but it can now be considered, if the diabetes is well managed. Tight glucose control delays the incidence and progression of microvascular disease (Diabetes Control and Complications Trial Research Group, 1993). The danger of trying to maintain glucose levels close to normal is that of going too low and bringing on hypoglycemic shock, coma, and death. One advance in control is the insulin pump, which delivers insulin without injection. If advanced atherosclerotic disease can be detected before clinical symptoms appear, vigorous risk factor management may avert a catastrophic cardiac event. Promising screening modalities include ankle blood pressure, subtle electrocardiographic changes, and measures of insulin resistance (Olson et al., in press).

Attempts to avoid the need for insulin administration have included pancreas or islet cell transplants, combined with immunosuppressant drugs. Newer gene therapy approaches are being investigated. These attempt to block the immunologic attack on islet cells before diabetes appears (Giannoukakis et al., 1999), and may be thought of as a vaccine.

Psoriasis

Psoriasis is a chronic, recurrent skin disease, of which several varieties are recognized. Severity varies from a few lesions to widespread scaling. In exfoliative psoriatic dermatitis, the entire skin is red and covered with scales. Pustular psoriasis is characterized by sterile pustules on the palms and soles, or may be generalized. Psoriatic arthritis resembles rheumatoid arthritis and may be crippling. When patients were asked to evaluate their quality of life, the impact of psoriasis on physical and mental functioning was comparable to that of cancer, arthritis, hypertension, heart disease, diabetes, and depression (Rapp et al., 1999).

Estimates of *psoriasis vulgaris* prevalence range from 0.3 to 2.5 percent, while incidence was 60.4 per 100,000 in one study (Plunkett and Marks, 1998). Excluding persons with RA, SLE, and several other arthritic conditions, the sex and age-adjusted incidence of psoriatic arthritis was 6.6 per 100,000 and the prevalence 0.1 percent, with an average age of 40.7 years at diagnosis in Olmstead County, Minnesota. There was little gender difference, and survival was not different from the general population, in contrast to referral-based studies (Shbeeb et al., 2000).

Psoriasis vulgaris is associated with T cell-mediated attack on skin components. Streptococcal throat infection can trigger guttate psoriasis, which is assumed to be a cross-reaction due to the resemblance of streptococcal proteins to skin proteins (Prinz, 2001). Atopic allergic diseases and contact sensitization were common among psoriasis patients in a study from Italy (Pigatto, 2000).

The initial treatment of psoriasis is often topical, with phototherapy for more advanced cases, and oral pharmaceuticals for the most serious cases. Tumor necrosis factor- α blockade with or without methotrexate is used in refractory psoriasis (Davidson and Diamond, 2001).

Lubricating creams are applied alone or with corticosteroids (Berkow and Talbot, 1977); some local treatments combine a corticosteroid and a noncorticosteroid (Feldman, 2000).

Although antistreptococcal drugs have been recommended, there is no evidence of benefit from them (Owen et al., 2000).

Phototherapy with ultraviolet radiation has been used with psoralen (PUVA) as a sensitizer, or without it (UVB). PUVA treatment, although very effective, was recently associated with increased risk of melanoma, and life-long follow-up is recommended for patients receiving PUVA (Wolf, 1997).

Few randomized trials have compared treatments for acute guttate psoriasis; however, an intravenous n-3 fatty acid rich emulsion was superior to placebo (Chalmers et al., 2000).

The combination of systemic treatments is controversial, and safety must be balanced against effectiveness (Van de Kerkof, 2001a). Various combinations of treatments involving calcipotriol, cyclosporin, PUVA, UVB, dithranol, acitretin, and coal tar may be useful, while other combinations are contraindicated. Rotation of therapies is a way to avoid toxicity (Van De Kerkhof, 2001b). Men are more likely than women to receive intensive therapies, at least in part because of potential teratogenesis from treatment during pregnancy (Hotard et al., 2000).

Monoclonal antibodies are being developed to target abnormal new blood vessels, which are characteristic of diseases such as RA and psoriasis (Halin and Neri, 2001).

Fibromyalgia and Chronic Fatigue Syndrome

Fibromyalgia (also called fibromyalgia syndrome, or FMS) and chronic fatigue syndrome are both mysterious conditions. They tend to be (and ought to be) diagnoses assigned only after attempts to establish better-known diagnoses fail. Little is known about their prevalence, causes, and treatment. They are generally considered autoimmune diseases although this link has not been established either. It is not unlikely that further research will identify subsets of individuals who are more alike in their disease characteristics, thus facilitating investigations into causes and cures.

Many people who meet the definition of an unexplained clinical condition (fibromyalgia, chronic fatigue syndrome, irritable bowel syndrome, multiple chemical sensitivity, temporomandibular disorder, tension headache, interstitial cystitis, postconcussion syndrome) also meet the definition for another unexplained condition, so that the diagnosis depends somewhat on which specialist first sees the patient (Aaron and Buchwald, 2001; Natelson, 2001). The diagnosis of fibromyalgia requires widespread pain persisting for 3 months or more in 10 or more specified muscles, tendons, or ligaments. Rheumatoid arthritis or Sjogren's syndrome may be present. The American College of Rheumatology estimates that 3 to 6 million Americans are affected. Allegations that a fibromyalgia diagnosis increases illness behavior and dependence on the medical system are not supported by research (White and Harth, 2001).

The treatment of fibromyalgia is directed toward the relief of symptoms (pain, sleep disturbance, mood disturbance, and fatigue), and should be individualized, while addressing physical fitness, work, and mental health (Barkhuizen, 2001). A variety of interventions involving pharmacological treatment of pain or depression, physical therapy, diet, meditation, cognitive therapy, and so on, have been helpful in some patients, but not all treatments may be covered by insurance. Examples of successful unusual treatments are the discontinuation of monosodium glutamate in the diet, which was theorized to excite neurotransmitters (Smith et al., 2001), and a randomized trial in which static magnetic fields in sleep pads brought improvement to fibromyalgia patients, though not significantly different than the improvement from placebo or usual care (Alfano et al., 2001). Participation in clinical studies and trials are ways to obtain potentially useful treatment or to contribute to knowledge about the causes of fibromyalgia. Fibromyalgia research is funded by the National Institutes of Health, especially the National Institute of Arthritis and Musculoskeletal and Skin Diseases.

Understandably, many sufferers turn to untenable theories and unproven remedies. It is

important to avoid unproven remedies which are either expensive or dangerous, and it would seem prudent to seek treatments that have rational bases, and come from reputable sources.

In a study of 127 twin pairs, one twin with chronic fatigue syndrome (CFS) and one without, fibromyalgia was present in more than 70 percent of the twins with CFS, versus less than 10 percent in the twins without CFS (Aaron et al., 2001). Chronic fatigue syndrome, while it overlaps with fibromyalgia, is also different in certain ways. A substance P is elevated in the spinal fluid of fibromyalgia patients only (Natelson, 2001). Whether a cardiovascular response is abnormal in chronic fatigue syndrome, is controversial (Natelson, 2001; Naschitz et al., 2001).

Support organizations often emphasize that CFS has appeared prior to the twentieth century, under different names. The diagnosis requires at least 6 months of new-onset severe fatigue accompanied by infectious, rheumatologic, or neuropsychiatric symptoms such as intense headache, sore throat, tender lymph nodes, muscle and joint aches (without redness or swelling), unrefreshing sleep, and inability to concentrate (Natelson, 2001). The case definition for chronic fatigue syndrome reflects the assumption, as yet unproved, of a viral origin. An initial hypothesis linking CFS to Epstein-Barr virus has been abandoned. Some data support an immunologic origin to CFS, but allergies are the only consistent immune system abnormality among CFS patients (WebMD, 2000). While many patients have psychiatric co-morbidities, CFS does not have the same profile as depression or somatization disorder. Magnetic resonance imaging of the brain found subtle changes, but only in those CFS patients without a psychiatric co-diagnosis (Natelson, 2001). Reports of association with HLA genes were not confirmed in a recent study (Underhill et al., 2001).

In the United States, the prevalence of CFS has been estimated at 0.52 percent in women and 0.29 percent in men (Natelson, 2001). A large number of Gulf War veterans reported CFS symptoms. While investigations continue, the causes of CFS remain unclear.

As with fibromyalgia, chronic fatigue syndrome is treated individually and symptomatically. According to one web source, children have better prognosis than adults, and 95 percent recover within four years. Adults with a sudden onset appear to recover faster than those with no sudden beginning to their symptoms, although a multidisciplinary program helped most people (WebMD, 2000).

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20. SEXUALLY TRANSMITTED DISEASES

INTRODUCTION

Sexually transmitted diseases (STDs) are infections passed from person to person during sexual contact or from mother to child. More than 20 STDs, caused by organisms ranging from viruses to insects, have been identified. Of these, Connecticut physicians are required by law to report cases of five STDs—syphilis, gonorrhea, chlamydia, chancroid, and neonatal herpes—to the Connecticut Department of Public Health. No cases of chancroid or neonatal herpes have been reported in Connecticut in recent years, and new cases of syphilis have diminished to historic low levels. In contrast, thousands of new gonorrhea and chlamydia cases are still reported in Connecticut women each year.

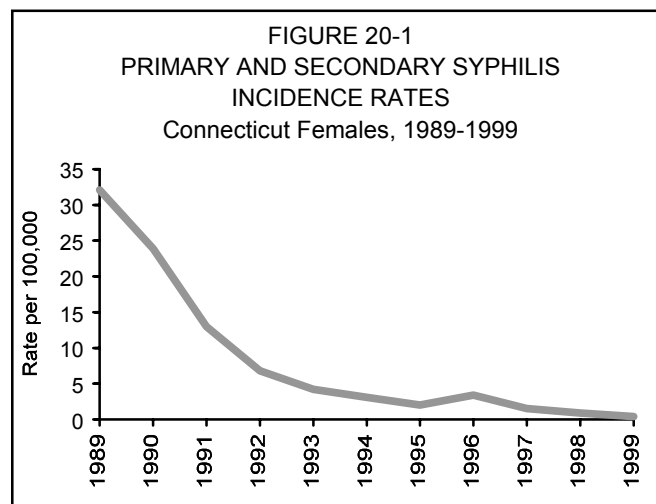
Because initial symptoms of gonorrhea and chlamydia typically are absent or transient in women, medical care is often sought only after serious complications have developed. Without treatment, gonorrhea and chlamydia infections can spread to the uterus and fallopian tubes, causing pelvic inflammatory disease (PID), other problems that may lead to infertility, or ectopic

(tubal) pregnancy. PID occurs less frequently and is associated with milder pelvic lesions in women who use oral contraceptives (Henry Suchet, 1997). Ectopic pregnancy is the leading cause of maternal death during the first trimester of pregnancy, and nearly half of ectopic pregnancies have been attributed to STDs (Coste et al., 1994). In pregnant women, gonorrhea and chlamydia infections increase the risk of preterm birth, and newborns can become infected during delivery, resulting in eye infections and pneumonia. Furthermore, a chlamydia infection increases the risk of developing invasive cervical cancer (Anttila et al., 2001), and its presence can facilitate the acquisition and transmission of the human immunodeficiency virus (Chin, 2000).

SCOPE OF THE PROBLEM

Syphilis

Syphilis control is a success story in the recent history of public health in Connecticut. Since peaking in 1989, syphilis has nearly been eradicated. Although 543 newly acquired infections, termed “primary and secondary syphilis,” were reported in Connecticut females



Source: Connecticut Department of Public Health, STD Control Program, 2000. Rate calculations from Division of Policy, Planning, and Analysis.

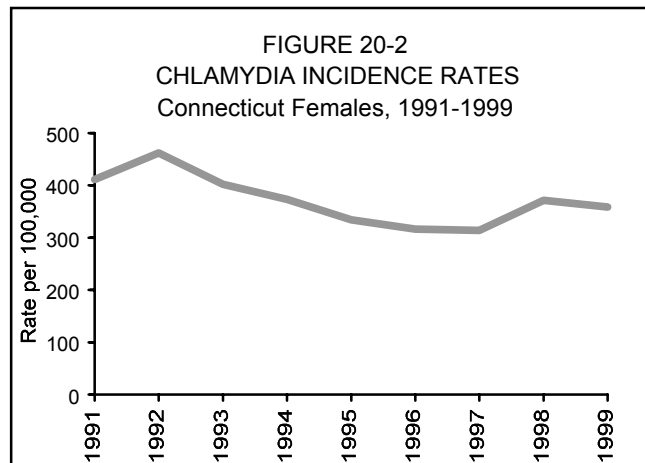
in 1989, only 6 cases were reported in 1999, with case rates dropping from 32 to 0.4 cases per 100,000 females (Fig. 20-1). During the same interval, the number of congenitally acquired cases dropped from 25 or more per year between 1990 and 1992 to 0 in 2000 (Connecticut Department of Public Health, STD Control Program, 1999, 2000, 2001). Nationally, syphilis rates in males and females peaked in 1990 and in 1999 were at the lowest level since reporting began in 1941. In 1999, the Centers for Disease Control and Prevention launched the National Plan to Eliminate Syphilis in the United States (Centers for Disease Control and Prevention, 2000).

In the absence of HIV co-infection, untreated syphilis infections can remain clinically latent for many years, then re-emerge as serious, late-stage diseases of the heart and central nervous system. Although 12 Connecticut women died with late manifestations of the disease since 1990, there were no deaths from 1995 to 1998.

Chlamydia

Chlamydia is the most common of all STDs, with 5,000 to 7,000 new cases reported in Connecticut women each year. Chlamydia incidence peaked in 1992, declined through 1997, then rose again in 1998 (Fig. 20-2). In 1999 there were 6,056 new cases in Connecticut women (359 per 100,000 population), representing 82 percent of total cases.

Chlamydia is found in females more often than in males because, until the recent advent of urine-based screening, only women were routinely screened for the infection. No deaths attributable to chlamydia infection have been reported in Connecticut. Nationally it is believed that chlamydia infections have declined from over 4 million annually in the early 1980's to 3 million annually in the late 1990's, primarily due to increased screening and treatment of women (Centers for Disease Control and Prevention, 2000).

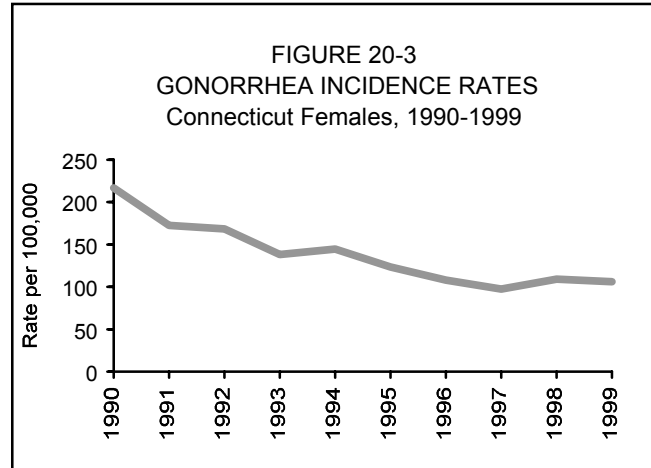


Source: Connecticut Department of Public Health, STD Control Program, 2000.
Rate calculations from Division of Policy, Planning, and Analysis.
Note: Chlamydia surveillance began 7/1/90, so 1990 data are not included.

Gonorrhea

Gonorrhea incidence among Connecticut women generally has been declining since peak levels were reached in the late 1980's (Fig. 20-3). In 1999 there were 1,796 reported new cases

females. In 1999, about 40 percent of the cases occurred in 15 to 19 year olds, and their infection rates exceeded those found in any other age group (2,446 and 684 per 100,000 females for chlamydia and gonorrhea, respectively).



Source: Connecticut Department of Public Health, STD Control Program, 2000.

among Connecticut women (106 per 100,000 population), which was an historic low. Since 1992, slightly more cases have been reported in women than in men, whereas the opposite had been true previously.

Age

Most chlamydia and gonorrhea infections are reported in adolescent and young adult

Race and Ethnicity

Disproportionate numbers of Connecticut's black and Hispanic women are reported with gonorrhea and chlamydia infections, evidenced by high incidence rates (Table 20-1). Although blacks represented only 10 percent of Connecticut's female population, they accounted for 64 percent of gonorrhea cases and 47 percent

TABLE 20-1
GONORRHEA AND CHLAMYDIA INCIDENCE
BY RACE & ETHNICITY
Connecticut Females, 1999

| | Gonorrhea | | Chlamydia | |
|-----------------|-----------|-------|-----------|---------|
| | Cases | Rate | Cases | Rate |
| All races | 1,789 | 105.9 | 6,005 | 355.5 |
| White | 316 | 21.3 | 1,301 | 87.9 |
| Black | 1,136 | 701.5 | 2,822 | 1,742.2 |
| Native American | 0 | 0 | 0 | 0 |
| Asian & PI | 3 | 6.5 | 32 | 72.5 |
| Hispanic | 334 | 234.1 | 1,850 | 1,296.3 |

Source: Connecticut Department of Public Health, STD Control Program, 2000

Notes: Rates expressed as reported cases per 100,000 females.

Numbers and rates include data where race/ethnicity was not indicated on the case report.

These data were distributed among the categories in proportion to known cases.

of chlamydia cases in 1999. Similarly, although only 8 percent of Connecticut women were Hispanic, Hispanics represented 19 percent of gonorrhea cases and 30 percent of chlamydia cases. Relative to white females, chlamydia rates were 20 times higher and gonorrhea rates were 33 times higher in black females; chlamydia rates were 15 times higher and gonorrhea rates were 11 times higher in Hispanic females.

Hospitalizations for STDs had similar patterns. Between 1993 and 1997, compared to white females, age-adjusted hospitalization rates of black females were 18 times higher for STDs and 4 times higher for PID, while rates for Hispanics were 5 times higher for STDs and twice as high for PID. All these differences were statistically significant (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001).

Town of Residence

The incidence of syphilis, gonorrhea, and chlamydia is disproportionately high among women residing in urban areas. In 1999, four towns—Hartford, New Haven, Bridgeport, and Waterbury—accounted for 58 percent of gonorrhea cases and 50 percent of chlamydia cases. These towns represent only about 16 percent of the state's female population. Hartford residents accounted for four of the six Connecticut women reported with primary and secondary syphilis (Connecticut Department of Public Health, STD Control Program, 2000).

PREVENTION AND RISK REDUCTION

Sexually transmitted diseases can be prevented by abstaining from sex with a partner, and the risk of acquiring them or passing them to others can be reduced by using barrier protection (male or female condoms) consistently and properly (See Chapter 21, HIV Infection and AIDS, for a more detailed discussion of prevention or reduction of risky sexual behavior). A study from an STD clinic estimated that the relative risk of gonorrhea in women where a spermicide and condom, or spermicide and diaphragm were used, was 55-59% lower than in

women where no spermicide, condom, or diaphragm were used (Austin et al, 1984).

The Connecticut Department of Public Health Abstinence-Only Education Initiative is designed to reach 9 to 14 year old children and to include parental or guardian involvement when possible. The initiative operates in five locations and is adding a sixth. The STD Control Program conducts urine-based STD testing through schools, correctional facilities, and Planned Parenthood clinics, and provides counseling, treatment, and public education.

Screening Tests

A simple and inexpensive blood-screening test exists for syphilis. Urine screening for chlamydia and gonorrhea, which costs about \$38 per sample and \$453 per case identified (Jones et al., 2000), is being used increasingly in Connecticut (Connecticut Department of Public Health, STD Control Program, 1999). Because gonorrhea and chlamydia often are asymptomatic, sexually active women—especially those with a new sex partner—should have regular check-ups for STDs performed by their doctors, even in the absence of symptoms. As STDs are highly infectious, the sex partners of infected women also should be tested.

Several analyses of cost-benefits indicate that the total cost of general testing for chlamydia in at-risk populations could save twice the cost of treating pelvic inflammatory disease and six times the cost of all the medical consequences of chlamydia infections, including tubal infertility and ectopic pregnancies (Henry Suchet et al., 1996). Therefore, programs designed to encourage or facilitate routine screening for STDs in high-risk populations (sexually active females between 15 and 24 years old, those who live in urban areas, and those with multiple sex partners) are likely to reduce costs in the healthcare delivery system.

TREATMENT

Syphilis, gonorrhea, and chlamydia can be treated successfully with antibiotics. The earlier a woman seeks treatment and warns her sex partners about the disease, the less likely it becomes that severe complications and passage to others, including her newborn, will occur.

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21. HIV INFECTION AND AIDS

INTRODUCTION

Acquired immunodeficiency syndrome (AIDS) is a severe, life-threatening disease representing the late stage of infection with the human immunodeficiency virus (HIV). HIV infection can result in progressive damage to the immune system, leading to opportunistic infections, cancers, and other diseases that characterize clinical AIDS. HIV transmission in women occurs mainly through heterosexual contact or by using contaminated needles and syringes for injecting drugs. The presence of an ulcerative sexually transmitted disease like chancroid facilitates HIV transmission during intercourse. HIV-infected pregnant women can pass the virus to their infants during pregnancy or birth and during breastfeeding.

