

# *The Social Structure, Stress, and Women's Health*

---

**KRISTI WILLIAMS, PhD,\* and LIANNE M. KURINA, PhD†**

*\*The Ohio State University, Columbus, Ohio, and †University of  
Chicago, Chicago, Illinois*

Of all the social determinants of health, gender is one of the most significant. Research spanning multiple disciplines indicates that women have higher rates of psychological distress, depression, and physical morbidity than men.<sup>1,2</sup> According to the social stress model,<sup>3,4</sup> group differences in mental and physical health partly reflect socially structured variations in exposure and vulnerability to stress. Thus, understanding the socio-environmental factors that influence the number and types of stressors to which women are exposed is essential to understanding women's health risks.

The structural context in which women live has changed significantly over the past 30 years. Gender roles, family roles, and family structure have undergone dramatic shifts, many of which have been particularly relevant to women. These changes have im-

portant implications for the types of stressors women face and, ultimately, for their health and well-being. This chapter reviews recent research and theory on the impact of three key demographic patterns on the number and types of stressors to which women are exposed and the consequences of these stressors for their physical and mental health. These three factors that have significantly increased stress in women are: (1) women's increased workforce participation, (2) the rise in divorce and single parenthood, and (3) the aging of the population. We also provide an overview of the psychosocial factors that influence women's vulnerability to stress, discuss the biologic pathways through which socioenvironmental stressors affect women's health and well-being, and explore disease outcomes of the stress process that are especially relevant to women. We conclude with a discussion of the ways in which interdisciplinary collaborations between sociologists, psychologists, and biomedical researchers can enhance our understanding of the processes through which stressors in the social environment affect women's health.

*Correspondence: Kristi Williams, Department of Sociology, The Ohio State University, 42 Bricker Hall, 190 N. Oval Hall, Columbus, OH 43210. E-mail: klw@mail.sociology.ohio-state.edu.*

*Funding for this work was provided by NIH/NIA grant 2T32AG00243 to K. Williams (NIA Specialized Training: Demography and Economics of Aging) and Alfred P. Sloan Foundation, Center on Parents, Children and Work grant 2000-6-14 to L. M. Kurina.*

---

### ***The Stress Model of Illness***

*Stress* refers to a state of emotional or physiological arousal that occurs when demands from the environment (ie, stressors) tax the individual's adaptive capacity.<sup>5,6</sup> The psychological and emotional changes evoked during the stress process can, over time, undermine mental and physical health. Stress has been shown to influence health through both biologic and behavioral mechanisms. Biologically, stress is associated with cardiovascular responses;<sup>7</sup> neuroendocrine changes, including catecholamine activity and increased cholesterol levels;<sup>8,9</sup> and impaired immune functioning.<sup>10</sup> Stress can also lead to negative health behaviors such as substance and alcohol abuse, smoking, and a lack of exercise.<sup>11</sup>

Psychological and sociological research on the stress process indicates that exposure to stressors does not always lead to a stress reaction.<sup>6</sup> In many ways, stress is in the eye of the beholder. Whether exposure to stressful life circumstances undermines health and well-being depends on how the stressor is perceived (eg, as threatening or benign), the salience of the stressor to the individual, and the strategies and resources available to the individual to respond to and cope with socioenvironmental challenges.<sup>6</sup> Thus, vulnerability to stress is distinct from exposure to stress.

A number of social and personal resources are thought to influence an individual's vulnerability to stress. Among these, social support has received the greatest attention. The advice, emotional support, and caring provided by interpersonal relationships not only helps individuals cope with stress but also reduces the probability that particular challenges will be perceived as unmanageable.<sup>12,13</sup> Personal control or mastery, "the belief that one's own intentions and behaviors can impose control over one's environment,"<sup>14</sup> influences vulnerability to stress through a similar process.<sup>15</sup> In addition, the salience of a socioenviron-

termines her vulnerability to stress. Research indicates that stressors that are related to social roles that are central to an individual's identity are more likely to undermine her mental and physical health.<sup>16</sup>

Although some research indicates that, compared with men, women are exposed to more chronic strains<sup>17-19</sup> and are more vulnerable to the negative consequences of the stressors that they encounter,<sup>18</sup> other studies find no gender differences in exposure or vulnerability to stress.<sup>20-23</sup> Clearly, both men and women experience stress, and the stress process is an important determinant of the health of all individuals, regardless of gender. However, because men and women have different social roles and structural positions, the types of stressors that they encounter and the resources available to cope with and adjust to these strains often differ considerably.

### ***Gender, Social Stress, and Health***

Much of the early biomedical research on the links between stress and health focused exclusively on men. This approach was due in part to attempts to understand men's greater risk of mortality from cardiovascular disease and other chronic health problems. This focus, however, left the impression that stress is an important risk factor for men only. The identification of the "Type A personality" and its greater prevalence among men<sup>24</sup> buttressed this view.

Because the gender distribution of morbidity has undergone significant shifts in the past several decades, there has been growing recognition of the need to more fully understand the link between stress and women's health. Indeed, some of the greatest changes in the gender distribution of chronic health problems are observed for conditions that are closely tied to stressful life circumstances. For example, despite a general decrease in the incidence of cardiovascular disease among men, it has actually increased

women.<sup>25</sup> Moreover, although women have longer life expectancies than men, this advantage has steadily declined over the past 10 years.<sup>26</sup>

Sociologists have long been attuned to the importance of stress in women's lives. A central tenet of the sociological stress model is that stressful experiences "don't spring out of a vacuum but typically can be traced back to surrounding social structures and people's locations within them."<sup>27</sup> According to this model, the differing structural contexts in which men and women live have important implications for the types of stressors they encounter, the way they perceive or appraise potential stressors, and the resources available to cope with environmental challenges. Social change can, therefore, impinge on all phases of the stress process.

### ***Women and Stress in the 21st Century***

The structural context in which women live has changed significantly over the past 30 years. Gender roles, family roles, and family structure have undergone dramatic shifts, many of which have been particularly relevant to women. Although these changes have had many positive consequences, they also influence the types of stressors women face, the resources available to women to manage these stressors, and ultimately their mental and physical health. We next review the impact of three key demographic shifts on women's exposure and vulnerability to socioenvironmental stressors: (1) women's increased workforce participation, (2) the rise in divorce and single parenthood, and (3) the aging of the population.

The sociological stress model, with its emphasis on structural and environmental stressors, is particularly relevant to an understanding of the unique stressors encountered by women. Sociological research on stress focuses on global mental and physical health outcomes of the stress process, including self-assessed health and psychological well-being or depression. Thus, in dis-

cussing the consequences of recent demographic shifts on women's exposure and vulnerability to stress, the health outcomes we examine are limited to these global measures of health and well-being. We strongly contend, however, that a full understanding of the impact of stress on women's lives necessitates an understanding of the specific disease outcomes that result from the socioenvironmental stressors that women encounter and also requires knowledge of the biologic mechanisms through which these effects are produced. We therefore conclude with a review of emerging research on a potential mechanism through which stress may uniquely affect women's health (impaired ovarian function and diminished estrogen levels) and consider evidence establishing links between this mechanism and women's risk of heart disease and osteoporosis.

### ***Women and Paid Work***

Women's paid labor force participation has increased dramatically in the past two decades. Today approximately 69% of women between the ages of 25 and 64 work in the paid labor force, up from approximately 51% in 1978.<sup>28</sup> The increase in the labor force participation of married women with children has been even more dramatic. In 1978, only 38% worked outside of the home, but by 1990 this figure had risen to 58%.<sup>29</sup> These trends are not due exclusively to an increase in part-time employment: approximately three quarters of all employed women work full-time, year-round.<sup>28</sup>

Although most research indicates that paid employment benefits a woman's mental and physical health,<sup>30-34</sup> employed women report more stress, depression, and psychological distress than their male counterparts.<sup>35-39</sup> In a recent review of women's occupational health, Messing<sup>40</sup> suggests that "stress is identified as *the* women's [occupational] health problem." Of course, women do not have a monopoly on occupational stress; men's jobs are often challenging and demanding as well. What is clear, however, is that paid employment is associ-

ated with a different set of demands and challenges for men and women. There are two primary reasons for this. First, men and women tend to be employed in different sectors of the economy. Second, the strains associated with balancing the demands of work and family fall disproportionately on women.

