Intergenerational transfers of infant mortality in 19th century northern Sweden

Elisabeth Engberg, Sören Edvinsson, and Göran Broström

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1 Introduction

For more than 25 years it has been observed in numerous studies of historical and contemporary populations that infant deaths seem to be clustered into high-risk families. In a study of infant mortality in two regions in 19th century Sweden, Skellefteå and Sundsvall, Edvinsson et al. (2005) found that fifty per cent of the infant deaths were found in approximately 10 per cent of the families. Despite the fact that infant mortality rates shifted over time, from about 200 per thousand in the beginning of the nineteenth century to approximately 100 per thousand in the 1890s, most families never experienced an infant death. About 67 per cent of all women that gave birth would see their offspring survive their first year of life regardless of how many children they conceived. Various explanations have been proposed to this apparently complex phenomenon, including different aspects of inter-generational transmissions: genetic, cultural, socio-economic and environmental factors or a combination of these. Today there seems to be consensus that the unequal distribution of infant death among families is a complex interplay of different factors, which can be difficult to identify and separate.

Although the clustering of infant mortality appears to be a salient phenomenon throughout history, there are also major regional differences in the strength of the intergenerational transmission of infant mortality. While in certain regions the mortality history of infants is strongly correlated with the survival of infants in the previous generation, in other regions this effect is weak or completely absent (Brändström et al., 2008). Vandezande (2012) suggests that this can be attributed to differences in local culture and family systems, but also proposes the hypothesis that the variations also might be related to strong local variants in gene defects. In practice, regional differences could also be related to the fact that most studies focus on a limited

number of rather small regions, and that different studies thus are hard to compare due to differences in methodology, both in terms of database management and in terms of statistical analysis.

2 Area

The area under study is the Skellefteå region in the province of Västerbotten in the northern part of Sweden, and analyses cover the period 1831–1900. The region was vast, and consisted at the outset of the study of one large rural parish, Skellefteå parish. In 1875, the northern part, Byske was detached into a separate administrative unit, but the population is nevertheless included in the study until 1900. Before 1834 the analyses also include the population in Jörn and Norsjö parishes, which until then were part of Skellefteå parish. The region was large, both in terms of area and of population. With an area of about 1700 square miles, Skellefteå was considerably larger than most rural parishes in Sweden. It was considered a one-day's journey to travel from the northern to the southern border, and a ride from the coast to the more remote and sparsely populated parts of the parish in the west could take even longer, especially in wintertime. The main part of the population was, however, concentrated in the coastal area and in river valleys. In the early 19th century the population was around 6900, and it increased rapidly during the first half of the century. By 1850 it had reached to about 14000 and at the turn of the century it had further doubled. Despite the large increase in population, which was mainly the result of a high natural growth, the population density on the whole remained low (Alm Stenflo, 1994).

Skellefteå was during the studied period a rural area with a mixed economy, based on animal husbandry, forestry and sidelines such as tar and saltpeter production. By the mid-19th century export of tar and lumber became an increasingly important part of the economy. The majority of the farmers in the region were smallholders and there were no large estates. Some small sawmills were established early in the century, but before 1900, industrialization had little impact on the local economy. In 1835, approximately 85 percent of the population made their living from farming. Although the distribution of economic resources was more equal than in several other Swedish regions, the social stratification became more pronounced throughout the 19th century. The increasing proletarianization was mainly a consequence of rapid population growth. The number of farming households remained fairly stable, while the number of landless households increased. The socioeconomic development was also influenced by to two devastating subsistence crises in the region, in the 1830s and in the 1860s (Engberg, 2005).

Mortality was comparatively low. Fertility was high, not only by Swedish standards, but also in an international comparison and there are no indications of family planning. Total fertility fluctuated around five children per woman and, although fertility did decline during the nineteenth century, the actual fertility transition occurred late in the district (Alm Stenflo, 1994). The rate of illegitimacy was low in comparison with many other parts of Northern Sweden, where frequent pre-nuptial conceptions and illegitimate births were common. The illegitimacy rate fluctuated between three and six per cent during the nineteenth century (Alm Stenflo, 1994).

3   Data sources

The analysis is based on information found in the Swedish church book records, that is, birth, death, migration, and catechetical registers. In the catechetical registers the clergy kept a continuous record of all demographic events for all individuals residing in a parish, making it possible to follow in- dividuals over time and to identify their relatives. Furthermore, information on literacy and occupation was recorded, including any changes in status. A selected number of parishes are digitalized by the [Demographic Data Base at Umeå University](http://www.cedar.umu.se/). The entries in the catechetical registers have been linked for each individual making possible longitudinal analyses.

4   Implementation of IDS

A closer look at the data created from `Episodes_file_creator`. It contains one record per birth and is used for the suvival analysis approach, where the influence of grandmother's infant deaths on her grandchild's survival chances is studied.