Except for transient flu-like symptoms that occur weeks or months after infection occurs, people with HIV can be free of symptoms for many months or years. Certain nonspecific symptoms (swollen glands, loss of appetite, fever, fatigue, diarrhea and weight loss) mark the onset of clinical illness and are called “symptomatic HIV infection.” In HIV-infected people, more than a dozen specific opportunistic infections, such as *Pneumocystis carinii* pneumonia, and several cancers, including Kaposi’s sarcoma, invasive cervical cancer, and non-Hodgkin lymphoma, constitute the case definition of AIDS (Chin, 2000). The original, 1982 case definition of AIDS was expanded twice, in 1987 and 1993, to encompass additional indicator diseases and conditions.

In the absence of treatment, more than 90 percent of HIV-infected persons ultimately develop AIDS, and of these, 80 to 90 percent die within 3 to 5 years of diagnosis (Chin, 2000). HIV infection is listed as the cause of death for AIDS and HIV- or AIDS-related diseases, whereas the incidence of HIV infection and AIDS are reported separately. Clinically-defined AIDS has been reported in Connecticut since

1981. In 1999, HIV infection also became reportable in the state.

SCOPE OF THE PROBLEM

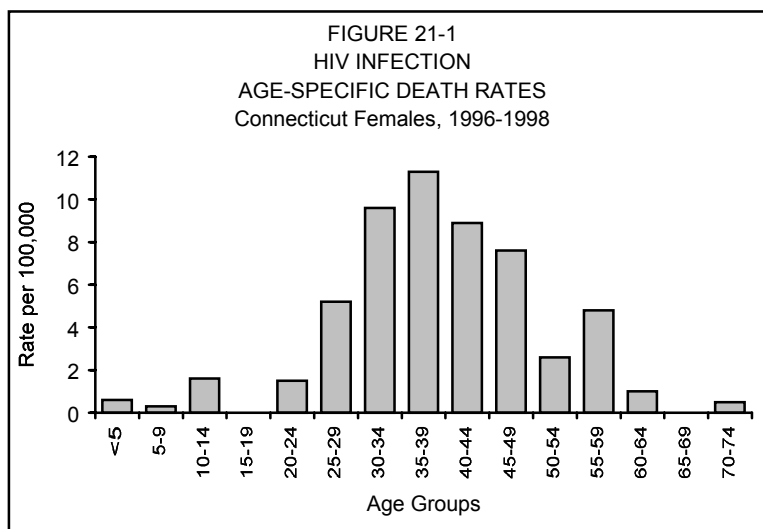
In 1999, 188 new cases of AIDS (the lowest number since 1992) were reported among Connecticut females, representing an incidence rate (both crude and age-adjusted) of 11 cases per 100,000 population. Cumulatively, through the end of 1999, AIDS was reported in 2,786 Connecticut females, and as of June 30, 2000, 1,610 Connecticut females with AIDS were still alive (Connecticut Department of Public Health, HIV/AIDS Surveillance Program, 2000a, 2000b).

Between 1993 and 1997 there were 1,576 hospitalizations of Connecticut women for HIV/AIDS. The annual, age-adjusted hospitalization rate was 18 per 100,000 women, and the average charge was \$18,640 (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001).

Between 1996 and 1998, 204 Connecticut women died of HIV/AIDS, for an annual, age-adjusted death rate of 7 deaths per 100,000 population (Mueller et al., in preparation).

Age

The rates of reported cases of both HIV infection and AIDS are greatest in women 30 to 39 years of age, and in 1999, about 90 percent of reported cases of HIV infection and AIDS in women were between the ages of 20 and 49 (Connecticut Department of Public Health, HIV/AIDS Surveillance Program, 2000b). From 1987 to 1998, HIV infection was one of the top five leading causes of death among Connecticut women 25 to 44 years of age, and from 1992 to 1996 it was the leading cause of death among women 25-34 years of age. Between 1996 and 1998, HIV infection was the fourth-ranked leading cause of premature deaths in Connecticut. The death rate for HIV infection



Source: Mueller et al., in preparation.

also is highest among women in their fourth decade (Fig. 21-1).

Race and Ethnicity

As with sexually transmitted diseases, disproportionate numbers of Connecticut's black and Hispanic females develop AIDS (Table 21-1). Although blacks make up only 10 percent of Connecticut's female population, they accounted for 39 percent of new AIDS cases in 1999. Similarly, although only 8 percent of Connecticut women are Hispanic, Hispanics represented 29 percent of AIDS cases. Relative to the rate for white females, the AIDS rates in 1999 were 12 times greater for blacks and 10 times greater for Hispanics. Since 1998, the proportions of AIDS cases represented by white and Hispanic women have increased, while cases among black women have decreased (Connecticut Department of Public Health, HIV/AIDS Surveillance Program, 2000b).

In Connecticut, hospitalization and death rates for HIV infection are also disproportionately high among minority women. Relative to non-Hispanic white females, the 1993-1997 age-adjusted hospitalization rate for HIV in non-Hispanic black females (98.0) was 18 times greater, and that for Hispanic females (63.2) was 12 times greater than white females (5.4 per 100,000). These differences were statistically significant (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001).

Between 1996 and 1998, the age-adjusted death rate for black females was more than 9 times greater than that for white females, and the rate for Hispanic females was nearly 6 times greater (Table 21-2). HIV infection was the fourteenth-ranked leading cause of death among white females. It was the fourth-ranked among Hispanic females and the fifth-ranked among black females (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001).

TABLE 21-1
AIDS INCIDENCE BY RACE & ETHNICITY
Connecticut Females, 1999

| | Population (1999 Est.) ^a | No. Cases | Case Rate ^b | % of Population | % of Cases | Relative Risk ^c |
|----------------------|--|-----------|------------------------|-----------------|------------|----------------------------|
| All races | 1,689,230 | 188 | 11.1 | 100.0 | 100.0 | |
| White | 1,479,617 | 58 | 3.9 | 87.6 | 30.9 | 1.0 |
| Black | 161,977 | 73 | 45.1 | 9.6 | 38.8 | 11.6 |
| Native American | 4,037 | | | 0.2 | 0.2 | 2.4 |
| Asian & Pacific Isl. | 43,599 | | | 2.6 | 0.2 | 0.2 |
| Hispanic | 142,732 | 54 | 37.8 | 8.4 | 28.7 | 9.7 |

Sources: Connecticut Department of Public Health, HIV/AIDS Surveillance Program, 2000d;
Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001.

Notes: ^a Population estimates from U.S. Census Bureau (2000).

^b Rate expressed as reported cases per 100,000 females.

^c Relative risk is estimated as the ratio of the minority group rate to the white group rate.

Percent of cumulative cases 1981-1999 were used to calculate relative risk for Native American and Asian or Pacific Islander, due to 3 or fewer cases in 1999

TABLE 21-2
HIV INFECTION DEATHS BY RACE AND ETHNICITY
Connecticut Females, 1989-1991 and 1996-1998

| Race/Ethnicity | 1989-1991 | | 1996-1998 | |
|-----------------|------------------|---------------------------------------|------------------|---------------------------------------|
| | Number of Deaths | Age Adjusted Death Rate (per 100,000) | Number of Deaths | Age Adjusted Death Rate (per 100,000) |
| All races | 192 | 3.6 | 204 | 3.9 |
| White | 86 | 1.8 | 102 | 2.3 |
| Black | 103 | 21.7 [‡] | 101 | 21.1 [‡] |
| Hispanic/Latina | 32 | 9.4 [‡] | 51 | 13.4 [‡] |

Source: Mueller et al., in preparation.

[‡] Rate significantly different from that of whites ($p < .05$).

Notes: U.S. Census Bureau population estimates used for rate calculations. Rates adjusted to the 2000 U.S. standard million population.

Geographic Region

Table 21-3 shows AIDS rates in females in the United States and Northeastern states from July 1999 through June 2000. Connecticut's rate was above the national average. Connecticut's rate was the third highest among the eight Northeastern states.

TABLE 21-3
AIDS INCIDENCE RATES
United States and Northeastern States
Females, July 1999 through June 2000

| State | Incidence per 100,000 |
|---------------|-----------------------|
| United States | 9.0 |
| Connecticut | 17.0 |
| Maine | 2.0 |
| Massachusetts | 14.5 |
| New Hampshire | 1.0 |
| New Jersey | 18.7 |
| New York | 27.8 |
| Rhode Island | 6.3 |
| Vermont | 0.8 |

Source: Centers for Disease Control, 2000.

TRENDS OVER TIME

The Centers for Disease Control and Prevention estimates that two-thirds of people living with HIV have been confidentially tested and know their status, and that HIV reporting is likely to become increasingly representative of trends in infections (Centers for Disease Control and Prevention, 2001). Annual HIV incidence has been roughly constant since 1992 in most US populations with time trend data (Karon et al., 2001).

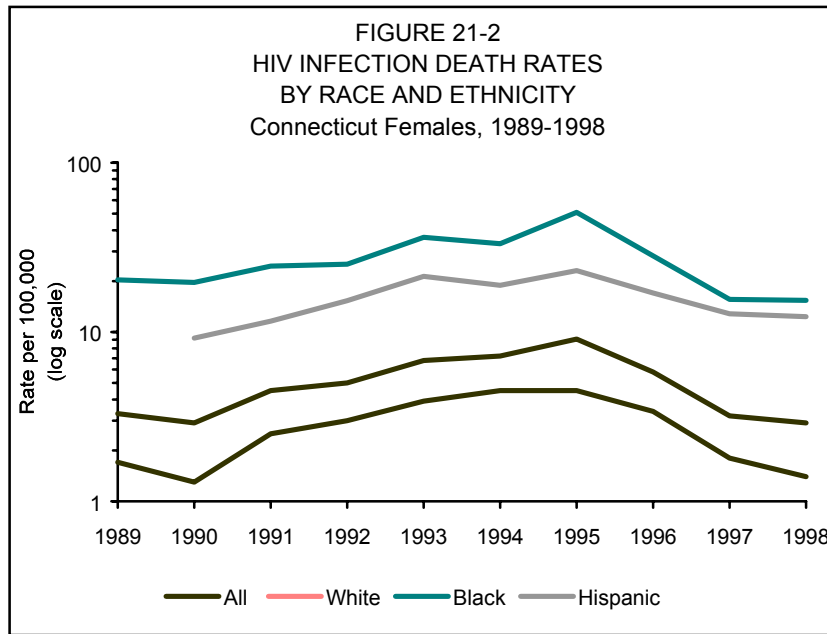
AIDS rates reflect both the acquisition of HIV and progression of HIV. Highly active antiviral therapy became available in the mid-1990's and had a dramatic impact on delaying the progression of HIV to AIDS (see the treatment section below). The AIDS case definition was expanded in 1993, causing a large increase in reported AIDS in the first quarter of 1993. Nationally, AIDS incidence in men began to decrease beginning in 1994, while it continued to increase in women through 1996. AIDS incidence began to drop earlier among whites than blacks, and dropped among men who have had sex with men, but continued to rise among intravenous drug users, and AIDS acquired through heterosexual transmission. For each of the groups: white, black, and Hispanic women, the annual number of AIDS cases acquired heterosexually surpassed the number acquired through intravenous drug use in the early 1990's (Centers for Disease Control and Prevention, 2001). There were only very slight declines in AIDS in 1998 and 1999. The epidemic

increasingly affects women, blacks, Hispanics, persons infected through heterosexual contacts, intravenous drug users, and the poor. Between 1990 and 1999, there was a four-fold increase in the number of Americans living with AIDS (Karon et al., 2001).

The numbers and rates of new AIDS cases in Connecticut women reported each year decreased between 1997 and 1999. However, for 2000, 225 AIDS cases in females were reported, which were 40 cases more than 1999. The proportion of cases represented by women rose from 14 percent in 1985 to 31 percent in 1999 (Connecticut Department of Public Health, HIV/AIDS Surveillance Program, 2000a).

HIV hospitalizations among Connecticut women more than tripled from 1994 to 1995, peaked in 1996, and then fell by more than one-third in 1997. This is similar to the patterns seen for AIDS incidence and mortality.

The first known deaths to Connecticut women from HIV infection occurred in 1987. From 1987 through 1998, HIV has claimed the lives of 945 women, or 36 percent of those reported with the disease (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001). The death rate for HIV infection peaked in 1995 and dropped by 68 percent (which was statistically significant) through 1998 (Figure 21-2), with the decline largely attributed to more effective antiviral therapy (Chin, 2000). Between 1995 and 1998, the death rates declined among white, black, and Hispanic females.



Source: Mueller et al., in preparation.
Rates are age-adjusted, except that crude rates were used for Hispanic females in 1990, 1991, and 1998 due to small numbers.

RISK FACTORS

HIV is spread mainly through sexual contact and sharing contaminated needles and syringes. It also can be acquired via transfusions of infected blood or blood components, and transplants of infected organs or tissues. Since the mid-1980's the United States has taken vigorous measures to safeguard the blood supply from HIV. The infants of pregnant women with HIV can become infected before, during, or shortly after birth. The epidemic spread of HIV in the black and Hispanic communities is fueled by drug use, including sex with an infected drug user and sex for money (Centers for Disease Control and Prevention, 2001).

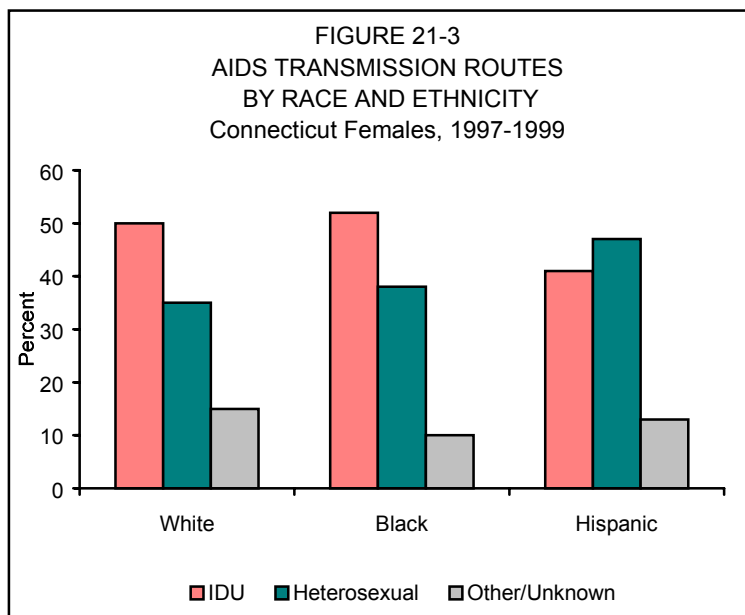
Although HIV has been found in the saliva, tears, urine, and bronchial secretions of infected persons, transmission by contact with these

substances has never been documented (Centers for Disease Control and Prevention, 1999a).

The most important risk factors for HIV infection among women are injecting-drug use and heterosexual sex with multiple partners and without condoms. The presence of a sexually transmitted disease facilitates the spread of the virus.

Injecting Drug Use

An IDU transmission was the most common route of HIV infection in white women, while heterosexual transmission was the most common route in Hispanic women (Fig. 21-3). Between 1997 and 1999 the AIDS incidence through IDU fell, with a 73 percent decrease among black women, 66 percent among Latinas, and 43 percent among whites (Connecticut Department of Public Health, HIV/AIDS Surveillance Program, 2000c).



Source: Connecticut Department of Public Health, HIV/AIDS Surveillance Program, 2000b.

The exact number of Connecticut women who use injecting drugs is not known, but estimates have been made using admissions to substance abuse treatment programs. In the most recent 12 months for which data are available (June, 1999 through May, 2000), 25 percent of the adult females admitted to such programs—more than 4,800 women or 0.4 percent Connecticut's adult female population—said they were current intravenous drug users (Connecticut Department of Mental Health and Addiction Services, 2000). Adolescents may represent an even larger risk group. In 1999, 1.6 percent of surveyed Connecticut high school girls said they had injected illegal drugs one or more times in their lives (Kann et al., 2000).

Heterosexual Sex

The percentage of AIDS cases associated with heterosexual sex has been increasing and is consistently higher for Hispanic females than for white or black females. Between 1997 and 1999, a significantly higher percentage of Hispanic females (47 percent) who developed AIDS had a heterosexual sex risk, compared to 38 percent of blacks and 35 percent of whites (Connecticut Department of Public Health, HIV/AIDS Surveillance Program, 2000b; Connecticut

Department of Public Health, Division of Policy, Planning, and Analysis, 2001).

In 1998, 8 percent of sexually active Connecticut women 18 to 64 years of age said they had more than one sex partner in the prior year (Connecticut Department of Public Health, AIDS Counseling and Testing Program, 2000). Of these, three out of four said they don't always use condoms, and one in three said they never use condoms. Women with multiple sex partners were almost twice as likely as those with only one sex partner to use condoms all the time (27 percent and 15 percent, respectively).

With regard to condoms usage, similar behavior was reported among Connecticut high school students (Kann et al., 2000). Even though more than nine out of ten girls in grades 9-12 said they were taught about HIV in school, 40 percent reported they had sexual intercourse at least once, 10 percent said they had four or more sex partners in their lifetime, and 30 percent were currently sexually active. Only half said they used a condom during their last sexual intercourse.

PREVENTION AND RISK REDUCTION

The only effective way to prevent HIV infection is to avoid exposure to the virus. Exposure to HIV can be avoided or reduced by abstaining from injecting-drug use or using only sterile drug paraphernalia, abstaining from sex or practicing low risk or non-risky sexual behaviors (using condoms consistently and correctly, having sex only with an uninfected partner, etc.).

The effectiveness of condoms in preventing heterosexual transmission of HIV has been estimated at 87 percent, which is comparable to or slightly lower than its effectiveness at preventing pregnancy (Davis and Weller, 1999). The spermicide nonoxynol-9 appears, from an observational study, to be at least as effective as condoms in reducing heterosexual transmission of HIV, but it may not be possible to conduct randomized clinical trials (Wittkowski et al., 2001). Studies suggest that condom use in at-risk heterosexuals increased in the 1990's (Catania et al., 2001), but that as many as one in three HIV-positive people continue to have unprotected sex after learning that they are infected (Kalichman et al., 2001).

Medical treatment after exposure to HIV is considered relatively ineffective for preventing infection, compared to preventing exposure altogether. An exception is the use of anti-retroviral medications to prevent transmission of HIV from infected mothers to their infants before and after delivery (Centers for Disease Control and Prevention, 1998).

The Connecticut Department of Public Health, AIDS Prevention and Treatment Services program, coordinates counseling and testing services, risk reduction education, outreach to drug users, needle exchange, referral to drug treatment and care, case management, social marketing, and public information campaigns.

The needle exchange program is established in Hartford, Bridgeport, New Haven, Danbury, and Stamford. The program, which also offers education and testing for tuberculosis and hepatitis, and referrals for STD testing and substance abuse treatment, enrolled 1,872 clients between July 1999 and June 2000.

The ATHENA Project at the Yale School of Nursing has begun an effort ("HIV University") to teach HIV positive women about treatment, life skills, peer education, and support groups.

TESTING AND TREATMENT

HIV Testing

Antibodies to HIV usually develop within one to three months after infection. In Connecticut, blood tests for HIV can be done by a woman's own doctor, at a reproductive health clinic, or at 25 publicly funded counseling and testing sites. Strict confidentiality is maintained, and some sites offer anonymous testing. In 1999 a law was passed by the state legislature to promote counseling and testing for pregnant women; the law also addresses the testing of newborns when their mothers have not been tested.

In 2000, 7,361 HIV tests were performed for Connecticut women, of which 9 percent were anonymous, and 90 percent were confidential. About 1 percent of tests was positive. Ninety-six percent of positive tests were followed by counseling sessions, as were 83 percent of negative test results.

Treatment

Effective combination therapy using three or more drugs, known as highly active anti-retroviral therapy or HAART, became available routinely in the U.S. in the mid-1990's, and new treatment regimens are still being developed. Specific treatments also are available for pneumonia and other opportunistic infections associated with HIV. Additionally, pregnant women can be treated with AZT and other drugs to prevent *in utero* and perinatal HIV transmission to their children (Chin, 2000).

Barriers to treatment for women include lack of knowledge about AIDS in women by health providers, family responsibilities, and the fear and burden of disclosure (Zorilla and Santiago, 1999).

Although access to care improved among HIV-infected persons in the U.S. in recent years,

women were among those groups with less desirable patterns of care. Relative to men, women were significantly more likely to use hospital emergency rooms and to be hospitalized. They also were significantly less likely to take prophylactic drugs to prevent *Pneumocystis carinii* pneumonia or to receive therapy using protease inhibitors and non-nucleoside reverse transcriptase inhibitors. Persons without insurance and those on Medicaid also were significantly less likely to receive anti-retroviral therapy (Shapiro et al., 1999).

More than half of Connecticut women reported with AIDS through 1999 were injection drug users (Connecticut Department of Public Health, HIV/AIDS Surveillance Program, 2000b). According to one report, while most infected IDUs receive care for HIV, less than half receive highly active antiretroviral therapy (Rogers, 2000). Barriers to HIV care among IDUs include: difficulty finding a health care provider, poverty, homelessness, and incarceration; negative experiences with providers (care refused because of drug use); equating treatment with the disease (having a constant reminder of illness); and concern about treatment drug and street drug interactions.

