



# Hypotheses Explaining the Sex Mortality Differential

*“In brief, a price is paid for a beard and the presence of functional testes” (Hamilton 1948).*

*“Women who smoke like men die like men who smoke” (Califano 1979).*

Hypotheses regarding the sex mortality differential fall into two general families: (1) constitutional (biological/genetic) and (2) external (social/cultural/environmental/behavioral). Many researchers support the external family of hypotheses, believing that external causes imply that it is likely excess male mortality can be decreased (Johnson 1977; K. Goldberg 1993). Madigan (1957), on the other hand, felt that this would require a profound cultural revolution in our society. He stated that the prognosis would be more hopeful if biological factors are primarily responsible for the differential. In that case, medical research may be able to *“isolate the factors responsible for greater female viability, and use this knowledge to advantage in the treatment of middle-aged and old men, assuming that this can be done without disturbing psychological balance or causing observable physical reactions.”*

The sex mortality differential is a difficult subject to investigate because ideal studies are not ethically possible. It is not morally practical for a researcher to take newborn infants of each sex, have half live as males and half as females, then compare mortality rates. Instead, investigators have been ingenious in designing various types of less-than-perfect studies in order to explore the sex mortality differential. As we shall see, evidence supports both schools of hypotheses as partial explanations of the sex mortality differential.

## 7.1 Constitutional—Biological/Genetic

As shown in the table in Appendix F, considerable data have shown that, in the vast majority of animal species studied, the males have higher mortality rates than do the females. This holds true for animals in the wild and in captivity, including nematodes, crustaceans, mollusks, insects, spiders, reptiles, fish, and mammals, including primates.

The mortality differential by sex in birds varies by species, which may be explained by the fact that in birds, unlike mammals, it is the male who has a pair of two like chromosomes (Smith and Warner 1989), and, as the table indicates, also appears to be related to the monogamous/polygamous/promiscuous behavior of the species (Trivers 1972). The majority of birds are monogamous (Murton and Westwood 1977) and the male is interested in his offspring in the great majority of birds (Skutch 1976). Six surviving species of birds are polyandrous (females having sexual relationships with more than one male), where the males incubate the eggs (Murton and Westwood 1977). It would be interesting to know what the sex mortality differential is for these species of birds, along with that of the seahorse, in which it is the male who is pregnant.

Mortality differences by sex are more pronounced in species with greater adult size differences between males and females. The sex mortality differences in mammals may be attributable to sex differences in nutritional requirements and/or sex differences in dispersal (Clutton-Brock 1994). Care should be taken in extrapolating animal sex mortality differences to humans since the causes of death are often quite differ-

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ent (D. Smith 1989). According to Hayflick (1982), “regardless of which sex might be favored, the fact that one is, strongly suggests a fundamental genetic basis for the difference.”

Analyzing mortality tables for several species of mammals, Caughley (1966) found that all shared the familiar J-shape, with the rate of mortality initially high but rapidly declining in the juvenile years, followed by a postjuvenile phase of initially low mortality but steadily increasing mortality.

Many have concluded that the male is the weaker or frailer of the sexes (Holmes and Goff 1923; Allen 1934; Crew 1937; Sowder 1952; Hammoud 1965; Potts 1970; Bhatia 1983; James V. Neel cited in Holden 1987; Christen 1991; Montagu 1992). Allen (1934) studied data from The Mayo Clinic and determined “that among males there is a higher incidence of most diseases which might permanently influence health or endanger life” and that “mere maleness influences unfavorably the resistance of the organism to disease at all ages.” He continued with:

*The factors which are usually set down in explanation of the greater mortality of males are overwork, alcoholism, venery [hunting or sexual indulgence], tobaccoism, exposure to the elements, industrial hazards, and irregular habits of eating and sleeping. . . . For each explanation of the lack of inherent vitality of the male there are objections, but these do not influence the fact; the male is, by comparison with the female, a weakling at all periods of life from conception to death. Venery, alcoholism, exposure, overwork, and various other factors may influence the susceptibility to disease and the greater mortality of the adult male, but they are only straws placed on the greater burden of his sex-linked weakness. There seems to be no doubt that, speaking comparatively, the price of maleness is weakness.*

Hamilton (1948), in a listing of pathological conditions in which the incidence differs by sex, found that the total number of conditions that occur predominantly in males is more than twice that which occur chiefly in females. Arranging these conditions according to body system, he found that the male is the preferred sex for all body systems, except for the endocrine system. Childs (1965) stated “the list of diseases to which the male succumbs more often than the female is depressingly long;” among the exceptions are the autoimmune diseases. Scheinfeld (1965) stated that the female is, in many respects, more variable biochemically than the male, which “may make the female better able to adjust to bodily stresses and ac-

*cidents and to rally defenses against infectious or other adverse outside influences.”*

Scheinfeld (1958) stated that a

*biological disadvantage of the male is his comparatively slow development in the early years. At every prenatal stage the female leads the male in rate of biological growth; at birth the male is 4 to 6 weeks behind the female. In that sense a full-term male baby can be considered “premature,” compared with a full-term female. (If a male is bigger and heavier than the female at birth, it is only because he is heading toward an ultimately greater size.) Since a male fetus or newborn infant is retarded with respect to the female, it is obviously exposed to greater hazard at any given stage. This may be one of the reasons why congenital abnormalities are much more common among male babies (Scheinfeld 1958).*

Torday et al. (1981) found evidence that, for a given gestational age, the lungs of male babies are less mature than the lungs of female babies. Males are more likely to be born with congenital abnormalities and, if a boy baby and a girl baby have exactly the same accident, the chances of fatality are considerably greater for the boy (Scheinfeld 1950 and 1965). Renkonen, Mäkelä and Lehtovaara (1962) found evidence suggesting that male pregnancies render couples less likely to have more baby boys, because male pregnancies immunize some mothers against male antigens, which is harmful to subsequent male fetuses.

A pivotal study supporting the biological hypothesis was performed by Madigan (1957) when he compared the mortality rates between teaching Catholic Brothers and Sisters. Social and cultural differentials were minimized between the two groups as follows: Entered the religious life before their 27th birthday, born in the United States, were unmarried and white, were members of the same faith and followed almost identical religious practices, were exempt from military service, possessed adequate housing and clothing and ate wholesome meals at regular hours, had easy access to good medical and dental care, had no familial financial and domestic worries and strains, served as teachers or administrators of schools, and were unable to lead a life of excess or dissipation. Madigan was not, however, able to control for smoking, alcohol use, or obesity. He found sex mortality differentials similar to those in the general population and greater differentials after age 45. Because of health selection (they needed to pass a health examination for admittance) and lifestyle, both the Brothers

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and Sisters tended to have better mortality rates than the general population.

A similar study was done by Leviatan and Cohen (1985) among male and female kibbutz members in Israel. The life experiences of male and female kibbutz members are similar in that men and women are all engaged full-time in the labor force outside their homes, have similar financial pressures and responsibilities, experience similar community life, community social life and daily schedules, are knowledgeable to a similar degree about the experiences of their spouses at work and community domains, and had similar experiences and similar stages in their life cycles. Noting that the life expectancy of a population is positively related to the size of the sex mortality differential of the population, Leviatan and Cohen found that the sex mortality differential of kibbutz members was less than would be expected on the basis of a regression analysis of data from 73 societies. From this finding, they concluded that at least a major part of the sex mortality differential is attributable to social and environmental causes. If, however, the sex mortality differential of the kibbutz members is compared to that of the Jewish population in Israel, the kibbutz members had a *greater* sex mortality differential. This comparison is evidence that the sex mortality differential is *not* completely attributable to social, environmental, and behavioral causes.

