Estimating the Effects of Smoking and Other Risk Factors on Mortality Slowdowns in Developed Countries

Atttention has been directed recently to observed slowdowns in gains in life expectancy in recent decades in the United States and some other developed countries, particularly for females at older ages, even as increases in life expectancy have continued to be robust in other countries (Meslé and Vallin, 2006; Ezzati et al., 2008). The effects of previous trends in cigarette smoking on these results merit considerable attention, given that decades of medical and epidemiological research have demonstrated that cigarette smoking is the largest cause of preventable mortality in the U.S. and many other developed countries (Rogers et al., 2005). One of the most common means of measuring the effects of cigarette use on mortality is the indirect method presented by Peto et al. that uses observed lung cancer mortality in a population as a proxy for previous smoking use and then estimates the overall mortality attributable to smoking based on the relative risks of mortality for various causes for smokers and nonsmokers (1992). This study modifies and extends this method in order to estimate mortality attributable to smoking for females at older ages and thus better understand the effect of previous cigarette use on recent differences between countries in female old-age mortality. In doing so, it introduces an adjustment factor into use of the indirect method to account for low levels of mortality in its study population, and this adjustment factor is shown to be useful in this particular case and advantageous more generally in the estimation of mortality from smoking.

Use of the indirect method as implemented here indicates that smoking exposure accounts for approximately half of the difference in e65 for females in

the U.S. and some other developed countries. With regard to the remaining differences, mixed effects models using longitudinal data on smoking exposure and other mortality risk factors suggest that dietary factors, in particular animal fat consumption, are the most easily identifiable cause, a conclusion that is similar to previous findings with regard to excess male mortality in many of the same countries in a previous period. Finally, differences in economic conditions, such as per capita GDP and income inequality, between countries do not appear to account for much of the remaining difference in life expectancy.

Research in the Field

Various researchers in the fields of demography and epidemiology have observed recent differences in mortality trends in older age groups in several developed countries, particularly for females. Meslé and Vallin, for example, noted that life expectancy at age 65 for females increased only slightly from 1984 to 2000 in the Netherlands and the United States after a period of more robust gains from 1968 to 1984, in contrast to trends in France and Japan where e65 values for females increased steadily in both periods. (2006). The authors discussed the possibility that reductions in age misreporting over time as well as aspects of the U.S. health system, such as the lack of preventative care and prescription drug coverage in the Medicare program, could account for some portion of these results for the U.S. and that attitudes toward end of life care at advanced ages could contribute to this trend in the Netherlands. Similarly, Janssen, Mackenbach,

and Kunst found slowing mortality declines for men and women above the age of 80 in the Northern European countries of Denmark and the Netherlands that they concluded were not due to cigarette smoking as well as continued strong mortality declines for men and women above this age in other European countries, particularly France (2004), although they concluded in a subsequent paper that smoking did have an important effect on old-age mortality decline (2007).

Another, more extensive, body of literature addresses the effect of cigarette smoking on mortality trends for all ages. Majid Ezzati and Alan D. Lopez estimated that smoking caused approximately 5 million deaths worldwide in 2000, roughly divided in half among developed and developing countries, using an indirect method proposed by Peto et al. that uses lung cancer mortality as a proxy for smoking exposure (2003). The Centers for Disease Control and Prevention estimated that smoking caused around 400,000 deaths in the U.S. each year from 1997 to 2001, a figure found by multiplying the number of deaths in disease categories by the proportion of deaths attributable to smoking for each of these disease groups (Centers for Disease Control and Prevention, 2005).

Numerous studies have shown that previous trends in cigarette smoking help explain changes in differences in mortality rates and life expectancy by sex within countries. Pampel, for example, used estimates of mortality rates attributable to smoking and not attributable to smoking calculated by Peto et al. using the indirect method for males and females in broad age groups in 21 high-income countries to examine the mortality differential by sex in these

countries (2002). He classified the countries into three groups based on their mean logged ratio of male to female mortality rates, with one group consisting of countries such as Japan, France, and Spain in which the ratio increased over time from 1975 to 1995, another group including countries such as Canada, Australia, and Italy in which the ratio remained about the same during the period, and a third group including the U.S., the U.K., the Netherlands, and Denmark in which the ratio decreased. He also showed that the changes in these ratios in each of these groups were largely due to changes in the ratio of logged mortality rates attributable to smoking, particularly for ages 35 to 69, thus indicating that changing smoking exposure among males and females accounted for much of the changes in the overall mortality differential by sex in these countries.

Preston and Wang likewise found that sex differentials in mortality rates in the U.S. at older ages in the second half of the twentieth century were largely due to cohort effects related to smoking (2006). They showed using U.S. vital statistics data that the direction of sex differences in mortality rate change for ages from 50 to 84 in the period from 1948 to 2003 changed on a cohort basis, with the sex differential increasing for every birth cohort group up to and including cohorts born between 1903 and 1908 and then decreasing for subsequent cohort groups. They then presented data from three sources that indicate that the main cause of this cohort effect was changes in cigarette smoking. First, they presented results from a 1955 national smoking survey that found that the difference between males and females who had become regular smokers by the

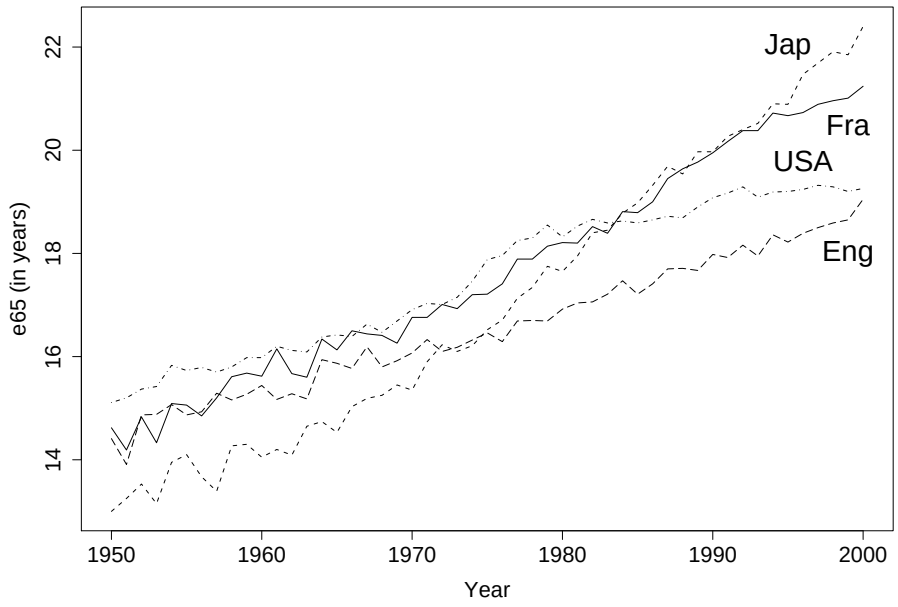
age of 35 peaked for cohorts born in the 1890s and 1900s at around 45% and then subsequently declined. Second, they showed using detailed reconstructions of smoking histories from National Health Interview Surveys that the greatest cohort difference for males and females in the mean number of years spent as cigarette smokers before the age of 40 again peaked for the birth cohorts of 1895 to 1899 and 1900 to 1904. Finally, they again used U.S. vital statistics data to show that the difference in male and female lung cancer mortality was greatest for cohorts born between 1903 and 1908, the same cohorts for which the sex differential in all-cause mortality was greatest. Preston and Wang then used an age-period-cohort smoking history model that included age and period effects as well a term for the mean number of years that members of birth cohorts by sex had smoked before the age of 40. Using this model, they estimated that the difference by sex in the increase in mortality due to smoking peaked with the 1900 to 1904 cohorts. The authors concluded by predicting that the mortality differential by sex for cohorts at older ages in the U.S. would narrow in future decades due to increases in smoking by women and decreases in smoking by men during the second half of the twentieth century.

Differences in Life Expectancy over Time and between Countries

Recent slowdowns in gains in life expectancy for females at older ages can be observed in various developed countries. Figure 1 presents e65 values for fe-

males over time for England and Wales, France, Japan, and the United States from the Human Mortality Database (2009). The data show that the U.S. performed rather well in female mortality trends at older ages compared to other developed countries prior to 1980, but that gains in e65 for females slowed in this country after this time. Reductions in mortality for older women appear to have slowed in a somewhat earlier period for England and Wales, although by the end of the period the gap in e65 between the U.S. and England and Wales had narrowed. Similar slowdowns in gains in female e65 occurred in some other developed countries, including Denmark and the Netherlands according to HMD data (2009).

