COMPARATIVE STUDIES OF HEALTH AND MORTALITY

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ABSTRACT

COMPARATIVE STUDIES OF HEALTH AND MORTALITY

Jessica Y. Ho

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This dissertation consists of three comparative studies of health and mortality which address major topics in the field: persistent mortality disparities within the U.S., how mortality in the U.S. compares to other high-income countries, and early life determinants of adult morbidity in developing countries. The design of these studies is predicated on the belief that we can draw meaningful inferences from comparisons across populations.

Chapter I examines the contribution of smoking to black-white mortality differences above age 50 from 1980-2005. This study shows that smoking-attributable mortality accounted for 20-40% of the black-white mortality gap among males between 1980-2005, but accounted for almost none of the black-white mortality gap among females. The results support the hypothesis that later initiation and lower rates of smoking cessation among black men may contribute to their higher levels of smoking-related mortality relative to white men.

Chapter II provides a comprehensive assessment of U.S. mortality relative to other high-income countries. This study demonstrates that mortality differences below age 50 account for the majority of the gap in life expectancy at birth between American males and their counterparts in other high-income countries. Among females, this figure is 41%. The major causes of death responsible for Americans' excess years of life lost below age

50 are unintentional injuries, noncommunicable diseases, perinatal conditions, and homicide. This study also finds that the U.S.'s unique pattern of age-specific mortality rankings holds for birth cohorts whose mortality experience spans the period 1935-2005.

Chapter III explores the association between two measures of early life conditions and adult morbidity in six countries. The findings from this study indicate that those born during the autumn in Ghana, Mexico, Russia, and South Africa and during the monsoon in India experience a health advantage. In China, the autumn-born experience a health disadvantage. This study also finds that pre- and postnatal rainfall and temperature conditions are associated with adult health outcomes, particularly height and blood pressure. The results provide support for the hypotheses that early life disease and nutritional conditions are important influences on later life health.

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CHAPTER I. The Contribution of Smoking to Black-White Differences in Mortality[1]

Introduction

Black-white mortality differentials in the United States are sizeable and persistent, and pose a significant, longstanding public health concern. The black-white gap in life expectancy at birth has fluctuated over time, reaching a recent peak of 8.5 years for men and 5.8 years for women in 1993 before declining to 5.2 years for men and 3.6 years for women in 2008 (National Center for Health Statistics [NCHS] 1997; Arias 2012). In contrast, after about 1990 the black-white gap in life expectancy at age 50 stagnated for males, and it declined only slightly for females (**Figure 1.1**). Given that 88% of black males, 93% of white males, 93% of black females, and 96% of white females survived to age 50 in 2008 (Arias 2012), an improved understanding of the factors contributing to this persistent black-white disparity in mortality at older ages is needed.

Despite the large literature on black-white health disparities, the reasons for their persistence have not been fully resolved (e.g., Smedley, Stith, and Nelson 2003; Williams et al. 2010). In this study, we focus on the contribution of smoking to black-white differences in mortality at ages 50 and above. Smoking is the leading preventable cause of premature morbidity and mortality in the United States and has strong links to chronic diseases that are prevalent at older ages (U.S. Department of Health and Human Services [DHHS] 2000). For example, in 1980, 87%-95% of lung cancer deaths occurred above age 50 among black and white males and females. By 2005, these percentages had

[1] Material from this chapter has appeared in Ho JY and Elo IT. 2013. "The Contribution of Smoking to Black-White Differences in U.S. Mortality." *Demography* 50(2): 545–568.

increased to 92%-96% (NCHS 2010a). Cohort smoking histories and period data on smoking behavior – smoking prevalence, intensity, cessation, and duration – suggest that the magnitude of smoking-attributable mortality at these ages may differ between blacks and whites, especially among males. Consistent with this speculation are the higher death rates from lung cancer and other smoking-related chronic diseases among blacks than whites (Haiman et al. 2006; Harper et al. 2007).

In this study, we integrate multiple approaches and data sources to provide a comprehensive assessment of the contribution of smoking to black-white mortality differences. To the best of our knowledge, no previous study has examined the contribution of smoking-attributable deaths to mortality trends among blacks or to trends in black-white mortality differences at older ages. Identifying whether smoking is a major contributor to black-white mortality differences is important. Smoking is a potentially modifiable health behavior, and lung cancer risk is sensitive to smoking intensity, duration, and time since quitting. Furthermore, determining the proportion of excess mortality due to smoking among blacks and whites will improve our understanding of its role in mortality trends and differentials. We find that smoking has contributed to the black-white gap in life expectancy at age 50 for males, accounting for 20%-40% of the gap between 1980 and 2005, but not for females. Smoking also explains a larger portion of the sex gap in life expectancy at age 50 for blacks than for whites. Estimates of the fraction of deaths attributable to smoking above age 50 are slightly greater for black males than for white males, and among males, current smoking status explains about 20% of the black excess risk in all-cause mortality above age 50 in the absence of adjustment for socioeconomic characteristics. These findings contribute to our understanding of the

role of smoking in driving contemporary mortality trends and differentials and reinforce the need for interventions that better address the needs of all groups.

Background

Black-White Differences in Smoking-Related Mortality

Smoking substantially increases the risk of death from cardiovascular diseases, including hypertension, ischemic heart disease, cerebrovascular disease, and atherosclerosis, and respiratory diseases such as pneumonia, influenza, bronchitis, emphysema, and chronic airway obstruction (DHHS 1989; DHHS 2001; Rogers et al. 2005). Smoking is also a risk factor for 15 cancer sites (Doll et al. 2005; IARC 2004).[2] For cancers of the esophagus, kidney, lung, larynx, oral cavity, pancreas, pharynx, and urinary bladder, estimates of the excess risks associated with smoking range from factors of 3 to 20 (IARC 2004). Furthermore, estimates based on the Cancer Prevention Study II (CPS-II), a prospective cohort study in the United States, suggest that at least 25% of deaths from nine cancers (bladder, esophagus, kidney, larynx, lip, lung, oral cavity, pancreas, pharynx) are attributable to smoking (DHHS 1989; Preston, Glei, and Wilmoth 2010). One recent study estimates that smoking cessation was responsible for at least 40% of the decline in male cancer mortality between 1991 and 2003 (DeLancey et al. 2008).

It is well-known that blacks suffer disproportionately from smoking-related diseases, with the exception of chronic obstructive pulmonary disease (COPD) (Burns et al. 1997; DHHS 1998; Haiman et al. 2006; Novotny et al. 1988; Williams and Collins 1995).

[2] These 15 sites are the kidney, larynx, liver, lung, myeloid leukemia, nasal cavity and paranasal sinuses, nasopharynx, oro- and hypopharynx, esophagus, oral cavity, pancreas, stomach, ureter, urinary bladder, and uterine cervix (IARC 2004).

Many of these smoking-related diseases have been implicated in the black-white gap in life expectancy. For example, Harper et al. (2007) identified cardiovascular diseases (heart disease, hypertension, stroke, and other cardiovascular diseases) as the leading causes of black-white differences in life expectancy at birth in 2003. Together, these causes accounted for 1.9 years of the 6.3 year gap in male life expectancy and 1.9 years of the 4.5 year gap in female life expectancy. Total cancer-related mortality explained close to an additional year of the black-white life expectancy gap among men, and slightly over half a year among women (Ibid.). Data from the Surveillance, Epidemiology and End Results (SEER) cancer registry show that in 2007, blacks had higher death rates than whites from most smoking-related cancers, with the exception of cancers of the kidney and urinary bladder among males and cancers of the lung and bronchus among females.[3] Data on smoking-related cancer incidence and survival are generally consistent with racial disparities in cancer mortality, with blacks having higher incidence and lower survival rates than whites (Clegg and Ries 2007; Edwards et al. 2010; Wong et al. 2009). Furthermore, blacks appear to be at greater risk of lung cancer than whites at lower levels of cigarette consumption (Haiman et al. 2006).

Black-White Differences in Smoking Behavior

Burns et al. (1997) have made the best reconstruction of cohort smoking histories by race and sex using data from the National Health Interview Survey (NHIS), including an adjustment for differential mortality by smoking status. For birth cohorts born between 1920 and 1945, black males were slightly more likely to be ever-smokers than white

[3] These are age-adjusted death rates for the population aged 50+. SEER provides statistics for the following smoking-related cancer sites: kidney, larynx, liver, lung and bronchus, oral cavity and pharynx, esophagus, pancreas, stomach, urinary bladder, and cervix uteri.

males. In contrast, white women from these birth cohorts were more likely than black women to be ever-smokers (**Figure 1.2**) (Ibid.). Burns et al. (1997) also found that black males had substantially lower smoking cessation rates than white males, with smoking cessation beginning 10 to 20 years later among blacks than whites. **Figure 1.3** shows annual quit rates among three 10-year cohorts of black and white males born between 1920 and 1949, which overlap with the cohorts in this analysis (those aged 50+ in 1980-2005 or aged 50-84 in 1997-2003). For each birth cohort, quit rates were substantially lower among blacks than whites. Similarly, smoking cessation among black females was lower than among white females (Ibid.). These differences in smoking cessation are unlikely to be due to differences in the desire to quit because surveys show that blacks appear as or more likely than whites to want to quit smoking (DHHS 1998).

Figure 1.4 shows current smoking prevalence (percent current smokers) at each age among two 5-year birth cohorts for black and white males. For the 1920-1924 birth cohorts, white males were slightly more likely to be current smokers until age 28, when a crossover occurred. Between ages 28 and 66, black males were more likely to be current smokers at every age, and the black-white difference widened with age, reaching a peak of 16% when males in these cohorts reached their late fifties and early sixties. For the 1940-1944 birth cohort, the crossover occurred earlier and widened more rapidly, maintaining a black-white gap of at least 10% for 16 years (between ages 30 and 46). We show only two cohorts here for clarity; the same general pattern of black-white differences in current smoking prevalence widening dramatically with age was observed for all of the cohorts born between 1920 and 1949.

The cohort smoking data are fairly consistent with period data on smoking prevalence, cessation, and duration: in general, whites are more likely to be ever smokers and have higher smoking intensities, whereas the prevalence of smoking cessation is much lower among black smokers (DHHS 1998; Fiore et al. 1989; Gilpin and Pierce 2002; King et al. 2004; NCHS 2010b; Siahpush et al. 2010). For example, Giovino et al. (1994), utilizing data from the NHIS, found that in the late 1980s and early 1990s, a larger percentage of whites than blacks of both sexes reported being ever smokers, but a higher percentage of black males than white males reported being current smokers. The authors further showed that between 1965 and 1991 the quit ratio (the percentage of adult ever smokers who were former smokers) was higher for whites than for blacks by 9.7%-16.8% (both sexes combined). More recent studies have documented a similar pattern (Gilpin and Pierce 2002), and King et al. (2004) showed that the racial disparity in the quit ratio persisted through 2000. These patterns have resulted in longer durations of smoking among blacks than whites. Using data from the 2003, 2006, and 2007 Tobacco Use Supplements from the Current Population Survey, Siahpush et al. (2010) estimated that the median duration of smoking among non-Hispanic black ever-smokers was 30 years, compared to 28 years among non-Hispanic whites (both sexes combined). Longer smoking durations and lower smoking cessation rates among blacks relative to whites may help explain their higher lung cancer death rates and higher mortality from other smoking-related diseases. Duration of smoking has been found in some studies to be a stronger predictor of lung cancer risk than the number of cigarettes smoked per day (Bach et al. 2003; Flanders et al. 2003; IARC 2004; Lubin and Caporaso 2006). Several studies have found that smoking cessation is beneficial at any age, with the reduction in the risk

of lung cancer mortality being particularly large for smokers who quit before middle age (Halpern, Gillespie, and Warner 1993; Peto et al. 2000). Smoking cessation has also been found to decrease the excess risks of stroke, COPD, myocardial infarction, and coronary heart disease (Kawachi et al. 1993; Kawachi et al. 1994; Oza et al. 2011; Rosenberg et al. 1985; Rosenberg, Palmer, and Shapiro 1990).

Black-White Differences in Nicotine Metabolism

Compared to whites, blacks smoke fewer cigarettes but inhale more deeply, are more likely to smoke menthol cigarettes and cigarettes with higher tar yields, achieve higher net indexes of smoke exposure, and may be at risk of greater physical dependence and exposure to more smoke toxins (Chen 1993; Sellers 1998). These differences may be due in part to differential environmental and occupational exposures and their interactions with biological mechanisms in how nicotine and other substances in tobacco smoke are metabolized (Williams at al. 2010). Pharmacogenetic differences in nicotine metabolism can affect the risk of becoming a smoker, amount smoked, degree of physical dependence, and absorption, distribution, excretion, and metabolism of carcinogens in tobacco smoke (Sellers 1998).

The measurement of cotinine, a nicotine metabolite, is a specific and sensitive test for exposure to tobacco smoke and can be used to distinguish active and passive smokers from nonsmokers (IARC 2004). It is used as a marker of both tobacco use and exposure to environmental tobacco smoke (ETS) because it has a longer half-life than nicotine (Caraballo et al. 1998). Using data from the Third National Health and Nutrition Examination Survey (1988-1991), Caraballo et al. (1998) found that black smokers had

significantly higher serum cotinine levels than white or Mexican American smokers at all intensities of cigarette smoking. This finding held even after adjustment for other sources of nicotine, such as the number of cigarettes smoked per day and exposure to ETS at home and at work, as well as age, sex, and body weight. It has been suggested that racial differences in serum cotinine levels may be attributable to racial differences in the accuracy of self-reported smoking. The authors of this study found that black and white self-reported smokers had serum cotinine levels consistent with their reported smoking levels, suggesting that their results were not biased by reporting differences between groups (Ibid.).

Pérez-Stable et al. (1998) examined the potential mechanism underlying higher cotinine levels among blacks in their hospital-based study of 40 black smokers and 39 white smokers. The participants received infusions of deuterium-labeled nicotine and cotinine, allowing for the accurate determination of daily nicotine intake from smoking. The authors found that although black smokers in this study smoked fewer cigarettes per day, they had higher overall levels of serum cotinine than white smokers. They attributed this difference to slower clearance of cotinine and higher intake of nicotine per cigarette smoked among blacks. A greater intake of nicotine and carcinogens from tobacco smoke per cigarette may be a partial explanation for the elevated burden of smoking-related diseases among blacks (Ibid.; Haiman et al. 2006).

Socioeconomic Status, Residential Context, Health Care, and Black-White Differences in Smoking-Attributable Mortality

Although racial identity is often conceptualized as an individual determinant of health and mortality, race captures important macro-level influences that condition individuals' life chances in the United States (e.g., Massey and Denton 1993; Smelser, Wilson and Mitchell 2000). Furthermore, U.S. racial classifications reflect prevailing political, ideological, and social forces rather than meaningful biological differences (Omi and Winant 1994; Williams et al. 2010; Zuberi 2001). Black-white differences in socioeconomic status (SES), residential context, and access to health care are prominent explanations for black-white differences in health and mortality (Hayward et al. 2000; Howard et al. 2000; Smedley, Stith and Nelson 2003; Williams et al. 2010), and they are also likely to contribute to black-white differences in smoking-attributable mortality at older ages.

Racial/ethnic differences in smoking behavior have been largely explained by racial/ethnic differences in SES (Flint and Novotny 1997; Kiefe et al. 2001; King 1997; King et al. 2004; Novotny et al. 1988). The benefits of smoking cessation may be viewed as marginal by low SES blacks due to their greater exposure to other health-eroding circumstances such as lack of material resources, residence in segregated neighborhoods, and the cumulative impact of disadvantage over the life course (Hayward et al. 2000; Link and Phelan 1995; Pampel, Krueger, and Denney 2010; Williams et al. 2010; Yao and Robert 2008). Smoking may serve as a "self-medicating mechanism" and a "form of relaxation" among low-income groups in the face of high levels of financial and other sources of stress (Cutler and Lleras-Muney 2008; Lawlor et al. 2003; Lutfey and Freese

2005; Pampel, Krueger, and Denney 2010). There is also evidence that cigarette companies target advertising toward black communities (Altman, Schooler, and Basil 1991; Landrine et al. 2005). The treatment effects of smoking cessation interventions appear to be weaker for blacks than for whites (Murray et al. 2001), and success in quitting smoking has been found to be higher among higher SES individuals, a pattern that holds for most racial/ethnic groups (Barbeau, Krieger, and Soobader 2004; Barbeau et al. 2005).

Researchers have speculated that smoking may be more harmful for low SES relative to high SES individuals because of their already poorer health status, whereas others have made the opposite argument – that because the baseline health stock of low SES individuals has already been diminished due to limited resources, the marginal impact of unhealthy behaviors like smoking is smaller for low SES than high SES individuals (Blaxter 1990; Pampel and Rogers 2004). That smoking might have a differential impact on mortality by SES and/or by race/ethnicity could also result in part from differential access to health care and the quality of that care. For example, smoking is a contributing factor to many forms of heart disease, the management of which is enhanced by high quality health care (Smedley, Stith, and Nelson 2003; Williams et al. 2010). Recent evidence points to the possibility that smoking is more detrimental to the health of disadvantaged groups. For example, Pampel and Rogers (2004), using data from the 1990 NHIS Health Promotion and Disease Prevention Supplement, concluded that smoking was more predictive of morbidity among low than high SES individuals, whereas no such interaction was found for mortality or between smoking and race/ethnicity for either morbidity or mortality controlling for socioeconomic status. Using the same data source,

Krueger and Chang (2008) found that high levels of former smoking increased the impact of stress on mortality for low SES individuals, but the interaction between smoking and stress was not significant for middle or high SES individuals.

AIMS OF THIS STUDY

In this study, we examine the contribution of smoking to black-white differences in mortality above age 50 using two complementary methods and data sources. First, we use indirect estimation techniques (Preston, Glei, and Wilmoth 2010) and vital statistics and census data from 1980-2005 to quantify the overall contribution of smoking-attributable mortality to trends in black and white mortality at ages 50 and above and to trends in the black-white gap in life expectancy at age 50. The indirect method has the potential for generating more reliable estimates of smoking-attributable mortality than direct methods relying on self-reported smoking behavior measured at a single point in time (Ibid.). Second, we use the National Health Interview Survey (NHIS) data from 1997-2003 linked to the National Death Index (NDI) through the end of 2006 to examine the extent to which differences in smoking behavior explain black-white differences in mortality and whether smoking exhibits a different association with mortality among blacks and whites (Denney et al. 2010; Flanders et al. 2003; IARC 2004; Pampel and Rogers 2004). Together, these estimates provide a comprehensive picture of the contribution of smoking to black and white mortality trends and to black-white mortality differences at older ages.

Data and Methods

I. Indirect Estimation of Smoking-Attributable Mortality, 1980-2005

Data

In the first part of the analysis, we use vital statistics data on deaths and population estimates available from the National Cancer Institute's SEER database (http://seer.cancer.gov/). These data are used to calculate age-specific death rates from lung cancer and from all other causes combined by race, sex, and 5-year age groups (50-54 to 85+). These data include all blacks and whites regardless of whether individuals identified as Hispanic because of the uneven quality of Hispanic ethnicity reporting in vital statistics and census data over the study period. States began to include a Hispanic-origin item on the death certificate in 1985, but this item was not reported by all 50 states and the District of Columbia until 1997 (NCHS 2006). We test the sensitivity of our results to the inclusion of Hispanic blacks and whites using the NHIS in the second part of the analysis (see below and **Table A1.1**).

Methodology

We use an indirect estimation method developed by Preston, Glei, and Wilmoth (2010) to estimate smoking-attributable mortality. The method is based on an assumption that lung cancer death rates can be used as a proxy to estimate the impact of smoking on mortality from all other causes of death (Preston, Glei, and Wilmoth 2011). The high proportion (approximately 90%) of lung cancer deaths attributable to smoking and the quality of lung cancer reporting on death certificates in the United States give us confidence that lung cancer mortality accurately proxies the impact of smoking on mortality from other

causes (Ibid.; Percy, Miller, and Ries 1990; Percy, Stanek, and Gloeckler 1981). Lung cancer is one of the most accurately reported cancers on the death certificate, and the quality of its reporting on death certificates has been high since at least the 1970s, a decade prior to the beginning of our study period (Ibid.).

In this model, age-specific lung cancer death rates are used to predict age-specific smoking-related mortality from all other causes of death. The model produces age- and sex-specific coefficients that, along with information on lung cancer death rates among non-smokers, can be used to estimate the fraction of deaths from all other causes that are attributable to smoking. Preston, Glei, and Wilmoth (2010) estimated this model using data from 21 developed countries for the period 1950-2006. The same model was subsequently applied by Fenelon and Preston (2012) to state-level data from the United States for the period 1996-2004. In this paper, we performed all analyses using the estimated age- and sex-specific coefficients from the above two studies and concluded that the results were not sensitive to the choice of the coefficients. The results reported here are based on the coefficients published in Fenelon and Preston (2012). Both Preston, Glei and Wilmoth (2011) and Fenelon and Preston (2012) demonstrated that this method produces results very similar to those obtained from an older, widely-used method developed by Peto et al. (1992).

To apply this method, we first estimated the fraction of deaths due to lung cancer in a specific age group that is attributable to smoking (A_L) as follows:

$$A_L = \frac{M_L - \lambda_L^N}{M_L} \qquad (1)$$

where M_L is the observed race-sex-specific lung cancer death rate and λ_L^N is the expected sex-specific lung cancer death rate among non-smokers, which is taken from the Cancer Prevention Study II (Preston, Glei, and Wilmoth 2010).

Second, we estimated the fraction of deaths from all other causes in a specific age group that is attributable to smoking (A_O) as follows:

$$A_O = 1 - e^{-\beta_L^{'}(M_L - \lambda_L^N)} \quad (2)$$

where $\beta_L^{'}$ are the age-sex-specific model coefficients from Fenelon and Preston (2012) and M_L and λ_L^N are as defined above.

The overall attributable fraction of deaths from all causes that is due to smoking, A, was then calculated as:

$$A = \frac{A_L D_L + A_O D_O}{D} \quad (3)$$

for each sex-race-age group, where D_L is the observed number of deaths from lung cancer, D_O is the observed number of deaths from all other causes, and D is the observed number of deaths from all causes combined (Preston, Glei, and Wilmoth 2010). Thus, the fraction of all deaths above age 50 that is attributable to smoking (A_{50+}), is:

$$A_{50+} = \frac{\sum_{i=50-54}^{85+} A_i D_i}{\sum_{i=50-54}^{85+} D_i} = \frac{\sum_{i=50-54}^{85+} A_i D_i}{D_{50+}} \quad (4)$$

where A_i is the overall fraction of deaths attributable to smoking in age group i and D_i is the number of deaths from all causes in age group i.

To translate smoking-attributable mortality by age into implications for life expectancy at age 50, we obtained adjusted age-specific death rates (i.e., death rates from which smoking-attributable mortality is removed), m_i^{-s}, as follows:

$$m_i^{-s} = m_i(1 - A_i) \quad (5)$$

where i = 50-54, 55-59, …, 80-84, 85+ and m_i is the age-specific death rate from all causes combined.

We then used standard life table procedures to calculate life expectancy at age 50 by race and sex with and without the inclusion of smoking-attributable deaths to determine the extent to which smoking contributes to the black-white gap in life expectancy at age 50 and whether this contribution has changed over time (Preston, Heuveline, and Guillot 2001). In these calculations, we used race-age-sex-specific ${}_n a_x$ and ${}_\infty m_{85}$ values from published U.S. life tables constructed for each year by NCHS (CDC/NCHS 2010) because estimates of old age mortality based on vital statistics and census data without adjustment are likely to be flawed (Elo 2001; Preston and Elo 2006). Our estimates of life expectancy at age 50 using all-cause mortality are very close to the published U.S. life table values.

In addition to comparing differences in smoking-attributable mortality between blacks and whites, we examine within-group differences in levels of smoking-attributable mortality for males and females and translate these differences into implications for the sex gap in life expectancy at age 50. This allows us to investigate how the impact of smoking on the sex gap in life expectancy at age 50 has changed over time and whether the magnitude of its contribution differs between blacks and whites.

II. Estimates of Smoking-Attributable Mortality Based on the NHIS, 1997-2003

Data

In the second part of the analysis, we used data from seven waves of the National Health Interview Survey (1997-2003), including the Sample Adult supplement, which collects information on smoking behavior for individuals ages 18+. These data have been linked to the NDI through the end of 2006. The NHIS is the most comprehensive, nationally representative data source for the study of socioeconomic and behavioral determinants of mortality in the United States and has been widely used for these purposes in previous studies (e.g., Hummer et al. 1999; Krueger and Chang 2008; Pampel and Rogers 2004; Rogers et al. 2005). The NHIS has several strengths for this analysis. The survey has a large sample size with an oversample of blacks, Hispanics, and Asians; provides high-quality information on the health and socio-demographic characteristics of respondents; is representative of the U.S. civilian noninstitutionalized population; and has achieved a lengthy mortality follow-up while maintaining a close correspondence between the survival experience of race-age-sex groups of NHIS cohorts and the U.S. population (CDC/NCHS 2009; Ingram, Lochner, and Cox 2008).

We restrict our sample to individuals aged 50-84 at the time of the survey, an age range similar to that employed in the indirect estimation of smoking-attributable mortality. These ages accounted for over 98% of the black-white difference in life expectancy at age 50 for both males and females in 1980 and 2005 (calculations by the author). We further limit the sample to non-Hispanic whites and non-Hispanic blacks, although we also test the sensitivity of our results by including all whites and blacks without the exclusion of Hispanics. We focus this part of the analysis on males since smoking-

attributable mortality appears to make a larger contribution to black-white differences in mortality among males than females (see **Figure 1.6** below).

The pooled 1997-2003 NHIS adult sample consists of 30,422 non-Hispanic white and non-Hispanic black males aged 50-84. We dropped 1,677 individuals who were ineligible for mortality follow-up due to a lack of information necessary for the linkage to the NDI or who were missing information on date of birth from this sample. We also excluded 1,087 males due to missing information on smoking behavior and 494 males due to missing information on other explanatory variables. The final sample consists of 23,701 non-Hispanic white males and 3,463 non-Hispanic black males aged 50-84 at the time of the interview, of whom 4,831 died during the follow-up period.[4]

Explanatory Variables

Our smoking variable distinguishes between never[5], current, and former smokers, taking into account smoking intensity (number of cigarettes smoked per day) among current smokers and time since stopping smoking among former smokers. Time since quitting is calculated from respondents' reported number of years since quitting. Among current smokers, we differentiate light smokers (who reported smoking less than 1 pack per day) from heavy smokers (who reported smoking 1 or more packs per day). Individuals who reported smoking two or more packs per day were rare in this sample of older adults. The smoking variable is coded as follows: never smoker, former smoker who quit 30+ years

[4] Sample sizes and number of deaths for other comparison groups (black and white males including Hispanics, non-Hispanic black and white females, and black and white females including Hispanics) used in sensitivity analyses are reported in **Table A1.2**.

[5] In the NHIS, ever smokers are defined as those who reported smoking at least 100 cigarettes in their lifetimes.

ago, former smoker who quit 20-29 years ago, former smoker who quit 10-19 years ago, former smoker who quit 5-9 years ago, former smoker who quit < 5 years ago, current smoker who smoked < 1 pack per day, and current smoker who smoked 1+ packs per day.

Most individuals (86.1%) began smoking by age 20 and nearly all (98.9%) had begun by age 30. Most current smokers were long-time smokers, with 93% having smoked for over 30 years (tabulations by the author). Thus, being a current smoker captures not only current smoking intensity but also long smoking duration. Furthermore, recent quitters (former smokers who had quit less than 10 years ago) were also long-time smokers, with 99% having smoked for more than 20 years. Smoking duration has been found to be a stronger predictor of lung cancer than the number of cigarettes smoked per day in several studies (Flanders et al. 2003; IARC 2004), and smoking-related mortality is known to vary by time since quitting (Ben-Shlomo et al. 1994; IARC 2004; Jacobs et al. 1999; Peto et al. 2000). Because smoking duration, intensity, and quit rates differ between blacks and whites, the smoking variable captures these differences in smoking behavior.

We also control for other individual-level attributes which vary by smoking status and between blacks and whites, and which can confound the relationship between smoking and mortality. These include body mass index (BMI), which is coded using standard categories (underweight, BMI <18.5 kg/m^2; normal, BMI 18.5-24.9 kg/m^2; overweight, BMI 25.0-29.9 kg/m^2; obese I, BMI 30.0-34.9 kg/m^2, and obese II/III, BMI $\geq$35.0 kg/m^2), and marital status, which is coded as never married, currently married, and widowed/divorced/separated. Our socioeconomic attributes include educational attainment (< 12 years, 12 years, 1-3 years of college, and 4+ years of college) and family

income. Because a large number of cases are missing information on family income in the NHIS, we used the imputed family income variable available from the Integrated Health Interview Series (IHIS) (Minnesota Population Center and State Health Access Data Assistance Center 2010).[6] We converted the categorical family income variable into a linear variable by taking the midpoint of each income interval and dividing it by 10,000. We estimated the value of the open ended category, which begins at $75,000, by estimating a median value for this category (Parker and Fenwick 1983; Rogers et al. 2005).[7] We include the natural log of family income in our models to capture the non-linear income association with mortality. In addition, we control for region of residence (northeast, north central, south, and west) to account for differences in residential distribution between blacks and whites and for differential mortality by region of residence.

Methods and Analytic Strategy

We used Cox proportional hazards regression models to estimate all-cause mortality. We focus on all-cause mortality because smoking, as discussed above, is a contributing factor for mortality from multiple causes of death. In addition, our indirect estimation of

[6] This variable was created using multiple imputation methods first developed by Rubin (1987). Imputation of family income was restricted to families containing at least one adult earner, and observed personal earnings were used as the lower bounds in these imputations. A large number of variables were used in the imputation, including variables pertaining to geographic location, household composition, employment, race/ethnicity, education, and reported health conditions or disability of family members.

[7] The median value in the open ended category is estimated using the Pareto curve. The slope of this curve, v, is estimated as $[\log(n_t + n_{t-1}) - \log(n_t)] / [\log(x_t) - \log(x_{t-1})]$, where n_t is the number of people in the open ended (last) income category, n_{t-1} is the number of people in the next-to-last income category, x_t is the lower bound of the open ended income category, and x_{t-1} is the lower bound of the next-to-last income category. The median value for the open ended category is then estimated as $10^{(0.301/v)*x_t}$.

smoking-attributable mortality captures mortality from all smoking-related causes. Our Cox proportional hazards model takes the form of:

$$\log h_i(t) = \log h(t) + \alpha Y_i + \sum_j \beta_j S_{ji} + \sum_k \gamma_k X_{ki}, \qquad (6)$$

where $h(t)$ is the unspecified baseline hazard function; t measures age; i refers to the individual; S to smoking behavior; X to other explanatory variables, including baseline age and race/ethnicity; and Y refers to the survey year. Individuals who were alive on December 31, 2006 were censored on this date (Allison 1995). Age was used as the analysis time, which allows for unrestricted non-linearity in its effects and, in the construction of the partial likelihood, makes comparisons only among people of the same age. Hazard ratios were calculated based on exponentiated coefficients from the proportional hazards models ($HR = e^{\beta}$). We used t-tests to assess the significance of individual coefficients. In the estimation, we took into account the complex sample design of the NHIS to obtain corrected standard errors. All models were weighted and estimated using Stata 11.

We estimated three models: Model 1 estimates the magnitude of the excess mortality among non-Hispanic black males controlling for age at baseline, survey year, BMI, and race. Model 2 adds smoking behavior to Model 1 to assess whether controlling for smoking helps explain the black-white difference in mortality. In Model 3, we introduce marital status, education, family income, and region of residence. These further adjustments allow us to assess whether introducing these additional controls further modifies the black and smoking status coefficients. We then interact race with smoking status to examine whether smoking has a differential impact on mortality among non-

Hispanic whites and non-Hispanic blacks. Finally, we used the hazard ratios from the Cox proportional hazards models to calculate population attributable risk fractions (PAF) due to smoking and compare these fractions to those obtained using the indirect estimation method described above.

To calculate the PAFs, we used the following equation recommended for use with a multicategory exposure variable and in the presence of confounding (Rockhill, Newman and Weinberg 1998):

$$PAF = \sum_{i=0}^{k} pd_i \left(\frac{RR_i - 1}{RR_i}\right) = 1 - \sum_{i=0}^{k} \frac{pd_i}{RR_i} \quad (7)$$

where i refers to smoking category, pd_i is the fraction of total deaths occurring in the ith smoking category, and RR_i is the hazard ratio from Model 3 for the ith smoking category. The PAF provides an estimate of the proportion of deaths that could be avoided if smoking were eliminated from the population (Flegal et al. 2005; Mehta and Chang 2009). In practice, the PAF estimates the proportion of deaths that could be avoided if all current and former smokers experienced the mortality risks of never smokers.

Results

I. Indirect Estimates of Smoking-Attributable Mortality, 1980-2005

Table 1.1 and **Figure 1.5** show the percentage of deaths attributable to smoking by race and sex between 1980 and 2005. Among males, this fraction increased until around 1990, and it was higher among black males than white males throughout the period. This black-white difference reached a peak (7.2%) around 1990, when 32.3% of deaths among black males and 25.1% of deaths among white males above age 50 were attributable to smoking. This gap subsequently declined to 3.7% by 2005, when 24.2%

and 20.5% of deaths were attributable to smoking among black males and white males, respectively.

Smoking-attributable deaths made up a much smaller percentage of deaths among females than males, but their contribution rose steadily throughout the period, reflecting differential smoking histories (Burns et al. 1997; Preston and Wang 2006). In contrast to the case of males, smoking-attributable deaths made up a somewhat smaller percentage of all deaths among black females than among white females except in the early 1980s, when this percentage was slightly higher among black females (**Table 1.1** and **Figure 1.5**). For example, in 1980 the percentage of deaths attributable to smoking was 6.4% for white females and 6.8% for black females. By 2005, these percentages had increased to 15.8% and 14.4%, respectively.

Table 1.2 and **Figure 1.6** illustrate the impact of smoking-attributable mortality on life expectancy at age 50 by race and sex and its contribution to black-white differences in life expectancy at age 50 over time. For example, for white males, smoking-attributable mortality reduced life expectancy at age 50 by 3.02 years in 1990 (29.78 without smoking versus 26.76 with smoking) and 2.41 years in 2005 (31.39 versus 28.98). For black males, smoking-attributable mortality reduced life expectancy at age 50 by between 4.47 years in 1990 (27.03 versus 22.56) and 3.18 years in 2005 (28.29 versus 25.11). In the absence of smoking-attributable mortality, the black-white gap in male life expectancy at age 50 would have been about 1.44 years smaller in 1990 and 0.78 years smaller in 2005. Thus, smoking-attributable mortality accounted for about 34% (1.44/4.20) of the black-white gap in male life expectancy at age 50 in 1990 and about 20% (0.78/3.87) of the gap in 2005.

Because smoking-attributable mortality made a smaller contribution to mortality above age 50 among females than among males, its contribution to life expectancy at age 50 was also much smaller for females. In 1980, it accounted for 0.79 years of white female life expectancy and 0.86 years of black female life expectancy at age 50. By 2005, these figures had increased to 1.90 years and 1.83 years, respectively. Because the impact of smoking on life expectancy at age 50 was similar for white females and black females, the elimination of smoking-attributable deaths had little impact on the black-white gap in female life expectancy at age 50.

Figure 1.6 also suggests that the contribution of smoking to sex differences in mortality differs between blacks and whites. The percentage of deaths attributable to smoking among males exceeds that among females for both blacks and whites; however, the magnitude of this difference is larger for blacks than for whites (**Table 1.1**). In 1990, the percentage of deaths attributable to smoking was roughly 12% for both black and white females, but these figures were 32.3% for black males and 25.1% for white males. The male-female difference in smoking-attributable mortality declined throughout the study period for both blacks and whites but remained larger for blacks in 2005 (a difference of 9.8% among blacks versus 4.7% among whites).

Table 1.3 and **Figure 1.7** demonstrate the impact of smoking-attributable mortality on the sex gap in life expectancy at age 50 for blacks and whites. In the absence of smoking-attributable mortality, the sex gap in life expectancy at age 50 for blacks would have been smaller by about 2.94 years in 1980 and 1.36 years in 2005, accounting for between 50-55% of the gap between 1980-1990 and 28% of the gap in 2005. For whites, the sex gap in white life expectancy at age 50 would have been between 0.51 (2005) and 1.94 (1980)

years smaller in the absence of smoking between 1980 and 2005, and smoking-attributable mortality accounted for approximately 14%-34% of the gap during this time period. Overall, the contribution of smoking-attributable mortality to sex differences in life expectancy at age 50 has been larger for blacks than whites.

In summary, the results from indirect estimation of smoking-attributable mortality show that smoking had a sizeable impact on life expectancy at age 50 for blacks and whites between 1980 and 2005, especially among males. Smoking-attributable mortality has also contributed to the black-white gap in male life expectancy at age 50, but it has had a negligible impact on the black-white gap in female life expectancy at age 50. These estimates are consistent with black-white differences in mortality from most smoking-related causes of death. However, differences in ever-smoking prevalence between black and white male birth cohorts do not appear to be especially large (**Figure 1.2**). Thus, it is somewhat puzzling why the contribution of smoking-attributable mortality to life expectancy at age 50 is larger for black males than for white males, whereas among women, the contribution of smoking-attributable mortality is very similar. In the second part of the analysis, we assess whether differences in smoking behavior, including smoking duration and smoking intensity (among current smokers), can help explain black-white differences in smoking-attributable mortality among males.

II. Smoking Behavior and Mortality Among Non-Hispanic Black and Non-Hispanic White Males: Results from the NHIS

Table 1.4 provides sample characteristics for the entire male sample and for non-Hispanic white and non-Hispanic black males separately.[8] As expected, there were significant differences in smoking status between the two groups. For example, a higher percentage of blacks (35.7%) than whites (32.8%) were never smokers and current smokers (28.2% and 18.6%, respectively), whereas a larger percentage of whites (48.6%) than blacks (36.2%) were former smokers. Furthermore, 26.5% of whites had quit 20+ years ago compared with only 15.9% of blacks. These smoking patterns are consistent with results from prior studies summarized above: blacks were more likely to be current smokers and to have stopped smoking more recently relative to whites.

There were also significant black-white differences in the other explanatory variables. Black males were on average slightly younger than white males, and they were less likely to be currently married and more likely to have been widowed, divorced, separated, or never married. About 35.7% of black males had less than high school education compared with 15.6% of white males, and blacks were less likely to have attended at least 4 years of college. Blacks also had significantly lower family incomes, and they were more likely to live in the south.

Table 1.5 presents the hazard ratios from the multivariate models for black males relative to white males and for former and current smokers relative to non-smokers. All models control for age at baseline, BMI, and survey year. Model 2 adds the smoking status

[8] In this section, from this point forward "black" refers to non-Hispanic black males and "white" refers to non-Hispanic white males unless otherwise specified.

variable, and Model 3 adjusts for marital status, educational attainment, family income, and region of residence. In Model 1, the hazard of dying is about 50% higher for black males than for white males controlling for age at baseline, BMI, and survey year. The introduction of smoking status decreases this relative risk by 20%[9], suggesting that smoking plays some role in the excess mortality of black males relative to white males at ages 50 and above. However, a far greater reduction is observed when we introduce controls for marital status, educational attainment, family income, and region of residence. The hazard ratio for non-Hispanic black males is reduced by 62%, from 1.39 to 1.15. These results support the notion that socioeconomic circumstances are the key explanations for the high mortality of black males.

As seen in **Table 1.5**, smoking is a highly significant predictor of mortality, even when controlling for potential confounding variables. The risks vary by time since quitting among former smokers and by smoking intensity among current smokers. Compared to never smokers, the hazard ratio is highest for current heavy smokers (2.66, Model 3), those who smoked 1+ packs per day. Current light smokers (< 1 pack per day) and former smokers who quit less than 5 years ago (2.28 and 2.37, respectively, Model 3) have similar relative risks compared to never smokers. The high risk of dying among recent quitters is consistent with other studies and may result from individuals quitting smoking due to illness (DHHS 1990). Among former smokers, the hazard ratios decline by time since quitting, and those who quit 30+ years ago experience risks that are not significantly different from those of never smokers. The introduction of other explanatory variables (Model 3 versus Model 2) results in small declines in the magnitudes of the

[9] (1.49-1.39)/(1.49-1.00)=0.20.

hazard ratios for the various smoking categories, but they remain highly significant (with the exception of the hazard ratio for those who quit 30+ years ago, which is not significant in either model).

We speculated that the associations between smoking status and mortality might vary between blacks and whites. To test this possibility, we included interaction terms between race and smoking status in our main effects Model 3. These interaction terms were not jointly significant, providing no support for the hypothesis that the effects of smoking differ between black males and white males.

Table 1.6 shows the population attributable risk fractions (PAF) due to smoking by smoking category for white and black males. Attributable risk fractions are presented as percentages, and they reflect the percentage of deaths that could be avoided if smoking were eliminated (Mehta and Chang 2009). The reference category for all estimates is never smokers. Because the effects of smoking did not vary between black males and white males, we re-estimated Model 3 without controlling for race and used the hazard ratios associated with the various smoking categories from this model to calculate the PAFs by smoking status.[10]

Overall, we estimate that 29.4% and 33.1% of deaths were attributable to smoking among white males and black males, respectively. Among whites, the largest contribution is made by current heavy smokers (9.8%); the contributions of other smoking categories are only about half or less than half this size. In contrast, among blacks, the largest contribution is made by current light smokers (13.2%) followed by current heavy

[10] The results are essentially the same if we use coefficients from a model that includes race.

smokers (7.8%). The contributions of most other smoking categories are less than half the size of those made by current smokers.

Discussion

Smoking has significantly impacted American mortality and continues to be a leading cause of morbidity and mortality today. To the best of our knowledge, this is the first study to investigate the contribution of smoking-attributable deaths to mortality trends among blacks or to black-white mortality differences at older ages over time. We employed multiple methods and data sources to provide a comprehensive assessment of this contribution.

We find that smoking has reduced life expectancy at age 50 considerably, especially among males. These reductions range from 3.02 years in 1990 to 2.41 years in 2005 among white males, and from 4.47 years in 1990 to 3.18 years in 2005 among black males. Smoking has also contributed to the black-white gap in male life expectancy at age 50. This contribution reached its peak in 1985 and 1990 at 1.45 years (approximately 34-40% of the black-white gap in male life expectancy at age 50), but has subsequently declined to 0.78 years (20% of the gap) by 2005. Trends in smoking-attributable mortality differ for females. The smoking epidemic began later among females, and this pattern is reflected in our results. Smoking-attributable mortality reduced life expectancy at age 50 for white females by only 0.79 years in 1980; the respective amount for black women was 0.86 years. By 2005, these contributions had increased to 1.90 years and 1.83 years among white and black females, respectively. In earlier years, smoking made only a minor contribution to the black-white gap in female life expectancy at age 50, but in more

recent years smoking has had a more detrimental impact on mortality at older ages for white females than black females.

We know of only one prior study that has estimated the contribution of smoking to mortality by race/ethnicity, in this case among eight population subgroups in the U.S. which were defined based on race and geography (Danaei et al. 2010). This study estimated relative risks associated with current and former smoking as well as other risk factors (e.g., obesity), and then used these relative risks to estimate the contribution of these risk factors to life expectancy at birth. The authors estimated that in the absence of smoking, white males stood to gain between 2.4-3.3 years in life expectancy at birth in 2005 depending on the subgroup, whereas black males stood to gain between 2.6-3.1 years. These estimates were 1.4-2.1 years for white females and 1.5-1.8 years for black females (Ibid.). Although these estimates apply to a single year (2005) and are not directly comparable to our results because we consider deaths and life expectancy gains only above age 50, they are generally consistent with our findings.

Preston and Wang (2006) predicted that sex differences in mortality would narrow dramatically in coming decades. Our analysis also points to the importance of smoking in explaining sex differences in mortality and suggests that the contribution of smoking to the sex gap in life expectancy has differed between population subgroups in the U.S. over time. We found that the contribution of smoking-attributable mortality to the sex gap in life expectancy at age 50 has been larger among blacks than whites. While the sex gap in life expectancy at age 50 had already narrowed substantially among whites by 2005, given that smoking still accounted for roughly 30% of the sex gap among blacks (nearly

twice the corresponding figure for whites) in 2005, we may expect to see a continued and even more dramatic narrowing of the sex gap in life expectancy at age 50 among blacks.

Based on the indirect estimation approach, we found that the percentage of deaths attributable to smoking above age 50 ranged from roughly 24%-29% among black males and 20%-23% among white males between 1997 and 2005. These estimates are consistent with those of Preston, Glei, and Wilmoth (2010), who estimated the smoking-attributable fractions among all males to be 24% in both 1980 and 2003 (these figures would necessarily be more similar to the fractions for white males, given that they make up a larger proportion of the total male population).

In the second part of our analysis, based on a regression approach and the NHIS data, we found that although the population attributable fractions of deaths due to smoking among black men (29.43%) and white men (33.08%) exceeded the estimates based on the indirect approach, the magnitude of the black-white difference was quite similar (3.65% compared to 4.89%, the black-white difference from the indirect approach in 2001, roughly the midpoint of the period 1997-2006). Although the fractions produced from direct estimation are somewhat larger than those produced from indirect estimation, they are in line with other published results. For example, Mehta and Chang (2009) found that smoking-attributable mortality was 50% for males and 35% for females using a sample aged 50-61 at baseline from the Health and Retirement Study (1992-2004), another nationally representative survey.

We also demonstrate that smoking contributes to the relative differences in mortality among black males and white males at ages 50 and above. The black excess risk was

reduced by 20% when smoking status at the start of the mortality follow-up was included in the model. At the same time, the reduction in the black excess risk was much smaller than the reduction resulting from the inclusion of socio-demographic characteristics (62%). Thus, while black-white differences in smoking behavior contribute to black-white disparities in male mortality, black-white differences in SES play a much larger role (see also Hayward et al. 2000). Finally, we speculated that the effects of smoking on mortality might differ between blacks and whites, but found no support for this hypothesis. This result is consistent with Pampel and Rogers (2004), who found that interactions between race and smoking status were not significant predictors of morbidity, mortality, or self-rated health. Danaei et al. (2010:4) reviewed several observational studies and trials and concluded that "the current evidence indicates that while the absolute effects (e.g., excess mortality rate) of risk factors vary by race, their proportional effects (i.e., relative risks) did not vary appreciably by race and ethnicity."

The deleterious effects of smoking change only modestly and are preserved after controlling for marital status, educational attainment, family income, and region. The excess risks associated with smoking are striking. Current smokers, 93% of whom have smoked for more than 30 years, are 2.3-3 times more likely to die than never smokers. Former smokers who have quit most recently have elevated risks that are slightly higher than those of current light smokers, with the risk diminishing considerably among former smokers as the time since quitting increases. In fact, those who stopped smoking 30 years prior to the baseline survey have mortality risks similar to never smokers. Most of these individuals had quit prior to age 40. These results are consistent with Peto et al. (2000),

who found that smokers who quit before middle age avoid over 90% of lung cancer risk attributable to smoking.

Overall, the results from this study demonstrate that former smokers are a highly heterogeneous group and should be differentiated when estimating the effects of smoking on mortality. Simply comparing levels of ever-smoking prevalence without taking into account differences in smoking duration, intensity, and cessation can mask differences among subgroups, and using a never, former, and current smoker categorization may not be sufficient to adequately capture the impact of smoking on mortality.

A number of factors may play a role in explaining why the burden of smoking-attributable mortality is greater among black males than white males. Our analysis supports the hypothesis that longer smoking durations and substantially lower smoking cessation rates among blacks may be contributing to their higher burden of smoking-related morbidity and mortality. Black-white differences in exposure to toxins per cigarette (reviewed above) and the types of cigarettes smoked may also be part of the explanation. Blacks are more likely to smoke cigarettes with high tar yields and mentholated cigarettes, although it has not been established whether mentholated cigarettes increase the risk of lung cancer and other smoking-related diseases. In addition, factors such as the experience of cumulative disadvantage over the life course and lack of access to timely and high quality health care and other resources are likely to result in higher mortality among black smokers, who may have lower success in quitting smoking and poorer outcomes once they have developed smoking-related diseases.

Strengths

The main strengths of this study lie in the pairing of direct and indirect estimation approaches used to estimate smoking-attributable mortality and our efforts to include detailed information on individual smoking histories. The indirect estimation method has the potential to produce more accurate estimates of smoking-attributable mortality because it does not rely on self-reported smoking behavior at a single point in time. For example, Stringhini et al. (2010) found that compared to studies using repeated measures of health behaviors (including smoking), studies using only a single assessment of health behaviors tend to substantially underestimate the contribution of these health behaviors to social inequalities in mortality. In addition, it is relatively straightforward to estimate the impact of smoking-attributable deaths by age group on life expectancy at age 50 using this method. It is particularly instructive to do so because smoking-attributable mortality varies by age and deaths at different ages contribute differentially to life expectancy (i.e., deaths occurring at younger ages contribute more to life expectancy than those occurring at older ages). Life expectancy is also an intuitive and interpretable measure which allows us to explore more fully the contribution of smoking to black-white mortality differentials.