Schmidt and Popham (1980) also performed a study minimizing the environmental differences between men and women. Their study involved alcoholics who were admitted to clinics for alcoholism treatment. Alcohol and cigarette smoking use were similar for both men and women in the sample. They found that the sex mortality differential was much smaller within this group than among the general population, particularly for ages 50 and under. Unless alcoholics experience mortality patterns different from the general population, this study is evidence for the hypothesis that the sex mortality differential is attributable to social, environmental, and behavioral causes.

## 7.2 Genetic/Immunologic Factors

Humans have 46 chromosomes, arranged in 22 pairs of autosomes and one pair of sex chromosomes, which are found in the nuclei of the cells of the body. The sex chromosome pair consists of an X sex chromosome inherited from the mother and either an X or a Y sex chromosome inherited from the father. Individuals who have a 46, XX chromosome constitution are females and those who have a 46, XY constitution

are males. There are equal numbers of two sperm types, X- or Y-bearing, divided equally. As previously discussed, it is estimated that there are 7–70% more male conceptions than female conceptions and, at birth, the male-to-female sex ratio is about 1.05 to 1.

The Y chromosome is small and apparently contains only a few genes, which are related to the determination of sex. In contrast, the X chromosome is one of the largest chromosomes and contains numerous genes that control biological processes not connected with sex determination. “*Thus, the possession of at least one X chromosome is essential to life, but this is not true for the Y chromosome*” (Rasmuson 1971).

Females respond more vigorously to infections and other conditions requiring activation of the immune system throughout their life span because the X chromosome carries genes that help control immunoresponsiveness; two X chromosomes increase this capacity, but only for ages between roughly 5 and 65 for the IgM class of immunoglobulins (Ramey 1997; Waldron 1983). Apparently, the Y chromosome contributes only to maleness and seems to have no other genes matching those on the X chromosome for such traits as blood clotting and a lively immune system. A male who receives from his mother abnormal genetic information on his X chromosome does not have the opportunity to neutralize the trait because he lacks a second X chromosome. If a female has a genetic abnormality on one of her X chromosomes, her other X chromosome can usually make up for it.

This makes male fetuses more vulnerable to errors of metabolism, producing higher male mortality both before and after birth (Ramey 1997). Typical conditions relating to abnormal genes on the X chromosome are the bleeding disease hemophilia and color blindness (Scheinfeld 1950). Hazzard (1999) speculates that “*the greater vulnerability of men than women to death from cancer may reflect a greater impairment in immune surveillance.*” While evidence supports the supposition that females of several species have greater immune reactivity than males, few studies have been done to determine if immune vigor is directly related to life span (Weksler 1990).

Some studies have suggested that aberrations on the long arm of the Y chromosome may be associated with greater male longevity (Kuznetsova 1987). Kirby Smith studied an Amish family whose male members are missing the long arm of their Y chromosomes. Fourteen male members of this family lived on average to age 82.3, while the women members lived on average to age 77.4. For comparison, in two nearby Amish families, the women lived into their mid-70s

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and the men died five or six years earlier. Although it is not known what genes are on the missing arm and this was only one small study, Smith hypothesized that “*too much Y and you die*” (Holden 1987).

### 7.3 Androgens

Androgens, such as testosterone, are a class of hormones that are more predominant in males than in females. At six weeks after conception, the Y chromosome induces the formation of the testes and their secretion of testosterone, a powerful androgen. In addition to causing the male sex organs to begin to mature, androgens alter brain development and enzyme patterns in organs such as the liver. Testosterone slightly slows the growth of the left side of the cerebral cortex, while the right side grows faster. Testosterone also slows the overall rate of maturation of the male brain. Both sides of female brains develop more equally and are more interconnected. This allows women to tolerate brain damage better later in life, enabling women to recover more fully from strokes (Ramey 1997).

Testosterone provokes higher blood pressure, which causes damage to blood vessels and makes the liver produce low-density lipoprotein (LDL). LDL, the so-called “bad” cholesterol, can be oxidized by free radicals, and oxidized LDL causes damage to blood vessels. Animal studies have shown that, if testosterone secretion is inhibited in young males, their life span is markedly increased despite stressful conditions (Ramey 1997). It has been found that “*testosterone markedly increases arachidonate-induced platelet aggregability and thrombus formation with a concomitant increase in mortality rates. Testosterone also sensitizes blood vessel strips to the constrictor effects of the endoperoxides released during stress*” (Ramey 1982).

Testosterone also has been shown to suppress high-density lipoprotein (HDL) levels, the so-called “good” cholesterol. In studies by Bagatell et al. (1992), normal men, who were given a gonadotropin-releasing hormone (GnRH) antagonist, had increasing levels of HDL. When they were simultaneously given testosterone replacement, their HDL levels did not increase, implying that androgen levels may contribute to the increased risk for coronary artery disease in men. Androgenic anabolic steroids have been shown to reduce HDL and increase LDL dramatically and reproducibly (Hazzard 1999).

Hamilton (1948) hypothesized that the sex mortality differential exists, in part, because of the increased

metabolism in males due to androgens. In his study of castrated men, he found that androgens increase rates of metabolism, which is associated with shortened life spans. The basal metabolic rate has been determined to be greater in men than in women. Also, there is evidence that, in many animals, the life span of both sexes can be regulated to some extent by increasing or decreasing metabolism (Hamilton 1948; Perls and Fretts 1998).

Further evidence that androgens increase mortality is noted in Hamilton and Mestler’s (1969) study of 735 intact and 297 castrated men in an institution for the mentally retarded. In their study, castrated men had a life expectancy at age 8 that was 10.2 years greater than that of intact men. In fact, the castrated males significantly outlived intact females. They also found that the younger castration was performed, the lower the mortality—castration between ages 8 and 39 was associated with the reduction of 0.28 years in age at death for every year of delay before castration.

Although these data provide strong evidence of the effect of androgens on longevity, deaths from infections were primarily responsible for the difference in mean age at death between castrated and intact males. There was no significant difference in the mean age at death attributed to cardiovascular disease or cancer between the castrated and intact men. Because infectious diseases are not a major cause of death in developed countries today, these results may not be an explanation of the current sex mortality differential.

Studies of castrated men have not all been so conclusive. A study comparing castrated and intact male singers during the period 1581–1858 found no significant increase in life span due to castration. Nieschlag et al. (1993) compared the life spans of 50 castrated singers and 50 intact singers and found that the mean life span of the castrates was 65.5 years while the mean life span of the intact singers was 64.3 years, results that were not statistically significant using unpaired *t*-tests ( $p = 0.65$ ). But as pointed out by Paternostro (1994) and Smith (1994), there are several statistical reasons to be skeptical of these conclusions, including lack of power. It should also be noted that studies in lower animal species have shown that castration has generally, but not always, prolonged life.

Other evidence supporting the testosterone theory of sex mortality differentials concerns women with elevated androgen levels. Björntorp (1996) states that such androgenic women develop secondary sex characteristics, psychological profiles, and stress reactions similar to males. They also have an increased risk of developing hypertension, noninsulin-dependent diabetes mellitus, and cardiovascular disease. Waldron

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(1983), however, noted that the available data do not support the theory that testosterone contributes to men's higher risk of heart disease, but the data are limited.

