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Let the Little Children Come to Me

Abstract

We document the relationship of a set of individual choices - including parenthood, marital state, and income - with an individual's cause of death. Using the data set of the Office for National Statistics Longitudinal Study (ONS-LS) which follows one percent of the population of England and Wales along five census waves 1971, 1981, 1991, 2001, and 2011, our competing risks analysis yields several striking results. 1) Females have only a 28% chance to die of cancer when they have children (compared to childless females); 2) males have a 71% increased chance of dying from cancer when they are married (compared to unmarried males); 3) females with children have only a 34% risk to die of heart disease and 4) a 53% chance of dying from infections (compared to females without children); 5) married men have an increased expectation of 23% to die of heart disease (compared to unmarried men); 6) high income and house ownership always is associated with higher survival but less so than having children.

JEL-codes: I100, J100.

Keywords: children, mortality. longevity.

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1 Motivation

While the relationships of lifestyle choices such as smoking, obesity, drinking, and other behavioral factors with life expectancy and causes of death are well-studied and understood (Rizzuto & Fratiglioni, 2014; Gutterman, 2017; Krokstad et al., 2017), the same is not true for individual decisions resulting in becoming a parent, being married, or ending up rich.[1]

Modig et al. (2017) study the association between parenthood and longevity by following Swedish men and women born between 1911 and 1925. They find (in age progressively) lower death risks for individuals having at least one child than for childless men and women. The study performs sensitivity analyses with respect to the gender of the child, parents' educational achievements, and geographical distance between parent and child and attributes the difference in death risks to the support children provide to their aging parents. Extending this approach, we are interested in the correlation of an individual's lifestyle choices with the causes of death. There are several recent investigations of the association of all-cause or cause-specific mortality with parity that are indirectly relevant to our project. For instance, Zeng et al. (2016) is a dose-response meta-analysis of 18 cohort studies including 2.813,418 participants. They find that moderate-level parity is inversely associated with all-cause mortality: participants with no live birth have a 19% higher relative risk of all-cause mortality compared with participants with one or more live births.[2]

Berntsen (2011) is an investigation into the association of cause-specific mortality with marital state in Norway. In tune with the previous literature, the paper finds that, "relative to married persons, those who are never married, divorced or widowed have significantly higher mortality for most causes of death." Kaplan & Kronick (2006) study the relation between marital status and survival and Manfredini et al. (2017) is a more focused investigation into the association of marital status and disruption with poor physical health outcomes, including all-cause mortality.

Our motivator for performing this study is to test what we call the "parental co-immunization hypothesis," the idea that a parent's or carer's immune system is refreshed by a child's infections at a time when their own protection starts wearing thin. With this boosted immune system, the parent has a better chance to fend off whatever infections might strike when old and weak and parenthood is rewarded in individual terms through an improved immunization against infections. Although we indeed find a reduced parental risk of dying from infections, we can document a similarly beneficial association of parenthood with other causes of death

[1] For a beautiful introduction to the theory of aging see Fabian & Flatt (2011). The classic aging theories balance longevity with reproduction and typically predict that, over a genotype's life span, there is a genetic trade-off between early reproduction and late fitness. Hence, these theories usually associate an increased number of children with *decreased* lifespan (Partridge & Barton, 1993; Kirkwood & Austad, 2000; Flatt & Promislow, 2007). An analysis of the association between the number of children (i.e., parity) and the mortality of mothers is Dior et al. (2013). They observe higher mortality rates for mothers than for women without children. Life circumstances and the presence of factors influencing socioeconomic pathways and health as well as lifestyle choices, however, may reasonably be expected to confound empirical analyses and contribute to non-monotonic associations between mortality and fertility (McNamee, 2003).

[2] Further recent and related studies include Einiö et al. (2016), Högnäs et al. (2016), and Kaptijn et al. (2015).

which are not compatible with the hypothesis and therefore cannot be explained by the theory.

