

# 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,

A handwritten signature in black ink, appearing to read "J. Garcia", written over the printed name and title.

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



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connecticut **women's** health

CONNECTICUT DEPARTMENT OF PUBLIC HEALTH  
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## INTRODUCTION

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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.

## REFERENCES

- 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

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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 21<sup>st</sup> 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.

## REFERENCES

- Abel, E.K. and M.K. Nelson. 1990. *Circles of Care*. Albany, NY: SUNY Press.
- American Medical Association Council on Ethical and Judicial Affairs. 1991. Gender disparities in clinical decisionmaking. *Journal of the American Medical Association* 266: 559-562.
- Auerbach, J.D. 1988. *In the Business of Child Care: Employer Initiatives and Working Women*. New York: Praeger.
- Auerbach, J.D. and A.E. Figert. 1995. Women's health research: Public policy and sociology. *Journal of Health and Social Behavior* (Extra Issue): 115-131.
- Avery, B.Y. 1996. Breathing life into ourselves: The evolution of the National Black Women's Health Project. Pp. 761-766 in P.Brown, ed. *Perspectives in Medical Sociology*. Prospect Heights, Illinois: Waveland Press.
- Bird, C.E. and P.P. Rieker. 1999. Gender matters: An integrated model for understanding men's and women's health. *Social Science & Medicine* 48: 745-755.
- Boston Women's Health Book Collective. 1971. *Our Bodies, Ourselves*. New York: Simon and Schuster.
- Boston Women's Health Book Collective. 1998. *Our Bodies, Ourselves for the New Century: A Book by and for Women*. New York: Simon and Schuster.
- Colditz, G.A. J.E. Manson, S.E. Hankinson. 1997. The Nurses' Health Study: 20 year contribution to the understanding of health among women. *Journal of Women's Health* 6: 49-61.
- Dubos, R. 1959. *Mirage of Health*. New York: Simon and Schuster.
- Ehrenreich, B. and D. English. 1978. *For Her Own Good*. New York: Doubleday.
- Evans, R.G., M.L. Barer, T.R. Marmor. 1994. *Why Are Some People Healthy and Others Not?* New York: Aldine De Gruyter.
- Hankinson, S.E., G.A. Colditz, F. Speizer, et al., eds. 2001. *Healthy Women, Healthy Lives: A Guide to Preventing Disease, from the Landmark Nurses' Health Study*. New York: Simon and Schuster.
- Institute of Medicine. 2001. *Exploring the Biological Contributions to Human Health: Does Sex Matter?* Washington, D.C.: National Academy of Sciences.
- Khan, S.S., S. Nessim, R. Gray, et al. 1990. Increased mortality of women in coronary artery bypass surgery: Evidence for referral bias. *Annals of Internal Medicine* 112(8): 561-567.
- Kirschstein, R.L. 1991. Research on women's health. *American Journal of Public Health* 81(3): 291-293.
- Kuhn and Rackley. 1993. Coronary artery disease in women. Risk factors, evaluation, treatment, and prevention. *Archives of Internal Medicine* 153(23): 2626-2636.
- Longino C.F. 1988. Who are the oldest Americans? *Gerontologist* 28(4):515-23.
- Lorber, J. 1997. *Gender and the Social Construction of Illness*. Thousand Oaks, CA: Sage.

- Manson, J.E., J.E. Buring, S. Satterfield, et al. 1991. Baseline characteristics of participants in the Physicians' Health Study: A randomized trial of aspirin and beta-carotene in U.S. physicians. *American Journal of Preventive Medicine* 7(3): 150-154.
- Matthews, K.A., S.A. Shumaker, D.J. Bowen, et al. 1997. Women's Health Initiative: Why now? What is it? What's new? *American Psychologist* 52(2): 101-116.
- McGowan, J.A. and L. Pottern. 2000. Commentary on the Women's Health Initiative. *Maturitas* 34(2): 109-112.
- McKinlay, J.B. 1996. Some contributions from the social system to gender inequalities in heart disease. *Journal of Health and Social Behavior* 37(1): 1-26.
- Mechanic, D., ed. 1982. *Symptoms, Illness Behavior, and Health-Seeking*. New Brunswick, NJ: Rutgers University Press.
- Multiple Risk Factor Intervention Trial Research Group. 1985. Exercise electrocardiogram and coronary heart disease mortality in the Multiple Risk Factor Intervention Trial. *American Journal of Cardiology* 55(1): 16-24.
- Nathanson, C. 1984. Sex differences in mortality. *Annual Review of Sociology* 10: 191-213.
- Ory, M.G. and H.R. Warner, eds. 1990. *Gender, Health, and Longevity: Multidisciplinary Perspectives*. New York: Springer.
- Smyke, P. 1991. *Women and Health*. London: Zed Books.
- Sontag, S. 1989. *Illness as Metaphor and AIDS and its Metaphors*. New York: Doubleday.
- Stillion, J.M. 1995. Premature death among males—extending the bottom line of men's health. Pp 46-67 in *Men's Health and Illness* D. Sabo and D.F. Gordon (eds.) Thousand Oaks, CA: Sage.
- Thoits, P.A. 1986. Multiple identities: Examining gender and marital status differences in distress. *American Sociological Review* 51: 259-272.
- Tobin, J.N., S. Wassertheil-Smoller, J.P. Wexler, et al. 1987. Sex bias in considering coronary bypass surgery. *Annals of Internal Medicine* 107(1): 19-25.
- U.S. Public Health Service, Gerontology Research Center, and National Institutes of Health; N.W. Shock, ed., 1984. *Normal Human Aging: The Baltimore Longitudinal Study of Aging*. Bethesda, MD: NIH.
- U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health. 2001. *Agenda for Research on Women's Health for the 21<sup>st</sup> Century. A Report of the Task Force on the NIH Women's Health Research Agenda for the 21<sup>st</sup> Century, Volumes 1 to 6*. Bethesda, MD: NIH.
- Verbrugge, L.M. 1976. Females and illness: Recent trends in sex differences in the United States. *Journal of Health and Social Behavior* 17: 387-403.
- Verbrugge, L.M. 1989. The twain meet: Empirical explanations of sex differences in health and mortality. *Journal of Health and Social Behavior* 30: 282-304.
- Verbrugge, L.M. and D.L. Wingard. 1987. Sex differentials in health and mortality. *Women & Health* 12(2): 103-145.
- Waldron, I. 1986. What do we know about causes of sex differences in mortality? *Population Bulletin of the U.N.* No. 18-1985, 59-76.
- Waldron, I. 1995. Contributions of changing gender differences in behavior and social roles to changing gender differences in mortality. Pp. 22-45 in *Men's Health and Illness* D. Sabo and D.F. Gordon (eds.) Thousand Oaks, CA: Sage.
- Worobey, J.L. and R.J. Angel. 1990. Poverty and health: Older minority women and the rise of the female-headed household. *Journal of Health and Social Behavior* 31: 370-383.
- Zola, I.K. 1966. Culture and symptoms: An analysis of patients presenting complaints. *American Sociological Review* 33: 615-630.

## 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.

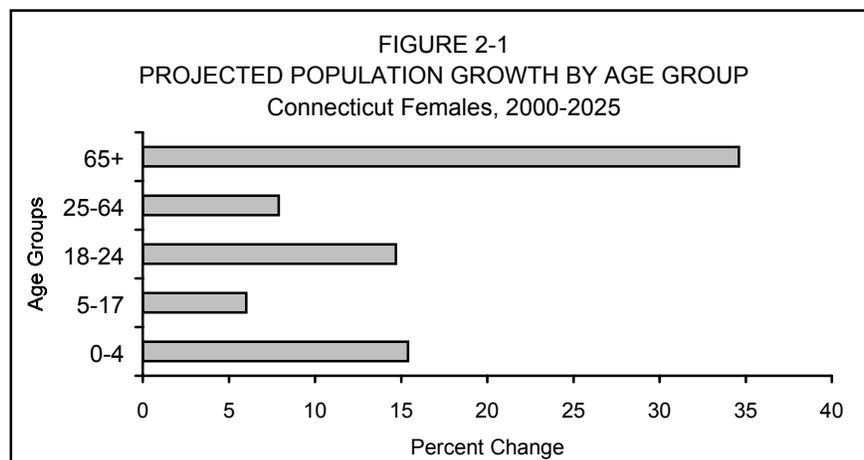
### AGE GROUPS

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

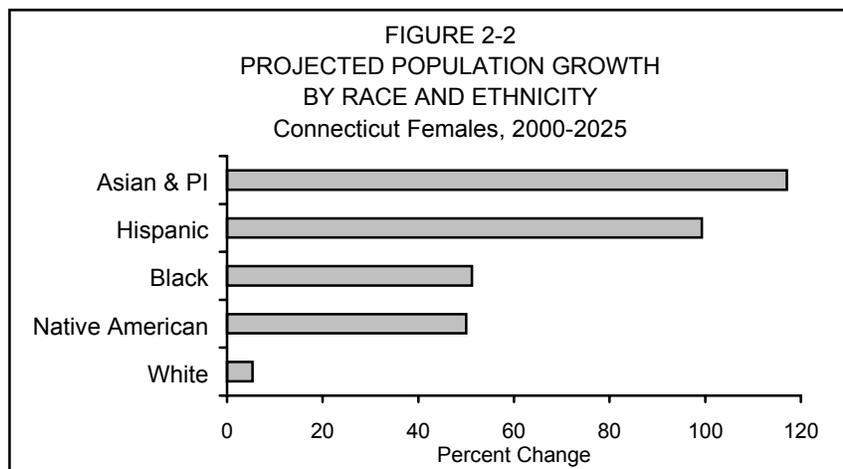
Race and ethnicity are strongly associated with health outcomes. The life expectancy at birth for white females in the U.S. is 5.2 years longer than that for black females. Women of color have higher mortality rates than white women for certain cancers, cardiovascular disease, diabetes, chronic obstructive 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*

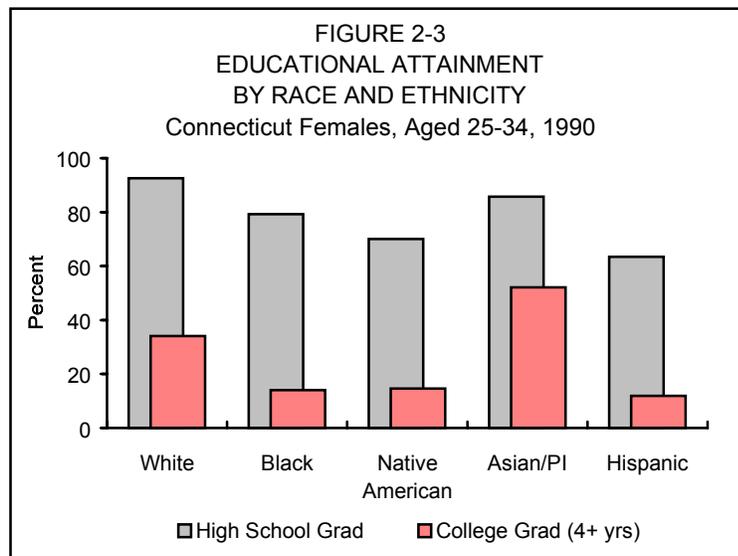
Level of education is linked closely to health, morbidity, and mortality; persons with more education have better health and lower mortality rates than those with less education (Pappas et al., 1993). Education also influences how people make health-related decisions. It was found, for example, that 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



Source: U.S. Census Bureau, 1993 (Table 47).