Because sex differences in risk-taking behavior are widespread cross-culturally, in both industrialized and nonindustrialized societies, genetics may be a contributing factor (Waldron 1983). There is evidence to suggest that exposure to higher levels of male sex hormones during the prenatal period may predispose males to greater levels of physical aggressiveness (Ehrhardt and Meyer-Bahlburg 1981). Studies of correlation between testosterone and aggression after puberty have yielded contradictory results, but androgens are clearly implicated in some forms of aggressive behavior in humans (Doering et al. 1974; Waldron 1983; Petersen 1980; Moyer 1987). But, as Smith (1993) stated, "*testosterone, despite its adverse effects, plays an essential role in human reproduction.*"

Studies in animals have shown that increases in testosterone levels can suppress the effectiveness of the immune system, thus increasing the risk of disease. This immunosuppression is more evident in low-quality males than in high-quality males (Geary 1998). Studies have shown that female infant rhesus monkeys, born of mothers who had testosterone injections during pregnancy, showed greater frequencies of threats, play, and play imitations than did normal female infants, but less frequencies than normal male infants (Goy and Phoenix 1971). This and other studies involving rhesus monkeys showed that prenatal, not postnatal, androgens influenced sex-differentiated behavior in infancy (Mitchell 1979). It has also been shown that artificially elevated levels of testosterone in male lizards (Marler and Moore 1988) and male brown-headed cowbirds (Dufty 1989) increased mortality, probably due to greater aggression. Studies of male birds with artificially elevated testosterone levels have shown decreases in over-winter survival rates for dark-eyed juncos, *Junco hyemalis*, but not for song sparrows (Beletsky et al. 1995).

## 7.4 Estrogens and Progestins

Estrogens, the hormones that promote the development of female secondary sex characteristics, act on the liver to produce more immune globulins (antibodies that fight infectious agents). Estrogens and progestins in females protect the heart and blood vessels from rapid aging while male hormones accelerate the aging of these tissues (Ramey 1997; Talal 1979).

Research has shown that testosterone treatment increased sudden death in mice by dramatically increasing thrombosis in both sexes. When estrogen was administered to these animals, thrombosis was significantly reduced. Estrogens give women a much greater ability to resist stress and a greater capacity for long-term energy expenditure. They induce the liver to produce more HDL, which removes cholesterol from the bloodstream and thus protects females against its harmful buildup in blood vessels (Ramey 1997). Studies have shown that estrogen lowers LDL and raises HDL in both men and women when given in high doses (McGill and Stern 1979). In addition, estrogens regulate the creation of prostaglandins, hormones that protect women from forming fatal blood clots or coronary artery damage (Ramey 1997). There is also circumstantial evidence that the scavenging of free radicals by estrogens may help slow the aging and degeneration processes in women as compared with men (Conn 1987).

A study of large numbers of women who had their ovaries removed in their 20s and did not receive hormonal replacement therapy found that they developed heart disease about 15 years before women with intact ovaries or women who received hormonal replacement therapy after removal (Ramey 1997). In fact, most, but not all, studies of women who had their ovaries removed found an increased risk of heart disease (Waldron 1983, 1992). Small doses of estrogens given to men who had had heart attacks decreased their mortality rates, although this treatment may result in some "feminizing" physical effects (Scheinfeld 1965). Studies of higher doses of estrogen therapy in men have yielded mixed results regarding the risk of heart disease (Rivin and Dimitroff 1954; Waldron 1983). Hazzard (1999) summarized that "*the sex hormones confer a gender differential in the risk factors to the major chronic diseases of middle and old age is incontrovertible.*"

A majority of the more than 30 observational studies of postmenopausal hormone replacement therapy demonstrated benefits from estrogen. Also, studies have shown that age-adjusted, all-cause mortality is lower among estrogen users. Although the reasons are not completely understood, it is in part attributable to estrogen-increasing HDL and estrogen-lowering LDL. Because of possible bias in observational studies, an extensive clinical study called the Women's Health Initiative is underway. The study is intended to test the hypothesis that women who receive estrogen replacement therapy will have lower rates of coronary heart disease. When this 15-year study is completed in

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2007, more answers will be available (The Women's Health Initiative Study Group 1998).

It has recently been found that while men react to stress with a "fight or flight" response, women react with a "tend and befriend" response. It is thought that the "tend and befriend" response is related to oxytocin, a powerful hormone whose effect seems to be amplified by estrogen and diminished by androgens. It is being studied whether this difference could help explain the sex mortality differential (Suplee 2000).

## 7.5 Iron Overload

Because of the iron loss through menstruation, women have less iron in their blood than do men. Sullivan (1996) and Perls and Fretts (1998) have speculated that increased iron raises the risk for heart disease because of the relation between iron and free radicals, and because of conditions such as hereditary hemochromatosis, a common hereditary iron overload disease. Studies have suggested that blood loss through whole blood donations may be associated with a reduced risk of cardiovascular events (myocardial infarction, angina, stroke) in men (Salonen et al. 1998; Meyers et al. 1997) and have found an association between stores of iron in the body and heme iron intake with excess risk of myocardial infarction (Tuomainen et al. 1998; Klipstein-Grobusch et al. 1999).

## 7.6 Natural Selection and Care of Offspring

A Darwinian natural selection hypothesis may help explain why females have greater longevity. Because bearing and rearing offspring is consistently much more a female role than a male role (Geary 1998), longer-lived women have a selective advantage over women who die young (Perls and Fretts 1998). Humans are unusual among animals in that the female's fertile life ends well before the duration of life under optimal conditions (Potts 1970; Perls et al. 1999). Menopause, it is speculated, promotes longer life of women by eliminating the mortality risk from childbirth, which allows post-menopausal women to care for their children and even their grandchildren (Potts 1970; Perls and Fretts 1998; Perls et al. 1999).

Further support for this hypothesis comes from the study of apes. Allman, Rosen, Kumar et al. (1998), in a study of nine anthropoid primates, found clear cor-

relation between male/female survival ratios and the amount of care the males provide to their young. Allman and Hasenstaub (1999) stated:

*Natural selection will tend to favor genes which enhance survival in adults in the sex that provides the most care for offspring. If the caretaking parent dies, the offspring will probably die as well, but if the noncaretaking parent dies this event will have little impact on the offspring's chances of survival. The death of a noncaretaking parent might even enhance the survival of its offspring by removing a competitor for scarce food resources.*

They also said that the mortality differential between caretakers and noncaretakers is in part attributable to caretakers' risk aversion and noncaretakers' risk seeking, because risk on the part of caretakers puts their offspring at risk as well. This risk aversion may be a conscious decision or may be attributable to genetically determined instincts. Estrogen may also be responsible for reducing risk-taking behavior because it enhances the actions of serotonin. Allman and Hasenstaub also found that the pattern of a peak in the sex mortality ratio in early adulthood, which is at the period of caregiving in women and the greatest risk seeking in men, is also present in chimpanzees and gorillas. Allman (1999) believes that noncaretaking males are aggressive and prone to risk taking because they then seek new opportunities that can be communicated back to their close kin, enhancing their kin's survival.

Studies in birds have shown that high testosterone levels in males reduce parental care in favor of aggression (Wingfield 1994), so perhaps there is a relationship between less parental care, testosterone, aggression, and greater mortality. Greater male survival is more prevalent in monogamous birds, while greater male mortality seems to occur among polygynous or promiscuous birds (Trivers 1972).

