

FACTORS AFFECTING RETIREMENT MORTALITY

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Abstract

This paper is the final report in a project commissioned by the Society of Actuaries Social Security Committee. The paper considers a variety of possible risk classification factors that could be used in estimating the value of any mortality based benefit for retirees.

As many countries consider mandatory individual retirement accounts as their answer to a secure Social Security system, the question arises as to whether all workers can get true “market value” annuities when they retire. It is clear today that private sector life annuities are priced assuming that the applicant is healthy—very healthy. Very little underwriting or risk classification now exists in the individual annuity marketplace. However, if a large percentage of the population were looking to annuitize their Social Security accounts upon retirement, there would be strong pressure for more risk classes in the annuity pricing structure.

Even without the advent of individual accounts for Social Security, the authors of this paper feel there may be real market opportunities for more risk classification in the individual annuity market and the offering of “impaired life annuities”.

In addition to refinements in annuity pricing, another issue to consider is proper valuation of liabilities held by defined benefit pension plans, whether privately or publicly sponsored. For example, is it appropriate for all plans to use the same mortality assumption? What variation might we expect from plan to plan, reflecting differences in location, industry, jobs covered, etc.? Poor estimates of mortality, and therefore liabilities, could risk plan solvency or, conversely, unduly constrict plan sponsors and contributing participant assets.

This paper reviews 45 recent research papers that look at factors that affect mortality after retirement. In particular, factors that seem to be important in predicting retirement mortality include: age, gender, race and ethnicity, education, income, occupation, marital status, religion, health behaviors, smoking, alcohol, and obesity. For each factor, this paper gives highlights relative to the named factor of the impact expected from that variable as described in the 45 reviewed research papers.

The authors believe there is a wealth of information contained in the summaries that follow, and it is our sincere hope that this paper will cause an increased interest in a more broadly-based risk classification structure for individual annuities and improved mortality assumptions for valuing all retirement benefits.

Factors Affecting Retirement Mortality

Introduction

This paper was commissioned by the Society of Actuaries Social Security committee.

Mortality assumptions are a basic input to valuing life insurance, annuities, pension plans and even Social Security. Generally, the mortality rates used differ by only age and gender. When are further refinements appropriate? Which of the factors that can influence mortality experience are appropriate to include? How can these factors be fairly and systematically reflected in the mortality assumptions used in the valuation of benefits paid to retirees? How might these factors be expected to influence future improvements in mortality? This paper is the starting point for answering these questions and others related to risk classification of retiree mortality.

One of the more interesting applications of the work presented herein relates to proposals to reform many Social Security systems into mandatory individual accounts. One of the problems with such individual accounts is the ability to annuitize the account values at retirement at “fair market” rates. At the moment, there is very little to no underwriting in the individual annuity market. If a person volunteers to buy an individual annuity, it is assumed that that person is healthy—in fact, very healthy. Except for structured settlements (income for people severely impaired mostly because of auto accidents) there

exists almost a single mortality assumption for individual annuities in Canada and the United States.

If more countries move to mandatory individual accounts to provide retirement income, or if annuitization of retirement savings is made mandatory (these are separate possibilities), then there will be a wider spectrum of mortality among the prospective annuitants than exists today. This, in turn, will create the need for more risk classification in the annuitant market.

This is already happening in the UK where pensioners who have used tax-advantaged systems to save for retirement must buy some form of life annuity by age 75. In response, there is now a growing “impaired annuity” market in the UK. Using UK mortality and an interest rate of 5 percent per annum, the following table shows the increase in annuity income justified by various increases in the mortality rate, q_x .

Increase in Annuity			
Increase in q_x	Age 65	Age 75	Age 85
+25%	+ 7%	+11%	+15%
+50	+13	+21	+30
+100	+24	+40	+58

“Impaired annuities” met a market that had become disappointed with ever-decreasing annuity income per dollar of premium because of decreasing interest rates combined with decreasing mortality rates (i.e. increasing life expectancy).

“Impaired annuities” have found demand in two separate market niches. It has enhanced incomes for those who retire with profiles of high mortality (e.g. those in poor health). It has also worked to provide annuities to fund nursing home care and other long term care costs for ill or frail elderly people who are near the end of their lives.

In a competitive annuity market, impaired annuities represent an opportunity for some insurers to carve out a profitable, sizeable niche at the expense of their competitors. New players in the market can do this without threatening the turnover of their existing book of business.

This is especially timely as the massive baby-boom generation is just now starting to think about the issue of retirement income security and the potential annuitization of retirement savings.

In the UK, risk classification in annuity pricing has created new rates for classes that include: smokers, those with medical impairments, (diabetics, high blood pressure, high cholesterol, stroke or heart attack victims), the overweight, and, more recently, manual workers living in geographic areas displaying higher than average mortality.

It is the hope of the authors of this paper that broader risk classification will come to North American annuity markets. If that were to occur, more retirees could buy life annuities at a fair market value. This is just not possible today and is a hindrance to the expansion of this important market. The review of the factors included in this paper will assist in the future creation of a wider variety of risk classes.

In addition to refinements in annuity pricing, there is also the issue of proper valuation of liabilities held by defined benefit pension plans, whether privately or publicly sponsored. For example, is it appropriate for all plans to use the same mortality assumption? What variation might we expect from plan to plan, reflecting differences in location, industry, jobs covered, etc.? Poor estimates of mortality, and therefore liabilities, could risk plan solvency or, conversely, unduly constrict plan sponsors and contributing participant assets.

The authors have reviewed 45 papers addressing 12 different risk factors that influence mortality after retirement. The factors reviewed include: age, gender, race and ethnicity, education, income, occupation, marital status, religion, health behaviors, smoking, alcohol, and obesity. For each factor, the pertinent results from the 45 papers reviewed are gathered and presented. The 45 papers have also been summarized and those summaries are available to interested readers (although not included directly in this paper) on the Society of Actuaries web site.

As is virtually always the case, this research is presented with one very large caveat. Our summary review of factors affecting retirement mortality focuses on 12 risk factors

as previously listed. This list could be expanded to include dozens of other factors -- both proximate to and remote from the time of death. For example, Hurd et al. (1999) evaluated the impact of 13 health indicators that are strong predictors of mortality.

This raises two questions: (1) What should one do about the potential lack of independence between the risk factors that are included in an actuarial model? (2) What should one do about important risk factors that are not included in an actuarial model? From an actuarial perspective, these questions relate to the construction of an initial risk classification system and any subsequent refinement of that system.

(1) Our review indicates that all 12 risk factors are important enough to be included in an actuarial model to the extent that such data are available for risk classification. Practically, one might expect to have information on age and gender, and possibly one or two others, but not on all 12 risk factors. This means that one needs to be careful to ensure that the numerical values of parameters used for select subsets of the 12 risk factors are consistent between the estimation model (in the published paper) and the actuarial model. For example, if the estimation model jointly included age, gender, marital status, education, income, and occupation while the actuarial model included age and occupation, the parameter values from the estimation model would likely not be directly applicable to the actuarial model. An important exception would occur if the risk effects of age and occupation were independent of the risk effects of gender, marital status, education and income.

