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Understanding the Declining Female Mortality Advantage in the United States: An Assessment Using Prospective Data

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ABSTRACT

Since about 1980, the longstanding female life expectancy advantage has stalled in the United States. While secular improvements have continued, U.S. women have lost ground relative to women in comparably wealthy nations and relative to American males. Evidence for this pattern is striking, but most work on this topic has examined aggregate data. While key contributing factors have been identified, including smoking and obesity among others, aggregate level data has not been available for all potentially relevant explanatory factors. We build on these findings with a cohort of 3617 U.S. adults first interviewed in 1986 and followed up for about twenty years, with detailed information about behaviors, stressful exposures, and health from up to four additional interviews. Initial results demonstrate the stalled female mortality advantage in individual-level data, show the relevance of smoking, indicate stronger

patterns of gender convergence among non-whites, and suggest a role for employment in these patterns.

INTRODUCTION

Since about 1980, the longstanding female life expectancy advantage has stalled in the United States. While secular improvements have continued, U.S. women have lost about 1 to 6 years of life expectancy relative to women in comparably wealthy nations, and about 2 to 3 years of life expectancy relative to American males (Crimmins, Preston, and Cohen 2010). This unfavorable trend is even more serious among U.S. women with low educational attainment; life expectancy at age 25 of low educated white women declined by 5 years in absolute terms between 1990 and 2008 (Olshansky, Antonucci, Berkman, Binstock, Boersch-Supan, Cacioppo, Carnes, Carstensen, Fried, Goldman, Jackson, Kohli, Rother, Zheng, and Rowe 2012). This weakening of the female life expectancy advantage in the U.S. relative to other wealthy nations was explored in two recent reports conducted by the U.S. National Research Council (Crimmins, Preston, and Cohen 2011; Crimmins, Preston, and Cohen 2010), and a range of recent papers have examined the weakening of the female advantage in a variety of countries and comparisons (e.g., Conti, Farchi, Masocco, Minelli, Toccaceli, and Vichi 2003; Glei and Horiuchi 2007; Trovato and Heyen 2006; Yang, Khang, Chun, Harper, and Lynch 2012).

While the evidence is for this pattern is striking, nearly all work on this topic has example life table estimates or other aggregate data, or on mortality follow up of a cross-sectional sample. Moreover, some key contributing factors have been explored, including smoking patterns, obesity, physical activity, hormone replacement therapy, health care access, and some aspects of stress and social inequality, but aggregate level data has not been available for all potentially

relevant explanatory factors (e.g., Crimmins, Preston, and Cohen 2010). To shed new light on U.S. women's weakening advantage in life expectancy, we propose to use detailed panel data on a cohort of about 3,600 U.S. adults who were first interviewed in 1986 and have been followed up for about twenty years, with detailed information about behaviors, stressful exposures, and health in up to four additional interviews.

The major explanations for the worsening of American women's health and mortality that currently prevail in the broader literature are the delayed impact of cohort patterns of smoking and perhaps other health behaviors on women's health and mortality. Men's relative life expectancy could be improving as cohort changes in men's behaviors – particularly smoking – have already exerted most of their impact because men took up smoking in earlier cohorts than did women. Many existing analyses of these explanatory factors, however, rely on national mortality and health data bases that cannot capture individual level detail and dynamics of these behaviors across the life course, and must rely on indirect estimation techniques. For example, some of the strongest evidence for the role of smoking in these gendered life expectancy patterns come from estimates derived from tobacco-related cause-specific death information (Rostron 2010). While powerful, these aggregate, population-level studies cannot explore individual level histories of smoking or other health behaviors. They are also limited in their ability to capture a range of other potentially meaningful changes that men and women have faced in recent decades. These range from changes in the likelihood of divorce and single parenthood to the increasing exposure to the "second" shift of unpaid work among the large numbers of women, even those parenting young children, who are working for pay (e.g., Annandale and Hunt 2000; Spijker, van Poppel, and van Wissen 2007). Data on such measures may not be available or reliable at the aggregate level, however. A handful of studies have suggested that some of these gendered role

transformations could be important in explaining why less educated women are falling behind their more educated female counterparts but there has been relatively little assessment of these factors in the context of gender differences in life expectancy in the US (Montez, Hummer, Hayward, Woo, and Rogers 2011).

