The Link between Parental and Offspring Longevity*

Preliminary

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Abstract

Studies of adult mortality typically examine the impact of individual characteristics, but ignores that the characteristics of people closely linked to individuals also influence mortality risk. This paper examines the effect of parental longevity on survival outcomes of adult offspring using survey data from the Health and Retirement Study (HRS) between 1992 and 2008 and 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. Furthermore, we find that behavior variables explain between 30 and 40 percent of the total effect of parental age on offspring longevity. Even after controlling for health and behavioral variables of the offspring, parental age of death has a substantial impact on the survival of the adult offspring.

1 Introduction

Higher standard of living, advances in medical technology and better knowledge about health has dramatically improved human longevity.[1] Despite a large literature on determinants of longevity there is little agreement on the relation between parental longevity and offspring longevity and whether the improvements in life expectancy has weaken the link between the two. This paper presents new evidence on the link between survival of parents and their offspring and the extent to which it depends on genetic or social factors.

Children's longevity is related to their parents' longevity through both genetics and behaviour. Parents pass on genetic material that either improve and worsen their offspring's survival. The most extreme example is genetic diseases that lowers survival chances but parents might also pass on genes that affect their children's survival chances directly or indirectly. Tall parents may, for example, be more likely to have tall children. Being tall can be a benefit in itself, but can also improve survival by leading to higher income in adult life. Behavioural factors also create to a link between parental and child survival. An example is smoking. Parental smoking has both a direct effect on the children who are exposed to second-hand smoking during their early life (including in-vitro) and an indirect effect by increasing the probability that the children will themselves become smokers later in life. The implications of parental longevity also include social effects that may increase or decrease longevity depending on the nature of the relationship with family members. For example, strong bonds with parents may provide positive psychological benefits, and increase longevity while caring for a sick parent may degrade health.

These examples also show why the link between parental and offspring longevity might weaken over time. Say a parent is genetically predisposed to high blood pressure and therefore faces an increased risk of dying from a heart attack. If predisposition to high blood pressure is transferred genetically from parent to child then the adult children also have an elevated risk of dying from a heart attack. With advances in medical technology and improvements in income the children might still have high blood pressure as adults but drugs are now available to control blood pressure. In

[1] Between 1900 and 1999 the average lifespan in the United States increased by 30 years (CDC 1999).

addition, if they do have a heart attack they have a higher chance of survival because of better access to emergency response and medical technology. Better knowledge about health and healthy behaviour can also break the link. There is now a better understanding of the health risks of smoking and that could lead to a break in the link between parental smoking and the smoking habits of offspring thereby improving survival chances of the offspring relative to their parents.

There are two motivations for studying the relation between parental and offspring longevity. First, it adds to the understanding of what determines human longevity. Second, people have been shown to make decisions on consumptions, savings and retirement based on their subjective mortality expectations rather than expected mortality based on life tables (Gan et al. 2004; Salm 2006). If people based their expectations of survival on the experiences of related family members it is important to understand the strength of the link between the two. With increasing life expectancy ensuring that retirees have accumulated enough wealth to last through their life becomes even more important. Gauging life expectancy earlier in the life cycle provides a more certain target for wealth at retirement and directs resources to policies achieving this goal.

The ideal data set for this study would be one where there is information on both parents' and offsprings' age at death. Unfortunately, that type of data is difficult to come by. Instead, the data used in this analysis is from the Health and Retirement Study (HRS). The HRS is a nationally representative sample of Americans tracked for the purpose of understanding the outcomes of individuals entering retirement. The survey completed its ninth wave in 2008 and tracks respondents and their families biannually, recording health, functional, socio-economic and expectational information. Because neither all offspring nor all parents are dead by the latest round of the HRS we use a competing risk model to estimate the effect of parental longevity on offspring survival probability.

When analyzing individual outcomes through a longitudinal study such as the HRS, non-response increases with the length of the study. For example, the non-response rate in the 2006 wave of the HRS was approximately 12%. The main issue with non-response is that it is unlikely to be non-random, specifically the probability of non-response is likely to be closely correlated with

the outcomes of interest. A respondent in poor health may, for example, be more likely to decline participation in the interview and being in poor health is also likely to be correlated with higher risk of dying. To overcome this problem we model respondent death and survey non-response as distinct exit states using competing risk hazard model.

Results indicate that increases in parental longevity have significant negative effects on both the probability of death and survey non-response, respectively. The positive effects exist for both the mother and father. 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 find that the significant effect of parental survival remains even after controlling for offspring's health behavior and health status, indicating that there still is a significant genetic component to the link between parental and offspring longevity.

2 Literature Review

Adult mortality in the United States has received considerable attention in the medical, demographic and economic literature.[2] One of the main findings is that individuals with higher socioeconomic status have lower mortality odds. Socioeconomic status encompasses a number of factors, including financial resources. Individuals with a higher income and wealth have better resources to reduce the risk of disease and have improved access to health information and medical care, which lowers mortality odds (Lantz et al. 1998). Education also has a negative effect on mortality, even after controlling for financial resources (Deaton and Paxson 2001; Lleras-Muney 2005). One hypothesis is that more educated individuals make better decisions and have better information related to health, but the effect of education may also arise from omitted variables that affect both education and health. Controlling for background characteristics of sisters eliminates the effect of education on mortality (Wolfe and Behrman 1987). Finally, african-americans face higher mortality, but this effect disappears when controlling for income (Menchik 1993).

[2]Rogers et al. (2000) and Sickles and Taubman (1997) provide comprehensive surveys.

