THE TRIUMPH OF COHORT-EFFECTS IN THE EXPLANATION OF MORTALITY CHANGE: A NEW AGE-PERIOD-COHORT ANALYSIS OF ADULT CAUSE-SPECIFIC MORTALITY IN THE UNITED STATES*

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Abstract

This study examined the temporal changes in U.S. adult mortality by cause of death and sex over a 40-year period in the second half of the 20th century. It applied the Intrinsic Estimator to log-linear APC models to simultaneously account for age, period, and cohort variations in mortality rates for four leading degenerative causes of deaths – heart disease, stroke, lung cancer, and female breast cancer. The results show that the large mortality reductions since the late-1960s continued well into late-1990s and these reductions were predominately contributed by cohort effects. Cohort effects are found to differ by specific causes of death examined, but they generally show substantial survival improvements, especially for cohorts born since 1900. Implications of these results are discussed with regard to epidemiological transition theory, the theory of technophysio evoluation, differential cohort accumulation of health capital and life time exposures to socioeconomic and behavioral risk factors, and period changes in diagnostic techniques and medical treatment.

The Triumph of Cohort-Effects in the Explanation of Mortality Change: A New Age-Period-Cohort Analysis of Adult Cause-Specific Mortality in the United States

INTRODUCTION

Recent increases in life expectancy at birth and at advanced ages have been substantial in the United States. Large declines in death rates have been documented for the period from the late 1960s to the 1980s and early-1990s. Temporal changes in mortality have not been the same for different causes of deaths and the two sexes. At present, the sources of these demographic variations in U.S. adult mortality are not well understood. One way to expand our insight is by distinguishing age, period, and cohort effects in temporal changes of overall and cause specific mortality. Age-period-cohort (APC) analyses play an important role in the search for particular agents or risk factors of disease and mortality by depicting the whole complex of social and environmental factors that create these risk factors. The theory of epidemiological transition (Olshansky and Ault 1986; Omran 1971, 1982) has been used to specify models of changes in morbidity and mortality patterns in developed countries in the past centuries. But after a number of industrialized societies, including the U.S., have achieved the final health and economic state depicted by the models, current theory does not indicate what further health and demographic changes to expect and how chronic disease morbidity and mortality patterns can be expected to change. APC analyses can help to generate theoretical insights into continuing mortality reduction patterns in countries that achieved extreme economic prosperity and high life expectancies.

The examination of mortality patterns by cause links total mortality patterns with underlying chronic diseases. The cause-of-death structure facilitates analyses of the components of recent mortality reductions and the mechanisms by which the advances of life expectancy

were achieved. The contribution made by different causes of death to the recent decline of mortality can be assessed by identifying the age-period-cohort patterns of cause-specific mortality. First, the composition of deaths by cause bears directly on the age patterns of mortality because the incidence of mortality by cause varies substantially with age (Preston 1976; Sullivan 1973; McNown and Rogers 1992). Second, the ebbs and flows of cause-specific mortality rates are the product of period and cohort factors (Manton, Stallard, and Corder 1997). U.S. mortality from 1954 to 1968 showed few declines, but declined steadily after 1968. Those changes represent public health and medical care effects for the period from 1950 to1990. Changes in deaths due to chronic diseases also reflect long-term health effects of the processes of differential accumulation of lifetime exposures to risk factors, e.g., education, physical activity, diet and nutrition, age at first pregnancy, and smoking (Hummer, Rogers, and Eberstein 1998), which suggest clues to the pathways responsible for cohort mortality differentials. Third, temporal trend analyses of disease-specific mortality rates are frequently utilized in etiological investigations to provide clues to diseases themselves (e.g., Holford 1991). When such analyses are conducted on a comparative basis between different diseases and populations such as two sexes, the results can be used to identify populations with significantly higher or lower incidence of disease so that the search for risk factors associated with the disease may be more selective and specific.

APC analyses have received considerable attention in previous demographic analyses. In a sweeping review of age-period-cohort analyses in demography, Hobcraft, Menken, and Preston (1982) emphasized the relevance of cohort effects to studies of human mortality and disease rates. They noted that “in mortality analyses, it seems clear that in many cases cohort effects are biologically plausible and have been demonstrated in a variety of ways that may not be statistically rigorous but are nevertheless convincing” (Hobraft et al.:19). While descriptive

approaches mainly documented the period or cohort changes of age-specific death rates, age-period-cohort models have been used to separately estimate the effects of these three variables. They noted that one problem in the early age models was that the cohort effects were not explicitly controlled. The other demographic work that attempted to identify period and cohort effects did so only loosely because the linear dependence among age, period, and cohort components creates a model identification problem for three-factor multivariate analysis. Therefore, previous findings regarding the relative importance of cohort effects in explaining mortality change are at best tentative.

In sum, examinations of recent mortality change require both up-to-date population level mortality data and a rigorous modeling effort. In this study, I analyze temporal patterns of adult mortality due to four leading causes of death – heart disease, stroke, lung cancer, and breast cancer – in the United States for the second half of the 20th century. Using population mortality data from men and women aged 20 to 95+ over a 40-year period from 1960 to 1999, this study aims to test hypotheses of role of period and cohort effects in recent mortality decline. It conducts a new age-period-cohort analysis based on the log-linear Intrinsic Estimator (IE) model (Yang, Fu, and Land 2004) and provides a model-based summary of temporal effects of mortality that can be delineated into changes across ages, over time and among birth cohorts.

TEMPORAL VARIATIONS IN U.S. ADULT MORTALITY

Substantial mortality reduction in the United States for a large part of the last 100 years has been widely documented (e.g., Crimmins 1981; Manton et al. 1997). We know, however, relatively less about the evolution of mortality experiences attributable to distinct impacts of age, period, and cohort. Our understanding of sources of recent mortality reductions is confined to

changes in one or two of these demographic components. The cohort effect is less frequently tested, but its presence implies that certain assumptions currently used by demographers and other social scientists to analyze factors contributing to mortality declines can be misleading. For instance, it is frequently assumed that rates of mortality declines over time are equal across birth cohorts. It is also assumed that these declines depend on rates of changes in period-specific conditions such as economic advance and health care technology that are exogenous to individuals and independent of the year of birth. These assumptions neglect cohort effects and greatly simplify estimations, but are increasingly inconsistent with accumulating evidence of cohort changes in a variety of health outcomes that predict mortality (Fogel 2004).

Proponents of cohort analysis argue that the mortality change process needs to be considered to depend on all three of age, period, and cohort and requires formulation of models in which outcomes are determined jointly by the three (Mason and Fienberg 1985; Mason and Smith 1985). Although efforts have been made in previous demographic studies to determine if there are distinct contributions of age, period, and cohort variation to these mortality changes, few of them succeeded in taking all three dimensions of temporal trends into account simultaneously in multivariate analysis. The methods on which previous studies of period and cohort effects are based may not be statistically rigorous and powerful. It follows that the findings are not complete and accurate, and consequently, difficult to interpret. For those studies that did explicitly model the APC effects, strong assumptions and sensitivities of results to choice of assumptions have largely prohibited reliable and consistent patterns to be revealed. Two interesting questions remain. First, to what extent did the large mortality declines since the 1960s continue into the last decade of the 20th century? Second, what are the patterns of mortality risks that are uniquely attributable to age, period, and cohort factors?

Period Effects and Cohort Effects

Age is undoubtedly the mostly studied source of variation in vital rates. Mortality risks increase with the biological process of aging and the age patterns of mortality change exhibit considerable regularities across nations and historical time (Hobcraft et al. 1982). Of central focus in the APC analyses is the distinction of the other two components of the temporal effects of mortality change, i.e., period and cohort differences, through the use of APC models.

The *period effect* relates to variation in mortality by time at which the death is recorded that influences all age groups. It subsumes a complex set of historical events and environmental factors such as world wars, economic crisis, famine and pandemics of infectious diseases, which influence mortality of all society members (Omran 1971, 1982). It may also arise with public health efforts and medical technology breakthroughs that lead to reductions in mortality rates of all ages (Frost 1939). In addition to these direct effects, there may also be changes in disease classification or diagnostic techniques that affect the mortality outcomes of certain diseases (Tarone, Chu, and Gaudette 1997).

The *cohort effect* represents the influences of endogenous (e.g., biological) and exogenous (e.g., socioeconomic and behavioral) factors that are present at the moment of exposure early in a birth cohort's life and continuously accumulate over the life course to produce health and mortality risk differences (Ryder 1965). This type of effect is particularly relevant in the examination of chronic diseases and cancer, wherein long-term exposure to a carcinogen is the major cause of the disease (Jemal et al. 2001).

To the extent that period and cohort effects are aggregates and proxies of different sets of structural correlates of mortality, such distinctions as these are especially valuable for better

identification and understanding of underlying social and environmental factors that are amenable for modifications or reversions.

Early empirical studies that explicitly tried to distinguish period and cohort effects of mortality are principally descriptive and rely on external information or reasoning, but they suggest the existence of independent cohort effects (Hobcraft et al. 1982). Distinct period and cohort patterns in general mortality have also been found in recent studies of several industrialized countries, including France (Wilmoth 1990), Italy (Caselli and Capocaccia 1989), and U.K. (Robertson and Ecob 1999), based on different APC modeling techniques. While no study tested of the distinct role of period versus cohort effects in recent changes in U.S. mortality, the weight of the evidence points to the possibility that period and cohort factors jointly contribute to survival improvements in the U.S. population. Findings, however, are not clear on the shapes of period and cohort effects and the relative importance of these effects as driving forces of mortality reduction.

Mortality decline in recent human history can be related to major advancement in economic development, standard of living, and medical measures (Mckeown 1976; Preston 1975). It has been argued that improving nutrition and reducing exposure to diseases that are associated with economic development are likely to produce cohort effects in a population, while the introduction of medical techniques is likely to produce period effects. But studies also suggest that the overall contribution of medical measure is small for period effects, but large for cohort effects. For example, specific birth cohorts in both Italy and England and Wales benefited substantially from these measures (e.g., Collins 1982). These findings are mostly based on data from the time period of 1900 to 1960s or restricted to tuberculosis mortality in a few European

countries. Less is known regarding period-specific and cohort-specific influences that operate on mortality in the U.S. in the latter half of the 20th century.

It can be hypothesized that period effects continued to contribute to recent mortality change, but the potency of such effects may have decreased relative to those in the first half of the last century. Although there were continuous mortality reductions during most periods in the last 100 years, there is also evidence that the decline was much more dramatic early in the 20th century than periods after 1960 (White and Preston 1996). This indicates that social forces that contributed to mortality decline in previous periods such as the nation's level of economic development, improved standard of living, or advancement in medical measures may have become much less responsible for improvement in survival during this period. This is largely due to reductions in or elimination of many infectious diseases as causes of death (Omran 1982). Because period effects result from variations in forces that affect the mortality rates of all age groups simultaneously, large period effects are more likely to occur if diseases being considered are more acute or infectious in nature and kill individuals of a wide age range alike. The advent and diffusion of new medical technology may effectively prevent the spread of such diseases and elevate survival chances of all affected individuals in a relatively short time. As the U.S. has reached the fourth and final stage of the epidemiological transition (Olshansky and Ault 1986), i.e., the age of delayed degenerative diseases, the leading causes of death have shifted to chronic diseases that disproportionately affect adults of older ages who have higher risks of mortality. And the effects of medical measures may take a longer period of time to translate into massive improvement in survival detectable at the societal level to the same extent as those for acute diseases.