A final important aspect of the stalled female life expectancy advantage in the U.S. is the possibility that intersections between gender and race are an important component of the explanation. Some recent studies have identified particularly severe health and mortality trends among the least educated white women (Olshansky et al. 2012). Other investigators have noted a particularly promising pattern of improvement among African American men (Harper, Rushani, and Kaufman 2012). No studies to our knowledge have considered how gender gaps among whites and blacks might be contributing to the overall female stalled advantage. Changes in health behaviors and conditions of work and life discussed above, or other factors, may have differentially impacted the survival prospects of white Americans and others over this period.

In this study, we consider whether the risk of death as a function of age and gender has changed during 21 years of follow-up in the American's Changing Lives Study. We use individual-level prospective data with time-varying indicators of smoking, body mass index, work and family responsibilities and associated time use and stressful experiences, and detailed mortality follow up. An oversample of African Americans allows for some exploration of gender gaps for whites and non-whites in these conditions and experiences, and their contribution to the weakening female mortality advantage. We ask several research questions: (1) do we observe the same stalling of the female mortality advantage for women in panel data on individuals that has been observed by those using life table estimates and aggregate data? (2) If we find this pattern, what are the contributions of smoking, body mass index and physical activity to the stalled

progress for women when we use individual-specific and time-varying measures? (3) Do responsibilities at home and work, time spent fulfilling them, and stressfulness of these major roles contribute to the pattern of stalled progress for women? (4) Do any of these associations operate differently by race/ethnicity?

DATA AND METHODS

Data

The American's Changing Lives (ACL) study began in 1986 with a national face-to-face survey of 3617 adults ages 25 and up in the continental U.S., with African Americans and people aged 60 and over over-sampled at twice the rate of the others. Face-to-face re-interviews were conducted in 1989 with 83% (n=2867) of survivors, and survivors since baseline have been re-interviewed by telephone, and where necessary face-to-face, in 1994 (83%), 2001/02 (74%), and 2011/12 (81%). For these analyses we use information from all respondents, even those who were lost to follow up. Over the course of the study, ACL respondents have been matched to the National Death Index (NDI) to verify reported deaths, to identify unreported deaths, and to obtain coded cause of death and access to death certificates. Our experience indicates that we ascertain at least 99% of all ACL deaths, mainly via NDI but also via other respondent tracking, and confirm more than 96% via death certificates (with other data indicating that those reported dead but without a death certificate are almost certainly dead). We have completed full death verification through 2006 and should have further deaths confirmed by the PAA meetings. Analyses use survey weights that make the sample representative of the U.S. population in 1986.

Measures

Our outcome measure is *death*, confirmed mainly using the NDI as described above. As predictors we include time-constant measures from baseline of *gender*, *race/ethnicity* (non-

Hispanic white, non-Hispanic black, Hispanic, and Other race/ethnicity), *educational attainment* (less than high school, high school completion, some college, bachelor's degree or more), and baseline *family structure* (never a single parent, ever a single parent before baseline, currently a single parent at baseline, indicated by having had biological child under 18 coincident with being unmarried/unpartnered). We use time-varying measures (from each survey wave) of the respondents' *age* in years and age in years squared, *smoking status* (never smoker, current smoker, former smoker), and *body mass index* category (underweight, normal weight, overweight, obese, morbidly obese, using the typical cutpoints). Time-varying measures of *physical activity* were obtained from a physical activity index created based on answers to questions regarding how often the respondent took walks, did gardening or yard work, or engaged in sports or exercise. Physical activity index scores were divided into quintiles to create 5 groups of approximately the same size. The group in the top quintile represents those who are the most physically active. In some models, time-varying measures of *stress* (frequency of being bothered or upset) were included for the domains of employment, parenthood, and partnership, and time-varying indicator variables for employment, parental, and partnership statuses were included in the relevant models.

Methods

Date of death is available on a monthly basis, so we use a discrete time-to-event model, predicting the probability of death P_{it} for respondent *i* at follow-up month *t* using a pooled complementary log-log model as follows:

