Is there a US Mortality Advantage at Older Ages?[1]

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Abstract

Americans experience higher mortality than their peers in other high-income countries for most of the life course, but recent work has shown that at the oldest ages they experience a mortality advantage—a phenomenon we call the "US mortality crossover." In this paper we document the crossover and time trends thereof. We find that the age of crossover increases linearly by about 0.5 years per year, a pattern of changes that, to our knowledge, has not been identified before. We then interrogate several potential explanations for a steadily increasing crossover age. While none is completely satisfactory, we rule out differential age misstatement, selection, and access to and quality of health care. We find that the most plausible explanation involves the deleterious effects of differential smoking patterns working through the life table.

1 Introduction

Recent reports by the National Academy of Sciences argue convincingly that Americans experience shorter lives and worse health than their peers in other high-income countries (Crimmins et al., 2011a,b; Woolf and Aron, 2013). This disadvantage is visible in mortality, across a broad array of health status measures, and (with one exception) throughout the life cycle (Woolf and Aron, 2013). The exception is that Americans fare relatively better if they survive to older ages. Whereas conditional life expectancies at ages 50-65 years are lower than in peer countries (World Health Organization, 2013), at very old ages (70-75 and 75-79 years) Americans have better survival prospects (Crimmins et al., 2011a,b; Ho, 2013; Ho and Preston, 2010). This advantage is a singularity in an otherwise bleak and persistent landscape of US unfavorable mortality and deserves special consideration. How large is this later-life advantage and how old is the population that experiences it? Is this an enduring feature of US adult mortality or is it a recent phenomenon? How can it be explained?

In this paper we estimate the magnitude of differences in mortality at older ages between the US and peer countries, trace the history of such differences, and test alternative hypotheses that account for them. We find that throughout the 55-year period examined here (1955-2010) there is no consistent US superiority in survival at older ages. The age after which US mortality is lower (i.e., better survival) than in peer countries has increased linearly since the late 1970's, is rapidly converging to ages that few people will ever attain in any population, and is associated with the passage and extinction of some cohorts, possibly connected to smoking histories. The details of this finding have not been thoroughly documented before and it is a rather baffling empirical regularity if only because its occurrence has important implications for the nearly two-thirds of newborns who will survive to age 75 (Arias et al., 2010).

2 Mortality in the US and other high-income countries

Although this is well trodden territory, we briefly review recent findings, use them to establish less well-known facts, and move on to describe the empirical regularities peculiar to US mortality at very old ages.

2.1 Previous research: US disadvantages and advantages throughout life

There is no clear consensus in the literature on the mortality crossover regarding the age beyond which the US older population experiences lower mortality. In some accounts this age is within the interval 80 to 85 (Manton and Vaupel, 1995), in others in the age group 70-75 (Crimmins et al., 2011a,b; Ho and Preston, 2010), and in others it is believed to be as low as 65, at least for females (Ho, 2013). Admittedly, even if the

US older population experiences better mortality conditions at older ages, there could be some variability in the crossover age as a result of transient period effects, changes in the composition of older cohorts, vagaries of measurement of mortality at older ages, and variability of mortality rates in benchmark countries. The estimates identified above span a wide range, perhaps the result of using estimates for different time periods or different data sources, and should be explained along with the survival advantage itself. We argue below that systematic variation in the crossover age—be it in the form of increases, decreases, or cyclical fluctuations—shifts the phenomenon that requires explanation: it is not just why Americans have experienced favorable conditions at older ages but, rather, why is it that the age at which these conditions prevail increases over time?

Before proceeding as if the US mortality crossover were an enduring feature, a first order task is to compute robust estimates of the crossover age over a long time interval, verify its existence, and ascertain the existence of time trends, if any. In what follows we use a standard database to show that the crossover age is not constant and instead increases linearly for males and females starting in the late 1970s. Systematic increases in the crossover age suggest that the US old age advantage is an evanescent phenomenon not a feature persistently engraved in US adult mortality trends. This finding adds a puzzling trait to those already uncovered by past empirical research and requires its own *sui generis* explanation.

2.2 Data and Measures

As most other research in this area, we use country age-specific deaths and population estimates from the Human Mortality Database (HMD) for the years 1955-2010, separately by sex. These data include information for the US and a set of 16 high-income comparison countries: Australia, Austria, Canada, Denmark, Finland, France, Germany, Italy, Japan, Netherlands, Norway, Portugal, Spain, Sweden, Switzerland, and United Kingdom.[1] There are a few instances of incomplete or missing data for a given year or small set of years. We correct these issues by applying the data from the nearest year. These corrections streamline our analyses but have virtually no effect on results.[2] For Germany we take data from West Germany (Former Federal Republic) through 1990 and then from 1991 to 2010 the current Federal Republic of Germany.

To establish the relative position of the US we use the ratios of US mortality rates to those in a benchmark for comparison. The use of mortality ratios versus ranks is important since the analysis of crossover ages requires computation of magnitudes of differences, a dimension blurred by indices based on rank order. We create a year- and age-specific "superpopulation," the pooled composite of all countries except the US.

[1] This set of countries included in the benchmark was chosen to be comparable to recent NAS reports (Crimmins et al., 2011a,b; Woolf and Aron, 2013) and other works (i.e., Ho and Preston (2010)).

[2] HMD corrections: Germany (1955 from 1956); Australia, Canada, Finland, Italy, Japan, Netherlands, Norway, Spain (2010 from 2009).

This population combines the mortality experiences of peer nations. This pooled composite is our primary reference population or benchmark. Our main indicators are mortality rates in the US, M_x, and functions of these (residual life expectancies at various ages), mortality rates in the pooled average, PM_x, and the ratios $RM_x = M_x/PM_x$. Ratios PR_x above 1 reflect higher US mortality rates than the benchmark and values below 1 correspond to lower US mortality rates.[3]

2.3 General results

We begin with a brief review of findings reported elsewhere. Figure 1 displays the difference between male and female life expectancy at birth relative to the pooled benchmark (i.e., pooled life expectancy at birth minus US life expectancy at birth; negative values indicate US *advantage* and positive values US *disadvantage*). To provide a sense of magnitude, the plot of these differences is bounded by plots of differences between the US life expectancies and those in an optimal benchmark life table (upper curve) and the worst benchmark life table (lower curve).[4] There is no evidence of a US disadvantage in 1955-60. Quite the contrary, in the mid-1950s the US female and male life expectancy were roughly 1.5 years and 1 year higher that the pooled benchmarks. This favorable situation reverses by 2010 as both US males and females trail by about 2 and 3 years respectively, a change that results from *deceleration* of life expectancy gains of about 0.08 years of life expectancy per year. The deceleration occurs gradually and triggers a convergence of US life expectancy at birth toward the worst benchmark.

The differences in Figure 1 are not the result of poor performance of a single or even a handful of age groups but rather reflect a pervasive contrast over the life span (Woolf and Aron, 2013). Figure 2 displays differences in life expectancy at age 50 relative to the pooled, worst, and optimal benchmarks. Note that the US male disadvantage with respect to peers at age 50 is already in place by the late 1950s (with a minor reprieve in the late 1970s) whereas among females it emerges only in the mid-1980s but grows much faster than that of males thereafter. As with life expectancy at birth, there is a wholesale, steady deterioration that has lasted thirty years.

A comparison of temporary life expectancy in the age interval 0 to 50 (not shown) reveals similar features, albeit with reduced magnitudes. US male and female temporary life expectancies in the interval 0-50 trail the pooled benchmarks starting as early as in 1965 and their relative contribution to differences in life

[3] We are cognizant that when rates attain very low values minor differences between observed and pooled rates can produce large ratios. However, since our analyses are mostly focused on ages at which the rates are high and growing, this drawback of the ratio index is less of a concern. Other alternative indicators are considerably more problematic (Woolf and Aron, 2013).