The existence of family relationships may decrease mortality through enhanced ties and positive networks that provide social, emotional and financial support. Married individuals have lower mortality (Lillard and Waite 1995). It is unclear if the effect is causal or the result of selectivity where healthier individuals are more likely to enter into marriage (Kisker and Goldman 1987). There is also evidence that better socioeconomic status of the spouse provides spillover effects to the individual. The same ties that provide positive support in some cases may generate emotional stress in others. For example, an individual caring for an ailing parent is an example of a family relationship which may have a negative impact on survival.

There is little consensus in the literature on the effects of family longevity on offspring mortality. In the Framingham Study parental survival to age 75 increases the probability among 50 year olds of surviving to age 75 (Goldberg et al. 1996). In [THE US/HOLLAND?], the number of living parents has a negative effect increases the 15 and 25 year survival rates for women, but have no effect on the longevity of men (Vandenbroucke et al. 1984). Using Australian data, the age of death (or current age if alive) for both parents does not significantly affect mortality, regardless of individual sex (van Doorn and Kasl 1998). Finally, siblings of centenarians in the US have a mortality advantage relative to other members of the 1900 cohort (Perls et al. 2002).

Existing studies have argued that improved parental longevity lowers mortality through genetic transmission, for example, by lowering the instance of chronic disease. Increases in parental age of death decrease the probability of individual death by coronary heart disease, but not death by stroke or cancer (Brand et al. 1992). Among Japanese individuals the risk of mortality, particularly 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 Britian longer parental longevity, in particular the mother's age of death, decreases the incidence of pulmonary disease, coronary heart disease and hypertension (Gjonca and Zaninotto 2008). The age of parents at conception also matters for the longevity of females through genetic transmission (Gavrilov and Gavrilova 1997). In particular, adult daughters born to fathers above the age of 45 have a higher mortality rate, but an analogous effect is absent in sons and for fathers who lived past the age of 81 the effect of late fatherhood is

eliminated. The results are attributed to higher mutation rates in the X chromosome passed from fathers only to daughters. Individuals with longer lived parents have improved health through reductions in the probability of developing chronic disease.

Parental longevity could also affect mortality through emotional and social channels, although the specific mechanisms are largely unknown. For community dwelling elderly in Spain, poor relationships with at least one child increased mortality by 30% (Zunzunegui et al. 2009). Families where adult children living with parents suggest a need for emotional, physical or financial dependence. For example, unmarried males living with his parents and two siblings are three times more likely to die than a married male living with his spouse and two children (Rogers et al. 2000). At later stages of the life cycle, the dependence is likely reversed as elderly parents, often in poor health, cohabitant with their children, potentially creating damaging social relations and increasing mortality risk.

A handful of studies look at mortality of respondents in the Health and Retirement Study. The data have been used to estimate a logistic model to develop a prognostic index for mortality (Lee et al. 2006). The final index consists of twelve variables and includes a mix of demographic, comorbidity, behavioral and functional measures that are significant in the logistic estimation. Taking advantage of variables capturing subjective measures and questions gauging physical health and functionality, absence of health insurance or intermittent coverage has been shown to be associated with the degradation of health and increases the probability of developing a difficulty in physical function (Baker et al. 2001). Finally, the data have been used to estimate the effect of body mass index (BMI) on mortality, showing that mortality risk increases only when individuals reach the class II and class III obese categories (Mehta and Chang 2009).

3 Data

The Health and Retirement Study (HRS) conducted by the University of Michigan and sponsored by the National Institute on Aging is a nationally representative sample of Americans. The study

tracks over 22,000 Americans over the age of 50 with the purpose of expanding on the theory concerning retirement, health and other economic decisions of older individuals. The study, which began in 1992 and surveys individuals and households biennially now consists of nine full waves of data. The HRS data set contains a broad set of health, functional, socio-economic and expectational variables. The HRS is also unique in the broad set data collected that pertains to the extended family of the primary respondent.

This study focuses on the longevity experience of the initial cohort of respondents that entered the HRS during the first wave in 1992. This initial cohort consisted of 12,652 respondents, of which 10,155 are in the target group between the ages of 50 and 61. Spouses of age eligible respondents account for the difference. The final sample consisted of 10,003 individuals after 152 were omitted because of missing covariate data. The top panel of Table 1 presents baseline summary statistics for the study sample. The average respondent is over 55 years old. Individuals possess 12 years of education and 3.287 children on average. Mean earnings are $20,343, which includes respondents who did not report labor income. Mean non-housing wealth is $149,374. Females make up the majority of respondents, representing 53.9% of the sample. Over 74% of respondents are married. The ethnic composition consists of 79% caucasian and 17% black. The HRS intentionally over sampled hispanics, blacks and residents from Florida during initial waves of the survey. In our analysis, sampling weights generated by the HRS were used to account for oversampling.

The second panel of Table 1 presents baseline health status. The self reported measure of health is a categorical response ranging from 1 (poor health) to 5 (excellent health). The mean response is between 2 (fair health) and 3 (good health). The average BMI is 27.18, within the overweight category. The remaining variables in the second panel are dummy variables indicating whether the respondent has a given health condition. The third panel presents behavioral factors. The majority of respondents have smoked at one time in their life (63.3%) and currently drink alcohol (60.3%). Vigorous exercise is defined as running, jogging, swimming, cycling, aerobics, gym workout, tennis, or digging with a spade. Over 19% of respondents report vigorous exercise three times or

more per week[3]. Table 2 provides summary statistics for the longevity experience of respondent's parents. The majority of parents are deceased by the ninth wave with 97% of fathers and 87% of mothers in the category. The average age of parental death is approximately 70.50 years for fathers compared to 74.62 years for mothers. The average maximum observed age for fathers is 85.37 and 85.89 for mothers conditional on survival to the ninth survey wave. The longevity experience of HRS respondents is tabulated in Table 3. Approximately 16% of the respondents in the study sample have died by wave 9. Also, 28.23% of respondents have at least one survey non-response. The number of respondents who die in each wave is around 200.