$$\log(-\log(1-P_{it})) = f(t) + \sum_{k=1}^{p} X_{ikt} * \beta_k \qquad (1)$$

where X_{ikt} are (possibly time-varying) covariates and β_k are fixed parameters associated with the k^{th} covariate. Since $-\log(1-P_{it})$ can be interpreted as the hazard (risk) of death during time t, β_k can be interpreted as the change in the log relative risk associated with a one-unit change in X_{ikt}, and $f(t)$ as the baseline log-hazard at time t (when all X_{ikt}=0) (Prentice and Gloeckler 1978). We fit this model by creating month-level observations for each subjects, with a death indicator $D_{it}=0$ until the month of death, when $D_{it}=1$, and X_{ikt} equal to its value at time *t* (constant if a baseline covariate). A binary regression model is then fit using a complementary log-log link to preserve a relative risk interpretation of the covariate effects. Because death is potentially observable for all subjects even if they have dropped out of the study, we utilize the baseline sampling weights to obtain point estimates using sampling weights and confidence intervals using Taylor Series approximations. We considered deaths through calendar year 2006, with survivors censored at this point, the last year with complete death identification available from the U.S. National Death Index. Time-varying covariate information was carried forward from each interview for all subsequent months until the next completed interview or death.

After considering a variety of functional forms for $f(t)$, including indicators of year and cubic splines with knots at 7 and 14 years (dividing the follow up period into tertiles), it appeared that a quadratic model was sufficient to capture baseline trends in mortality risk. We considered age as a time-varying covariate and preliminary model selection indicated a quadratic specification of age was sufficient. Next, we considered interactions between age and gender, between age and follow-up time, and between gender and follow-up time. Only the last reached statistical significance. Based on model fit criteria that compared predicted age-gender-specific

mortality rates from our models to age-gender-specific population-level mortality rates obtained from life table data, it appeared that a linear model for time had the best fit.

PRELIMINARY RESULTS

Results for the main effect and interaction model are given in Model 1 in Table 1. The log-risk of death increases with age in an approximately linear fashion, with some evidence of acceleration even on the log scale at older ages (age-squared term is positive and significant in some models). Men are at greater risk of death than women, and supporting evidence from prior studies using life table estimates, we find evidence of a secular decline in this difference (β = -0.031, $p<.01$). Based on the coefficients in Model 1, the relative risk of mortality for men compared to women declines from 2.44 (95% CI 1.90-3.14) in 1986 to 1.31 (95% CI 0.98-1.74) in 2006, the end of follow up. Figure 1 illustrates what is occurring for both groups: viewing this interaction from the perspective of the secular trend, we see evidence of a declining age-adjusted risk death for males, but no corresponding decline for females (see Figure 1). An alternative approach to visualizing this secular trend in the gender difference is provided in Figure 2, which shows the relationship between log-risk of death and age for men and women in 1986, 1996, and 2006, starting at the youngest age cohort in each of these years (age 25 in 1986, 35 in 1996, and 45 in 2006). Male and female risks of death are clearly delineated in 1986, but move closer together and begin to overlap by 2006.

We next consider the degree to which the stalling of female mortality risk and continued decline of male mortality risk may be explained by individual-level changes in smoking, obesity, and physical activity, after adjusting for baseline education and race/ethnicity. Model 2 in Table 1 shows that little of the gender-by-time interaction is explained by baseline differences in education and race. Model 3 shows that approximately 10% of this key interaction is explained

by differences in smoking levels (earlier and relatively more rapid declines in being a current smoker among men relative to women). However, further adjustments for time-varying measures of obesity together with physical activity in Model 4 show that these activities actually had a slight suppressor effect on the interaction between gender and time, such that the gender-time interaction in Model 4 is virtually identical to that in the unadjusted Model 1. We also considered whether the effects of smoking or obesity interacted with age or time. Model 5 shows that only the interaction of smoking by the linear term of age even approaches significance, and its inclusion has only a very modest mediating effect on the interaction between gender and time, explaining approximately 3% of the effect relative to the unadjusted model. Finally, Model 6 adjusts for work stress, parental stress, and marriage stress as well as single parent status at baseline (currently single parent, previously single parent). The employed indicator compares those working but reporting the lowest level of stress with those not working, while the work stress measures estimate the impact of each unit increase in work stress among those working. A similar explanation applies to marital and parental status indicators and their related stress measures. We find that the work and family stress measures explain approximately 10% of the gender-time interaction term, suggesting that they contribute to the stalled female mortality advantage.

