Comparing the observed and unobserved components of the long arm of childhood – Evidence from Finnish register data on midlife mortality from siblings and their parents

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Abstract

In this study, we argue that the 'long arm of childhood' that determines adult mortality should be thought of as comprising both an observed part and its unobserved counterpart, reflecting, on the one hand, the observed socioeconomic position of individuals and their parents, and on the other the unobserved factors shared within a family. Our estimates of the observed and unobserved part of the long arm of childhood are based on family-level variance in a survival analytic regression model, using siblings nested within families as the units of analysis. The study uses a sample of Finnish siblings born between 1936 and 1950 obtained from Finnish census data. Individuals are followed from age 35 up to age 72. To explain familial influence on mortality, we use demographic background factors, the socioeconomic position of the parents, and the individuals' own socioeconomic position at age 35 as predictors of all-cause and cause-specific mortality. The observed part – comprised of demographic and socioeconomic factors, including region, number of siblings, native language, parents' education and occupation, and individuals' income, occupation, tenancy status, and education – makes up between 10 and 25% of the total familial influence on mortality. The larger part of the influence of the family on mortality is not explained by observed individual and parental socioeconomic position or demographic background, and thus remains an unobserved component of the arm of childhood. This highlights the need to investigate the influence of childhood circumstances on adult mortality in a comprehensive framework including demographic, social, behavioral, and genetic information from the family of origin.

Introduction

The influence of the family of origin on adult mortality has been established in many studies (Galobardes et al. 2008; Turrell et al. 2007). The common approach in estimating the social influence of the family is to take observed socioeconomic characteristics like parental education, occupation, or income to predict the child's mortality. Within a life course approach, the effects of childhood on adult health outcomes and mortality are sometimes referred to as the *long arm of childhood* (Hayward and Gorman 2004). Socioeconomic position (SEP) in adulthood is, in this perspective, seen as an important mediator of childhood SEP, as well as an independent predictor of mortality. Research within this tradition shows that people from disadvantaged social backgrounds in childhood have higher mortality and lower life expectancy, and that a considerable proportion of the effects of these early life conditions is mediated by achieved social status (Pakpahan et al. 2017; Palloni 2006; Pudrovska and Anikputa 2014).

Midlife mortality is of relevance when assessing the importance of childhood, because it is the first major period in which many individuals are no longer under the direct influence of their family of origin. Mortality differences in this age range – due to practical constraints defined in this study as deaths occurring between the age 35 and 72 – are of particular interest from a health equity perspective, but also as a focal point for social policy. Further, it has been recognized that midlife is the period in the life course in which health is most stratified by social characteristics (House et al. 1994).

Theoretical models of previous studies have focused on narrow ranges of observable childhood characteristics, putting the spotlight on different features of childhood depending on discipline and research question. However, it is increasingly accepted that health is influenced by complex interactions of individuals' social and biological conditions through the life course (Ben-Shlomo and Kuh 2002; Blane et al. 2013; Galea et al. 2010; Shanahan and Hofer 2005). In focusing only on observed characteristics of the family of origin, previous studies have often deliberately chosen more parsimonious models to reduce analytic complexity. To the best of our knowledge, we present here the first systematic attempt to give an estimate of the total familial influence on midlife mortality, decompose it into unobserved and observed family factors, direct and indirect pathways, and relate it

back to the total childhood influence on adult mortality. We will use Finnish register data and a family approach to give an estimate of how much of the total childhood influence on adult mortality can be explained with a parsimonious set of childhood socioeconomic and demographic variables, as well as how much is mediated through adulthood characteristics in a pathway model.

Observed and unobserved parts of the long arm of childhood

We propose that, within the framework of childhood influences on adult mortality, the total effect of the long arm of childhood (CH_t) should be thought of as comprising two components. The first is the *observed component (*Δ_{obs}*)*, widely investigated in previous studies; it is estimated by the joint influence of *observed measures* for SEP and family characteristics. Its counterpart is the *component of the arm* which is not observed (Δ_{uno}), reflecting the influence of unobserved childhood characteristics. This is a crucial addition, because the influence of childhood and the family of origin can extend far beyond the socioeconomic and demographic factors that are typically observed and used in studies implementing parsimonious models of the long arm of childhood.

We therefore define the total childhood influence as the sum of the observed and unobserved parts:

$$CH_t = \Delta_{\text{obs}} CH_t + \Delta_{uno} CH_t \ (1)$$

$$\Delta_{\text{obs}} + \Delta_{\text{uno}} = 1$$

$$0 \leq \{\Delta_{\text{obs}}; \Delta_{\text{uno}}\} \leq 1$$

To obtain a better understanding of what is observed and unobserved in the study of childhood influences on midlife mortality we draw on two complementary theoretical frameworks. The first divides childhood influences into four different dimensions; in the second, we divide the influence of childhood into direct and indirect effects according to two different life course perspectives. We will apply the division into observed and

unobserved factors to both approaches by employing family-based design. We will see below that it is impossible to obtain a direct estimate of Δ_{obs} and Δ_{uno}. The family approach is therefore necessary in order to enable indirect inferences about the two parts of the long arm of childhood.

The four dimensions of the long arm

First, we adapt the approach of Pescosolido et al. (2008) and divide the influence of the long arm of childhood on health and mortality in factors attributable to fundamental cause theory ($\Delta_{FC_{ch}}$) (Link and Phelan 1995), stress process theory ($\Delta_{ST_{ch}}$) (Pearlin 1989; Szanton et al. 2005), social safety net theory ($\Delta_{SN_{ch}}$), including social support and coping (Pescosolido and Levy 2002; Turner et al. 2014), and genetic influences (Δ_{GE}), as well as the interaction of all four dimensions ($g(FC_{ch}, ST_{ch}, SN_{ch}, GE)$).

Consequently, the total influence of childhood on mortality can be defined as the additive components plus an unknown function of the interactions of the four dimensions:

$$CH_t = \Delta_{FC_{ch}} CH_t + \Delta_{ST_{ch}} CH_t + \Delta_{SN_{ch}} CH_t + \Delta_{GE} CH_t + \Delta_{g(FC_{ch},ST_{ch},SN_{ch},GE)} CH_t \quad (2)$$

$$\Delta_{FC_{ch}} + \Delta_{ST_{ch}} + \Delta_{SN_{ch}} + \Delta_{GE} + \Delta_{f(FC_{ch},ST_{ch},SN_{ch},GE)} = 1$$

$$0 \leq \left\{\Delta_{FC_{ch}}; \Delta_{ST_{ch}}; \Delta_{SN_{ch}}; \Delta_{GE}; \Delta_{f(FC_{ch},ST_{ch},SN_{ch},GE)}\right\} \leq 1$$

While such a framework is necessarily a strong simplification we can link most previous research to one or more of the four dimensions. Parental education and occupation or the financial or material situation of the household are investigated in several studies (Agahi et al. 2014; Case and Paxson 2010; Elo et al. 2014; Hayward and Gorman 2004; Link et al. 2017; Palloni 2006; Turner et al. 2016) and can clearly be thought of as representing factors attributed to fundamental cause theory (FC_{ch}).