In the first part of the analysis, which uses indirect estimation, we are able to address concerns of age misreporting at the older ages by borrowing life table quantities from the NCHS life tables for age groups above 85. We also examine the number of expected years lived between ages 50 and 84 ($\frac{T_{50}-T_{85}}{l_{50}}$) as an alternate measure to life expectancy at age 50 which should be less affected by age misreporting at the oldest ages (results not shown). Our results are robust to the age range used in the analysis. They are also robust

to the use of model coefficients estimated based on U.S. data and data from multiple developed countries. Estimates of smoking-attributable mortality among older females are slightly lower using the Fenelon and Preston (2012) coefficients, which are based on U.S. data and which the authors recommend for analyses in the U.S. because of its more mature smoking epidemic relative to other developed countries.

An additional strength is our ability to compare results obtained from the indirect estimation to those obtained from survey data with detailed information on smoking histories for a similar age range and overlapping study periods. Using the NHIS data, we can also control for potential confounders of the relationship between smoking and mortality. In addition, we are able to examine whether including or excluding Hispanics affects our conclusions. As noted above, we were unable to restrict our analyses to non-Hispanic whites and non-Hispanic blacks in the vital statistics and census data. Our conclusions are unaffected by the inclusion of Hispanics, including estimates of the population attributable fraction of deaths due to smoking (see **Table A1.1**). While the coefficients on smoking status become slightly smaller, they remain very close to those obtained from regressions based on a sample of non-Hispanic whites and non-Hispanic blacks. Finally, our smoking status variable incorporates more information on smoking behavior than most other studies, which tend to control only for whether the individual is a never, former, or current smoker. We were able to capture both smoking duration and intensity among current smokers, as well as recency of quitting among former smokers.

Limitations

There are also some potential limitations to our analysis. The indirect estimation method relies on the strength of the association between smoking and lung cancer mortality. If the quality of death certification for lung cancer varies between blacks and whites, some bias could be introduced. There is some evidence that racial identity may affect the classification of certain causes of death (e.g., cirrhosis and homicide) on death certificates (Noymer, Penner, and Saperstein 2011). However, because of the strong link between lung cancer and smoking and the high quality of death certification for lung cancer in the United States, we do not believe this should be a major problem. For example, despite their lower health care access (which may make them less likely to be diagnosed), black males have a higher incidence of lung cancer. Their higher mortality rates are also consistent with lower survival from lung cancer relative to white males. Together, these observations suggest that the bias in certification may not be large if present. Another potential source of bias may be introduced, however, if the coefficients used to estimate smoking-attributable mortality differ between blacks and whites. We do not expect this to be a major issue.

Another concern is that the expected lung cancer death rates among non-smokers were drawn from the CPS-II, which had a study population composed of volunteers who were more likely to be white, middle-class, and college-educated than the general U.S. population. Thus, the method may produce overestimates of smoking-attributable mortality. However, the rates of lung cancer among non-smokers observed in this study were similar to those observed in other samples, and the CPS-II is likely to be the current best source of these estimates given that it “remains the largest epidemiological study of

its kind ever attempted in the history of medical science," having enrolled a total of 1.2 million participants (Klausner 1997: iv; Preston, Glei, and Wilmoth 2010).

The primary limitation of the direct estimation approach based on the NHIS linked mortality files is the reliance on self-reported smoking information from a cross-sectional survey. It is possible that recall bias and misclassification of smoking status may result in underestimates of smoking-attributable mortality. Smoking status is measured only at baseline and therefore we do not know whether this behavior changed during the follow-up period and how accurately it captures individuals' past smoking behavior. For example, individuals entering the survey at ages 50 and above may now smoke fewer cigarettes than they did at points earlier in their lifetimes, and the number of cigarettes they currently report smoking may not reflect lifetime smoking intensity. It is possible that smokers have altered their smoking behavior over time in response to factors such as illness, changes in cigarette taxation levels, and changing tar and nicotine yields in manufactured cigarettes (Bach et al. 2003).

The extent to which these factors vary between blacks and whites is fairly ambiguous. It may be the case that smoking information is more accurate for blacks than for whites since on average, black smokers start smoking at older ages and have quit more recently compared to whites. However, the implications for the direction of potential biases and how this would affect differences in smoking-attributable mortality between the groups is unclear. We were not able to control for intensity of smoking among former smokers, as the questions eliciting this information were not asked during the survey waves under consideration. We are also unable to control for other potentially relevant exposures such as cigars, smokeless tobacco, or occupational exposures using the NHIS.

Black-white differences in occupational exposures may contribute to higher smoking-attributable mortality among blacks. For example, exposure to asbestos, certain metals, radon, and ionizing radiation elevates lung cancer risk (Tyczynski et al. 2003). Estimates of the attributable risk from occupational exposures are highly time- and context-specific. Most U.S. case-control studies estimate that the fraction of lung cancer attributable to workplace exposures lies in the range of 6-17% among males (Steenland et al. 2003). However, not all studies control for the confounding effects of smoking, and others have found an interaction between smoking and other lung carcinogens, particularly asbestos (Tyczynski et al 2003). Sample characteristics and sizes and the ability to capture the intensity and cumulative burden of exposures differ widely between studies (Ibid.; IARC 1992). Although the contribution of occupational exposures to lung cancer is large relative to most other exposure classes, it is small compared to that of cigarette smoking in industrialized countries (Alberg and Samet 2003). There is wide agreement that 90% of lung cancer mortality in the U.S. is attributable to smoking (Oza et al. 2011; Tyczynski et al 2003).

Finally, the PAF estimates are interpreted as the proportion of deaths that could be avoided if smoking were eliminated from the population; in other words, this is the proportion of deaths that could be avoided if all current and former smokers experienced the mortality risks of never smokers. However, even with the extensive demographic and socioeconomic controls included in the full model, unobserved heterogeneity between smokers and non-smokers may remain. It may be the case that smokers would experience worse health and higher mortality risks even if they had not smoked.

Conclusion

Overall, our findings are highly consistent with the previous literature, which finds that blacks suffer disproportionately from smoking-related diseases despite lower levels of smoking prevalence and lower intensities of smoking among current smokers. Some have suggested that these trends are due to longer smoking durations and lower smoking cessation rates among blacks. This study, which builds both duration and time since quitting into the measure of smoking status, supports this hypothesis. It also emphasizes the need to consider additional factors beyond levels of smoking initiation and ever-smoking prevalence (**Figure 1.2**) by using more detailed categorizations of smoking status. The elevated mortality risks among recent quitters and current smokers are substantial and highlight the need for smoking cessation interventions that better address the needs of all groups.

Tables

Table 1.1. Percentage of Deaths Attributable to Smoking at Ages 50+ by Sex Using the Indirect Estimation Method, Blacks and Whites, United States, Select Years Between 1980 and 2005

	Male		Female	
Year	**Whites**	**Blacks**	**Whites**	**Blacks**
1980	22.8	28.3	6.4	6.8
1985	24.0	30.8	8.8	9.0
1990	25.1	32.3	12.0	11.7
1995	23.8	30.5	14.2	12.9
2000	22.0	27.0	15.3	13.4
2005	20.5	24.2	15.8	14.4

Table 1.2. Life Expectancy at Age 50 and the Black-White Gap in Life Expectancy at Age 50 With and Without Smoking-Attributable Deaths Using the Indirect Estimation Method, United States, Select Years Between 1980 and 2005

Males	***With smoking***			***Without smoking***			
Year	**Whites**	**Blacks**	**Gap (W-B)**	**Whites**	**Blacks**	**Gap (W-B)**	**% Gap due to Smoking**
1980	25.21	21.69	3.52	27.95	25.49	2.46	30.15
1985	25.80	22.15	3.65	28.68	26.48	2.20	39.73
1990	26.76	22.56	4.20	29.78	27.03	2.76	34.34
1995	27.41	23.11	4.30	30.26	27.22	3.05	29.09
2000	28.23	24.30	3.93	30.83	27.85	2.98	24.17
2005	28.98	25.11	3.87	31.39	28.29	3.09	20.12

Females	***With smoking***			***Without smoking***			
Year	**Whites**	**Blacks**	**Gap (W-B)**	**Whites**	**Blacks**	**Gap (W-B)**	**% Gap due to Smoking**
1980	30.92	27.37	3.55	31.71	28.23	3.48	1.95
1985	31.19	27.93	3.26	32.31	29.13	3.18	2.46
1990	31.69	28.25	3.44	33.18	29.78	3.39	1.19
1995	31.80	28.50	3.30	33.54	30.15	3.39	-2.76
2000	32.06	29.17	2.89	33.94	30.91	3.03	-4.81
2005	32.64	29.99	2.65	34.54	31.82	2.72	-2.66

Table 1.3. Life Expectancy at Age 50 and the Sex Gap in Life Expectancy at Age 50 With and Without Smoking-Attributable Deaths Using the Indirect Estimation Method, United States, Select Years between 1980 and 2005

Blacks	***With smoking***			***Without smoking***			
Year	**Females**	**Males**	**Gap (F-M)**	**Females**	**Males**	**Gap (F-M)**	**% Gap due to Smoking**
1980	27.37	21.69	5.68	28.23	25.49	2.74	51.66
1985	27.93	22.15	5.78	29.13	26.48	2.65	54.17
1990	28.25	22.56	5.69	29.78	27.03	2.75	51.64
1995	28.50	23.11	5.39	30.15	27.22	2.94	45.57
2000	29.17	24.30	4.87	30.91	27.85	3.06	37.28
2005	29.99	25.11	4.88	31.82	28.29	3.52	27.84

Whites	***With smoking***			***Without smoking***			
Year	**Females**	**Males**	**Gap (F-M)**	**Females**	**Males**	**Gap (F-M)**	**% Gap due to Smoking**
1980	30.92	25.21	5.71	31.71	27.95	3.77	34.02
1985	31.19	25.80	5.39	32.31	28.68	3.63	32.66
1990	31.69	26.76	4.93	33.18	29.78	3.39	31.22
1995	31.80	27.41	4.40	33.54	30.26	3.28	25.40
2000	32.06	28.23	3.84	33.94	30.83	3.11	19.00
2005	32.64	28.98	3.66	34.54	31.39	3.15	13.95

Table 1.4. Descriptive Statistics, Means and Standard Deviations or Percentages for Individual-level Characteristics, Males, National Health Interview Survey, 1997-2003

	Total	**Non-Hispanic Whites (91%)**	**Non-Hispanic Blacks (9%)**	**p-value**
Variable	N=27,164	N=23,701	N=3,463	
Smoking Behavior				
Never smoker	33.1	32.8	35.7	
Former smoker, quit 0-4 years ago	5.6	5.5	5.7	
Former smoker, quit 5-9 years ago	4.4	4.4	4.5	
Former smoker, quit 10-19 years ago	12.0	12.2	10.1	0.0000
Former smoker, quit 20-29 years ago	11.7	12.0	8.8	
Former smoker, quit 30+ years ago	13.8	14.5	7.1	
Current smoker, <1 pack per day	7.3	6.2	18.1	
Current smoker, 1+ packs per day	12.2	12.4	10.1	
Body Mass Index				
Underweight, BMI < 18.5	0.7	0.7	1.0	
Normal, BMI 18.5-24.9	27.8	27.6	30.0	0.0000
Overweight, BMI 25-29.9	46.7	47.1	42.8	
Obese I, BMI 30-34.9	18.5	18.5	18.5	
Obese II and III, BMI 35+	6.3	6.1	7.8	
Socio-demographic Characteristics				
Mean age at baseline (SD)	62.776 (0.069)	62.894 (0.074)	61.610 (0.190)	0.0000
Marital status				
Never married	5.0	4.5	9.5	0.0000
Currently married	76.3	78.1	58.6	
Widowed/divorced/separated	18.7	17.3	32.0	
Educational attainment				
Less than high school	17.4	15.6	35.7	
High school graduate	31.4	31.5	30.0	0.0000
1-3 years of college	23.7	24.0	20.5	
4+ years of college	27.5	28.9	13.9	
Mean family income (10,000s) (SD)	5.510 (0.035)	5.653 (0.037)	4.087 (0.105)	0.0000
Region				
Northeast	20.2	20.6	16.1	
North Central	26.6	27.4	18.4	0.0000
South	37.2	35.2	57.4	
West	16.0	16.8	8.1	
Mean Years of Follow-up				
Alive (censored)	6.130	6.136	6.071	
Dead	3.807	3.817	3.732	

Source: Author's calculations based on 1997-2003 NHIS data linked to the National Death Index through 2006; p-values correspond to t-tests and chi-square tests for the equality of means/distributions of variables between non-Hispanic black and white males.

Table 1.5. Hazard Ratios from Cox Regression Models Predicting Mortality, National Health Interview Survey, Non-Hispanic White and Non-Hispanic Black Males aged 50-84, 1997-2003 (reference category in parentheses)

	Model 1	**Model 2**	**Model 3**
Race (Non-Hispanic white)			
Non-Hispanic black	1.49***	1.39***	1.15**
Smoking Status (never smoker)			
Former smoker, quit 30+ years ago		1.06	1.06
Former smoker, quit 20-29 years ago		1.21**	1.19**
Former smoker, quit 10-19 years ago		1.70***	1.66***
Former smoker, quit 5-9 years ago		2.30***	2.14***
Former smoker, quit 0-4 years ago		2.57***	2.37***
Current smoker, <1 pack per day		2.52***	2.28***
Current smoker, 1+ packs per day		3.05***	2.66***
N (unweighted)	27,164	27,164	27,164

Notes: All models control for survey year, age at baseline, and BMI. Model 3 also includes controls for marital status, educational attainment, family income, and region of residence.
* p<0.05; ** p<0.01; *** p<0.001

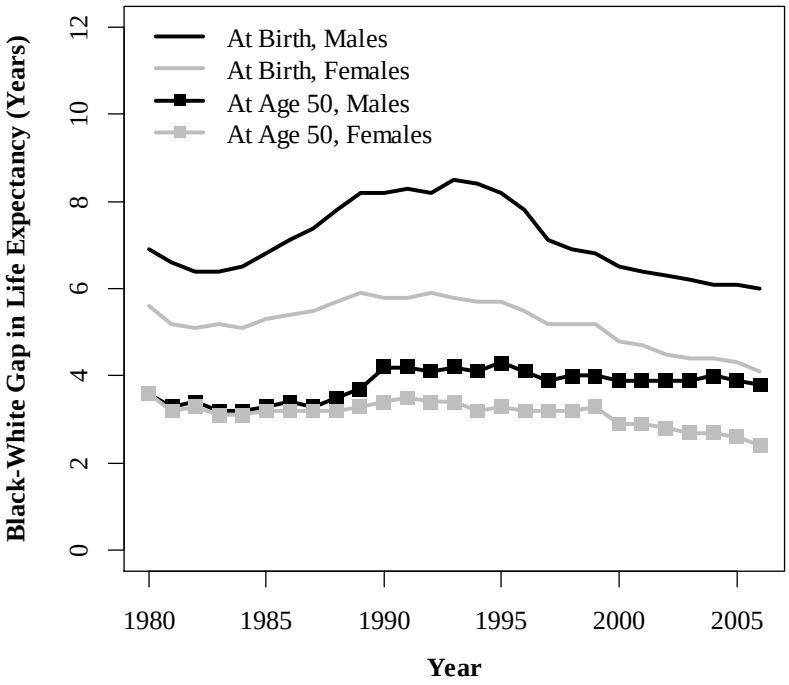
Table 1.6. Distribution of Population Attributable Risk Fractions (PAF) by Smoking Status, National Health Interview Survey, Non-Hispanic White and Non-Hispanic Black Males aged 50-84, 1997-2003

Reference: never smoker	Non-Hispanic White Males	Non-Hispanic Black Males
Former smoker, quit 30+ years ago	0.77	0.33
Former smoker, quit 20-29 years ago	1.60	0.94
Former smoker, quit 10-19 years ago	5.49	4.27
Former smoker, quit 5-9 years ago	3.22	2.50
Former smoker, quit 0-4 years ago	4.41	4.02
Current smoker, <1 pack per day	4.13	13.19
Current smoker, 1+ packs per day	9.82	7.82
Total	29.43	33.08

Notes: Based on hazard ratios from a model controlling for survey year, age at baseline, BMI, marital status, educational attainment, family income, and region of residence.

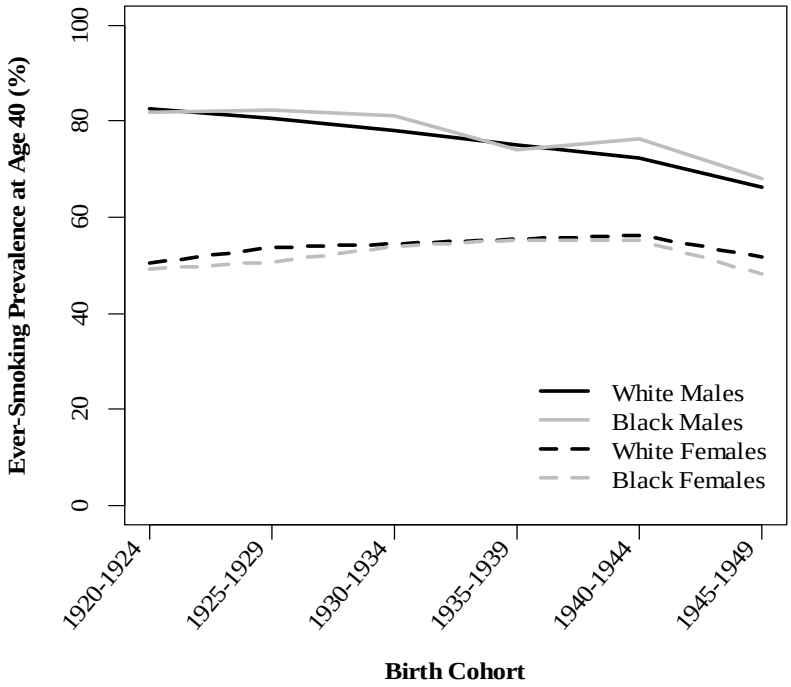
Figures

Figure 1.1. Black-White Gap in Life Expectancy at Birth and at Age 50 by Sex, 1980-2006



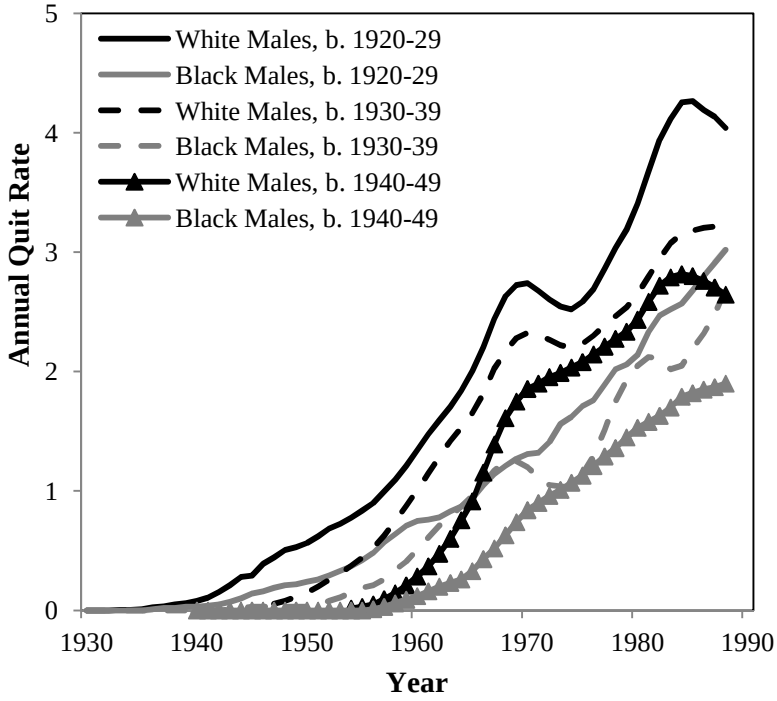
Source: NCHS Life Tables 1980-2006.

Figure 1.2. Ever-Smoking Prevalence at Age 40 by Sex and Birth Cohort, Blacks and Whites Born Between 1920 and 1949



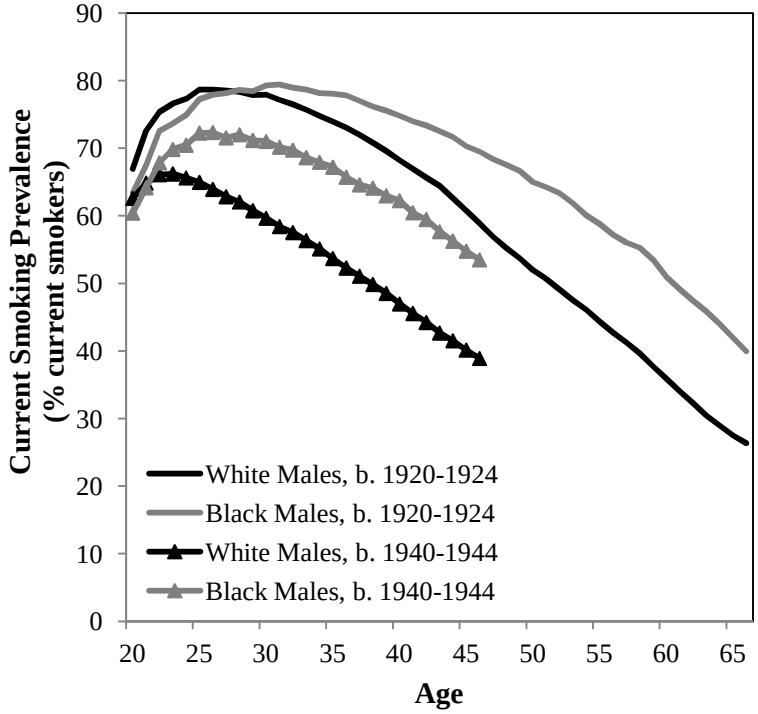
Source: Burns et al. (1997).

Figure 1.3. Annual Quit Rates for Males by Birth Cohort, Blacks and Whites Born Between 1920 and 1949, 1930-1990



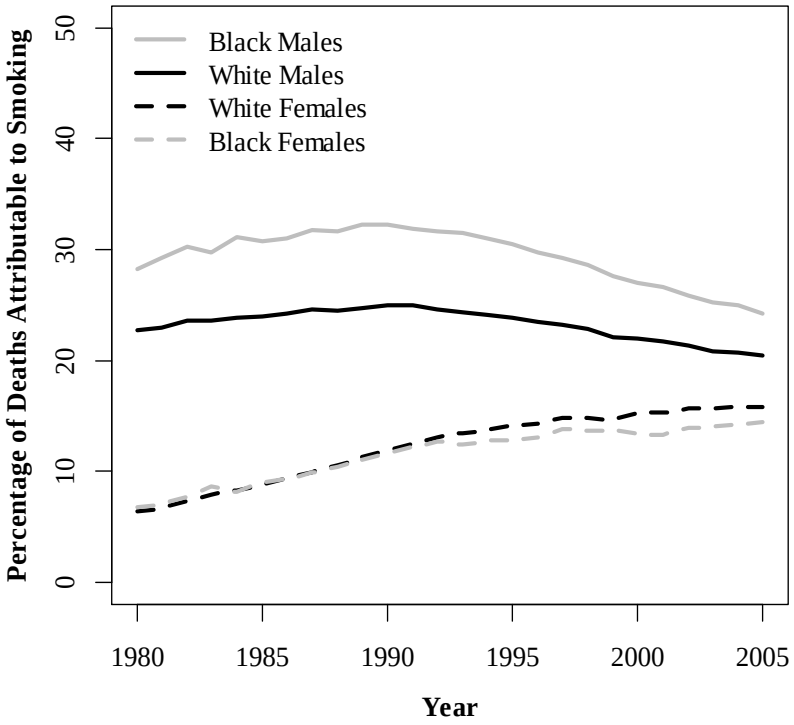
Source: Burns et al. (1997).

Figure 1.4. Current Smoking Prevalence by Age and Birth Cohort, Black and White Males Born Between 1920-1924 and 1940-1944



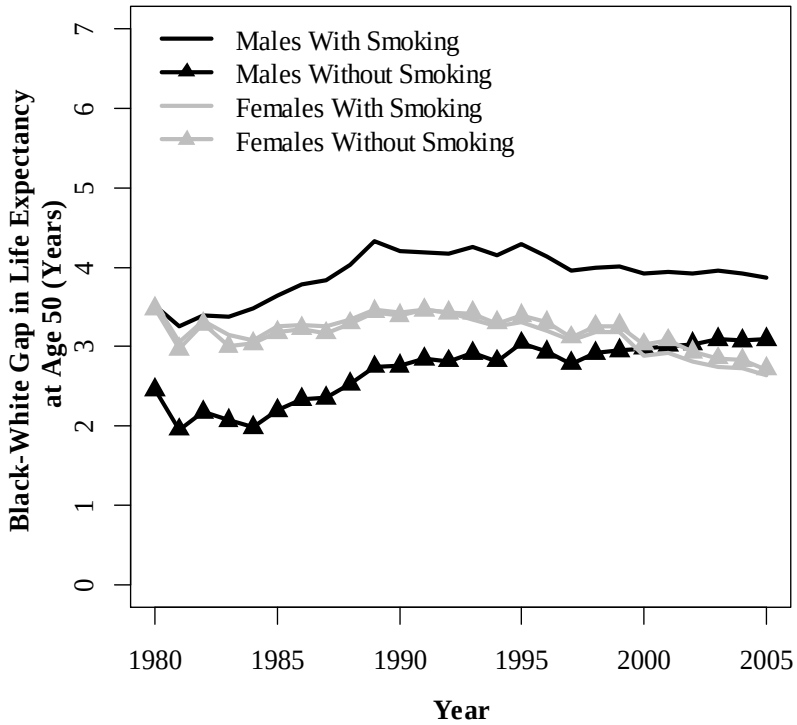
Source: Burns et al. (1997).

Figure 1.5. Percentage of Deaths Attributable to Smoking Above Age 50 by Sex Among Blacks and Whites, United States, 1980-2005



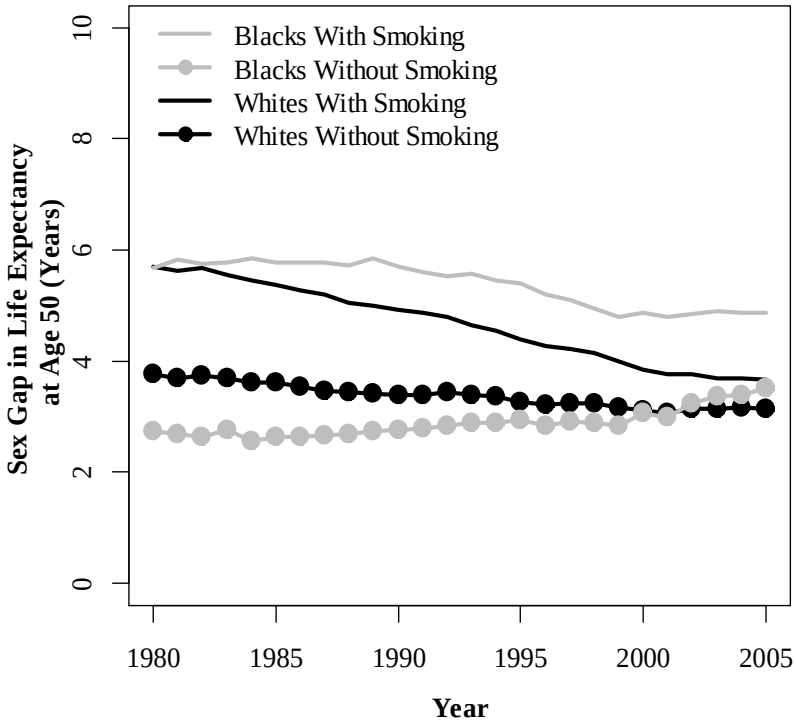
Source: Calculations by the author.

Figure 1.6. Black-White Gap in Life Expectancy at Age 50 With and Without Smoking-Attributable Deaths by Sex, United States, 1980-2005



Source: Calculations by the author.

Figure 1.7. Sex Gap in Life Expectancy at Age 50 With and Without Smoking-Attributable Deaths by Race, United States, 1980-2005



Source: Calculations by the author.

CHAPTER II. International Comparisons of U.S. Mortality[11]

Introduction

Life expectancy in the United States is currently among the lowest of all high-income countries. Research and policy discussions have focused largely on cross-national mortality differences at older ages (e.g., at ages 50 and above). This chapter provides a comprehensive assessment of U.S. mortality relative to a set of 16 high-income peer countries in the most recent period for which data are available. In the first part of the analysis, I use cross-national mortality data to identify age and cause of death contributions to the U.S. mortality disadvantage. This study finds that mortality differences below age 50 account for 67% of the gap in life expectancy at birth between American males and their counterparts in the comparison countries. Among females, this figure is 41%. The major causes of death responsible for Americans' excess years of life lost below age 50 are unintentional injuries, noncommunicable diseases, perinatal conditions, and homicide. Together, these causes account for 84% and 85% of the difference in years of life lost below age 50 between the U.S. and the mean of other countries for males and females, respectively. In the second part of the analysis, I examine how the age-specific mortality rates of cohorts of Americans born between 1850 and 2004 compare to that of their counterparts in the comparison countries. Period rankings of U.S. death rates reveal a distinctive age pattern: American males and females perform very poorly before age 75, experiencing among the highest death rates among

[11] Material from this chapter has appeared in Ho JY. 2013. "Mortality Under Age 50 Accounts For Much of the Fact That US Life Expectancy Lags That of Other High-Income Countries." *Health Affairs* 32(3): 459–467.

the comparison countries, but perform exceptionally well at the oldest ages, experiencing among the lowest death rates among the comparison countries. I find that this pattern is replicated for over a century's worth of American birth cohorts. This study highlights the importance of mortality at the younger ages and injury-related mortality as major contributors to low life expectancy in the United States and calls attention to an enduring pattern of Americans' poor mortality performance prior to age 75 and improved performance thereafter which holds for both periods and cohorts.

Background

Americans reside in one of the richest and most technologically-advanced countries in the world, yet they are less healthy than their counterparts in other wealthy industrialized countries. Life expectancy in the United States is among the lowest of all high-income countries. Not only can Americans expect to live fewer years, on average, but they also experience a greater burden of disease during those years (Thorpe, Howard, and Galactionova 2007; Banks, Muriel, and Smith 2010; Martinson, Teitler, and Reichman 2011). On the cusp of the 21st century, the United States ranks poorly in terms of both life expectancy at birth and mortality through most of the lifespan, and its position has been deteriorating over time (National Research Council [NRC] 2011).

Life expectancy at birth in the United States has increased over time, but not as quickly as in its peer countries. Between 1960 and 2007, American males gained 9.0 years in life expectancy at birth, while males in a set of 16 peer countries[12] gained 10.2 years on average. Over the same period, life expectancy at birth for American males went from

[12] These countries are: Australia, Austria, Canada, Denmark, Finland, France, Germany, Italy, Japan, the Netherlands, Norway, Portugal, Spain, Sweden, Switzerland, and the United Kingdom.

being ranked 12th to 17th among this set of countries. American females gained 7.5 years in life expectancy at birth between 1960 and 2007, nearly three years less than females in the comparison countries, who gained an average of 10.2 years over the same period. Life expectancy at birth for American females has gone from being ranked 10th to 16th. Similarly, the rankings of U.S. age-specific mortality rates for both males and females have deteriorated over time, most dramatically for females between 1975 and 2005. The move towards poorer ranks has been equally striking for males, but the deterioration in their rankings was heavily concentrated in the decade between 1995 and 2005 (Ho and Preston 2010).

In 2007, American males had the lowest life expectancy at birth (75.6 years) among the set of comparison countries (**Table 2.1**). This figure was 3.7 years lower than the world leader, Switzerland, and 2.2 years lower than the average of the comparison countries. In 2007, American females had the second lowest life expectancy at birth (80.8 years), just ahead of Danish females. Their life expectancy was 5.2 years lower than Japanese females, the world leader, and 2.3 years lower than the average of the comparison countries.[13] These life expectancy differences are non-trivial.

The following thought experiment illustrates just how far Americans have fallen behind their peers in other high-income countries. Best-practice female life expectancy has increased at a rate of roughly 2.5 years per decade for the past 160 years (Oeppen and

[13] This picture remains largely unchanged in 2009, the latest year for which data for all 17 countries is currently available. Among males, Australia have overtaken Switzerland as the world leader at 79.7 years, 3.6 years greater than life expectancy at birth among American males in 2009. The average difference in life expectancy at birth between American males and males in the comparison countries remained at 2.2 years. Among females, Japan remained the world leaders in 2009. Their life expectancy exceeded that of American females by 5.4 years, and the average difference in life expectancy at birth between American females and females in the comparison countries increased to 2.4 years.

Vaupel 2002). Even if the U.S. were able to achieve this rate of increase and if the other countries did not experience any further gains in life expectancy, it would still take nearly 15 years for males and over two decades for females to catch up to the current world leaders. The U.S. would require nearly a decade to catch up to the average of the other 16 countries. Again, this exercise assumes that life expectancy in the comparison countries would remain at its current levels and that the U.S. would be able to match the best-practice rate of increase in life expectancy. It seems highly unlikely that either condition would be met in reality. In the decade between 1997 and 2007, American males and females gained 1.8 and 1.0 years in life expectancy at birth, respectively, far less than the best-practice rate of 2.5 years per decade (Arias 2011). At these rates (and assuming no additional life expectancy improvements in the other countries), it would take the U.S. two and five decades to catch up to the world leaders for males and females, respectively.

Proposed Explanations for Lagging Life Expectancy in the United States

Over time, U.S. life expectancy has fallen further and further behind other developed countries, and mortality improvements have been slower for both males and females. This is occurring on the heels of phenomenal mortality declines experienced over the course of the 20th century, strong economic growth and performance, and among the highest levels of per capita health care spending in the world (Reinhardt, Hussey, and Anderson 2004). A substantial body of research has focused on addressing why the U.S. performs so poorly relative to other developed countries. By and large, these studies have investigated health and mortality differences at older ages. For example, the recent National Research Council Panel on Understanding Divergent Trends in Longevity in

High-Income Countries focused exclusively on ages 50 and above (NRC 2011). One of the most consistent findings from these studies has been that Americans experience a greater burden of disease than their European counterparts (Avendano et al. 2009; Banks, Muriel, and Smith 2010; Crimmins, Garcia, and Kim 2010; Martinson, Teitler, and Reichman 2011; Thorpe, Howard and Galactionova 2007).

In general, explanations of the U.S. life expectancy disadvantage fall within the following categories: (1) poorer functioning of and lack of access to the U.S. health care system, (2) a greater prevalence of unhealthy behaviors in the U.S., (3) higher levels of income inequality and poverty coupled with weaker social safety nets in the U.S., and (4) lower levels or quality of social capital and interactions in the U.S. relative to other countries. Ideally, a tight linkage between the proposed mechanism and health and mortality should exist, and there should be a greater prevalence or severity of the factor in the United States. For several of these proposed explanations, much of the evidence base remains speculative due to the difficulty of establishing one or both of these conditions.

Income Inequality and Social Safety Nets

Whether differences in income inequality can explain differences in health across nations remains contested (e.g., Wilkinson 1992; Wilkinson 1996; Lynch et al. 2004; Beckfield 2004). Higher levels of income inequality can be related to poorer national health through a purely mechanistic effect, also referred to as the aggregation effect or a statistical artifact. Drawing on the work of Preston (1975), Deaton (2002) points out that if the curvilinear relationship between income and health holds within as well as across countries, in the scenario where two countries have the same average income but

different distributions of income, all else being equal, the society with the higher level of income inequality would have poorer health due to purely mechanical effects. What remains debatable is whether income inequality itself affects health apart from the individual-level relationship between income and health.

Given the levels of development the countries in this analysis have achieved, income inequality is hypothesized to act primarily through relative rather than absolute deprivation. People's negative perceptions of their positions within the social hierarchy may be translated into negative emotions and poorer health through biological pathways induced by the stress response and psychosocial strain (Wilkinson 1992; Lynch et al. 2000; Marmot 2005; Schnittker and McLeod 2005). Social comparisons may contribute to psychosocial strain and social isolation for lower-income residents of wealthier neighborhoods, and institutions like hospitals and schools may be of lower quality or more responsive to the needs and demands of wealthier residents (Vartanian and Houser 2010). Income inequality is also hypothesized to act through decreased social capital and social cohesion and the adoption of negative health behaviors as coping mechanisms for stress (Wilkinson 1992; Lynch et al. 2000; Schnittker and McLeod 2005.). The neo-material interpretation gives greater attention to structural factors and highlights potential linkages between income inequality and underinvestment in human resources such as education and medical expenditures (Davey Smith 1996). While underinvestment in these areas may help to explain within-country differences, it is unlikely that they can account for the U.S.'s poor life expectancy ranking. Compared to other high-income countries, the U.S. population is very well educated and has among the highest per capita health expenditures in the world (NRC 2011). A systematic review of 98 studies concluded that

income inequality does not explain international differences in population health (Lynch et al. 2004).

Related explanations point to higher levels of poverty and weaker social safety nets in the U.S. relative to other high-income countries. For example, Canada experienced more rapid life expectancy gains than the U.S. during the 1980s despite similar or slightly worse macroeconomic conditions (in terms of GDP growth and unemployment rates). Dow and Rehkopf (2010) suggested that this may be related to stronger safety nets encompassing more generous unemployment benefits, sickness and maternity leave policies, and eligibility and benefits for means-tested cash assistance in Canada relative to the United States. Along with its weaker social safety nets, the U.S.'s labor and child support policies have also been suggested as important contextual factors that may contribute to its low life expectancy ranking (NRC 2011).

Social Capital and Interactions

There has been a recent resurgence of interest in whether differences in social capital, cohesion, interactions, and networks can explain cross-national differences in health (e.g., Lynch et al. 2000; Kennelly, O'Shea, and Garvey 2003; Mansyur et al. 2008). Two main avenues through which social networks are believed to influence health are the promotion of health-enhancing or risky health behaviors and the provision of financial, instrumental, or emotional resources and support (Berkman and Glass 2000). While the social capital and social cohesion explanation is usually presented as a corollary of the psychological consequences of income inequality (Kennelly, O'Shea, and Garvey 2003), the neo-materialist perspective suggests that differences in social capital may be more strongly

related to material conditions than psychosocial factors. For example, some studies have found that proposed indicators of social capital such as levels of trust, belonging to organizations, and doing unpaid work for such organizations are more strongly related to GDP per capita than to income inequality (Lynch et al. 2000).

Establishing the linkage between social interactions and health is further complicated by issues of reverse causality and endogeneity. In general, the current body of evidence does not support differences in social capital and networks as an explanation of cross-national health differentials. Both Kennelly, O'Shea, and Garvey (2003) and the NRC (2011) concluded that there is no evidence for a positive effect of social capital on population health. The findings from studies of cross-national differences in social networks are complex. In a comparison of the U.S. and England, Banks et al. (2011) found no evidence of weaker networks or less support in the U.S. than England among older adults; instead, social support and networks are similarly distributed in the two countries. They also found that the quality of social relationships differed between the countries, but not in a way that suggested a clear-cut U.S. advantage or disadvantage – for example, Americans appear to receive more positive *and* negative support from their children relative to their English counterparts (Ibid.).

Health Care System

The performance of the U.S. health care system is often blamed for its low life expectancy ranking (Nolte and McKee 2008; Muennig and Glied 2010). The main critiques of the U.S. health care system include lack of universal access, fragmented delivery of care, high costs of care, inefficiency, and a greater focus on specialist rather

than primary care. Evidence regarding the adequacy of primary care in the U.S. is mixed: Macinko, Starfield, and Shi (2003) find that the U.S. scores in the bottom of a group of 18 Organisation for Economic Co-operation and Development (OECD) countries for primary care, but the U.S. also appears to perform well in terms of pneumonia and influenza vaccination and cancer screening for the older population (Howard, Richardson, and Thorpe 2009; Preston and Ho 2011).

A nuanced view must be taken of what, precisely, lies within the jurisdiction of the health care system. For example, if personal health practices are unusually deleterious, a country could still exhibit poor measured health even if the health care system is performing exceptionally well in identifying and treating disease. Compared to other OECD countries, the U.S. health care system typically functions well in the identification and treatment of cancer and heart disease, the two leading causes of death at older ages (NRC 2011). This conclusion was based in part on an analysis of diseases for which effective methods of identification and treatment have been developed and where behavioral factors do not play a dominant role. Results from other studies (Ho and Preston 2010, Banks, Muriel, and Smith 2010) are consistent with these findings. For example, in their comparison of disease prevalence, incidence, and mortality between the U.S. and England, Banks, Muriel, and Smith (2010) conclude that their findings support higher quality medical treatment in the U.S. than in England. Relative to the English, Americans become sicker at earlier ages and spend more money treating their greater burden of disease, but this does, on average, have payoffs in reducing mortality. In addition, Wang et al. (2007) and Crimmins, Garcia, and Kim (2011) find that aggressive treatment and

identification has resulted in cholesterol and blood pressure being well-controlled in the U.S. relative to other countries.

Unlike the other high-income countries included in this analysis, the U.S. does not have universal health care. Roughly 16.7% of the U.S. resident population was estimated to lack insurance coverage in 2009 (De Navas-Walt, Proctor, and Smith 2010). It is possible that lack of access to timely and high quality health care is contributing to excess mortality in the U.S. relative to other countries. For example, the uninsured are more likely to forego care and less likely to receive preventive services. The salience of this factor varies with age – access alone is less likely to matter for adults over the age of 65 due to coverage under Medicare, but it may be more important at the younger ages, which are a key focus of this chapter.

Health Behaviors

It has been suggested that health behaviors such as smoking, diet, and lack of physical activity or sedentary lifestyle may account for the poorer health status of Americans. These behaviors are grounded in contextual influences including national policies regarding cigarette and gasoline taxation, availability and quality of public transportation systems, and food policies (NRC 2011). In general, studies agree that while a greater prevalence of some deleterious health behaviors in the U.S. is an important part of the explanation, they are not the whole explanation. Among the most important of these behaviors is smoking. During the 20th century, Americans had a history of heavier smoking relative to other developed countries. Preston, Glei, and Wilmoth (2011) found that smoking accounts for 41% and 78% of the gap in life expectancy at age 50 between

the U.S. and 9 high-income countries among males and females, respectively. The U.S. has among the highest prevalence of obesity among OECD countries (OECD Health Data 2012). While estimates of mortality attributable to obesity remain highly variable, studies have suggested that it may be a substantial contributor to the U.S. shortfall in life expectancy at age 50 (Preston and Stokes 2011). Evidence on whether cross-national differences in diet and physical activity can explain life expectancy differentials remains inconclusive. This is in large part related to measurement issues, reporting differences across countries, and a paucity of high quality, international data on physical activity (NRC 2011).

Contribution

Recent studies of the U.S. life expectancy disadvantage have concentrated largely on mortality at older ages. Examining differences at older ages makes sense given that in 2008, 92% of male newborns and 95% of female newborns in the U.S. could expect to survive to age 50 (Arias 2012). However, the importance of mortality differences at younger ages should not be dismissed in explaining life expectancy differences between the United States and other developed countries. The life course approach posits that reducing premature mortality at the younger ages may have important consequences for health and mortality at the older ages, and these effects may accumulate over time (Elo and Preston 1992; Kuh and Ben-Shlomo 1997; Hayward and Gorman 2004). It is likely that common factors are contributing to the U.S. life expectancy shortfall across all ages, and looking across a wider age range may help us identify these factors. In addition, Americans have the highest or second-highest mortality rates in every age group below

50 among a set of 17 high-income countries. In contrast, recent research has shown that the U.S. performs very well in terms of mortality at the older ages (e.g., at ages above 75) in cross-national comparisons (Ho and Preston 2010). Together, these observations suggest the need for an increased focus on mortality conditions at the younger ages. A rich body of literature has examined youth health and mortality, and particularly the burden of injury-related deaths, within the United States. For example, it is well-known that the U.S. experiences high infant mortality rates and high death rates from homicide and HIV. In this chapter, I draw on these studies to provide a comprehensive assessment of the relative importance of the main causes of death operating at younger ages in explaining cross-national mortality differences.

This study examines how U.S. mortality compares to that of a set of high-income peer countries in a very recent period, 2006-2008. First, I use decomposition methods to answer the question, "How much of the gap in life expectancy at birth between the United States and other developed countries is due to mortality differences below age 50?" Next, I use cause-deleted life table techniques to identify the major causes of death contributing to Americans' excess years of life lost below age 50. Finally, I examine two indicators of age-specific mortality performance for U.S. birth cohorts born between 1850 and 2004 relative to their counterparts in the comparison countries.

Data and Methods

Comparison Countries

In these analyses, I compare the U.S. to a set of 16 high-income comparison countries: Australia, Austria, Canada, Denmark, Finland, France, Germany, Italy, Japan, the

Netherlands, Norway, Portugal, Spain, Sweden, Switzerland, and the United Kingdom. This group of countries is a subset of OECD countries chosen for best comparability to the United States.[14] The main criteria for inclusion in the sample were that the countries had to have sufficient population size to ensure stability of estimates (above 4.5 million), achieved high levels of development for a long period of time, and maintained acceptable levels of data quality and availability. These countries transitioned from high to low mortality regimes in the same period as the U.S. and share more similar cause-of-death profiles compared to countries that transitioned more recently or are still in the process of transitioning. High-income countries that were excluded from the sample fail to fulfill one or more of these conditions. For example, Belgium has typically had data issues and its latest year of available data is 2005, which is outside the recent three-year span used in the first part of the analysis; Greece and Korea are not included in the Human Mortality Database; and several other countries are former Soviet countries whose mortality experience has been somewhat unique.

I. Age- and Cause-Specific Contributions to the U.S. Life Expectancy Shortfall, 2006-2008

Data

Three main data sources were used for this analysis: the Human Mortality Database (HMD), the World Health Organization (WHO) Mortality Database, and Statistics Canada. Country- and sex-specific life table all-cause death rates ($_nm_x$'s) for age groups 0-1, 1-4, 5-9, 10-14, …, and 45-49 were drawn from the HMD. Deaths from all causes

[14] The U.S.'s low life expectancy ranking is not a result of the selection of countries used in this study. Among all 34 OECD countries, life expectancy at birth in the United States was ranked 24th for males and 27th for females in 2005 (HMD 2012; OECD 2010).

and from the codes making up the cause of death categories of interest for the same age groups were obtained from the WHO Mortality Database to produce the proportion of deaths in each age group due to the causes of interest ($_nD_x^i/_nD_x$), where i indicates a specific cause of death category. Since the latest year of mortality data available from the WHO differed across countries, the latest year of data available between 2006 and 2008 was extracted for each country in order to cover the most recent period possible and to maximize coverage of comparison countries (see **Table A2.1** for the country-year pairs used in this analysis and their corresponding life expectancies). The results are not sensitive to the choice of year used. When the analysis was repeated using data from 2006 for each country, the results were nearly identical to those reported here.[15]

These data were then aggregated according to the major cause of death categories of interest (see **Table A2.2** for the list of categories and corresponding ICD-10 codes). These categories were specified per the Global Burden of Disease Study (GBD) classification (Mathers, Lopez, and Murray 2006) and the standard categories used in the U.S. National Vital Statistics Reports (NVSR) (e.g., Xu et al. 2010). The proportions of total deaths attributable to each cause category were taken from the WHO and applied to the HMD all-cause age-specific death rates to obtain age-specific death rates by cause, sex, and country.

Special Cases: Canada and Switzerland

For Canada, the latest year mortality data were available from the WHO Mortality Database was 2004. I used mortality data for 2007 from Statistics Canada. These data were aggregated using the same cause of death categorizations specified in **Table A2.2**

[15] Portugal was excluded from this robustness check since its 2006 cause of death data were not available.

and merged with the master data set. For Switzerland, cause of death data were coded using less detailed four-digit numeric codes denoting larger cause of death categories instead of the customary four-digit alphanumeric codes available for the other 16 countries. It was not possible to extract all codes for certain cause of death categories for Switzerland. In cases where it was possible to use identical cause of death codes, Switzerland was included in the computation of the mean of the comparison countries, excluding the United States. Thus, the U.S. was compared to a composite of comparison countries calculated as the average of either 15 or 16 countries. The results were not sensitive to the inclusion or exclusion of Switzerland.

Methods

Age Decomposition

Decomposition methods can be used to determine the contribution of mortality differences in specific age groups to differences in life expectancy at birth between two populations (Preston, Heuveline, and Guillot 2001). The following identity was used to estimate the contribution of mortality differences at ages below 50 and at ages above 50 to the difference in life expectancy at birth between the U.S. and each of the comparison countries, as well as between the U.S. and the composite of the other countries:

$$(1)\ e_0 = {}_{50}L_0 + p(50) * e_{50},$$

where the contribution of mortality differences above age 50 is the weighted difference between the two populations' life expectancy at age 50 (e_{50}), and the weight is the mean of the two countries' probability of survival to age 50 ($p(50)$) (Ho and Preston 2010).

This method produces results nearly identical to those obtained using Arriaga (1984)'s decomposition. The difference in life expectancy at birth between the U.S. and each of the individual comparison countries, as well as the difference in life expectancy at birth between the U.S. and the mean of the comparison countries, were decomposed using both methods. Three different ways of generating a composite life table for the mean of the comparison countries were used:

1. Taking the mean of the individual countries' l_x, ${}_nL_x$, and T_x columns,
2. Deriving a mean life table from the mean of the individual countries' l_x and ${}_na_x$ columns, and
3. Deriving a mean life table from the mean of the individual countries' ${}_nm_x$ and ${}_na_x$ columns.