[4] The "optimal" life table for each year is constructed by chaining together the lowest age specific mortality rates (terminated with the 110+ age group) in the set of countries we use for comparison. The "worst" life table is similarly constructed using the highest age specific mortality rates. These two life tables are the boundaries of the space occupied by all empirical life tables. Like the pooled benchmark life table, neither the "optimal" nor the "worst" life tables correspond to the experience of a real country.

expectancy at birth decreases over time. Thus, although deceleration of survival occurs at all ages, those involving early childhood and adulthood (ages 0-49) are ubiquitous throughout the period under study but become gradually smaller whereas those that apply to ages over 50 are of more recent vintage but exert a heavier tug in the last two decades or so.

Figure 3 displays age-specific ratios (RM_x) of mortality rates observed in the US to those in the pooled benchmark for selected years by sex. It shows a consistent pattern reproduced over time and characterized by three features. First, the US disadvantage between ages 35 and 60 has been increasing for well over 50 years. Second, the US disadvantage at early ages (before age 10) did not exist before 1975 but grew rapidly since then and spread to other age groups, particularly 10 to 50. Third, the US does indeed experience an advantage at older ages but with a perverse twist: over time, the advantage in this age group is progressively displaced toward older ages and more so among females. If past trends were to continue, the crossover age would be over 100 by the year 2020 and thus only a lucky few US older people will enjoy it.

2.4 US advantage at older ages: A shifting landscape

A comparison of time trends of differences in life expectancy at age 75 is useful to summarize the nature of the advantage enjoyed by Americans older than 50. Figure 5 displays the differences in life expectancy at age 75 relative to the pooled benchmark for the entire period under study.[5] Initially the US enjoyed an advantage equivalent to about 1 extra year of life expectancy at age 75 for both males and females; this amounts to approximately 13% of the average residual expected years of life after age 75. Over time, however, the US position gradually deteriorates and by 2008-2010 there is scarcely any advantage left for females and no advantage for males. Figure 5 is consistent with a progressive *relative* deterioration of US mortality rates at older ages and an upward displacement of the crossover age.

To obtain estimates of crossover ages for the time period examined we use a simple procedure that consists of computing crossover ages for each year using simple linear interpolation of the observed ratios. For a few years there are multiple crossover ages contained in narrow ranges, and in those cases we use the median values of the ranges.[6] We then fit a cubic spline to the interpolated and median values and generate a smoothed time trend of crossover ages for the time interval 1955-2010. Figure 6 displays both the observed median and the fitted values of crossover ages for males and females. The fitted trends are smoother and more regular than the computed values, but the inferences from each set are the same: prior to 1970 crossover ages are nearly invariant and from 1970 onward there is a sharp, unmistakable increasing trend.

[5] We use e_{75} because it is a good summary indicator of M_x for $x \geq 75$. However, the regularities we find are, with some minor variation, applicable to all M_x after age 75.

[6] In figures not shown we plot the median, maximum and minimum values for years with multiple crossovers. Since the frequency of years with multiple crossover ages is small and the ranges, when they apply, are very narrow we only examine the median values.

Although the male and female patterns are not identical, gender differences are subdued. The smoothed trend for males implies that the crossover age is increasing at a rate of about 0.6 years per year whereas among females the rate of increase is about 0.4 years per year. If these trends persists into the future, the crossover age will become 100 years in the year 2020 (approximately).

A systematically increasing crossover age is a new feature added to those uncovered by past empirical research. These findings usually imply either an unchanging crossover age or one that varies but follows no systematic trend that could translate into a stronger or weaker advantage for the US (Crimmins et al., 2011a,b). Past literature on the subject recognizes some variability in the crossover age, but in all cases the analyses rest on the premise that while the US has done badly at ages over 50, it has done much better at ages over 75 or so. Previous research on the subject attempts to explain this pattern but, in general, does not identify or explain the fact that the segment of the life span where there is an advantage is shrinking—and rapidly so. That the landscape of mortality advantages and disadvantages at older ages is shifting is important for two reasons. First, because it has tangible implications for the growing number of older people who will become members of cohorts implicated by the phenomenon. Second, because it alters the grounds for hypothesis testing as it automatically rules out explanations that are plausible *only when the crossover age is time invariant*. That is, we make the conceptual distinction between factors that may explain the existence of a crossover from factors that may explain *a systematically increasing crossover*.

3 Explanations of a shifting crossover age

Why should a crossover age exist at all? Why should the US experience better mortality at ages that become progressively older? The most obvious possibility is that the entire pattern of RM_x ratios shifts upwards every year more or less uniformly at all ages. This, in turn, points to the presence of an exogenous force that worsens the US *relative* mortality experience compared to peer countries at all ages.[7] The evidence examined before does indeed suggest the existence of such a process (see Figure 3). This is consistent with a second interpretation, namely, that in addition to the impact of forces that retard US progress in mortality at younger ages, there are other factors that slow-down the relative progress of survival at very old ages and that the strength of these effects intensifies over time.

In what follows we review five alternative hypotheses that could plausibly account for the crossover phenomenon.[8] The first, changing age overstatement at older ages, interprets the crossover as a product of data errors that become less serious over time. The second, within-cohort selection, rests on the idea that

[7] Because our evaluation rests on a comparison between the US and a potentially changing benchmark, US progress or deterioration is always a relative matter. If the US relative condition worsens it may be because the US experiences shocks that peer countries do not or because peer countries gain from sources that the US is not exposed or cannot take advantage.

[8] We investigate these possible explanations individually, but acknowledge that one or more may be operating together.

the observed increasing crossover age is a result of changing composition of cohorts by frailty and that these changes are associated with past mortality improvements. The final three explanations identify mechanisms that affect the health status of older individuals, alter exposure to illnesses, or enhance the capacity to resist and/or recover from chronic conditions.

3.1 Age misstatement

It is known that US mortality rates at older ages contain downward biases due to overstatement of ages of population and deaths. Identification of these biases has proven useful to partially account for the so-called Black-White mortality crossover (Coale and Kisker, 1986; Condran et al., 1991; Hill et al., 2000; Preston et al., 1996; Rosenwaike, 1981). Although some degree of age overstatement is also present in European countries that rely on vital registrations and censuses (but much less so in countries with population registries) errors seem to be of either smaller magnitude (Condran et al., 1991) or affect a much older population than in the US. To the extent that US benchmark differentials in age overstatement bias are of some consequence at older ages, a crossover of the mortality rates (M_x's) is possible since we would observe deceivingly low values of M_x's for the US and, consequently, RM_x ratios lower than 1—or at least downward biased. This could explain the sustained US old age advantage over peer countries. But could this explanation account for the *increasing trend* of the age above which the US experiences mortality advantage? We do not think that is the case for two reasons. First, because the biases associated with age overstatement are influential only at ages over 70 or 75, the age overstatement biases cannot account for time trends of crossing ages before 1990 or so (see Figure 3). Second, for years after 1990 it could only be a satisfactory explanation if the propensity toward age overstatement decreases in the US *more rapidly* than it does in peer countries, a result perhaps of improved age declaration in population censuses and ages at death in vital registration in the US relative to peer countries.