Treatment for AIDS is recommended to begin before a person's CD4 count falls below a certain threshold. CD4 cells are an important part of the immune system, and people with AIDS have fewer CD4 cells than do healthy people. At the same CD4 count, women have a lower viral load than men—sometimes half as great. Women also have a higher risk of becoming ill at the same CD4 count. These findings have two important implications. First, women may systematically be excluded from clinical trials in which enrollment criteria are based on viral load. Second, it may be important for women to start treatment earlier than men (National Institute of Allergy and Infectious Disease, 2000).

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22. PNEUMONIA AND INFLUENZA

INTRODUCTION

Pneumonia and influenza are highly contagious respiratory infections. Influenza is caused by constantly evolving influenza viruses, whereas pneumonia may be caused by a host of microorganisms, including bacteria (notably *Streptococcus pneumoniae*, which causes pneumococcal pneumonia), viruses, mycoplasma, protozoa, and chlamydia. Although influenza usually does not cause pneumonia, pneumonia is a frequent complication of influenza, and most influenza fatalities result from secondary bacterial pneumonia (Kilbourne, 1994). The role of seasonal influenza epidemics in increasing flu and pneumonia deaths among the elderly is the reason why disease monitoring systems typically track influenza and pneumonia deaths together, instead of separately.

SCOPE OF THE PROBLEM

Between 1996 and 1998, pneumonia and influenza (P & I) caused 1,988 deaths among Connecticut women, resulting in an age-adjusted death rate of 27 deaths per 100,000 population

(Table 22-1). About 98 percent of these deaths were attributed to pneumonia (Connecticut Department of Public Health, 2001b). It was the fifth leading cause of death among women of all ages and the eleventh leading cause of premature deaths (Mueller et al., in preparation).

Pneumonia is a leading cause of hospitalization among Connecticut women. Between 1993 and 1997, there were about 6,000 hospital admissions of Connecticut women each year for pneumonia and influenza, resulting in an age-adjusted hospitalization rate (AAHR) of 283 hospitalizations per 100,000 women. About 98 percent of hospitalizations were also attributed to pneumonia (Connecticut Department of Public Health, 2001a). In 1997, P&I resulted in total hospital charges of \$79.5 million dollars (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001a). Pneumococcal pneumonia may account for up to 55 percent of all cases of adult pneumonia that require hospitalization (Heffelfinger et al., 2000).

TABLE 22-1
PNEUMONIA AND INFLUENZA DEATHS
BY RACE AND ETHNICITY
Connecticut Females, 1989-1991 and 1996-1998

| Race/Ethnicity | 1989-1991 | | 1996-1998 | |
|------------------------|------------------|---------------------------------------|------------------|---------------------------------------|
| | Number of Deaths | Age Adjusted Death Rate (per 100,000) | Number of Deaths | Age Adjusted Death Rate (per 100,000) |
| All races | 1,884 | 29.6 | 1,988 | 26.6* |
| White | 1,808 | 29.5 | 1,892 | 26.4* |
| African American/Black | 59 | 22.7 | 91 | 30.9 |
| Asian/Pacific Islander | 4 | † | 4 | † |
| Native American | 1 | † | 1 | † |
| Hispanic/Latina | 20 | 19.1‡ | 32 | 18.4‡ |

Source: Mueller et al., in preparation.

* Change in rates from 1989-91 to 1996-98 period is statistically significant ($p < .05$).

† Statistics not calculated for fewer than 15 events.

‡ Rate significantly different from that of whites ($p < .05$)

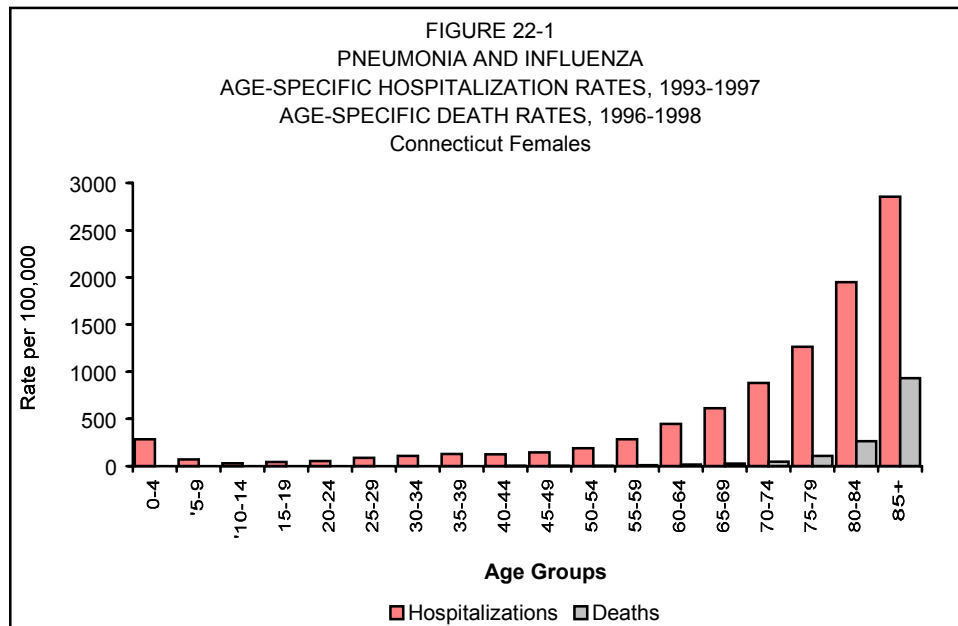
Notes: U.S. Census Bureau population estimates used for rate calculations. Rates adjusted to the 2000 U.S. standard million population.

Age

Both hospitalizations and deaths due to pneumonia and influenza increase with age (Fig. 22-1). Women 65 years of age and older, who represent less than 17 percent of Connecticut's female population, accounted for 95 percent of P & I deaths between 1996 and 1998, and 65 percent of pneumonia hospitalizations between 1993 and 1997.

TRENDS OVER TIME

Age-adjusted death rates for pneumonia and influenza among Connecticut women fell an average of 1.5 percent per year from 1989 to 1998. This change resulted from a decrease in the death rate for P & I among white females. Both the annual and overall changes were statistically significant (Mueller et al., in preparation).



Source: Mueller et al., in preparation; Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001.

Race and Ethnicity

Between 1989 and 1998, the death rate for Hispanic females was significantly lower than that for white females (Table 22-1), whereas for the period 1993 to 1997, hospitalization rates for Hispanic females and for non-Hispanic black females were significantly higher relative to the rate for non-Hispanic white females (403, 436, and 256 per 100,000 females, respectively) (Mueller et al., in preparation; Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001a). The reasons for the reversed white to Hispanic relationship for hospitalization and mortality rates due to pneumonia are not clear.

RISK FACTORS AND GROUPS

The influenza virus is spread by air and direct contact, particularly in crowded or enclosed spaces. Pneumonia is spread by direct or indirect contact with respiratory discharges (coughing, sneezing) from infected persons. Those at high risk for pneumonia and influenza and their complications include the elderly, younger persons with underlying chronic medical conditions, and pregnant women (Table 22-2).

TABLE 22-2
 GROUPS AT HIGH RISK FOR PNEUMONIA OR INFLUENZA
 AND CHRONIC CONDITIONS UNDERLYING PNEUMONIA & INFLUENZA

| Influenza High-Risk Groups |
|--|
| <ul style="list-style-type: none"> ◆ All persons over 65 years of age ◆ Residents of chronic care facilities ◆ Persons with chronic underlying conditions (see below) ◆ Children receiving long-term aspirin treatment ◆ Pregnant women beyond the first trimester of pregnancy (during the flu season) ◆ Organ transplant recipients ◆ Healthcare workers (who pose a transmission risk to others, including patients) |
| Pneumonia High-Risk Groups |
| <ul style="list-style-type: none"> ◆ Persons over age 65 ◆ Persons age 2-64 years with chronic underlying conditions (see below) ◆ Alcoholics ◆ Persons with sickle cell disease ◆ Immunocompromised persons |
| Chronic Underlying Conditions That Increase Pneumonia & Influenza Risk |
| <ul style="list-style-type: none"> ◆ Diabetes mellitus ◆ Chronic lung, liver, kidney, and heart diseases ◆ HIV infection ◆ Cancer |

Source: National Coalition for Adult Immunization, 1999.

PREVENTION AND TREATMENT

Improved personal hygiene (hand washing, shielding coughs and sneezes) and minimizing contacts with infected individuals are important ways to control the spread of influenza and pneumonia. Vaccines and certain medications are effective at preventing and treating many forms of the diseases.

Vaccines

A single dose of pneumococcal vaccine and a yearly flu shot are recommended for all adults 65 years of age and over, and for certain at-risk groups under age 65 (Table 22-2). Vaccines are especially important in long term care facilities, where infectious diseases can spread rapidly.

Although both vaccines are safe, effective, and free to Medicare beneficiaries, they are still underutilized, especially among the elderly and minorities. In 1999, Connecticut was below the national median for both influenza and pneumococcal vaccination coverage (Centers for

Disease Control and Prevention, 2001); more than one-third (34 percent) of non-institutionalized Connecticut women age 65 and over reported they did not get a flu shot during the previous year, and nearly half (48 percent) said they never had a pneumonia vaccination (Centers for Disease Control and Prevention, 2000). Nationally, non-Hispanic whites have the highest vaccination coverage for pneumonia and influenza, whereas non-Hispanic blacks have the lowest coverage (Centers for Disease Control and Prevention, 2001).

According to a survey of Medicare beneficiaries, the main reasons for not receiving a flu shot were a lack of knowledge that it was needed, not thinking of or missing it, and the misconceptions that it could cause or not prevent the flu. The main reasons for not receiving pneumococcal vaccination were lack of knowledge that it was needed and lack of recommendations from physicians (Drociuk, 1999). In older populations, all of these reasons could be eliminated by stronger educational efforts on the part of health care providers, especially physicians.

Medications

Two medications, amantadine hydrochloride and rimantadine hydrochloride, are used to prevent and treat of certain types of influenza. They are recommended mainly for high-risk individuals who were not vaccinated or when a supplement to vaccine is desirable for maximum protection.

Antibiotics like penicillin and erythromycin are effective for treating pneumococcal pneumonia and pneumonia caused by other bacteria and microorganisms. The emergence of antibiotic-resistant bacteria presents a growing challenge, however, to the medical and public health communities and highlights the importance of wider deployment of existing vaccines.

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23. SUICIDE

Suicide is the taking of one's own life and attempted suicide is an unsuccessful attempt to take one's own life through intentional harm. The number of suicides may reflect only 5 percent of suicide attempts (Centers for Disease Control and Prevention, 2000). This bears particular importance for female suicide prevention because individuals who complete suicide are most likely to be male, but those who survive a suicide attempt are most likely to be female (Moscicki, 1994).

PREVALENCE

Between 1996 and 1998, suicide was the 7th leading cause of premature death for all Connecticut women with a total of 188 suicides (age-adjusted rate of 3.6 per 100,000). Connecticut's suicide rate is below the national rate (4.3 per 100,000) (Mueller et al., in preparation; U.S. Department of Health and Human Services, 2000). Female suicide rates have remained stable over the past decade with no significant variations between 1989 and 1998 (Mueller et al., in preparation).

Similar to national trends, female suicides in Connecticut represent only 3 percent of suicide attempts that require hospitalization. Between 1993 and 1997, there were nearly 6,000 hospitalizations (age-adjusted rate of 73 per 100,000) for self-inflicted injury by Connecticut

females (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001). The number of suicide attempts may be underestimated by hospitalization data because some suicide attempts require medical attention provided in clinics or doctor's offices. These encounter data are not collected by any single agency. Other suicide attempts are not reported to any health professional and, therefore, not measurable.

Among the northeastern states, Connecticut ranks 4th in female suicides behind New Hampshire, Vermont, and Maine. Connecticut is also ranked 4th in female self-inflicted injury hospitalizations behind New Hampshire, Maine, and Massachusetts (Table 23-1).

Connecticut females most often attempt and commit suicide by poisoning. In 1997, one-third of all suicides and 85 percent of all self-inflicted injury hospitalizations were caused by drug or gas poisoning (Mueller et al., in preparation; Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001). Not surprisingly, more lethal methods, such as hanging or firearms, represent a greater proportion of female suicides (29 and 14 percent, respectively) than hospitalizations (< 1 percent and 0, respectively).

TABLE 23-1
SUICIDE DEATHS AND SELF-INFLICTED INJURY HOSPITALIZATIONS
Northeastern States, Females Aged 10 and Older, 1992-1996

| State | 1992-1996 Death | | 1996 Hospitalization | |
|---------------|------------------|------|----------------------|------|
| | Rate per 100,000 | Rank | Rate per 100,000 | Rank |
| Connecticut | 4.2 | 4 | 85.1 | 4 |
| Maine | 4.8 | 3 | 93.7 | 2 |
| Massachusetts | 4.0 | 5 | 89.9 | 3 |
| New Hampshire | 5.3 | 1 | 97.7 | 1 |
| New Jersey | 3.6 | 7 | 80.3 | 6 |
| New York | 3.8 | 6 | 59.5 | 8 |
| Rhode Island | 3.8 | 6 | 80.6 | 5 |
| Vermont | 5.0 | 2 | 77.0 | 7 |

Source: Education Development Center, Inc. 2000.

Age

Female suicide and self-inflicted injury hospitalization rates are highest among adolescents. Suicide was the second leading cause of death for Connecticut females between 15 and 24 years of age, exceeding cancer and homicide deaths. Similar to national proportions, nearly one in three female high school students in Connecticut have considered suicide, one out

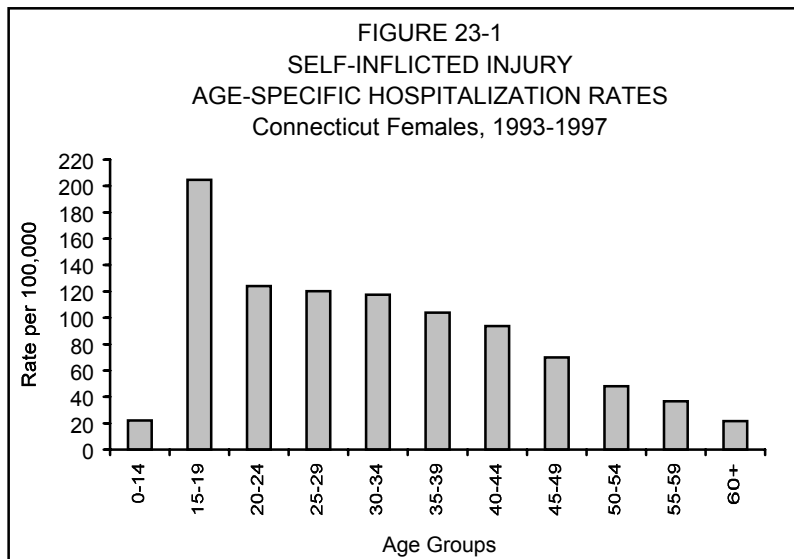
of five have been serious enough to create a plan to carry it out, and one in ten have attempted suicide (Table 23-2).

Self-inflicted injury hospitalization rates were two-thirds higher for female adolescents aged 15 to 19 years than the rates of women between 20 and 34 years (Fig. 23-1). After age 19, self-inflicted injury hospitalization rates drop steadily with age.

TABLE 23-2
SUCIDE ATTEMPTS
U.S. and Connecticut Females, Grades 9-12, 1997

| Suicidal Behaviors | U.S. % | CT % | CT # (estimated) |
|--|-----------|---------|---------------------|
| Seriously considered suicide | 27 | 28 | 27,000 |
| Made a suicide plan | 20 | 20 | 20,000 |
| Attempted suicide | 12 | 13 | 12,000 |
| Suicide attempt required medical attention | 3 | 4 | 4,000 |

Source: Centers for Disease Control and Prevention, 1998.



Source: Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001.

Race and Ethnicity

Suicide deaths and hospitalization rates for white and black women have not changed significantly over the past decade. There were insufficient numbers of suicide deaths among Hispanic, Asian and Pacific Islander, and Native American women during this period to calculate reliable mortality rates. Over 90 percent of the female suicides in Connecticut were among white women (Mueller et al., in preparation).

However, there are reported ethnic disparities in self-inflicted injury hospitalizations. From 1993 to 1997 in Connecticut, Hispanic females had a significantly higher hospitalization rate than white women (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001) (Table 23-3). Nationally, twice as many Hispanic female adolescents (21 percent) attempted suicide than black or white female adolescents (11 percent, and 10 percent, respectively) and twice as many Hispanic women (11 percent) reported being depressed than did black and white women (nearly 6 percent and 5 percent, respectively) (Rouse, 1995). Research has not identified the cause of these disparities but it has been suggested that there are added stresses, such as single parenting, poverty, and the lack of health insurance that place many Hispanic women at risk for mental health problems (Office of Research on Women's

Health, 1998).

RISK FACTORS

The most significant risk factors for suicide include a prior suicide attempt and mental disorders, particularly mood disorders such as depression and bipolar disorder, in combination with alcohol or substance abuse (Regier et al., 1990; U.S. Department of Health and Human Services, 1999). Research indicates that at least 90 percent of all people who kill themselves have a mental or substance abuse disorder (Regier, et al., 1993) and over 90 percent of children and adolescents who commit suicide have a mental disorder before their death (Shaffer & Craft, 1999). Depression is the most significant risk factor among adolescent females and is suggested to increase the risk of suicide twelve-fold, whereas a previous suicide attempt may triple the risk. (Shaffer et al., 1996). Major depressive disorders associated with suicide risk are shown to continue from adolescence into adulthood (Weissman et al., 1999).

Other risk factors for suicide include access to alcohol, illicit drugs, and firearms; and stressful life events, such as getting into trouble at school, an estranged relationship, or a fight among friends (U.S. Department of Health and Human Services, 1999). Exposure to suicide in the family, among friends, and even of a real or

TABLE 23-3
SELF-INFLICTED INJURY HOSPITALIZATIONS
BY RACE AND ETHNICITY
Connecticut Females, 1993-1997

| Race/Ethnicity | Number of Hospitalizations | Age Adjusted Rate per 100,000 |
|--------------------------------------|----------------------------|-------------------------------|
| All races | 5,981 | 73.7 |
| White, non-Hispanic | 4,664 | 71.6 |
| Black, non-Hispanic | 515 | 65.1 |
| Asian/Pacific Islander, non-Hispanic | 32 | 16.8 [†] |
| Native American, non-Hispanic | 5 | † |
| Hispanic | 654 | 97.9 [‡] |

Source: Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001.

[†] Statistics not calculated for fewer than 25 events.

[‡] Rate significantly different from that of whites (p < .05)

Notes: U.S. Census Bureau population estimates used for rate calculations. Rates adjusted to the 2000 U.S. standard million population.

fictional celebrity can contribute to suicidal thoughts or behavior (Gould & Shaffer, 1986). Research also links sexual orientation and depression with suicidal tendencies (Garofolo et al., 1999).

a medical emergency and requires appropriate treatment from mental health and medical professionals. The community of medical professionals (e.g., internists, pediatricians, nurse practitioners) are often the only source of health

TABLE 23-4
SUICIDE RISK FACTORS

| | |
|---|--|
| ◆ Previous suicide attempt | ◆ Mental disorders, such as depression and bipolar disorder |
| ◆ Adverse life events | ◆ Co-occurring mental illness and alcohol or substance abuse |
| ◆ Impulsive or aggressive tendencies | ◆ Family history of mental illness or substance abuse |
| ◆ Access to lethal methods, especially firearms | ◆ Exposure to the suicidal behavior of others, including family, peers, or in the news or fiction stories |
| ◆ Family history of suicide | ◆ Family violence, including physical or sexual abuse |
| ◆ Hopelessness | ◆ Unwillingness to seek help due to stigma attached to mental and substance abuse disorders or suicidal thoughts |
| ◆ Physical illness | |
| ◆ Feelings of isolation | |
| ◆ Cultural and religious beliefs | |
| ◆ Barriers to accessing mental health treatment | |

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

ECONOMIC BURDEN

Attempted and completed suicides in Connecticut were estimated to cost over \$8 million in medical expenses and over \$216 million in lost wages during 1996 (Education Development Center, 2000). However, these figures are substantially underestimated. Hospitalization charges for self-inflicted injury at acute care facilities alone totaled over \$8 million in 1997 for Connecticut females, an average of \$7,000 per hospitalization (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001). This does not include inpatient or outpatient care at psychiatric institutions.

PREVENTION AND TREATMENT

Suicide prevention strategies should be comprehensive and include a focus on increasing the recognition and appropriate response to suicide risk factors. The Surgeon General recommends broadening the public's awareness of suicide and enhancing public health and clinical services (U.S. Department of Health and Human Services, 1999). Every suicide attempt is

services for many adolescents and adults, including mental health services. Nationally, 27 percent of young people aged 9 to 17 years with a psychological condition receive treatment in the medical services sector (Howard et al., 1996), and 20 percent use mental health services in their schools (Reiger et al., 1990).