Table 2 considers models that assess race and education interactions with gender and gender*time effects on risk of death, unadjusted and adjusted for smoking, obesity, and physical activity. Model 7 is the base model and replicates the findings from Model 2 in Table 1. Model 8a added interactions between gender and race/ethnicity, between time and race/ethnicity, and between gender, race/ethnicity, and time. There was little evidence of any change in the effects of race among females over time. All non-white groups showed a larger gender difference in

mortality than whites in 1986, although none of these differences reach statistical significance. Similarly, all non-white groups saw a more rapid decline in this gender difference over time than whites, visible in the three-way interaction terms, although these differences did not reach statistical significance. Adjusting for smoking, obesity, and physical activity had little effect on the race*gender*time interactions (Model 8b); adjusting further for work and family stress seemed to reduce the race*gender*time interaction somewhat, at least among Hispanics and other races (Model 8c). Because the race*gender*time interactions for all non-white groups were similar, we fit a model with a common interaction term, unadjusted (Model 9a), adjusted for smoking, obesity, and physical activity (Model 9b), and adjusted further for work and family stress (Model 9c). Both the unadjusted and adjusted models suggest that the gender difference among non-whites has been declining; in the unadjusted Model 9a it was falling 5.0% faster than the gender difference among whites (95% CI 0.3%-9.3%, p=.036;). Adjusting for smoking, obesity, and physical activity decreased this gap between non-whites and whites to 4.8% (95% CI 0.1%-9.2%, p=.043), and adjusting for smoking, obesity, physical activity, work stress, and family stress decreased this gap to 4.5% (95% CI 0.0%-9.1%, p=.064). However, but the stalled female mortality advantage appears even stronger among non-whites even after all these adjustments.

Figure 3 illustrates these differences across race/ethnic groups. For all groups, women maintain a fairly constant death rate over the follow-up period. Male rates decline over follow-up for all groups, but this decline is far more pronounced among minority men, particularly African-American men (small sample sizes preclude accurate estimates among Hispanic and other minorities but we present figures here to show that their patterns are similar to those for African Americans). We examined a similar series of models that substituted education for

race/ethnicity, but found no evidence for any education*gender, education*time, or education*gender*time interactions.

These results are all preliminary, and will be elaborated further before the PAA 2013 meetings. In the interim, we also plan to explore additional psychosocial covariates, including time-varying measures of time spent in paid and unpaid work, and potentially other stressors such as discrimination, social integration, and other factors for which the ACL data have rich, time-varying measures. We will also explore gender and race differences in the influences of these exposures. Initial results are promising and signal the potential contributions that prospective individual-level data can make to understanding this critical population health problem.

Table1. Coefficients and standard errors from discrete time hazard models of mortality, log-relative risk of death, American's Changing Lives respondents.

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Time in years	-.005 (.007)	-.002 (.007)**	-.004 (.007)	.007(.007)	.006(.007)	.011(.008)
Age in years	.061 (.015)**	.057 (.015)**	.055 (.015)**	.067 (.016)**	.079 (.020)**	.056 (.020)**
Age-sqared in years	.00031 (.00017)	.00033 (.00016)*	.00042 (.00016)**	.00021 (.00017)	.00018 (.00019)	.00026 (.00019)
Male	.924 (.118)**	.925 (.140)**	.784 (.140)**	1.000 (.143)**	1.000 (.143)**	1.033 (.158)**
Male*Time	-.031 (.012)**	-.031 (.017)**	-.028 (.012)*	-.031 (.012)*	-.030 (.011)*	-.028 (.013)*
Race (Non-Hispanic White omitted)						
African American		.299 (.078)**	.283 (.079)**	.264 (.080)**	.260 (.080)**	.246 (.086)**
Hispanic		.117 (.189)	.129 (.189)	.043 (.190)	.027 (.191)	.076 (.229)
Other		.196 (.252)	.159 (.252)	.021 (.253)	.025 (.255)	.219 (.271)
Educational Attainment (BA+ omitted)						
< High School		.498 (.138)**	.380 (.138)**	.302 (.140)*	.298 (.140)*	.193 (.148)
High School		.258 (.143)	.168 (.145)	.100 (.145)	.093 (.145)	.050 (.156)
Some College		.251 (.155)	.127 (.156)	.100 (.156)	.099 (.156)	.101 (.166)
Smoking Status (Never smoker omitted)						
Current Smoker			.815 (.106)**	.650 (.1067)**	1.228 (.422)**	1.296 (.492)**
Former Smoker			.354 (.080)**	.314 (.079)**	1.165 (.409)**	1.333 (.451)**
Body Mass Index (Normal weight omitted)						
Underweight				.744(.1501)**	.753(.150)**	.724(.160)**
Overweight				-.126(.086)	-.128(.086)	-.112(.095)
Obese				-.168(.122)	-.173(.122)	-.262(.138)
Morbidly Obese				-.067(.172)	-.078(.173)	-.077(.186)
Physical Activity Score				-.289(.039)**	-.291(.039)**	-.309(.044)**
Current Smoker*Age					-.011(.008)	-.013(.010)
Former Smoker *Age					-.016(.007)*	-.018(.008)*
Employed						-.542(.216)*
Work Stress score						-.160(.131)
Parent						-.004(.118)
Parent Stress score						-.087(.051)
Married/Partnered						-.122(.105)
Marriage Stress score						-.039(.087)
Single Parent at Baseline						.131(.224)
Ever a Single Parent before baseline						.087(.090)