3.1.1 A simple counterfactual

An empirical test of this possibility is demanding, for it requires estimates of time trends of biases in older age mortality rates in the US and in countries included in the benchmark. In lieu of this, we compute a counterfactual for the period 1995-2010 and evaluate its plausibility. To justify the counterfactual we use Figure 4 that displays mortality rates at ages 70 and over during the entire period under observation. The thick line in each graph traces the trajectory of mortality rates at the crossing ages for each year. Thus, for example, the mortality rates at the age of crossover in 1990 among females was of the order of 0.025 and in 2000 about 0.07 (for males it was 0.08 and 1.20 respectively). If more recent data are closer to the truth then

the true age of crossover throughout the period should have been close to 90—a scenario that essentially negates the existence of a persistent US health mortality advantage at older ages during the period under observation.[9] This means that mortality rates at ages below 90 years before 2010 must be biased downward by more a large amount. Thus, for example, the female mortality rates at ages 80 and 90 in 2005 were 0.07 and 0.15, respectively, implying shifts due to (relative) biases of the order of 2.1. It is unlikely that age overstatement in the US can produce errors of this magnitude.[10]

3.1.2 Medicare data

There is a more precise and convincing test we can carry out: to recalculate US mortality rates at older ages using Medicare registries where the likelihood of systematic age overstatement is low or non-existent.[11] The Medicare data available to us have two shortcomings. First, they only include the period 1993-2001, forcing us to focus on only a fraction of the time over which the crossover takes place. Second, the data we use exclude the Hispanic White (HW) population. This could cause a problem only if two conditions are met: the first is that there is a substantial fraction of the HW population at older ages and the second is that the HW population propensity to overestimate ages is higher than among the Non-Hispanic White (NHW) and African American (AA) populations. While there is some indirect evidence for the latter condition (Dechter and Preston, 1991; Palloni and Pinto, 2004), the first condition is not met as the fraction of HW among those older than 60 is considerably less than the fraction of AA population. We show elsewhere that the US crossover phenomenon takes place irrespective of whether or not we consider only NHW or both (the NHW and AA) populations (Palloni and Yonker, 2014). Figure 7 displays observed crossover ages by year after recalculating the RM_x ratios using Medicare data. We also show estimated values derived from fitting a model analogous to the one described above. Because for males we identify multiple crossover ages for some years, we use box plots centered on the median values and each spanning the entire range of estimates to provide an idea of the level of uncertainty. The graphs show that even if one uses the noisiest observed values (for males) there is still a marked increasing trend—which is confirmed by the model-based estimates. For females there is no uncertainty, as the estimated and observed values coincide almost perfectly, with a minor exception in the year 2001. Furthermore, the observed and fitted crossover ages are tightly associated: the R^2 of the relation between the two is about 0.95 for males and 0.94 for females.

To sum up: while there are admissible patterns of age overstatement that could partially explain time

[9] The conjecture of age overstatement as an explanation for an increasing crossover ages is inconsistent with a true age of crossover close to, say, 70, observed around 1980.

[10] Since the mortality rates in the benchmark are also likely to be downwardly biased, the errors in the US should induce biases that are equivalent to something less acute than halving the true rates.

[11] We are grateful to Felix Elwert and Elizabeth Wrigley-Field who provided us with the Medicare mortality rates we used in these computations.

trends in crossover ages during part of the time period examined, the counterfactual implies a rather extreme pattern of age exaggeration. More convincing is the empirical evidence from Medicare data—a data source that should be virtually immune to biases associated with age declaration—that produces results identical to those derived from the HMD life tables. We conclude that it is highly unlikely that the old age mortality crossover and the linear increase in the crossover age are artifacts of bad data.

3.2 Selection

Selection processes—whereby members of a birth cohort who survive to older ages are disproportionately drawn from among the sturdiest members—produce mortality patterns that rise less rapidly at older ages. Because all populations experience selection processes, any explanation for the pattern of US advantages/disadvantage relying on selection alone is tenable only if these processes are different or exaggerated/diminished in the US compared to peer countries.

The first mechanism that may produce this result is one whereby US selection processes become increasingly weaker over time (due to health and mortality improvements at younger ages among progressively more recent birth cohorts). If so, the pace of US mortality rate improvements at very old ages will decelerate and more so among younger cohorts. When a similar process is *not occurring at all* in peer countries, US mortality rates at older ages could exceed those elsewhere even if the average mortality patterns are slightly more favorable to the US population. The crossover age will, on average, increase in proportion to the rate of growth of the new birth cohorts that attain older ages with higher than average frailty levels. But if similar selection processes occur in other countries then the only way this mechanism could explain increasing crossover age is if selection in the US is relaxing more rapidly than in peer countries or, equivalently, that improvements in mortality at younger ages and experienced by the more recent birth cohorts that attain older ages took place more slowly (or with significant time lags) than in peer countries. The only way to verify that selection processes are weakening at a faster rate (or are taking place later) than in peer countries is to observe a declining time trend of the within-cohort correlation between mortality at older and younger ages.[12] There is no suitable information to confirm or refute that this pattern is in fact the one that prevails.

A second mechanism that could produce the same outcome involves the variance (and higher moments) of the frailty distribution at birth: the higher the variance, the stronger the force of selection and the lower the slope of old age mortality rate increases (Vaupel, 2010). To verify the presence of this mechanism we must show that the US population's frailty distribution at birth has larger variance than in peer countries. In addition, to explain the increasing trend in cross over ages we should prove that the frailty distribution

[12] In fact, if the idea is correct the correlation should gradually increase from negative values to positive values and should do so more rapidly in the US than elsewhere.

is either being reduced more rapidly in the US than in peer countries or, alternatively, that it is increasing less rapidly in the US than in peer countries.

Although the foregoing are unverifiable propositions, an indirect test can be performed. Since an important contributor to increasing variability in frailty in the US is rooted in its relatively diverse ethnic composition, we can focus on M_x patterns for either the NHW or AA sub-populations separately. If the variance-of-frailty mechanism explains observed patterns of crossover ages it must the case that they disappear or, at the very least, are attenuated in observed crossover ages in each sub-population. Figure 8 shows that precisely the opposite occurs: the patterns of crossover ages are *stronger* within groups than in the combined population. Note that the observed crossover ages involve almost no uncertainty among AA's and White females. Virtually all the uncertainty is concentrated in the White male population but, even there, the linearly increasing trend stands out above the noise surrounding the estimates. The tightening of the linear trend in crossover ages, particularly among Blacks, is difficult to reconcile with the variance-of-frailty argument, which would lead us to expect a blurring of any observed pattern as homogeneity of the population increases. Admittedly, Figure 8 alone cannot be the basis to reject *in toto* the selection hypothesis since there is more than one way in which pure selection can produce the observed regularities. A final blow to the selection hypothesis can only be delivered with information that is not readily available—namely, detailed times series of cohort life tables.

In summary, although at least one of the selection mechanisms identified above could produce patterns of increasing crossover ages, it is impossible to verify with the data available to us. That said, the empirical evidence gathered here is inconsistent with the second selection mechanism and we conclude it is unlikely to be operating at all.

We turn now to explanations involving health behaviors and health care. To do so we loosely follow arguments by Ho and Preston (2010) with the caveat that theirs is mostly an effort to explain sustained and uniform advantages at ages over 70 rather than to account for shifts in the age at which mortality patterns in the US are less severe than in peer countries. Ho and Preston make a convincing case for one of the explanations (quality of health care) that is unsuitable to account for an upwardly shifting crossover age. We argue that the quality of health care hypothesis is plausible *only if the crossover age remains approximately fixed over time.*

3.3 Access to health care

This explanation is as follows: the US does better at older ages because of almost universal health care coverage made possible by the adoption of Medicare after 1968. As argued by Ho and Preston (2010), if this

were the correct explanation, the US old age advantage should vanish for older cohorts who did not have access to Medicare. This is exactly the *opposite* of what the data show (see above). If anything, relative conditions worsen for the more recent cohorts who have had more extensive Medicare coverage. In addition, one could argue that there is no plausible reason for the US to have an advantage at older ages solely because of implementation of quasi-universal coverage at ages past 65 since a number of peer countries have had such coverage in place for a longer period of time than the US. The argument is defensible only if one confirms that access, use, and quality of care has been and is better than in peer countries. Furthermore, and most important for our purposes, this explanation cannot possibly account for an *increasing trend* in the crossover age. If the explanation were correct, one would expect to see a crossover age located around age 65 *holding steady after* 1968-1970, the date of Medicare implementation.