When the decomposition methods were applied to each of these three composites, highly similar results are obtained. Weighting the ${}_nm_x$ values by population size would be inappropriate in these and the subsequent analyses since the country, not the individual, is considered to be the unit of analysis.

Cause-Deleted Life Tables

The remainder of this portion of the analysis focuses on ages below 50 because I find that mortality differences at these ages make substantial contributions to Americans' overall life expectancy shortfall relative to the comparison countries among both males and females and because cross-national mortality differences at these ages are relatively understudied. In addition, the main assumption used to generate the cause-deleted life

tables (Chiang's assumption) becomes much more tenuous at the older ages due to competing risks.

Cause-deleted life table methods were used to estimate what temporary life expectancy – the expected number of years lived between ages 0 and 50 out of a maximum possible 50 years – would be in the absence of a particular cause of death (Preston, Heuveline, and Guillot 2001). This approach uses Chiang's assumption, which assumes that the force of decrement function from cause i is proportional to the force of decrement function from all causes combined in each age interval.

The proportion of deaths due to a specific cause of death category in the age group x to $x+n$ was calculated as:

(2) ${}_nD_x^i/{}_nD_x$, where i=communicable and nutritional conditions, HIV, …, and homicide.

Country-, year-, age-, and sex-specific survival probability (${}_np_x$) and mean age at death (${}_na_x$) columns were drawn from the HMD. The cause-specific proportions from the WHO were used to generate the R^{-i} quantities:

(3) $R^{-i} = 1 - {}_nD_x^i/{}_nD_x.$

The following life table columns were then generated to produce cause-deleted life tables (life tables in the absence of a specific cause of death category i) for each country and separately by sex:

(4) ${}_n^*p_x^{-i} = ({}_np_x)^{R^{-i}}$

(5) ${}_n^*l_{x+n}^{-i} = {}^*\dot{l}_x^{-\iota} \cdot {}_n^*p_x^{-i}$

(6) ${}_n^*d_x^{-i} = {}^*l_x^{-i} \cdot (1 - {}_n^*p_x^{-i})$

$$(7a)\ {}^{*}_{n}a_x^{-i} = n + R^{-i} \cdot ({}_nq_x / {}^{*}_{n}q_x^{-i}) * ({}_na_x - n) \text{ for } x = 0, 1, 5, 45.$$

$$(7b)\ {}^{*}_{n}a_x^{-i} = \frac{-\frac{5}{24} \cdot {}^{*}_{5}d^{i}_{x-5} + 2.5 \cdot {}^{*}_{5}d^{i}_{x} + \frac{5}{24} \cdot {}^{*}_{5}d^{i}_{x+5}}{{}^{*}_{5}d^{i}_{x}} \text{ for } x = 10 \text{ to } 40.$$

$$(8)\ {}^{*}_{n}L_x^{-i} = n \cdot {}^{*}l_{x+n}^{-i} + {}^{*}_{n}a_x^{-i} \cdot {}^{*}_{n}\, d_x^{-i}.$$

Temporary life expectancy between ages 0 and 50 in the absence of a specific cause of death was obtained by summing the number of life-years that would have been lived between those ages in the absence of that particular cause (${}^{*}_{n}L_x^{-i}$'s). Years of life lost below age 50 due to each specific cause i were calculated as:

$$(9)\ \sum_{x=0}^{45} {}^{*}_{n}L_x^{-i} - \sum_{x=0}^{45} {}_nL_x.$$

These quantities were then used to calculate the percentage contributions of specific causes of death to the total difference in years of life lost below age 50 between the U.S. and the comparison countries. All analyses were performed for each country and separately for males and females.

II. Cohort Rankings and Ratios of Age-Specific Death Rates

In the second part of the analysis, I compare 5-year birth cohorts born between 1850 and 2004 in the United States to their mirror birth cohorts in the other 16 high-income countries. I assess the mortality performance of these birth cohorts observed during the period 1960-2005.

For each country, period age-specific death rates are drawn from the HMD and aligned to correspond to each 5-year birth cohort. For example, the death rates of the 1900-1904 birth cohort correspond to the period death rates for age group 55-59 in 1960, 60-64 in 1965, 65-69 in 1970, …, 95-99 in 2000, and 100-104 in 2005. The cohort death rate

series is constructed this way for all other birth cohorts. Due to issues of data availability and the fact that we have yet to observe the full mortality experience of the youngest birth cohorts, many of these age-specific profiles are necessarily incomplete. I use the maximum amount of data possible for each birth cohort ages 0-4 and 105-109. Cohorts born in the first half of the 20th century have the most complete series.

For each cohort, the age-specific death rates are ranked from 1 to 17, such that the country with the lowest mortality rate in each age group receives a rank of 1 and the country with the highest mortality rate in each age group receives a rank of 17. In addition, the ratio of the U.S. death rate to the average of the other 16 death rates is calculated for each birth cohort-age-specific death rate. The presentation of the ratios provides an alternate measure of the U.S.'s mortality performance that allows for a greater sense of the variation in the death rates.

Results

Ia. Age-Specific Contributions to the U.S. Life Expectancy Shortfall, 2006-2008

There is a tendency to overlook mortality at younger ages since by and large, these high-income countries have long completed their epidemiological transitions, and most deaths in these countries now occur at ages above 50. However, the decomposition results indicate that mortality differences below age 50 are crucial in explaining life expectancy differentials between the United States and other high-income countries.

On average, life expectancy at birth for American males is 2.2 years lower than the comparison countries. **Figure 2.1** shows the contribution of specific age groups to this 2.2-year difference. All of the U.S. life expectancy disadvantage for American males is

concentrated entirely below age 80. The contributions of ages 80 and above are all negative, indicating that in these age groups, American males experience lower death rates than males in the comparison countries. Differences in infant mortality account for 12% of the male life expectancy gap, and half of the gap is accounted for by mortality differences between ages 0 and 39. Mortality differences between ages 20 and 69 are particularly important in explaining the male life expectancy gap. Each of the age groups in this age range contribute between 6.3%-10.4% of the gap in life expectancy at birth.

Life expectancy at birth for American females is 2.3 years lower than the average of the comparison countries. The life expectancy disadvantage for females is concentrated entirely below age 85 (**Figure 2.2**). On average, mortality for American females at ages 85 and above is lower than in the comparison countries. Compared to males, we see that among females, mortality differences between ages 40 and 79 account for the bulk of the gap in life expectancy at birth. Differences in infant mortality account for 10% of the gap, and half of the gap is accounted for by mortality differences between ages 0 and 54.

These figures showed the contribution of specific age groups to the gap in life expectancy at birth between the United States and the average of the comparison countries. In this case, mortality differences below age 50 accounted for 67% and 41% of the U.S. shortfall in life expectancy for males and females, respectively. We are also interested in whether this holds if we compare the United States to each of these countries individually.

Figures 2.3 and 2.**4** show the relationship between the size of the gap in life expectancy at birth between the U.S. and each of the comparison countries and the percentage contribution of mortality differences below age 50. For example, life expectancy for

Swedish males is 3.3 years higher than that of American males, and differences in mortality below age 50 between the two countries accounted for 59% of this 3.3-year gap. Mortality below age 50 accounts for over half of the gap in life expectancy at birth between American males and males in 14 of the 16 comparison countries. For the remaining two countries, Australia and Canada, the contribution of mortality below age 50 is 41% and 49%, respectively. Thus, ages below 50 make a substantial contribution to the gap in male life expectancy at birth between the U.S. and other developed countries. These ages account for two-thirds of the gap on average, and their contribution is always in excess of two-fifths of the gap for all the comparison countries.

For females, the U.S. shortfall in life expectancy at birth is less concentrated at ages below age 50, with the contribution of mortality differences at these ages ranging between 22%-80%. However, mortality differences at these ages remain important contributors. Mortality differences below age 50 account for over a third of the life expectancy gap between American females and females in 11 of the 16 comparison countries. They are particularly important contributors to the difference in life expectancy at birth between American females and females in the United Kingdom, the Netherlands, and Germany, accounting for 80%, 62%, and 60% of these gaps, respectively.

One intuitive way of interpreting these results is that even if life expectancy at age 50 were equalized among all countries, on average 67% and 41% of the gap in life expectancy at birth between the U.S. and other countries would remain.[16] Thus, it is clear that, particularly for males but also for females, mortality at ages below 50 should not be overlooked in examining differences in life expectancy between the U.S. and other high-

[16] This assumes that changing mortality at ages above 50 would not affect mortality at ages below 50.

income countries.[17] It is also interesting to note that a negative relationship exists between the size of the gap in life expectancy at birth and the contribution of mortality differences below age 50. This correlation is -0.84 for males and -0.88 for females. In other words, the larger the difference in life expectancy at birth between the U.S. and any given country, the smaller the proportion of the gap that is attributable to ages below 50.

Ib. Cause of Death Contributions to the U.S. Excess in Years of Life Lost Below Age 50, 2006-2008

Given the importance of mortality differences below age 50 in explaining the gap in life expectancy at birth between the U.S. and other developed countries, I now focus on identifying the primary causes of death responsible for Americans' excess mortality in this age range. Males and females in the United States lose the most years of life below age 50 in this set of high-income countries (**Figures 2.5** and 2.**6**). American males lose 1.36 years below age 50 compared to the comparison country average of 0.77 years, while American females lose 0.80 years below age 50 compared to the comparison country average of 0.45 years. As expected, females lose fewer years of life before age 50 than males. Compared to the best performers, Swedish males and females, Americans are losing over twice as many years of life below age 50. Notably, the other English-speaking countries (Australia, Canada, and the United Kingdom) are clustered at the left side of these figures, indicating that they, along with the U.S., tend to lose more years of life at younger ages than the other comparison countries. This has two implications: first, factors common to social structures in these countries may be contributing to this

[17] This finding is robust to analyses that incorporate a larger set of 21 comparison countries (Belgium, Ireland, Iceland, Luxembourg, and New Zealand are the additional countries).

phenomenon. For example, the U.S., Canada, and Australia have been classified as liberal welfare states, which are characterized by weaker social safety nets and private provision of services compared to corporatist-statist or social democratic regimes (Esping-Anderson 1990). Second, studies comparing the United States and the United Kingdom are examining differences between the U.S. and one of its nearest, relatively low-performing neighbors. These studies often find large health differences (Banks, Muriel, and Smith 2010; Martinson, Teitler, and Reichman 2011), suggesting that comparisons between the U.S. and its higher-performing peers would be even more dramatic.

The cause-deleted life table results for males are shown in **Figures 2.7a** and 2.**7b**. Compared to the mean of the other countries, American males lose more years of life below age 50 from communicable diseases, and the differential is similar for HIV and all other communicable diseases. They also lose more years of life below age 50 from noncommunicable diseases. Looking at specific noncommunicable diseases, American males do well where cancer is concerned but worse for diabetes, cardiovascular diseases, respiratory diseases, digestive diseases, genitourinary diseases, and congenital anomalies at these ages. The largest differential between the U.S. and the mean of other countries exists for unintentional injuries, which is composed of transport and non-transport injuries. The U.S. performs poorly for both of those subcategories. Most transport injuries are motor vehicle accidents. Nontransport injuries consist of deaths from accidental poisoning and exposure to noxious substances (i.e., accidental drug overdose), falls, accidental firearm discharge, accidental drowning, and exposure to smoke, fire, and flames. **Table 2.2** shows the breakdown of nontransport deaths occurring among American males at all ages and below age 50. In 2007, 64% of nontransport injuries

below age 50 were due to accidental drug overdose. Another 20% of these deaths were due to falls, accidental firearm discharge, accidental drowning, and exposure to smoke, fire, and flames, with falls and accidental drowning being the largest contributors. American males also lose more years of life below age 50 from intentional injuries, which consist of suicide and homicide. The differential in homicide mortality is particularly large. Finally, American males also lose more years of life below age 50 from perinatal conditions and drug-related causes compared to males in the comparison countries.

Figures 2.8a and 2.**8b** present the contribution of various causes of death to years of life lost below age 50 for females. On average, American females also lose more years of life at these ages from communicable diseases and noncommunicable diseases than their counterparts in the comparison countries. This differential exists for cancer, diabetes, cardiovascular disease, endocrine disorders, digestive diseases, genitourinary diseases, musculoskeletal diseases, and congenital anomalies. Differences in years of life lost below age 50 between the U.S. and the mean of other countries for unintentional injuries are substantial and similar in magnitude for transport and nontransport injuries. Deaths from accidental drug overdose accounted for 72% of nontransport deaths among American females below age 50 in 2007 (**Table 2.2**). Another 15% of nontransport deaths at these ages were distributed roughly equally among falls, accidental drowning, and exposure to smoke, fire, and flames. American females also lose more years of life below age 50 from intentional injuries. This difference comes entirely from homicide since they lose fewer years of life from suicide. Finally, American females lose more

years of life before age 50 from drug-related causes, maternal conditions, and perinatal conditions.

These findings are best summarized in **Figures 2.9** and **2.10**, which show the contributions of mutually exclusive and exhaustive causes to the difference in years of life lost below age 50 between the U.S. and the mean of other countries (0.59 years for males and 0.35 years for females). Among males, unintentional injuries (transport and nontransport injuries, 34%), intentional injuries (homicide and suicide, 23%), noncommunicable diseases (18%), and perinatal conditions (13%) are the largest contributors to the difference in years of life lost below age 50 between the U.S. and the mean of the comparison countries. Together, these four categories account for 88% of American males' excess years of life lost below age 50. Unintentional injuries are the largest contributing category, with transport and nontransport injuries contributing 18% and 16%, respectively. Just under half of the noncommunicable disease contribution comes from cardiovascular disease. Communicable and nutritional conditions, including HIV, and suicide make relatively small contributions to the American males' excess in years of life lost below age 50.

Among females, unintentional injuries (30%), noncommunicable diseases (29%), and perinatal conditions (19%) are the main contributors to Americans' excess years of life lost below age 50. Along with homicide (7%), these causes of death account for 85% of the difference in excess years of life lost below age 50 between the U.S. and the comparison countries. Suicide is not included in this figure since American females actually perform better than their counterparts in other developed countries where suicide mortality is concerned. As was the case for males, transport and nontransport injuries

make substantial contributions (16% and 14%, respectively), accounting for just under a third of American females' excess years of life lost below age 50. Noncommunicable diseases contribute another third of American females' excess years of life lost below age 50, with just under a third of this contribution coming from cardiovascular disease. American females perform poorly in terms of most noncommunicable diseases, particularly respiratory diseases and digestive diseases.

In summary, transport injuries, nontransport injuries, and perinatal conditions are key contributors to U.S. excess years of life lost between ages 0 and 50 for both males and females. The contribution of cardiovascular disease is similar for males and females, at about a tenth. Differences between males and females are observed for homicide and noncommunicable diseases besides cardiovascular disease: homicide makes a larger contribution for males, while noncommunicable diseases matter relatively more for females.

While there are advantages to comparing the United States to the average of the comparison countries, it is also interesting to examine the contributions of the causes of death to the difference in years of life lost below age 50 between the U.S. and each of the individual comparison countries. **Tables A2.3** and **A2.4** show these comparisons for males and females, respectively. Overall, communicable diseases, suicide, and maternal conditions (among females) do not emerge as important contributors in any cases. In general, the contributions of cardiovascular disease and all noncommunicable diseases are similar to the mean scenario. One notable exception is the United Kingdom-U.S. comparison, where the total contribution of noncommunicable disease is smaller than in the mean case (3% vs. 18% among males and 15% vs. 29% among females). Among

males, suicide is more somewhat important than in the mean case for the differences between the U.S. and Italy, Portugal, and Spain, although its contribution never exceeds 15%. Homicide is an important contributor in all cases for males, with its contribution ranging between 15%-25%. Among females, the contributions are fairly similar to the mean case. The contributions of transport and nontransport injuries are also fairly similar to the mean, with the exceptions of Italy, where the contribution of transport injuries to the Italy-U.S. differences are roughly half the contribution of the mean-U.S. differences, and Finland, where the contribution of nontransport injuries to the Finland-U.S. differences are much smaller than in the mean-U.S. comparisons. The contributions of perinatal conditions in the individual comparisons are also similar to the mean scenario with the exception of the Canada-U.S. comparisons, where perinatal conditions make a much smaller contribution. Thus, while there is some heterogeneity when the individual country comparisons are considered, the main findings are robust: unintentional injuries, noncommunicable conditions, perinatal conditions, and homicide are the key contributors to the U.S. shortfall in years of life lost below age 50 in nearly all cases, while communicable diseases, suicide, and maternal conditions are not major contributors in any of the comparisons.

II. Cohort Rankings and Ratios of Age-Specific Death Rates

Figure 2.11 shows the ranking of U.S. age-specific mortality rates between ages 0 and 109 among the 17 countries. Until roughly age 75, American males and females perform poorly, ranking last or close to last in every age group. After age 75, the U.S. rankings improve dramatically until American males and females experience the second lowest

death rates at ages 95 and above. This pattern was first documented by Ho and Preston (2010), who considered four possible explanations for the unique pattern of U.S. mortality rankings at ages 40 and above. They concluded that the evidence most strongly supports the hypothesis that the U.S. health care system performs especially well for older patients. This pattern and the poor performance of the U.S. is not due to higher mortality experienced by disadvantaged minority groups. Non-Hispanic whites are arguably the most privileged racial/ethnic group in the United States, and when the rankings are repeated using the U.S. non-Hispanic white population, the picture remains the same. It is crucial to address the large and persistent racial and socioeconomic health inequalities that exist in the U.S. However, alone they are not sufficient to explain to explain the U.S.'s low life expectancy ranking.

Figure 2.12 shows the ratio of U.S. mortality rates to the average of the other 16 comparison countries by age. The pattern is roughly similar for males and females: the ratio is highest at the infant and young adult ages, peaking at just under 2 for males aged 20-24 and females aged 25-29. The male ratio lies above the female ratio until age 25-29; between ages 25 and 85 the female ratio lies above the male ratio. The ratios for both males and females decline between roughly ages 25 and 85, with a hump for females between ages 60-70 that disappears once smoking-attributable mortality is removed among all countries (Ho and Preston 2010). The ratios fall below one at age 80 for males and age 85 for females, and remain below one through the highest age group (105-109).

In this analysis, I extend this work by exploring rankings and ratios for birth cohorts whose mortality is observed in the period 1960-2005. Examining the mortality experience of birth cohorts is particularly instructive because it allows for the comparison of

American cohorts who have experienced similar exposures at the same ages over the life course to their counterparts in other high-income countries. **Figures 2.13** and 2.**14** show the rankings for U.S. birth cohorts of males and females, respectively, among the set of 17 countries. First, it is evident that the shape of these cohort patterns is very similar to that of the period figure shown in **Figure 2.11**. For these male and female birth cohorts, Americans perform exceptionally well at the oldest ages, exhibiting among the lowest mortality rates in this set of countries. Between roughly ages 30 and 74, however, nearly all cohorts rank in the bottom half of the 17 countries, with females doing somewhat worse than males. The most interesting divergence between the cohort and period rankings occurs at the younger ages, between ages 0 and 34. Cohorts born around the middle of the 20th century actually perform quite well in these younger age groups (**Figures 2.13** and **2.14**, panels B and C). In contrast, the patterns for cohorts born in the last quarter of the 20th century are very similar to the period pattern. They exhibit among the highest mortality rates in this set of high-income countries, as far as we are able to observe them. Overall, the rankings patterns are highly similar for males and females.

Figures 2.15 and **2.16** show the ratios of U.S. age-specific death rates to the average of the other comparison countries for the same birth cohorts. The improvement in U.S. mortality relative to the comparison countries is echoed in these figures when the ratio drops below 1 around age 65, earlier than observed in the period figure. For both male and female birth cohorts, Americans at the oldest ages experience mortality rates up to 20% lower than the average of the comparison countries. In the middle age range, this ratio decreases with age. Americans in these birth cohorts may experience mortality rates up to 40% higher than the average of the comparison countries in their thirties, but this

ratio gradually converges towards 1 with age. The patterns are noisier at the younger ages, but it is clear that all cohorts born between 1950 and 2004 experience elevated mortality rates relative to their counterparts in the comparison countries (**Figures 2.15** and **2.16**, panel C). The ratios are highest in these adolescent and young adult ages, where American males and females in these birth cohorts experience death rates up to 80%-90% higher than their peers in the comparison countries. While the pattern in the ratios is roughly similar for males and females, the ratios are generally smaller in magnitude for females than males. In addition, the pattern in the ratios for birth cohorts born in the first half of the 20th century (**Figure 2.16**, panel B) is more differentiated among the female birth cohorts, with two sets of peaks observed at ages 20-29 and ages 60-69. This is particularly noticeable for the 1940-1944 and adjacent birth cohorts, which were the heaviest-smoking birth cohorts of American women (Preston and Wang 2006).

Discussion

This study demonstrates that deaths occurring at younger ages – below age 50 – account for 67% and 41% of the gap in life expectancy at birth between American males and females, respectively, and a set of high-income comparison countries. The main causes of death responsible for the U.S.'s excess years of life lost below age 50 are unintentional injuries (transport and nontransport injuries), noncommunicable diseases, perinatal conditions, and homicide. While unintentional injuries and perinatal conditions make similar contributions among both males and females, homicide is more important for males and noncommunicable diseases are more important for females. American females in this age range also experience an advantage in terms of suicide mortality relative to the

comparison countries, while American males do not. Cohort patterns of rankings and ratios illustrating the mortality performance of American birth cohorts born between 1850 and 2004 relative to their counterparts in other high-income countries reveal that these cohorts consistently perform exceptionally well at the oldest ages and very poorly in the middle ages.

Infant Mortality

It is well-known that infant mortality rates in the U.S. exceed those in other developed countries. Previous observers have noted that variations in the definition of a live birth across countries may result in underestimates of infant mortality in other countries relative to the United States. Efforts to standardize the definition of live births and infant deaths across countries attenuate but do not eliminate the U.S. disadvantage in infant mortality (MacDorman and Mathews 2009; Joseph et al. 2012). MacDorman and Mathews (2009) concluded that the high percentage of preterm births (births that occur before 37 weeks of completed gestation) in the U.S. is a key contributor to its infant mortality disadvantage. They demonstrate that if the U.S. had the same distribution of births by gestational age as Sweden, its infant mortality rate would be 33% lower. Compared to European countries, the U.S. has very high rates of preterm births. In 2004, 12% of all births in the U.S. were preterm births, while this figure ranged from 6.3% (Sweden and France) to 11% (Austria) among the 12 comparison countries considered in a recent U.S. National Center for Health Statistics Data Brief (Ibid.).[18] Observers have noted that neonatal intensive care units in the U.S. perform very well in saving preterm

[18] Although preterm births have declined over time in the United States, the preterm birth rate remained at 12% in 2010 (Martin et al. 2012).

and low birth weight babies. An examination of gestational age-specific infant mortality rates shows that the U.S. performs very well relative to other high-income countries for infants born between 24 and 37 weeks of gestation (MacDorman and Mathews 2009). For term infants (born at 37 or more weeks of gestation), however, U.S. infant mortality rates exceeded those of other European countries (Ibid.). The causes of preterm births are less well understood than the causes of intrauterine growth restriction. So far, the most important causes of preterm births that have been identified include genitourinary tract infections, multiple births, and smoking (Kramer et al. 2010).

Homicide

Homicide accounts for roughly a fifth of excess U.S. years of life lost below age 50 for males and less than a tenth for females. Gun ownership is much higher in the United States than other countries, although precise estimates are difficult to come by. The U.S. owns roughly 35%-50% of civilian-owned guns worldwide, with a lower bound estimate of 83 firearms per 100 for the U.S. compared to 31 per 100 for Switzerland, the comparison country with the second-highest rate of gun ownership (Small Arms Survey 2007). Studies have documented that gun ownership and availability are associated with greater risk of violent death among both children and adults within the United States. (Kellermann et al. 1993; Cummings et al. 1997; Miller, Azrael, and Hemenway 2002). The U.S. is clearly an outlier in the percentage of its homicides that are due to firearm discharge. In 2007, about 73% of homicides occurring below age 50 in the U.S. were homicides by firearm. Among the comparison countries, the average was 25%, ranging from 1.5% in Japan to 52% in Italy. Homicide is the cause of death that has been most

strongly linked to income inequality and social disorganization (Lynch et al. 2004; Kennedy, Kawachi, and Prothrow-Stith 1996). Within the United States, homicide has also been linked to poverty, structural disadvantage, residential segregation, and community levels of social capital and trust (Massey 1995; Messner, Baumer, and Rosenfeld 2004).

Unintentional Injuries

One of the most striking findings from this study is the sizeable contribution of unintentional injuries, which make the largest single contribution of any cause of death category. Unintentional injuries account for nearly a third of U.S. excess years of life lost below age 50 among both males and females. Transport (mainly motor vehicle accidents) and nontransport (mainly accidental drug overdose) injuries make roughly equal contributions. A recent report by the Transportation Research Board (2010) found that the fatality rate per 100 million vehicle kilometers traveled is highly similar for the United States and a set of 15 comparison countries. In fact, until 2004, the fatality rate per 100 million vehicle kilometers traveled was lower for the U.S. than for the composite. The annual number of vehicle kilometers driven in the U.S. far exceeds that in the comparison countries, with the difference widening over time and reaching roughly 1.4 trillion kilometers or over 870 billion miles driven in 2007 (Ibid.). The total population of these 15 comparison countries exceeds that of the U.S.; thus, the difference in annual kilometers driven per capita would be even more dramatic. The excess mortality from motor vehicle accidents in the U.S. is therefore attributable to a greater amount of driving rather than a higher fatality rate per mile driven.

Accidental drug overdose has increased dramatically since the early 1990s and particularly in the 2000s in the United States (Okie 2010). Accidental drug overdoses were responsible for 91% of unintentional poisoning deaths in the U.S. in 2009 (CDC 2012). Prescription painkillers (e.g., methadone, hydrocodone, and oxycodone) were the most commonly implicated in overdose deaths, followed by heroin and cocaine (CDC 2011). After opioid analgesics, benzodiazepines, antidepressants, and antiepileptic and antiparkinsonism drugs were the most commonly involved in pharmaceutical overdose deaths (Jones, Mack, and Paulozzi 2011).[19] Nonmedical use of prescription drugs (use of drugs without a prescription, in ways other than prescribed, or solely for inducing an experience or feeling) is a significant contributor to these deaths. Trends in accidental drug overdose mirror increases in prescription drug use in the United States. Between 1990 and 2008, spending on prescription drugs within the U.S. increased nearly sixfold, from $40.3 billion to $234.1 billion (Kaiser Family Foundation 2010). Although age-specific death rates are highest between the ages of 20 and 60 (CDC and NCHS 2012), prescribing – particularly the use of stimulants, antidepressants, and antipsychotics – has also increased among children over the past decade (Horwitz 2010).

The high burden of deaths from accidental drug overdose in the U.S. relative to the comparison countries is consistent with international estimates of drug use. Data from the WHO World Mental Health Surveys conducted between 2001-2005 suggest that the U.S. is an outlier in terms of lifetime incidence of drug use (Degenhardt et al. 2008). Compared to 7 other high-income countries, the U.S. had the highest levels of cocaine

[19] Three of these – benzodiazepines, antidepressants, and antiepileptic and antiparkinsonism drugs – are commonly prescribed for mental health conditions. Studies have documented that those with mental health disorders are at greater risk of heavy therapeutic use, nonmedical use, and overdose from prescription opioids (Becker et al. 2008).

use (16.2% versus 0.3%-4.1% among the comparison countries), despite its severe regulatory environment.[20] Addressing drug use at young ages is important given that the median ages of drug use, abuse, and dependence all occur prior to age 20 and given its potential to negatively impact later life outcomes (Swendsen et al. 2008).

Mental Health

One observation about these main contributors to excess mortality below age 50 is their potential relationships with mental health. Similar to their poor performance on other indicators in this age range, Americans had the highest prevalence of having a mental disorder in the past year among 8 high-income countries according to surveys fielded between 2001-2003 (WHO World Mental Health Survey Consortium 2004). This finding held for anxiety, mood, impulse-control, and substance disorders (Ibid.). While these estimates are based on diagnostic interviews (rather than asking respondents whether they have ever been diagnosed with these conditions), controversy remains over the reliability of international rankings due to cross-national differences in the reporting of symptoms. Studies have found that a number of chronic physical conditions, including diabetes, hypertension, and heart disease, are associated with affective disorders (Scott et al. 2007). In addition, findings from longitudinal studies indicate that several mental disorders are associated with an increased risk of later substance use (Swendsen et al. 2010). These findings highlight the importance of considering a wide range of health outcomes and linkages among them.

[20] These seven countries are: Belgium, France, Germany, Italy, Japan, the Netherlands, and Spain.

Cohort Patterns

The cohort patterns of international mortality rankings are generally consistent with the pattern observed in the most recent period, 2006-2008. Americans in these birth cohorts perform poorly in the middle ages but exceptionally well at the older ages, with the shift occurring between ages 60 and 75. In 2006-2008, the U.S. had among the highest death rates in the set of comparison countries at below age 30. In contrast, birth cohorts from the middle of the 20th century performed quite well at these ages, exhibiting a U-shaped pattern of rankings rather than the elongated S-shaped pattern of period rankings. While further work remains to be done to explain these trends, it is possible that the positive performance of these birth cohorts may be related to more favorable conditions in the United States relative to conditions in Europe during the first and second World Wars. Americans born in the latter half of the 20th century perform particularly poorly at the younger ages in terms of both low rankings (often experiencing the highest or second-highest death rates in this set of countries) and high mortality ratios (reaching a maximum of 1.9 for both males and females). Among females, no birth cohorts since the 1965-1969 birth cohort have been ranked in the top half of the comparison countries in the ages at which we are able to observe them. Among males, the picture is highly similar, with the exception of the 5-9 age group. All cohorts of American males born since 1975-1979 have been ranked in the bottom half of the comparison countries. These trends are consistent with Heuveline (2002)'s assessment of the U.S. as an outlier among industrialized nations in terms of adolescent and young adult mortality. It also supports the conclusion drawn from the first set of analyses presented in this paper that greater attention should be paid to mortality conditions at younger ages in the United States.

Limitations

There are some important limitations to this analysis. First, health is a multidimensional construct and covers many domains of life, including the physical, affective, cognitive, social, and functional. Mortality measures alone do not capture the full range of health. However, linkages between mental and physical health exist, and it is likely that conditions contributing to high levels of excess mortality are also contributing to greater morbidity in the U.S. population.

Additionally, this study necessarily relies on the comparability of mortality data across countries. The set of countries under analysis are all high-income countries with well-developed, complete vital registration systems (Mathers et al. 2005). The World Health Organization has made international recommendations regarding the coding of the underlying cause of death, which have helped standardize death certification across countries (Désesquelles et al. 2010). While some variation in coding practices remains, this is mitigated through the use of broader but still informative cause of death categories. It is not expected that minor variations in coding practices would change the results substantially.[21] In addition, restricting the analysis to ages below 50 means that much of the difficulty associated with ascertaining cause of death among the elderly is avoided.

In the second part of the analysis, which evaluates how U.S. mortality rates compare to those of the comparison countries between ages 0 and 109, age misreporting may be an issue. Preston, Elo, and Stewart (1999) found that for three typical patterns of age misreporting, age misstatement results in downwards-biased mortality estimates at the

[21] For example, it seems unlikely that a death from firearms would be classified under communicable and nutritional conditions or noncommunicable diseases rather than as a homicide. Deaths from drug-related causes are expected to fall under nontransport injuries (as an accidental poisoning) or noncommunicable diseases, both of which are important contributors.

oldest ages. If age misreporting is more severe in the United States than in the comparison countries, it is possible that the U.S.'s superior performance in rankings and ratios observed at the oldest ages may be artifactual.

Conclusion

We have arrived at a picture of how mortality in the United States compares to other high income countries in a recent period. American males and females have the lowest and second lowest life expectancy at birth, respectively, among a set of 17 high-income countries. Among males, the majority of the U.S. shortfall in life expectancy at birth is attributable to mortality differences below age 50, and these ages also account for a substantial proportion of the female disadvantage. The major causes of death contributing to excess years of life lost below age 50 in the U.S. relative to the comparison countries are: unintentional injuries (particularly accidental drug overdose and motor vehicle fatalities), noncommunicable diseases, perinatal conditions, and homicide. Diverse factors including increased prescribing, commuting patterns, public transportation systems, widespread automobile ownership, lower population density, health behaviors, access to health insurance, access to firearms, and residential segregation may be contributing to the U.S. mortality disadvantage at these ages.

Identifying which ages and causes of death are most responsible for the U.S.'s low life expectancy ranking is essential for developing an informed analysis to adjudicate among the proposed explanations. Many of these explanations, at their core, are concerned with distinctive features of American society. It is likely that a comprehensive explanation lies at the intersection of a broad and diverse set of social forces. That the U.S. appears to

perform poorly across a diverse set of diseases and conditions suggests that attention should be paid to social and contextual factors that shape day-to-day life in the United States and have the potential to affect outcomes across many domains.

Tables

Table 2.1. Life Expectancy at Birth, Difference with United States, and Ranking for Males and Females in 17 High-Income Countries, 2007

	Males			Females		
Country	e_0	Difference with U.S.	Rank	e_0	Difference with U.S.	Rank
Switzerland	79.32	3.68	1	84.09	3.31	3
Australia	79.27	3.63	2	83.78	3.00	6
Japan	79.20	3.56	3	85.98	5.20	1
Sweden	78.96	3.32	4	82.95	2.17	7
Italy	78.82	3.18	5	84.09	3.31	3
Canada	78.35	2.71	6	82.95	2.17	7
Norway	78.25	2.61	7	82.68	1.90	11
Netherlands	78.01	2.37	8	82.31	1.53	13
Spain	77.62	1.98	9	84.03	3.25	5
United Kingdom	77.43	1.79	10	81.68	0.90	15
France	77.41	1.77	11	84.43	3.65	2
Austria	77.33	1.69	12	82.86	2.08	9
Germany	77.11	1.47	13	82.44	1.66	12
Denmark	76.13	0.49	14	80.53	-0.25	17
Portugal	75.87	0.23	15	82.19	1.41	14
Finland	75.86	0.22	16	82.86	2.08	9
United States	75.64		17	80.78		16
Non-U.S. Average	77.81	2.17		83.12	2.34	

Source: Human Mortality Database (2012).

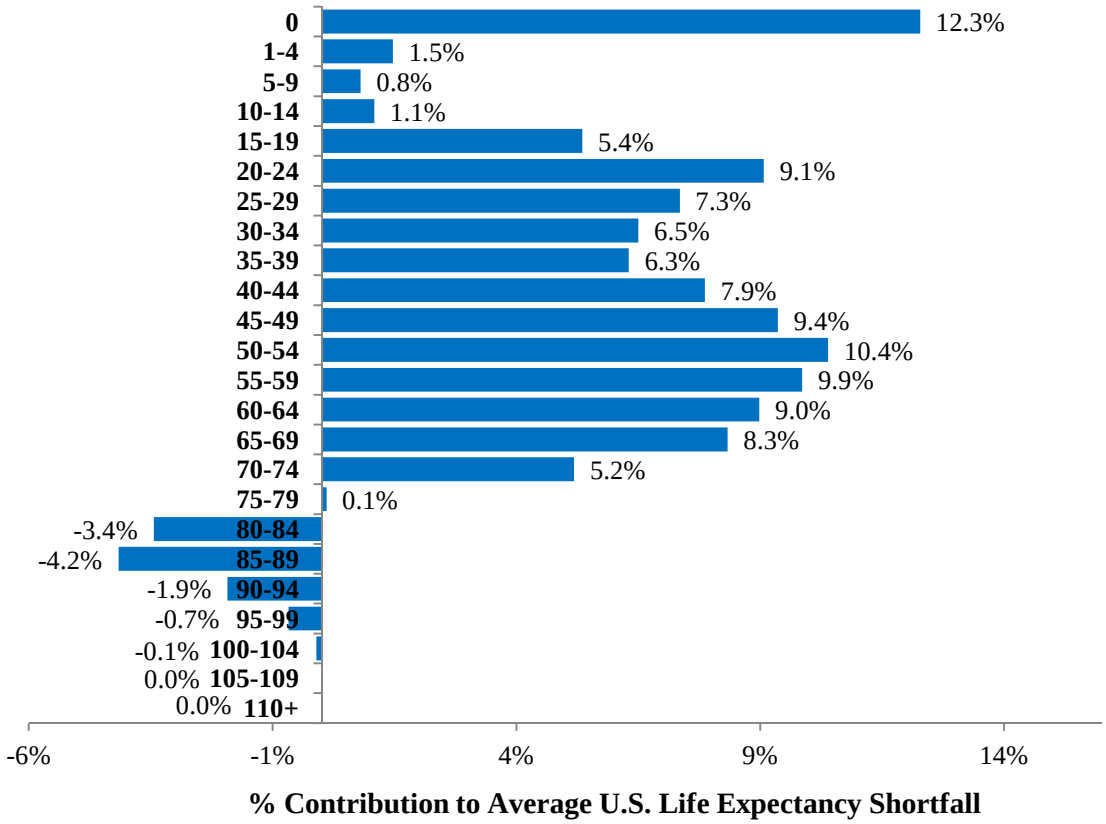
Table 2.2. Nontransport Injuries by Type (%), Males and Females, United States, 2007

	Males		Females	
	All Ages	Below Age 50	All Ages	Below Age 50
Accidental drowning	5.8	8.3	2.5	5.2
Accidental firearm discharge	1.2	1.7	0.2	0.6
Accidental poisoning and exposure to noxious substances	42.3	63.8	33.5	72.0
Exposure to smoke, fire, and flames	4.2	3.8	4.4	5.6
Falls	25.0	6.6	36.2	3.8
Residual	21.5	15.8	23.1	12.8

Source: Author's calculations based on World Health Organization (WHO) Mortality Database data.

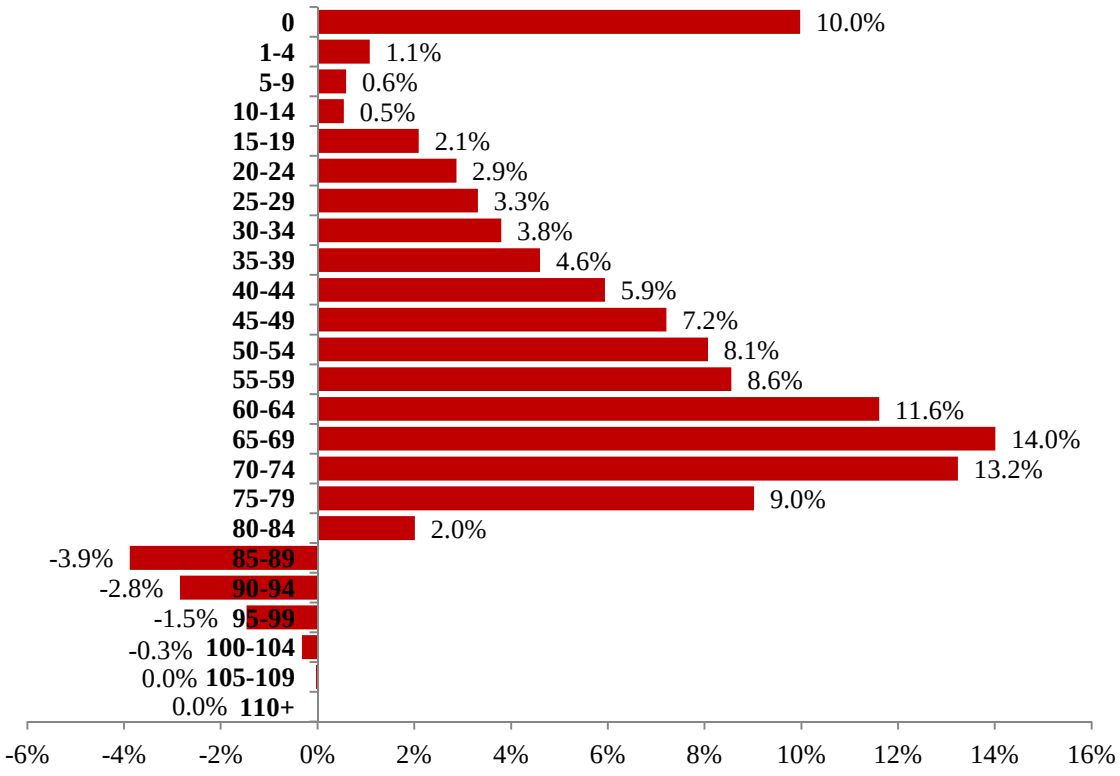
Figures

Figure 2.1. Contribution of Mortality Differences by Age (%) to the Gap in Life Expectancy at Birth Between the United States and the Average of 16 OECD Countries, Males, 2007



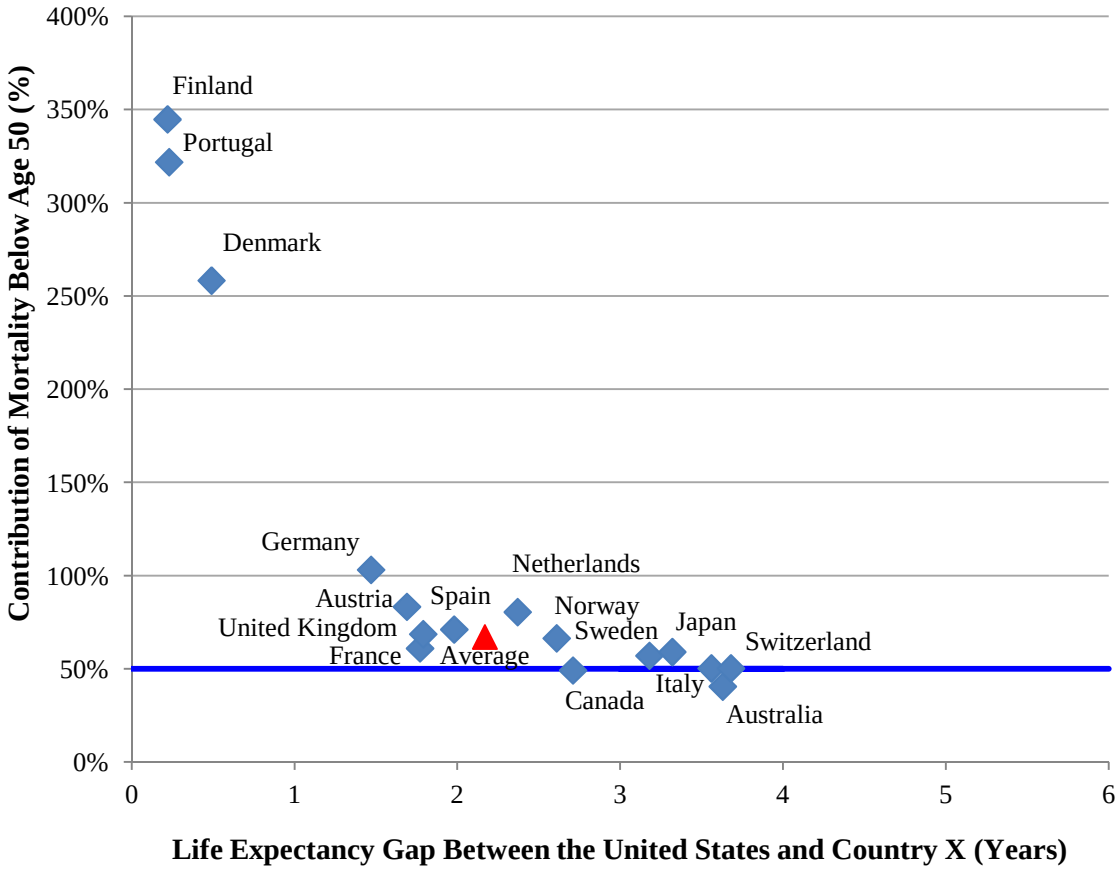
Source: Author's analysis based on data from the Human Mortality Database (2012).

Figure 2.2. Contribution of Mortality Differences by Age (%) to the Gap in Life Expectancy at Birth Between the United States and the Average of 16 OECD Countries, Females, 2007



Source: Author's analysis based on data from the Human Mortality Database (2012).

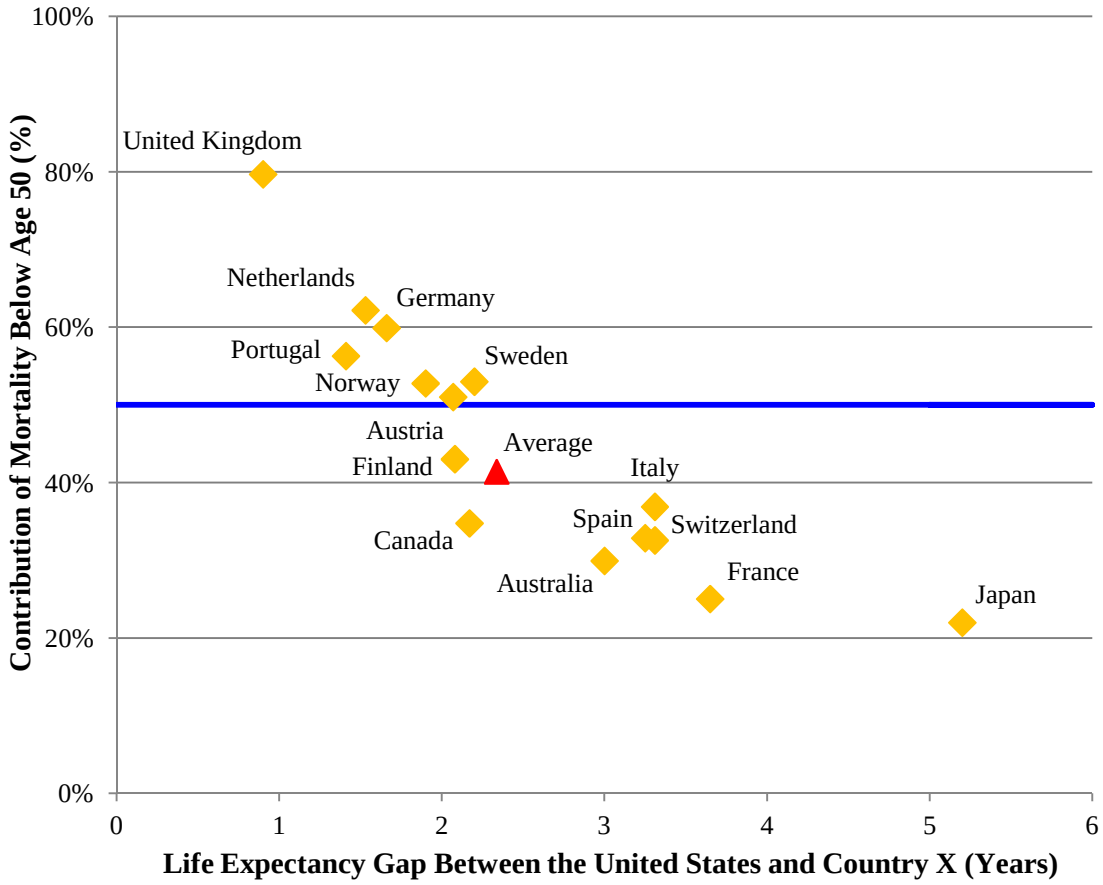
Figure 2.3. Contribution of Mortality Differences Below Age 50 (%) to the Gap in Life Expectancy at Birth Between the United States and 16 OECD Countries, Males, 2007[22]



Source: Author's analysis based on data from the Human Mortality Database (2012).

[22] The red triangle indicates the average of the 16 comparison countries. If a country lies above the blue 50% line, over half of its advantage in life expectancy at birth relative to the U.S. is attributable to mortality differences below age 50. Percentages in excess of 100% result from cases where life expectancy at age 50 is higher in the U.S. than in the comparison country. For example, if the U.S. experienced Finnish mortality rates above age 50 (i.e., if only differences in mortality below age 50 remained), the gap in life expectancy at birth between the U.S. and Finland would grow to 345% of its current value.

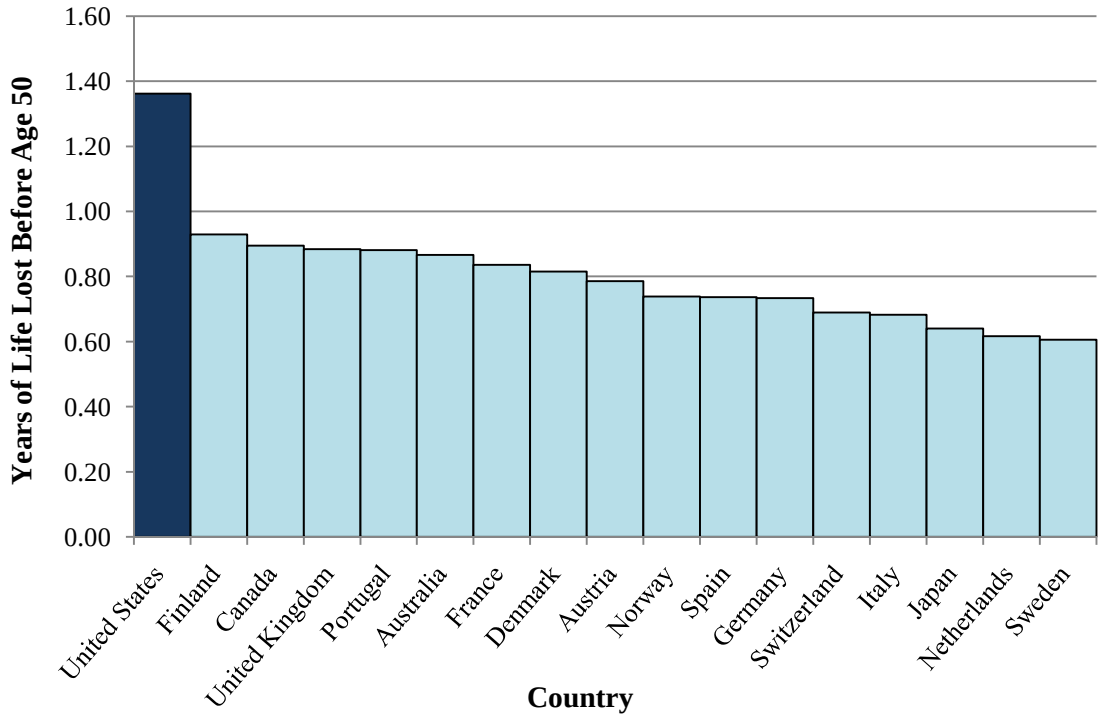
Figure 2.4. Contribution of Mortality Differences Below Age 50 (%) to the Gap in Life Expectancy at Birth Between the United States and 15 OECD Countries, Females, 2007[23]



Source: Author's analysis based on data from the Human Mortality Database (2012).