Stressors (ST_{ch}) can be such measures as crowded housing (Falkstedt et al. 2011) or family experience of imprisonment or substance abuse (Kelly-Irving et al. 2013), and direct indicators of childhood health or illness that have been used previously (Case and Paxson 2010; Pakpahan et al. 2017; Palloni 2006). In particular, exposure to infectious diseases has been suggested as a specific mechanism of the way in which childhood health affects adulthood health and mortality (Bengtson and Lindström 2000; Bengtsson and Lindström 2003; Dowd et al. 2009). These infections are expected to leave a direct (scarring) effect (Bengtsson and Broström 2009), with a negative impact in particular on cardio-vascular related mortality in later life, although some demographic evidence calls their importance into question (Gagnon and Mazan 2009). Another related pathway through which early life stressors might negatively impact adult mortality is cognitive ability (Kuh et al. 2009). Examples for the safety net (SN_{ch}) include family structure, such as the early death of parents (Campbell and Lee 2009), the influence of peer groups, or the strength of parent-child relationships (Andersson 2016).

The last part of the framework is genetic endowment (GE). Studies that have quantified the degree of heritability of longevity (based on twins studies) come to the conclusion that between 15%-30% of the variation in longevity may be due to genetic heritability, with another 25% the result of environmental factors that are fixed by the age of 30 (Beekman et al. 2013; McGue et al. 1993; Vaupel et al. 1998). The framework further acknowledges that genetic endowment is always interacting with early life social environment ($FC; ST; SN$), from the in-utero stage, but also postnatally. We can thus expect a complex interaction of genes and social environment to determine of longevity. Such gene-environment interactions have been shown in health-related outcomes like smoking (Boardman 2009) , physical activity (Aaltonen et al. 2016) or obesity (Boardman et al. 2014; Bouchard 2008; Qi and Cho 2008). These results speak against interpreting genetic family influences on mortality as a result of the purely mechanistic heredity of genes and in favor of finding possible evidence and explanations for gene-environment interactions and the related processes (Freese 2008; Freese and Shostak 2009). However, as defined above, we can also find environment-environment interactions, for example if the influence of parental SEP is moderated by parent-child relationships (Andersson 2016).

The observed part of the long arm of childhood is therefore the explanatory power of the observed variables (OV) used to represent the four dimensions (D) making up the long arm of childhood (CH_t).

$$\Delta_{obs} = \sum_{\substack{D\in \\ \{FC;ST,SN,GENE\}}} \Delta_{OV_{D_{ch}}} + \Delta_{g(OV_{D_{ch}})} \quad (3)$$

The unobserved part is by definition a residual category and should be seen as a benchmark of the explanatory power that different approaches to the study of childhood circumstances and their relation to adult mortality provide. It is therefore conditional on data, research focus, and the state of research in the field in general.

A life course perspective on the long arm of childhood

The second perspective that is important in the study of the long arm of childhood is the life course approach in the study of health, disease, and mortality. It stresses the concepts of critical period, accumulation, and pathways through the life course (Ben-Shlomo and Kuh 2002). Childhood is a critical period in which influences on the child can have scarring effects, leading to an underdevelopment of organs and the metabolic system which only manifests itself in an increased risk of (for example) cardio-vascular disease in midlife and consequently in a higher risk of premature mortality. The ideas of cumulative (dis)advantage (Dannefer 2003; DiPrete and Eirich 2006) and the pathway model can be seen as an analog to sociological models that link an individual's family of origin to their own socioeconomic status (Blau and Duncan 1967). Both accumulation and the pathway model suggest that early childhood disadvantages are translated into midlife (socioeconomic) disadvantages, and might therefore have an increasing impact on mortality risk throughout the life course. To distinguish between the idea of critical period and the pathway model, we can divide childhood impact on adult mortality into a direct effect (Δ_{direct}), an indirect ($\Delta_{indirect}$) effect, and the effects of possible interactions between childhood and adulthood status ($g(CH, ADULT)$), signifying diverging development trajectories.

$$CH_t = \Delta_{direct} CH_t + \Delta_{indirect} CH_t + \Delta_{g(CH,ADULT)} \ (4)$$

$$\Delta_{\text{direct}} + \Delta_{\text{indirect}} = 1$$

$$0 \leq \{\Delta_{\text{direct}}; \Delta_{\text{indirect}}\} \leq 1$$

Interaction patterns ($g(CH, ADULT)$) have been investigated in previous studies, but have been found to have little or no impact on adult health or mortality compared to the critical period or pathway model (Hayward and Gorman 2004; Kröger et al. 2016; G. Mishra et al. 2009; G. D. Mishra et al. 2013). To reduce the complexity of our study we therefore disregard such a pattern of interaction for the remainder of the study and assume that $\Delta_{g(CH,ADULT)} = 0$. Estimates of the extent to which childhood influences are mediated through adulthood characteristics (Δ_{indirect}) have been conducted in many studies, often taking adulthood SEP, health behavior, or health status as ways in which childhood influences mortality or adult health (Hayward and Gorman 2004; Link et al. 2017; Pakpahan et al. 2017).

A family perspective on the long arm of childhood

In order to get an estimate of the observed and unobserved part of the long arm of childhood, we need to superimpose another approach on the domain-specific approach to childhood influences on adult mortality. In our study, we take a family perspective on the childhood effects, based on the assumption that family and family-related characteristics are the most important compound factor for determining mortality in adulthood. We therefore further define the total childhood influence as the sum of the shared family component (Δ_{fam}) plus the individual influences (Δ_i) that are specific to the individual and not shared within the family.

$$CH_t = \Delta_i CH_t + \Delta_{fam} CH_t \ (5)$$

$$\Delta_{\text{i}} + \Delta_{fam} = 1$$

$$0 \leq \{\Delta_i; \Delta_{fam}\} \leq 1$$

The family part of the childhood influence (Δ_{fam}) will be central to our analyses. It can be divided into an observed and unobserved part in a similar manner as the total childhood influence on mortality.

$$\Delta_{fam} = \Delta_{fam,obs}\Delta_{fam} + \Delta_{fam,uno}\Delta_{fam} \ (6)$$

$$\Delta_{fam,obs} + \Delta_{fam,uno} = 1$$

$$0 \leq \{\Delta_{fam,obs}; \Delta_{fam,uno}\} \leq 1$$

As defined above, the observed part is the explanatory power we get from the observed variables (OV) representing the four domains that make up CH_t. However, as we are calculating the observed part on the *family level,* only differences *between* families are taken into account.

$$\Delta_{fam,obs} = \sum_{\substack{D\in \\ \{FC;ST,SN,GENE\}}} \Delta_{fam,OV_{D_{ch}}} + \Delta_{fam,g(OV_{D_{ch}})}$$

We calculate the direct and indirect (mediated by adulthood characteristics) effect on the family level in a fashion similar to the decomposition into indirect and direct effect on the observed variable ($OV_{D_{ch}}$) level.

$$\Delta_{fam} = \Delta_{fam,direct}\Delta_{fam} + \Delta_{fam,indirect}\Delta_{fam} (7)$$

$$\Delta_{fam,direct} + \Delta_{fam,indirect} = 1$$

$$0 \leq \{\Delta_{fam,direct}; \Delta_{fam,indirect}\} \leq 1$$

Before we turn to the question of how we derive estimates for Δ_{fam} and its components, we will discuss the kind of conclusions we can draw from such estimates on their own, and how this lets us draw conclusions about $\Delta_{obs}, \Delta_{uno}, \Delta_{direct}, \Delta_{indirect}$, the elements that make up the whole long arm of childhood that affects adult mortality (CH_t).