(2) As a general rule, the larger the number of risk factors used in an actuarial model, the less significant are the effects of any omitted variables, and the greater the likelihood that the assumption of independence of the omitted variables would be a reasonable approximation to reality. If an important risk factor is missing from an actuarial model, then the most direct response is to re-estimate the published estimation model with that risk factor omitted (assuming that the risk factor was originally included in the published model). This would yield parameter estimates for the actuarial model that were consistent with the retained risk factors, even though it was recognized that the parameter estimates were somewhat biased. This would be satisfactory if the distribution of the omitted factor in the estimation sample was the same (or approximately the same) as in the insured population. An important exception would occur if the omitted risk factor were subject to significant antiselection (which would be more likely for life insurance than retirement annuity models).

Because of the complex issues involved in estimating numerical values for parameters of actuarial models, including choice and measurement of risk factors, assumptions about independence/dependence and antiselection, and other related issues, we focused our review on the qualitative impact of the 12 main risk factors, on the consistency of the evidence supporting conclusions about that impact, and on specific interactions between the risk factors that have been identified and documented to date.

Factors Affecting Retirement Mortality

Age

It is widely accepted that mortality rates increase with age. Horiuchi and Wilmoth (1998), Brown (1997) and Brown (1988) state that the rate of mortality increases almost exponentially with age. This reflects the decline in physiological functional capacity after age 30.

However, the pattern of mortality changes at older ages. Brown (1997) comments that the exponential form only applies between the ages of 30 and 85, beyond which the force of mortality levels off and becomes almost constant. Horiuchi and Wilmoth (1998) also note the slow down of increases in mortality rates at very old ages. They propose that this is due to the 'heterogeneity hypothesis', i.e. selective survival of healthier individuals to older ages. Their results supported this, however it was also suggested that factors other than heterogeneity might play a role.

Brown (1997) comments that there are problems with data reliability at advanced ages, the size of the population is relatively small and there are also potential errors due to accidental and deliberate misstatements of age in census data. Kestenbaum (1997) agrees with this, but comments that information on death certificates has improved substantially.

Deaton and Lubotsky (2001) comment that cause of death differs by age. In considering cause of death Horiuchi and Wilmoth noted that mortality rate deceleration by age was observed for most major causes. However, it was found that mortality deceleration started at relatively young ages for degenerative diseases, but deceleration for infectious diseases was at relatively old ages. Age 75 was the dividing point to distinguish between younger old ages and older old ages. They distinguished between two types of adult mortality; senescent mortality, which results from the age-related deterioration of physiological functions and background mortality, which is relatively independent of age. Mortality deceleration tended to shift to older ages as the level of senescent mortality declined.

A number of other factors have been found to interact with age.

Trovato and Lalu (1998) found a consistent age pattern within sex differences, with smaller gender gaps at younger ages and then generally increasing until peaking in the oldest age category.

There are well-documented racial differences in mortality, with blacks generally having higher mortality than whites. However, Kallan (1997) found the significant effects of race on all-cause mortality were weaker for the older ages. Kestenbaum (1997) noted that these differentials narrowed with increasing age and that black-white mortality crossovers occurred at ages 88 for males and 87 for females. He does not consider these crossovers to be artifacts of excessive age mis-reporting. He also found a Hispanic-black crossover for women and both Hispanic-black and Hispanic-white crossovers for men. Kestenbaum

also noted that data reliability problems at older ages were more significant for blacks than whites. The case for Hispanics is slightly more complex due to less reliable recording of race. In general, death rates for Hispanics in all age groups over 45 were below comparable rates for both non-Hispanic whites and non-Hispanic blacks.

Hummer (1996) found some suggestion of a skin color by age interaction term in analyses. Williams and Collins (1996) supported this, commenting that the age patterning of racial disparities suggest that age may be a proxy for the cumulative exposure to racism and adverse living conditions.

Williams and Collins (1996) noted an age pattern in the association between SES (socio-economic status) and mortality. SES mortality differentials were largest during middle age and relatively small at older ages. A number of other studies obtained similar results. Hurd et al (1999), using a population aged 70 or over, found that there was evidence of differential mortality for the SES indicators of income, wealth and education but that these differences decreased with age. Kallan found that a range of sociodemographic variables had independent effects on all-cause mortality. However, the effects for both all-cause and specific causes of death were weaker for the older age group (65+). The Statistical Bulletin (1975) also found that at older ages, mortality differences due to social class leveled off. It suggested that biological and genetic influences increased in importance as the relation between socioeconomic factors and mortality weakened. Vaillant and Mukamal (2001) comment that although the socially disadvantaged are less

likely to survive until age 75, having reached 75 there are then no obvious effects of social class.

In Deaton and Paxson (1999), education and income were found to affect mortality differently for young adults (those under 65), versus older adults (those aged 65 and over). Rogers (1995) highlights the substantial changes in income for those over 65 with Hurd et al (1999) also noting that wealth and income fall with age. Brown (1997) supports this by mentioning that there are indications that at advanced ages mortality does not vary by work history. Knox and Tomlin (1997) also found that the disparities in death rates between high and low income earners were not as pronounced after age 74. Wolfson et al (1990) note that the clear mortality gradients by income and educational attainment are not as strong for those over age 65. In contrast, Preston and Elo (1995) found that educational inequality trends are more adverse for people aged 65+ than for those aged 25-64.

In Judge (1995), Wilkinson (in the commentary) suggests that a consequence of the age differences in income effects, combining all ages in life expectancy masks the large impact of income inequality on younger lives. His later paper with Lobmayer (2000) supported this by finding that greater inequality was associated with higher premature mortality but that this did not extend for ages over 65.

Allison et al (1999) refers to another paper by Stevens et al which found that hazard ratios for obesity decreased steadily with advancing age.

Hurd et al (1999) considered the impact of an individual's subjective view of their health for mortality. They found that subjective survival probabilities have considerable explanatory power for mortality, even after controlling for socioeconomic and health indicators. However, they noted some overestimation of subjective survival probabilities among the oldest age group.

In recent years, people have been living to older ages. Vaillant and Mukamal (2001) consider what factors are predictors of successful aging. They propose that a high level of education and having an extended family network are the most important predictors, and that absence of alcohol and cigarette abuse are the more important protective factors. They state that the increasing number of people living to age 85 and older is not so much the result of greater longevity among the elderly, but of more people living to age 65. Indeed, their analyses suggested that "good" and "bad" ageing from age 70-80 could be predicted by variables assessed before age 50. They highlight that greater longevity is resulting in fewer, not more, years of disability as generally those surviving to 85 are more likely to be active.

Alcohol

Alcohol has been shown to have an impact on mortality, but the direction and extent of this impact varies. Vaillant and Mukamal (2001) found that the absence of alcohol abuse was one of the most important protective factors for successful aging. However, Thun et al (1997) found that moderate alcohol consumption is associated with a small reduction in mortality. They showed that deaths from all causes were lowest for those consuming about one drink daily, but for more than one drink, the balance of adverse and beneficial effects was dependent on age and background cardiovascular risk.