### Employment

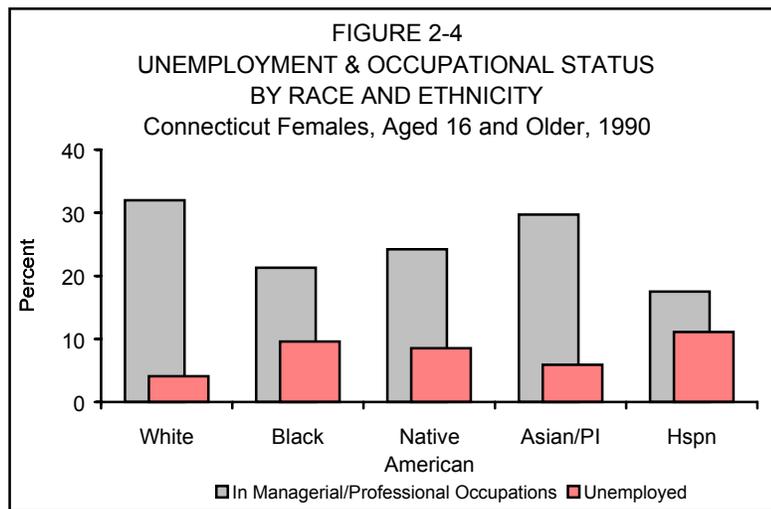
A strong relationship also exists between employment and health, not only through the effects of the resulting income but also because of the contributions of the work environment to health and health behaviors (work-related stress, social support networks, etc.). Women in clerical positions, for example, 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 level<sup>1</sup> 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).

## REFERENCES

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

- Auerbach, J.A., B.K. Krimgold, and B. Lefkowitz, 2000. *Improving Health: It Doesn't Take a Revolution*. Washington, D.C.: National Policy Association.
- 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.
- Davern, M.E. and P.J. Fisher. 2001. *Household Net Worth and Asset Ownership: 1995*. U.S. Census Bureau Current Population Reports, Series P70-71. Washington, DC: U.S. Government Printing Office  
<http://www.census.gov/hhes/www/wealth/1995/wealth95.html> (3/09/01).
- Haan, M., G.A. Kaplan, and T. Camacho. 1987. Poverty and health - prospective evidence from the Alameda county study. *American Journal of Epidemiology*; 125(6):989-998.
- Hemingway, H., M.J. Shipley, S. Stansfeld, and M. Marmot. 1997. Sickness absence from back pain, psychosocial work characteristics and employment grade among office workers. *Scandinavian Journal of Work and Environmental Health* 23: 121-129.
- Hynes, M.M., L.M. Mueller, C.E. Bower, and M.J. Hofmann. 1999. *Multicultural Health: The Health Status of Minority Groups in Connecticut*. Hartford, CT: Connecticut Department of Public Health.
- Lantz, P.M., J.S. House, J.M. Lepkowski, D.R. Williams, R.P. Mero, and J. Chen. 1998. Socioeconomic factors, health behaviors, and mortality. *Journal of the American Medical Association* 279:1703-1707.
- Lyons B., A. Salganicoff, and D. Rowland. 1996. Poverty, access to health care, and Medicaid's critical role for women, in *Women's Health: The Commonwealth Fund Survey*. Falik MM, Collins K, eds., Baltimore, MD: Johns Hopkins University Press.
- Marmot, M.G., H. Bosma, H. Hemingway, E. Brunner, and S. Stansfeld. 1997. Contribution of job control and other risk factors to social variations in coronary heart disease incidence. *Lancet* 350: 235-239..
- Mishel, L., J. Bernstein, and J. Schmitt. 2001. *The State of Working America 2000-2001*. Ithaca, NY: Cornell University Press.
- Murphy, S.L. 2000. Deaths: Final data for 1998. *National Vital Statistics Reports* 48(11): 1-108.
- National Center for Health Statistics, 2000. *Health, United States, 2000*, with Adolescent Health Chartbook. Hyattsville, MD: U.S. Department of Health and Human Services.
- National Institute on Aging. 1999. Socioeconomic status, health and longevity. *Research Highlights in the Demography and Economics of Aging* 4: 1-4.
- Pappas, G., S., W. Queen, W. Hadden, and G. Fisher. 1993. The increasing disparity in mortality between socioeconomic groups in the United States, 1960 and 1986. *The New England Journal of Medicine*, 329(2):103-109.
- Permanent Commission on the Status of Women, Lifetime Television for Women, and the Center for Policy Alternatives. 2000. *Connecticut Women's Voices 2000*. Hartford, CT: Permanent Commission on the Status of Women.

<sup>1</sup> 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).

- Polednak, A.P. 1997. Segregation, Poverty, and Mortality in Urban African Americans. New York: Oxford University Press.
- Schulz, R. and S.R. Beach. 1999. Caregiving as a risk factor for mortality – The caregiver health effects study. *Journal of American Medical Association* 282(23):2215-2219.
- U.S. Census Bureau. 1993. *1990 Census of Population. Social and Economic Characteristics. Connecticut*. Washington, D.C.: U.S. Department of Commerce Publ. No. 1990 CP-2-8.
- U.S. Census Bureau. 1999. Estimated Connecticut population by sex, race, and Hispanic origin. ST-99-43, Internet release date 12/29/99 <http://www.census.gov/population/estimates/state/sasrh/sasrh99.txt> (9/19/00).
- U.S. Census Bureau. 2000. Projected Connecticut population by sex, race, and Hispanic origin: 1995-2025. <http://www.census.gov/population/projections/state/stpjrace.txt> (6/18/01).
- U.S. Census Bureau. 2001. Poverty thresholds in 1989 and 1999, by size of family and number of related children under 18 years. *Current Population Survey*. Washington, D.C.: U.S. Department of Commerce. <http://www.census.gov/hhes/poverty/threshld.html> (3/2/01).
- U.S. Department of Health and Human Services. 2000. *Healthy People 2010: Understanding and Improving Health*. 2nd ed. Washington, D.C.: U.S. Government Printing Office.
- Verbrugge, L.M. and J.H. Madans. 1985. Social roles and health trends of American women. *Milbank Memorial Fund Quarterly*. Fall; 63(4):691-735.
- Waldron, I., M.E. Hughes, and T.L. Brooks. 1996. Marriage protection and marriage selection - prospective evidence for reciprocal effects of marital status and health. *Social Science and Medicine* Jul;43(1):113-23.
- Wray, L.A., A.R. Herzog, R.J. Willis, and R.B. Wallace. 1998. The impact of education and heart attack on smoking cessation among middle-aged adults. *Journal of Health and Human Behavior* 39: 271-294.

### 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.

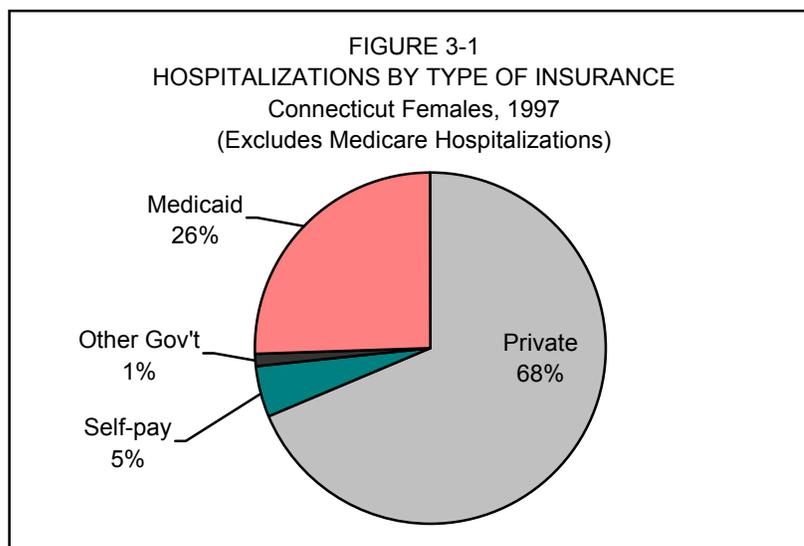
#### HEALTH INSURANCE COVERAGE

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<sup>a</sup>  
Connecticut, 2000

Geographic Areas	Populations <sup>b</sup>
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.

<sup>a</sup> Geographic areas and populations do not necessarily include all census tracts within the named town.

<sup>b</sup> All Health Professional Shortage Populations qualify as low-income.

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

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

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

<sup>a</sup> Counts are for those without sub-specialties.

<sup>b</sup> 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.

## REFERENCES

- Aaron, D.J., N. Markovic, M.E. Danielson, J.A. Honnold, J.E. Janosky, and N.J. Schmidt. 2001. Behavioral risk factors for disease and preventive health practices among lesbians. *American Journal of Public Health* 91: 972-975.
- Bailey, J.V., J. Kavanagh, J. Owen, et al. 2000. Lesbians and cervical screening. *British Journal of General Practice* 50: 481-482.
- Bayne-Smith, M. 1996. Health and women of color: A contextual overview. In: *Race, Gender, and Health*, M. Bayne-Smith, Ed. London: Sage Publications.
- Bureau of Health Professions. 2000. *HRSA State Health Workforce Data Resource Guide and Workforce Profile—Connecticut*. Washington, DC: National Center for Health Workforce Information & Analysis, Health Resources and Services Administration, U.S. Department of Health and Human Services.
- Bureau of Primary Health Care. 1989. Criteria for Designation of Areas Having Shortages of Primary Medical Care Professional(s). [http://www.bphc.hrsa.gov/dsd/hpsa\\_fr2.htm](http://www.bphc.hrsa.gov/dsd/hpsa_fr2.htm). (6/06/01).
- Bureau of Primary Health Care. 2001. Selected statistics on health professional shortage areas as of March 31, 2001. Division of Shortage Designation, Bureau of Primary Health Care, HRSA.
- Burnett, C.B., C.S. Steakley, R. Slack, et al. 1999. Patterns of breast cancer screening among lesbians at increased risk for breast cancer. *Women Health* 29: 35-55.
- Center for Health Economics Research. 1993. *Access to Health Care. Key Indicators for Policy*. Princeton, NJ: Robert Wood Johnson Foundation.