Meanwhile, it can be hypothesized that cohort effects contributed to recent mortality change independent of period effects. Whereas period-specific conditions affect death rates at all ages, cohort-specific conditions tend to take effect at early ages and depend on subsequent length of exposure to risk factors. In a study of English death rates of 19th century, Kermack, McKendrick, and McKinlay (1934) attributed the regularity in cohort mortality patterns to a cohort's health risk as determined by environmental conditions in its first 15 years of life. The well-known study of tuberculosis mortality conducted by Frost (1939) also points to the importance of early life influences on cohort experiences rather than current conditions in the case of a disease that has long latency. This conceptual characteristic of cohort effects has important implications for chronic diseases as causes of death that span longer time intervals since the age of onset.

The theory of technophysio evolution, recently postulated by Fogel and colleagues (Fogel 2004; Fogel and Costa 1997), also provides alternative explanations to a period effects hypothesis that attributes the mortality decline between 1970 and 1990 to health interventions during that period. The theory implies that over the course of 20th century, individuals' health capital changed with the year of birth. More recent cohorts fared substantially better in the initial endowment of health capital at birth and have lower rates of depreciation of the stock of health capital. These led to improved physiological capacities in later cohorts that also bold well for effectiveness of medical treatments. This is consistent with mounting evidence in recent research in demography of aging that successive birth cohorts experience later onset of chronic diseases and disabilities (Crimmins, Reynolds, and Saito 1999; Freedman and Martin 1998). Fogel (2004) argued that improvements in life expectancy may not be exclusively due to medical advancements during those years, but may also reflect improved physiologies by later cohorts as

a result of exposures to improvements in technologies in health care, nutritious diets, personal and public health practices long before 1970.

In addition, socioeconomic differentials, such as difference in education, affect mortality differentials (Kitagawa and Hauser 1973). Large increments in average years of education can be interpreted as both a period and cohort phenomenon. But from the vantage point of health capital, the increasing educational attainment in successive birth cohorts found in recent studies (e.g., Hughes and O'Rand 2004) suggests corresponding large reductions in cohort mortality levels that are distinct from those due to period-specific conditions. Increased education not only is related to engagement in lifestyle choices and behaviors that lower the exposure to stress and risk factors of chronic illness and mortality, but also enhances other socioeconomic resources that allow greater accesses to health insurance and medical care (Land and Yang 2005). All these may increase the amount of health capital available to later birth cohorts early on in life and have lasting impact on their mortality outcomes. In fact, cohorts with more years of schooling have substantial survival advantages (Lauderdale 2001).

Mortality Caused by Degenerative Diseases

Cardiovascular diseases (CVD) and cancer together cause more than two-thirds of all deaths in the U.S. As both direct and indirect indicators of the influences of a myriad of social and behavioral, economic, and environmental factors on population health, CVD and cancer mortality rates are crucial for understanding the longevity of men and women. Recent analyses show that cancer has taken the place of heart disease to become the No. 1 killer of Americans under age 85, based on age-adjusted death rates (Jemal, Murray, Ward et al. 2005). Among all the cancer death rates, lung cancer is the leading cause of deaths for both sexes and is expected to account for 31% and 27% of all cancer deaths in 2005 for men and women, respectively.

Breast cancer has receded to be the second killer for women since 1987 (Jemal et al. 2005: Figures 1, 4 and 5). The four degenerative causes of mortality that represent particular chronic conditions: heart disease, stroke, lung cancer, and female breast cancer, are of interest to the current analysis also because their temporal variations have different public health and clinical characteristics and different implications for socioeconomic changes during the past 40 years.

Previous studies of changes in CVD mortality are mainly descriptive. Recent analyses of period changes of age-specific mortality patterns suggest declines in heart disease and stroke mortality for both genders from 1962 to 1995, but there were more rapid declines for males (e.g., Manton 2000: Figure 2). The declines by cohort were monotonic and continuous for both heart disease and stroke mortality in the U.S. (Manton 2000) and stroke mortality in Sweden (Peltonen and Asplund 1996). As with total mortality rates, declines in CVD mortality rates have been projected to continue to the post-1990 period until 2000 (Crimmins 1981). Since the launch of Framingham Heart Study in 1948, several major risk factors for CVD have been established. These include hypertension, high low-density lipoprotein (LDL) or bad cholesterol levels, diabetes, low socioeconomic status, cigarette smoking, a lack of physical activities, and obesity (Barrett-Connor 1997; Lerner and Kannel 1986). Increasing knowledge about prevention, treatment, and diagnosis of hypertension, together with diffusion of current medical techniques should largely decrease CVD mortality (CDC-MMWR 1999). The obesity epidemic in recent years, on the other hand, may reduce the rate of decline (Mokdad et al. 1999; Rogers, Hummer, and Krueger 2003). Period and cohort changes in distributions of specific risk factors may lead to corresponding rise and fall of period and cohort effects of CVD mortality.

Among recent studies of various causes of death, lung cancer and female breast cancer mortality have received the most attention in temporal analyses using APC regression models.

They both have been shown to have characteristic period and cohort patterns. Inconsistencies in these patterns, however, preclude conclusive inferences of the role of period and cohort effects.

Compared to descriptive studies, analyses fitting APC models to lung cancer mortality data were better able to delineate the temporal effects. The power of APC modeling is best illustrated by findings of the unique *cohort patterns* that were ambiguous otherwise, that is, inverse-U shaped curves for the cohort effects across developed countries, with varying peaks (Gardner and Osmond 1984; Jemal, Chu, and Tarone 2001; Lee and Lin 1996; Tarone and Chu 2000a). Cohort effects on lung cancer mortality suggest the heavy influence of gender-specific risk factor exposure and behavioral differences, especially cigarette smoking, in the life experience of specific birth cohorts. Studies have found diverse experiences and patterns of smoking across cohorts and the presence of sex differences in cohort smoking patterns, with recent increases in smoking in later cohorts of females (Harris 1983; Zang and Wynder 1996). The cohort and gender differences in cigarette use have been related to social patterns of cigarette diffusion processes. Earlier cohorts experienced periods of growing cigarette use at early adulthood and adopted the addictive behavior that would shape later patterns of smoking (Pampel 2005). Male cohorts adopted cigarettes in large numbers earlier than women and the pattern preceded women's by a decade or two (Lopez 1995). A gender gap in lung cancer mortality that first increased and then decreased reflected such a gap in diffusion of smoking (Pampel 2003). Therefore, it can be expected that the peak of the inverse U-shaped male cohort mortality curve precedes that of the females and the mortality decline in male cohorts started earlier than that in females.

Ambiguities exist between descriptive and model-based results as well as among the model-based results themselves regarding *period effects* of lung cancer mortality. Descriptive

studies generally show that lung cancer mortality tended to increase over the period of last 40 years (Jemal et al. 2005; Manton 2000; Pampel 2003; Trovato and Lalu 1998). In contrast, Gardner and Osmond (1984) found virtually no period effect when cohort effects are considered in APC models of lung cancer mortality in England and Wales from 1950 to 1980. They concluded that the period of time appeared to be not the major influence and offered a "much modified interpretation", i.e., the experience of the different birth generations contributed to the death rates at different time periods, which accorded with known changes in smoking behavior in women. Clayton and Schifflers (1987a) examined Belgian female lung cancer mortality and found monotonically increasing period effects that suggests a log-linear trend. As for studies of the United States, Kupper and colleagues (1985) estimated APC models of white male lung cancer mortality and found large fluctuations in numerical estimates of the effect coefficients using different arbitrary constraints on model parameters. Recently, Jemal and colleagues (2001) found inverse U-shaped period effects for both genders that showed sharp decreases around 1990. The absolute magnitudes of the effects, however, are based on very small scales.

Descriptive studies documented fairly small *period changes* of age-specific and age-standardized breast cancer mortality rates (Jemal et al. 2005; Manton 2000). Model-based results show different trends of the period effects: there were continuous declines in mortality rates from the 1950 to 1970s in the U.S., Japan, and Canada (Clayton and Schifflers 1987b, Tarone and Chu 2000b; Tarone et al. 1997), slight increases in 1980s in both the U.S. and Canada, and decreases in recent years from 1990 to 1995 for Canadian and white U.S. females (Tarone and Chu 2000b; Tarone et al. 1997). Two period factors may be responsible for the temporal trends in female breast cancer mortality. First, the increase in the 1980s coincided with a rise in diagnosis via mammography. Second, medical interventions such as increased use of tamoxifen therapy and

early detection can temper the increases and even lead to decreases in deaths from breast cancer (Tarone et al. 1997).

There is also evidence of large *cohort effects* from regression studies that were not found by descriptive studies. In a study of Japanese women age 25 to 79 from 1955 to 1979, Clayton and Schifflers (1987b) found M-shaped cohort effects with two peaks in 1900 and 1935, increases before 1900, decreases in between, and a slight decline after 1940. The cohort effects in the U.S. were found to be concave, with steep increases in cohort mortality risks until the peak in 1925, followed by moderations until mid 1940s, and a marked decrease for white women and moderate decrease for black women born after 1950 (Tarone and Chu 2000b; Tarone et al. 1997).

Fertility related behaviors, such as reductions in completed pregnancies, delaying of childbearing to later ages, are socially and culturally regulated and determine the length of hormonal exposure. The exposure of breast tissue to endogenous estrogens is a known risk factor for breast cancer (MacMahon, Cole, and Brown 1995). Recent demographic changes of women may be consequential for cohort changes in the exposure to this risk factor. There were large cohort declines in the proportion ever marrying and increases in the mean age at marriage among female cohorts born 1888 – 1950 (Schoen, Urton, Woodrow, and Baj 1985). Delayed first marriages and decreased durations of marriages imply delayed births and fewer life-time births. In addition, labor force attachment accelerated among more recent cohorts, especially baby boom women (Spain and Bianchi 1996). And increased labor force participation is inversely related to fertility (Schoen et al. 1997). There is evidence that recent cohorts of women had first births as well as high-order births at progressively later ages (Spain and Bianchi 1996; Yang and Morgan 2004). It is, therefore, possible that large changes in female cohort fertility patterns lead to cohort

variations in breast cancer mortality (Tarone et al. 1997). But this hypothesis was not strongly supported in previous studies. Manton (2000) found only small cohort differences in age-specific death rates from breast cancer. Tarone and colleagues (1997, 2000b) found a decrease in cohort mortality for mothers of Baby Boomers (cohorts 1924 – 1938) that coincided with increasing fertility rates following the World War II, but stable or decreasing rates for late Baby Boomers (cohorts born1950 – 1964) who experienced the reverse trend in fertility due to increasing female labor force participation and uses of oral contraceptives.