It is important to recognize that screening within the medical, mental health, and school community (e.g., school nurses, youth program staff, teachers, counselors) can be effective for early detection and referral to appropriate treatment of depression and suicidal behavior.

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24. VIOLENCE AGAINST WOMEN

INTRODUCTION

Violence is a physical or sexual act carried out with the intention or perceived intention of causing physical pain or injury to another person. The U.S. Department of Health and Human Services includes psychological and emotional abuse as a form of violence when there has been prior physical or sexual violence (Centers for Disease Control and Prevention, 1999).

There are three forms of violence that predominantly victimize women: sexual assault, family violence, and intimate partner violence (IPV). Family violence and IPV are defined by the relationship of the victim to the perpetrator, whereas sexual assault is defined according to the type of violence.

Sexual assault, including rape, is the use of physical force to attempt or compel a person to engage in a sexual act against their will. Victims include persons without the ability to communicate unwillingness to engage in the act because of age, illness, disability, the influence of alcohol or other drugs, or intimidation (Petersen, 1998). The rate of female rape and sexual assault was 7.5 times greater than the rate for males (Rennison, 2000).

Family violence includes the physical or sexual assault or abuse of children, partners, siblings, or parents living together in a household. Over 75 percent of victims of family violence are female (Connecticut Department of Public Safety, 1998).

Physical, sexual, and psychological violence against women is perpetrated most often by an intimate partner, but includes assault by a relative, acquaintance, or stranger. Two out of every five women in the United States have experienced violence once in their lifetime (Tjaden & Thoennes, 1998; The Commonwealth Fund, 1999) and two-thirds (68 percent) knew their offender as a partner, friend, or relative (Rennison, 2000). Approximately one in five

female students reported being physically or sexually abused by a dating partner (Silverman et al., 2001). Nationally, there are an estimated 1.5 million women who are physically or sexually assaulted by a current or former intimate partner every year (Centers for Disease Control and Prevention, 2000).

SCOPE OF THE PROBLEM

The full extent of violence against women is difficult to assess due to inconsistent reporting methods and the limited number of data sources. There are different measures of violence against women, ranging from an assessment of self-reported prevalence to a review of police reports and arrests. These methods are further complicated when the research considers the relationship between the victim and the assailant or the type of violence inflicted. Secondly, there are gaps in the surveillance of injury and trauma services provided by private practitioners and emergency departments, where over 80 percent of medical care is provided to victims of violence (Tjaden and Thoennes, 2000). Lastly, most existing data sources used to determine the prevalence of violence against women rely on the victims' willingness to disclose their victimization. This willingness is often justifiably limited due to the fear of reprisal, of not being believed, or of being blamed for instigating the act (Hadley, 1996). Despite these limitations, the data presented below establish violence against women as a significant public health problem in Connecticut.

Self-reported

In Connecticut in 1995, an estimated 1,500 per 100,000 (20,000) women reported that they were victims of physical violence from a current or former spouse, boyfriend, or girlfriend during the past year (Adams, 1998) (Table 24-1).

Crisis Services

Nearly 40,000 women and children requested the services of 18 domestic violence shelter programs in Connecticut from July 1, 1998 to June 30, 1999 (Table 24-1). Approximately 2,500 of them required the use of an emergency shelter because they were in serious physical danger and had no other safe haven (Connecticut Coalition Against Domestic Violence, 1999). During that same time period, nearly 3,000 women reported a sexual assault to a crisis service. Two-thirds of these women were between the ages of 18 and 44 years and 86 percent knew their assailant. Only 20 percent of the sexual assault victims that contacted a crisis service reported the assault to the police or received medical assistance (Connecticut Sexual Assault Crisis Services, 1999).

Criminal Offenses

Eighty percent of the victims of family violence offenses that involved police intervention during the past decade were women. Over two-thirds of all family violence victims were intimate partners of the offenders (Connecticut Department of Public Safety, 1988-1999).

Rape is the crime least often reported to law enforcement, often due to fear of retribution and shame of discussing the incident (Rennison, 2000). Marital rape is often overlooked since women are more reluctant to reveal rape by their spouses, even more so than to disclose physical abuse (Geffner and Pagelow, 1990). The number of attempted and completed rapes of females reported in Connecticut has decreased during the past decade from 842 in 1988 to 727 in 1998 but the rate remained stable at 60 per 100,000 women aged 16 years and older (Connecticut Department of Public Safety, 1988-1998). These rates do not reflect the number of sexual assaults other than rape or statutory rape of females under 16 years of age.

Hospitalizations

In 1997, over 22,000 Connecticut women 18 years and older were hospitalized for all injuries, 152 were identified as victims of assault, 32 were victims of abuse, and 6 were rape victims. Twenty of the 32 victims of abuse were assaulted by their current or former intimate partner (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001).

TABLE 24-1
SUMMARY MEASURES OF VIOLENCE AGAINST WOMEN
Connecticut Females, Selected Years and Age Groups

| Violence Measure | Number | Rate/100,000 females ^a |
|---|---------------------|-----------------------------------|
| Family violence victims receiving shelter services, all ages, 1999 | 38,617 ^b | 2,290 |
| Self-reported domestic violence victims, aged 18+ years, 1995 | 20,000 ^c | 1,540 |
| Family violence victims involving police intervention, all ages, 1999 | 12,906 ^d | 780 |
| Rape victims receiving crisis services, all ages, 1999 | 2,921 ^e | 170 |
| Rape victims involving police intervention, aged 16+ years, 1998 | 727 ^d | 60 |
| Assault hospitalizations, all ages, 1997 | 189 ^f | 10 |

^a Rates calculated using U.S. Census Bureau population estimates.

^b Connecticut Coalition Against Domestic Violence, 1999.

^c Adams, 1998.

^d Connecticut Department of Public Safety, 1998.

^e Connecticut Sexual Assault Crisis Services, Inc., 1999.

^f Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001.

Homicides

One in three female homicides in the U.S. and Connecticut were the result of intimate partner violence (Saltzman, et al., 1999; Fox and Zawitz, 2000; Connecticut Department of Public Safety, 1999). From 1996 to 1998, homicide was the fifteenth leading cause of death and the ninth leading cause of premature death for all women in Connecticut. There was no significant change in homicide rates over the past decade (Table 24-2).

Younger women are at greater risk of violent death. Homicide was the third leading cause of death for women aged 15-24 years of age and the leading cause of death for black women in that age group. From 1996 to 1998, the rate of homicide deaths among black females was significantly higher than the rate for white females. There were insufficient numbers of homicide deaths among Hispanic, Asian and Pacific Islander, and Native American women in Connecticut during this period to calculate reliable rates (Table 24-2).

HEALTH CONSEQUENCES

Women who suffer from physical and sexual violence are more likely to report general

health problems, exhibit depressive symptoms, or have a mental health diagnosis. Women who experience ongoing abuse may not be able to make appropriate choices to reduce their health risks or obtain complete, effective care for other conditions (Cohen et al., 2000). Violence is associated with unintended pregnancy, gynecological diagnosis, abortion, and sexually transmitted disease, including HIV (Campbell et al., 2000; United National Children's Fund, 2000).

Fatal outcomes from violence include homicide, suicide, maternal mortality, and AIDS-related deaths. Nonfatal outcomes include head and facial fractures, burns, broken bones, internal bleeding, neurological damage, and permanent physical disability. Mental health consequences of violence include post-traumatic stress disorder, eating disorders, alcohol or substance abuse, and major depression (Centers for Disease Prevention and Control, 1998; United National Children's Fund, 2000). Of the 20 female Connecticut residents hospitalized for abuse in 1997, 12 were admitted for broken bones or facial contusions, and 11 were diagnosed with alcohol or drug dependence (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001).

TABLE 24-2
HOMICIDE AND LEGAL INTERVENTION DEATHS
BY RACE AND ETHNICITY
Connecticut Females, 1989-1991 and 1996-1998

| Race/Ethnicity | 1989-1991 | | 1996-1998 | |
|------------------------|------------------|---------------------------------------|------------------|---------------------------------------|
| | Number of Deaths | Age Adjusted Death Rate (per 100,000) | Number of Deaths | Age Adjusted Death Rate (per 100,000) |
| All races | 127 | 2.4 | 106 | 2.1 |
| White | 89 | 1.9 | 61 | 1.4 |
| African American/Black | 35 | 6.9 | 42 | 8.3 [‡] |
| Asian/Pacific Islander | 1 | † | 1 | † |
| Native American | 0 | † | 2 | † |
| Hispanic/Latina | 13 | † | 9 | † |

Source: Mueller et al., in preparation.

† Statistics not calculated for fewer than 15 events.

‡ Rate significantly different from that of whites ($p < .05$)

Notes: U.S. Census Bureau population estimates used for rate calculations. Rates adjusted to the 2000 U.S. standard million population.

RISK FACTORS

Violence touches women from all races, religions, socioeconomic backgrounds, and lifestyles living in cities, suburbs, and rural areas. However, some women are at greater risk than others for sexual assault, family violence, and intimate partner violence.

History of Abuse

Women with a childhood history of abuse are at greater risk of experiencing violence later in life (Ammerman and Hersen, 1990; Cohen et al., 2000). Nearly two-thirds of women reporting childhood abuse experienced family violence as an adult, compared with only one-quarter of women without a history of childhood abuse (The Commonwealth Fund, 1999).

The history of abuse is also a significant risk factor for the offender. Violence is most common in adults who, as children or adolescents, witnessed or were victims of family violence (Hotaling and Sugarman, 1986; Tjaden and Thoennes, 2000). Connecticut police report that children were present or participated in 45 percent of the family violence incidents during the past decade (Connecticut Department of Public Safety, 1989-1999).

Income

Women living on low incomes or experiencing economic stress present a higher risk for violence. Nearly one-half of women with incomes of \$16,000 or less reported at least once incident of family violence or abuse in their lifetime, but only one-third with incomes of more than \$50,000 (Rennison, 2000). More than half (52 percent) of women who said they have “a lot of trouble” paying for food, phone, gas and electricity reported being victims of domestic abuse (The Commonwealth Fund, 1999).

Urban Residence

Connecticut’s rates of family violence and rape were 80 percent higher in cities with

populations greater than 65,000 persons¹ than the statewide rates (518 vs. 930 per 100,000, respectively). Similarly, the rate of rape offenses in urban areas was higher (79 per 100,000 women) than the statewide rate of 44 rape offenses per 100,000 women (Connecticut Department of Public Safety, 1998). Urban populations represent 25 percent of the state’s total population but over 40 percent of all reported family violence and rape offenses in Connecticut. The higher rates of violence against women in urban areas may be attributable to many factors, including the concentration of persons in poverty and the proximity of neighbors and witnesses who call the police when violence erupts (Miles-Doan and Kelly, 1997).

ECONOMIC BURDEN

The economic burden of violence against women includes the cost of medical and psychological treatment, police services, criminal justice (prison, prosecution), emergency shelters, social services, and productivity loss for working women, their families, and the offenders. Annual medical costs and lost productivity resulting from violence against women are estimated at \$5 to \$10 billion per year (U.S. Bureau of National Affairs, 1990; Meyer, 1992; Laurence and Spalter-Roth, 1996). Applying national per capita estimates (\$19-38) to Connecticut’s 1998 population estimates, \$62-124 million was spent in Connecticut for medical costs and lost productivity resulting from violence against women.

PREVENTION AND TREATMENT

Current prevention strategies focus on the medical and criminal justice communities’ assessment and intervention for both offenders and victims. Screening for offenders requires the recognition of violence risk factors such as: alcohol and drug abuse, mental illness, social isolation, lack of close family ties, low self-

¹ Bridgeport, Danbury, Hartford, New Britain, New Haven, Norwalk, Stamford, and Waterbury.

esteem, lack of financial resources, and aggressive or hostile behavior (Heise and Gottemoeller, 1999). Interventions include anger management counseling and violence prevention programs. Connecticut courts try to deter domestic violence with 6-month restraining orders to legally keep offenders away from victims when physical pain or injury is threatened or present in a household. However, restraining orders do not always prevent a recurrence of domestic violence. For example, Connecticut police reported a prior court order existed in 17 percent of the 18,948 domestic violence incidents in 1999 (Connecticut Department of Public Safety, 1999).

Female victims of intimate partner violence use a disproportionate share of health care services, including more visits to emergency departments, primary care facilities, and mental health agencies than nonabused women (Coker et al., 2000). Therefore, the opportunity exists for assessment and early intervention by the medical community for victims of violence to decrease the risk of further injuries and death. Connecticut law allows the Department of Social Services to administer shelter services and temporary family assistance for victims of domestic violence (C.G.S. 17b-112 and 17b-850).

Confidential 24-hour hotlines provide domestic violence and sexual assault victims with crisis information services, information about laws, protections, counseling support, and referrals to community services. There are 2 statewide 24-hour hotlines for violence against women sponsored by the Connecticut Coalition Against Domestic Violence (CCADV) and the Connecticut Sexual Assault Crisis Service (CONNSACS). CCADV received over 23,000 hotline calls in fiscal year 1999 while CONNSACS received over 14,000 calls during the same time period. This averages out to nearly 100 telephone calls a day seeking crisis services for domestic violence or sexual assault.

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25. FALLS AND FALL-RELATED INJURIES

INTRODUCTION

Falls, related injuries, and their complications are a serious public health concern among older women. It is estimated that every year about 30 percent of people over age 65 experience a fall (Tinetti, et al., 1989; Sattin, 1992) and that 20 percent to 30 percent of these individuals (28,000 to 42,000 women and men in Connecticut) sustain injuries that reduce their independence and mobility and increase their risk for premature death (Alexander, et al., 1992). Hip fractures, the most serious fall-related injury, can drastically reduce a person's quality of life by imposing functional limitations that lead to gradual physical deterioration. Over 90 percent of hip fractures are the direct result of a fall (Grisso et al., 1991; Stevens and Olson, 2000). Because the Connecticut population is aging, the number of hip fractures is likely to increase substantially in the next two decades.

SCOPE OF THE PROBLEM

Deaths

Between 1989 and 1998, 888 Connecticut females died of fall or fall-related injuries. Ninety percent of these deaths occurred among women aged 65 and over. Falls and fall-related injuries were the second-ranked cause of unintentional injury death among all Connecticut women during this period and they were the leading cause of unintentional injury death among women 70 and over. Fall and fall-related death rates increase sharply with age with the highest death rates occurring among women aged 85 and older (Mueller et al, in preparation).

The vast majority of female deaths due to falls and fall-related injuries occur among white women (Table 25-1). Between 1989 and 1998, 98 percent of all such deaths occurred among white women. There were too few deaths among black, Hispanic, Asian and Pacific Islander, and Native American women during this period to calculate reliable rates. Death rates due to falls and fall-related injuries among Connecticut females remained about the same between 1989 and 1998.

TABLE 25-1
FALL AND FALL-RELATED INJURY DEATHS BY RACE AND ETHNICITY
Connecticut Females, 1989-1991 and 1996-1998

| Race/Ethnicity | 1989-1991 | | 1996-1998 | |
|------------------------|------------------|---------------------------------------|------------------|---------------------------------------|
| | Number of Deaths | Age Adjusted Death Rate (per 100,000) | Number of Deaths | Age Adjusted Death Rate (per 100,000) |
| All races | 282 | 4.4 | 293 | 4.0 |
| White | 275 | 4.5 | 287 | 4.1 |
| African American/Black | 5 | † | 3 | † |
| Asian/Pacific Islander | 0 | † | 1 | † |
| Native American | 0 | † | 2 | † |
| Hispanic/Latina | 4 | † | 3 | † |

Source: Mueller et al, in preparation.

† Statistics not calculated for fewer than 15 events.

Notes: U.S. Census Bureau population estimates used for rate calculations. Rates adjusted to the 2000 U.S. standard million population.

During the 1996 to 1998 period, Connecticut rates were not significantly different from U.S. rates (Mueller et al, in preparation). Connecticut data are consistent with 1996 national figures showing that white women were twice as likely as black women aged 65 and older to die of fall-related deaths (Centers for Disease Control and Prevention, 1999).

Hospitalizations

The most common fall-related injuries among older Americans are fractures of the hip, spine, upper arm, forearm, and bones of the pelvis, hand, and ankle. Of these, hip fractures are the most serious injury. Fifty percent of all Americans hospitalized for hip fractures do not regain their previous level of functioning (Stevens et al., 1999; Stevens and Olson, 2000). Between 1993 and 1997, there were more than 33,000 hospitalizations for fall and fall-related injuries among Connecticut females. About 78 percent of these hospitalizations occurred among women aged 65 and over. The vast majority of fall and fall-related hospitalizations were among white women. The age-adjusted hospitalization rate of white women was about four times higher than that of Asian and Pacific Islanders, more than twice that of Latinas, and 1.5 times higher than that of black women. There were too few fall and fall-related hospitalizations among Native American women to calculate reliable

rates (Table 25-2). The median charge for all fall and fall-related injury hospitalizations was about \$10,700 in 1997 (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001). Connecticut data are consistent with 1996 national figures indicating that white women aged 65 and over were significantly more likely than black women to be hospitalized for hip fractures (Centers for Disease Control and Prevention, 1999).

RISK FACTORS

Several physiological, behavioral, and environmental factors increase the risk for falls and related injuries. The risk of falling increases with age with the highest fall and fall-related mortality rate occurring in the oldest age group (85 and older). Health conditions that limit the performance of routine daily activities such as dressing or bathing, muscle weakness or problems with balance, vision problems, and osteoporosis, a condition that increases bone fragility, are highly associated with increased risk for falls (Table 25-3). Also, the presence of underlying chronic diseases such as cerebrovascular, cardiovascular, and neurologic disorders may increase the number of falls among older persons (Sattin, 1992). Behavioral factors include the use of multiple medications or

TABLE 25-2
FALL AND FALL-RELATED INJURY HOSPITALIZATIONS
BY RACE AND ETHNICITY
Connecticut Females, 1993-97

| Race/Ethnicity | Number | Age Adjusted Rate (per 100,000) |
|------------------------|--------|------------------------------------|
| All races | 33,196 | 311.6 |
| White | 30,917 | 316.4 |
| African American/Black | 1,158 | 208.9 [‡] |
| Asian/Pacific Islander | 57 | 78.7 [‡] |
| Native American | 7 | † |
| Hispanic/Latina | 610 | 152.4 [‡] |

Source: Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001.

† Statistics not calculated for fewer than 25 events.

‡ Rate significantly different from that of whites ($p < .05$).

Notes: U.S. Census Bureau population estimates used for rate calculations. Rates adjusted to the 2000 U.S. standard million population.

psychoactive medications, sedentary lifestyle, and a history of falls. Environmental factors include home hazards like floor clutter, slippery surfaces, and poor lighting (Stevens and Olson, 2000; Centers for Disease Control and Prevention, 2000). The risk of falling has been shown to increase with the number of risk factors present (Tinetti, et al., 1988; Nevitt et al., 1989); therefore, programs that target several of these modifiable risk factors offer promise in reducing fall incidence.

White and Asian females are considered

(Tinetti et al., 1994). These include regular exercise to improve strength, balance, mobility, and flexibility (Judge et al., 1993; Lord et al., 1993; Lord et al., 1996). Tai Chi is a commonly used exercise in intervention studies. Other strategies include 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). Early detection and treatment of

TABLE 25-3
RISK FACTORS FOR FALLS AND FALL-RELATED INJURY AMONG OLDER ADULTS

| | |
|------------------------------------|---|
| • Increasing age | • Low body mass index |
| • Muscle weakness | • Use of psychoactive medications (tranquilizers or antidepressants) |
| • Lack of physical activity | • Some combinations of medications |
| • Difficulties in gait and balance | • A history of falls |
| • Visual impairment | • Home environmental hazards (e.g. poor lighting, loose rugs, and unstable furniture) |
| • Osteoporosis | |

Source: Stevens and Olson, 2000.

high risk groups for osteoporosis (U.S. Department of Health and Human Services, 2001). White females are a high risk group for fall-related hospitalizations and deaths (Centers for Disease Control and Prevention, 1999). State and national data indicate that white women incur significantly more fall-related hospitalizations and deaths than do black women. Researchers have suggested that greater bone mass among black compared to white females may partially explain their lower prevalence of fall-related injuries (Snelling et al., 2001). 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, 2001).

PREVENTION AND TREATMENT

Combinations of strategies have been shown to reduce the risk of falling among older women

common vision conditions such as cataracts and glaucoma might reduce falls. Prevention and appropriate treatment of chronic illnesses such as cardiovascular disease could decrease the number of falls and related injuries (Sattin, 1992).

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). Basic clinical screening tests can accurately identify seniors who are at high risk for falls (Centers for Disease Control and Prevention, 2000). Other promising prevention strategies include identifying footwear that promotes stability and balance, developing more effective home lighting systems, and designing undergarments with absorbent hip pads (Stevens and Olson, 2000).

Such multifactorial approaches have been shown to be cost-effective (Rizzo et al. 1996).