- Centers for Disease Control and Prevention. 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.
- Clark, M.E., S. Landers, R. Linde, and J. Sperber. 2001. The GLBT Health Access Project: A state-funded effort to improve access to care. *American Journal of Public Health* 91: 895-896.
- Cochran, S.D., V.M. Mays, D. Bowen, et al. 2001. Cancer-related risk indicators and preventive screening behaviors among lesbians and bisexual women. *American Journal of Public Health* 91: 591-597.
- Connecticut Commission on the Deaf and Hearing Impaired. 2001. Estimates of hard of hearing and profoundly deaf persons in Connecticut.
- Connecticut Department of Public Health, Bureau of Regulatory Services, Division of Health Systems Regulation. 2001. Unpublished analysis of data from the *Connecticut Physician Profile Survey*.
- Connecticut Department of Public Health, Division of Policy, Planning, and Analysis. 2001. Unpublished data.
- Dean, L., I.H. Meyer, K. Robinson, et al. 2000. Lesbian, gay, bisexual, and transgender health: Findings and concerns. *Journal of the Gay and Lesbian Medical Association* 4: 101-151.
- Diamant, A.L., C. Wold, K. Spritzer, and L. Gelberg. 2000. Health behaviors, health status, and access to and use of health care: a population-based study of lesbian, bisexual, and heterosexual women. *Archives of Family Medicine* 9: 1043-1051.
- Foland, J. 2000. Avoidable hospitalizations: An indicator of inadequate primary care. *Issue Briefs*, No. 2000-1. <http://www.state.ct.us/dph/OPPE/brief20001.htm> (3/15/01).
- Lauver, D.R., S.L. Karon, J. Egan, et al. 1999. Understanding lesbians' mammography utilization. *Women's Health Issues* 9: 264-274.
- Marrazzo, J.M., L.A. Koutsky, N.B. Kiviat, et al. 2001. Papanicolaou test screening and prevalence of genital human papillomavirus among women who have sex with women. *American Journal of Public Health* 91: 947-952.
- Meyer, I.H. 2001. Why lesbian, gay, bisexual, and transgender public health? *American Journal of Public Health* 91: 856-858.
- Millman, M., Ed. 1993. *Access to Health Care in America*. Washington, DC: Institute of Medicine.
- Office of Health Care Access. 1999. Administrative Regulations & Rules of Practice, Effective February 26, 1999. <http://www.state.ct.us/ohca/indinfo/indinfoframes.htm> (3/18/01).
- Office of Health Care Access. 2000. Preventable hospitalizations during the 1990's. *ACHIEVE Issue Brief*, May, 2000.
- Office of Health Care Access. 2001. Estimates of Connecticut's uninsured using different methods. *ACHIEVE Issue Brief*, April, 2001.
- Office of Minority, Health. 2000. Assuring cultural competence in health care: Recommendations for national standards and outcomes-focused research agenda. Part One: Recommendations for national standards. <http://www.omhrc.gov/clas/po.htm> (3/16/01).
- Scout, M.A., J. Bradford, and C. Fields. 2001. Removing the barriers: Improving practitioners' skills in providing health care to lesbians and women who partner with women. *American Journal of Public Health* 91: 989-1990.
- True, R.H., and T. Guillermo. 1996. Asian/Pacific Islander American Women. In: *Race, Gender, and Health*, M. Bayne-Smith, Ed. London: Sage Publications.
- Turner, C., and E. Campbell. 1999. Counting the uninsured using state-level hospitalization data. *Public Health Reports* 114: 149-156.
- U.S. Census Bureau. 1993. *1990 Census of Population. Social and Economic Characteristics. Connecticut*. Washington, D.C.: U.S. Department of Commerce Publ. No. 1990 CP-2-8.
- U.S. Census Bureau. 2001. DP-2 social characteristics: 1990. Data set: 1990 summary tape file 3 (STF3) – Sample data.
- U.S. Department of Health and Human Services. 2000. *Healthy People 2010*. 2nd ed. 2 vols. Washington, D.C.: U.S. Government Printing Office.
- U.S. Department of Health and Human Services, Health Resources and Services Administration. 2000. Lists of designated primary medical care, mental health, and dental health professional shortage areas. *Federal Register* 65(180):56028-56154 (September 15, 2000).

## 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.

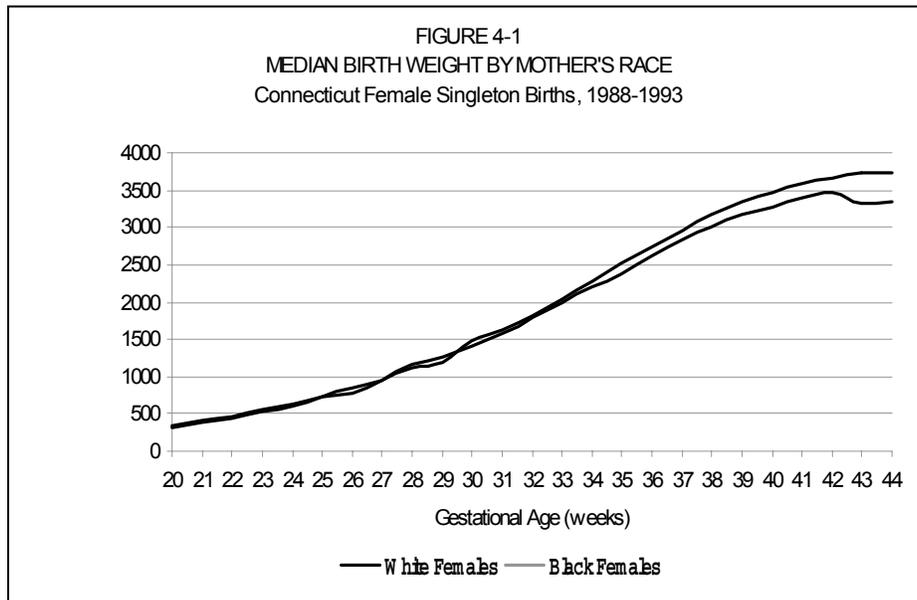
## MENOPAUSE

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

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**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.

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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.

REFERENCES

Adair L.S., P. Gordon-Larsen. 1991. Maturation timing and overweight prevalence in U.S. adolescent girls. *American Journal of Public Health*. April (4):642-4.

American Medical Society. 1997. Women's Health Menopause. [http://www.ama-assn.org/insight/h\\_focus/wom\\_hlth/menopaus/menopaus.htm](http://www.ama-assn.org/insight/h_focus/wom_hlth/menopaus/menopaus.htm).

Endocrinology Society. 1998. *Endocrinology and Menopause*. Bethesda, MD: <http://www.endo-society.org> (7/10/01).

Gallop Organization for ACOG. 1993. *A Gallop Study of Attitudes About the Use of Obstetrician/Gynecologists for Primary Care*, Princeton, N.J.

Gold E.B., J. Bromberger, S. Crawford, et al. 2001. Factors Associated with Age at Natural Menopause in a Multiethnic Sample of Midlife Women, *American Journal of Epidemiology*, 153(9): 865-874.

Kaiser Family Foundation. 2000. *Issue Brief: State Policies on Access to Gynecological Care and Contraception*. <http://www.kff.org/content/2000/1556b/statepol3.pdf> (8/11/2001).

Kaiser Family Foundation, Essence, Latina, and Los Angeles Times. 1999. *A National Survey of Women About Their Reproductive Health Care*.

Murray C.J., A.D. Lopez, Eds. 1996. *The Global Burden of Disease: a comprehensive assessment of mortality and disability from diseases, injuries and risk factors in 1990 and projected to 2020*, Vol-1. Cambridge, Mass: Harvard School of Public Health on behalf of the World Health Organization and the World Bank.

- National Center for Health Statistics. 1997. *Fertility, Family Planning, and Women's Health: New Data From the 1995 National Survey of Family Growth*. Atlanta: Centers for Disease Control and Prevention. Series 23, No. 19.
- North American Association for the Study of Obesity. 2000. *Weight Gain Risk in Women Linked to Key Stages of Life*. Silver Spring, MD.  
<http://www.naaso.org/newsflash/women.htm>.
- Roberts, C., L. Mueller, J. Hadler. 1996. Birthweight percentiles by gestational age, Connecticut 1988-1993, *Connecticut Medicine*, 60(3):131-140.
- U.S. Preventive Services Task Force. 1996. *Guide to Clinical Preventive Services, Second Edition*. Washington D.C.: U.S. Department of Health and Human Services.
- Wilcox A., R. Skjaeven, P. Buekens, et al. 1995. Birthweight and perinatal mortality. A comparison of the United States and Norway. *Journal of the American Medical Association* 273: 709-711.

## 5. PREGNANCY AND BIRTH

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### 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

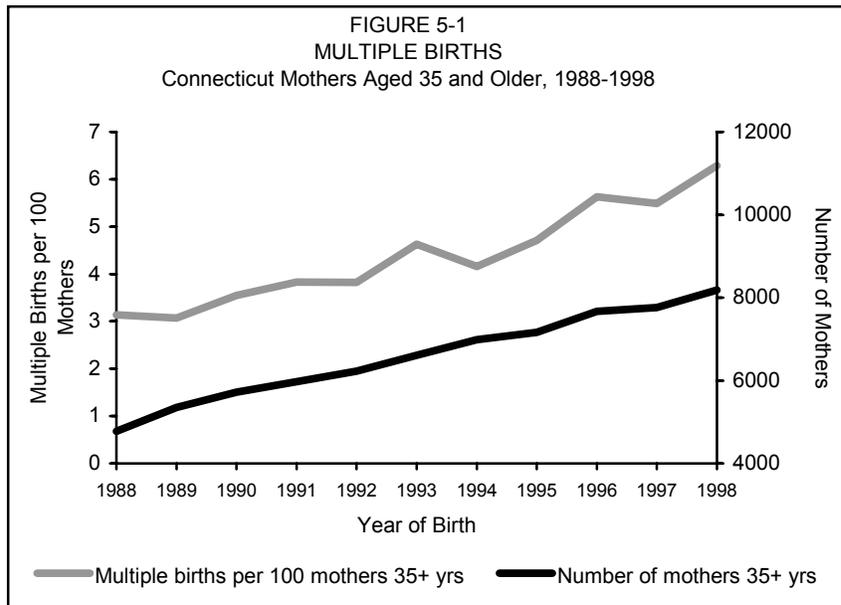
rate for black women, 154.6, was higher than the target value. This finding is consistent with other reports of higher rates of sexually transmitted diseases in Connecticut's black population. As noted in Chapter 20 of this report, black women have higher rates of gonorrhea and chlamydia incidence when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

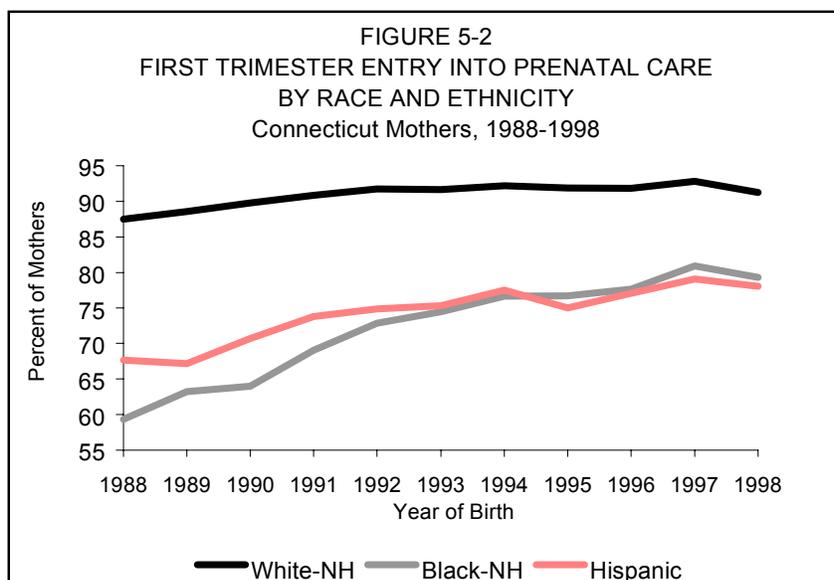
## PREGNANCY

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.