2 Design of the empirical strategy

Our event of interest is "death" associated with a set of specific causes or diseases. Death is most adequately modeled as the probability of dying given that the person survived until that time and, hence, time until failure (duration or survival) models are most appropriate. In the following discussion we assume that time is described by a continuous random variable.

For some subjects in our sample the total survival time cannot be accurately determined. Although for part of the sample the duration data has information on the time from a well-defined starting point until the event of interest occurs, it is also the case that for specific individuals we only know the time until the end of the data collection process. This could happen because the subject drops out, is lost to follow-up, or because the study ends before the subject experiences the event of interest. In the latter case, the individual survived at least until the end of the study and we face right censoring as the individual is removed from the study before the event occurs.

Each individual is characterized by *(i)* survival time or spell, *(ii)* status at the end of the survival time (event occurrence or censored), and, in some cases, *(iii)* the study group (s)he is in. In our case the groups are "Alive," dead due to a "Specific cause" and dead for "Other reason". The specific causes of death we consider are Infections, Pulmonary disease, Cancer, Heart disease, Accidents/Homicides/Suicides (Acc/Hom/Suic), and Other causes. We will split the analysis into a sequence of steps, where in each step we classify each one of those six causes as a "Specific cause," while aggregating all the other causes under "Other reason."

One can interpret the group "Other reason" as a competing risk that occurs instead of the failure event of interest. One needs to specifically deal with different competing events, which implies that the model has to account for the fact that the number of failures from any competing risk (of failure) will condition on the number of failures from the main failure, which, in turn, implies changes in the estimated failure probability. Failures from any competing risk reduce the number of individuals at risk of failure from the cause under analysis (Gooley et al., 1999). This implies that we cannot treat it as censored, which renders a one-risk-type model, like for instance the Cox model, infeasible to deal with our survival analysis. As such, a competing risks framework becomes a natural solution for our estimation strategy.

Formulation of the competing risks model. In a general setting, for each individual in a competing risks model, the type of failure is identified by the index j, where $j = 1, \ldots, k$. The random duration variable is defined by $T^{(j)}$, where $T^{(j)}$ is the time to exit/failure to state j after the elimination of all other possible states. A spell ends when individuals leave for one of the k possible states. The states are mutually exclusive and exhaustive. We assume that there exists only one period of duration.

The k random variables, $T^{(1)}$,$T^{(2)}, \ldots, T^{(k)}$, can be interpreted as latent durations. These

are abstract time periods used in the construction of the econometric model underlying our empirical analysis. Entry into a certain state is dictated by the smallest latent time period (the smallest $T^{(j)}$), so the time to failure can be specified as $T^{(j)} = min[T^{(1)}, \ldots, T^{(k)}]$. For each individual, only one $T^{(j)}$ is observed in the data and others are considered censored. We consider a competing risks model with independent risks under the assumption that the random variables $T^{(1)}, \ldots, T^{(k)}$ are independent.

Under this setup it is possible to estimate conditional and unconditional probability functions that characterize the variables T and J. The expression

$$\lambda_J(t,x) = \lim_{dt \to 0} \frac{P(t \leq T < t + dt, J = j | T \geq t, x)}{dt} \tag{1}$$

is the transition intensity into state j, and x is a vector of explanatory variables consisting of individual characteristics.[3] These functions are designated as cause-specific hazard functions, which can be empirically interpreted as the fraction of survivors at time t that subsequently leave for state j.[4]

Similarly to concentrating on the *cause–specific hazard function*, we also focus on the *cumulative incidence function* (CIF) rather than the survivor function.[5] A CIF is just the probability that a specific type of event is observed before a given time, and can be defined as

$$CIF_J(t) \ = P(T < t, J = j). \tag{2}$$

The CIF gives the proportion of individuals at time t who have died of cause j accounting for the fact that patients can die of other causes. For example, the CIF for death due to Infection (which is one of the possible states discussed below) depends not only on the hazard for death by infection but also on the remaining hazards associated with other causes of death. This implies that it is no longer possible to define a direct relation between cause-specific hazard rate and the probability of death.