3.4 Quality of health care

A heterogeneous assortment of data suggest that the older US population benefits more than peer countries from better screening for and treatment of chronic conditions responsible for mortality at older ages: cancer (particularly breast, colon, and prostate), Ischemic Heart Disease (HD), Acute Myocardial Infarction (AMI) and, finally, cholesterol and hypertension control and surveillance. The empirical evidence is not as strong as one would desire but the conjecture emerges as the most convincing explanation of the US mortality advantage (Ho and Preston, 2010).

Two issues remain to be sorted out. The first is that, under a strict interpretation, the empirical evidence supporting this explanation should lead us to expect a *declining not an increasing (or even a steady) crossover age*. If better health care applies equally to all (and there is universal access to health care after age 65), and those who benefit from superior health care are no more frail than the rest of the population, then old-age mortality rates for more recent birth cohorts should decrease relative to peer countries. By contrast, mortality at very old ages—when marginal benefits due to better screening and improved treatment begin to wane—should at best remain steady. But if this is so, the crossover age should shift to *younger ages*, exactly the opposite of what observed patterns show.

The second issue is as follows: a well-known result from the standard mortality heterogeneity model implies that better screening and treatment at some (early) ages should generate a sub-population of survivors with higher than average frailty. Superior quality of health care at younger ages in the US must then be associated with a larger inflow of a more-frail sub-population into very old ages. This implies that while US mortality should be lower than peer countries at younger ages, it should be higher at older ages. If the effects of a US superior quality of health care unfold over, say, the last two to three decades, we should

observe faster mortality improvements at younger ages and slower ones at very old ages *relative to peer countries*. In the absence of other changes, these conditions alone should bend the pattern of US M_x's and produce two crossover ages: one at relatively young ages (say 60-65), after which the US begins to look better than peer countries due to better screening and treatment, and a second one at older ages (say 80-85) beyond which the perverse effects of increased heterogeneity begin to be felt. Over time, and as the effects of better quality of health care slowly accumulate, there will be increasing benefits accruing among the youngest segment of the old population and growing (relative) deterioration among the oldest old. But the data contradict this expectation as we observe widespread relative deterioration everywhere at older ages. Indeed, when projected into the future, current patterns imply that any traces of a US old-age mortality advantage will vanish altogether in less than twenty years. This is inconsistent with the better quality of health care argument.

3.5 Smoking

Could smoking trends and the changing composition of cohorts by past smoking explain the existence of worse mortality and a linearly increasing crossover age? Given the contrasts in the history of smoking in the US and peer countries, one might suspect that smoking could turn out to be a key contributor to the US disadvantage in mortality at ages 50-70 among both males and females. There is evidence demonstrating that the US disadvantage is partially explained by excess smoking-attributable mortality (Preston et al., 2010, 2011), but can past smoking account for an increasing trend in crossover ages? Forerunners in the US smoking epidemic are men who entered their teens around 1935-40, reached their 50s in the 1970s, and attained their 65th birthday around 1985 (for Disease Control, 1999). One would expect this and subsequent birth cohorts with high prevalence of smoking to experience relatively worse mortality than equivalent cohorts in peer countries.[13] If so, the age at which the US advantage surfaces for the first time must begin to shift upwards as soon as the cohorts with heavy smoking uptake attain ages beyond which smoking-attributable mortality risks increase sharply (60-65). The experience for US females should be analogous but lagging behind by ten to fifteen years.

To assess the role of smoking in the observed trends we compute counterfactual M_x's for the US and peer countries from 1955 on and re-estimate the crossover ages for each year of observation.[14] This test is analogous to one performed by Preston and Ho (2010) for a single calendar year but we add an important modification: we estimate a time trend on the fraction of deaths attributable to smoking from 1975 to 2010. For each year after 1975 and age above 50 we deflate the mortality rates on both the US and the pooled

[13] With the exception of the UK, the smoking epidemic in other high-income countries is believed to have started a decade or so later than in the US.

[14] We use estimates of smoking attributable risks described in the National Academy of Science report (Preston et al., 2011).

benchmark by an amount equal to the estimated fraction of all deaths attributable to smoking derived from the estimated trend. We then compute the RM_x ratios using the new, adjusted quantities. If smoking were the only explanation for the crossover, the adjusted RM_x ratios should display no trend in the crossover age. Figure 9 shows that this is partially the case for both males and females. As expected, adjusted-for-smoking estimated trends of crossover age are flatter than the unadjusted trends and more so for males than for females. A simple linear time trend fitted to the adjusted-for-smoking crossover age among males (not shown) produces a slope of 0.1, down from 0.6 for the unadjusted estimates. For females, the slope declines from 0.4 to 0.2, a more than trivial reduction but only half as large as the reduction for males.

That adjustments for smoking reduce the time dependence of crossover age is comforting, for it suggests that smoking prevalence and its sequelae are important factors behind the upward displacement of ages at which the US does better than peer countries. But while this explanation is satisfactory for males, it is insufficient to account for crossover ages among females. This is puzzling because if smoking is the main explanation behind observed patterns of US old age mortality, it should work for males and females alike. It is possible that the procedure to compute smoking attributable mortality rates underestimates the target quantity for females, but this cannot be the only or even the main explanation. This is because the increase in smoking attributable female mortality needed to attenuate the time trend in female crossover ages to levels equivalent to those observed among males implies implausible high values of lung cancer mortality rates and/or much stronger effects of smoking on mortality due to other causes of death.

Two last caveats. First, all explanations for the crossover age summarized above are cohort-based in the sense that the phenomenon singled out as responsible for the crossover is attributed to changes *across birth cohorts*. If this is indeed the case, it follows that the mortality rates at the point of crossover *increase over time*. Figure 4 above shows that this expectation is supported by the data. Had this not been the case, one could have questioned the validity of any cohort-based explanation. Conversely, the fact that Figure 4 reveals an increasing trend of mortality rates at crossover ages is not sufficient to validate cohort-based explanations. Second, in addition to smoking, obesity and associated excess mortality risks are also plausible explanations for relative deterioration among recent older US cohorts. In fact, Ho and Preston (2010) consider it as an alternative explanation and promptly dismiss it as an unlikely mechanism. We follow their lead since to make much of a dent on the crossover age phenomenon we identify here, the obesity epidemic would have to have started much earlier than when it really did and produce gradually increasing effects in mortality that are inconsistent with the finding that obesity-related mortality has steadily decreased (Ho and Preston, 2010).

To summarize: cohort trends in differential smoking uptake and in the progression of its deleterious effects is an important factor partially accounting for the increasing trend in crossover age. But it is not a

complete explanation, for it leaves a residual, unexplained slope and cannot successfully account for observed gender differentials in the pattern of crossover ages.