DeVore and Lovejoy (1985), relying on fundamental biological principles, determined by induction the natural superiority of women, including their capacity for greater longevity. They argued that various species are capable of direct female replication with no male contribution. "If there is only one gender capable of physical replication, that gender is primary. Thus the female gender is fundamental and the male auxiliary. Males were 'invented' by natural selection as a means of providing genetic variety." The basic strategy of both males and females is the same—to produce as many offspring as possible. Selection favors increased longevity in women, because the woman's reproduc-

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tive rate is linear with age until menopause. Menopause does not alter this conclusion, they said, because “*for the greater part of that time our species has existed, so few women have survived to that age, that selection has been powerless to extend the length of the reproductive period.*” Men, though, are fitter if they father their offspring early in life, even at the expense of longevity. These authors concluded that “*while males can never be naturally superior to women, they can approach equality. It is most instructive that realization of equality requires intensification of male-parenting behavior.*”

## 7.7 Social and Medical Advances

Hamilton (1948) stated that males have been less able than females to take advantage of better living conditions. Lopez (1983), in discussing modern life for men, said “*the implication, therefore, is that males have adapted less well to the more comprehensive process of modernization with its attendant behavioural changes.*” With respect to the increase of the sex mortality differential over time, Scheinfeld (1950) said “*the more that environmental factors for the two sexes have been improved and equalized, the proportionately greater has been the advantage to women, and the more apparent it has become that females are genetically better constructed, have a more efficient internal chemical system, and in various other ways are biologically better adapted to resist most of the modern human afflictions.*”

As mortality rates have decreased, the relative importance of causes of death have changed, and those causes affecting females have declined more than those affecting males. In particular, maternal deaths, defined as deaths from complications of pregnancy, childbirth, and the puerperium, have declined substantially. In the United States, the maternal death rate was 610 per 100,000 live births in 1915, increasing to 920 per 100,000 live births in 1919, “*largely because of the influenza epidemic and the medical and social disorganization of World War I*” (Health Information Foundation 1958). By 1998, the rate had toppled to 7.1 per 100,000 live births; in fact, only 281 women died in the United States in 1998 from maternal causes (Murphy 2000).

The maternal death rate in England and Wales fell from 500 per 100,000 births in 1860 to 25 per 100,000 births in 1960; maternal mortality, as a percentage of all female deaths in the 15- to 44-year-old age group, declined from 9% to 2% (Potts 1970). The decrease of maternal deaths as a percentage of all female deaths

is attributable to improvements in obstetrics, the reduction in the total number of births, and their concentration at the physiologically most advantageous time. It is estimated that maternal mortality in parts of Asia or Africa may exceed 1,000 per 100,000 live births (Potts 1970). The enormous decrease in maternal death and disability has been called “*one of the most significant achievements of modern medical science*” (Health Information Foundation 1958).

## 7.8 Risk-Taking and Other High-Risk Behaviors

Nathanson (1977) presented data showing that men take more risks than women, while women tend to exhibit more preventive behaviors. Table 4 summarizes Nathanson’s risk-taking behavior data, together with the ratios of female-to-male rates for the behaviors. All such rates from 1977 are based on sample surveys, except for Nathanson’s rates for opiate addiction and use of LSD. This table also shows ratios of these rates based on more recent data.

### 7.8.1. Cigarette Smoking

Tobacco smoking has been called “*hateful to the nose, harmful to the brain, dangerous to the lungs*” (James I, King of England, 1604). The National Center for Health Statistics (2000b) said “*tobacco use is the single leading preventable cause of death in the United States.*” And Pierce (1997) said “*Tobacco is one of the few products on the legal market which, when used explicitly as the manufacturer intended, leads to untimely death and disability. . . . If the product were new to the market today, it could never meet the regulatory requirements for legal production and marketing for sale. However, tobacco was introduced into Europe five centuries ago, and it now plays an important role in many national and provincial economies. No country has sought to delegalize all tobacco products.*”

Cigarette smokers have greater mortality than nonsmokers (Benjamin 1982; Hammond 1966, 1969), and actuaries are well aware of this. U.S. life insurance experience for 1990–91 shows that male mortality for smokers averaged 2.32 times as great as for male nonsmokers; the ratio was 2.19 for female smokers compared to female nonsmokers (Individual Life Insurance Experience Committee 2000, Society of Actuaries 1982). Also, there are separate 1986–92 Canada CIA Basic tables by sex and smoking status

**TABLE 4**  
**COMPARISON OF SEX DIFFERENCES IN EXPOSURE TO SELECTED POTENTIAL RISKS**

Class of Risk	Nathanson (1977)			Current		
	Year	Risk-Exposure	Ratio (Female/Male)	Year	Risk-Exposure	Ratio (Female/Male)
Cigarette smoking	1975	Has smoked at least 100 cigarettes during lifetime and who now smokes	0.74	1998	Smokes cigarettes now	0.83
Alcohol consumption	1967	On basis of frequency of drinking alcoholic beverages and amount consumed on each occasion	0.24	1997	At risk for alcohol-related illnesses (acute drinking)	0.30
				1997	At risk for alcohol-related illnesses (chronic drinking)	0.15
				1997	At risk for injury from drinking and driving	0.30
				1998	Arrested for driving under the influence	0.24
Illegal drug use	1965–1972	Seeking treatment for opiate use	0.24	1997	Ever used any illicit drug	0.77
				1997	Ever used heroin	0.46
				1997	Ever used crack	0.60
				1997	Ever used cocaine	0.60
				1970	Use of LSD one or more times	0.41
				1973	Use of marijuana one or more times	0.52
Legal drug use	1970–1971	Use of prescription psychotherapeutic drugs in past year	2.27	1997	Ever used any psychotherapeutic drug	0.73
Automobile driving	1969–1970	Estimated average annual miles driven per licensed driver	0.48	1995	Estimated average annual miles driven per licensed driver	0.61

Source: Nathanson 1977, originally published in *Journal of Community Health*, reprinted with permission. All current data is from Centers for Disease Control and Prevention 1995–1999; except arrest data from Washington State Patrol, Breath Alcohol Test Section, licensed driver information from State of Washington, Department of Licensing, drug use data from Substance Abuse and Mental Health Services Administration, Office of Applied Studies 1998, automobile driving from Hu and Young 1999.

(Canadian Institute of Actuaries 1995). The expectation of life at age 16,  $e_{16}$ , for both these sets of mortality tables is shown in Table 5. Because of the select nature of the tables, the actual expectations vary between the two countries' sets of data, but the ratios are relatively similar, although the U.S. data show greater differences in mortality by smoking status for both sexes than do the Canadian. On the other hand, the Canadian data show greater mortality differences by sex for both smoking statuses. In both sets of data,

nonsmoking males have greater life expectancy than do smoking females.

As demonstrated by the United States life insurance experience for 1990–91, the excess mortality among female smokers appears to be only 0.94 (2.19/2.32) times the excess mortality among male smokers. This ratio does not necessarily imply that the effects of smoking are relatively less severe for women, since women smokers tend to have lower overall exposure to cigarette smoke. Relative to men, women started

smoking at older ages, women smoke cigarettes with lower tar and nicotine content, and they smoke fewer cigarettes per day (U.S. Department of Health, Education, and Welfare 1979). As stated by Joseph Califano Jr., former Secretary of the Department of Health, Education, and Welfare, “*women who smoke like men die like men who smoke*” (Califano 1979). In 1979, the U.S. Surgeon General (U.S. Department of Health, Education, and Welfare 1979) found that, in the general population, male smoker/nonsmoker mortality was on the order of 1.7 to 1, whereas the Society of Actuaries (1985) found the ratio to be 2 to 1 for the insured population. The results of the U.S. life insurance experience for 1990–91 were that smoker/nonsmoker mortality was 2.32 for males and 2.19 for females.