Figure 1: Life Expectancy at Age 65, Females in Various Countries



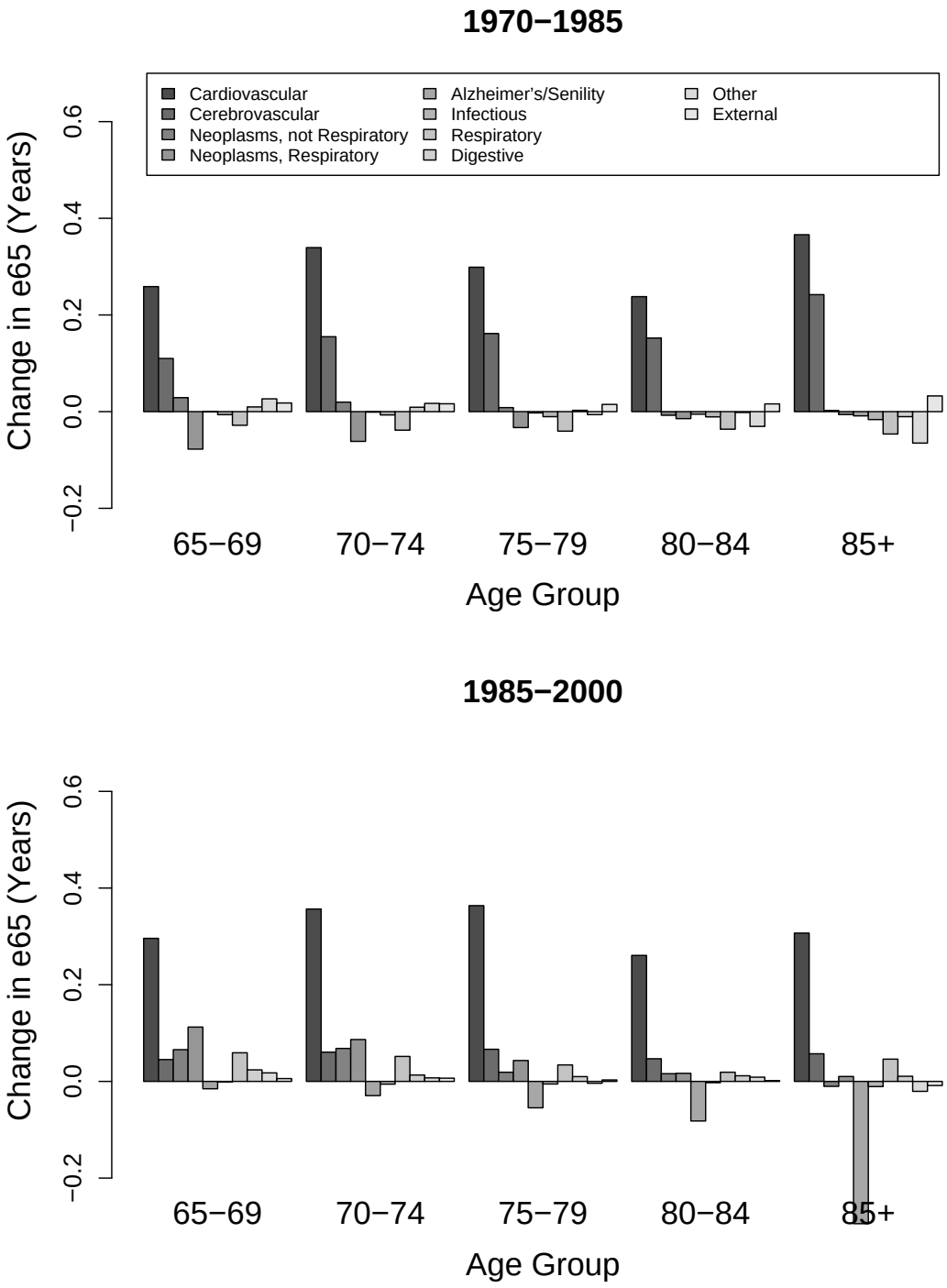
Source: Human Mortality Database

These differences in life expectancy between countries can be better understood by decomposing them by age group and cause. Figure 2 presents results from the decomposition of changes in life expectancy for females in the United States from 1970 to 2000 by age group and cause produced using the method presented by Arriaga for discrete age groups (1984) and mortality by cause of death data available in the World Health Organization Mortality Database (2009a). Table 1 provides codes from the relevant revisions of the International Classification of Diseases (World Health Organization, 2009b) for the diseases in each class of causes. These ten classes of causes are similar to the seven classes of causes used by Christine Himes in her analysis of mortality by cause of death in Japan, Sweden, and the U.S. (1994) as well as the eight classes used by Meslé and Vallin in their analysis (2006).[1]

The plots indicate that large decreases in mortality from cardiovascular and cerebrovascular diseases in the period from 1970 to 1985 as well as decreases in mortality from cardiovascular disease from 1985 to 2000 contributed to gains in female e65 in the U.S. during these periods. The figure also indicates an increase in observed mortality rates in Alzheimer's disease and senility in the open-ended age group for U.S. women from 1985 to 2000, a change that is probably due in some part to increased classification of deaths in this age range as being due to Alzheimer's disease during the period, as has been suggested previously (Meslé and Vallin, 1996), in addition to some increase in mortality itself from this cause.

Decompositions of differences in life expectancy between countries can help

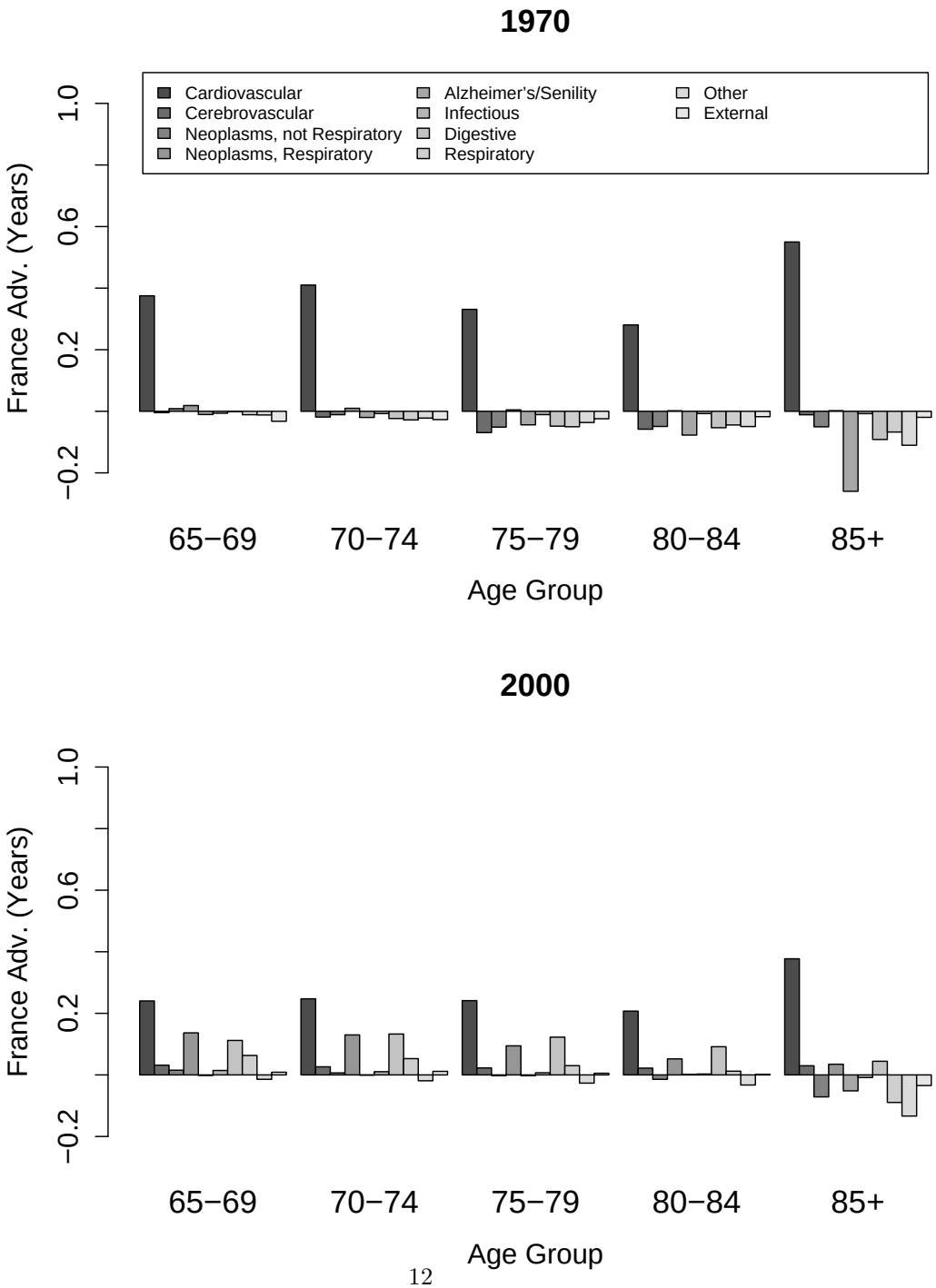
Figure 2: Decomposition of Changes in e65, USA Females by Age and Cause



Source: WHO Mortality Database data

clarify the nature of this increase in mortality attributed to Alzheimer's disease and senility. Figure 3 presents the decomposition of differences in e65 values for females in France and the U.S. in the years of 1970 and 2000 by age group and cause. The figure shows that France maintained an advantage over the U.S. in mortality trends from cardiovascular disease for females at older age groups in both years, some portion of which may be attributable to differences in classification of causes of death given that various studies have suggested that deaths from some cardiovascular causes such as ischaemic heart disease are under-reported in countries such as France and Japan relative to other developed countries such as the U.K. and the U.S. (Murray and Lopez, 1997), but that France also gained an advantage in life expectancy due to mortality from other classes of causes over the period. The figure also graphically shows a convergence in mortality trends by the end of the period for the class of Alzheimer's Disease and senility for females at oldest ages in France and the U.S. This trend can be explained by the decrease in classification over time of large numbers of deaths due to senility in France, which in the 1950s and 1960s was a commonly cited cause of death at older ages in that country, and increases in classification of deaths to Alzheimer's Disease in the U.S. during the period, and indicates that the increase in mortality attributable to Alzheimer's Disease in the U.S., although an important trend in mortality at older ages in that country, is not as important in explaining differences in life expectancy overall or by cause at older ages between the U.S. and other developed countries.

Figure 3: Decomposition of Differences in e65, France and U.S. Females by Age and Cause



Source: WHO Mortality Database data

The Effect of Cigarette Smoking on Mortality Trends

Mortality from lung cancer increased for females at older ages in the U.S. in the second half of the twentieth century, as suggested by Figure 2, an increase largely explainable by previous increases in cigarette smoking among women at younger ages in that country, given that excess lung cancer mortality is largely attributable to smoking. Smoking trends could also explain other mortality trends within and between countries, given that women began smoking in large numbers in various countries at different points in time. As an example of such trends, national surveys show that the prevalence of smoking among women ages 25 to 29 was around 50% in the U.K., 45% in the U.S., 15% in France, and less than 10% in Japan around 1965. By 1990, smoking prevalence in this age group was approximately 35% in France and the U.K., 30% in the U.S., and 20% in Japan (Forey et al., 2002).

The effect of cigarette smoking on life expectancy at older ages can be estimated from vital statistics data using a method suggested by Peto et al. (1992). Pampel provided a useful summary of the procedure as well as a review of criticisms of its methodology and support from some empirical studies of its validity as an estimate of mortality from smoking (Pampel, 2005; Sterling et al., 1993; Valkonen and van Poppel, 1997).

