The Link between Parental and Offspring Longevity*

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February 2013

*Partial support for this research came from a Eunice Kennedy Shriver National Institute of Child Health and Human Development research infrastructure grant, 5R24HD042828, to the Center for Studies in Demography and Ecology at the University of Washington.

Abstract

Studies of adult mortality typically examine the impact of individual characteristics, but ignore the fact that the characteristics of people closely linked to those individuals also influence mortality risk. This paper examines the effect of parental longevity on survival outcomes of adult offspring using survey data from the University of Michigan Health and Retirement Study (HRS) between 1992 and 2008. It employs a competing risk model that controls for correlation between individual death and survey non-response. There is strong evidence that individuals with longer-lived parents exhibit lower mortality risk. Even after controlling for health conditions and behavioral variables of the offspring, parental age at death has a substantial impact on the survival of the adult offspring, suggesting a strong genetic component that must be considered as important in determining longevity.

1 Introduction

Higher standards of living, advances in medical technology, and better knowledge about positive health behaviors have dramatically increased human longevity, especially in the Western world.[1] Despite a large body of literature on determinants of longevity, there is little agreement on the relation between parental longevity and offspring longevity, and whether improvements in life expectancy have weakened the link between the two. This paper presents new evidence examining the link between the survival of parents and their offspring and the extent to which this connection depends on genetic or social factors.

The relation between parental and offspring longevity is important for two reasons. First, we still know little about parental longevity as a determinant of offspring longevity. Second, people make decisions on consumption, savings, and retirement planning based on subjective mortality expectations rather than on expected mortality rates as cited in life tables (*Gan et al.* 2004; *Salm* 2010). If people base their survival expectations on the longevity of family members, it is important to understand the link between the two. Individuals need to understand the determinants of their life expectancyhow long they expect to live and how healthy they will be, among other factorsin order to make informed decisions about how they spend their money and how much they plan to work. By doing so, they stand a greater chance of ensuring they retain sufficient resources for their projected years in retirement. If methods are implemented to increase the precision with which life expectancy estimates are determined, the government will be able to reference a more useful and definitive target estimate when designing retirement policy.

Children's longevity is related to their parents' longevity through genetics and behavior. Parents pass on genetic material that either directly or indirectly improves or worsens their offspring's survival. Genetically-transmitted diseases may directly lower an individuals survival chances. Offspring whose parents have had a history of health conditions that increase mortalitysuch as high blood pressure and diabetesare at greater risk of acquiring the same disease (*Hakonarson et al.*

[1]Between 1900 and 1999 the average life span in the United States increased by 30 years (*CDC* 1999). Adult mortality in the United States has received considerable attention in the medical, demographic, and economic literature. *Rogers et al.* (2000) and *Sickles and Taubman* (1997) provide comprehensive surveys.

2007; *Scott et al.* 2007; *Newton-Cheh et al.* 2009). Conversely, tall parents are more likely to have tall children, demonstrating a genetic component. Taller people have higher average income, which has been shown to increase longevity. (*Blakely et al.* 2004; *Judge and Cable* 2004; *Persico et al.* 2004; *Dowd et al.* 2011). Behavioral factors also support a link between parental and child survival. For example, parental smoking not only directly exposes children to second-hand smoke during early life (including in utero) it also makes it more likely that these offspring will become smokers themselves later in life (*Tobacco and Genetics Consortium* 2010). But at the same time, relationships with family members may also affect parental longevity. While strong bonds with parents may provide positive psychological benefits to offspring and may increase longevity, the stress associated with caring for a sick parent may degrade health.

These examples also illustrate the reasons why the link between parental and offspring longevity might weaken over time. While a parent with high blood pressure faces a higher individual risk of dying from a heart attack, the genetic transmission of a predisposition to high blood pressure also means that the child faces a higher risk of dying from a heart attack (*Newton-Cheh et al.* 2009). However, with medical advances, mortality risk may be lowered; the child might still have a predisposition to high blood pressure as an adult, but drugs are now available to control blood pressure. In addition, todays improved access to emergency response and medical technology increases the chance for survival in the event of a heart attack. The link between parental and offspring longevity is also potentially weakened by improved knowledge about health and healthy behavior. There is now a better understanding of the health risks of smoking, which could weaken or break the link between parental smoking and the smoking habits of offspring, thereby improving the survival chances of the offspring of smokers relative to their parents.

The literature on the effects of family longevity on offspring mortality indicates a wide range of results, depending on the country studied and the study method employed. A number of studies examined individuals with exceptional longevity, focusing on whether their relatives lived longer than the relatives of those with typical life spans. Siblings of centenarians in the United States have a mortality advantage relative to others in the study (*Perls et al.* 2002). A related study

shows that both parents and siblings of "supercentenarians," i.e., those who live to age 110 or above, have substantial survival advantages over the general population, although the reason for these advantages remains unidentified. (*Perls et al.* 2007). Using the entire population of Iceland, along with a genealogy database, researchers examined whether survival is inheritable by following ancestors of those who survived to the 95th percentile, and found strong familial relation in survival rates (*Gudmundsson et al.* 2000). The Framingham study in the United States showed that parental survival to age 75 increases the probability among 50-year-olds of surviving to age 75 (*Goldberg et al.* 1996). Similarly, in Japan, the older the age of death of both mothers and fathers, the lower the mortality risk for their offspring between 40 and 79 years of age (*Ikeda et al.* 2006). A study of civil servants in Amsterdam, the Netherlands, found that the number of living parents increases the 15- and 25-year survival rates for middle-aged women, but shows no effect on the longevity of men (*Vandenbroucke et al.* 1984). Danish twins showed only moderately inherited longevity (*Herskind et al.* 1996). In Australia, the age at death of either parent had no significant effect on mortality, regardless of an individual's gender (*van Doorn and Kasl* 1998).

There is a larger degree of consensus with respect to morbidity and cause-specific mortality. Increases in parental age of death decrease the probability of an offsprings death from coronary heart disease, but not death by stroke or cancer (*Brand et al.* 1992). In a Japanese prospective study, the risk of mortalityparticularly as a result of cardiovascular disease decreases when fathers reach the age of 80 and when mothers reach the age of 85 (*Ikeda et al.* 2006). In Britain, longer parental longevity, especially for the mother, decreases the incidence of pulmonary disease, coronary heart disease, and hypertension (*Gjonca and Zaninotto* 2008). The longevity of females relative to the age of their parents at conception supports an argument for genetic transmission (*Gavrilov and Gavrilova* 1997). Adult daughters born to fathers older than 45 have a higher mortality risk, but an analogous effect is absent in sons born to older fathers. The effect of late fatherhood is eliminated for fathers who lived past the age of 81. These results are attributed to higher mutation rates in the X chromosome, passed only from fathers to daughters.

Even if one accepts that longevity is inheritable from parents, the reasons for this conclusion

remain unknown. Specifically, the extent to which a positive relationship between survival rates of parent and child is caused by genetics or by behavioral or environmental factors must be identified (*Cournil and Kirkwood* 2001). One approach uses adoptees to examine whether biological or adoptive parents mortality explains the adoptees mortality. Data on Danish adoptees born between 1924 and 1926 and monitored until age 58 shows that premature mortality is mainly determined by biological parents (when focusing on parents who died before age 50 and whose main causes of death were possibly genetic in nature) (*Sø rensen and Nielsen* 1988). Also in Denmark, an analysis of monozygotic and dizygotic twins showed that longevity was only moderately heritable, and that there was no evidence of impact from a shared family environment (*Herskind et al.* 1996). Both of these studies suffer from relatively small sample size. The Iceland study referenced above shows a statistically significant impact related to genetic transmission, whereas the environmental impact appears to be small and difficult to predict with the data (*Gudmundsson et al.* 2000).

Parental longevity could also affect mortality through emotional and social channels, although the specific mechanisms are largely unknown. For the non-institutionalized elderly in Spain, a poor relationship with at least one child was shown to increase mortality by 30% (*Zunzunegui et al.* 2009). Survival rates for adult children living with parents suggest a need for emotional, physical or financial dependence. That is to say, an unmarried adult man who lives with his parents and two siblings is three times more likely to die younger than a married man who lives with his spouse and two children (*Rogers et al.* 2000). Strong family relationships and positive networks provide social, emotional, and financial support,effectively reducing mortality. One example is the lower mortality rate among married individuals (*Lillard and Waite* 1995). It is unclear, however, if the effect is causal or is the result of healthier individuals being more likely to marry (*Kisker and Goldman* 1987).

