SOCIAL, BEHAVIORAL, AND BIOLOGICAL FACTORS, AND SEX DIFFERENCES IN MORTALITY*

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Few studies have examined whether sex differences in mortality are associated with different distributions of risk factors or result from the unique relationships between risk factors and mortality for men and women. We extend previous research by systematically testing a variety of factors, including health behaviors, social ties, socioeconomic status, and biological indicators of health. We employ the National Health and Nutritional Examination Survey III Linked Mortality File and use Cox proportional hazards models to examine sex differences in adult mortality in the United States. Our findings document that social and behavioral characteristics are key factors related to the sex gap in mortality. Once we control for women's lower levels of marriage, poverty, and exercise, the sex gap in mortality widens; and once we control for women's greater propensity to visit with friends and relatives, attend religious services, and abstain from smoking, the sex gap in mortality narrows. Biological factors—including indicators of inflammation and cardiovascular risk—also inform sex differences in mortality. Nevertheless, persistent sex differences in mortality remain: compared with women, men have 30% to 83% higher risks of death over the follow-up period, depending on the covariates included in the model. Although the prevalence of risk factors differs by sex, the impact of those risk factors on mortality is similar for men and women.

Women generally live longer than men, and although research has consistently shown that the U.S. sex gap in mortality has fluctuated over time, much of the literature has overlooked important covariates and has not examined recent demographic patterns. One of the main objectives of the *Healthy People 2010* public health initiative is to eliminate health disparities, with a specific emphasis on sex differences in health (U.S. Department of Health and Human Services [U.S. DHHS] 2000). We use hazard models and a current nationally representative data set to examine numerous factors that affect sex differences in adult mortality in the United States. Specifically, we seek to explain sex differences in mortality by (1) accounting for differences in the distribution of both social and biological protective and risk factors by sex, including socioeconomic status (SES), social relationships, health behaviors, and biological indicators of health; (2) examining whether specific protective and risk factors have unique relationships with mortality among men and women; (3) determining sex differences in cause-specific mortality; and (4) estimating potential sex-specific gains with cause-elimination models.

Both sexes have experienced tremendous but uneven increases in life expectancy at birth over the past century. Between 1900 and 2005, male U.S. life expectancy rose from

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46.3 to 75.2 years, and female life expectancy rose from 48.3 to 80.4 years (Arias 2007; Kung et al. 2008). From 1920, when the sex gap in life expectancy at birth was just 1.0 year, the sex gap slowly increased to a peak of 7.8 years in 1975 and again in 1979 (Arias 2007). Thus, the proliferation of articles published in the mid-1980s—on the heels of this peak—should come as no surprise (Nathanson 1984; Waldron 1986; Wingard 1982). Since 1979, the U.S. sex gap in life expectancy at birth has steadily declined to 5.2 years in 2005, the lowest level in nearly 60 years (Kung et al. 2008). The persistence of the sex gap in mortality, even at the reduced level, coupled with the potential for further narrowing and for additional mortality improvement among both sexes, warrants additional research.

Wingard (1982) used Alameda County data to find that compared with females aged 30–69, similarly aged males had a 50% higher risk of death over the nine-year follow-up period in the baseline model. Adjusting for risky behaviors (including smoking and alcohol consumption) reduced the sex gap, but adjusting for physical activity and marital status increased the gap. Adjusting for all factors in the multivariate model increased the relative risk of death for males from 50% to 70%. But these relationships vary across analytic samples and calendar periods. Wingard, Suarez, and Barrett-Connor (1983) employed a sample from Rancho Bernardo, California, an upper-middle-class community, to demonstrate that the sex gap in mortality closed from 70% to 30% over the follow-up period once they controlled for age; behavioral factors; and biological characteristics, including systolic blood pressure and cholesterol. Rogers, Hummer, and Nam (2000) used the 1990 National Health Interview Survey to demonstrate that among adults aged 18 and older, and compared with females, mortality among males was 73% higher in the baseline model that controlled for age and race/ethnicity, twice as high in a model that additionally controlled for other social and economic factors, but 75% higher in the full model that also controlled for health behaviors over the five-year follow-up period.

Examining sex differences in cause-specific mortality provides added insight into the mechanisms that create the sex gap in overall mortality. Compared with females, males experience higher risk of death from almost all causes (Kalben 2000; Kung et al. 2008). The National Center for Health Statistics (NCHS) reported that in 2005, males had higher age-adjusted death rates for 12 of the top 15 causes of death. The male-to-female ratio in the age-adjusted death rates, which was 1.4 for all causes, was similar for cerebrovascular diseases and essential (primary) hypertension and hypertensive renal disease (1.0); lower for Alzheimer's disease (0.7); and largest for chronic liver disease and cirrhosis (2.1) and external causes, including accidents (2.2), homicides (3.8), and suicides (4.1). Even though the sex ratio for diseases of the heart was just 1.4, this cause of death is a major contributor to sex differences in mortality because it is the primary cause of death and accounts for over a quarter of all deaths (Kung et al. 2008). Because males experience higher mortality risk than females from most causes of death, eliminating certain causes often generates greater male than female gains in life expectancy at birth. For example, life expectancy at birth could increase by 0.4 years for males and 0.1 years for females through the elimination of human immunodeficiency virus (HIV), by 1.0 year for males and 0.7 years for females through the elimination of lung cancer, and by 1.2 years for males and 0.6 years for females through the elimination of accidents and adverse effects (Anderson 1999).

Both social/environmental and biological factors have been invoked to explain the sex gap in mortality.[1] Social perspectives tend to emphasize the importance of social relationships, health-related behaviors, and socioeconomic factors in accounting for sex differences in survival, whereas biological models emphasize the role of biological markers,

1. Researchers often distinguish between sex differences, which are biologically determined, and gender differences, which are socially and culturally constructed (Rieker and Bird 2000). Although we examine social, cultural, economic, and behavioral factors, we do not have direct measures of gender roles and therefore focus on sex differences.

hormones, and genetics on health outcomes. Biological research has offered little insight into why sex differences vary over time and place and by various social categories. Similarly, past demographic research has focused primarily on social characteristics and has often inadequately controlled for biological risk factors. Rather than juxtaposing opposing biological and social characteristics approaches, we maintain that the sex gap in mortality is best explained by examining differential distributions of demographic, social, SES, health behaviors, *and* biological factors by sex. The following sections highlight the relationships between demographic characteristics, social relationships, SES, health behaviors, and biological factors on sex differences in mortality.

SOCIAL AND BEHAVIORAL FACTORS

Age shapes social status, social relations, and behaviors. Sex differences in mortality are greatest among younger adults (Kung et al. 2008), in part because young males are more apt to engage in risky and aggressive behaviors that generally attenuate with age. Nevertheless, sex differences in mortality persist even among older individuals. Fried et al. (1998) used the Cardiovascular Health Study to show that among individuals aged 65 and older, males were more than twice as likely as females to die over the five-year follow-up period, even net of other control variables.

Higher SES increases access to health insurance and health care, knowledge, social connections, social support, and safer neighborhoods, and creates a buffer against financial hardship (Kalben 2000; Passannante and Nathanson 1985). Although men are more likely to be employed and earn higher incomes, sex gaps in SES have continued to narrow with time (Schnittker 2007). Between 1980 and 2006, among full-time workers, women's earnings as a percentage of men's earnings increased from 64% to 80% (U.S. Department of Labor 2008). Much of women's improvement in health over the past several decades is due to gains in SES, especially increasing education, and increasing returns to women's education, such as better earnings and employment prospects, higher occupational statuses, and lower odds of falling into poverty (DiPrete and Buchman 2006; Schnittker 2007).