4 Summary and Conclusion

Time trends in crossover ages above which Americans enjoy an advantage in survival are puzzling and consequential. If proven correct, this empirical regularity weakens the case for US exceptionalism in mortality at very old ages. It is also has harsher implications for the more than two-thirds of birth cohorts who will soon attain ages over 75. In this paper we verify the existence of an upward, linear shift of the (old) age above which the US experiences lower mortality relative to peer countries, a fact confirmed by two independent estimation procedures and two different data sets. Though each of the methods and data sets has drawbacks, it is unlikely that the tight relation uncovered between crossover ages and time is a computational or a data artifact. Nor can the phenomenon be attributable to deceptive regularities induced by age misstatement, for it is equally visible in data that are possibly affected by it and in data where such errors are absent. It is unlikely that the two most salient selection mechanisms that operate across birth cohorts might play an important role but we cannot dismiss them altogether for lack of solid empirical evidence. Universal access to old age health care is neither a convincing explanation of a general US advantage at old ages nor, least of all, a proper accounting of the systematic increase of the age at which such advantage appears. Better quality health care in the US, on the other hand, has important virtues as an explanation for the overall advantage of US old age mortality but has less traction accounting for a progressive reduction of the range of ages within which the advantage is manifested. Finally, differential trends in smoking uptake and its delayed deleterious effects can explain a non-trivial fraction of the crossover ages time trend among the oldest old, particularly males. However, the smoking-based explanation is not the magic bullet we sought, for it does not account for all the trends we observe nor does it satisfactorily resolves the issue of persistent sex differentials in the phenomenon.

There are at least two large question marks that need to be removed. First, a full explanation of the increasing of crossover ages must be based on a thorough analysis of causes of death. The best way to do this, but also the most complex, is to decompose the yearly increase in crossover ages by causes of death—that is, to determine how much of the increase in crossover is associated with changes in well-defined causes of death. Second, the explanation based on obesity needs to be explored further. In particular, one could use past trends in obesity prevalence and alternative estimates of excess mortality due to obesity to compute counterfactual mortality rates at older ages. These estimates should shed light on whether or not the trend in crossover ages is sensitive (or not) to changes in either the cohort-specific prevalence of obesity

or obesity-related excess mortality rates.

Throughout we emphasized a single explanation model, that is, we only consider arguments and empirical evidence for one of the explanations at a time and pay no notice to the possibility that a combination of some of them produces the observed patterns. Although plausible, this is an empirically unverifiable explanation.