To estimate the effects of parental longevity on offspring longevity, we use data from the Health and Retirement Study (HRS),[2] a longitudinal study conducted by the University of Michigan that tracks a representative sample of the civilian, non-institutionalized US population over 50 years

[2]Other studies that have examined mortality using the HRS are *Lee et al.* (2006) and *Mehta and Chang* (2009).

of age. HRS tracks respondents and their families biannually, recording health, functional, socio-economic, and expectational information. The ninth wave of this study was completed in 2008. A major advantage of the HRS compared to other studies is that it collects information on both the respondents and their parents. In particular, the survey asks whether a respondent's parents are still living, and if so, how old they are, and if not, how old they were when they died.

Two main statistical issues need to be accounted for when using the HRS to analyze the effect of parental longevity on offspring mortality: censoring and non-response. Censoring occurs because not all respondents and parents are dead at the time the latest round of the HRS occurs. Non-response increases with the length of the study. Non-response is an issue if it is non-random and correlated with the outcomes of interest. A respondent in poor health may, for example, be more likely to decline participation, and poor health is correlated with a higher risk of dying. To address these two issues, we model respondent death and survey non-response as distinct exit states using a discrete competing risk hazard model.

This paper makes three main contributions to the literature. First, it uses a rich data set with information on both adult offspring and their parents that allows us to examine the population at large, rather than focusing on just those individuals with "exceptional longevity." Second, we directly address the selection issues that arise from survey attrition and employ more flexible estimation methods than have previously been used. Finally, we identify the effects associated with an offspring having a parent still alive and control for the censoring associated with unobserved parental longevity.

We find that longer parental longevity significantly reduces both the probability of death and survey non-response. The positive effects on survival hold for both the mother and father's longevity and for both males and female. Consistent with previous studies, maternal longevity is a stronger predictor of mortality than paternal longevity (*Preston and Taubman* 1994; *Rogers et al.* 2000, 2005). We also find that the significant effect of parental survival remains even after controlling for offspring's health behavior and health status. This indicates that a significant genetic component to the link between parental and offspring longevity remains present.

2 Data

The Health and Retirement Study (HRS), conducted by the University of Michigan and sponsored by the National Institute on Aging, tracks over 22,000 Americans over the age of 50 longitudinally, with the purpose of expanding our knowledge of retirement, health, and other economic decisions made by older individuals. The study began in 1992, surveys individuals and households biennially, and has completed nine full waves of data to date. The data contain a broad set of health, functional, socio-economic, and expectational variables, and are unique due to the information collected on the characteristics of the primary respondent's extended family.

Our sample consists of HRS's initial cohort of respondents, surveyed during the first wave in 1992. The initial cohort consisted of 12,652 respondents, of which 10,155 are in our target group between the ages of 50 and 61. The remaining 2,497 respondents are mainly spouses of participants in our selected age group. After dropping 152 individuals because of missing data, the final sample consisted of 10,003 individuals, with 4,616 males and 5,387 females.

The top panel of Table 1 presents baseline summary statistics for the sample. The average respondent is approximately 55 years old. The HRS over-sampled Hispanics, blacks, and Florida residents during initial waves of the survey. We use the sampling weights provided by the HRS to account for over-sampling.

The second panel of Table 1 presents baseline health status. The variables are dummy variables that indicate whether the respondent has a particular health condition. Women are more likely to have arthritis, cancer, lung disease, and psychiatric disorders, whereas men are more likely to have a heart condition. The third panel presents behavioral factors. The average education level of the sample is roughly equivalent to a high school degree. A majority of respondents has smoked cigarettes at one time in their life and currently drinks alcohol. Men are more likely than women to engage in both behaviors. Vigorous exercise is defined as running, jogging, swimming, cycling, aerobics, gym workout, tennis, or digging with a spade. Over 19% of respondents report doing vigorous exercise three times or more per week.[3]

[3]Because of question framing, in waves 7 through 9, the indicator for vigorous exercise is coded as one if activities

Table 1: Baseline Summary Statistics for Respondents in the first HRS Wave

	Males		Females	
Variable	**Mean**	**Std. Dev.**	**Mean**	**Std. Dev.**
		Demographics		
Age	55.573	3.244	55.324	3.333
White	0.810	0.392	0.777	0.417
Black	0.153	0.36	0.185	0.389
		Health Factors		
Arthritis	0.31	0.463	0.442	0.497
Cancer	0.032	0.177	0.074	0.262
Diabetes	0.109	0.312	0.108	0.311
Heart condition	0.149	0.356	0.110	0.313
High blood pressure	0.400	0.490	0.391	0.488
Lung disease	0.076	0.265	0.085	0.279
Psychiatric disorder	0.08	0.271	0.139	0.346
Stroke	0.035	0.183	0.024	0.153
		Behavioral Factors		
Married	0.810	0.393	0.687	0.464
Education (years)	12.195	3.43	11.935	3.031
Number of children	3.169	2.113	3.389	2.139
Non-housing wealth	162496.492	449069.533	138129.816	387890.923
Earnings	29711.187	41092.646	12315.87	15718.427
Ever smoked	0.742	0.438	0.539	0.499
Alcohol use (1=yes)	0.682	0.466	0.535	0.499
Vigorous exercise	0.196	0.397	0.196	0.397

Note. The samples consist of 4616 males and 5387 females. Alcohol use is whether the respondent currently drinks alcohol. Vigorous exercise is defined as running, jogging, swimming, cycling, aerobics, gym workout, tennis, or digging with a spade three or more times a week.

Table 2: Parental Longevity Summary Statistics for Respondents in the Initial Wave of the HRS

	Males		Females	
Variable	**Mean**	**Std. Dev.**	**Mean**	**Std. Dev.**
Mother Alive (1=yes)	0.141	0.348	0.130	0.337
Father Alive (1=yes)	0.037	0.189	0.031	0.174
Mother Age (Died)	74.873	15.236	74.405	15.624
Father Age (Died)	70.997	14.315	70.076	14.878
Mother Age (Alive)	85.663	7.132	86.095	7.004
Father Age (Alive)	84.684	6.312	86.071	6.372

Note. Parents' maximum age and whether still alive is measured at the respondent's last observed survey.

Table 2 provides summary statistics on the longevity of respondents' parents. Most of the respondents parents were deceased by the ninth wave, with the exception of approximately 13% of mothers and 3% of fathers. Parents' ages of death are similar for both men and women; the average age of parental death is around 70 years for fathers and 75 years for mothers, and the average age of still-living parents is approximately 85.

Table 3 shows the longevity experience of HRS respondents. Approximately 20% of the 4,616 males and 13% of the 5,387 females in our sample have died by wave 9. At least one survey non-response was recorded in 29% of men and 28% of women.

3 Empirical Model

We use a competing risk discrete hazard model to analyze factors that influence respondent exits (*Allison* 1982; *Jenkins* 1995). For each individual, $(i = 1, \ldots, n)$, in the data we observe the spell from the date of the first survey to either death, survey non-response, or wave 9 of the survey. An individual's spell is measured in years $(t = 1, 2, 3, \ldots)$. Respondents are all between 50 and 61 years of age at the beginning of the spell. All analyses are stratified by gender because of inherent biological differences.

We assume there are two distinct failure types, $k = 1, 2$: death and survey non-response. We

are performed more than once per week.

Table 3: Tabulation of Survival Outcomes by Wave for Respondents in the Initial HRS Sample

	Wave							
Status	2	3	4	5	6	7	8	9
Males								
Alive	4,121	3,575	2,949	2,832	2,812	2,601	2,388	2,191
Died	103	124	84	105	131	112	140	123
Non-response	392	262	193	145	126	81	63	70
Total	4,616	3,961	3,226	3,082	3,069	2,794	2,591	2,384
Females								
Alive	4,929	4,392	3,707	3,559	3,589	3,394	3,172	2,975
Died	72	69	74	85	120	71	123	104
Non-response	386	292	195	209	168	107	81	86
Total	5,387	4,753	3,976	3,853	3,877	3,572	3,376	3,165

Note. Alive is the number of respondents in a given survey round that are alive at the time of the survey. Died is the number of respondents who have died between two survey rounds and non-response is the number of respondents who for any reason did not respond to a given survey.

record failure by non-response as the first wave a respondent fails to participate in a survey. Should a respondent reenter the survey, subsequent exits are eliminated. The reason we drop respondents even if they reenter the survey is to ensure that information on parental death and other important factors related to behavior and health are not missed during the time the respondent was out of the survey. Approximately 15.5% of all respondents do not respond to at least one survey and return in a future wave, compared to 13.1% who are never re-interviewed.