### ***Sex Segregation of the Labor Force***

Despite the dramatic increase in women's labor force participation, employment in specific occupations and industries remains highly segregated by sex.<sup>41</sup> Because men and women tend to work in different sectors of the economy, the types of stressors to which they are exposed differ considerably.<sup>42</sup> For example, the impact of financial strain on mental and physical health is well established,<sup>43</sup> and the gender gap in earnings places women, especially unmarried women, at greater risk of experiencing this type of stress. On average, the yearly earnings of women employed full-time in the paid labor force are approximately 71% of men's.<sup>44</sup>

Women are also concentrated in service sector jobs that are characterized by low levels of autonomy and high levels of routinization.<sup>43,45,46</sup> Although there is a paucity of research on the effects of these job characteristics on women per se, general population studies indicate that these factors are positively associated with job strain and psychological distress.<sup>32,47</sup> Compared with men's, women's work roles appear to provide fewer psychosocial rewards (eg, personal fulfillment and recognition from others), and these gender differences appear to partly explain women's poorer self-assessed health status.<sup>43</sup> Moreover, the characteristics of the jobs that women tend to occupy have been shown to undermine personal control,<sup>48,49</sup> and lower personal control is, in turn, associated with greater vulnerability to stress.<sup>14</sup> In addition, women are concentrated in jobs that involve the provision of

care to others (eg, nursing, social work, teaching, customer service).<sup>50</sup> Providing care, whether in the context of interpersonal relationships or paid labor, often requires the sublimation of one's own needs to the needs of others, a process that is itself associated with psychological distress and poorer health.<sup>50-52</sup> Moreover, research on gender and personal relationships suggests that women are more vulnerable to the negative effects of interpersonal stressors.<sup>53</sup> This could mean that in addition to being exposed to more demands associated with providing care to others, women may also be especially vulnerable to the stressors that they encounter in this domain.

Very little research has examined whether women have heightened vulnerability to work-related stressors. Gender role theory suggests that stressors associated with the work role should be more salient to men than to women, while family stressors should be more salient to women.<sup>16</sup> If, as identity theory suggests, stressors that occur in roles that are highly salient to one's identity more strongly undermine health,<sup>54</sup> women should be less vulnerable to the negative effects of work-related stressors than men. The available evidence, however, suggests that women are actually more psychologically vulnerable than men to the negative effects of job demands and high levels of routinization at work.<sup>39</sup> Although the reasons for this pattern deserve further investigation, Roxburgh<sup>39</sup> speculates that employed women's vulnerability to workplace stressors is amplified by exposure to additional stressors associated with housework and childcare. We explore this possibility in more depth in the following section.

### ***The "Second Shift" and Work/Family Strain***

The substantial increase in women's labor force participation has not been accompanied by parallel decreases in their work within the home. Estimates indicate that employed married women perform approxi-

mately twice the amount of housework as men—what amounts to about two thirds of all household chores.<sup>32,38</sup> This discrepancy is amplified among those with children: women perform more than 75% of all child-care tasks.<sup>55,56</sup> The result is that many employed women face a “second shift” when they return home from the workplace.<sup>57</sup> Employed married women spend an average of 64.7 hours per week engaged in paid work and housework.<sup>58</sup>

Theories of role strain “propose that women’s multiple roles result in role overload or role conflict, which contribute to increased stress and excessive demands on time, energy, and psychologic resources—resulting in poorer health.”<sup>59</sup> Although studies of the health consequences of women’s multiple roles have not produced a consistent pattern of findings, the best evidence indicates that women’s family roles and work roles interact to affect their psychological well-being. For example, women’s mental health is most negatively affected by exposure to job stressors, such as low levels of control, when they are also exposed to family demands.<sup>32</sup> Furthermore, Rosenfield<sup>60</sup> reports that although employed women report more distress and depression than employed men, this gap is closed when the division of labor in the home is equal.

### **HEALTH CONSEQUENCES**

Although very little is known about the physical health consequences of women’s “second shift,” some evidence indicates that the challenges associated with balancing the roles of worker and wife/parent undermine women’s mental and physical health.<sup>61,62</sup> Further, research on men and women in management positions finds that although men and women have similar levels of epinephrine, a compound secreted by the sympathetic adrenal medullary system during a stress response, during the workday, women have higher levels of norepinephrine than men after returning home in the evening.<sup>63</sup> These findings suggest that among employed individuals with similar levels of

work stress, the stress of household labor and maintenance is greater among women than men. The implications of this research for women’s health are far-reaching. According to the allostatic load model,<sup>64</sup> sustained stress reactions have particularly detrimental consequences for biologic systems in the body and, ultimately, for the risk of disease and illness.<sup>65</sup> Thus, women’s higher rates of morbidity may, in part, reflect their exposure to stressors in multiple domains that result in sustained stress reactions and an increased allostatic load.

### ***Divorce and Single Parenthood***

Patterns of marriage and parenthood have also undergone significant shifts in the past 30 years. Between 1975 and 1990, the percentage of ever-married women who had divorced by midlife increased from 20% to 32% for white women and from 29% to 45% for black women.<sup>66</sup> The rise in the divorce rate has been accompanied by an increase in nonmarital childbearing. The proportion of children born to single mothers increased from 18.4% in 1980 to 33.2% in 2000.<sup>67</sup> A consequence of these trends is that an increasing number of women are raising children on their own, a situation that exposes them to a range of chronic strains that can ultimately affect their health and well-being.

### **EXPOSURE TO POVERTY AND FINANCIAL STRAIN**

A lack of economic resources is one of the most significant stressors associated with divorce and single parenthood for women. Indeed, poverty and financial strain are arguably the most pervasive of all stressors that disproportionately affect women in the United States. Although the “feminization of poverty” is rooted in a number of social structural factors, divorce and nonmarital childbearing are two of the quickest routes to poverty for women. On average, divorce is associated with a 27% decline in available

income for women and a 37% decline among women with children.<sup>68,69</sup> Even more striking is the fact that approximately 46% of single mothers live below the poverty line.<sup>70</sup>

Single mothers often have sole responsibility for providing financial support to children. Recent estimates indicate that half of single mothers have a court order for child support, but only 25% receive the mandated amount.<sup>71,72</sup> Divorced and never-married mothers who live in poverty encounter additional stressors associated with providing care and financial support to children, goals that are often incompatible given the high cost of childcare. Moreover, poverty and financial strain, although chronic stressors themselves, expose individuals to a number of secondary stressors, including residential crime and frequent residential mobility.<sup>73,74</sup>

### **PARENTING STRAINS**

Regardless of women's marital, employment, or socioeconomic status, parenting minor children is often stressful. Mothers report greater levels of psychological distress and lower levels of well-being compared with those who do not have children.<sup>75,76</sup>

The stressors encountered by average parents are exacerbated for single and divorced custodial mothers, who typically assume sole responsibility for providing emotional support and care to children. However, because most research has focused on the effects of single parenthood on children's well-being, very little is known about the specific stressors that single mothers encounter in their daily lives. The available evidence indicates that balancing the demands of work and family is particularly challenging for single mothers. For example, Avison<sup>77</sup> found that single mothers are more likely than their married counterparts to be exposed to caregiving and work strain. Given the prevalence of single parenthood in the United States, future research should attempt to describe the unique parenting-related stressors to which single mothers are exposed and identify the psy-

chosocial resources that help them manage these challenges. Understanding the stress process among single mothers can not only help to improve women's health, but can also enhance the well-being of their children.

### **VULNERABILITY TO STRESS**

Divorce and single parenthood not only increase women's exposure to stress, but also the poverty that they engender may increase women's vulnerability to additional stressors. For example, Turner and Noh<sup>78</sup> found that even if poor women and more advantaged women were exposed to similar levels of stress, disadvantaged women would continue to have lower psychological well-being. This evidence indicates that poor women are particularly reactive to the socio-environmental challenges that they encounter. Research on socioeconomic differentials in personal and social resources supports this possibility. The social networks of poor women tend to be smaller and less supportive than those of more advantaged women. Poor women also have lower levels of personal control.<sup>78</sup> Divorce itself may also increase vulnerability to secondary stressors such as financial strain. For example, Doherty<sup>79</sup> found that the transition to divorce is associated with declines in personal control.