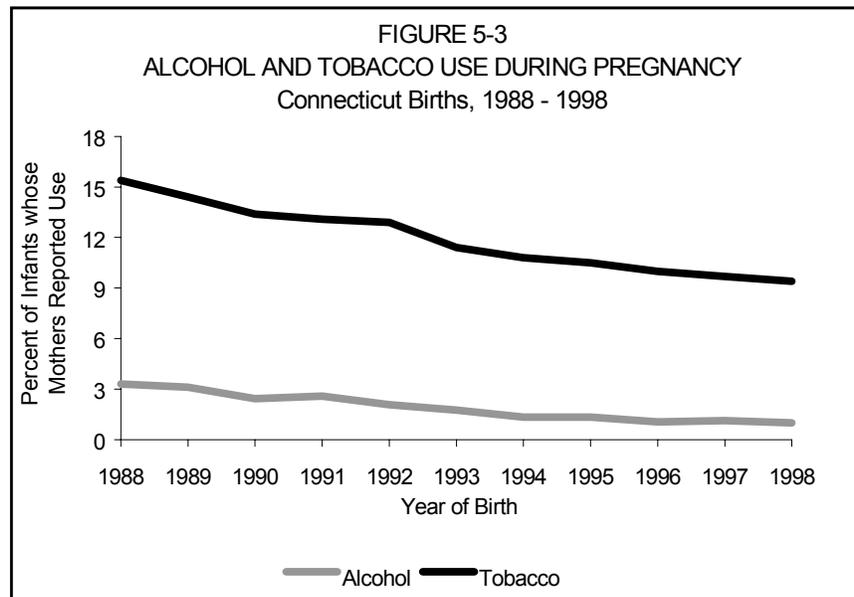
For mothers with singleton births, the percentage receiving early care improved through 1994, when it reached a plateau at 88 percent, just below the *Healthy People 2000* target (90 percent). Fortunately, prenatal care utilization is slightly higher for women with multiple gestation pregnancies, since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

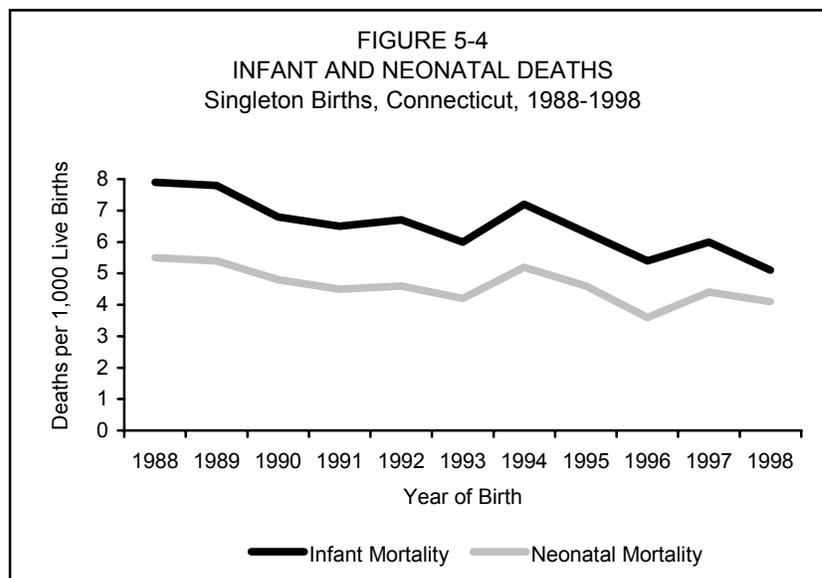
## PERINATAL PERIOD

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.

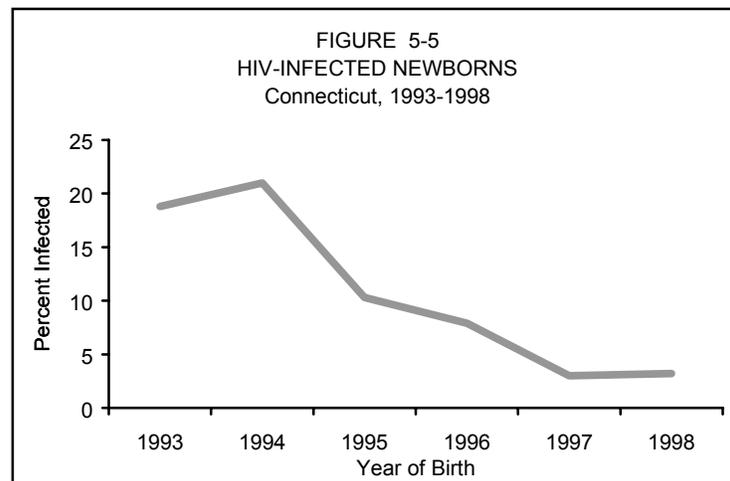
*Perinatal transmission of HIV*

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.

## REFERENCES

- Beaudry, M., R. Dufour, S. Marcoux. 1995. Relation between infant feeding and infections during the first six months of life. *Journal of Pediatrics*. 126:191-197.
- Brett, K.M., D.S. Strogatz, D.A. Savitz. 1997. Employment, the job strain, and preterm delivery among women in North Carolina. *American Journal of Public Health* 87: 1999-2004.
- Brown, S.S., and L. Eisenberg, eds. 1995. *The Best Intentions: Unintended Pregnancy and the Well-Being Of Children And Families*. Washington, D.C.: National Academy Press.
- Callahan, T.L., J.E. Hall, S.L. Ettner, et al. 1994. The economic impact of multiple-gestation pregnancies and the contribution of assisted-reproduction techniques to their incidence. *New England Journal of Medicine*. July 28, 331(4): 244-9.
- Centers for Disease Control and Prevention. Recommendations of the U.S. Public Health Service Task Force on the Use of Zidovudine to Reduce Perinatal Transmission of Human Immunodeficiency Virus. *Morbidity and Mortality Weekly Report*. 1994; 43(RR11);1-20
- Centers for Disease Control and Prevention, 1998. Pediatric Nutrition Surveillance System Annual Summaries.
- Centers for Disease Control and Prevention. 1999a. Ten Great Public Health Achievements- United States 1990-1999. *Morbidity and Mortality Weekly Report*. 48(12): 241-243.

- Centers for Disease Control and Prevention. 1999b. Achievements in Public Health, 1990-1999: Family Planning- United States 1990-1999. *Morbidity and Mortality Weekly Report*. 48(47): 1073-1080.
- Centers for Disease Control and Prevention. 1999c. Achievements in Public Health, 1990-1999: Healthier Mothers and Babies. *Morbidity and Mortality Weekly Report* 48(38): 849-858.
- Centers for Disease Control and Prevention. 2000. Contribution of Assisted Reproductive Technology and ovulation-inducing drugs to triplet and higher-order multiple births—United States, 1980-1997. *Morbidity and Mortality Weekly Report*, 49:535-8.
- Chua, S., S. Arulkumaran, I. Lim, et al. 1994. Influence of breastfeeding and nipple stimulation on postpartum uterine activity. *British Journal of Obstetrics and Gynecology*. 101:804-805.
- Cohen, B.B., D.J. Friedman, Z.Zhang, et al. 1999. Impact of multiple births on low birthweight—Massachusetts, 1989-1996. *Morbidity and Mortality Weekly Report*, 48(14): 289-292.
- Connecticut Department of Public Health. 1997. *Connecticut Registration Report*. Hartford, CT: Connecticut Department of Public Health.
- Cumming, R.G. and R.J. Klineberg. 1993. Breastfeeding and other reproductive factors and the risk of hip fracture in elderly women. *International Journal of Epidemiology*. 22:684-691.
- Dewey, K.G., M.J. Heinig, L.A. Nommsen. 1993. Maternal weight-loss patterns during prolonged lactation. *American Journal of Clinical Nutrition*. 58:162-166.
- Duncan, B., J.Ey, C.J. Holberg, et al. 1993. Exclusive breast-feeding for at least 4 months protects against otitis media. *Pediatrics*. 91(5):867-872.
- Egeland, G.M., K.A. Perham-Hester, B.D. Gessner et al. 1998. "Fetal alcohol syndrome in Alaska, 1977 through 1992: an administrative prevalence derived from multiple data sources," *American Journal of Public Health*. 88(5):781-6.
- Elster, Nanette, and the Institute for Science, Law and Technology Working Group on Reproductive Technology, 2000. Less is more: the risks of multiple births. *Fertility & Sterility* 74(4): 617-623.
- Enger, S.M., R.K. Ross, A. Paganini-Hill et al. 1998. Breastfeeding experience and breast cancer risk among postmenopausal women. *Cancer Epidemiology, Biomarker & Prevention*. 7(5):365-369.
- Ettner, S.L., C.L. Christiansen, T.L. Callahan, et al. 1998. How low birthweight and gestational age contribute to increased inpatient costs for multiple births. *Inquiry*. 34(4): 325-39.
- Faber, K. 1997. IVF in the US: multiple gestation, economic competition, and the necessity of excess. *Human Reproduction*. 12(8): 1614-6.
- Goldenberg, R.L., W.W. Andrews. 1996. Intrauterine infection and why preterm prevention programs have failed. *American Journal of Public Health*. 86: 781-783.
- Goldenberg, R.L., S.P. Cliver, F.X. Mulvihill, et al. 1996. Medical, psychosocial, and behavioral risk factors do not explain the increased risk for low birthweight among black women. *American Journal of Obstetrics and Gynecology*. 175: 1317 -- 1324.
- Goldman, A.S. 1993. The immune system of human milk: antimicrobial, antiinflammatory and immunomodulating properties. *Pediatrics Infectious Disease Journal*. 12(8):664-672.
- Goldman, A.S., R.M. Goldblum, L.A.Hanson. 1990. Antiinflammatory systems in human milk. *Advances in Experimental Medicine and Biology*. 262:69-76.
- Guttmacher, A. 1994. The Alan Guttmacher Institute. *Sex and Americas Teenagers*. New York, NY.
- Guyer, B., M.A. Freedman, D.M. Strobino, et al. 2000. Annual summary of vital statistics: trends in the health of Americans during the 20th century, *Pediatrics*. December, 106(6): 1307-17.
- Guyer, B, M.F. MacDorman, J.A. Martin, et al. 1998. Annual summary of vital statistics-1997, *Pediatrics*. December, 102(6): 1333-49.
- Hauth, J.C., R.L. Goldenberg, W.W. Andrews, et al. 1995. Reduced incidence of preterm delivery with metronidazole and erythromycin in women with bacterial vaginosis. *New England Journal of Medicine* 333: 1732-1736.
- Howie, P.W., J.S. Forsyth, S.A. Ogston. 1990. Protective effect of breast feeding against infection. *British Medical Journal*. 300:11-16.
- Hoyert, D.L., I. Danel, P. Tully. 2000. Maternal mortality, United States and Canada, 1982-1997. *Birth*. (27)1: 4-11.

- Institute of Medicine, Committee to Study the Prevention of Low Birth Weight, 1985. *Preventing Low Birthweight*. Washington, DC: National Academy Press.
- Klerman, L. 1988. The Intervention programs prevent subsequent births to teenage mothers? Reactions to use recent surge." Paper for program on preventing second first to teenage mothers: Demonstration findings, sponsored by the American Enterprise Institute For Public Policy Research, March 6, 1998.
- Kuzela, A.L., C.A. Stifter, J. Worobey. 1990. Breastfeeding and mother-infant interactions. *Journal of Reproduction and Infant Psychology*. 8:185-194.
- Lightwood, J.M., C.S. Phibbs, S.A. Glantz. 1999. "Short-term health and economic benefits of smoking cessation: Low birth weight," *Pediatrics*, 104(6):1312-1320.
- Lundsberg, L.S., M.B. Bracken, A.F. Saftlas. 1997. "Low-to-moderate gestational alcohol use and intrauterine growth retardation, low birthweight, and preterm delivery," *Annals of Epidemiology*, 7(7):498-508.
- Mamelle, N., M. Segueilla, F. Munoz, et al. 1997. Prevention of preterm births in patients with symptoms of preterm labor -- the benefit of psychological support. *American Journal of Obstetrics and Gynecology* 177: 947-952.
- Marks, J.S., J.P. Koplan, C.J.Hogue, et al. 1990. "A cost-benefit/cost-effectiveness analysis of smoking cessation for pregnant women," *American Journal of Preventive Medicine*, 6:282-289.
- Melton, 3d, L.J, S.C. Bryant, H.W. Wahne, et al. 1993 Influence of breastfeeding and other reproductive factors on bone mass later in life. *Osteoporos International*. 3:76-83.
- Mills, J.L., B.I. Graubard, E.E. Harley, et al. 1984. "Maternal alcohol consumption and birth weight. How much drinking during pregnancy is safe?," *Journal of the American Medical Association*. 252(14):1875-9.
- Mothers Survey. 1998. Ross Products Division, Abbott Laboratories.
- Mueller, L.M. 2001a. Connecticut Birth data 1988-1998. Unpublished data.
- Mueller, L.M. 2001b. *Connecticut Behavioral Risk Factor Surveillance System, 1999*. Unpublished data.
- National Center for Health Statistics (NCHS). 1997. *Healthy People 2000 Review*, Hyattsville, MD, Public Health service.
- National Center for Health Statistics (NCHS). 1999. Births: final data for 1997. *National Vital Statistics Reports*. 47 (18): 10-11.
- Newcomb, P.A., K.M. Egan, L. Titus-Ernstoff, et al. 1999. Lactation in relation to postmenopausal breast cancer. *American Journal of Epidemiology*. 150(2):174-182.
- Office of Technology Assessment, US Congress, 1988. *Healthy Children: Investing in the Future*. Washington, DC: US Congress, Office of Technology Assessment.
- Pollack, H., P. Lantz, J. Frohna. 2000. "Maternal smoking and adverse birth outcomes among singletons and twins," *American Journal of Public Health*. 90(3): 395-400.
- Rosenblatt, K.A., D.B. Thomas, and the WHO Collaborative Study of Neoplasia and Steroid Contraceptives. 1993. Lactation and the risk of epithelial ovarian cancer. *International Journal of Epidemiology*. 22(2):192-197.
- Rothberg, A.D., B. Lits. 1991. The psychosocial support for maternal stress during pregnancy: Effect on birthweight. *American Journal of Obstetrics and Gynecology* 165: 403-407.
- Scariati, P.D., L.M. Grummer-Strawn, S.B. Fein. 1997. A longitudinal analysis of infant morbidity and the extent of breastfeeding in the United States. *Pediatrics*. 99(6):5.
- Shu, X.O., M.C. Hatch, J. Mills, et al. 1995. "Maternal smoking, alcohol drinking, caffeine consumption, and fetal growth: results from a prospective study," *Epidemiology*. 6(2):115-20.
- Teitelman, A.M., L.S. Welch, K.G. Hellenbrand, et al. 1990. Effect of maternal work activity on preterm births and low birthweight. *American Journal of the Epidemiology* 131: 104-113.
- U.S. Department of Health and Human Services, 1991. *Healthy People 2000: National Health Promotion and Disease Prevention Objectives*. Washington, D.C.: U.S. Government Printing Office.
- U.S. Department of Health and Human Services, 1999. The Healthy People 2010 objectives for Proving pregnancy planning and spacing. *Family Planning* (Chapter 9). Washington, D.C.: U.S. Government Printing Office.
- U.S. Department of Health and Human Services, 2000. *HHS Blueprint for Action on Breastfeeding*. Washington, D.C.: USDHHS Office on Women's Health, 2000.