While extensive methodological discussions of APC multiple classification/accounting model specifications and identification problems are available elsewhere (see e.g., Mason and Wolfinger 2002; Robertson, Gandini, and Boyle 1999), the empirical studies reviewed above give examples of both the power of such models in delineating cohort effects from the age and period effects and the problems associated with inconsistencies of findings due to different arbitrary identifying constraints used. This study employs a new statistical approach – the Intrinsic Estimator (IE) method for APC regression analyses (Yang et al. 2004) – to provide new evidence on the age, period, and cohort effects of the U.S. adult mortality. These APC analyses aim to improve previous estimates of temporal effects of U.S. adult mortality in several regards. First, the improved modeling framework yields results that can serve as objective criteria for selecting the best among alternative summaries of data on mortality and provides a test of whether the observed pattern revealed in descriptive analysis is real or random. Second, the empirical assessment of the evidence for recent mortality trends may help generate theoretical insights into continuing mortality reductions in the United States. Delineation of different sources of variations in death rates brings clarity to the specific components of mortality dynamics in recent historical period and highlight important period and cohort specific factors

that are operational in the declines in the population risks of chronic diseases and improvement in general health conditions. Third, monitoring trends in mortality using the most recent data available helps to reveal previously unknown forces affecting health and mortality conditions in the U.S. that likely will continue into the 21st century. If major period changes and cohort differentials are identified, we may be able to determine if recent declines in mortality are likely to continue and to what levels.

DATA AND METHODS

Data

The data on numbers of deaths are from the Multiple Cause of Death public use files of all U.S. death certificates reported to the Centers for Disease Control and Prevention/National Center for Health Statistics (NCHS) for the period from 1960 to 1999. Deaths at all ages are recorded in those files and this analysis of adult mortality uses the data on ages 20 and above (95+). The underlying cause of death is reported on the death certificate by the physician or other certifier. The medical condition for each cause of death is numerically coded according to the manual of the International Classification of Diseases (ICD), which changed during the 40-year period. The changes of the ICD codes from the seventh to the tenth revision are summarized in Appendix A for the four causes of deaths examined in this study, namely, heart disease, stroke, lung cancer, and female breast cancer. Previous coding studies from NCHS suggest that specific diagnostic categories across different ICD revisions are comparable (e.g., Klebba 1980). Recent studies also found ICD revisions have little effect on the temporal trends for the four causes in that these cause-of-death categories are leading causes of deaths in the U.S. and represent

catastrophic health events, so they are much less subject to major diagnostic errors over time (CDC-MMWR 1999; Manton 2000).

Data on population exposure-to-risk by five-year age intervals and sex from 1960 to 1999 were taken from U.S. Census Bureau estimates provided by the Human Mortality Database (HMD) of the University of California, Berkeley.[1] For most of the age range, the HMD used either linear interpolation of population estimates or intercensal survival methods. At ages 80 and older, HMD used population estimates computed using the methods of extinct cohorts and survivor ratios. For details of calculations, see the Methods Protocol for the HMD (Wilmoth 2002). Death rates were calculated as the ratio of number of deaths reported to population exposure for five-year age groups and five-year time periods.[2] There are 16 five-year age groups, from 20 – 24 to 95+ and eight five-year time periods, from 1960 – 1964 to 1995 – 1999. This yields 23 successive 10-year birth cohorts whose death counts and rates fall in diagonal cells of the arrays. Each birth cohort is labeled by its central birth year that ranges from 1865 to 1975.

Analytic Approaches: Descriptive and Statistical APC Analysis

Descriptive Analysis

The majority of previous studies rely on two analytic approaches that attempt to depict the time trends by age, period, and birth cohort. One common practice uses summary measures that are independent of age composition, such as standardized indices (*age standardized death rates*) arrayed by time periods. This is exemplified in Figure 1 which shows female age-standardized death rates due to four causes calculated for each period by adjusting the crude

[1] Although the NCHS data on death counts by causes are available until 2002, 1999 is the last year for which the population exposure at risk is present in HMD. Therefore, the current analyses can only use the data to the most recent five-year periods for which data are available, i.e., 1995 - 1999.

[2] The rates were also computed for single-year of age and for each year to assess the consistency of the aggregated rates. The results are similar.

death rates to the 1960-1964 female population age 20 and above. There were large declines in the total mortality rates (not shown) and rates of mortality due to heart disease and stroke and a slight decrease in breast cancer death rates. During the same period, there was a sharp increase in female lung cancer death rates. The trends hold for males, except that the increase in male lung cancer mortality was much less pronounced.

An alternative device is to produce a *graphical display of the table of age-period-specific rates* or *age-cohort-specific rates*. The former is illustrated in Figure 2 that shows age-specific rates of mortality from the four causes by sex for the beginning five-year period, 1960 – 1964, and ending period, 1995 – 1999. Changes shown in the figure using the logarithmic transform of the rates can be interpreted as the proportional increase in rates with age. Both sexes experienced declines in age-specific death rates from heart disease and stroke over time for all adult ages, but the declines decreased in old ages for heart disease mortality. There is evidence for female heart disease mortality advantage before middle ages and stroke mortality disadvantages at old ages, a phenomenon noted in many studies of different national populations that indicates the effect of some biological factors such as hormonal changes that favor premenopausal women (Barrett-Connor 1997; Manton 2000). Graphs of cancer mortality rates show drastically different pictures. Both sexes experienced increases in death rates from lung cancer over the 40-year period. Whereas the increases for males were moderate and did not occur until the age of 55, female lung cancer death rates increased substantially and continuously from early adulthood through old age with no marked decrease. By contrast, the period changes in age-specific female breast cancer death rates were fairly small. A modest cross-over of the two age curves in two periods indicates improvement of survival that was restricted to ages of 20 to 65 and a slight increase in the postmenopausal breast cancer mortality.

The above two approaches do not explicitly consider an age-period-cohort modeling framework. Both are descriptive and share two disadvantages that are evident in Figures 1 and 2. Firstly, they only describe variation in the rates attributable to factors associated with either period of death or birth cohort. Secondly, neither summarizes the rate table satisfactorily. Standardization ignores different trends at different ages and graphical methods represent all available rates providing no summary at all (Osmond 1985). In addition, they each have their own limitations. Standardized crude rates depend on the selection of an appropriate standard population age composition and give more weight to the older ages (Schoen 1970). The substantial population aging makes the choice even more difficult. Graphs of rates from two-way age-by-period tables are helpful for *qualitative* impressions about temporal patterns, but they provide no *quantitative* assessment of the source of mortality change (Kupper et al. 1985). For example, in Figure 3, the curve of age-specific lung cancer death rates for females in any given time period, say 1995-99, cuts cross a number of birth cohort curves, such as 1900, 1905, 1910, and 1920. Therefore, the shape of the period curve is affected by both varying age effects and cohort effects. The question of how these effects operate simultaneously to shift period curve motivates the use of statistical regression modeling.

[Figures 1 – 3 about here]

APC Accounting Models and Intrinsic Estimator Analyses

The multivariate technique known as APC multiple classification/accounting models (e.g., Mason et al. 1973) has been used as a popular tool to address the question: what are the net trends in the data across age, period and cohort categories? The model for the tabular mortality data can be written in log-linear regression form as

$$\log(R_{ijk}) = \log(D_{ijk} / N_{ijk}) = \mu + \alpha_i + \beta_j + \gamma_k \qquad (1)$$

where R_{ijk} denotes the death rate in age-period-cohort cell (i, j, k), D_{ijk} denotes the number of deaths and is assumed to be distributed as a Poisson variate, and N_{ijk} is the population or exposure-at-risk, the log of which is also termed as the offset or adjustment for the log-linear contingency table model, μ denotes the intercept or adjusted mean rate, α_i denotes the *i-th* row age effect for $i = 1,\ldots,a$ age groups, β_j denotes the *j-th* column period effect for $j = 1, \ldots, p$ periods, and γ_k denotes the *k-th* diagonal cohort effect for $k = 1,\ldots,(a+p-1)$ cohorts, with $k=a-i+j$. In conventional practice, one of each of the α_i, β_j , and γ_k coefficients is set to zero as a "reference" age, period, or cohort category against which the estimated coefficients for the other categories can be compared. The key problem in APC analysis using model (1) is the "identification problem" that arises in the application of model (1) to tables of deaths rates tabulated by age and period. The linear relationship between the age, period and cohort variables (period = age + birth year) translates to a design matrix that is singular and the estimator of the parameter vector of model (1) that is not unique.[3] Therefore, it is not feasible to separately estimate the three effects without assigning certain identifying constraints.

One possible solution is to just estimate a reduced age and period two-factor model that contains no cohort effects and can be written as

$$\log(R_{ij}) = \log(D_{ij} / N_{ij}) + \mu + \alpha_i + \beta_j \qquad (2)$$

Because cohort effects can be interpreted as a special form of interaction effect between the categorical age and period variables, model (2) rests on the assumption of no interaction effect (Fienberg and Mason 1985). In particular, the expected rate in age by period cell (i, j) is modeled a function of the *marginal* or gross effects of age i and period j only, and not also of the cell-

[3] Denote the design matrix of model (1) as X and the outcome as Y, because the exact linearity between age, period, and cohort variables leads to an X that is one less than full rank, the inverse of $X^T X$ does not exist. It follows that the least square estimator, $(X^T X)^{-1} X^T Y$, does not have a unique solution.

specific effect, such as γ_{a-i+j}, which is a function of both *i* and *j*. Violation of this assumption can be detected by plots of age-specific death rates by time period and a lack of parallelism among these curves suggest birth cohort effects that are operating (Kupper et al. 1985). The same applies to the period effect as a particular type of age-cohort interaction (Holford 1991). Figure 4 shows evidence of non-parallelism among age curves by period in plots of female cause-specific mortality, confirming the existence of birth cohort effects. Age curves by cohort (not presented) show non-parallelism only in plots of lung cancer and breast cancer mortality, but not in those of heart disease and stroke mortality. Therefore, it is likely that period effects are more important in some causes of deaths than others. So results of preliminary graphical analyses argue against the plausibility of reduced two-factor models.

[Figure 4 about here]

The most widely used approach to solving the identification problem in full APC models is constraining two or more of the age, period, or cohort coefficients to be equal (e.g., Mason et al. 1973; Fienberg and Mason 1985). This conventional approach and other variations of this approach place arbitrary constraints on the coefficients and yield coefficient estimates that are highly sensitive to the choice of model identifying constraints (Mason and Wolfinger 2002; Robertson et al. 1999). Therefore, analysts have been advised that any statistical modeling of APC data should be carried out in conjunction with a detailed descriptive analysis (Kupper et al. 1985; Mason and Smith 1985).

Recently, a promising alternative modeling approach was described and evaluated by Yang and colleagues (2004) and is termed the *intrinsic estimator* (IE). Using vector space projection, this approach yields a unique solution to model (1) that is determined by the Moore-Penrose generalized inverse and removes the arbitrariness of linear constraints on parameters. It

has been shown that each of the infinite estimator of parameter vector of model (1), denoted as $\hat{b}$, can be decomposed into two parts that are orthogonal or independent to each other in the parameter space and written as

$$\hat{b} = B + tB_0, \qquad (3)$$

where *t* is an arbitrary real number, B_0 is the eigen-vector corresponding to the zero eigen-value of the design matrix, [4] and *B* is a special estimator called the *intrinsic estimator*. In vector space terminology, the identifying constraint imposed by the IE approach to estimating the model is a constraint such that the direction in parameter space defined by the unique eigen-vector B_0 in the null space of the design matrix has zero influence on the parameter vector to be estimated (i.e., by setting $t = 0$ in Eq. 3). This constraint of removing the influence of null vector B_0 is plausible in that B_0 is completely a function of the dimension of the age by period data matrix and does not relate to mortality rates themselves. Projection of the estimators of (3) onto to the non-null space yields the IE:

$$B = (I - B_0 B_0^T)\hat{b} \qquad (4)$$

For a fixed number of time periods of observations, Yang et al. (2004) showed that the IE has desirable statistical properties such as unbiasedness and relative efficiency in estimating the projected parameter vector compared to conventional estimators based on linear constraints on the coefficients.

