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The Sex Differential in Mortality Rates and Underlying Factors

Leonie Tickle

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ltickle@efs.mq.edu.au
School of Economic and Financial Studies
Macquarie University
Sydney NSW 2109 Australia

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School of Economics and Financial Studies
Macquarie University NSW 2109
Fax No: 61 2 9850 9481
Email: lschalch@efs.mq.edu.au

THE SEX DIFFERENTIAL IN MORTALITY RATES AND UNDERLYING FACTORS

by

Leonie Tickle

Macquarie University

This paper examines the sex differential in population mortality in Australia, and the possible factors underlying this differential. The five main cause of death groups which contribute to the current sex differential in mortality in Australia are ischaemic heart disease, lung and related cancers, respiratory disease, cancers except lung and related cancers, and accidents and suicide. For each of these causes of death, the sex differential in mortality in different countries and over time in Australia has been presented. Research on the various genetic/biological and lifestyle/environmental factors which are thought to be associated with each of these causes of death is also presented. The overall conclusion is that the data and research suggest that environmental and lifestyle factors make an important contribution to the sex differential in mortality in Australia.

CONTENTS

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Section 1: INTRODUCTION

In all developed countries males have higher overall rates of mortality than females. The extent of the excess male mortality varies by age and cause of death, although in most countries male rates exceed female rates in all age groups and for the majority of causes (UN Secretariat, 1988).

Numerous studies have attempted to isolate the factors underlying the excess male mortality. These factors may be broadly grouped into **biological or genetic** factors and **environmental or lifestyle** factors. Most explanations offered for the observed sex difference recognise that both sets of factors play a part; however, researchers disagree on the relative importance of innate versus environmental factors, with no consensus having being reached.

The fact that excess male mortality is so widespread in the developed world may seem to indicate that the sex differential is largely innate. However, the size of the differential varies markedly over time, between countries, and by cause. In addition, causes thought to have a strong behavioural component often display the widest sex difference. Hence, although the focus of this paper is the current Australian situation, mortality data for previous years and for other countries are also be presented in an attempt to illuminate the underlying causes of the sex differential. Because environmental and genetic factors vary in importance according to the cause of death, several of the most important causes are considered separately.

Sources of data and measurement of the sex differential are outlined in section 2, and data on sex differences in mortality around the world and in Australia are presented in sections 3 and 4 respectively. An analysis of data and research relating to the specific causes of death which make a major contribution to the sex differential in Australia is then presented, including ischaemic heart disease, lung cancer, respiratory diseases, other cancers, and accident and suicide in sections 5 to 9 respectively. Finally, an assessment of the overall contribution of environmental versus biological factors is made in section 10.

Section 2: MEASUREMENT OF THE SEX DIFFERENTIAL

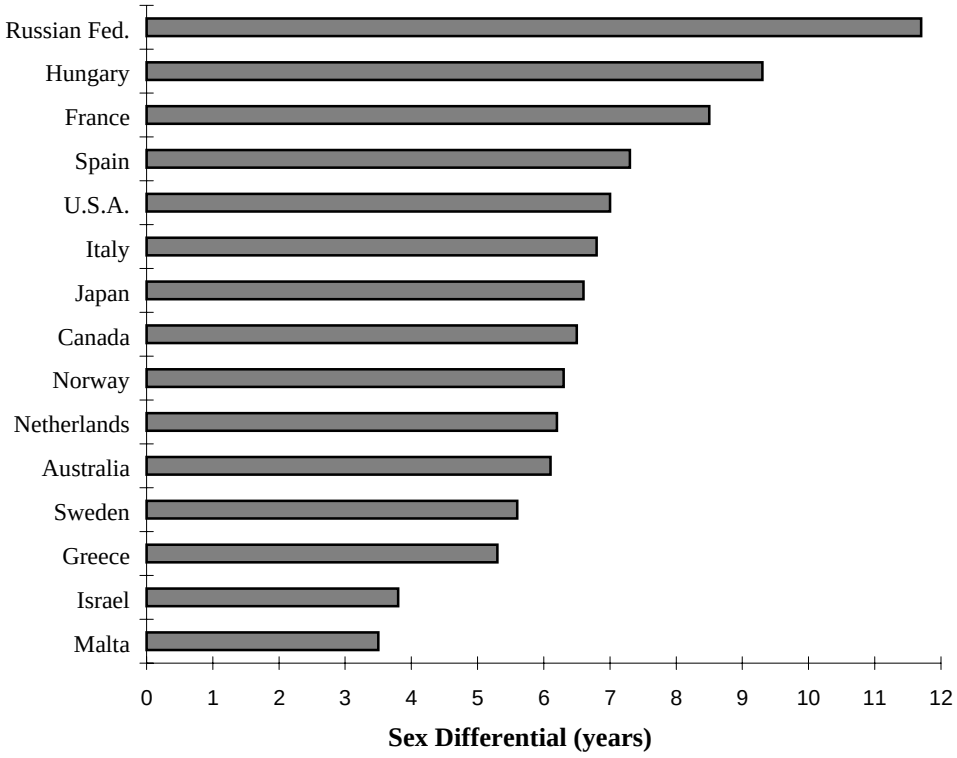
The sex differential can be measured or illustrated in various ways; in this paper two main measures are used. The first is the difference between female and male expectation of life, which is the expected excess future lifetime in years for the average female as compared to the average male, usually measured from birth. The second measure is the ratio of the male mortality rate to the female mortality rate, with the sex difference in mortality indicated by the extent to which this ratio is different from one. This measure may be applied to overall rates, age-specific rates or cause-specific rates.

The two measures differ in their emphasis. The expectation of life measure attaches more importance to causes of death which act earlier in the lifespan, as these causes of death result in the loss of a larger expected future lifetime. In addition, the expectation of life measure reflects mortality at all future ages, whereas the ratio of mortality rates may be used to reflect the sex difference in mortality for a particular age group. The measures should give similar results when considering trends between countries or over time.

Section 3: SEX DIFFERENTIALS IN MORTALITY AROUND THE WORLD

A distinction is often made between developed and developing countries; the sex differential in expectation of life at birth is larger in the developed regions, averaging 7.0 years compared to 2.8 years for developing regions (Jain, 1994). Figure 1 shows the sex differential in the expectation of life at birth for various developed countries.

Figure 1: Sex differential in the expectation of life at birth for various developed countries, 1991-1993.



Source: WHO (1995)

The sex differential in the expectation of life at birth for developed countries has increased markedly over this century - prior to the 1930s the differential was typically only two or three years (Lopez, 1983). For a number of countries, including Japan, Bulgaria, Hungary, Denmark, the Netherlands, Ireland, Italy and Spain, the sex differential was less than two years during some part of the period 1900-1930 (UN

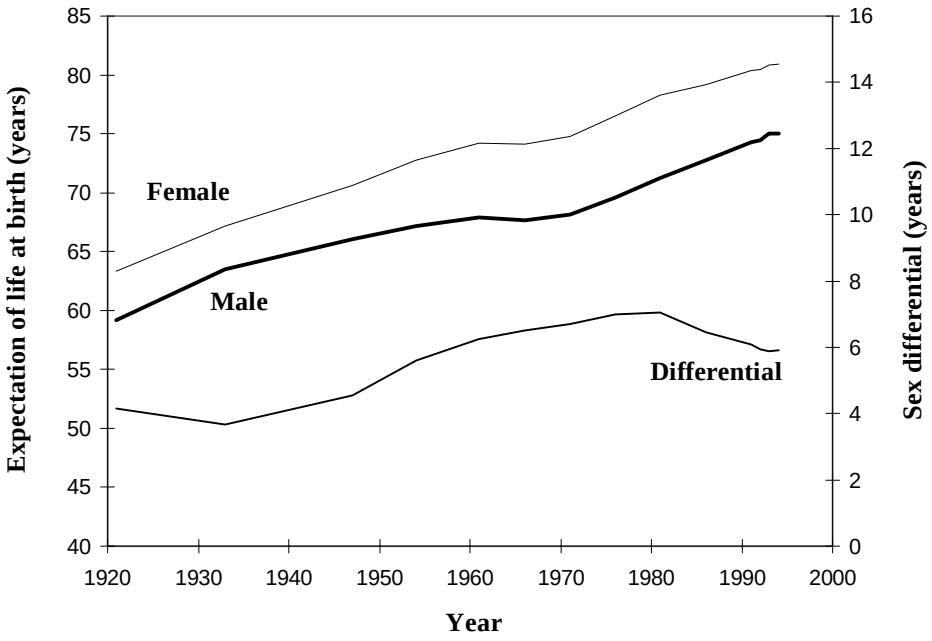
Secretariat, 1988). Between the 1930s and the 1970s, the sex differential increased in all developed countries (UN Secretariat, 1988). The marked increase in the sex differential has been attributed to increasing male excess mortality from cardiovascular diseases, cancers, respiratory diseases and motor vehicle accidents, along with the reduction of maternal deaths among women (Lopez, 1983). During the 1970s and 1980s, the increasing trend in the sex differential in many countries has either reversed - for example, the UK (Tickle, 1996), USA (Knudsen & McNown, 1993), Australia (Lee & Smith, 1988), Canada, Finland and Portugal (UN Secretariat, 1988) - or stabilised (UN Secretariat, 1988).

Section 4: THE SEX DIFFERENTIAL IN MORTALITY IN AUSTRALIA

Historical Trends in the Sex Differential

The most recently released Australian population data indicates that the expectation of life at birth is 80.9 years for females and 75.0 years for males, a difference of 5.9 years (ABS, 1995a). However, the sex differential has not always been so wide. Figure 2 shows the expectation of life at birth for males and females for the period 1921-1994, together with the sex differential in the expectation of life at birth.

Figure 2: Expectation of life at birth for Australian males and females, and the sex differential in the expectation of life at birth, 1921-1994.

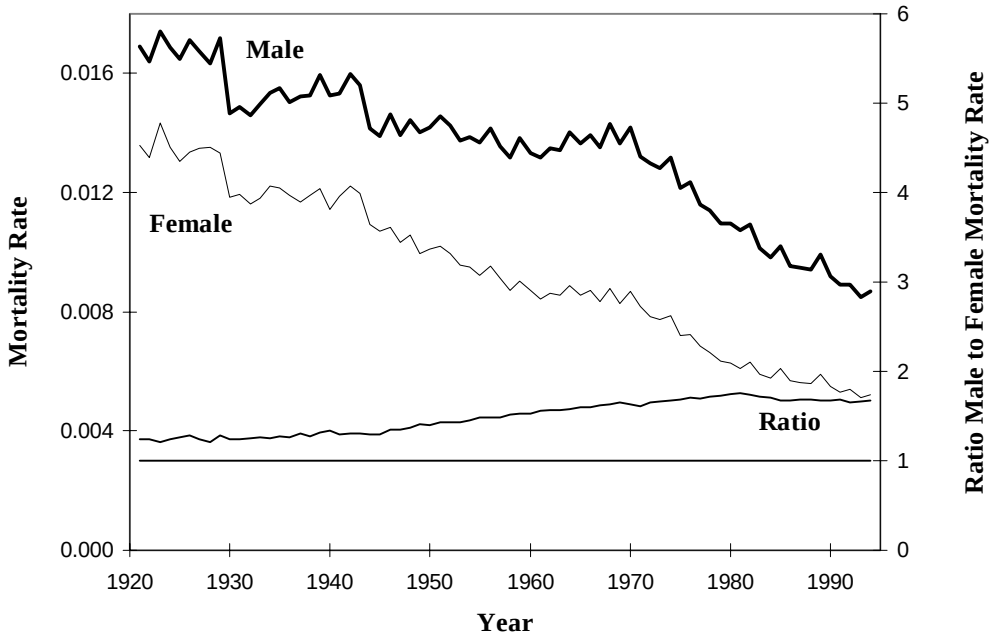


Sources: Office of the Australian Government Actuary (1995) for years 1921-91 (based on three year averages)
ABS (1995a) for years 92-94

The sex differential was 4.2 years in 1921, dropped to 3.7 years in 1933 and then increased steadily until the early 1950s, after which time it began to increase more rapidly. It reached a maximum of around 7.0 years in the late 1970s and early 1980s and since that time has declined by about 1.1 years.

As mentioned previously, the sex differential in mortality may also be indicated by the ratio of the male to female mortality rates. Figure 3 shows the all cause age-standardised mortality rates for males and females as well as the ratio of the male to female rates. The mortality rates have been age-standardised using the 1988 mid-year population, so are not distorted by changes in the age-structure of the population.

Figure 3: All cause age-standardised mortality rates for Australian males and females, and the ratio of the male to female rate, 1921-1994.