Rich, intricate, and strong prosocial relationships buffer against mortality risk through social integration and regulation (Durkheim [1897] 1951; Moen, Dempster-McClain, and Williams 1989). Marriage is linked to greater social, emotional, instrumental, and financial support; increased compliance with medical regimens; less depression and anxiety; eating balanced and healthy meals; regular sleep habits; and healthier behaviors (Ross, Mirowsky, and Goldsteen 1990). Married individuals are less likely than unmarried individuals to smoke, drink excessive amounts of alcohol, use illicit drugs, drive recklessly, and engage in violence (Umberson 1992). Married individuals also benefit from higher incomes, more stable employment, safer neighborhoods, greater social prestige, and increased access to private health insurance (South and Crowder 2000; Umberson 1992). Although both sexes benefit from the financial security of marriage, the income benefit is greater for females than for males (Ross et al. 1990; Waite and Gallagher 2000).

Historically, the mortality gap between the married and the nonmarried has been higher for men than for women (Ross et al. 1990; Waite and Gallagher 2000). This is attributed to unmarried men's dangerous activities and risky lifestyles and the healthier lifestyles of women of all marital statuses. Over time, the adverse health associated with marital dissolution has increased more for women than for men, although widowhood and divorce remain more detrimental for men than for women, potentially because men rely more heavily on their spouses for their social ties, whereas women more often accumulate social ties both through and outside of marriage (Liu and Umberson 2008; Nathanson 1984; Umberson et al. 1996).

Religious participation also reduces mortality risk, in part through social, emotional, financial, and instrumental support; social integration; and social regulation (Hummer et al. 2004; McCullough et al. 2000). Attendance at religious services is associated with lower

levels of smoking and drinking and more extensive and positive social connections, although religious attendance is also more common among those with fewer years of education (Hummer et al. 1999). Females are more likely than males to be involved in religious activities. Other forms of social support that increase survival prospects include contacts with friends and club membership (Berkman and Glass 2000). Compared with females, males are more likely to be involved in clubs but less likely to be involved with friends and family.

For decades, women have been more likely than men to engage in such preventive health behaviors as annual doctor visits for routine physical checkups, whereas men have been more likely to partake in excessive drinking, drunken driving, illegal drug use, physical fights and violence, and high tobacco consumption (Kalben 2000; Pampel 2002, 2005; Rieker and Bird 2000; Rogers et al. 2000; Waldron 1985; Waldron, McCloskey, and Earle 2005). As women's behavior has become less restricted and as more women enter and remain in the labor force, there has been a convergence in unhealthy behaviors, which a United Nations publication (1991:56) labeled "the dark side of equality."

Cigarette consumption, the single most important preventable cause of death in the United States, is one of the greatest contributors to sex differences in mortality (Pampel 2002; Preston and Wang 2006; Retherford 1972; Rogers et al. 2005). The sex ratio (male/female) in U.S. adult cigarette smoking rates declined from 2.1 in 1955 (Kalben 2000) to 1.3 in 2003 (Centers for Disease Control and Prevention [CDC] 2008). Men have a higher rate of ever smoking, and the pattern for women lags that of men by a decade or two and peaks at a lower level (Pampel 2002). Compared with women, men's greater exposure to smoking for longer periods of time contributes to a number of smoking-related diseases—including lung cancer, emphysema, and cardiovascular disease—increasing the risk of overall mortality (Case and Paxson 2005). The faster declines in smoking among men than women in recent decades have lead to a convergence in smoking patterns by sex: in 2007, 22% of adult males and 17% of adult females were current cigarette smokers (CDC 2008). The closing sex gap in smoking not only reduces the sex gap in mortality but also reduces cigarettes' impact on the sex gap (Pampel 2005). Retherford (1972) determined that among individuals aged 37 and older, cigarette smoking contributed to 47% of the sex gap in life expectancy in 1962 and to 75% of the increase in that gap between 1910 and 1962. Rogers et al. (2000) used U.S. data from 1990–1995 to show that cigarette smoking contributed to 25% of the sex gap in overall mortality. Furthermore, Preston and Wang (2006) examined smoking histories of males and females to conclude that the sex gap in mortality will continue to close in the coming decades.

The association between alcohol consumption and mortality is complex. A J-shaped curve best fits the relationship, with both abstainers and heavy drinkers experiencing higher mortality than light to moderate drinkers (Klatsky and Udaltsova 2007). Even though excessive drinking is linked to higher mortality, self-reports of excessive drinking are very low and thus may not correlate highly with sex differences in mortality (Nathanson 1984). Although males are more likely than females to be light to moderate drinkers, which confers some health and longevity benefits, they are also more likely to drink excessively, which may lead to higher male mortality. Heavy drinking results in increased mortality from several causes of death, including some cancers, chronic alcoholism, cirrhosis of the liver, and external causes (see Corrao et al. 2004).

Increased levels of physical activity are associated with increased survival (Fried et al. 1998; Paffenbarger et al. 1986). Compared with females, males are generally less likely to be obese and more likely to exercise (Caspersen, Pereira, and Curran 2000; National Center for Health Statistics [NCHS] 2007a; Wingard 1982). In 1997, 43% of women but just 36% of men engaged in no leisure-time physical activity (U.S. DHHS 2000). Recent research has highlighted the importance of examining sex differences in exercise and fitness as opposed to obesity because physical fitness is a better predictor of mortality, the body mass index does not directly measure body fat, and the relationship between body mass and mortality

has diminished over time (Flegal et al. 2005). It is also important to adjust for functional impairment when modeling sex differences in mortality because, in addition to higher reported rates of disability among females, restrictions in basic movement can constrain physical and social activity; strain marriages; limit employment; increase the risk of accidents, including falls; and increase the risk of chronic conditions and overall mortality (Alley and Chang 2007; Freedman, Martin, and Schoeni 2002; Fried et al. 1998; Jagger et al. 2007).

Social characteristics are important, but sex differences in mortality persist and in some instances widen in select populations, wherein both sexes engage in healthy behaviors and have more similar SES (see Lyon et al. 1978; Merrill and Lyon 2005). Thus, while sex differences in mortality are partly due to social characteristics, we must also consider biological factors.

BIOLOGICAL FACTORS

Demographers have increasingly recognized the importance of inflammation and cardiovascular risk factors on population health (Crimmins et al. 2003). Cardiovascular risk factors such as blood pressure, cholesterol levels, and glycosylated hemoglobin are key predictors of cardiovascular disease mortality, the leading cause of death for both men and women in the United States. Hypertension, or high blood pressure, affects about a third of the U.S. adult population and contributes to an increased risk of circulatory diseases, disability, and overall mortality (Fields et al. 2004; Wingard et al. 1983). Hypertension is more common among men than women in youth and middle age but is more frequent among women than among men at older ages (Waldron 1995).

Glycosylated hemoglobin provides information about an individual's long-term glucose control (Nathan et al. 1984) and has been linked to increased risk of cardiovascular diseases in multiple studies (Selvin et al. 2004). Total cholesterol is also associated with higher rates of coronary heart disease and mortality (Verschuren et al. 1995) and has a U-shaped relationship with mortality (Jacobs et al. 1992). Cholesterol levels have improved for both sexes in recent years, in part due to improved diets and the widespread availability of cholesterol-lowering drugs (Gregg et al. 2005). While these two factors may be related to coronary heart disease, it has been suggested that glycosylated hemoglobin is independently associated with the risk of death (Slevin et al. 2004).

Although short-term inflammation helps the body deal with acute infections or trauma, social stressors and negative affect can lead to persistent inflammation and increased levels of cardiovascular disease, arthritis, diabetes, Alzheimer's disease, some cancers, and ultimately, death (Kiecolt-Glaser et al. 2002). Low albumin concentration, a marker of inflammation, has been shown to be significantly associated with increased risk for functional decline (Visser et al. 2005) and mortality (Goldwasser and Feldman 1997). High levels of C-reactive protein (CRP) reduce the body's ability to heal wounds and fight viruses, and are associated with both medical and behavioral risk factors such as smoking, low levels of exercise, obesity, diabetes, high blood pressure, heart attack, and stroke (Ridker 2003). A meta-analysis summarized data from multiple prospective studies and found an increased risk for coronary heart disease among individuals in high-risk groups for CRP (with an odds ratio [OR] of 1.7) and albumin (OR = 1.5), compared with individuals in the low-risk groups (Danesh et al. 1998).