This study employs the IE method to a substantive analysis of cause-specific mortality data. Comparisons of modeling results with those displayed in the descriptive analysis can be used as an empirical assessment of the utility and sensibility of the new modeling approach. The intrinsic estimates of model regression coefficients and standard errors are computed using the

[4] The explicit form of B_0 is given in Yang et al. (2004):106.

principal components regression algorithm introduced by Yang et al. (2004) and S-Plus software program (Venables and Ripley 2000). The computational algorithm is included as Appendix B.

RESULTS AND FINDINGS

For purpose of model selection, I first estimated six reduced log linear models: three gross effects models, namely, model A for age effects, model P for period effects, and model C for cohort effects; and three two-factor models, one for each of three possible pairs of effects, namely, AP, AC, and PC effects models. I then estimated the full APC model where all three factors are simultaneously controlled. The IE method was employed for model identification. Goodness-of-fit statistics were calculated and used to select the best fitting models for male and female mortality data. Because likelihood ratio tests tend to favor models with a larger number of parameters, I calculated two most commonly used penalized-likelihood model selection criteria, namely, Akaike's information criterion (AIC) and Bayesian information criterion (BIC), which adjust the impact of model dimensions on model deviances. Recent methodological literature suggests that although the two criteria have different foundations and penalty terms[5], useful information for model selection can be obtained from using both together and selecting models favored by both criteria (Kuha 2004).

[Table 1 about here]

Table 1 presents the comparisons of the goodness-of-fit statistics from above models using the AIC and BIC. The smaller the AIC and BIC, the better the model fit. Because age is the most important source of variation in mortality, models with no age effects (P, C, and PC) have shown to be significantly lack of fit and are omitted from Table 1. For both male and female data and all

[5] AIC = Deviance + 2DF; BIC = Deviance + log(N)*DF, where DF is the degree of freedom used by model parameters.

causes, the AIC and BIC statistics converged in the results that the full APC models fit the data significantly better than any of the reduced models.

The results from the full APC models of total mortality using the IE method are displayed in Table 2. It summarizes the model coefficients, their standard errors, model deviance, and overdispersion coefficient. To facilitate the comparisons of the age, period, and cohort effects for different causes by sex, Figures 5 to 9 plot the point estimates of the effect coefficients estimated from each model.[6] These plots compare the coefficients of successive categories within the age, period, and cohort classifications, thus showing the net trends of mortality along the three dimensions. The results largely accord with the observed patterns from descriptive displays of the data, but they also offer additional insights of distinct sources of mortality change. A caveat for interpreting the separate results for males and females is that these are not absolute levels of mortality rates, but model effect coefficients. The former can be compared directly based on the same scale, such as deaths per 100,000 persons. But the latter only indicate relative trends within each gender-specific population.

Total Mortality

The IE analysis of the total mortality change summarized in Figure 5 shows the dominance of age and cohort effects over the period effect. The shapes of the age effects bear close resemblance for males and females, but the females show steeper increases in (but not necessarily higher) mortality in old ages. Similar to the finding of Yang et al. (2004), when all three factors are considered in the APC models, there seems to be only minimal period effects, but substantial cohort effects. The finding of mortality declines by cohorts largely supports the contention that nutrition, reduced exposure to diseases, and medical measures all contributed to increasing health capital in more recent cohorts that improved cohort survival. And cohort trends

[6] The tables of modeling results for the four specific causes are omitted in the interest of space.

in educational attainment may be one important mechanism through which these influences were realized.

In fact, the cohort differentials in total mortality mirror the cohort changes in education levels. A recent study by Hughes and O'Rand (2004) shows that baby boomers (1946 – 1964 cohorts) had significantly higher levels of high school and college education than their Jazz Age (1916 – 1925 cohorts) and Depression Kid (1926 – 1935) parents, but the educational achievement leveled off across the boomer cohorts. Cohort effects on mortality were similarly characterized by persistent declines in mortality from early cohorts until the early baby boomers (1945 – 1950 cohorts) and evident decreases in the rates of declines for both sexes. The same study also indicated a cross-over of gender-specific attainments that shows a slight drop of education level among late-boomer men and an educational advantage for late-boomer women. Furthermore, there is evidence that this trend has continued among cohorts younger than the boomers (Gamoran 2001). These patterns correspond well with recent cohort mortality patterns: while male boomer cohorts appeared to experience little decrease, females had a smoother progressive decline in successive cohorts, but the slow-down in such decline is also evident for females starting from the baby boomer cohorts.

[Table 2 and Figure 5 about here]

Heart Disease Mortality

Figure 6 presents model estimates of the trends of heart disease mortality change. The age effects of mortality due to heart disease generally exhibit exponential increases and this is consistent with previous findings. There is also a notable sex difference in the rates of changes in mortality risks with age. While women's mortality risks show log-linear increases with age, those for men show a faster increase before the age of 55, but a slower increase afterwards. The

slowing of the acceleration rate occurring in middle-aged men is intriguing. A conventional view relates this change in sex difference to hormonal effects that may protect premenopausal women against higher cholesterol levels, a benefit lost in postmenopausal women (Witteman, Grobbee, Kok et al. 1989). But recent reviews of studies indicate that the menopause has no discernible effect on the age pattern of coronary heart disease and the male midlife mortality change is more compatible with a change in male hormones than with an effect of the female menopause (Barrett-Connor 1997:256). It could also be a result of selection due to differential survival of men at highest risks prior to middle ages.

Both males and females had large monotonic declines in heart disease deaths for most of the pre-boomer cohorts. The declines slowed down considerably for female cohorts starting from the late boomers. And there was even a slight upward trend for the same male cohorts. These mortality changes for late baby boomers and more recent cohorts are similar to those found in the analysis of total mortality and therefore also reflect the cohort differentials in socioeconoimc attainment. This is consistent with the position of heart disease as the leading cause of adult mortality in recent decades. The modest increase in heart disease mortality rates for the most recent male cohorts also suggests the effects of changes in other major heart disease risk factors. Large increases in obesity rates, for instance, have been shown to increase the risk of overall and circulatory disease mortality (Rogers et al. 2003).

Stroke Mortality

Figure 7 shows that the age effects of stroke mortality are similar to those of heart disease mortality. The gender differences in the growth rates differ across the adult ages. It has been suggested that estrogen has both neuroprotective and neurodeleterious effects, which may

contribute to the variable sex mortality differentials at different ages (Fullerton, Chetkovich, Wu et al. 2002).

In contrast to the cohort effects of heart disease mortality, the declines in stroke mortality are largely continuous from the earliest to the most recent cohorts with no evident decrease in the rate of decline. This result is surprising in that heart disease and stroke deaths share several medical and social risk factors and should demonstrate similar patterns of mortality rates. Among the classic risk factors for heart disease, only LDL cholesterol has not been proven to be a strong risk factor for stroke (Barrett-Connor 1997). It can thus be inferred that cohort differences in LDL cholesterol play a role in the different cohort effects for the two causes of deaths. It is possible that the late-boomers and recent cohorts, especially males, have been disproportionately affected by behavioral changes such as diet high in animal fat that led to higher levels of LDL cholesterol.

[Figures 6 and 7 about here]

Lung Cancer Mortality

The APC model estimates for lung cancer mortality are displayed in Figure 8. The age effects of lung cancer death rates resemble those found in descriptive analyses. Both male and female age curves show rapid increases from early adulthood to peaks around ages 80 to 85 and then level off.

The cohort effects of lung cancer mortality are consistent with expectations: mortality curves increased first and decreased for more recent cohorts, following an inverse-U shape. The decrease in the male cohort mortality that started after the turn of the 20th century correlates with reduced initiation of cigarette smoking in young men during the Great Depression (1929 – 1933), as suggested by Tarone and Chu (2000a). But the argument relating economic deprivation to

reduced smoking does not seem to explain increased initiation of smoking and lung cancer deaths for the same female birth cohorts. Men and women also differ in birth cohorts that experienced the highest levels of lung cancer mortality: 1895 – 1900 for men and 1920 – 1925 for women. The lag of 25 years between the peaking male and female birth cohorts can be attributed to the sex differences in stages of cigarette diffusion. The subsequent decreases in mortality for more recent males and females may reflect the long-term benefits of increases in smoking cessation.

Compared to the relatively flat period effects of heart disease and stroke mortality, the period effects of lung cancer mortality show monotonic increases. And females had a steeper increase than males throughout the 40-year time period. There were signs of reductions in tar and nicotine yield per cigarette in the United States from 1950 to 1995 (Hoffman, Djordjevic, and Brunnemann 1996), which should decrease the harmful effects of smoking. It is, therefore, possible that the changes in cigarette composition do not affect lung cancer death rates until after a sufficiently long latency. It is also possible that the changes in mortality simply reflect more accurate diagnosis of lung cancer. But no evidence shows dramatic increases in diagnosis of lung cancers during this period. Other period factors should be considered.

Breast Cancer Mortality

The final set of results pertains to the female breast cancer mortality change and is summarized in Figure 9. The age effects are large and fit the general description of the age variations of breast cancer risks that are described as the “Clemensen’s hook” (Clayton and Schifflers 1987b). Such an age pattern is indicative of the etiology of the disease: early (pre-menopausal) disease risk is more strongly genetically determined and the increases in risk slow down at the age of menopause. The period effects are fairly small and indicate a similar upward

trend with those of the lung cancer mortality. The increases in breast cancer mortality since the 1980s may be related to the marked increases in breast cancer incidence rates due to the expanded use of mammography (Breen and Kessler 1994).

The cohort effects show a steady downward trend, suggesting continuous improvement in cohort survival from breast cancer. Such monotonic declines in mortality cannot be entirely related to changes in female fertility behaviors. Fertility rates dropped for the Depression and World War II cohorts, increased afterwards until 1964 (Baby boom cohorts), and decreased again since then (Easterlin 1987: Figure 1.1). But mothers of the baby boomer cohorts who had historically high fertility rates did not have lower mortality rates than their children or more recent cohorts of women who had lower fertility rates. Both delays of childbearing and fewer numbers of children affect the exposure of breast tissue to endogenous estrogen levels and may elevate the risks for breast cancer. But recent female cohorts still had progressively lower breast cancer mortality rates. This may well imply that the effects of biological risk factors have been dampened by those associated with improvements in socioeconomic conditions. Increased female labor force participation may delay or reduce childbearing on one hand, and boost economic well-being on the other. It is likely that the positive effect of more social roles and income outweighed the negative effect of hormonal exposure and acted to sustain the continuous mortality reductions.

[Figures 8 and 9 about here]

DISCUSSION

This study employed a new approach to APC analysis to examine the U.S. adult mortality change during the second half of the 20th century by four degenerative causes of deaths. It shows

that the large mortality reductions from 1960's to 1970's continued well into late1990's. The analysis relates the temporal patterns of mortality to specific demographic components by delineating age, period, and cohort effects. Related to each of these components are epidemiologic conditions determined by a host of biological, socioeconomic and behavioral factors, and public health and personal medical services. Model-based assessments of the age, period, and cohort effects, therefore, help to determine the distinct role of these factors in mortality change. Whereas differences associated with age may primarily indicate different etiological factors underlying specific degenerative diseases, period and cohort variations reflect the effects of social change. There are three major findings.