What can we learn from the family part of the long arm of childhood?

One of the key features of the family approach to the long arm of childhood is that it is possible to link insight from Δ_{fam}back to CH_t and its components (which are the original theoretical interest of our study). The kind of conclusions we can draw depend on the assumption we make regarding the relationship between the components of Δ_{fam} and those of Δ_i. As we cannot estimate Δ_i or its components directly, we cannot verify any of the assumptions, but we think it is helpful to divide them into three comprehensive scenarios listed in table 1.

With the strong assumption **1** stating that the proportion that is observed is equal for the individual and family components, our estimate of the family components is a direct estimate of the observed and unobserved components of the total childhood influence. The same argument holds if the direct effect is equal for the family and individual components.

If we assume that the observed component of the family part is *larger* than the observed component of the individual part (assumption **2**), we get an upper bound estimate for the observed effect and a lower bound estimate for the unobserved effect. This means that the observed part of the total influence of the long arm of childhood cannot be larger than the observed family part and will be smaller to a certain degree, meaning that for the total influence the unobserved factors are even more important than for the family level. Assumption **3** posits that the observed component of the family part is *smaller* than the observed component of the individual part. In this case, we get a lower bound estimate for the observed effect and an upper bound estimate for the unobserved effect.

All three assumptions apply equally to the relationship between the direct and indirect part of the long arm of childhood, with indirect being the equivalent to observed and direct being the equivalent to unobserved (see table 1).

In sum, it is the intention of this study to give estimates of Δ_{fam} – and its components, defined in equations (6) and (7) – to assess how much of the long arm of childhood can be observed and how much of the effects of the long arm of childhood are direct effects and thus not mediated by adulthood characteristics.

When we assess what the family part (Δ_{fam}) of the long arm of childhood can teach us, it is also important to note what we are *excluding* with the choice of our focus. We are disregarding all influences from $FC_{ch}, ST_{ch}, SN_{ch}$ and GE on adult mortality that are not shared in the family, but might be different for different members of the family. For example, parental investment of resources from FC_{ch} can vary between children in one family (Becker and Tomes 1976). Illnesses might strike one sibling but not the other, leading to differential stress exposure (ST_{ch}). Family relations might be different, with parents having closer or weaker ties and support (e.g. related to birth order) for particular children (SN_{ch}). Lastly, genetic endowment varies by definition between siblings and between families. Together, these elements constitute important influences on mortality acquired in childhood that are individual (Δ_i) and not family specific, but these effects are disregarded when focusing on the family component.

Causes of death

We will stratify our analyses by groups of causes of deaths. Previous research has established that childhood circumstances are related in different ways to different causes of death (Galobardes et al. 2004). It is therefore of interest to investigate whether this also holds true for the family component of the total influence of childhood. Cardio-vascular disease, and related mortality, has often been argued to build up over the life course starting in childhood with both scarring (critical period) effects (Bengtson and Lindström 2000; Bengtsson and Lindström 2003), but also the accumulation of risk factors (Davey Smith et al. 1997); this is also true for lung cancer, with smoking as a naturally cumulating behavioral risk factor (Lynch et al. 1997). In the age range under investigation (35-72) major groups of causes of deaths in Finland in addition to cardiovascular disease and cancer are to an almost equal proportion the combined deaths related to accidents, violence or alcohol (see table A2 in the appendix). These causes warrant special attention, because their development through the life course and the link to childhood might be more indirect and thus mediated by adulthood social risks and health behaviors.

Data and Methods

We use a 10% sample from the Finnish 1950 census for our analyses. Statistics Finland linked the individuals to the death register between 1970 and 2007 using personal identification codes. Siblings are identified as persons aged 0-14 at the time of the 1950 census (birth cohorts from 1936 to 1950) and having the status of child in the same family. This excludes all siblings living in different households, orphans, and institutionalized children, and treats adopted children as full siblings. This way of identifying siblings is in line with the wider social notion of siblings, meaning being raised by at least one common parent in the same family, instead of a biological definition of siblings (although in the majority of cases these definitions converge).

All surviving individuals are censored at the end of year 2007. As there is no mortality information before 1970, the analyses exclude all deaths in early life (here, before the age of 35) and refer only to survivors past this age. This restriction reduces the age range from which we can draw inference, but avoids the problem of variation in left truncation that can create biased inference of the estimated parameters (Berg and Drepper 2015; Hoffmann 2008). This design also means that our results refer to midlife and early old age mortality (deaths in the age range 35-72). Not only are those who died before 1970 not present in the analysis; individuals who emigrated before this date are also not included. As a result, 15,065 of those individuals included in the 1950 census sample make no contribution to the mortality analysis. This is largely due to extensive emigration to Sweden in the 1960s. Prior studies on the same data set have shown that this leads to a minor overrepresentation of women, those born before 1945, individuals from low SES backgrounds, and mother-only families in the sample (Elo et al. 2014). This bias is so small that it is unlikely to impact on our results. The sample results in 94,042 individuals nested in 32,544 families, resulting in 2,598,805 person-years of analysis time.

We divide mortality into all-cause mortality and mortality due to a) cancers of the lung, larynx, trachea, and bronchus (referred to hereafter, for brevity's sake, as *lung cancer*), b) other forms of cancer, c) cardiovascular diseases, d) alcohol-related deaths, and e) accidents and violence-related deaths. Other groups of mortality do not provide sufficient numbers of

deaths in the data set to analyze them separately. Alcohol-related causes include, among others, alcoholic liver disease, accidental alcohol poisoning, alcoholic diseases of the pancreas, alcoholic cardiomyopathy, alcohol dependence syndrome, and other mental and behavioral disorders resulting from alcohol use. They are important causes of midlife (male) mortality in Finland (Elo et al. 2014; Herttua et al. 2008; Tarkiainen et al. 2016). Accidents and violence include, among other causes, suicides, traffic accidents, poisoning (excluding alcohol poisoning), and homicide. The coding of causes of death in the Finnish death register, especially in broader categories such as these, has been shown to be reliable (Lahti and Penttilä 2001).