- U.S. Department of Health and Human Services, 2000. *Healthy People 2010*. 2nd ed. 2 vols. Washington, D.C.: U.S. Government Printing Office.
- U.S. Public Health Service. 2000. *Healthy People 2010*. Washington, D.C.: Department of Health and Human Services, Public Health Service.
- Viriden, S.F. 1988. The relationship between infant feeding method and maternal role adjustment. *Journal of Nurse-Midwifery*. 33(1):31-35.
- Zhu, B., R. Rolfs, et al. 1999. Effects of interval between pregnancies on perinatal outcomes. *New England Journal of Medicine* 340 (8): 589-594, 1999.

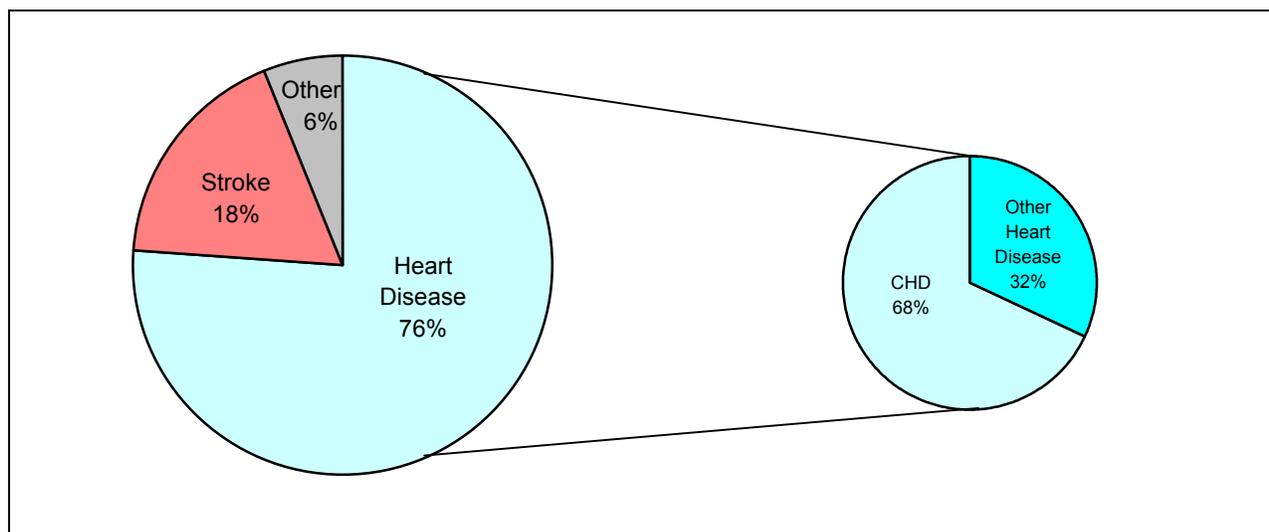
## 6. CORONARY HEART DISEASE

### 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.

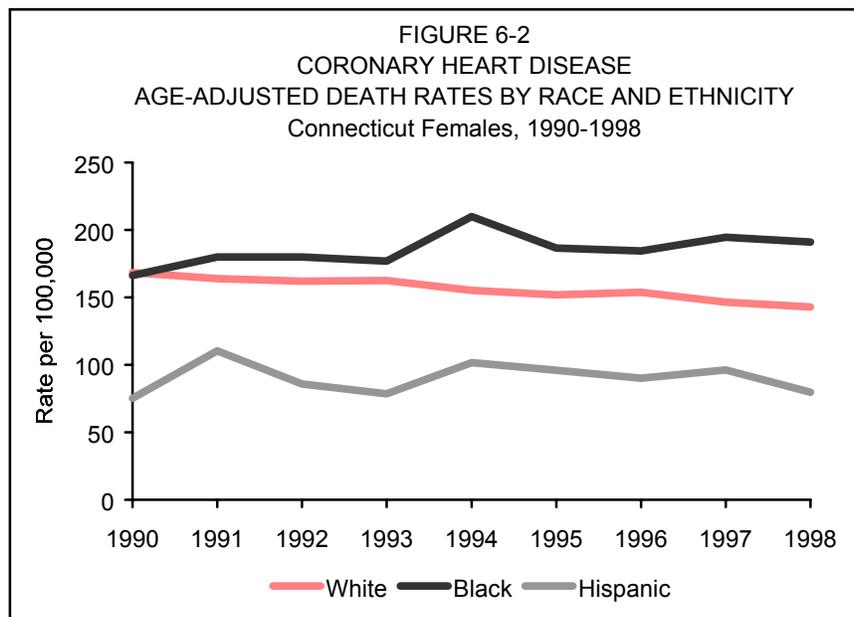
## SCOPE OF THE PROBLEM

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 <sup>‡</sup>
Asian/Pacific Islander	10	†	22	46.8 <sup>‡</sup>
Native American	13	†	2	†
Hispanic/Latina	118	97.6	157	88.2 <sup>‡</sup>

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  $> 27.3$ ).<sup>1</sup>

<sup>1</sup> 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.

## REFERENCES

- Abraido-Lanza A.F., B.P. Dohrenwend, D.S. Ng-Mak, J.B. Turner. 1999. The Latino mortality paradox: a test of the "salmon bias" and healthy migrant hypotheses. *American Journal of Public Health* 89(10):1543-1548.
- American Heart Association. 1996. *1997 Heart and Stroke Facts: Statistical Update*. Dallas, Texas: American Heart Association.

- Appel L.J., E.R. Miller, S.H. Jee, R Stolzenberg-Solomon, P. Lin, T. Erlinger, M.R. Nadeau, J. Selhub. 2000. Effect of dietary patterns on serum homocysteine. *Circulation* 102:852.
- Barrett-Connor E., N.K. Wenger, D. Grady, et al. 1998. Hormone and nonhormone therapy for the maintenance of postmenopausal health: the need for randomized controlled trials of estrogen and raloxifene. *Journal of Womens Health* 7:839-847.
- Bickell N.A., K.S. Pieper, K.L Lee, et al. 1992. Referral patterns for coronary artery disease treatment: gender bias or good clinical judgment? *Annals of Internal Medicine* 116:791-797.
- Bittner V. 2000. Heart disease in women. *Clinical Reviews*: 62-66.
- Bittner V. 1994. Cardiovascular disease in women. *Journal of Women's Health* 3:369-376.
- Centers for Disease Control and Prevention. 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.
- Connecticut Department of Public Health, Division of Policy, Planning, and Analysis. 2001. Unpublished data (see *Appendix D*).
- Fuchs F.C., M.J. Stampfer, G.A. Colditz, E.L. Giovannucci, J.E. Manson, I. Kawachi, D.J. Hunter, P.E. Hankinson, C.H. Hennekens, B. Rosner, F.E. Speizer, W.C. Willett. 1995. A prospective study of alcohol consumption and mortality among women. *New England Journal of Medicine* 332:1245-1250.
- Hemingway H., M. Marmot. 1999. Psychosocial factors in the etiology and prognosis of coronary heart disease: systematic review of prospective cohort studies. *British Medical Journal* 318:1460-1467.
- Hulley S., D. Grady, T. Bush, et al. 1998. Randomized trial of estrogen plus progestin for secondary prevention of coronary heart disease in postmenopausal women. *Journal of the American Medical Association* 280:605-613.
- Kudenchuk P., C. Maynard, J. Martin, M. Wirkus, W.D. Weaver. 1996. Comparison of presentation, treatment, and outcome of acute myocardial infarction in men versus women (the Myocardial Infarction Triage and Intervention Registry). *American Journal of Cardiology* 78:9-14.
- Love R.R., D.A. Wiebe, P.A. Newcomb, et al. 1991. Effects of tamoxifen on cardiovascular risk factors in postmenopausal women. *Annals of Internal Medicine* 115:860-864.
- Manson J.E., A. Spelsberg. 1996. Risk modification in the diabetic patient. In: Manson J.E., Ridker P.M., Gaziano J.M. (eds). *Prevention of Myocardial Infarction*. New York, NY: Oxford University Press; 241-273.
- Matthews K.A., L.H. Kuller, R.R. Wing, E.N. Meilahn, P. Plantinga. 1996. Prior to use of estrogen replacement therapy, are users healthier than nonusers? *American Journal of Epidemiology* 143:971-978.
- Mosca L., P. Collins, D. Herrington, et al. 2001. Hormone replacement therapy and cardiovascular disease: a statement for healthcare professionals from the American Heart Association. *Circulation* 104:499-503.
- 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 (see *Appendix C*).
- Oberman, A. Spring 2000. Role of lipids in the prevention of cardiovascular disease. *Clinical Reviews*: 10-15.
- Rich-Edwards J.W., J.E. Manson, C.H. Hennekens, J.E. Buring. 1995. The primary prevention of coronary heart disease in women. *New England Journal of Medicine* 332:1758-1766.
- Rosenberg H.M., J.D. Maurer, P.D. Sorlie, N.J. Johnson, M.F. MacDorman, D.L. Hoyert, J.F. Spitler, C. Scott. 1999. Quality of death rates by race and hispanic origin: a summary of current research, 1999. National Center for Health Statistics. *Vital Health Stat* 2(128).
- Stampfer M.J., F.B. Hu, J.E. Manson, E.B. Rimm, W.C. Willett. 2000. Primary prevention of coronary heart disease in women through diet and lifestyle. *New England Journal of Medicine* 343:16-22.