Sources: d'Espaignet et al. (1991) for years 1921-1988 (1988 population as standard)
ABS (1995a) for years 1989-1994 (1991 population as standard)

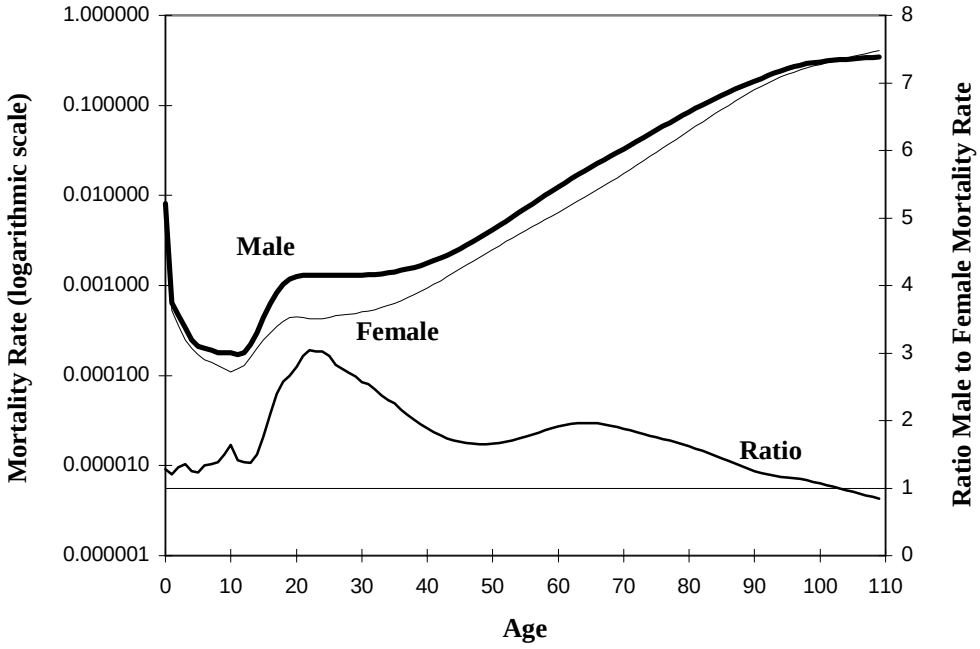
The male to female ratio was 124% in 1921, and remained relatively constant until 1930, after which time it began to increase. The rate of increase became more rapid after about 1950, and the ratio reached a maximum of 176% in 1981. Since that time the ratio has been reducing, the figure for 1994 being 167%.

It is evident from the previous figures that the excess male mortality has varied markedly over time. These wide variations would seem to indicate that environmental factors are having an influence on mortality experience.

The Sex Differential by Age

The previous section reviewed changes in the sex differential in overall mortality. However, the extent to which male mortality exceeds that for females varies over the lifespan. Figure 4 shows male and female mortality rates by age based on Australian population data for the period 1990-92, as well as the ratio of male to female mortality rates at each age.

Figure 4: All cause age-specific mortality rates for Australian males and females by age, and the ratio of the male to female rate, 1990-1992.



Source: Office of the Australian Government Actuary (1995).

It can be seen that male mortality exceeds female mortality for all but the very oldest ages. The differential is greatest in early adulthood where it reaches a maximum of 305% at age 22. The differential is also high between ages 50 and 80.

Another way to illustrate the importance of the sex differential at various ages is to use the decomposition method of Pollard (1982). This method allows the total sex difference in the expectation of life to be apportioned according to the contribution of each age group. Table 1 (based on figures from Pollard, 1996) shows the contribution of each age group to the total sex difference in the expectation of life at birth for the year 1992. For example, heavier male mortality for the age group 50-69 contributed 2.04 years (or 34%) to the 5.99 year sex differential.

Table 1: Contribution of sex mortality differentials by age to the total sex differential in expectation of life at birth in Australia, 1992.

Age Group	Contribution by age group to mortality differential in expectation of life at birth, in	
	years	% of total differential
0	0.15	2.5
1 - 4	0.01	0.2
5 - 14	0.04	0.7
15 - 29	0.57	9.5
30 - 49	0.68	11.3
50 - 69	2.04	34.1
70 and over	2.50	41.7
Total	**5.99**	**100.0**

Source: Pollard (1996)

Figure 4 and Table 1 both demonstrate that a large portion of the total sex differential in mortality is due to excess male mortality for ages 50 and over. It should be noted that even though the ratio of male to female mortality rates in the age group 20 to 30 years is very high (as illustrated in Figure 4), this large differential does not have a major impact on the expectation of life because the numbers of deaths at these ages is small.

The Sex Differential by Cause

Figure 3 indicates that male mortality for all causes and ages combined exceeded female mortality by about 67% in 1994; however, the sex differential varies markedly between causes. Pollard (1996) has analysed male and female mortality rates by cause for Australia in 1992, and the following table is based on figures from his analysis. Table 2 shows the cause-specific mortality rates for Australian males and females in 1992, standardised by age using the total 1992 population, as well as the ratio of the male to female mortality rate for each cause. The causes of death are arranged in order of the male to female ratio from highest to lowest, and are based on the World Health Organisation International Classification of Diseases (WHO, 1977).

Table 2: Standardised mortality rates per 100,000 by cause for Australian males and females, 1992, and ratio of the male rate to the female rate.

Cause	Standardised Male Rate (per 100,000)	Standardised Female Rate (per 100,000)	Ratio Male to Female Rate
AIDS	7	0.25	28.0
Suicide	21	5	4.2
Cancer of lung, mouth, throat, etc.	71	20	3.5
Cirrhosis of the liver	10	3	3.3
Motor and non-motor accident	38	15	2.5
Respiratory diseases	84	37	2.3
Other violence	4	2	2.0
Ischaemic heart disease (IHD)	233	125	1.9
Infectious diseases	6	4	1.5
Other circulatory disease	73	52	1.4
Cancers except lung etc.	169	119	1.4
Cerebrovascular disease (eg. stroke) (CVD)	70	59	1.2
Congenital/perinatal	10	8	1.2
Obstetrics	0.0	0.1	-
All other causes	93	70	1.3
Total	**887**	**521**	**1.7**

Source: Pollard (1996)

The ratio is very high for AIDS as expected, since the majority of sufferers in Australia are males. Male excess mortality is also high for suicide, lung and related cancers, cirrhosis, motor and non-motor accidents and respiratory disease, the male rate being more than twice the female rate for these causes.

Whilst the above analysis is useful, it does not indicate which causes make a major impact on the sex differential in the expectation of life. The ratio for cirrhosis, for example, is high, but this cause makes only a small contribution to the sex differential because the number of deaths by this cause are limited. The importance of each cause can be illustrated by apportioning the total sex difference in the expectation of life by cause (Pollard, 1982). Table 3 shows the contribution of each cause grouping to the total sex difference in the expectation of life at birth for the years 1982 and 1992 (Pollard, 1996). For example, ischaemic heart disease (mainly "heart attack") contributed 1.69 years (or 28%) of the 5.99 year sex differential in 1992. The causes are listed according to the impact on the 1992 sex differential.

Table 3: Contribution of sex mortality differentials by cause to the total sex differential in expectation of life at birth in Australia, 1982 and 1992.

Cause of death	Contribution by age group to mortality differential in expectation of life at birth, for			
	1982		1992	
	years	% total	years	% total
Ischaemic heart disease (IHD)	2.38	33.9	1.69	28.2
Cancer of lung, mouth, throat etc.	0.85	12.1	0.81	13.5
Respiratory diseases	0.80	11.4	0.59	9.9
Motor and non-motor accident	1.06	15.1	0.58	9.7
Cancers except lung etc.	0.34	4.8	0.56	9.4
Suicide	0.29	4.1	0.42	7.0
Other circulatory disease	0.39	5.5	0.30	5.0
AIDS	0.00	0.0	0.18	3.0
Cerebrovascular disease (CVD)	0.19	2.7	0.15	2.5
Cirrhosis of the liver	0.15	2.1	0.14	2.3
Congenital / perinatal	0.12	1.7	0.10	1.7
Other violence	0.05	0.7	0.04	0.7
Infectious diseases	0.02	0.3	0.04	0.7
Obstetrics	-0.01	-0.1	0.00	0.0
All other causes	0.40	5.7	0.38	6.4
Total	**7.03**	**100.0**	**5.99**	**100.0**

Source: Pollard (1996)

It can be seen from Table 3 that the sex differential in the expectation of life narrowed from 7.03 years to 5.99 years over the period 1982 to 1992. The contribution (in years) to the sex differential from IHD, respiratory disease, motor and non-motor accident and lung cancer all declined over the decade, indicating a relatively greater improvement in male mortality for these causes. By way of contrast, the contribution from other cancers and suicide increased, indicating more favourable experience for females.

It has been noted previously that the narrowing of the sex differential is a relatively recent phenomenon in Australia, and that the differential widened until the late 1970s or early 1980s. Table 4 shows the contribution of various causes of death to the change in the sex differential in Australia between 1911 and 1983.

Table 4: Changes in the sex differential in the expectation of life in Australia 1911-1983 by cause.

Sex differential in expectation of life in 1911 in years	**3.83**
Change in sex differential in years (% of total) 1911-1983 due to:	
Circulatory diseases (including IHD, CVD)	2.55 (86%)
Cancer	1.36 (46%)
Accident, suicide and violence	-0.32 (-11%)
Maternal mortality	0.58 (19%)
Infectious/parasitic diseases; pneumonia; bronchitis; influenza; diarrhoeal disease; certain diseases of infancy	-0.66 (-22%)
All other causes	-0.53 (-18%)
Total change in sex differential	**2.98 (100%)**
Sex differential in expectation of life in 1983 in years	**6.81**

Source: UN Secretariat (1988).

It can be seen that the main causes of the increasing differential in Australia over the period to 1983 have been the relatively greater female improvements for circulatory diseases and cancer, and declining maternal mortality. These sources of increase have been partly offset by relatively greater improvements for males for deaths due to accident, suicide and violence, infectious diseases and other causes.

From Table 3, it is evident that over three-quarters of the sex differential in the expectation of life at birth in 1992 was due to heavier male mortality for IHD, lung and related cancers, respiratory disease, accident, other cancers and suicide. The extent to which the mortality experience for each of these causes of death depends upon environmental versus biological factors will now be considered, the causes covered in order of importance as indicated by Table 3.

Section 5: ISCHAEMIC HEART DISEASE

Introduction

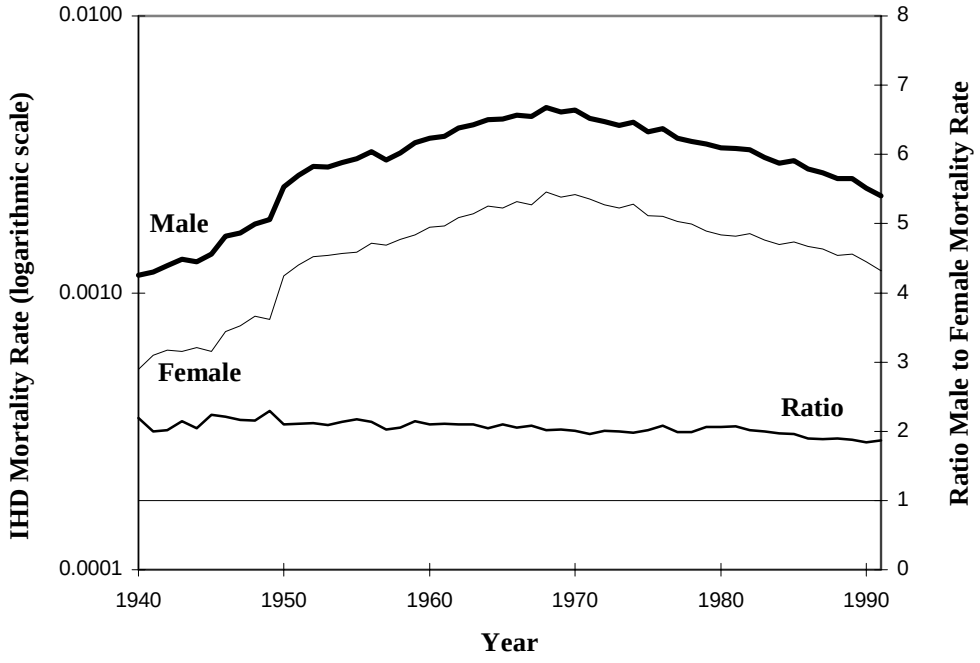
Ischaemic heart disease (previously called coronary heart disease) results from a blockage to coronary artery blood flow. An acute blockage results in a myocardial infarction or heart attack (d'Espaignet, 1994). Ischaemic heart disease (IHD) is the major single cause of death in Australia for both males and females, and accounted for 16,515 male deaths (24% of total male deaths) and 14,058 female deaths (24% of total female deaths) in Australia in 1994 (ABS, 1995b). As seen in Table 3, the higher rate of mortality for males due to IHD accounts for about 28% of the total sex differential in the expectation of life at birth. The ratio of male to female overall mortality due to this cause is about 1.9 (Table 2).

Australian Data and Trends

Historical Trends in IHD Mortality

Figure 5 shows age-standardised mortality rates due to ischaemic heart disease for Australian males and females for the period 1940-1991, as well as the ratio of the male to female rate.

Figure 5: Age-standardised mortality rates due to ischaemic heart disease for Australian males and females, and the ratio of the male to female rate, 1940-1991.