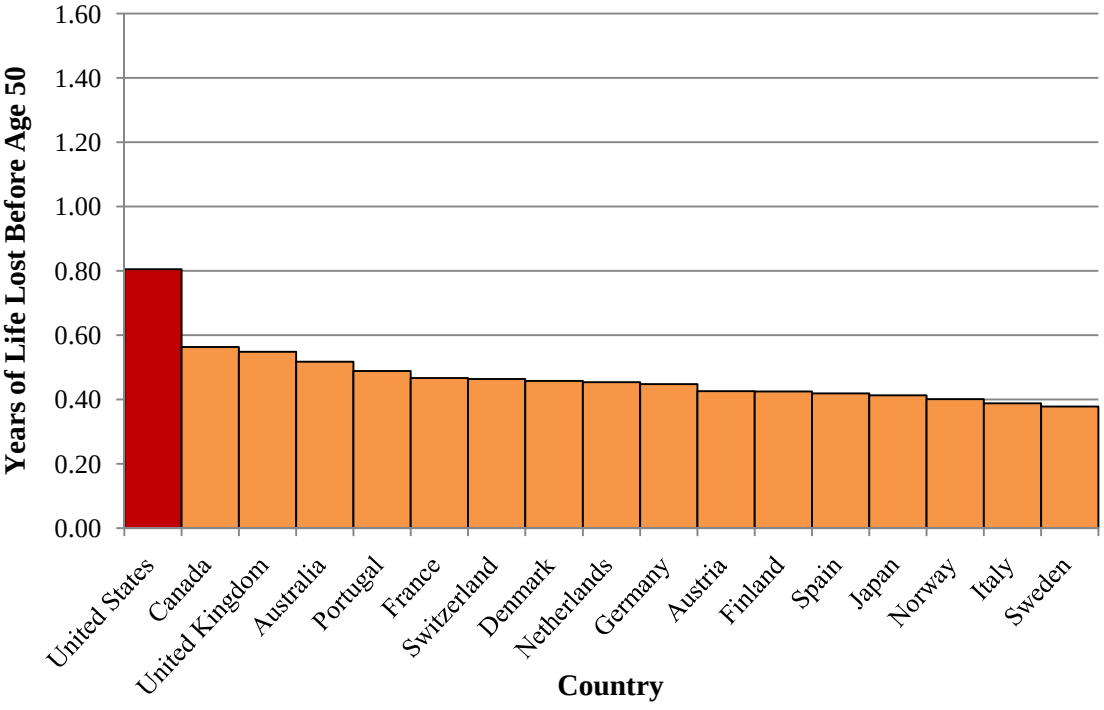
[23] The red triangle indicates the average of the 16 comparison countries. If a country lies above the blue 50% line, over half of its advantage in life expectancy at birth relative to the U.S. is attributable to mortality differences below age 50. Denmark is not shown here since the life expectancy of American females exceeds that of Danish females.

Figure 2.5. Years of Life Lost Before Age 50, Males, 17 OECD Countries, 2006-2008



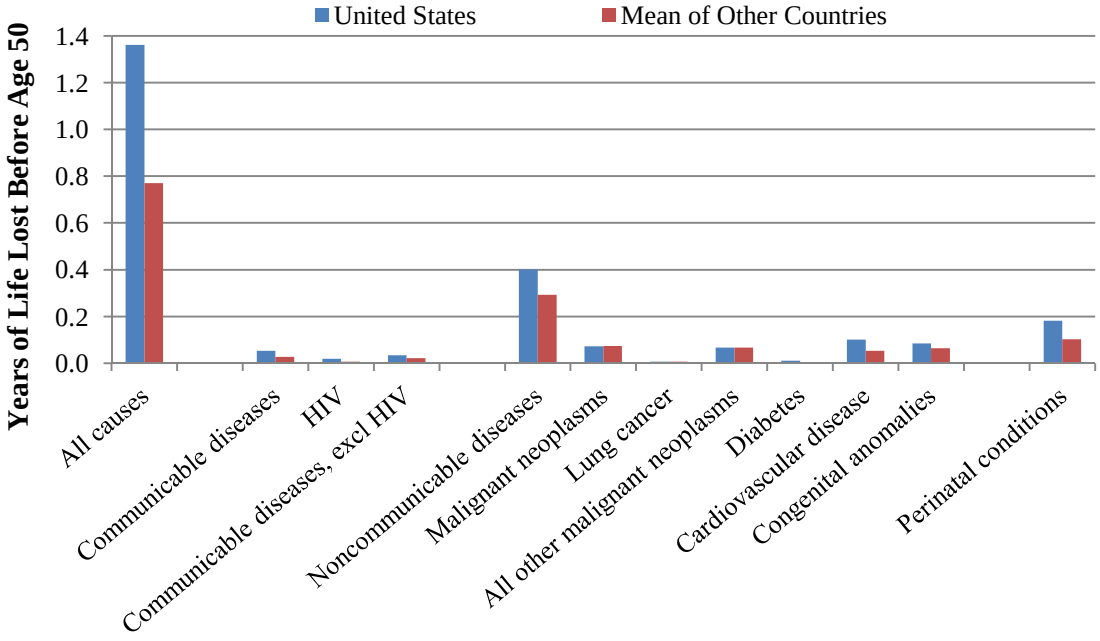
Source: Author's analysis based on data from the Human Mortality Database (2012).

Figure 2.6. Years of Life Lost Before Age 50, Females, 17 OECD Countries, 2006-2008



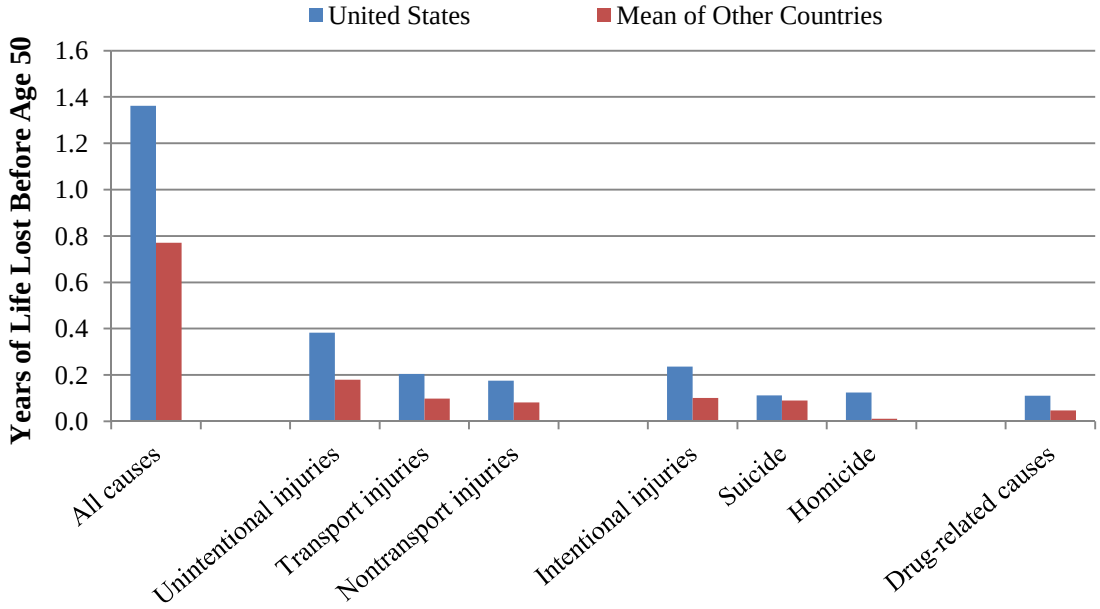
Source: Author's analysis based on data from the Human Mortality Database (2012).

Figure 2.7a. Years of Life Lost due to Specific Causes of Death, Males, 2006-2008[24]



Source: Author's analysis based on data from the HMD (2012) and the WHO (2011).

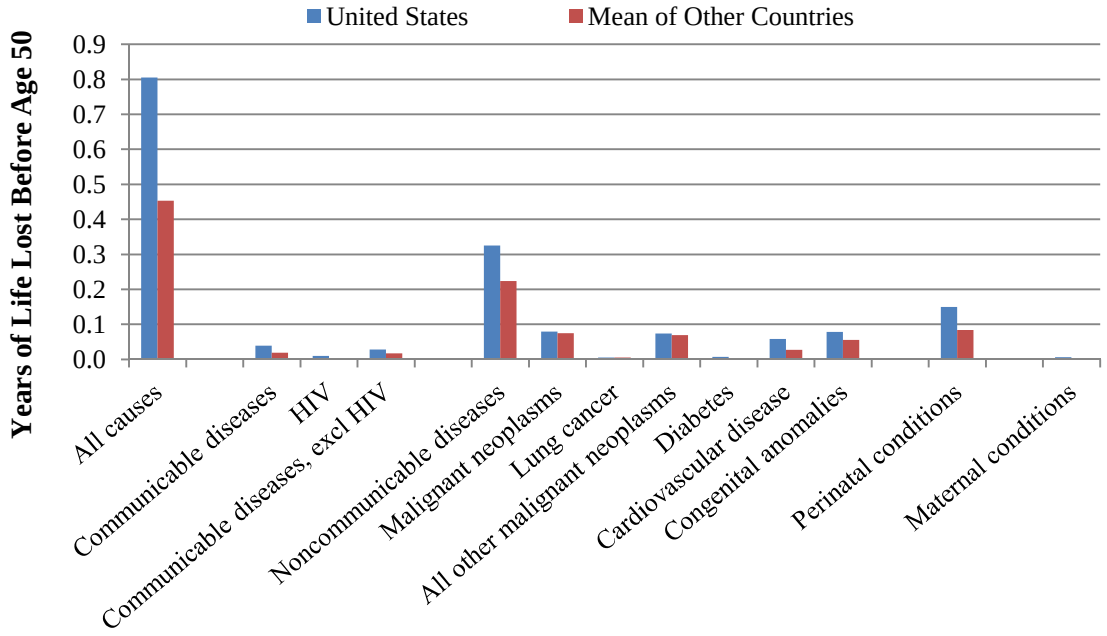
Figure 2.7b. Years of Life Lost due to Specific Causes of Death, Males, 2006-2008



Source: Author's analysis based on data from the HMD (2012) and the WHO (2011).

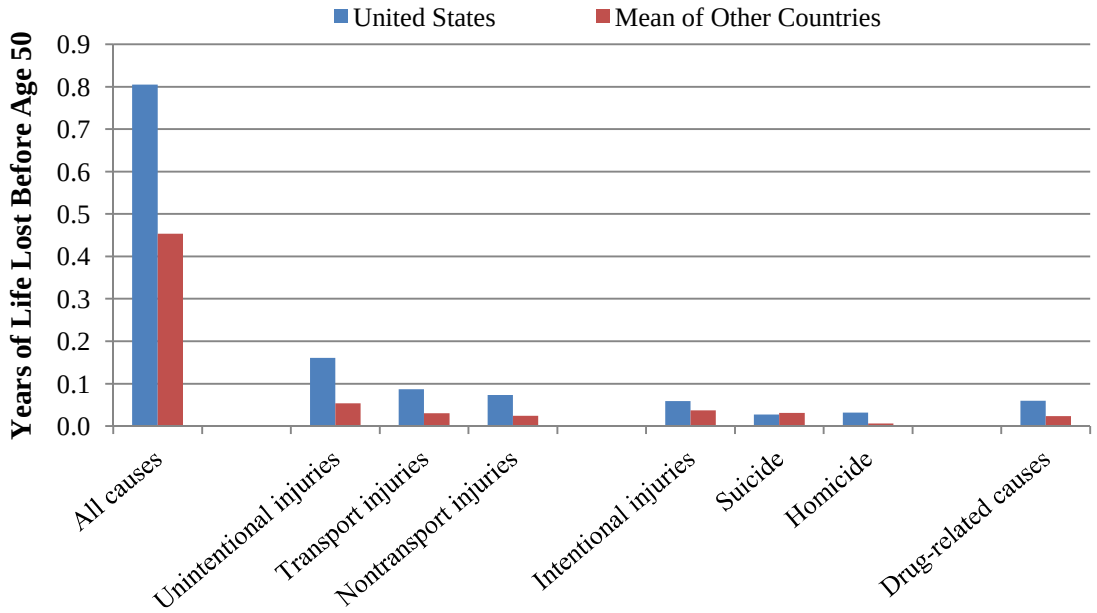
[24] The following causes of death categories are mutually exclusive: communicable diseases, noncommunicable diseases, perinatal conditions, unintentional injuries, and intentional injuries. Drug-related causes overlap with some of these categories, notably noncommunicable diseases and unintentional injuries (see **Table A2.2** for further detail).

Figure 2.8a. Years of Life Lost due to Specific Causes of Death, Females, 2006-2008[25]



Source: Author's analysis based on data from the HMD (2012) and the WHO (2011).

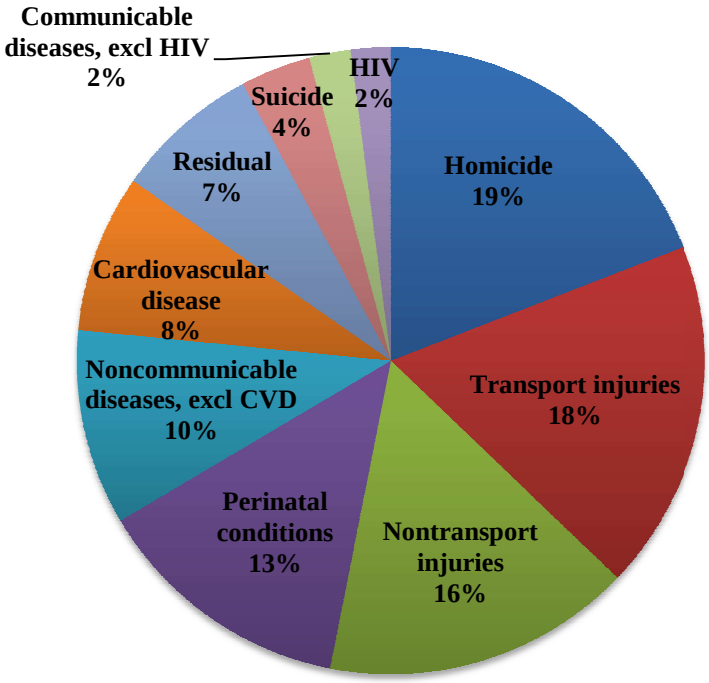
Figure 2.8b. Years of Life Lost due to Specific Causes of Death, Females, 2006-2008



Source: Author's analysis based on data from the HMD (2012) and the WHO (2011).

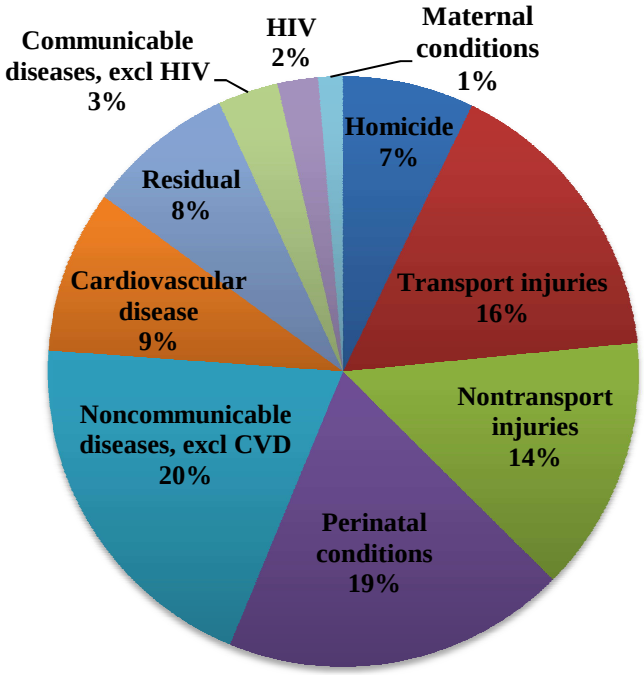
[25] The following causes of death categories are mutually exclusive: communicable diseases, noncommunicable diseases, perinatal conditions, unintentional injuries, and intentional injuries. Drug-related causes overlap with some of these categories, notably noncommunicable diseases and unintentional injuries (see **Table A2.2** for further detail).

Figure 2.9. Contribution of Causes of Death to Difference in Years of Life Lost Below Age 50 Between the U.S. and the Mean of Other Countries, Males, 2006-2008



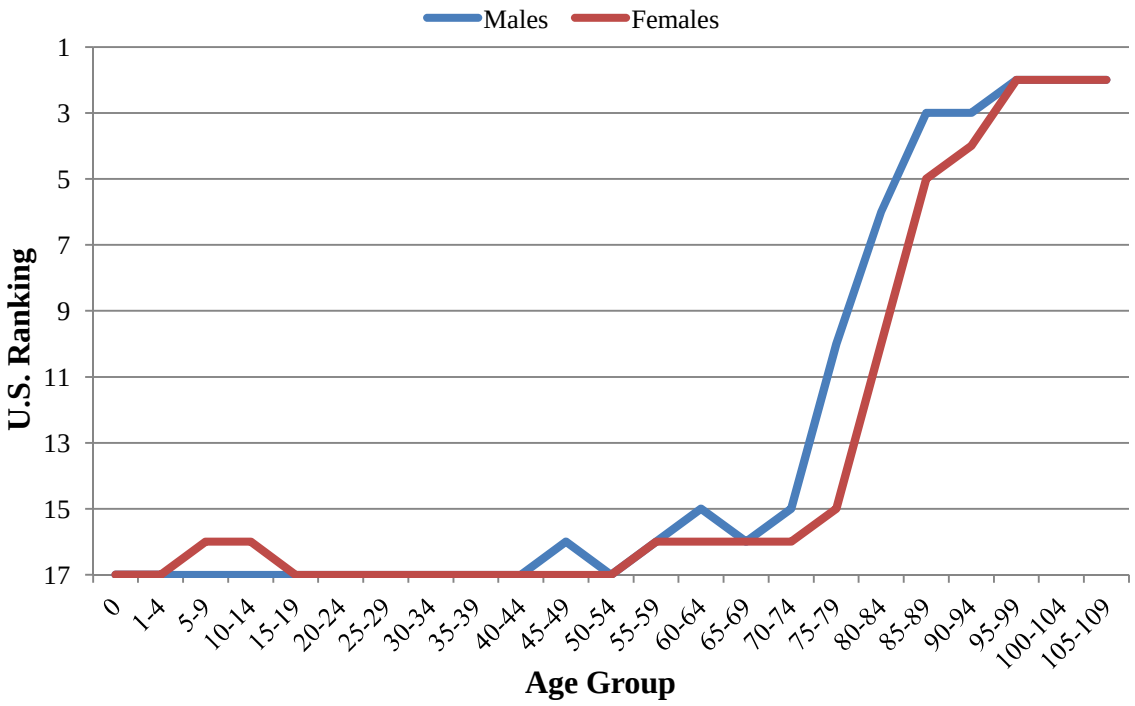
Source: Author's analysis based on data from the HMD (2012) and the WHO (2011).

Figure 2.10. Contribution of Causes of Death to Difference in Years of Life Lost Below Age 50 Between the U.S. and the Mean of Other Countries, Females, 2006-2008



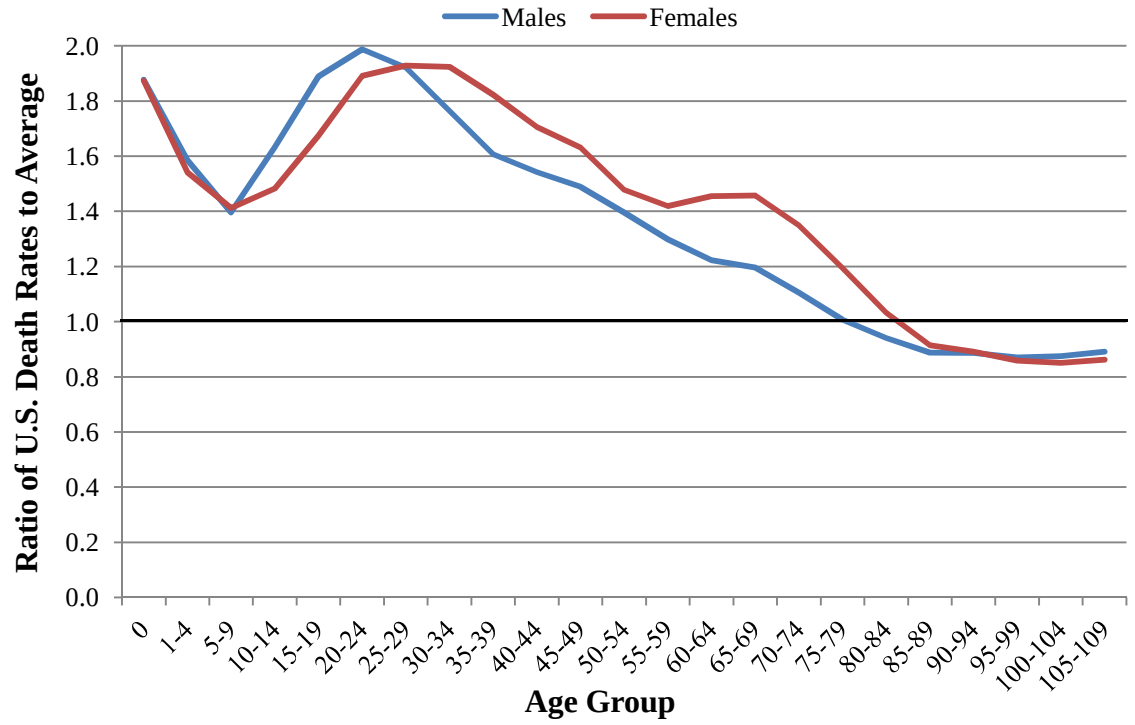
Source: Author's analysis based on data from the HMD (2012) and the WHO (2011).

Figure 2.11. Ranking of U.S. Age-Specific Death Rates Among 17 Countries, 2006-2008



Source: Author's analysis based on data from the Human Mortality Database (2012).

Figure 2.12. Ratio of U.S. Age-Specific Death Rates to Average of 16 Countries, 2006-2008



Source: Author's analysis based on data from the Human Mortality Database (2012).

Figure 2.13. Cohort Rankings of U.S. Age-Specific Mortality Rates, Male Birth Cohorts Born Between 1850 and 2004

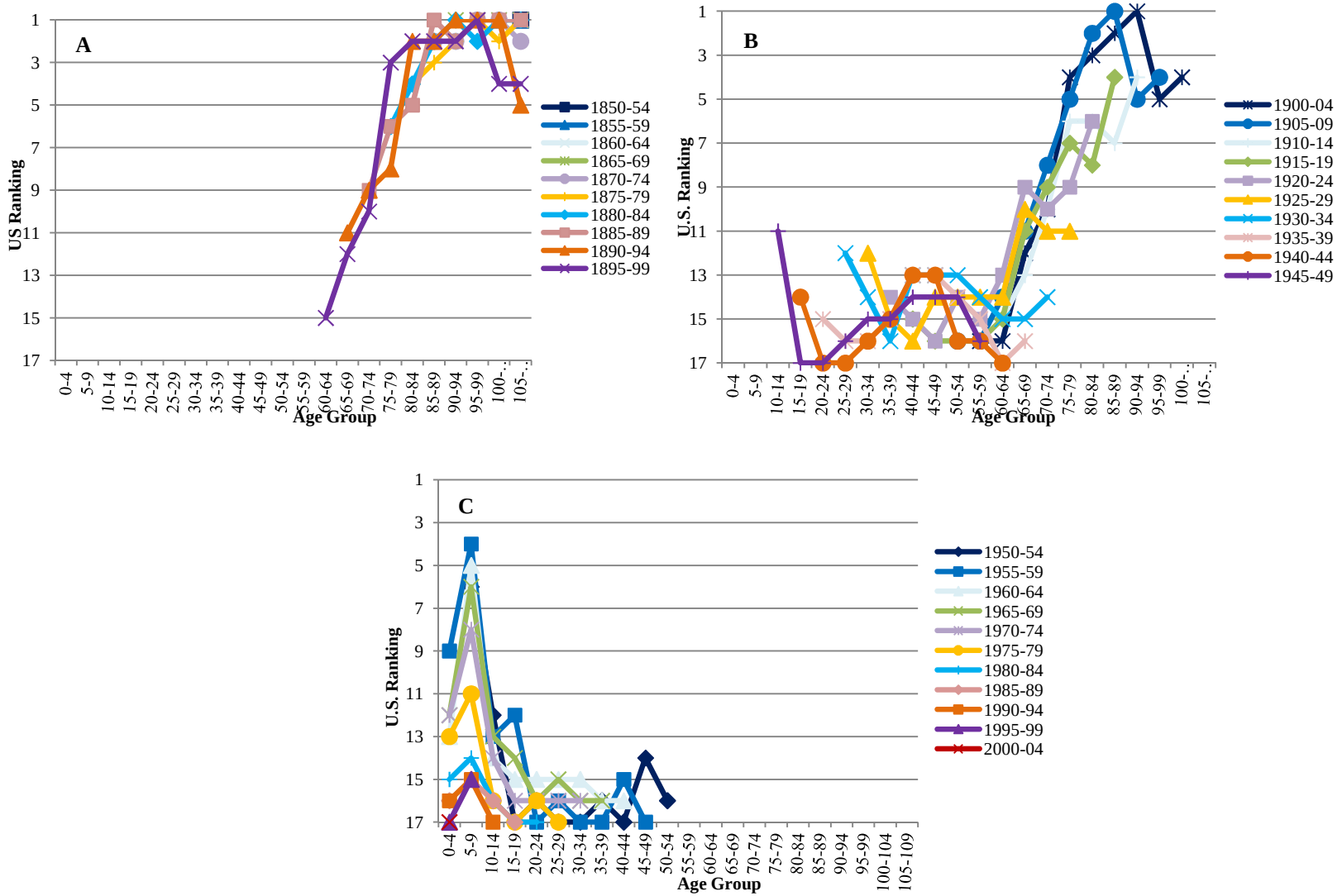


Figure 2.14. Cohort Rankings of U.S. Age-Specific Mortality Rates, Female Birth Cohorts Born Between 1850 and 2004

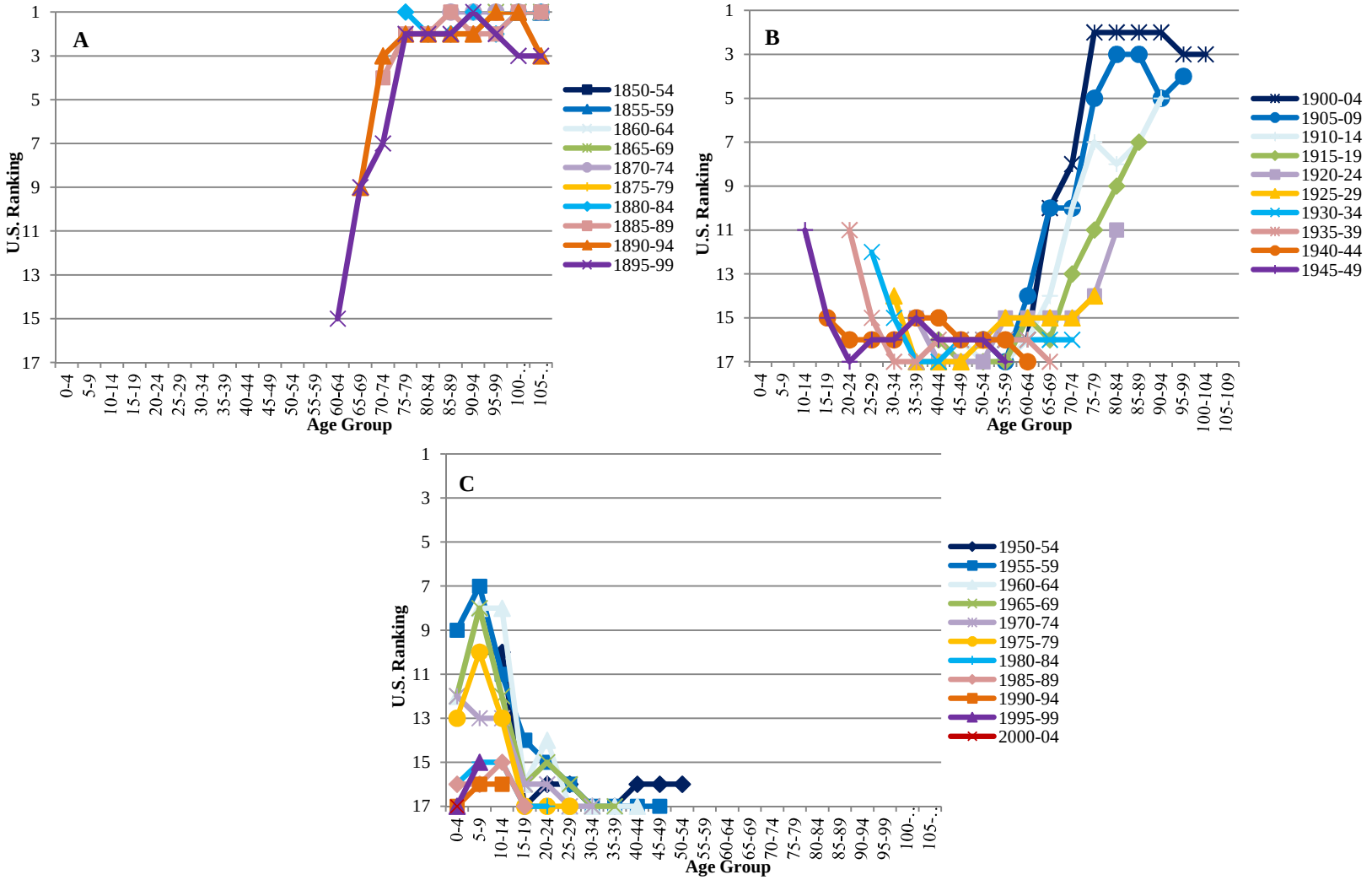


Figure 2.15. Cohort Ratios of U.S. Age-Specific Mortality Rates, Male Birth Cohorts Born Between 1850 and 2004

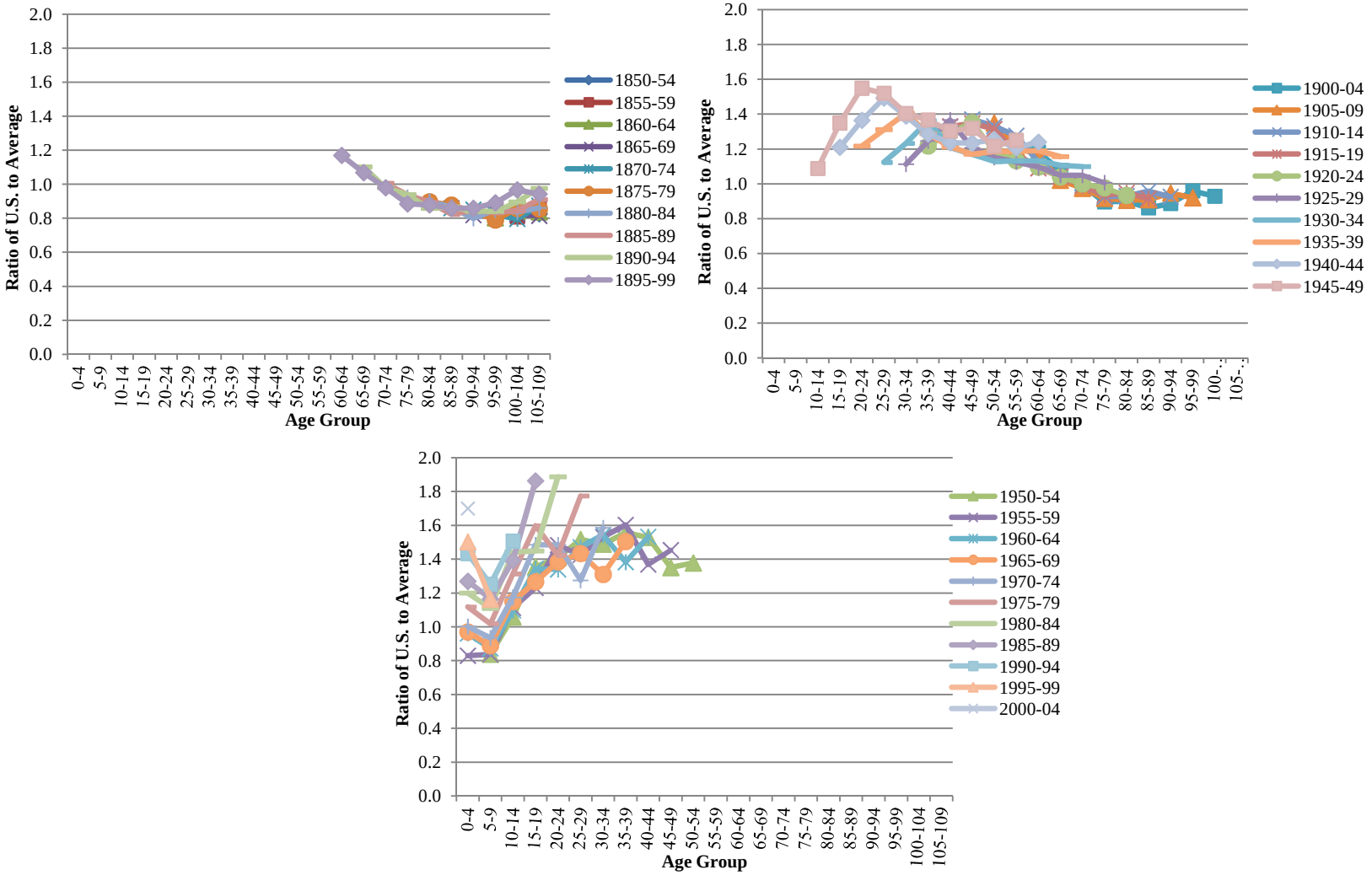
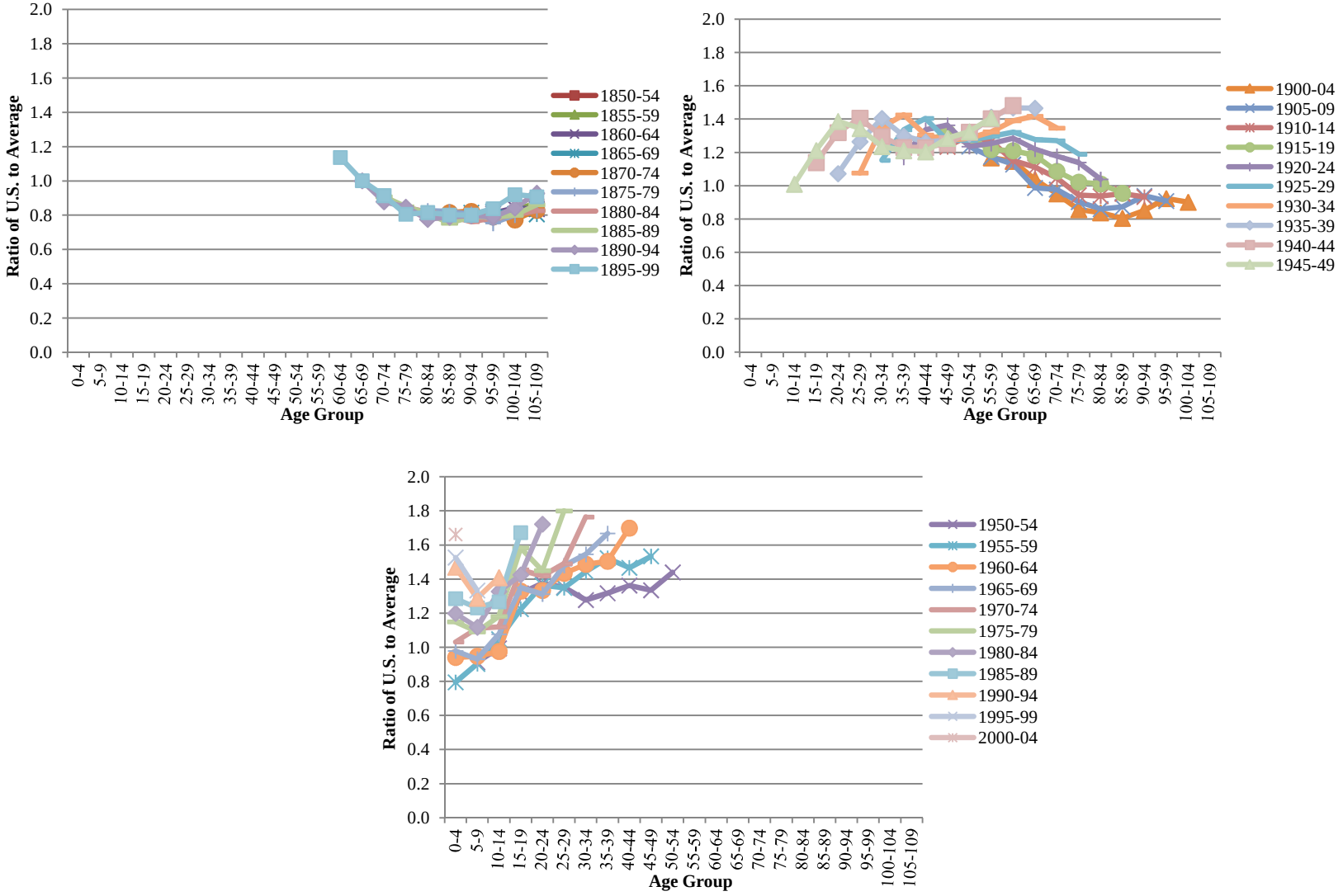


Figure 2.16. Cohort Ratios of U.S. Age-Specific Mortality Rates, Female Birth Cohorts Born Between 1850 and 2004



CHAPTER III. Season of Birth, Environmental Exposures, and Adult Health in Six Developing Countries

Introduction

Chronic disease has become increasingly important as a major cause of morbidity and mortality worldwide. Estimates from the Global Burden of Disease Study indicate that noncommunicable diseases accounted for approximately 65.5% of all deaths in 2010 (Lozano et al. 2012). Out of the top 10 and 25 causes of death, five and 13, respectively, were noncommunicable diseases (Ibid.). Increasingly, researchers have taken a life course approach to unpacking the determinants of the development of chronic disease and adult mortality. Many studies have concentrated on the early life origins of adult health, how exposures experienced *in utero* or in infancy and early childhood affect morbidity and mortality later in life.

However, the importance of environmental conditions experienced in early life for health over the life course remains relatively understudied. Infectious disease exposure and nutrition are two of the most important early life conditions thought to influence health and mortality in adulthood, and environmental conditions have the potential to influence both disease environment and nutritional intake in the prenatal and postnatal periods. For example, certain temperature and rainfall levels can produce conditions that are particularly conducive to the spread of infectious and parasitic diseases such as malaria, dengue, dysentery, cholera, and diarrheal diseases (Binka et al. 1994; Craig et al. 1999; Craig et al. 2004; McEniry and Palloni 2010). Rainfall and temperature levels also play a major role in determining crop yield and the quality of the harvest, which in turn affect

maternal and infant nutrition. Environmental conditions are likely to have exerted particularly strong effects in developing countries which, for much of the 20th century, lacked widespread access to refrigeration, clean water, and sanitation and experienced very high burdens of infectious and parasitic diseases. Several prior studies have examined the effects of extreme, one-time shocks such as famines, recessions, and pandemics experienced early in life on cardiovascular disease, disability, socioeconomic outcomes, and mortality in adulthood, primarily in the United States and other developed countries. In this chapter, I am interested in whether it is possible to detect the effects of exposure to routinely-experienced climate conditions early in life on later life health outcomes in developing countries. Developing countries, along with developed countries prior to the epidemiological transition, are the ideal settings to examine these associations because many of the mechanisms relating early life exposures and adult health operate only in conditions of high infectious disease burdens (Crimmins and Finch 2006).

In part due to limited data availability, few direct tests of the effects of environmental conditions on later life health have been performed in developing countries. Other studies have used season of birth as a proxy for these exposures because there exists seasonal variation in disease prevalence and in food availability. While season and month of birth patterns in health and mortality in developed countries are well-documented, relatively less is known about whether these patterns exist in developing countries and whether they differ from those observed in developed countries. This study aims to fill these gaps in the existing literature by: (1) estimating the associations between season of birth and adult health outcomes in six developing and newly industrialized countries (India, China, Ghana, Mexico, Russia, and South Africa), and (2) testing whether environmental

conditions (rainfall and temperature levels) experienced around the time of birth affect adult morbidity in India. These analyses have the potential to contribute to further theoretical refinements and to shed light on early life determinants of health among aging populations in developing countries.

In four of these countries (Ghana, Mexico, Russia, and South Africa), I find that individuals born in the autumn may experience a health advantage relative to those born in the other three seasons. This is consistent with season of birth patterns in longevity observed in developed countries (Doblhammer 2004; Doblhammer and Vaupel 2001; Gavrilov and Gavrilova 2011). According to the existing literature, the primary explanations for this autumn-born advantage are reduced exposure to infectious diseases and more favorable maternal and fetal nutritional conditions during the third trimester (coinciding with the harvest season) for autumn-born individuals compared to those born in other seasons. In contrast, I find that in China, the autumn-born appear to experience a health disadvantage relative to those born in other seasons. In India, which has slightly different seasons due to the monsoon, individuals born during the monsoon season appear to be healthier than their counterparts born in other seasons.

In India, I observe the most consistent associations between rainfall and temperature conditions prevailing during the pre- and postnatal periods and blood pressure and height in adulthood. Based on the existing literature, these are two of the health outcomes we would expect to be most strongly associated with early life conditions.[26] In general,

[26] Barker's fetal origins hypothesis and a substantial body of literature in this vein relates adverse early life conditions to an increased risk of cardiovascular disease and mortality from cardiovascular disease in adulthood. Three of the primary risk factors for cardiovascular disease are high blood pressure, high cholesterol, and diabetes. Unfortunately, no biological measures of the latter two risk factors are available in the datasets used in this chapter.

higher levels of rainfall and temperature around the time of birth appear to be deleterious for these adult health outcomes. Supplemental analyses of temperature and rainfall shocks and considering a longer post-birth window (up through 5 years after birth) support the importance of nutritional conditions during gestation and infectious diseases experienced during the weaning period for adult health outcomes.

Background

Early Life Conditions and Health Over the Life Course

In recent decades, renewed interest in early life conditions was spurred in part by observations that adult risk factors could not entirely predict individual risks of developing chronic disease or account for between-group differences in chronic disease burdens (Kuh and Ben-Shlomo 1997). Although the life course framework encompasses exposures that accumulate throughout the life course (e.g., those that occur during gestation, childhood, adolescence, young adulthood, etc.), the most relevant frameworks for this chapter focus on conditions experienced during gestation and early infancy, regarded as a "critical period" of exposure.

Prenatal Exposures: Nutrition and Infection

Barker's fetal origins hypothesis (Barker and Osmond 1986; Barker 1995; Barker et al. 2002; Eriksson 2005) focuses on the relationship between early life nutritional conditions and later life health. This model posits that adverse nutritional conditions experienced *in utero* can "program" organ systems (referred to as developmental plasticity), predisposing the individuals experiencing unfavorable conditions during critical

developmental periods to develop cardiovascular disease in adulthood. Birthweight and length at birth are the most commonly used markers of fetal nutrition (Barker et al. 2002; Forsén et al. 1999; Roseboom et al. 2001). In its most specific form, undernutrition in mid- to late gestation leading to small size and low weight at birth, combined with catch-up growth in childhood, increases the risk of adult coronary heart disease (CHD) (Barker 1995; Eriksson 2005). For example, using data from a Helsinki hospital cohort born in 1924-1933, Eriksson et al. (1999) and Barker et al. (2002) documented negative associations between birthweight and deaths from CHD and between birthweight and the incidence of CHD, type 2 diabetes, and hypertension. In both of these studies, individuals who had low birthweights and who experienced accelerated weight gain between ages 3-11 had the worst health outcomes, experiencing the highest risk of dying from CHD and the highest incidence of CHD, type 2 diabetes, and hypertension (Ibid.).

However, most other studies testing the Barker hypothesis focus on associations between measures of intrauterine conditions and adult health outcomes without considering compensatory growth in childhood. The majority of these studies examined effects of early life exposure to famines and to the 1918 influenza pandemic on later life outcomes since these two types of events are considered to be exogenous shocks or "natural experiments." Findings from famine studies have been mixed, while findings from studies of the 1918 influenza pandemic generally support the deleterious impact of pre- and post-natal exposure to the pandemic on height, cardiovascular disease prevalence, disability, socioeconomic outcomes, and mortality in adulthood. The main strength of these studies is their use of large, unexpected health shocks to arrive at internally valid estimates of the long-run health impacts of early life exposures. However, the limitations

of these studies are that these shocks are often rare and extreme forms of exposures, and they often (but not always) rely on small, nonrepresentative (e.g., hospital-based) samples. In addition, there may be selection into conception during these adverse health conditions, mortality selection that occurs *in utero* due to the health shocks (influencing the composition of live births occurring in these periods), and mortality differences post-birth that influence the composition of the populations surviving to older ages.

Famine Studies

Studies have considered the effects of early life exposure to five famines that occurred between the mid-1800s and mid-1900s on later life health outcomes: the 1846-1847 Dutch Potato famine, the 1866-1868 Finnish famine, the 1941-1944 siege of Leningrad, the 1944-1945 Dutch famine, and the 1959-1961 Chinese famine. Using historical data from three provinces in the Netherlands, Lindeboom, Portrait, and van den Berg (2010) considered three cohorts: those who were born before, during, and after the Dutch Potato famine, when the potato, rye, and wheat crops failed. Individuals exposed to the famine at birth or for at least 6 gestational months had significantly lower life expectancy at age 50 than individuals born before the famine, and the effects were stronger for men than women (Ibid.). Kannisto, Christensen, and Vaupel (1997) used vital statistics data to examine the long-run mortality impacts of the 1866-1868 Finnish famine, during which 8% of the total population died. Similarly, they considered three groups of cohorts born immediately preceding, during, and immediately following the famine. They found no significant differences in older age mortality among these groups; in fact, “the survival curves for the famine cohorts and the control cohorts were virtually identical after

childhood" (Ibid., p. 989). Stanner et al. (1997) focused on the impact of intrauterine nutritional deprivation experienced during the 1941-1944 siege of Leningrad, where roughly 31-42% of the city's population died (primarily of starvation), on diabetes and CHD in adulthood. They considered three groups: individuals exposed to the siege *in utero*, individuals exposed to the siege as infants, and an unexposed group of individuals born in the province of Leningrad but outside the siege limits during the same time period. Overall, this study found no evidence for long-term impacts of intrauterine malnutrition. Exposure to this famine was not associated with glucose intolerance, dyslipidaemia, hypertension, or cardiovascular disease in adulthood (Ibid.). Roseboom et al. (2001) and Painter et al. (2005) examined the impact of early life exposure to the 1944-1945 Dutch famine on later life health and mortality, respectively, using the Dutch famine birth cohort study (based on all singleton live births occurring between November 1, 1943 and February 28, 1947 in a hospital in Amsterdam). Official daily rations for the adult population fell from roughly 1800 calories to between 400-800 calories at the height of the famine (Roseboom et al. 2001). Roseboom et al. (2001) summarized a number of studies finding that individuals exposed to the Dutch famine in mid- to late gestation had reduced glucose tolerance and an increased risk of developing type 2 diabetes, while those exposed in early gestation had worse lipid profiles, higher body mass index, an increased risk of developing CHD, and worse self-rated health. However, there was no impact of prenatal famine exposure on blood pressure in adulthood (Ibid.). In contrast, Painter et al. (2005) found no effect of prenatal famine exposure on all-cause or CHD mortality using the same dataset. None of the three groups of individuals who were exposed to the famine in early, mid, or late gestation had elevated mortality

compared to those born before or conceived after the famine (Ibid.). Finally, Chen and Zhou (2007) and Huang et al. (2010) examined the long-term health and economic consequences of the 1959-1961 Chinese famine, which resulted in 15-30 million excess deaths. Chen and Zhou (2007) used data from the China Health and Nutrition Surveys and found that cohorts born in rural China in 1959, 1960, and 1962 were shorter and had lower labor supply and income in adulthood (Ibid.). Based on a sample of Chinese women born in 1957-1963, Huang et al. (2010) found that rural cohorts exposed to the famine had lower height, lower BMI, and an increased risk of hypertension in adulthood compared to the unexposed 1963 birth cohort.