Hammond (1966) and Wingard (1980) found that smoking is more of a risk factor for men, and Johnson (1977) similarly concluded that heart disease mortality is considerably higher for male smokers than for female smokers, controlling for age, blood pressure and cholesterol level. Hammond (1966) found that, as a group, men smoked more cigarettes per day than women, inhaled to a greater degree, and started smoking at earlier ages. But even controlling for number of cigarettes smoked per day, degree of inhalation, and age cigarette smoking began, male cigarette smokers in Hammond’s study had relatively greater mortality than women.

In the United States, more men than women smoke, but the difference is getting smaller. Smoking prevalence among adults has decreased in recent years, as shown in Table 6. A general indication of the effect of smoking on the sex mortality differential can be estimated by applying smoking prevalences at different points in time, as shown in Table 6, to expectation of life at age 16 for the U.S. life insured population, as shown in Table 5. Using the data in these tables, the smoking prevalences in 1955, 1978, and 1997 yield expectation of life at age 16 differentials by sex of 8.2 years, 6.3 years, and 5.8 years, respectively.

Retherford (1975) compared female-male differences in  $e_{37:\overline{50}}$ , temporary life expectancy between ages 37 and 87, and obtained a difference of 2.71 years for nonsmokers and 5.13 years for the total sample, concluding that if no one in the sample smoked, the female-male difference in  $e_{37:\overline{50}}$  would be reduced by 47.2%. He also concluded that up to 74.5% of the increase in the female-male difference in  $e_{37:\overline{50}}$  from 1919 to 1962 could be accounted for by changes in smoking habits.

Rogers et al. (1999) estimated that cigarette smoking accounts for about 25% of the current overall sex

**TABLE 5**  
**EFFECTS OF SMOKING ON EXPECTATION OF LIFE AT AGE 16**

1990–91 US Life Insurance Experience*			
	Nonsmoker	Smoker	Ratio (Smoker/ Nonsmoker)
Male	63.5	54.1	0.85
Female	68.7	60.6	0.88
Ratio (Male/Female)	0.93	0.89	
1986–89 Canada CIA Basic**			
	Nonsmoker	Smoker	Ratio (Smoker/ Nonsmoker)
Male	70.8	66.7	0.94
Female	72.6	70.0	0.96
Ratio (Male/Female)	0.98	0.95	

\*Calculated using age last birthday, ultimate of SOA Basic 1975–80 Tables for males and females, adjusted by the following factors:

Male nonsmoker	67.3%
Male smoker	156.4%
Female nonsmoker	65.8%
Female smoker	144.1%

\*\*Calculated using age last birthday, the first year of the select period for ages 16 to 80, the 15 year select period for ages 81 to 95, and ultimate for ages 96 to 104.

Source of data: SOA 1975–80 Basic from Society of Actuaries 1982, and 1986–89 Canada CIA Basic from Canadian Institute of Actuaries 1995, both in Society of Actuaries mortality tables library [online database]. SOA smoker/nonsmoker adjustment factors from Individual Life Insurance Experience Committee 2000.

mortality difference. This writer found that, as shown in the table in Appendix G, about 30% of the sex mortality differential in 44 developed countries combined is accounted for by cigarette smoking, by correlating the difference in female and male life

**TABLE 6**  
**ADULT CIGARETTE SMOKING PREVALENCE IN THE UNITED STATES**

	Year		
	1955	1978	1998
Men	54%	38%	25%
Women	25%	30%	21%
Ratio (Men/Women)	2.16	1.27	1.19

Source: 1955 & 1978 from United States Department of Health and Human Services 1988; 1997 from Centers for Disease Control and Prevention 1995–1999.

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expectancy at birth by country with the difference in percentage of male and female deaths attributable to smoking by country.

In determining causes of greater cigarette smoking among males during the 20th century, consideration should be given to the fact that smoking was indirectly encouraged by the U.S. military, since cigarettes were routinely included as part of C and K field rations at a time when military service was compulsory for males but not for females (Personal Communication, John F. Kalben).

### 7.8.2. Other Risk-Taking Behavior

As shown in Table 4, men are exposed to more selected potential risks than are women, but the differences appear to be narrowing for cigarette smoking, illegal drug use, legal psychotherapeutic drug use, and automobile driving.

How much of the male's risk-taking behavior is due to biology and how much to environment is a matter of debate among scientists. Some feel that testosterone is necessary for aggression. There is some evidence that the pattern of male aggression is influenced by sex hormones, but the evidence is not consistent (Geary 1998). Castrated male animals lose aggressiveness and become docile, while treatment with testosterone reverses the changes brought about by castration. There is some evidence that testosterone, or one of its hormonal metabolites, is necessary during fetal development in order to lay down the neural substrate for aggression. There is also evidence that aggressive behavior itself boosts testosterone secretion (Greenstein 1993). Van Goozen et al. (1995) found that the administration of antiandrogens and estrogens to men (male-to-female transsexuals) resulted in a decrease in anger and aggression proneness, while the administration of testosterone to women (female-to-male transsexuals) resulted in an increase in anger and aggression proneness. In every known human society, the level of lethal violence among men is much greater than among women, typically 30–40 times greater. The fact that competition among males is much more likely to escalate to homicide or serious but nonlethal physical assaults than is competition among females, is also true in most, if not all, other primates (Geary 1998).

Even in chimpanzees, males show a greater propensity toward alcohol use than do females. In studies of voluntary alcohol use in chimpanzees and orangutans (*Pongo pygmaeus*), male chimpanzees drank more alcohol than did female chimpanzees—54% of the males consumed enough vodka to become intox-

icated at least once, while only 25% of the females did. None of the orangutans, males or females, showed intoxication (Fitz-Gerald et al. 1968). The international data discussed previously also suggests that cigarette smoking and alcohol use account for some of the sex mortality differential.

There is some evidence that males have been less able to adapt to modern Western-style life. The Seventh-day Adventist religion prohibits smoking and drinking of alcoholic beverages and recommends, but does not require, avoidance of meat, poultry, fish, coffee, tea, other beverages containing caffeine, rich and highly refined foods, and hot condiments and spices. The mortality differences are greater between Seventh-day Adventist males and non-smoking non-Seventh-day Adventist males than between similar groups of females, particularly for coronary heart disease (Phillips et al. 1980). In fact, a Dutch study found that the sex mortality differential almost disappeared among Seventh-day Adventists. In this study, at the age of 26.8, which was the mean age at baptism of the Seventh-day Adventists, the life expectancy was 52.3 years for males and 52.5 years for females (Berkel and de Waard 1983). Studies of migrants from Japan to the United States have shown similar results—the increasing risk of coronary heart disease has been much less in women than men (Gordon 1967).

Waldron (1983) hypothesizes that women's role in bearing and nursing children has led societies to assign dangerous tasks, and other duties incompatible with child rearing, to men. Because alcohol intoxication is also incompatible with child rearing, she also suggests that social pressures developed against women's heavy drinking, thereby reducing women's risk of death.

Coronary artery disease, other vascular diseases (such as strokes), and diabetes have a marked correlation with excess weight. Although females have a greater propensity to be overweight than do males, overweight men have about 25% more excess mortality (expressed as a percentage of the average) than do overweight women (Potts 1970).