4 Empirical Model

The method used to estimate factors that influence respondent exits is the competing risk model (Allison 1982; Jenkins 1995). An advantage of the methods proposed for this study in comparison with the previous literature is that it is possible to directly deal with the problem of sample selection due to attrition. For each individual $(i = 1, \ldots, n)$ in the data we observe at least one spell. All spells are measured in years $(t = 1, 2, 3, \ldots)$. We analyze the mortality experience of respondents present in the first wave of the survey. The starting age of these respondents is between 50 and 61 years of age. Assume there are two distinct failure types, $k = 1, 2$ which are death and survey non-response, respectively. In the analysis, we record failure by non-response as the first wave respondents fail to participate in a survey, and eliminate subsequent exits should a respondent re-enter the survey. Approximately 13% of respondents do not respond to at least one survey and return in a future wave. Define the latent variable $Y_k^* > 0$ as the length of the spell before failure of risk 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).[4] The variable δ_i is equal to one if individual i is uncensored (the individual dies or drops out in the

[3]Because of question framing, in waves 7 and 8, the indicator for vigorous exercise is set to one if activities are performed more than once per week.

[4]The time of censoring is assumed independent of the hazard rate as is standard in the literature on hazard models.

given period); otherwise it is zero. In addition to information about the spell length, there is also information about various individual and household characteristics that may vary over time and 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 at which an exit occurs. It is the distribution of T_i which is of primary interest here.

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. Allison (1982) shows that the likelihood specification is the same form as the standard binary logit model if the data are transformed so the unit of analysis is spell year rather than the individual.

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

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

The advantage of this specification is that it leads to the same likelihood function as a 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 (the hazard when $\mathbf{X_{it}} = 0$). One issue to keep in mind with this approach is that interpretation of the results is no longer straightforward. 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. Secondly, an increase in a variable with a positive coefficient may not increase the probability that the associated event occur since the probability of another event(s) may increase even more.[5]

A more significant problem with the competing risks model is that it assumes that alternative exit states are stochastically independent, also know as the Independence of Irrelevant Alternatives (IIA) assumption. This 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.[6] If people leave the sample due to, for example, health reasons (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 an approach proposed in Han and Hausman (1990). This approach specifies that the errors in expression (4) are distributed bivariate standard normal, and is identified under standard regularity conditions, the most important being the inclusion of at least one continuous variable in the vector $\mathbf{X_{it}}$. We estimate (3) as a bivariate probit model.

4.1 Baseline Hazard

To estimate (3) one must also specify the functional form for the baseline hazard function. The possible forms run from a simple constant to completely non-parametric. The non-parametric form consists of as many dummies as there are time periods and is the most flexible, leading to the best fitting model. The main drawbacks of the non-parametric version are that it requires events to occur in each period, that it may fluctuate erratically across periods because of nothing more than

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

[6] See Hill (1993) for a more thorough and formal discussion of the issues involved and a suggested method for estimating a more general model.

sampling variation and that with long spells it requires inclusion of a large number of unknown parameters. Alternatives to the non-parametric form are the piece-wise constant baseline hazard rate, which includes dummies equal to one for periods that are expected to have the same hazard, and polynomial specifications.

Survival times are recorded as years in the HRS. As a result, we choose a non-parametric baseline hazard that includes a dummy variables for all possible respondent ages. As a robustness check, we estimate the competing risk model assuming a continuous baseline hazard function and apply fractional polynomials (Royston et al. 1999) to determine the best fitting functional form.

The preceding models assume a non-proportional baseline hazard. However, for certain characteristics, the assumption that hazard risk is increasing proportionally may not be plausible. As a second robustness check, we also estimate a non-proportional hazard model for gender. For non-proportional hazard models, the baseline hazard function includes age bins and interaction terms for age and gender.

4.2 Individual Characteristics

To identify the effects of family longevity, we include variables that decompose the underlying survival effect derived from the parent as well as the effect from social relations when parents remain alive. Variables capturing family longevity include a dummy variable for the father currently alive, the maximum observed age of the father and an interaction between survival and father's age. The maximum age is either the father's age of death, or the last recorded father's age if both the respondent and father are still alive at the last wave. Analogous variables are included for the respondent's mother. The dummy for a parent alive identifies the network effect of parental survival on the probability of death. On the one hand, healthy relationships with living parents may improve health through financial resources from the parents, or enhance emotional health. However, strained relationships with parents may have a negative impact, particularly if an individual must care for a debilitated parent.

Including parental age captures two effects. If a parent is deceased, the maximum age captures

the underlying propensity to survive that is derived from that parent. This may include genetic and behavioral components. When the parent is still alive, the maximum observed age is an informational component that arises from knowledge that a parent has reached a certain age. Continued increases in the age of a living parent likely provide positive information about an individual's longevity. The information may not only affect individuals on an psychological level, but also affect lifestyle and behavior in a manner that improves longevity. We hypothesize that the increases in maximum observed age are associated with decreases in mortality risk regardless if a parent is alive or deceased. Similarly, we hypothesize that increases in parental longevity decrease the probability of survey non-response through a positive social effect.

For parents who remain alive at the end of the sample, the age of death is censored. In these cases, the dummy variable for parent alive likely captures part of the survival propensity from age of death. A dummy variable for parent alive at the last survey wave and an interaction with the maximum age are included to correct for the bias caused by the censoring of parental age.