Although there are established connections between mortality and cardiovascular risk factors and inflammation, little work has specifically examined whether these factors might contribute to sex differences in mortality in the U.S. population, especially within the context of a broader set of social, socioeconomic, and behavioral risk factors.

AIMS

The primary goal of this article is to determine which social and biological factors are associated with increases or decreases in the sex gap in U.S. adult mortality. We expect that

controlling for women's lower rates of risky behaviors but greater social connections will reduce the sex gap in mortality. On the other hand, controlling for women's lower SES, lower rates of exercise, and higher rates of disability should widen the sex gap in mortality. Next, we determine whether these relationships are affected by specific age groups or by sex-specific interactions (that is, whether some factors are riskier for men than for women). Our cause-specific analyses reveal how much of the sex gap is linked to a few specific causes, including HIV, cirrhosis of the liver, and external causes of death. Last, we calculate potential reductions in the sex gap through cause-elimination models.

DATA AND METHODS

Data

We employ the National Health and Nutritional Examination Survey (NHANES III) Linked Mortality File (NCHS 2007b), a valuable demographic resource that is ideal for our analyses because the data are current and nationally representative, and include a long (up to 13-year) follow-up of survival status, sociodemographic indicators as well as self-reported and clinical measures of health status, and a substantial number of deaths. NHANES III uses a multistage sampling frame to draw a nationally representative sample from the noninstitutionalized U.S. population. Because of the substantial amount of detailed information obtained from interviews, physical examinations, and laboratory tests, the NCHS spent six years (1988–1994) collecting NHANES III data. Both non-Hispanic blacks and Mexican Americans were oversampled in the survey. Individuals were asked to complete a home interview that was administered by trained bilingual interviewers, take a follow-up medical examination, and provide blood and urine samples for laboratory analyses. Seventy-seven percent of all persons who participated in the home interview also completed the laboratory exam. Those aged 17 and older completed individual and family questionnaires (NCHS 1994).

NHANES III is linked to the National Death Index (NDI) through the year 2000 via a computer matching algorithm that examines combinations of social security number; day, month, and year of birth; first and last name; middle initial; and father's surname (NCHS 2005). Although the NHANES III files are linked to the NDI beginning at age 17, our sample is limited to individuals who were aged 20 and older at the time of the interview and were matched to mortality records (n = 17,017; 2,857 deaths). Because NHANES III participants who were not determined to have "true" matches with the NDI are assumed to be alive, there exists the potential for underreporting of deaths within the NHANES III sample (NCHS 2005). We drop 230 individuals (1.35%), of whom 67 have died, because of missing information on the variables included in the analysis, resulting in a final sample size of 16,787 individuals and 2,790 deaths.

Variables and Measurement

The key independent variable, sex, is coded as 0 (female) and 1 (male). Race/ethnicity, based on self-identification, is coded as non-Hispanic black and nonblack (referent). SES includes poverty and education. We code education into 12 or fewer, 13 to 15, and 16 or more years of education (referent). The poverty-income ratio is computed by NHANES as the ratio of family income to the Census Bureau–defined poverty threshold, which varies across the number and age of family members in each calendar year. To retain observations, we imputed missing poverty-income ratio information with linear regression, and incorporated random variation (drawn from the residuals) into the estimates to better reflect our uncertainty about the missing values (Gelman and Hill 2007).

Social relations are assessed with four variables. Marital status is coded as currently married (referent), widowed, separated or divorced, and never married. We distinguish between widowed and separated or divorced because of the substantial sex differences in

these categories. Visiting with friends or relatives is coded as once or more per week (referent) versus none. Religious attendance is coded in a series of dummy variables that identify whether, in the past year, persons have never attended church, have attended at least once but less than once per week, weekly, or have attended more than once per week (referent). Individuals are coded as belonging (referent) or not belonging to clubs and organizations.

Health behaviors and conditions include cigarette smoking, alcohol consumption, physical activity, and physical impairment. Smoking status is coded categorically as never, former, or current smokers. Individuals are defined as never drinkers if they have consumed fewer than 12 drinks in their lifetime, as former drinkers if they have consumed at least 12 drinks in their lifetime but have had fewer than 12 drinks in the last year, and as current drinkers if they have had at least 12 drinks in the past year. We subdivide current drinkers into those who average 1–4, and those who drink 5 or more drinks per day on the days that they drink. Physical activity is classified as more than 7 hours per week of exercise (referent), 7 or fewer hours per week, or not at all. Functional impairment is coded dichotomously as no impairment (referent) or 1 or more impairments, as indicated by difficulty or an inability to walk one-quarter mile; walk up 10 steps without resting; walk from one room to another on the same floor; stoop, kneel, or crouch; lift or carry 10 pounds; or stand from an armless chair (for similar coding, see Alley and Chang 2007).

Biological factors include inflammation, hypertension, cholesterol, and glycosylated hemoglobin. We assess inflammation through albumin (greater than 4.5 g/dL [referent], greater than 4.0 to less than or equal to 4.5 g/dL, greater than 3.5 to less than or equal to 4.0 g/dL, and less than or equal to 3.5 g/dL) and CRP (less than 0.3 mg/dL [referent] compared with greater than or equal to 0.3 mg/dL), following the American Heart Association and Centers for Disease Control and Prevention recommendations (Pearson et al. 2003). Because individuals who are fighting an existing infection will have artificially low albumin and high CRP levels, we created a separate category: missing because of infection. We do not include fibrinogen, even though other studies have demonstrated its importance (see Danesh et al. 1998), because NHANES III collected that information only for individuals aged 40 and older.

Hypertension is calculated from the average of up to six blood pressure measurements taken during the examination. Following the guidelines of the Joint National Committee on Prevention (U.S. DHHS 2003), we code the hypertension variable as normotensive, or normal blood pressure (less than 120 systolic blood pressure [SBP] and less than 80 diastolic blood pressure [DBP], referent), prehypertensive (120–139 SBP or 80–89 DBP), hypertensive stage 1 (140–159 SBP or 90–99 DBP), and hypertensive stage 2 (SBP of 160 or more, DBP of 100 or more, or currently taking antihypertensive medication).

Total cholesterol is coded as a series of dummy variables to capture the U-shaped relationship between total cholesterol and mortality. Individuals are coded as having total cholesterol levels less than 150, 150 to 249 (referent), 250 to 349, and greater than or equal to 350. We also include glycosylated hemoglobin (less than 6.4% [referent] compared with 6.4% or more; for similar coding, see Osei et al. 2003). To retain the entire sample, we include missing values on biomarker data. Although the percentage of individuals with missing biomarker information is quite small, missing values may indicate information that was collected but deemed unusable, additional risk factors (e.g., frail and sick individuals may have been unable to complete parts of the examination), or refusals to provide samples (NCHS 1994).

Analyses of Overall and Cause-Specific Mortality

The dependent variable for our analyses is dichotomous: survival versus death. Following Kom, Graubard, and Midthune (1997), we use age to indicate the time to death, which ensures that our mortality analyses are age-adjusted. Respondents enter the sample at age 20 or older, and they increase in age over the follow-up period. Because age has

such a profound effect on mortality and on sex differences in mortality, we disaggregate our analyses by age at baseline. We use conventional cutpoints and focus on those aged 20–44, who are moving into the labor force, completing their education, and getting married; those aged 45–64, who are likely to be fully engaged in the labor force but who may be beginning to experience age-related declines in poor health; and those aged 65 and older, who may be retiring and experience substantially higher risks of death. One distinct advantage of prospective data sets is that there is a clear temporal order: all covariates are measured before death (Bhrolcháin and Dyson 2007). Individuals who survived the entire follow-up period are right-censored.