Wingard (1980, 1982) studied the effects of 16 demographic and behavioral factors (including smoking, occupation, use of health services, and alcohol consumption) on mortality by sex using multiple logistic analysis. She found that adjustment for some factors (smoking and alcohol) decreased the relative mortality risk for men compared with women, while others (physical activity, physical health status, and marital status) increased the relative risk. Adjustment for all 16 factors actually *increased* the relative mor-

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tality risk for men, compared with women, from 1.5 to 1.7, implying that there is an inherent difference between men and women that explains the sex mortality differential. Wingard's study has been called "*undoubtedly the best individual-level analysis of sex mortality differentials published to date*" (Nathanson 1984).

A similar prospective study, which also used multiple logistic analysis, controlled for age, marital status, education, cigarette smoking, cholesterol, systolic blood pressure, fasting plasma glucose, and obesity. Adjustment decreased the relative mortality risk (male/female) from 1.7 to 1.3 for all causes and from 4.8 to 2.4 for ischemic heart disease. When the analysis was limited to healthy individuals, the relative mortality risk was 1.2 for all causes and 2.0 for ischemic heart disease. So these biological and behavioral risk factors explain a good portion of the sex mortality differential from all causes, but a substantial sex differential remains in mortality due to heart disease (Wingard et al. 1983).

Rogers et al. (1999) also studied the effects of behavioral factors on mortality by sex and found that smoking, alcohol use, exercise and body mass accounts for some, but not nearly all, of the sex mortality gap. Belloc (1982) found that good health practices are associated with lower mortality rates, especially among men. In this study, good health practices included not smoking, drinking moderately, if at all, maintaining body weight within desirable limits, exercising in leisure activities, sleeping seven or eight hours per night, eating breakfast, and not eating between meals.

Nathanson (1977) also cited sex differences in preventive behaviors which, along with more current data, is shown in Table 7. The table contains sex-distinct ratios of those who participated in preventive behaviors compared with the total population. As shown in this table, sex ratios have narrowed considerably since the 1970s. In fact, several preventive health behaviors and procedures, such as sigmoidoscopy, proctoscopic or digital rectal examination, and flu or pneumonia shots, are now utilized more by men than by women.

## 7.9 Utilizing Health Care Services

One hypothesis for the sex mortality differential involves differences between how the sexes care for themselves when they become ill. Sowder (1954) speculated that "*women possibly have a greater tendency to stay away from work for mild illnesses than*

*men, to go to bed sooner and stay longer, to go to their physician earlier and return more often, and to follow their physician's instructions more faithfully.*" In describing how women take better care of their bodies, Madigan (1956) said, "*When they fall ill, they are more apt to give the trouble instant attention and thus nip it in the bud. . . Men often push themselves too far physically in pursuing their various ends, and ride the machine until it is metaphorically out of gas. It seems likely that these different outlooks have some relation to the divergent death rates.*"

There is little evidence regarding differential health attitudes between the sexes, but some believe that women are more sensitive to physical discomforts; more likely to act on such discomforts; more willing to change activities, seek medical care, and take drugs; and, more able to alter responsibilities when experiencing chronic conditions. Some think, because women are more familiar with health care for obstetric, gynecological, and pediatric purposes, their access to health care is increased, so they are more likely to have an established health care source (Verbrugge 1983).

In a summary of utilization of health care, Waldron (1976) found both differences and similarities between the sexes. She found that men and women both omitted mention of many conditions found clinically, but men were more likely than women to underestimate their illness. Both sexes were similar in the extent of agreement between conditions reported by a respondent and those found clinically. Of those reporting a symptom, there was little difference between the sexes in the proportion of people who subsequently visited a doctor. Men and women both delayed seeking medical attention the same length of time after the first symptoms of cancer or a myocardial infarction. Women visited doctors more, and made more use of preventive services than men did.

Waldron found little difference between the sexes in the proportion of patients who complied with doctors' recommendations. She later reported that sex differences in the use and effectiveness of medical care do *not* contribute to men's higher rates of ischemic heart disease mortality (Waldron 1992). As shown in Tables 7 and 8, there is some evidence that females use medical care more than males do, although the differences appear to be narrowing.

## 7.10 Socioeconomic Status

The sex mortality difference varies by socioeconomic status; typically the difference is at its lowest

**TABLE 7**  
**COMPARISON OF SEX DIFFERENCES IN PREVENTIVE BEHAVIORS**

Class of Behavior	Nathanson (1977)			Current		
	Year	Preventive Health Behavior	Ratio (Female/Male)	Year	Preventive Health Behavior	Ratio (Female/Male)
Preventive medical examination	1964	Preventive physical examination in past year	1.63	1999	Visited a doctor for a routine checkup in past year	1.25
	1964	Preventive visits	1.58	1999	Visited a doctor for a routine checkup in past 2 years	1.14
	1973–1974	Medical and special examination without illness	1.28			
Preventive tests	1964	Chest x-ray in past year	0.97	1999	Blood pressure taken by a health professional in past 6 months	1.11
				1999	Blood pressure taken by a health professional in past year	1.08
				1999	Blood pressure taken by a health professional in past 2 years	1.04
				1999	Ever had blood cholesterol checked	1.06
				1999	Ever had a sigmoidoscopy or proctoscopic examination	0.96
				1995	Ever had a digital rectal exam	0.92
Immunization	1960	Polio immunization status	1.21	1999	Flu shot during last year	1.04
	1957	One or more polio inoculations	1.50	1997	Flu shot during last year of adults 65+	0.97
				1999	Ever had a pneumonia vaccination	0.96
				1997	Ever had a pneumonia vaccination of adults 65+	1.06
Dental care	1964	Dental care during past year	1.11			
	1974	Dental visits per person per year	1.10			
Eye examination	1964	Eye care during last year	1.16			
	1973	Eye examination during past year	1.01			
Seatbelt use				1997	When driving or riding in a car, use seatbelts always or nearly always	1.12

Source: Nathanson 1977, originally published in *Journal of Community Health*, reprinted with permission. All current data is from Centers for Disease Control and Prevention 1995–1999.

**TABLE 8**  
**HEALTH CARE UTILIZATION RATIOS BY SEX**

	Year of Data	Ratio (Female/Male)
<b>Physician contact, based on interview, by poverty status</b>		
Poor	1994-96	1.33
Near poor	1994-96	1.26
Nonpoor	1994-96	1.29
<b>Ambulatory care visits to physician offices and hospital outpatient and emergency departments</b>		
All places	1997	1.30
Physician visits	1997	1.33
Hospital outpatient departments	1997	1.42
Hospital emergency departments	1997	1.09
<b>Short-stay hospitals</b>		
Discharges	1996	1.01
Days of care	1996	0.94
Average length of stay	1996	0.93

All are age-adjusted

Source of data: National Center for Health Statistics 1999.

for the highest socioeconomic status (Rogers et al. 1999). Also, a Finnish study showed that women's mortality varies less by socioeconomic status than men's mortality (Koskinen and Martelin 1994). As shown by Rogers et al. (1999), women in the United States, on average, have lower social and economic characteristics—such as employment status, family income, education, and marital status—than do men. Thus, the sex mortality differential in the U.S. would be even wider if men and women had the same social and economic characteristics.