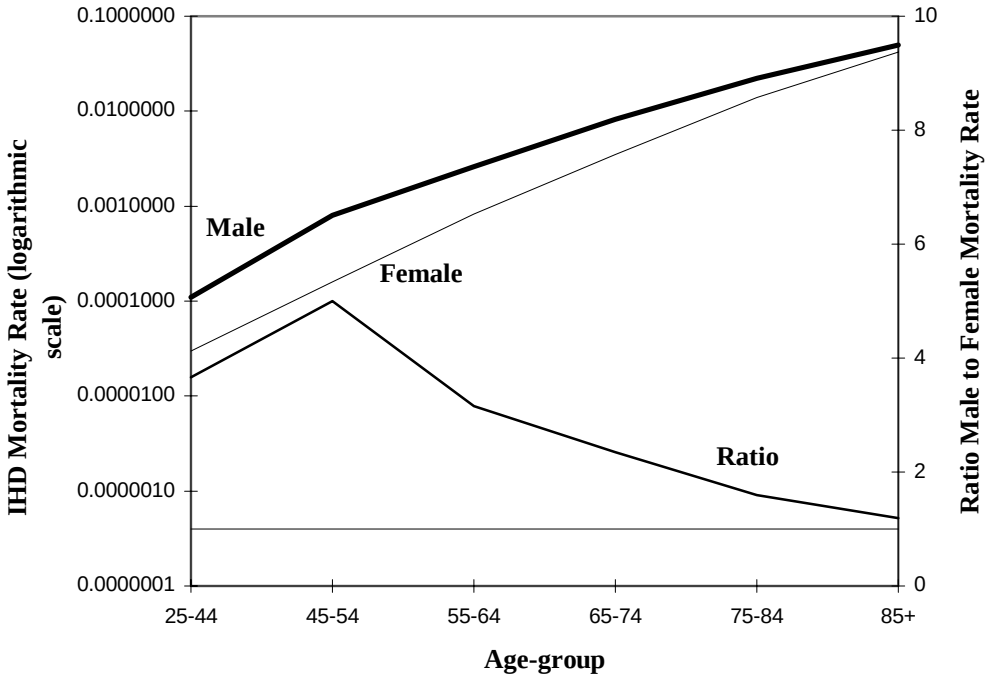
Source: d'Espaignet (1994) for all years (1988 population as standard)

The age-standardised mortality rates for males and females reached a peak in 1968, having increased between 1940 and 1968 at a rate of 5.1% per year for males, and 5.4% per year for females. The rates have consistently declined since 1968 at a rate of 3.1% and 2.8% per year for males and females respectively. A number of researchers have stated that the declining death rates are not an artefact of changes in classification but reflect true declines in rates (d'Espaignet, 1994; Dwyer and Hetzel, 1980; Dobson, Gibberd, Wheeler and Leeder, 1981). The ratio of male to female mortality has remained stable during the period, especially considering the significant changes in the mortality rates. The ratio decreased slightly from 2.2 in 1940 to 1.9 in 1991.

IHD Mortality by Age

Figure 6 shows IHD mortality rates by age for males and females for the year 1994, as well as the ratio of male to female mortality rates at each age.

Figure 6: Age-specific IHD mortality rates for Australian males and females by age group, and the ratio of the male to female rate, 1994.



Source: ABS (1995b)

It can be seen that the mortality rates increase rapidly with age. The ratio of male to female mortality is highest in the 45-54 age group. Although not shown here, a consideration of the age-specific rates of mortality over time reveals that the commencement of declining IHD mortality in 1968 occurred uniformly for most age groups for both males and females (Dobson et al., 1981). The fact that the decline occurred simultaneously for all generations (or cohorts) indicates that the reason for the improvement must have been a factor which affected the entire population at the same time; for example, a decline in risk factors which cause IHD (a decline in incidence), or improved medical management of the disease (a decline in mortality following incidence) (Dobson, et al., 1981). Evidence suggests that both these influences have been present, although the fall in incidence is thought to have been the more important factor (Waters and Bennett, 1995).

Data and Trends for Other Developed Countries

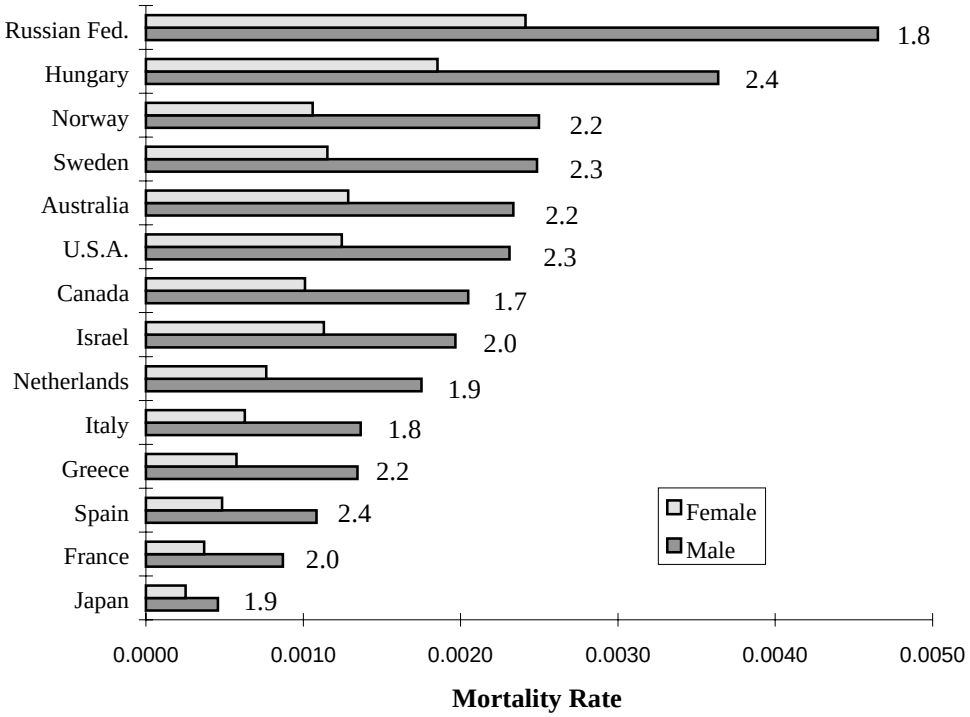
Historical Trends in IHD Mortality

Thom (1989) has analysed international trends in mortality due to IHD for the period 1950-1985. Most developed countries experienced increasing rates until the 1960s, followed by peaks during the 1960s or 1970s and then declining rates. Male IHD mortality rates began to decline in 1966 in Australia, 1968 in the USA (Dwyer & Hetzel, 1980), the late 1960s in New Zealand, Finland and Portugal, the early to mid 1970s in Israel, Belgium, the Netherlands, Scotland, Northern Ireland and Norway, and the late 1970s in Switzerland, Denmark, Austria, England and Wales, France, Italy, Sweden and Spain (Thom, 1989). In contrast to this pattern, IHD death rates have risen significantly in most of the Eastern European countries (Lopez, 1990).

International Comparison of IHD Mortality

Figure 7 shows IHD mortality rates for males and females for a number of developed countries in 1988, as well as the sex ratio.

Figure 7: Age-standardised male and female mortality rates for IHD for various developed countries, and the ratio of the male to female rate, 1991-1993.



Source: WHO (1995) - standard population is European standard population

It can be seen that the rates of IHD mortality for different countries of the world vary markedly, although caution should be exercised in interpreting these rates due to possible differences in diagnosis and/or coding of IHD and other heart diseases (Lopez, 1990). The ratio of IHD mortality rates in the highest IHD mortality country to those in the lowest IHD mortality country is 10.2 for males and 9.5 for females (Russian Federation having the highest and Japan the lowest IHD mortality in both cases). Note that the rate of mortality for Japanese males is lower than the female mortality rates for the majority of countries, emphasising the importance of factors other than gender for this cause of death.

International Comparison of the Sex Differential in IHD Mortality

Figure 7 shows the ratio of male to female IHD mortality for a number of developed countries. The ratios vary from 1.7 for Canada to 2.4 for Hungary and Spain, and do not appear to vary in any uniform way with the absolute level of IHD mortality rates. These results are consistent with those reported by Kalin and Zumoff (1990) who found that sex mortality ratios for IHD were constant and close to 2 for 52 countries with a very wide range of IHD prevalence (based on 1987 WHO data). Other researchers have reported a ratio of around 2 for the whole age range (Waldron, 1976), and higher figures for restricted sections of the age range (Koskenvuo, Kaprio, Lönnqvist & Sarna, 1986; Thom, 1989).

The Contribution of Genetic and Biological Factors

The fact that IHD sex mortality ratios have been close to 2 over a number of years in Australia and for a number of developed countries around that world suggests that innate gender differences contribute to IHD mortality. In addition, Wingard, Suarez and Barrett-Connor (1983) have found that the sex difference is reduced but not eliminated after controlling for various IHD risk factors.

Most research on genetic factors has focussed on the role of male and female sex hormones, and there is disagreement among researchers as to the effects of hormones on IHD risk (Waldron, 1983). An extensive review of the clinical studies has been carried out by Kalin and Zumoff (1990) who believe that poorly constructed studies and other experimental weaknesses have contributed to the apparent disparate findings. After critically reviewing studies of hormone levels in persons with IHD, as well as studies of IHD risk in groups with different sex hormone patterns, the authors conclude that no final answers have been reached, but they propose the following hypotheses to account for the research findings. Firstly, endogenous female sex hormones (estrogens and progesterone) decrease the risk of IHD. Secondly, and in contrast, exogenous estrogens and progesterone appear to increase the risk of developing IHD. Thirdly, male androgens favour the development of IHD. These findings are broadly consistent with the conclusion reached by Waldron (1983) that endogenous female hormones appear to decrease the risk (however she notes that these findings are controversial) and that exogenous female hormones do not decrease the risk.

It has also been suggested that sex differences in the distribution of body fat may contribute to the risk of IHD. A high waist-to-hip ratio is more common among men and has been associated with risk of IHD (Wingard, 1990).

The Contribution of Environmental and Lifestyle Factors

The fact that IHD mortality rates have varied markedly over time in Australia (Figure 5) and vary widely between countries (Figure 7) suggests that environmental factors play some part in the development of the illness.

The Role of Diet

Many studies have considered the role of diet, and particularly the level of dietary fat, in the development of IHD. The impact of diet may be assessed by several types of study; firstly by contrasting IHD mortality and diet for different countries or regions, secondly by contrasting IHD mortality and diet for individuals or different sub-groups of a population, thirdly by looking at the effects of dietary intervention on IHD rates, fourthly by correlating trends in IHD mortality over time with trends in diet, and finally by comparing IHD mortality for migrants against the rates in the old and new countries. The evidence for the impact of diet on IHD mortality for each of these types of study will now be presented.

A comparison of IHD mortality rates by country reveals very low rates in Japan and the countries of Southern Europe (Figure 7) where the diet has traditionally been low in fat (Lopez, 1990). A study in Belgium found that IHD mortality rates were higher in the southern regions where butter consumption was higher and soft margarine consumption lower, although differences in smoking habits and personality traits were also found (Kornitzer, De Backer, Dramaix and Thilly, 1979).

Studies comparing IHD incidence or mortality for different sub-groups of a population have also found correlations with diet. It has been shown that IHD hospital admission rates for US male Seventh Day Adventists (SDAs), who generally follow a vegetarian lifestyle and do not use tobacco, alcohol or caffeine, are only about 60% of those for the general white US male population. In addition, the sex difference in the rates of IHD incidence normally observed in the general population was not evident for the SDAs (Wynder, Lemon & Brass, 1959, cited in Walden, Schaefer, Lemon, Sunshine & Wynder, 1964). It has also been found that the rate of IHD mortality for males is significantly inversely related to the degree of adherence to the SDA lifestyle, even when non-smoking SDAs and non-SDAs are compared (Phillips, Kuzma, Beeson & Lotz, 1980). A study of English men found significantly lower incidence of IHD associated with a high intake of cereal dietary fibre (Morris, Marr and Clayton, 1977) which supports research showing that worsening relative IHD mortality among the working class in England between 1931 and 1971 has been associated with higher consumption of sugar, increased smoking and lower consumption of wholemeal bread in this class (Marmot, Adelstein, Robinson & Rose, 1978).

The effects of dietary intervention on IHD mortality have also provided support for the role of diet. A twelve year study involving the introduction of a cholesterol-lowering diet into two hospitals found a significant reduction in IHD mortality to less than half the control levels for men, and reductions in IHD mortality for women although these were not statistically significant (Miettinen, Turpeinen, Karvonen, Elosuo & Paavilainen, 1972). Studies have also shown that reduced fat diets can lead to lower serum total cholesterol levels in the blood (Walden et al., 1964); elevated serum total cholesterol level is a risk factor for IHD (Waters and Bennett, 1995).

Many studies have attempted to correlate trends in IHD mortality over time with changes in diet. Epstein (1989) compared trends in rates of mortality due to IHD and trends in nutrition, smoking and alcohol consumption for 27 countries over a 10-25 year period prior to 1985. He found that countries experiencing increasing IHD mortality had also experienced increases in animal fat consumption, and that declining IHD mortality was generally associated with reductions in animal fat intake. Differences in rates of change in IHD mortality between countries could not be accounted for by changes in alcohol or smoking habits. Heller, Hayward and Hobbs (1983) also found that declines in IHD mortality in the United Kingdom were preceded by decreased total fat consumption about five years earlier.

Migrants provide valuable information with which to assess the influence of environmental factors on IHD mortality. Stenhouse and McCall (1970) have studied death rates due to IHD during 1962-66 for migrants to Australia from England and Wales, Scotland and Italy compared with rates in the Australian-born population. They found that death rates due to arteriosclerotic and degenerative heart diseases (including IHD) for migrants from England and Wales and Italy (where mortality rates are lower than in Australia) were lowest among those resident in Australia for short periods of time, and generally increased as length of residence increased until significantly higher than the rates in their country of origin. Although this study does not specifically consider diet, it indicates the important role of environmental and cultural factors as a whole in the development of IHD.