We expect greater sex differences in mortality due to external causes, infectious and parasitic diseases (including HIV), and chronic liver disease and cirrhosis; substantial differences in mortality from circulatory diseases; and more modest differences in cancer deaths (Kung et al. 2008). NCHS recoded all deaths in the NHANES III Linked Mortality File to the 10th revision of the *International Classification of Diseases* (ICD-10), the most current classification scheme (World Health Organization 2007). We examine seven major causes of death: diseases of the circulatory system (ICD10 I00–I78, I80–I99); malignant neoplasms (ICD10 C00–C97); infectious and parasitic diseases (ICD10 A00–B99); diseases of the respiratory system, including acute upper respiratory infections, influenza and pneumonia, and chronic lower respiratory diseases (ICD10 J00–J98); chronic liver disease and cirrhosis (ICD10 K70, K73–K74); external causes, including accidents, suicides, and homicides (ICD10 U01–U03, V01–Y09, Y85–Y86, Y87.0, and Y87.1); and all other causes of death (residual), which include "events of undetermined intent" and "complications of medical and surgical care."

Although limited by the modest number of deaths, we are able to examine heart (ICD10 I00–I09, I11, I13, and I20–I51) and cerebrovascular diseases (ICD10 I60–I69) separately from diseases of the circulatory system; malignant neoplasms of the trachea, bronchus, and lung (or lung cancer, for brevity; ICD10 C33–C34) separately from all cancers; HIV (ICD10 B20–B24) separately from infectious and parasitic diseases; pneumonia (ICD10 J12–J18) and chronic lower respiratory diseases (ICD10 J40–J47) separately from diseases of the respiratory system; and accidents (ICD10 V01–X59, Y85–Y86) and suicides and homicides (ICD10 X60–Y09, Y87.0–Y87.1) separately from all external causes. Furthermore, we were able to examine motor vehicle accidents and falls (ICD10 W00–W19) separately from all accidents. It is important to examine lung cancer separately because a very high proportion of all lung cancer deaths are due to smoking (Pampel 2002; Preston and Wang 2006) and because 90% of the sex differences in lung cancer mortality may be due to smoking (Waldron 1986). In models of cause-specific mortality, respondents are right-censored when dying of other causes or at the end of the follow-up period.

Compared with cause-specific analyses, cause-elimination models can be advantageous because they (1) retain the large base (most causes of death) and thus produce stable estimates, (2) can reveal the cumulative effects of the elimination of multiple causes of death, (3) document the share of the sex gap in overall mortality that results from rare causes of death that are much more common among one sex than the other, or common causes of death that exhibit more subtle differences by sex, and (4) can show the impact of predominately sex-specific causes, such as prostate and breast cancer. Unlike traditional cause-elimination models, which mathematically show the potential life expectancy gains associated with the elimination of a particular cause (see Anderson 1999), our cause-elimination models demonstrate the possible reduction in the sex gap in mortality through the statistical elimination (right-censoring) of particular causes within a multivariate framework. Because males are more likely than females to die from most causes, our models statistically eliminate causes of death that would create the largest reduction of the sex gap in mortality.

We use Cox proportional hazards models to examine the risk of death (Allison 1984). We use Stata 10.0 (StataCorp 2007) to produce descriptive and multivariate results, adjusted

for the sample weights and the complex sampling frame of NHANES III using the Stata "svy" commands. We test for, but do not find, significant calendar year or follow-up year effects on overall mortality or on the sex gap in mortality within the multivariate analyses. Although NCHS designed NHANES III so that the first three years, the last three years, and the entire period are all national probability samples, we follow NCHS recommendations and analyze the entire six-year period (NCHS 1994).

We employ a model-building strategy that begins with the baseline model and progressively adds covariates for different sets of social characteristics, including SES, social relations, health behaviors, and health conditions. We then test for interactions, differences by age, and differences by cause of death. A significant interaction would indicate that a given factor (such as education) confers different survival advantages for men than for women. Separate analyses by age will reveal whether the sex gap persists for all ages and, if so, whether the gap within age groups closes with controls for covariates.

RESULTS

Table 1 presents some striking differences in the distributions of the covariates by sex and broad age group. Compared with males, females tend to have lower SES, especially at older ages. Compared with young adult males, young adult females are much more likely to be divorced or widowed. Among those aged 65 and older, only 43% of women but 78% of men are married, although females are more likely than males to attend religious services. All females, but especially older females, are more likely than their male counterparts to have never smoked cigarettes and to have never consumed alcohol. Nevertheless, more men than women exercise regularly and experience no functional impairment. Compared with men, women have higher levels of inflammation, as measured by albumin and CRP. Compared with women, men are more likely to be prehypertensive and hypertensive stage 1 at all ages. But at the older ages, women exhibit higher rates of stage 2 hypertension (for similar results, see Waldron 1995). Compared with men, a slightly greater percentage of women have cholesterol levels of 350 or higher, but a slightly smaller percentage of women have hemoglobin levels of 6.4% or higher.

Table 2 presents hazard ratios of the covariates. Compared with females, males are 49% more likely to die over the follow-up period, with controls for race/ethnicity (baseline Model 1). Higher levels of education, lower levels of poverty, and being married are each associated with lower risks of death (Model 2). Controlling for marital and socioeconomic status increases the sex gap in mortality from 49% to 63% (compare Models 1 and 2), a difference that can be attributed largely to men's higher levels of education, higher incomes, and higher propensities to be married (see Table 1). Adjusting for visits with friends and attendance at religious services—activities that are higher among women—substantially reduces the sex gap in mortality from 63% to 55% (Models 2 and 3). Adding a control for club membership does not further affect the sex gap in mortality (compare Models 3 and 4).

Females live longer than males, in part owing to their lower cigarette consumption. With controls for smoking, the sex gap declines from 55% to 43% (Models 4 and 5). Because males are more physically active than females, adjusting for physical activity (Model 6) increases the sex gap in mortality. Although former and heavy drinking places individuals at higher mortality risk, light to moderate alcohol consumption bestows survival advantage. Thus, controlling for males' greater likelihood to drink moderately reduces their mortality and thereby increases the sex gap in mortality (compare Models 5 and 6).

Controlling for functional impairment—which is more common among women—widens the sex gap in mortality. This measure of functional limitation also partially captures the selection of healthier individuals into health-promoting roles and behaviors. Controlling for functional limitations slightly reduces but does not eliminate the significant association between marital status, religious attendance, and physical activity and mortality.

Table 1. Descriptive Statistics of Covariates by Sex and Age: U.S. Adults Aged 20 and Older, 1988–1994

	All Ages		Ages 20–44		Ages 45–64		Age 65 and Older	
Variable	Male (%)	Female (%)	Male (%)	Female (%)	Male (%)	Female (%)	Male (%)	Female (%)
Demographic Factors								
Race/ethnicity (nonblack)	89.5	87.7	88.1	85.9	91.5	89.5	92.1	91.4
Non-Hispanic black	10.5	12.3	11.9	14.1	8.5	10.5	7.9	8.6
Socioeconomic Status								
Education (≥ 16 years)	23.6	18.0	23.5	21.3	26.4	15.5	18.6	9.7
≤ 12 years	56.3	60.5	52.8	53.5	57.2	67.0	69.6	75.5
13–15 years	20.1	21.5	23.7	25.1	16.4	17.5	11.7	14.9
Poverty-income ratio (mean)	3.6	3.4	3.4	3.3	4.1	3.9	3.6	3.1
Social Relations								
Marital status (married)	71.2	62.1	64.6	63.8	82.6	70.2	78.3	42.8
Divorced/separated	7.9	13.7	6.9	14.3	11.4	16.3	5.2	7.1
Never married	18.6	14.3	28.3	21.3	4.3	4.4	3.8	4.9
Widowed	2.3	9.9	0.2	0.7	1.6	9.2	12.7	45.2
Visit with friends/relatives (≥ 1 visit per week)	95.6	95.7	96.5	96.4	95.4	94.8	92.2	94.9
No visits	4.4	4.3	3.5	3.6	4.6	5.2	7.8	5.1
Religious attendance (> once per week)	7.6	9.7	5.7	8.2	11.3	11.6	9.0	12.3
No attendance	44.7	33.7	46.2	35.0	43.6	33.6	40.9	29.9
< Once per week	25.3	26.4	28.7	31.7	24.0	21.4	16.9	16.5
Once per week	22.4	30.1	19.3	25.3	21.2	33.5	33.2	41.4
Club involvement (yes)	60.3	64.1	64.9	68.5	52.6	59.5	55.5	55.6
Health Behaviors								
Smoking status (never)	37.0	55.0	43.2	54.9	27.7	50.8	27.9	62.1
Former	30.8	19.5	19.9	15.0	42.0	24.7	56.8	27.7
Current	32.2	25.6	37.0	30.2	30.2	24.6	15.3	10.3
Drinks per day (1–4)	47.9	38.3	49.4	44.4	48.4	34.9	40.6	21.7
Never	5.9	18.7	5.1	15.0	6.0	20.6	9.2	29.4
Former	26.3	36.5	19.1	32.1	33.3	40.6	44.1	46.3
≥ 5 drinks	17.7	4.2	24.0	6.3	10.3	2.1	4.5	0.1
Missing	2.2	2.2	2.4	2.2	1.9	1.9	1.6	2.5
Hours of exercise per week (> 7 hours)	36.2	31.8	39.3	36.6	33.7	28.4	28.2	21.3
None	29.1	36.6	25.0	29.8	30.0	38.1	43.8	56.9
≤ 7 hours	34.8	31.6	35.8	33.6	36.3	33.5	28.0	21.8