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MOVING FORWARD ON WOMEN'S HEALTH

Connecticut Women's Health was developed to provide baseline data and a context for assessing the overall health and well being of Connecticut women at the beginning of this new millennium. It is hoped that health professionals, policymakers, academics, and advocates will be able to build upon this foundation to improve the health of all women in Connecticut in the coming years. *Connecticut Women's Health* examines more than 24 selected health conditions important to women across their life span and establishes, wherever possible, the status of women with respect to these conditions. It describes the social context of women's health in the United States, profiles the demographics of Connecticut's women, and discusses the threats to health posed by access barriers to health care services for women.

Although the health of Connecticut women compares well to the health of women nationally, some feel that the health of women everywhere in the United States could be significantly improved (National Women's Law Center et al., 2000). The findings from *Connecticut Women's Health* do not contradict that opinion, underlining the fact that women have a long way to go to achieve the definition of health articulated by the World Health Organization, "a state of complete well being, physical, social, and mental, and not merely the absence of disease or infirmity" (World Health Organization, as quoted in Committee for the Study of the Future of Public Health, 1988, pg. 39).

But where should Connecticut begin? With so many issues of concern to women, political leaders, scientists, practitioners, and advocates can debate endlessly which issues should have priority. However, one thing is certain. An ongoing discourse about the condition and progress of women's health is needed, and it should be led by the women of Connecticut, because their health and well-being depend on the outcome.

Since more than 1.7 million residents are directly affected, there is no shortage of opinion about what should be done to improve women's health in Connecticut. To move the discussion forward, consider some of this report's notable findings:

- ◆ From the perspective of mortality, the leading causes of death for women in Connecticut are cardiovascular disease and cancer, led by lung and breast cancer (age-adjusted mortality rates of 286, 41, and 30 deaths per 100,000 women, respectively; Mueller et al., in preparation). These diseases share a number of modifiable risk factors in common: smoking, diet, overweight, and physical inactivity. Although the Behavioral Risk Factor Surveillance System provides much information on these conditions and risk factors, greater investment in this important public health tool would improve our knowledge of these and other health conditions by allowing more precise estimates of risk for local communities and small population groups.
- ◆ From the perspective of sheer magnitude, i.e., the number of people affected, violence against women is a serious health threat. Estimates are that 40 percent of all women, or 640,000 Connecticut residents, are touched by violence at some point in their lives. This statistic includes only female victims of violence, and would be greater if all those persons significantly involved in the violence were also counted, for example, the perpetrators, family members, co-workers, friends, and neighbors. Estimates of the social and economic costs are proportionally high. This problem is widely believed to be underreported and underestimated. In counterpoint to the issue's magnitude, the data sources that are used to track violence against women are fragmented, and

Connecticut currently has no organized or integrated surveillance system.

- ◆ Once again, from the perspective of magnitude, one woman in five over the age of 50 has clinically defined osteoporosis, and one in two over 50 will sustain an osteoporosis-related fracture in her lifetime. Osteoporosis is a condition that progresses steadily after menopause, and leads to significant mortality as a consequence of hip fracture, a common complication, and to permanent disability. As with violence, Connecticut has very little precise, population-based data on the prevalence of the condition, and no organized or integrated surveillance system.
- ◆ Disparities in health status, the utilization and outcomes of medical treatments, health insurance status, and access to health services have been widely documented between white Americans and Americans of color in the public health literature. Connecticut is not an exception to these findings, with significant health disparities across several indicators of mortality, chronic diseases, infectious and sexually-transmitted diseases, pregnancy and birth outcomes, and hospitalizations (Hynes et al., 1999). This report documents similar patterns among Connecticut women. For all 13 health conditions presented in this report where reliable, comparative statistics could be calculated, there were significant differences between the age-adjusted death rates of whites and at least one racial-ethnic group (Mueller et al., in preparation). Furthermore, for all 20 health conditions presented in this report where reliable, comparative statistics could be calculated, there were significant differences between the age-adjusted hospitalization rates of whites and at least one racial-ethnic group (Connecticut Department of Public Health, Division of Policy, Planning, and Analysis, 2001). The magnitude and pervasiveness of the health disparities based on race and ethnicity affect every woman in the state, even though the causes of these disparities

are not clear. For black women, these disparities translate into 5 lost years of life (in 1998, life expectancy for white women at birth was 79.9 years, for black women, 74.7 years; Kramarow et al., 1999).

- ◆ The relationship between our social environment and health has been a core theme of public health for more than a century. The influence of gender, income, education, occupation, family structure, and other factors on health is well documented. But in this country, the dominant biomedical model of health views these social factors as contextual aspects of society, which, although affecting health, are not subject to intervention through the traditional tools of medicine. As a result, the social determinants of health are not viewed as characteristics that we, as a society, can decide to change for the benefit of all (Ruzek, 1993). As a result of the gender inequities in our society, women earn less, have less education, and hold lower status occupations than men. This affects women's access to safer and more sanitary housing, better nutrition, healthier foods, safer working conditions, better employee benefits, and better access to health care. Because of society's traditional gender roles, women are more likely to care for children, elders, and other family members, and this affects their health negatively. Women are more likely to be single heads of household, a family structure that has increased in proportion from one in ten to one in five families since 1970. Sixty percent of these families contain young children. Households headed by single females are twice as likely to be living in poverty as households headed by single men, and five times as likely as families headed by two parents (U.S. Bureau of the Census, 1995). Although many epidemiological studies have demonstrated the importance of these social determinants on health, few states have incorporated social indicators into their primary surveillance systems so that their effect on the health of the population can be routinely monitored and studied.

How will these priorities, and others, be addressed? First, through enhanced surveillance and research to improve our current epidemiological knowledge of the social, behavioral, and biological factors which underlie these conditions. Second, through the development of public policies which support and strengthen the social and biological foundations for good health. And third, through the implementation of programs and activities which address the specific needs of individuals and communities throughout the state.

Research and Surveillance

Historically, women's health research focused on reproductive functions, and drew conclusions about non-reproductive conditions largely from studies based on men. But with the lengthening of women's life expectancy and the advent of the modern women's health movement in the 1960's, researchers and academicians have developed a greater interest in and knowledge about women's experience of health and disease across the life span. The scope and volume of research focused on women have increased considerably during the last decade as the federal government reorganized its health administration and funding priorities. Progress remains to be made, however, to improve our understanding of the relationships between health and the biological and social determinants of health: age, sex, genetics, family history, race, gender, ethnicity, culture, socioeconomic status, education, and occupation (Office of Disease Prevention and Health Promotion, 1997; Weisman, 1997).

The Connecticut Department of Public Health recognizes the need for additional research into women's health, and particularly for expanded disease surveillance. Efforts are currently underway to expand the surveillance of multicultural health; the social determinants of health; the incidence of certain chronic diseases and their risk factors; and health services. In this report discussions of certain health conditions, for example osteoporosis, violence, and autoimmune diseases, were limited because of a paucity of state-specific data. Some conditions, for example eating disorders and carpal tunnel

syndrome, were not discussed. Mental health, especially depression, and substance use, including alcohol use, are important health issues for women, but statewide, population-based data are limited.

Public Policy Development

Over the last decade, the federal Department of Health and Human Services (DHHS) recognized women's health as a unique and important domain for research and programmatic activity. This is reflected in the formation of three distinct administrative units. DHHS established the Office on Women's Health, which addresses gender inequities in research, the provision of health care services, and health education. The National Institutes of Health created the Office of Research on Women's Health, which promotes biomedical and behavioral research to improve women's health. And the Food and Drug Administration established the Office of Women's Health, which provides information specifically directed to women on nutrition and cosmetics. In addition, the importance of women's health is reflected in the goals of *Healthy People 2010*, the federal government's statement of its national health goals for the next decade. These goals are to eliminate health disparities related to gender, race, ethnicity, education, income, and sexual orientation (U.S. Department of Health and Human Services, 2000).

The Connecticut Department of Public Health has identified women's health as a statewide priority for policy and program development. In April 2000, the Department commenced this policy direction by hosting its first Women's Health Summit to encourage public and private health professionals to discuss women's health issues. In August 2000, the Division of Policy, Planning, and Analysis began to develop *Connecticut Women's Health* to provide a foundation for the discussion of women's health issues. And recently, the Department launched its Woman to Woman initiative, which promotes health and disease prevention among the state's less advantaged women.

Program Implementation and Population Needs

Changing policy priorities and epidemiological knowledge related to women's health has direct implications for public health programs. Traditionally, public health programs for women focused on reproductive and maternal functions. But as women's life spans increased and attitudes toward health changed, chronic and other health conditions took on an added importance.

To address these issues, the Department of Public Health's Breast and Cervical Cancer Early Detection Program is providing underserved Connecticut women with screening and diagnostic services for breast and cervical cancer. Recently, the Department formed the Connecticut Cancer Consortium, a partnership with the Yale Cancer Center, the University of Connecticut Health Center, and the Connecticut State Medical Society. The Consortium is developing a statewide cancer control plan designed to reduce the burden of lung, breast, and colorectal cancer through primary and secondary prevention, including surveillance, screening, and public and professional education. The Department's Women and Health program provides cardiovascular risk assessment, screening, and intervention to women between the ages of 50 and 64 who are below 200 percent of the federal poverty line, have no health insurance, or whose health insurance does not cover screening. One goal of the program is to provide women a low-cost intervention which they can follow if they are at risk, in contrast to the typical, high-cost pharmacological intervention.

The control of infectious diseases, particularly STDs and HIV/AIDS, has been a health priority for women for some time. The Department's AIDS Prevention and Treatment Services program coordinates counseling and testing services, risk reduction education, outreach to drug users, needle exchange, referral to drug treatment and care, case management, social marketing, and public information campaigns. The STD Control Program conducts

urine-based STD testing through schools, correctional facilities, and Planned Parenthood clinics, and provides counseling, treatment, and public education.

To address violence against women, the Department supports both community programs targeted to battered women for early medical treatment and referral to support services, and the training of health care professionals to identify, treat, and help plan for the safety of battered women.

Finally, the Department's maternal and child health programs continue to provide an array of reproductive- and family-related programs designed to address women's needs. For example, family planning programs designed for low-income, uninsured, underinsured, minority, or adolescent women address pregnancy prevention, responsible parenthood, and safe sex practices. Access to early prenatal care is encouraged through free pregnancy testing, options counseling, and assistance in referrals for care where appropriate. The Maternal PKU program, in collaboration the Department and the University of Connecticut Health Center and Yale University School of Medicine, provides genetic and nutritional counseling and high-risk pregnancy care. The Pregnancy Exposure Information Service at the University of Connecticut Health Center is funded by the Department and provides information concerning the potential teratogenic effects of prescription drugs, maternal illness, infections, and occupational exposures. The Comadrona program is supported by the Department to facilitate access to comprehensive and continuous prenatal and pediatric services for low-income families of Hispanic origin. The Department, in collaboration with the University of Connecticut Health Center, directs the Pregnancy-Related Mortality Surveillance Program, which is designed to identify preventable causes of pregnancy-related mortality and facilitate educational programs to prevent such deaths. Finally, the Department administers WIC, the supplemental food program for women, infants, and children sponsored by the U.S. Department of Agriculture.

As the discussion of women's health in Connecticut moves forward, the Department of Public Health encourages health care providers, policy makers, and consumers to consider policy, program, and research initiatives that will improve the health status of all Connecticut's women.

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APPENDIX A TECHNICAL NOTES

Age-adjusted rates are used to compare relative risk across groups and over time. They represent weighted statistical averages of the age-specific rates, in which the weights represent the fixed population proportions by age. The age-adjusted rates in this report were computed by the direct method using the 1940 and 2000 U.S. standard populations. The total of expected deaths or hospitalizations divided by the total of the standard population and multiplied by 100,000 yields the age-adjusted rate per 100,000. The 1940 and 2000 U.S. standard million population distributions are shown below:

| Age group | 1940 | 2000 |
|-----------|-----------|-----------|
| 0-4 | 80,057 | 69,136 |
| 5-9 | 81,151 | 72,533 |
| 10-14 | 89,209 | 73,032 |
| 15-19 | 93,665 | 72,169 |
| 20-24 | 88,002 | 66,478 |
| 25-29 | 84,280 | 64,530 |
| 30-34 | 77,787 | 71,044 |
| 35-39 | 72,501 | 80,762 |
| 40-44 | 66,744 | 81,851 |
| 45-49 | 62,696 | 72,118 |
| 50-54 | 55,116 | 62,716 |
| 55-59 | 44,559 | 48,454 |
| 60-64 | 36,129 | 38,793 |
| 65-69 | 28,519 | 34,264 |
| 70-74 | 19,519 | 31,773 |
| 75-79 | 11,423 | 27,000 |
| 80-84 | 5,878 | 17,842 |
| 85+ | 2,765 | 15,508 |
| Total | 1,000,000 | 1,000,000 |

Age-specific rates can reveal age-related differences that are hidden in overall age-adjusted rates. For this report, the age-specific rate was calculated based on the number of deaths among individuals within a specific age group and calendar year, divided by the mid-year population of all residents in that same age group and then multiplied by 100,000.

Age standardization is a technique that allows for the comparison of rates in two or more populations. The National Center for Health Statistics (NCHS) has used the 1940 standard million population in reporting national mortality statistics for over 50 years. Implementation of the year 2000 population will be implemented with deaths occurring in 1999. Age-adjustment based on the year 2000 standard results in age-adjusted death rates that are larger than those based on the 1940 standard. The new standard will affect trends in age-adjusted death rates for certain causes of death and will decrease race and ethnicity differentials in age-adjusted death rates.

Age-standardized incidence rates are used to express the number of new cancers of a specific site/type occurring in a specified population during a year, expressed as the number of cancers per 100,000 population. These rates refer to the numbers of cancers, not to the numbers of people with cancers. All incidence rates are adjusted to the 1970 U.S. standard population. Rates are for invasive cancers only, unless otherwise specified.

Body Mass Index (BMI) is a measure of obesity. $BMI = \text{weight in kilograms} / \text{height in meters}^2$

Cause-of-death or -hospitalization classification: Mortality and morbidity statistics were compiled in accordance with the World Health Organization (WHO) regulations, which specify that mortality and morbidity data be classified by the current manual of the International Statistical Classification of Diseases, Injuries, and Causes of Death. The *Ninth Revision of the International Classification of Diseases (ICD-9)* identifies the classification used to code and classify mortality data from death certificates. The *International Classification of Diseases, 9th Revision, Clinical Modification (ICD-9-CM)* is used to code and classify morbidity data from inpatient records. Tabulations of cause-of-death and hospitalization statistics in this report are based solely on the underlying cause of death or the principal diagnosis, respectively, unless otherwise stated. The “underlying” cause is the disease or injury that initiated the series of events leading directly to death or hospitalization, or the circumstances of the event that resulted in the injury.

| Disease | ICD-9 Mortality | ICD-9-CM Morbidity |
|---------------------------------------|---------------------------|--|
| Birth-related | | V30-V39, DRGs 370-384 |
| Cardiovascular | 390-459 | 390-459 |
| Coronary heart disease | 402, 410-414, 429.2 | 402, 410-414 |
| Cerebrovascular | 430-438 | 430-438 |
| Lung cancer | 162.2-.9 | 162, 197.0, 197.3 |
| Female breast cancer | 174 | 174, 198.81 |
| Colorectal cancer | 153.0-154.3, 154.8, 159.0 | 153-154, 197.5 |
| Cervical cancer | 180 | 180 |
| Ovarian cancer | 183 | 183, 198.6 |
| Endometrial cancer | 182 | 182 |
| Asthma | 493 | 493 |
| Chronic obstructive pulmonary disease | 490-496 | 490-496 |
| Diabetes mellitus | 250 | 250 |
| Osteoporosis | | 733.0, 733.1 |
| Osteoarthritis | | 715 |
| Auto-immune diseases | | |
| Fibromyalgia | | 729.1 |
| Graves disease | | 648.8 |
| Hashimoto's thyroiditis | | 245.2 |
| Multiple sclerosis | | 340 |
| Rheumatoid arthritis | | 714.0 |
| Scleroderma | | 710.1 |
| Sjogren's syndrome | | 710.2 |
| Systemic lupus | | 710.0 |
| Auto-immune not otherwise specified | | 279.4 |
| Sexually transmitted diseases | | 090-099 |
| Pelvic inflammatory disease | | 614.0-614.5, 614.7-614.9, 615.0, 615.1, 615.9, 098.10, 098.16, 098.17, 098.30, 098.36-098.37, 098.39, 098.86 |
| HIV infection | 042-044 | 042, V08 (1995 and forward) 042-044, 279.19, 795.8 (Prior to 1995) |
| Pneumonia & influenza | 480-487 | 480-487 |
| Suicide & self-inflicted injury | E950-E959 | E950-E959 |
| Sexual assault | | E960.1, V71.5 |
| Abuse | | |
| Child | | 995.5 |
| Adult | | 995.8 |
| By intimate partner | | E967.3 |
| Falls & fall-related injuries | E880-E888 | E880-E888 |

Cause-of-death rankings are based on the National Center for Health Statistics List of 72 Selected Causes of Death, HIV infection, and Alzheimer's disease (National Center for Health Statistics, 1999). Ranks are based on the total number of deaths occurring during a specific time period. This report ranks number of deaths by age group, and race and ethnicity for the period 1996-98.

Hispanic origin refers to people whose origins are from Spain, the Spanish-speaking countries of Central America, South America, and the Caribbean, or persons of Hispanic origin identifying themselves as Spanish, Spanish-American, Hispanic, Hispano, or Latino. The death certificate has a separate line item for Hispanic ethnicity. Individuals identifying themselves as “Hispanic” can be of any race, and are also counted in the race breakdown as either “white,” “black,” “Asian or Pacific Islander,” “American Indian,” or other.

Hospitalization or discharge refers to any patient discharged from a non-federal, short-stay, acute care general hospital in Connecticut as recorded in the state’s hospital discharge abstract and billing data base. A person may have multiple hospitalizations and thus may be counted more than once.

Population bases for computing rates are taken from the U.S. Census Bureau *Estimates of the population of states by age, sex, race, and Hispanic origin*. These data are estimates of the population of Connecticut by 5-year age groups (age 0 to 4, 5 to 9, ... 85 and over), sex (male, female), modified race (white; black; Native American including Alaska Natives; Asian and Pacific Islander) and Hispanic origin (Hispanic, non-Hispanic) for each year, July 1, 1989 through July 1, 1998. Population estimates for 1989 are taken from the series *1981 to 1989*; estimates for 1990 through 1997 are taken from the series *1990 to 1997* released on September 4, 1998; and estimates for 1998 are taken from the series *1990 to 1998* released on September 15, 1999.

Population rates in this report are on an annual basis and are per 100,000 estimated population in a specified group.

Premature mortality refers to deaths that occur before 65 or 75 years of age. (See *Years of Potential Life Lost*).

Race refers to a population of individuals who identify themselves from a common history, nationality, or geographical place. When responses in the ‘race’ line item on vital records are associated with the definition of Hispanic origin, they are re-coded to ‘white race,’ as described in the National Center for Health Statistics instruction manuals for coding vital records. Individuals identifying themselves as either ‘white,’ ‘black,’ ‘Asian,’ ‘American Indian’ or ‘other’ race can be of any ethnic group.) In mortality data in this report, ‘Hispanic’ may include women who are also included under either ‘black’ and ‘white.’ Hospitalization and incidence data classify white, black, and Hispanic women into mutually exclusive categories, with Hispanic ethnicity having priority.

Random variation: the mortality data in this report represent all Connecticut female resident deaths and are, therefore, not subject to sampling error. Mortality data, however, may be affected by random variation. When the number of events is often considered to be small (less than 100) and the probability of such an event is small, random variation may be relatively large, and thus considerable caution must be used in interpreting the data.

Relative survival rate: Relative survival rates for cancer are calculated using a method that adjusts observed survival for expected mortality. The 5-year relative survival rate represents the likelihood that a patient will not die from causes related specifically to cancer for 5 years after diagnosis. It always exceeds the observed survival rate for the same group of patients.

SEER is the Surveillance, Epidemiology, and End Results Program based within the Cancer Surveillance Research Program at the National Cancer Institute. Comparisons to “national” cancer incidence and survival rates in the Women’s Health Report refer to the 9 SEER population-based registries, comprising 9% of the US population, which include Connecticut, and which have provided high quality and comparable data since 1973. SEER cancer incidence rates and national cancer mortality rates are age-adjusted to the 1970 US population, as are all Connecticut rates when reported by SEER or the Connecticut Tumor Registry.

Statistical significance levels are routinely used in research to screen findings and minimize the attention we might otherwise focus on spurious results that are due to chance alone. The statistical significance levels reported in this document all use a cutoff point of $p < .05$ to identify pairwise comparisons of rates that are deemed “significant.” The “ $p < .05$ ” criterion will reduce the number of chance differences that we label as “significant” to no more than 5 out of every 100 comparisons. In a study such as this one many statistical tests are performed. The overall chance of reporting spurious findings increases in proportion to the number of tests made. This problem is often referred to as the problem of “multiple inferences” or “multiple comparisons.” Even if we treat our analysis of each disease or condition as a separate investigation, multiple statistical comparisons are found within each report section.