Although nonparametric estimation of CIFs is flexible, it cannot be adjusted for relevant regressors as they are associated with the cause-specific hazard. The efficient (and correct) way to run CIF covariate analysis is to implement a competing risks regression, according to the model of Fine & Gray (1999). They propose an alternative to cause-specific hazards: a semiparametric model for the hazard of the subdistribution for the failure event of interest, known as the subhazard. The *subhazard function* for failure cause j can be defined as

$$\bar{\lambda}_J(t,x) = \lim_{dt \to 0} \frac{P\left\{(t \leq T < t + dt, J = j | T > t \text{ or } (T \leq t \text{ and } J \neq j), x)\right\}}{dt}. \tag{3}$$

Under this formulation, there is a one-to-one correspondence between subhazards and CIFs for respective event types; that is, the CIF for a specific cause of death is a function of only the

[3] We design our empirical analysis as single–record data and time–invariant covariates and coefficients.

[4] In our survival analysis we ignore the role of possible unobserved heterogeneity due to currently unsurmountable technical difficulties in our environment (Deng et al., 2000).

[5] The Kaplan-Meier statistic would be inadequate for estimating the survival function from lifetime data. Berry et al. (2010) summarize the argument: "Kaplan-Meier survival analysis and Cox proportional hazards regression [...] can overestimate risk of disease by failing to account for the competing risk of death."

subhazard for that cause of death. Covariates affect the subhazard proportionally, similar to the Cox regression. From the relation between the hazard and survival functions, Fine & Gray (1999) define a subdistribution function.[6]

3 Data

We use census data from England and Wales provided by the Office for National Statistics Longitudinal Study (ONS-LS) which follows one percent of the respective populations along five census waves 1971, 1981, 1991, 2001, and 2011. From this data set we use information on age (`age`), time & cause of death (we split the ICD-coded causes of death into the seven categories of Table 1), child births (`yngkids`), marital status (`married`), profession, income & class status (`highclass_track`), home ownership (`house_owner`), and gender.[7] From the professional information we identify individuals working with children (`working_yngkids`). We define the variable `female`=1 for females; it takes the value of 0 otherwise. The precise descriptive statistics are provided in Table 2.

The actual data we use has the following characteristics. We start with 788,558 observations of individuals which we restrict to 547,957 by keeping only those alive in 1971. We drop visitors and perform several consistency checks on the data. From this set we only consider those aged between 16 and 50 in 1971. The combined sample is 204,277 individuals consisting of 99,520 females and 104,757 males (from starting figures of 403,968 and 384,590, respectively).

Table 1: Health status

Status	Females (%)		Males (%)		Frequency	Share (%)
Alive	65·9	52·3	57·2	47·7	125,502	61·4
Infection	2·3	46·6	2·5	53·4	4,832	2·4
Pulmonary disease	1·9	43·9	2·4	56·1	4,402	2·2
Cancer	9·0	47·9	9·3	52·1	18,616	9·1
Heart disease	6·8	33·5	12·8	66·5	20,230	9·9
Acc/Hom/Suic	0·8	33·9	1·5	66·1	2,311	1·1
Other diseases	4·9	50·5	4·6	49·5	9,708	4·8
Errors, Open, Others	8·4	44·9	9·8	55·1	18,676	9·1
	100·0	48·7	100·0	51·3	204,277	100·0

Notes: Status indicating alive, or cause of death. Within each pair of columns, Females & Males, the left column represents the share of each status, while the right column shows the share of females and males with this specific attribute. Source: Own computations based on ONS-LS.

In this sample, roughly 39% of the individuals have died. Details on the causes of death are provided in Table 1. The most common cause of death is Heart disease (25.7%), followed by

[6] For a more detailed discussion of competing risks models see, for instance, Kalbfleisch & Prentice (2002).
[7] For easier identification we use the names in the data set `monospaced`.

Cancer (23.6%). Infection attributes to about 6.1% of deaths.[8] Females represent about 49% of the sample being over-represented among those who are Alive. By contrast, they are clearly under-represented in Heart disease (34%) and Acc/Hom/Suic (34%).