## 7.11 Labor Force Participation

The conventional explanation that excess male mortality is attributable to greater male labor force participation (Wallace 1996; Graney 1979; Haynes et al. 1984; Rosenberg and Luckner 1998) is *not* supported by evidence (Waldron 1991, 1992; Pampel and Zimmer 1989). It has been estimated that occupational exposures account for 5–10% of the total sex difference in mortality (Waldron 1991). Ramey (1982) observed, “*It is ironic that. . . women are now being warned that if they move into roles of power and*

*achievement they will kill themselves. The very opposite effect actually occurs.*”

Passannante and Nathanson (1987) found that female labor force participants experience substantially lower death rates than the total female population. In addition, the population as a whole experiences narrower sex mortality ratios than does the labor force at every age and marital status, with the exception of the nonmarried age 55–59 population. Similarly, the sex mortality ratios for the total population were smaller than for most major occupational categories. They also found that causes of death that are presumably affected by occupational affiliation do not have smaller sex mortality differentials than those causes that are not presumably affected by occupational affiliation.

A study in Texas showed that employed women had more favorable blood lipid profiles (important in heart disease) than nonemployed women, regardless of smoking, exercise, or alcohol habits (Hazuda et al. 1986). Although not specifically addressing mortality, Waldron et al. (1982) presented evidence that labor force participation has little effect on the general health of middle-aged, married women in the United States. Haynes et al. (1984) found that employment per se was not associated with the incidence of coronary heart disease in women, but working women who had ever been married, had raised children, and had been employed in clerical work were at increased risk of developing coronary heart disease.

Further evidence can be found by considering that the 1971 Group Annuity Mortality Table, which included only those people working outside the home, shows greater sex mortality differentials than does the 1971 Individual Annuity Mortality Table (Lautzenheiser 1976; Greenlee and Keh, 1971; Cherry 1971).

Lower mortality observed among women in the labor force, compared with women not in the labor force, exists despite the fact that, among women, smoking prevalence currently tends to be higher among the employed than among those not in the labor force (Brackbill et al. 1988; U.S. Department of Health and Human Services 1985; Sterling and Weinkam 1978; Schuman 1977). Unexpectedly, Sorenson and Pechacek (1986) found that the prevalence of smoking was higher among employed women than employed men. If smoking prevalence by sex among the employed was the same as smoking prevalence by sex in the population, one would expect larger sex mortality differentials among the employed.

Sowder (1954) noted that the widening of the sex mortality differential was occurring at a time when

more and more women were becoming employed and in occupations once monopolized by men. Herdan (1952) observed that working conditions, specifically safety measures, hygiene, and reduced working hours, have improved considerably during the 20th century; as such, one would expect a decrease in excess male mortality. In fact the opposite has happened. Also during this period, industrial occupation by women has increased, thereby exposing them more to occupational risk. The increasing female labor force participation in the United States is illustrated in Table 9.

## 7.12 Sex Roles in a Social/Cultural Context

Male and female sex roles have been cited as reasons for the sex mortality differential (Graney 1979; Jourand 1971; Harrison 1978; Ortmeyer 1979). Graney (1979) states, without evidence, that the sex mortality differential in infants and young children is in part attributable to increased protectiveness and decreased exposure of female compared with male children. Differences in the socialization of boys and girls are widespread cross-culturally, which prepare boys to participate in more dangerous activities (Waldron 1983). Ortmeyer (1979) states that “*the learned denial of pain and the hesitancy to seek help in childhood might be responsible for the adult male’s low utili-*

*zation of the health care system.*” There is, however, little evidence to either support or refute this hypothesis, primarily because it is difficult to isolate sex roles from other possible causes.

Berin et al. (1990) note that the widening of the sex mortality differential primarily took place during the 19th and 20th centuries when the role of females was changing. Sowder (1954), a public health physician, speculated that reasons for the widening of the sex mortality differential may include differences in the reaction of men and women to modern life, including work. “*It is possible that women escape the consequences of worry, frustration, disappointment, and tension to a greater degree than men by being more vocal about these conditions, through tears, or occasionally hysterics. The reaction of men, on the other hand, may be in the form of coronary disease, hypertension, or ulcers.*” In response to the widening sex mortality differential, Bond (1957), an epidemiologist, tried to call “*to your attention a problem that is essentially man’s doing, and should, therefore, be his for undoing. I should like to induce a little more humble, relaxed, and objective attitude of men toward themselves, for it is here perhaps more than anywhere else that the key to the solution of this problem is to be found.*”

The scientists queried by Moriyama et al. (1958) felt that the increasing sex differential in cardiovascular-renal mortality may have been due to a poorer tolerance in men than in women for high-fat, high-caloric diets and obesity, or to men eating proportionately more fats and tending more toward a sedentary mode of life. Other possibilities mentioned that could have been increasing more for men than for women were stress, tobacco use, and exposure to noxious gases (such as gasoline fumes). Also advantageous to women was less childbearing and better medical care.

Haynes et al. (1980) found that Type A behavior (enhanced aggressiveness, ambitiousness, competitive drive and a chronic sense of time urgency), a risk factor for heart disease, is more prevalent among men than women, but the relative risk of heart disease with Type A behavior is slightly higher for women than for men. Therefore, it is questionable if the Type A behavior has a significant effect on the sex mortality differential (Wingard 1982).

Jourand (1971) cites research that men typically have lower self-disclosure than women—that is, they reveal less information about themselves to others. He concluded from this that men must be more tense, and this added burden of stress and expenditure of energy can be a factor in higher male mortality. Jourand also

**TABLE 9**  
**HISTORICAL FEMALE LABOR FORCE**  
**PARTICIPATION IN THE UNITED STATES**

Year	Percent of Female Population Employed in the Labor Force
1900	18.8
1910	23.4
1920	21.0
1930	22.0
1940	25.4
1950	30.9
1960	34.9
1970	43.3*
1980	51.5
1990	57.5
1998	59.8

\*The methodology used to calculate the rate changed as of 1970. Using the previous method, the rate for 1970 would have been 41.9%.

Source of data: Jacobs 1999 for 1970–1998; United States Bureau of the Census 1975 for 1900–1970.

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stated a hypothesis that men are less likely to be sensitive to weak “all-is-not-well” signals, so they do not take action on them as soon as women do. He continued that men, after retirement or through another loss of masculine identity, may lose their reason for living and die, while women in a similar situation manage to find new meaning and go on living.

Harrison (1978) said that “*alcohol serves both as a symbolic manifestation of compensatory masculinity and as an escape mechanism from the pressure to achieve*,” but gives no evidence to support this hypothesis. He also noted that “*any biogenetic factor [for sex mortality differentials] is exacerbated by male role socialization*” and “*male anxiety about the achievement of masculine status seems to result in a variety of behaviors [such as smoking, alcohol consumption] which can be understood as compensatory.*”

Goldberg (1976) advanced an intriguing hypothesis to explain why men die younger than women. He believed that the male is unconsciously afraid that he can’t survive without the woman. Goldberg cited statistics regarding 1) higher male than female mortality among the divorced and widowed populations, 2) studies of high male mortality following the death of their wives, 3) higher suicide rates following the death of their mothers, 4) higher suicide rates among bachelors compared with spinsters, and 5) higher male than female remarriage rates soon after divorce. Married men and women have lower mortality rates than the unmarried—including those who never married, divorced, and became widowed. The marital status effects vary by sex and have changed over time.

In 1960, the beneficial effects of marriage on mortality were consistently stronger for males than for females, especially compared with that of the divorced. By 1998, the beneficial effects of marriage were still stronger for males than for females, but only in the 25–74 age group, and were not as strong as they had been in 1960 (Trowbridge 1995, Murphy 2000). Other evidence supporting this hypothesis comes from a study of centenarians, which found several lifelong single female centenarians, but all of the men in the study had been married, and many of the oldest old men had been married 70 or 80 years (Perls et al. 1999).