Thun et al (1997) noted that the relation between drinking and mortality varied according to specific causes of death. Deaths from cirrhosis, alcoholism, cancers of the mouth, esophagus, pharynx, larynx and liver were 3 to 7 times higher if at least four drinks are consumed per day, compared with non-drinkers. Deaths from external causes e.g. injuries, suicide, were also increased for males. For females, death from breast cancer increased by 30% if one or more alcoholic drinks were consumed daily. In contrast, death from cardiovascular causes was 30-40% lower for men and women consuming one or more drinks daily, compared with non-drinkers and the percentage of deaths due to cardiovascular causes decreased as alcohol consumption increased.

Thun et al (1997) commented that alcohol consumption varied with demographic characteristics and was positively correlated to cigarette smoking. This was supported by Lantz et al (1998), who found that the distribution of four behavioral risk factors

(cigarette smoking, alcohol drinking, sedentary lifestyle and relative body weight) significantly varied by educational attainment and annual household income. Those with the least education and lowest income were significantly more likely to be current smokers, overweight and in the lowest quintile for physical activity. However, the effects of smoking and drinking were no longer significant once they were adjusted for demographic, socioeconomic and other health behavior variables.

Education

Vaillant and Mukamal (2001) found that one of the most important predictors of successful aging was a high level of education.

Sorlie et al (1995) found that higher education level was associated with lower mortality in men and women. Similar results were obtained in Pappas et al (1993) and in Rogers et al (1999). Pappas et al (1993) found that death rates fell consistently with increasing levels of education. Adjustments for other variables reduced the risks but they remained significant. Rogers et al (1999) state that the continuous inverse relationship between education and mortality is robust to controls for age, sex, race, marital status, cigarette smoking, adequacy of housing and income. In contrast, Attanasio and Emmerson (2001) found that education was significant for morbidity, but not for mortality and Lantz et al (1998) found that education was related to mortality through its association with income.

Education is one possible measure of socioeconomic status. Deaton and Paxson (1999) show that at the individual level, both income and education are separately protective against mortality. However, Vaillant and Mukamal (2001) suggest that education is a more significant cause of differential mortality than other differences in socioeconomic status. In comparing college students and core-city youth over a long period, they found that the core-city men who had completed 16 or more years of education had very similar health to the college cohort. Preston and Elo (1995) also consider education to be advantageous relative to occupation and income. They state that information on educational attainment is available for people who are not currently in the labor force and its value is less influenced by health problems that develop in adulthood. Rogers et al (1999) also suggest that education may be the best measure as it is determined early in life and so can be assessed for all individuals. However, the Statistical Bulletin (1975) states that occupation, education and income are associated with health and longevity, but highlights that the interdependence of these factors mean that the effect of any one is affected by the presence of the others.

The level of education can also affect the cause of death. Kallan (1997) found that education affected every cause of death in the younger age group, but particularly those having a large behavioral component. Bucher and Ragland (1995) found that education was inversely related to blood pressure, cholesterol and smoking. It was also inversely associated with mortality from coronary heart disease, stroke and all causes. However, no significant association was found between death from non-lung cancer and education.

Deaton and Paxson (1999) comment that education affects mortality differently for men and women. For men, education influences mortality only through its association with income, whereas for women, education has a separate protective effect. Lantz et al (1998) found that the relationship between education and mortality and between income and mortality is stronger for females.

Sorlie et al (1995) found that the strongest relationships between higher education and lower mortality were in the under 65 age group, as did Deaton and Paxson (1999).

Rogers et al (1999) state that not only does education affect mortality through its link to employment, income generation and information gathering, it also affects mortality by influencing health behavior and the use of health services. This would support Kallan's (1997) finding that education particularly affected the causes of death having a large behavioral component. In another paper, Rogers et al (1999) comment that those with less education and lower incomes were more likely to be smokers and less likely to quit. However, in their study, income and education did not alter the effects of smoking significantly. Lantz et al (1998) also found that the distribution of four behavioral risk factors (cigarette smoking, alcohol drinking, sedentary lifestyle and relative body weight) significantly varied by educational attainment. Those with the least education were significantly more likely to be current smokers, overweight and in the lowest quintile for physical activity. Bucher and Ragland (1995) also found those with less education had higher risk factors. They noted that those with less education were in the older age groups, were less tall and smoked more.

Some of the papers also identified increases in educational differentials in recent years. Pappas et al (1993) found that absolute death rates declined for people of all educational levels, but the reduction was greater for those with more education than for those with less, highlighting an increasing disparity in mortality rates for those of different education levels. Preston and Elo (1995) found that educational inequalities have widened for males but contracted for working-age females. For both sexes, inequality trends are more adverse for people aged 65+ than for those aged 25-64. They state that the reasons for these changes are not easily explained and are likely to be multi-factorial and some or all of the factors must be highly differentiated by sex.

Gender

Rogers (1995) comments that gender interacts with mortality for both overall mortality and cause-specific mortality. Trovato and Lalu (1998) state that there is an accepted difference in life expectancy at birth between the two sexes, which favors females and that the sex gap has been narrowing in highly advanced Western nations. Paradoxically, Vallin (1995) states that the gender mortality gap has increased in recent years and states that gender is not a factor in excess male mortality. Gender is not acquired, but predetermined at conception; it is not because they are males that men die earlier than women.

Vallin notes that the recent increase in the gender mortality gap has coincided with an increasing disparity in socioeconomic groups and comments that this is despite men and women's roles in society having grown considerably closer. He suggests that some of the mortality differences are a function of differential social roles. This is supported by the findings of Preston and Elo (1995). They found that educational inequalities have widened for males but contracted for working-age females. They state that the reasons for these changes are not easily explained and are likely to be multi-factorial but some or all of the factors must be highly differentiated by sex.

In Deaton and Paxson (1999), education and income were found to affect mortality differently for men and women. They noted a striking positive effect of income on mortality among young men, but the effect was large and negative for young women. They also found that it was income, not education, that was protective for men's health, whereas income and education had separate protective effects for women. Lantz et al (1998) found that the relationship between education and mortality and between income and mortality is stronger for females. However, Pappas et al (1993) found that the SES-mortality relationship was not as strong for females. Hurd et al (1999) found few differences between males and females for the effects of SES or health conditions on mortality. It should be noted that subjects in this study were aged 70 or over.

Attanasio and Emmerson (2001) considered wealth rankings and found that, even after conditioning on initial health status, they were important determinants of mortality and

morbidity, but that the effects on female mortality and morbidity were marginally lower relative to those for males.

Vallin (1995) considered socioeconomic groups, based on occupation, and found that occupation was related to excess male mortality. The highest probability of death for women was far lower than the lowest mortality level for men and the range of death probabilities is also much greater for men compared to women. Although male and female occupations are quite different, the study determined that if working men had the same occupational structure as women, this would actually increase excess mortality. Vallin suggests that the gender mortality gap may be explained to an extent by factors similar to those that contribute to socioeconomic differentials in mortality. For example, the widening of the mortality gap between socioeconomic groups is partially a result of the greater responsiveness of the higher social groups to health promotional campaigns and a more pervasive sense of health consciousness.