Table 2: Descriptive statistics

Variable	Females (%)	Males (%)	Overall (%)
`age`	67·2	65·5	66·3
	(13·1)	(12·9)	(13·0)
`yngkids`	90·9	88·1	89·5
`working_yngkids`	4·9	2·5	3·7
`married`	90·5	88·1	89·3
`highclass_track`	64·1	52·9	58·3
`house_owner`	80·9	79·9	80·4

Notes: The total number of observations is 204,277 (the share of females is about 49%). Age is computed in years. (Standard deviations in parentheses.) Source: Own computations based on ONS-LS.

From Table 2 we observe that females live on average by about 1.7 years longer. Roughly 90% of our sample have young children, `yngkids`. The share of females who have at some point in their lives worked with young children, `working_yngkids`, is 4.9% (2.5% for males). The share of individuals who were married, `married`, were in white collar professions, `highclass_track`, or own a house, `house_owner`, are 90%, 58%, and 80%, respectively.

4 Results

Tables 3 & 4 show our results that were estimated using the competing risks model, separately for females and males. Values colored green show a risk-reduction of more than one third. A value of 1.0 would indicate no effect (baseline of 100%). All significant values greater than 1 are colored red and correspond with increased risk.

Hence, the value 0.528 in the first line `yngkids` of Table 3, column Infection, indicates that the hazard of dying for those with young kids is only 52.8% of the hazard of females without kids. Moreover, all remaining characteristics in the first (Infection) column are also associated with a lowered risk of dying, ranging from 69% to 75%. Thus, the effect of children has roughly twice the magnitude of the other lifestyle variables listed below. All results in the column are statistically significant. The observed beneficial effect of children is highest for cancer (72.5%) but is also strong and significant for all other categories except pulmonary disease.

[8] The ONS-LS data set uses International Classification of Diseases (ICD) codes to categorize the main and, if applicable, contributory reasons of death. These codes come in several revisions of which 8, 9, and 10 are relevant for the census waves we study; for details see World Health Organization (2010). The exact definition of infectious disease we use, for instance, is the following combination of ICD-9 codes (and their earlier and later equivalents): Infectious Diseases 001–139, Chronic Obstructive Pulmonary Disease 490–496, Occupational or Environmental Lung Disease 500–508, Other Diseases of Respiratory System 510–519. The other reasons for death listed in our tables are defined similarly according to the ICD system.

Table 3: Contribution to Cause of Death – Female sample

Variable	Infection	Pulmonary	Cancer	Heart disease	Acc/Hom/Suic	Others
Failed	*2,254*	*1,931*	*8,912*	*6,770*	*783*	*4,904*
`yngkids`	0·528***	0·933	0·275***	0·344***	0·380***	0·735***
	(0·028)	(0·063)	(0·007)	(0·010)	(0·034)	(0·030)
`working_yngkids`	0·746**	0·643***	0·955	0·877	0·789	0·836**
	(0·107)	(0·111)	(0·060)	(0·071)	(0·178)	(0·072)
`married`	0·751***	1·056	1·176***	1·066	0·565***	0·819***
	(0·047)	(0·082)	(0·042)	(0·043)	(0·054)	(0·036)
`highclass_track`	0·689***	0·645***	0·721***	0·588***	0·694***	0·807***
	(0·032)	(0·033)	(0·017)	(0·016)	(0·055)	(0·025)
`house_owner`	0·735***	0·606***	0·726***	0·704***	0·797***	0·911***
	(0·036)	(0·031)	(0·019)	(0·020)	(0·067)	(0·031)

Notes: 8,380 Errors and open cases are not shown. Reported parameters are subhazard ratios; for each column, the heading indicates the main risk, all others are aggregated into a single competing risk. (Robust standard errors are in parentheses.) Significance levels: 10%*, 5%**, 1%***. Green color indicates a reduced hazard of more than one third; significant increased risk is shown red. Source: Own computations based on ONS-LS.