Other hypotheses of contributions to declining female mortality are the liberation of women from prolonged and exhausting household drudgery, new job opportunities, and the possibility to slow down at ages 40–60, after children are grown (Moriyama et al. 1958). In discussing the male’s greater mortality from

violence, Moriyama (1983) stated that “*the life style of the male is such that he takes more chances than the female, is less stable in the face of adversity, and more frequently the victim of violence.*”

It has been speculated that, as women’s roles move from primarily domestic to more public, the mortality of women will be adversely affected (United Nations 1991). Nathanson studied female mortality and several indices of women’s position, including education, employment, fertility, political power, marriage age, divorce rate, and smoking for 22 developed countries. She concluded that “*neither the data we have presented nor the scenarios for the future that we can anticipate are consistent with the existence of a causal relationship between movement toward gender equality and women’s mortality*” (Nathanson 1995).

## 7.13 Environmental Factors

Environment plays an important role in mortality, so it also affects the sex mortality differential. For example, Sorenson et al. (1988) studied mortality rates by cause for adult adoptees. They found that the risk of dying from cancer for adoptees is five times as great if one of the adoptive parents has died of cancer before age 50, compared with adoptees whose adoptive parents were both alive at that age. But the risk of dying from cancer for adoptees was not significantly increased if one of the biological parents has died from cancer before age 50.

## 7.14 Interaction Between Factors

Because of the interaction between biological factors and environmental factors, it can be difficult, if not impossible, to determine the relative contribution to the sex mortality differential of the two sets of factors. Although not necessarily related to the sex mortality differential, a classic example of the interaction between biological and environmental factors is melanoma, a deadly form of skin cancer. Melanoma is strongly associated with the interaction of the biological factor of a fair complexion with the environmental factor of exposure to ultraviolet light (Personal Communication, Robert Fineman).

Breast cancer is an example of the interaction between biological and environmental factors, as related to the sex mortality differential. Because of sex differences in anatomy, breast cancer is predominately female, but environmental factors, such as diet, influ-

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ence the occurrence of the disease. Changes in diet can influence breast cancer mortality rates, thereby affecting the sex mortality differential (Tickle 1997; Waldron 1983). Evidence has shown that changes in diet are associated with estrogen and androgen metabolism, which alters the risk of heart disease (Hill et al. 1980). Even prenatal sex mortality differentials, which are likely primarily due to biological factors, may change if nutritional or other medical improvements tend to reduce prenatal mortality more for one sex than the other.

In fact, Kitagawa (1977) stated that “*There is general agreement. . . that biological and environmental factors are so interdependent that it is not possible to determine the separate influence of each.*” This view was amplified by Lopez and Ruzicka (1983) who, in discussing a predominately constitutional versus a predominately environmental explanation of sex mortality differential, stated that “*these determinants are generally closely intertwined with one another in their influence on the sex pattern of survival and, consequently, any disaggregation of them is bound to be potentially misleading.*”

The interdependency between the biological and environmental factors has been likened to the Heisenberg Uncertainty Principle (Personal Communication, Robert Fineman). This principle states that the location and speed of subatomic particles cannot both be measured exactly, at the same time, even in theory. The result has nothing to do with shortcomings in the measuring instruments, the technique, or the observer. An accurate measurement of one necessarily implies uncertainty in the measurement in the other. Any attempt to precisely measure the speed of a subatomic particle, such as an electron, will move it about in an irregular manner, so that a concurrent measurement of its position is impossible. Applying this concept analogously to the biological and environmental factors’ effects on the sex mortality differential, it has been speculated that it is impossible, even in theory, to measure the effects of both the biological factors and the environmental factors at the same time, because any attempt to measure one necessarily alters the other.

## 7.15 Putting It All Together

As we have seen, evidence supports both biological and environmental causes in explaining the sex mortality differential. Palmore (1980) stated “*Based on the ratio of male to female mortality rates at various ages, I would estimate that about half the greater lon-*

*gevity of women is due to genetic differences and about half is due to difference in life-style.*” Pressat (1973) suggested that because women had an excess life expectancy of less than two years in pre-industrial societies, biological factors may account for that amount of greater female life expectancy, but the rest is due largely to environmental factors.

Pinnelli (1997), in discussing what she calls “male supermortality,” stated that a difference of life expectancy at birth between males and females of five years is considered normal. She maintained that a greater difference indicates that males are disadvantaged with respect to females, in part because of their behavior; which is more aggressive, they take more risks, and are not very protective of their own health. Lower difference indicates that women are disadvantaged regarding medical care, diet, and distribution of employment.

“*The factors accounting for sex differentials in mortality, and their widening over time, is a mix of environmental impact (both man-made and natural), social structures and individual behaviours, interacting with biological susceptibilities*” (Kirmeyer and Heligman 1985). The fact that the sex mortality differential has changed over time is an indication that biological differences are not the sole reason for the differential.

What about the difference in mortality between the sexes due to cardiovascular disease? Is it biological/genetic or environmental/behavioral? The historical study of mortality from cardiovascular disease showed that the sex mortality differential due to cardiovascular disease deaths did not emerge until the 1920s. As seen in Figure 3, the sex mortality differential in the United States also began to widen in the 1920s. Nikiforov and Mamaev (1998) suggested that factors that affect males and females differently began entering industrialized societies in the 1920s, and when the influence of these factors stabilized in the 1960s, the sex mortality differential also stabilized. Thus none of the hypotheses described above entirely explains this phenomenon. The genetic and biological hypotheses do not account for how the changes occurred so rapidly. The hormone, iron overload, and Type A personalities hypotheses have similar shortcomings. The hypotheses that females have a more favorable diet and take better care of their health do not explain how cardiovascular disease mortality increased for males from the 1920s to the 1960s.

Smoking can explain part of the sex mortality differential, since the sex mortality differential from ischemic heart disease in the 45–54 age group is 4.55

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for nonsmokers and 6.39 for smokers, but it is not the complete answer. Even studies that jointly consider several variables, such as Wingard (1982) and Johnson (1977), cannot explain the sex mortality differential from ischemic heart disease. Although many of these hypotheses may help explain the sex mortality differential, the shortcomings of all the current hypotheses indicate that the definitive cause of the sex mortality differential is not presently known (Nikiforov and Mamaev 1998).

Scheinfeld (1950) felt that the sex mortality differential has changed over time because, as environments have improved, the genetic disadvantages of the male have become more marked, compared with the female. He continued that the female's extra margin of resistance isn't sufficient to make much difference when conditions are very bad, but the more conditions improve, the more the slight advantage of the female comes to the fore. "*Under like conditions, females are better adapted to cope with most human afflictions because they are genetically better constructed and*

*have a more efficient chemical system*" (Scheinfeld 1965).

Because of the evidence that genetic, biological, hormonal, and iron overload factors have on the sex mortality differential, it is possible that they interact with behavioral and environmental factors that manifest the changes in the sex mortality differential during the 20th century. As pointed out by Madigan (1956), it is possible that different explanations account for the sex mortality differential at different ages. Differentials in infants may be primarily biological in origin, while social/behavioral reasons may explain more of the differentials in adults.

After much study and research, the conclusion of the author is that there are significant biological reasons for the sex mortality differential. These basic biological differences can be masked, as has been the case during much of mankind's history because of the poor environment and poor maternal and childbirth practices, and can be exacerbated, as by cigarette smoking in the male.