(continued)

(Table 1, continued)

Variable	All Ages		Ages 20–44		Ages 45–64		Age 65 and Older	
	Male (%)	Female (%)	Male (%)	Female (%)	Male (%)	Female (%)	Male (%)	Female (%)
Impairment								
Functional impairment								
None	54.8	52.8	50.0	49.9	54.0	52.6	77.1	63.7
≥ 1 impairment	5.9	9.7	1.6	2.2	6.7	10.4	22.9	36.3
Missing	39.3	37.5	48.4	47.9	39.3	37.0	0.0	0.0
Inflammation								
Albumin (> 4.5 g/dL)	20.9	6.1	29.9	8.3	10.2	7.6	5.1	1.9
> 4.0 to ≤ 4.5 g/dL	52.0	44.3	51.6	46.1	56.6	45.8	44.8	36.1
> 3.5 to ≤ 4.0 g/dL	18.2	31.1	11.5	27.2	24.7	34.2	33.4	39.2
≤ 3.5 g/dL	1.1	3.9	0.4	3.6	1.4	0.0	3.0	6.0
Missing because of infection	2.7	7.5	1.7	7.3	3.2	7.4	6.0	8.3
Missing	5.1	7.1	5.0	7.6	3.9	5.0	7.7	8.6
CRP (< 0.3 mg/dL)	73.7	62.0	79.6	65.9	69.7	55.1	57.5	55.6
≥ 0.3 mg/dL	13.3	19.9	9.0	15.5	17.1	25.7	23.6	26.0
Missing because of infection	8.4	12.4	6.5	11.6	10.2	14.9	12.4	11.5
Missing	4.6	5.7	4.9	7.0	3.0	4.4	6.4	6.9
Cardiovascular Risk Factors								
Hypertension (normotensive < 120 SBP and < 80 DBP)	62.5	71.4	73.6	89.0	53.6	58.8	36.1	32.2
Prehypertensive (120–139 SBP or 80–89 DBP)	13.5	5.8	15.4	4.9	12.5	8.9	8.1	4.2
Hypertensive stage 1 140–159 SBP or 90–99 DBP	10.2	6.2	7.4	2.6	12.4	8.8	17.6	14.1
Hypertensive stage 2 ≥ 160 SBP or ≥ 100 DBP	13.3	16.3	3.6	3.5	21.3	23.4	37.7	48.0
Missing	0.5	0.3	0.0	0.0	0.2	0.1	0.5	1.5
Total cholesterol 150–249	73.0	68.1	72.4	71.3	74.0	65.9	73.8	59.7
< 150	9.1	8.0	12.3	12.2	3.8	2.4	5.4	1.3
250–349	11.3	14.3	7.9	5.7	18.1	24.0	13.0	29.9
≥ 350	3.4	5.1	3.7	5.5	2.2	4.3	4.1	5.1
Missing	3.2	4.6	3.7	5.2	1.9	3.4	3.8	4.1
Glycosylated hemoglobin < 6.4%	91.7	91.3	95.5	94.1	88.5	89.1	81.5	84.7
≥ 6.4%	5.9	5.1	1.8	1.6	9.9	8.5	15.6	12.0
Missing	2.4	3.6	2.7	4.3	1.5	2.3	2.9	3.2

Source: Derived from NHANES III Linked Adult Interview, Examination, and Laboratory Files.

Table 2. Hazard Ratios for Sex Differences in Mortality: U.S. Adults Aged 20 and Older, 1988–2000

Variable	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 8	Model 9	Model 10	Model 11
Sex (female)	1.49***	1.63***	1.55***	1.55***	1.43***	1.51***	1.58***	1.65***	1.60***	1.54***	1.62***
Demographic Factors											
Race/ethnicity (nonblack)											
Non-Hispanic black	1.51***	1.27**	1.32***	1.30***	1.30***	1.26**	1.26**	1.14†	1.23**	1.20*	1.08
Socioeconomic Status											
Education (≥ 16 years)											
≤ 12 years		1.36**	1.31*	1.27*	1.20†	1.13	1.13	1.14	1.13	1.13	1.13
13–15 years		1.20	1.19	1.19	1.14	1.11	1.11	1.11	1.11	1.12	1.09
Poverty-income ratio		0.92***	0.92***	0.93***	0.93**	0.96*	0.97	0.97	0.97	0.97	0.97
Social Relations											
Marital status (married)											
Divorced/separated		1.49***	1.42***	1.42***	1.36**	1.34**	1.32**	1.31**	1.32**	1.32**	1.31**
Never married		1.22	1.17	1.16	1.20	1.14	1.15	1.15	1.15	1.13	1.15
Widowed		1.14†	1.13	1.12	1.11	1.09	1.07	1.08	1.07	1.07	1.07
Visit with friends/relatives (≥ 1 per week)			1.38**	1.35**	1.35**	1.25*	1.20†	1.20†	1.19	1.17	1.17
Religious attendance (> once per week)											
No attendance			1.60***	1.54***	1.39**	1.35*	1.27†	1.28†	1.26†	1.26†	1.26†
< Once per week			1.30**	1.27*	1.19	1.19	1.15	1.16	1.14	1.12	1.12
Once per week			1.17	1.16	1.13	1.14	1.12	1.13	1.12	1.09	1.11
Club involvement (yes)				1.17*	1.16*	1.12	1.11	1.08	1.11	1.09	1.08
Health Behaviors											
Smoking (never)											
Former					1.27**	1.28**	1.27**	1.27**	1.27**	1.25**	1.27**
Current					1.84***	1.80***	1.80***	1.77***	1.84***	1.79***	1.82***
Drinks (1–4 drinks)											
Never						1.27*	1.25*	1.23†	1.25†	1.20	1.20
Former						1.36**	1.32**	1.30*	1.31*	1.28*	1.26*
≥ 5 drinks						1.43*	1.42*	1.43*	1.40*	1.42*	1.40*
Missing						1.62***	1.52**	1.39**	1.49**	1.39*	1.31*
Hours of exercise per week (> 7 hours)											
None						1.48***	1.40***	1.38***	1.40***	1.40***	1.38***
≤ 7 hours						1.11	1.11	1.11	1.11	1.11	1.11