A standard response to this problem is to make the test criterion for a single test more stringent, so that the overall p-value for all tests still remains at the $p < .05$ level. There are both advantages and disadvantages to this remedy and the discussion of these issues is complicated (Thomas, 1985) and well beyond the scope of the report. We have chosen to report all comparisons as "significant" if they reach the $p < .05$ threshold. In adopting this threshold we are attempting to preserve a level of simplicity in our presentation of results that is appropriate for a general non-technical audience. For those readers interested in more detailed significance test results, they will be provided upon request.

In the process of deciding how to present significance test results in this report, we made the following observations with respect to the mortality data. (These points should also apply to other data sources.)

- In many cases the significant differences reported already satisfy a more stringent test criterion than $p < .05$.
- Some comparisons between race/ethnicity subgroups in 1996-1998 were significant at $p < .05$ but not lower (i.e. not $p < .01$). However these differences followed the same pattern in earlier years (e.g. 1989-1991), so if data from more than three years had been combined these significance levels would satisfy a more stringent test criterion than $p < .05$.
- Some comparisons between 1989-1991 and 1996-1998 were significant at $p < .05$ but not lower (i.e. not $p < .01$). In each case, the use of a more sophisticated method to estimate the annual percent change in the risk of dying (based on logistic regression models) resulted in lower significance levels.

Years of potential life lost (YPLL) represents the number of years of potential life lost by each death before a predetermined end point (e.g., 75 years of age). Whereas adjusted death rates are heavily influenced by the large number of deaths among the elderly, the YPLL measure provides a picture of premature mortality by weighting deaths that occur at younger ages more heavily than those occurring at older ages. It thereby emphasizes different causes of death. Age-adjusted YPLLs are calculated using the methodology of Romeder and McWhinnie (1977).

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APPENDIX B. SOCIODEMOGRAPHIC PROFILE

SUMMARY OF SOCIODEMOGRAPHIC DATA
Connecticut Females, 1980-2000

| Connecticut Females, (unless otherwise noted) | 1980 ^a | 1990 ^a | 1998 ^e | 1999 ^e | 2000 ^a |
|--|-------------------|-------------------|-------------------|-------------------|-------------------|
| Population | | | | | |
| Male & Female | 3,107,576 | 3,287,116 | 3,274,069 | 3,282,031 | 3,405,565 |
| Female | 1,609,571 | 1,694,276 | 1,685,462 | 1,689,230 | 1,756,246 |
| Age | | | | | |
| 0-14 | 316,138 | 308,856 | 324,910 | 340,475 | 346,237 |
| 15-24 | 279,193 | 223,678 | 187,421 | 188,820 | 197,804 |
| 25-44 | 440,921 | 553,915 | 527,778 | 511,194 | 524,747 |
| 45-64 | 352,306 | 339,341 | 366,830 | 370,217 | 407,277 |
| 65-74 | 124,709 | 144,640 | 130,715 | 128,626 | 127,563 |
| 75+ | 96,275 | 124,823 | 147,808 | 149,898 | 152,618 |
| Race | | | | | |
| White/Caucasian | 1,454,045 | 1,474,842 | 1,480,247 | 1,479,617 | |
| Black/African American | 114,489 | 143,664 | 159,551 | 161,977 | |
| Asian & Pacific Islander | 10,801 | 24,325 | 41,685 | 43,599 | |
| Native American | 2,441 | 3,718 | 3,979 | 4,037 | |
| Hispanic Origin | | | | | |
| Hispanic | 64,698 | 103,781 | 137,210 | 142,732 | |
| Non-Hispanic | 1,544,844 | 1,591,472 | 1,548,272 | 1,546,498 | |
| Education (25 yrs+) | | | | | |
| High School graduates | 708,933 | 916,222 | -- | -- | |
| College graduates (4-yr) | 161,877 | 275,564 | -- | -- | |
| Marital Status | | | | | |
| Married | 684,052 | 715,165 | | | |
| Marriage Rate (per 1,000) | 16.8 | 15.8 | 13.9 ^d | | |
| Divorce Rate (per 1,000) | 8.7 | 7.1 | 6.6 ^d | | |
| Family Structure | | | | | |
| Female head, no husband present (FHNHP) | 115,018 | 136,381 | -- | -- | 157,411 |
| White | 87,730 | 95,786 | -- | -- | |
| Black | 21,482 | 28,886 | -- | -- | |
| Hispanic | 9,722 | 18,312 | -- | -- | |
| FHNHP w/ children <18yrs | 66,331 | 71,720 | -- | -- | 91,114 |
| White | 44,408 | 43,216 | -- | -- | |
| Black | 16,935 | 19,167 | -- | -- | |
| Hispanic | 8,385 | 14,783 | -- | -- | |
| FHNHP w/ children <6 yrs | 22,111 | 28,168 | -- | -- | |
| White | 11,879 | 14,418 | -- | -- | |
| Black | 7,627 | 9,171 | -- | -- | |
| Hispanic | 4,598 | 7,403 | -- | -- | |

| Connecticut Females | 1980 ^a | 1990 ^a | 1998 | 1999 | 2000 |
|----------------------------------|-------------------|-------------------|----------------------|----------------------|------|
| Labor Force (15 yrs +) | | | | | |
| In civilian labor force | 677,689 | 832,431 | 836,000 ^c | 810,000 ^f | |
| Employed | 645,792 | 793,450 | 800,000 ^c | 787,000 ^f | |
| Median Income^b | | | | | |
| Females 15+ w/ income | \$5,892 | \$14,028 | -- | -- | |
| White | \$5,879 | \$14,308 | -- | -- | |
| Black | \$5,997 | \$13,698 | -- | -- | |
| Asian & PI | \$6,204 | \$11,072 | -- | -- | |
| Native American | \$4,764 | \$13,068 | -- | -- | |
| Hispanic | -- | \$9,649 | -- | -- | |
| Female-headed family | \$11,624 | \$25,739 | -- | -- | |
| Working Mothers | | | | | |
| Females 16+ | 1,265,121 | 1,367,775 | -- | -- | |
| w/ children <6 yrs | 160,432 | 193,621 | -- | -- | |
| in labor force | 65,531 | 114,818 | -- | -- | |
| w/ children 6-17 yrs | 248,177 | 201,090 | -- | -- | |
| in labor force | 165,479 | 158,338 | -- | -- | |

-- Data not available

Data not available at time of publication.

Sources:

^a U.S. Census Bureau, 1970, 1980, 1990, and 2000 Census of Population.

^b Census data reflect prior year's income and poverty levels. For example, 1990 data reflects 1989 income.

^c U.S. Census Bureau, Statistical Abstract of the United States: 1999.

^d Connecticut Department of Public Health, Office of Policy, Planning, and Evaluation, vital statistics.

^e U.S. Census Bureau, Population Estimates by Age, Race, and Ethnicity, 1998 and 1999.

^f U.S. Census Bureau, Statistical Abstract of the United States: 2000.

POPULATION ESTIMATES
Connecticut Females, 1990-1999

| Age (yrs) | 1990 | 1991 | 1992 | 1993 | 1994 | 1995 | 1996 | 1997 | 1998 | 1999 |
|-----------|-----------|-----------|-----------|-----------|-----------|-----------|-----------|-----------|-----------|-----------|
| 0-14 | 309,734 | 315,801 | 319,391 | 324,089 | 326,309 | 327,177 | 326,326 | 325,052 | 332,229 | 340,475 |
| 15-24 | 226,515 | 216,722 | 207,880 | 201,435 | 195,926 | 191,039 | 187,223 | 186,512 | 187,299 | 188,820 |
| 25-44 | 553,683 | 555,665 | 546,811 | 541,872 | 538,759 | 536,596 | 536,200 | 532,651 | 522,219 | 511,194 |
| 45-64 | 336,030 | 334,814 | 339,799 | 342,442 | 345,271 | 348,169 | 352,961 | 359,320 | 364,011 | 370,217 |
| 65-74 | 144,202 | 143,701 | 142,761 | 142,021 | 140,576 | 138,881 | 136,690 | 133,614 | 131,250 | 128,626 |
| 75-84 | 90,483 | 92,491 | 94,844 | 96,738 | 98,363 | 99,696 | 101,273 | 102,756 | 103,655 | 104,611 |
| 85+ | 34,536 | 35,856 | 36,979 | 38,177 | 39,236 | 40,640 | 41,983 | 42,932 | 44,177 | 45,287 |
| Total | 1,695,000 | 1,695,000 | 1,689,000 | 1,687,000 | 1,684,000 | 1,682,000 | 1,683,000 | 1,683,000 | 1,685,000 | 1,689,000 |

Source: U.S. Census Bureau. "Estimates of the Female Population of Connecticut" PE-65, 1990-1999.

POPULATION PROJECTIONS BY RACE & ETHNICITY
Connecticut Females, 2000-2025
(Rounded to the nearest thousand)

| | 2000 | 2005 | 2015 | 2025 |
|------------------|-------|-------|-------|-------|
| Total Population | 3,284 | 3,317 | 3,506 | 3,739 |
| Females | 1,688 | 1,702 | 1,793 | 1,906 |
| White | 1,474 | 1,464 | 1,498 | 1,553 |
| Black | 170 | 183 | 220 | 257 |
| American Indian | 4 | 4 | 5 | 6 |
| Asian & PI | 41 | 50 | 70 | 89 |
| Hispanic | 146 | 169 | 227 | 291 |

Source: U.S. Census Bureau. Projected State Populations by Sex, Race, and Hispanic Origin: 1995-2025. PPL #47, 2000.

POPULATION PROJECTIONS BY AGE GROUPS
Connecticut Females, 2000-2025
(Rounded to the nearest thousand)

| | 2000 | 2005 | 2015 | 2025 |
|------------------|-------|-------|-------|-------|
| Total Population | 3,284 | 3,317 | 3,506 | 3,739 |
| Females | 1,688 | 1,702 | 1,793 | 1,906 |
| 0-4 | 104 | 100 | 111 | 120 |
| 5-17 | 281 | 278 | 272 | 298 |
| 18-24 | 136 | 148 | 161 | 156 |
| 25-64 | 895 | 910 | 954 | 966 |
| 65+ | 272 | 266 | 295 | 366 |

Source: U.S. Census Bureau. Projected State Populations, by Sex, Race, and Hispanic Origin: 1995-2025. PPL #47, 2000.

APPENDIX C. MORTALITY DATA SUMMARY

LEADING CAUSES OF DEATH Connecticut Females, 1996-1998

| Cause of death | Deaths | Rank |
|--|--------|------|
| Diseases of the heart (390-398,402,404-429) | 15,669 | 1 |
| All cancer (140-208) | 10,698 | 2 |
| Cerebrovascular disease (430-438) | 3,654 | 3 |
| Chronic obstructive pulmonary disease (COPD) (490-496) | 2,005 | 4 |
| Pneumonia & influenza (480-487) | 1,988 | 5 |
| Unintentional injuries (E800-E949) | 1,142 | 6 |
| Diabetes mellitus (250) | 1,058 | 7 |
| Septicemia (038) | 638 | 8 |
| Nephritis, nephrotic syndrome/nephrosis (580-589) | 551 | 9 |
| Alzheimer's disease (331.0) | 541 | 10 |
| Atherosclerosis (440) | 351 | 11 |
| Chronic liver disease and cirrhosis (571) | 334 | 12 |
| HIV infection (042-044) | 204 | 13 |
| Suicide (E950-E959) | 188 | 14 |
| Homicide and legal intervention (E960-E978) | 106 | 15 |

Source: Mueller, L.M., M.M. Hynes, H. Li, and F. Amadeo. In preparation. *Mortality and Its Risk Factors in Connecticut, 1989-1998*. Hartford, CT: Connecticut Department of Public Health, Division of Policy, Planning, and Analysis.

LEADING CAUSES OF DEATH BY AGE GROUPS Connecticut Females, 1996-1998

| Cause of death | Age groups | | | | | | |
|---|------------|-------|-------|-------|-------|-------|-------|
| | 0-14 | 15-24 | 25-44 | 45-64 | 65-74 | 75-84 | 85+ |
| Unintentional injuries (E800-E949) | 46 | 87 | 203 | 160 | 126 | 250 | 270 |
| All cancer (140-208) | 22 | 15 | 401 | 2,341 | 2,681 | 3,368 | 1,870 |
| Diseases of the heart (390-398,402,404-429) | 19 | 14 | 160 | 1,006 | 1,935 | 4,708 | 7,827 |
| Homicide and legal intervention (E960-E978) | 16 | 15 | 45 | 23 | 2 | 4 | 1 |
| HIV infection (042-044) | 8 | 4 | 143 | 48 | 1 | 0 | 0 |
| Pneumonia & influenza (480-487) | 5 | 1 | 20 | 80 | 144 | 536 | 1,202 |
| Cerebrovascular disease (430-438) | 5 | 1 | 46 | 174 | 392 | 1,219 | 1,817 |
| Septicemia (038) | 4 | 1 | 16 | 50 | 80 | 220 | 267 |
| Chronic obstructive pulmonary disease (490-496) | 2 | 3 | 21 | 182 | 462 | 831 | 504 |
| Nephritis, nephrotic syndrome/nephrosis (580-589) | 2 | 1 | 13 | 49 | 99 | 179 | 208 |
| Suicide (E950-E959) | 2 | 20 | 81 | 49 | 18 | 13 | 5 |
| Atherosclerosis (440) | 0 | 0 | 1 | 9 | 26 | 85 | 230 |
| Alzheimer's disease (331.0) | 0 | 0 | 0 | 7 | 36 | 182 | 316 |
| Chronic liver disease and cirrhosis (571) | 0 | 0 | 32 | 99 | 89 | 98 | 16 |
| Diabetes mellitus (250) | 0 | 0 | 22 | 176 | 224 | 333 | 303 |

Source: Mueller, L.M., M.M. Hynes, H. Li, and F. Amadeo. In preparation. *Mortality and Its Risk Factors in Connecticut, 1989-1998*. Hartford, CT: Connecticut Department of Public Health, Division of Policy, Planning, and Analysis.

LEADING CAUSES OF DEATH
BY RACE AND ETHNICITY
Connecticut Females, 1996-1998

| Causes of death | White | Black/ African American | Asian/ PI* | Native American | Hispanic |
|---|--------|-------------------------------|---------------|--------------------|----------|
| Diseases of the heart (390-398,402,404-429) | 14,764 | 864 | 33 | 4 | 248 |
| All cancer (140-208) | 10,027 | 614 | 44 | 10 | 181 |
| Cerebrovascular disease (430-438) | 3,451 | 182 | 17 | 3 | 59 |
| Chronic obstructive pulmonary disease (COPD) (490-496) | 1,955 | 50 | | | 31 |
| Pneumonia & influenza (480-487) | 1,892 | 91 | 4 | 1 | 32 |
| Unintentional injuries (E800-E949) | 1,047 | 80 | 8 | 5 | 48 |
| Diabetes mellitus (250) | 941 | 115 | 1 | 1 | 33 |
| Septicemia (038) | 584 | 52 | | | 18 |
| Alzheimer's disease (331.0) | 528 | 12 | 1 | | 6 |
| Nephritis, nephrotic syndrome/nephrosis (580-589) | 491 | 57 | 3 | | 20 |
| Atherosclerosis (440) | 339 | 12 | | | 2 |
| Chronic liver disease and cirrhosis (571) | 311 | 22 | 1 | | 22 |
| Suicide (E950-E959) | 173 | 12 | 2 | 1 | 5 |
| HIV infection (042-044) | 102 | 101 | | | 51 |
| Homicide and legal intervention (E960-E978) | 61 | 42 | 1 | 2 | 9 |

* Asian and Pacific Islander.

Source: Mueller, L.M., M.M. Hynes, H. Li, and F. Amadeo. In preparation. *Mortality and Its Risk Factors in Connecticut, 1989-1998*. Hartford, CT: Connecticut Department of Public Health, Division of Policy, Planning, and Analysis.

AGE-ADJUSTED MORTALITY RATES (AAMR) FOR SELECTED CAUSES
Connecticut Females, 1989 – 1998

CARDIOVASCULAR DISEASE

| Year | CT AAMR (2000) | CT AAMR (1940) | US AAMR (1940) |
|------|-------------------|-------------------|-------------------|
| 1989 | 316.1 | 127.0 | 147.5 |
| 1990 | 302.1 | 120.9 | 143.0 |
| 1991 | 296.3 | 117.4 | 139.4 |
| 1992 | 298.5 | 118.4 | 136.3 |
| 1993 | 297.4 | 116.4 | 137.9 |
| 1994 | 293.3 | 116.6 | 134.5 |
| 1995 | 289.6 | 115.6 | 133.8 |
| 1996 | 296.6 | 118.6 | 131.3 |
| 1997 | 280.9 | 109.8 | 128.1 |
| 1998 | 279.6 | 109.0 | 125.3 |

LUNG CANCER*

| Year | CT AAMR (2000) | CT AAMR (1940) | US AAMR (1940) |
|------|-------------------|-------------------|-------------------|
| 1989 | 35.8 | 24.8 | 25.0 |
| 1990 | 35.4 | 24.3 | 25.6 |
| 1991 | 37.8 | 25.5 | 25.8 |
| 1992 | 39.1 | 26.6 | 26.3 |
| 1993 | 37.8 | 25.2 | 26.5 |
| 1994 | 39.2 | 26.1 | 26.6 |
| 1995 | 39.1 | 25.6 | 26.9 |
| 1996 | 41.2 | 26.6 | 26.8 |
| 1997 | 42.4 | 27.3 | 27.0 |
| 1998 | 40.0 | 25.7 | 27.0 |

CORONARY HEART DISEASE

| Year | CT AAMR (2000) | CT AAMR (1940) | US AAMR (1940) |
|------|-------------------|-------------------|-------------------|
| 1989 | 180.5 | 71.2 | 87.0 |
| 1990 | 169.3 | 66.5 | 84.1 |
| 1991 | 166.6 | 64.9 | 81.6 |
| 1992 | 163.2 | 63.7 | 79.1 |
| 1993 | 163.5 | 62.2 | 79.2 |
| 1994 | 157.7 | 61.1 | 76.6 |
| 1995 | 153.8 | 60.9 | 75.2 |
| 1996 | 155.7 | 61.1 | 73.3 |
| 1997 | 148.9 | 58.0 | 70.0 |
| 1998 | 145.6 | 56.4 | 68.0 |

FEMALE BREAST CANCER*

| Year | CT AAMR (2000) | CT AAMR (1940) | US AAMR (1940) |
|------|-------------------|-------------------|-------------------|
| 1989 | 35.5 | 24.0 | 23.1 |
| 1990 | 31.8 | 21.4 | 23.0 |
| 1991 | 32.6 | 22.5 | 22.8 |
| 1992 | 30.9 | 21.1 | 21.8 |
| 1993 | 31.2 | 20.8 | 21.4 |
| 1994 | 30.9 | 21.1 | 21.1 |
| 1995 | 31.9 | 21.4 | 21.1 |
| 1996 | 30.9 | 20.9 | 20.2 |
| 1997 | 29.6 | 19.6 | 19.4 |
| 1998 | 29.7 | 19.7 | 18.8 |

CEREBROVASCULAR DISEASE

| Year | CT AAMR (2000) | CT AAMR (1940) | US AAMR (1940) |
|------|-------------------|-------------------|-------------------|
| 1989 | 53.5 | 20.9 | 26.1 |
| 1990 | 52.2 | 19.9 | 25.5 |
| 1991 | 48.1 | 18.1 | 24.6 |
| 1992 | 49.0 | 18.2 | 24.1 |
| 1993 | 51.1 | 19.3 | 24.4 |
| 1994 | 49.2 | 18.9 | 24.5 |
| 1995 | 48.7 | 18.1 | 24.7 |
| 1996 | 51.3 | 19.6 | 24.6 |
| 1997 | 49.6 | 18.4 | 24.1 |
| 1998 | 49.9 | 18.8 | 23.6 |

COLORECTAL CANCER*

| Year | CT AAMR (2000) | CT AAMR (1940) | US AAMR (1940) |
|------|-------------------|-------------------|-------------------|
| 1989 | 23.3 | 12.0 | 11.7 |
| 1990 | 21.5 | 11.2 | 11.4 |
| 1991 | 20.8 | 11.3 | 11.3 |
| 1992 | 19.0 | 9.9 | 10.9 |
| 1993 | 20.2 | 10.9 | 11.1 |
| 1994 | 19.0 | 9.5 | 10.7 |
| 1995 | 18.4 | 9.8 | 10.6 |
| 1996 | 16.9 | 8.8 | 10.3 |
| 1997 | 17.9 | 9.8 | 10.1 |
| 1998 | 18.5 | 9.7 | 10.0 |

CERVICAL CANCER

| Year | CT AAMR (2000) | CT AAMR (1940) | US AAMR (1940) |
|------|-------------------|-------------------|-------------------|
| 1989 | 2.8 | 2.0 | 2.6 |
| 1990 | 2.4 | 1.7 | 2.8 |
| 1991 | 3.2 | 2.4 | 2.6 |
| 1992 | 2.0 | 1.5 | 2.7 |
| 1993 | 1.8 | 1.3 | 2.6 |
| 1994 | 2.5 | 2.1 | 2.6 |
| 1995 | 2.0 | 1.5 | 2.5 |
| 1996 | 3.0 | 2.2 | 2.5 |
| 1997 | 1.8 | 1.5 | 2.5 |
| 1998 | 1.9 | 1.4 | 2.3 |