We arrange the factors explaining mortality differences between families into three categories. The first category contains demographic factors that have been shown to be associated with mortality in Finland. This category includes the native language (be it Swedish or Finnish), parental age at conception (Gavrilov and Gavrilova 2001; Hubbard et al. 2009; Myrskylä et al. 2014), the number of siblings (Hart and Davey Smith 2003), and region of residence (Blomgren et al. 2004; Saarela and Finnäs 2009).

The second category contains information on parental SEP from the 1950 census and includes the highest educational level attained by both parents (no schooling, primary, or past primary education), as well as the occupational class of the father, categorized according to the Erikson-Goldthorpe-Portocarero scheme (EGP). If paternal information was not available, the occupational status of the mother was used. Further, housing conditions – measured as persons per heated room – are used as an indicator of the socioeconomic resources of the parents.

The third category of variables measures the individuals' own SEP at age 35. We use the highest educational degree of each sibling. The degrees are categorized into "basic" (ISCED 2011 code: 2), "upper secondary (lower track ISCED: 3)", "upper secondary (higher track ISCED: 3-4)", "lowest and lower level tertiary (ISCED: 5-6)", and "highest-degree tertiary (ISCED: 7-8)". Again, occupational status is measured based on occupational coding comparable to the EGP class scheme. The categories used are "employers and self-employed", "upper white-collar workers", "lower white-collar workers", "blue-collar

workers". Tenancy status distinguishes between individuals who are renting and those who own or part-own their home. Personal income before taxes is categorized into deciles for those who earn taxable income, plus a category for those who do not earn taxable income. This represents the relative income position in the year of the census closest to the year when the individual turned 35, and not necessarily the relative position within the sample.

Table A2 in the appendix shows the summary statistics for all variables used in the sample. As our sample excludes all only children, table A4 in the appendix shows the differences between the sample of individuals from families with at least 2 siblings and the thus excluded only children (21,902 individuals). With respect to the relevant characteristics, we can say that the samples are fairly similar. Only children tend to have a higher probability of having Swedish as their native language, and are slightly better educated than those who have siblings.

Statistical Approach

Identifying the familial influence on mortality

As an identification strategy for Δ_{fam} we propose to estimate the variance of the shared frailty parameter based on a multi-level survival model that uses siblings nested within families. This approach of estimating total familial influence is widespread in the study of the transmission of socioeconomic status (Björklund and Jäntti 2012; Duncan et al. 2001; Solon et al. 1991) and has also been used in research on health inequalities (Johnson et al. 2012; Merlo 2011).

In a second step we conduct a step-by-step introduction of sets of observed factors representing childhood and early adulthood conditions. Adding the observed demographic and socioeconomic characteristics of the parents to the basic model, we can show how much of this total familial influence on mortality can be attributed to these observed characteristics (*observed part of the family part long arm of childhood,* $\Delta_{fam,obs}$) and how much of the familial influence is left unexplained (*unobserved component of the family part*

of the long arm, $\Delta_{fam,no}$). The same approach applies to the introduction of the later-life SEP of a family's children into the model, which identifies the direct ($\Delta_{fam,direct}$) and indirect ($\Delta_{fam,indirect}$) family pathways.

Quantifying the familial influence on mortality

We use the median hazard ratio (MHR) to quantify the total familial influence on mortality. The MHR is a relative measure of dissimilarity in mortality risk between families, and is reported in the hazard ratio metric. In the appendix we report similar analyses for two other ways of estimating the total family influence, namely equivalent years of aging and sibling similarity.

The MHR is based on the variance of the shared frailty parameter derived from a multilevel survival model (Θ). The frailty parameter is shared between siblings, making families the higher level (level 2) units. It should be noted that frailty is used here in the statistical sense of survival analysis, which takes variation between different levels into account (Hougaard 1995; Vaupel 1988; Vaupel et al. 1979; Wienke 2010). It is not the measurement of frailty as a clinical indicator for health often used in ageing research (Aalen et al. 2015; Gobbens et al. 2010).

We estimate a parametric survival model with an exponential distribution of the underlying hazard, the explicit introduction of analysis time (t) as a covariate, and a shared frailty parameter for siblings within the same family. Using exponential distribution and analysis time as covariate is equivalent to specifying a Gompertz distribution for the hazard.

$$h_t = \exp\left(a + b * t\right)$$

The advantage of this approach is that it allows us to include both men and women in the model, but still estimate the shape parameter of the Gompertz model separately for men and women, as is appropriate due to the much higher mortality risk of men in midlife.

In the proportional hazards metric, the model is defined as:

$$h_{fs}(t) = Z_f * \exp\left(-\left(a + b_w * t_{fs} + c * male_{fs} + b_m * t_{fs} * male_{fs} + \boldsymbol{X_{ds}\gamma}\right)\right) h_0(t)$$

As the distributional assumption of this model is equivalent to the Gompertz distribution, its use for midlife mortality seems appropriate. See methods appendix for a non-parametric test that supports this assumption.

Our central measure of familial influence on mortality is the median hazard ratio (Merlo et al. 2006). It is a measure of *dissimilarity between* groups. It is the average increase in mortality that would occur if a random individual from a random family were to be put in another higher risk family. The MHR can be estimated based on the variance term on the family level, and we therefore do not need to make each and every comparison, as suggested above (Merlo et al. 2006, p. 294):

$$MHR = \exp\left(\sqrt{2 * \Theta} * 0.6745\right)$$

Our baseline model includes only two variables: the birth cohort and gender of the individuals. After estimating the baseline model, the second model introduces the demographic characteristics of the individuals and their families (demography model). The third model includes parental SEP variables (parental SEP model). The last model includes the individuals' own achieved socioeconomic characteristics at age 35 (own SEP model). This last model provides information on the contribution to the total familial influence resulting not from common parental SEP, but from similarities between siblings in their individual, adult SEP.

We then compare the MHR from the null model to the subsequent three models. Comparing the difference in MHR after introducing demographic and socioeconomic characteristics of the parents gives us an estimate of the observed part of the family part of the long arm of childhood ($\Delta_{fam,obs}$). The change after introducing adulthood characteristics gives an estimate of the indirect effect on the family level ($\Delta_{fam,indirect}$).

All data preparation and all analyses are performed using Stata version 14.1 with the mestreg command and additional user written commands (Jann 2007).

Results

The baseline model contains gender, cohort, a gender-specific shape factor, and a random-intercept term (shared frailty) for each family (group of siblings). Table 2 contains the estimates of individual and family-level characteristics on all-cause mortality. The variance estimate for frailty is 0.36 on the hazard scale, which translates into a median hazard ratio of 1.77. This means that, on average, between a pair of families randomly drawn from the population, the difference in mortality risk is 77% higher in the higher risk family than in the lower risk family.

In the demography model, we add variables for the age of parents at the individual's birth, differences between regions in Finland, the number of siblings in the family, and an indicator for individuals with Swedish as their mother tongue. The only major difference in mortality risk is between children whose mother tongue is Swedish compared to the Finnish speaking majority (HR 0.61). Overall, MHR (1.75) is not influenced notably, meaning that familial influence on mortality risk cannot be traced back to similarity of siblings with regard to language, regional parity, or parental age at birth.