### **HEALTH CONSEQUENCES**

Despite the pervasive stressors engendered by divorce and single parenthood, very little is known about their direct health consequences for women. Some research confirms that separated and divorced mothers report more stress and generally poorer health than their married counterparts.<sup>80</sup> Other studies have found that compared with married women, divorced women have higher levels of cholesterol, a pattern that researchers attribute to divorce-related stressors.<sup>81</sup> As noted, divorce and single parenthood are associated with poverty for many women, and the effects of poverty on morbidity and mortality are well established.<sup>75</sup>

Divorce per se and the stressors that it engenders do not appear to undermine women's health more than men's. In fact, research indicates that divorce is associated with poorer health outcomes and greater mortality risk for men compared with women.<sup>82-84</sup> Most studies on which this conclusion is based, however, control for factors that characterize divorce and that may amplify its negative effects on health, particularly among women. Controlling for socioeconomic status and/or the presence of children in the household may result in an underestimation of the negative effects of divorce for women because it is these factors that shape the challenges that divorced women face. More research is needed to determine whether socioeconomic status and, especially, custody of a minor child moderates the effect of divorce on women's health. It is likely that divorce leads to the most negative health consequences for socioeconomically disadvantaged women with children.

### **Population Aging**

The age structure of the U.S. population has changed dramatically in the past 40 years. Between 1960 and 2000, there was a 65% increase in the proportion of the population aged 65 and older. In 1960, adults over 65 represented only 9.2% of the population, but this figure had risen to 12.8% by 2000. Moreover, the older population is expected to explode in the next 40 years as baby boomers reach retirement age. Recent projections indicate that by 2040, more than 20% of the U.S. population will be over age 65.<sup>85</sup>

Because of their greater life expectancy, women substantially outnumber men at all ages over 65.<sup>85</sup> The most dramatic sex difference is seen among the oldest-old: approximately 72% of those over age 85 are women.<sup>85</sup> These trends have important implications for the types of stressors that women encounter throughout their lives.

Widowhood and advanced age accrue disproportionately to women, but midlife and younger women are also increasingly likely to provide care to an aging parent, a role that itself engenders multiple stressors.

### **WIDOWHOOD**

Women's longer life expectancy, combined with the tendency of women to marry somewhat older men, means that women are much more likely than men to be widowed in late life.<sup>85</sup> Only 40% of women over the age of 65 live with a spouse, compared with 74% of men.<sup>85</sup> The death of a spouse is a stressful life event that is associated with increases in psychological distress and declines in self-assessed health.<sup>86,87</sup> Although most evidence indicates that widowhood is worse for men's mental health than for women,<sup>87,88</sup> some research suggests that women's physical health may be more negatively affected than men's.<sup>89</sup> Although there is little consensus on gender differences in the negative effects of widowhood, researchers generally recognize that the primary structural and psychosocial mechanisms responsible for the negative effect of widowhood on health likely differ for men and women.

After the strain of bereavement, financial strain is perhaps the greatest stressor faced by widowed women. Estimates indicate that widowhood is associated with an 18% reduction in women's living standards.<sup>90</sup> As a result, approximately one third of elderly women live near or below the poverty line.<sup>91</sup> The financial strain associated with widowhood is largely responsible for the poorer mental health of widowed compared with married women.<sup>87</sup> Widowhood also exposes women to a number of secondary stressors, including the loss of social support provided by the marital relationship, the strains of residential relocation (eg, to be near a caregiving child or family member) or institutionalization, and loneliness associated with

### **HEALTH PROBLEMS AND FUNCTIONAL DECLINE IN LATE LIFE**

Regardless of their marital status, women are especially likely to experience poor health and chronic disease in later life. Because the health problems experienced by women tend to be less immediately life-threatening than men's, women spend a greater amount of time during late life coping with the strains of chronic illness and functional limitations.<sup>1,92</sup> Among those over age 80, a significantly greater proportion of women compared with men report having two or more chronic conditions.<sup>93</sup> A recent report issued by the U.S. Bureau of the Census<sup>85</sup> indicates that 34% of elderly women need assistance with everyday activities compared with only 22% of men. Moreover, functionally dependent women are more likely to live alone than their male counterparts.<sup>85</sup>

### **VULNERABILITY TO STRESS IN LATE LIFE**

Research on gender and life course differences in depression suggests that exposure and/or vulnerability to late-life stressors is particularly great for women. For, example, Mirowsky<sup>94</sup> found that although women tend to be more depressed than men at all ages, the gender gap increases significantly with age. Age-related changes and late-life stressors may increase vulnerability to additional stress. For example, Ensel and Lin<sup>95</sup> found that stressful life events such as the death of significant others are more strongly associated with physical health problems of older compared with younger adults. Although the causes for increased vulnerability to stress in late life are not well understood, they likely include the decline in autonomy and personal control that accompanies health problems and functional limitations. Moreover, socioenvironmental stressors appear to more strongly undermine the psychological well-being of functionally disabled individuals,<sup>96</sup> perhaps because of their reduced autonomy.

stressor for women in late life, results in a decline in socially supportive networks that might otherwise buffer individuals from the negative effects of stress exposure. In general, however, older women tend to have more close relationships outside of marriage than men.<sup>97</sup> Although these close personal relationships no doubt protect women's health, they can also involve strains associated with providing emotional support and practical assistance.<sup>98</sup> Moreover, some evidence indicates that women are particularly vulnerable to the negative effects of stressful life events experienced by friends and relatives.<sup>53</sup>

Very little is known about the potentially stress-provoking aspects of women's close relationships in later life or the impact of demands and strains in this domain on women's health and well-being. In addition to exploring these issues, future research should seek to identify the factors responsible for life course variations in vulnerability to stress and determine whether these processes differ for men and women. Moreover, the role of exposure and vulnerability to late-life stressors in precipitating or exacerbating the health problems and functional limitations of older women deserves much more investigation.

### **PROVIDING CARE TO AGING PARENTS**

As the population ages, the proportion of adults requiring care due to illness or disability increases dramatically. A recent report by the U.S. Census Bureau<sup>85</sup> states, "there is no historical precedent for the experience of most middle-aged and young-old persons having living parents." The result of this demographic shift is a dramatic increase in what is commonly described as the parent-support ratio, the number of persons aged 85 and over compared with those aged 50 to 64 years. The parent-support ratio has tripled in the past 40 years, and estimates suggest that it will triple again in the coming 60 years.<sup>85</sup>

Providing care to an ill or disabled family

deavor and one that is overwhelmingly undertaken by women.<sup>99-101</sup> Estimates indicate that over 50% of women will provide care to an aging parent at some point in their lives.<sup>102</sup> In addition to providing more frequent care to aging parents than their male counterparts, women's caregiving responsibilities tend to be more demanding and often include daily care and household maintenance.<sup>101</sup> Moreover, some evidence suggests that female caregivers receive less socioemotional support from the care recipient than their male counterparts, perhaps because the provision of care by a daughter is more expected, and therefore less appreciated, than the provision of care by a son.<sup>100</sup> The result is that among those who provide care to an aging parent, women report more stress than men.<sup>100</sup>

In addition to the primary stressors directly associated with caregiving, providing care to an aging parent often exposes women to secondary stressors associated with balancing the roles of parent, worker, and caregiver. The aging of the population, combined with trends toward later childbearing, has resulted in what some have called the "sandwich generation"—baby boomers, who, in midlife, find themselves simultaneously raising children and providing care to aging parents. Research indicates that women's psychological distress in midlife is strongly related to the challenges of balancing roles of parent, worker, and care provider to an aging parent.<sup>103</sup> Moreover, women are more likely than their male counterparts to miss work or quit their jobs due to responsibilities of providing care to a parent,<sup>104,105</sup> a situation that may lead to financial strain associated with reduced work hours or unemployment. Regardless of the existence of other roles, however, it is clear that the strains of caregiving undermine health and well-being. Caregivers report more psychological distress and greater physical and psychiatric symptoms than

## ***Pathways from Stress to Illness***

The relationship between stress and ill health is commonly accepted, but how, exactly, does stress produce adverse effects on the body? What are the biologic pathways through which stress affects health, and might some of these pathways differ for men and women? In this section, we review the primary physiological responses to stress and discuss some of the potential disease-related effects of these responses that are particularly relevant to women. Specifically, we focus on the relationship between stress and ovarian dysfunction and consider how suppression of reproductive hormones may be related to common and disabling conditions among women, including osteoporosis and heart disease.