Vallin (1995) also suggests that some of the mortality differences are a function of behavioral differences. Behavior affects mortality in two ways, directly through detrimental health practices and indirectly through the use of health services. This can enhance or mitigate positive or negative effects of other factors affecting mortality. Women are increasingly large consumers of health services and have benefited from medical progress earlier than men. Trovato and Lalu (1998) also deemed lifestyle and personal behaviors as important for cause of death.

Rogers et al (1999) comment that a smaller proportion of women than men are currently smokers and women were more likely to have never started smoking and more likely, if smoking, to be smoking a smaller number of cigarettes. They suggest that smoking is a major contributor to the current sex gap in mortality.

Thun et al (1997) found that the relation between drinking and mortality varied for males and females according to specific causes of death. If at least four drinks are consumed per day male deaths from external causes (e.g. injuries, suicide) increased significantly. However, for females, death from breast cancer increased if one or more alcoholic drinks were consumed daily.

Rogers (1995) found marriage was more advantageous for men as did Brown and Di Meo (1995). However, Rogers (1995) suggested marriage might be more psychologically constraining for women whereas Brown and Di Meo (1995) only found that marriage was less protective for females. They suggested that marriage offers protection to males through social support, but for females it is mainly through economic factors and that the lifestyle change is less significant for females. Hurd et al (1999) found there was no differential effect of marital status for men compared with women. However, again, it should be noted that subjects in this study were aged 70 or over.

Trovato and Lalu (1998) found a consistent age pattern in the gender differences, with small gaps in younger ages and generally peaking in the oldest age category.

Kark et al (1996) found that belonging to a religious collective was associated with a strong protective effect and that the protective effect of religiousness was stronger in women than in men.

LeClere et al (1997) suggest that residential segregation influences individual mortality and that the effects are larger for men than women. The mechanisms also differed by gender. It was suggested that neighborhood income and ethnic concentration affects male mortality risk and that physical and financial stress affected female mortality, but the educational attainment of neighbors protected all women.

Trovato and Lalu (1998) also noted that the amount of the gender mortality difference depends on the time period and the country. Despite finding that gender mortality differentials had narrowed in Western countries, i.e. Australia, the United States, Sweden and England/Wales, they found the gap had widened in Hungary, Japan and Portugal.

(Aside: for readers wanting an end-depth discussion of the impact of gender on mortality, see: Kalben, Barbara Blatt (2002): “Why Men Die Younger: Causes of Mortality Differences by Sex, Society of Actuaries Monograph M-LI01-1, Schaumburg. Mortality in retirement is not the focus of this monograph, however.)

Health Behaviors

Adler et al (1994) found that health behaviors are also closely tied to both SES and health. Vaillant and Mukamal (2001) suggested that the absence of alcohol and cigarette abuse was the most important protective factor for successful aging. Lantz et al (1998) noted a high degree of stability in health behaviors for all individuals. Vallin (1995) comments that behavior affects mortality in two ways, directly through detrimental health practices and indirectly through the use of health services. Therefore behavior can enhance or mitigate positive or negative effects of other factors affecting mortality.

Wei et al (1999) found that compared to normal-weight men obese men, had much higher risk of cardiovascular disease mortality and all-cause mortality. Overweight men had intermediate death rates between normal-weight and obese men. They also highlighted that men who were overweight or obese were also more likely to have baseline disease, smoke cigarettes, be sedentary and have a family history of cardiovascular disease. In contrast, Vaillant and Mukamal (2001) found body mass index to be only marginally significant. Lantz et al (1998) did not find being overweight was significant in any of their models.

Wei et al (1999) stated that low cardio-respiratory fitness was a strong and independent predictor of cardiovascular disease and all-cause mortality.

Rogers et al (1999) comment that high-risk smokers were not very likely to simultaneously be high-risk drinkers and abnormally overweight. This did not change with age. Their study found that neither exercise nor alcohol consumption had much impact on smoking patterns, however, being underweight or overweight increased the effects of smoking on mortality.

Lantz et al (1998) commented that some health behaviors are associated with a significantly higher risk of death for specific causes of death. They found that the distribution of four behavioral risk factors (cigarette smoking, alcohol drinking, sedentary lifestyle and relative body weight) significantly varied by educational attainment and annual household income. Those with the least education and lowest income were significantly more likely to be current smokers, overweight and in the lowest quintile for physical activity. Pappas et al (1993) also noted differences in the social distribution of health-risk behavior. They commented that people of higher socioeconomic status tended to adopt healthy lifestyles more rapidly.

Adler et al (1994) state that smoking rates vary inversely with SES. Rates of smoking initiation are also inversely related to SES and rates of cessation are positively related to SES. In addition, among smokers, the number of cigarettes smoked is inversely related to SES.

Lantz et al (1998) noted that education was strongly related to health behaviors, whereas income was more predictive of mortality. Education was related to mortality through its

association with income. Kallan also found that education particularly affected causes of death that have a large behavioral component. However, Lantz et al (1998) found that the effects of smoking and drinking were no longer significant once they were adjusted for demographic, socioeconomic and other health behavior variables.

Adler et al (1994) found that the SES-health association is reduced, but not eliminated, when health behaviors are statistically controlled.

Lantz et al (1998) also concluded that health behaviors and socioeconomic factors are important determinants of mortality, but that they only explain a small proportion of the socioeconomic differences in mortality and are therefore not the primary mechanisms linking socioeconomic status and mortality.

Rogers (1995) found that marital status mortality differentials were predominantly due to behavioral components. Brown and Di Meo (1995) also noted that marriage restricts the extent of unhealthy behaviors such as smoking and alcohol consumption, and that those who are married generally take better care of themselves.

Kark et al (1996) found that belonging to a religious collective was associated with a strong protective effect and that tobacco consumption and fat intake are generally lower in religious kibbutzim. However, there were no significant differences in the lifestyle factors of diet, smoking, obesity, alcohol intake, exercise and exposure to accidents. Similarly, Musick (1996) found that lower levels of unhealthy behaviors were associated

with religiousness, but these behaviors did not mediate the relationship between religion and self-rated health for either blacks or whites.

Vallin (1995) noted that men and women exhibit differences in health behavior and in their attitudes towards their bodies. Women are increasingly large consumers of health services and have benefited from medical progress earlier than men. Trovato and Lalu (1998) suggested that lifestyle and personal behaviors were important in respect of the causes of death relating to the gender gap in mortality.

Wilkinson (1997) suggests that income inequality may effect society in a wider context. He proposes that less egalitarian societies may have higher levels of homicide, accidents and alcohol related deaths, but these have not yet been discerned.