The parental SEP model includes the education and occupation of the parents. A lower parental education level ("less than primary school or no information" compared to "past primary school") is associated with higher mortality (HR 1.16). We can further see that parental occupational status is also associated with midlife mortality. Compared to professionals (higher white collar), the HR for blue-collar and farm workers is 1.16; other differences are smaller and not statistically significant. Our measure of total familial influence, MHR, is minimally reduced to 1.73 after inclusion of parental SEP variables. Substantively, these changes are very small. Taken together, the observed part ($\Delta_{fam,obs}$) is just 5.2% of the total familial influence. We thus conclude that parental SEP has some association with mortality, but does not contribute substantially to the explanation of total familial influence on midlife mortality.

The individual SEP model adds education, income, home ownership, occupational position, and employment status at age 35. All of the dimensions of individuals' SEP exert an

influence on mortality separately. For example, individuals in the lowest income decile have a mortality risk 1.99 times higher than those in the highest decile. Compared to those with higher tertiary education, individuals with only basic or unknown education have a mortality risk which is 1.57 times higher. Individuals who rent have significantly increased mortality risk compared to those who own or part-own a house at the age of 35 (HR 1.31). Lastly, compared to upper white collar workers, blue collar workers' mortality risk is 1.23 times higher.

The socioeconomic stratification variables of individuals at age 35 explain a larger proportion of the total family influence. The median hazard ratio is 1.64. The indirect part ($\Delta_{fam,indirect}$) thus makes up an additional 11.7% of the total familial influence.

For all-cause mortality we can conclude that, first, the average difference in mortality risk between families is almost as large as the strongest differences we find between social groups and, second, only the indirect pathway ($\Delta_{fam,indirect}$) through individuals' own SEP contributes a relevant portion to the explanation of familial influences on all-cause mortality. As we proposed in the theoretical section, the *unobserved arm* is of greater magnitude than the *observed long arm of childhood*.

Cause-specific familial influence

In this section, we examine differences in the magnitude of sibling similarity, and the proportion of similarity explained by the demography, parental, and individual SEP models between causes of death. Table A3 in the appendix lists the relative frequency of causes of death in the sample. Figure 1 shows MHR by cause of death.

The highest MHR is found for alcohol-related deaths (2.49), but the median hazards in CVD (2.37) and accidental and violent (MHR 2.04) deaths are also markedly higher than for all-cause mortality. Lung cancer (MHR 2.19) also shows higher total family influence than all-cause mortality. Other types of cancer show a similar total familial influence to all-cause mortality (MHR1.78).

Similar to the result for all-cause mortality above, parental and individuals' own SEP can only explain a small proportion of the familial influence on mortality. The largest fraction

(21.5%) is explained by the indirect effect ($\Delta_{fam,indirect}$) of individuals' SEP on mortality due to lung cancer, in addition to only 7% of the observed part of total family influence ($\Delta_{fam,obs}$). The cumulative explanatory power ($\Delta_{fam,indirect} + \Delta_{fam,obs}$) for other causes of death lies between 10% (alcohol related) and 15.41% (accidents and violence), which is smaller than the explicable familial influence on all-cause mortality. Despite the fact that we can find clear and strong social gradients in all cause-of-death groups, we can only attribute mortality differences between families to a maximum of one quarter of our measures of social stratification. The analyses show that the differences in the level of familial influence between causes of death are much higher than the share of familial influence that can be explained by SEP (the differences between models within each cause of death), indicating that there is much more variation in the strength of the long arm of childhood across causes of death than there is between the observed and unobserved components of the arm.

We conducted several sensitivity analyses that show that our results are not sensitive to gender (analyses solely of brother-sister sibling pairings, see Fig. A8-A13) or to alternative choices of distributional assumption about the shared frailty parameter (inverse Gaussian, Gamma distribution). The results are reported in Fig. A6 and Fig. A7 in the appendix.

Discussion

We set out to establish whether we can find evidence that the long arm of childhood influences adult mortality in Finland. We have shown that midlife mortality exhibits clear social gradients with respect to achieved income, education, occupation, and measures of wealth at age 35, but to a lesser degree, however, with the socioeconomic characteristics of an individual's parents during childhood. Based on these analyses alone, we would have found little evidence for the long arm of childhood, with it acting mostly indirectly via individuals' own achieved SEP. The exception was Swedish as mother tongue with showed a strong gradient favoring the Swedish minority, which could only partly be explained by adult SEP.

However, we proposed that – in addition to the *observed long arm* of childhood (Δ_{obs}) – there is an *unobserved counterpart* (Δ_{uno}) that is of even greater importance. We find

substantial unobserved familial influences ($\Delta_{\mathrm{fam,uno}}$) in all-cause and cause-specific mortality, measured as median hazard ratio (MHR) and reflecting the family component of the total influence of childhood on adult mortality. On average, the mortality risk more than doubles for CVD, alcohol, and lung cancer-related deaths, and would be about 70% higher for cancers other than lung cancer and all-cause mortality if an individual were to change to a randomly chosen higher risk family. This confirms that there is an unobserved counterpart to the arm of childhood that has substantial stratifying effects on midlife mortality. Further, we show that only about 20% (up to 28% for lung cancer) of the total familial influence on mortality can be explained by the joint effect of observed sociodemographic characteristics of parents ($\Delta_{\mathrm{fam,obs}}$) and the indirect pathway through individuals' adult SEP ($\Delta_{\mathrm{fam,indirect)}}$), confirming our hypothesis that the unobserved counterpart of the long arm of childhood is in fact of greater importance for midlife mortality than the visible arm. We also found that the larger part of the observed arm is the indirect pathway which is mediated by adulthood SEP. It contributes much more to the explanation of the family component of the long arm than observed childhood characteristics. Nevertheless, the remaining direct pathway of family influence ($\Delta_{\mathrm{fam,direct}}$) is more than twice the size of the mediated pathway, indicating potential for unrecognized scarring effects in childhood, making it an even more sensitive or critical period (or as yet unobserved factors of individuals in early adulthood).

The major part of the explained familial influence in all groups of causes is related to the indirect effect through individuals' own SEP, showing strong support for the pathway model. In comparison, observed parental factors are of much smaller importance. We think that several explanations are possible. First, the increase in economic and educational status from the parents' to the children's generation leads to a higher (observable) variation, especially in educational degrees. Due to the reduction in size of the lowest educational categories in the parent's generation, the variance in mortality risk between families can be explained only to a small degree by differences in education. Second, adulthood characteristics might be more important for health behaviors in the context of a rapidly changing economic, social, and technological situation in Finland after the 1950s. Finally, it might be that, especially for premature mortality, current living conditions, including economic and working conditions – but also level of education – have a more direct relation

to mortality, while childhood conditions and their latent effects do not (yet) show their influence in the age group under observation.