### **PHYSIOLOGICAL RESPONSES TO STRESS**

There are two distinct physiological pathways, directed by the sympathetic nervous system (SNS) and the hypothalamus respectively, that are activated in response to stress. Short-term stressors mobilize the SNS, resulting in the "fight or flight" response: increased blood sugar levels, a constriction of the blood vessels, an increase in heart rate (together increasing blood pressure), and a diversion of blood from nonessential organs to the heart, brain, and skeletal muscles.<sup>107</sup> Concurrently, the SNS stimulates the adrenal medulla to release catecholamines (epinephrine and norepinephrine) into the bloodstream, which has the effect of reinforcing and prolonging the fight or flight response. The SNS acts quickly and generates a brief, strong physiological response to a perceived threat.

In contrast, the hypothalamus activates a slower-acting but much longer-lasting stress response through the hypothalamus-pituitary-adrenal (HPA) axis. Stress stimulates the hypothalamus to manufacture and release corticotropin releasing factor (CRF), which causes the pituitary gland to release

adrenal cortex to produce and release corticosteroids (primarily cortisol) into the body. The increase in cortisol levels after activation of the HPA axis promotes glucose formation from noncarbohydrate molecules and enhances epinephrine's vasoconstrictive effects, thereby helping to sustain the rise in blood pressure necessary to promote efficient distribution of nutrients to the cells.<sup>107</sup> In addition to its effects on the cardiovascular system, cortisol also affects the immune system; acute stressors are associated with an upregulation of the immune system, in which lymphocytes are mobilized into the bloodstream, but prolonged increases in cortisol levels, which may be typical in chronically stressed individuals, have been shown to depress immunologic function.<sup>64</sup> Finally, the HPA axis also interacts with and strongly influences the other neuroendocrine axes in the body, including the hypothalamus-pituitary-thyroid (HPT) axis, critical to normal metabolism, and the hypothalamus-pituitary-gonadal (HPG) axis, responsible for the regulation of reproductive function, effects of which we will consider in more detail below.

The physiological mechanisms activated by stress are presumably an adaptation to help people cope with the threat at hand; energy resources are mobilized, the immune system is put on alert, and unnecessary functions are temporarily shut down. This process is not in and of itself pathologic. Realization of the pathway from stress to physical illness is strongly contingent upon the severity and/or persistence of the stress response; too-frequent threats, nonadaptation to repeated stressors of the same type, or a breakdown in the negative feedback system that normally regulates the stress response can generate problems.<sup>64</sup> Somewhat counterintuitively, health-damaging effects can also occur when the cortisol stress response is not sufficiently strong—a condition that promotes the secretion of inflammatory cytokines (normally suppressed by cortisol) and hence an enhanced inflammatory re-

Here, we mainly consider the first alternative: chronic or severe stress leading to persistent HPA activity.

### **STRESS, DEPRESSION, AND ILLNESS**

Stress is a strong risk factor for depression; indeed, the endocrine abnormalities common in major depression (hypercortisolism, in particular) mimic those of stress.<sup>108–111</sup> Depression impacts physical well-being both directly, via persistent hypercortisolism, and indirectly, by affecting health-related behaviors. Depression is, on average, twice as common in women as it is in men: it is estimated that 12% of the women in the United States suffer from depression each year,<sup>112</sup> and the majority of these women go untreated.<sup>113,114</sup> Given how common an exposure depression is (and stress that much more so!), we suggest that the etiological pathways to illness discussed below have important implications for women's health.

### **STRESS AND OVARIAN FUNCTION**

Persistently elevated levels of cortisol and catecholamine exposure have effects on multiple organ systems in the body, including the brain, the immune system, and the cardiovascular system. The best evidence suggests that the biologic pathways through which stress affects health may differ by gender. A comprehensive meta-analysis of research on gender differences in stress responses indicates that among adults, men show greater blood pressure and urinary epinephrine reactions to laboratory-induced stressors, while women show greater heart rate reactivity.<sup>115</sup> These gender differences, however, are not observed among prepubertal children,<sup>116</sup> suggesting that differences in estrogen levels (which do not become pronounced until puberty) may be partly responsible for gender differences in stress reactions.

Although these findings are commonly interpreted as evidence that estrogen pro-

health consequences of stress exposure (particularly the risk of CHD), emerging evidence indicates that among women, estrogen levels themselves may be affected by stress. Thus, impaired ovarian function resulting in diminished estrogen levels may itself be a unique pathway through which stress adversely affects women's health. The limited data that exist suggest that ovarian dysfunction may be quite common among premenopausal women<sup>117</sup> and that, in the absence of other endocrine abnormalities, stress and depression are among the most important causes of functional hypothalamic hypogonadism.<sup>118,119</sup> As shown in Figure 1, stress and persistent HPA activation can effectively shut down the HPG axis, inhibiting ovarian function and decreasing levels of circulating estrogen.<sup>119</sup> Such an effect has been observed in cynomolgus macaques, a primate species with a complex social structure resulting in strong dominance hierarchies. Kaplan et al<sup>118</sup> showed that in comparison with dominant females, subordinate females were characterized by hypercortisolemia, behavior dysfunction, impaired ovarian function, and low circulating levels of estrogen. In women, ovarian dysfunction consists of a spectrum of conditions from amenorrhea to occult anovulation. Although more extreme forms of hypogonadism are typically treated with estrogen supplementation, occult anovulation will generally go unnoticed and untreated (except in cases where women are trying to have children). Diminished estrogen levels,

in turn, have been linked to specific health risks among women, including heart disease and osteoporosis, as discussed below. Given emerging evidence of the links between stress exposure and estrogen levels, researchers have begun to explore the role of ovarian function and estrogen in mediating the effects of stress on specific disease outcomes. We consider these etiologic pathways in greater detail below.

#### HEART DISEASE

Research in a variety of animal models and in humans has established that stress and depression are significant risk factors for heart disease.<sup>118,120,121</sup> Multiple etiological pathways have been proposed for these relationships, including: (1) acceleration of atherosclerosis as a result of frequent or sustained increases in blood pressure,<sup>122</sup> (2) increases in the likelihood of clotting, due to HPA-induced changes in platelet reactivity,<sup>123</sup> and (3) increased storage of fat in the abdominal cavity.<sup>111,123</sup>

Research on heart disease in women has been undertaken only in recent decades, but there is growing interest in the relationship between endogenous estrogen levels and heart disease. There are several reasons to suspect that estrogen levels are related to the risk of heart disease among women. First, women have a much lower risk of heart disease than men,<sup>124,125</sup> but their risk increases steadily with age. Menopause, in particular, is associated with changes in important heart disease risk factors, such as increased serum

Prolonged/acute stress → HPA activation → HPG inhibition → Ovarian dysfunction →

Decreased estrogen levels



Increased risk of heart disease / increased risk of osteoporosis

**FIG. 1.** Stress, estrogen, and women's health: potential etiologic pathways.

levels of low-density lipoprotein and decreased serum levels of high-density lipoprotein,<sup>126</sup> changes that are mitigated by estrogen supplementation in the form of hormone replacement therapy (HRT). Early menopause (whether natural or surgical) is also associated with an increased risk of heart disease.<sup>127,128</sup>

These findings suggest that estrogen may play a role in protecting young women against heart disease. Might diminished estrogen exposure before menopause predispose women to heart disease later in life? Several case-control studies suggest that this may be the case. Irregular menstruation has been linked both to risk factors for cardiovascular disease like increased plasma fibrinogen concentrations and thickened or sclerotic arterial intima<sup>129</sup> and to risk of myocardial infarction.<sup>130</sup> A smaller study showed that premenopausal women with CHD had significantly lower levels of plasma estradiol.<sup>131</sup> Results of a nested case-control study in the Netherlands suggested that anovulatory cycles were more frequent in women who later developed CHD, although no relationship was observed between urinary sex hormone excretions and CHD risk.<sup>132</sup>

These findings, combined with emerging evidence of the effects of stress on estrogen levels, suggest that impaired ovarian function may be a pathway through which stress affects heart disease risk among women. The pathway from stress or depression through ovarian dysfunction and diminished estrogen levels to heart disease is a plausible etiological pathway, strongly supported by research with the cynomolgus macaques;<sup>118</sup> premenopausal subordinate females developed atherosclerosis at about the rate of male macaques (whereas atherosclerosis progression was very slight in dominant females), but this process was inhibited by exogenous estrogen. To date, however, ovarian function and atherosclerosis progression and their potential links to socioenvironmental stressors have not been studied prospectively in premenopausal women.