The method suggested by Peto et al. was implemented in this study by first estimating the standardized cigarette smoking exposure in a country by year

for sex and age groups. This estimation was done by calculating the proportion of smokers and nonsmokers in a group that would produce the observed lung cancer mortality rates for that group in that year. It was assumed that smokers and nonsmokers by group had the same lung cancer mortality rates as their counterparts in the American Cancer Society's Cancer Preventive Study-II, a large prospective cohort study that included more than one million adults in the United States in the mid-1980s. This standardized calculation of exposure can be expressed as:

$$M_{lc} = P \cdot M_{lc}^{S} + (1 - P) \cdot M_{lc}^{N}, \tag{1}$$

where M_{lc} is lung cancer mortality for a national population, P is proportion exposed as smokers in that population, and M_{lc}^{S} and M_{lc}^{N} are lung cancer mortality for smokers and nonsmokers in a study population, in this case the CPS-II study. Peto et al. calculated mortality rates for nonsmokers from data for individuals who reported at the beginning of the CPS-II study that they had never smoked regularly. They calculated rates for smokers from data for individuals who reported that they were current smokers at the beginning of the CPS-II study. Peto et al. noted that most of these current smokers were lifelong adult smokers who smoked on average approximately 20 cigarettes a day.

Smoothed mortality rates for lung cancer obtained from the data presented in the appendix of the paper by Peto et al. were used in this study to estimate smoking exposure, similar to the procedure used in the original paper, although

in this case exposure was consistently estimated for five-year age groups through 75-79. Smoking exposure was also estimated for the age groups of 80-84 and 85+, calculated from the data in the appendix of the paper for those 80+ with the lung cancer mortality rates for smokers and nonsmokers assumed to be the same in both groups, even though Peto et al. attributed the same proportion of mortality to smoking for those 80+ as was estimated for those 75-79 because of concern about the reliability of lung cancer mortality rates at older ages. More specific estimates of smoking exposure for older age groups were calculated here because of the particular attention being directed to mortality at older ages as well as because of the changes in smoking prevalence that occurred over time for cohorts leading to appreciable differences in smoking exposure for different age groups at advanced ages. Population groups for which the observed lung cancer mortality rates were lower than the incidence of lung cancer mortality among their CPS-II nonsmoker counterparts were assumed to have had no smoking exposure.

Lung cancer mortality rates among nonsmokers have tended to be rather consistent during the period in the U.S., according to results from the ACS CPS-I and CPS-II (Thun et al., 2006), thus making possible plausible estimation of smoking exposure for these years. Moreover, the period of estimation is similar to the interval for which Peto and his colleagues estimated the effects of smoking on mortality for various developed countries using their method (Peto et al., 1994).

Table 2 provides estimates of standardized smoking exposure for females in

four developed countries by age groups in 1955, 1970, 1985, and 2000. Standardized smoking exposure may not be accurately estimated for the youngest age groups in some countries, particularly those with relatively small populations, an imprecision resulting in part from the low levels of lung cancer generally found among smokers and nonsmokers at these ages. Exposure may also be somewhat overestimated for the oldest age groups in some countries because of variation in lung cancer mortality rates and classification at advanced ages in populations as well as because of the lack of specific mortality data for age groups above 80 from the CPS-II study data. This overestimation is most apparent for those 85+ in Japan in 2000.

In general, trends in estimated standardized smoking exposures for cohorts in these and other developed countries are generally consistent with survey results. Smoking exposure as measured here by the indirect method does tend to be higher for females in the U.S. and some other developed countries than self-reported prevalence figures for cohorts in published compilations of smoking statistics (Forey et al., 2002). To some extent, variation in results from the two methods may occur because of differences in the way in which smoking exposure is measured, given that survey data often provide an estimate of prevalence of some form of smoking at a particular time whereas the indirect method attempts to estimate cumulative exposure to smoking as measured by its impact. On the other hand, these differences may also result from certain characteristics of the indirect method's study population, given that the method standardizes smoking exposure based on the mortality of CPS-II nonsmokers and smokers

by age and sex group and that the overall mortality rates for smokers and nonsmokers in the CPS-II were, as noted by Peto et al. and others (Sterling et al., 1993), lower than those of the U.S. population for these age groups, perhaps because of a tendency of people in good health or concerned about their health to participate in such a study.

It is possible that these low mortality rates among participants in the CPS-II study population could lead to overestimation of exposure to smoking, and consequently to overestimation of the mortality effects of smoking, when these rates are applied to national population data, as has been suggested (Wilmoth, 2008). The indirect method assumes, as expressed in Equation 1, that lung cancer mortality by age and sex group in a population is a function of exposure to smoking and the mortality of smokers and nonsmokers from that cause. If it is assumed that mortality was lower by age and sex group by a consistent factor for lung cancer and for all causes for smokers and nonsmokers in the CPS-II study population compared to mortality for these groups in the U.S. population during the study period, then mortality for all causes, M, and lung cancer in the U.S. population at that time can be expressed as:

$$M = \dot{P} \cdot \lambda M^S + (1 - \dot{P}) \cdot \lambda M^N \text{ and} \tag{2}$$

$$M_{lc} = \dot{P} \cdot \lambda M_{lc}^S + (1 - \dot{P}) \cdot \lambda M_{lc}^N, \tag{3}$$

where λ is an adjustment factor that is applied to rates from the study population in order to produce rates equal to those of the national population and $\dot{P}$

is an adjusted measure of exposure in the national population. These equations imply that estimates P of population smoker exposure are in some sense inflated in the absence of such an adjustment. These equations can be solved for the adjusted exposure $\dot{P}$:

$$\dot{P} = \frac{M_{lc}\frac{M^N}{M} - M_{lc}^N}{M_{lc}^S - M_{lc}^N - \frac{M_{lc}}{M}(M^S - M^N)} \tag{4}$$

and λ:

$$\lambda = \frac{M}{\dot{P} \cdot M^S + (1 - \dot{P}) \cdot M^N} = \frac{M_{lc}}{\dot{P} \cdot M_{lc}^S + (1 - \dot{P}) \cdot M_{lc}^N} \tag{5}$$

to produce estimates of exposure for the U.S. population at the time of the CPS-II study. Values for λ for females in the U.S. in 1986, approximately the midpoint of the CPS-II study, are as follows:

$45-49$	$50-54$	$55-59$	$60-64$	$65-69$	$70-74$	$75-79$	$80-84$	$85+$
1.62	1.97	1.73	2.11	1.95	2.04	1.94	2.13	2.61

This adjustment factor can be applied to other national populations or the U.S. in other years to produce a comparable adjusted proportion exposed to smoking, denoted by $\hat{P}$:

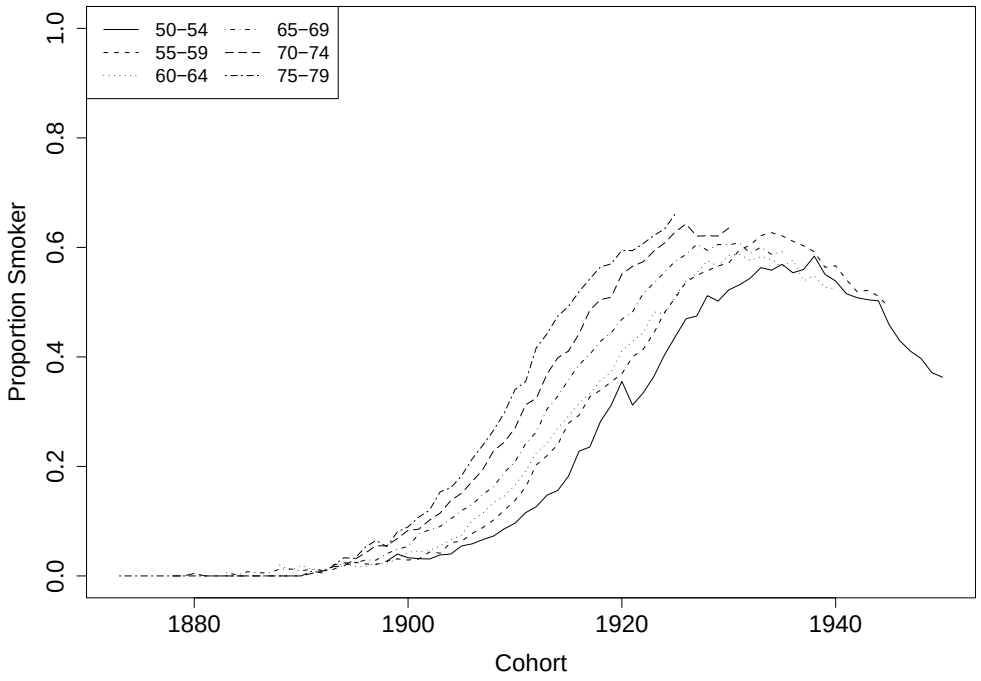
$$M_{lc} = \hat{P} \cdot \lambda M_{lc}^S + (1 - \hat{P}) \cdot \lambda M_{lc}^N. \tag{6}$$

These adjusted smoking exposure estimates tend to be more consistent for cohorts across periods in various countries. To illustrate this point, Figure 4 shows smoking exposure for female cohorts in the U.S. over time estimated with