In summary, studies of the 1866-1868 Finnish famine, 1941-1944 siege of Leningrad, and 1944-1945 Dutch famine found no support for the effects of prenatal exposure to famines on adult mortality (Finnish and Dutch famines) or on diabetes or cardiovascular disease (siege of Leningrad). However, studies found that early life exposure to the Dutch Potato famine of 1846-1847, the 1944-1945 Dutch famine, and the 1959-1961 Chinese famine had negative consequences for life expectancy at age 50, diabetes and CHD morbidity, and height and socioeconomic outcomes, respectively.

1918 Influenza Pandemic Studies

In the United States, the influenza pandemic infected approximately 30% of the population, with three waves occurring between March 1918 and March 1919 (Myrskylä, Mehta, and Chang 2013). Influenza deaths peaked in October 1918 and remained elevated through the first quarter of 1919 (Almond 2006). Almond (2006) found evidence that individuals who were *in utero* during the pandemic had increased

rates of physical disability, reduced educational attainment, lower income, and higher transfer payments compared to the surrounding birth cohorts. Those who were born in the first two quarters of 1919 had lower high school graduation rates and higher disability rates compared to those born in the surrounding birth quarters (Ibid.).[27] Using data from the National Health Interview Survey (NHIS), Mazumder et al. (2010) found that individuals born in 1919 had an increased prevalence of cardiovascular disease (particularly ischemic heart disease) and were shorter in adulthood compared to adjacent cohorts. The effects were strongest for those born during the first quarter of 1919. Myrskylä, Mehta, and Chang (2013) also used data from the NHIS to examine the effect of exposure to the pandemic on mortality but considered a more detailed coding of pre- and postnatal exposure to the three waves of the pandemic. Compared to unexposed cohorts (those born between 1920-1924), cohorts who were exposed in the third trimester and at birth to the influenza pandemic (those born in the second quarter of 1918 and the first quarter of 1919) experienced significantly elevated mortality from noncancer causes, the majority of which are due to cardiovascular and respiratory diseases (Ibid.). The main mechanisms suggested for these findings include elevated levels of maternal stress, infection, and nutritional deprivation; infection-related increases in chronic inflammation; deleterious effects on lung maturation; and elevated risk of preterm birth (Ibid.; Mazumder et al. 2010).

[27] Since individuals born in the first six months of 1919 could have been exposed to the height of the epidemic during the first, second, or third trimesters, it is difficult to tell if exposure during any specific trimester was most important. However, Almond (2006) notes that on average, welfare payments were highest for individuals who were in the first trimester during the peak of the pandemic.

Postnatal Exposures: Infections

Specific examples of early-life infections that have been linked with later life morbidity include tuberculosis, hepatitis B, streptococcal infections, and diarrhea and enteritis (Elo and Preston 1992). Streptococcal infections cause both upper and lower respiratory tract infections. The former have been linked to acute rheumatic fever and rheumatic heart disease in adulthood through damage to the heart valves, while the latter have been linked to chronic obstructive lung disease through impaired lung development and function (Ibid.). *Helicobacter pylori* infection is spread through person-to-person, oral-oral, and fecal-oral modes of transmission and has been linked to peptic ulcer disease and stomach cancer (Go 2002). It is common among children living in crowded and unsanitary conditions (including contaminated food and water) in developing countries (Ibid.; Elo and Preston 1992; Monteverde, Noronha, and Palloni 2009). Height is a measure of both nutritional and infectious disease exposures in childhood. Studies conducted in Bangladesh, Brazil, the Gambia, and Guatemala have documented associations between chronic parasitic and gastrointestinal tract infections such as diarrheal diseases and dysentery and slower height increases (Stephensen 1999). The mechanisms operating in these cases are hypothesized to be both physiological scarring and malnutrition caused by infections (resulting from reduced food intake, reduced nutrient absorption, nutrient losses, increased metabolic requirements, and the diversion and reallocation of nutrients from routine developmental processes).

More broadly, Finch and Crimmins (2004) relate exposure to infectious diseases and poor nutrition early in life to height, chronic disease morbidity, and mortality in adulthood. The primary mechanisms believed to be operating are chronic inflammation and energy

reallocation, which are set off by the adverse early life infections and in turn lead to lower height and the development of a diverse set of chronic diseases including heart disease, stroke, and cancer. One criticism of this hypothesis is that support for this mechanism remains restricted to a connection between inflammation caused by periodontitis and *Chlamydia pneumonia* infections in late childhood and early adolescence and adult coronary artery disease (Monteverde, Noronha, and Palloni 2009).

Several studies have explored the impact of postnatal early life conditions on a wide array of adult health outcomes in both developed and developing countries. While most studies find that adverse childhood conditions increase the risk of morbidity, disability, negative socioeconomic outcomes, and mortality in adulthood, at times, the associations between early life conditions and adult health outcomes have operated in unexpected directions. For example, early infectious exposures are hypothesized to result in increased inflammation and the development of chronic conditions later in life. Thus, we would expect to observe a positive association between early infectious exposures and C-reactive protein (CRP), a marker of inflammation. However, McDade et al. (2010) found that in the Philippines, higher levels of microbial exposures in childhood were associated with lower levels of adult C-reactive protein.

Developed Country Studies

Support for the chronic inflammation hypothesis proposed by Finch and Crimmins (2004) comes mainly from studies examining the association between early life mortality and adult mortality, most likely due to limited measures of infectious disease exposure in historical populations. These studies are often based on historical mortality data for

cohorts born in Europe (e.g., Sweden, France, Switzerland, and England) between the mid-1700s and early 1900s. Beltrán-Sánchez, Crimmins, and Finch 2012, Finch and Crimmins (2004), and Crimmins and Finch (2006) documented positive associations between early life cohort mortality and adult mortality levels but negative associations between early life cohort mortality and rates of mortality acceleration. Based on data from France and Sweden during the 19th century, Crimmins and Finch (2006) also found a strong inverse association between cohort childhood mortality and cohort adult height, such that taller cohorts had lower early-age mortality. Bengtsson and Lindstrom (2000) used Swedish parish data to examine the associations between four indicators of early life conditions (rye prices 9 months prior to birth as a proxy for maternal nutrition, mortality at ages 20-50 years for disease load of the mother, and mortality at ages 0-1 and 0-5 for disease load of the child) and adult mortality at ages 55-80 between 1760-1895. Out of these indicators, only mortality in the first year of life demonstrated a significant (positive) association with overall adult mortality and particularly mortality from airborne infectious diseases in adulthood (Ibid.). These results were supported by a follow-up study also using Swedish parish data (from 1766-1894) where the authors found that greater exposure to airborne infectious diseases in the first year of life (primarily from smallpox, whooping cough, pneumonia, and measles) was associated with higher mortality at ages 55-80 (Bengtsson and Lindstrom 2003).

Using data from Denmark, England and Wales, Finland, the Netherlands, and Sweden, Myrskylä (2010) found weak positive associations between cohort mortality shocks experienced during the first year of life and mortality at older ages and stronger positive associations between period mortality shocks and mortality at older ages. Bozzoli,

Deaton, and Quintana-Domeque (2009) examined the association between postneonatal mortality (occurring between ages 1 month-1 year), used as a proxy for disease and nutritional conditions in childhood, and adult height among cohorts born between 1950-1980 in the U.S., England, and 10 continental European countries. They find that postneonatal mortality predicts adult height in these countries, with mortality from pneumonia (versus mortality from congenital anomalies, intestinal disease, or other causes) emerging as the strongest predictor. The authors interpret their results as support for evidence of scarring and inflammation as key pathways linking early life conditions and later life outcomes (Ibid.).

Studies focusing on more recent populations in developed countries have generally relied on individual-level datasets containing retrospective self-reports of specific diseases experienced in childhood or other markers of childhood disease and nutritional conditions (e.g., height). Using data from the Health and Retirement Study (HRS), Blackwell et al. (2001) examined the associations between infectious, non-infectious, and autoimmune childhood illnesses on adult chronic diseases. Infectious childhood illnesses were associated with significantly elevated risks of cancer, cardiovascular disease, lung conditions, and arthritis in adulthood, but not diabetes. Non-infectious childhood illnesses were only associated with a significantly elevated risk of cancer, and autoimmune childhood illnesses were never significant predictors of adult morbidity (Ibid.). Haas (2008), also using data from the HRS, found that poorer self-rated childhood health between birth and age 16 and poorer childhood socioeconomic conditions were associated with higher levels of functional limitations at baseline and worse trajectories (higher rates of increase) in functional limitations in adulthood.

Using height as a marker of childhood health, Case and Paxson (2010) examined the associations between height and schooling, employment, earnings, health, and cognitive ability in adulthood. Using five British and American surveys[28], they found significant positive associations between height and educational attainment, employment, and earnings. Taller individuals in these samples had better self-rated health, were less likely to report being disabled, and had fewer functional limitations (Ibid.).

Developing Country Studies

Monteverde, Noronha, and Palloni (2009) used data from the Puerto Rican Elderly: Health Conditions survey (PREHCO) and Salud, Bienestar y Envejecimiento en América Latina y El Caribe (SABE), which was fielded in seven cities in Barbados, Argentina, Cuba, Mexico, Uruguay, Chile, and Brazil, to investigate the effects of early life conditions on disability in Latin America and the Caribbean. Deleterious early life conditions were based on retrospective reports of poor socioeconomic conditions, poor health, and infectious diseases (hepatitis, tuberculosis, rheumatic fever, chronic bronchitis, nephritis, typhus, polio, malaria, dengue, pneumonia, and asthma) experienced during the first 15 years of life. In Puerto Rico and the seven SABE cities, poor early conditions increased the probability of having mental, respiratory, circulatory, and musculoskeletal chronic diseases among those aged 60 and above. These chronic conditions, in turn, were significant predictors of disability (Ibid.). Based on the Mexican Health and Aging Study (MHAS), Kohler and Soldo (2005) found that having a serious health condition before age 10 (tuberculosis, rheumatic or typhoid fever, polio, or any

[28] These surveys were: the National Child Development Study, the British Cohort Study, the Panel Study of Income Dynamics, Whitehall II, and the Health and Retirement Study.

other serious health problem) and going to bed hungry as a child were positively associated with having diabetes in adulthood. In contrast, having a toilet inside the house before age 10 (a proxy for early life exposure to infectious and parasitic diseases) lowered the risk of diabetes in adulthood (Ibid.). Also using the MHAS, Huang, Soldo, and Elo (2011) found that experiencing serious health conditions before age 10 and going to bed hungry as a child significantly increased the risk of lower-body functional limitations. This finding was robust to the inclusion of both childhood and adult socioeconomic conditions (Ibid.).

The two frameworks discussed above propose a positive relationship between early life exposures and adult health, where health insults experienced in infancy and childhood lead to poorer health and elevated mortality in adulthood. However, the typology offered by Preston, Hill, and Drevenstedt (1998) allows for greater heterogeneity in the effects of early life conditions. Two of the four mechanisms, physiological scarring and correlated early-life and adult social environments, lead to positive relationships between mortality risks in childhood and adulthood. The majority of the pathways discussed in the preceding paragraph could be considered examples of physiological scarring. In contrast, the other two mechanisms, acquired immunity and selection, result in negative relationships between mortality risks in childhood and adulthood.

In summary, a large number of studies have suggested that undernutrition and infections *in utero* and infections contracted in the immediate post-birth period are related to worse health and increased mortality in adulthood. The most consistent associations between early life conditions and adult health have been found for the following outcomes: height, cardiovascular diseases (e.g., CHD, hypertension, and stroke), and respiratory diseases.

In general, the main limitations of these studies are that they often rely on retrospective self-reports of childhood conditions (which may be subject to recall bias) and self-reporting of adult health outcomes.

Environmental Conditions and the Disease Environment

Environmental conditions such as temperature and rainfall can exert strong influences on the disease environment. For example, the incidence of waterborne infectious diseases affecting the gastrointestinal tract is correlated with flooding and warmer temperatures (Doblhammer 2004). Higher temperatures can shorten pathogens' incubation periods, contributing to the spread of foodborne diseases. Diarrheal disease, a major cause of morbidity and mortality among children, was historically referred to as the "summer complaint" (Preston and Nelson 1974). The incidence of other gastrointestinal diseases such as cholera, dysentery, and typhoid also peaks in summer. Temperature and rainfall levels can also affect the proliferation and range of disease vectors like mosquitoes, flies, and rodents. Malaria is particularly sensitive to climate conditions since temperature affects both parasite and vector development, while rainfall influences mosquito breeding sites and survival (Craig et al. 1999; Craig et al. 2004). As a result, climate data is commonly used to predict malaria epidemics.

If environmental conditions increase the incidence of infectious and parasitic diseases, the synergy between infections and the absorption of nutrients provides an additional pathway through which environmental conditions affect child nutrition and health. Mata (1992) documented the striking effects of repeated infections (predominantly from diarrhea) on weight gain in children. Environmental conditions and their associated

effects on infectious disease burdens are likely to be particularly relevant for this analysis, which considers samples of older individuals born prior to 1960. For example, refrigeration is still not widespread in India: as of 2002, only 3.8% of rural households and 30.0% of urban households reported having a refrigerator (Sharma and Haub 2008).

Several recent studies have examined the effects of environmental conditions on child outcomes (mainly height and infant mortality) in developing countries. While these studies generally find that rainfall and temperature conditions are significantly associated with child outcomes, the direction of the effect varies depending on the outcome and context. Few of these studies discuss specific mechanisms connecting climate conditions and health outcomes, although the most commonly mentioned are nutrition, infectious disease prevalence, income shocks, and epidemiological pathways in general. Thai and Myrskylä (2012) suggest that rainfall shocks may affect health by increasing labor demand and consequently decreasing breastfeeding. Using data from the Vietnam Demographic Health Survey, they find support for this hypothesis – excess rainfall in the birth year decreased the proportion breastfed for more than 12 months and for more than 18 months by 11% and 9%, respectively, among children aged 12-36 months (Ibid.).

Rainfall and Temperature Studies

Skoufias and Vinha (2011) examined the effects of weather shocks (deviations from long-run averages) on height-for-age among children aged 12-47 months surveyed in 2000 in rural Mexico. They found that positive (higher) rainfall shocks in 1999 and negative (lower) temperature shocks in 1998 were significantly associated with lower height-for-age in most regions; negative (lower) rainfall shocks were significantly

associated with taller height-for-age in some areas; but positive (higher) temperature shocks were not significantly associated with height-for-age (Ibid.). Hoddinott and Kinsey (2001) examined the effects of the 1994-1995 drought on changes in height among children aged 12-60 months in rural Zimbabwe. They specify the "drought cohort" as children who were aged 12-24 months in 1995-1996, since failed rains in the 1994-1995 agricultural year were expected to result in food shortages during the subsequent 12 months. Compared to the older children in this sample, the drought cohort experienced growth faltering on the order of 1.5-2 cm, with no evidence of catch-up growth, and the impact was greatest for children in poor households (Ibid.). While the main focus of Bhalotra's (2010) study was on the effect of income shocks on infant mortality, the author also included rainfall shocks (deviations from state-level means) as a predictor.[29] A one standard deviation increase in rainfall was associated with a 0.004 decline in infant mortality risk among children born between 1961-1999 whose mothers were surveyed in the National Family Health Survey of India (NFHS-2) (Ibid.).

One historical study of the associations between rainfall and temperature and overall mortality rates in England in 1665-1834 found evidence of temperature effects but no evidence of rainfall effects on mortality during this period (Lee 1981). Cold winters (December-May) and hot summers (June-November) were associated with increases in mortality. The results suggest that increasing winter temperatures by 1° Celsius and lowering summer temperatures by 1° Celsius would reduce annual mortality by roughly 2% and 4% respectively, resulting in an increase in period life expectancy of

[29] This study used rainfall data from indiastat.com, which requires a membership fee to access the data. Their rainfall data appear to pertain mainly to the post-1950 period and may only be available at the state level, which would not be useful to my analyses.

approximately 2 years (Ibid.). Lee (1981) suggests that these associations may be due to mortality from respiratory tract diseases (e.g., pneumonia, bronchitis, influenza) among the elderly in winter months and mortality from digestive tract diseases among infants and children in summer months.

I know of only one study which examines the effect of rainfall on later life health outcomes. Maccini and Yang (2009) used the Indonesian Family Life Surveys (IFLS) and data from rainfall stations to examine the association between birth year rainfall and adult outcomes among Indonesian men and women born between 1953-1974. Among women, higher levels of rainfall in the birth year were significantly and positively associated with self-rated health, height, schooling, and socioeconomic status in adulthood; however, no significant effects are observed for men (Ibid.).

Season of Birth Patterns in Health and Mortality

While there is general agreement regarding the importance of early life conditions, data capturing these conditions have often been scarce. As a result, many scholars have focused on the effects of month or season of birth as a proxy or "indicator for environmental factors that are linked to seasons of the year" (Doblhammer and Vaupel 2001: 2934). Relative to birthweight, month of birth is regarded as being less susceptible to selection biases and to influence by socioeconomic factors. In addition, while birthweight captures only prenatal influences, month or season of birth serves as an indicator of seasonal influences operating during pregnancy and the first year of life. In general, studies have found that longevity is highest for those born between September and December and lowest for those born between March and June (Doblhammer and

Vaupel 2001; Doblhammer 2004; Gavrilov and Gavrilova 2011) for countries located in the Northern Hemisphere, although the peaks and troughs vary from study to study.

Using Swedish parish data from 1766-1894, Bengtsson and Lindstrom (2003) found strong season of birth influences on the association between infectious disease load in the first year and mortality at ages 55-80. The associations between infant mortality and older age mortality were stronger for individuals born in the winter and summer than those born in spring and autumn. The authors note that historically in Sweden, infectious diseases peaked in the winter and summer months, with respiratory diseases and smallpox being particularly important in winter months and water- and airborne infectious diseases being highly prevalent in the summer months (Ibid.). Based on more recent data, Doblhammer (2004)'s comprehensive study found distinctive patterns in life expectancy at age 50 by season of birth in Australia, Austria, Denmark, and the United States. The data were based on individuals aged 50 and above who died between 1988-1996 in Austria, between 1993-1997 in Australia, between 1968-1998 in Denmark, and between 1989-1997 in the United States. In Austria and Denmark, located in the Northern Hemisphere, individuals born between September and December had the highest life expectancy at age 50, while those born between March and June had the lowest life expectancy at age 50. In the U.S., those born between September and November had the highest life expectancy at age 50, and those born between April and July had the lowest life expectancy at age 50. This pattern is mirrored in Australia, located in the Southern Hemisphere, where those born between March and June had the highest life expectancy at age 50 and those born between September and December had the lowest life expectancy at age 50. Differences in adult lifespan between those born in October-

December and April-June were statistically significant and ranged from 0.3 to 0.6 years in Denmark, Austria, and Australia (Ibid.).

For regions located somewhat closer to the equator (e.g., Hawaii and Queensland, Australia), temperature appears to influence longevity. In these areas, mean age at death is highest for months coinciding with lower temperatures (January-April in Hawaii and May-June in Queensland) (Doblhammer 2004). Season of birth patterns by cause of death were also identified in Austria and the United States. Compared to those born in October-December, those born in April-June in Austria were significantly more likely to die at younger ages from ischemic heart disease, cerebrovascular disease, diabetes, certain cancers (stomach cancer and the residual group of "Other neoplasms"), chronic respiratory diseases, pneumonia and influenza, and injuries (Doblhammer and Vaupel 2001). In the U.S., those born in September-November died later from several cancers (breast, prostate, lung, colorectal, stomach, pancreatic, liver, and the residual group of "Other neoplasms"), circulatory diseases, diabetes, chronic respiratory diseases, pneumonia, influenza, and injuries compared to those born in April- June (Doblhammer 2004). In Doblhammer (2004)'s analyses, the hypothesis that these patterns were related to food availability and infectious disease exposures experienced early in life and which differ by season of birth received the strongest support. For example, autumn months generally coincide with the harvest season, so individuals born in autumn and early winter were likely to have experienced the most favorable nutrition conditions during the third trimester (the period of peak growth) and directly after birth (Ibid.).

These findings for the U.S. are consistent with those of Costa and Lahey (2005) and Gavrilov and Gavrilova (2011). Using historical data on white male Union Army recruits

who survived to 1900, Costa and Lahey (2005) found that individuals born in the second and third quarters of the year experienced elevated mortality rates between ages 60-79 compared to those born in the fourth quarter. They ruled out differences in household wealth, father's occupation, and mortality selection as explanations for these patterns and suggested that maternal diet and early life exposures to infectious diseases were the likely drivers of these patterns (Ibid.). Gavrilov and Gavrilova (2011) examined month of birth patterns among validated centenarians born in the U.S. in 1880-1895. Compared to their shorter-lived siblings and spouses, centenarians were more likely to be born in September-November and less likely to be born in March, May, and July (Ibid.).

Two recent studies of season of birth effects on health in developing countries focused on Puerto Rico and India. McEniry and Palloni (2010) examined the effects of seasonal exposures to poor nutrition and infectious diseases during late gestation in a sample of older Puerto Ricans. In this study, season of birth effects were hypothesized to derive from seasonal variation in parental employment and exposure to infectious and parasitic diseases during the hurricane season. Controlling for childhood socioeconomic conditions, childhood health conditions, and adult risk factors, individuals born in rural areas during high exposure periods were at greater risk of developing heart disease (Ibid.). Lokshin and Radyakin (2012) examined the relationship between month of birth and child height in India using the NFHS.[30] They found that children born in May, June, and July (during the monsoon months) had lower anthropometric scores (height-for-age z-scores) compared to children born during November, December, and January (Ibid.).

[30] They also examine but do not present rainfall coefficients from models controlling for both month of birth and district-level rainfall (taken from the same data source used in this analysis). They note that the month of birth coefficients remain largely unchanged after controlling for rainfall and that prenatal rainfall does not predict height-for-age, but they do not describe the direction of effects of rainfall at the month of birth or postnatal rainfall (1-2 months after birth) on height-for-age.

To the best of my knowledge, however, no studies have examined the effects of being born during the monsoon season on later life health outcomes in India.

Studies in developed countries have found month and season of birth patterns in adult mortality, while studies in developing countries have documented month and season of birth patterns on height among children and cardiovascular disease among adults. The main criticisms of such studies are that month or season of birth is not free of socioeconomic influences (e.g., those in certain occupations or social classes are more likely to have births in certain seasons than others). However, Doblhammer (2004) concluded that the month-of-birth patterns in longevity observed in developed countries were not due to differences in the seasonal distribution of births by parental socioeconomic status or age at entry into school, seasonality of death, or differential infant survival. Finally, as with many of previous studies discussed above, reliance on self-reported morbidity in adulthood may be an issue.

Studying season of birth effects in developing countries is particularly appropriate given that these effects are expected to be strongest in populations with greater susceptibility to seasonal cycles in disease prevalence and food availability and with higher disease loads (e.g., prior to mortality transitions) (Becker and Weng 1998; Costa and Lahey 2005; Crimmins and Finch 2006). This is supported by Doblhammer and Vaupel (2001) and Doblhammer (2004), who found that in developed countries, differences in adult lifespan by month of birth were significantly smaller in more recent cohorts that had experienced substantial improvements in maternal and infant mortality relative to older cohorts.

Data from the Annual Reports of the Public Health Commissioner of India (1937) indicate that the prevalence of and mortality from several diseases were highly seasonal in the first half of the 20th century. For example, in 1931-1935, cholera deaths peaked between July and October, which makes since given that these months coincide with the monsoon season and cholera is a waterborne disease (**Figure 3.1**). It is also notable that in two of the years, 1932 and 1933, cholera mortality was much lower and did not exhibit pronounced seasonal variation. In contrast, plague deaths in 1923-1935 peaked between February and May, during what are considered the winter and summer seasons (**Figure 3.2**). Plague mortality during this time period appears to have been lowest between June and November, during the monsoon season. Smallpox cases also peaked between February and May in 1931-1935, with 1931 and 1932 being somewhat more favorable years in terms of lower smallpox incidence (**Figure 3.3**). These figures of seasonality in disease prevalence and mortality from the 1920s-1930s provide a snapshot of disease conditions to which individuals in the analytical sample would have been exposed.[31]

Hypotheses

In the first part of the analysis, I examine associations between season of birth and adult health in six developing and newly industrialized countries. Prior studies, mostly conducted in developed countries, suggest that individuals born between September and December have a mortality advantage relative to individuals born in the other months of the year. It is likely that this pattern also holds for morbidity. The standard meteorological definitions for autumn are September-November in the Northern Hemisphere and March-May in the Southern Hemisphere, which I employ in this analysis

[31] Respondents in the India sample were born between 1913 and 1957.

(see **Table 3.1** for the season of birth classifications in each of the six countries).[32] Thus, I hypothesize that chronic disease prevalence will be lower among those born in September-November in the Northern Hemisphere (China, Ghana, Mexico, Russia) and among those born in March-May in the Southern Hemisphere (South Africa) compared to individuals born in the remaining months. Specifically, I test for an autumn-born health advantage by comparing those born in autumn to those born in all other months.[33] The India Meteorological Department (Attri and Tyagi 2010) designates the seasons in India as: winter (January-February), summer (March-May), monsoon (June-September), and post-monsoon (October-December). One previous study (Lokshin and Radyakin 2012) has examined the effect of being born during monsoon months on height-for-age among children. In this study, I examine the association between adult health outcomes and being born in the monsoon season versus being born in all other months. It is indeterminate which direction the effect will be in since higher levels of rainfall may promote the transmission of water- and vector-borne diseases like cholera and malaria, but temperatures are highest during the summer months, when the incidence and mortality from diseases such as smallpox and plague peak.

In the second part of the analysis, I examine the impact of pre- and postnatal environmental conditions (rainfall and temperature) on adult health in India. While environmental conditions are hypothesized to be important influences on early life disease environment and nutritional conditions, relatively few direct tests of their

[32] I also test the robustness of the results to defining autumn as October-December in the Northern Hemisphere and April-June in the Southern Hemisphere following Doblhammer (2004).
[33] In addition, **Appendix Tables A3.5-A3.10** present results comparing those born in autumn to those born in the winter, spring, and summer seasons separately in China, Ghana, Mexico, Russia, and South Africa and those born in the monsoon season to those born in the winter, summer, and post-monsoon seasons in India.

importance for later life health outcomes have been performed. In addition, many prior studies of the effects of early life conditions on adult health focus on the long-term impacts of large, one-time shocks (e.g., pandemics, famines, droughts). I am interested in testing whether routinely-experienced environmental conditions affect adult health outcomes. The expected direction of the association between rainfall and adult health outcomes is indeterminate: more rain can be beneficial or deleterious in its effects. On the one hand, it is very important to have sufficient rain for crops, which affects both fetal and infant nutrition. On the other hand, too much rain can be deleterious for crops, cause flooding, and contribute to the spread of water- and vector-borne diseases. However, I hypothesize that rainfall shocks during the monsoon will be associated with worse health. Excess rain (above the 90^{th} percentile) may lead to flooding, poor harvests, and greater spread of infectious and parasitic diseases, while insufficient rain (below the 10^{th} percentile) may result in failed harvests. I hypothesize that higher temperatures will be unfavorable for health because they may contribute to food spoilage and expand the range of disease vectors. I also hypothesize that particularly high temperatures in the summer will be associated with worse health, potentially by promoting diarrheal diseases through food spoilage and by providing ideal conditions for mosquitoes, which transmit malaria and dengue. These hypotheses are summarized in **Table 3.2**.

Based on the existing literature (summarized above), studies have found the most consistent associations between early life exposures and height, cardiovascular diseases, respiratory diseases, disability, and mortality. Thus, out of the outcomes considered in this analysis, I expect the associations between season of birth and adult health and between early life environmental exposures and adult health to be strongest for: height;

blood pressure, hypertension, angina, and stroke (all risk factors for or types of cardiovascular disease); and chronic lung disease (a respiratory disease).

Data and Methods

Data

The WHO Study on Global Ageing and Adult Health (known as SAGE) is the primary data source for this study. SAGE is a longitudinal survey modeled after the HRS, the English Longitudinal Study of Ageing, and the World Health Survey, with waves 2 and 3 to be fielded in the coming years.[34] The first wave was fielded in 2007-2010 in six countries: India, China, Ghana, Mexico, Russia, and South Africa. The samples are nationally representative of the population aged 50+ in each country, although they also include younger adults aged 18-49 (Kowal et al. 2012). The total number of respondents aged 50+ in each survey were: 13,367 (China), 4,724 (Ghana), 7,150 (India), 2,315 (Mexico), 3,933 (Russia), and 3,840 (South Africa). The following number were dropped due to missing information on month of birth: 623 (China), 3,540 (Ghana), 5,196 (India), 9 (Mexico), 64 (Russia), and 569 (South Africa). Next, 6,769 (China), 346 (Ghana), 578 (India), 1,369 (Mexico), 1,609 (Russia), and 1,940 (South Africa) cases were dropped due to missing information on the other key variables of interest (i.e., respondents in the final sample were not missing information on any of the adult health outcomes, state, education, father's education, and father's occupation). The final sample sizes for each country were as follows: 5,975 (China), 838 (Ghana), 1,376 (India), 937 (Mexico), 2,260

[34] While the analyses in the present study examine the association between season of birth and adult health at a point in time, the longitudinal design of this study allows for the possibility of looking at the relationship between season of birth and changes in health status over time once data from subsequent waves become available.

(Russia), and 1,331 (South Africa). For India, an additional subsample was used: individuals who were not missing information on rainfall and temperature near the time of birth and who had always lived in their current district of residence (N=1,044).

This dataset has many strengths for the present analysis: it includes information on respondents' geographic location and month of birth, which are rare and essential to this study; it collects measured as well as self-reported health indicators; and it is very recent, allowing me to assess the current state of health and aging in developing countries. In addition to collecting information on respondents' sociodemographic characteristics, SAGE also collects information on self-reported chronic conditions, functional limitations, anthropometrics, performance tests, and biomarkers. The questionnaires were designed to be fielded consistently across the six survey countries.

The second dataset used in this analysis is the Climate Research Unit (CRU) TS2.1 dataset, which is publicly available through the Indian Meteorological Department (IMD) and collected by the Tyndall Centre for Climate Change Research, School of Environmental Sciences, University of East Anglia. This dataset contains monthly temperature and rainfall measures from rainfall stations in selected districts in 35 Indian states and union territories from 1901-2002. While variation in climate conditions within districts may remain, districts are a much finer unit of analysis than other larger administrative divisions (e.g., states). District-wise data are produced from interpolations based on 0.5 degree latitude-longitude climate grids (Mitchell and Jones 2005). These data are linked to the SAGE India sample by matching on month, year, state, and district of birth. The sample for these analyses consists of 1,044 respondents from six states and 118 districts who are considered non-movers.

Health Outcomes

A set of measured and self-reported health outcomes are the main outcomes of interest in these analyses. Respondents are coded as falling into either the "normal" or "high-risk" category for the measured health outcomes (see **Table 3.3** for the measures and high-risk cutpoints used in this study). Most of the high-risk cutpoints are taken from the recommendations of the World Health Organization (WHO), National Institutes of Health, or previously published studies. There is some evidence that the associations between biomarkers and health outcomes may differ across populations. For example, WHO Asia Pacific (WHO 2000), Misra et al. (2006), and Mohan et al. (2007) suggest that Asian populations may have lower cutpoints for body mass index (BMI) and waist circumference because they may have less skeletal muscle mass and greater abdominal obesity and visceral fat at lower BMIs. Although the WHO has not issued standard cutpoints for specific Asian populations (e.g., WHO 2004; WHO 2013), WHO Asia Pacific (WHO 2000) and several epidemiological studies proposed alternate thresholds based on examinations of cutpoints that best identify cardiovascular risk factors. I test the sensitivity of these results for the India sample to two alternate cutpoints for BMI (≥23.0 kg/m^2 and ≥25.0 kg/m^2) and waist circumference (≥ 90 cm for males, ≥ 80 cm for females and ≥ 87 cm for males, ≥ 82 cm for females) from epidemiologic studies conducted using data on Asian Indian adult populations (Misra et al. 2006), and Mohan et al. 2007).

The blood pressure and pulse rate measures are based on the average of three measures taken.[35] For systolic and diastolic blood pressure, individuals were coded as falling into the high-risk categories if they met the high-risk cutpoints or if they reported taking

[35] This was true for respondents in all of the SAGE countries except for Mexico, where blood pressure and pulse rate measures were based on the average of two measures taken per individual.

antihypertensive medication within the past two weeks. A third blood pressure measure (referred to as overall blood pressure) was created, with respondents falling in the high-risk category if they met the high-risk cutpoints for *both* systolic and diastolic blood pressure (also adjusted for hypertensive medication usage in the past two weeks). For height, I consider two outcome measures: whether the respondents fell below the 10^{th} or 25^{th} height percentiles, where the thresholds are specific to each sex and country. These thresholds are based on all SAGE respondents aged 50+ for whom height measurements were taken. **Tables A3.1-A3.4** show the percentages of adults in each sample who fall into the normal and high-risk categories for these health outcomes.

Questions eliciting information on chronic diseases are in the form of: "Have you ever been diagnosed with" or "Have you ever been told by a health professional that you have had" a particular condition. These conditions are: hypertension, diabetes, angina, stroke, chronic lung disease, arthritis, and asthma. The reporting of chronic conditions and other health measures is dependent on respondents' interactions with health care professionals and health knowledge and the level of health system infrastructure within each country. However, the collection of measured health outcomes allows for the examination of the relationship between early life conditions and health outcomes which are not dependent on individuals' interactions with the health care system. **Tables A3.1-A3.4** show the percentages of adults in each sample who report having been diagnosed with each of these chronic conditions.

Explanatory Variables

The main social and demographic variables considered are age, sex, region of residence, father's education, father's occupation, and respondent's education (see **Tables A3.1-A3.4** for the summary statistics for each of the samples). Region, father's education, and respondent's education are coded on a country-specific basis. Father's occupation is coded as a binary variable (agricultural vs. non-agricultural) capturing those who are classified as Skilled Agricultural and Fishery Workers based on the current version of the International Standard Classification of Occupations (ISCO-88).[36]

Season of Birth

The first predictor variable of interest is season of birth. Based on the existing literature, the autumn-born are hypothesized to have a health advantage relative to those born in other seasons. I use a binary variable that defines an autumn births as occurring in September-November and all other births as occurring in December-August in the Northern Hemisphere countries (China, Ghana, Mexico, and Russia) and autumn births as occurring in March-May and all other births as occurring in June-February in the

[36] Ideally, we would like to be able to determine whether the respondent's father was a farmer since the mechanisms through which these early life exposures are hypothesized to act (e.g., seasonal variation in food availability and disease prevalence, effects of rainfall and temperature on nutrition and disease transmission) may be most relevant for respondents from farming backgrounds and/or rural areas. However, this is difficult to ascertain since occupations are classified according to skill level and specialization rather than industrial sector under the ISCO-88 classification (Elias and Birch 1994). Farmers may be classified in three major groups (1 - Legislators, Senior Managers, and Officials, 6 - Skilled Agricultural and Fishery Workers, and 9 - Elementary Occupations) under the ISCO-88. Most farmers classified under major group 1 are not the type we are thinking of, since they would be managing very large farms. This leaves us with major groups 6 and 9, with most skilled market-oriented farmers and agricultural workers and subsistence agricultural occupations falling within major group 6 and unskilled farmhands falling within major group 9 (Ganzeboom and Treiman 1996). However, major group 9 is a highly heterogeneous group, including domestic cleaners, food vendors, and mining and construction laborers in addition to unskilled farmhands. Since it is not possible to separately identify unskilled farmhands from other occupations within major group 9 in all of the SAGE countries, the present study defines agricultural father's occupation as major group 6 alone for the sake of maintaining comparability across countries.

Southern Hemisphere countries (South Africa). **Tables A3.5-A3.10** show additional results using a four-category season of birth variable (defined as shown in **Table 3.1**). In India, the distinction is between births occurring in June-September (the monsoon season) and all other births occurring in October-May. While parents may be aware that certain seasons provide more or less favorable conditions for infants, month of birth is plausibly exogenous. In the full models, I test whether the effects of season of birth are robust to the inclusion of controls for father's education and father's occupation.

Temperature and Rainfall Conditions

The two main measures of climate conditions in India are constructed from monthly rainfall (in meters) and monthly minimum temperature (in °Celsius) measures capturing both pre- and post-birth conditions. I create three variables capturing climate conditions during the 9-month gestational period (total rainfall and average minimum temperatures observed during the first, second, and third trimesters) and three variables capturing conditions during a 9-month post-birth period (total rainfall and average minimum temperatures observed during the first 1-3, 4-6, and 7-9 months after birth). Respondents are assumed to have been born at the end of the month. For example, a respondent born in May 1930 would be linked to rainfall and temperature conditions observed in May 1930, April 1930, and March 1930 in the third trimester; February 1930, January 1930, and December 1929 in the second trimester, and November 1929, October 1929, and September 1929 in the first trimester. For the post-birth period, the climate conditions for this respondent would correspond to June 1930, July 1930, and August 1930 in the first

1-3 months; September 1930, October 1930, and November 1930 in the first 4-6 months, and December 1930, January 1931, and February 1931 in the first 7-9 months.

In supplementary analyses, I also examine the effects of rainfall and temperature shocks in the monsoon and summer seasons and rainfall and temperature conditions through the first 60 months after birth to test whether climate conditions are particularly important during the weaning period. I define an excess rainfall or temperature shock as occurring when rainfall or temperature in a given month and district is $\geq 90^{th}$ percentile of rainfall or temperature observed in that month and district between 1901-2002. I define an insufficient rainfall shock as occurring when rainfall in a given month and district is $\leq 10^{th}$ percentile of rainfall observed in that month and district between 1901-2002. I focus on whether rainfall shocks during the monsoon season and temperature shocks during the summer matter for adult health.

To capture rainfall and temperature conditions occurring during the first 60 months after birth, I create five variables capturing total rainfall and average minimum temperature during the first 1-12, 13-24, 25-36, 37-48, and 49-60 months after birth. I also consider whether rainfall and temperature shocks in each of these 12 month periods are associated with adult morbidity.

There is substantial temporal and spatial variation in India's climate, allowing for the examination of the effects of experiencing a wide range of temperature and rainfall conditions. **Figures 3.4-3.7** provide an illustration of this variability, considering climate conditions in 1907-1957 in the two most populous districts in the states of Rajasthan and West Bengal: Jaipur and North 24 Parganas. Rajasthan has a tropical desert climate.

Rainfall in Jaipur generally peaks in June-September, coinciding with the monsoon season, but the timing and magnitude of the peak vary considerably from year to year. During this time period, monthly rainfall ranged from 0 to 0.518 meters. In contrast, West Bengal is more coastal and has a more moderate climate. North 24 Parganas receives much more rainfall spread over a greater number of months, and the month of peak rainfall occurs slightly later than in Jaipur. Monthly rainfall in this district ranged from 0 to 0.704 meters in 1907-1957. The range in minimum temperatures is much wider in Jaipur, ranging from 5.6-29.7 °Celsius over this period, compared to 12.2-27.6 °Celsius in North 24 Parganas. Temperatures peak in May-September in Jaipur, with higher temperatures observed closer to May, whereas the level of peak temperatures is fairly stable over a greater number of months in North 24 Parganas.

Methods

In the first set of analyses, I estimate logistic regression models using season of birth (autumn vs. all other or monsoon vs. all other) to predict adult health outcomes in each of the six SAGE countries. In these models, being born in the autumn or during the monsoon season is specified as the reference category. In the second set of analyses, which focus on India, I consider the gestational and post-birth periods separately. First, I estimate logistic regression models using rainfall or temperature conditions in the three trimesters to predict adult health outcomes. Next, I estimate logistic regression models using rainfall or temperature conditions in the first 1-3, 4-6, and 7-9 months after birth to predict adult health outcomes. The dependent variables in these models are the set of the measured health outcomes and self-reported chronic conditions described above.

All models are estimated using sample weights to account for the complex survey design and include controls for age and sex. I specify five models: Model 1 (M1) includes controls for sex and 5-year age group. Model 2 (M2) adds region, Model 3 (M3) adds father's education, Model 4 (M4) adds father's occupation, and Model 5 (M5) adds respondent's education. In this chapter, I focus on and present results from Model 1, referred to as the basic model, and Model 5, referred to as the full model.

Results

I. Season of Birth and Adult Morbidity in Six Countries[37]

Results from these models are summarized in **Table 3.4** and detailed in the following text. The main hypothesis tested in these models is whether being born during the autumn or monsoon seasons confers a health advantage relative to being born in all other seasons.

South Africa

South Africa is the only one of the six SAGE countries located in the Southern Hemisphere, with its seasons proceeding from summer to spring during the calendar year. **Table 3.5 Part A** presents odds ratios from logistic regression models predicting measured health outcomes using autumn as the reference category. Individuals born in the autumn appear to be healthiest (the odds ratios for those born in the other seasons exceed one for all measured health outcomes). Compared to the autumn-born, those born in other seasons have significantly higher odds of falling below the 10^{th} height percentile (OR=1.96, M1). These findings are robust to the inclusion of controls including province,

[37] Significant differences refer to significance levels of $p<0.05$. Borderline significance refers to significance levels of $p<0.10$.

education, and father's education and occupation (M1 vs. M5). When the autumn-born are compared to those born in each of the other three seasons separately (**Table A3.5 Part A**), we see that in addition to having higher odds of falling below the 10^{th} percentile, the summer-born have significantly higher odds of having a high-risk pulse rate and the winter-born have significantly higher odds of falling below the 25^{th} height percentile compared to the autumn-born in the basic models. The odds ratios reach borderline significance for high-risk waist-to-hip ratio (spring vs. autumn).

For self-reported chronic conditions, we do not observe strong season of birth effects (**Table 3.5 Part B**). Those born in all other seasons have higher odds of reporting diabetes, arthritis, and asthma but lower odds of reporting hypertension, angina, stroke, and chronic lung disease compared to the autumn-born. The results are highly similar for the basic and the full models. In the full models considering a more detailed season of birth variable (**Table A3.5 Part B**), the results for hypertension, angina, and stroke reach borderline significance, with the spring-born being less likely to report hypertension and angina and the winter-born being less likely to report stroke compared to the autumn-born. The odds ratios for season of birth do not attain significance in the basic models or for the other health outcomes.

Thus, in South Africa, the results for the measured health outcomes provide weak support for a health advantage for the autumn-born relative to those born in other seasons, while the results for self-reported chronic conditions are mixed.

Ghana

In Ghana, there is some evidence to support a health advantage among the autumn-born for most of the measured health outcomes (**Table 3.6 Part A**). In general, the odds ratios for the other seasons exceed one, with the exceptions of waist-to-hip ratio, pulse rate, and height. Those born in all other seasons have significantly higher odds of having high-risk diastolic blood pressure (OR=1.46, M1), although this result reaches only borderline significance in the full model. Odds ratios for the other measured health outcomes are mostly similar in the basic and the full models with the exception of pulse rate and height, which are less than one in the basic models but exceed one in the full models. Consideration of the more detailed season categorization (**Table A3.6 Part A**) indicates that the finding for diastolic blood pressure is mainly due to a health advantage of the autumn-born relative to the summer-born.

Season of birth is not a significant predictor of any self-reported chronic conditions in Ghana (**Table 3.6 Part B**). Relative to those born in all other seasons, the autumn-born experience a health advantage for hypertension (basic and full models) and asthma (full model only) but a health disadvantage for diabetes, stroke, angina, and asthma. Results from models comparing the autumn-born to those born in each of the other three seasons separately indicate that the summer-born have significantly lower odds of reporting diabetes than the autumn-born (**Table A3.6 Part B**).

In Ghana, there appears to be weak support for a health advantage for the autumn-born relative to those born in other seasons for the measured health outcomes. However, there is no support for an autumn-born health advantage for self-reported chronic conditions.

Russia

In Russia, the autumn-born experience a health advantage relative to those born in other seasons for diastolic blood pressure, pulse rate, and height (**Table 3.7 Part A**). Compared to the autumn-born, those born in other seasons are more likely to fall below the 25th height percentile (OR=1.56, M1), but the results reach only borderline significance in both the basic and full models. In contrast, those born in other seasons appear to experience a health advantage relative to the autumn-born for obesity, waist circumference (significant at $p<0.10$), waist-to-hip ratio, systolic blood pressure (basic model only), and overall blood pressure. Results from models using a detailed season of birth categorization indicate that the autumn-born are less likely to fall below the 25th height percentile relative to the winter-born and are more likely to have high-risk waist circumference relative to the winter- and summer-born (all odds ratios significant at $p<0.05$ in both the basic and full models) (**Table A3.7 Part A**).

The results for self-reported chronic conditions provide stronger support for an autumn-born health advantage in Russia (**Table 3.7 Part B**). Compared to the autumn-born, those born in other seasons have higher odds of having diabetes (OR=1.84, M1), angina (OR=1.81, M1), and stroke (OR=3.32, M1). For diabetes and angina, the odds ratios are significant in both the basic and the full models. For stroke, the odds ratio is borderline significant in the basic model and significant in the full model. These results are supported by models comparing the autumn-born to those born in each of the other three seasons separately, where the odds ratios for those born in the other seasons all exceed one (**Table A3.7 Part B**). Compared to the autumn-born, the spring-born have significantly higher odds of diabetes and stroke and the summer-born have significantly

higher odds of stroke. Those born in other seasons also have higher odds of having hypertension and chronic lung disease relative to the autumn-born, although these results do not reach significance in the main models. However, those born in other seasons have higher odds of having arthritis and asthma than the autumn-born, although these differences are not significant.

In Russia, the autumn-born experience a health advantage for measured health outcomes such as blood pressure and height but not for waist circumference relative to those born in all other seasons. An autumn-born health advantage is observed for diabetes, angina, and stroke among the self-reported chronic conditions.

Mexico

In Mexico, the results for measured health outcomes generally support the existence of a health advantage for the autumn-born (**Table 3.8 Part A**). With the exceptions of waist-to-hip ratio (basic model only), systolic blood pressure, and pulse rate, the autumn-born experience a health advantage relative to those born in all other seasons. Compared to the autumn-born, those born in other seasons are significantly more likely to fall below the 25^{th} height percentile (OR=2.53, M1; the odds ratio reaches borderline significance in the full model) and more likely to have high-risk waist circumference (the odds ratio reaches borderline significance only in the full model). Results from models considering the more detailed season of birth variable are similar to those from the main models (**Table A3.8 Part A**). Compared to the autumn-born, the winter-born are more likely to fall below the 10^{th} and 25^{th} height percentiles and the summer-born are more likely to fall below the 25^{th}

height percentile (results significant at the 10% level in both the basic and full models in nearly all cases).

In terms of self-reported chronic conditions, however, the picture is more mixed (**Table 3.8 Part B**). The autumn-born appear to be better off in terms of hypertension and chronic lung disease but not diabetes, stroke, or arthritis. Compared to the autumn-born, those born in all other seasons have significantly higher odds of reporting chronic lung disease (OR=6.87, M1) in both the basic and full models. However, they have significantly lower odds of reporting diabetes (OR=0.41, M1) in both the basic and full models and lower odds of reporting stroke (borderline significant in the full model only). These findings are supported by results from models comparing the autumn-born to those born in each of the other three seasons separately (**Table A3.8 Part B**). Both the spring- and particularly the summer-born have higher odds of reporting chronic lung disease than the autumn-born (significant for the summer-born in both the basic and full models and borderline significant in the full model for the spring-born). These models indicate that those born in each of the other seasons have lower odds of reporting diabetes than the autumn-born (all odds ratios are significant in the full models and significant at the 5% and 10% levels for summer and winter, respectively, in the basic model).

In Mexico, the results for the measured health outcomes generally support a health advantage for the autumn-born relative to those born in other seasons, but the results for self-reported chronic conditions are mixed.

China

In China, the autumn-born appear to be the most unhealthy in terms of measured health outcomes (**Table 3.9 Part A**). With the exception of waist-to-hip ratio, the odds ratios are less than one for all other measured health outcomes. Compared to the autumn-born, those born in all other seasons have significantly lower odds of having high-risk systolic blood pressure (OR=0.80, M1) and overall blood pressure (OR=0.83, M1) in the basic and full models. They also have lower odds of being obese, having high-risk waist circumference, and having high-risk diastolic blood pressure than the autumn-born (results are borderline significant in the basic and full models). Results from models comparing the autumn-born to those born in each of the other three seasons separately are similar to the main models (**Table 3A.9 Part A**). Although the winter- and summer-born are also better off than the autumn-born for many of the measured health outcomes, the spring-born in particular experience a strong health advantage for obesity, systolic blood pressure, diastolic blood pressure, and overall blood pressure relative to the autumn-born (significant in all basic and full models).

Results from models for self-reported chronic conditions are mixed but also tend to indicate a health disadvantage for the autumn-born (**Table 3.9 Part B**). Relative to the autumn-born, those born in all other seasons are significantly less likely to report having hypertension (OR=0.89, M1; the odds ratio is borderline significant in the full model) and less likely to report having diabetes, angina, or asthma. However, those born in other seasons are more likely to report having stroke, chronic lung disease, or arthritis compared to the autumn-born. Results from models using a more detailed season of birth categorization are consistent with those from the main models (**Table A3.9 Part B**).