We find considerable evidence to suggest that familial influence is strongest in accidents, violence, and alcohol-related deaths (as well as lung cancer), which reflects results from previous studies on childhood influences based on observed characteristics (Galobardes et al. 2004, p. 15). The familial influence on lung cancer should be given special consideration, because the overall familial influence on this cause of death is larger than for all-cause mortality, and the relative importance of the observed part of the arm – that is, the part that can be attributed to the observed sociodemographic characteristics of parents and their children – is considerably larger compared to other groups of cause of death. This indicates that determinants of lung cancer mortality, primarily smoking (Fenelon and Preston 2012), are especially subject to observable social influences, a result that has also been found in other studies (Geyer 2008; Kulik et al. 2013; Mackenbach et al. 2004). Note that while cancer other than lung cancer shows the smallest familial influence, there is still a considerable link between total family circumstances and these forms of cancer, suggesting that previous studies finding no evidence for links with observed SEP variables (Galobardes et al. 2004, 2008) might have taken a too narrow or specific view on childhood influences. In future studies, more in-depth analyses regarding familial influence on more specific groups of causes of death would be interesting, as previous results indicate particular causes like stomach cancer or hemorrhagic stroke which have especially strong links to observed childhood characteristics (Galobardes et al. 2004).

When relating our estimates of $\Delta_{\mathrm{fam,uno}}$ to Δ_{uno} and $\Delta_{\mathrm{fam,indirect}}$ to $\Delta_{\mathrm{indirect}}$ our conclusions depend on which of the three assumptions (1-3) we can defend. If we made the very unrealistic assumption **1** that our observed variables would explain the same amount for the individual part of the long arm of childhood as for the family part, we could generalize our statements to the total family influence on adult mortality. If our observed variables have more explanatory power on the family than the individual level (assumption **2**) then our estimates for the unexplained direct family effects are conservative or lower bound estimates. For the unexplained part of childhood effects, we think this is likely because almost all variables vary only between families, and only to a minor degree within (an exception is e.g. age of parents at birth). It is difficult to form definitive conclusions

regarding the indirect pathway, but we think that the results shown here are strong enough to conclude that, for the total childhood influence, the set of observed variables is only capturing at best half of the long arm of childhood, and probably less. At the very least, we can say that there is a substantial total childhood influence left unexplained in a very parsimonious model, even if we cannot specify the exact proportion (assumption **3**).

Overall, this indicates that calls for more complex models of interaction between social and biological factors in the life course (Galea et al. 2010) are not merely aiming at minor improvements of existing parsimonious models, but could potentially have strong additional predictive power when considering childhood circumstances and adult mortality.

This does not imply that the differences between socioeconomic groups are unimportant. On the contrary – the models showed that there are significant differences between them. Nevertheless, it is clear that other characteristics of the family of origin – ones which we have been unable to observe directly – are extremely powerful in determining midlife mortality. Depending on cause of death, these unobserved factors contribute between three and five times more than observed factors to the differences in midlife mortality between families.

Comparing our study results to previous research shows similarities in the sense that most childhood socioeconomic influences can be explained by the pathway model. In the original study that gave the 'long arm of childhood' research its name, Hayward and Gorman (2004) find that the effects of socioeconomic and demographic variables of the parents are mediated through individuals' own achieved SEP and health behaviors. Also, Case et al. (2010) find that the differences in adult health between different levels of childhood socioeconomic position are completely explained by attained adult social position. Similar results of (almost) complete mediation when investigating adult health instead of adult mortality have been found by several other studies as well (Link et al. 2017; Pakpahan et al. 2017; Turner et al. 2016; Zajacova et al. 2015), although there is also some evidence to suggest that neither the effects of childhood socioeconomic conditions, nor the effects of early life health conditions, on adult health can fully be accounted for by adulthood characteristics (O'Rand and Hamil-Luker 2005). A systematic review of the literature on childhood SEP and its association with adult mortality also corroborates the view that a

large part of the effects of childhood SEP is mediated via adult characteristics (Galobardes et al. 2004, 2008). The key addition to previous studies is that we show – while using a data set that yields similar results on observed socioeconomic variables as previous research – that the total childhood influence may exceed what can typically be observed several times over. We therefore argue that the general direction of our results would also hold in other contexts. There is convincing evidence from many developed countries that adult SEP has strong predictive power for mortality (Elo 2009; Mackenbach et al. 2008). Therefore, our results showing an association with observed parental characteristics should be replicable across countries, time, and cohorts, even if the exact strength of the associations may vary. It is thus reasonable to expect sizable differences in mortality between families (sibling similarity) in other contexts as well.

Our results indicate that the factors that are not observed are important in determining adult mortality. In future studies, their relative contribution to the total childhood influence can be assessed in one of the following ways. Based on our results we know that the combined effects of all factors not measured is about three to four times larger than observed adult SEP variables, which were the strongest predictors of mortality. Therefore, a rough guide for future studies would be to assess whether a new set of explanatory variables is as predictive or more predictive. For example, the estimates in our final model for all-cause mortality, with a family level variance of 0.27, mean that a potential new predictor that is standardized to have a variance of 1 on the family level needs to show a HR of 1.68[1] to completely explain differences between families in mortality hazard. While we should not take these estimates as exact guidelines, as they are conditional on data and modelling differences, they can be seen as an order of magnitude estimate for the future introduction of new variables or for modeling complex interactions and assessing their contribution to the study of childhood influences on adult mortality.

[1] See appendix for the calculation.

Limitations

The advantages of using register data also come with certain disadvantages. When comparing the influence of the observed and unobserved arm of childhood, we run the systematic risk of underestimating the contribution of the visible arm, due to poor measurement of our observed socioeconomic and demographic characteristics. For example, we do not have information on household income when the individuals were young, although our results show that there are substantial differences in mortality risk between income groups in adult age. This might lead to an underestimation of some of the effects of parental SEP, especially because parental education is also only measured in three broad categories. Consequently, measurement error might be a driver of the low estimate of the influence of the visible arm of childhood and parental social characteristics on mortality, a finding that has been observed in previous studies as well (Hayward and Gorman 2004; Kröger, Kroh, Kroll, & Lampert, 2017). However, we can also give a complementary explanation for the relatively minor contribution of parental SEP to the visible arm of childhood. When parental characteristics are compressed into only three educational categories, and there is much less variation in occupational class positions in the parental generation than there is in the children's generation, it is a sign that stratification across these dimensions is much smaller than in the children's generation. This does not mean that there were no educational or occupational inequalities in the pre-war generation in Finland, but that the advantaged groups (well-educated, upper white-collar workers) made up such a small part of the population that these dimensions can make only a modest contribution to the explanation of differences in mortality risk in midlife. In our assessment, it is very likely that both measurement error and lower stratification play some role in the relatively minor contribution of parental characteristics to total familial influence. Another limitation of the measurement of parental characteristics stems from the fact that we only have one point of observation in childhood, at an age that is dictated by the timing of the census and not by theoretical choice. While the educational degree of the parents can be assumed to be quite stable throughout childhood, economic conditions and occupations can change, but we cannot observe these changes or their implications for later life mortality. Note, however, that socioeconomic position in adulthood is arguably well measured from

the register data and still cannot account for the majority of variation in mortality risk between families.