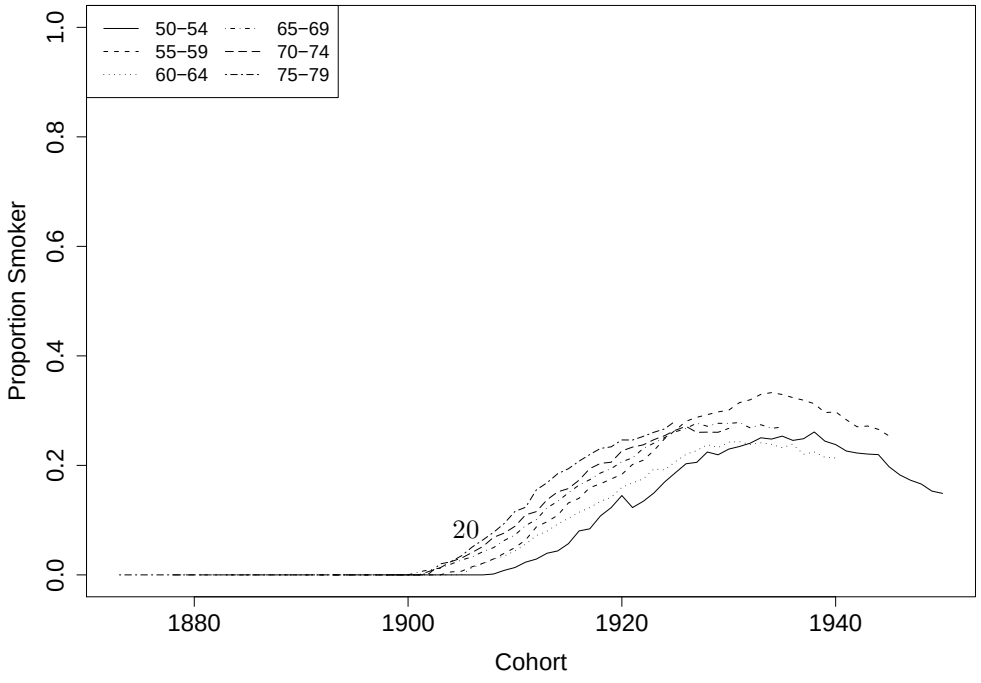
and without the adjustment factor. Use of the adjustment factor significantly decreases the variability of the estimates for specific cohorts and produces results that are more consistent with published figures of self-reported smoking prevalence for cohorts.

Figure 4: Estimated Smoking Exposure for U.S. Females, by Cohort

(a) Without Adjustment Factor



(b) With Adjustment Factor



Source: WHO Mortality Database data

The second half of the indirect method was then implemented by using these estimates of the standardized proportion of smokers and nonsmokers in each age and sex group to calculate mortality attributable to smoking. Standard population attributable risk formulas were used (Kahn and Sempos, 1989), and relative risks of mortality for various causes for five-year age groups were calculated from the detailed ACS-CPS II data provided in the appendix of the Peto et al. rather than the more general relative risks presented in the paper because of the emphasis here on mortality at older ages. In this particular case, the proportion of mortality attributable to changes in smoking exposure over time was calculated, so the population attributable risk formula can be expressed as:

$$PAR = \frac{N\Delta P_e I_e[(RR-1)/RR]}{NP_e I_e + N\Delta P_e I_e + N(1-P_e-\Delta P_e)I_o} \tag{7}$$

where N is the number of people in a population at a particular time, P_e is the proportion exposed as smokers in a previous time, ΔP_e is the change in the proportion of smokers over time, RR is the relative risk of mortality from a cause due to smoking exposure, and I_e and I_o are the incidence rates, in this case expressed as mortality rates, for that cause among smokers and nonsmokers. Relative risk and incidence rates were assumed to be constant over time. The formula simplifies to:

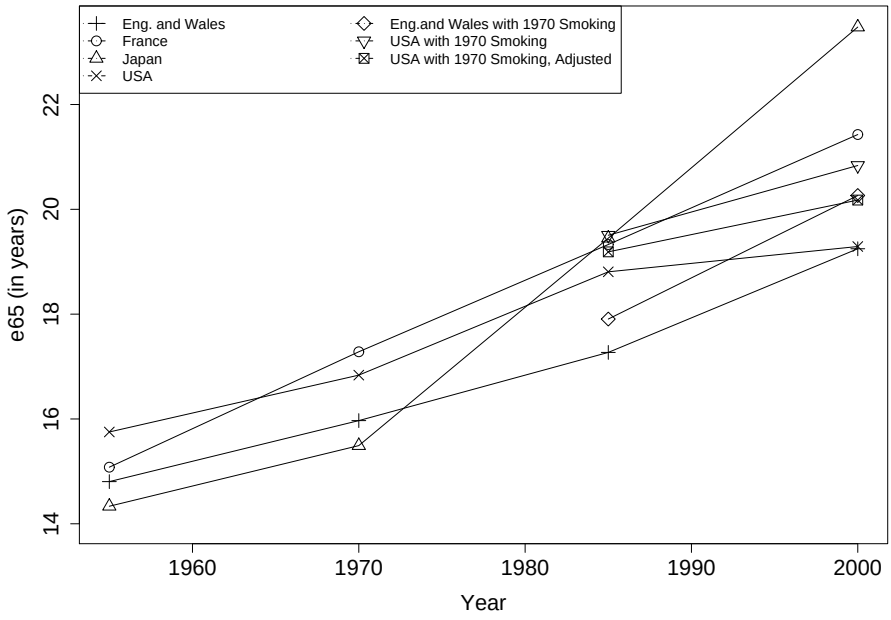
$$PAR = \frac{\Delta P_e(RR-1)}{P_e RR + \Delta P_e RR + (1-P_e-\Delta P_e)} \tag{8}$$

Following Peto et al., only half of the increase in relative risk of mortality for

smokers for causes other than lung cancer was attributed to smoking, due to the possible confounding effect of other risk factors associated with smoking such as alcohol consumption or behavioral factors. It was also assumed that smokers have no increased risk of mortality from external causes and cirrhosis of the liver, as was the case in the procedure presented by Peto et al.

Alternative estimates of life expectancy in the absence of all or some fraction of smoking can be calculated, using estimates of smoking exposure with and without adjustment using the indirect method. In this case, life expectancy for countries was calculated for females at older ages under the assumption that smoking rates by age group had stayed the same over time in these countries from one period to another, a method chosen in order to try to control for differences other than smoking that existed between countries during these times. Figure 5 presents e65 values for females in England and Wales and the U.S. calculated with the indirect method using WHO data assuming that smoking exposure had remained at 1970 levels in subsequent years, including estimates for the U.S. made with and without the adjustment factor. The plot shows an appreciable increase in e65 for females in England and Wales and the U.S. in 2000 if smoking exposure had remained at the same levels by age group as in 1970, although the improvement estimated using the adjustment factor for the U.S. is more modest.

Figure 5: Life Expectancy at Age 65, Females in Various Countries with Changed Smoking Exposure



Source: WHO Mortality Database data

Note: Alternative values for e65 with continued 1970 exposure are based on the indirect method and the alternative value for e65 with adjusted 1970 exposure is based on the indirect method using the adjustment factor λ.

These methods and results can be evaluated with reference to another analysis of the effect of smoking on mortality for females at older ages in the U.S. in 2000 conducted by Richard Rogers, Robert Hummer, Patrick Krueger, and Fred Pampel (2005). Rogers and his colleagues calculated the mortality risks of smoking for different lengths and intensities compared to not smoking, controlling for possible confounding variables, using a discrete-time hazard model with seven years of follow-up data from the 1990 National Health Interview Survey Health Promotion and Disease Prevention supplement. These mortality risks were then applied to the number of people in each smoker category in the U.S. in 2000, using smoking prevalence figures from the NHIS and population figures from the U.S. Census Bureau, to produce an estimate of the number of deaths attributable to smoking by sex and age group in that year. Removing the number of deaths attributable to smoking for females in age groups above 65 reported by Rogers et al. from the total number of deaths for these groups in the WHO mortality data for the U.S. in 2000 produces a value of e65 for females of approximately 20.1 years. The comparable figures derived from the indirect method for the U.S. in 2000 using the indirect method are 20.19 years with the adjustment factor and 20.96 years without it, suggesting that use of the adjustment factor with the indirect method produces much more accurate estimates of smoking exposure in the U.S. and thus mortality attributable to smoking than does use of the indirect method without this adjustment.

A similar analysis can be performed for life expectancy at older ages for males in developed countries. HMD data show that e65 values for males in many de-

veloped countries, including Australia, Canada, Denmark, England and Wales, Italy, and the U.S., either did not increase significantly or in fact decreased between 1955 and 1970, although e65 did increase for males in the period in France and Japan. The effect of changes in smoking exposure on mortality can again be estimated using population attributable risk calculations. In this case, smoking exposure increased for males in most developed countries during the period, so mortality rates based on higher levels of smoking exposure can be calculated for earlier years by multiplying observed rates by an appropriate factor, which is calculated in the following manner:

$$\text{Attributable Risk factor} = 1 - \frac{N\Delta P_e I_e(\frac{RR-1}{RR})}{NP_e I_e + NI_o(1-P_e)} \tag{9}$$

where ΔP_e is the difference between an observed level of smoking exposure and an alternative level of exposure. ΔP_e in this case has a negative value because the observed smoking exposure in the earlier period was less than the smoking exposure in a later period that is used as the alternative level of exposure. This formula simplifies to:

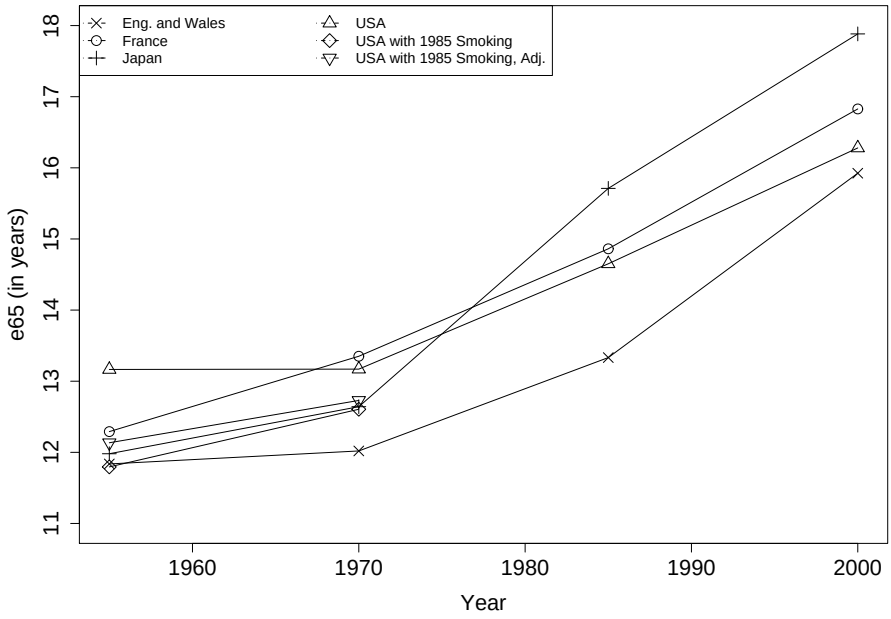
$$\text{Attributable Risk factor} = 1 - \frac{\Delta P_e(RR-1)}{P_e RR + 1 - P_e} \tag{10}$$