- Thoma M.S., D.B. Diercks, C.S. Hampikian, J.D. Kirk. 2001. Gender and clinical presentation influence aspirin therapy in patients with acute myocardial infarction. *Academic Emergency Medicine* 8(5):556.
- Thomas R.J., N.H. Miller, C. Lamendola, K. Berra, B. Hedback, J.L. Durstin, W. Haskell. 1996. National survey on gender differences in cardiac rehabilitation programs: patient characteristics and enrollment patterns. *Journal of Cardiopulmonary Rehabilitation* 16:402-412.
- Walsh B.W., L.H. Kuller, R.A. Wild, et al. 1998. Effects of raloxifene on serum lipids and coagulation factors in healthy postmenopausal women. *Journal of the American Medical Association* 279:1445-1451.
- Whelton P.K., J. He, L.J. Appel. 1996. Treatment and prevention of hypertension. In: Manson J.E., P.M. Ridker, J.M. Gaziano, C.H. Hennekens, eds. *Prevention of Myocardial Infarction*. New York, NY: Oxford University Press 154-171.
- Willett W.C., A. Green, M.J. Stampfer, F.E. Speizer, G.A. Colditz, B. Rosner, R.R. Monson, W. Stason, C.H. Hennekens. 1987. Relative and absolute excess risks of coronary heart disease among women who smoke cigarettes. *New England Journal of Medicine* 317:1303-1309.
- Willett W.C., E.B. Lenart. 1996. Dietary factors. In: Manson J.E., P.M. Ridker, J.M. Gaziano, C.H. Hennekens, eds. *Prevention of Myocardial Infarction*. New York, NY: Oxford University Press 351-383.
- Willett W.C., M.J. Stampfer, J.E. Manson, G.A. Colditz, F.E. Speizer, B.A. Rosner, L.A. Sampson, C.H. Hennekens. 1993. Intake of trans-fatty acid and risk of coronary heart disease among women. *Lancet* 341:581-585.
- Yusuf S., R. Peto, J. Lewis, R. Collins, P. Sleight. 1985. Beta blockage during and after myocardial infarction: an overview of the randomized trials. *Progress in Cardiovascular Diseases* 27:335-371.

## 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.

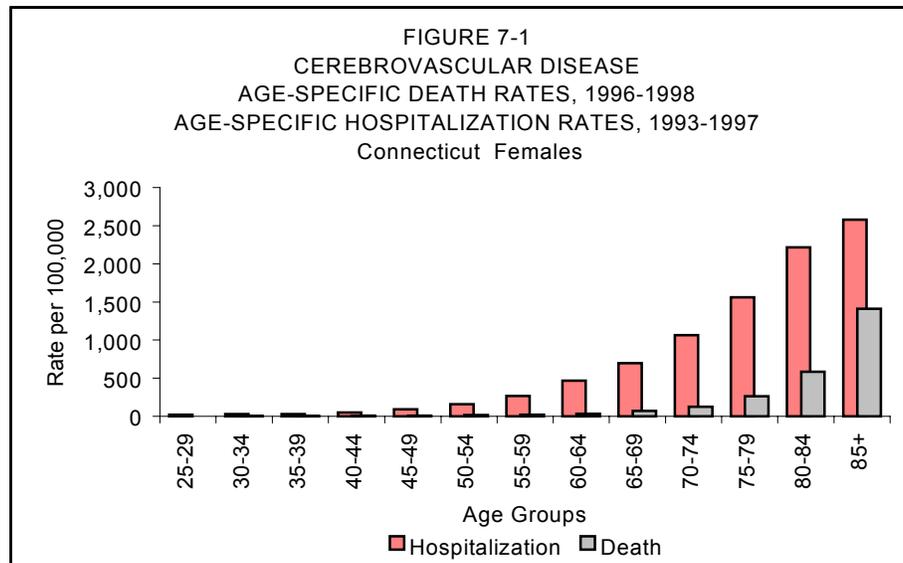
There has not been a significant change in mortality rates over the last decade for cerebrovascular disease in Connecticut females. The mortality rate from 1989 to 1991 was 51 per 100,000 females and was only slightly lower at 50 per 100,000 from 1996 to 1998. However, over the five-year period from 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 <sup>†</sup>
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.

<sup>†</sup> Statistics not calculated for fewer than 15 events.

<sup>‡</sup> 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

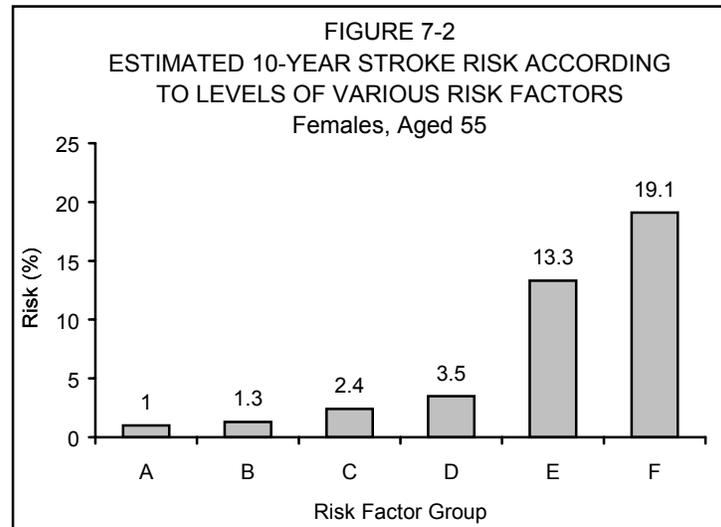
men (Wolf et al., 1991). Other types of cardiac disease that increase the risk of stroke are heart attack (also associated with atrial fibrillation), congestive heart failure, valvular heart disease, and intracardiac congenital defects (Benjamin et al., 1998). Based on the Framingham Heart Study, 11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

## REFERENCES

- Benjamin E.J., P.A. Wolf, R.B. D'Agostino, et al. 1998. Impact of atrial fibrillation on the risk of death: the Framingham Heart Study. *Circulation* 98:946-952.
- Bonita R., J. Duncan, T. Truelsen, et al. 1999. Passive smoking as well as active smoking increases the risk of acute stroke. *Tobacco Control* 8:156-160.
- Connecticut Department of Public Health, Division of Policy, Planning, and Analysis. 2001. Unpublished data (see *Appendix D*).
- Goldstein, L.B., R. Adams, K. Becker, et al. 2001. Primary prevention of ischemic stroke: a statement for healthcare professional from the stroke council of the American Heart Association. *Circulation* 103:163-182.
- Hu, F.B., M. Stampfer, G. Colditz, et al. 2000. Physical activity and risk of stroke in women. *Journal of the American Medical Association* 283:2961-2967.
- Hart R.G., O. Benavente, R. McBride, L.A. Pearce. 1999. Antithrombotic therapy to prevent stroke in patients with atrial fibrillation: a meta-analysis. *Annals of Internal Medicine* 131(7):492-501.
- Heart Outcomes Prevention Evaluation Study Investigators. 2000. Effects of ramipril on cardiovascular and microvascular outcomes in people with diabetes mellitus: results of the HOPE study and MICRO-HOPE substudy. *Lancet* 355:253-259.
- Kawachi I., G.A. Colditz, M.J. Stampfer, et al. 1993. Smoking cessation and decreased risk of stroke in women. *Journal of the American Medical Association* 269:232-236.
- MacMahon S., R. Peto, J. Cutler, et al. 1990. Prolonged differences in blood pressure: prospective observational studies corrected for the regression dilution bias. *Lancet* 335:765-774.
- 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 (see *Appendix C*).
- Sacco, R.L., E.J. Benjamin, J.P. Broderick, et al. 1997. Risk factors. *Stroke* 28:1507-1517.
- Stamler J., P. Greenland, J.D. Neaton. 1998. The established major risk factors underlying epidemic coronary and cardiovascular disease. *CVD Prevention* 1:82-97.
- Whisnant, J.P. 1996. Effectiveness versus efficacy of treatment of hypertension for stroke prevention. *Neurology* 46:301-307.
- Wolf P.A., R.B. D'Agostino, A.J. Belanger, W.B. Kannel. 1991. Probability of stroke: a risk profile from the Framingham Study. *Stroke* 22:312-318.
- Yusuf H.R., W.H. Giles, J.B. Croft, R.F. Anda, M.L. Casper. 1998. Impact of multiple risk factor profiles on determining cardiovascular risk. *Preventive Medicine* 27:1-9.

## 8. LUNG CANCER

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### 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 6<sup>th</sup> highest of the 8 states listed for the prevalence of ever smoking, and 7<sup>th</sup> 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).

### REFERENCES

- Adams, M.L. 2000. *Connecticut Behavioral Health Risks: Factors Related to Cancer*. Hartford, CT: Connecticut Department of Public Health.
- Anderson, C., and D.M. Burns. 2000. Patterns of adolescent smoking initiation rates by ethnicity and sex. *Tobacco Control* 9 (Suppl 2):II4-8.
- Au, W.W., H.Y. Oh, J. Grady, S.A. Salama, and M.Y. Heo. 2001. Usefulness of genetic susceptibility and biomarkers for evaluation of environmental health risk. *Environmental and Molecular Mutagenesis* 37:215-25.
- Bower, C.E. (Ed.). 1995. *Connecticut Health 1994: Selected Data Concerning Public Health Programs in Connecticut and the Health of Connecticut's Residents*. Hartford, CT: Connecticut Department of Public Health and Addiction Services. 128 pp.
- Brownson, R.C., P.L. Remington, and J.R. Davis, Eds. 1998. *Chronic Disease Epidemiology and Control, 2nd Edition*. Washington, DC: American Public Health Association.
- Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Behavioral Surveillance Branch. 1999 and 2000. *Behavioral Risk Factor Surveillance System: Connecticut Statewide Survey Data, Weighted*. Atlanta, GA: Centers for Disease Control and Prevention.
- Connecticut Department of Public Health, Division of Policy, Planning, and Analysis. 2001. Unpublished data (see *Appendix D*).
- Connecticut Tumor Registry. 1999. *Cancer Incidence in Connecticut, 1997*. Hartford, CT: Connecticut Department of Public Health.
- Connecticut Tumor Registry. 2001. *Cancer Incidence in Connecticut, 1980-1998*. Hartford, CT: Connecticut Department of Public Health.
- Forgacs, E., S. Zochbauer-Muller, E. Olah, and J.D. Minna. 2001. Molecular genetic abnormalities in the pathogenesis of human lung cancer. *Pathology Oncology Research* 7:6-13.
- Holford, T.R., Z. Zhang, T. Zheng, and L.A. McKay. 1996. A model for the effect of cigarette smoking on lung cancer incidence in Connecticut. *Statistics in Medicine* 15:565-80.