Define the latent variable $Y_k^* > 0$ as spell length before failure of type k occurs. The starting point for each spell is $t = 1$ and it continues until time $t_i = \min(Y_1^*, Y_2^*)$ at which point one of the failure types occur or the last survey takes place (the observation is censored). In addition to information about the spell length, there is information about individual and household characteristics that may vary over time and which are included in the vector of explanatory variables $\mathbf{X_{it}}$.

The discrete time hazard rate $h_{k,it}$ for individual i at time t for exit type k is defined as

$$h_{k,it} = \Pr(T_i = t \mid T_i \geq t; \mathbf{X_{it}}), \tag{1}$$

where T_i is a discrete random variable that captures the year an exit occurs. Of primary interest is

the distribution of T_i.

As a reference case, we estimate the discrete time hazard rate for exit by death, without accounting for survey non-response. The hazard rate in logit form is

$$\log\left[\frac{h_{1,it}}{1-h_{1,it}}\right] = c(t) + \beta' \mathbf{X_{it}} + \varepsilon_1, \tag{2}$$

where $c(t)$ is the baseline hazard function (the hazard when $\mathbf{X_{it}} = 0$). The likelihood specification of (2) has the same form as the standard binary logit model if the data are transformed, so the unit of analysis is person-years *Allison* (1982).

Allowing for survey non-response, the complete model specifies the hazard rate as

$$h_{k,it} = \frac{\exp(\alpha_{kt} + \beta_k' \mathbf{X_{it}})}{1 + \sum_l \exp(\alpha_{lt} + \beta_l' \mathbf{X_{it}})} \quad k = 1, 2. \tag{3}$$

To estimate (3) one must also specify the functional form for the baseline hazard function. Survival times are recorded as years in the HRS. As a result, we use a non-parametric baseline hazard that includes one-year age dummies for respondent's age at time t.

The advantage of specification (3) is that likelihood function is identical to the likelihood function for the multinomial logit model. In the reorganized data, the outcome variable is set to zero if the individual survives and participates in the next wave, one if the individual dies between survey waves, and two if the individual fails to respond during the next wave. Define survival to the next wave as the reference outcome $k = 0$, then the log odds ratio for type k exit is

$$\log\left[\frac{Pr(Y_{it} = k)}{Pr(Y_{it} = 0)}\right] = c_k(t) + \beta_k' \mathbf{X_{it}} + \varepsilon_k, \tag{4}$$

where $c_k(t)$ is the baseline hazard for event type k.

Interpreting parameter estimates from (4) presents two issues. First, the estimated parameters measure the change in probabilities relative to the censored outcome, rather than simply the probability of an event, as in the basic model. Second, an increase in a variable with a positive

coefficient may not increase the probability that the associated event occurs, because the probability of the other event may increase even more.[4] Parameter estimates are therefore accompanied by marginal effects that measure the change in the estimated probability of death resulting from an incremental change in an explanatory variable.

A more significant problem with the competing risk model is that it assumes that alternative exit states are stochastically independent, also known as the Independence of Irrelevant Alternatives (IIA) assumption. This assumption rules out any individual-specific unmeasured or unobservable risk factors that affect both the hazard of dying and the hazard of leaving the sample. In other words, the assumption requires that the hazard of dying relative to staying in the sample is uncorrelated with the corresponding relative hazard of attrition to staying in the sample.[5] For example, if people leave the sample due to health reasons (they are too sick to participate), then clearly that is correlated with the risk of dying. Hence, the simple competing risk model might lead to biased estimates. To account for dependence between exit states, we implement the approach proposed in *Han and Hausman* (1990). This approach specifies that the errors in expression (4) are distributed bivariate standard normal, with parameters identified under standard regularity conditions, with the most important being the inclusion of at least one continuous variable in the vector $\mathbf{X_{it}}$. The dependent exit model is estimated as a bivariate probit model. As with the competing risk model, we report marginal effects in addition to parameter estimates.

3.1 Specifying Parental Longevity

For ease of exposition we focus on the longevity of the father. Our specifications include similar variables for the mother. Let FA_i be the maximum observed age of the respondent's father and FC_i a dummy for whether the father was alive at the respondent's final survey. Our initial specification for paternal longevity is then

$$\alpha_1 FA_i + \alpha_2 FC_i + \alpha_3 FC_i \times FA_i, \tag{5}$$

[4] See *Thomas* (1996) for an illustration of this problem in a continuous time setting.

[5] See *Hill et al.* (1993) for a more thorough and formal discussion of the issues involved.

where FC captures the censoring of the father's age and the interaction captures the effect of the father's expected remaining years of life. Both FC and FA are time invariant variables.[6]

To evaluate the impact of father's age on mortality, we compute marginal effects conditional on the father's age being censored and uncensored, holding all other covariates at the sample mean. Standard error estimates for conditional marginal effects are obtained using the delta method. We also computed projections for life expectancy for 55-year-old males and females. Mortality rates are generated from the bivariate probit model and are conditional on individual gender, parent maximum age, and whether the parent was alive at the respondent's last survey. We apply Lowess smoothing to reduce noise associated with mortality estimates at older ages, where we have fewer observations. Years of remaining life are computed using standard life table methods. Mortality rates for ages 74 and above are extrapolated using the Gompertz method (*Vallin and Caselli* 2005).

Observable variables correlated to both parental longevity and individual mortality will moderate estimated marginal effects from (5). For example, mortality risk from heart disease has a strong genetic component (*Newton-Cheh et al.* 2009). To assess the relative importance of these characteristics on the estimate parental longevity marginal effects, we perform four analyses to disentangle the different mechanisms through which parental and offspring longevity are connected. First, we control only for variables capturing parental longevity. Second, in addition to parental longevity variables, we control for respondent health conditions. These health conditions include arthritis, cancer, diabetes, heart conditions, high blood pressure, lung disease, psychological disorders and stroke. Third, in addition to parental longevity variables, we control for respondent behavioral variables: marital status, education (years attained), number of children, household non-housing wealth, individual earnings, whether the respondent has ever smoked, whether the respondent drinks alcohol, and whether the respondent does vigorous exercise. Finally, the full model simultaneously controls for respondent health conditions, behavioral variables, and parental longevity. In all analyses, we control for race (white, black and other).

[6] We experimented with more complex specifications that allowed for differential effects of parental age, depending on whether the parents dies younger or older than the respondent's current age, but there is unfortunately not enough variation in the data to identify these effects.

4 Effects of Parental Longevity on Offspring Mortality

Table 4 presents coefficient estimates for parental longevity variables, controlling for race and age of respondent through the non-parametric baseline hazard that includes one year age dummies for respondent's age. In both the male and female samples, the higher the maximum age of parents, the lower the mortality risk for their offspring. This holds for both mothers' and fathers' ages. All parental maximum age coefficients are statistically significant. Hence, greater parental longevity is associated with reductions in mortality, with maternal effects larger than paternal effects. Stronger effects of maternal longevity are consistent with previous studies (*Gjonca and Zaninotto* 2008; *Brand et al.* 1992).

When a parent is dead, the maximum observed parental age is fixed and the marginal effect simply captures the effect of parental longevity on offspring longevity. For parents who were still alive at the respondent's last survey, the effect of maximum observed age does not represent a pure survival effect. Instead, an increase in parental age combines the survival benefit of an additional year gained and the benefit from the parent's future years of survival. In this case, the combination of coefficient estimates for maximum parent age, the dummy for parent age censored, and the interaction between parent age and the dummy for parent age censored captures the impact of parental longevity on respondent mortality. The effect is analogous to the fact that in life tables the expected age of death continues to increase as an individual ages.

Consistent with the low number of fathers still alive, none of the interactions are statistically significant and only for some specifications are the dummies for still alive statistically significant. The added survival benefit is, however, strongly present for maternal longevity with coefficients on dummies for still alive and interaction terms all statistically significant. Furthermore, the effects are large. The coefficient for the interaction between the mothers age censored and the maximum observed age of the mother is approximately 10 times larger than the coefficient for the direct effect of maximum observed age of the mother.[7]

Table 5 presents conditional marginal effects for parental longevity on mortality hazard rates.

[7]The combined effect of censoring and interaction is negative from around age 18.