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Tables and Figures

Figure 1: *Differences in Life Expectancy at Birth by Sex (1955-2010)*

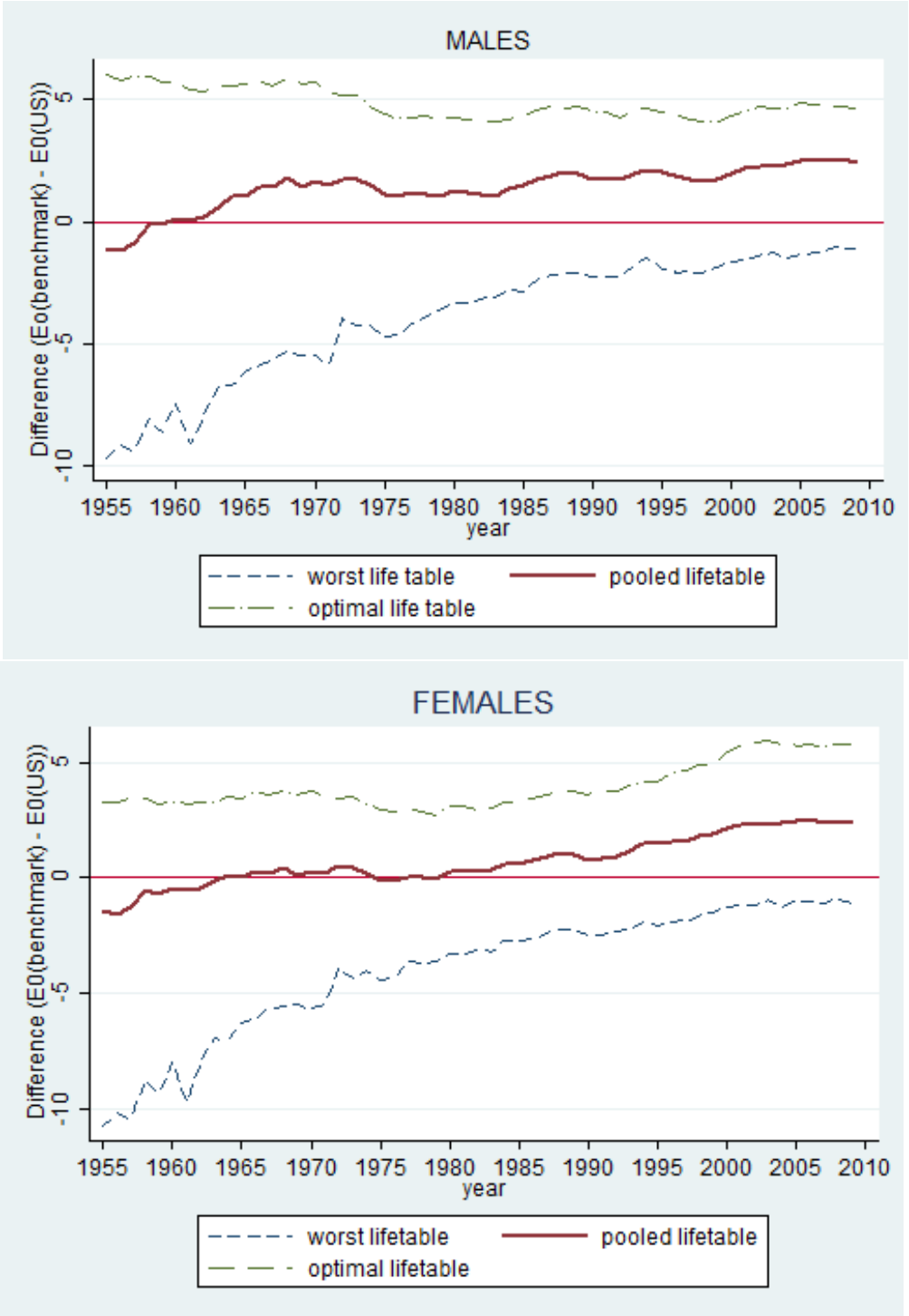


Figure 2: *Differences in Life Expectancy at Age 50 by Sex (1955-2010)*

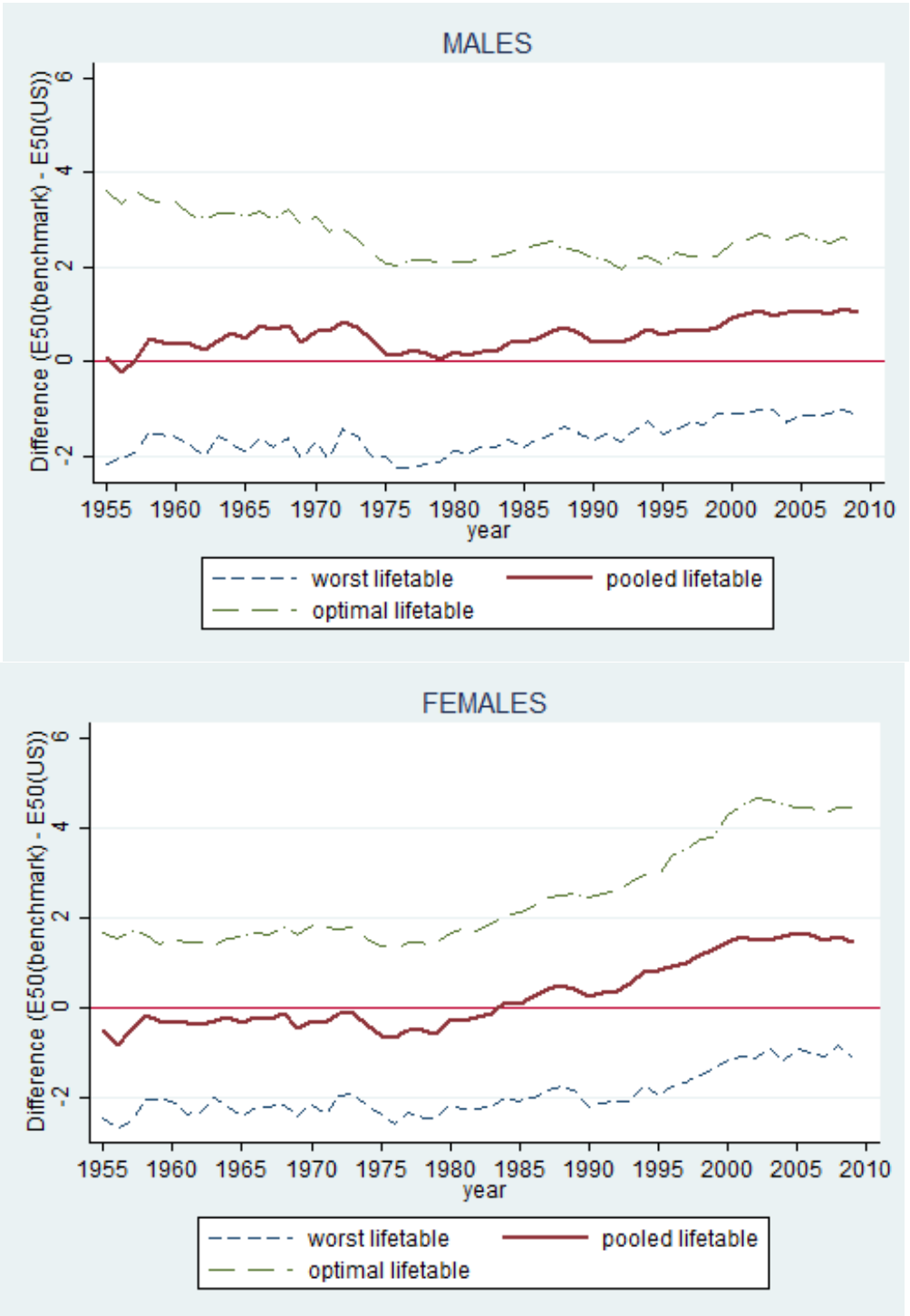