Note: *p<.05, **p<.01, ***p<.001

Table 2. Coefficients and standard errors from discrete time hazard models of mortality with assessment of racial and socioeconomic disparities, log-relative risk of death, American's Changing Lives respondents.

	Model 7	Model 8a	Model 8b	Model 8c	Model 9a	Model 9b	Model 9c
Time in years	-.002 (.007)	-.002 (.009)	.007 (.009)	.012 (.010)	-.002 (.009)**	.007 (.009)	.012 (.010)
Age in years	.057 (.015)**	.059 (.015)**	.081 (.020)**	.058 (.020)**	.059 (.015)**	.081 (.020)**	.058 (.020)**
Age-sqared in years	.00033 (.00016)*	.00031 (.00016)	.00015 (.00020)	.00024 (.00019)	.00031 (.00016)	.00016 (.00020)	.00024 (.00019)
Male	.925 (.140)**	.842 (.166)**	.961 (.168)**	.990 (.185)**	.842 (.166)**	.961 (.168)**	.990 (.185)**
Male*Time	-.031 (.017)**	-.021 (.014)	-.021 (.013)	-.020 (.015)	-.021 (.014)	-.021 (.013)	-.020 (.015)
Race (Non-Hispanic White omitted)							
African American	.299 (.078)**	.294 (.176)	.354 (.178)*	.313 (.192)	.259 (.184)	.321 (.185)	.320 (.195)
Hispanic	.117 (.189)	.083 (.441)	.144 (.444)	.288 (.421)	.183 (.386)	.154 (.397)	.237 (.410)
Other	.196 (.252)	.164 (.522)	.204 (.572)	.489 (.549)	.336 (.498)	.460 (.487)	.543 (.565)
Educational Attainment (BA+ omitted)							
< High School	.498 (.138)**	.503 (.138)**	.304 (.140)*	.200 (.148)	.503 (.138)**	.304 (.140)*	.200 (.148)
High School	.258 (.143)	.262 (.144)	.100 (.146)	.055 (.157)	.262 (.144)	.101 (.146)	.056 (.157)
Some College	.251 (.155)	.259 (.156)	.115 (.157)	.116 (.166)	.260 (.156)	.115 (.157)	.116 (.166)
Black*Male		.241(.270)	.028(.270)	.091(.287)	.312(.274)	.093 (.275)	.075(.288)
Hispanic*Male		.941(.629)	.671(.631)	.670(.650)	.858(.420)	.654 (.422)	.748(.480)
Other*Male		.500(.773)	.300(.779)	.156(.798)	.209(.532)	-.132 (.537)	.073(.584)
Black*Time		.0077(.0147)	.0081(.0148)	.0091(.0158)	.0110(.0151)	.0110(.0152)	.0084(.0160)
Hisp*Time		-.0161(.0340)	-.0211(.0342)	-.0486(.0359)	-.0208(.0284)	-.0210(.0287)	-.0431(.0318)
Other*Time		.0187(.0366)	.0182(.0374)	-.0029(.0387)	.0032(.0364)	.0054(.0368)	-.0078(.0390)
Black*Male*Time		-.043(.024)	-.042(.024)	-.048(.025)			
Hisp*Male*Time		-.059(.054)	-.050(.055)	-.036(.061)			
Other*Male*time		-.079(.070)	-.091(.070)	-.055(.072)			
Nonwhite*Male*Time					-.051(.024)*	-.049(.024)*	-.047(.025)
Smoking Status (Never smoker omitted)							
Current Smoker			1.239(.425)**	1.286(.496)**		1.236(.424)**	1.286(.496)**
Former Smoker			1.154(.411)**	1.320(.453)**		1.154(.411)**	1.322(.453)**
Body Mass Index (Normal weight omitted)							
Underweight			.761(.151)**	.761(.160)**		.763(.151)**	.731(.160)**
Overweight			-.136(.086)	-.118(.095)		-.135(.086)	-.118(.095)
Obese			-.180(.122)	-.261(.139)		-.179(.122)	-.261(.139)
Morbidly Obese			-.098(.175)	-.089(.198)		-.097(.175)	-.088(.189)
Physical Activity			-.293(.039)**	-.307(.045)**		-.293(.039)**	-.307(.045)**
Curr Smoke*Age			-.0113(.0082)	-.0121(.0096)		-.0113(.0082)	-.0121(.0096)
Past Smoke *Age			-.0163(.0074)*	-.0178(.0081)*		-.0163(.0074)*	-.0178(.0081)*
Employed				-.542(.217)*			-.542(.217)*
Work Stress score				-.159(.131)			-.159(.131)
Parent				.007(.117)			.008(.117)
Parent Stress score				-.091(.052)			-.091(.052)
Married/Partnered				-.132(.106)			-.132(.106)
Marriage Stress score				-.031(.090)			-.031(.090)
Single Parent at Baseline				.129(.227)			.130(.226)
Ever a Single Parent before baseline				.076(.092)			.076(.092)