In summary, various types of studies have provided support for the hypothesis that diet, and particularly the level of fat in the diet (or the relative consumption of animal versus vegetable fats), is related to IHD mortality. Correlations between dietary fat and blood cholesterol have also been found. In Australia, the proportion of people aged 25 to 64 years with high blood cholesterol is higher in men (17.6%) than women (15.3%) after controlling for age (Mathers, 1994, cited in Waters and Bennett, 1995).

The Role of Smoking

Cigarette smoking is one of the major, modifiable causes of IHD (US DHHS, 1989). Declines in IHD mortality in the USA have been greater for non-smokers than for current smokers (US DHHS, 1989).

A prospective study over the forty years to 1991 involving over 30,000 male British doctors found that IHD mortality rates increased according to amount smoked, and that this relationship was very highly statistically significant. The IHD mortality rate for heavy smokers (≥ 25 cigarettes per day) was roughly double that for non-smokers (Doll, Peto, Wheatley, Gray & Sutherland, 1994a). The relationship between smoking and IHD mortality was strongest at the younger ages - the relative IHD mortality risk for heavy smokers compared with non-smokers for the first twenty years of follow-up was 15:1 at ages under 45 years, 3:1 at ages 45-54 years, and 2:1 at ages 55-64 years (Doll & Peto, 1976).

The same researchers also studied female British doctors over a twenty-year period, and found that the IHD mortality rate for heavy smokers (≥ 25 cigarettes per day) was again roughly double that for non-smokers (Doll, Gray, Hafner & Peto, 1980). The relative excess risk was thus the same as in the male study, however the absolute risk and hence the absolute excess risk was lower than for men, possibly due to the fact that women generally smoke fewer cigarettes, inhale less deeply and on average smoke lower tar cigarettes (Ford, 1994). A prospective study over six years of almost 120,000 female nurses in the USA found a relative risk for fatal IHD of 5.5 for heavy smokers (≥ 25 cigarettes per day) compared with non-smokers (Willett et al., 1987), underlining the importance of smoking as a risk factor for IHD in women.

Studies of correlations between IHD mortality rates and smoking patterns over time have found trends which are consistent with the above findings. Worsening relative IHD mortality among the working-class in England and Wales has been found to be correlated with relatively more smoking in this class (Marmot et al., 1978). Research on correlations between smoking and IHD mortality would seem to suggest that the change to low tar cigarettes has little or no influence on the risk of IHD (US DHHS, 1989), consistent with the hypothesis that it is the gaseous components of tobacco smoke which contribute to IHD (Wald, 1976).

In summary, there is no doubt that smoking causes IHD; the risk for smokers is about double that for non-smokers. In Australia and overseas, smoking prevalence is, and has been in the past, higher for males than for females (see section 6), so higher levels of smoking among males has contributed to the sex differential in mortality due to IHD.

The Role of Alcohol

There is now substantial evidence that low to moderate alcohol consumption is associated with decreased risk of IHD (Waters and Bennett, 1995). A study of roughly 12,000 male British doctors over the thirteen years to 1991 found that IHD mortality rates were significantly lower in regular moderate drinkers compared to non-drinkers after standardising for age, year and smoking habits (Doll, Peto, Hall, Wheatley & Gray, 1994b). A study of US men over the seventeen years to 1975 found that mortality rates from IHD decreased as alcohol intake increased up to a level of five drinks per day (Dyer, Stamler, Paul, Lepper, Shekelle, McKean & Garside, 1980).

Other Factors

Physical fitness has been found to be a protective factor for IHD (Waters and Bennett, 1995); in a study of British men over a decade, Morris, Marr and Clayton (1977) found a lower rate of occurrence of IHD among men with a high energy intake (associated with greater physical activity). Some researchers have suggested that "Coronary Prone Behaviour Pattern" or Type A behaviour is associated with increased risk of IHD and can account for part of the sex differential in mortality (Waldron, 1976), although Nathanson (1984) points out that more research is needed to provide sufficient evidence for this hypothesis. It has also been suggested that occupation and socioeconomic status influence in the risk of IHD - studies have found that IHD mortality rates are declining more quickly for the professional occupations in both Australia (Dobson, Gibberd, Leeder & O'Connell, 1985) and England and Wales (Marmot et al., 1978). However, Dobson et al. (1985) suggest that the lower mortality among the higher status groups is due to a pattern of behaviour incorporating several factors associated with low IHD mortality, including diet, alcohol use, exercise and smoking. There is clear evidence that use of oral contraceptives in women is a risk factor for IHD (US DHHS, 1989), and that this interacts synergistically with other risk factors, including smoking (Mann, Vessey, Thorogood & Doll, 1975).

Conclusion

Smoking has been shown to be one risk factor for IHD, with smokers experiencing roughly double the IHD mortality rate of non-smokers. The higher prevalence of smoking among males has thus contributed to the sex differential in IHD mortality. However, other risk factors are also important; a comparison of male and female British doctors found that the ratio of male non-smoker to female non-smoker IHD mortality rates was 5.3 for ages less than 65 years and 2.9 for ages 65 and over (Doll et al., 1980).

Various studies outlined previously have indicated that in addition to smoking, diet, physical exercise, alcohol consumption and use of oral contraceptives play a role. The sex difference in IHD mortality is consistent with gender differences in risk factor prevalence. As stated previously, Australian men are more likely than women to have higher cholesterol levels and to smoke. The proportion of Australian men and women aged 25 to 64 undertaking physical exercise appears to be similar after adjusting for age (Mathers, 1994, cited in Waters and Bennett, 1995). A study has found that the proportion of men with multiple risk factors for IHD (high blood pressure, high cholesterol and smoking) is higher than that for women (National Heart Foundation of Australia and Australian Institute of Health, cited in Waters and Bennett, 1995).

Wingard, Suarez and Barrett-Connor (1983) found that the sex ratio for IHD mortality over the age range 30-69 reduced from 4.8 to 2.4 when a number of demographic, behavioural and biologic risk factors were controlled for (including age, marital status, education, smoking, cholesterol, blood pressure, plasma glucose and obesity). The observed reduction indicates that these factors contribute to the sex differential in IHD mortality. However, the fact that a substantial sex difference remains suggests factors other than those controlled for are also important. There is evidence that sex hormones influence IHD risk, and it is possible that this biological factor may account for part of the remaining gender difference. The fact that the sex ratio for IHD mortality is similar for different countries and has been very stable over time in Australia is consistent with the presence of some biological contributing factor.

It is difficult to assess the relative importance of genetic versus environmental factors. Moriyama (1983) states that "there appears to be general agreement that the improvement in ischaemic heart disease mortality can largely be attributed to significant decreases in cigarette smoking and the decline in consumption of saturated fat and cholesterol" (pp. 288-290). Anderson and Halliday (1979) also argue that the great increase in IHD mortality since 1921 has occurred so rapidly that it can only be explained by environmental and not genetic factors.

Section 6: LUNG AND RELATED CANCERS

Introduction

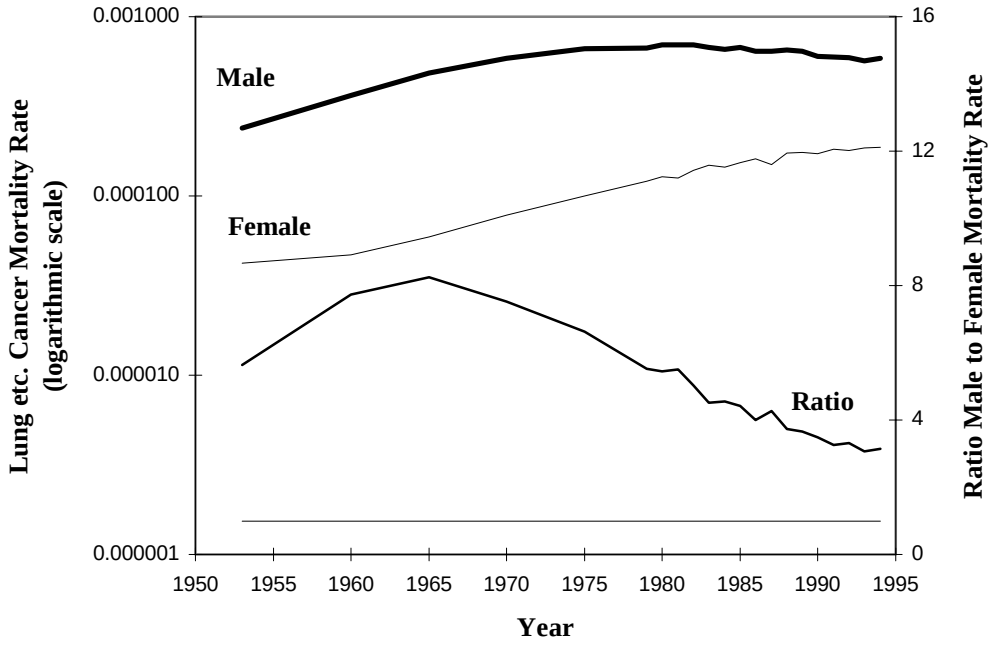
Cancers of the respiratory organs (including lung, bronchus, trachea, larynx) and lip, oral cavity and pharynx, accounted for 5,704 male deaths (8.5% of total male deaths) and 2,159 female deaths (3.6% of total female deaths) in Australia in 1994 (ABS, 1995b). As seen in Table 3, the higher rates of mortality for males due to lung and related cancers accounts for about 13.5% of the total sex differential in the expectation of life at birth. The ratio of male to female overall mortality due to this cause is about 3.5 (Table 2).

Australian Data and Trends

Historical Trends in Lung and Related Cancer Mortality

Figure 8 shows age-standardised mortality rates due to cancer of the lung, bronchus and trachea for Australian males and females for the period 1950-1994, and the ratio of the male to female rate. These cancers form the bulk of those mentioned above.

Figure 8: Age-standardised mortality rates due to cancer of the lung, bronchus and trachea for Australian males and females, and the ratio of the male to female rate, 1950-1994.



Note: **Rates are plotted for 1953 (representing the period 1950-57), 1960 (1958-62), 1965 (1963-67), 1970 (1968-72), 1975 (1973-77) and for individual years from 1979-94.**

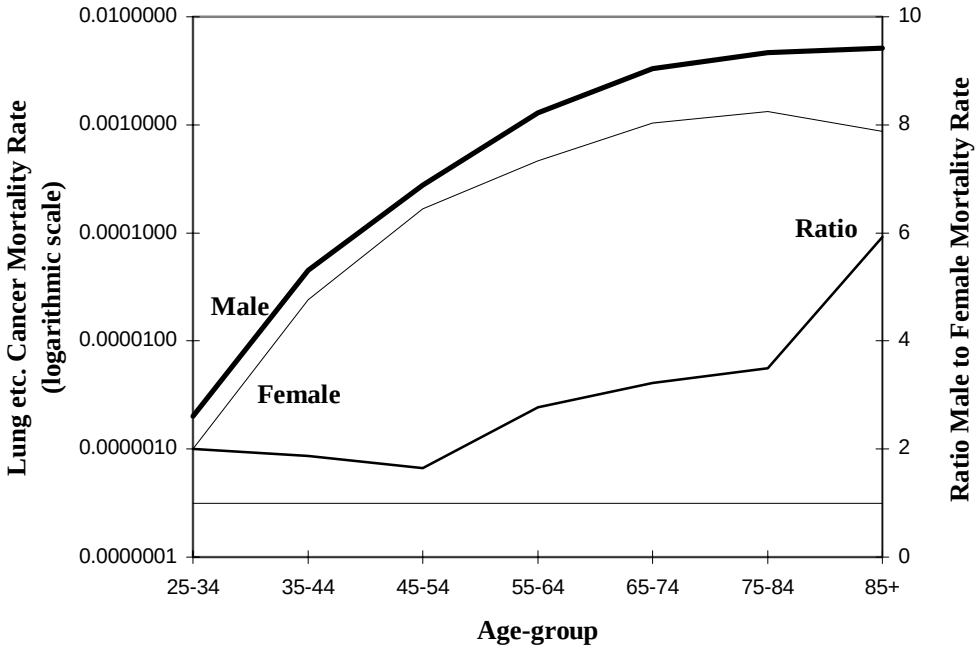
Source: **Rohan, T & Christie, D. (1980) for years 1950-1977 age-specific rates - standardised using Australian mid-year 1991 population from ABS (1992). ABS (1996) for 1979-1994 - standard population is Australian mid-year 1991 population.**

The age-standardised mortality rates for males reached a peak in the early 1980s and have been falling ever since. In contrast to the male rates, female rates have been increasing throughout the entire period; and increasing at a faster rate than males since the mid to late 1960s. The ratio of male to female mortality has varied enormously over the period, from a starting value of 5.6 to a peak of 8.2 for the period 1963-67 to a value of 3.1 in 1994. The pattern is very different to that for IHD where the ratio has been very stable over time, despite marked changes in the underlying mortality rates.

Lung and Related Cancer Mortality by Age

Figure 9 shows lung cancer mortality rates by age for males and females for the year 1994, as well as the ratio of male to female mortality rates at each age.