First, the most striking finding is the dominance of cohort effects in recent trends of mortality reductions. If the finding on cohort effects is true, then the assumption that the declines in death rates are fixed for different birth cohorts is violated and standard models of period changes in age-specific death rates such as those shown in Figures 3 and 4 and Equation (2) are misspecified. The role of cohort effects in mortality decline undeniably has serious implications for measurement and analysis.

The analyses provide new evidence on persistent cohort differences in mortality rates of all causes of deaths examined. The results largely support the theory of technophysio evolution that implies large cohort improvements in health capital and physiological capacities. Cohort effects differ by specific causes of death examined, but they generally show substantial survival improvements. For total mortality and mortality due to heart disease, the declines were monotonic for pre-baby boomers and leveled off for late-boomer cohorts and more recent cohorts. For mortality due to stroke and female breast cancer, cohorts born from 1865 to 1975 had monotonic declines in mortality rates. Cohort effects for lung cancer mortality are

characterized by inverse-U shapes, with continuous declines occurring for male cohorts born since 1900 and female cohorts born since 1925. The effects associated with birth cohorts reflect processes of differential cohort accumulation of health effects of lifetime exposures to risk factors: e.g., education, diet and nutrition, physical activity, and smoking.

Second, sex differences in the cohort effects are most evident for heart disease and lung cancer mortality. Cohort trends in heart disease mortality have shown similar declines for earlier cohorts and diverged for recent male and female cohorts since the late baby boomers (1960 – 1964), with men experiencing a small increase and women continuing to decline at a slower rate. This pattern corresponds well with the differential cohort changes in educational attainments by sex. It may also be due to cohort differences in exposures to risk factors that are specific for heart disease, such as LDL cholesterol. The sex differences are largest for lung cancer mortality. The differential timing of initiations of cohort declines in lung cancer mortality by sex are consistent with the recent demographic finding that there was a lag of reduction in lung cancer deaths between men and women. This finding, therefore, sheds light on remote sources of gender differences in lung cancer mortality such as stages of diffusion of cigarette consumption.

Third, period effects are generally small or modest when birth cohort and age effects are simultaneously controlled. Specifically, there is virtually no period effect for heart disease mortality and a very moderate decrease in stroke mortality since 1975. Lung cancer and breast cancer mortality show monotonically increasing, albeit small period effects that suggest log-linear trends. Whereas the increases in breast cancer mortality may be influenced by changes in diagnostic techniques that increased breast cancer incidence rates, the increases in lung cancer mortality are not likely to be affected by such changes. Future research should further investigate the roles of smoking cessation, drug use, and air pollution in explaining the lung cancer trends.

The finding of weak period effects relative to the cohort effects is surprising. Dramatic improvement in medical technology and diffusion of medical innovations for the past few decades can be expected to lead to decreases in mortality rates at each successive historical time period. These anticipated mortality declines, however, have been mostly captured by the cohort trends rather than period effects. The insights gleaned from the descriptive plots suggest that this is not likely to be an artifact of the modeling. As discussed earlier in Figure 4, whereas there is evident non-parallelism among age curves across time periods, which suggests strong cohort effects, evidence for non-parallelism among age curves across cohorts, which suggests period effects, is restricted to cancer-related mortality and the degree of non-parallelism is smaller. The lack of declines in period effects of cancer related mortality may signal the lack of marked improvement or breakthroughs in improving cancer treatments.

The result is also comprehensible in light of the hypotheses on independent period and cohort effects. Because successive cohorts experienced more favorable historical and social conditions, they had not only lower exposure levels to socioeconomic, behavioral, and environmental risk factors, but also benefited from reduction in the exposure earlier in life course than earlier cohorts. In this sense, the mortality reduction as a consequence of advancement in medical measures manifests itself as cohort declines because the cumulative effects have been more pronounced in successive cohorts compared to periods.

An alternative explanation relates to shifts in the age structure of the adult population in recent decades. That is, the rapid increase in recent periods of older adults who have higher mortality risks for certain diseases mitigated the magnitudes of period effects. For example, the proportions of elderly ages 65 and over in the adult populations increased from 13.9% to 15.3% for males and from 16.3% to 20.2% for females during 1960 and 1999. There were a

corresponding 14.6%-increase in the proportion of deaths contributed by older males and a 15.6%-increase by older females. The increases by cause for males are 18.4% in heart disease, 6.4% in stroke, and 49.7% in lung cancer. And the respective increases for females are 11.8% in heart disease, 9.8% in stroke, 51.8% in lung cancer, and 41.7% in breast cancer. In supplementary analyses, the full APC models were estimated separately for populations age 20 – 64 and age 65 and over. The results for period effects generally hold in both magnitude and direction for both sexes. But period changes in population age compositions play a bigger role on the period effects coefficients of total mortality and heart disease mortality than those of the others. Whereas the period curves of total mortality and heart disease mortality estimated for all age groups combined show slightly upward trends, those estimated separately for the two age groups show either flat or slightly downward trends, indicating mortality declines over the period net of cohort changes. Although period effects are fairly small compared to cohort effects of mortality, the sensitivity of the estimates of period effects to the changing age composition of the adult population needs to be explored quantitatively in future research.

This study has focused on adult mortality within a specific country. Additional studies are needed to systematically examine the temporal components of other demographic outcomes such as fertility and in multiple countries. Comparison of results may shed light on a more general demographic theory of period and cohort dependence of change in vital rates. There has been relatively little analytic work explicitly directed at the delineation of age, period, and cohort effects in fertility (Hobcraft et al. 1982). The majority of previous demographic analyses of sources of variations in fertility rates found period effects dominate and that cohort effects are minimal (see e.g., Namboodiri 1981; Ni Bhrolchain 1992). It is well worth our effort to conduct a fertility trend analysis using the new analytic tool to test such a conventional wisdom. The

goal-directed behavior of fertility makes the linearity assumption of temporal variations in fertility rates less tenable (Hobcraft et al. 1982), but the statistical delineation of period and cohort effects provides a good starting point for developing in-depth investigations of fertility jointly with the theories of reproductive behavior.

In sum, the findings of sex-specific patterns of mortality change for all major causes of deaths in the U.S. suggest that the health capital, presence of early-life and life time cumulative exposure differences, and social behavioral influences are important in reducing mortality risks for successive birth cohorts. Of greatest concerns for the prospect of future mortality reductions are the stagnation of recent cohort declines in heart disease mortality and period increases in cancer rates in spite of the large cohort improvements. These trends are related to social patterns of health behaviors such as an increasingly high-fat and high-cholesterol diet, decreasing physical activities, and increases in rates of smoking, especially in teenagers and women of lower SES class. Although the smoking patterns are most directly related to lung cancer incidence and mortality, they could also be responsible for some mortality differences in cardiovascular diseases. These factors are unfavorable conditions that act as both debilitative and selective forces that shape the levels of cohort mortality. Even without major biomedical advances (e.g., in stem cell and genomic therapies), if greater social and public health efforts are directed to modifications of these behaviors and environmental factors, then the mortality reductions will continue into the 21st century.

Appendix A. International Classification of Diseases (ICD) Coding Classifications

Cause of Death	ICD Revision Number and Date Applicable			
	7th 1960 - 1967	**8th 1968 - 1979**	**9th 1980 - 1998**	**10th 1999**
Heart Disease[1]	400 - 402, 410 - 443	390 - 398, 402, 404, 410 - 429	390 - 398, 402, 404 - 429	I00 - I09, I11, I13, I20 - I51
Stroke[2]	330 - 334	430 - 438	430 - 438	I60 - I69
Lung Cancer[3]	162	162	162	C33 - C34
Breast Cancer[4]	170	174	174	C50

[1]Disease of the heart, i.e., coronary heart disease, hypertensive heart disease, and rheumatic heart disease
[2]Cerebrovascular disease
[3]Malignant neoplasms of trachea, bronchus, and lung
[4]Malignant neoplasm of breast

Appendix B: Computational Algorithm of Intrinsic Estimator

i) Compute the eigen-vectors $u_1,\ldots,u_r$ of matrix X^TX, where X denotes the design matrix of model (1). Normalize them with $\|u_m\| = 1,\ldots,r$ and denote the orthonormal matrix as $U = (u_1,\ldots u_r)^T$;

ii) Identify the special eigenvector B_0 corresponding to eigenvalue 0. Denote $u_1 = B_0$ without loss of generality;

iii) Select the principal components to be the remaining eigen-vectors $u_2,\ldots,u_r$ with non-zero eigen-values;

iv) Fit a principal components regression (PCR) model using a design matrix V whose column vectors are the principal components $u_2,\ldots,u_r$, i.e., $V = (u_2,\ldots,u_r)$, to obtain the coefficients $(w_2,\ldots,w_r)$;

v) Set coefficient $w_1 = 0$ and transform the coefficients vector $w = (w_1,\ldots,w_r)^T$ by the orthonormal matrix $U = (u_1,\ldots u_r)^T$ to obtain the intrinsic estimator $B = Uw$.

Note: Instead of using reference categories, the IE uses the "usual ANOVA-type constraints" that the sums of the respective age, period, and cohort coefficients equal zero: $\sum_{i=1}^{a}\alpha_i = \sum_{j=1}^{p}\beta_j = \sum_{k=1}^{a+p-1}\gamma_k = 0$. The computational algorithm used by the IE estimates effect coefficients for each of the $a - 1$, $p - 1$, and $a + p - 2$ age, period, and cohort categories, respectively, which is consistent with the definition of the parameter vector in Eq. (1). Then the IE uses the zero-sum constraints to obtain the numerical values of the deleted age, period, and cohort categories.