Table 4: Coefficient Estimates for Parental Longevity Variables

	Logit[a]	Competing Risk[b]	Dependent Exit[c]
Males			
		Paternal longevity	
Max age	-0.007^{**}	-0.007^{**}	-0.003^{**}
	(0.002)	(0.002)	(0.001)
Age censored	0.858^{*}	0.978^{*}	0.459^{*}
	(0.390)	(0.412)	(0.199)
Censored × max age	0.010	0.006	0.002
	(0.021)	(0.022)	(0.011)
		Maternal longevity	
Max age	-0.012^{***}	-0.013^{***}	-0.006^{***}
	(0.002)	(0.002)	(0.001)
Age censored	2.621^{***}	2.854^{***}	1.240^{***}
	(0.184)	(0.185)	(0.090)
Censored × max age	-0.144^{***}	-0.160^{***}	-0.070^{***}
	(0.014)	(0.015)	(0.007)
Females			
		Paternal longevity	
Max age	-0.011^{***}	-0.011^{***}	-0.005^{***}
	(0.002)	(0.002)	(0.001)
Age censored	0.888	1.730^{**}	0.359
	(0.597)	(0.652)	(0.295)
Censored × max age	−0.030	−0.071	−0.012
	(0.033)	(0.037)	(0.016)
		Maternal longevity	
Max age	-0.018^{***}	-0.018^{***}	-0.008^{***}
	(0.002)	(0.002)	(0.001)
Age censored	2.611^{***}	2.880^{***}	1.166^{***}
	(0.196)	(0.199)	(0.098)
Censored × max age	-0.132^{***}	-0.151^{***}	-0.060^{***}
	(0.016)	(0.016)	(0.008)

Note. Standard errors in parentheses; * significant at 10%; ** significant at 5%; *** significant at 1%. All models control for respondent's race and and age of respondent through the non-parametric baseline hazard that includes one year age dummies for respondent's age. Parameters are not directly comparable because of the different estimation methods.

[a] Discrete hazards logit model with death as only exit.

[b] Discrete hazards multinomial logit model with death and survey non-response as the two possible exit states.

[c] Discrete hazards bivariate probit model with death and survey non-response as the two possible exit states.

Table 5: Marginal Effects of Parental Maximum Age by Parental Survival Status

	Father's Age		Mother's Age	
	Deceased	Alive	Deceased	Alive
Males	$-2.3 \times 10^{-4***}$	-1.4×10^{-4}	$-3.7 \times 10^{-4***}$	$-1.1 \times 10^{-2***}$
	(7.2×10^{-5})	(1.5×10^{-3})	(6.3×10^{-5})	(1.4×10^{-3})
Females	$-2.2 \times 10^{-4***}$	-1.4×10^{-3}	$-3.4 \times 10^{-4***}$	$-7.3 \times 10^{-3***}$
	(4.9×10^{-5})	(1.9×10^{-3})	(4.3×10^{-5})	(1.1×10^{-3})

Note. Standard errors in parentheses; * significant at 10%; ** significant at 5%; *** significant at 1%. Race and age are set to sample means when computing marginal effects.

Each cell shows the marginal effects by parent, conditional on parent's survival status. Race and offspring age are set to the sample means when computing marginal effects. Marginal effects for father's age are statistically significant when age is uncensored. The decrease in the probability of death is, for example, associated with an increase in father's age ranges from 0.00024 to 0.00025 for sons and 0.00020 to 0.00021 for daughters. Mother's age when uncensored is also associated with reductions in mortality risk. Marginal effect estimates for mother's age range from -0.00033 to -0.00035 among sons and -0.00031 to -0.00034 among daughters. Marginal effects for mother's maximum observed age when censored are statistically significant and relatively larger than those from uncensored age. These marginal effects range from -0.011 to -0.017 among sons and -0.0073 to -0.0140 among daughters.

Figures 1 and 2 show predicted hazard curves for different ages of parental death.[8] In each diagram, the lower parental age corresponds to the mean observed in data. The plots provide a graphical representation of the mortality benefit from increased parental longevity over time. Hazard curves are estimated using the full dependent competing risk model–assuming an initial respondent age of 50–and are evaluated using the mean characteristics in each sample. Mortality when evaluated at respective sample means is higher for sons than for daughters. Sons and daughters with older parents face lower hazard curves. Hazard curves within a subplot diverge with age, suggesting that larger survival benefits are gained as the individual increases in age. One possible

[8]Because we use a non-parametric baseline hazard function, the number of deaths is low for age bins with few observations, resulting in noisy estimates. Hence, Lowess smoothing with a bandwidth parameter of 0.4 was applied to all predicted hazard curves.

explanation for the lower difference at younger ages is that causes of death that are not directly related to genetics and health behavior, such as motor vehicle accidents, are more important at younger ages.

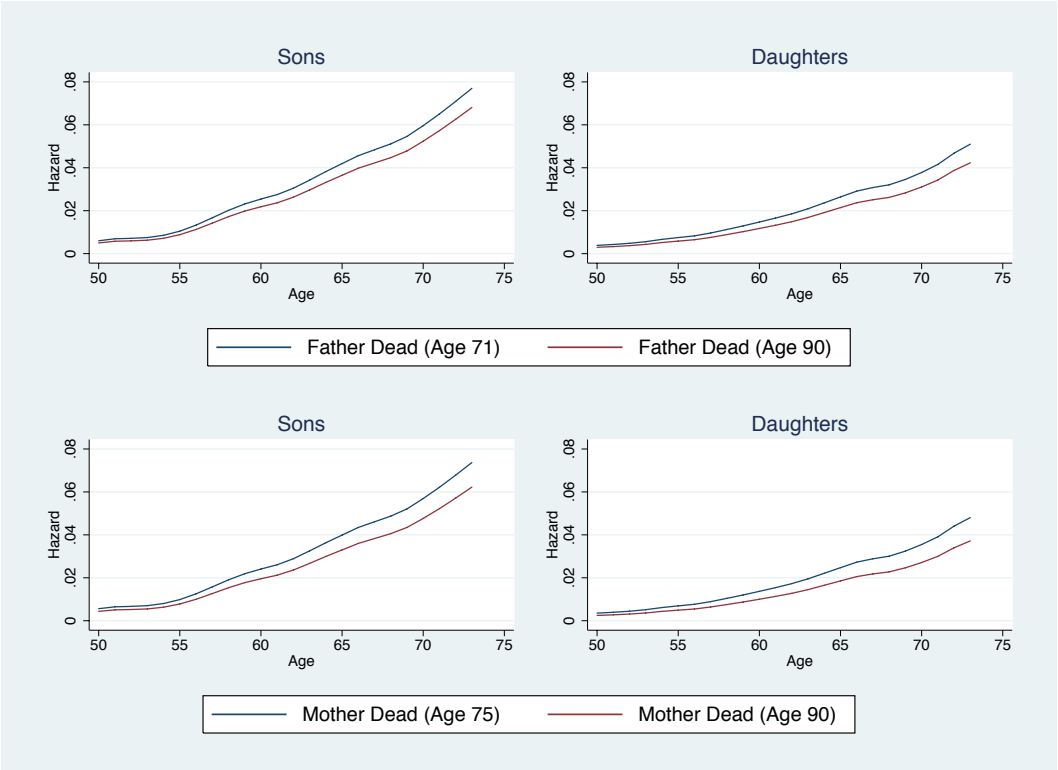

Figure 1: Comparison of predicted hazard curve for the average respondent by parent age of death.

Table 6 presents life expectancy estimates for 55year-old males and females, based on mortality rates from 1 and 2. When maximum parental age is uncensored, greater paternal and maternal longevity improved individual life expectancy. The impact of parental longevity is greater among women. For example, life expectancy for a daughter with a father 90 years of age at death is 1.6 years greater compared to a daughter with a father 71 years of age at death. For males, the difference in life expectancy is 1.1 years. The results in Table 6 also indicate that maternal longevity has a greater impact on individual mortality. Increasing maternal death from age 75 to age 90 resulted in 1.5- and 2.2-year differences in life expectancy for men and women, respectively. When maximum parental age is censored, increases in parental and maternal longevity have substantial impacts on life expectancy. Increasing maternal age from 88 to 100 results, for example, in 15.7-