- Humphrey, E.W., H.B. Ward, and R.T. Perri. 1995. Lung cancer. In: G.P. Murphy, W. Lawrence, Jr., and R.E. Lenhard, Jr., eds. *American Cancer Society textbook of clinical oncology*, 2<sup>nd</sup> ed. Atlanta: American Cancer Society, 220-35.
- Kann, L., S.A. Kinchen, B.I. Williams, et al. 2000. Youth Risk Behavior Surveillance --United States, 1999. In: CDC Surveillance Summaries, June 9, 2000. *Morbidity and Mortality Weekly Report* 49(SS-5), 96 pp.
- Krieger, N., C. Quesenberry, Jr., T. Peng, et al. 1999. Social class, race/ethnicity, and incidence of breast, cervix, colon, lung, and prostate cancer among Asian, Black, Hispanic, and White residents of the San Francisco Bay Area, 1988-92 (United States). *Cancer Causes and Control* 10:525-37.
- Melloni, B., A. Vergnenegre, P. Lagrange, and F. Bonnaud. 2000. Radon and domestic exposure (French with English abstract). *Revue des Maladies Respiratoires* 17:1061-71.
- 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 (see Appendix C).
- National Cancer Institute. 2001. <http://www.cancerNet.nci.nih.gov> (31 July 2001).
- National Research Council. 1999. Health effects of exposure to radon: BEIR VI, Committee on Health Risks of Exposure to Radon (BEIR VI). *National Academy Press*.
- Polednak, A.P. 1994. Trends in cancer incidence in Connecticut, 1935-1991. *Cancer* 74:2863-72.
- Ries, L.A.G., C.L. Kosary, B.F. Hankey, et al. 1997. *SEER Cancer Statistics Review, 1973-1994*. Bethesda, MD: National Cancer Institute, NIH Publ. No. 97-2789.
- Ries, L.A.G., M.P. Eisner, C.L. Kosary, et al., eds. 2001. *SEER Cancer Statistics Review, 1973-1998*. Bethesda, MD: National Cancer Institute. [http://seer.cancer.gov/publications/csr1973\\_1998](http://seer.cancer.gov/publications/csr1973_1998).
- Satcher, D. 2001. Why we need an international agreement on tobacco control. *American Journal of Public Health* 91:191-3.
- Sly, D.F., R.S. Hopkins, E. Trapido, and S. Ray. 2001. Influence of a counteradvertising media campaign on initiation of smoking: the Florida "truth" campaign. *American Journal of Public Health* 91:233-8.
- Tockman, M.S. 2001. Lung cancer: chemoprevention and intermediate effect markers. *IARC Scientific Publications* 154:257-70.

## 9. BREAST CANCER

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### 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).

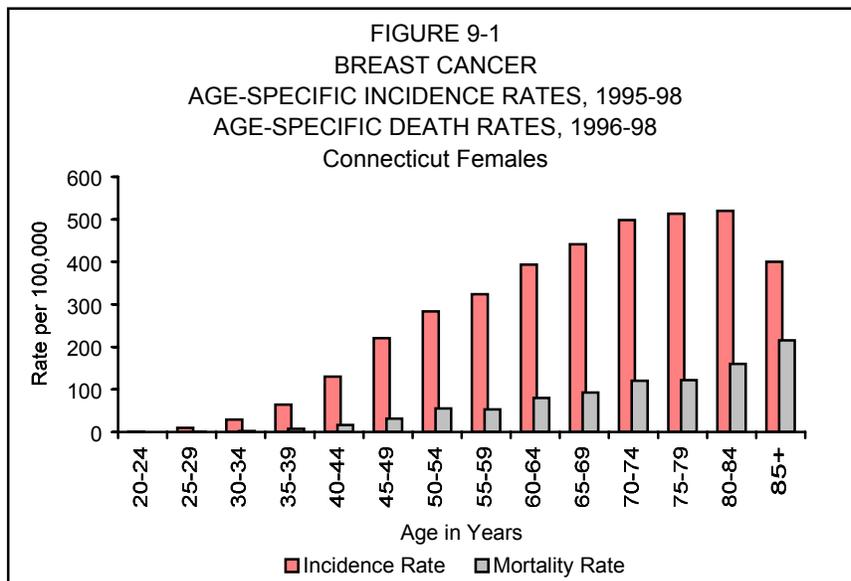
Between 1996 and 1998, 1,796 Connecticut women died of breast cancer (age-adjusted mortality rate 30.0 per 100,000 women per year).

#### *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 12<sup>th</sup> 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 <sup>‡</sup>
Asian/Pacific Islander	1	†	5	†
Native American	1	†	1	†
Hispanic/Latina	16	9.3	33	13.2 <sup>‡</sup>

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 8<sup>th</sup> highest for women age 40 and over who never had mammography, and 7<sup>th</sup> 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).

## REFERENCES

- Adams, M.A. 2000. *Connecticut Behavioral Health Risks: Factors Related to Cancer*. Hartford, CT: CT Department of Public Health.
- Barlow, W.E., S.H. Taplin, C.K. Yishida, D.S. Buist, D. Seger, and M.Brown. 2001. Cost comparison of mastectomy versus breast-conserving therapy for early stage breast cancer. *Journal of the National Cancer Institute* 93:447-55.
- Bonfill, X., M. Marzo, M. Pladevall, J. Marti, and J.I. Emparanza. 2001. Strategies for increasing women participation in community breast cancer screening. *Cochrane Database of Systematic Reviews* 1:CD002943.
- Brownson, R.C., P.L. Remington, and J.R. Davis, Eds. 1998. *Chronic Disease Epidemiology and Control, 2nd Edition*. Washington, DC: American Public Health Association.
- Centers for Disease Control and Prevention. 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.
- Connecticut Department of Public Health, Breast and Cervical Cancer Early Detection Program. 1999. *Breast Cancer in Connecticut: A Handbook for Health Care Providers*. Hartford, CT: CT Department of Public Health.
- Connecticut Department of Public Health, Division of Policy, Planning, and Analysis. 2001. Unpublished data (see *Appendix D*).
- Connecticut Tumor Registry. 1999a. *Connecticut Tumor Registry*. Hartford, CT: Connecticut Department of Public Health.
- Connecticut Tumor Registry. 1999b. *Cancer Incidence in Connecticut, 1997*. Hartford, CT: Connecticut Department of Public Health.
- Connecticut Tumor Registry. 2000. *Cancer Incidence in Connecticut, 1998*. Hartford, CT: Connecticut Department of Public Health.
- Connecticut Tumor Registry. 2001a. *Cancer Incidence in Connecticut, 1980-1998*. Hartford, CT: Connecticut Department of Public Health.
- Connecticut Tumor Registry, 2001b. Hartford, CT: Connecticut Department of Public Health. Unpublished data.
- Cummings, S.R., S. Eckert, K.A. Dureger, et al. 1999. The effect of raloxifene on risk of breast cancer in postmenopausal women: results from the MORE randomized trial. Multiple Outcomes of Raloxifene Evaluation. *Journal of the American Medical Association* 281:2189-2197.
- Dravet, F., J. Belloin, P.F. Dupre, et al. 2000. (French.) Role of outpatient surgery in breast surgery. Prospective feasibility study. *Annales de Chirurgie* 125:668-76.
- Evans, W.K., B.P. Will, J.M. Berthelot, D.M. Logan, D.J. Mirsky, N. Kelly. 2000. Breast cancer: better care for less cost. Is it possible? *International Journal of Technology Assessment in Health Care* 16:1168-78.
- Ferrante, J., E. Gonzalez, N. Pal, and R. Roetzheim. 2000. The use and outcomes of outpatient mastectomy in Florida. *American Journal of Surgery* 179:253-9.
- Fink, D.J., and C.J. Mettlin. 1996. Cancer detection: the cancer-related checkup guidelines. In: G.P. Murphy, W. Lawrence, Jr., and R.E. Lenhard, Jr., eds. *American Cancer Society textbook of clinical oncology*, 2<sup>nd</sup> ed. Atlanta: American Cancer Society, 178-93.
- Fisher B., J.P. Constantino, L. Wickerham, et al. 1998. Tamoxifen for prevention of breast cancer: report of the National Surgical Adjuvant Breast and Bowel Project P-1 Study. *Journal of the National Cancer Institute* 90:1371-88.
- Galante, E., A.M. Cerrotta, and A. Crippa. 1994. Outpatient treatment of clinically node-negative breast cancer in elderly women. *Cancer Control* 1:344-9.
- Henderson, I.C. 1995. Breast cancer. In: G.P. Murphy, W. Lawrence, Jr., and R.E. Lenhard, Jr., eds. *American Cancer Society textbook of clinical oncology*, 2<sup>nd</sup> ed. Atlanta: American Cancer Society, 198-219.
- Henderson, B.E., M.C. Pike, L. Bernstein, and R.K. Ross. 1996. Breast cancer. In: D. Schottenfeld, and J.F. Fraumeni, Jr., eds. *Cancer epidemiology and prevention*, 2<sup>nd</sup> ed. New York: Oxford Univ. Press, 1022-39.
- Howe, H.L., P.A. Wingo, M.J. Thun, et al. 2001. Annual report to the nation on the status of cancer (1973 through 1998), featuring cancers with recent increasing trends. *Journal of the National Cancer Institute* 93:824-42.

- Janerich, D.T. 1994. The fetal antigen hypothesis for breast cancer, revisited. *Medical Hypotheses* 43:105-10.
- Jones, B.A., S.V. Kasl, M.G.M. Curnen, P.H. Owens, and R. Dubrow. 1997. Severe obesity as an explanatory factor for the black/white difference in stage at diagnosis of breast cancer. *American Journal of Epidemiology* 146:394-404.
- Krieger N., C. Quesenberry, Jr., T. Peng, et al. 1999. Social class, race/ethnicity, and incidence of breast, cervix, colon, lung, and prostate cancer among Asian, Black, Hispanic, and White residents of the San Francisco Bay Area, 1988-92 (United States). *Cancer Causes and Control* 10:525-37.
- Lambe, M., H. Chung-cheng, D. Trichopoulos, A. Ekblom, M. Pavia, and H. Adami. 1994. Transient increase in the risk of breast cancer after giving birth. *New England Journal of Medicine* 331:5-9.
- Lawson, H.W., R. Henson, J.K. Bobo, and M.K. Kaeser. 2000. Implementing recommendations for the early detection of breast and cervical cancer among low-income women. *Morbidity and Mortality Weekly Report* 49(RR02):35-55.
- Margolese, R.G., and J.C. Lasry. 2000. Ambulatory surgery for breast cancer patients. *Annals of Surgical Oncology* 7:181-7.
- McCarthy, E.P., R.B. Burns, K.M. Freund, et al. 2000. Mammography use, breast cancer stage at diagnosis, and survival among older women. *Journal of the American Geriatrics Society* 48:1226-33.
- McCooey, L., P.P. Mitchell, C.B. Parker, and J.Simpson. 1999. A comprehensive breast and cervical cancer screening program for medically underserved women in Connecticut. *Connecticut Medicine* 63: 17-21.
- Mitchell, P. 2001. Connecticut Department of Public Health, Connecticut Breast and Cervical Cancer Early Detection Program. Unpublished data.
- 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 (see Appendix C).
- National Cancer Institute. 2001. <http://www.cancerNet.nci.nih.gov> (31 July 2001).
- Newman, B., R.C. Millikan, and M.C. King. 1997. Genetic epidemiology of breast and ovarian cancers. *Epidemiologic Reviews* 19:69-79.
- Parker, R.G., K.M. Leung, K.S. Rees, L.W. Bassett, and A.P. Legorreta. 1999. Mammographic screening downstages breast carcinoma at time of diagnosis: a community-based experience. *Breast Journal* 5:359-63.
- Polednak, A.P. 1999. Epidemiology of breast cancer in Connecticut women. *Connecticut Medicine* 63:7-16.
- Polednak, A.P. 2000a. Later-stage cancer in relation to medically underserved areas in Connecticut. *Journal of Health Care for the Poor and Underserved* 11:301-9.
- Polednak, A.P. 2000b. Trends in late-stage breast, cervical and colorectal cancers in blacks and whites. *Ethnicity and Disease* 10:60-8.
- Ries, L.A.G., C.L. Kosary, B.F. Hankey, et al. 1997. *SEER Cancer Statistics Review, 1973-1994*. Bethesda, MD: National Cancer Institute, NIH Publ. No. 97-2789.
- Ries, L.A.G., M.P. Eisner, C.L. Kosary, et al., eds. 2001. *SEER Cancer Statistics Review, 1973-1998*. Bethesda, MD: National Cancer Institute. [http://seer.cancer.gov/publications/csr1973\\_1998](http://seer.cancer.gov/publications/csr1973_1998)
- Rockhill, B. 2001. The privatization of risk. *American Journal of Public Health* 91:365-8.
- Schairer, C., J. Lubin, R. Troisi, et al. 2000. Menopausal estrogen and estrogen-progestin replacement therapy and breast cancer risk. *Journal of the American Medical Association* 283:485-91.
- Tabar L., B. Vitak, H.H. Tony, M.F. Yen, S.W. Duffy, and R.A. Smith. 2001. Beyond randomized controlled trials: organized mammographic screening substantially reduces breast carcinoma mortality. *Cancer* 91:1724-31.
- Walter, L.C., and K.E. Covinsky. 2001. Cancer screening in elderly patients: a framework for individualized decision making. *Journal of the American Medical Association* 285:2750-6.
- Willett, W.C., G. Colditz, and M. Stampfer. 2000. Postmenopausal estrogens—opposed, unopposed, or none of the above. *Journal of the American Medical Association* 283:534-5.
- Zheng, T., T.R. Holford, Y. Chen, B.A. Jones, J. Flannery, and P. Boyle. 1997. Time trend of female breast carcinoma in situ by race and histology in Connecticut, U.S.A. *European Journal of Cancer* 33:96-100.