To model the relationship between sibling longevity and individual mortality, we add variables capturing the number of brothers, number of sisters, respectively. In the empirical analysis, we also focus on variables related to demographics and comorbidity. Demographic variables in the vector $\mathbf{X_{it}}$ include individual earnings, household non-housing wealth and dichotomous variables for marital status, gender and ethnicity (caucasian and black).

To capture the effects of health and comorbidity, we include indicators for the existence of health conditions recorded by the HRS. These health conditions include arthritis, cancer, diabetes, heart conditions, high blood pressure, lung disease, psychological disorders, and stroke. Additionally, we add indicator variables for BMI that include normal, overweight, obese (class I-III) catagories[7]. The underweight group is taken as the reference. Finally, we include a set of behavioral variables that include whether respondents have smoked at any time in their life, whether the respondent drinks alcohol and engagement in vigorous exercise.

[7]For the definition of BMI catagories, see `http://apps.who.int/bmi/index.jsp?introPage=intro_3.html`.

5 Results

5.1 Parental Age

Table 4 summarizes average marginal effects for parental age. Columns 2 and 3 provide marginal effects conditional on the parents deceased and alive, respectively. Greater parental longevity is associated with reductions in hazard. In the case where the parent is deceased, the maximum observed parental age is fixed and the marginal effect captures the propensity for survival derived from the parent. Marginal effects are nearly identical across models for parents who are deceased. A marginal increase in father's age is associated with a 0.00014 decrease in hazard. The marginal effect for mother's age is 0.00022.

For parents who are alive, the marginal effect does not represent a pure survival effect because the age of death is censored. Instead, a marginal increase in age combines the survival benefit from the year gained in the present and the benefit from a parent's expected future years of survival. The effect is analogous to the fact that in life tables the expected age of death continues to increase as an individual ages. As a result, the marginal effects for parent age conditional on survival are larger in magnitude relative to the effects conditional on parent death. The marginal effect ranges from -0.00008 to -0.00078 for father's age and from -0.0072 to -0.012 for mother's age. All effects with the exception of paternal age in the censored case are statistically significant at the 1% level. Stronger effects from maternal longevity are consistent with previous studies (Gjonca and Zaninotto 2008; Brand et al. 1992).

Because individual hazard, irrespective of parent's longevity, differs across the age distribution for our sample, we hypothesized that the marginal effect of parental age would also differ across time. Figure 1 displays the marginal effect of parent age as a function of respondent age and stratified by parent survival status. The survival benefit of paternal longevity is relatively constant across age. In contrast, the benefit of maternal longevity is decreases with age if the mother is deceased, but is U-shaped if alive. One potential reason for this pattern is that health conditions associated with mortality and have a hereditary component (i.e. heart disease, cancer) manifest at younger

ages. For example, Chow et al. (2011) show that individuals with parents who have a history of myocardial infarction are themselves at at a higher risk of myocardial infarction. However, the risk is greater for men below age 55 and women below age 65.

Predicted hazard curves in Figure 2 and 3 provide a comparison of the mortality benefit of parental longevity over time. An individual with a father or mother who was older at death faces lower hazard curves. Larger survival benefits are gained as the individual increases in age. The benefit is particularly large in the case where the mother's age of death is censored (Figure 3), and results in the high mortality rates among respondents with a mother who is alive.

5.2 Hazard Model

Primary results from the semi-parametric estimation of the three hazard models (2) for survey exit by death are in Tables 5 and 6. Reported results are coefficient estimates and standard errors for the models specified in Section 4. Also, the marginal effects of covariates for the average individual on the probability of death are displayed in italics. 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 in Table 5, we obtain results 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 smaller 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. In addition, there exist non-linearities for the effect of BMI, as the mortality risk is initially decreasing, but increases at the right tail of the BMI distribution. For example, there is a 0.020 to 0.025 decrease in the probability of death for individuals

classified as overweight relative to those classified as underweight. As individuals enter the obese categories, mortality risk increases and is related to the onset of comorbid conditions that decrease life expectancy.

For behavioral characteristics, respondents who currently or previously smoked also face increases in mortality risk. Finally, those who consume alcohol and engage in vigorous exercise reduce their chances of death. All behavioral characteristics are significant at the 1% level.

5.3 Correlation Between Exit States

The bivariate probit model simultaneous estimates hazard due to death and survey attrition allowing for correlation across the exit states. The estimated correlation coefficient for the error terms is -0.780, significant at the 1% level suggesting that there exist unobservable factors that affect both individual death and non-response.

Without allowing for correlation between outcomes, the magnitude of the marginal effects are generally biased downward, particularly for health characteristics. For example, the marginal effect for cancer is 0.021 in the standard competing risk model compared to 0.025 in the dependent exit model. Parental longevity effects are nearly identical across models when the parent's maximum age is observed. When the age of death is censored there is a modest attenuation when allowing for a second competing risk and correlation across exit states. Figure 4 displays the predicted hazard curve across models when estimates for the average respondent. The single exit model underestimates mortality risk relative to the two competing risk models with differences increasing with age. Comparing the two competing risk models, mortality is lower in the dependent risk model for at younger ages, but is reversed for respondents over the age of 60.

5.4 Components of Parental Age Effects

The effect of parental longevity influences individual mortality through both improved health and behavior [REFS]. For example, a long lived parent may have a robust survival effect derived from the absence of hereditary conditions, but also maintain a healthy lifestyle that includes exercise

which is shared by other family members. The results in the previous section show that parental longevity, independent of health and behavior, significantly reduce mortality risk. However, to determine whether parental age captures the survival benefit from health and behavior, we compared marginal effects for maximum parent age after including different combinations of characteristic sets. Table 7 provides a comparison using the dependent exit model. The baseline model in the fourth row does not include health or behavioral variables. For deceased mothers, the difference between the marginal effect without health variables only is 0.00009, suggesting 39% of the parental effect is due to behavior variables. Approximately 9% of the maternal effect is due to health factors. Paternal effects are only significant when the father is deceased. About 30% of the paternal effect is due to behavioral characteristics and 10% due to health.