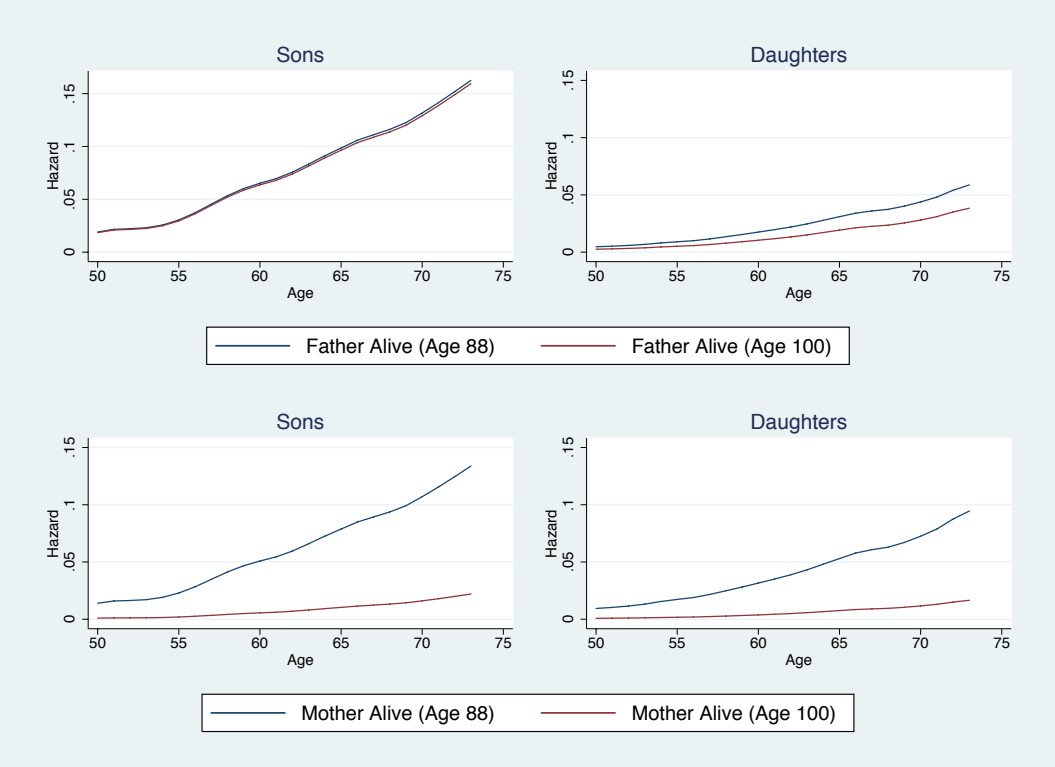

Figure 2: Comparison of predicted hazard curve for the average respondent by maximum observed parent age conditional on parent survival.

and 14.6-year increases in life expectancy for sons and daughters, respectively.

4.1 Components of Parental Age Effects

The results in the previous section show that the longevity of both fathers and mothers significantly reduce mortality risk of offspring. In other words, there is a positive correlation between how long your parents live and how long you live. The results do not, however, explain why we observe this relationship. At its simplest a person's mortality risk is determined by genetics, behaviors, and a random component. The problem in understanding how parental and offspring longevity are connected is the fact that each of these factors is only partially the result of genetic transmission from parents. Peoples genes are a mix of their biological father's and mother's genes and any spontaneous mutations that might occur. Behaviors are partly learned from parents during childhood, but may also be shaped by peers and ones own choices later in life. To further complicate matters,

Table 6: Life Expectancy Estimates for 55 Year Old Males and Females

	Males	Females
Father's Age		
Non-Censored - 90	24.460	28.829
Non-Censored - 71	23.374	27.225
Difference	1.112	1.604
Censored - 100	16.509	29.628
Censored - 88	16.330	25.953
Difference	0.179	3.674
Mother's Age		
Non-Censored - 90	25.262	29.893
Non-Censored - 75	23.747	27.715
Difference	1.515	2.178
Censored - 100	33.855	36.199
Censored - 88	18.187	21.596
Difference	15.668	14.603

Note. Standard errors in parentheses; * significant at 10%; ** significant at 5%; *** significant at 1% Predicted mortality rates at each age are from the bivariate probit model and are estimated holding covariates at sample means.

interactions between genetics and behavior may also be present. For example, depending on their genes, two people who smoke may have different health outcomes.

With these caveats, we compare marginal effects of changes in paternal and maternal age when controlling for different combinations of individual characteristics, in order to provide evidence on how parental longevity affects offspring longevity. We do this by dividing respondent characteristics into two groups: health variables and behavioral variables. Observed health variables are arthritis, cancer, diabetes, heart condition, high blood pressure, lung disease, psychiatric disorders, and stroke. Behavioral variables, which can also be called social variables, are marital status, education, number of children, non-housing wealth, earnings, smoking history, alcohol use, and participation in vigorous exercise. Very broadly speaking, health variables can be assigned in part as genetic transmission from parents to child, while behavioral variables may be more affected by socializing during upbringing. Clearly this division is not ideal; diabetes and high blood pressure may, for example, be the result of behaviors, and educational attainment and whether a respondent chooses to marry may also partly be the result of genetics. Table 7 shows a comparison of

Table 7: Marginal Effects of Parental Maximum Age when Including Health and Behavioral Variables

	Father's Age		Mother's Age	
	Deceased	Alive	Deceased	Alive
Males				
Base[a]	$-2.3\times10^{-4***}$	-1.4×10^{-4}	$-3.7\times10^{-4***}$	$-1.1\times10^{-2***}$
	(7.2×10^{-5})	(1.5×10^{-3})	(6.0×10^{-5})	(1.4×10^{-3})
Health Variables[b]	$-1.3\times10^{-4*}$	6.9×10^{-4}	$-2.6\times10^{-4***}$	$-9.8\times10^{-3***}$
	(7.3×10^{-5})	(1.1×10^{-3})	(6.0×10^{-5})	(1.3×10^{-3})
Behavioral Variables[c]	$-1.9\times10^{-4***}$	5.0×10^{-4}	$-2.9\times10^{-4***}$	$-1.0\times10^{-2***}$
	(7.2×10^{-5})	(1.3×10^{-3})	(6.0×10^{-5})	(1.4×10^{-3})
All Variables[d]	$-1.2\times10^{-4*}$	9.1×10^{-4}	$-2.3\times10^{-4***}$	$-9.2\times10^{-3***}$
	(7.3×10^{-5})	(1.1×10^{-3})	(6.0×10^{-5})	(1.3×10^{-3})
Females				
Base[a]	$-2.2\times10^{-4***}$	-1.4×10^{-3}	$-3.4\times10^{-4***}$	$-7.3\times10^{-3***}$
	(4.0×10^{-5})	(1.0×10^{-3})	(4.0×10^{-5})	(1.1×10^{-3})
Health Variables[b]	$-1.5\times10^{-4***}$	-2.0×10^{-3}	$-2.5\times10^{-4***}$	$-6.2\times10^{-3***}$
	(4.0×10^{-5})	(2.0×10^{-3})	(4.0×10^{-5})	(1.1×10^{-3})
Behavioral Variables[c]	$-1.7\times10^{-4***}$	-3.7×10^{-4}	$-2.8\times10^{-4***}$	$-6.5\times10^{-3***}$
	(4.0×10^{-5})	(1.0×10^{-3})	(4.0×10^{-5})	(1.1×10^{-3})
All Variables[d]	$-1.3\times10^{-4***}$	-9.0×10^{-4}	$-2.2\times10^{-4***}$	$-5.6\times10^{-3***}$
	(4.0×10^{-5})	(1.0×10^{-3})	(4.0×10^{-5})	(1.1×10^{-3})

Note. Standard errors in parentheses; * significant at 10%; ** significant at 5%; *** significant at 1%. Marginal effects based on dependent competing risk bivariate probit model. Covariates are set to sample means when calculating marginal effects. Results for base model and the model with all variables are in Appendix.

[a] Base model controls for respondent's race and age of respondent through the non-parametric baseline hazard that includes one year age dummies for respondent's age. Using separate regressions for men and women controls for respondents sex.

[b] This specification includes observed health variables in addition to the base model variables. Observed health variables are arthritis, cancer, diabetes, heart condition, high blood pressure, lung disease, psychiatric disorder, and stroke.

[c] This specification includes behavioral variables (can also be called social variables in addition to the base model variables. Behavioral variables are married, education, number of children, non-housing wealth / 100, earnings / 10,000, ever smoke, alcohol use, and vigorous exercise.

[d] This specification includes all base variables, observed health variables, and all behavioral variables.

parental age effects using the bivariate probit model.[9] The baseline model in the first row is the one presented above and does not include health or behavioral variables.

For men, the difference between the marginal effects of uncensored father's age in the baseline model and the model with health variables is 0.0001. This suggests that 42% of the baseline paternal age effect is attributable to observed health factors. Similarly, the paternal age effect is reduced by 19% when controlling for behavioral factors. For uncensored mother's age, health and behavioral factors constitute 30% and 20% of the estimated effect from the baseline model. Among daughters, 32% and 20% of the paternal effect is due to health and behavioral factors, respectively,

[9] Full results for the models are presented in the Appendix.

while health and behavioral factors constitute 26% and 19% of the maternal effect.