Figure 9: Age-specific lung, bronchus and trachea cancer mortality rates for Australian males and females by age group, and the ratio of the male to female rate, 1994.



Source: ABS (1996)

Mortality rates for lung and related cancers increase dramatically with age, and are very low at the younger ages. The ratio of male to female mortality tends to increase with age from about age 50, possibly due to the cumulative effects of heavier male smoking.

Data and Trends for Other Developed Countries

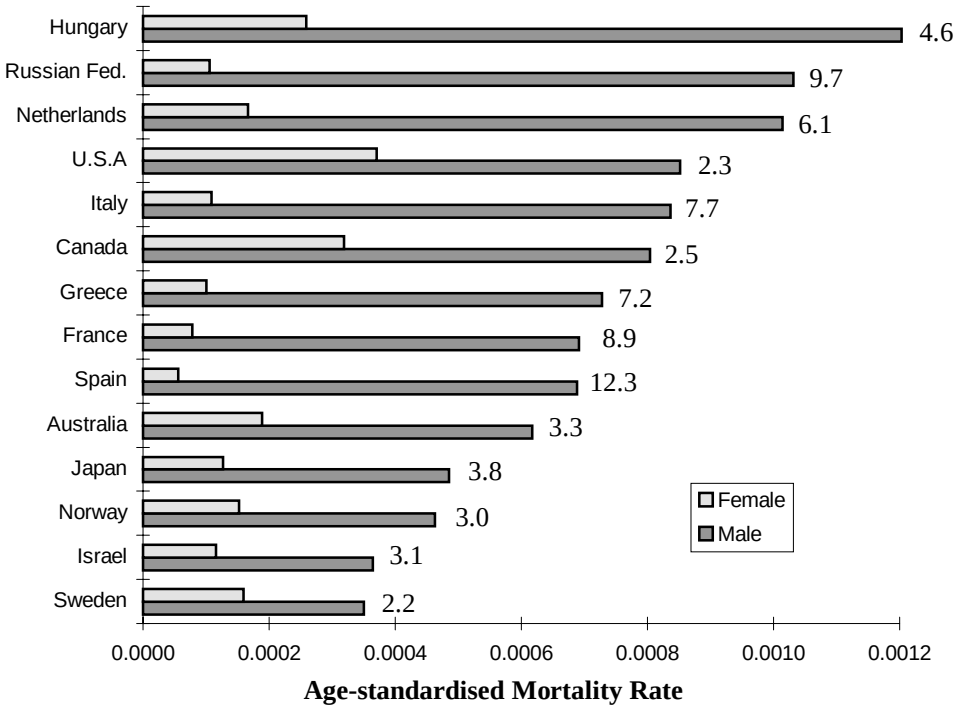
Historical Trends in Lung and Related Cancer Mortality

Lopez (1990) has included lung cancer in his analysis of international mortality trends in developed countries. Lung cancer mortality rates for males in Finland and the UK peaked in the 1970s and have since been falling at all ages; for many other countries including Australia, Belgium, Canada, Czechoslovakia, Germany, the Netherlands, New Zealand, Sweden, Switzerland, and the USA, the increase in male lung cancer mortality rates had slowed at the time of writing, and were beginning to fall at the younger ages in some countries. For males, rates peaked at a level on average 165% higher than those in 1950. Among females lung cancer mortality rates in developed countries are rising, and rates in the 1980s are on average 200% higher than they were in 1950. Lopez states that there is no evidence of a slowing in the rates of increase of lung cancer mortality in women.

International Comparison of Lung and Related Cancer Mortality

Figure 10 shows lung cancer mortality rates for males and females for a number of developed countries in 1991-93, as well as the sex ratio.

Figure 10: Age-standardised male and female mortality rates for cancer of the trachea, bronchus and lung for various developed countries, and the ratio of the male to female rate, 1991-93.



Source: WHO (1995) - standard population is European standard population

Male rates of lung and related cancer mortality for the developed countries in Figure 10 vary from 120.3 per 100,000 (Hungary) to 35.0 per 100,000 (Sweden), a ratio of 3.4. Female rates of lung and related cancer mortality vary from 37.1 per 100,000 (USA) to 5.6 per 100,000 (Spain), a ratio of 6.6.

International Comparison of the Sex Differential in Lung and Related Cancer Mortality

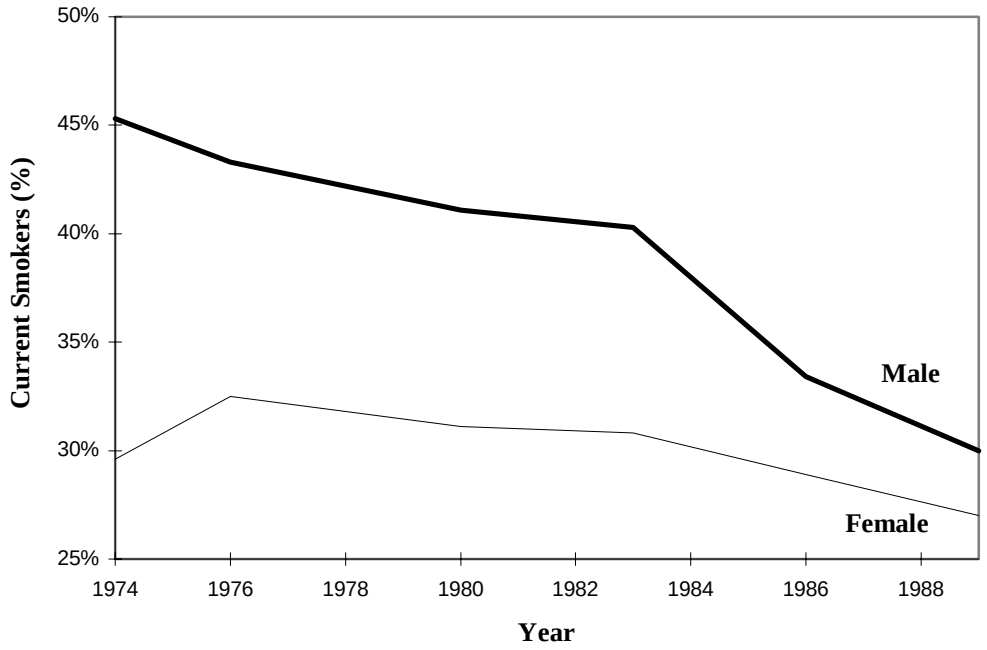
Figure 10 shows the ratio of male to female lung and related cancer mortality for a number of developed countries. The ratios vary from 2.2 in Sweden to 12.3 in Spain, and do not appear to vary in any uniform way with the absolute level of mortality.

Smoking Prevalence and Trends

In subsequent sections, the relationship between lung cancer mortality and smoking is examined, so it is worthwhile to review trends in smoking prevalence in Australia and overseas.

Smoking Trends in Australia

Figure 11: Smoking prevalence rates for Australian males and females, 1974-1989.



Source: Hill, White and Gray (1989) cited in Waters and Bennett (1995). Rates have been age-standardised to the 1986 sample.

Prevalence of smoking has reduced substantially over the period 1974-1989, particularly during the period 1983-1986 coinciding with anti-smoking campaigns (Hill, White & Gray, 1988). Male prevalence is falling at a faster rate than female prevalence, resulting in a narrowing sex difference. Females are taking up smoking at a greater rate than previously (in 1989 smoking prevalence for 18-24 year olds was 36% for both males and females; ABS, 1992b) and they state weaker intentions of giving up smoking (Hill & Gray, 1984). However, females smoke fewer cigarettes on average, as shown in Table 5, and a higher proportion of females smoke low tar cigarettes (ABS, 1992b).

Table 5: Smoking statistics in Australia for males and females, 1989

	Males	**Females**
Number of cigarettes smoked per day		
No more than ten	22.5 %	33.4 %
Eleven to twenty	35.9 %	38.2 %
More than twenty	41.6 %	28.4 %

Source: ABS (1992b)

Smoking Trends in Other Developed Countries

In general, smoking has been more prevalent among males than females in a variety of regions, including Europe, the USA, Australia, Latin America, and South Asia (Waldron, 1985). Pierce (1989) reviewed smoking trends in a number of developed countries between 1974 and 1987 and found that male cigarette smoking prevalence had declined for all countries, and that female prevalence had declined for all countries except Norway. With the exception of Sweden, in all countries considered males smoked more than females over the period, but this gap narrowed over time. In Sweden male and female prevalence rates between 1974 and 1987 were similar - in light of this fact it is interesting to note that Sweden has the lowest sex ratio for lung cancer of all countries considered in Figure 10.

Historical data on smoking prevalence shows that for UK males the average cigarette consumption per person per day reached two in the first decade of this century, increased to a peak of about twelve in the mid to late 1940s, averaged around ten for the next thirty years, and since around 1975 has decreased to reach about six in 1985. In contrast, women started smoking later, reaching two cigarettes per day in about 1940, consumption increased to a peak of about seven in 1975 and since that time has declined slightly to around five in 1985 (Ford, 1994). Hence, males commenced smoking about 30 years before females, and male cigarette consumption also levelled-off before female consumption.

Filter cigarettes became popular in the 1950s and by 1970 accounted for 80% of sales in the USA and UK (Ford, 1994). By estimating previous smoking prevalence in the USA, Harris (1983) suggests that the peak exposure to cigarette smoking for men occurred for the cohorts born around 1903-1923, but for women for the cohorts born around 1923-1943. These results are broadly similar to those based on data from England and Wales (Lee, Fry & Forey, 1990).

The Contribution of Genetic and Biological Factors

Evidence has been presented which suggests that genetic factors play a less important role than environmental factors for mortality due to all types of cancer. A prospective study of 960 children born between 1924 and 1926 and placed with unrelated adoptive parents found that the death of an adoptive parent from any type of cancer before the age of 50 increased the rate of mortality from cancer fivefold among the adoptees, a statistically significant result. In contrast, the death of a biological parent had no significant effect on the risk (Sorenson et al., 1988).

There is irrefutable evidence that smoking causes lung cancer, some of which is reviewed in the following sections. On the basis of some studies which have shown a higher excess risk of lung cancer for smokers among males than among females, some researchers have hypothesised that there may be a gender difference in susceptibility to lung cancer from smoking. However, at least part of the difference in absolute excess risk is due to the fact that women start smoking later, inhale less and smoke less of each cigarette (Waldron, 1976). In a study of lung cancer mortality and cigarette consumption trends, Burbank (1972) concluded that gender differences in lung cancer mortality could be explained by past cigarette usage. It has also been suggested that biological or genetic sex differences in the propensity to take up or to quit smoking may exist, but there is no evidence that physiological factors have made anything more than a minor contribution to sex differences in smoking adoption or cessation (Waldron, 1991). The fact that the proportions of Australian 18-24 year olds who smoke is equal for males and females (ABS, 1992b) would also seem to argue against this hypothesis.

The Contribution of Environmental and Lifestyle Factors

The Role of Smoking

In Australia, lung cancer is the most common form of cancer for males, and one of the most common causes of cancer deaths for females. The extent to which mortality due to lung cancer has risen this century is indicated by the fact that in 1912 authorities on lung cancer agreed that it was “among the rarest forms of disease” (Ford, 1994, pg. 30). In a review of causes of death in developed countries, Lopez (1990) states that “unquestionably, the most significant feature of postwar mortality in the developed

countries has been the extraordinary and sustained rise in lung cancer mortality, the vast majority of which can be attributed to smoking" (pg. 108). Research into the effects of cigarette smoking on lung cancer has included large prospective studies and attempts to correlate lung cancer mortality rates and smoking prevalence trends.

Evidence for the fact that cigarette smoking causes lung cancer was presented as early as 1950 (Doll & Hill, 1950) and large prospective studies were set up at this time to assess the risk. A forty year study to 1991 of over 30,000 male British doctors found that mortality rates due to cancer of the lung for heavy smokers (≥ 25 cigarettes per day) were about 25 times the rates for non-smokers, a very highly statistically significant result. The ratio of heavy smoker to non-smoker mortality exceeded ten for cancers of the oesophagus and upper respiratory sites (Doll et al., 1994a). Similar results were found for the American Cancer Society Cancer Prevention million person prospective study, in which lung cancer mortality rates for current smokers were about 25 times those for non-smokers (US DHHS, 1989, cited in Peto, 1994). A prospective study of female British doctors over the 22 years to 1973 found that lung cancer rates for heavy smokers were 30 times those for non-smokers; the absolute excess risks were lower than for males which could be due to the fact that women commence smoking later than men and inhale less often (Doll et al., 1980).

A comparison of the male and female prospective studies revealed a ratio of non-smoker male to non-smoker female age-standardised mortality rates for diseases "closely related to smoking" (including cancer of the lung, oesophagus and upper respiratory sites as well as other respiratory diseases) of only 1.3 (Doll et al., 1980), which is close to 1 considering the small numbers of deaths on which these rates are based (17 male non-smoker deaths and 14 female non-smoker deaths). Hence, apart from the risk due to smoking, male and female lung and related cancer mortality rates are similar in these studies.