Further, our analyses are limited to midlife and early old age mortality. Early life mortality and old age mortality might show different patterns regarding total familial influence and sibling similarity. It is hard to predict their magnitude relative to midlife mortality. On the one hand, genetic research shows that inheritance of mortality grows with age(Gentilini et al. 2013; Murabito et al. 2012). On the other hand, intra-cohort differentiation during the life course, and individual paths and influences from outside the family, might lead to higher heterogeneity between families and within families at older ages (Dannefer 1997; A. O'Rand and Henretta 1999). It would therefore be an interesting undertaking for future research to compare total familial influence on mortality in different stages of the life course and for different cohorts.

We are also only able to analyze causes of death by very broad groups, due to the limited number of deaths per family per cause. In terms of statistical models, we have to rely on the parametric assumptions of a Gompertz distribution of the hazard and normal distribution of the shared frailty parameter. The former yields a very similar prediction of the hazard as a non-parametric approach, and the latter is insensitive to specifying gamma or inverse Gaussian distributions for the frailty parameter.

A further limitation derives from our inability to determine the exact degree of relatedness of all individuals in the register data. While for each individual in the sibling data we are at least able to identify a common mother or father, it is not always clear whether the individuals share both parents. It is therefore not possible to differentiate clearly between full, half, and step siblings. This misclassification is likely to lead to the underestimation of shared frailty. Additionally, orphans and institutionalized children are not included in the analyses; however, in the cohorts under investigation, they make up only 4.6% of the population. Furthermore, using a sibling approach excludes, by definition, all only children. While we could not find substantive differences in terms of SEP between siblings and only children, the long arm of childhood might manifest itself differently for only children, because of their only child status per se.

Finally, we should remember that the unobserved contribution of shared family effects is conditional on data and research question. We could not cover many other important factors that we included in our theoretical model. Other studies have shown that childhood health status is especially predictive of adult health, even beyond achieved adulthood characteristics (Case and Paxson 2010; Haas 2008; Link et al. 2017; O'Rand and Hamil-Luker 2005; Pakpahan et al. 2017; Zajacova et al. 2015), which makes it an important stressor that could explain the unobserved family component. We also had only very limited information on family structure and relationships inside the family, which might be important for the development of health and mortality risk in later life (Campbell and Lee 2009). Finally, we have no genetic information. If such information cannot be directly collected, one way to indirectly assess genetic endowment reducing the mortality risk could be to calculate family excess longevity (if family members can be linked), which has been shown to explain a substantial part of the correlations in mortality hazards between same-sex siblings (Smith et al. 2009). Finally, we did not consider complex interactions of any of the dimensions that influence mortality.

Conclusion

The midlife mortality hazard of Finnish cohorts born between 1936 and 1950 shows considerable variation between families, which to a significant extent is due to unobserved factors. Thus, to get a more comprehensive picture of the influence of childhood and family circumstances on mortality, the observed part of the long arm of childhood needs to be supplemented with the unobserved counterpart of the same arm. The degree of familial influence varies between causes of death, with alcohol-related causes showing the strongest influence from the family, and all-cause mortality and cancer (except lung cancer) the lowest total familial influence. All types of mortality show strong social gradients, mostly with respect to the individuals' own SEP, but parental social background also plays a stratifying role. In combination with demographic characteristics, these observed social characteristics account for about a fifth of the total variation of all-cause mortality between families, and up to 28% of lung cancer mortality differences between families. Because a

large proportion of the total familial effect is left unexplained, other family-related factors that are shared within families are immensely important in determining the mortality risk in midlife and early old age, highlighting the potential for complex models of social biological interactions in a life course framework.

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Fig. 1 – Differences in median hazard ratio between models and by cause of death

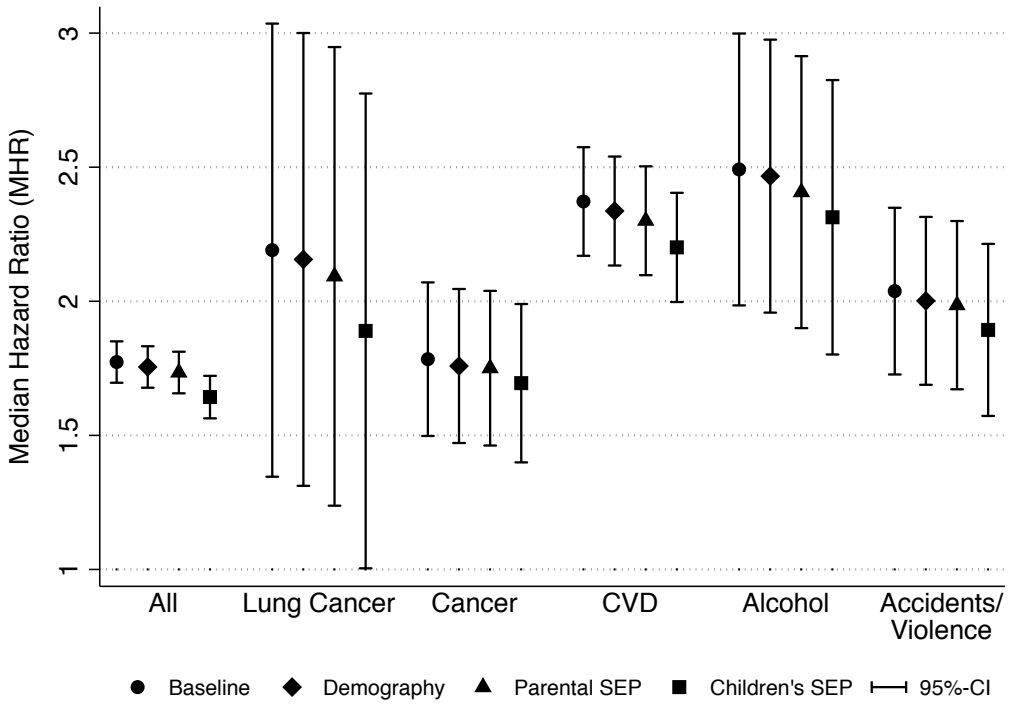

Table 1 – The relation between family component and total childhood influence on adult mortality depending on different assumptions