For censored maternal age, the changes in marginal effects are consistently lower than for uncensored maternal age.[10] Adding health or behavioral variables makes little difference for the marginal effects of maternal longevity on men's survival probability. The baseline marginal effect is -1.1×10^{-2} and controlling for health variables only reduces the marginal effect to -9.8×10^{-3} or around 10%. The change from including behavioral variables is even smaller, at less than 10%. For women, controlling for health variables reduces the marginal effect of maternal longevity by 15%, while controlling for behavioral variables reduces the effect by around 11%.

Overall, behavioral and health factors do reduce the parental longevity effects, with observed health factors appearing to explain a larger component of the parental longevity effects than behavioral factors. Combining health and behavioral variables in the last row of each panel in Table 7 provides an indication of the extent to which observable characteristics overall explain the relationship between parental and offspring longevity. For paternal age, the reductions are 48% for men and 41% for women. For uncensored maternal age, the reductions are 38% for men and 35% for women, and for censored maternal age the reductions are 16% for men and 23% for women.

Two things stand out in this analysis. First, health and behavioral characteristics appear to overlap, in the sense that the change in marginal effects is not simply the sum of the changes when including health variables and when including behavior variables. This is not surprising. People who smoke may, for example, be more likely to have cancer and those with a psychiatric disorder may have lower income and be less likely to be married. Second, there are substantial unexplained effects of parental longevity. The largest change is for the effect of paternal longevity on men where including both health and behavioral characteristics reduce the marginal effect by almost 50%. The smallest change is for censored maternal age on men's survival where combining both health and behavior only reduces the effect by 16%. Hence, we are left with between 52% and 74% of the overall effect of parental longevity that cannot be accounted for by the variables available in the data set. What, then, explains the remaining effect of parental longevity? It is possible that

[10]None of the marginal effects of censored paternal age are statistically significant and we therefore do not consider those changes.

there are important omitted variables in our analysis, or that the health and behavior variables in the data do not fully capture the underlying the complexity of the underlying health conditions and behaviors. Another possibility is that even with the duration of the HRS, the length of follow-up is insufficient to completely capture the incidence of conditions such as heart disease. Finally, the genetic transmission of longevity may, indeed, be largely unobserved.

There is some circumstantial evidence that a longer follow-up does affect how much of the effect is explained. At the conclusion of the last round of our data, 53% of men had died, while only 48% of women had died. The percentage of the parental longevity effect explained is larger for men than for women, although only by 3 percentage points. It is difficult to assess how important better measures of health and behavior would be in influencing our estimate, but given that about half of our sample has already died, it is unlikely that this will have a large enough effect to explain away the effect of parental longevity. As a result, this leaves unobserved genetic transmission of longevity as the most likely explanation for the observed positive relationship between parental longevity and offspring longevity.

4.2 Does Survey Dropout Matter?

The advantage of the dependent competing risk model compared to those in previous literature is the ability to directly deal with the problem of sample selection due to attrition. The dependent competing risk model simultaneously estimates hazards due to death and survey attrition, allowing for correlation across the exit states. The estimated correlation coefficients for the error terms across equations are -0.820 and -0.755 for the male and female samples, respectively. Both correlation coefficients are significant at the 1% level, suggesting that there exist unobservable factors that simultaneously affect both individual death and survey non-response.

Table 8 presents a comparison of marginal effects across different model specifications. Marginal effects conditional on the father having died are nearly identical across models for both males and females. The differences in maternal effects are more substantial. The dependent competing risk models estimate is approximately 10% larger than the single exit model estimate for both men and

Table 8: Marginal Effects for Parental Maximum Age for Different Models

	Father's Max Age		Mother's Max Age	
Model type	Uncensored	Censored	Uncensored	Censored
Males				
Discrete Hazard	-0.239^{***}	0.165	-0.325^{***}	-17.083^{***}
	(0.070)	(1.253)	(0.059)	(1.271)
Competing Risk	-0.240^{***}	-0.136	-0.325^{***}	-16.636^{***}
	(0.070)	(1.485)	(0.059)	(1.438)
Dependent Exit	-0.248^{***}	-0.240	-0.353^{***}	-10.905^{***}
	(0.071)	(1.538)	(0.062)	(1.308)
Females				
Discrete Hazard	-0.195^{***}	-1.617	-0.308^{***}	-14.042^{***}
	(0.048)	(2.413)	(0.041)	(1.352)
Competing Risk	-0.195^{***}	-3.799	-0.307^{***}	-12.904^{***}
	(0.048)	(4.202)	(0.041)	(1.733)
Dependent Exit	-0.206^{***}	-1.230	-0.336^{***}	-7.292^{***}
	(0.048)	(1.811)	(0.043)	(1.126)

Note. Standard errors in parentheses; * significant at 10%; ** significant at 5%; *** significant at 1%. Marginal effect and standard error estimates multiplied by 1000.

women. Differences in maternal marginal effects are even larger when the mother was still alive at the most recent survey, although here the dependent exit model shows the smallest effect and the single exit model the largest.

5 Conclusion

Differences in demographic characteristics affect an individual's mortality risk through numerous channels. The primary purpose of this paper is to illustrate how the longevity experiences of an individual's parents affect mortality risk. The results indicate that increased paternal or maternal longevity decreases the probability of death among Americans between the age of 50 and 75, with maternal longevity showing a larger effect than paternal longevity. Having longer-lived parents lowers an individual's mortality risk, even when controlling for observed behaviors and health conditions. This indicates that longevity is, in large part, the result of genetic factors. In addition, we find decreased mortality risk among aging parents who are alive, suggesting parental survival generates a positive informational effect on individual longevity, independent of the age of the

parents.

In longitudinal studies, survey non-response is often ignored. Non-response is a particular difficulty in the HRS, where up to 14% of eligible respondents do not complete the survey in a given wave. A second purpose of this paper is to evaluate the importance of non-response, and to identify the factors that result in non-response. Using a competing risk model allowing for survey exit by death and non-response, we find that individuals with higher vitalityspecifically, younger respondents who are in better healthwho presumably have a higher opportunity cost of completing the survey, are more likely to not respond than others in the survey. Ignoring survey non-response mostly biases downward the magnitude of the marginal effects of parental longevity.

This study leads to a number of important conclusions. First, there is clearly a strong relationship between parental and offspring longevity. Second, this connection remains, despite advances in medical technology and an increasing sophistication in health knowledge over time. Finally, there is a strong genetic component to the link between parental and offspring longevity. Our results raise two important questions: to what extent do offspring take account of their parents' longevity when forming expectations about their own survival, and to what extent does this affect offspring behaviors such as saving, labor force participation, purchasing of life insurance, and retirement timing. These remaining questions suggest important areas for future research.

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A Hazard Model Results

This appendix presents the results from the semi-parametric estimation of the three hazard models. Tables 9 and 10 show the result for the base model for men and women and Tables 11 and 12 show the results when also controlling for health and behavioral factors. Reported results are coefficient estimates and standard errors for the models specified in Section 3. The marginal effects of covariates for the average individual on the probability of death are displayed in square brackets. Under the assumption of a non-parametric proportional hazard functional form for time, the probability of death is decreasing with age although the relationship is not monotonic as a result of random sampling.

With regard to individual demographic characteristics, results are consistent with the literature on adult mortality (*Rogers et al.* 2000). Males and single respondents are at a higher risk of death. Black respondents face a higher mortality risk relative to the reference group, although the effect is not statistically significant. Also consistent with previous studies is the result that respondents that have higher earnings and hold larger stocks of wealth have a significantly lower mortality risk. Interestingly, we are unable to find any significant effect for education.

The presence of health conditions increases the probability of death. The exception is arthritis, which has a small negative effect. Diagnosis of cancer has the largest impact, increasing hazard by 0.021 to 0.025, significant at the 1% level. For behavioral characteristics, respondents who currently smoke or previously smoked also face increases in mortality risk. Finally, those who consume alcohol and engage in vigorous exercise reduce their chances of death. These behavioral characteristics are significant at the 1% level.