CHRONIC OBSTRUCTIVE PULMONARY DISEASE*

| Year | CT AAMR (2000) | CT AAMR (1940) | US AAMR (1940) |
|------|-------------------|-------------------|-------------------|
| 1989 | 22.9 | 12.2 | 14.8 |
| 1990 | 23.3 | 12.2 | 14.6 |
| 1991 | 22.6 | 11.1 | 15.4 |
| 1992 | 25.8 | 12.4 | 15.4 |
| 1993 | 26.5 | 12.9 | 17.2 |
| 1994 | 28.5 | 14.3 | 17.0 |
| 1995 | 27.8 | 13.7 | 17.1 |
| 1996 | 28.6 | 14.2 | 17.6 |
| 1997 | 31.5 | 15.5 | 17.8 |
| 1998 | 29.5 | 14.5 | 18.1 |

OVARIAN CANCER

| Year | CT AAMR (2000) | CT AAMR (1940) | US AAMR (1940) |
|------|-------------------|-------------------|-------------------|
| 1989 | 9.3 | 6.5 | 6.3 |
| 1990 | 8.7 | 6.0 | 6.3 |
| 1991 | 9.4 | 6.3 | 6.5 |
| 1992 | 9.1 | 5.8 | 6.3 |
| 1993 | 10.1 | 6.4 | 6.1 |
| 1994 | 9.0 | 5.7 | 6.3 |
| 1995 | 9.3 | 5.9 | 6.1 |
| 1996 | 7.2 | 4.5 | 6.0 |
| 1997 | 8.8 | 5.7 | 5.9 |
| 1998 | 9.7 | 6.3 | 5.8 |

ASTHMA

| Year | CT AAMR (2000) | CT AAMR (1940) | US AAMR (1940) |
|------|-------------------|-------------------|-------------------|
| 1989 | 1.8 | 1.2 | 1.6 |
| 1990 | 1.4 | 1.0 | 1.4 |
| 1991 | 1.1 | 0.6 | 1.4 |
| 1992 | 1.7 | 1.1 | 1.6 |
| 1993 | 1.7 | 1.2 | 1.8 |
| 1994 | 1.5 | 1.1 | 1.6 |
| 1995 | 2.0 | 1.6 | 1.7 |
| 1996 | 1.7 | 1.3 | 1.8 |
| 1997 | 2.3 | 1.4 | 1.7 |
| 1998 | 1.8 | 1.2 | 1.5 |

ENDOMETRIAL CANCER

| Year | CT AAMR (2000) | CT AAMR (1940) | US AAMR (1940) |
|------|-------------------|-------------------|-------------------|
| 1989 | 2.2 | 1.3 | 1.2 |
| 1990 | 2.1 | 1.3 | 1.2 |
| 1991 | 2.2 | 1.4 | 1.2 |
| 1992 | 1.8 | 1.1 | 1.3 |
| 1993 | 2.0 | 1.2 | 1.3 |
| 1994 | 2.2 | 1.3 | 1.2 |
| 1995 | 2.0 | 1.3 | 1.1 |
| 1996 | 2.4 | 1.4 | 1.1 |
| 1997 | 1.8 | 1.0 | 1.2 |
| 1998 | 2.1 | 1.2 | 1.2 |

DIABETES MELLITUS*

| Year | CT AAMR (2000) | CT AAMR (1940) | US AAMR (1940) |
|------|-------------------|-------------------|-------------------|
| 1989 | 12.7 | 6.8 | 10.9 |
| 1990 | 14.7 | 7.5 | 11.0 |
| 1991 | 13.1 | 6.6 | 11.0 |
| 1992 | 13.9 | 7.4 | 11.2 |
| 1993 | 15.4 | 8.5 | 11.6 |
| 1994 | 16.5 | 8.8 | 12.1 |
| 1995 | 16.1 | 8.9 | 12.5 |
| 1996 | 18.1 | 10.1 | 12.6 |
| 1997 | 14.4 | 7.7 | 12.3 |
| 1998 | 16.0 | 8.5 | 12.3 |

HIV INFECTION

| Year | CT AAMR (2000) | CT AAMR (1940) | US AAMR (1940) |
|------|-------------------|-------------------|-------------------|
| 1989 | 3.3 | 3.3 | 1.8 |
| 1990 | 2.9 | 3.0 | 2.0 |
| 1991 | 4.5 | 4.5 | 2.6 |
| 1992 | 5.0 | 4.9 | 3.1 |
| 1993 | 6.8 | 6.8 | 3.8 |
| 1994 | 7.2 | 7.1 | 4.6 |
| 1995 | 9.1 | 8.8 | 5.3 |
| 1996 | 5.8 | 5.5 | 4.3 |
| 1997 | 3.2 | 3.2 | 2.3 |
| 1998 | 2.9 | 2.8 | 2.2 |

HOMICIDE & LEGAL INTERVENTION

| Year | CT AAMR (2000) | CT AAMR (1940) | US AAMR (1940) |
|------|-------------------|-------------------|-------------------|
| 1989 | 2.8 | 2.9 | 4.2 |
| 1990 | 2.2 | 2.2 | 4.3 |
| 1991 | 2.1 | 2.3 | 4.5 |
| 1992 | 2.2 | 2.2 | 4.4 |
| 1993 | 3.0 | 3.3 | 4.5 |
| 1994 | 2.9 | 3.2 | 4.0 |
| 1995 | 1.8 | 2.2 | 4.1 |
| 1996 | 2.4 | 2.4 | 3.6 |
| 1997 | 1.8 | 1.8 | 3.3 |
| 1998 | 2.3 | 2.6 | 3.2 |

PNEUMONIA & INFLUENZA*

| Year | CT AAMR (2000) | CT AAMR (1940) | US AAMR (1940) |
|------|-------------------|-------------------|-------------------|
| 1989 | 28.6 | 9.8 | 10.9 |
| 1990 | 32.1 | 10.7 | 11.1 |
| 1991 | 28.1 | 8.9 | 10.5 |
| 1992 | 28.1 | 9.3 | 10.0 |
| 1993 | 29.1 | 10.0 | 10.8 |
| 1994 | 27.7 | 9.4 | 10.6 |
| 1995 | 26.2 | 9.3 | 10.6 |
| 1996 | 26.4 | 9.1 | 10.5 |
| 1997 | 26.4 | 9.0 | 10.6 |
| 1998 | 27.0 | 9.0 | 11.0 |

FALLS & FALL-RELATED INJURY

| Year | CT AAMR (2000) | CT AAMR (1940) | US AAMR (1940) |
|------|-------------------|-------------------|-------------------|
| 1989 | 4.0 | 1.5 | 1.6 |
| 1990 | 5.2 | 1.8 | 1.6 |
| 1991 | 4.1 | 1.4 | 1.6 |
| 1992 | 4.3 | 1.5 | 1.6 |
| 1993 | 4.1 | 1.5 | 1.6 |
| 1994 | 4.4 | 1.7 | 1.6 |
| 1995 | 4.7 | 1.8 | 1.6 |
| 1996 | 4.5 | 1.7 | 1.7 |
| 1997 | 3.0 | 1.1 | 1.7 |
| 1998 | 4.5 | 1.7 | 1.9 |

SUICIDE

| Year | CT AAMR (2000) | CT AAMR (1940) | US AAMR (1940) |
|------|-------------------|-------------------|-------------------|
| 1989 | 3.4 | 3.1 | 4.6 |
| 1990 | 3.4 | 3.2 | 4.4 |
| 1991 | 3.6 | 3.4 | 4.4 |
| 1992 | 2.9 | 2.7 | 4.3 |
| 1993 | 3.7 | 3.4 | 4.2 |
| 1994 | 4.1 | 3.8 | 4.1 |
| 1995 | 3.7 | 3.6 | 4.1 |
| 1996 | 3.3 | 3.2 | 4.0 |
| 1997 | 3.8 | 3.5 | 4.0 |
| 1998 | 3.7 | 3.4 | 4.0 |

Source: Mueller, L.M., M.M. Hynes, H. Li, and F. Amadeo. In preparation. *Mortality and Its Risk Factors in Connecticut, 1989-1998*. Hartford, CT: Connecticut Department of Public Health, Division of Policy, Planning, and Analysis.

Notes:

* The annual percent change from 1989 to 1998 is significant ($p < .05$).

Rates adjusted to the 2000 and 1940 U.S. standard million population. U.S. Census Bureau population estimates used for rate calculations are presented in Appendix B. See Appendix A for definition of terms used in mortality analyses.

AGE-ADJUSTED MORTALITY RATES (AAMR) FOR SELECTED CAUSES
BY RACE & ETHNICITY
Connecticut Females, 1989-1991 and 1996-1998

CARDIOVASCULAR DISEASE

| Race/Ethnicity | 1989-1991 deaths | 1989-1991 AAMR | 1996-1998 deaths | 1996-1998 AAMR |
|--------------------------|---------------------|--------------------|---------------------|--------------------|
| All Females | 19,155 | 304.7 | 20,586 | 285.7* |
| White | 18,124 | 299.9 | 19,401 | 280.5* |
| Black & African American | 902 | 351.3 [‡] | 1,118 | 368.4 [‡] |
| Asian & Pacific Islander | 21 | 111.8 [‡] | 54 | 120.1 [‡] |
| Native American | 19 | 270.3 | 8 | † |
| Hispanic | 233 | 185.8 [‡] | 319 | 176.4 [‡] |

CORONARY HEART DISEASE

| Race/Ethnicity | 1989-1991 deaths | 1989-1991 AAMR | 1996-1998 deaths | 1996-1998 AAMR |
|--------------------------|---------------------|-------------------|---------------------|--------------------|
| All Females | 10,857 | 172.1 | 10,826 | 150.0* |
| White | 10,318 | 170.1 | 10,224 | 147.5* |
| Black & African American | 454 | 179.7 | 575 | 189.9 [‡] |
| Asian & Pacific Islander | 10 | † | 22 | 46.8 [‡] |
| Native American | 13 | † | 2 | † |
| Hispanic | 118 | 97.6 [‡] | 157 | 88.2 [‡] |

CEREBROVASCULAR DISEASE

| Race/Ethnicity | 1989-1991 deaths | 1989-1991 AAMR | 1996-1998 deaths | 1996-1998 AAMR |
|--------------------------|---------------------|-------------------|---------------------|-------------------|
| All Females | 3,219 | 51.3 | 3,654 | 50.3 |
| White | 3,060 | 50.7 | 3,451 | 49.5 |
| Black & African American | 140 | 57.4 | 182 | 62.1 [‡] |
| Asian & Pacific Islander | 5 | † | 17 | 42.2 |
| Native American | 3 | † | 3 | † |
| Hispanic | 48 | 39.0 | 59 | 32.9 [‡] |

LUNG CANCER

| Race/Ethnicity | 1989-1991 deaths | 1989-1991 AAMR | 1996-1998 deaths | 1996-1998 AAMR |
|--------------------------|---------------------|-------------------|---------------------|-------------------|
| All Females | 2,109 | 36.4 | 2,503 | 41.2* |
| White | 2,004 | 36.6 | 2,378 | 41.8* |
| Black & African American | 88 | 29.9 | 119 | 35.8 |
| Asian & Pacific Islander | 2 | † | 5 | † |
| Native American | 1 | † | 1 | † |
| Hispanic | 19 | 13.4 [‡] | 25 | 13.6 [‡] |

FEMALE BREAST CANCER

| Race/Ethnicity | 1989-1991 deaths | 1989-1991 AAMR | 1996-1998 deaths | 1996-1998 AAMR |
|--------------------------|---------------------|-------------------|---------------------|-------------------|
| All Females | 1,885 | 33.3 | 1,796 | 30.0* |
| White | 1,777 | 33.3 | 1,653 | 29.7* |
| Black & African American | 92 | 30.1 | 135 | 39.5 [‡] |
| Asian & Pacific Islander | 1 | † | 5 | † |
| Native American | 1 | † | 1 | † |
| Hispanic | 16 | 9.3 [‡] | 33 | 13.2 [‡] |

COLORECTAL CANCER

| Race/Ethnicity | 1989-1991 deaths | 1989-1991 AAMR | 1996-1998 deaths | 1996-1998 AAMR |
|--------------------------|---------------------|-------------------|---------------------|-------------------|
| All Females | 1,325 | 21.8 | 1,169 | 17.8* |
| White | 1,265 | 21.9 | 1,097 | 17.5* |
| Black & African American | 52 | 20.0 | 65 | 19.9 |
| Asian & Pacific Islander | 1 | † | 7 | † |
| Native American | 0 | – | 0 | – |
| Hispanic | 16 | 13.1 [‡] | 15. | 7.2 [‡] |

CERVICAL CANCER

| Race/Ethnicity | 1989-1991 deaths | 1989-1991 AAMR | 1996-1998 deaths | 1996-1998 AAMR |
|--------------------------|---------------------|-------------------|---------------------|-------------------|
| All Females | 150 | 2.8 | 127 | 2.2 |
| White | 126 | 2.5 | 101 | 1.9 |
| Black & African American | 22 | 6.6 [‡] | 22 | 5.8 [‡] |
| Asian & Pacific Islander | 1 | † | 2 | † |
| Native American | 0 | – | 2 | † |
| Hispanic | 7 | † | 11 | † |

OVARIAN CANCER

| Race/Ethnicity | 1989-1991 deaths | 1989-1991 AAMR | 1996-1998 deaths | 1996-1998 AAMR |
|--------------------------|---------------------|-------------------|---------------------|-------------------|
| All Females | 526 | 9.2 | 522 | 8.6 |
| White | 506 | 9.4 | 505 | 8.9 |
| Black & African American | 17 | 5.9 | 15 | 4.6 [‡] |
| Asian & Pacific Islander | 1 | † | 1 | † |
| Native American | 0 | – | 1 | † |
| Hispanic | 9 | † | 10 | † |

ENDOMETRIAL CANCER

| Race/Ethnicity | 1989-1991 deaths | 1989-1991 AAMR | 1996-1998 deaths | 1996-1998 AAMR |
|--------------------------|---------------------|-------------------|---------------------|-------------------|
| All Females | 133 | 2.2 | 135 | 2.1 |
| White | 127 | 2.2 | 126 | 2.1 |
| Black & African American | 6 | † | 9 | † |
| Asian & Pacific Islander | 0 | – | 0 | – |
| Native American | 0 | – | 0 | – |
| Hispanic | 0 | – | 2 | † |

CHRONIC OBSTRUCTIVE PULMONARY DISEASE

| Race/Ethnicity | 1989-1991 deaths | 1989-1991 AAMR | 1996-1998 deaths | 1996-1998 AAMR |
|--------------------------|---------------------|-------------------|---------------------|-------------------|
| All Females | 1,412 | 23.0 | 2,005 | 29.9* |
| White | 1,369 | 23.3 | 1,955 | 30.7* |
| Black & African American | 43 | 15.3 [‡] | 50 | 15.1 [‡] |
| Asian & Pacific Islander | 0 | -- | 0 | -- |
| Native American | 0 | -- | 0 | -- |
| Hispanic | 19 | 14.7 [‡] | 31 | 16.2 [‡] |

ASTHMA

| Race/Ethnicity | 1989-1991 deaths | 1989-1991 AAMR | 1996-1998 deaths | 1996-1998 AAMR |
|--------------------------|---------------------|-------------------|---------------------|-------------------|
| All Females | 85 | 1.5 | 114 | 1.9 |
| White | 73 | 1.3 | 100 | 1.8 |
| Black & African American | 12 | † | 14 | † |
| Asian & Pacific Islander | 0 | -- | 0 | -- |
| Native American | 0 | -- | 0 | -- |
| Hispanic | 7 | † | 12 | † |

DIABETES MELLITUS

| Race/Ethnicity | 1989-1991 deaths | 1989-1991 AAMR | 1996-1998 deaths | 1996-1998 AAMR |
|--------------------------|---------------------|-------------------|---------------------|-------------------|
| All Females | 822 | 13.5 | 1,058 | 16.2* |
| White | 735 | 12.6 | 941 | 15.1* |
| Black & African American | 83 | 30.7 [‡] | 115 | 36.3 [‡] |
| Asian & Pacific Islander | 1 | † | 1 | † |
| Native American | 0 | -- | 1 | † |
| Hispanic | 25 | 20.6 | 33 | 17.8 |

HIV INFECTION

| Race/Ethnicity | 1989-1991 deaths | 1989-1991 AAMR | 1996-1998 deaths | 1996-1998 AAMR |
|--------------------------|---------------------|-------------------|---------------------|-------------------|
| All Females | 192 | 3.6 | 204 | 3.9 |
| White | 86 | 1.8 | 102 | 2.3 |
| Black & African American | 103 | 21.7 [‡] | 101 | 21.1 [‡] |
| Asian & Pacific Islander | 0 | -- | 0 | -- |
| Native American | 0 | -- | 0 | -- |
| Hispanic | 32 | 9.4 [‡] | 51 | 13.4 [‡] |

PNEUMONIA & INFLUENZA

| Race/Ethnicity | 1989-1991 deaths | 1989-1991 AAMR | 1996-1998 deaths | 1996-1998 AAMR |
|--------------------------|---------------------|-------------------|---------------------|-------------------|
| All Females | 1,884 | 29.6 | 1,988 | 26.6* |
| White | 1,808 | 29.5 | 1,892 | 26.4* |
| Black & African American | 59 | 22.7 | 91 | 30.9 |
| Asian & Pacific Islander | 4 | † | 4 | † |
| Native American | 1 | † | 1 | † |
| Hispanic | 20 | 19.1 [‡] | 32 | 18.4 [‡] |

SUICIDE

| Race/Ethnicity | 1989-1991 deaths | 1989-1991 AAMR | 1996-1998 deaths | 1996-1998 AAMR |
|--------------------------|---------------------|-------------------|---------------------|-------------------|
| All Females | 181 | 3.5 | 188 | 3.6 |
| White | 165 | 3.5 | 173 | 3.7 |
| Black & African American | 10 | † | 12 | † |
| Asian & Pacific Islander | 1 | † | 2 | † |
| Native American | 1 | † | 1 | † |
| Hispanic | 7 | † | 5 | † |

HOMICIDE AND LEGAL INTERVENTION

| Race/Ethnicity | 1989-1991 deaths | 1989-1991 AAMR | 1996-1998 deaths | 1996-1998 AAMR |
|--------------------------|---------------------|-------------------|---------------------|-------------------|
| All Females | 127 | 2.4 | 106 | 2.1 |
| White | 89 | 1.9 | 61 | 1.4 |
| Black & African American | 35 | 6.9‡ | 42 | 8.3‡ |
| Asian & Pacific Islander | 1 | † | 1 | † |
| Native American | 0 | – | 2 | † |
| Hispanic | 13 | † | 9 | † |

FALLS AND FALL RELATED INJURY

| Race/Ethnicity | 1989-1991 deaths | 1989-1991 AAMR | 1996-1998 deaths | 1996-1998 AAMR |
|--------------------------|---------------------|-------------------|---------------------|-------------------|
| All Females | 282 | 4.4 | 293 | 4.0 |
| White | 275 | 4.5 | 287 | 4.1 |
| Black & African American | 5 | † | 3 | † |
| Asian & Pacific Islander | 0 | – | 1 | † |
| Native American | 0 | – | 2 | † |
| Hispanic | 4 | † | 3 | † |

Source: Mueller, L.M., M.M. Hynes, H. Li, and F. Amadeo. In preparation. *Mortality and Its Risk Factors in Connecticut, 1989-1998*. Hartford, CT: Connecticut Department of Public Health, Division of Policy, Planning, and Analysis.

Notes:

Racial groupings (African American/Black, Asian & Pacific Islander, Native American, and White) include persons of Hispanic ethnicity. Less than 1 percent of the mortality data are missing race or ethnicity identifiers.

* Change in rates from 1989-1991 to 1996-1998 period is statistically significant ($p < .05$).

† Statistics not calculated for fewer than 15 events.

‡ The AAMR is significantly different from that of whites ($p < .05$)

Rates are per 100,000 persons based on race and ethnicity specific population estimates, and age-adjusted to the 2000 U.S. standard million population.

See Appendix A for definition of terms used in mortality analyses.