This formula can be used to estimate the value of e65 for males in the U.S. in 1955 and 1970 with the smoking exposure of 1985 using the indirect method with and without adjustment. These results, shown in Figure 6, indicate that life expectancy at age 65 for males would have been lower in 1955 and 1970

in the U.S. with either of the estimated 1985 smoking exposure levels rather than the observed values due to subsequent increases in smoking exposure for age groups above 65. The results also again show that the unadjusted exposure estimates tend to produce larger estimates of the effect of smoking on mortality, as expected. The estimates also show that with constant 1985 smoking levels e65 for males would have increased in the U.S. from 1955 to 1970 at a rate consistent with the increase observed in Japan, rather than remaining relatively unchanged during the period. They also suggest that e65 for U.S. males would have increased at a somewhat greater rate from 1970 to 1985 with constant smoking exposure than the observed appreciable increase, suggesting that observed increases in life expectancy at older ages for males in the U.S. and other developed countries during this period were principally due to factors such as advances in medical care and technology rather than advantageous changes in smoking exposure among older age groups.

Overall, this analysis suggests that differences in previous smoking trends account for some portion of the observed differences across countries in life expectancy at older ages and that use of an adjustment factor with the indirect method produces substantially more accurate estimates of the effect of smoking on mortality in countries such as the U.S. As an example, e65 for females calculated from WHO data for France was 2.14 years higher than the corresponding value for the United States in 2000. Use of the indirect method without adjustment estimates that 93% of this difference would have been eliminated if U.S. females had had the same smoking exposure as French females by age group,

Figure 6: Life Expectancy at Age 65, Males in Various Countries



Source: WHO Mortality Database

whereas the comparable estimate obtained from the indirect method with adjustment is 63%. Given the greater accuracy introduced by the adjustment factor with regard to the mortality effect of smoking in the U.S., it would appear the latter is a better estimate.

Evaluation of the Proposed Method

Three types of evidence suggest that use of the adjustment factor with the indirect method produces improved estimates of the effect of smoking on mortality. First, estimates of smoking exposure made with the method and adjustment factor are more consistent with self-reported smoking prevalence than those estimates made without the adjustment factor. For example, the 1975 Adult Use of Tobacco Survey conducted by the U.S. Department of Health, Education, and Welfare and Public Health Service found that 36% of U.S. women in their 30s, 33% of women in their 40s, and 26% of women in their 50s reported that they were smokers (Forey et al., 2002), proportions that are much closer to the estimates of standardized smoking exposure produced using the indirect method with adjustment than estimates produced without adjustment as seen in Figure 4.

Second, estimates of mortality attributable to smoking made with the indirect method and adjustment factor are close to estimates of the mortality effects of smoking found in other studies. As stated, estimates of e65 for females in the U.S. in 2000 made with the adjustment factor are close to those obtained from

results from the more detailed study of mortality attributable in that country in that year conducted by Rogers et al. and much closer than estimates obtained through use of the indirect method without adjustment. Similarly, the indirect method with adjustment factor can be used to estimate smoking exposure for use in age-period-cohort smoking history models, similar to the the models employed by Preston and Wang in a study that measured smoking exposure based on the mean number of years spent as a smoker before the age of 40 for cohorts obtained from reconstructions of National Health Interview Survey data (2006). The results, seen in Table 3, demonstrate that the indirect method as implemented in this study with adjustment produces estimates of the mortality effect of smoking that are quite similar to those produced by Preston and Wang from more detailed smoking histories, particularly for cohorts who experienced at least moderate levels of smoking and for more recent cohorts, and that the estimates produced with adjustment are much closer to these published results than those produced without adjustment.

Finally, estimates of the mortality attributable to smoking obtained from this method are similar to detailed national estimates produced for various causes and age groups. Table 4a presents estimates of mortality due to smoking in the U.S. from 1997 to 2001 produced by the CDC by multiplying estimates of the smoking-attributable fractions of deaths, obtained by using relative risks of mortality for causes of death from the ACS CPS-II study and current and former smoking prevalence for the age groups 35-64 and 65+, by total mortality for 18 adult and four infant causes of death (2005), as well as estimates ob-

tained with the indirect method as implemented in this study with and without adjustment. The results show that the indirect method, with or without adjustment, produces estimates of lung cancer mortality attributable to smoking that are close to the estimates produced by the CDC. The results also indicate that the indirect method without adjustment produces estimates of overall mortality attributable to smoking that are closer to those of the CDC than the estimates produced without adjustment, although there have been suggestions that estimates by the CDC and other organizations may sometimes overestimate the overall mortality effects of smoking. As has been noted, Rogers et al. produced detailed estimates of mortality due to smoking in the U.S. in 2000 from data from the NHIS-HPDP supplement with mortality follow-up, and found that 338,000 deaths could be attributed to smoking in the country in that year, a figure substantially lower than CDC estimates (2005). Estimates produced with the indirect method as implemented in this study, with and without adjustment, and with the indirect method as specified by Peto et al., with and without adjustment for the age groups used in that method, are shown in Table 4b, along with estimates from Rogers et al. The results indicate that use of an adjustment factor with the indirect method, whether with the method as implemented here or as presented by Peto et al., produces estimates of mortality due to smoking that are closer to the detailed estimates produced by Rogers et al. than estimates produced without adjustment. The results also show that although the indirect method with adjustment produces estimates of mortality from smoking for females in the U.S. in 2000 that are lower than those produced by Rogers et

al., the estimates of mortality for females above the age of 65, which is the focus of this work, are quite similar from both methods, this despite concerns about the reliability of data used with the indirect method for advanced age groups.

The Effect of Smoking and Other Risk Factors

Other factors that could account for differences in life expectancy at older ages in these countries in addition to smoking include environmental exposures, genetic factors, behavior, diet, and social and health care programs and policies. One aspect of the U.S. health care system that could affect morbidity and mortality at older ages is that although most U.S. residents have access to health care coverage from the age of 65 through the Medicare program, some U.S. residents lack health care coverage prior to the age of 65. Researchers have shown, for example, using a prospective cohort study with data from the U.S. Health and Retirement Study that individuals in the U.S. in the age range from 51 to 61 with no or intermittent health insurance coverage were more like to suffer serious declines in health over a four-year period than were individuals with continuous health care coverage during this time (Baker et al., 2001). Other researchers have shown using the U.S. Health and Retirement Study and the English Longitudinal Study of Aging that the health of U.S. residents aged 55 to 64 is generally worse than that of their English counterparts based on self-reported prevalence of several chronic diseases including heart disease and diabetes and biological markers such as c-reactive protein, even for residents of the two countries in

similar income or eduction levels and when results were adjusted to account for differences in other risk factors such as obesity and smoking (Banks et al., 2006). The study was restricted to non-Hispanic whites in both countries to eliminate any possible effects due to health differences among minority racial or ethnic populations within a country, particularly for African-Americans and Hispanics in the U.S. This result is consistent with data that show that although a disparity in e65 values exists between racial and ethnic groups in the U.S., with the value of e65 for African-American females being 17.7 years as opposed to 19.4 years for white females in 2000, white females are a sufficiently large proportion of the female population above the age of 65 in the U.S. that results for the national population, in this case an e65 value of 19.3 for females in 2000, are very similar to those of whites (National Center for Health Statistics, 2006), and thus these differences for racial and ethnic minority populations do not account for much of the difference in life expectancy at older ages between the U.S. and other developed countries. Moreover, the correspondence between female e65 values for the white and total population in the U.S. has been observed extending back to at least 1960 in data from the annual *Statistical Abstract of the United States* and *NCHS Report* (Meslé and Vallin, 2006).

These studies suggest that aspects of the U.S. health care and social systems could account for some portion of the differences observed between life expectancy at older ages for females in the U.S. and other developed countries. It is interesting to note that, as seen in Figure 1, the value for e65 for females in 2000 was approximately a year lower in the U.S. than in Canada, even though

both countries have similar characteristics in terms of smoking prevalence, diet, and ethnic composition, although they do have differences in their health care and social welfare systems with Canada providing more comprehensive care for its residents throughout the life course.

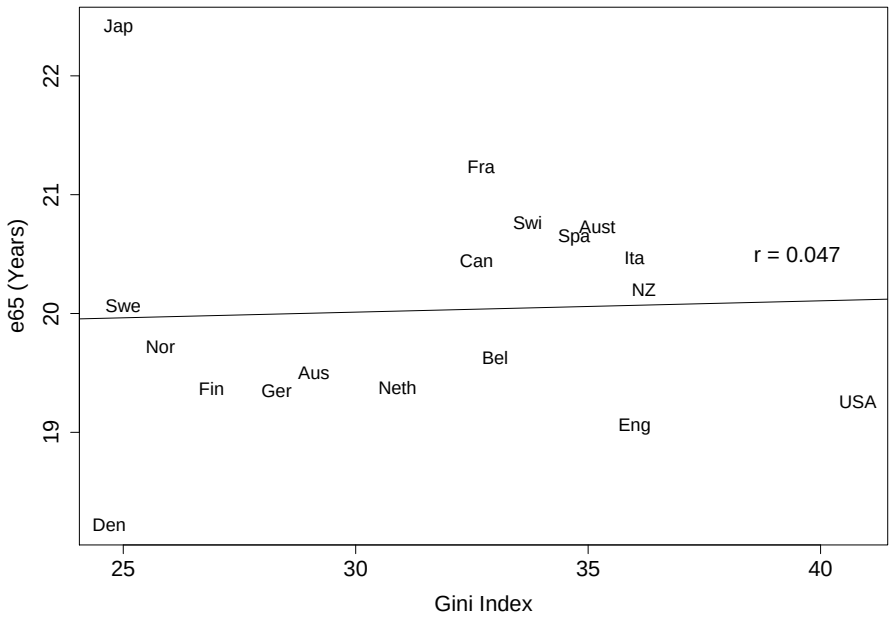
Differences in health and mortality based on social and health care systems between the U.S. and other industrialized countries could result from specific differences in health care coverage between the countries or more generally from differences in the level of economic and social equality between them. Numerous researchers have argued that income inequality accounts for some portion of mortality differentials between countries (Wilkinson, 1992) and U.S. states (Kaplan et al., 1996), although other researchers question aspects of these findings (Judge, 1995; Mackenbach, 2002).