Table 9: Baseline Model for Males

	Logit	Competing Risk	Dependent Exit
White	-0.036	-0.073	-0.010
	(0.205)	(0.205)	(0.092)
	$[-9.2\times10^{-4}]$	$[-1.1\times10^{-3}]$	$[-5.8\times10^{-4}]$
Black	0.399	0.393	0.191
	(0.218)	(0.218)	(0.099)
	$[1.2\times10^{-2}]$	$[1.2\times10^{-2}]$	$[1.3\times10^{-2}]$
Father alive at last survey	0.858^{*}	0.978^{*}	0.459^{*}
	(0.390)	(0.412)	(0.199)
	$[3.2\times10^{-2}]$	$[3.8\times10^{-2}]$	$[4.2\times10^{-2}]$
Father's maximum observed age	-0.007^{**}	-0.007^{**}	-0.003^{**}
	(0.002)	(0.002)	(0.001)
	$[-1.8\times10^{-4}]$	$[-1.8\times10^{-4}]$	$[-2.0\times10^{-4}]$
Father alive $\times$ father's max age	0.010	0.006	0.002
	(0.021)	(0.022)	(0.011)
	$[2.6\times10^{-4}]$	$[1.4\times10^{-4}]$	$[1.4\times10^{-4}]$
Mother alive at last survey	2.621^{***}	2.854^{***}	1.240^{***}
	(0.184)	(0.185)	(0.090)
	$[2.0\times10^{-1}]$	$[2.1\times10^{-1}]$	$[1.8\times10^{-1}]$
Mother's maximum observed age	-0.012^{***}	-0.013^{***}	-0.006^{***}
	(0.002)	(0.002)	(0.001)
	$[-3.1\times10^{-4}]$	$[-3.1\times10^{-4}]$	$[-3.5\times10^{-4}]$
Mother alive $\times$ mother's max age	-0.144^{***}	-0.160^{***}	-0.070^{***}
	(0.014)	(0.015)	(0.007)
	$[-3.6\times10^{-3}]$	$[-3.9\times10^{-3}]$	$[-4.2\times10^{-3}]$
Constant	-2.848^{***}	-2.780^{***}	-1.596^{***}
	(0.519)	(0.519)	(0.243)

Note. Standard errors in parentheses; * significant at 10%; ** significant at 5%; *** significant at 1%. Marginal effects evaluated at means in square brackets. Number of observations for all models is 25,723. Results for age dummies available upon request.

Table 10: Baseline Model for Females

	Logit	Competing Risk	Dependent Exit
White	-0.153	-0.180	-0.088
	(0.216)	(0.216)	(0.092)
	$[-2.4\times10^{-3}]$	$[-2.6\times10^{-3}]$	$[-3.7\times10^{-3}]$
Black	0.279	0.259	0.106
	(0.224)	(0.224)	(0.095)
	$[4.7\times10^{-3}]$	$[4.6\times10^{-3}]$	$[4.5\times10^{-3}]$
Father alive at last survey	0.888	1.730^{**}	0.359
	(0.597)	(0.652)	(0.295)
	$[2.1\times10^{-2}]$	$[5.1\times10^{-2}]$	$[2.0\times10^{-2}]$
Father's maximum observed age	-0.011^{***}	-0.011^{***}	-0.005^{***}
	(0.002)	(0.002)	(0.001)
	$[-1.6\times10^{-4}]$	$[-1.6\times10^{-4}]$	$[-1.8\times10^{-4}]$
Father alive $\times$ father's max age	-0.030	-0.071	-0.012
	(0.033)	(0.037)	(0.016)
	$[-4.5\times10^{-4}]$	$[-1.1\times10^{-3}]$	$[-4.8\times10^{-4}]$
Mother alive at last survey	2.611^{***}	2.880^{***}	1.166^{***}
	(0.196)	(0.199)	(0.098)
	$[1.3\times10^{-1}]$	$[1.4\times10^{-1}]$	$[1.2\times10^{-1}]$
Mother's maximum observed age	-0.018^{***}	-0.018^{***}	-0.008^{***}
	(0.002)	(0.002)	(0.001)
	$[-2.7\times10^{-4}]$	$[-2.7\times10^{-4}]$	$[-3.1\times10^{-4}]$
Mother alive $\times$ mother's max age	-0.132^{***}	-0.151^{***}	-0.060^{***}
	(0.016)	(0.016)	(0.008)
	$[-2.0\times10^{-3}]$	$[-2.2\times10^{-3}]$	$[-2.3\times10^{-3}]$
Constant	-2.305^{***}	-2.255^{***}	-1.307^{***}
	(0.435)	(0.435)	(0.209)

Note. Standard errors in parentheses; * significant at 10%; ** significant at 5%; *** significant at 1%. Marginal effects evaluated at means in square brackets. Number of observations for all models is 31,959. Results for age dummies available upon request.

Table 11: Full Model for Males

	Logit	Competing Risk	Dependent Exit
		Base Factors	
White	0.073	0.040	0.021
	(0.217)	(0.217)	(0.100)
	$[1.2\times10^{-3}]$	$[1.1\times10^{-3}]$	$[9.2\times10^{-4}]$
Black	0.314	0.307	0.137
	(0.229)	(0.229)	(0.106)
	$[6.2\times10^{-3}]$	$[6.3\times10^{-3}]$	$[6.7\times10^{-3}]$
Father alive at last survey	0.761	0.871^{*}	0.383
	(0.415)	(0.438)	(0.212)
	$[1.9\times10^{-2}]$	$[2.2\times10^{-2}]$	$[2.4\times10^{-2}]$
Father's maximum observed age	−0.004	−0.004	−0.002
	(0.003)	(0.003)	(0.001)
	$[-7.3\times10^{-5}]$	$[-7.4\times10^{-5}]$	$[-8.5\times10^{-5}]$
Father alive × father's max age	0.023	0.019	0.010
	(0.023)	(0.024)	(0.012)
	$[3.9\times10^{-4}]$	$[3.2\times10^{-4}]$	$[4.5\times10^{-4}]$
Mother alive at last survey	2.530^{***}	2.754^{***}	1.217^{***}
	(0.186)	(0.189)	(0.093)
	$[1.3\times10^{-1}]$	$[1.4\times10^{-1}]$	$[1.4\times10^{-1}]$
Mother's maximum observed age	-0.009^{***}	-0.009^{***}	-0.004^{***}
	(0.002)	(0.002)	(0.001)
	$[-1.5\times10^{-4}]$	$[-1.5\times10^{-4}]$	$[-1.7\times10^{-4}]$
Mother alive × mother's max age	-0.130^{***}	-0.144^{***}	-0.063^{***}
	(0.015)	(0.015)	(0.007)
	$[-2.2\times10^{-3}]$	$[-2.5\times10^{-3}]$	$[-2.8\times10^{-3}]$
		Health Factors	
Arthritis	-0.251^{**}	-0.257^{**}	-0.107^{**}
	(0.080)	(0.080)	(0.036)
	$[-4.3\times10^{-3}]$	$[-4.4\times10^{-3}]$	$[-4.6\times10^{-3}]$
Cancer	1.040^{***}	1.026^{***}	0.509^{***}
	(0.099)	(0.100)	(0.048)
	$[2.8\times10^{-2}]$	$[2.8\times10^{-2}]$	$[3.5\times10^{-2}]$
Diabetes	0.586^{***}	0.567^{***}	0.279^{***}
	(0.086)	(0.087)	(0.040)
	$[1.2\times10^{-2}]$	$[1.2\times10^{-2}]$	$[1.5\times10^{-2}]$
Heart Condition	0.473^{***}	0.468^{***}	0.234^{***}
	(0.087)	(0.087)	(0.040)
	$[9.4\times10^{-3}]$	$[9.5\times10^{-3}]$	$[1.2\times10^{-2}]$
High Blood Pressure	−0.011	−0.009	−0.012
	(0.080)	(0.080)	(0.036)
	$[-1.8\times10^{-4}]$	$[-2.0\times10^{-4}]$	$[-5.3\times10^{-4}]$
Lung Disease	0.513^{***}	0.518^{***}	0.258^{***}
	(0.105)	(0.105)	(0.050)
	$[1.1\times10^{-2}]$	$[1.1\times10^{-2}]$	$[1.4\times10^{-2}]$
Psychiatric Disorder	0.302^{**}	0.296^{**}	0.154^{**}
	(0.109)	(0.109)	(0.051)
	$[5.9\times10^{-3}]$	$[5.9\times10^{-3}]$	$[7.7\times10^{-3}]$