6 Conclusion [Needs updating]

Differences in demographic characteristics affect an individual's mortality risk that operate through numerous channels. The primary purpose of this paper is to illustrate how the longevity experience of family members, particularly an individual's parents, affects mortality risk. The results indicate that increases in both paternal and maternal longevity decrease the probability of death among Americans between the age of 50 and 75. Longer lived parents decrease an individual's mortality risk through increases in the predisposition for longer life, which may include genetic factors. The results also indicate that survival propensity is transmitted through maternal longevity only. In addition, we find that decreases in mortality risk through aging parents who are alive, suggesting parental survival generates a positive informational effect on individual longevity. Finally, we find evidence that parental survival influences mortality though the social relationships individuals have with parents. Unlike the underlying survival propensity, the informational and social effects are present for both 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 the chapter is to evaluate the importance of account for non-response, while identifying 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 vitality and have a higher opportunity cost to completing the survey are more likely to not respond. Specifically, this includes younger respondents in better health. Ignoring survey non-response biases downward the magnitude of the marginal effects pertaining to death, particularly in variables capturing parental longevity.

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7 Appendix A: Results

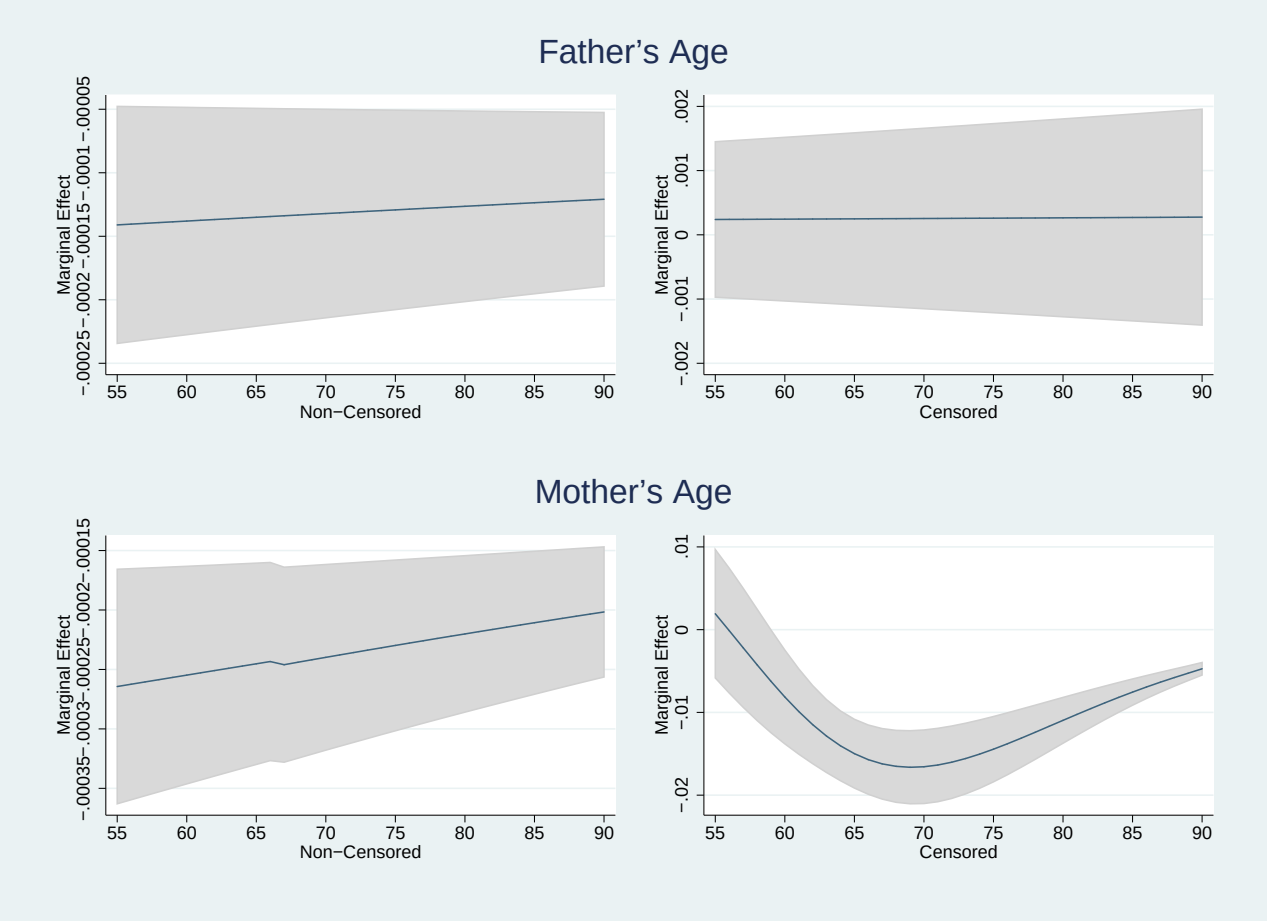

Figure 1: Marginal effects of respondent's father's and mother's age, respectively, on death hazard in the dependent competing risk model.