Comparison of income inequality across countries is difficult and inexact because the data are often obtained by household from surveys that vary in methodology and data collection. That stated, some comparison of such measures will be made here in an attempt to understand the general relationship between income inequality and life expectancy at advanced ages. Figure 7 plots e65 values for females in 17 developed countries against their Gini index value, a measure of income inequality that is the ratio, multiplied by 100, of the area between the cumulative income distribution function in a population and the uniform distribution function divided by the area under the uniform distribution function, as reported by the World Bank for various developed countries in the year closest to 2000 (World Bank, 2006). The figure shows essentially

no relationship between these two variables, perhaps because of confounding by other variables such as smoking exposure, and the correlation coefficient is equal to 0.047. Figure 8 plots e85 values against Gini index values for these same countries and shows a much stronger relationship between these two variables. The correlation coefficient for these countries excluding Japan, which appears as an outlier in the plot and is the only non-European, non-English-speaking country in the group as well as the developed country for which published estimated of the Gini index vary most widely (Central Intelligence Agency, 2005; World Bank, 2006), is 0.64, indicating a positive relationship between life expectancy at advanced age and income inequality. To some extent, this relationship may result from the greater ability of individuals to obtain advanced, and in some cases very expensive, medical procedures and care in countries with greater income inequality and emphasis on individualism such as the U.S. than in some Northern and Central European countries that provide comprehensive health course throughout the life course and have a tradition of more collectivist policies and programs. The figure also shows graphically the superior position that the U.S. enjoys in terms of life expectancy for females at age 85 compared to age 65, a result that is consistent with research over time that has found that the U.S has done well in life expectancy at very advanced ages compared to other developed countries (Manton and Vaupel, 1995; Hill et al., 2000).

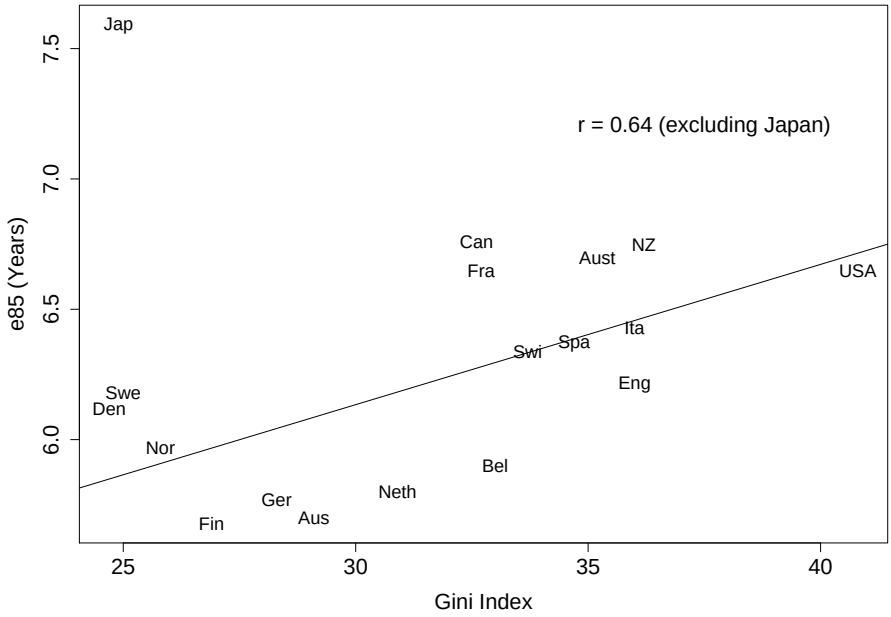
Finally, other differences in factors such as diet and behavior could account for a portion of differences in life expectancy for females at older ages in developed countries. Some researchers have suggested that differences in consump-

Figure 7: e65 for Females and Gini Index, Various Countries 2000



Source: Human Mortality Database and *World Bank World Development Indicators 2006*

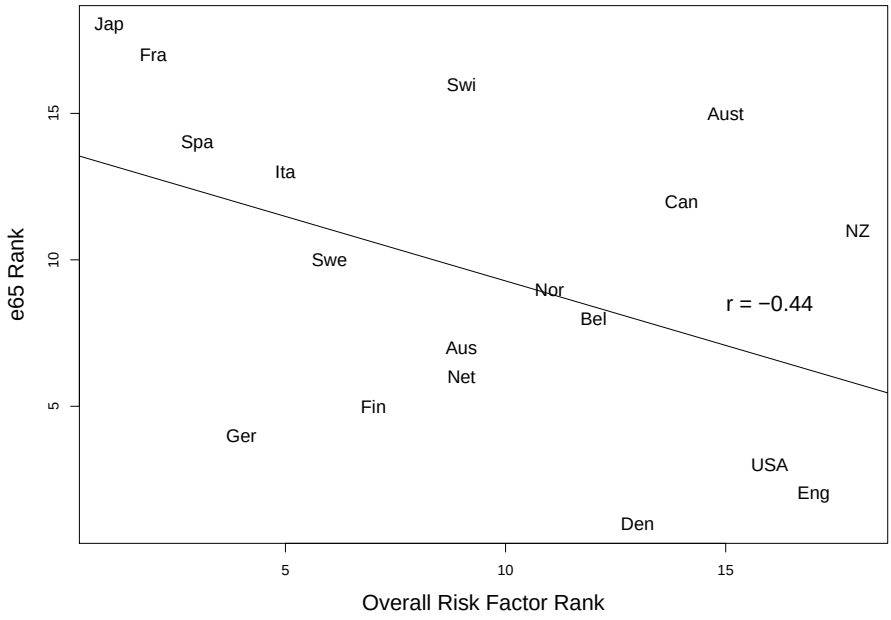
Figure 8: e85 for Females and Gini Index, Various Countries 2000



Source: Human Mortality Database and *World Bank World Development Indicators 2006*

tion of saturated fats, animal products, or alcohol (Criqui and Ringel, 1994; Law and Wald, 1999) could affect life expectancy for various nations. Table 5 displays data and ranks for three important risk factors, estimated smoking exposure, the Gini Index, and animal fat consumption for a group of 17 countries. These countries were selected according to the criteria, similar to the methodology used by (Criqui and Ringel, 1994) in a comparable analysis, of having per capita GDP in 2000 of at least $18,000, a population of at least one million residents, and inclusion of data in the Human Mortality Database. The figure for estimated smoking exposure for each country is the mean of the estimated smoking exposure with adjustment for 2000 for the age groups 65-84 derived from the WHO data. Values for the Gini Index for 2000 come from the World Bank. Animal fat consumption is measured by the proportion of daily caloric intake obtained from animal fat in 1965 according to published data derived from U.N. Food and Agricultural Organization food balance sheets (Criqui and Ringel, 1994), a value that to some extent reflects the type of diet that women at older ages in these countries would have commonly consumed during much of the lifetimes. The table also presents an overall risk factor ranking based on the means of the three risk factor ranks. Figure 9 plots ranks for female e65 values for these developed countries against these overall risk factor ranks and suggests a negative association between these two sets of ranks, with their correlation coefficient being -0.44. The plot also shows that the model somewhat over-penalizes countries such as Australia and New Zealand that have relatively high income inequality in its prediction of life expectancy

Figure 9: Female e65 and Risk Factor Rankings, Various Countries 2000



Source: See Table 5

rank and under-penalizes countries such as Denmark with high female smoking exposure at older ages. These deviations are not entirely unexpected given the large effect of smoking on mortality trends that has been demonstrated here as well as the weak relationships seen previously between income inequality and life expectancy at older ages. A similar model that employs a weighted mean of rankings, with twice the weight assigned to smoking exposure and half the weight assigned to the Gini index as the weight assigned to animal fat consumption produces a correlation coefficient that is somewhat larger in magnitude at -0.55.

In general, these results suggest that these factors, particularly smoking and fat consumption, have an appreciable effect on life expectancy at older ages in these countries. This notion can be further tested by analyzing the effects of these risk factors as well as those of other economic and health characteristics on life expectancy over time in a longitudinal data analysis. Estimates of the effect of smoking on mortality obtained from the indirect method as implemented in this study are useful in such an analysis, given that this method produces measures of the effect of smoking for countries over time that are comparable to those obtained from more detailed data sources such as smoking histories.

Longitudinal data analysis of risk factors and life expectancy in developed countries demonstrates the effect of smoking and certain dietary factors on mortality at older ages. Table 6 presents results of analyses of the effect of mortality risk factors and economic conditions on e65 values over time for a group of developed countries using mixed effect models with random intercepts for each country of the form:

$$y_i = a_j[i] + b_1 x_{i1} + b_2 x_{i2} + ..., \tag{11}$$

where $a_j[i]$ is the intercept term for country j and b_1, b_2, and so on are the estimated effects of covariates x_1, x_2, etc. The group of countries is the same as that used previously with the exception of Germany, which was not included in this analysis because of its divided status prior to 1990. Life expectancy figures come from the Human Mortality Database, smoking exposure figures are esti-

mated using the indirect method with adjustment from WHO data, total caloric intake and animal fat consumption data come from UN Food and Agricultural Organization food balance sheets, alcohol consumption figures come from the WHO (2004), values for the Gini Index for countries over time come from the World Bank's World Income Distribution Gini Database (World Bank, 2009), and real per capita GDP data come from the Penn World Table (Heston et al., 2006). [2]

Overall, these results show that life expectancy at 65 for females consistently increased with time in each period as expected and clearly decreased with increased smoking exposure from 1970 to 1984 and 1985 to 1999, periods when smoking levels for females were increasing to moderate and high levels in many developed countries.