Figure 3: *Ratio of US Mortality Rates to Pooled Benchmark by Sex (1955-2010)*

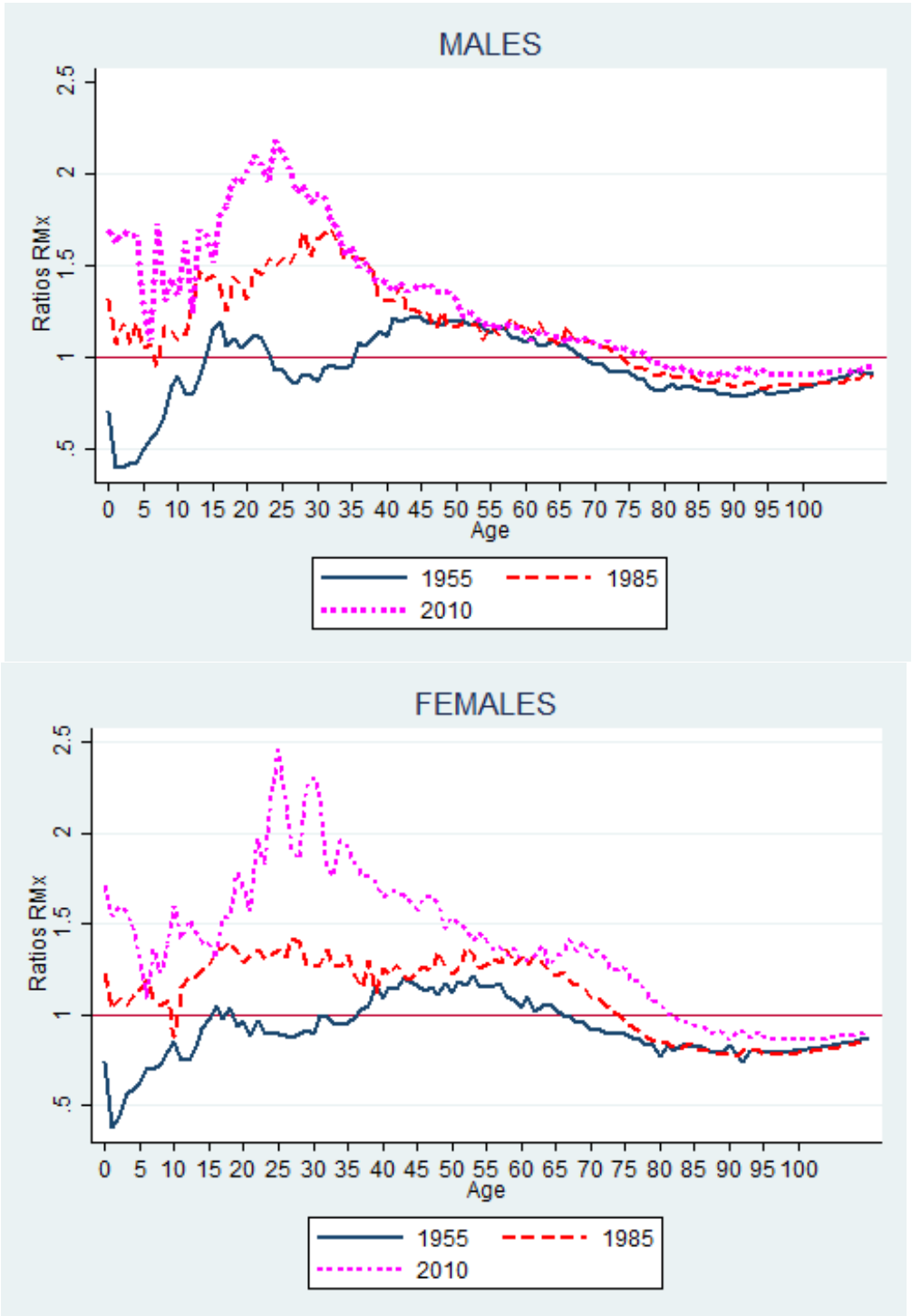


Figure 4: *Mortality Rates at Crossover Age by Sex (1955-2010)*

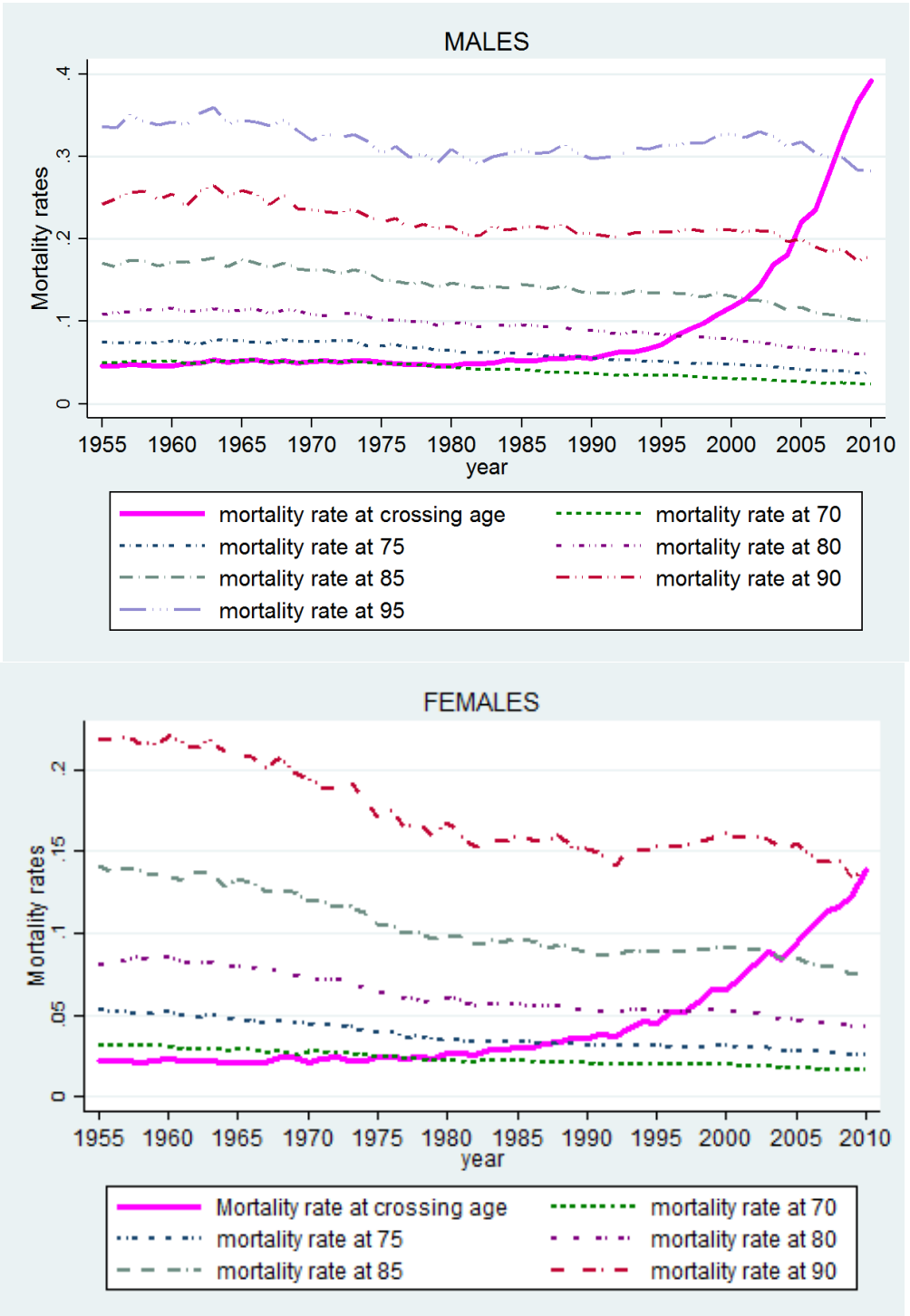


Figure 5: *Difference in Life Expectancy at Age 75 Compared to Pooled Benchmark by Sex (1955-2010)*