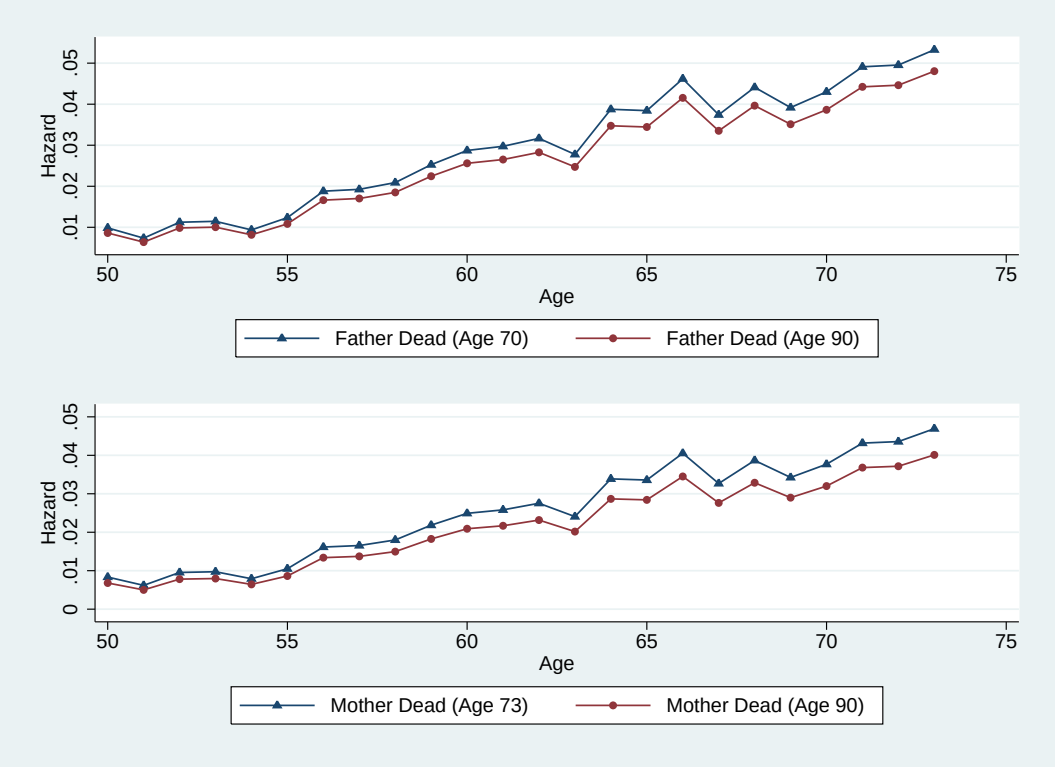

Figure 2: Comparison of predicted hazard curve for the average respondent by parent age of death.

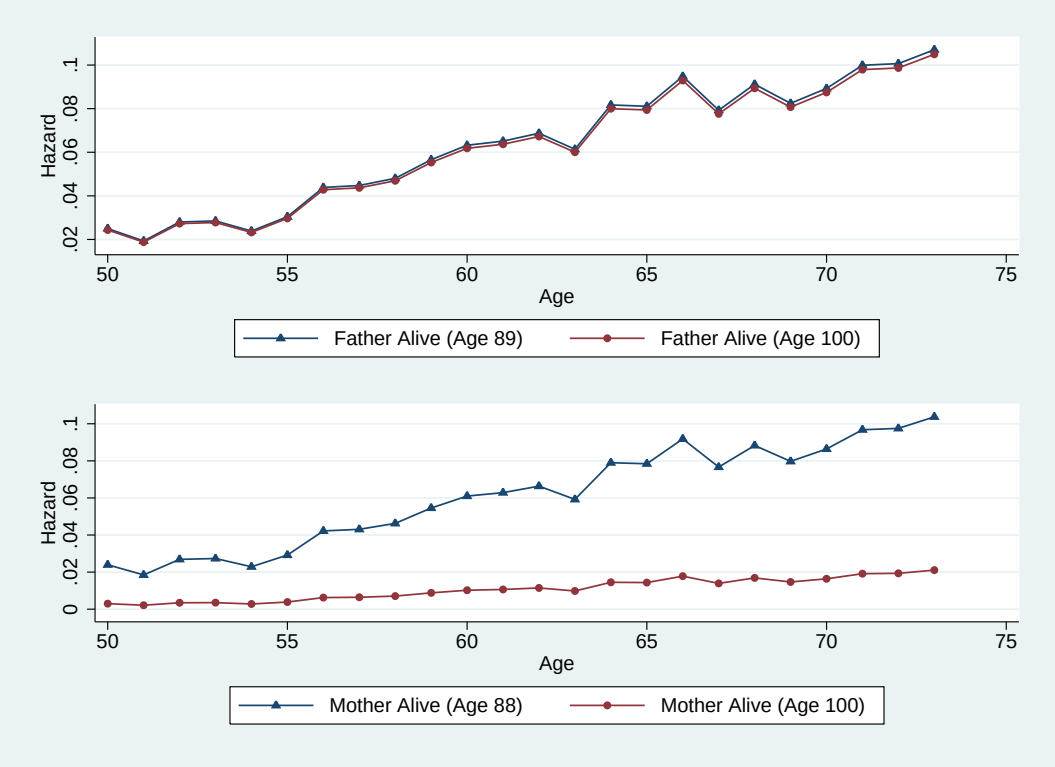

Figure 3: Comparison of predicted hazard curve for the average respondent by maximum observed parent age conditional on parent survival.