Results from the most recent period suggest that certain factors particularly influence national life expectancy trends. In addition to the observed positive association between time and GDP and e65 and the negative association between smoking exposure and e65 in this period, these results indicate a negative association between fat consumption and e65, which is consistent with previous results in this study, although no such association is found between total caloric intake and e65 values. No strong relationship is found between alcohol consumption and life expectancy at this age, which is generally consistent with findings that find only small associations between alcohol consumption and mortality rates compared with other risk factors such as smoking (Thun et al., 1997), and the estimated effects of the Gini index and per capita GDP on e65 are positive,

although somewhat small in magnitude, and approaching conventional levels of statistical significance.

Table 7 presents similar results for e85. Once again, time is generally associated with improvement in life expectancy, and the Gini index is positively associated with e85 in the most recent period, which is consistent with results from previous analysis. Estimates of the effect of smoking exposure vary by period, a result probably due in large part to the instability of estimates of smoking exposure at advanced ages obtained from the indirect method, although the effect is estimated to be negative in the most recent period, particularly in the absence of additional covariates such as alcohol consumption. Fat consumption is negatively associated with e85 values for the period from 1970 to 1984, although less so in the subsequent period. Results from the more extensive set of risk factors in the most recent period show a positive association between low to moderate alcohol consumption and e85 values, a result consistent with various studies that suggest that moderate, but not excessive, alcohol consumption may be inversely associated with mortality rates at older ages (Doll et al., 1994; Lin et al., 2005).

Interestingly, the finding that cigarette smoking accounts for much of the excess mortality for females at older ages in some developed countries and that dietary factors such as animal fat consumption perhaps also play some role is not new. Samuel Preston, for example, studied excess mortality among males at older ages in some countries in a monograph published in 1970 (1970). He concluded that "by far the most promising explanation of recent excesses in

older male death rates is cigarette smoking behavior" and that "one can only speculate as to the nature of other contributing factors, but increases in cholesterol consumption and decreases in exercise levels, perhaps associated with the greater use of automobiles, seem reasonable prospects." This conclusion, based on analysis of mortality data for a similar trend, provide useful support for the findings presented here.

Conclusion

This study has specifically examined the effect of smoking and other risk factors on mortality trends for females at older ages in developed countries, given the observed slowdowns in recent decades in mortality declines for this group in various countries. It has modified and extended the indirect method to estimate mortality attributable to smoking based on lung cancer mortality rates for women at older ages by implementing the method for more specific ages groups, particularly at older ages, and introduced an adjustment factor to account for low mortality in the method's study population. Results from the method implemented in this manner are similar to those in published studies, and smoking is estimated to account for approximately half of the difference in life expectancy at age 65 for females in the U.S. and some other developed countries. For the remaining differences, longitudinal data analyses suggest that dietary factors, particularly animal fat consumption, are the most identifiable cause of differences between countries.

More generally, the study has evaluated the use of an adjustment factor with the indirect method in the attempt to better estimate smoking exposure and mortality attributable to smoking. Various studies and results presented here suggest that the indirect method without adjustment tends to overestimate the mortality effects of smoking, due to low mortality in its ACS CPS-II study population. This study has found that use of an adjustment factor produces more consistent estimates of smoking exposure in previous cohorts and improved estimates of mortality due to smoking than does the indirect method without adjustment. Precise implementation of the method may vary with the particular study, but one possibility for improvement could be to use an adjustment factor with the age groups used in the original method presented by Peto et al. In the case of females, the relevant factors would be 1.73 for ages 35-59, 2.08 for 60-64, 1.99 for 65-69, 2.00 for 70-74, and 1.92 for 75-79, which would also be used for those 80+, or a common adjustment factor of, say, 1.9 or 2 could be used for all age groups. Use of such adjustment factors could produce more consistent and reliable estimates of the mortality effects of smoking, as this study has demonstrated.

This study has also shown the usefulness of the indirect method, particularly for comparisons between countries and over time. The method as presented by Peto et al. makes various assumptions, such as the halving of elevated risks of mortality for causes other than lung cancer, and modifications made here have sometimes introduced additional assumptions, such as that mortality was lower for both lung cancer and total mortality in the ACS CPS-II study by a consistent

factor. That stated, the results produced by the method as implemented here are plausible and consistent with published findings. The indirect method is particularly useful, as demonstrated here, in cases in which detailed smoking exposure and relative risks of smoking may not be readily available but lung cancer and total mortality figures are available. The indirect method, as a result, is very useful in comparisons of the mortality effects of smoking in countries over appreciable periods of time or between numerous countries.

Finally, these results confirm the importance of the effect of previous smoking behavior on trends in mortality and life expectancy. Beginning in the 1970s, mortality declines began to slow for females at older ages in countries such as the U.S., U.K., the Netherlands, and Denmark, in large part due to previous increases in the number of women smoking at younger ages in these countries. As the change in the proportion of women smoking in successive cohorts in these countries began to slow or in some cases even decline, then the effect of smoking on mortality for females at older ages also began to stabilize or decrease. As an example, e65 for females in the U.S. increased from 19.3 to 20.0 years from 2000 to 2005, this after only increasing by 0.2 years in the preceding decade (University of California, Berkeley, 2009). On the other hand, an eventual slowdown in gains in life expectancy should be expected in countries in which women began smoking in large numbers at a later date such as France, Italy, and Spain, thus producing trends similar to those that have already been observed in some countries.

Table 1: Classification of Causes of Death According to Various Revisions of the International Classification of Diseases

Class of Causes	ICD7 List A	ICD8 List A	ICD9 Basic Training List	ICD10 Mortality Tabulation List 1
Infectious	A001 - A043	A001 - A044	B01 - B07	A00 - B99
Malignant Neoplasms, Not Respiratory	A045 - A048, A051 - A059	A047 - A049, A052 - A060	B09, B11 - B14	C15 - C31, C35 - C99
Malignant Neoplasms, Respiratory	A044, A049 - A050	A045, A050 - A051	B08, B10	C00 - C14, C32 - C34
Cardiovascular Diseases	A079 - A086	A080 - A084, A086 - A088	B25 - B28, B30	I00 - I59, I70 - I99
Cerebrovascular Diseases	A070	A085	B29	I60 - I69
Respiratory Diseases	A087 - A097	A089 - A096	B31 - B32	J00 - J99
Digestive	A098 - A107	A097 - A104	B33 - B34	K00 - K99
Alzheimer's, Senility, and Psychoses	A067, A136	A069, A136	B210-B213, B465	F00 - F09, F20 - F29, G30 - G39, R54
Other Diseases	A060 - A066, A068 - A069, A071 - A078, A108 - A135	A061 - A068, A070 - A079, A105 - A135	B15 - B20, remainder of B21, B22 - B24, B35 - B45,	D00 - E99, F10 - F19, G40 - H93, F20 - G29, L00 - Q99
Ill-Defined Causes	A137	A137	remainder of B46	remainder of R
External Causes	A138 - A150	A138 - A150	B47 - B56	W00 - Y99

Source: ICD codes from *WHO Mortality Database Documentation*.

Table 2: Estimated Standardized Smoking Exposure for Females in Developed Countries, by Year and Age Group

Country	45-49	50-54	55-59	60-64	65-69	70-74	75-79	80-84	85+
England and Wales									
1955	0.17	0.13	0.12	0.08	0.07	0.04	0.00	0.00	0.00
1970	0.34	0.34	0.28	0.22	0.18	0.15	0.12	0.04	0.04
1985	0.31	0.27	0.42	0.46	0.43	0.39	0.32	0.24	0.17
2000	0.26	0.27	0.34	0.32	0.39	0.50	0.48	0.40	0.27
France									
1955	0.03	0.01	0.01	0.01	0.00	0.00	0.00	0.00	0.00
1970	0.01	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
1985	0.03	0.04	0.05	0.02	0.02	0.01	0.00	0.00	0.00
2000	0.24	0.15	0.12	0.06	0.06	0.05	0.03	0.00	0.00
Japan									
1955	0.01	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
1970	0.07	0.04	0.05	0.06	0.05	0.04	0.01	0.00	0.00
1985	0.07	0.06	0.07	0.08	0.09	0.11	0.15	0.16	0.15
2000	0.08	0.10	0.08	0.07	0.09	0.10	0.15	0.23	0.38
United States									
1955	0.07	0.04	0.03	0.02	0.01	0.00	0.00	0.00	0.00
1970	0.39	0.31	0.24	0.15	0.10	0.07	0.03	0.00	0.00
1985	0.55	0.56	0.56	0.48	0.43	0.37	0.27	0.18	0.10
2000	0.36	0.40	0.52	0.55	0.60	0.62	0.62	0.58	0.43

Source: WHO Mortality Database data

Table 3: Comparison of Estimated Effect of Smoking History on Female Mortality

Birth Cohort	Mortality Increase from Cigarette Smoking Histories	Mortality Increase from Indirect Method with Adjustment	Mortality Increase from Indirect Method without Adjustment
1895 - 1899	3.2%	0.0%	1.4%
1900 - 1904	4.2%	0.0%	2.0%
1905 - 1909	7.1%	0.1%	3.9%
1910 - 1914	10.2%	2.1%	8.0%
1915 - 1919	12.3%	5.8%	15.8%
1920 - 1924	12.9%	8.1%	20.9%
1925 - 1929	14.0%	12.4%	30.5%
1930 - 1934	14.3%	13.9%	34.0%
1935 - 1939	14.6%	14.5%	35.5%
1940 - 1944	14.6%	12.2%	30.0%
1945 - 1949	12.7%	9.0%	22.9%

Note: Mortality increase estimates from the indirect method come from an age-period-cohort smoking exposure model, as presented by Preston and Wang (2006), using Human Mortality Database data for deaths and population. Increases estimated with the indirect method use lung cancer mortality rates from the WHO Mortality Database for the age group 50-54 to estimate smoking exposure. Increases estimated from cigarette smoking histories are those presented by Preston and Wang.