Income

Knox and Tomlin (1997) found that there was very strong evidence to suggest that mortality rates were not equal at different income levels. Kallan (1997) concludes that income and employment status have significant net effects on both all-cause mortality and cause-specific mortality for all age-sex groups, with the exception of older females. Montgomery and Pappas (1996) found that poverty had the strongest consistent effect on children's health. Rogers et al (1999) comment that family income is a primary link between education and mortality. The inverse relation between income and mortality has

been identified, regardless of sex, age, race or marital status, and is apparent even after adjustments for variations in education, occupation, health status and other demographic characteristics. Pappas et al (1993) also found that not only did poor people have higher death rates than wealthier people, death rates fell consistently as levels of income increased. The mortality disparity has increased between 1960 and 1986. Similar findings are reported in Attanasio and Emmerson (2001), Williams and Collins (1996) Sorlie et al (1995), Deaton and Paxson (1999), Wolfson et al and the Statistical Bulletin (1975).

In Judge (1995), Wilkinson states (in the commentary) that income is a determinant and indicator of a wide range of material factors that not only affect standard of living but also have a crucial impact on psychosocial factors such as sense of control, security and status. He highlights that the association between income and mortality is independent of fertility, maternal literacy and education in developing countries and of average incomes, absolute levels of poverty, smoking, racial differences and the provision of medical services in developed countries.

It has been argued that rather than income affecting health, health may impact on income. However, Deaton and Paxson (1999) show that only some of the effect of income was removed when allowing for reverse causality. Wolfson et al (1990) agree.

Deaton (1999) concluded that income exerts a strong protective effect, with permanent income being more protective than current income. Wolfson et al (1990) has similar

findings. In fact, Deaton and Paxson (1999) noted that the effects of income are dynamic, and although permanent income is negatively correlated with mortality, transitory income was positively correlated to mortality and that it positively followed the business cycle. They suggest that long-run income was a better predictor of health than current income. Wolfson et al also found that permanent rather than transitory earnings is the key variable. Thus earnings have long term effects on mortality. Indeed, they found that higher earnings in late middle age were associated with significantly lower mortality two decades later.

Rogers (1995) comments that there are substantial changes in income for those over 65. He argues that the lower mortality rates of married couples may be attributable to the economic advantage of double income. Wolfson et al (1990) found that while there are clear mortality gradients by income within virtually all age ranges, the relationships are not as strong for those over age 65. However, Knox and Tomlin (1997) found that for those aged 60-79 mortality was still related to income, with a trend towards lower mortality rates as income increases. They highlight that the disparities in death rates between high and low income earners were not as pronounced for the older age groups. The disparities diminished markedly after age 74. Deaton and Paxson (1999) found income mattered most in middle age (40-54) and also that the effect diminished with age. Wolfson et al (1990), Williams and Collin (1995), Sorlie et al (1995), and Rogers et al (1999) have similar findings. They highlighted a striking positive correlation between income and mortality among young men, which was not observed for young women.

Both Knox and Tomlin (1997) and Vaillant and Mukamal (2001) find that the mortality and the health status of persons over 80 are not significantly related to pre-retirement income.

Deaton and Lubotsky (2001) state that blacks have higher mortality rates than whites and, on average have lower incomes. Williams and Collins (1995) also comment that gains in economic status have stagnated for blacks. In contrast, Montgomery and Pappas (1996) found that the effect of poverty on children's health did not differ by race.

Rogers et al (1999) comment that those with lower incomes were more likely to be smokers and less likely to quit. However, in their study, income did not alter the effects of smoking significantly. Lantz et al (1998) found that the distribution of four behavioral risk factors (cigarette smoking, alcohol drinking, sedentary lifestyle and relative body weight) significantly varied by annual household income. Those with the lowest income were significantly more likely to be current smokers, overweight and in the lowest quintile for physical activity. However, income was predictive of mortality, rather than strongly related to health behaviors.

Bucher and Ragland (1995) found that income was inversely related to blood pressure, cholesterol and smoking, but none were significant. The correlation of income with age and height was significant. Income was significantly related to mortality from all causes and lung cancer.

Pappas et al (1993) noted that over time absolute death rates declined for people of all income levels, but the reduction was greater for those with higher incomes. This highlighted the increasing disparity in mortality rates for those of different income levels.

Williams and Collins (1996) mention that it has been suggested that mortality rates are not strongly related to a country's overall economic status, but to the level of income inequality within the country.

Several authors also investigate the connection between mortality and income distribution.

Wilkinson (1997) argues that the relation between individual income and mortality is primarily an effect of relative income. He states that absolute income levels are no longer important in the developed world, rather there is a relation with income distribution, whereby health is worse when there is greater inequality across the social gradient. He does acknowledge that within a small area of analysis, income is related to mortality and income distribution is not, due to the lack of heterogeneity in a small area. Kennedy et al (1996) also suggest a significant positive relation between income distribution and life expectancy and infant mortality. These results were robust to adjustments for poverty, median household income and household size. Kennedy et al found that the relation of income inequality to total mortality was higher for the black population but comment that the association between income inequality and mortality is not completely explained and that income distribution may be a proxy for other social indicators.

This last comment is supported by Judge (1995). In this paper, Judge refutes Wilkinson's initial claims that income inequality is the key determinant of variations in average life expectancy among developed countries. He views variations in income distribution to be the product of many influences such as dietary influences, cultural factors and national perceptions of self-esteem, that interact over long periods of times.

In a later paper, Judge et al (1998) reach the same conclusion. However, the authors state that the existence of a link between income inequality and average levels of population health is a plausible proposition and comment that they cannot confidently state that income inequality is not associated with national health. They considered whether changes over time in income inequality and population health are associated with each other and although the coefficients for life expectancy were in the expected direction, none were significant. They highlight that any link between income inequality and health is exposed to the risk of confounding variables.

Deaton (1999) also challenges Wilkinson's hypothesis and analyses. Higher income was shown to lead to lower mortality and higher income inequality was shown to lead to higher mortality. However, the effects were not robust when the mortality was age standardized. Nor did Deaton (1999) find a clear and consistent correlation between inequality and life expectancy when tracking on a consistent basis over a period of years. He concluded that there was no evidence that higher income inequality raises the risk of

dying. He suggests there is a risk of spurious correlations being made with income inequality being a proxy for a wide range of social ills.

In contradiction to the Wilkinson hypothesis, Deaton and Paxson (1999) actually found a protective effect whereby higher inequality is associated with lower mortality.

A further paper by Lobmayer and Wilkinson (2000) found that both higher median income and greater inequality were associated with higher premature mortality. This suggests that wider income distribution is related to higher premature mortality. The results did not extend for ages over 65 and they suggest that this is due to a shift in relative poverty from older people to young families with children. They conclude that there may be a large impact of income inequality on younger lives and that combining all ages in life expectancy masks this.

The paper notes that the USA, having higher and more unequal incomes, was an outlier highlighting that the USA is very different from the other OECD countries. A similar result was found in Judge et al (1998).

In contrast to earlier papers, Deaton and Lubotsky (2001) showed that income inequality is correlated to mortality for both blacks and whites separately. The fraction of the population that is black was found to be positively correlated with average white incomes, and negatively correlated with average black incomes. Once the fraction black was controlled for, there was no relationship between income inequality and mortality for

the whole population or for whites. This implies that the component of income inequality that matters for mortality is income inequality between races, not income inequality within them. They concluded that conditional on racial composition, income inequality does not raise the risk of mortality. They highlighted that the effects of inequality are inconsistent and more often estimated to be protective than hazardous.