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	Logit	Competing Risk	Dependent Exit
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Stroke	0.601^{***}	0.591^{***}	0.309^{***}
	(0.123)	(0.123)	(0.059)
	$[1.4\times10^{-2}]$	$[1.4\times10^{-2}]$	$[1.8\times10^{-2}]$
		Behavioral Factors	
Married	-0.486^{***}	-0.505^{***}	-0.229^{***}
	(0.089)	(0.089)	(0.041)
	$[-9.9\times10^{-3}]$	$[-1.0\times10^{-2}]$	$[-1.2\times10^{-2}]$
Education (years)	-0.003	-0.005	-0.003
	(0.012)	(0.012)	(0.005)
	$[-5.7\times10^{-5}]$	$[-7.1\times10^{-5}]$	$[-1.1\times10^{-4}]$
Number of Children	-0.008	-0.011	-0.005
	(0.018)	(0.018)	(0.008)
	$[-1.3\times10^{-4}]$	$[-1.5\times10^{-4}]$	$[-2.2\times10^{-4}]$
Non-Housing Wealth / 100	0.001	0.001	0.000
	(0.004)	(0.004)	(0.002)
	$[1.4\times10^{-5}]$	$[1.4\times10^{-5}]$	$[1.1\times10^{-5}]$
Earnings / 10k	-1.038^{***}	-1.044^{***}	-0.382^{***}
	(0.273)	(0.274)	(0.105)
	$[-1.8\times10^{-2}]$	$[-1.8\times10^{-2}]$	$[-1.7\times10^{-2}]$
Ever Smoke	0.634^{***}	0.632^{***}	0.278^{***}
	(0.105)	(0.105)	(0.045)
	$[9.7\times10^{-3}]$	$[9.7\times10^{-3}]$	$[1.1\times10^{-2}]$
Alcohol use (1=yes)	-0.293^{***}	-0.290^{***}	-0.131^{***}
	(0.080)	(0.080)	(0.036)
	$[-5.2\times10^{-3}]$	$[-5.2\times10^{-3}]$	$[-5.9\times10^{-3}]$
Vigorous Exercise	-0.707^{***}	-0.716^{***}	-0.308^{***}
	(0.093)	(0.093)	(0.040)
	$[-1.2\times10^{-2}]$	$[-1.2\times10^{-2}]$	$[-1.3\times10^{-2}]$
Constant	-3.494^{***}	-3.361^{***}	-1.865^{***}
	(0.563)	(0.564)	(0.266)

Note. Standard errors in parentheses; * significant at 10%; ** significant at 5%; *** significant at 1%. Marginal effects evaluated at means in square brackets. Number of observations for all models is 25,723. Results for age dummies available upon request.

Table 12: Full Model for Females

	Logit	Competing Risk	Dependent Exit
		Base Factors	
White	-0.064	-0.083	-0.032
	(0.223)	(0.223)	(0.097)
	$[-6.1\times10^{-4}]$	$[-6.6\times10^{-4}]$	$[-8.1\times10^{-4}]$
Black	0.107	0.096	0.047
	(0.230)	(0.231)	(0.101)
	$[1.1\times10^{-3}]$	$[1.1\times10^{-3}]$	$[1.2\times10^{-3}]$
Father alive at last survey	0.933	1.782^{**}	0.370
	(0.628)	(0.684)	(0.317)
	$[1.4\times10^{-2}]$	$[3.5\times10^{-2}]$	$[1.4\times10^{-2}]$
Father's maximum observed age	-0.007^{*}	-0.007^{**}	-0.003^{**}
	(0.003)	(0.003)	(0.001)
	$[-6.3\times10^{-5}]$	$[-6.4\times10^{-5}]$	$[-7.5\times10^{-5}]$
Father alive $\times$ father's max age	-0.025	-0.067	-0.010
	(0.036)	(0.040)	(0.017)
	$[-2.3\times10^{-4}]$	$[-6.1\times10^{-4}]$	$[-2.6\times10^{-4}]$
Mother alive at last survey	2.582^{***}	2.830^{***}	1.167^{***}
	(0.208)	(0.211)	(0.103)
	$[8.1\times10^{-2}]$	$[8.7\times10^{-2}]$	$[8.9\times10^{-2}]$
Mother's maximum observed age	-0.012^{***}	-0.012^{***}	-0.006^{***}
	(0.003)	(0.003)	(0.001)
	$[-1.1\times10^{-4}]$	$[-1.1\times10^{-4}]$	$[-1.5\times10^{-4}]$
Mother alive $\times$ mother's max age	-0.122^{***}	-0.140^{***}	-0.056^{***}
	(0.018)	(0.018)	(0.008)
	$[-1.2\times10^{-3}]$	$[-1.3\times10^{-3}]$	$[-1.4\times10^{-3}]$
		Health Factors	
Arthritis	-0.170	-0.188	-0.068
	(0.096)	(0.096)	(0.041)
	$[-1.6\times10^{-3}]$	$[-1.7\times10^{-3}]$	$[-1.7\times10^{-3}]$
Cancer	1.079^{***}	1.072^{***}	0.499^{***}
	(0.102)	(0.102)	(0.046)
	$[1.6\times10^{-2}]$	$[1.6\times10^{-2}]$	$[2.0\times10^{-2}]$
Diabetes	0.418^{***}	0.411^{***}	0.200^{***}
	(0.098)	(0.098)	(0.044)
	$[4.6\times10^{-3}]$	$[4.6\times10^{-3}]$	$[5.9\times10^{-3}]$
Heart Condition	0.430^{***}	0.431^{***}	0.193^{***}
	(0.100)	(0.100)	(0.044)
	$[4.7\times10^{-3}]$	$[4.8\times10^{-3}]$	$[5.7\times10^{-3}]$
High Blood Pressure	0.172	0.173	0.071
	(0.096)	(0.096)	(0.041)
	$[1.6\times10^{-3}]$	$[1.6\times10^{-3}]$	$[1.8\times10^{-3}]$
Lung Disease	0.523^{***}	0.518^{***}	0.251^{***}
	(0.108)	(0.108)	(0.049)
	$[6.1\times10^{-3}]$	$[6.1\times10^{-3}]$	$[8.0\times10^{-3}]$
Psychiatric Disorder	0.014	0.008	0.014
	(0.102)	(0.102)	(0.044)
	$[1.4\times10^{-4}]$	$[1.2\times10^{-4}]$	$[3.4\times10^{-4}]$

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	Logit	Competing Risk	Dependent Exit
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Stroke	0.669^{***}	0.659^{***}	0.335^{***}
	(0.127)	(0.128)	(0.061)
	$[8.7\times10^{-3}]$	$[8.7\times10^{-3}]$	$[1.2\times10^{-2}]$
		Behavioral Factors	
Married	-0.216^{*}	-0.212^{*}	-0.102^{*}
	(0.094)	(0.094)	(0.040)
	$[-2.1\times10^{-3}]$	$[-2.1\times10^{-3}]$	$[-2.6\times10^{-3}]$
Education (years)	-0.006	-0.009	-0.003
	(0.016)	(0.016)	(0.007)
	$[-5.6\times10^{-5}]$	$[-6.6\times10^{-5}]$	$[-7.5\times10^{-5}]$
Number of children	0.001	-0.002	-0.000
	(0.020)	(0.020)	(0.009)
	$[1.1\times10^{-5}]$	$[-1.0\times10^{-6}]$	$[-9.3\times10^{-6}]$
Non-Housing Wealth / 100	-0.068^{**}	-0.068^{**}	-0.025^{**}
	(0.024)	(0.024)	(0.009)
	$[-6.4\times10^{-4}]$	$[-6.4\times10^{-4}]$	$[-6.2\times10^{-4}]$
Earnings / 10k	-1.820^{***}	-1.802^{***}	-0.719^{***}
	(0.497)	(0.497)	(0.195)
	$[-1.7\times10^{-2}]$	$[-1.7\times10^{-2}]$	$[-1.8\times10^{-2}]$
Ever Smoke	0.684^{***}	0.686^{***}	0.288^{***}
	(0.096)	(0.096)	(0.040)
	$[6.4\times10^{-3}]$	$[6.4\times10^{-3}]$	$[7.1\times10^{-3}]$
Alcohol use (1=yes)	-0.469^{***}	-0.478^{***}	-0.197^{***}
	(0.100)	(0.100)	(0.042)
	$[-4.4\times10^{-3}]$	$[-4.5\times10^{-3}]$	$[-4.9\times10^{-3}]$
Vigorous Exercise	-0.736^{***}	-0.731^{***}	-0.298^{***}
	(0.123)	(0.123)	(0.049)
	$[-6.2\times10^{-3}]$	$[-6.2\times10^{-3}]$	$[-6.6\times10^{-3}]$
Constant	-2.942^{***}	-2.833^{***}	-1.598^{***}
	(0.492)	(0.492)	(0.234)

Note. Standard errors in parentheses; * significant at 10%; ** significant at 5%; *** significant at 1%. Marginal effects evaluated at means in square brackets. Number of observations for all models is 31,959. Results for age dummies available upon request.