Note: *p<.05, **p<.01, ***p<.001

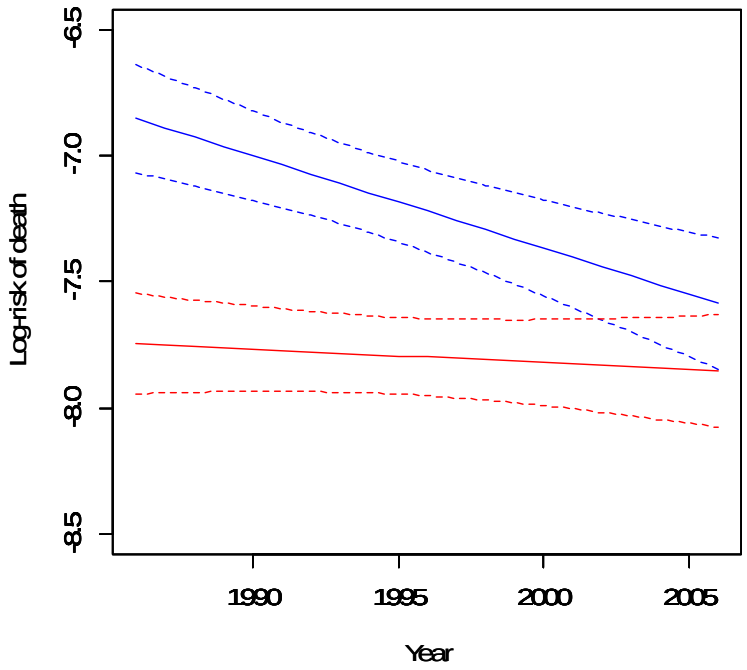

Figure 1: Predicted log-risk of death for 55 year old males (blue) and females (red) over ACL follow-up period: point estimates (solid line) and 95% confidence intervals (dotted line).