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Table 1. Goodness-of-Fit Statistics for Age-Period-Cohort Log Linear Models of U.S. Adult Mortality

Cause of Death		Male				Female			
	Models	***A***	***AP***	***AC***	***APC_IE***	***A***	***AP***	***AC***	***APC_IE***
	DF	112	105	90	84	112	105	90	84
Total	***Deviance***	978783	108289	45427	25828	695527	40443	72089	18903
	AIC	979007	108499	45607	25996	695751	40653	72269	19071
	BIC	979019	108510	45617	26005	695763	40664	72279	19080
Heart Disease	***Deviance***	1110502	63080	14811	5533	782210	52225	18638	9243
	AIC	1110726	63290	14991	5701	782434	52435	18818	9411
	BIC	1110738	63301	15000	5711	782446	52446	18827	9420
Stroke	***Deviance***	543470	7260	17947	1027	655622	12660	25967	1480
	AIC	543694	7470	18127	1195	655846	12870	26147	1648
	BIC	543706	7482	18137	1204	655858	12881	26157	1657
Lung Cancer	***Deviance***	115651	51520	7073	611	320050	42126	5296	245
	AIC	115875	51730	7253	779	320274	42336	5476	413
	BIC	115887	51741	7262	788	320286	42347	5486	422
Breast Cancer	***Deviance***					9748	7403	1553	512
	AIC					9972	7613	1733	680
	BIC					9984	7625	1743	689

Table 2. Intrinsic Estimates for the U.S. Adult Mortality Rates for All Causes by Sex

Male

Intercept	*s.e.*	*Age*	*effect*	*s.e.*	*Period*	*effect*	*s.e.*	*Cohort*	*effect*	*s.e.*
-3.969	0.007	**20-24**	-1.661	0.023	**1960-64**	-0.150	0.009	**1865**	0.852	0.096
		25-29	-1.736	0.022	**1965-69**	-0.058	0.008	**1870**	0.835	0.046
		30-34	-1.675	0.021	**1970-74**	-0.011	0.008	**1875**	0.804	0.029
		35-39	-1.498	0.020	**1975-79**	-0.033	0.007	**1880**	0.727	0.022
		40-44	-1.221	0.017	**1980-84**	-0.007	0.007	**1885**	0.654	0.019
		45-49	-0.883	0.015	**1985-89**	0.052	0.008	**1890**	0.590	0.016
		50-54	-0.532	0.013	**1990-94**	0.087	0.008	**1895**	0.526	0.015
		55-59	-0.205	0.011	**1995-99**	0.120	0.008	**1900**	0.440	0.014
		60-64	0.112	0.010				**1905**	0.351	0.013
		65-69	0.407	0.009				**1910**	0.258	0.013
		70-74	0.703	0.009				**1915**	0.141	0.012
		75-79	0.997	0.009				**1920**	0.011	0.013
		80-84	1.323	0.010				**1925**	-0.093	0.014
		85-89	1.651	0.012				**1930**	-0.223	0.015
		90-94	1.957	0.016				**1935**	-0.359	0.017
		95+	2.263	0.025				**1940**	-0.494	0.018
								1945	-0.572	0.020
								1950	-0.567	0.021
								1955	-0.577	0.022
								1960	-0.646	0.026
Deviance		25828.4						**1965**	-0.757	0.032
DF		84						**1970**	-0.890	0.042
Overdispersion		306.0						**1975**	-1.009	0.068

Female

Intercept	*s.e.*	*Age*	*effect*	*s.e.*	*Period*	*effect*	*s.e.*	*Cohort*	*effect*	*s.e.*
-4.571	0.007	**20-24**	-2.032	0.032	**1960-64**	-0.115	0.009	**1865**	0.939	0.060
		25-29	-1.995	0.029	**1965-69**	-0.064	0.008	**1870**	0.937	0.031
		30-34	-1.819	0.026	**1970-74**	-0.042	0.007	**1875**	0.900	0.021
		35-39	-1.555	0.023	**1975-79**	-0.081	0.007	**1880**	0.834	0.016
		40-44	-1.245	0.020	**1980-84**	-0.034	0.007	**1885**	0.748	0.014
		45-49	-0.915	0.017	**1985-89**	0.044	0.007	**1890**	0.653	0.012
		50-54	-0.596	0.015	**1990-94**	0.094	0.007	**1895**	0.556	0.011
		55-59	-0.295	0.013	**1995-99**	0.197	0.008	**1900**	0.449	0.011
		60-64	0.026	0.011				**1905**	0.331	0.011
		65-69	0.349	0.010				**1910**	0.243	0.011
		70-74	0.701	0.009				**1915**	0.148	0.012
		75-79	1.072	0.008				**1920**	0.060	0.013
		80-84	1.484	0.008				**1925**	-0.018	0.015
		85-89	1.896	0.009				**1930**	-0.124	0.017
		90-94	2.273	0.011				**1935**	-0.249	0.019
		95+	2.652	0.014				**1940**	-0.416	0.022
								1945	-0.567	0.024
								1950	-0.678	0.026
								1955	-0.732	0.030
								1960	-0.805	0.035
Deviance		18903.2						**1965**	-0.918	0.044
DF		84						**1970**	-1.063	0.060
Overdispersion		224.3						**1975**	-1.230	0.103

Figure 1. Age Standardized Death Rates by Causes of Deaths and Sex, 1960 - 1999

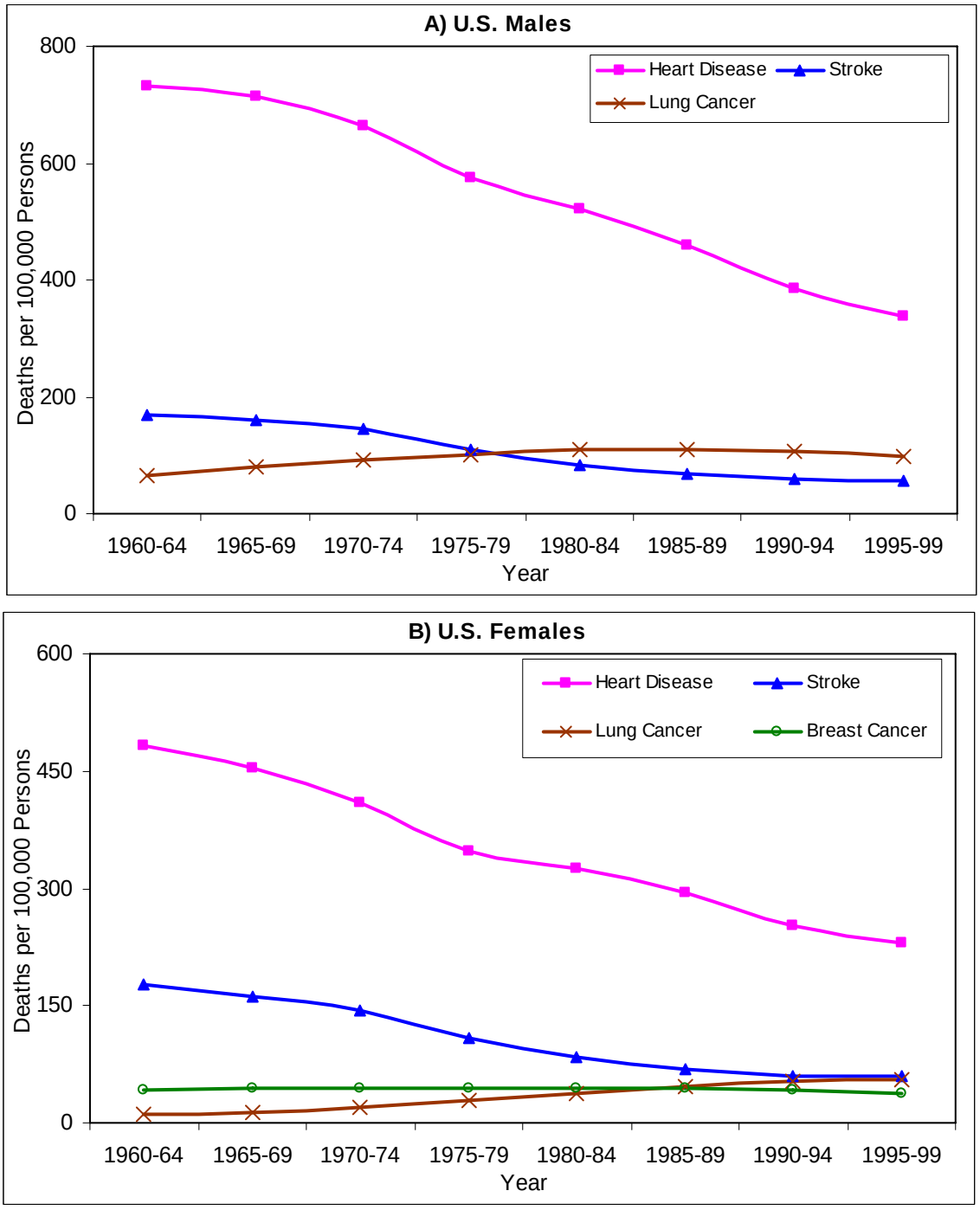

Figure 2. Age-Specific Death Rates (Logarithmic Scale) by Sex for U.S. Adult Population by Four Causes: 1960 – 1999

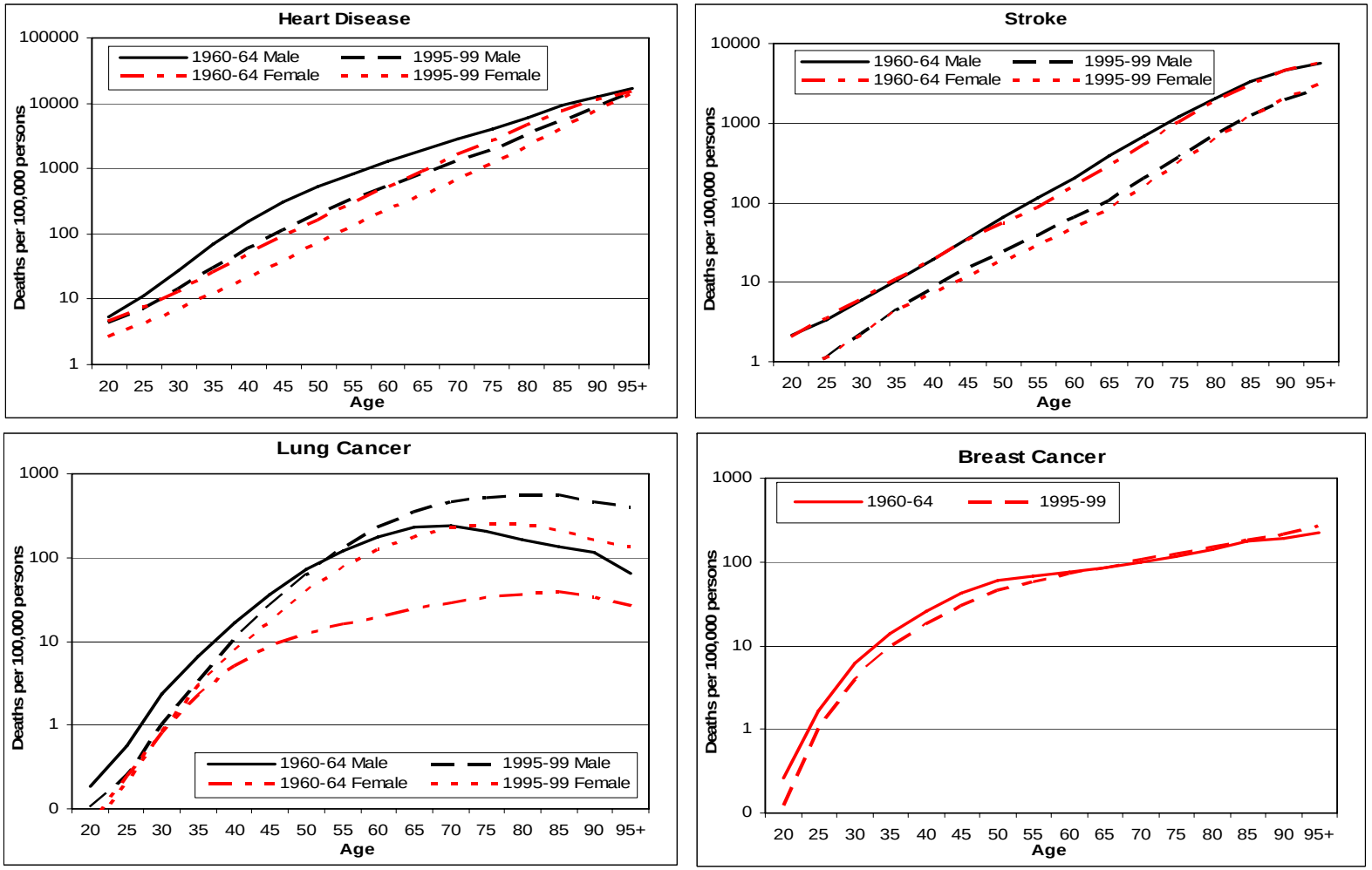

Figure 3. U.S. Female Lung Cancer Mortality Rates per 100,000 by Age at Death, Period of Death, and Birth Cohort