Attempts to correlate lung cancer mortality and trends in smoking prevalence have also supported the strong link between lung cancer mortality and cigarette smoking. Burbank (1972) studied lung cancer mortality rates between 1950 and 1968 and tobacco consumption and smoking prevalence between 1920 and 1968 in the USA and found that lung cancer mortality was a simple function of past cigarette use with a lag period of about thirty years. Wald (1976) found that lung cancer mortality trends for men and women in the UK between 1956 and 1973 were consistent with trends in cigarette consumption and the change to reduced tar cigarettes. Cross-country comparisons provide further support for the link between smoking and lung cancer. Since 1976, male smoking prevalence in Sweden has been low (Pierce, 1989) which is consistent with low rates of lung cancer mortality shown in Figure 10. Lopez (1990) points out that female lung cancer mortality is high in countries where females began smoking earlier and where prevalence of female smoking is high (UK, USA, Australia and Denmark). In addition, the continuing increase in lung cancer mortality rates in women in many countries of the world is thought to be primarily due to increased cigarette consumption among women (Lopez, 1990).

Other Factors

In addition to smoking, occupational hazards can contribute to the development of lung cancer. Exposure to asbestos and radon has been shown to influence the risk of lung cancer, although it seems that most deaths occur among smokers and hence that the risk factors have a multiplicative effect (Ford, 1994). It has been estimated that roughly 10% of male lung cancer deaths are due to asbestos (Waldron, 1982).

Passive smoking is also associated with increased risk of certain diseases, including lung cancer and respiratory infections (US DHHS, 1989) and in children, asthma, reduced lung growth, SIDS, bronchitis and pneumonia (Ford, 1994).

Conclusion

All the evidence suggests that the majority of lung cancer deaths are due to cigarette smoking. According to the American Cancer Society study, about 90% of male and 80% of female lung cancer deaths are due to cigarette smoking (US DHHS, 1989, cited in Ford, 1994) and still others are due to passive smoking, occupational hazards and pipe and cigar smoking (Ford, 1994). Past differences in smoking prevalence and practices between men and women would be expected to account for a large part of the sex difference in lung cancer rates, and increased male exposure to occupational hazards to at least part of the remaining difference. That smoking contributes to the sex differential in lung cancer mortality has been supported by a study of 23 developed countries which found that sex differentials in lung cancer mortality decreased between 1979 and 1987, in line with decreasing sex differences in cigarette smoking in previous years (Waldron, 1993). Whether these differences in smoking prevalence have any physiological basis is not completely clear; however it would seem that innate genetic or biological factors do not play a major role.

Section 7: RESPIRATORY DISEASES

Introduction

Respiratory diseases include chronic obstructive pulmonary disease (COPD), as well as pneumonia, influenza, and other respiratory illnesses. COPD includes asthma, emphysema, chronic bronchitis, and chronic airways obstruction, and accounts for roughly 60%-70% of the total age-standardised death rate due to all respiratory diseases. Diseases such as emphysema cause prolonged disability but not death, and so mortality rates may understate the prevalence of the disease (Ford, 1994).

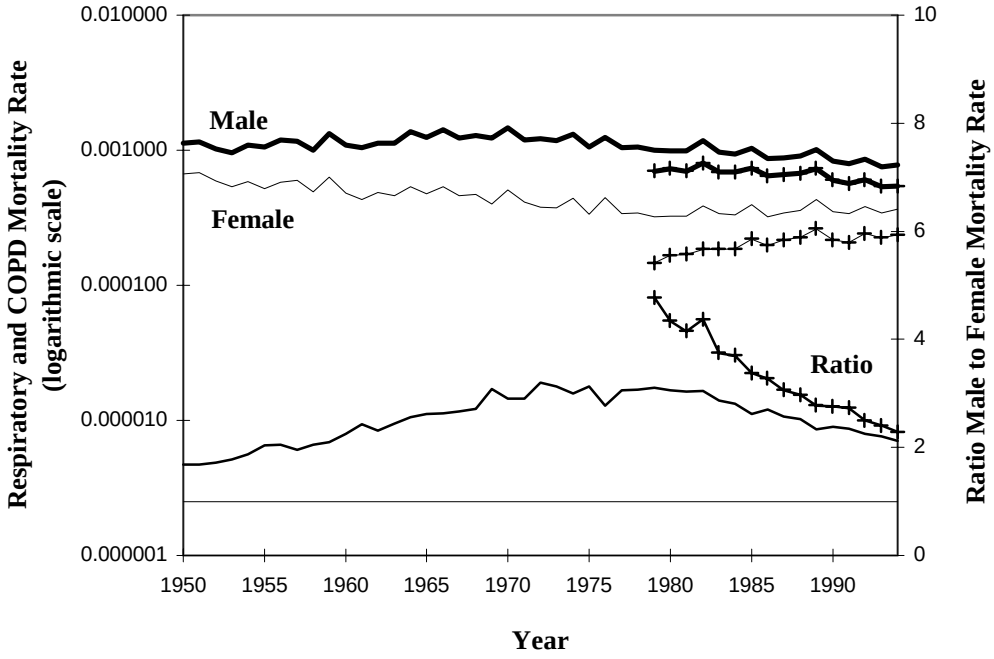
As seen in Table 3, the higher male mortality rates for respiratory illnesses as a whole account for about 9.9% of the total sex differential in the expectation of life at birth. The ratio of male to female overall mortality due to this cause is about 2.3 (Table 2).

Australian Data and Trends

Historical Trends in Respiratory Disease Mortality

Figure 12 shows age-standardised mortality rates due to the entire class of respiratory diseases for 1950-1994, and for COPD deaths for 1979-1994, as well as the ratio of the male to female rates.

Figure 12: Age-standardised mortality rates due to respiratory diseases, 1950-1994 and COPD 1979-1994 for Australian males and females, and the ratio of the male to female rate.



Note: **COPD rates and ratios are denoted by the crossed lines**
Total respiratory disease mortality is denoted by the plain lines.

Source: **d'Espaignet et al. (1991) for respiratory diseases 1950-1978 - standard population is Australian mid-year 1988 population.**
ABS (1996) for respiratory diseases 1979-1994 and COPD 1979-1994 - standard population is Australian mid-year 1991 population.

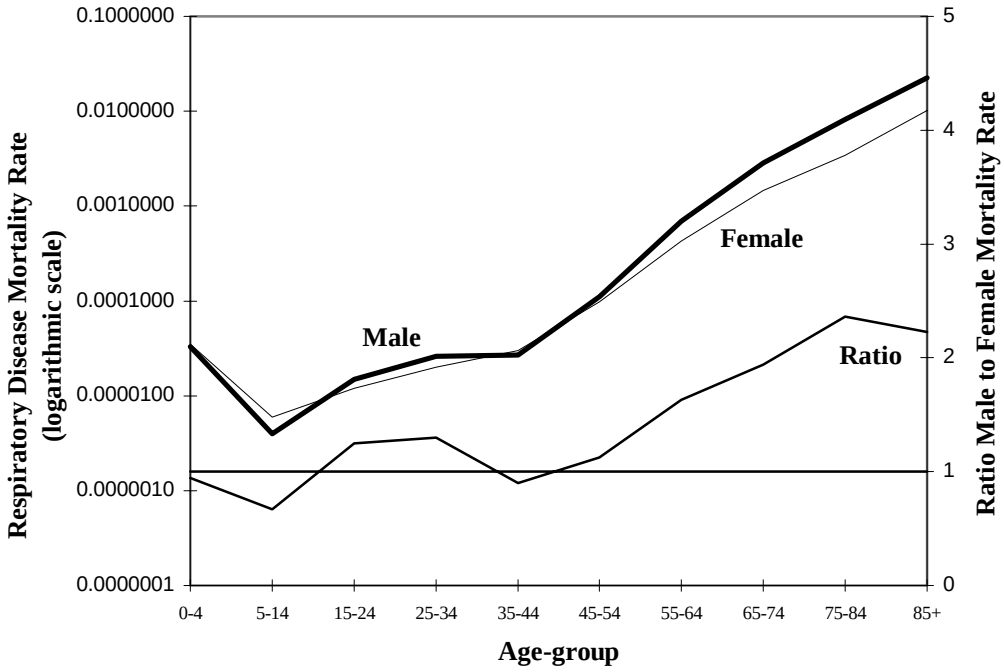
For the entire class of respiratory illnesses, male rates increased very slightly over the period 1950 to the early 1970s and since that time have declined (the rate in 1950 was 113.1 per 100,000 compared with 78.0 per 100,000 in 1994). Female rates declined between 1950 and around 1980, and since that time have increased slightly (the rate in 1950 was 67.0 per 100,000 compared with 36.8 per 100,000 in 1994). The ratio of male to female mortality rose from 1.7 in 1950 to a peak of 3.2 in 1972, and then declined to reach 2.1 in 1994. Note that the small year-to-year peaks for respiratory disease mortality often coincide for males and females - these peaks are due to new strains of the influenza virus (ABS, 1996).

For COPD deaths the age-standardised mortality rates for males have generally been falling over the 15 year period (69.7 per 100,000 in 1979 to 54.4 per 100,000 in 1994, a fall of 1.6% pa.), whereas the rates have been rising for females quite markedly (14.6 per 100,000 in 1979 to 23.8 per 100,000 in 1994, a 3.3% pa. increase). The ratio of male to female mortality has thus declined rapidly over the whole period, from a value of 4.8 in 1979 to 2.3 in 1994, reflecting worsening experience for females relative to males.

Respiratory Disease Mortality by Age

Figure 13 shows respiratory disease mortality rates by age for males and females for the year 1994, as well as the ratio of male to female mortality rates at each age.

Figure 13: Age-specific respiratory disease mortality rates for Australian males and females by age group, and the ratio of the male to female rate, 1994.



Source: ABS (1996)

Respiratory disease mortality rates increase dramatically with age especially from the 35-44 year age group onwards. Male and female rates are similar up to the 45-54 year age group, but male mortality exceeds female mortality after this age. The ratio of male to female mortality increases over the lifespan to a maximum of about 2.2 for the 85 and over age group. Although not shown here, the ratio for COPD also increases rapidly over age, reaching a maximum of 3.4 for the 85 and over age group. This pattern is very similar to that for the sex ratio by age for lung cancer, and could again be due to the cumulative effects of heavier male smoking.

Data and Trends for Other Developed Countries

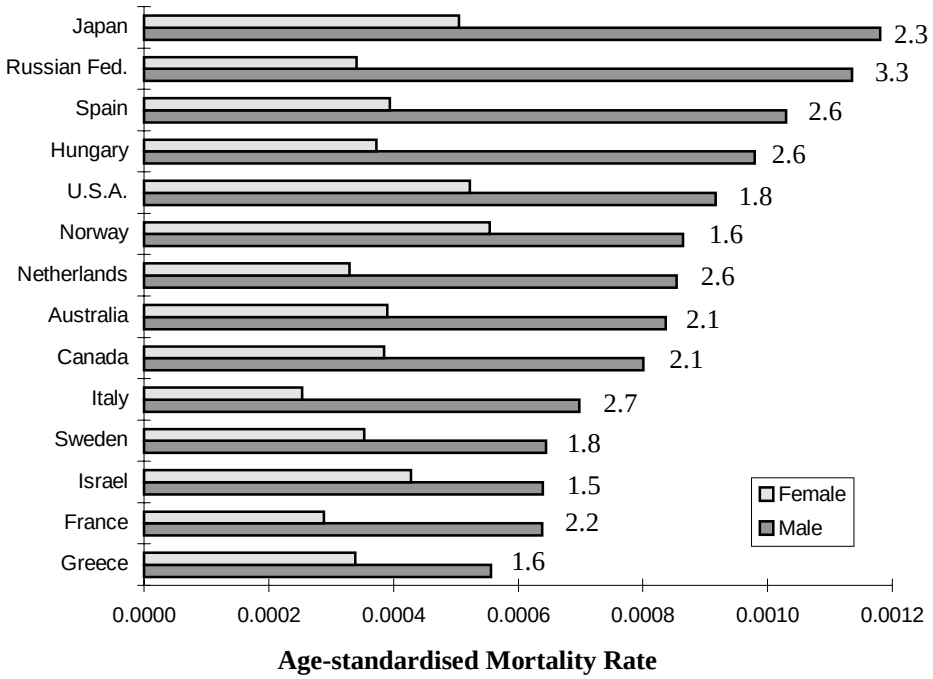
Historical Trends in Respiratory Disease Mortality

Lee et al. (1990) have analysed mortality rates due to various respiratory diseases using data for England and Wales for the period 1941-1985. They found that age-specific mortality rates in men and women followed a similar pattern to those for lung cancer, with evidence of a peak in mortality rates for men born around the turn of the century and for women born in about 1925. For one subgroup of respiratory diseases, this cohort effect was superimposed on a sharp downward trend in rates.

International Comparison of Respiratory Disease Mortality

Figure 14 shows mortality rates for all diseases of the respiratory system for males and females for a number of developed countries in 1991-93, as well as the sex ratio.

Figure 14: Age-standardised male and female mortality rates for respiratory diseases for various developed countries, and the ratio of the male to female rate, 1991-93.



Source: WHO (1995) - standard population is European standard population

Male rates of respiratory disease mortality for the developed countries in Figure 14 vary from 118.0 per 100,000 (Japan) to 55.6 per 100,000 (Greece), a ratio of 2.1. Female rates of respiratory disease mortality vary from 55.4 per 100,000 (Norway) to 25.4 per 100,000 (Italy), a ratio of 2.2.