## OSTEOPOROSIS

Osteoporosis, defined operationally as a significant reduction in bone mineral density (BMD) compared with that of average young women,<sup>133</sup> is extremely common among postmenopausal women in the United States.<sup>134</sup> Hip and vertebral fractures are directly related to low BMD<sup>135,136</sup> and are strongly associated with future morbidity and mortality.<sup>134</sup> The presence of biologically active estrogen receptors in normal human osteoblast cells was demonstrated unequivocally in 1988,<sup>137</sup> suggesting a biologic mechanism for the observed relationship between estrogen deficiency and BMD, seen both in postmenopausal women (not taking HRT) and in premenopausal women with ovulatory disturbances.<sup>134</sup> A review of the literature at the time of this discovery emphasized the importance of gynecologic disorders like amenorrhea, gonadal dysgenesis, and premenopausal oophorectomy for BMD; however, the impact of other ovulatory disturbances on BMD was uncertain.<sup>138</sup> Although findings have not been entirely consistent, subsequent studies have shown links between endogenous estrogen exposure, as affected by short luteal phase, anovulatory cycles, history of premenstrual symptoms, menstrual irregularities, or oligomenorrhea, and vertebral osteoporosis or bone loss.<sup>117,139-144</sup>

The results of these studies suggest that less severe estrogen deficiencies could also operate to enhance the risk of osteoporosis and fracture later in life, via the same pathway described above for heart disease (see Fig. 1). Although the relationship between stress and osteoporosis or fractures has not been studied directly, there is emerging evidence that depression is associated with decreased BMD and an increased risk of osteoporotic fracture in older women.<sup>145-148</sup> The high prevalence of depression and stress in women in the United States and the small changes in bone density associated with important increases in fracture rates together suggest the public health importance of fur-

ther investigating the links between stress, depression, and osteoporosis.<sup>146-149</sup>

### ***Avenues for Interdisciplinary Research***

Sociological research clearly indicates that the roles and statuses that women occupy structure the number and types of stressors to which they are exposed. There is little doubt that the strains associated with balancing the demands of work and family and providing care to aging parents, as well as the challenges associated with aging, divorce, poverty, and single parenthood, have important implications for women's health risks. Moreover, emerging research in health psychology and psychoneuroendocrinology indicates that the primary biologic pathways through which these stressors affect health differ for men and women. An integration of these lines of research has great potential for the development of a more complete understanding of the processes underlying gender differences in specific disease risks.

Although sociologists, psychologists, and biomedical researchers commonly agree that the health of all individuals, including women, results from a complex interaction of biologic, social, and psychological factors, researchers in each discipline emphasize different aspects of this process and rarely combine their efforts. The result is a somewhat fragmented view of the causes and health consequences of the stressors that women encounter in their daily lives. Although social scientists have made great contributions by identifying socially structured variations in exposure and emotional vulnerability to stress, they rarely investigate the effects of these stressors on specific disease outcomes. In contrast, epidemiologists are interested in differences between the sexes in disease risk, but they generally do not have the theoretical framework or methodological tools to consider social context in sufficient detail to incorporate it into their causal disease models. Thus, although physiological and epidemiological

research has greatly enhanced our understanding of the specific mechanisms through which stress affects health and has shed light on sex differences in health outcomes of the stress process, it rarely links these mechanisms and outcomes back to the varying social environments of men and women. In fact, many biomedical studies of the stress process focus exclusively on laboratory-induced stressors, an approach that precludes a consideration of gender differences in biologic reactivity to stressors based upon their salience or relevance to one's identity. Making these links is essential to a complete understanding of the stress process as experienced by women.

The field of health psychology has made great strides in integrating the sociological, psychological, and biologic dimensions of the stress process. Not only do health psychologists recognize that social and psychological influences on group differences in health are mediated by biologic processes, but they also attempt to explain individual or group differences in stress responses by examining the influence of the social environment on the way that particular stressors are appraised.<sup>150</sup> Psychoneuroimmunology, which emphasizes the interaction of psychosocial processes with nervous, endocrine, and immune systems,<sup>151</sup> shows great promise for understanding how social-psychological factors influence biologic stress responses and, ultimately, disease risks. The positive association of social support with mental and physical health is a central component of the sociological model of health and illness, but the mechanisms through which social support protects individuals were previously not well understood. Psychoneuroimmunologists have made important contributions in this regard by establishing that social support has direct beneficial effects on immunocompetence and on cardiovascular and endocrine systems.<sup>152</sup>

Psychoneuroimmunology has also been successful in identifying gender differences in the links between socioenvironmental

stressors and biologic processes. For example, in a series of studies, Kiecolt-Glaser and Newton<sup>153</sup> convincingly demonstrated that men and women respond differently to different kinds of stress. Marital conflict, for example, results in much stronger and more persistent stress responses among women than men in terms of changes in blood pressure, stress hormone levels, and immune function; this is at odds with the common observation of stronger responses among males to acute stressors in laboratory studies.<sup>154</sup> Gender differences in stress responses are thought to reflect socially patterned variations in the perception or salience of marital conflict. As sociologists have observed, women tend to be more psychologically reactive to interpersonal stressors than men.<sup>153</sup> The work of Kiecolt-Glaser demonstrates that these gender differences in vulnerability to particular social stressors are also manifested in biologic processes.

In addition, an intriguing new theory posits a novel biobehavioral response to stress in females called "tend-and-befriend."<sup>154</sup> This theory draws on neuroendocrine evidence from animal models and behavioral observations in humans and primates to suggest that oxytocin and endogenous opioids could mediate the stress response in women, promoting caring and socializing behaviors to cope with the current challenge and to guard against future challenges. Although empirical confirmation of the theory awaits further evidence, it is consistent with sociological theories of gender differences in psychological development and socioemotional behavior that result, in part, from gender socialization.<sup>155</sup>

In sum, interdisciplinary collaborations between psychologists, sociologists, and biomedical researchers represent the cutting edge of research on the stress process and its link to group differences in health risks. Understanding the interaction of the social environment with individual and group-specific biologic processes offers great promise for advancing knowledge about the stress-related processes through which gen-

der differences in morbidity and mortality are produced. In doing so, however, researchers should recognize that multiple sociodemographic characteristics may modify gender differences in the link between stress and health. Thus, in addition to developing causal models that consider gender differences in socioenvironmental exposure, psychological vulnerability, and biologic reactivity to stress, researchers should strive to understand how other social and material circumstances, such as race, class, and age, interact with gender at each stage of the stress process.