Table 4: Contribution to Cause of Death – Male sample

Variable	Infection	Pulmonary	Cancer	Heart disease	Acc/Hom/Suic	Others
Failed	*2,578*	*2,471*	*9,704*	*13,460*	*1,528*	*4,804*
`yngkids`	0·654***	1·008	0·325***	0·385***	0·444***	0·778***
	(0·034)	(0·060)	(0·008)	(0·001)	(0·028)	(0·031)
`working_yngkids`	0·677**	0·488***	0·796***	0·923	0·909	1·103
	(0·121)	(0·110)	(0·067)	(0·060)	(0·186)	(0·105)
`married`	0·753***	0·860**	1·707***	1·230***	0·437***	0·669***
	(0·043)	(0·054)	(0·060)	(0·035)	(0·027)	(0·028)
`highclass_track`	0·735***	0·650***	0·791***	0·807***	0·776***	0·969
	(0·032)	(0·029)	(0·018)	(0·015)	(0·045)	(0·030)
`house_owner`	0·806***	0·614***	0·652***	0·661***	0·664***	0·827***
	(0·038)	(0·028)	(0·016)	(0·014)	(0·042)	(0·029)

Notes: 10,296 Errors and open cases are not shown. Reported parameters are subhazard ratios; for each column, the heading indicates the main risk, all others are aggregated into a single competing risk. (Robust standard errors are in parentheses.) Significance levels: 10%*, 5%**, 1%***. Green color indicates a reduced hazard of more than one third; significant increased risk is shown red. Source: Own computations based on ONS-LS.

A similarly beneficial effect can be seen in the line `working_yngkids` for those who work with young children. Being `married` is generally beneficial when significant. A surprising result is, however, that females are associated with a roughly 18% increase in death risk if they are married (when compared to non-married females). The status and income variables `highclass_track` and `house_owner` exhibit the expected positive regularities.

Qualitatively similar results are documented in Table 4 for males. Two noteworthy differences are the fact that for Pulmonary disease, being married is associated with a 14% decrease

of death risk compared to unmarried males (the corresponding variable is insignificant for females). Also for Cancer, working with children lowers male death risk by 20% (while females are not distinguished along this dimension).

Figure 1 illustrates the cumulative incidence functions (CIF) built from the competing-risks regression results for the combined samples of males and females. With the exception of Pulmonary disease we see the consistent result that the CIF of those with young children show a lower risk of dying at every age. The relative vertical distance between the lines exhibits a natural correspondence with the parameters estimated with the competing-risks models.

By themselves, the results shown in the Infection columns of Tables 3 & 4 and the corresponding graphs of Figure 1 are compatible with the causal explanation provided by the parental co-immunization hypothesis. The demonstrated association of children with most other causes of death, however, cannot be explained by the hypothesis. This suggests the presence of behavioral effects which we cannot causally disentangle from a co-immunization hypothesis. Hence, we are led to reject the parental co-immunization hypothesis as the sole causal explanation of the beneficial effect of children on death risks.