International Comparison of the Sex Differential in Respiratory Disease Mortality

Figure 14 shows the ratio of male to female respiratory disease mortality for a number of developed countries. The ratios vary from 1.5 for Israel to 3.3 for the Russian Federation. A comparison of Figures 10 and 14 reveal consistent patterns in the sex ratios between countries for lung cancer and respiratory disease. Sweden, Israel, Norway and the USA have low ratios in both cases, and the Russian Federation, Italy and Spain have high ratios in both cases. It could be that this consistency reflects gender differences in smoking prevalence in each country.

The Contribution of Genetic and Biological Factors

The fact that mortality rates due to lung and related cancers and some respiratory illnesses are similar for male and female non-smokers (recall that Doll et al., 1980, found a sex ratio of 1.3 for this group of causes), along with the strong support for smoking as a cause of COPD indicates that genetic and biological factors are of secondary importance for this cause of death.

The Contribution of Environmental and Lifestyle Factors

The previously mentioned study of British male doctors over the forty year period to 1991 found increased rates of mortality for smokers compared to non-smokers for respiratory diseases as a whole and for the sub-categories, pneumonia and chronic bronchitis and emphysema. The ratio of mortality rates for heavy smokers compared to non-smokers was 4.4, 2.4 and 22.5 for these three causes respectively (Doll et al., 1994a), all highly statistically significant results. For the 22 year follow-up of female British doctors the ratio of mortality rates due to chronic bronchitis and emphysema for heavy smokers to non-smokers was 32, again a highly significant result (Doll et al. 1980). The ratios for chronic bronchitis and emphysema (which form a large proportion of COPD deaths) are thus of the order of those for lung cancer mentioned previously. As mentioned in section 6, the ratio of male to female mortality rates for lung and related cancers and certain respiratory diseases among non-smokers was only 1.3 (Doll et al., 1980), indicating that rates are similar in the absence of smoking.

Studies of correlations between smoking trends and mortality due to COPD also support the view that smoking is a cause of these respiratory illnesses. Lee et al. (1990) found that mortality rates in England and Wales for emphysema, chronic bronchitis and chronic airways obstruction were consistent with trends in cigarette consumption, although apart from emphysema the rates were declining more rapidly than expected, possibly due to decreased exposure to occupational hazards and air pollution. Lopez (1990) states that the contribution of cigarette smoking is reflected to some extent in differences in mortality rates due to COPD between countries, but warns that international comparisons are difficult due to diagnostic and coding differences.

Conclusion

A review of evidence on smoking and COPD has concluded that smoking is the major cause of COPD, accounting for 80-90% of deaths (US DHHS, 1984, cited in US DHHS, 1989). Lopez (1990) states that cigarette smoking is the leading cause of COPD, together with some influence from air pollution. Deaths due to COPD account for roughly 60-70% of total respiratory disease mortality and rates of death due to other respiratory diseases are very similar for males and females (19 per 100,000 for males compared to 18 per 100,000 for females in 1994 from ABS (1995b)). Hence smoking accounts for a large portion of the sex differential in the expectation of life due to respiratory diseases.

Section 8: CANCERS EXCEPT LUNG AND RELATED CANCERS

Introduction

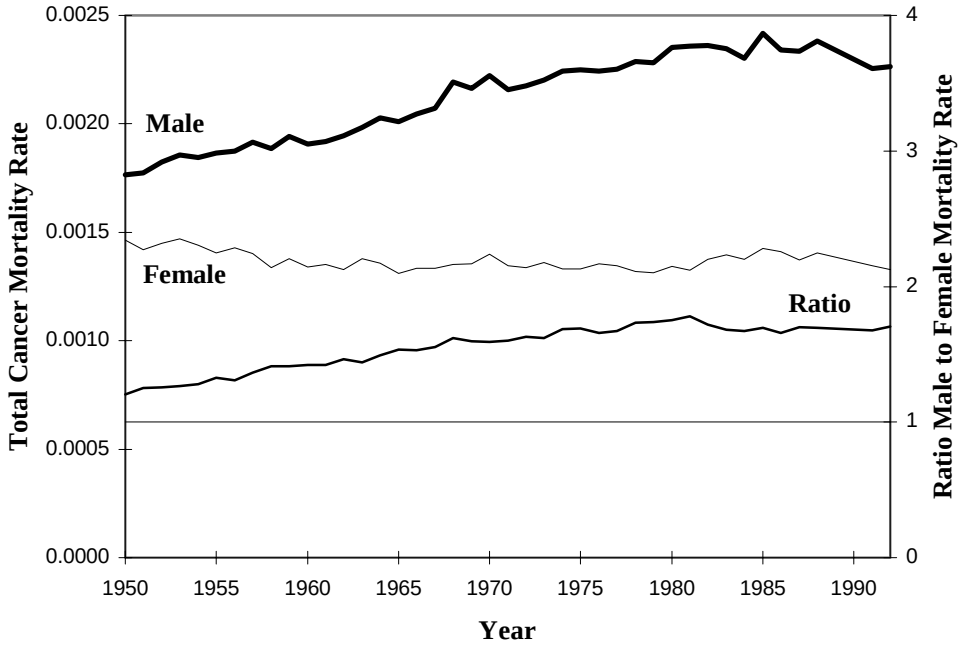
Other cancers include cancers of the digestive organs (stomach, colon, etc.), bone and connective tissue, breast, skin, genitourinary organs, other organs and lymphatic tissue. Rates for Australian males are highest for lung cancer, followed by genitourinary organs (including prostate) and digestive organs. Rates for Australian females are highest for the breast followed by the digestive system, lung and genitourinary organs. As seen in Table 3, the higher male mortality rates for other cancers as a whole account for about 9.4% of the total sex differential in the expectation of life at birth. The ratio of male to female overall mortality due to this cause is about 1.4 (Table 2). Removing breast cancer from this category gives a total contribution to the differential of 17.4% (-8.0% for breast cancer) and a ratio of 1.7.

Australian Data and Trends

Historical Trends in Other Cancer Mortality

Figure 15 shows age-standardised mortality rates due to all cancers (including lung and related cancers) for Australian males and females, and the ratio of the male to female rate, for the period 1950-1992. Note that the scale for the mortality rates is not logarithmic in this case.

Figure 15: Age-standardised mortality rates due to cancer, 1950-1992 for Australian males and females, and the ratio of the male to female rate.



Source: d'Espaignet et al. (1991) for 1950-1988 - standard population is 1988 population Jain (1994) for 1991 and 1992 - standard population is 1986 population.

For all cancers, male rates increased over the period from 176.4 per 100,000 in 1950 to 226.4 per 100,000 in 1992 (an increase of 0.6% pa.) and female rates declined slightly over the period from 146.5 per 100,000 in 1950 to 132.8 in 1992 (a decrease of 0.2% pa.). The ratio of male to female mortality rose over the period from 1.2 in 1950 to 1.7 in 1992. Although not shown here, male rates were also rising continually over the period 1921 to 1950, whereas female rates remained relatively stable over this period (d'Espaignet, 1991). In 1921 the ratio of male to female mortality was 100% (d'Espaignet, 1991), indicating equal mortality for males and females due to this cause. Around 1900, the sex ratio for all cancers was generally less than 100% for developed countries (UN Secretariat, 1988), indicating excess female mortality.

Data and Trends for Other Developed Countries

International Comparison of Other Cancer Mortality

Davis, Hoel, Fox and Lopez (1990) have reviewed international trends in cancer mortality for Italy, Japan, the Federal Republic of Germany, England and Wales, France and the USA for the period 1968 to 1988. They found that mortality rates for all cancers combined excluding stomach cancer and lung cancer increased for ages

above 54 years in all countries for this period. Stomach cancer rates were found to be declining for most countries, while lung cancer rates had started to decline in some age groups for some countries (see section 6 for a more detailed analysis of lung cancer trends). Cancer rates varied by factors as high as 30 between all countries, and up to 5 for industrialised countries (Davis et al., 1990). A detailed analysis of cancer mortality by particular types of cancer is beyond the scope of this paper.

The Contribution of Genetic and Biological Factors

Some cancers are predominantly or entirely sex-specific, including cancers of the breast, ovary, uterus and cervix for women, and cancers of the prostate and testis for males. Hence, genetic and biological factors contribute to the sex difference in mortality through these causes of death. However, despite the fact that there is a biological or genetic basis to these cancers, the **extent** to which each of these causes contributes to the sex differential depends on their importance as a cause of death, and this may be influenced by environmental factors.

For example, the majority of sufferers of breast cancer are females, due to innate gender differences in anatomy. However, it has also been found that environmental factors, for example the level of fat in the diet, play a role in this disease (see the following section). If consumption of fat increases over time, causing breast cancer rates to increase, then breast cancer will have a larger impact on the sex differential in mortality, and this effect can be attributed to both innate genetic differences and environmental factors. This interaction between genetic and environmental factors also applies to other causes of death and can make it difficult to ascertain the relative contribution to the sex differential of each set of factors (Waldron, 1983).

A detailed analysis of causes of specific types of cancer is beyond the scope of this paper. However, a study showing that the total cancer mortality rate for adoptees is increased fivefold if the adoptive parent has died of cancer before age 50 but is not significantly increased if the biological parent has died, provides support for the importance of environmental factors in the development of cancer (Sorenson et al., 1988). Waldron (1976) has stated that hormonal and genetic differences between men and women make little or no contribution to the higher overall cancer rates for males.

The Contribution of Environmental and Lifestyle Factors

The Role of Smoking

The forty year study of British male doctors found significantly increased rates of mortality for smokers compared to non-smokers for cancers of the pancreas, bladder, stomach, and rectum; and found a significant trend in mortality for myeloid leukaemia according to the amount smoked (Doll et al., 1994a). For the twenty-two year follow-up for British female doctors, a significant relationship was found between smoking and cancer of the ovary (Doll et al., 1980). An association between smoking and cancer of the cervix has also been found (US DHHS, 1989).

The Role of Diet

Breast cancer rates in North America are more than thirty times those in western Africa and central America and also vary markedly between developed countries (Davis et al., 1990). Lopez (1990) has stated that high breast cancer mortality rates in countries including the UK, Denmark and Belgium and low rates in countries including Japan, Romania and Greece are consistent with a high fat diet being a risk factor for this disease.

Stomach cancer is thought to be associated with diet, food storage and refrigeration (McMichael, McCall, Hartshorne, & Woodings, 1980). McMichael et al. (1980) compared mortality rates among migrants in Australia with those for the Australian-born population for various types of gastro-intestinal cancers. For stomach cancer, rates of mortality in the country of origin for all countries studied (England, Scotland, Ireland, Poland, Yugoslavia, Greece and Italy) were higher than those in Australia. In line with the hypothesis that environmental factors play a role in this disease, the rates of stomach cancer mortality for migrants were lower than the rates in the country of origin but significantly higher than the rates for the Australian-born, and reduced with increasing duration of residence. For colon cancer, migrants from European countries with rates of colon cancer lower than those in Australia experienced increased risk with increasing duration of residence in Australia, whereas migrants from Scotland, where rates are higher, experienced a decline in risk. Overall, the study supported the view that dietary change associated with migration is a factor influencing the rates of gastro-intestinal cancer.

The Role of Alcohol

It is known that alcohol plays a role in cancer of the liver, oral cavity, oesophagus, larynx and pharynx (Doll et al., 1994b). Studies have also suggested that beer consumption may be a risk factor for cancer of the rectum (McMichael, Potter and Hetzel, 1979). In a prospective study of US males over 17 years, Dyer et al. (1980) found a significant relationship between alcohol consumption and mortality due to all cancers combined, even after adjustment for age, blood pressure and smoking.

Other Factors

In a review of cancer risk factors, Doll and Peto (1981) list among the "firmly established causes" of cancer, older age at first pregnancy (breast), zero or low parity (ovary), sexual promiscuity (cervix), exposure to UV light (skin, lip), overnutrition (endometrium, gallbladder), parasites (bladder, liver) and the Hepatitis B virus (liver) together with a number of medical and occupational risk factors.

Conclusion

There is evidence that smoking, diet, alcohol and other environmental factors play a role in cancer mortality. The fact that the sex ratio for total cancer mortality in Australia in 1921 was 100% (indicating equal male and female mortality) suggests that environmental changes over the century (specifically cigarette smoking, especially among males, and changes in diet) may have contributed to the current male excess mortality. The importance of environmental factors is also suggested by the wide variations in cancer mortality rates around the world. In a comprehensive study of the various factors thought to contribute to cancer mortality, Doll and Peto (1981) estimated that in the USA in 1970, 75% to 80% of total cancer deaths for both sexes might have been avoidable, largely through avoidance of smoking and changed dietary habits.

Section 9: MOTOR AND NON-MOTOR ACCIDENT AND SUICIDE

Introduction

This category includes motor vehicle traffic accidents, non-motor accidents (including transport accidents, accidental poisoning, medical misadventure, accidental falls, fire, drugs and other accidents), homicide, violence and suicide.