Impairment					
Functional impairment (none)					
≥ 1 impairment	1.60***	1.51***	1.57***	1.54***	1.45***
Missing	1.21	1.30*	1.22	1.22	1.31*
Inflammation					
Albumin (> 4.5 g/dL)					
≤ 3.5 g/dL		2.72***			2.66***
> 3.5 to ≤ 4.0 g/dL		1.42*			1.40*
> 4.0 to ≤ 4.5 g/dL		1.19			1.20
Missing because of infection		1.67**			1.60**
Missing		2.29***			2.25***
CRP (< 0.3mg/dL)					
≥ 0.3 mg/dL		1.07			1.04
Missing because of infection		1.19			1.13
Missing		0.78			0.66
Cardiovascular Risk Factors					
Hypertension (< 120 SBP and < 80 DBP)					
Prehypertensive (120–139 SBP or 80–89 DBP)			0.99		0.98
Hypertensive stage 1 (140–159 SBP or 90–99 DBP)			1.04		1.05
Hypertensive stage 2 (≥ 160 SBP or ≥ 100 DBP)			1.20*		1.20*
Missing			1.35		1.28
Total cholesterol (150–249)					
< 150				1.60***	1.48**
250–349				1.01	1.03
≥ 350				1.70†	1.75*
Missing				1.14	1.06
Glycosylated hemoglobin (< 6.4%)					
≥ 6.4%				1.54***	1.42***
Missing				1.27	1.34

Notes: Reference groups are listed in parentheses. The sample size is 16,787 individuals and 2,709 deaths.

Source: Derived from NHANES III Linked Mortality File (NCHS 2007b).

†$p \leq .10$; *$p \leq .05$; **$p \leq .01$; ***$p \leq .001$

Adjusting for inflammation—which is more prevalent among females—widens the sex gap in mortality (compare Models 7 and 8). Compared with individuals who are normotensive, those who are hypertensive stage 2 experience higher mortality risk, which slightly widens the sex gap in mortality (compare Models 7 and 9). Total cholesterol and glycosylated hemoglobin are significantly related to mortality and reduce the sex gap in mortality (compare Models 7 and 10). Compared with women, a greater proportion of men have high glycosylated hemoglobin and total cholesterol below 150 (see Table 1), which impart greater mortality risks.[2] In separate analyses (not shown), we tested for but did not find statistically significant interactions between sex and any of the independent variables, including SES, social relation, health behavior, impairment, inflammation, or cardiovascular risk factors, which is consistent with other studies (see, e.g., Fried et al. 1998; Marang-van de Mheen et al. 2001). Thus, even though males and females may differ in their distributions of risk factors, those risk factors may have the same relationship with mortality for both sexes.

To examine whether the relationship between mortality and the other model covariates is the same (i.e., proportional) across ages, Table 3 estimates our models separately for three age groups. The sex gap in mortality is smallest among middle-aged adults and largest among young adults. Compared with females aged 20–44, males are 88% more likely to die over the follow-up period (Model 1). After adjustment for SES, social relations, and health behaviors (Model 2), the sex gap widens and men have more than twice the risk of death as women. The sex differences in mortality widen further with controls for functional impairment, measures of inflammation, and cardiovascular risk factors (Model 3). We find no statistically significant sex gap in mortality among individuals aged 45–64. At older ages, males are 77% more likely to die over the follow-up period than females in the fully adjusted model (Model 3), which is similar to but slightly lower than the over twofold gap in mortality reported by Fried et al. (1998).

Table 4 shows sex differences by cause of death among U.S. adults. Diseases of the circulatory system are the leading causes of death in the United States, and heart and cerebrovascular (stroke) diseases are the two primary circulatory diseases. Sex differences in circulatory diseases increase from Models 1 to 2, suggesting that differences in SES, social relations, and health behaviors suppress this sex difference. Controlling for all risk factors, Model 3 shows that compared with females, males are twice as likely to die from circulatory diseases and 2.4 times as likely to die of heart disease over the follow-up period. The risk of death from stroke is similar for both sexes (for similar results, see Kung et al. 2008).

The models for all cancers show that significant sex differences in Model 1 are no longer significant in Model 3. Lung cancer displays a large and persistent gap. Compared with females, males have a 90% higher risk of lung cancer mortality in Model 1. Because cigarette smoking contributes to a large proportion of all cancers, especially lung cancer, and because males have historically smoked at greater levels for longer periods, controlling for smoking statistically eliminates the sex gap in overall cancer and in lung cancer (Model 2). Furthermore, Model 3 shows that once we control for all covariates, males are 48% more likely than females to die from lung cancer. Compared with females, males are 3.6 times as likely to die from infectious and parasitic diseases, 7.5 times as likely to die from HIV, 3.2 times as likely to die from pneumonia, and 6.5 times as likely to die from chronic liver disease and cirrhosis, net of other controls (Model 3). Compared with females, males are 2.3 times as likely to die from external causes (Model 3).[3] Falls, a leading external cause

2. Stata's "svy" commands fit models with maximum pseudo-likelihood methods, which limit our ability to consider the complex survey design when calculating the fit statistics. Nevertheless, when we adjust for sample weights and strata but not the primary sampling units, each model shows improvement in fit based on the pseudo-log-likelihood, AIC, and BIC.

3. In cause-specific analyses not shown, we allowed the hazard ratio of sex to vary by age. The sex gap in mortality is driven in large part by circulatory diseases and external causes at younger ages, whereas it is more heavily driven by cancer and respiratory diseases (factors that are due in part to men's poor health behaviors) at older ages.

of death, also exhibit a greater mortality risk among males than females, with a hazard ratio of 3.5. Although modest numbers of deaths from accidents, homicides, and suicides limit our statistical power, the sex differences we find are comparable to those documented with U.S. vital statistics data (Kung et al. 2008).

Table 5 reveals the potential closure in the sex gap in mortality through the elimination of selected single (panel a) and multiple (panel b) causes of death. Because the sex differences in external causes, HIV, and chronic liver disease and cirrhosis are large (see Table 4), the elimination of any of these causes would reduce mortality for both sexes and would close the sex gap in mortality by 5%. Each of these causes has a similar impact on the sex gap in mortality in the cause-elimination models, even though they make up different percentages of all deaths and exhibit different impacts on the sex differences in mortality in the cause-specific models (see Table 4). Thus, the elimination of a cause of death, such as HIV, that has small prevalence but a disproportionate impact on male mortality, results in a relatively large reduction in the sex difference in mortality. Eliminating prostate cancer substantially reduces sex differences in mortality—by 8%—because it affects only males. The elimination of heart disease, which contributes to 28% of all deaths in the sample, could reduce the sex gap in mortality by 29%. The elimination of external causes, chronic liver disease and cirrhosis, and HIV would reduce the sex gap in mortality by 10% (panel b), and the elimination of these causes and heart disease would reduce the sex gap in mortality by 59%.

CONCLUSION

In general, we find that a large part of the sex gap in mortality can be attributed to differing sex-specific *distributions* of social relations, functional impairments, SES, health behaviors, and biological markers. We expect that the gap will continue to close for the next few years, as males increasingly engage in preventive behaviors, reduce their risky behaviors (especially tobacco consumption), and continue to benefit from the lagged effect of smoking cessation (Pampel 2005; Preston and Wang 2006). Still, it is reasonable to expect that the sex gap in mortality will continue to vary with changes in the prevalence of demographic characteristics, SES, social relations, health behaviors, and biological factors.

We find a sex gap in U.S. adult mortality of 62%, net of other covariates, which is larger than the gap found by some studies (e.g., Wingard 1982), but smaller than the gap identified by others (e.g., Fried et al. 1998; Rogers et al. 2000). These differences are likely due to different samples, time periods, age groups, and control variables. Over time, females have attained higher levels of SES and males have engaged in fewer risky behaviors, especially smoking. Thus, improved health behaviors among males may account for part of the narrowing of the sex gap in mortality.