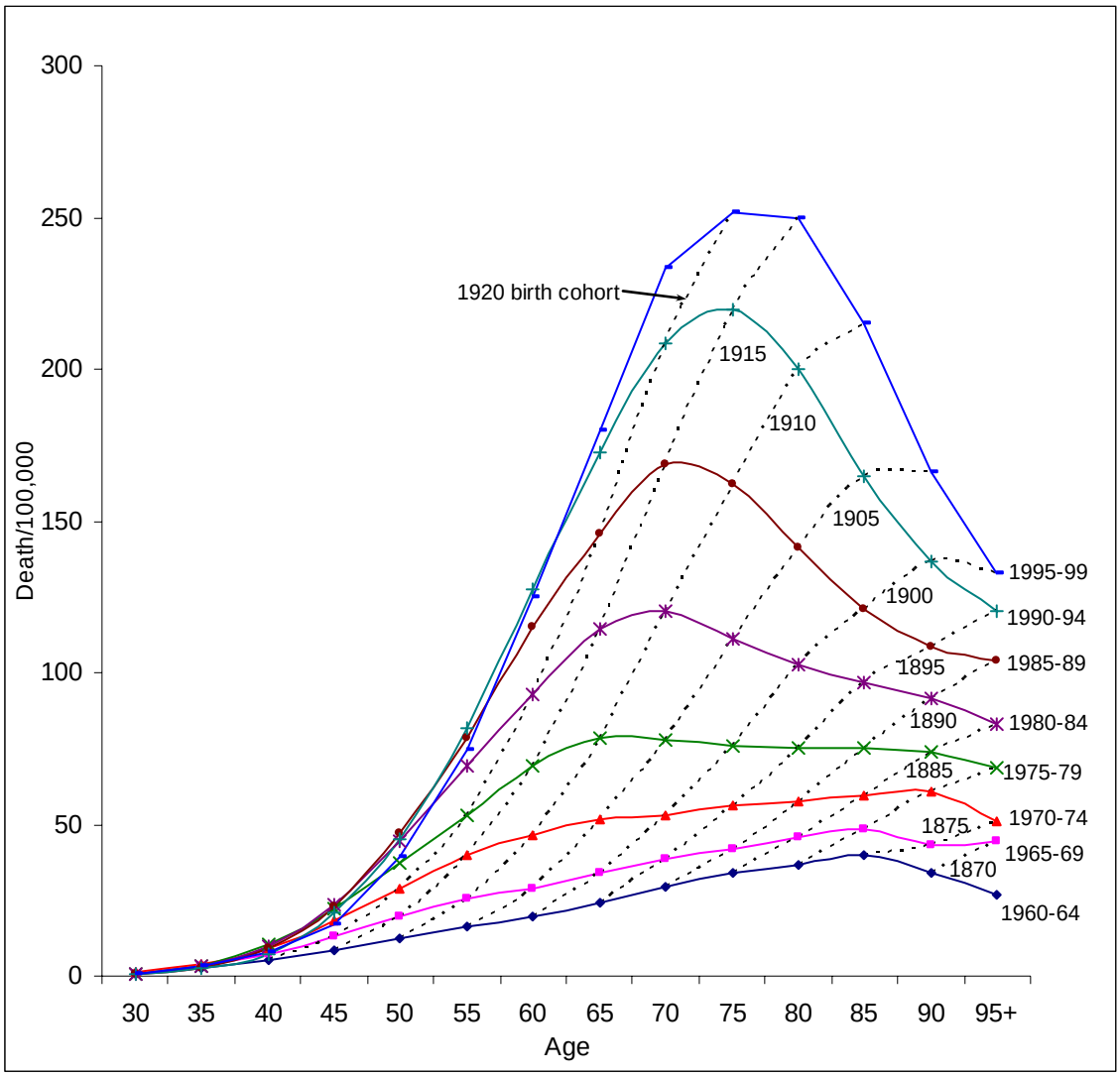

Figure 4. U.S. Female Age-Specific Death Rates by Period of Death

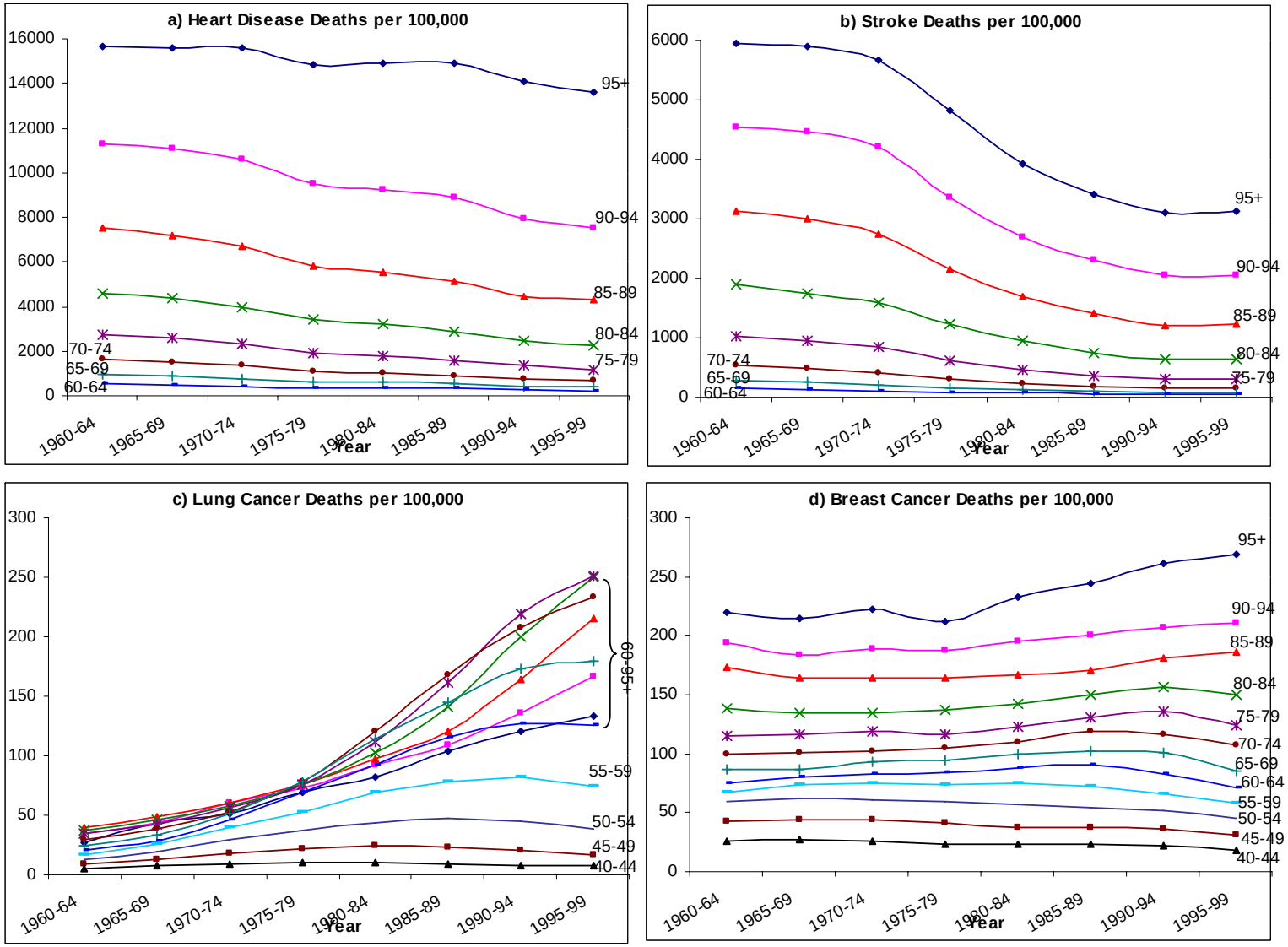

Figure 5. Intrinsic Estimates of Age, Period, and Cohort Effects of Mortality for All Causes by Sex

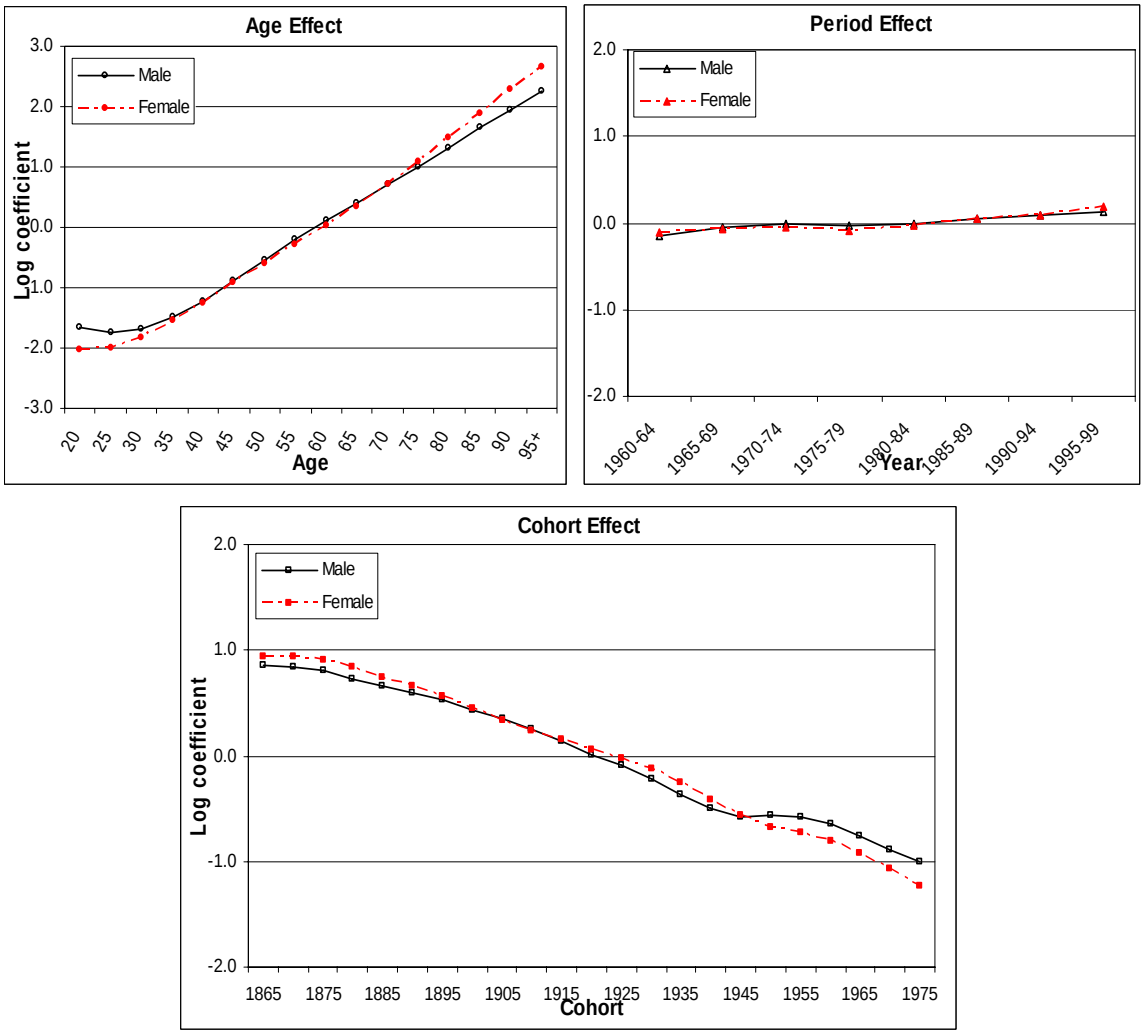

Figure 6. Intrinsic Estimates of Age, Period, and Cohort Effects of Heart Disease Mortality by Sex