Compared to the autumn-born, the spring-born have significantly lower odds of reporting hypertension in the basic and full models, and the winter-born have borderline significantly lower odds of reporting hypertension in the basic and full models.

In China, I find no support for an autumn-born health advantage for either measured or self-reported adult health outcomes. It is unclear why the season of birth pattern may be different in China, but looking across different cohorts, provinces, and urban/rural status may help to shed light on these results.

India

In India, individuals born during the monsoon appear to be healthiest in terms of measured health outcomes compared to those born in all other seasons (**Table 3.10 Part A**). The odds ratios for those born in the other seasons exceed one for almost all measured health outcomes. The exceptions are obesity defined using the lowest alternate cutpoint ($\geq$23 kg/m^2) and high-risk waist circumference defined using both of the alternate cutpoints, although these differences are not significant. Compared to those born during the monsoon, those born in all other seasons are significantly more likely to meet the high-risk cutpoints for both systolic and diastolic blood pressure (OR=1.59, M1) in both the basic and full models. They are also more likely to have high-risk systolic blood pressure (borderline significant in the basic model), high-risk diastolic blood pressure (borderline significant in the basic and full models), and high-risk pulse rate (borderline significant in the basic and full models). Models comparing those born in the monsoon season to those born in the winter, summer, and post-monsoon seasons separately indicate that this disadvantage is mostly concentrated among individuals born in the

summer and, to a lesser extent, in the winter (**Table A3.10 Part A**). Compared to those born during the monsoon season, those born in summer have significantly higher odds of having high-risk overall blood pressure (significant in the basic model borderline significant in the full model) and high-risk pulse rate (significant in both the basic and full models). They also have higher odds of having high-risk systolic blood pressure and diastolic blood pressure (borderline significant in the basic models). The winter-born also have higher odds of having high-risk overall blood pressure (significant at the 10% level in the basic model and at the 5% level in the full model).

For the self-reported chronic conditions, the results are more mixed, although there is general support for a health advantage for individuals born during the monsoon season (**Table 3.10 Part B**). Compared to those born during the monsoon, those born in all other seasons have higher odds of reporting hypertension, angina (full model only), stroke (OR=3.38, M1; significant in the basic model only), chronic lung disease (OR=2.35, M1; significant in the basic model and borderline significant in the full model), and asthma (borderline significant in the full model only). However, they have lower odds of reporting angina (basic model only), diabetes (OR=0.58, M1; significant in the basic and full models), and arthritis. Models using a more detailed categorization for season of birth indicate that the summer-born are particularly disadvantaged in terms of chronic lung disease (significant in the basic and full models) (**Table 3.10 Part B**). The winter-born have higher odds of reporting stroke (significant at the 5% level in the basic model and at the 10% level in the full model) and lower odds of reporting diabetes relative to those born during the monsoon season (significant in the basic and full models). Those born

during the post-monsoon season have lower odds of reporting diabetes relative to those born during the monsoon season (significant only in the full model).

Thus, in India, those born during the monsoon appear to experience a health advantage for measured health outcomes and for some of the self-reported chronic conditions, particularly compared to those born during the summer. One potential explanation for the health advantage of those born during the monsoon season is that those born during the winter and summer seasons would have been exposed to the highest temperatures of the year and monsoon conditions shortly after birth. During the 1920s-1930s in India, cholera deaths peaked in the monsoon season, while plague deaths and smallpox cases clustered in the winter and summer months (**Figures 3.1-3.3**; Public Health Commissioner of India 1937).

II. Pre- and Postnatal Climate Conditions and Adult Morbidity in India

I now discuss results from models using climate conditions in an 18-month window around birth to predict measured and self-reported health outcomes in India. The main hypotheses being tested in these models is whether more rainfall and higher temperatures during the gestational and post-birth periods are associated with adult morbidity. Models are estimated separately for the gestational and post-birth periods. I present results from models based on the sample of individuals with complete data on all predictor variables of interest and who report always having lived in their current place of residence to ensure that the climate conditions correspond to those experienced by the respondents around the time of birth. In the summary of the results, I focus on the health outcomes hypothesized to most strongly connected to early life conditions (e.g., height, risk factors

for or types of cardiovascular disease, and respiratory disease) although all of the health outcomes are included in the results tables.

A. Rainfall Conditions and Adult Morbidity in India

Results from these models are summarized in **Table 3.11** and detailed in the following text. The main hypothesis being tested in these models is whether higher levels of rainfall in an 18-month window around birth are associated with adult morbidity in India.

Gestation

Barker's fetal origins hypothesis is the main theory regarding the effects of exposures experienced *in utero* and later life health outcomes. According to this theory, the main pathway connecting early life exposures and adult health is nutrition (although it has also been used in studies considering the long-term impacts of *in utero* exposure to the influenza pandemic), and the outcomes we would expect to be most strongly affected by adverse conditions during gestation are cardiovascular disease and height.

More rainfall during each of the three trimesters increases the odds that an individual falls below the 10^{th} and 25^{th} height percentiles (**Table 3.12 Part A**). For falling below the 10^{th} height percentile, the odds ratios for rainfall in the second trimester are borderline significant in the basic and full models, and the odds ratio for rainfall in the third trimester is borderline significant in the basic model. For falling below the 25^{th} height percentile, the odds ratios for rainfall in the second trimester are significant at the 5% level in the basic model and at the 10% level in the full model, and the odds ratio for rainfall in the third trimester is significant at the 5% level in the basic model.

Among the measured health outcomes, obesity, waist circumference, waist-to-hip ratio, and blood pressure are risk factors for cardiovascular disease. In general, higher amounts of rainfall during gestation appear to be favorable for these adult health outcomes (e.g., the odds ratios are less than one), although the results are sensitive to the high-risk cutpoints used for obesity and waist circumference. More rain in the third trimester is only a significant predictor of obesity (in both the basic and full models) when it is defined as ≥BMI of 25 kg/m^2. More rain in the first and third trimesters has the strongest association with high-risk waist circumference using the lowest cutpoint for waist circumference (specification c in **Table 3.12 Part A**). Finally, higher amounts of rainfall in the third trimester are associated with significantly lower odds of having high-risk systolic, diastolic, and overall blood pressure in the full models (the odds ratios are borderline significant in the basic models for diastolic and overall blood pressure).

Among the self-reported chronic conditions, hypertension, diabetes, angina, and stroke are risk factors for or types of cardiovascular disease. There is no clear pattern of advantage or disadvantage associated with higher rainfall levels for these health outcomes (**Table 3.12 Part B**). Significant associations are observed only for rainfall in the third trimester and hypertension in the full model and rainfall in the first trimester and diabetes in the full model, where higher rainfall levels are associated with lower odds of reporting these conditions.

Post-Birth

In the post-birth period, infectious diseases (and the synergy between infection and nutrition) are considered to be the most important early life exposures associated with

later life health outcomes. Thus, we may expect to see the strongest influences on height, a measure of childhood disease and nutritional conditions; chronic lung disease, which may be related to early respiratory infections; and the risk factors for and types of cardiovascular disease discussed above, which may result from chronic inflammation due to childhood infections.

Higher levels of rainfall in the post-birth period are generally unfavorable for the measured health outcomes (**Table 3.12 Part A**). Higher levels of rainfall during the first 1-3 months after birth are associated with significantly increased odds of falling below the 25^{th} height percentile in the basic model (OR=1.70, M1), and higher levels of rainfall during the first 7-9 months after birth are associated with significantly increased odds of falling below the 25^{th} height percentile in the basic and full models (OR=2.01, M1). Among the measured health outcomes that pertain to cardiovascular disease, the most consistent associations are observed for blood pressure and waist-to-hip ratio. Higher levels of rainfall during the first 1-3 months after birth (significant in the basic model only) and the first 7-9 months after birth (significant in the basic model, borderline significant in the full model) are associated with higher odds of having high-risk waist-to-hip ratio. Higher levels of rainfall during the first 4-6 months after birth are significantly associated with higher odds of having high-risk systolic, diastolic, and overall blood pressure in the basic models (odds ratios are borderline significant for diastolic and overall blood pressure in the full models). Rainfall during the post-birth period is only significantly associated with obesity defined as a BMI $\geq$30 kg/m^2 and is not significantly associated with any of the three waist circumference measures.

Among the self-reported chronic conditions, we may expect to see stronger associations between rainfall and chronic lung disease, hypertension, diabetes, angina, and stroke. Similar to the case for rainfall during the gestational period, we do not observe strong associations between rainfall during the post-birth period and self-reported chronic conditions (**Table 3.12 Part B**). The odds ratios for these health outcomes are not consistently less than or greater than one. The only significant association is observed between rainfall in the first 7-9 months after birth and angina in the basic model (OR=0.24, M1).

I also estimate models which include both season of birth and rainfall conditions as predictors of adult health outcomes (results not shown). In these models, season of birth is generally not a significant of measured health outcomes, and the associations between rainfall and adult morbidity are largely robust to the inclusion of season of birth as a predictor variable.

B. Temperature Conditions and Adult Morbidity in India

Results from models examining the associations between minimum temperature conditions in an 18-month window around birth and adult morbidity are summarized in **Table 3.13** and detailed in the following text. The main hypothesis being tested in these models is whether higher temperatures during the gestational and post-birth periods are associated with adult morbidity in India.

Gestation

Similar to the hypotheses for rainfall, we may expect temperatures in gestation to be most strongly related to cardiovascular disease and height. Among the measured health outcomes, the most consistent associations are observed for height and, to a lesser extent, blood pressure (**Table 3.14 Part A**). Higher temperatures during gestation are uniformly unfavorable for height (odds ratios for temperatures in all trimesters exceed one in both the basic and full models). Higher temperatures during the first and third trimesters are associated with significantly higher odds of falling below the 10^{th} height percentile in the basic models (significant at the 10% level in the full model for temperature in the first trimester and at the 5% level in the full model for temperature in the third trimester). Higher temperatures during the first and third trimesters are associated with significantly higher odds of falling below the 25^{th} height percentile in the basic models only. For blood pressure, there is some evidence that higher temperatures during the first trimester may be unfavorable for systolic and overall blood pressure (significant at the 5% level in the basic models only), but results are mixed for higher temperatures during the other trimesters and for diastolic blood pressure. Temperatures during gestation do not appear to be associated with waist-to-hip ratio or any of the measures of obesity or waist circumference.

Among the self-reported chronic conditions, there is weak support for the influence of temperature during gestation and conditions related to cardiovascular disease (**Table 3.14 Part B**). The odds ratios for temperature in all three trimesters exceed one for angina and stroke, although the odds ratios are only significant for first trimester temperature and stroke (significant in the basic model and borderline significant in the full model). The

results are more mixed for hypertension and diabetes, although higher temperatures during the first trimester are associated with significantly higher odds of reporting hypertension in the basic model only.

Post-Birth

In the post-birth period, we are most interested in the associations between temperature and height, chronic lung disease, and risk factors for and types of cardiovascular disease. As was the case with temperatures during gestation, temperatures during the post-birth period are most strongly associated with height and blood pressure among the measured health outcomes (**Table 3.14 Part A**). For these outcomes, the odds ratio for temperatures during all of the three post-birth periods exceed one. Higher temperatures during the first 7-9 months after birth are significantly associated with falling below the 10^{th} (OR=1.14, M1) and 25^{th} (OR=1.11, M1) height percentiles in both the basic and full models. The odds ratio for temperature during the first 1-3 months after birth is borderline significant for falling below the 25^{th} height percentile in the basic model only (OR=1.08, M1). For blood pressure, most, but not all, of the odds ratios for temperatures in the three post-birth periods are greater than one. Higher temperatures during the first 4-6 months after birth are associated with significantly higher odds of having high-risk overall blood pressure in the basic and full models (OR=1.05, M1). The odds ratios for temperature during the first 4-6 months after birth are significant and borderline significant in the basic models for systolic and diastolic blood pressure, respectively. Temperatures during the post-birth period do not appear to be associated with waist-to-hip ratio or any of the measures of obesity or waist circumference.

For the self-reported chronic conditions, it is unclear whether higher temperatures in the post-birth period are favorable or unfavorable for adult morbidity (**Table 3.14 Part B**). Temperature in any of the post-birth periods is not a significant of hypertension, diabetes, or angina, and there is no clear pattern to the odds ratios. However, higher temperatures in all three post-birth do appear to be associated with higher odds of reporting stroke. The results are significant for temperature in the first 4-6 months after birth in both the basic and full models (OR=1.16, M1) and borderline significant for temperature in the first 1-3 and 7-9 months after birth in the basic models. In contrast, higher temperatures appear to be associated with lower odds of reporting chronic lung disease. The odds ratios are significant for temperature in the first 7-9 months after birth (OR=0.83, M1) in both the basic and full models and borderline significant for temperature in the first 1-3 months after birth in the basic model only.

I also estimate models which include both season of birth and temperature conditions as predictors of adult morbidity (results not shown). In these models, season of birth is generally not a significant predictor of measured health outcomes, and the associations between temperature and adult morbidity are largely robust to the inclusion of season of birth as a predictor variable.

III. Climate Conditions in the First Five Years After Birth and Shocks in Pre- and Postnatal Climate Conditions and Adult Morbidity in India

I conducted a number of supplementary analyses examining whether climate conditions up to 60 months after birth are associated with adult health outcomes, and whether additional insights can be gleaned from investigating the impacts of pre- and postnatal

climate shocks on adult morbidity. This resulted in a very large set of results, in which the most consistent associations were generally observed for height and blood pressure measure. Instead of detailing all of these results, I discuss three cases that are best motivated by the theories and existing literature on early life exposures on adult health.

a. Rainfall Shocks During Gestation

During the gestational period, insufficient rain during the monsoon months is associated with worse adult health outcomes for all three blood pressure measures.[38] Insufficient rain is defined as when a given month fell during the monsoon season and when rainfall during that month fell below the 10^{th} percentile observed for that particular month and district over the period 1901-2002. The predictor variable of interest here is the fraction of each trimester for which monsoon rains were abnormally low (e.g., a failed monsoon), with possible values ranging from 0-1 in increments of 1/3.

Insufficient monsoon rains during all three trimesters are associated with higher odds of falling in the high-risk categories for systolic, diastolic, and overall blood pressure. The odds ratios are significant at the 5% level for all trimesters in the basic models except for insufficient monsoon rains during the first trimester, which is significant at the 10% level. The odds ratios are at least borderline significant for nearly all trimesters in the full models. The strongest effects are observed for insufficient monsoon rains during the third trimester. Experiencing one month of insufficient monsoon rains during the third trimester is significantly associated with two- to three-fold increases in the odds of having high-risk blood pressure (systolic: OR=3.04, M1; diastolic: OR=2.01, M1;

[38] Insufficient monsoon rains do not display consistent associations with other health outcomes.

overall: OR=2.84, M1). Given that failed monsoon rains are likely to have a substantial negative impact on the harvest and that the third trimester is considered by many to be the period of peak growth, these results are consistent with the Barker hypothesis that adverse nutritional conditions *in utero* may be related to an increased risk of cardiovascular disease in adulthood.

b. Rainfall Shocks in the First Five Years After Birth

Excess rain during the post-birth period is associated with worse adult health outcomes for height and diastolic blood pressure.[39] Excess rain experienced during the 1st year (1-12 months) and 3rd year (24-36 months) after birth is associated with worse adult health outcomes for diastolic blood pressure and height, respectively.[40] Each model uses five 12-month measures of excess rain to predict adult health outcomes, where the other controls in the basic and full models are as described above. Excess rain is defined as the fraction of each 12-month period when rainfall exceeded the 90th percentile of rainfall observed for the particular month and district over the period 1901-2002 (thus, the possible values range from 0-1 in increments of 1/12).

Excess rain during the 1st year is significantly associated with high-risk diastolic blood pressure in both the basic and full models. Experiencing rainfall above the 90th percentile in one month during the 1st year is associated a 29% increase in the odds of having high-risk diastolic blood pressure in the basic model (M1). Excess rain during the 3rd year is associated with higher odds of falling below the 10th (odds ratios are significant in the basic and full models) and 25th (odds ratios are borderline significant in the basic and full

[39] Excess rain during the post-birth does not display consistent associations with other health outcomes.
[40] Odds ratios for the other 12-month periods are not significant.

models) height percentiles. Experiencing rainfall above the 90th percentile in one month during the 3rd year is associated with a 39% increase in the odds of falling below the 10th height percentile in the basic model (M1). These results are highly similar to results from models that use total rainfall (rather than rainfall shocks) in each of the five 12-month periods to predict height and blood pressure in adulthood.[41]

While data on the duration of breastfeeding are scarce for the period when individuals in this sample were born, the earliest available estimates for India from the 1940s and 1950s suggest that breastfeeding lasted for approximately 2 years (see the Discussion section below for more detail). The associations between excess rainfall shocks in the 3rd year and height suggest that environmental conditions may take on increased importance during the weaning or immediate post-weaning period.

c. Temperature Shocks in the First Five Years After Birth

The third case considers the impact of exceptionally hot summers during the first 60 months after birth. In this case, a temperature shock is defined as occurring when the minimum temperature in a particular month and district exceeded the 90th percentile for minimum temperature observed for that month and district over the period 1901-2002. The predictor variable of interest here is the fraction of the summer months in a given year that were abnormally hot, with possible values ranging from 0-1 in increments of

[41] These models indicate that higher levels of rainfall in the first year are associated with higher odds of having high-risk diastolic blood pressure and that higher levels of rainfall in the first 25-36 months after birth are associated with higher odds of falling below the 10th and 25th height percentiles (results are at least borderline significant in all basic and full models).

1/3. Exceptionally hot summers in the 1st year (1-12 months) and 2nd year (13-24 months) after birth are associated with higher odds of falling below the lowest height percentiles.[42]

Exceptionally hot summers in the 2nd year after birth are associated with significantly higher odds of falling below the 10th height percentile in both the basic and full models. Exceptionally hot summers in the 1st year and 2nd year after birth are associated with higher odds of falling below the 25th height percentile (significant at the 5% and 10% levels only in the full models, respectively). Based on the existing literature, two pathways that are likely to connect high summer temperature shocks in first 24 months after birth and adult height are: (1) the contribution of higher summer temperatures to food spoilage and increased infections during the weaning period (e.g., diarrheal diseases) and (2) the contribution of higher summer temperatures to ideal conditions for the transmission of vector-borne diseases (e.g., malaria, dengue, and other mosquito-borne diseases).

Discussion

Researchers have become increasingly interested in the impacts of early life conditions on later life morbidity, socioeconomic outcomes, and mortality, motivated in part by growing importance of chronic disease as a major cause of morbidity and mortality worldwide. In general, studies have found support for long-term negative health consequences of pre- and postnatal exposure to infectious diseases, including the 1918 influenza pandemic. Findings from studies examining the effects of prenatal exposure to famines are mixed, with some studies finding no long-term impacts and others

[42] For height, odds ratios for the other 12-month periods are not significant. Excess summer temperatures during the post-birth period do not display consistent associations with other health outcomes.

documenting adverse impacts on adult morbidity and mortality. The main pathways theorized to connect these early life conditions and later life health outcomes are physiological changes that occur in response to unfavorable nutritional conditions *in utero* (developmental plasticity) and physiological scarring and chronic inflammation due to infectious diseases (Barker 1995; Elo and Preston 1992; Finch and Crimmins 2004). Among the outcomes hypothesized to be most strongly linked to early life exposures, the most consistent associations have been observed between early life conditions and later life height, cardiovascular disease, respiratory disease, and mortality.

Data on early life exposures are often scarce, leading some researchers to focus on season of birth as a proxy for environmental exposures that influence early life nutrition and exposure to infectious diseases. Several studies have documented season of birth patterns in developed countries indicating a longevity advantage for those born in the autumn relative to those born in all other seasons. To the best of my knowledge, however, no studies have systematically examined whether these season of birth patterns exist in developing countries.

In this chapter, I examined whether season of birth is associated with adult morbidity in six developing and newly industrialized countries: India, China, Ghana, Mexico, Russia, and South Africa. I also tested whether environmental exposures (rainfall and temperature) experienced around the time of birth have a direct influence on adult morbidity in India. Climate exposures are hypothesized to have strong effects on the infectious disease environment and nutrition early in life, particularly in developing countries.

Season of Birth and Adult Morbidity

Studies in developed countries (e.g., Doblhammer and Vaupel 2001, Doblhammer 2004, and Gavrilov and Gavrilova 2011) have documented an autumn-born longevity advantage. The health advantage of the autumn-born is hypothesized to be related to the harvest season and the availability of food, as maternal nutrition during the third trimester is generally expected to be better for those born in the autumn and early winter compared to those born in spring and early summer (Doblhammer 2004). In addition, the incidence of infectious disease varies seasonally.

In this chapter, I consider whether season of birth patterns in adult morbidity exist in developing countries. The findings from these analyses (summarized in **Table 3.4**) are inconclusive. I do not find evidence of strong season of birth patterns in adult morbidity in the six countries considered in these analyses. In these models, the coefficients rarely reach statistical significance and at times the effects are not in the expected direction. This may be related to limited power due to small sample sizes, the consideration of different adult health outcomes compared to earlier studies (e.g., morbidity versus mortality), or different processes operating in developing versus developed countries. Compared to the United States and other Western European countries for which an autumn-born longevity advantage has been documented, the countries considered in this chapter may have very different climates and seasonal patterns in disease incidence and agricultural production. It is possible that future studies may be able to further explore whether season of birth patterns exist in developing countries.

Pre- and Postnatal Rainfall and Temperature Conditions and Adult Morbidity

In the second set of analyses, I examine whether rainfall and temperature conditions during the pre- and post-birth periods affect adult health outcomes in India. These results are summarized in **Tables 3.11** and **3.13** for rainfall and temperature, respectively. I find that rainfall and temperature conditions around the time of birth are most strongly associated with adult height and blood pressure, two of the outcomes we would expect to be most related to early life exposures. Height is a measure of disease and nutritional conditions in childhood, and high blood pressure is a risk factor for cardiovascular disease. The results are more consistent for measured health outcomes than for self-reported chronic conditions. Supplementary cases provide support for the importance of environmental conditions influencing nutrition *in utero* and influencing the disease environment during the weaning period.

Rainfall

During the gestational period, higher amounts of rainfall appear to be somewhat beneficial for blood pressure but deleterious for height. The most consistent associations are observed between rainfall during the third trimester and adult morbidity. Although higher amounts of rainfall during gestation are associated with lower odds of high-risk blood pressure in the main models, supplementary analyses indicate that exposure to failed monsoon rains during gestation is associated with higher odds of high-risk blood pressure. Similarly, the effects are strongest for failed monsoon rains experienced during the third trimester, the period of peak fetal growth. Failed monsoon rains are likely to have a substantial negative impact on agricultural production. In 2002, for example, the

drought caused by failed monsoon rains resulted in a 7.2% contraction in the agricultural sector and a 2% decline in GDP growth ("Monsoon Blues" 2009). The finding that failed monsoon rains during gestation are associated with adverse later life outcomes for blood pressure is consistent with the hypothesis that adverse nutritional conditions *in utero* are related to an increased risk of cardiovascular disease in adulthood (Barker 1995).

During the post-birth period, higher levels of rainfall are consistently associated with worse health outcomes for height, blood pressure, and waist-to-hip ratio. One possible mechanism that may be operating here is that higher levels of rainfall contribute to greater infectious and parasitic disease exposure in early life, which in turn lead to greater adult morbidity through scarring and chronic inflammation. This possibility is supported by supplementary analyses examining the importance of climate conditions during an extended post-birth window, discussed below.

Temperature

With very few exceptions, higher temperatures around the time of birth are associated with worse adult health outcomes. During gestation, higher temperatures are associated with higher odds of having high-risk blood pressure and higher odds of falling below the lowest height percentiles. These associations are generally most consistent for conditions experienced during the first and third trimesters. It is possible that higher temperatures during gestation may influence maternal stress, infection, and nutrition.

During the post-birth period, higher temperatures are also associated with higher odds of having high-risk blood pressure and higher odds of falling below the lowest height percentiles. These associations are stronger in the later two post-birth periods (4-6 and 7-

9 months after birth) than in the earliest post-birth period. The most likely explanation for these associations are the contribution of higher temperatures to diarrheal disease and other gastrointestinal infections through food spoilage and to the spread of vector-borne diseases.

Temperature and Rainfall Shocks During the First Five Years After Birth

I also examine whether temperature and rainfall conditions during the first 60 months after birth are associated with adult health outcomes. I find that abnormally high levels of rainfall during the third year and abnormally high summer temperatures during the second year after birth are associated with significantly higher odds of falling below the lowest height percentiles. These findings provide suggestive evidence of the increased importance of environmental conditions during the weaning period.

Estimates of the duration of breastfeeding in India, especially for the period prior to the 1960s, are scarce. According to Visaria, Visaria, and Jain (1995), even at present, few surveys in India have collected information on breastfeeding. However, the majority of the literature indicates that up until very recently, breastfeeding in India was considered to be universal and quite lengthy in duration, typically lasting up until the next pregnancy (Jain and Adlakha 1982; Nath, Land, and Singh 1994; Visaria 2004; Visaria, Visaria, and Jain 1995). The earliest period estimates suggest that the average duration of breastfeeding was approximately two years. Based on observation of a village in Karnataka, Caldwell, Reddy, and Caldwell (1982) state that “in the 1940s, only women who had no milk or were very sick failed to breastfeed for two years, and many did so for three to five years” (694). Other estimates from 1958 and 1959, the first based on patients

in a birth control clinic in Bombay and the second on 11 villages in Punjab, placed the mean duration of lactation at 21 and 19.8 months, respectively (Peters et al. 1958; Potter et al. 1965). One study from 1957-1969 found the average duration of breastfeeding to be 28 months (Rao and Mathen 1970).

Given that the average duration of breastfeeding has tended to decline over time in developing countries, estimates from more recent periods may serve as lower bounds. From their review of data from six studies covering both rural and urban areas in several regions of India conducted during the 1960s and 1970s, Jain and Adlakha (1982) concluded that the average duration of breastfeeding during the 1970s was 20-22 months. Estimates of the average duration of breastfeeding from the six studies ranged from 19.6-30.3 months (Ibid.). A 1967 study based in rural Uttar Pradesh estimated the average duration of breastfeeding to be 22.6 months (Visaria, Visaria, and Jain 1995). Estimates from 1981-1982 suggest that breastfeeding lasted 21.8 and 22.9 months in urban and rural areas of Rajasthan, respectively, and 23.8 and 28.6 months in urban and rural areas of Orissa, respectively (Ibid.).

More recently, data from a 1992 survey indicated that children in rural West Bengal and an urban slum in Calcutta were breastfed for 12-15 and 20-23 months, respectively (Sen and Biswas 1993). Even through the late 1990s, the duration of breastfeeding seems to have been quite long. Visaria (2004) estimated the median duration of breastfeeding to be 25.4 months. Although urban women had shorter durations of breastfeeding than rural women, on average the median duration of breastfeeding among urban women was still in excess of 21 months. Data from the National Family Health Survey 1998-9 indicate

that for the six states included in this analysis, estimates of the duration of breastfeeding ranged from 20 months in Karnataka to 36 months in Assam (Ibid.).

Since breastfeeding is combined with the introduction of solid foods and other supplements as the child ages, ideally we would like to have estimates of the average duration of exclusive breastfeeding. According to Visaria (1988), "Women continue to feed their children for up to 24-36 months, and do not introduce supplementary foods very systematically," although supplementary milk (diluted with water) may be introduced around 6 months (86). A study in 1982 of low-income women in Hyderabad found that the total duration of lactation was 21.4 months, but exclusive breastfeeding lasted for 8.9 months (Prema and Ravindranath 1982).

Overall, these studies suggest that breastfeeding may have lasted for roughly 2 years for the individuals in my sample. With regards to the findings from my analyses, it is striking that rainfall and temperature conditions in the weaning or post-weaning period (in the second and third years after birth) show strong associations with height, a marker of infectious disease and nutritional conditions during childhood. The associations observed between rainfall and temperature conditions during the 4-6 month and 7-9 month post-birth periods and blood pressure and height in adulthood may reflect the end of exclusive breastfeeding, the introduction of weaning foods, and waning protection from maternal antibodies, all of which would result in increased susceptibility to infectious and parasitic diseases in childhood. For example, Motarjemi et al. (1993) noted the relationship between higher ambient temperatures and the contamination of weaning foods, which in turn are strongly associated with diarrheal disease and malnutrition among children. In addition, based on longitudinal studies conducted in Matlab, Bangladesh, Black et al.

(1982) found that 41% of samples of food fed to children of weaning age and 50% of drinking water specimens contained *E. coli*. The proportion of children's food samples containing *E. coli* was strongly associated with incidence of diarrheal disease, and higher *E. coli* levels were related to cooked foods stored at higher temperatures (Ibid.).

Robustness of the Results

In general, results summarized above are robust to the inclusion of controls for district of residence, education, and father's occupation and education (all models control for age and sex). Additional analyses indicate that the findings are also robust to controlling for race in South Africa and religion in each of the six countries. For countries located in the Northern Hemisphere, autumn is defined as occurring from September-November. Results from models in which autumn is defined as occurring from October-December are highly similar to those presented here.

Although I also explored whether interactions between father's occupation and season of birth and interactions between father's occupation and climate conditions around the time of birth are significant predictors of adult morbidity, the results from these models did not show a clear pattern. In addition, these interactions rarely reached statistical significance.

Limitations

There are a number of important limitations to this study. First, I necessarily rely on the accuracy of respondents' reporting of their month and year of birth, and age misreporting may be an issue for these samples. In addition, the proportions of the total samples who report their month of birth vary by country and may result in a select sample (e.g., a

lower fraction of women than men report their month of birth in India and Ghana). However, it is unlikely that nonresponse to the month of birth question varies systematically by month of birth (e.g., individuals born in June should not be any less likely to report their month of birth than individuals born in December). Measurement error in month of birth is thus expected to be mean zero; generally, this should result in attenuation bias and the estimates may be considered conservative (Stefanski and Carroll 1985).

In addition, it is uncertain how and to what extent these patterns may be influenced by selective survival to age 50.[43] For example, if we expect certain seasons to be particularly unfavorable for infant survival and survival to age 50, we may expect that individuals who were born in those seasons and who survive to age 50 may be particularly hardy. In contrast, if being born in certain seasons is health-promoting, the group of individuals who were born in those seasons and who survive to age 50 may be more heterogeneous in terms of frailty. However, given that being born in autumn is considered to be most favorable for early *and* later life health outcomes, we would expect the group of autumn-born survivors to be more heterogeneous than the hardier survivors born in other seasons and that any health advantage for the autumn-born should be muted at older ages. However, I still observe a consistent health advantage in adult morbidity for the autumn-born in most countries. It is also possible that there may be countervailing effects which cancel out on average (e.g., if both acquired immunity and scarring are operating), resulting in null findings.

[43] The month of birth distributions for the six analytical samples in the season of birth models are shown in **Figure A3.1**.

Finally, data limitations result in fairly small sample sizes for some countries (e.g., Ghana, Mexico). Although many of the associations are in the hypothesized direction, they do not always reach significance. It may be the case that there is insufficient power to detect more robust relationships between the predictor variables of interest and health outcomes.

Conclusion

In this chapter, I examined the associations between early life conditions, captured by season of birth and climate conditions experienced around the time of birth, and both measured and self-reported adult health outcomes. In the first part of the analysis, I do not find strong season of birth patterns in adult morbidity in the six developing and newly industrialized countries considered in this chapter (China, Ghana, India, Mexico, Russia, and South Africa).

In the second part of the analysis, I find evidence that both pre- and postnatal rainfall and temperature conditions are associated with adult morbidity in India. The most consistent associations are observed for height and blood pressure, two adult health outcomes we would expect to be most strongly related to early life exposures based on the existing literature. While more rainfall is associated with both beneficial and deleterious adult health outcomes, higher temperatures are nearly always associated with worse adult health outcomes. Furthermore, supplementary analyses examining rainfall and temperature shocks and extending the post-birth observation window through 5 years support the importance of nutritional conditions during gestation and infectious disease exposure during the weaning period.

Tables

Table 3.1. Four-Category Season of Birth Classifications

	China, Ghana, Mexico, Russia	South Africa	India
January	Winter	Summer	Winter
February			
March	Spring	Autumn	Summer
April			
May			
June	Summer	Winter	Monsoon
July			
August			
September	Autumn	Spring	
October			Post Monsoon
November			
December	Winter	Summer	

Table 3.2. Hypotheses for the Associations Between Season of Birth and Environmental Exposures and Adult Health

Variable of Interest	**Expected Association with Adult Health**
Season of Birth (China, Ghana, India, Mexico, Russia, South Africa)	
Autumn vs. All other	Better
Monsoon vs. All other	Indeterminate
Environmental Exposures (India)	
I. Rainfall	
a. Higher levels	Indeterminate
b. Shocks	
$\geq 90^{th}$ percentile during monsoon	Worse
$\leq 10^{th}$ percentile during monsoon	Worse
II. Minimum temperature	
a. Higher levels	Worse
b. Shocks	
$\geq 90^{th}$ percentile during summer	Worse

Table 3.3. Measured Health Outcomes and High-Risk Cutpoints

Measure	High-Risk Cutpoint	Source
Body mass index (BMI)[44]	a. BMI ≥ 30 kg/m^2	1
India	b. BMI ≥ 25 kg/m^2	2
	c. BMI ≥ 23 kg/m^2	3
Waist circumference	a. > 102 cm (M), > 88 cm (F)	4
India	b. ≥ 90 cm (M), ≥ 80 cm (F)	2
	c. ≥ 87 cm (M), ≥ 82 cm (F)	3
Waist-to-hip ratio	> 0.90 (M), > 0.85 (F)	5
Systolic blood pressure[25]	> 140 mm Hg	6
Diastolic blood pressure[45]	> 90 mm Hg	6
Blood pressure[25]	> 140 mm Hg systolic and > 90 mm Hg diastolic	6
Pulse rate[25]	≥ 90 beats/min	5
Height, ≤10th percentile	China: ≤ 1.555 m (M), 1.46 m (F)	
	Ghana: ≤ 1.55 m (M), ≤ 1.49 m (F)	
	India: ≤ 1.545 m (M), ≤ 1.418 m (F)	
	Mexico: ≤ 1.568 m (M), ≤ 1.426 m (F)	
	Russia: ≤ 1.62 m (M), ≤ 1.52 m (F)	
	South Africa: ≤ 1.56 m (M), ≤ 1.49 m (F)	
Height, ≤25th percentile	China: ≤ 1.6 (M) m (M), ≤ 1.5 m (F)	
	Ghana: ≤ 1.61 m (M), ≤ 1.533 m (F)	
	India: ≤ 1.587 m (M), ≤ 1.461 m (F)	
	Mexico: ≤ 1.593 m (M), ≤ 1.443 m (F)	
	Russia: ≤ 1.68 m (M), ≤ 1.55 m (F)	
	South Africa: ≤ 1.5 m (M), ≤ 1.44 m (F)	

Sources: (1) WHO (2013); (2) WHO (2000) and Misra et al. (2006); (3) Mohan et al. (2007); (4) NHLBI; (5) Seeman et al. (2008); (6) Crimmins et al. (2005).

[44] Calculated from respondents' measured height and weight.
[45] Calculated from the average of three measurements taken.

Table 3.4. Summary of Results from Logistic Regression Models Using Season of Birth (Autumn vs. All other or Monsoon vs. All other) to Predict Measured Health Outcomes (Part A) and Self-Reported Chronic Conditions (Part B), Males and Females Aged 50+, India (2007-2008)

Part A: Measured Health Outcomes

		High-Risk						Height	
	Obese	Waist Circ.	WHR	Systolic BP	Diastolic BP	BP	Pulse	≤10th Percentile	≤25th Percentile
South Africa								✓	
Ghana					✓				
Russia									
Mexico									✓
China									
India						✓			

Part B: Self-Reported Chronic Conditions

	Hypertension	Diabetes	Angina	Stroke	CLD	Arthritis	Asthma
South Africa							
Ghana					~~		
Russia		✓	✓				
Mexico			~~		✓		~~
China							
India				✓			

Notes: WHR refers to waist-to-hip ratio, BP refers to blood pressure, CLD refers to chronic lung disease. Shaded boxes refer to support for a health advantage for those born in the autumn or monsoon seasons relative to those born in all other seasons (OR>1) and check marks indicate that these associations are significant at $p<0.05$ in the basic models (M1). Crossed out boxes indicate that models were not run for a particular health outcome due to very low incidence of the health outcome in that sample.

Table 3.5. Odds Ratios and 95% Confidence Intervals from Logistic Regression Models Predicting Measured Health Outcomes (Part A) and Self-Reported Chronic Conditions (Part B), Males and Females Aged 50+, South Africa (2007-2008)

Part A	Season of Birth (ref=Autumn)		*N*
Obese (BMI ≥ 30 kg/m^2)			
M1	1.22	[0.85,1.75]	1,331
M5	1.18	[0.83,1.70]	1,331
High-Risk Waist Circumference			
M1	1.02	[0.68,1.52]	1,331
M5	1.06	[0.72,1.55]	1,331
High-Risk Waist-to-Hip Ratio			
M1	1.32	[0.90,1.93]	1,331
M5	1.29	[0.88,1.90]	1,331
High-Risk Systolic Blood Pressure			
M1	1.06	[0.66,1.72]	1,331
M5	1.03	[0.63,1.68]	1,331
High-Risk Diastolic Blood Pressure			
M1	1.25	[0.81,1.93]	1,331
M5	1.17	[0.77,1.79]	1,331
High-Risk Blood Pressure			
M1	1.31	[0.85,2.02]	1,331
M5	1.28	[0.83,1.98]	1,331
High-Risk Pulse Rate			
M1	1.47	[0.89,2.42]	1,331
M5	1.48	[0.88,2.49]	1,331
Height, ≤ 10th Percentile			
M1	1.96*	[1.06,3.61]	1,331
M5	2.16*	[1.14,4.09]	1,331
Height, ≤ 25th Percentile			
M1	1.34	[0.88,2.03]	1,331
M5	1.25	[0.82,1.90]	1,331

Part B	Season of Birth (ref=Autumn)		*N*
Hypertension			
M1	0.85	[0.57,1.25]	1,331
M5	0.80	[0.54,1.18]	1,331
Diabetes			
M1	1.39	[0.69,2.78]	1,331
M5	1.22	[0.60,2.51]	1,331
Angina			
M1	0.76	[0.31,1.85]	1,331
M5	0.72	[0.32,1.63]	1,331
Stroke			
M1	0.88	[0.36,2.19]	1,331
M5	0.65	[0.28,1.51]	1,331
Chronic Lung Disease			
M1	0.94	[0.26,3.36]	1,331
M5	0.92	[0.23,3.62]	1,331
Arthritis			
M1	1.38	[0.87,2.18]	1,331
M5	1.18	[0.71,1.95]	1,331
Asthma			
M1	1.10	[0.45,2.68]	1,331
M5	1.05	[0.43,2.56]	1,331

+ p <0.10, * p < 0.05, ** p < 0.01, *** p < 0.001

Notes: Model 1 includes controls for age and sex. Model 5 adds controls for region, father's education, father's occupation, and respondent's education. See **Table 3.3** for the high-risk cutpoints.

Table 3.6. Odds Ratios and 95% Confidence Intervals from Logistic Regression Models Predicting Measured Health Outcomes (Part A) and Self-Reported Chronic Conditions (Part B), Males and Females Aged 50+, Ghana (2007-2008)

Part A	Season of Birth (ref=Autumn)		*N*	**Part B**	Season of Birth (ref=Autumn)		*N*
Obese (BMI ≥ 30 kg/m^2)				*Hypertension*			
M1	1.44	[0.80,2.62]	838	M1	1.21	[0.77,1.91]	838
M5	1.25	[0.68,2.29]	838	M5	1.05	[0.64,1.75]	838
High-Risk Waist Circumference				*Diabetes*			
M1	1.20	[0.74,1.96]	838	M1	0.79	[0.34,1.80]	838
M5	1.01	[0.63,1.61]	838	M5	0.70	[0.33,1.49]	838
High-Risk Waist-to-Hip Ratio				*Angina*			
M1	0.85	[0.58,1.26]	838	M1	0.47	[0.14,1.61]	838
M5	0.79	[0.52,1.20]	838	M5	0.47	[0.14,1.55]	838
High-Risk Systolic Blood Pressure				*Stroke*			
M1	1.19	[0.84,1.69]	838	M1	0.97	[0.30,3.08]	838
M5	1.09	[0.75,1.57]	838	M5	0.91	[0.28,2.94]	838
High-Risk Diastolic Blood Pressure				*Chronic Lung Disease*			
M1	1.46*	[1.06,2.01]	838	M1			
M5	1.38+	[0.99,1.92]	838	M5			
High-Risk Blood Pressure				*Arthritis*			
M1	1.26	[0.90,1.76]	838	M1	0.86	[0.54,1.37]	838
M5	1.16	[0.82,1.64]	838	M5	0.84	[0.52,1.35]	838
High-Risk Pulse Rate				*Asthma*			
M1	0.97	[0.55,1.72]	838	M1	0.95	[0.38,2.40]	838
M5	1.06	[0.60,1.86]	838	M5	1.14	[0.42,3.10]	838
Height, ≤ 10th Percentile							
M1	0.98	[0.46,2.09]	838				
M5	1.09	[0.53,2.26]	838				
Height, ≤ 25th Percentile							
M1	0.99	[0.66,1.49]	838				
M5	1.02	[0.68,1.52]	838				

+ p <0.10, * p < 0.05, ** p < 0.01, *** p < 0.001

Notes: Model 1 includes controls for age and sex. Model 5 adds controls for region, father's education, father's occupation, and respondent's education. See **Table 3.3** for the high-risk cutpoints. Chronic lung disease is omitted due to very low incidence of this condition in this sample.

Table 3.7. Odds Ratios and 95% Confidence Intervals from Logistic Regression Models Predicting Measured Health Outcomes (Part A) and Self-Reported Chronic Conditions (Part B), Males and Females Aged 50+, Russia (2007-2010)

Part A	Season of Birth (ref=Autumn)		*N*
Obese (BMI ≥ 30 kg/m^2)			
M1	0.89	[0.45,1.74]	2,260
M5	0.98	[0.55,1.78]	2,260
High-Risk Waist Circumference			
M1	0.56+	[0.31,1.01]	2,260
M5	0.56+	[0.30,1.03]	2,260
High-Risk Waist-to-Hip Ratio			
M1	0.78	[0.40,1.55]	2,260
M5	0.78	[0.38,1.60]	2,260
High-Risk Systolic Blood Pressure			
M1	0.99	[0.61,1.62]	2,260
M5	1.01	[0.65,1.57]	2,260
High-Risk Diastolic Blood Pressure			
M1	1.08	[0.74,1.58]	2,260
M5	1.16	[0.80,1.67]	2,260
High-Risk Blood Pressure			
M1	0.82	[0.49,1.37]	2,260
M5	0.86	[0.53,1.39]	2,260
High-Risk Pulse Rate			
M1	1.18	[0.44,3.16]	2,260
M5	1.37	[0.55,3.43]	2,260
Height, ≤ 10th Percentile			
M1	1.17	[0.50,2.71]	2,260
M5	1.12	[0.54,2.31]	2,260
Height, ≤ 25th Percentile			
M1	1.56+	[0.94,2.59]	2,260
M5	1.53+	[0.96,2.45]	2,260

Part B	Season of Birth (ref=Autumn)		*N*
Hypertension			
M1	1.14	[0.57,2.26]	2,260
M5	1.24	[0.64,2.41]	2,260
Diabetes			
M1	1.84**	[1.25,2.70]	2,260
M5	2.06**	[1.27,3.36]	2,260
Angina			
M1	1.81*	[1.08,3.03]	2,260
M5	1.89*	[1.16,3.09]	2,260
Stroke			
M1	3.32+	[0.95,11.56]	2,260
M5	3.53*	[1.18,10.58]	2,260
Chronic Lung Disease			
M1	1.13	[0.49,2.59]	2,260
M5	1.22	[0.68,2.19]	2,260
Arthritis			
M1	0.96	[0.55,1.68]	2,260
M5	0.93	[0.52,1.67]	2,260
Asthma			
M1	0.52	[0.13,2.10]	2,260
M5	0.78	[0.32,1.87]	2,260

+ p <0.10, * p < 0.05, ** p < 0.01, *** p < 0.001

Notes: Model 1 includes controls for age and sex. Model 5 adds controls for region, father's education, father's occupation, and respondent's education. See **Table 3.3** for the high-risk cutpoints.

Table 3.8. Odds Ratios and 95% Confidence Intervals from Logistic Regression Models Predicting Measured Health Outcomes (Part A) and Self-Reported Chronic Conditions (Part B), Males and Females Aged 50+, Mexico (2009-2010)

Part A	Season of Birth (ref=Autumn)		*N*
Obese (BMI ≥ 30 kg/m^2)			
M1	1.73	[0.73,4.12]	937
M5	1.71	[0.86,3.39]	937
High-Risk Waist Circumference			
M1	1.55	[0.68,3.52]	937
M5	1.67+	[0.93,2.99]	937
High-Risk Waist-to-Hip Ratio			
M1	0.90	[0.41,2.00]	937
M5	1.10	[0.49,2.51]	937
High-Risk Systolic Blood Pressure			
M1	0.57	[0.22,1.43]	937
M5	0.80	[0.36,1.74]	937
High-Risk Diastolic Blood Pressure			
M1	1.22	[0.52,2.85]	937
M5	1.33	[0.72,2.45]	937
High-Risk Blood Pressure			
M1	1.40	[0.62,3.17]	937
M5	1.59	[0.88,2.88]	937
High-Risk Pulse Rate			
M1	0.99	[0.19,5.17]	937
M5	0.87	[0.29,2.57]	937
Height, ≤ 10th Percentile			
M1	2.01	[0.70,5.83]	937
M5	2.47	[0.57,10.71]	937
Height, ≤ 25th Percentile			
M1	2.53*	[1.07,5.98]	937
M5	2.39+	[0.89,6.37]	937

Part B	Season of Birth (ref=Autumn)		*N*
Hypertension			
M1	1.29	[0.54,3.07]	937
M5	1.37	[0.73,2.57]	937
Diabetes			
M1	0.41*	[0.18,0.94]	937
M5	0.23***	[0.10,0.55]	937
Angina			
M1			
M5			
Stroke			
M1	0.59	[0.16,2.21]	937
M5	0.43+	[0.17,1.06]	937
Chronic Lung Disease			
M1	6.87**	[1.79,26.45]	937
M5	8.37*	[1.42,49.41]	937
Arthritis			
M1	0.51	[0.17,1.52]	937
M5	0.66	[0.31,1.42]	937
Asthma			
M1			
M5			

+ p <0.10, * p < 0.05, ** p < 0.01, *** p < 0.001

Notes: Model 1 includes controls for age and sex. Model 5 adds controls for region, father's education, father's occupation, and respondent's education. See **Table 3.3** for the high-risk cutpoints. Angina and asthma are omitted due to very low incidence of these conditions in this sample.

Table 3.9. Odds Ratios and 95% Confidence Intervals from Logistic Regression Models Predicting Measured Health Outcomes (Part A) and Self-Reported Chronic Conditions (Part B), Males and Females Aged 50+, China (2007-2010)

Part A	Season of Birth (ref=Autumn)		*N*
Obese (BMI ≥ 30 kg/m^2)			
M1	0.80+	[0.62,1.04]	5,975
M5	0.79+	[0.60,1.04]	5,975
High-Risk Waist Circumference			
M1	0.87+	[0.74,1.02]	5,975
M5	0.86+	[0.74,1.01]	5,975
High-Risk Waist-to-Hip Ratio			
M1	1.13	[0.96,1.34]	5,975
M5	1.14	[0.96,1.35]	5,975
High-Risk Systolic Blood Pressure			
M1	0.80**	[0.70,0.92]	5,975
M5	0.79**	[0.69,0.91]	5,975
High-Risk Diastolic Blood Pressure			
M1	0.90+	[0.82,1.00]	5,975
M5	0.92+	[0.84,1.01]	5,975
High-Risk Blood Pressure			
M1	0.83***	[0.75,0.92]	5,975
M5	0.84**	[0.76,0.93]	5,975
High-Risk Pulse Rate			
M1	0.93	[0.75,1.17]	5,975
M5	0.94	[0.75,1.18]	5,975
Height, ≤ 10th Percentile			
M1	0.88	[0.69,1.13]	5,975
M5	0.89	[0.69,1.14]	5,975
Height, ≤ 25th Percentile			
M1	0.96	[0.81,1.13]	5,975
M5	0.95	[0.81,1.13]	5,975

Part B	Season of Birth (ref=Autumn)		*N*
Hypertension			
M1	0.89*	[0.80,1.00]	5,975
M5	0.90+	[0.80,1.00]	5,975
Diabetes			
M1	0.90	[0.70,1.15]	5,975
M5	0.90	[0.70,1.15]	5,975
Angina			
M1	0.94	[0.77,1.16]	5,975
M5	0.93	[0.77,1.13]	5,975
Stroke			
M1	1.10	[0.72,1.67]	5,975
M5	1.09	[0.71,1.66]	5,975
Chronic Lung Disease			
M1	1.19	[0.93,1.51]	5,975
M5	1.18	[0.93,1.51]	5,975
Arthritis			
M1	1.03	[0.85,1.25]	5,975
M5	1.04	[0.86,1.26]	5,975
Asthma			
M1	0.77	[0.48,1.25]	5,975
M5	0.74	[0.46,1.18]	5,975

+ p <0.10, * p < 0.05, ** p < 0.01, *** p < 0.001

Notes: Model 1 includes controls for age and sex. Model 5 adds controls for region, father's education, father's occupation, and respondent's education. See **Table 3.3** for the high-risk cutpoints.