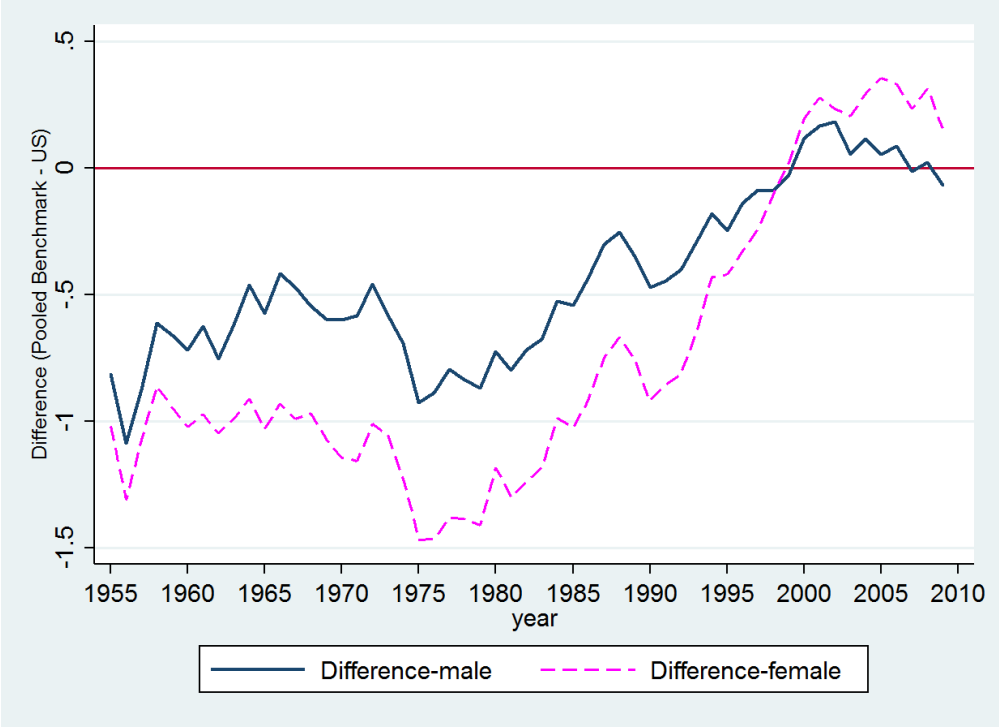


Figure 6: *Observed and Model-Dependent Crossing Ages by Sex (1955-2010)*

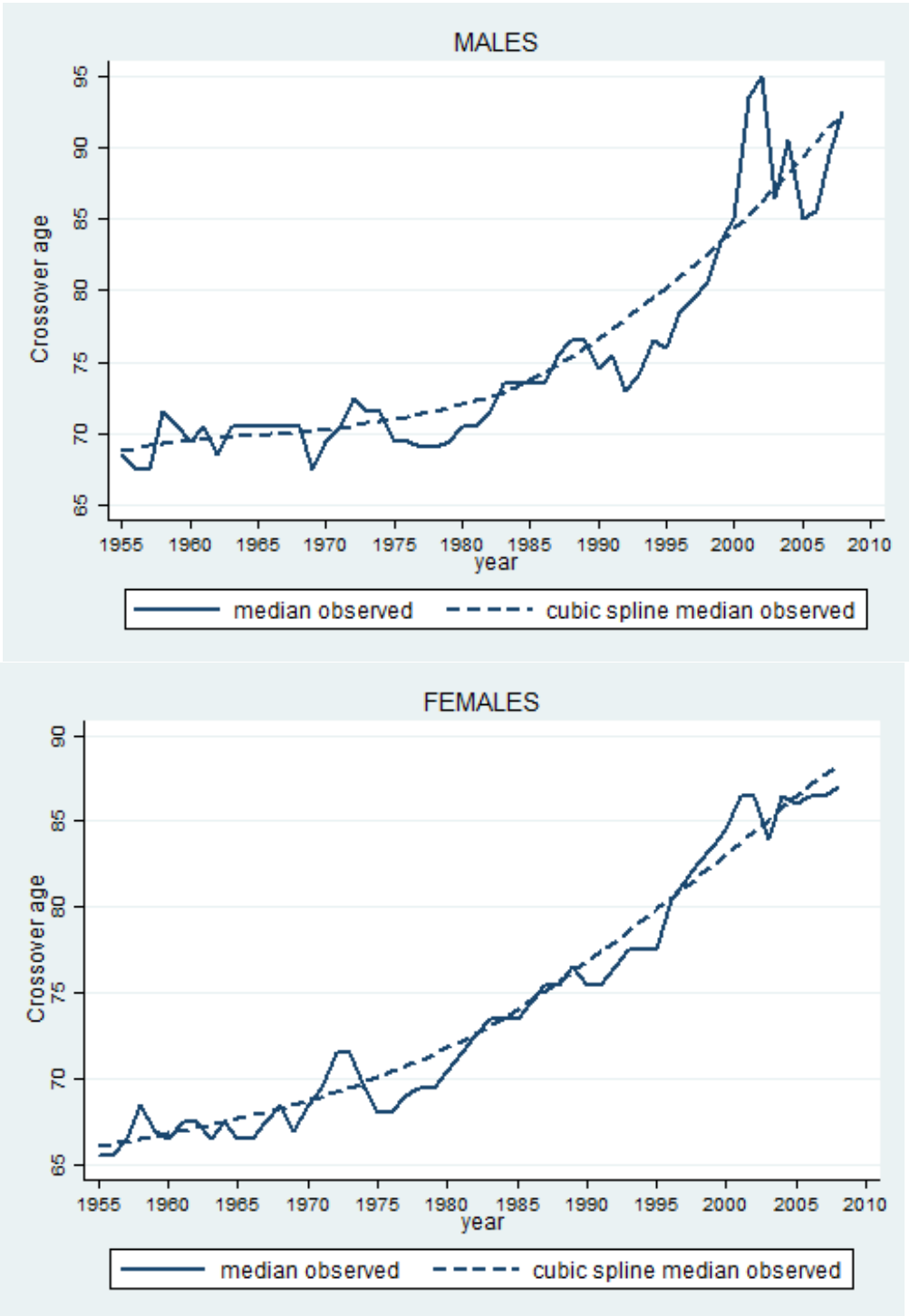


Figure 7: *Crossover Age from Medicare Data, Total US Population*

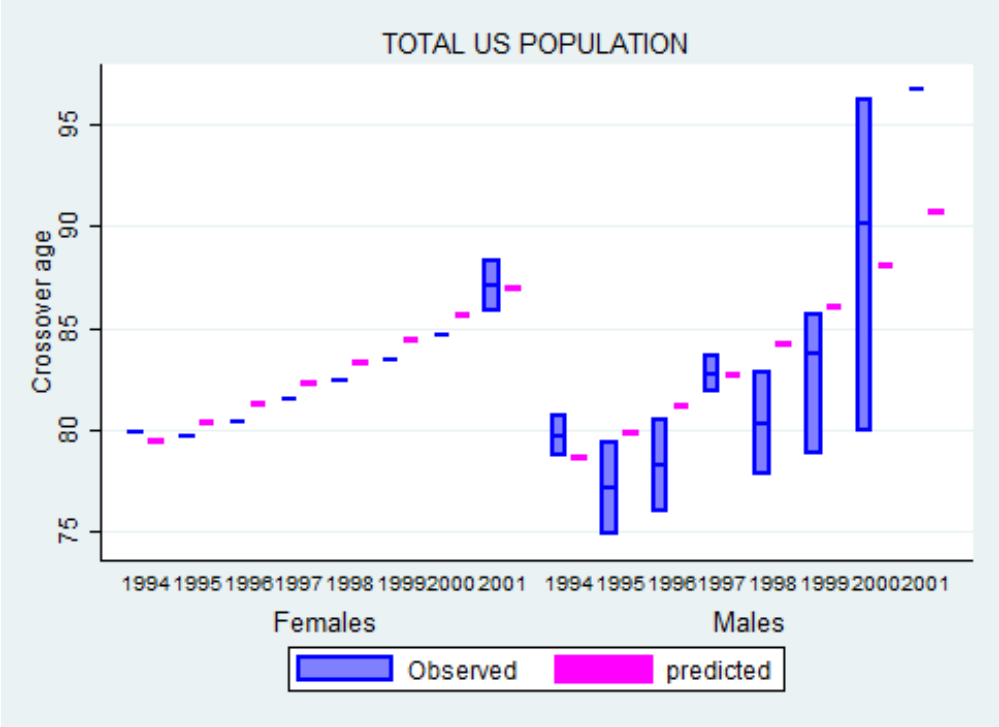
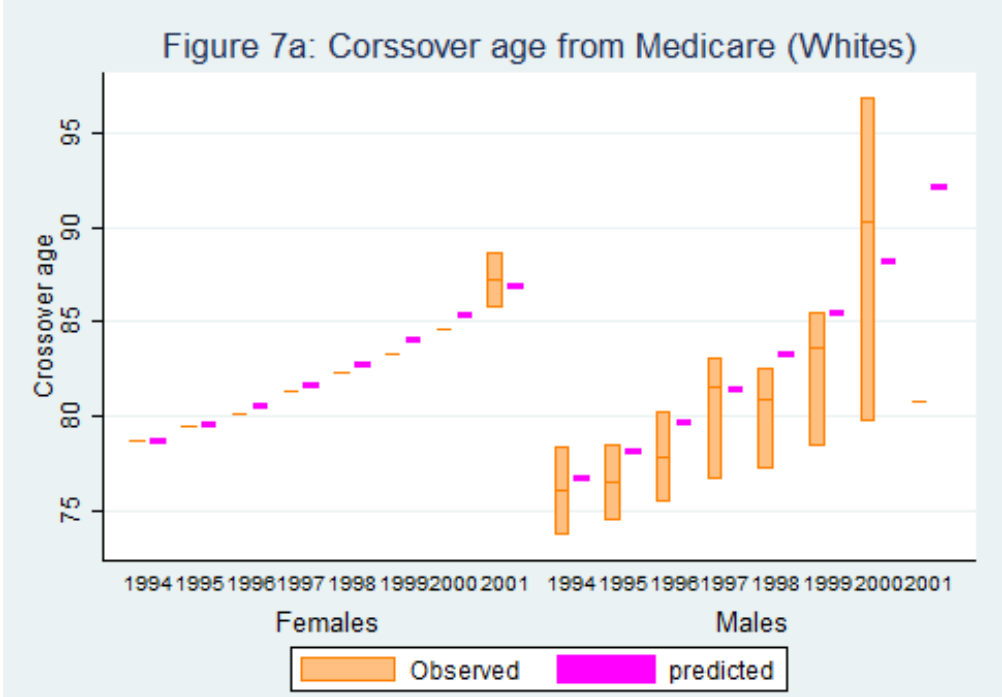


Figure 8: *Crossover Age from Medicare Data, By Race*



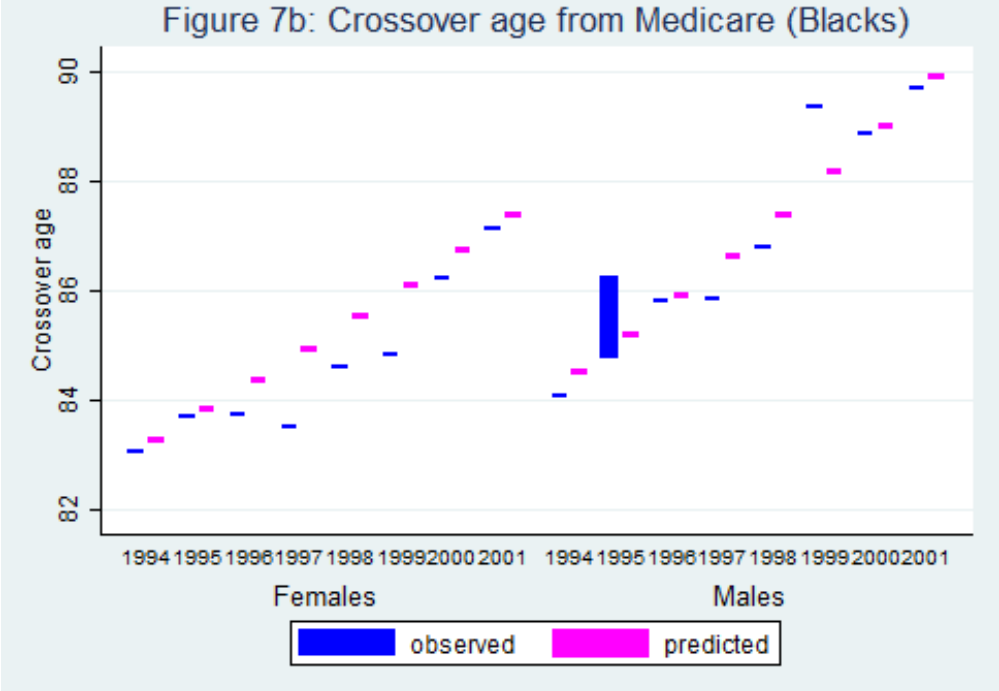


Figure 9: *Crossing Ages, Observed and Adjusted for Smoking by Sex (1955-2010)*