Attanasio and Emmerson (2001) could not determine whether the relevant concept is relative or absolute wealth.

Marital Status

Rogers (1995) found that marital status differentially affects mortality, with married people consistently exhibited lower mortality than those who are not married. The same result was reported in Hurd et al (2001), Kallan (1997), Sorlie et al (1995) and Brown and Di Meo (1995). In contrast, Attanasio and Emmerson (2001) found that, marital status was not significant for mortality or morbidity. Rogers (1996) found that marriage was protective, but only for people in certain kinds of families.

In the accompanying literature review to Brown and Di Meo's (1995) paper, socioeconomic status, gender, education and income are discussed as factors that are correlated with both marital status and mortality.

In Kallan (1997), males were found to benefit more from marriage. Rogers (1995) and Brown and Di Meo (1995) supported this view, but Hurd et al (2001) found no differential effect of marital status for men compared with women. It should be noted that the subjects in Hurd et al's study were all aged over 70. However, the explanations for the gender differences varied. Rogers (1995) suggested that marriage might be more psychologically constraining for women, whereas Brown and Di Meo (1995) found marriage offers protection to males through social support, but for females it is mainly through economic factors as the lifestyle change is less significant for females.

Kallan (1997) found the lower risk of all-cause mortality for married people was especially strong in the younger ages. Sorlie et al (1995) agreed with this finding and suggested that some of the association was due to economic variables, particularly for women. However, although those under 65 who lived with others had lower mortality than those who lived alone, for those over 65, women who lived alone had lower mortality, whereas men living alone had higher mortality.

Brown and Di Meo (1995) state that generally, there are two reasons proposed for these mortality differentials: the selection of marriage theory and the theory of social support. The selection of marriage theory suggests that the physically and psychologically unhealthy are predisposed to higher mortality and are also less likely to be chosen into marriage. The effect of the selection theory is likely to be small and difficult to quantify. The theory of social support suggests that the obligation, expectations and responsibilities of marriage lead to a certain level of protection. This implies that marriage leads to a

different lifestyle due to the reduction in unhealthy behaviors and the increased means for social integration.

Rogers (1995) also proposed that the mechanisms for marital mortality differentials were superior integration into society, natural selection and psychological and lifestyle protection. He comments that little difference has been shown to be due to selection, rather differences are predominantly due to social or behavioral components.

Rogers (1995) found that gender interacts with mortality for overall mortality and cause-specific mortality with the mortality gap being greatest for social pathologies and diseases that require health regulation, suggesting marriage protects individuals by focusing attention on health, reducing risk and increasing medical compliance. Brown and Di Meo (1995) also found that death rates based on social and psychological factors were lower in married people while there was no significant difference between marital states with regard to mortality which is largely unaffected by social factors. However, Rogers et al (1999) found that the effects of smoking were not altered significantly by marital status.

Rogers (1995) also found that the lower mortality of married people did not exist in a social vacuum. He noted that marital status and income both influence mortality. Similarly, Kallan (1997) suggested that the total effects of marital status operate partly through income and employment status.

As mentioned in the first paragraph, Rogers (1996) found that marriage was protective, but only for people in certain kinds of families. In addition to married couples, previously married individuals who lived with their young children and never-married individuals, who lived alone or who lived with other relatives, were found to enjoy low mortality. Indeed, married individuals who lived with other relatives experience high mortality, as did previously married individuals if they lived alone, with older children or others.

Rogers (1996) also comments that mortality is affected by family composition and health and also informal and formal social support. He showed that death was less likely for those with social ties and that contacts with relatives, friends and the community reduced mortality and were as, if not more, important than marital status.

Montgomery and Pappas (1996) found that mortality is tangentially related to marital status, in that they found that family structure was one of a number of factors that affected the health of children. Children in families headed by single mothers were much more likely to be in poor or fair health compared with children in two-parent families. Although the risk was reduced when adjusting for race and poverty, it remained highly significant.

Rogers (1995) suggests that family structure among racial and ethnic groups is determined as much by economic as by cultural factors.

Vaillant and Mukamal (2001) found that one of the most important predictors of successful aging was having an extended family network. This would support the above findings of lower mortality for married people and certain family structures.

Obesity

Allison et al (1999) found that more than 80% of the estimated obesity-attributable deaths occurred among individuals with a BMI¹ of more than 30. The hazard rates generally increased with BMI, although this was not consistent until the BMI was in the upper 20s. Wei et al (1999) found that, compared to normal-weight men, obese men had an almost 3-fold higher risk of cardiovascular disease mortality and a 2-fold higher risk of all-cause mortality. Overweight men had intermediate death rates between normal-weight and obese men. In contrast, Vaillant and Mukamal (2001) found body mass index to be only marginally significant.

Allison et al (1999) found that in the higher BMI categories, hazard rates tended to be higher in non-smokers or never-smokers, however, over all categories the difference was slight and inconsistent. However, Wei et al (1999) comment that men who were overweight or obese were also more likely to have baseline disease, smoke cigarettes, be sedentary and have a family history of cardiovascular disease. Contrary to both of these,

¹ BMI (Body Mass Index) is calculated as weight (in kilograms) divided by height (in metres) squared. The unit of measurement is kg/m².

Rogers et al (1999) comment that being underweight or overweight increased the effects of smoking on mortality.

Wei et al (1999) found that low cardio-respiratory fitness was a strong and independent predictor of cardiovascular disease and all-cause mortality and is an important risk factor that, along with overweight and obesity, adversely influences mortality. They note that cardio-respiratory fitness has a genetic component, which explains 25% to 40% of the mortality variation in fitness levels. However, habitual physical activity is the other major determinant of fitness.

Lantz et al's (1998) found that those with the least education and lowest income were significantly more likely to be current smokers, overweight and in the lowest quintile for physical activity. However, the effects of being overweight were not significant once it was adjusted for demographic, socioeconomic and other health behavior variables.

Allison et al (1999) refer to another paper by Stevens et al which found that hazard ratios for obesity decreased steadily with advancing age. They also comment that there is continued growth in the population proportion that is obese and severely obese.

Occupation

Lee (1995) suggests that occupation should be the core socioeconomic variable as it reflects educational attainment and income and also highlights the economic structure of a country. Sorlie et al (1995) also found that employment status showed the largest correlation with mortality. They suggested this relationship could be due to both economic and health factors. In contrast, the Statistical Bulletin (1975) stated that occupation, education and income are associated with health and longevity, but highlighted that the interdependence of these factors mean that the effect of any one is affected by the presence of the others. Lee (1995) acknowledges that additional supplementary information is required in order to include those not in the labor force.

Williams and Collins (1995) highlight that the unemployed have higher mortality rates. Similarly, Vallin (1995) found that the risks of dying are much higher among the economically inactive than among the total working population and that this applied to males and females.

Rogers et al (1999) comment that employment status influences mortality as working not only provides income, but other benefits such as health insurance, social relations and camaraderie. The Statistical Bulletin (1975) notes that same inverse relationship exists for infant mortality and for married females indicating that it is not only occupation, but also lifestyle and living environment that account for mortality differentials.