## References

1. Verbrugge LM. Gender and health: an update on hypotheses and evidence. *J Health Soc Behav.* 1989;26:156–82.
2. Waldron I. Sex differences in illness, incidence, prognosis, and mortality: issues and evidence. *Soc Sci Med.* 1983;17:1107–23.
3. Dohrenwend BS, Dohrenwend BP. *Stressful life events and their contexts.* New York: Prodist, 1981.
4. Pearlin LI, Lieberman MA, Menaghan EG, Mullan JT. The stress process. *J Health Soc Behav.* 1981;22:337–56.
5. Aneshensel CS. Social stress: theory and research. *Annu Rev Sociol.* 1992;18:15–38.
6. Thoits P. Stress, coping, and social support processes: where are we? what next? *J Health Soc Behav.* 1995;Extra Issue:53–79.
7. Krantz DS, Manuck, SB. Acute psychophysiological reactivity and risk of cardiovascular disease: a review and methodologic critique. *Psych Bull.* 1984;96:435–64.
8. Frankenhaeuser M. The sympathetic-adrenal and pituitary-adrenal response to challenge: comparison between the sexes. In Dembroski TM, Schmidt TH, Blumchen G, eds. *Biobehavioral Bases of Coronary Heart Disease.* New York: Basel Karger, 1983.
9. Van Doornen LJP, van Blockland R. Serum cholesterol: sex specific psychological correlates during rest and stress. *J Psychosom Res.* 1987;31:239–49.
10. Kiecolt-Glaser JK, Glaser R. Psychosocial

- moderators of immune function. *Ann Behav Med.* 1987;9:16-20.
11. Steptoe A, Lipsey Z, Wardle J. Stress, hassles, and variations in alcohol consumption, food choice, and physical exercise: a diary study. *Br J Health Psychol.* 1998;3:51-63.
  12. Cohen S. Psychosocial models of the role of social support in the etiology of physical disease. *Health Psychol.* 1988;7:269-97.
  13. House JS, Umberson D, Landis KR. Structures and processes of social support. *Ann Rev Soc.* 1988;14:293-318.
  14. Williams K, Umberson D. Women, stress, and health. In Goldman MB, Hatch MC, eds. *Women's Health.* San Diego, CA: Academic Press; 2000:553-62.
  15. Mirowsky J, Ross CE. Social patterns of distress. *Annu Rev Sociol.* 1986;12:23-45.
  16. Simon R. Parental role strains, salience of parental identity and gender differences in psychologic distress. *J Health Soc Behav.* 1992;33:25-35.
  17. Radloff LS, Rae DS. Susceptibility and precipitating factors in depression: sex differences and similarities. *J Abnorm Psychol.* 1979;88:174-81.
  18. Turner RJ, Wheaton B, Lloyd DA. The epidemiology of social stress. *Am Sociol Rev.* 1995;60:104-25.
  19. McDonough P, Walters V. Gender and health: reassessing patterns and explanations. *Soc Sci Med.* 2001;52:547-59.
  20. Henderson S, Byrne DG, Duncan-Jones P, et al. Social relationships, adversity and neurosis: a study of associations in a general population sample. *Br J Psychiatry.* 1980;136:574-83.
  21. Markush RE, Favero R. Epidemiologic assessment of stressful life events, depressed mood, and psychophysiological symptoms. In Dohrenwend BS, Dohrenwend BP, eds. *Stressful Life Events: Their Nature and Effects.* New York: Wiley, 1974: 171-90.
  22. Personn G. Life event ratings in relation to sex and marital status in a 70-year-old urban population. *Acta Psychiatr Scand.* 1980;29:679-87.
  23. Vingerhoets AJJM, Van Heck GL. Gender, coping, and psychosomatic symptoms. *Psychol Med.* 1990;20:125-35.
  - et al. Hostility patterns and health implications: correlates of Cook-Medley hostility scale scores in a national survey. *Health Psychol.* 1991;10:18-24.
  25. Das BN, Banka VS. Coronary artery disease in women: how it is and isn't unique. *Postgrad Med.* 1992;91:197-207.
  26. Rodin J, Ickovics JR. Women's health: Review and research agenda as we approach the 21st century. *Am Psychol.* 1990;45: 1018-34.
  27. Pearlin LI, Jacobson D, Lennon MC. The sociological study of stress. *J Health Soc Behav.* 1989;30:241-56.
  28. U.S. Department of Labor, Bureau of Labor Statistics. *Employment and earnings.* Washington DC: U.S. Government Printing Office, 1997.
  29. Cohen, Bianchi SM. Marriage, children, and women's employment: what do we know? *Monthly Labor Review.* 1999; Dec: 22-31.
  30. Baruch GK, Barnett RC. Role quality, multiple role involvement, and psychologic well-being in mid-life women. *J Pers Soc Psych.* 1986;51:578-85.
  31. Bird CE, Freemont AM. Gender, time use, and health. *J Health Soc Behav.* 1991;32: 114-29.
  32. Lennon MC, Rosenfield S. Women and mental health: the interaction of job and family conditions. *J Health Soc Behav.* 1992;33:316-27.
  33. Passannante MR, Nathanson CA. Female labor force participation and female mortality in Wisconsin, 1974-1978. *Soc Sci Med.* 1985;21:655-65.
  34. Repetti RL, Matthews KA, Waldron I. Employment and women's health: effects of paid employment on women's mental and physical health. *Am Psychol.* 1989;44: 1394-401.
  35. Barnett RC, Marshall NL, Raudenbush SW, Brennan RT. Gender and the relationship between job experiences and psychologic distress: a study of dual-earner couples. *J Pers Soc Psych.* 1993;64:794-806.
  36. Cleary PD, Mechanic D. Sex differences in psychologic distress among married people. *J Health Soc Behav.* 1983;24: 111-21.

- work, and depression among husbands and wives. *J Health Soc Behav.* 1994;35:179-91.
38. Pleck JH. *Working Wives/Working Husbands.* Beverly Hills, CA: Sage, 1985.
  39. Roxburgh S. Gender differences in work and well-being: Effects of exposure and vulnerability. *J Health Soc Behav.* 1996;37:265-77.
  40. Messing K. Multiple roles and complex exposures: hard-to-pin-down risks for working women. In Goldman MB, Hatch MC, eds. *Women's Health.* San Diego, CA: Academic Press, 2000:455-62.
  41. Reskin B, Hartmann H, eds. *Women's Work, Men's Work: Sex Segregation on the Job.* Washington DC: National Academy Press, 1986.
  42. Lennon MC. Sex differences in distress: the impact of gender and work roles. *J Health Soc Behav.* 1987;28:290-305.
  43. Ross CE, Bird CE. Sex stratification and health lifestyle: consequences for men's and women's perceived health. *J Health Soc Behav.* 1994;35:161-78.
  44. U.S. Bureau of the Census. *Child support for custodial mothers and fathers.* 1991. *Curr Population Rep. Ser. P60-187.* Washington DC: U.S. Government Printing Office, 1995.
  45. England P, Farkas G, Kilbourn BS, Dou T. Explaining occupational sex segregation and wages: findings from a model with fixed effects. *Am Sociol Rev.* 1988;53:544-58.
  46. Karasek RA, Gardell B, Lindell J. Work and non-work correlates of illness and behavior in male and female Swedish white-collar workers. *J Occup Behav.* 1987;8:187-207.
  47. Karasek RA, Theorell T. *Healthy Work: Stress, Productivity, and the Reconstruction of Working Life.* New York: Basic Books, 1990.
  48. Kohn ML, Schooler C. Job conditions and personality: a longitudinal assessment of their reciprocal effects. *Am J Sociol.* 1982;87:1257-86.
  49. Wheaton B. The sociogenesis of psychological disorder: an attributional theory. *J Health Soc Behav.* 1980;21:100-24.
  50. Marshall NL, Barnett RC, Baruch GK, stress in caregiving occupations. *Cur Res Occup Prof.* 1991;6:61-81.
  51. Bulan HF, Erickson RJ, Wharton AS. Doing for others on the job: the affective requirements of service work, gender, and emotional well-being. *Soc Prob.* 1997;44:235-56.
  52. Pavalko EK, Woodbury S. Social roles as process: Caregiving careers and women's health. *J Health Soc Behav.* 2000;41:91-105.
  53. Kessler RC, McLeod JD. Sex differences in vulnerability to undesirable life events. *Am Sociol Rev.* 1984;49:620-31.
  54. Thoits PA. On merging identity theory and stress research. *Soc Psych Q.* 1991;54:101-12.
  55. Bird CE. Gender differences in the social and economic burdens of parenting and psychologic distress. *J Marriage Fam.* 1997;59:809-23.
  56. Ross CE, Van Willigen M. Gender, parenthood and anger. *J Marriage Fam.* 1996;58:572-84.
  57. Hochschild AR. *The Second Shift: Working Parents and the Revolution at Home.* New York: Viking, 1989.
  58. Lennon MC. Domestic arrangements and depressive symptoms: an examination of housework conditions. In Dohrenwend BP, ed. *Adversity, Stress, and Psychopathology.* New York: Oxford University Press, 1998:409-21.
  59. Waldron I, Weiss CC, Hughes ME. Marital status effects on health: are there differences between never-married women and divorced or separated women? *Soc Sci Med.* 1998; 45:1387-97.
  60. Rosenfield S. The effects of women's employment: personal control and sex differences in mental health. *J Health Soc Behav.* 1989;30:77-91.
  61. O'Neil R, Greenberger E. Patterns of commitment to work and parenting: implications for role strain. *J Marriage Fam.* 1994; 56:101-18.
  62. Repetti RL. Short-term effects of occupational stressors on daily mood and health complaints. *Health Psychol.* 1993;12:126-31.
  63. Lundberg U, Frankenhaeuser M. The total workload of male and female white-collar