Table 3.10. Odds Ratios and 95% Confidence Intervals from Logistic Regression Models Predicting Measured Health Outcomes (Part A) and Self-Reported Chronic Conditions (Part B), Males and Females Aged 50+, India (2007-2008)

Part A	Season of Birth (ref=Monsoon)		*N*
Obese (BMI ≥ 30 kg/m^2)			
M1	1.09	[0.48,2.50]	1,376
M5	1.21	[0.51,2.85]	1,376
Obese (BMI ≥ 25 kg/m^2)			
M1	1.06	[0.70,1.62]	1,376
M5	1.04	[0.66,1.66]	1,376
Obese (BMI ≥ 23 kg/m^2)			
M1	0.92	[0.64,1.33]	1,376
M5	0.88	[0.61,1.28]	1,376
High-Risk Waist Circumference[a]			
M1	1.16	[0.68,2.00]	1,376
M5	1.27	[0.76,2.14]	1,376
High-Risk Waist Circumference[b]			
M1	0.90	[0.59,1.37]	1,376
M5	0.87	[0.57,1.31]	1,376
High-Risk Waist Circumference[c]			
M1	0.95	[0.62,1.44]	1,376
M5	0.97	[0.63,1.49]	1,376
High-Risk Waist-to-Hip Ratio			
M1	1.10	[0.73,1.67]	1,376
M5	1.02	[0.68,1.52]	1,376
High-Risk Systolic Blood Pressure			
M1	1.41+	[1.00,1.99]	1,376
M5	1.28	[0.92,1.78]	1,376
High-Risk Diastolic Blood Pressure			
M1	1.42+	[0.95,2.11]	1,376
M5	1.39+	[0.96,2.00]	1,376
High-Risk Blood Pressure			
M1	1.59*	[1.11,2.30]	1,376
M5	1.47*	[1.04,2.10]	1,376
High-Risk Pulse Rate			
M1	1.55+	[0.98,2.45]	1,376
M5	1.57+	[0.99,2.47]	1,376

Part B	Season of Birth (ref=Monsoon)		*N*
Hypertension			
M1	1.24	[0.83,1.84]	1,376
M5	1.14	[0.80,1.63]	1,376
Diabetes			
M1	0.58*	[0.35,0.96]	1,376
M5	0.49*	[0.28,0.85]	1,376
Angina			
M1	0.96	[0.55,1.65]	1,376
M5	1.12	[0.66,1.92]	1,376
Stroke			
M1	3.38*	[1.07,10.67]	1,376
M5	2.96	[0.80,10.91]	1,376
Chronic Lung Disease			
M1	2.35+	[0.90,6.14]	1,376
M5	2.35*	[1.02,5.42]	1,376
Arthritis			
M1	0.82	[0.53,1.27]	1,376
M5	0.73	[0.47,1.11]	1,376
Asthma			
M1	1.48	[0.75,2.90]	1,376
M5	1.74+	[0.92,3.32]	1,376

Height, ≤ 10th Percentile			
M1	1.41	[0.76,2.60]	1,376
M5	1.36	[0.75,2.47]	1,376
Height, ≤ 25th Percentile			
M1	1.13	[0.75,1.72]	1,376
M5	1.08	[0.72,1.61]	1,376

+ p <0.10, * p < 0.05, ** p < 0.01, *** p < 0.001

Notes: Model 1 includes controls for age and sex. Model 5 adds controls for region, father's education, father's occupation, and respondent's education. See **Table 3.3** for the high-risk cutpoints.

[a] > 102 cm (males), > 88 cm (females)

[b] ≥ 90 cm (males), ≥ 80 cm (females)

[c] ≥ 87 cm (males), ≥ 82 cm (females)

Table 3.11. Summary of Results from Logistic Regression Models Using Rainfall in Gestation (Part A) and the Post-Birth Period (Part B) to Predict Measured Health Outcomes and Self-Reported Chronic Conditions, Males and Females Aged 50+, India (2007-2008)

Part A: Gestational Period

	Measured Health Outcomes								Self-Reported Chronic Conditions				
		High-Risk					Height						
	Obese	WC	WHR	SBP	DBP	BP	$\leq 10^{th}$	$\leq 25^{th}$	Hypertension	Diabetes	Angina	Stroke	
1^{st} Trim.	(✓)												
2^{nd} Trim.							(X)	(X)					
3^{rd} Trim.					(✓)	(✓)	(X)	X					

Part B: Post-Birth Period

	Measured Health Outcomes								Self-Reported Health Outcomes				
		High-Risk					Height						
	Obese	WC	WHR	SBP	DBP	BP	$\leq 10^{th}$	$\leq 25^{th}$	Hypertension	Diabetes	Angina	Stroke	CLD
1-3 Months			X					X					
4-6 Months	✓			X	X	X						(X)	
7-9 Months			X					X			✓		

Notes: WC refers to waist circumference, WHR refers to waist-to-hip ratio, SBP refers to systolic blood pressure, DBP refers to diastolic blood pressure, BP refers to overall blood pressure, and CLD refers to chronic lung disease. Height measures refer to falling below the 10^{th} and 25^{th} height percentiles. ✓'s indicate that higher levels of rainfall are associated with a significant health advantage (OR<1) and X's indicate that higher levels of rainfall are associated with a significant health disadvantage (OR>1) for a particular health outcome. Parentheses indicate that the odds ratios are significant at $p<0.10$, otherwise ✓'s and X's indicate that odds ratios are significant at $p<0.05$ in the basic models (M1). For obesity and waist circumference, this table summarizes results from models using the standard cutpoints.

Table 3.12. Odds Ratios from Logistic Regression Models Using Total Rainfall (m) to Predict Measured Health Outcomes (Part A) and Self-Reported Chronic Conditions (Part B), Males and Females Aged 50+, India (2007-2008)

Part A: Measured Health Outcomes									
	Gestation					Post-Birth			
	1st Trimester	2nd Trimester	3rd Trimester	*N*		1-3 Months	4-6 Months	7-9 Months	*N*
Obese (BMI ≥30 kg/m^2)					*Obese (BMI ≥30 kg/m^2)*				
M1	0.22+	0.45	0.52	1044	M1	1.46	0.20*	0.64	1044
M5	0.25	0.44	0.55	1044	M5	1.55	0.22*	0.68	1044
Obese (BMI ≥25 kg/m^2)					*Obese (BMI ≥25 kg/m^2)*				
M1	0.70	1.01	0.44*	1044	M1	1.02	0.95	0.88	1044
M5	0.66	1.04	0.41*	1044	M5	1.01	1.03	0.92	1044
Obese (BMI ≥23 kg/m^2)					*Obese (BMI ≥23 kg/m^2)*				
M1	1.02	1.01	0.91	1044	M1	1.19	0.98	1.04	1044
M5	1.15	1.02	0.97	1044	M5	1.23	1.11	1.05	1044
High-Risk Waist Circumference[a]					*High-Risk Waist Circumference*[a]				
M1	0.59	0.77	0.57	1044	M1	1.12	0.69	0.68	1044
M5	0.64	0.71	0.54	1044	M5	0.97	0.85	0.62	1044
High-Risk Waist Circumference[b]					*High-Risk Waist Circumference*[b]				
M1	0.44*	1.18	0.52+	1044	M1	1.16	0.58+	1.19	1044
M5	0.51+	1.27	0.58	1044	M5	1.41	0.72	1.59	1044
High-Risk Waist Circumference[c]					*High-Risk Waist Circumference*[c]				
M1	0.48*	1.2	0.49*	1044	M1	1.00	0.72	1.18	1044
M5	0.51*	1.24	0.47*	1044	M5	1.05	0.92	1.42	1044
High-Risk Waist-to-Hip Ratio					*High-Risk Waist-to-Hip Ratio*				
M1	0.85	1.40	0.94	1044	M1	1.82*	0.81	2.68*	1044
M5	0.56	1.23	0.65	1044	M5	1.66	0.75	2.36+	1044

High-Risk Systolic Blood Pressure				
M1	1.25	1.21	0.66	1044
M5	0.86	0.94	0.46*	1044
High-Risk Diastolic Blood Pressure				
M1	1.23	1.19	0.57+	1044
M5	0.83	1.05	0.39*	1044
High-Risk Blood Pressure				
M1	1.11	1.28	0.49+	1044
M5	0.76	1.02	0.34*	1044
High-Risk Pulse Rate				
M1	1.26	0.61	1.00	1044
M5	1.37	0.73	1.13	1044
Height, ≤10th Percentile				
M1	1.38	2.18+	1.72+	1044
M5	1.12	2.82+	1.44	1044
Height, ≤25th Percentile				
M1	1.70	1.69+	1.75*	1044
M5	1.43	2.04*	1.55	1044

High-Risk Systolic Blood Pressure				
M1	1.20	1.55*	1.44	1044
M5	1.00	1.49	1.04	1044
High-Risk Diastolic Blood Pressure				
M1	0.91	1.66*	1.33	1044
M5	0.73	1.60+	1.08	1044
High-Risk Blood Pressure				
M1	0.91	1.70*	1.25	1044
M5	0.77	1.68+	0.94	1044
High-Risk Pulse Rate				
M1	0.75	1.29	0.46+	1044
M5	0.90	1.28	0.63	1044
Height, ≤10th Percentile				
M1	1.18	1.06	1.99	1044
M5	0.94	0.86	1.89	1044
Height, ≤25th Percentile				
M1	1.70*	1.16	2.01*	1044
M5	1.61	1.00	2.19*	1044

Part B: Self-Reported Chronic Conditions

	Gestation			
	1st Trimester	2nd Trimester	3rd Trimester	*N*
Hypertension				
M1	1.07	1.54	0.68	1044
M5	0.54	1.12	0.37*	1044

	Post-Birth			
	1-3 Months	4-6 Months	7-9 Months	*N*
Hypertension				
M1	0.98	1.17	1.60	1044
M5	0.59	0.92	0.82	1044

Diabetes					*Diabetes*			
M1	0.61	0.90	0.89	1044	M1	1.32	0.77	0.93	1044
M5	0.28*	0.68	0.52	1044	M5	0.77	0.57	0.49	1044
Angina					*Angina*			
M1	0.58	0.51	0.69	1044	M1	0.59	0.59	0.24*	1044
M5	0.87	0.74	0.80	1044	M5	0.43	0.74	0.27+	1044
Stroke					*Stroke*			
M1	1.23	1.96	0.18	1044	M1	0.60	2.28+	1.62	1044
M5	0.37	1.66	0.03+	1044	M5	0.42	2.00	1.10	1044
Chronic Lung Disease					*Chronic Lung Disease*			
M1	0.50	0.50	0.05+	1044	M1	0.77	1.54	0.45	1044
M5	0.43	0.46	0.05*	1044	M5	1.00	1.77	0.73	1044
Arthritis					*Arthritis*			
M1	1.71	0.54*	1.64	1044	M1	1.71+	1.14	0.94	1044
M5	1.00	0.55+	1.31	1044	M5	1.45	0.80	0.85	1044
Asthma					*Asthma*			
M1	0.25*	2.10	0.16*	1044	M1	0.60	0.55	1.41	1044
M5	0.31+	2.97+	0.19+	1044	M5	0.85	0.75	2.78	1044

+ p <0.10, * p < 0.05, ** p < 0.01, *** p < 0.001

Notes: Model 1 includes controls for age and sex. Model 5 adds controls for region, father's education, father's occupation, and respondent's education. See **Table 3.3** for the high-risk cutpoints. Models are based on a sample of non-movers who are not missing information on rainfall and temperature around the time of birth.

[a] > 102 cm (males), > 88 cm (females)

[b] ≥ 90 cm (males), ≥ 80 cm (females)

[c] ≥ 87 cm (males), ≥ 82 cm (females)

Table 3.13. Summary of Results from Logistic Regression Models Using Average Minimum Temperature in Gestation (Part A) and the Post-Birth Period (Part B) to Predict Measured Health Outcomes and Self-Reported Chronic Conditions, Males and Females Aged 50+, India (2007-2008)

Part A: Gestational Period

	Measured Health Outcomes								Self-Reported Chronic Conditions				
		High-Risk					Height						
	Obese	WC	WHR	SBP	DBP	BP	$\leq 10^{th}$	$\leq 25^{th}$	Hypertension	Diabetes	Angina	Stroke	
1^{st} Trim.				X		X	X	X	X			X	
2^{nd} Trim.						(X)							
3^{rd} Trim.				(X)			X	X					

Part B: Post-Birth Period

	Measured Health Outcomes								Self-Reported Health Outcomes				
		High-Risk					Height						
	Obese	WC	WHR	SBP	DBP	BP	$\leq 10^{th}$	$\leq 25^{th}$	Hypertension	Diabetes	Angina	Stroke	CLD
1-3 Months								(X)				(X)	(✓)
4-6 Months				X	(X)	X						X	
7-9 Months							X	X				(X)	✓

Notes: WC refers to waist circumference, WHR refers to waist-to-hip ratio, SBP refers to systolic blood pressure, DBP refers to diastolic blood pressure, BP refers to overall blood pressure, and CLD refers to chronic lung disease. Height measures refer to falling below the 10^{th} and 25^{th} height percentiles. ✓'s indicate that higher temperatures are associated with a significant health advantage (OR<1) and X's indicate that higher temperatures are associated with a significant health disadvantage (OR>1) for a particular health outcome. Parentheses indicate that the odds ratios are significant at $p<0.10$, otherwise ✓'s and X's indicate that odds ratios are significant at $p<0.05$ in the basic models (M1). For obesity and waist circumference, this table summarizes results from models using the standard cutpoints.

Table 3.14. Odds Ratios from Logistic Regression Models Using Average Minimum Temperature (°Celsius) to Predict Measured Health Outcomes (Part A) and Self-Reported Chronic Conditions (Part B), Males and Females Aged 50+, India (2007-2008)

Part A: Measured Health Outcomes									
	Gestation					Post-Birth			
	1st Trimester	2nd Trimester	3rd Trimester	*N*		1-3 Months	4-6 Months	7-9 Months	*N*
Obese (BMI ≥30 kg/m²)					*Obese (BMI ≥30 kg/m²)*				
M1	1.00	0.94	1.05	1044	M1	0.96	0.96	0.92	1044
M5	1.10	0.94	1.12	1044	M5	0.96	0.98	0.92	1044
Obese (BMI ≥25 kg/m²)					*Obese (BMI ≥25 kg/m²)*				
M1	1.06	0.99	1.06	1044	M1	1.01	1.01	1.01	1044
M5	1.06	0.99	1.05	1044	M5	1.00	1.02	1.00	1044
Obese (BMI ≥23 kg/m²)					*Obese (BMI ≥23 kg/m²)*				
M1	1.04	0.99	1.05	1044	M1	1.02	1.00	1.02	1044
M5	1.02	0.99	1.02	1044	M5	1.02	1.00	1.02	1044
High-Risk Waist Circumference[a]					*High-Risk Waist Circumference*[a]				
M1	1.03	0.98	1.07	1044	M1	0.93	0.99	0.93	1044
M5	1.06	0.97	1.09	1044	M5	0.90	0.98	0.90	1044
High-Risk Waist Circumference[b]					*High-Risk Waist Circumference*[b]				
M1	1.00	1.00	1.01	1044	M1	0.97	0.99	0.98	1044
M5	1.04	1.00	1.05	1044	M5	1.01	1.00	1.02	1044
High-Risk Waist Circumference[c]					*High-Risk Waist Circumference*[c]				
M1	1.02	0.98	1.01	1044	M1	0.95	1.01	0.95	1044
M5	1.07	0.99	1.05	1044	M5	0.98	1.01	0.97	1044
High-Risk Waist-to-Hip Ratio					*High-Risk Waist-to-Hip Ratio*				
M1	1.07	1.00	1.04	1044	M1	1.01	1.03	1.01	1044
M5	1.04	0.99	1.02	1044	M5	0.99	1.03	0.98	1044

High-Risk Systolic Blood Pressure				
M1	1.10**	0.97	1.07+	1044
M5	1.05	0.96+	1.02	1044
High-Risk Diastolic Blood Pressure				
M1	1.06	0.97	1.02	1044
M5	1.00	0.97	0.96	1044
High-Risk Blood Pressure				
M1	1.07*	0.96+	1.03	1044
M5	1.03	0.96+	0.99	1044
High-Risk Pulse Rate				
M1	1.01	0.94+	0.98	1044
M5	1.02	0.95	0.98	1044
Height, ≤10th Percentile				
M1	1.16*	1.06	1.18*	1044
M5	1.15+	1.07+	1.15*	1044
Height, ≤25th Percentile				
M1	1.12**	1.03	1.12**	1044
M5	1.08	1.04	1.07	1044

High-Risk Systolic Blood Pressure				
M1	1.04	1.04*	1.00	1044
M5	1.02	1.03	0.98	1044
High-Risk Diastolic Blood Pressure				
M1	1.02	1.04+	1.00	1044
M5	1.00	1.03	0.98	1044
High-Risk Blood Pressure				
M1	1.02	1.05*	0.98	1044
M5	1.00	1.04*	0.96	1044
High-Risk Pulse Rate				
M1	0.98	1.03	0.93	1044
M5	0.99	1.03	0.95	1044
Height, ≤10th Percentile				
M1	1.09	1.01	1.14**	1044
M5	1.07	1.02	1.13*	1044
Height, ≤25th Percentile				
M1	1.08+	1.01	1.11**	1044
M5	1.07	1.02	1.11**	1044

Part B: Self-Reported Chronic Conditions

	Gestation			
	1st Trimester	2nd Trimester	3rd Trimester	*N*
Hypertension				
M1	1.08*	1.00	1.06	1044
M5	1.00	0.99	0.98	1044

	Post-Birth			
	1-3 Months	4-6 Months	7-9 Months	*N*
Hypertension				
M1	1.01	1.03	1.01	1044
M5	0.94	1.01	0.95	1044

Diabetes					Diabetes				
M1	1.05	0.98	1.07	1044	M1	1.05	0.99	1.02	1044
M5	0.92	0.95	0.96	1044	M5	0.98	0.95	0.95	1044
Angina					*Angina*				
M1	1.03	1.01	1.07	1044	M1	0.94	0.97	0.95	1044
M5	1.01	1.03	1.02	1044	M5	0.94	0.98	0.97	1044
Stroke					*Stroke*				
M1	1.32*	1.00	1.12	1044	M1	1.23+	1.16**	1.20+	1044
M5	1.24+	1.00	1.01	1044	M5	1.20	1.20**	1.18	1044
Chronic Lung Disease					*Chronic Lung Disease*				
M1	0.99	0.90+	0.94	1044	M1	0.90+	1.04	0.83*	1044
M5	1.02	0.89+	0.98	1044	M5	0.90	1.03	0.82*	1044
Arthritis					*Arthritis*				
M1	1.04	0.99	1.06	1044	M1	1.07	1.00	1.04	1044
M5	0.99	0.98	1.01	1044	M5	1.03	0.99	1.01	1044
Asthma					*Asthma*				
M1	1.00	0.98	0.97	1044	M1	0.94	1.02	0.95	1044
M5	1.07	0.99	1.03	1044	M5	0.99	1.04	1.00	1044

+ p <0.10, * p < 0.05, ** p < 0.01, *** p < 0.001

[a] > 102 cm (males), > 88 cm (females)

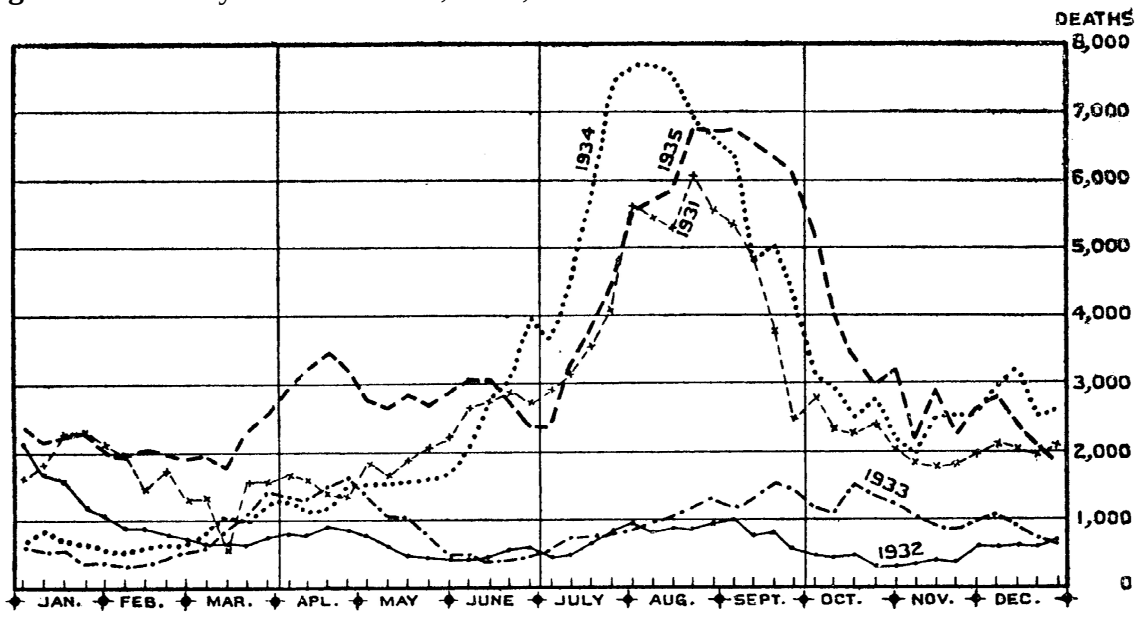
[b] ≥ 90 cm (males), ≥ 80 cm (females)

[c] ≥ 87 cm (males), ≥ 82 cm (females)

Notes: Model 1 includes controls for age and sex. 5 adds state, father's education, father's occupation, and respondent's education. See **Table 3.3** for the high-risk cutpoints. Models are based on a sample of non-movers who are not missing information on rainfall and temperature around the time of birth.

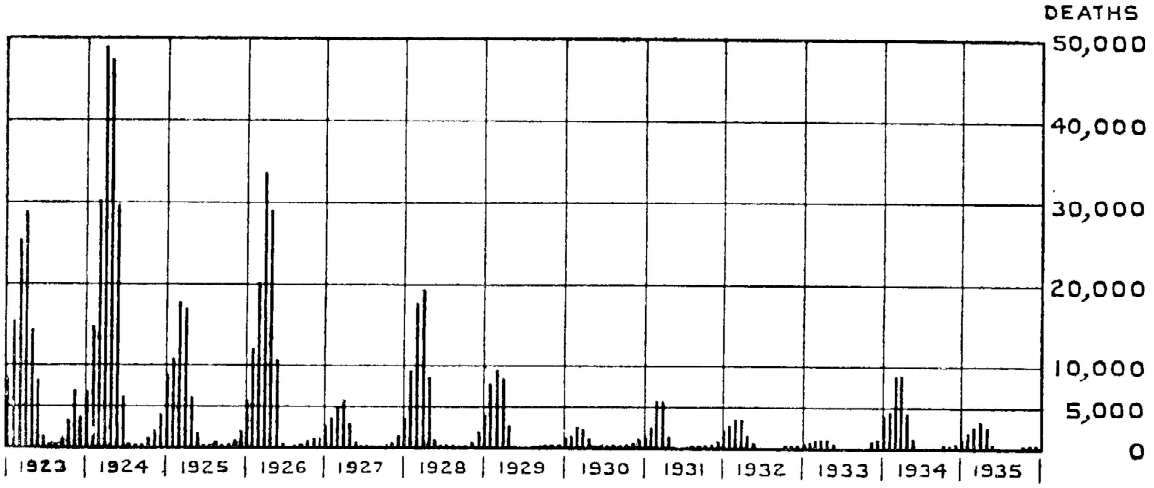
Figures

Figure 3.1. Weekly Cholera Deaths, India, 1931-1935



Source: Public Health Commissioner of India (1937).

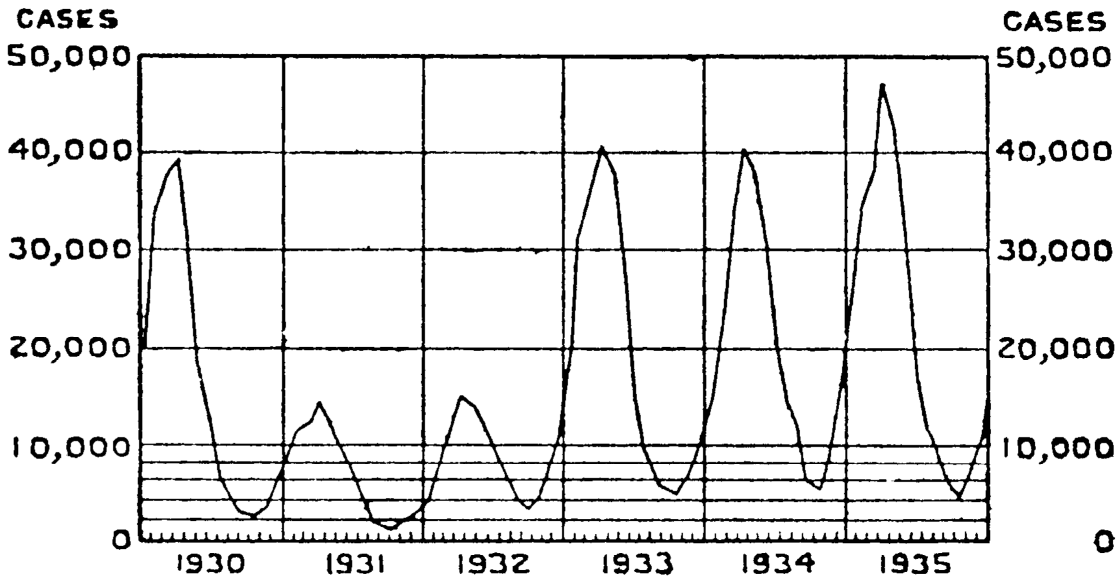
Figure 3.2. Reported Plague Deaths, Northern India[46], 1923-1935



Source: Public Health Commissioner of India (1937).

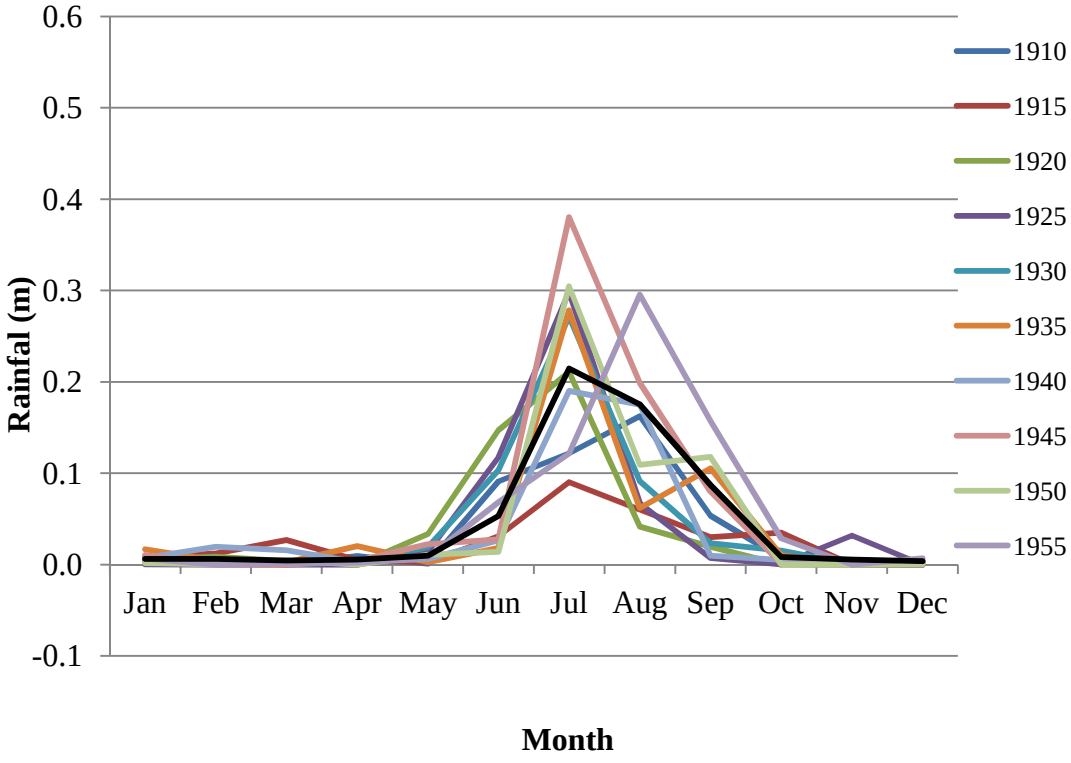
[46] North-West Frontier Province (now Pakistan), Punjab, Punjab States, Uttar Pradesh, Bihar and Orissa, and Kashmir

Figure 3.3. Reported Smallpox Cases, India, 1930-1935



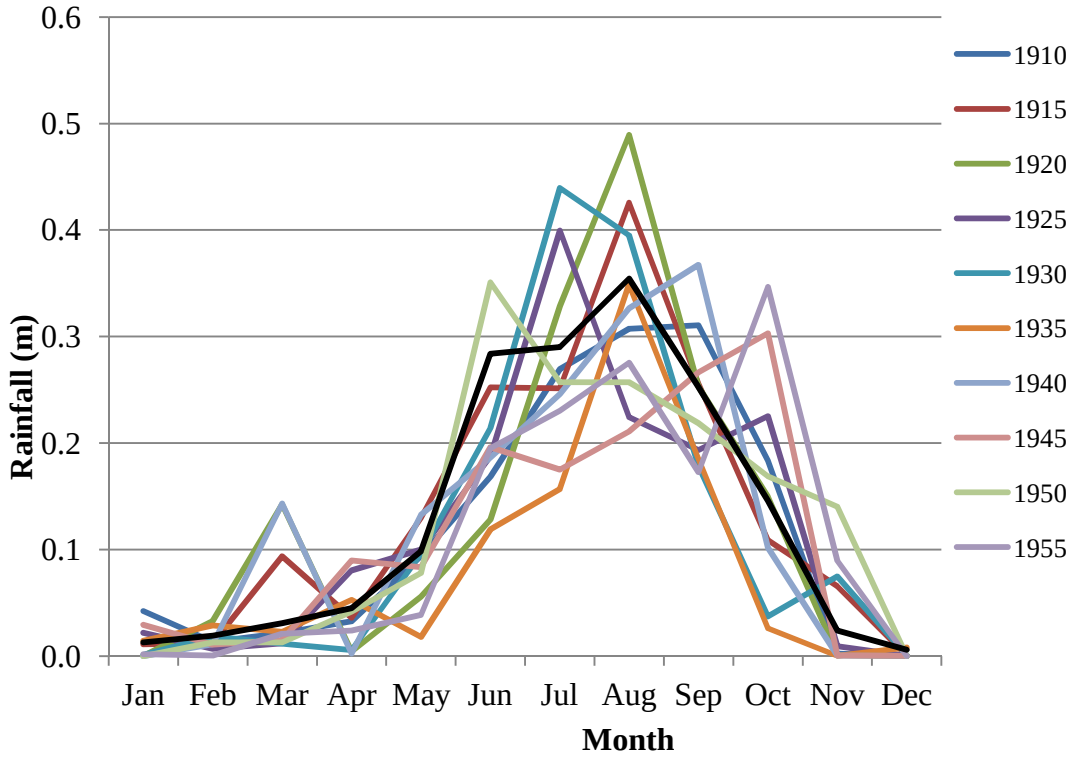
Source: Public Health Commissioner of India (1937).

Figure 3.4. Monthly Rainfall (m), Jaipur, Rajasthan, 1907-1957



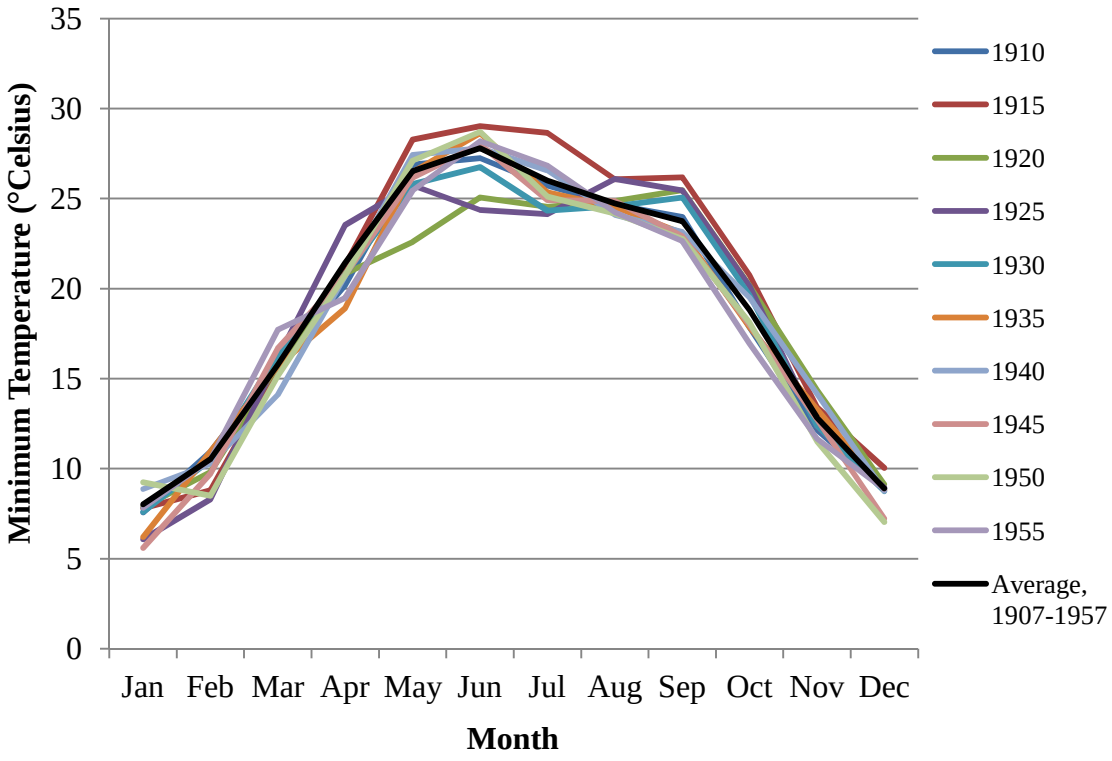
Source: India Water Portal (2012).

Figure 3.5. Monthly Rainfall (m), North 24 Parganas, 1907-1957



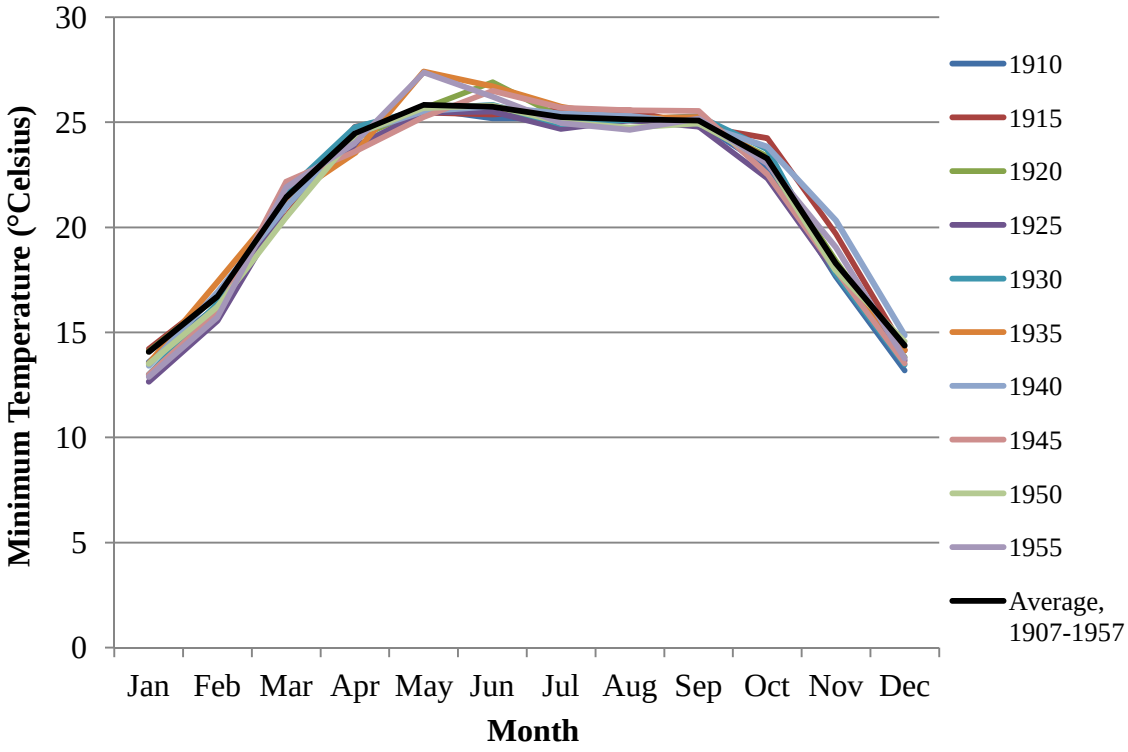
Source: India Water Portal (2012).

Figure 3.6. Monthly Minimum Temperature (°Celsius), Jaipur, Rajasthan, 1907-1957



Source: India Water Portal (2012).

Figure 3.7. Monthly Minimum Temperature (°Celsius), North 24 Parganas, 1907-1957



Source: India Water Portal (2012).

Appendix

Chapter 1

Table A1.1. Comparison of Population Attributable Fractions (%) from Indirect Estimation and Cox Regression Models, Black and White Males and Females Aged 50+ (Indirect Estimation) and Aged 50-84 at Baseline (Direct Estimation)

Males

	Blacks	Whites	Difference (B-W)
Indirect Estimation (2001)	26.64	21.76	4.89
Direct Estimation (1997-2006)			
• 8-category smoking status (non-Hispanics)†	33.08	29.43	3.65
• 8-category smoking status (including Hispanics)†	32.32	28.90	3.42
• 3-category smoking status (non-Hispanics)‡	31.16	29.08	2.08

Females

	Blacks	Whites	Difference (B-W)
Indirect Estimation (2001)	13.31	15.35	-2.04
Direct Estimation (1997-2006)			
• 8-category smoking status (non-Hispanics)†	22.41	23.62	-1.22
• 8-category smoking status (including Hispanics)†	22.00	22.85	-0.85
• 3-category smoking status (non-Hispanics)‡	21.48	23.42	-1.94

† Categories: never smoker, former smoker quit 30+ years ago, former smoker quit 20-29 years ago, former smoker quit 10-19 years ago, former smoker quit 5-9 years ago, former smoker quit 0-4 years ago, current smoker <1 pack per day, current smoker 1+packs per day

‡ Categories: never smoker, former smoker, current smoker

Table A1.2. Final Sample Sizes and Number of Deaths, National Health Interview Survey, Respondents Aged 50-84 at Baseline, 1997-2003

	Total	Whites	Blacks
Non-Hispanic Males			
Sample size	27,164	23,701	3,463
Number of deaths	4,831	4,082	749
Males, including Hispanics			
Sample size	29,922	26,411	3,511
Number of deaths	5,215	4,456	759
Non-Hispanic Females			
Sample size	36,646	31,156	5,490
Number of deaths	5,046	4,228	818
Females, including Hispanics			
Sample size	40,528	34,966	5,562
Number of deaths	5,427	4,603	824

Chapter 2

Table A2.1. Life Expectancy at Birth, Difference with United States, and Ranking for Males and Females in 17 High-Income Countries, 2006-2008

		Males			Females		
Country	Year	e0	Difference with U.S.	Rank	e0	Difference with U.S.	Rank
Switzerland	2007	79.33	3.69	1	84.09	3.31	4
Japan	2008	79.31	3.67	2	86.04	5.26	1
Australia	2006	79.17	3.53	3	83.79	3.01	6
Sweden	2008	79.09	3.45	4	83.12	2.34	7
Italy	2007	78.82	3.18	5	84.09	3.31	4
Canada	2007	78.35	2.71	6	82.95	2.17	11
Norway	2008	78.34	2.70	7	82.97	2.19	9
Netherlands	2008	78.32	2.68	8	82.28	1.5	13
Spain	2008	78.03	2.39	9	84.2	3.42	3
United Kingdom	2008	77.63	1.99	10	81.74	0.96	15
Austria	2008	77.62	1.98	11	82.97	2.19	9
France	2008	77.62	1.98	11	84.39	3.61	2
Germany	2006	76.93	1.29	13	82.27	1.49	14
Finland	2008	76.32	0.68	14	83.01	2.23	8
Portugal	2008	76.18	0.54	15	82.36	1.58	12
Denmark	2006	75.91	0.27	16	80.52	-0.26	17
United States	2007	75.64		17	80.78		16
Non-U.S. Average		77.94	2.30		83.17	2.39	

Source: Human Mortality Database (2012)

Table A2.2. Cause of Death Categories and Corresponding ICD-10 Codes[47]

Category	ICD-10 Codes	Source
1. Communicable and nutritional conditions	1. A00-B99, D50-D53, D64.9, E00-E02, E40-E46, E50, E51-E64, G00-G04, H65-H66, J00-J06, J10-J18, J20-J22, N70-N73	
a. HIV	a. B20-B24	GBD
b. All other communicable and nutritional conditions, excluding HIV/AIDS	b. A00-B99 (excluding B20-B24), D50-D53, D64.9, E00-E02, E40-E46, E50, E51-E64, G00-G04, H65-H66, J00-J06, J10-J18, J20-J22, N70-N73	
2. Noncommunicable diseases	2. C00-C97, D00-D48, D55-D64 (minus D64.9), D65-D89, E03-E07, E10-E16, E20-E34, E65-E88, F01-F99, G06-G98, H00-H61, H68-H93, I00-I99, J30-J98, K00-K14, K20-K92, N00-N64, N75-N98, L00-L98, M00-M99, Q00-Q99	
a. Malignant neoplasms	a. C00-C97	
i. Lung cancer	i. C33-C34	
ii. All other cancers, excluding lung cancer	ii. C00-C97 (excluding C33-C34)	
b. Other neoplasms	b. D00-D48	GBD
c. Diabetes mellitus	c. E10-E14	
d. Endocrine disorders	d. D55-D64 (minus D64.9), D65-D89, E03-E07, E15-E16, E20-E34, E65-E88	
e. Neuropsychiatric disorders	e. F01-F99, G06-G98	
f. Sense organ diseases	f. H00-H61, H68-H93	
g. Cardiovascular disease	g. I00-I99	
h. Respiratory diseases	h. J30-J98	
i. Digestive diseases	i. K20-K92	
j. Genitourinary diseases	j. N00-N64, N75-N98	

[47] GBD indicates that cause-of-death groupings are specified per the Global Burden of Disease Study conventions, NVSR indicates that cause-of-death groupings are specified following the conventions of the U.S. National Vital Statistics Reports.

k. Skin diseases	k. L00-L98	
l. Musculoskeletal diseases	l. M00-M99	
m. Congenital anomalies	m. Q00-Q99	
n. Oral conditions	n. K00-K14	
3. Maternal conditions	3. O00-O99	GBD
4. Perinatal conditions	4. P00-P96	GBD
5. Unintentional injuries	5. V01-X59,Y85-Y86	
a. Transport injuries	a. V01-V99,Y85	NVSR
b. Nontransport injuries	b. W00-X59,Y86	
6. Intentional injuries	6. X60-Y09, Y87.0-Y87.1	
a. Suicide	a. X60-X84,Y87.0	NVSR
b. Homicide	b. X85-Y09, Y87.1	
7. Drug-related causes[48]	7. D52.1, D59.0, D59.2, D61.1, D64.2, E06.4, E16.0, E23.1, E24.2, E24.4, E27.3, E66.1, F10, F11.0-F11.5, F11.7-F11.9, F12.0-F12.5, F12.7-F12.9, F13.0-F13.5, F13.7-F13.9, F14.0-F14.5, F14.7-F14.9, F15.0-F15.5, F15.7-F15.9, F16.0-F16.5, F16.7-F16.9, F17.0, F17.3-F17.5, F17.7-F17.9, F18.0-F18.5,F18.7-F18.9, F19.0-F19.5, F19.7-F19.9, G21.1, G24.0, G25.1, G25.4, G25.6, G31.2, G44.4, G62.0, G62.1, G72.0, G72.1, I42.6, I95.2, J70.2–J70.4, K29.2, K70, K85.2, K85.3, K86.0, L10.5, L27.0-L27.1, M10.2, M32.0, M80.4, M81.4, M83.5, M87.1, R50.2, R78.0, R78.1-R78.5, X40-X44, X45, X60-X64, X65, X85, Y10-Y14, Y15	NVSR

[48] Composed of the “drug-induced causes” and “alcohol-induced causes” categories from the NVSR.

Table A2.3. Percentage Contribution of Causes of Death to the Difference in Years of Life Lost Between the U.S. and 15 OECD Countries, Males, 2006-2008[49]

	Communicable Diseases		Intentional Injuries		Noncommunicable Diseases		Unintentional Injuries			
Country	HIV	All other	Suicide	Homicide	CVD	All other	Transport	Nontransport	Perinatal conditions	Residual
Australia	3	3	4	23	9	14	15	11	11	7
Austria	3	3	4	21	11	-1	17	18	13	12
Canada	3	2	1	20	12	15	16	20	4	7
Denmark	3	3	7	21	10	2	20	14	10	10
Finland	4	3	-16	24	8	12	24	3	25	14
France	2	2	0	22	9	11	17	18	16	1
Germany	2	2	6	19	6	10	17	21	12	4
Italy	1	2	11	16	7	13	9	17	12	11
Japan	2	1	-7	16	4	15	21	17	20	9
Netherlands	2	2	7	15	7	11	20	19	9	8
Norway	3	4	1	19	11	16	18	8	16	4
Portugal	-6	0	14	23	11	7	19	28	17	-12
Spain	1	1	10	18	7	9	16	15	12	10
Sweden	2	2	2	15	8	16	19	13	17	5
United Kingdom	3	0	8	25	6	-3	26	17	8	9
Mean	**2**	**2**	**4**	**19**	**8**	**10**	**18**	**16**	**13**	**7**

[49] Contributions sum to 100% across the rows of this table. The mean row corresponds to the data presented in **Figure 2.9**.

Table A2.4. Percentage Contribution of Causes of Death to the Difference in Years of Life Lost Between the U.S. and 15 OECD Countries, Females, 2006-2008[50]

	Communicable Diseases		Intentional Injuries		Noncommunicable Diseases		Unintentional Injuries				
Country	HIV	All other	Suicide	Homicide	CVD	All other	Transport	Non-transport	Perinatal conditions	Maternal conditions	Residual
Australia	4	4	1	9	9	23	17	11	14	2	6
Austria	2	3	1	7	9	17	14	15	16	1	13
Canada	3	4	-4	9	14	26	15	17	3	2	11
Denmark	2	3	0	7	11	16	14	15	16	1	14
Finland	3	4	-9	4	9	23	16	8	27	1	14
France	3	4	-1	8	9	22	17	14	20	1	4
Germany	2	3	2	7	6	21	16	16	17	1	9
Italy	2	4	4	7	8	20	13	14	15	1	12
Japan	3	1	-12	7	7	23	19	12	30	1	9
Netherlands	2	3	1	7	7	14	19	17	17	1	12
Norway	2	5	-5	7	9	22	16	10	22	1	10
Portugal	-2	3	5	8	10	19	15	19	22	2	1
Spain	1	3	4	7	9	18	15	14	18	1	11
Sweden	2	2	-4	6	10	25	17	13	23	1	4
United Kingdom	3	-1	3	11	9	6	24	16	13	2	13
Mean	**2**	**3**	**-1**	**7**	**9**	**20**	**16**	**14**	**19**	**1**	**9**

[50] Contributions sum to 100% across the rows of this table. The mean row corresponds to the data presented in **Figure 2.10**.