Table 4: Estimates of Deaths Attributable to Smoking, 1997-2001

(a) 1997-2001

	Females			Males		
	CDC Estimates	Indirect Method	Indirect Method with Adjustment	CDC Estimates	Indirect Method	Indirect Method with Adjustment
- Trachea, Lung, Bronchus Neoplasms	44810	53265	41915	79026	81642	76318
Neoplasms	54310	63730	46671	104219	113918	96462
- Cardiovascular Diseases	44719	33909	18371	75824	62794	37270
- Cerebrovascular Disease	8893	7512	4085	8543	8942	5411
Circulatory Diseases	53612	41421	22455	84367	71736	42681
Respiratory Diseases	47135	45855	21072	54319	41459	31202
Other Diseases	23351	37592	13746	16589	16710	2825
Total	178408	188598	103944	259494	243823	173170

(b) 2000

	Females					Males				
	Rogers et al. Estimates	Indirect Method	Indirect Method with Adjustment	Peto et al. Method	Peto et al. Method w/ Adjustment	Rogers et al. Estimates	Indirect Method	Indirect Method with Adjustment	Peto et al. Method	Peto et al. Method w/ Adjustment
Total	133000	195378	106957	257617	158334	200000	237715	167374	269821	205214
35-64	70000	47005	31159	59613	36556	105000	75855	43749	72922	54395
65+	63000	148373	75798	198004	121778	95000	161860	123626	196899	150818

Note: CDC estimates come from (2005). Estimates from the indirect method with and without adjustment were obtained by use of the indirect method with five-year age groups as implemented in this study. Estimates from the Peto et al. method were obtained through implementation of the method as explained in their work (1994), with the adjustment factor λ explained in this paper used for the appropriate age groups in the estimates with adjustment. Estimates from Rogers et al. come from their study (2005).

Table 5: Mortality Risk Factors and Ranks for Various Developed Countries

Country	2000 Gini Index	1965 Fat Consumption	Smoking Exposure	Gini Rank	Fat Rank	Smoking Rank	Overall Rank
Australia	35.2	30.8	7.8	14	16	12	15.0
Austria	29.1	27.1	1.9	7	11	8	9.0
Belgium	33.0	28.9	1.6	11	12	6	12.0
Canada	32.6	28.9	19.0	9	12	16	14.0
Denmark	24.7	32.0	23.2	1	17	17	13.0
Eng. & Wales	36.0	30.8	16.2	16	16	15	17.0
Finland	26.9	30.5	1.0	5	14	4	7.0
France	32.7	20.5	0.0	10	4	2	2.0
Germany	28.3	25.4	1.9	6	6	7	4.0
Italy	36.0	11.7	0.7	16	2	3	5.0
Japan	24.9	6.6	1.3	2	1	5	1.0
Netherlands	30.9	26.0	6.1	8	7	11	9.0
Norway	25.8	26.7	7.9	4	10	13	11.0
New Zealand	36.2	32.1	11.6	17	18	14	18.0
Spain	34.7	12.0	0.0	13	3	2	3.0
Sweden	25.0	26.5	4.7	3	8	10	6.0
Switzerland	33.7	23.6	2.5	12	5	9	9.0
USA	40.8	26.7	24.3	18	10	18	16.0

Source: Gini Index - World Bank *World Development Indicators 2006*, 1965 Animal Fat Consumption as Percentage of Total Energy Intake - UN Food and Agricultural Organization food balance sheets presented in Criqui and Ringel (1994), Smoking Exposure - mean of standardized smoking exposure for age groups 65-84 in 2000 estimated with adjustment from WHO Mortality Database data, Overall Rank - rank of mean of Gini coefficient, fat consumption, and smoking exposure ranks.

Table 6: Results of Regressing e65 on Risk Factors for Females in 16 Developed Countries

	Estimate	Std. Error	Pr(> |t|)	Estimate	Std. Error	Pr(> |t|)
1955 - 1969						
(Intercept)	14.4123	0.9477	0.0000	14.1283	0.9136	0.0000
Time (Year - 1955)	0.0823	0.0040	0.0000	0.0577	0.0146	0.0001
Smoking Exposure (%)	-0.2599	0.2697	0.3364	-0.3557	0.2749	0.1973
Fat Consumption (%)	0.0177	0.0305	0.5728	0.0099	0.0287	0.7350
Gini Index (%)	0.0012	0.0181	0.9453	0.0070	0.0180	0.6975
Per Capita GDP ($1000's)				0.1839	0.1047	0.0806
1970 - 1984						
(Intercept)	16.0874	0.4808	0.0000	16.3474	0.4864	0.0000
Time (Year - 1970)	0.1737	0.0044	0.0000	0.2256	0.0171	0.0000
Smoking Exposure (%)	-0.0889	0.0165	0.0000	-0.0765	0.0166	0.0000
Fat Consumption (%)	-0.0132	0.0153	0.3890	-0.0179	0.0152	0.2410
Gini Index (%)	0.0102	0.0077	0.1858	0.0133	0.0076	0.0817
Per Capita GDP ($1000's)				-0.0787	0.0251	0.0020
1985 - 1999						
(Intercept)	18.2136	0.9062	0.0000	18.6402	1.4707	0.0000
Time (Year - 1985)	0.1479	0.0044	0.0000	0.1240	0.0145	0.0000
Smoking Exposure (%)	-0.1128	0.0085	0.0000	-0.1198	0.0105	0.0000
Fat Consumption (%)	-0.0492	0.0179	0.0066	-0.0506	0.0195	0.0103
Total Calories (100's)				0.0063	0.0310	0.8401
Alcohol (liters)				-0.0724	0.1056	0.4941
Alcohol^2 (liters)				0.0010	0.0043	0.8164
Gini Index (%)	0.0482	0.0253	0.0587	0.0382	0.0258	0.1393
Per Capita GDP ($1000's)				0.0281	0.0185	0.1308

Source: e65 - Human Mortity Database, Smoking Exposure - mean of standardized smoking exposure for age groups 65-84 estimated from WHO Mortality Database data, Fat Consumption as Percentage of Total Energy Intake and Total Calories per Capita per Day - UN FAO Food Balance Sheets, Adult per Capita Consumption of Alcohol (Ethanol) per Year - WHO *Global Alcohol Report 2004*, Gini Index - World Bank *World Development Indicators 2006*, and Per Capita Real GDP - Penn World Table 6.2.

Table 7: Results of Regressing e85 on Risk Factors for Females in 16 Developed Countries

	Estimate	Std. Error	Pr(> |t|)	Estimate	Std. Error	Pr(> |t|)
1955 - 1969						
(Intercept)	4.9149	0.5442	0.0000	4.7131	0.5230	0.0000
Time (Year - 1955)	0.0243	0.0023	0.0000	0.0057	0.0083	0.4891
Smoking 85+ (%)	-0.2537	0.9099	0.7807	-0.3591	0.9049	0.6920
Fat Consumption (%)	-0.0020	0.0175	0.9103	-0.0079	0.0164	0.6409
Gini Index (%)	-0.0131	0.0104	0.2105	-0.0090	0.0103	0.3840
Per Capita GDP ($1000's)				0.1368	0.0587	0.0208
1970 - 1984						
(Intercept)	5.1206	0.2935	0.0000	5.2024	0.2994	0.0000
Time (Year - 1970)	0.0621	0.0024	0.0000	0.0775	0.0104	0.0000
Smoking 85+ (%)	0.4931	0.3879	0.2052	0.5739	0.3894	0.1422
Fat Consumption (%)	-0.0231	0.0092	0.0129	-0.0252	0.0093	0.0074
Gini Index (%)	0.0071	0.0045	0.1188	0.0083	0.0046	0.0715
Per Capita GDP ($1000's)				-0.0229	0.0151	0.1305
1985 - 1999						
(Intercept)	4.4710	0.6186	0.0000	4.2078	0.9599	0.0000
Time (Year - 1985)	0.0477	0.0031	0.0000	0.0462	0.0108	0.0000
Smoking 85+ (%)	-0.7742	0.2924	0.0088	-0.3990	0.3165	0.2090
Fat Consumption (%)	-0.0028	0.0125	0.8243	-0.0161	0.0128	0.2111
Total Calories (100's)				-0.0165	0.0205	0.4227
Alcohol (liters)				0.1916	0.0715	0.0081
Alcohol^2				-0.0089	0.0029	0.0025
Gini Index	0.0407	0.0168	0.0159	0.0477	0.0165	0.0043
Per Capita GDP ($1000's)				-0.0045	0.0137	0.7459

Source: e85 - Human Mortality Database, and Smoking Exposure - standardized smoking exposure for age group 85+ estimated from WHO Mortality Database data. For remaining data sources, see Table 6.

Notes

[1]The principal differences between this classification of causes and the classification systems employed by Himes and Meslé and Vallin are that here mortality from malignant neoplasms is divided into deaths from respiratory and non-respiratory cancers to help identify any possible changes due to causes such as lung cancer that are strongly associated with cigarette use and that deaths from Alzheimer's Disease and senility are included in a single, separate category to help track changes in the classification and occurrence of deaths from these causes over time and in various countries.

[2]Mean estimated smoking exposure for the age groups from 65 to 84 is used in the analyses for e65 and the estimated exposure for the age group 85+ is used in the analyses for e85. Total caloric intake and animal fat consumption data from UN FAO food balance sheets and alcohol consumption figures from the WHO for 1965, 1975, 1985, and 1995 are used as estimates of consumption for countries for the periods 1955 to 1969, 1970 to 1979, 1980 to 1989, and 1990 to 1999. The conversion factor of 1 gm animal fat = 9 kCal is used for the calculation of the proportion of per capita daily calories obtained from animal fat. Values for the Gini Index for are calculated as the mean of figures from the World Bank's World Income Distribution Gini Database designated as being of "accept" data quality for each country for the periods 1955 to 1965, 1966 to 1975, 1976 to 1985, and 1986 to 1999.

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