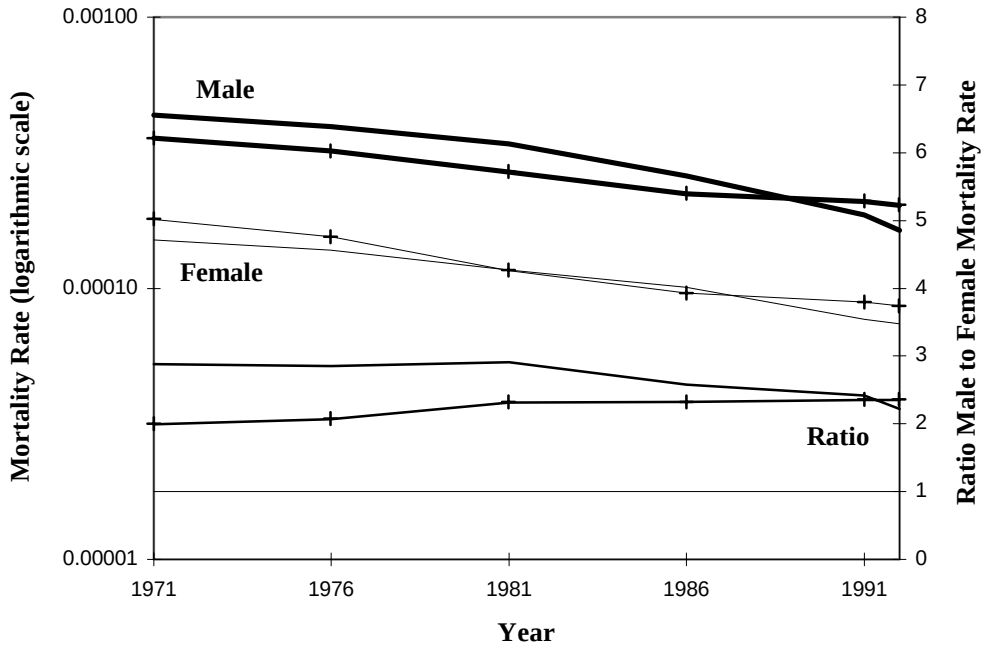
Results in Table 3 indicate that the higher male mortality rates for this group of causes accounts for 16.7% of the total sex differential in the expectation of life at birth. The ratio of male to female overall mortality due to accident is about 2.5 and suicide 4.2 (Table 2).

Australian Data and Trends

Historical Trends in Accident Mortality

Figure 16 shows age-standardised mortality rates due to motor accidents and non-motor accidents for Australian males and females, and the ratios of the male to female rates, for the period 1971-1992.

Figure 16: Age-standardised mortality rates due to motor and non-motor accident, 1971-1992 for Australian males and females, and the ratio of the male to female rate.



Note: **Crossed lines represent non-motor accident mortality. Plain lines represent motor vehicle accident mortality.**

Source: **Jain (1994) for 1970-72 (plotted as 1971), 1975-77 (1976), 1980-82 (1981), 1985-87 (1986), 1991 and 1992 - standard population is the Australian mid-year 1986 population.**

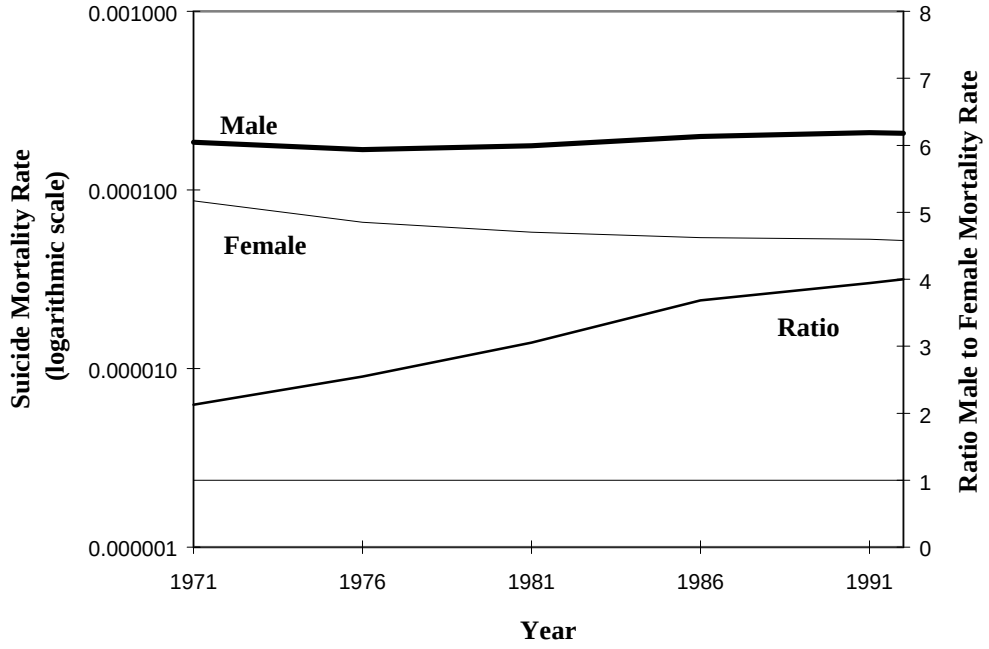
Motor vehicle accident mortality declined over the period for both males (43.5 per 100,000 in 1971 to 16.4 per 100,000 in 1992) and females (15.1 per 100,000 in 1971 to 7.4 per 100,000 in 1992). The relative rates of decrease were similar for males and females in the first decade, but have since been greater for males. The sex ratio remained stable at about 2.9 until 1981 and subsequently decreased to 2.2 in 1992.

For non-motor accident, male rates have declined over the period (35.7 per 100,000 in 1971 to 20.2 in 1992) and female rates have done likewise (17.9 per 100,000 in 1971 to 8.6 in 1992). The rate of decrease was faster for females in the first decade; the sex differential was 2.0 in 1971, increased to 2.3 by 1981 and has remained around this level to 1992.

Historical Trends in Suicide Mortality

Figure 17 shows age-standardised mortality rates due to suicide for Australian males and females, and the ratios of the male to female rates, for the period 1971-1992.

Figure 17: Age-standardised mortality rates due to suicide, for Australian males and females, and the ratio of the male to female rate, 1971-1992.



Source: Jain (1994) for 1970-72 (plotted as 1971), 1975-77 (1976), 1980-82 (1981), 1985-87 (1986), 1991 and 1992 - standard population is the Australian mid-year 1986 population.

Male rates have increased over the period 1971-1992, whilst female rates have generally declined. The ratio of male to female mortality has increased from 2.1 in 1971 to 4.0 in 1992.

Data and Trends for Other Developed Countries

International Comparison of Accident Mortality

In a review of causes of death in developed countries, Lopez (1990) found that countries with the highest male mortality rates due to motor accident included Portugal, New Zealand, Greece, Australia, France and the USA (all between 26 and 42 per 100,000). The lowest male motor accident mortality rates were for Malta (8 per 100,000) with low rates also observed for the UK, Sweden, the Netherlands, Norway and Japan (all between 13 and 15 per 100,000). Mortality rates for females were lower than for males in all countries and the highest death rate for females (12 per 100,000 in New Zealand) was still lower than all male rates except Malta. Mortality due to motor vehicle accidents has been declining rapidly in the developed countries since the mid 1960s (Lopez, 1990).

International Comparison of Suicide Mortality

In the international comparison of mortality by Lopez (1990), male suicide mortality rates were highest for Hungary (68 per 100,000) followed by a group of countries including Finland, the USSR, Switzerland and France (34 to 43 per 100,000) and lowest for Malta, Greece, Spain, Israel, and the UK (1 to 12 per 100,000). For females the highest rates were observed for Hungary and Denmark (19 to 23 per 100,000) and the lowest mortality countries were similar to those for males. Suicide mortality has generally been increasing for males and females in the developed countries since the early 1950s (Lopez, 1990). In 1990 in all developed countries male suicide rates exceeded those for females; the ratio of male to female mortality varying between about 1.3 and 5.0 with a median of 2.9, Australia being at the upper end of this range (Ruzicka and Choi, 1993).

The Contribution of Genetic and Biological Factors

It has been suggested that males hormones may contribute to higher levels of aggression and physical activity and thus to increased chance of accident, however, evidence for this is inconclusive (Waldron, 1985). Suicide has been linked with depressive and other mental illnesses, which may have some genetic or biological basis. However, some studies have indicated that mental illness may not be a necessary precursor of suicide (see Ruzicka and Choi, 1993 for a brief review).

The Contribution of Environmental and Lifestyle Factors

Higher male motor accident mortality is due in part to the fact that males make more use of cars for occupational reasons; however, at least some studies have shown that for each mile driven males have more fatal accidents and they also appear to drive less safely (Waldron, 1976). Motor vehicle accident mortality is influenced by blood alcohol level; studies in some countries have found that 30-50% of those responsible for fatal motor accidents and 30% or more of pedestrians involved in motor accidents have blood alcohol levels in excess of the legal limit (Lopez, 1990). In addition, correlations between alcohol consumption and motor vehicle accidents in Australia are consistent with an association between alcohol and road deaths (McMichael, 1980). Pollard (1996) suggests that random breath testing, changed attitudes to drink driving, compulsory use of seat-belts and speed checks have all contributed to the substantial decline in road deaths in Australia. Lopez also states that enforcement of stricter drink-driving, speed and seat-belt rules have been responsible for the declining motor accident mortality since the mid 1960s for all developed countries (Lopez, 1990). That these factors are important is supported by the fact that countries with the most stringent speed and drink-driving rules have lower motor accident mortality (Lopez, 1990).

Mortality rates due to other accidents are also higher for men. This excess is due, in part, to occupational hazards, which have more impact on men because they work more and generally have more physically hazardous jobs (Waldron, 1976). Higher alcohol consumption and use of firearms among men would also be expected to contribute to increased accident mortality (Waldron, 1976).

Hence, alcohol consumption has been shown to be associated with accident mortality, and it is known that alcohol consumption is higher for males in most countries. This factor has been included here as an environmental/lifestyle factor. However, it should be noted that higher alcohol consumption (and more "risky" behaviour in general) may be more socially acceptable for men because women have more often had the role of taking care of children. Hence there may be an indirect relationship between the fact that only women can bear children and the fact that alcohol consumption has been more acceptable for males (Waldron, 1983). This interaction between genetic and environmental factors makes it difficult to apportion the sex difference in mortality into these two discrete categories; however, we would consider alcohol consumption to be predominantly a lifestyle choice.

Risk factors for suicide are more difficult to assess. The fact that suicide rates are higher for the single, widowed and divorced; that high rates have in some studies been associated with prolonged unemployment and lack of meaningful social relations; that suicide during pregnancy has decreased dramatically as pregnancy outside marriage has become more socially acceptable; and that male suicide rates in Australia over this century have shown some correlation with changes in the economic cycle, all suggest that the social environment has an important influence on suicide (see Ruzicka & Choi, 1993, for a review of these factors and Burvill, 1980, for comment on high Australian male suicide rates during the Depression). Lopez (1990) also notes that countries with a strong Catholic tradition have lower rates of suicide mortality. Waldron (1976) suggests that higher suicide rates for males may be due, in part, to lower rates of suicide for housewives compared with those in other occupations, and the fact that men, possibly in an effort to appear "dominant" or "powerful", are more likely to make a fatal suicide attempt.

Conclusion

Genetic factors may lead to increased aggression and activity in males, although the evidence for this is inconclusive. Environmental factors which may contribute to higher male accident and suicide rates include the fact that males experience more occupational hazards, consume more alcohol, may drive less safely, are more likely to use firearms, and may be more influenced by aspects of the social or economic environment. There are also interactions between genetic sex differences and behaviour which occur due to the different social roles for males and females.

Section 10: OVERALL ASSESSMENT OF GENETIC VERSUS ENVIRONMENTAL INFLUENCES

There is evidence that various behaviours or lifestyle choices - including cigarette smoking, alcohol consumption, diet and fat consumption, physical activity and occupation - contribute to mortality by various causes. The fact that there are gender differences in the distribution of these risk factors means that they will make some contribution to the sex differential in mortality. Many authors believe that social/ environmental/ lifestyle factors are the primary cause of the large increases in the sex differential in developed countries in this century, and for differences in the sex differential between countries (Lopez, 1983; Zhang et al., 1995; Knudsen & McNown, 1993). There is also some evidence for genetic or biological influences on mortality, including the effect of sex hormones on the risk of ischaemic heart disease.

To assess the relative impact of behavioural versus innate risk factors is difficult, and some of the problems which arise in attempting to do this have been mentioned previously. As Waldron (1983) notes, the relative impact of each set of factors must be assessed at a particular point in time and under a particular set of environmental conditions, and hence any estimate of the importance of each set of factors cannot be generalised to a different country or point in time. In addition, there are interactions between environmental and innate factors which make apportioning the sex difference very difficult, if not impossible.

Because of these limitations, few researchers have attempted to estimate the relative proportions of the overall sex differential which are due to each set of factors, although some have made estimates for particular risk factors or causes of death (e.g. Doll and Peto, 1981, for cancer). Retherford (1975) has suggested that 75% of the change in the sex mortality differential between 1910 and 1962 can be attributed to cigarette smoking; however, this figure does not allow for interactions between smoking and other risk factors. In one of the few studies which do allow for multiple risk factors, Wingard (1982) found that adjustment for 16 biological and behavioural factors (including smoking and alcohol consumption) actually increased the differential. However, a second study using different risk factors found a decrease in the differential after adjustment from 1.7 to 1.3 (Wingard et al., 1983).

Hence, assessment of the cause of the sex differential in mortality is difficult and no firm conclusions have been reached. Despite this, there is clear evidence that environmental factors do have an important impact on mortality.

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