## 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)

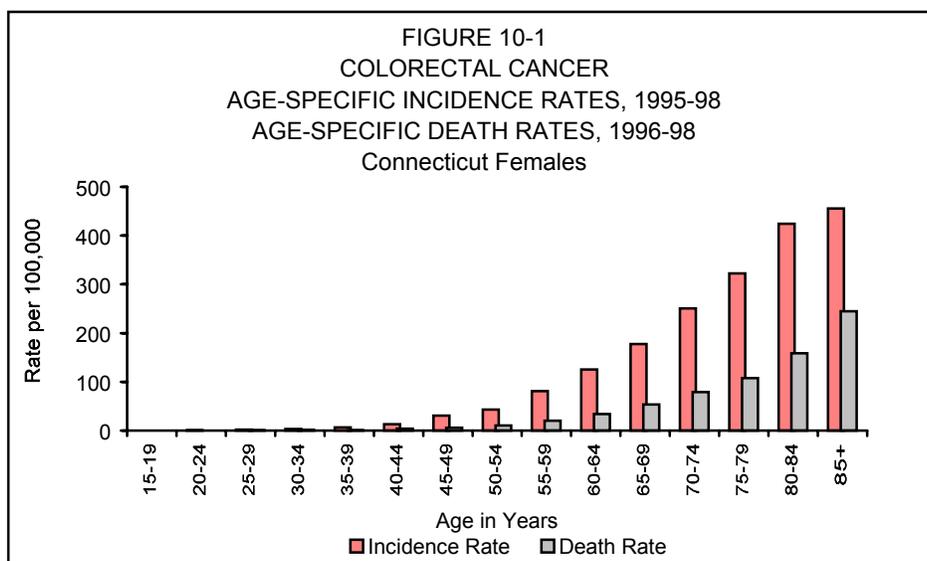
Age

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 5<sup>th</sup> 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 5<sup>th</sup> 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 7<sup>th</sup> (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 6<sup>th</sup> 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).

## REFERENCES

- 10 Steps: Implementation Guide. 1998. Put Prevention into Practice. Adapted from *The Clinician’s Handbook of Preventive Services*, 2<sup>nd</sup> Edition, Publication No. APPIP 98-0025. Agency for Healthcare Research and Quality, Rockville, MD. A 3<sup>rd</sup> edition of the handbook (2000-2002) is now available. <http://www.ahrq.gov/ppip/impsteps.htm>
- Adams, M.A. 2000. *Connecticut Behavioral Health Risks: Factors Related to Cancer*. Hartford, CT: CT Department of Public Health.

- American Cancer Society. 2000. *Selected Cancer Facts & Figures 1999-2000: Colon and Rectum*. [http://www.cancer.org/statistics/cff2000/selected\\_cancers.html](http://www.cancer.org/statistics/cff2000/selected_cancers.html) (22 March 2000).
- Baquet, C.R., and P. Commiskey. 1999. Colorectal cancer epidemiology in minorities: a review. *Journal of the Association for Academic Minority Physicians* 10:51-8.
- Borras, J.M., A. Sanches-Hernandez, M. Navarro, et al. 2001. Compliance, satisfaction, and quality of life of patients with colorectal cancer receiving home chemotherapy or outpatient treatment: a randomized controlled trial. *British Medical Journal* 322:826.
- Borum, M.L. 1999. Colorectal cancer surveillance in African-American and white patients at an urban university medical center. *Journal of the National Medical Association* 91:505-8.
- Brownson, R.C., P.L. Remington, and J.R. Davis, Eds. 1998. *Chronic Disease Epidemiology and Control, 2nd Edition*. Washington, DC: American Public Health Association.
- Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Behavioral Surveillance Branch, 1999. *Behavioral Risk Factor Surveillance System*. Atlanta: Centers for Disease Control and Prevention.
- Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Behavioral Surveillance Branch, 2000. *Behavioral Risk Factor Surveillance System*. Atlanta: Centers for Disease Control and Prevention.
- Connecticut Department of Public Health, Division of Policy, Planning, and Analysis. 2001. Unpublished data (see *Appendix D*).
- Connecticut Tumor Registry. 1999a. *Connecticut Tumor Registry*. Hartford, CT: CT Department of Public Health.
- Connecticut Tumor Registry. 1999b. *Cancer Incidence in Connecticut, 1997*. Hartford, CT: CT Department of Public Health.
- Connecticut Tumor Registry. 2001. *Cancer Incidence in Connecticut, 1980-1998*. Hartford, CT: Connecticut Department of Public Health.
- Cress, R.D., C.R. Morris, and B.M. Wolfe. 2000. Cancer of the colon and rectum in California: trends in incidence by race/ethnicity, stage, and subsite. *Preventive Medicine* 31:447-53.
- Efficacy of Interventions to Modify Dietary Behavior Related to Cancer Risk*. 2000. Summary, Evidence Report/Technology Assessment: Number 25. AHRQ Publication No. 01-E028. Agency for Healthcare Research and Quality, Rockville, MD. A final report no. 01-E029 is expected in 2001. <http://www.ahrq.gov/clinic/dietsumm.htm>.
- Elston Lafata, J., C. Cole Johnson, T. Ben-Menachem, and R.J. Morlock. 2001. Sociodemographic differences in the receipt of colorectal cancer surveillance care following treatment with curative intent. *Medical Care* 39:361-72.
- Fleming, I.D., L.W. Brady, G.B. Mieszkalski, M.R. Cooper, and M.R. Cooper. 1995. Basis for current therapies for cancer. In: G.P. Murphy, W. Lawrence, Jr., and R.E. Lenhard, Jr., eds. *American Cancer Society textbook of clinical oncology*, 2<sup>nd</sup> ed. Atlanta: American Cancer Society, 96-134.
- Gonzalez, E.C., R.G. Roetzheim, J.M. Ferrante, and R. Campbell. 2001. Predictors of proximal vs. distal colorectal cancers. *Diseases of the Colon and Rectum* 44:251-8.
- Holt, P.R. 1999. Dairy foods and prevention of colon cancer: human studies. *Journal of the American College of Nutrition* 18:5 Suppl., 379S-391S.
- Inciardi, J.F., J.G. Lee, and T. Stijnen. 2000. Incidence trends for colorectal cancer in California: implications for current screening practices. *American Journal of Medicine* 109:277-81.
- Krieger, N., C. Quesenberry, Jr., T. Peng, et al. 1999. Social class, race/ethnicity, and incidence of breast, cervix, colon, lung, and prostate cancer among Asian, Black, Hispanic, and White residents of the San Francisco Bay Area, 1988-92 (United States). *Cancer Causes and Control* 10:525-37.
- Management of Cancer Pain*. 2001. Summary, Evidence Report/Technology Assessment: Number 35. AHRQ Publication no. 01-E033. Agency for Healthcare Research and Quality, Rockville, MD. <http://www.ahrq.gov/clinical/canpainsum.htm>
- Manne, U., H.L. Weiss, and W.E. Grizzle. 2000. Racial differences in the prognostic usefulness of MUC1 and MUC2 in colorectal adenocarcinomas. *Clinical Cancer Research* 6:4017-25.
- Marcella, S., and J.E. Miller. 2001. Racial differences in colorectal cancer mortality. The importance of stage and socioeconomic status. *Journal of Clinical Epidemiology* 54:359-66.

- McMahon, L.F., Jr., R.A. Wolfe, S. Huang, P. Tedeschi, W. Manning, Jr., and M.J. Edlund. 1999. Racial and gender variation in use of diagnostic colonic procedures in the Michigan Medicare population. *Medical Care* 37:712-7.
- Medicare. 2001. [http://www.medicare.gov/publications/pubs/pdf/p\\_revent.pdf](http://www.medicare.gov/publications/pubs/pdf/p_revent.pdf) (6 August 2001).
- Michels, K.B., et al. 2000. Prospective study of fruit and vegetable consumption and incidence of colon and rectal cancers. *Journal of the National Cancer Institute* 92: 1740-1752.
- 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 (see Appendix C).
- Murphy, T.K., E.E. Calle, C. Rodriguez, H.S. Khan, and M.J. Thun. 2000. Body mass index and colon cancer mortality in a large prospective study. *American Journal of Epidemiology* 152:847-54.
- National Cancer Institute. 2001. <http://www.cancerNet.nci.nih.gov> (31 July 2001).
- Nelson, R.L., V. Persk, and M. Turyk. 1999. Determination of factors responsible for the declining incidence of colorectal cancer. *Diseases of the Colon and Rectum* 42:741-52.
- Pace, B., and R.M. Glass. 2000. Colon cancer. *Journal of the American Medical Association* 284: 3086.
- Polednak, A.P. 2000. Inpatient hospital admission through an emergency department in relation to stage at diagnosis of colorectal cancer. *Cancer Detection and Prevention* 24:283-9.
- Rex, D.K., A.M. Khan, P. Shah, J. Newton, and O.W. Cummings. 2000. Screening colonoscopy in asymptomatic average-risk African Americans. *Gastrointestinal Endoscopy* 51:624-7.
- Ries, L.A.G., C.L. Kosary, B.F. Hankey, et al. 1997. *SEER Cancer Statistics Review, 1973-1994*. Bethesda, MD: National Cancer Institute, NIH Publ. No. 97-2789.
- Ries, L.A.G., M.P. Eisner, C.L. Kosary, et al., eds., 2001. *SEER Cancer Statistics Review, 1973-1998*. Bethesda, MD: National Cancer Institute. [http://seer.cancer.gov/publications/csr1973\\_1998](http://seer.cancer.gov/publications/csr1973_1998)
- Roetzheim, R.G., N. Pal, C. Tennant, et al. 1999. Effects of health insurance and race on early detection of cancer. *Journal of the National Cancer Institute* 91:1409-15.
- Roetzheim, R.G., N. Pal, E.C. Gonzalez, J.M. Ferrante, D.J. Van Durme, and J.P. Krischer. 2000. Effects of health insurance and race on colorectal cancer treatments and outcomes. *American Journal of Public Health* 90:1746-54.
- Schottenfeld, D., and S.J. Winawer. Cancers of the large intestine. 1996. In: D. Schottenfeld, and J.F. Fraumeni, Jr., eds. *Cancer epidemiology and prevention*, 2<sup>nd</sup> ed. New York: Oxford University Press, 813-40.
- Steele, G. Jr. Colorectal cancer. 1995. In: G.P. Murphy, W. Lawrence, Jr., and R.E. Lenhard, Jr., eds. *American Cancer Society textbook of clinical oncology*, 2<sup>nd</sup> ed. Atlanta: American Cancer Society, 236-50.
- Woolf, S.H. 2000. The best screening test for colorectal cancer—a personal choice. *New England Journal of Medicine* 343:1641-2.