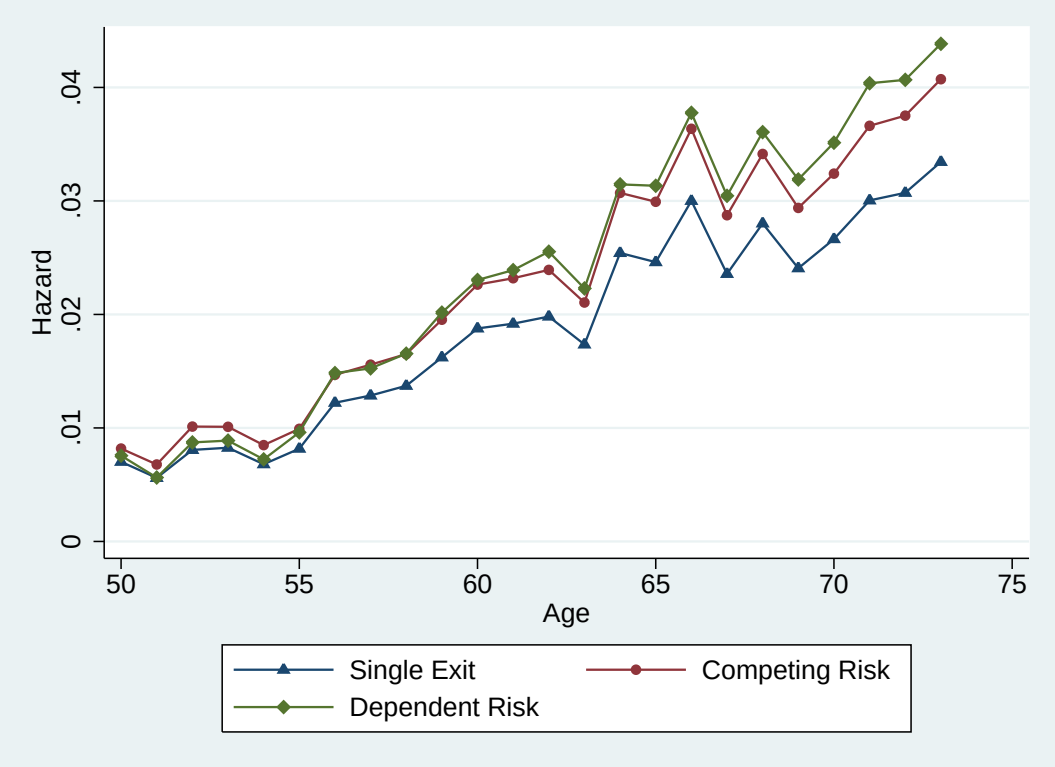

Figure 4: Comparison of predicted hazard curve across models.

Variable	Mean	Std. Dev.	N
Demographics			
Age	55.439	3.295	10003
Male (1=yes)	0.461	0.499	10003
Married (1=yes)	0.744	0.437	10003
White	0.792	0.406	10003
Black	0.171	0.376	10003
Education (years)	12.055	3.224	10003
Num. Children	3.287	2.13	10003
Non-Housing Wealth	149374.1	417394.116	10003
Earnings	20343.14	31422.726	10003
Health Factors			
Self Reported Health	2.592	1.204	10003
BMI	27.158	5.16	10003
Arthritis	0.381	0.486	10003
Cancer	0.055	0.228	10003
Diabetes	0.109	0.311	10003
Heart Condition	0.128	0.334	10003
High Blood Pressure	0.395	0.489	10003
Lung Disease	0.081	0.273	10003
Psychiatric Disorder	0.112	0.315	10003
Stroke	0.029	0.168	10003
Behavioral Factors			
Ever Smoked	0.633	0.482	10003
Ever Drink	0.603	0.489	10003
Vigorous Exercise (1=yes)	0.196	0.397	10003

Table 1: Baseline summary statistics for respondents in the first wave of the HRS.

Variable	Mean	Std. Dev.	N
Mother Alive	0.135	0.342	10003
Father Alive	0.034	0.181	10003
Mother Age (Died)	74.62	15.448	8652
Father Age (Died)	70.499	14.628	9663
Mother Age (Alive)	85.887	7.067	1351
Father Age (Alive)	85.374	6.37	340

Table 2: Parental longevity summary statistics for respondents in the initial wave of the HRS.

	Wave								
Status	2	3	4	5	6	7	8	9	Total
Alive	9,050	7,897	6,570	6,323	6,317	5,916	5,504	5,105	52,682
Dead	175	193	157	190	250	178	258	223	1,624
Drop Out	778	547	388	346	288	182	142	153	2,824
Total	10,003	8,637	7,115	6,859	6,855	6,276	5,904	5,481	57,130

Table 3: Tabulation of survival outcomes by wave for respondents in the initial HRS sample.

	Mortality	
Model	Dead	Alive
	Father's Maximum Age	
Discrete Hazard	-1.4e-04***	-1.1e-04
	(4.3e-05)	(9.0e-04)
Competing Risk	-1.4e-04***	-7.8e-04
	(4.3e-05)	(1.3e-03)
Dependent Exit	-1.3e-04***	-8.2e-05
	(4.2e-05)	(8.9e-04)
	Mother's Maximum Age	
Discrete Hazard	-2.2e-04***	-1.2e-02***
	(3.6e-05)	(1.0e-03)
Competing Risk	-2.2e-04***	-1.1e-02***
	(3.6e-05)	(1.3e-03)
Dependent Exit	-2.2e-04***	-7.2e-03***
	(3.7e-05)	(8.5e-04)

Table 4: Marginal effects for parental maximum age conditional on the parent alive and dead, respectively. Note, * denotes 10% significance, ** denotes 5% significance and *** denotes 1% significance.