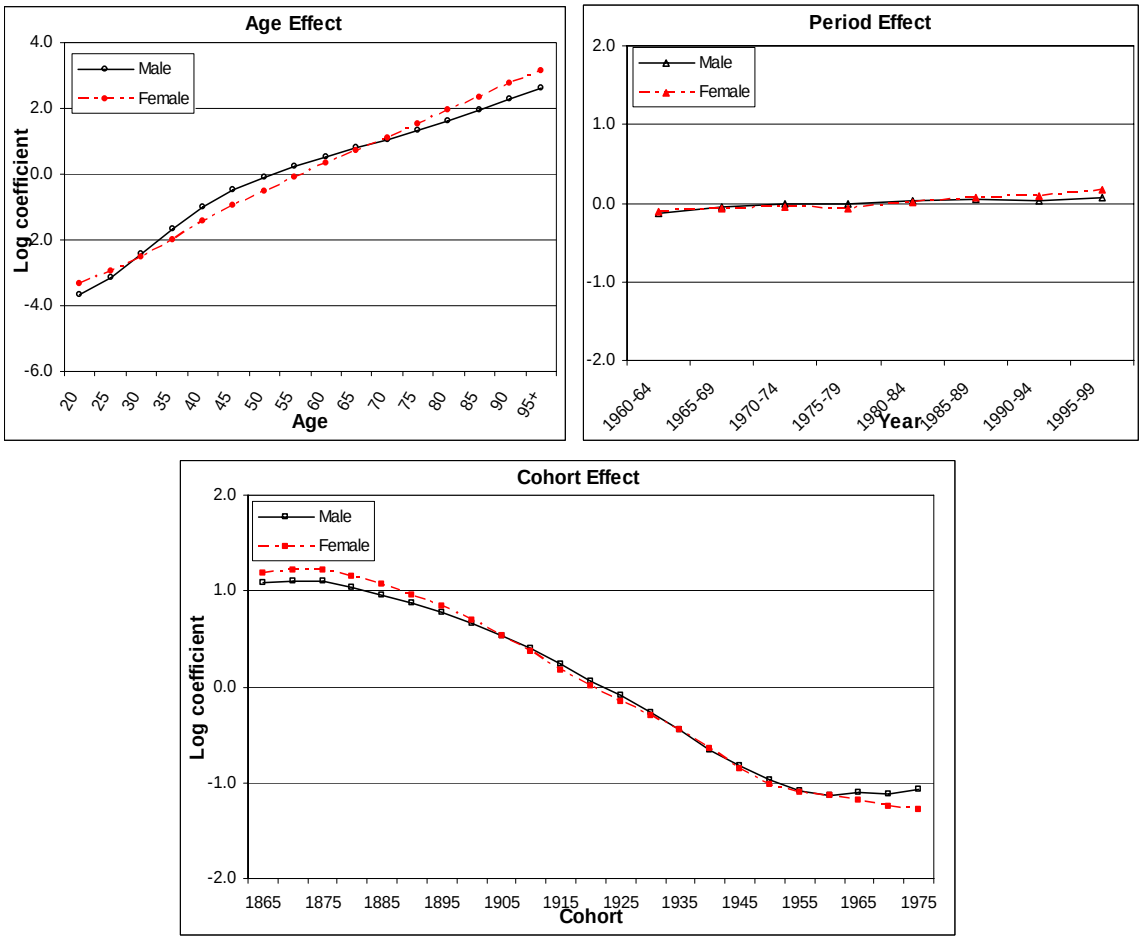

Figure 7. Intrinsic Estimates of Age, Period, and Cohort Effects of Stroke Mortality by Sex

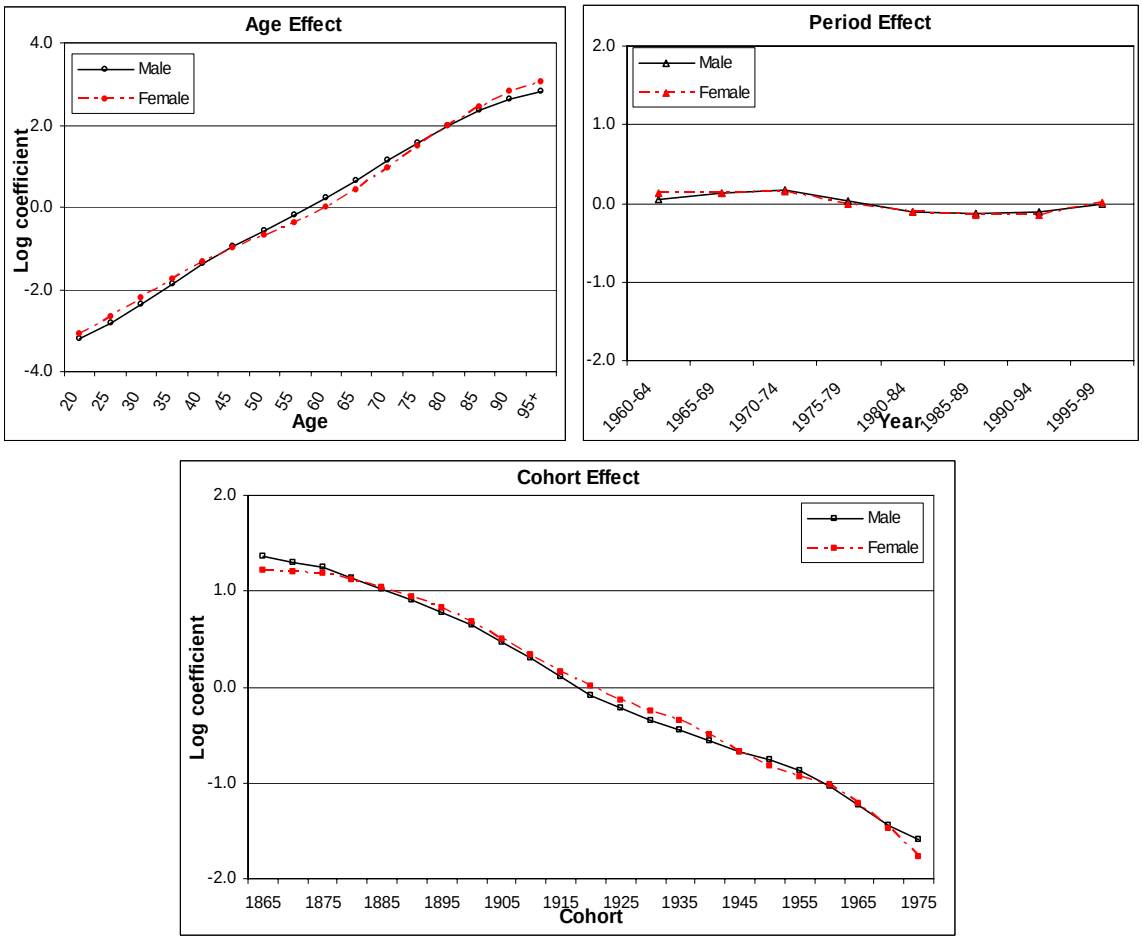

Figure 8. Intrinsic Estimates of Age, Period, and Cohort Effects of Lung Cancer Mortality by Sex

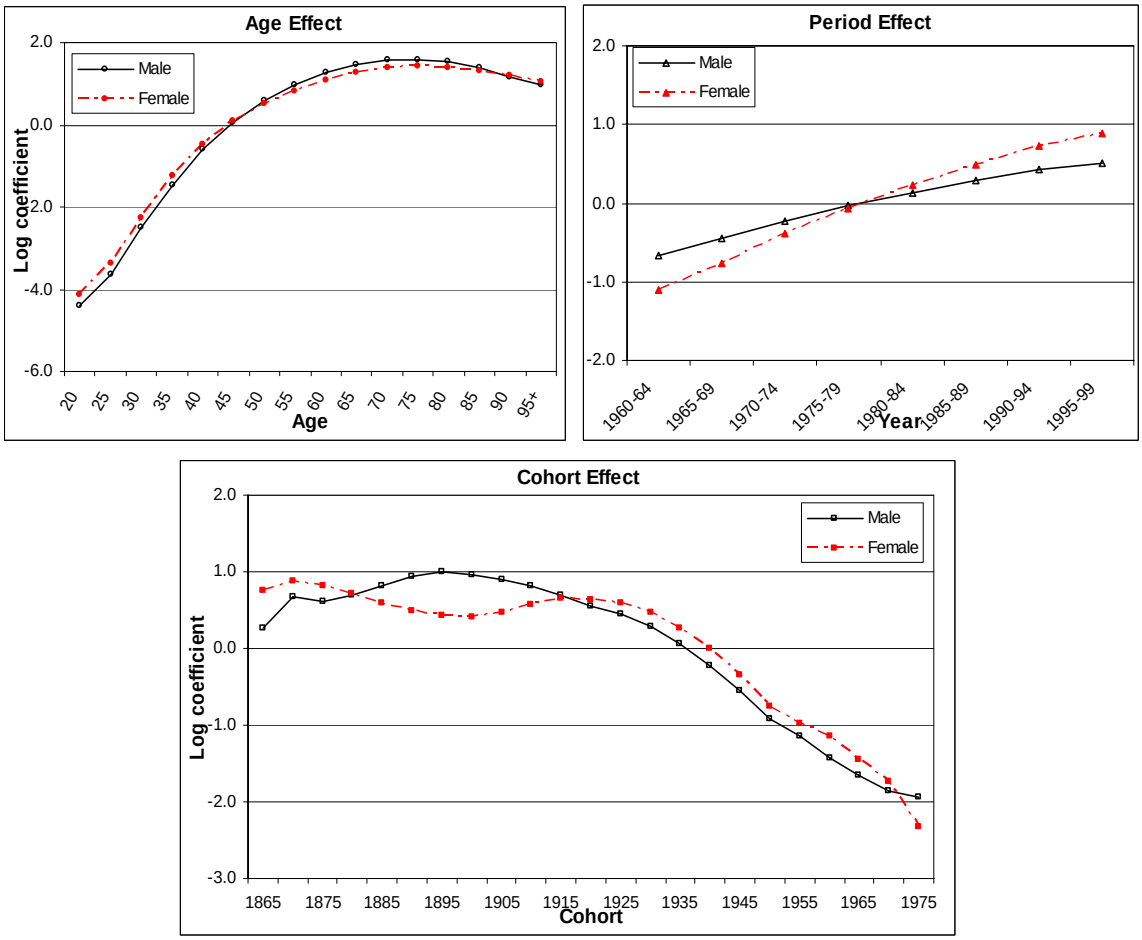

Figure 9. Intrinsic Estimates of Age, Period, and Cohort Effects of Female Breast Cancer Mortality