We can determine the potential range in the sex gap in mortality through statistical adjustment and confidence intervals. We cannot statistically eliminate the gap—which also persists in other studies (see Rogers et al. 2000; Wingard 1982; Wingard et al. 1983), even in select subpopulations (see Merrill and Lyon 2005)—but we can increase or decrease it, over a range of 30% to 83%, by changing the variables included in the model or the order in which they are applied. For example, separate models (not shown) that added smoking status, visits with friends and relatives, religious attendance, total cholesterol, and glycosylated hemoglobin to the baseline model produced a 30% higher risk of death among males than among females (hazard ratio = 1.30), thereby closing the sex gap in mortality by 37% ((1.30 – 1.49) / (1.49 – 1) × 100) from the baseline model. Although informative, this artificially low sex gap does not take into account women's higher rates of impairment; lower levels of physical activity, marriage, and SES; and different distributions of hypertension and measures of inflammation. Selectively including those variables on which women experience higher mortality risks widens the sex gap in mortality to 83%. An alternative method of determining the potential range in sex differences in mortality is to produce confidence

Table 3. Hazard Ratios for Sex Differences in Mortality, Disaggregated by Age Group: U.S. Adults, 1988–2000

	Ages 20–44			Ages 45–64			Ages 65 and Older		
Variable	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
Sex (female)	1.88***	2.16**	2.83**	1.12	1.00	1.12	1.63***	1.65***	1.77***
Demographic Factors									
Race/ethnicity (nonblack)									
Non-Hispanic black	2.10**	1.48†	1.06	1.90***	1.51**	1.21	1.23**	1.06	0.95
Socioeconomic Status									
Education (≥ 16 years)									
≤ 12 years		1.60	1.46		1.22	1.17		0.98	0.99
13–15 years		1.32	1.27		1.16	1.19		1.02	0.99
Poverty-income ratio		0.95	0.96		0.97	0.99		0.95**	0.96*
Social Relations									
Marital status (married)									
Divorced/separated		2.49***	2.18**		1.09	1.11		1.25†	1.20
Never married		1.97*	1.86*		0.67	0.59		1.03	1.08
Widowed		0.43	0.39		0.98	0.93		1.14†	1.13†
Visit with friends/relatives (≥ 1 per week)		1.25	1.14		1.27	1.25		1.26†	1.16
Religious Attendance (> once per week)									
No attendance		1.43	1.39		1.30	1.16		1.39**	1.28†
< Once per week		1.01	0.95		1.13	1.03		1.26†	1.20
Once per week		0.96	0.99		0.95	0.89		1.22†	1.19
Club involvement (yes)		1.40	1.40		1.09	1.14		1.11	1.06
Health Behaviors									
Smoking (never)									
Former		1.11	1.16		1.17	1.11		1.34***	1.32**
Current		1.27	1.45		1.99**	1.90**		1.73***	1.77***
Drinks (1–4 drinks)									
Never		1.43	1.34		0.86	0.76		1.32**	1.25*
Former		2.20*	2.10*		1.35†	1.22		1.25*	1.15
≥ 5 drinks		1.55	1.43		1.60*	1.57†		1.35	1.36
Missing		1.99	1.73		2.94***	2.59***		1.43**	1.16
Hours of exercise per week (> 7 hours)									
None		1.86*	1.86**		1.52*	1.35		1.38***	1.27**
≤ 7 hours		1.16	1.13		1.01	0.94		1.13	1.14

Impairment			
Functional impairment (none)			
≥ 1 impairment	1.88	1.54**	1.49***
Missing[a]	1.54†	1.40†	
Inflammation			
Albumin (> 4.5 g/dL)			
≤ 3.5 g/dL	8.50**	2.92**	2.32***
> 3.5 to ≤ 4.0 g/dL	2.44†	1.16	1.40†
> 4.0 to ≤ 4.5 g/dL	0.92	1.14	1.25
Missing because of infection	2.12	1.40	1.65*
Missing	5.81*	2.83**	1.90**
CRP (< 0.3mg/dL)			
≥ 0.3 mg/dL	1.30	1.48*	0.90
Missing because of infection	0.42	1.20	1.17
Missing	0.14*	0.32	0.90
Cardiovascular Risk Factors			
Hypertension (< 120 SBP and < 80 DBP)			
Prehypertensive (120–139 SBP or 80–89 DBP)	1.28	0.76	1.02
Hypertensive stage 1 (140–159 SBP or 90–99 DBP)	2.05†	1.39†	0.90
Hypertensive stage 2 (≥ 160 SBP or ≥ 100 DBP)	1.36	1.51*	1.05
Missing[b]		1.26	1.32
Total cholesterol (150–249)			
< 150	1.77†	2.16*	1.26†
250–349	1.26	1.04	1.00
≥ 350	0.00***	1.65	1.77*
Missing	1.65	2.29	0.79
Glycosylated hemoglobin (< 6.4%)			
≥ 6.4%	1.20	1.32†	1.42***
Missing	1.97	0.96	1.43†

Notes: Reference groups are listed in parentheses. The sample size (with deaths in parentheses) is 8,291 (219) for ages 20–44; 4,079 (468) for ages 45–64; and 4,417 (2,103) for ages 65 and older.

Source: Derived from NHANES III Linked Mortality File (NCHS 2007b).

[a]There are no missing values for functional impairment among individuals aged 65 and older.

[b]There are no missing values for hypertension among individuals aged 20–44.

†$p \leq .10$; *$p \leq .05$; **$p \leq .01$; ***$p \leq .001$

Table 4. Hazard Ratios for Sex Differences in Cause-Specific Mortality: U.S. Adults, 1988–2000

Underlying Cause of Death (based on ICD10 codes)	Model 1[a]	Model 2[b]	Model 3[c]
Diseases of the Circulatory System (ICD10 I00–I78, I80–I99)	1.68***	1.81***	2.01***
Heart disease (ICD10 I00–I09, I11, I13, I20–I51)	1.90***	2.14***	2.42***
Cerebrovascular disease (ICD10 I60–I69)	1.05	1.00	1.04
Malignant Neoplasms (ICD10 C00–C97)	1.27†	1.09	1.17
Malignant neoplasms of trachea, bronchus, and lung (ICD C33–C34)	1.90**	1.36	1.48*
Infectious and Parasitic Diseases (ICD10 A00–B99)	2.83***	3.74***	3.56***
Human immunodeficiency virus (HIV) (ICD10 B20–B24)	7.32***	7.24**	7.50***
Diseases of Respiratory System (ICD10 J00–J98)	1.38†	1.31	1.42†
Pneumonia (ICD10 J12–J18)	2.16***	2.56**	3.17***
Chronic lower respiratory diseases (ICD10 J40–J47)	1.16	1.07	1.18
Chronic Liver Disease and Cirrhosis (ICD10 K70, K73–K74)	4.66**	6.30*	6.46***
External Causes	2.10**	2.03*	2.27**
Accidents (ICD10 V01–X59, Y85–Y86)	1.95*	1.70	2.02†
Motor vehicle accidents (ICD10 V02–V04, V09.0, V09.2, V12–V14, V19.9–V19.2, V19.4–V19.6, V20–V79, V80.3–V80.5, V81.0–V81.1, V82.0–V82.1, V83–V86, V87.0–V87.8, V88.0–V88.8, V89.0, V89.2)	1.45	1.31	1.92
Falls (ICD W00–W19)	2.69*	3.10*	3.51**
Suicides and homicides (ICD10 X60–Y09, Y87.0–Y87.1)	2.44	2.46	2.58
Other Causes (ICD10 residuals)	1.03	1.06	1.12

Source: Derived from NHANES III Linked Mortality File (NCHS 2007b).

[a]Controls for race/ethnicity.

[b]Controls for race/ethnicity, socioeconomic status, social relations, and health behaviors.

[c]Controls for variables in Model 2 and also impairment, inflammation, and cardiovascular risk factors.

†$p \leq .10$; *$p \leq .05$; **$p \leq .01$; ***$p \leq .001$

intervals; in Table 2, the 90% confidence intervals are 1.38 and 1.63 for Model 1 (and 1.48 and 1.79 for Model 11), and the 95% confidence intervals are 1.35 and 1.65 for Model 1 (and 1.46 and 1.82 for Model 11). These two approaches establish a compelling empirical range for future sex differences in adult mortality.