Chapter 3

Table A3.1. Summary Statistics, Distribution (%) or Mean (SE), Males and Females Aged 50+, South Africa (2007-2008) and Ghana (2007-2008) SAGE Wave 1

	South Africa				Ghana		
	Males	Females	Total		Males	Females	Total
	568 (40%)	763 (60%)	1,331		581 (69%)	257 (31%)	838
Sociodemographic Characteristics				**Sociodemographic Characteristics**			
Season of Birth				*Season of Birth*			
Summer	25.5	22.4	23.7	Winter	24.6	23.6	24.3
Autumn	22.3	23.7	23.1	Spring	24.9	29.0	26.2
Winter	28.4	28.3	28.4	Summer	28.2	26.6	27.7
Spring	23.8	25.6	24.8	Autumn	22.3	20.8	21.9
Age Group				*Age Group*			
50-54	29.6	33.4	31.8	50-54	26.7	32.5	28.5
55-59	24.3	16.5	19.6	55-59	25.7	30.5	27.2
60-64	17.5	15.0	16.0	60-64	16.4	14.1	15.7
65-69	14.3	14.9	14.7	65-69	13.9	10.0	12.7
70-74	6.1	11.1	9.0	70-74	9.1	5.7	8.1
75-79	3.6	6.4	5.3	75-79	5.9	3.3	5.1
80-84	2.8	1.4	2.0	80-84	1.9	2.8	2.2
85+	1.9	1.3	1.5	85+	0.5	1.2	0.7
Ethnic Background							
African/Black	69.8	67.2	68.2				
White	13.3	13.7	13.5				
Coloured	12.6	14.5	13.8				
Other	4.3	4.6	4.5				
Religion				*Religion*			
Christianity	86.7	90.9	89.2	Christianity	89.9	95.9	91.7
Other	13.3	9.1	10.8	Islam	4.4	2.9	3.9
				Other	5.8	1.2	4.4
Province				*Region*			
Eastern	13.0	11.6	12.1	Ashanti	23.7	14.4	20.8
Free State	8.2	7.6	7.8	Brong-Ahafo	7.4	6.6	7.1
Gauteng	31.9	27.4	29.2	Central	7.2	7.9	7.4
KwaZulu-Natal	11.8	18.0	15.5	Eastern	15.4	11.3	14.1
Limpopo	6.4	5.2	5.7	Greater Accra	22.5	38.3	27.3
Mpumalanga	7.3	6.6	6.9	Northern	0.7	0.0	0.5
North-West	8.3	8.3	8.3	Upper East	0.8	0.0	0.6
Northern Cape	2.9	2.9	2.9	Upper West	0.5	0.3	0.5
Western Cape	10.3	12.4	11.6	Volta	11.8	14.5	12.6
				Western	10.1	6.7	9.1
Education				*Education*			
Less than primary	29.1	31.2	30.4	Less than primary	5.9	13.2	8.1
Primary	26.6	29.3	28.2	Primary	19.1	27.1	21.5
Secondary	18.6	21.5	20.3	Secondary	13.5	8.8	12.0
High school +	25.6	18.1	21.1	High school	48.4	39.5	45.6
				College +	13.2	11.5	12.7

Father's Education			
No formal education	38.4	37.0	37.5
Less than primary	24.3	21.8	22.8
Primary	19.6	19.5	19.5
Secondary +	17.6	21.8	20.1
Father's Occupation			
Non-agricultural	78.8	79.1	79.0
Agricultural	21.2	20.9	21.0
Measured Health Outcomes			
Body Mass Index			
Underweight	4.3	1.9	2.8
Normal	28.8	17.1	21.9
Overweight	29.2	30.2	29.8
Obese I	24.9	22.9	23.7
Obese II/III	12.8	27.9	21.8
High-Risk Waist Circumference			
Normal	75.1	34.5	50.9
High-risk	24.9	65.6	49.1
High-Risk Hip-to-Waist Ratio			
Normal	40.3	25.6	31.6
High-risk	59.7	74.4	68.5
High-Risk Systolic Blood Pressure			
Normal	38.3	31.3	34.2
High-risk	61.7	68.7	65.9
High-Risk Diastolic Blood Pressure			
Normal	30.1	23.6	26.3
High-risk	69.9	76.4	73.8
High-Risk Blood Pressure			
Normal	44.4	36.1	39.5
High-risk	55.6	63.9	60.5
High-Risk Pulse Rate			
Normal	85.7	83.9	84.7
High-risk	14.3	16.1	15.4
Height			
Mean (SE)	1.646 (0.007)	1.578 (0.007)	1.605 (0.006)
$\leq 10^{th}$ percentile			
No	95.5	94.1	94.6
Yes	4.5	5.9	5.4
$\leq 25^{th}$ percentile			
No	82.3	75.5	78.3
Yes	17.7	24.5	21.7
Chronic Conditions			
Hypertension			
No	72.0	65.2	68.0
Yes	28.0	34.8	32.1
Diabetes			
No	92.6	86.9	89.2
Yes	7.4	13.1	10.8

Father's Education			
No formal education	71.9	49.6	65.1
Less than primary	8.4	7.3	8.0
Primary	4.8	11.6	6.9
Secondary	3.0	4.0	3.3
High School +	11.9	27.6	16.7
Father's Occupation			
Non-agricultural	30.6	51.0	36.9
Agricultural	69.4	49.0	63.1
Measured Health Outcomes			
Body Mass Index			
Underweight	13.3	5.0	10.7
Normal	57.1	35.0	50.3
Overweight	20.9	29.0	23.4
Obese I	6.5	12.7	8.4
Obese II/III	2.2	18.3	7.1
High-Risk Waist Circumference			
Normal	91.8	40.0	75.9
High-risk	8.2	60.0	24.1
High-Risk Hip-to-Waist Ratio			
Normal	34.1	13.6	27.8
High-risk	65.9	86.4	72.2
High-Risk Systolic Blood Pressure			
Normal	55.5	48.8	53.5
High-risk	44.5	51.2	46.6
High-Risk Diastolic Blood Pressure			
Normal	42.2	40.7	41.8
High-risk	57.8	59.3	58.2
High-Risk Blood Pressure			
Normal	57.4	53.3	56.1
High-risk	42.7	46.7	43.9
High-Risk Pulse Rate			
Normal	87.6	91.8	88.9
High-risk	12.4	8.2	11.1
Height			
Mean (SE)	1.671 (0.004)	1.592 (0.006)	1.647 (0.004)
$\leq 10^{th}$ percentile			
No	94.5	95.6	94.9
Yes	5.5	4.4	5.1
$\leq 25^{th}$ percentile			
No	81.6	83.7	82.2
Yes	18.4	16.3	17.8
Chronic Conditions			
Hypertension			
No	82.7	67.8	78.1
Yes	17.3	32.2	21.9
Diabetes			
No	94.7	93.0	94.2
Yes	5.3	7.0	5.8

Angina				*Angina*			
No	96.0	95.0	95.4	No	98.2	95.6	97.4
Yes	4.0	5.0	4.6	Yes	1.8	4.4	2.6
Stroke				*Stroke*			
No	96.3	96.5	96.4	No	97.2	98.2	97.5
Yes	3.7	3.5	3.6	Yes	2.8	1.8	2.5
Chronic Lung Disease				*Chronic Lung Disease*			
No	98.4	96.5	97.3	No	99.4	100.0	99.6
Yes	1.7	3.5	2.7	Yes	0.6	0.0	0.4
Arthritis				*Arthritis*			
No	83.9	72.9	77.3	No	89.3	82.4	87.2
Yes	16.1	27.1	22.7	Yes	10.8	17.6	12.9
Asthma				*Asthma*			
No	94.4	96.3	95.5	No	96.2	96.8	96.4
Yes	5.6	3.7	4.5	Yes	3.8	3.2	3.6

Table A3.2. Summary Statistics, Distribution (%) or Mean (SE), Males and Females Aged 50+, Russia (2007-2010) and Mexico (2009-2010) SAGE Wave 1

	Russia				Mexico		
	Males	Females	Total		Males	Females	Total
	765 (42%)	1,495 (58%)	2,260		397 (50%)	540 (50%)	937
Sociodemographic Characteristics				**Sociodemographic Characteristics**			
Season of Birth				*Season of Birth*			
Summer	28.9	26.9	27.7	Summer	13.1	20.7	16.9
Autumn	25.2	21.8	23.2	Autumn	28.9	22.5	25.8
Winter	17.1	25.5	22.0	Winter	19.1	21.6	20.3
Spring	28.9	25.7	27.1	Spring	38.9	35.2	37.1
Age Group				*Age Group*			
50-54	22.4	26.5	24.8	50-54	31.6	17.8	24.7
55-59	25.7	21.2	23.1	55-59	29.9	30.3	30.1
60-64	16.0	12.0	13.6	60-64	12.6	15.8	14.2
65-69	17.6	11.5	14.1	65-69	9.2	12.0	10.6
70-74	9.3	10.9	10.2	70-74	5.1	6.9	6.0
75-79	4.9	11.3	8.6	75-79	6.9	12.4	9.6
80-84	1.7	3.8	2.9	80-84	1.7	4.0	2.8
85+	2.5	2.8	2.7	85+	2.9	0.9	1.9
Religion				*Religion*			
None	27.9	13.2	19.3	Catholic	95.0	95.3	95.1
Christianity	68.2	80.6	75.4	Other	5.1	4.7	4.9
Islam	3.4	5.4	4.6				
Other	0.6	0.8	0.7				
Federal District				*Region*			
Central	30.6	27.8	29.0	Center-North	11.2	5.7	8.4
Far Eastern	7.3	5.1	6.0	Center-South	34.2	27.5	30.9
North Caucasian	4.1	4.5	4.3	East	3.3	5.8	4.6
Northwestern	23.8	20.9	22.1	Northeast	9.0	17.3	13.1
Siberian	5.8	7.3	6.7	Northwest	20.1	16.9	18.5
Southern	2.1	2.4	2.3	Southeast	6.2	3.2	4.7
Ural	5.7	6.7	6.3	Southwest	10.8	8.4	9.6
Volga	20.6	25.4	23.4	West	5.3	15.2	10.2
Education				*Education*			
Primary or less	4.9	7.1	6.2	Less than primary	35.3	42.5	38.9
Secondary	10.3	14.6	12.8	Primary	34.3	28.4	31.3
High school	61.3	59.6	60.3	Secondary	10.8	17.4	14.1
College +	23.5	18.7	20.7	High school	3.3	4.9	4.1
				College +	16.4	6.8	11.6
Father's Education				*Father's Education*			
No formal education	9.8	10.8	10.4	No formal education	42.7	42.2	42.5
Less than primary	9.5	11.0	10.4	Less than primary	26.9	32.7	29.8
Primary	21.2	18.4	19.6	Primary	14.7	16.0	15.3
Secondary	17.6	18.8	18.3	Secondary +	15.7	9.1	12.4
High school	31.5	30.2	30.7				
College +	10.4	10.8	10.6				
Father's Occupation				*Father's Occupation*			
Non-agricultural	95.8	94.8	95.2	Non-agricultural	75.6	71.2	73.4
Agricultural	4.2	5.2	4.8	Agricultural	24.4	28.8	26.6

Measured Health Outcomes				**Measured Health Outcomes**			
Body Mass Index				*Body Mass Index*			
Underweight	1.5	1.3	1.4	Underweight	0.0	0.8	0.4
Normal	28.3	21.3	24.2	Normal	18.6	25.0	21.8
Overweight	43.2	37.7	40.0	Overweight	54.1	40.3	47.3
Obese I	23.0	24.1	23.6	Obese I	24.8	25.2	25.0
Obese II/III	3.9	15.8	10.8	Obese II/III	2.5	8.6	5.5
High-Risk Waist Circumference				*High-Risk Waist Circumference*			
Normal	76.3	41.9	56.4	Normal	61.0	39.1	50.1
High-risk	23.7	58.1	43.6	High-risk	39.0	60.9	49.9
High-Risk Hip-to-Waist Ratio				*High-Risk Hip-to-Waist Ratio*			
Normal	33.8	43.3	39.3	Normal	3.6	27.9	15.7
High-risk	66.3	56.7	60.7	High-risk	96.4	72.1	84.3
High-Risk Systolic Blood Pressure				*High-Risk Systolic Blood Pressure*			
Normal	51.1	39.3	44.3	Normal	51.5	40.5	46.0
High-risk	48.9	60.7	55.7	High-risk	48.5	59.5	54.0
High-Risk Diastolic Blood Pressure				*High-Risk Diastolic Blood Pressure*			
Normal	46.7	40.9	43.3	Normal	72.5	62.8	67.6
High-risk	53.3	59.1	56.7	High-risk	27.6	37.2	32.4
High-Risk Blood Pressure				*High-Risk Blood Pressure*			
Normal	58.7	44.9	50.7	Normal	72.7	65.7	69.2
High-risk	41.3	55.1	49.3	High-risk	27.3	34.3	30.8
High-Risk Pulse Rate				*High-Risk Pulse Rate*			
Normal	91.5	91.4	91.4	Normal	91.3	95.1	93.2
High-risk	8.5	8.6	8.6	High-risk	8.7	4.9	6.8
Height				*Height*			
Mean (SE)	1.717	1.597	1.648	Mean (SE)	1.650	1.511	1.581
	(0.006)	(0.005)	(0.004)		(0.010)	(0.007)	(0.010)
$\leq 10^{th}$ percentile				$\leq 10^{th}$ percentile			
No	93.2	91.0	91.9	No	93.4	94.8	94.1
Yes	6.8	9.0	8.1	Yes	6.6	5.2	5.9
$\leq 25^{th}$ percentile				$\leq 25^{th}$ percentile			
No	72.8	74.6	73.8	No	82.8	88.6	85.7
Yes	27.2	25.4	26.2	Yes	17.2	11.4	14.3
Chronic Conditions				**Chronic Conditions**			
Hypertension				*Hypertension*			
No	57.2	42.4	48.6	No	77.5	63.5	70.5
Yes	42.8	57.6	51.4	Yes	22.5	36.5	29.5
Diabetes				*Diabetes*			
No	94.9	91.5	93.0	No	81.4	81.2	81.3
Yes	5.1	8.5	7.0	Yes	18.6	18.8	18.7
Angina				*Angina*			
No	67.3	67.2	67.2	No	97.4	95.4	96.4
Yes	32.7	32.9	32.8	Yes	2.6	4.7	3.6
Stroke				*Stroke*			
No	92.6	96.4	94.8	No	96.0	95.2	95.6
Yes	7.4	3.6	5.2	Yes	4.0	4.8	4.4
Chronic Lung Disease				*Chronic Lung Disease*			
No	84.6	86.3	85.6	No	97.5	96.9	97.2
Yes	15.4	13.7	14.4	Yes	2.5	3.1	2.8
Arthritis				*Arthritis*			
No	79.7	72.1	75.3	No	97.6	88.1	92.9
Yes	20.3	27.9	24.7	Yes	2.4	11.9	7.1
Asthma				*Asthma*			
No	97.2	92.9	94.7	No	98.9	98.8	98.9
Yes	2.8	7.1	5.3	Yes	1.1	1.2	1.1

Table A3.3. Summary Statistics, Distribution (%) or Mean (SE), Males and Females Aged 50+, China (2007-2010)

	China		
	Males	Females	Total
	3,202 (57%)	2,773 (44%)	5,975
Sociodemographic Characteristics			
Season of Birth			
Winter	24.5	25.7	25.0
Spring	23.8	21.4	22.7
Summer	25.3	25.8	25.5
Autumn	26.4	27.2	26.7
Age Group			
50-54	26.6	29.4	27.8
55-59	26.5	27.7	27.0
60-64	18.7	19.3	18.9
65-69	13.8	12.7	13.3
70-74	7.8	7.2	7.6
75-79	4.5	2.8	3.8
80-84	1.7	0.8	1.3
85+	0.4	0.2	0.3
Religion			
None	96.9	91.5	94.6
Buddhism or Chinese Traditional	2.4	5.7	3.9
All other	0.6	2.8	1.6
Province			
Guangdong	21.1	16.4	19.0
Hubei	14.6	15.4	14.9
Jilin	2.7	3.1	2.9
Shaanxi	11.4	8.2	10.0
Shandong	27.8	32.6	29.9
Shanghai	4.9	5.4	5.1
Yunnan	8.8	9.7	9.2
Zhejiang	8.8	9.3	9.0
Education			
Less than primary	22.2	29.5	25.4
Primary	28.9	25.3	27.4
Secondary	26.8	25.2	26.1
High school +	22.0	19.9	21.1
Father's Education			
No formal education	60.4	56.5	58.7
Less than primary	17.0	15.2	16.2
Primary	10.3	13.3	11.6
Secondary	6.1	7.8	6.8
High school +	6.2	7.2	6.6
Father's Occupation			
Non-agricultural	27.7	32.4	29.7
Agricultural	72.3	67.7	70.3
Measured Health Outcomes			
Body Mass Index			
Underweight	3.5	2.7	3.1
Normal	65.0	55.1	60.7
Overweight	27.5	33.5	30.1

Obese I	2.9	7.4	4.8
Obese II/III	1.2	1.3	1.2
High-Risk Waist Circumference			
Normal	96.9	62.8	82.1
High-risk	3.1	37.2	17.9
High-Risk Hip-to-Waist Ratio			
Normal	53.7	35.1	45.6
High-risk	46.3	64.9	54.4
High-Risk Systolic Blood Pressure			
Normal	47.7	44.9	46.5
High-risk	52.3	55.1	53.5
High-Risk Diastolic Blood Pressure			
Normal	58.8	61.7	60.1
High-risk	41.2	38.3	39.9
High-Risk Blood Pressure			
Normal	65.3	65.7	65.5
High-risk	34.7	34.3	34.5
High-Risk Pulse Rate			
Normal	89.2	90.8	89.9
High-risk	10.8	9.2	10.1
Height			
Mean (SE)	1.649 (0.002)	1.556 (0.002)	1.609 (0.002)
$\leq 10^{th}$ percentile			
No	92.3	94.5	93.2
Yes	7.8	5.5	6.8
$\leq 25^{th}$ percentile			
No	79.3	83.4	81.1
Yes	20.7	16.6	18.9
Chronic Conditions			
Hypertension			
No	77.7	72.4	75.4
Yes	22.3	27.6	24.6
Diabetes			
No	95.0	92.9	94.1
Yes	5.0	7.1	6.0
Angina			
No	94.6	90.8	92.9
Yes	5.4	9.2	7.1
Stroke			
No	96.8	98.0	97.3
Yes	3.2	2.0	2.7
Chronic Lung Disease			
No	90.7	94.7	92.4
Yes	9.3	5.3	7.6
Arthritis			
No	81.8	73.7	78.3
Yes	18.2	26.3	21.7
Asthma			
No	98.2	98.7	98.4
Yes	1.8	1.3	1.6

Table A3.4. Summary Statistics, Distribution (%) or Mean (SE), Males and Females Aged 50+, India (2007-2008) SAGE Wave 1

	Season of Birth Sample			Climate Conditions Sample[51]		
	Males	Females	Total	Males	Females	Total
	1,071 (84%)	305 (16%)	1,376	854 (87%)	190 (13%)	1,044
Sociodemographic Characteristics						
Season of Birth						
Winter	19.6	19.5	19.6	19.9	19.0	19.8
Summer	23.3	31.0	24.6	22.9	32.3	24.1
Monsoon	41.0	31.8	39.5	41.0	33.1	40.0
Post Monsoon	16.1	17.8	16.4	16.2	15.6	16.1
Age Group						
50-54	25.5	31.8	26.5	25.7	32.1	26.5
55-59	29.8	25.3	29.1	30.9	30.1	30.8
60-64	12.3	15.9	12.9	11.2	13.3	11.5
65-69	13.9	12.4	13.6	13.5	13.0	13.5
70-74	13.5	10.2	13.0	13.7	10.8	13.3
75-79	3.0	2.6	2.9	3.1	0.3	2.7
80-84	1.4	1.9	1.5	1.3	0.4	1.1
85+	0.7	0.0	0.6	0.7	0.0	0.6
Religion						
Hinduism	89.3	90.3	89.5	88.5	88.6	88.5
Islam	5.8	5.9	5.9	6.5	7.7	6.7
Other	4.8	3.8	4.7	5.0	3.7	4.8
State						
Assam	2.1	2.8	2.3	2.1	2.2	2.1
Karnataka	13.3	26.8	15.5	10.6	19.7	11.9
Maharashtra	27.6	27.1	27.5	24.8	20.8	24.2
Rajasthan	9.5	5.0	8.8	9.7	4.7	9.1
Uttar Pradesh	25.3	6.9	22.3	28.8	8.1	26.0
West Bengal	22.2	31.5	23.7	24.1	44.6	26.8
Always Lived in Current Region						
Yes	80.2	93.8	86.8	100.0	100.0	100.0
No	19.8	6.2	13.2	0.0	0.0	0.0
Education						
Less than primary	8.6	17.2	10.0	8.8	16.5	9.9
Primary	17.6	33.4	20.2	17.0	33.7	19.2
Secondary	24.2	15.4	22.7	23.4	15.0	22.2
High school	28.5	18.2	26.8	30.0	15.3	28.0
College +	21.1	15.8	20.2	20.8	19.6	20.7
Father's Education						
No formal education	41.1	16.8	37.1	42.4	13.4	38.5
Less than primary	19.1	16.2	18.7	18.2	13.8	17.6
Primary completion	21.7	24.7	22.2	22.0	25.5	22.4
Secondary completion	8.4	16.1	9.7	8.3	16.5	9.4
High school +	9.6	26.3	12.4	9.1	30.9	12.0
Father's Occupation						
Non-agricultural	52.2	71.8	55.4	49.5	69.4	52.2
Agricultural	47.8	28.2	44.6	50.5	30.6	47.8

[51] Respondents who are non-missing on all predictor variables (including rainfall and temperature around the time of birth) and who have always lived in their current place of residence.

Measured Health Outcomes						
Body Mass Index						
Underweight	27.0	14.4	25.0	28.2	18.2	26.8
Normal	58.8	47.6	56.9	58.5	44.0	56.6
Overweight	11.4	26.9	14.0	10.7	26.6	12.8
Obese I	2.0	9.6	3.3	2.0	9.2	3.0
Obese II/III	0.8	1.5	0.9	0.6	2.1	0.8
Obese (BMI≥ 30 kg/m^2)						
Normal	97.2	88.9	95.8	97.4	88.7	96.3
High-risk	2.8	11.1	4.2	2.6	11.3	3.7
Obese (BMI≥ 25 kg/m^2)						
Normal	85.8	62.0	81.9	86.7	62.1	83.4
High-risk	14.2	38.0	18.1	13.3	37.9	16.6
Obese (BMI≥ 23 kg/m^2)						
Normal	73.0	46.3	68.6	74.1	47.5	70.5
High-risk	27.0	53.7	31.4	25.9	52.5	29.5
High-Risk Waist Circumference[a]						
Normal	95.4	43.0	86.8	96.4	49.1	90.0
High-risk	4.6	57.1	13.2	3.6	50.9	10.0
High-Risk Waist Circumference[b]						
Normal	70.6	24.9	63.1	71.4	30.3	65.9
High-risk	29.4	75.1	36.9	28.6	69.7	34.1
High-Risk Waist Circumference[c]						
Normal	58.9	27.0	53.6	59.7	32.1	56.0
High-risk	41.2	73.0	46.4	40.3	68.0	44.0
High-Risk Hip-to-Waist Ratio						
Normal	20.1	10.2	18.5	20.7	9.4	19.1
High-risk	79.9	89.8	81.5	79.4	90.7	80.9
High-Risk Systolic Blood Pressure						
Normal	74.1	60.8	71.9	74.6	58.1	72.4
High-risk	25.9	39.2	28.1	25.4	41.9	27.6
High-Risk Diastolic Blood Pressure						
Normal	69.2	59.7	67.6	69.8	60.2	68.5
High-risk	30.8	40.3	32.4	30.2	39.8	31.5
High-Risk Blood Pressure						
Normal	78.0	67.1	76.2	78.3	66.2	76.6
High-risk	22.0	32.9	23.8	21.7	33.8	23.4
High-Risk Pulse Rate						
Normal	82.6	81.3	82.4	82.6	78.6	82.1
High-risk	17.4	18.7	17.6	17.4	21.4	17.9
Height (m)						
Mean (SE)	1.650 (0.004)	1.515 (0.005)	1.628 (0.005)	1.651 (0.004)	1.515 (0.007)	1.633 (0.005)
≤10th percentile						
No	92.7	92.5	92.7	93.4	94.4	93.5
Yes	7.3	7.5	7.4	6.6	5.6	6.5
≤25th percentile						
No	80.5	82.9	80.9	81.1	83.5	81.5
Yes	19.5	17.1	19.1	18.9	16.5	18.6
Chronic Conditions						
Hypertension						
No	79.6	68.9	77.9	80.0	65.3	78.0
Yes	20.4	31.1	22.1	20.0	34.7	22.0

Diabetes						
No	86.5	86.9	86.6	87.6	88.5	87.7
Yes	13.5	13.1	13.4	12.4	11.5	12.3
Angina						
No	89.8	95.8	90.8	89.1	95.6	90.0
Yes	10.2	4.2	9.2	10.9	4.4	10.0
Stroke						
No	97.6	99.7	98.0	97.7	100.0	98.0
Yes	2.4	0.3	2.0	2.3	0.0	2.0
Chronic Lung Disease						
No	94.0	97.8	94.6	93.6	96.8	94.1
Yes	6.0	2.2	5.4	6.4	3.2	5.9
Arthritis						
No	88.0	70.7	85.2	88.2	66.2	85.3
Yes	12.0	29.3	14.8	11.8	33.8	14.7
Asthma						
No	94.8	92.0	94.3	94.8	90.2	94.2
Yes	5.2	8.0	5.7	5.2	9.8	5.8

[a] > 102 cm (males), > 88 cm (females)
[b] ≥ 90 cm (males), ≥ 80 cm (females)
[c] ≥ 87 cm (males), ≥ 82 cm (females)

Table A3.5. Odds Ratios from Logistic Regression Models Predicting Measured Health Outcomes (Part A) and Self-Reported Chronic Conditions (Part B), Males and Females Aged 50+, South Africa (2007-2008)

Part A: Measured Health Outcomes					
	Summer	**Autumn**	**Winter**	**Spring**	*N*
Obese (BMI ≥ 30 kg/m2)					
M1	1.23	1.00	1.17	1.27	1331
M5	1.24	1.00	1.13	1.19	1331
High-Risk Waist Circumference					
M1	1.33	1.00	1.06	0.76	1331
M5	1.34	1.00	1.13	0.77	1331
High-Risk Waist-to-Hip Ratio					
M1	1.07	1.00	1.35	1.61+	1331
M5	1.08	1.00	1.31	1.56+	1331
High-Risk Systolic Blood Pressure					
M1	1.33	1.00	0.96	0.97	1331
M5	1.25	1.00	0.94	0.94	1331
High-Risk Diastolic Blood Pressure					
M1	1.23	1.00	1.10	1.50	1331
M5	1.10	1.00	1.06	1.44	1331
High-Risk Blood Pressure					
M1	1.53	1.00	1.21	1.23	1331
M5	1.44	1.00	1.21	1.22	1331
High-Risk Pulse Rate					
M1	2.09*	1.00	1.39	1.02	1331
M5	2.05*	1.00	1.41	1.03	1331
Height, ≤10th Percentile					
M1	2.25*	1.00	1.76	1.93	1331
M5	2.29*	1.00	1.90+	2.35*	1331
Height, ≤25th Percentile					
M1	1.03	1.00	1.76*	1.19	1331
M5	0.98	1.00	1.66+	1.07	1331

Part B: Self-Reported Chronic Conditions					
	Summer	**Autumn**	**Winter**	**Spring**	*N*
Hypertension					
M1	1.01	1.00	0.88	0.68	1331
M5	0.94	1.00	0.85	0.61+	1331

Diabetes					
M1	1.45	1.00	1.62	1.09	1331
M5	1.26	1.00	1.48	0.91	1331
Angina					
M1	0.98	1.00	0.91	0.41	1331
M5	0.81	1.00	0.90	0.42+	1331
Stroke					
M1	1.52	1.00	0.50	0.76	1331
M5	1.20	1.00	0.36+	0.56	1331
Chronic Lung Disease					
M1	0.64	1.00	0.89	1.26	1331
M5	0.58	1.00	1.05	1.17	1331
Arthritis					
M1	1.17	1.00	1.47	1.46	1331
M5	0.98	1.00	1.35	1.17	1331
Asthma					
M1	0.78	1.00	1.34	1.14	1331
M5	0.78	1.00	1.16	1.18	1331

+ p <0.10, * p < 0.05, ** p < 0.01, *** p < 0.001

Notes: Model 1 includes controls for age and sex. Model 5 adds region, father's education, father's occupation, and respondent's education. See **Table 3.3** for the high-risk cutpoints.

Table A3.6. Odds Ratios from Logistic Regression Models Predicting Measured Health Outcomes (Part A) and Self-Reported Chronic Conditions (Part B), Males and Females Aged 50+, Ghana (2007-2008)

Part A: Measured Health Outcomes					
	Winter	**Spring**	**Summer**	**Autumn**	*N*
Obese (BMI ≥ 30 kg/m2)					
M1	1.12	1.86+	1.36	1.00	838
M5	1.12	1.49	1.15	1.00	838
High-Risk Waist Circumference					
M1	1.28	1.13	1.21	1.00	838
M5	1.20	0.84	1.00	1.00	838
High-Risk Waist-to-Hip Ratio					
M1	0.79	0.81	0.96	1.00	838
M5	0.74	0.74	0.89	1.00	838
High-Risk Systolic Blood Pressure					
M1	1.20	1.22	1.17	1.00	838
M5	1.12	1.10	1.05	1.00	838
High-Risk Diastolic Blood Pressure					
M1	1.36	1.41	1.60*	1.00	838
M5	1.30	1.29	1.53+	1.00	838
High-Risk Blood Pressure					
M1	1.23	1.35	1.21	1.00	838
M5	1.18	1.22	1.09	1.00	838
High-Risk Pulse Rate					
M1	0.61	1.26	1.05	1.00	838
M5	0.63	1.39	1.18	1.00	838
Height, ≤10th Percentile					
M1	0.91	1.30	0.80	1.00	838
M5	1.03	1.42	0.89	1.00	838
Height, ≤25th Percentile					
M1	1.10	1.05	0.86	1.00	838
M5	1.11	1.11	0.88	1.00	838

Part B: Self-Reported Chronic Conditions					
	Winter	**Spring**	**Summer**	**Autumn**	*N*
Hypertension					
M1	1.36	1.13	1.17	1.00	838
M5	1.31	0.90	1.00	1.00	838

Diabetes					
M1	0.48	1.66	0.35*	1.00	838
M5	0.42	1.60	0.30*	1.00	838
Angina					
M1	0.43	0.42	0.54	1.00	838
M5	0.41	0.48	0.51	1.00	838
Stroke					
M1	1.20	1.08	0.71	1.00	838
M5	1.13	1.17	0.61	1.00	838
Chronic Lung Disease					
M1					
M5					
Arthritis					
M1	0.91	0.74	0.92	1.00	838
M5	0.87	0.72	0.91	1.00	838
Asthma					
M1	1.47	1.14	0.32	1.00	838
M5	1.75	1.49	0.36	1.00	838

+ p <0.10, * p < 0.05, ** p < 0.01, *** p < 0.001

Notes: Model 1 includes controls for age and sex. Model 5 adds region, father's education, father's occupation, and respondent's education. See **Table 3.3** for the high-risk cutpoints. Chronic lung disease is omitted due to very low incidence of this condition in this sample.

Table A3.7. Odds Ratios from Logistic Regression Models Predicting Measured Health Outcomes (Part A) and Self-Reported Chronic Conditions (Part B), Males and Females Aged 50+, Russia (2007-2010)

Part A: Measured Health Outcomes					
	Winter	**Spring**	**Summer**	**Autumn**	*N*
Obese (BMI ≥ 30 kg/m^2)					
M1	0.83	1.22	0.66	1.00	2260
M5	0.95	1.28	0.73	1.00	2260
High-Risk Waist Circumference					
M1	0.44*	0.86	0.47*	1.00	2260
M5	0.43*	0.85	0.46*	1.00	2260
High-Risk Waist-to-Hip Ratio					
M1	0.67	1.09	0.67	1.00	2260
M5	0.63	1.12	0.68	1.00	2260
High-Risk Systolic Blood Pressure					
M1	0.99	1.07	0.91	1.00	2260
M5	1.00	1.12	0.91	1.00	2260
High-Risk Diastolic Blood Pressure					
M1	1.28	1.16	0.80	1.00	2260
M5	1.45	1.22	0.82	1.00	2260
High-Risk Blood Pressure					
M1	0.87	0.88	0.69	1.00	2260
M5	0.92	0.92	0.71	1.00	2260
High-Risk Pulse Rate					
M1	0.74	1.66	1.32	1.00	2260
M5	0.77	1.78	1.89	1.00	2260
Height, ≤10th Percentile					
M1	1.21	1.25	1.02	1.00	2260
M5	1.12	1.22	1.01	1.00	2260
Height, ≤25th Percentile					
M1	2.06*	1.39	1.18	1.00	2260
M5	2.01*	1.43	1.14	1.00	2260

Part B: Self-Reported Chronic Conditions					
	Winter	**Spring**	**Summer**	**Autumn**	*N*
Hypertension					
M1	0.94	1.14	1.48	1.00	2260
M5	1.02	1.22	1.65	1.00	2260
Diabetes					
M1	1.44	2.81***	1.39	1.00	2260
M5	1.63	3.02***	1.52	1.00	2260

Angina					
M1	1.57	1.59	2.48**	1.00	2260
M5	1.69	1.54	2.72**	1.00	2260
Stroke					
M1	2.76	5.16**	1.95	1.00	2260
M5	2.92	5.41***	2.20	1.00	2260
Chronic Lung Disease					
M1	0.93	0.74	1.92	1.00	2260
M5	0.97	0.78	2.34*	1.00	2260
Arthritis					
M1	0.79	1.10	1.05	1.00	2260
M5	0.77	1.08	1.01	1.00	2260
Asthma					
M1	0.34+	0.20*	1.06	1.00	2260
M5	0.50	0.29+	1.77	1.00	2260

+ p <0.10, * p < 0.05, ** p < 0.01, *** p < 0.001

Notes: Model 1 includes controls for age and sex. Model 5 adds region, father's education, father's occupation, and respondent's education. See **Table 3.3** for the high-risk cutpoints.

Table A3.8. Odds Ratios from Logistic Regression Models Predicting Measured Health Outcomes (Part A) and Self-Reported Chronic Conditions (Part B), Males and Females Aged 50+, Mexico (2009-2010)

Part A: Measured Health Outcomes					
	Winter	**Spring**	**Summer**	**Autumn**	*N*
Obese (BMI ≥ 30 kg/m2)					
M1	1.84	1.44	2.06	1.00	937
M5	1.74	1.45	1.89	1.00	937
High-Risk Waist Circumference					
M1	1.54	1.51	1.60	1.00	937
M5	1.45	1.81	1.76	1.00	937
High-Risk Waist-to-Hip Ratio					
M1	0.75	0.99	0.97	1.00	937
M5	0.90	1.68	1.07	1.00	937
High-Risk Systolic Blood Pressure					
M1	0.47	0.48	0.81	1.00	937
M5	0.50	0.72	1.28	1.00	937
High-Risk Diastolic Blood Pressure					
M1	1.18	1.12	1.37	1.00	937
M5	1.19	1.35	1.43	1.00	937
High-Risk Blood Pressure					
M1	1.31	1.39	1.50	1.00	937
M5	1.37	1.60	1.78	1.00	937
High-Risk Pulse Rate					
M1	1.79	0.43	1.08	1.00	937
M5	1.79	0.23	0.93	1.00	937
Height, ≤10th Percentile					
M1	3.57+	1.33	1.84	1.00	937
M5	3.89+	1.60	2.21	1.00	937
Height, ≤25th Percentile					
M1	2.43+	2.28	2.91+	1.00	937
M5	2.58	1.81	2.77+	1.00	937

Part B: Self-Reported Chronic Conditions					
	Winter	**Spring**	**Summer**	**Autumn**	*N*
Hypertension					
M1	1.52	1.66	0.87	1.00	937
M5	1.60	1.70	1.02	1.00	937

Diabetes					
M1	0.42+	0.64	0.22**	1.00	937
M5	0.33*	0.25**	0.15***	1.00	937
Angina					
M1					
M5					
Stroke					
M1	0.79	0.57	0.44	1.00	937
M5	0.58	0.35	0.36	1.00	937
Chronic Lung Disease					
M1	2.47	3.34	13.64***	1.00	937
M5	2.42	5.67+	15.72**	1.00	937
Arthritis					
M1	0.84	0.26*	0.53	1.00	937
M5	0.99	0.33+	0.63	1.00	937
Asthma					
M1					
M5					

+ p <0.10, * p < 0.05, ** p < 0.01, *** p < 0.001

Notes: Model 1 includes controls for age and sex. Model 5 adds region, father's education, father's occupation, and respondent's education. See **Table 3.3** for the high-risk cutpoints. Angina and asthma are omitted due to very low incidence of these conditions in this sample.

Table A3.9. Odds Ratios from Logistic Regression Models Predicting Measured Health Outcomes (Part A) and Self-Reported Chronic Conditions (Part B), Males and Females Aged 50+, China (2007-2010)

Part A: Measured Health Outcomes					
	Winter	**Spring**	**Summer**	**Autumn**	*N*
Obese (BMI ≥ 30 kg/m2)					
M1	0.71+	0.65**	1.03	1.00	5975
M5	0.70+	0.66**	0.99	1.00	5975
High-Risk Waist Circumference					
M1	0.91	0.80	0.89	1.00	5975
M5	0.92	0.82	0.85	1.00	5975
High-Risk Waist-to-Hip Ratio					
M1	1.16	1.10	1.14	1.00	5975
M5	1.19	1.10	1.13	1.00	5975
High-Risk Systolic Blood Pressure					
M1	0.86	0.76**	0.78*	1.00	5975
M5	0.83+	0.77**	0.77*	1.00	5975
High-Risk Diastolic Blood Pressure					
M1	0.89	0.86*	0.95	1.00	5975
M5	0.91	0.88*	0.97	1.00	5975
High-Risk Blood Pressure					
M1	0.83*	0.80**	0.87+	1.00	5975
M5	0.83*	0.81**	0.87	1.00	5975
High-Risk Pulse Rate					
M1	0.90	0.94	0.96	1.00	5975
M5	0.90	0.94	0.98	1.00	5975
Height, ≤10th Percentile					
M1	0.81	0.90	0.93	1.00	5975
M5	0.82	0.88	0.97	1.00	5975
Height, ≤25th Percentile					
M1	0.89	0.94	1.05	1.00	5975
M5	0.88	0.91	1.07	1.00	5975

Part B: Self-Reported Chronic Conditions					
	Winter	**Spring**	**Summer**	**Autumn**	*N*
Hypertension					
M1	0.88+	0.82**	0.97	1.00	5975
M5	0.88+	0.84*	0.97	1.00	5975

Diabetes					
M1	0.87	0.78	1.02	1.00	5975
M5	0.88	0.79	1.01	1.00	5975
Angina					
M1	0.92	0.82	1.08	1.00	5975
M5	0.92	0.81	1.06	1.00	5975
Stroke					
M1	1.00	1.29	1.02	1.00	5975
M5	0.99	1.29	1.00	1.00	5975
Chronic Lung Disease					
M1	1.16	1.20	1.20	1.00	5975
M5	1.15	1.16	1.24	1.00	5975
Arthritis					
M1	0.99	1.18	0.96	1.00	5975
M5	0.99	1.20	0.96	1.00	5975
Asthma					
M1	0.81	0.74	0.77	1.00	5975
M5	0.76	0.70	0.76	1.00	5975

+ p <0.10, * p < 0.05, ** p < 0.01, *** p < 0.001

Notes: Model 1 includes controls for age and sex. Model 5 adds region, father's education, father's occupation, and respondent's education. See **Table 3.3** for the high-risk cutpoints.

Table A3.10. Odds Ratios from Logistic Regression Models Predicting Measured Health Outcomes (Part A) and Self-Reported Chronic Conditions (Part B), Males and Females Aged 50+, India (2007-2008)

Part A: Measured Health Outcomes					
	Winter	**Summer**	**Monsoon**	**Post Monsoon**	*N*
Obese (BMI ≥30 kg/m^2)					
M1	0.49	1.65	1.00	1.04	1376
M5	0.50	1.95	1.00	1.21	1376
Obese (BMI ≥25 kg/m^2)					
M1	1.08	1.16	1.00	0.91	1376
M5	1.09	1.14	1.00	0.87	1376
Obese (BMI ≥23 kg/m^2)					
M1	1.00	0.94	1.00	0.82	1376
M5	1.01	0.88	1.00	0.74	1376
High-Risk Waist Circumference[a]					
M1	1.00	1.14	1.00	1.42	1376
M5	1.11	1.30	1.00	1.42	1376
High-Risk Waist Circumference[b]					
M1	0.83	0.81	1.00	1.10	1376
M5	0.81	0.79	1.00	1.06	1376
High-Risk Waist Circumference[c]					
M1	0.73	0.99	1.00	1.21	1376
M5	0.75	1.03	1.00	1.22	1376
High-Risk Waist-to-Hip Ratio					
M1	1.15	1.00	1.00	1.23	1376
M5	1.13	0.88	1.00	1.11	1376
High-Risk Systolic Blood Pressure					
M1	1.38	1.57+	1.00	1.22	1376
M5	1.38	1.37	1.00	1.07	1376
High-Risk Diastolic Blood Pressure					
M1	1.40	1.58+	1.00	1.22	1376
M5	1.48	1.46	1.00	1.18	1376
High-Risk Blood Pressure					
M1	1.59+	1.75*	1.00	1.38	1376
M5	1.61*	1.55+	1.00	1.22	1376
High-Risk Pulse Rate					
M1	1.27	2.21**	1.00	1.10	1376
M5	1.33	2.19**	1.00	1.16	1376
Height, ≤10th Percentile					
M1	1.48	1.17	1.00	1.73	1376
M5	1.70	1.00	1.00	1.62	1376

Height, ≤25th Percentile					
M1	1.12	0.99	1.00	1.38	1376
M5	1.24	0.86	1.00	1.27	1376

Part B: Self-Reported Chronic Conditions					
	Winter	**Summer**	**Monsoon**	**Post Monsoon**	*N*
Hypertension					
M1	1.18	1.29	1.00	1.23	1376
M5	1.14	1.13	1.00	1.15	1376
Diabetes					
M1	0.46*	0.67	1.00	0.58	1376
M5	0.42*	0.54	1.00	0.49*	1376
Angina					
M1	0.60	1.12	1.00	1.16	1376
M5	0.77	1.34	1.00	1.29	1376
Stroke					
M1	4.88*	2.52	1.00	2.85	1376
M5	4.77+	2.03	1.00	2.31	1376
Chronic Lung Disease					
M1	1.22	3.98*	1.00	1.06	1376
M5	1.12	4.65**	1.00	1.12	1376
Arthritis					
M1	1.03	0.77	1.00	0.65	1376
M5	0.90	0.62+	1.00	0.68	1376
Asthma					
M1	1.80	1.08	1.00	1.76	1376
M5	2.18*	1.21	1.00	2.10	1376

+ p <0.10, * p < 0.05, ** p < 0.01, *** p < 0.001

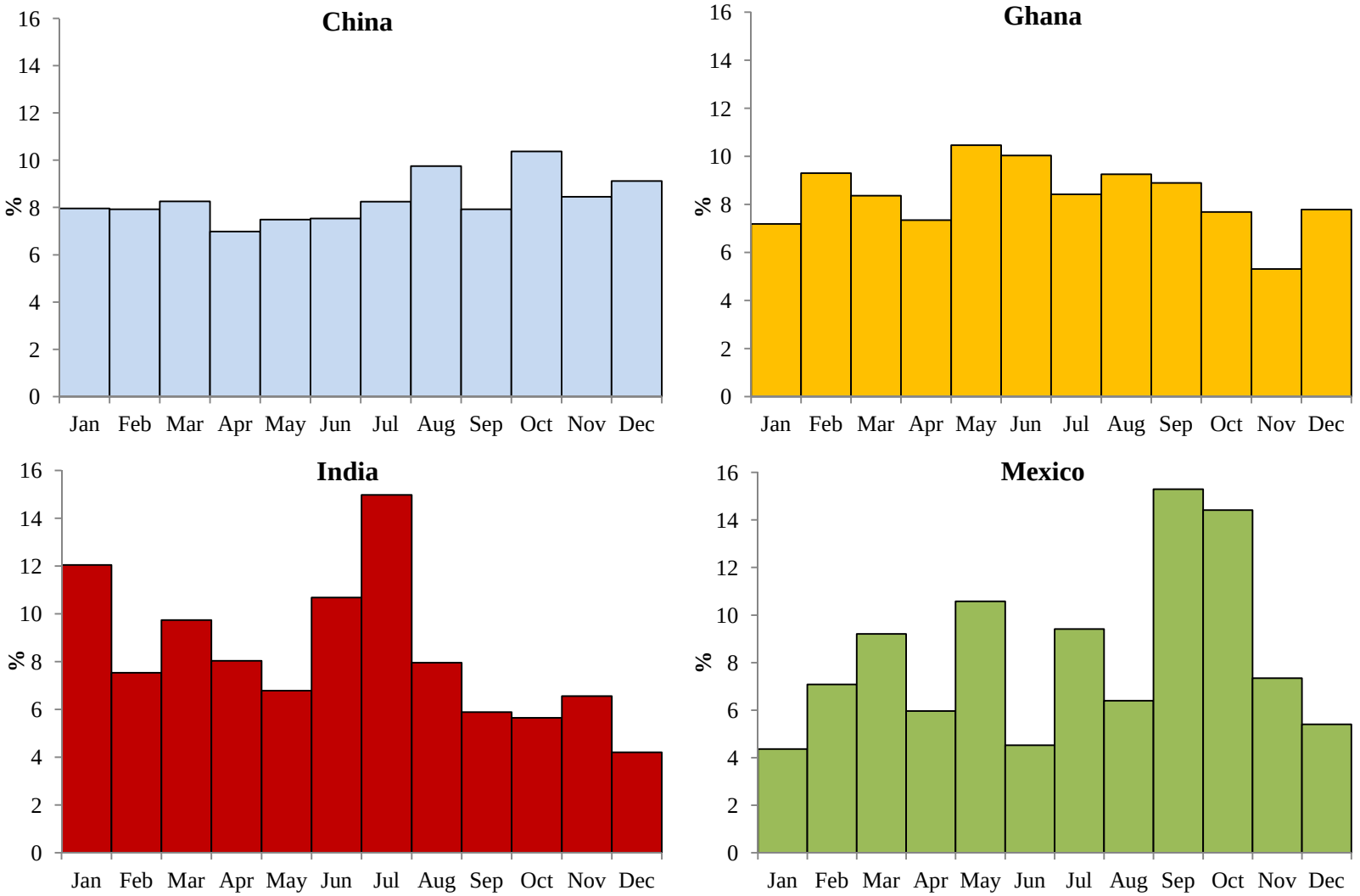
Notes: Model 1 includes controls for age and sex. Model 5 adds region, father's education, father's occupation, and respondent's education. See **Table 3.3** for the high-risk cutpoints.

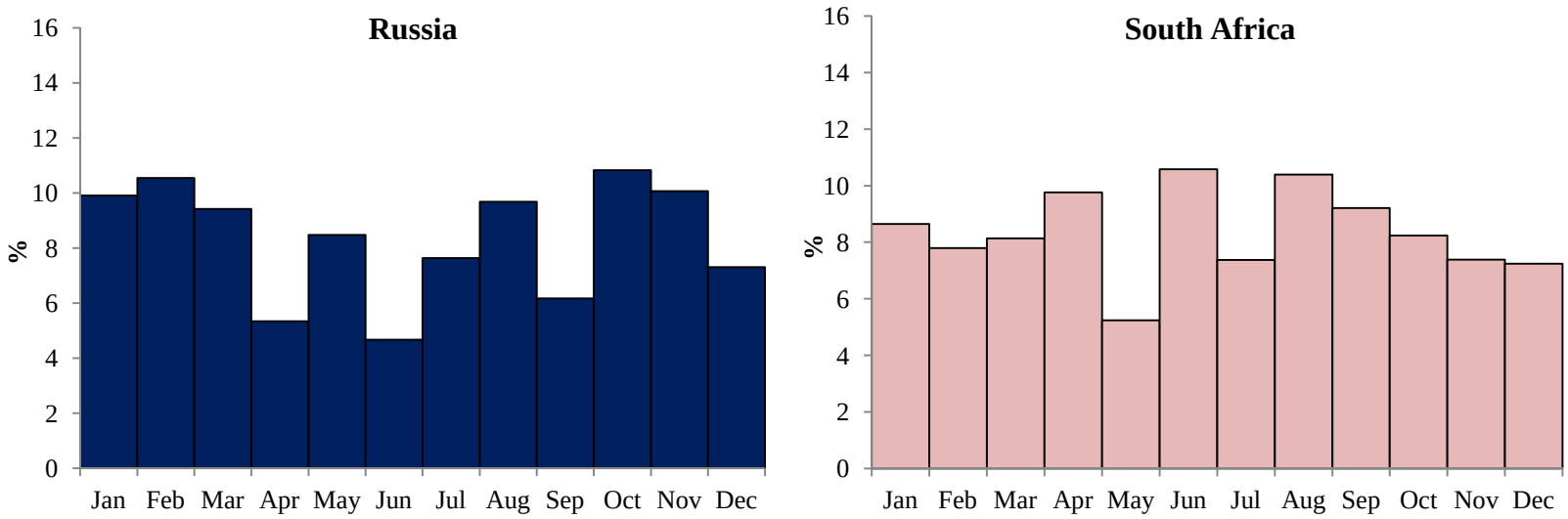
[a] > 102 cm (males), > 88 cm (females)

[b] ≥ 90 cm (males), ≥ 80 cm (females)

[c] ≥ 87 cm (males), ≥ 82 cm (females)

Figure A3.1. Month of Birth Distribution, Males and Females Combined, Ages 50+, SAGE 2007-2010





Note: Figures are based on samples of individuals aged 50+ non-missing on key characteristics of interest (see text and **Tables A3.1-A3.4** for summary statistics and sample sizes).

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