Rogers et al (1999) comment that specific occupational status can also have an impact on mortality. In their study, Sorlie et al (1995) found that professionals were found to have the lowest relative mortality, but adjustments for other variables reduced the occupational effects considerably. Williams and Collins (1995) also comment that there are also differences in occupational exposure to hazards, with low SES persons more likely to be in occupations with an elevated risk of exposure.

Vallin (1995) found that the occupational structures for men and women are quite different, with respect to physically demanding jobs and high mortality groups. However, his study determined that sex differences in socio-occupational structure do not play a central role in promoting excess male mortality. Men and women have different health behavior, which contributes to the widening mortality gap.

Brown (1997) mentions that there are indications that at advanced ages mortality does not vary by work history.

Race and Ethnicity

Kallan (1997) found race had significant effects on all-cause mortality among all age-sex groups. Hummer (1996) highlights that there are a host of health and mortality measures that mark the severe disadvantages experienced by the African American population in comparison to non-Hispanic whites. Williams and Collins (1995) state that the racial and ethnic differences in health are potent predictors of variations of infant mortality and age-

adjusted death rates in the U.S. They highlight that blacks generally have the highest mortality rates, followed by whites and Hispanics have the lowest rates. Elo and Preston (1997) also note that Hispanics and Asian and Pacific Islanders have lower mortality than whites.

Hummer (1996) comments that the black-white gap remains wide. However, Williams and Collins (1995) suggest that in recent years, black health has worsened with the gaps in life expectancy, infant mortality and rates of sexually transmitted diseases increasing. They also mention that gains in economic status have also stagnated for blacks.

Kallan (1997) found the effects of race on mortality were weaker at older ages. However, Sorlie et al (1995) found that blacks under age 65 had significantly higher mortality rates than Whites, but Blacks over 65 had similar or even slightly lower mortality than Whites. This was supported by Elo and Preston (1997), who commented that black and white death rates converge at older ages and some data sources show a crossover. Kestenbaum (1997) also found that mortality differentials among the extreme aged by race, narrowed with increasing age. He reports that black-white mortality crossovers occurred at ages 88 for males and 87 for females and states that these crossovers are not artefacts of excessive age mis-reporting. The data also displayed a Hispanic-black crossover for women and both Hispanic-black and Hispanic-white crossovers for men.

Williams and Collins (1995) state that the age patterning of racial disparities suggests that age may be a proxy for the cumulative exposure to racism and adverse living conditions.

The three most common explanations for black-white health and mortality differentials are racial/genetic, cultural/behavioral and socioeconomic.

Hummer (1996) states that there is little evidence that the racial population distribution of genes accounts for the numerous health and mortality differences exhibited between blacks and whites, and Williams and Collins (1995) state that explanations for racial differences based on biological or genetic factors have been scientifically discredited. They suggest interactions between biological and environmental variables are important.

The cultural behavioral approach, as detailed in Hummer (1996), suggests that different values, beliefs, attitudes, traditions and lifestyles are the important factors. However, whilst these factors reduce the difference, they do not close the gap and should be placed in the context of their partial dependence on social, economic and historical circumstances.

Hummer (1996) states that the socioeconomic approach considers the higher risks associated with poor education, poverty and unemployment. The Statistical Bulletin (1975) also notes that in the US, the mortality gradient by social class is affected by the high correlation between race and social class with non-whites being concentrated at the lower end of the social class scale. Williams and Collins (1995) suggest that racism may also transform social status, such that SES indicators are not equivalent across race, restricting access to health-related services and inducing psychological distress.

Deaton and Lubotsky (2001) state that blacks have higher mortality rates than whites and, on average, have lower incomes. Sorlie et al (1995) found a small interaction between race and employment status, but did not find that race interacted significantly with income, education or marital status.

LeClere et al (1997) state that social disadvantage does not completely explain the higher age-specific mortality differentials experienced by non-whites. This is particularly the case for Hispanics. Williams and Collins (1995) also note that race is strongly correlated with SES but the differentials in health status associated with race are smaller than those associated with SES. Similarly, in examining if the effects of race/ethnicity can be explained by other socioeconomic variables, Kallan (1997) found a nontrivial residual race effect.

Williams and Collins (1995) comment that evidence suggests smoking is increasingly concentrated among the lowest socioeconomic groups and minority populations. They also highlight that adverse changes in health behaviors of blacks have had a profound adverse effect on their health. However, Rogers et al (1999) comment that non-Hispanic Whites are more likely to have smoked or to smoke a larger number of cigarettes than Blacks or Hispanics. Despite this, their study found race to have a negligible impact on the effects of smoking.

Musick (1996) mentions that there are racial variations in religious activity, however none of the effects for religion, health, health behavior or social interaction variables were found to be different for Blacks and Whites.

Hummer (1996) considers multiple forms of racism to be crucial sociological determinants of racial health and mortality differentials. He proposes a conceptual framework whereby race is an important social determinant of health and mortality, operating through three primary pathways and five intervening sets of factors. The three primary pathways are institutional forms of racism, racial socioeconomic stratification and individual-level forms of racism and the five intervening factors are health care, physical environment, health and coping behaviors, stress and social roles and social support. These are the mechanisms through which the primary pathways can affect health and they may operate additively, interactively or even counter one another.

Hummer (1996) highlights the problems associated with residential segregation, racial isolation and political under-representation. Williams and Collins (1995) found that the concentration of black poverty in inner cities had a profound adverse effect on health. LeClere et al (1997) also suggest that residential segregation influences individual mortality, but in opposite ways for African Americans and Hispanics although the effects were not as dramatic for Hispanics. However, neighborhood linguistic isolation and mobility did not affect mortality risk. LeClere et al (1997) noted the effects are larger for men than women and the mechanisms also differed by gender. Neighborhood income and ethnic concentration affected male mortality risk, but it was suggested that physical and

financial stress increased female mortality although the educational attainment of neighbors protected all women. LeClere et al (1997) comment that although individual characteristics were robustly related to mortality, the community was also found to affect individual mortality risk. Williams and Collins (1995) also highlight that low-SES people and racial minorities are also more likely to be exposed to environmental risks in residential environments.

Kestenbaum (1997) states that, in general, death rates for Hispanics in all age groups are below comparable rates for both non-Hispanic whites and non-Hispanic blacks. Williams and Collins (1995) highlight that the Hispanic population, despite having low SES and low rates of utilization of medical care, has rates of infant mortality and overall mortality lower than blacks and comparable to whites. They suggest that the foreign-born have a better health profile, or that traditional Hispanic cultural factors enhance the lives of Hispanic immigrants, but these have not been clearly identified. Elo and Preston (1997) also comment on the gap between Hispanics and whites and noted that this increased with age. However, the Asian/Pacific Islander advantage was larger at younger ages. They also considered the lower mortality to be due to the low mortality of the foreign-born, but suggested this could either be due to a healthy migrant effect or omission of these deaths.

Kennedy et al (1996) suggested a relation between income distribution and life expectancy and that this relation was higher for the black population.