Over the past three decades, women's educational levels have increased and in some instances have surpassed men's levels of education, labor force participation rates by sex have converged, and women's incomes have become a greater proportion of family incomes: working wives contributed up to 36% of their families' incomes in 2006, up from 27% in 1970 (U.S. Department of Labor 2008). Women's increasing SES may contribute to their longer lives, ultimately widening the sex gap in mortality (for a similar view, see Nathanson 1995). But high-SES women may also contribute to the health and long life of others in their households. Thus, the convergence in SES may improve the quality—and even the length—of life for both sexes.

The higher mortality risk associated with widowed and divorced or separated statuses, combined with the additional time women spend in these statuses, relative to men (Schoen and Weinick 1993), contributes to the sex gap in mortality. If women reduced their time in the divorced and widowed states and increased their time in the married state—say, by marrying males of similar or younger ages, or remarrying sooner after a divorce or spousal death—the sex gap in mortality would likely widen.

Table 5. Hazard Ratios for Sex Differences in Mortality Based on Multivariate Cause-Elimination Models: U.S. Adults, 1988–2000

Elimination of Selected Underlying Causes of Death	Hazard Ratio	Percentage of Deaths Eliminated
A. Single-Cause Elimination		
Full model,[a] all causes	1.63***	0.0
Eliminate:		
Prostate cancer	1.58***	1.7
External causes	1.60***	4.5
Chronic liver disease and cirrhosis	1.60***	1.1
HIV	1.60***	1.3
Heart diseases	1.45***	28.4
B. Multiple-Cause Elimination		
Eliminate:		
External causes and cirrhosis	1.57***	5.6
External causes, cirrhosis, and HIV	1.54***	6.9
External causes, cirrhosis, HIV, and heart diseases	1.26*	35.3

Source: Derived from NHANES III Linked Mortality File (NCHS 2007b).

[a]Controls for race/ethnicity, socioeconomic status, social relations, health behaviors, impairment, inflammation, and cardiovascular risk factors.

*$p \leq .05$; ***$p \leq .001$

Unobservable characteristics, such as a person's overall outlook and larger life choices, may affect the relationship between religious attendance and mortality (Moffitt 2005): those who engage in healthier and safer behaviors may also be more likely to attend religious services. This argument suggests that even if more males attended religious services, there is no guarantee that their life expectancy would increase or that the sex gap in mortality would close. Indeed, our analyses found that the association between religious attendance and mortality was substantially attenuated after we adjusted for health behaviors, impairment, and biomarkers. Even so, both sexes experience survival advantages through increased social relations, including marriage, visits with friends and relatives, religious attendance, and involvement in clubs.

We calculate that cigarette smoking contributes to 22% of the sex difference in mortality (compare Models 4 and 5 in Table 2). This is smaller than but similar to the 25% estimate that Rogers et al. (2000) produced with data from 1990–1995, and follows the declining sex gap of 30% in 1985–1989, 40% in 1970–1974 (Valkonen and van Poppel 1997, with data from Denmark, Finland, the Netherlands, Norway, and Sweden), and 47% in 1962 (Retherford 1972). As male and female rates of smoking decline and become more similar, smoking will exert a smaller influence on sex differences in mortality, and other factors will exert stronger influences (Hummer et al. 2009; Pampel 2005).

The male exercise advantage may wane if males become more sedentary or if females continue to increase their physical activity. Males can continue to enhance their survival prospects through light to moderate alcohol consumption, and females might further increase their survival prospects with modest drinking. Of course, all individuals should avoid heavy drinking, with its attendant risk of some cancers (especially of the oral cavity, pharynx, esophagus, and larynx), chronic liver disease, cirrhosis, accidents, and violence (see Corrao et al. 2004).

Inflammation and cardiovascular risk factors retained statistical significance in our final models, and the social, behavioral, and socioeconomic factors largely retained their

relationships with mortality, even after we adjusted for the biomarkers. We focused on biomarkers that are frequently cited in the clinical literature and that are potentially amenable to intervention. The inflammation and cardiovascular risk measures were statistically related to mortality among adults, even in the final model. But many biological effects can be minimized or eradicated through diet; exercise; and advances in medical intervention, including health screening, surgery, and pharmacologic treatment, especially antihypertensive and cholesterol-lowering medication (Gregg et al. 2005; Waldron 1995)—factors that are seldom distributed equally by sex.

Even if the risk of external causes of death for males does not change, the sex gap could close if females increase their risky behavior. Passannante and Nathanson (1985:655) stated that "the coping mechanisms used by females to adjust to stressful situations may include assuming traditional 'male behaviors' associated with high male mortality (e.g. smoking, drinking, driving, suicide, homicide, low levels of health care utilization and little indulgence in the sick role)." The similarity in mortality in the middle adult years may be due to life course factors: the two sexes are more similar in their social relations, SES, and health behaviors in this age group than in other age groups. Trovato and Lalu (1996) demonstrated that among individuals aged 25–59, between the early 1970s and 1990s, males experience greater mortality reduction than females, which would contribute to a constriction of the sex gap in mortality among these ages. If the sex gap in mortality at older ages is capturing higher levels of lifetime cigarette smoking among males, then it should decline as healthier males (i.e., those who are aged 45–64 in our data) age into this group. In fact, the sex gap at older ages may have already witnessed some closure over time. Our finding of a sex gap of 77% among individuals aged 65 and older is already lower than the more than twofold gap found by Fried and colleagues (1998).

Cause-specific mortality varies by sex, with the largest gap for HIV, pneumonia, chronic liver disease and cirrhosis, and external causes, especially falls; a large gap for cardiovascular diseases and lung cancers; and a small gap for cerebrovascular diseases. Our cause-elimination models show that eliminating external causes, HIV, or chronic liver disease and cirrhosis could each reduce the sex gap in mortality by 5%. These causes contribute to a small portion of all deaths (e.g., HIV contributes to just 1.3% of all deaths) but a substantial portion of the sex differences in mortality. Prevention efforts aimed at reducing homicides, suicides, and accidents could have an immediate effect on overall mortality and the sex gap; diseases such as HIV and chronic liver disease and cirrhosis may be more difficult to prevent because of multiple risk factors and the longer lag between risky behavior, the onset of the disease, and subsequent death.

Fortunately, many disease processes can be halted or reversed. For example, individuals reduce their immediate and long-term risk of death by quitting smoking (U.S. DHHS 1990), eliminating excessive drinking, and reducing excess weight through better diet and exercise. Among external causes of death, men's higher rates of suicide may be due in part to their greater use of guns, which are quite lethal, compared with women's greater use of drugs, which are less lethal (Waldron 1985). As males and females converge in their driving patterns (including obtaining driver's licenses, driving similar numbers of miles, and wearing seat belts), in the risks of drowning, and in occupational accidents, we would expect further convergence in the sex differences in accidental mortality (Waldron et al. 2005).

Future research could extend our results by examining how changes in health behaviors, SES, and social relationships are related to overall mortality and to the sex gap in mortality. Although NHANES is the only nationally representative data set with information on social, behavioral, and biological factors, the Second Longitudinal Study of Aging, the Panel Study of Income Dynamics, and the Health and Retirement Study would allow researchers to directly examine the impact of changing social and economic factors on sex differences in mortality. Moreover, there would be tremendous benefits in developing a future longitudinal NHANES.

Reducing health disparities is central to the *Healthy People 2010* objectives (U.S. DHHS 2000). The nation should strive to increase longevity for both sexes, even if some interventions—for example, encouraging income parity by sex or increasing women's levels of physical activity—could increase sex disparities in survival. Improvements in health behaviors among men, especially at advanced ages, could substantially increase men's longevity; this could have the salubrious effect of increasing time in marriage for both sexes, thereby decreasing the percentage of time women spend as widows and resulting in greater gains in life expectancy for women. Such nuanced patterns highlight the value of fully understanding the many factors that shape sex differences in mortality.

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