	Assumptions	Possible conclusions	
1	$\Delta_{fam,obs} = \Delta_{i,obs}$ $\Delta_{fam,indirect} = \Delta_{i,indirect}$	$\Delta_{obs} = \Delta_{fam,obs}$ $\Delta_{uno} = \Delta_{fam,uno}$ $\Delta_{indirect} = \Delta_{fam,indirect}$ $\Delta_{direct} = \Delta_{fam,direct}$	Direct estimates of Δ_{obs}, Δ_{uno} and Δ_{direct} and $\Delta_{indirect}$
2	$\Delta_{fam,obs} > \Delta_{i,obs}$ $\Delta_{fam,indirect} > \Delta_{i,indirect}$	$\Delta_{obs} < \Delta_{fam,obs}$ $\Delta_{uno} > \Delta_{fam,uno}$ $\Delta_{indirect} < \Delta_{fam,indirect}$ $\Delta_{direct} > \Delta_{fam,direct}$	Upper bound estimate for the observed Δ_{obs} and indirect effect $\Delta_{indirect}$ and lower bound estimate for the unobserved and direct effect
3	$\Delta_{fam,obs} < \Delta_{i,obs}$ $\Delta_{fam,indirect} < \Delta_{i,indirect}$	$\Delta_{obs} > \Delta_{fam.obs}$ $\Delta_{uno} < \Delta_{fam,uno}$ $\Delta_{indirect} > \Delta_{fam,indirect}$ $\Delta_{direct} < \Delta_{fam,direct}$	Lower bound estimate for the observed Δ_{obs} and indirect effect Δ_{direct} and upper bound estimate for the unobserved and direct effect

Table 2 – Influences of observed and unobserved family characteristics on all-cause mortality

	Baseline		Demography		Parental SEP		Own SEP	
	HR	SE	HR	SE	HR	SE	HR	SE
Age (Gompertz shape parameter b)	1.07***	(0.00)	1.07***	(0.00)	1.07***	(0.00)	1.07***	(0.00)
Male	2.94***	(0.16)	2.94***	(0.16)	2.94***	(0.16)	3.15***	(0.17)
Male # Age (Gompertz shape parameter b)	0.99**	(0.00)	0.99**	(0.00)	0.99**	(0.00)	1.00	(0.00)
Demography								
Native language (ref. Finnish):								
Swedish			0.61***	(0.03)	0.62***	(0.03)	0.72***	(0.04)
Mother's age at birth (ref. 14-24)								
25-35			1.00	(0.03)	1.00	(0.03)	1.01	(0.03)
35+			1.07	(0.04)	1.06	(0.04)	1.06	(0.04)
no valid info			1.23*	(0.10)	1.19*	(0.10)	1.21*	(0.10)
Father's age at birth (ref. 14-24)								
25-35			0.98	(0.04)	1.00	(0.04)	0.98	(0.04)
35+			0.96	(0.04)	0.98	(0.04)	0.94	(0.04)
no valid info			1.07	(0.06)	1.04	(0.06)	1.00	(0.05)
Region (ref. Western Finland)								
Eastern Finland			1.13***	(0.03)	1.11***	(0.03)	1.10***	(0.03)
Lapland			1.03	(0.05)	0.99	(0.05)	1.06	(0.05)
Uusimaa			1.15***	(0.04)	1.14***	(0.04)	1.14***	(0.04)
Number of siblings (ref.: 2)								
3			1.02	(0.03)	1.02	(0.03)	1.01	(0.03)
4			1.05	(0.03)	1.04	(0.03)	1.01	(0.03)
5+			1.04	(0.03)	0.99	(0.03)	0.97	(0.03)
Parental SEP								
Education (ref. more than primary)								
Did not go to school, unknown					1.16**	(0.06)	1.00	(0.05)
Primary school					1.06	(0.05)	0.97	(0.04)
Occupational status (ref. Professionals)								
Workers & agriculture workers					1.16***	(0.04)	1.04	(0.04)
Farmers					1.01	(0.04)	0.89**	(0.04)
Farmer (10+ ha)					0.89*	(0.05)	0.83***	(0.04)
Employer/self-employed					1.04	(0.05)	0.96	(0.05)
Other, unknown					1.33**	(0.12)	1.14	(0.10)
Persons per heated room (ref. less than 1)								
1-2 persons					1.01	(0.05)	0.95	(0.05)
2-3 persons					1.02	(0.06)	0.93	(0.05)
3 and more persons					1.10	(0.06)	0.95	(0.06)
Unknown					0.99	(0.11)	0.89	(0.10)
Own SEP								
Education (ref: Highest tertiary)								
Basic or unknown							1.57***	(0.09)
Upper secondary (lower track)							1.33***	(0.08)
Upper secondary (higher track)							1.26***	(0.08)
Lower-degree tertiary							1.07	(0.08)

Income (ref. 10th decile)								
1st decile							1.99***	(0.10)
2nd decile							1.72***	(0.09)
3rd decile							1.53***	(0.08)
4th decile							1.43***	(0.08)
5th decile							1.31***	(0.07)
6th decile							1.23***	(0.06)
7th decile							1.07	(0.05)
8th decile							1.01	(0.05)
9th decile							1.00	(0.05)
No income							1.72***	(0.12)
Home ownership (ref: Home owner)								
No owner							1.31***	(0.03)
Unknown							0.10***	(0.01)
Occupational status (ref. Higher white collar)								
Self-employed							0.94	(0.05)
Lower white-collar							1.14**	(0.05)
Blue-collar							1.23***	(0.06)
Other/unknown							1.12	(0.07)
Employment status (ref: Employed)								
Unemployed							1.88***	(0.12)
Homemakers							0.85**	(0.05)
Others/Unknown							1.92***	(0.08)
Birth Cohort (ref: 1936)								
1937	0.98	(0.07)	0.99	(0.05)	0.99	(0.05)	0.98	(0.05)
1938	0.97	(0.07)	1.02	(0.05)	1.02	(0.05)	0.95	(0.05)
1939	0.88	(0.06)	0.95	(0.05)	0.96	(0.05)	0.88**	(0.04)
1940	0.86*	(0.06)	0.92	(0.05)	0.92	(0.05)	0.84***	(0.04)
1941	0.89	(0.06)	0.96	(0.05)	0.96	(0.05)	0.89*	(0.04)
1942	0.86*	(0.07)	0.91	(0.05)	0.92	(0.05)	0.86**	(0.05)
1943	0.84*	(0.06)	0.87**	(0.05)	0.88*	(0.05)	0.80***	(0.04)
1944	0.88	(0.07)	0.89*	(0.05)	0.90*	(0.05)	0.81***	(0.04)
1945	0.94	(0.07)	0.92	(0.05)	0.92	(0.05)	0.83***	(0.04)
1946	0.84*	(0.06)	0.88*	(0.05)	0.89*	(0.05)	0.79***	(0.04)
1947	0.77***	(0.06)	0.90	(0.05)	0.91	(0.05)	0.78***	(0.04)
1948	0.88	(0.07)	0.94	(0.05)	0.94	(0.05)	0.84**	(0.05)
1949	0.90	(0.07)	0.94	(0.05)	0.93	(0.05)	0.81***	(0.05)
1950	0.82*	(0.07)	0.90	(0.06)	0.90	(0.06)	0.77***	(0.05)
Family-level variance ($\bar{\theta}$)	0.36***	(0.03)	0.35***	(0.03)	0.33***	(0.03)	0.27***	(0.03)
MHR	1.77***	(0.04)	1.75***	(0.04)	1.73***	(0.04)	1.64***	(0.04)
Total person years at risk	2598805							
Individuals	94042							
Families	32544							
Deaths	10948							

Note: * p <0.05, ** p <0.01, *** p <0.001