	Logit	Competing Risk	Dependent Exit
Demographic Variables			
Male	0.723***	0.728***	0.327***
	(0.063)	(0.063)	(0.028)
	9.6e-03	*9.7e-03*	*1.1e-02*
Married	-0.344***	-0.349***	-0.156***
	(0.066)	(0.066)	(0.029)
	-4.7e-03	*-4.8e-03*	*-5.5e-03*
White	0.116	0.092	0.038
	(0.159)	(0.159)	(0.071)
	1.4e-03	*1.3e-03*	*1.2e-03*
Black	0.314	0.306	0.135
	(0.166)	(0.166)	(0.075)
	4.5e-03	*4.5e-03*	*4.9e-03*
Education	-0.010	-0.013	-0.005
	(0.010)	(0.010)	(0.004)
	-1.2e-04	*-1.4e-04*	*-1.7e-04*
Num. Children	0.006	0.002	0.002
	(0.014)	(0.014)	(0.006)
	7.1e-05	*5.7e-05*	*7.6e-05*
Financial Variables			
Non-Housing Wealth / 100	-0.004	-0.004	-0.001
	(0.011)	(0.010)	(0.003)
	-5.0e-05	*-5.0e-05*	*-4.9e-05*
Earnings / 10k	-1.036***	-1.038***	-0.407***
	(0.240)	(0.240)	(0.094)
	-1.3e-02	*-1.3e-02*	*-1.3e-02*
Behavioral Variables			
Ever Smoked	0.631***	0.629***	0.268***
	(0.071)	(0.071)	(0.030)
	7.5e-03	*7.5e-03*	*8.2e-03*
Ever Drink	-0.376***	-0.379***	-0.161***
	(0.063)	(0.063)	(0.027)
	-4.8e-03	*-4.9e-03*	*-5.4e-03*
Vigorous Exercise	-0.713***	-0.717***	-0.301***
	(0.074)	(0.075)	(0.031)
	-8.2e-03	*-8.3e-03*	*-9.0e-03*
Observations	57130	57130	57130

Table 5: Results from the semi-parametric estimation of mortality as a function of demographic, financial and behavioral variables. Marginal effects are in italics. Note, * denotes 10% significance, ** denotes 5% significance and *** denotes 1% significance.

	Logit	Competing Risk	Dependent Exit
Body Mass Index			
Normal	-1.232***	-1.232***	-0.602***
	(0.143)	(0.145)	(0.072)
	-1.3e-02	*-1.3e-02*	*-1.6e-02*
Overweight	-1.708***	-1.714***	-0.805***
	(0.145)	(0.147)	(0.072)
	-2.0e-02	*-2.1e-02*	*-2.5e-02*
Obese Class I	-1.984***	-1.994***	-0.926***
	(0.158)	(0.159)	(0.077)
	-1.6e-02	*-1.6e-02*	*-1.8e-02*
Obese Class II	-1.871***	-1.889***	-0.873***
	(0.181)	(0.183)	(0.087)
	-1.2e-02	*-1.2e-02*	*-1.3e-02*
Obese Class III	-1.468***	-1.479***	-0.661***
	(0.196)	(0.197)	(0.095)
	-1.0e-02	*-1.0e-02*	*-1.1e-02*
Health Variables			
Arthritis	-0.136*	-0.147*	-0.058*
	(0.062)	(0.062)	(0.027)
	-1.7e-03	*-1.8e-03*	*-1.9e-03*
Cancer	1.043***	1.033***	0.492***
	(0.071)	(0.071)	(0.033)
	2.1e-02	*2.1e-02*	*2.5e-02*
Diabetes	0.640***	0.628***	0.290***
	(0.067)	(0.068)	(0.031)
	1.0e-02	*1.0e-02*	*1.2e-02*
Heart Condition	0.475***	0.474***	0.227***
	(0.066)	(0.066)	(0.030)
	7.0e-03	*7.1e-03*	*8.8e-03*
High Blood Pressure	0.180**	0.182**	0.074**
	(0.063)	(0.063)	(0.028)
	2.3e-03	*2.3e-03*	*2.4e-03*
Lung Disease	0.443***	0.442***	0.220***
	(0.076)	(0.076)	(0.035)
	6.7e-03	*6.7e-03*	*8.8e-03*
Psychiatric Disorder	0.120	0.112	0.067
	(0.076)	(0.076)	(0.034)
	1.6e-03	*1.6e-03*	*2.3e-03*
Stroke	0.576***	0.566***	0.297***
	(0.090)	(0.090)	(0.043)
	9.5e-03	*9.5e-03*	*1.3e-02*
Corr. Coeff			-0.780***
			(0.052)
Observations	57130	57130	57130

Table 6: Results from the semi-parametric estimation of mortality as a function of BMI and health variables. Note, * denotes 10% significance, ** denotes 5% significance and *** denotes 1% significance.

	Father's Age		Mother's Age	
	Dead	Alive	Dead	Alive
All Variables	-1.3e-04***	-8.20e-05	-2.2e-04***	-7.2e-03***
	(4.20e-05)	(8.90e-04)	(3.70e-05)	(8.50e-04)
Health Variables	-1.4e-04***	-5.40e-04	-2.3e-04***	-7.1e-03***
	(4.20e-05)	(1.10e-03)	(3.70e-05)	(8.40e-04)
Behavioral Variables	-1.8e-04***	-2.10e-04	-2.9e-04***	-8.1e-03***
	(4.10e-05)	(9.60e-04)	(3.60e-05)	(8.60e-04)
None	-2.0e-04***	-9.70e-04	-3.2e-04***	-8.2e-03***
	(4.10e-05)	(1.30e-03)	(3.60e-05)	(8.50e-04)

Table 7: Comparison of marginal effects for parental maximum age with the inclusion of individual characteristic sets. Note, * denotes 10% significance, ** denotes 5% significance and *** denotes 1% significance.