- level, and number of children. *Scand J Psychol.* 1994;35:315-27.
64. McEwen BS. Protective and damaging effects of stress mediators. *N Engl J Med* 1998;338:171-9.
65. Lundberg U, Parr D. Neurohormonal factors, stress, health, and gender. In Eisler RM, Herson M, eds. *Handbook of Gender, Culture, and Health*. Mahwah, NJ: Lawrence Erlbaum Associates, 2000:21-41.
66. U.S. Bureau of the Census. Marriage, divorce and remarriage in the 1990s. *Curr Population Rep. Ser. P23-180*. Washington DC: U.S. Government Printing Office, 1992.
67. Martin JA, Hamilton BE, Ventura SJ, et al. Births: final data for 2000. *Natl Vital Stat Rep, Vol 50, No. 5*. Washington DC: National Center for Health Statistics, 2002.
68. Peterson RR. A re-evaluation of the economic consequences of divorce. *Am Sociol Rev.* 1996;61:528-36.
69. U.S. Bureau of the Census. Survey of income and program participation. *Curr Population Rep, Ser. P70-23*. Washington DC: U.S. Government Printing Office, 1991.
70. U.S. Bureau of the Census. Income, poverty, and valuation of noncash benefits, 1993. *Curr Population Rep, Ser. P60-188*. Washington DC: U.S. Govt. Printing Office, 1995.
71. U.S. Bureau of the Census. Child support and alimony: 1989. *Curr Population Rep, Ser. P60-173*. Washington DC: U.S. Govt. Printing Office, 1991.
72. Lin I. Perceived fairness and compliance with child support obligations. *J Marriage Fam.* 2000;62:388-99.
73. Garfinkel I, McLanahan S. *Single Mothers and Their Children: A New American Dilemma*. Washington DC: Urban Institute Press, 1986.
74. Williams DR. Socioeconomic differentials in health: a review and redirection. *Soc Psychol Q.* 1990;53:81-99.
75. McLanahan S, Adams J. Parenthood and psychologic well-being. *Annu Rev Sociol.* 1997;13:237-57.
76. Umberson D. Parenting and well-being: the importance of context. *J Fam Iss.* 1989;10:427-39.
77. Avison WR. Roles and resources: the effects of family structure and employment on women's psychosocial resources and psychologic distress. *Res Comm Mental Health.* 1995;8:233-56.
78. Turner RJ, Noh S. Class and psychologic vulnerability among women: the significance of social support and personal control. *J Health Soc Behav.* 1983;24:2-15.
79. Doherty WJ. Impact of divorce on locus of control orientation in adult women: a longitudinal study. *J Pers Soc Psych.* 1983;44:834-40.
80. Tchong-Laroche F, Prince R. Separated and divorced women compared with married controls: selected life satisfaction, stress and health indices from a community survey. *Soc Sci Med.* 1983;17:5-105.
81. Kushnir T, Kristal-Boneh E. Blood lipids and lipoproteins in married and formerly married women. *Psychosomatic Med.* 1995;57:116-20.
82. Gove WR. Sex, marital status, and mortality. *Am J Soc.* 1973;79:45-67.
83. Lillard LA, Waite LJ. Till death do us part: marital disruption and mortality. *Am J Soc.* 1995;100:1131-56.
84. Zick CD, Smith KR. Marital transitions, poverty, and gender differences in mortality. *J Marriage Fam.* 1994;53:327-36.
85. U.S. Bureau of the Census. Sixty-five plus in the U.S. *Curr Population Rep, Ser. P23-190*. Washington DC: U.S. Govt. Printing Office, 1996.
86. Bettis S, Scott F. Bereavement and grief. In Eisendorfer C, ed. *Annual Review of Gerontology and Geriatrics*. New York: Springer, 1981:144-59.
87. Umberson D, Wortman CB, Kessler RC. Widowhood and depression: explaining long-term gender differences in vulnerability. *J Health Soc Behav.* 1992;33:10-24.
88. Stroebe W, Stroebe MS. *Bereavement and Health: The Psychological and Physical Consequences of Partner Loss*. New York: Cambridge University Press, 1987.
89. Ferraro KF. The effect of widowhood on the health status of older persons. *Intl J Aging Hum Devel.* 1985;21:9-25.
90. Bound J, Duncan GJ, Laren DS, Oleinick L. Poverty dynamics in widowhood. *J Gerontol Soc Sci.* 1995;46:S115-24.

91. Malveaux J. Race, poverty, and women's aging. In Allen J, Pifer A, eds. *Women on the Front Lines: Meeting the Challenges of an Aging America*. Washington DC: Urban Institute, 1993:167-90.
92. Penning MJ, Strain LA. Gender differences in disability, assistance, and subjective well-being in later life. *J Gerontol Soc Sci*. 1994;49:S202-8.
93. Guralnik JM, Lacroix AZ, Everett DF, Kovar MG. *Aging in the Eighties: The Prevalence of Comorbidity and its Association With Disability*. Washington DC: National Center for Health Statistics, 1989.
94. Mirowsky J. Age and the gender gap in depression. *J Health Soc Behav*. 1996;37:362-80.
95. Ensel WM, Lin N. Age, the stress process, and physical disease. *J Aging Health*. 2000;12:139-68.
96. Roberts BL, Dunkle R, Haug M. Physical, psychologic, and social resources as moderators of the relationship of stress to mental health of the very old. *J Gerontol Soc Sci*. 1994;49:S35-43.
97. Antonucci TC. A life-span view of women's social relations. In Turner BF, Troll LE, eds. *Growing Older Female: Theoretical Perspectives in the Psychology of Aging*. New York: Sage Publications, 1994.
98. Rook KS. Detrimental aspects of social relationships: taking stock of an emerging literature. In: Veiel HOF, Bauman A, eds. *The Meaning and Measurement of Social Support*. New York: Hemisphere, 1992:157-69.
99. Pearlin LI, Mullan JT, Semple SJ, Skaff MM. Caregiving and the stress process. In Kasl SV, Kooper CL, eds. *Stress and Health: Issues in Research Methodology*. New York: Wiley, 1990:143-54.
100. Starrels ME, Ingersoll-Dayton B, Dowler DW, Neal MB. The stress of caring for a parent: effects of the elder's impairment on an employed adult child. *J Marriage Fam*. 1997;59:860-72.
101. Walker AJ, Pratt CC, Eddy L. Informal caregiving to aging family members. *Fam Relat*. 1995;44:402-11.
102. Himes C. Parental caregiving by adult women: A demographic perspective. *Res Aging*. 1995;16:191-211.
103. McKinlay JB, McKinlay SM, Brambilla D. The relative contributions of endocrine changes and social circumstances to depression in mid-aged women. *J Health Soc Behav*. 1987;28:345-63.
104. Anastas JW, Gibeau JL, Larson PJ. Working families and eldercare: a national perspective in an aging America. *Soc Work*. 1990;35:405-11.
105. Stone RI, Short PF. The competing demands of employment and informal caregiving to disabled elders. *Med Care*. 1990;28:513-26.
106. Schultz R, Visintainer P, Williamson GM. Psychiatric and physical morbidity effects of caregiving. *J Gerontol Psychol Sci*. 1990;45:181-91.
107. Marieb EN. *Human Anatomy and Physiology*. San Francisco: Benjamin Cummings, 2001:1249.
108. Carroll BJ, Curtis GC, Davies BM, et al. Urinary free cortisol excretion in depression. *Psychol Med*. 1976;6:43-50.
109. Gold PW, Chrousos GP. The endocrinology of melancholic and atypical depression: relation to neurocircuitry and somatic consequences. *Proc Assoc Am Physicians*. 1999;111:22-34.
110. Nemeroff CB, Widerlov E, Bissette G, et al. Elevated concentrations of CSF corticotropin-releasing factor-like immunoreactivity in depressed patients. *Science*. 1984;226:1342-4.
111. Thakore JH, Richards PJ, Reznick RH, et al. Increased intra-abdominal fat deposition in patients with major depressive illness as measured by computed tomography. *Biol Psychiatry*. 1997;41:1140-2.
112. Narrow WEU. One-year prevalence of depressive disorders among adults 18 and over in the U.S.: NIMH ECA prospective data. Population estimates based on U.S. Census estimated residential population age 18 and over on July 1, 1998. Unpublished data.
113. Regier DA, Narrow WE, Rae DS, et al. The de facto US mental and addictive disorders service system. Epidemiologic catchment area prospective 1-year prevalence rates of disorders and services. *Arch Gen Psychiatry*. 1993;50:85-94.
114. Kessler RC, Zhao S, Katz SJ, et al. Past-year use of outpatient services for psychiatric problems in the National Comorbidity