APPENDIX D. HOSPITALIZATION DATA SUMMARY

AGE-ADJUSTED HOSPITALIZATION RATES (AAHR) FOR SELECTED CAUSES Connecticut Females, 1993 – 1997

CARDIOVASCULAR DISEASE

| Year | Discharges | AAHR |
|------|------------|--------|
| 1993 | 27,937 | 1378.1 |
| 1994 | 28,599 | 1400.6 |
| 1995 | 28,984 | 1407.9 |
| 1996 | 28,868 | 1391.0 |
| 1997 | 29,317 | 1399.2 |
| 5-yr | 143,705 | 1395.9 |

COLORECTAL CANCER

| Year | Discharges | AAHR |
|------|------------|------|
| 1993 | 1,002 | 48.8 |
| 1994 | 1,017 | 49.3 |
| 1995 | 1,068 | 51.2 |
| 1996 | 955 | 45.8 |
| 1997 | 1,100 | 53.0 |
| 5-yr | 5,142 | 50.1 |

ASTHMA

| Year | Discharges | AAHR |
|------|------------|-------|
| 1993 | 2,830 | 165.5 |
| 1994 | 2,656 | 156.1 |
| 1995 | 2,710 | 159.7 |
| 1996 | 2,704 | 160.9 |
| 1997 | 2,503 | 148.7 |
| 5-yr | 13,403 | 158.6 |

CORONARY HEART DISEASE

| Year | Discharges | AAHR |
|------|------------|-------|
| 1993 | 9,826 | 493.8 |
| 1994 | 10,017 | 500.4 |
| 1995 | 10,087 | 501.3 |
| 1996 | 9,945 | 491.0 |
| 1997 | 9,813 | 481.3 |
| 5-yr | 49,688 | 493.9 |

CERVICAL CANCER

| Year | Discharges | AAHR |
|------|------------|------|
| 1993 | 168 | 9.4 |
| 1994 | 171 | 9.6 |
| 1995 | 184 | 9.8 |
| 1996 | 207 | 11.3 |
| 1997 | 188 | 10.2 |
| 5-yr | 918 | 10.4 |

DIABETES MELLITUS

| Year | Discharges | AAHR |
|------|------------|-------|
| 1993 | 1,827 | 100.5 |
| 1994 | 1,889 | 103.2 |
| 1995 | 1,935 | 105.7 |
| 1996 | 1,936 | 104.5 |
| 1997 | 1,875 | 101.8 |
| 5-yr | 9,462 | 103.7 |

CEREBROVASCULAR DISEASE

| Year | Discharges | AAHR |
|------|------------|-------|
| 1993 | 4,680 | 224.0 |
| 1994 | 4,799 | 228.3 |
| 1995 | 5,142 | 242.3 |
| 1996 | 5,169 | 242.0 |
| 1997 | 5,343 | 247.2 |
| 5-yr | 25,133 | 237.2 |

OVARIAN CANCER

| Year | Discharges | AAHR |
|------|------------|------|
| 1993 | 295 | 15.7 |
| 1994 | 311 | 16.5 |
| 1995 | 289 | 15.8 |
| 1996 | 324 | 17.3 |
| 1997 | 321 | 17.2 |
| 5-yr | 1,540 | 17.0 |

OSTEOPOROSIS

| Year | Discharges | AAHR |
|------|------------|------|
| 1993 | 640 | 29.3 |
| 1994 | 620 | 28.2 |
| 1995 | 547 | 24.5 |
| 1996 | 516 | 23.2 |
| 1997 | 504 | 22.0 |
| 5-yr | 2,827 | 25.8 |

LUNG CANCER

| Year | Discharges | AAHR |
|------|------------|------|
| 1993 | 828 | 43.4 |
| 1994 | 833 | 43.3 |
| 1995 | 852 | 43.6 |
| 1996 | 893 | 46.5 |
| 1997 | 979 | 49.8 |
| 5-yr | 4,385 | 45.8 |

ENDOMETRIAL CANCER

| Year | Discharges | AAHR |
|------|------------|------|
| 1993 | 520 | 27.6 |
| 1994 | 515 | 27.0 |
| 1995 | 513 | 27.7 |
| 1996 | 516 | 27.2 |
| 1997 | 494 | 26.0 |
| 5-yr | 2,558 | 27.5 |

OSTEOARTHRITIS

| Year | Discharges | AAHR |
|------|------------|-------|
| 1993 | 2,080 | 103.9 |
| 1994 | 2,202 | 110.1 |
| 1995 | 2,353 | 117.1 |
| 1996 | 2,591 | 128.8 |
| 1997 | 2,800 | 140.3 |
| 5-yr | 12,026 | 120.5 |

FEMALE BREAST CANCER

| Year | Discharges | AAHR |
|------|------------|-------|
| 1993 | 1,862 | 102.5 |
| 1994 | 1,822 | 99.8 |
| 1995 | 1,754 | 95.2 |
| 1996 | 1,647 | 90.2 |
| 1997 | 1,588 | 86.0 |
| 5-yr | 8,673 | 95.0 |

COPD

| Year | Discharges | AAHR |
|------|------------|-------|
| 1993 | 5,052 | 276.4 |
| 1994 | 4,967 | 272.6 |
| 1995 | 5,161 | 283.1 |
| 1996 | 5,173 | 284.0 |
| 1997 | 5,110 | 277.9 |
| 5-yr | 25,463 | 279.4 |

AUTO-IMMUNE DISEASES

| Year | Discharges | AAHR |
|------|------------|------|
| 1993 | 462 | 25.6 |
| 1994 | 449 | 25.3 |
| 1995 | 424 | 24.1 |
| 1996 | 464 | 25.9 |
| 1997 | 495 | 27.7 |
| 5-yr | 2,294 | 26.3 |

| SEXUALLY TRANSMITTED DISEASES | | | HIV INFECTION | | | SELF-INFLICTED INJURY | | |
|-------------------------------|------------|------|---------------|------------|------|-----------------------|------------|------|
| Year | Discharges | AAHR | Year | Discharges | AAHR | Year | Discharges | AAHR |
| 1993 | 49 | 2.6 | 1993 | 70 | 3.7 | 1993 | 1,181 | 72.1 |
| 1994 | 46 | 2.6 | 1994 | 147 | 8.0 | 1994 | 1,237 | 76.1 |
| 1995 | 46 | 2.7 | 1995 | 498 | 27.8 | 1995 | 1,169 | 71.6 |
| 1996 | 47 | 2.7 | 1996 | 523 | 29.5 | 1996 | 1,221 | 74.7 |
| 1997 | 31 | 1.6 | 1997 | 338 | 19.2 | 1997 | 1,173 | 71.8 |
| 5-yr | 219 | 2.8 | 5-yr | 1,576 | 18.0 | 5-yr | 5,981 | 73.7 |

| PELVIC INFLAMMATORY DISEASE | | | PNEUMONIA & INFLUENZA | | | FALLS & FALL-RELATED INJURY | | |
|-----------------------------|------------|------|-----------------------|------------|-------|-----------------------------|------------|-------|
| Year | Discharges | AAHR | Year | Discharges | AAHR | Year | Discharges | AAHR |
| 1993 | 538 | 31.5 | 1993 | 5,522 | 279.1 | 1993 | 5,710 | 276.1 |
| 1994 | 566 | 33.9 | 1994 | 5,661 | 283.7 | 1994 | 7,288 | 350.1 |
| 1995 | 492 | 29.1 | 1995 | 5,949 | 294.4 | 1995 | 6,755 | 315.3 |
| 1996 | 421 | 25.4 | 1996 | 5,714 | 281.0 | 1996 | 6,797 | 312.1 |
| 1997 | 380 | 23.1 | 1997 | 5,717 | 276.6 | 1997 | 6,646 | 300.3 |
| 5-yr | 2,397 | 29.1 | 5-yr | 28,563 | 283.6 | 5-yr | 33,196 | 311.6 |

Source: Connecticut Department of Public Health, Division of Policy, Planning & Analysis. 2001. Unpublished data.

Notes: Rates adjusted to the 2000 U.S. standard million population. U.S. Census Bureau population estimates used for rate calculations are presented in Appendix B. See Appendix A for definition of terms used in hospitalization analyses.

**AGE-ADJUSTED HOSPITALIZATION RATES (AAHR) FOR SELECTED CAUSES
BY RACE AND ETHNICITY
Connecticut Females, 1993-1997**

CARDIOVASCULAR DISEASE

| Race/Ethnicity | Discharges | AAHR |
|------------------------|------------|---------|
| All Females | 143,705 | 1395.9* |
| White, Non-Hispanic | 126,553 | 1329.3 |
| Black, Non-Hispanic | 10,720 | 2080.0* |
| Asian/PI, Non-Hispanic | 230 | 316.2* |
| Native American, Non-H | 45 | 349.8* |
| Hispanic | 4,277 | 1330.2 |

CORONARY HEART DISEASE

| Race/Ethnicity | Discharges | AAHR |
|------------------------|------------|--------|
| All Females | 49,688 | 493.9* |
| White, Non-Hispanic | 44,280 | 478.5 |
| Black, Non-Hispanic | 2,881 | 556.7* |
| Asian/PI, Non-Hispanic | 65 | 93.6* |
| Native American, Non-H | 12 | † |
| Hispanic | 1,633 | 519.8* |

CEREBROVASCULAR DISEASE

| Race/Ethnicity | Discharges | AAHR |
|------------------------|------------|--------|
| All Females | 25,133 | 237.2* |
| White, Non-Hispanic | 22,314 | 226.4 |
| Black, Non-Hispanic | 1,952 | 396.1* |
| Asian/PI, Non-Hispanic | 60 | 90.7* |
| Native American, Non-H | 8 | † |
| Hispanic | 501 | 164.9* |

LUNG CANCER

| Race/Ethnicity | Discharges | AAHR |
|------------------------|------------|-------|
| All Females | 4,385 | 45.8 |
| White, Non-Hispanic | 4,002 | 46.3 |
| Black, Non-Hispanic | 266 | 49.3 |
| Asian/PI, Non-Hispanic | 5 | † |
| Native American, Non-H | 0 | |
| Hispanic | 63 | 18.1* |

FEMALE BREAST CANCER

| Race/Ethnicity | Discharges | AAHR |
|------------------------|------------|-------|
| All Females | 8,673 | 95.0 |
| White, Non-Hispanic | 7,834 | 96.9 |
| Black, Non-Hispanic | 515 | 90.3 |
| Asian/PI, Non-Hispanic | 34 | 30.0* |
| Native American, Non-H | 4 | † |
| Hispanic | 158 | 40.1* |

COLORECTAL CANCER

| Race/Ethnicity | Discharges | AAHR |
|------------------------|------------|-------|
| All Females | 5,142 | 50.1 |
| White, Non-Hispanic | 4,695 | 49.8 |
| Black, Non-Hispanic | 288 | 56.2 |
| Asian/PI, Non-Hispanic | 10 | † |
| Native American, Non-H | 1 | † |
| Hispanic | 80 | 26.0* |

CERVICAL CANCER

| Race/Ethnicity | Discharges | AAHR |
|------------------------|------------|-------|
| All Females | 918 | 10.4* |
| White, Non-Hispanic | 663 | 8.7 |
| Black, Non-Hispanic | 142 | 23.3* |
| Asian/PI, Non-Hispanic | 12 | † |
| Native American, Non-H | 0 | † |
| Hispanic | 77 | 18.5* |

OVARIAN CANCER

| Race/Ethnicity | Discharges | AAHR |
|------------------------|------------|-------|
| All Females | 1,540 | 17.0 |
| White, Non-Hispanic | 1,420 | 17.7 |
| Black, Non-Hispanic | 50 | 8.7* |
| Asian/PI, Non-Hispanic | 5 | † |
| Native American, Non-H | 0 | † |
| Hispanic | 44 | 10.7* |

ENDOMETRIAL CANCER

| Race/Ethnicity | Discharges | AAHR |
|------------------------|------------|-------|
| All Females | 2,558 | 27.5 |
| White, Non-Hispanic | 2,360 | 28.3 |
| Black, Non-Hispanic | 115 | 21.8* |
| Asian/PI, Non-Hispanic | 7 | † |
| Native American, Non-H | 0 | † |
| Hispanic | 33 | 9.3* |

CHRONIC OBSTRUCTIVE PULMONARY DIS.

| Race/Ethnicity | Discharges | AAHR |
|------------------------|------------|--------|
| All Females | 25,463 | 279.4* |
| White, Non-Hispanic | 18,486 | 225.9 |
| Black, Non-Hispanic | 3,278 | 489.1* |
| Asian/PI, Non-Hispanic | 57 | 40.8* |
| Native American, Non-H | 13 | † |
| Hispanic | 3,413 | 664.7* |

ASTHMA

| Race/Ethnicity | Discharges | AAHR |
|------------------------|------------|--------|
| All Females | 13,403 | 158.6* |
| White, Non-Hispanic | 7,452 | 104.4 |
| Black, Non-Hispanic | 2,708 | 380.4* |
| Asian/PI, Non-Hispanic | 48 | 30.5* |
| Native American, Non-H | 11 | † |
| Hispanic | 3,043 | 546.4* |

DIABETES

| Race/Ethnicity | Discharges | AAHR |
|------------------------|------------|--------|
| All Females | 9,462 | 103.7* |
| White, Non-Hispanic | 6,723 | 83.8 |
| Black, Non-Hispanic | 1,967 | 337.3* |
| Asian/PI, Non-Hispanic | 14 | † |
| Native American, Non-H | 4 | † |
| Hispanic | 646 | 156.6* |

OSTEOPOROSIS

| Race/Ethnicity | Discharges | AAHR |
|------------------------|------------|------|
| All Females | 2,827 | 25.8 |
| White, Non-Hispanic | 2,707 | 26.6 |
| Black, Non-Hispanic | 39 | 8.0* |
| Asian/PI, Non-Hispanic | 4 | † |
| Native American, Non-H | 1 | † |
| Hispanic | 25 | 7.5* |

OSTEOARTHRITIS

| Race/Ethnicity | Discharges | AAHR |
|------------------------|------------|-------|
| All Females | 12,026 | 120.5 |
| White, Non-Hispanic | 11,070 | 121.6 |
| Black, Non-Hispanic | 626 | 123.9 |
| Asian/PI, Non-Hispanic | 9 | † |
| Native American, Non-H | 7 | † |
| Hispanic | 143 | 45.3* |

SEXUALLY TRANSMITTED DISEASES

| Race/Ethnicity | Discharges | AAHR |
|------------------------|------------|-------|
| All Females | 219 | 2.8* |
| White, Non-Hispanic | 59 | 0.9 |
| Black, Non-Hispanic | 120 | 15.8* |
| Asian/PI, Non-Hispanic | 18 | † |
| Native American, Non-H | 1 | † |
| Hispanic | 35 | 4.5* |

PELVIC INFLAMMATORY DISEASE

| Race/Ethnicity | Discharges | AAHR |
|------------------------|------------|-------|
| All Females | 2,397 | 29.1* |
| White, Non-Hispanic | 1,356 | 20.3 |
| Black, Non-Hispanic | 656 | 83.2* |
| Asian/PI, Non-Hispanic | 18 | † |
| Native American, Non-H | 1 | † |
| Hispanic | 324 | 46.5* |

HIV

| Race/Ethnicity | Discharges | AAHR |
|------------------------|------------|-------|
| All Females | 1,576 | 18.0* |
| White, Non-Hispanic | 390 | 5.4 |
| Black, Non-Hispanic | 757 | 98.0* |
| Asian/PI, Non-Hispanic | 0 | |
| Native American, Non-H | 0 | |
| Hispanic | 413 | 63.2* |

PNEUMONIA & INFLUENZA

| Race/Ethnicity | Discharges | AAHR |
|------------------------|------------|--------|
| All Females | 28,563 | 283.6* |
| White, Non-Hispanic | 23,698 | 256.1 |
| Black, Non-Hispanic | 2,714 | 435.6* |
| Asian/PI, Non-Hispanic | 53 | 63.1* |
| Native American, Non-H | 11 | † |
| Hispanic | 1,783 | 403.3* |

SELF-INFLICTED INJURY

| Race/Ethnicity | Discharges | AAHR |
|------------------------|------------|-------|
| All Females | 5,981 | 73.7 |
| White, Non-Hispanic | 4,664 | 71.6 |
| Black, Non-Hispanic | 515 | 65.1 |
| Asian/PI, Non-Hispanic | 32 | 16.8* |
| Native American, Non-H | 5 | † |
| Hispanic | 654 | 97.9* |

FALLS & FALL-RELATED INJURY

| Race/Ethnicity | Discharges | AAHR |
|------------------------|------------|--------|
| All Females | 33,196 | 311.6 |
| White, Non-Hispanic | 30,917 | 316.4 |
| Black, Non-Hispanic | 1,158 | 208.9* |
| Asian/PI, Non-Hispanic | | |
| Native American, Non-H | | |
| Hispanic | 610 | 152.4* |

Source: Connecticut Department of Public Health, Division of Policy, Planning & Analysis. 2001. Unpublished data.

Notes:

* There is a statistically significant difference from the White, Non-Hispanic AAHR.

† Statistics not calculated for fewer than 25 events.

Rates adjusted to the 2000 U.S. standard million population. U.S. Census Bureau population estimates used for rate calculations are presented in Appendix B. See Appendix A for definition of terms used in hospitalization analyses.

APPENDIX E. SUMMARY OF BEHAVIORAL RISK FACTORS

PREVALENCE OF HEALTH-RELATED RISK FACTORS Connecticut Females, Age 18+, 1994-2000

| Risk Factor | 1994 | 1995 | 1996 | 1997 | 1998 | 1999 | 2000 |
|--|------|------|------|------|------|------|------|
| Health status poor or fair | 11.4 | 10.1 | 11.4 | 11.0 | 12.2 | 11.3 | 14.2 |
| No health plan/insurance (18-64 yrs) | 8.7 | 6.6 | 10.9 | 9.9 | 9.7 | 9.7 | 9.0 |
| No leisure time physical activity ¹ | 26.4 | | 27.2 | | 29.8 | | 27.7 |
| No regular or sustained physical activity ² | 76.1 | | 79.1 | | 81.0 | | 77.5 |
| Not eating fewer calories and exercising to lose weight | 67.1 | | 50.0 | | 48.3 | | 47.1 |
| Not eating fruits and vegetables ³ | 61.3 | | 66.7 | 61.8 | 67.3 | | 67.5 |
| Overweight ⁴ | 19.2 | 21.9 | 21.9 | 26.0 | 24.7 | 27.2 | 29.4 |
| Ever smoked ⁵ | 44.2 | 48.2 | 46.2 | 49.5 | 46.3 | 43.4 | 45.7 |
| Current smoker ⁶ | 19.1 | 20.6 | 20.9 | 21.9 | 20.4 | 20.6 | 19.4 |
| Current drinker ⁷ | | 58.2 | | 55.5 | | 53.2 | |
| Binge drinker ⁸ | | 6.6 | | 8.0 | | 6.3 | |
| Chronic drinker ⁹ | | 1.8 | | 1.5 | | 0.4 | |
| Never had clinical breast exam & mammogram (40+ yrs) | 21.7 | 20.7 | 16.5 | 19.6 | 20.4 | 16.7 | 13.4 |
| No mammogram in 2 yrs (50+ yrs) | 33.1 | 23.5 | 20.1 | 22.3 | 22.0 | 13.9 | 15.4 |
| Never had pap smear (w/ intact cervix) | 7.9 | 6.2 | 6.4 | 7.8 | 7.3 | 5.7 | 5.9 |
| No pap smear in 3 yrs (w/ intact cervix) | 15.1 | 13.2 | 10.2 | 17.0 | 13.8 | 11.7 | 12.0 |
| Blood pressure not checked w/in 2 years | | 3.2 | | 4.3 | | 2.4 | |
| Told of high blood pressure (of those tested) | | 19.5 | | 20.7 | | 21.4 | |
| Never had cholesterol screening | 27.7 | 23.5 | | 21.1 | | 18.1 | |
| Cholesterol not checked within 5 years | 31.2 | 27.5 | | 25.8 | | 22.5 | |
| Told of high cholesterol (of those checked) | 20.6 | 18.6 | | 23.7 | | 26.9 | |
| Ever told has diabetes | 5.3 | 4.1 | 5.5 | 5.2 | 3.8 | 4.7 | 5.1 |
| Never tested for HIV within 1 year (18-64 yrs) | 75.8 | 65.1 | 61.9 | 60.2 | 58.7 | 61.7 | 64.4 |
| Medium or high chance of getting HIV (18-64 yrs) | 7.3 | 5.8 | 6.2 | 5.0 | 6.5 | 5.9 | 5.1 |
| No flu shot in past year (65+ yrs) | | 41.2 | | 34.7 | | 35.3 | |
| Never had pneumonia vaccine (65+ yrs) | | 61.6 | | 55.1 | | 50.0 | |
| Did not use home blood stool test within 2 yrs (50+ yrs) | | | | 67.6 | | 69.2 | |
| Never had a sigmoidoscopy (50+ yrs) | | | | 57.4 | | 49.5 | |

Source: Centers for Disease Control and Prevention. 1994, 1995, 1996, 1997, 1998, 1999, 2000. *Behavioral Risk Factor Surveillance System Connecticut Statewide Survey Data – Weighted*. Atlanta: Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Behavioral Surveillance Branch.

¹ No leisure time physical activity in the previous month.

² Physical activity for 30+ minutes a day at least 5 times per week, regardless of intensity.

³ Do not consume 5 or more servings of fruits and vegetables per day.

⁴ Body mass index greater than or equal to 27.3.

⁵ Smoked at least 100 cigarettes in their lifetime.

⁶ Smoked at least 100 cigarettes in lifetime and currently smoke some or every day.

⁷ Had alcoholic beverages during the past month.

⁸ Consumed 5 or more drinks on at least 1 occasion in the last month.

⁹ Consumed 60+ drinks in the last month.