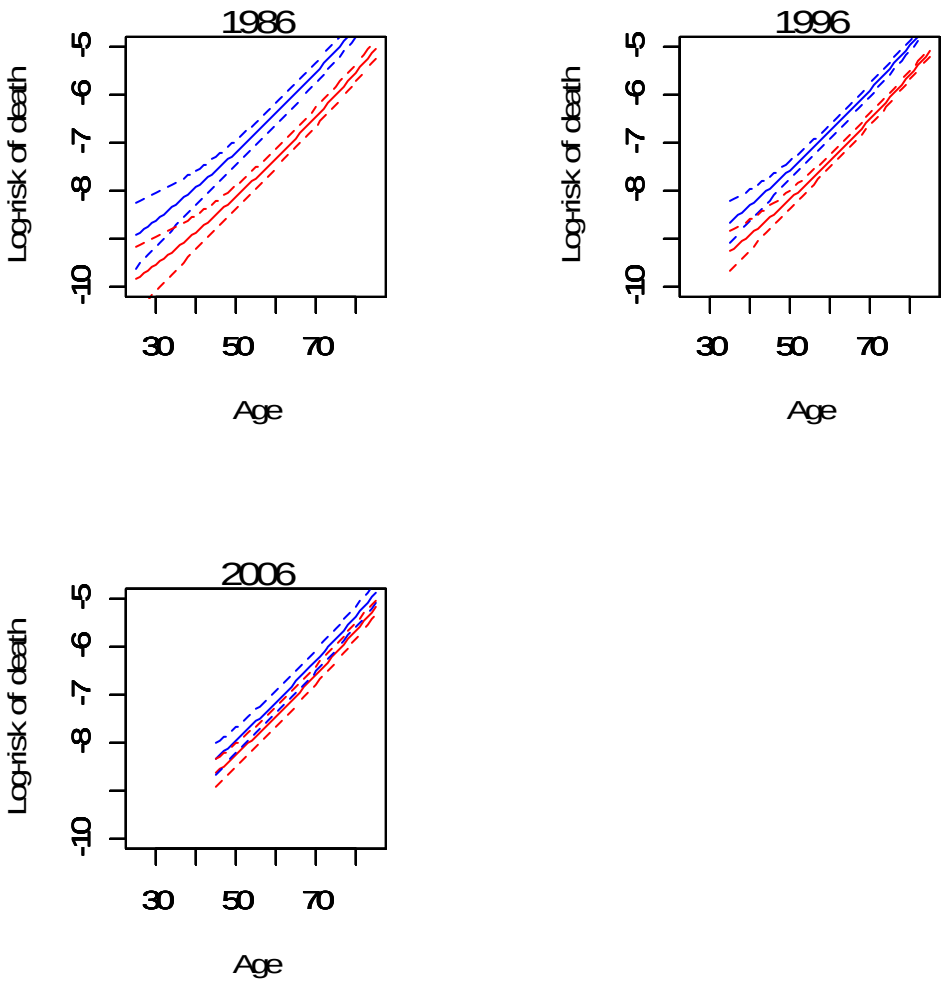

Figure 2: Log-risk of death by gender (blue=male, red=female) and age: point estimates (solid lines) and 95% confidence intervals (dotted lines). 1986 starts at age 25, 1996 at age 35, and 2006 at age 45.

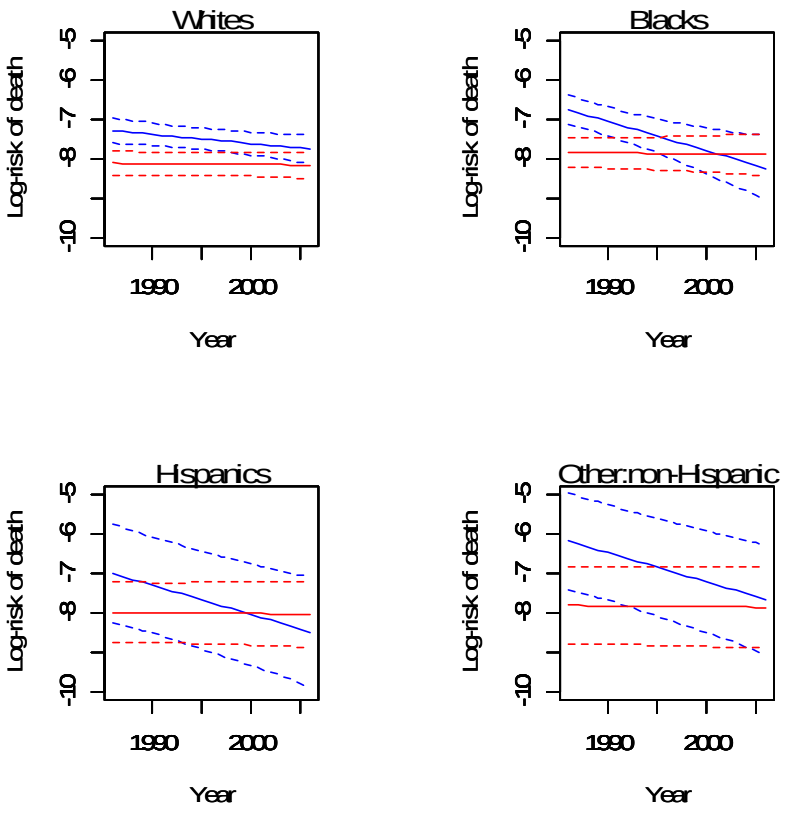

Figure 3: Predicted log-risk of death for college-educated 55 year old males (blue) and females (red) over ACL follow-up period, by race-ethnicity: point estimates (solid line) and 95% confidence intervals (dotted line).

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