- Survey. *Am J Psychiatry*. 1999;156:115-23.
115. Stoney CM, Davis MC, Matthews KA. Sex differences in physiological responses to stress and in coronary heart disease: a causal link? *Psychophysiology*. 1987;24:127-31.
  116. Matthews, KA, Stoney CM. Influences of sex and age on cardiovascular responses during stress. *Psychosom Med*. 1988;50:46-56.
  117. Prior J, Vigna Y, Schechter M, Burgess A. Spinal bone loss and ovulatory disturbances. *N Engl J Med*. 1990;323:1221-7.
  118. Kaplan JR, Adams MR, Clarkson TB, et al. Psychosocial factors, sex differences, and atherosclerosis: lessons from animal models. *Psychosom Med*. 1996;58:598-611.
  119. Berga SL. Behaviorally induced reproductive compromise in women and men. *Semin Reprod Endocrinol*. 1997;15:47-53.
  120. Krantz DS, McCeney MK. Effects of psychologic and social factors on organic disease: A critical assessment of research on coronary heart disease. *Annu Rev Psychol*. 2002;53:341-69.
  121. Rozanski A, Blumenthal JA, Kaplan J. Impact of psychologic factors on the pathogenesis of cardiovascular disease and implications for therapy. *Circulation*. 1999;99:2192-217.
  122. Musselman DL, Tomer A, Manatunga AK, et al. Exaggerated platelet reactivity in major depression. *Am J Psychiatry*. 1996;153:1313-7.
  123. Bjorntorp P. Visceral fat accumulation: the missing link between psychosocial factors and cardiovascular disease? *J Intern Med*. 1991;230:195-201.
  124. Meilahn E. Hormonal milieu and heart disease. In Goldman MB, Hatch MC, eds. *Women's Health*. San Diego: Academic Press, 2000:782-8.
  125. Tunstall-Pedoe H. Myth and paradox of coronary risk and the menopause. *Lancet*. 1998;351:1425-7.
  126. Matthews KA, Meilahn E, Kuller LH, et al. Menopause and risk factors for coronary heart disease. *N Engl J Med*. 1989;321:641-6.
  127. Colditz GA, Willett WC, Stampfer MJ, et al. heart disease in women. *N Engl J Med*. 1987;316:1105-10.
  128. Joakimsen O, Bonna KH, Stensland-Bugge E, Jacobsen BK. Population-based study of age at menopause and ultrasound-assessed carotid atherosclerosis: The Tromso Study. *J Clin Epidemiol*. 2000;53:525-30.
  129. Punnonen R, Jokela H, Aine R, et al. Impaired ovarian function and risk factors for atherosclerosis in premenopausal women. *Maturitas*. 1997;27:231-8.
  130. La Vecchia C, Decarli A, Franceschi S, et al. Menstrual and reproductive factors and the risk of myocardial infarction in women under fifty-five years of age. *Am J Obstet Gynecol*. 1987;157:1108-12.
  131. Hanke H, Hanke S, Ickrath O, et al. Estradiol concentrations in premenopausal women with coronary heart disease. *Coron Artery Dis*. 1997;8:511-5.
  132. Gorgels WJ, van der Graaf Y, Blankenstein MA, et al. Urinary sex hormone excretions in premenopausal women and coronary heart disease risk: a nested case-referent study in the DOM cohort. *J Clin Epidemiol*. 1997;50:275-81.
  133. World Health Organization. Assessment of fracture risk and its application to screening for postmenopausal osteoporosis. WHO Technical Report Series. Vol. 843. Geneva: WHO, 1994.
  134. Ross PD. Osteoporosis. Frequency, consequences, and risk factors. *Arch Intern Med*. 1996;156:1399-411.
  135. Mazess RB, Barden H, Ettinger M, Schultz E. Bone density of the radius, spine, and proximal femur in osteoporosis. *J Bone Min Res*. 1988;3:13-8.
  136. Melton LJ 3rd, Kan SH, Wahner HW, Riggs BL. Lifetime fracture risk: an approach to hip fracture risk assessment based on bone mineral density and age. *J Clin Epidemiol*. 1988;41:985-94.
  137. Eriksen EF, Colvard DS, Berg NJ, et al. Evidence of estrogen-receptors in normal human osteoblast-like cells. *Science*. 1988;241:84-6.
  138. Lam SY, Baker HW, Seeman E, Pepperell RJ. Gynaecological disorders and risk factors in premenopausal women predisposing to osteoporosis. A review. *Br J Obstet Gynaecol*. 1988;95:963-72.

- Reproductive correlates of bone mass in elderly women. Study of Osteoporotic Fractures Research Group. *J Bone Min Res.* 1993;8:901-8.
140. Lee SJ, Kanis JA. An association between osteoporosis and premenstrual symptoms and postmenopausal symptoms. *Bone Miner.* 1994;24:127-34.
141. Nguyen TV, Jones G, Sambrook PN, et al. Effects of estrogen exposure and reproductive factors on bone mineral density and osteoporotic fractures. *J Clin Endocrinol Metab.* 1995;80:2709-14.
142. Waller K, Reim J, Fenster L, et al. Bone mass and subtle abnormalities in ovulatory function in healthy women. *J Clin Endocrinol Metab.* 1996;81:663-8.
143. Cooper GS, Sandler DP. Long-term effects of reproductive-age menstrual cycle patterns on peri- and postmenopausal fracture risk. *Am J Epidemiol.* 1997;145:804-9.
144. Nicodemus K, Folsom A, Anderson K. Menstrual history and risk of hip fractures in postmenopausal women: the Iowa women's health study. *Am J Epidemiol.* 2001;153:251-5.
145. Halbreich U, Rojansky N, Palter S, et al. Decreased bone mineral density in medicated psychiatric patients. *Psychosom Med.* 1995;57:485-91.
146. Michelson D, Stratakis C, Hill L, et al. Bone mineral density in women with depression. *N Engl J Med.* 1996;335:1176-81.
147. Whooley MA, Browner WS. Association between depressive symptoms and mortality in older women. Study of Osteoporotic Fractures Research Group. *Arch Intern Med.* 1998;158:2129-35.
148. Schweiger U, Weber B, Deuschle M, Heuser I. Lumbar bone mineral density in patients with major depression: evidence of increased bone loss at follow-up. *Am J Psychiatry.* 2000;157:118-20.
149. Dinan TG. The physical consequences of depressive illness. *Br Med J.* 1999;318:826.
150. Umberson D, Williams K, Sharp S. Medical sociology and health psychology. In Bird C, Conrad P, Freemont A, eds. *Handbook of Medical Sociology.* NY: Plenum, 2000:353-64.
151. Sarafino EP. *Health Psychology: Biopsychosocial Interactions.* New York: Wiley, 1998.
152. Uchino BN, Cacioppo JT, Kiecolt-Glaser JK. The relationship between social support and physiological processes: a review with emphasis on underlying mechanisms and implications for health. *Psychol Bull.* 1996;119:488-531.
153. Kiecolt-Glaser JK, Newton TL. Marriage and health: his and hers. *Psychol Bull.* 2001;127:472-503.
154. Taylor SE, Klein LC, Lewis BP, et al. Biobehavioral responses to stress in females: tend-and-befriend, not fight-or-flight. *Psychol Rev.* 2000;107:411-29.
155. Gilligan C. In *a Different Voice: Psychological Theory and Women's Development.* Cambridge, MA: Harvard University Press, 1982.