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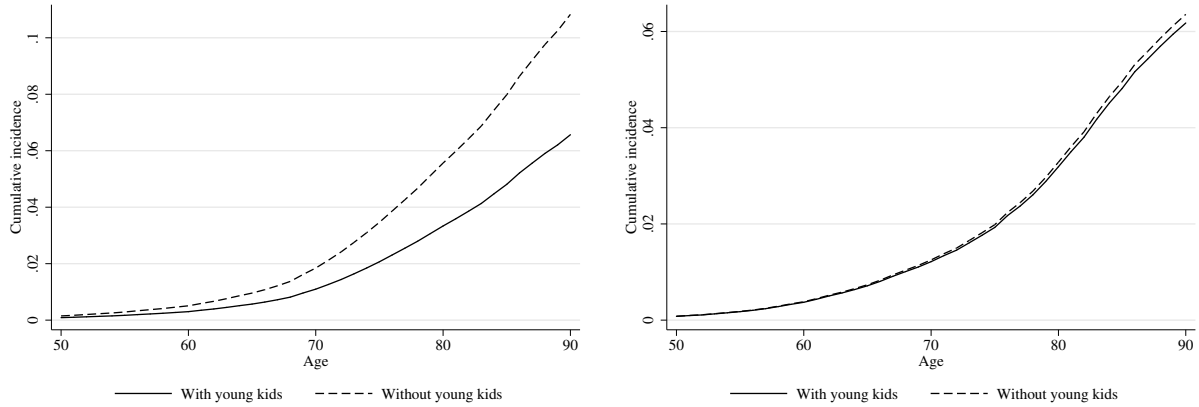
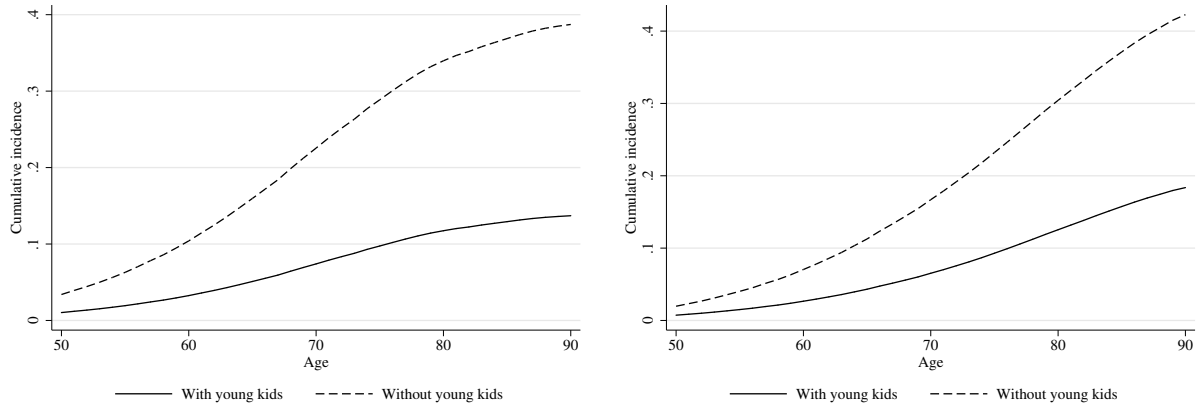
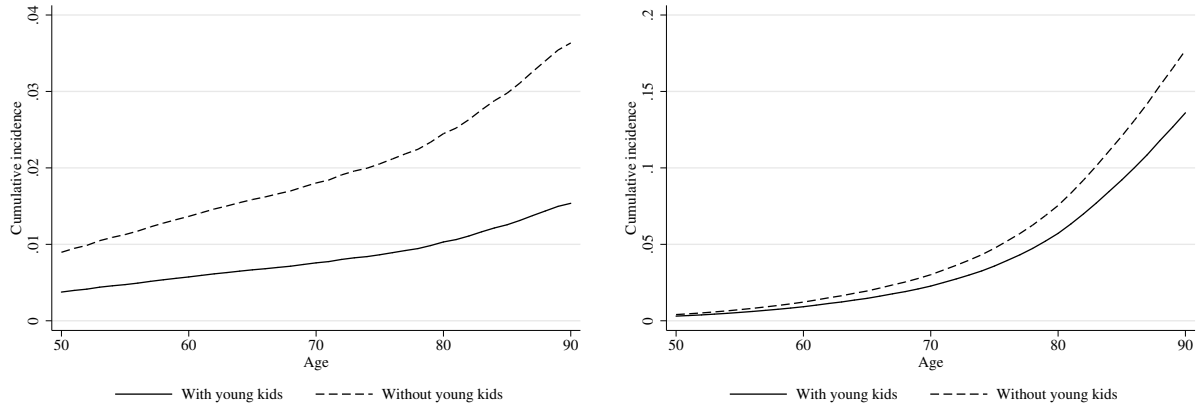
Figure 1: Competing-risks regressions: Cumulative Incidence Functions. Source: Own computations based on ONS-LS.



Left: Infection — Right: Pulmonary disease



Left: Cancer — Right: Heart disease



Left: Acc/Hom/Suic — Right: Other diseases

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