Deaton and Lubotsky (2001) also considered inequality and obtained a different intervening factor.

Deaton and Lubotsky (2001) show that income inequality is correlated to mortality for both blacks and whites separately. The fraction of the population that is black was found to be positively correlated with average white incomes, and negatively correlated with average black incomes. Once the fraction black was controlled for, there was no relationship between income inequality and mortality for the whole population or for whites. This implies that the component of income inequality that matters for mortality is income inequality between races, not income inequality within them. They concluded that conditional on racial composition, income inequality does not raise the risk of mortality but that the fraction of the population that is black is a significant risk factor for mortality, for the population as a whole and for blacks and whites separately. Deaton and Lubotsky (2001) could not determine why the fraction black was associated with higher white mortality. They did find that the effect was robust to conditioning on education, it was present for almost all age groups, within all geographical regions of the US and not attributed to variations in local public health provision or income inequality.

Elo and Preston (1997) found that data for estimating adult and old age mortality in the U. S, were more reliable for whites in all cases. There are also problems of inconsistencies in race classification. Kestenbaum (1997) comments that there is some support for the premise of net understatement of age on death certificates with age agreement among blacks much poorer than among whites. He states that the case for

Hispanics is slightly more complex due to poorer record keeping in the past. Williams and Collins (1995) also note that there are reliability and validity problems with the measurement of race and ethnicity, particularly between interview-observed race and respondent self-report. The classification of people of mixed race is an increasingly important issue. In a footnote, Elo and Preston (1997) comment that an increase in inter-racial marriages raises the potential for inconsistent race reporting for the offspring of such marriages. Such inconsistencies do not affect the results they discussed, but should be a consideration in future studies.

Religion

Kark et al (1996) found that belonging to a religious collective was associated with a strong protective effect. Musick (1996) used subjective, i.e. self-rated health as an indicator of mortality and found that religious activity was found to have modest main and interactive effects on subjective health. The effects were greatest for those suffering from physical health problems, emphasizing the comfort role of religion.

Kark et al (1996) comment that the protection from religion was not attributable to confounding by socio-demographic factors. The protection was found to be the result of collective attributes of religion rather than by individual religiosity. Religion resulted in a less stressful social environment that promoted overall well-being and a positive health status. Musick (1996) found that church attendance had a significant positive effect on

subjective health. However, when functional impairment or chronic conditions were considered, church attendance became insignificant but private religious devotion increased to significance. This would imply that failure to control for health status would conceal the effects of these activities. Musick's study did not find that social interaction and satisfaction from social interaction lessened the relationship between church attendance and subjective health.

Kark et al (1996) found there was also a stronger protective effect of religiousness in women than in men.

Musick (1996) mentions that there are racial variations in religious activity, however none of the effects for religion, health, health behavior or social interaction variables were found to be different for Blacks and Whites.

Although Kark et al (1996) noted tobacco consumption and fat intake are generally lower in religious kibbutzim, there were no significant differences in the lifestyle factors of diet, smoking, obesity, alcohol intake, exercise and exposure to accidents. Musick (1996) also found lower levels of unhealthy behaviors were associated with religiousness, but these did not mediate the relationship between religion and self-rated health.

Kark et al (1996) highlight that they compared religion with non-religion and comment that the effects of religion on mortality may actually vary according to the faith or denomination.

Smoking

Kennedy et al (1996) found smoking to be an independent predictor of total mortality and deaths from cancer. Thun et al (1997) commented that smoking approximately doubles the risk of death. Rogers et al (1999) also found the odds of dying for both heavy and light smokers were about twice that of those who never smoked. This also applied to those who quit smoking but at a late stage. These results were robust to controls for demographic, socioeconomic and other behavioral factors. These conclusions support Vaillant and Mukamal (2001) who state that the absence of cigarette abuse was one of the most important protective factors for successful aging.

Rogers et al (1999) comment that although all smoking categories had significantly higher odds of dying compared with non-smokers, the highest were for current heavy smokers. The mortality of former smokers depended on the volume of their previous smoking and whether they quit before or after contracting a serious illness.

There were high odds ratios of deaths from a large number of diseases, but circulatory diseases was by far the disproportionate underlying cause of death associated with smoking. This may reflect the fact that circulatory disease is a very common cause of death. Smokers are also more likely to have multiple causes of deaths. These findings were largely independent of age and sex.

Women were shown to be less likely to be smokers or have smoked at all, and those that did, smoked fewer cigarettes than men. Rogers et al (1999) suggested that smoking is a major contributor to the current sex gap in mortality.

Rogers et al (1999) comment that earlier research stated that non-Hispanic Whites are more likely to have smoked or to smoke a larger number of cigarettes than Blacks or Hispanics. However, they found that race had a negligible effect on the effects of smoking.

Williams and Collins (1995) states that evidence suggests smoking is increasingly concentrated among the lowest socioeconomic groups and minority populations. Although earlier research showed that those with less education and lower incomes were more likely to be smokers and less likely to quit, in Roger et al (1999) the socioeconomic factors of education and income did not alter the odds significantly. This study also found that marital status had a negligible impact on the effects of smoking.

There is some suggestion that smoking is related to other health behaviors. Thun et al (1997) commented that alcohol is positively correlated to cigarette smoking, but Rogers et al (1999) found that high-risk smokers were not very likely to be high-risk drinkers, nor did alcohol have any significant impact on smoking patterns. Allison et al (1999) found that mortality hazard rates generally increased with BMI². However, in the higher BMI categories hazard rates tended to be higher in non-smokers or never-smokers.

² BMI (Body Mass Index) is calculated as weight (in kilograms) divided by height (in metres) squared. The unit of measurement is kg/m².

Rogers et al (1999) also found that high-risk smokers were not abnormally overweight. However, being overweight or underweight increased the effects of smoking on mortality.

Lantz et al (1998) found that the distribution of four behavioral risk factors (cigarette smoking, alcohol drinking, sedentary lifestyle and relative body weight) significantly varied by educational attainment and annual household income. Those with the least education and lowest income were significantly more likely to be current smokers, overweight and in the lowest quintile for physical activity. However, they found that the effects of smoking were no longer significant once they were adjusted for demographic, socioeconomic and other health behavior variables. This contradicts Roger et al's (1999) final conclusion as mentioned in the first paragraph.

Wald and Watt (1997) considered smoking from a slightly different perspective. They found that all smokers have a greater risk of lung cancer than lifelong non-smokers. They also found that those who switch from cigarettes do not achieve the lower risk of pipe and cigar smokers who have never smoked cigarettes. Pipe and cigar smokers smoked less tobacco than cigarette smokers, but former cigarette smokers who had switched inhaled tobacco to a greater extent and therefore were at a higher risk. It was shown that men who had stopped smoking cigarettes for at least 20 years had death rates similar to lifelong non-smokers.

Similarly, Rogers et al (1999) concluded that as well as smoking being a crucial mortality risk, the volume of cigarettes smoked also has a significant impact. They suggested that other aspects of smoking status, e.g. age at initiation, age at quitting, type of cigarettes smoked and extent of inhaling may have an effect, but they were not captured.

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