5   Data

We use the data set that is created from the IDS data base with a standard extraction script (Quaranta, 2016). In the analyses presented here we are using **R**, a free software environment for statistical computing and graphics (R Development Core Team, 2016).

Some variables need to be redefined, centered and categorized, and the data set needs to be restricted in calendar time. The study is limited to mothers born between Sunday, January 1, 1826 and Tuesday, December 31,
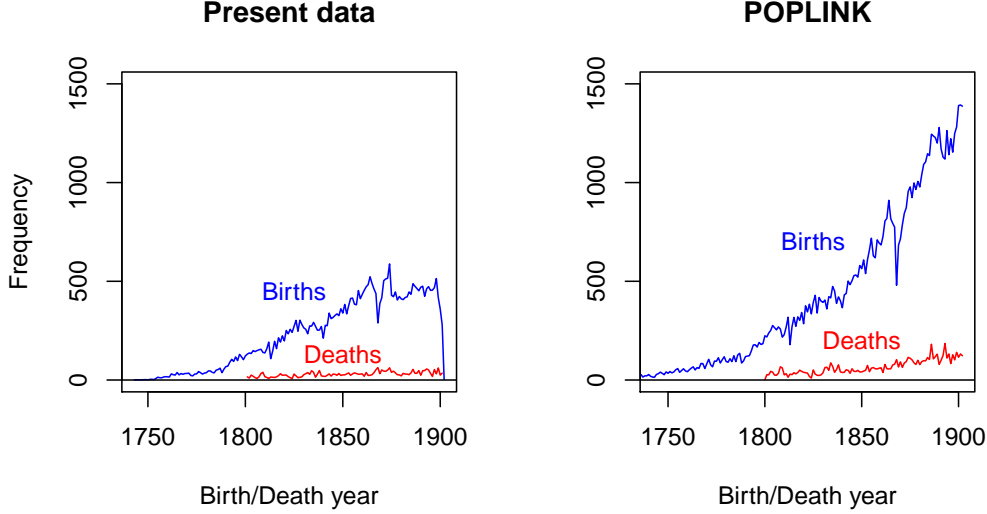
Figure 1: Number of births and infant deaths by year.

1850. *Grandmother's number of births* is categorized into "2", "3", "4-6", "7+" (named *gmBirths*), and *Mother's age at child birth* is centered around 30 (named *mAge*), close to the mean age at delivery. *Birth_order* is categorized into "1", "2", "3", "4-6", "7+" (named *parity*) and *Mother's birthdate* is centered around Wednesday, January 1, 1840 (named(*mBirthdate*). The new variable *gmIMR* is defined as the ratio between grandmother's number of infant deaths and her number of births, or G0_InfD/G0_Births.

6 Results

6.1 Descriptive statistics of the IDS extraction

The yearly numbers of births and deaths for the *mothers in the data set* and, as a comparison, for the data in POPLINK, are shown in Figure 1.

The difference betwen the two data sets is of course explained by the fact that in the present data file there are restrictions on which births to include: Mother and grandmother present, grandmother must have at least two children, etc.
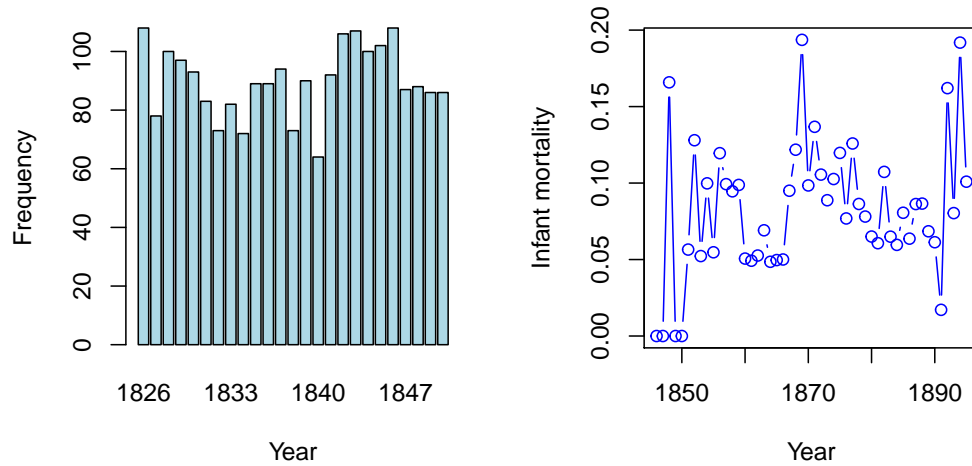
Figure 2: Distribution of mother birth years and mothers' infant mortality rate by infant death year.

The covered time period

Our study sample consists of all mothers born 1826–1950. The distribution of their birth years and their infant mortality (by year of child death) are shown in Figure 2.

Grandmothers, mothers and mother–sisters

There are 2247 mothers and 1384 grandmothers in the data, so obviously there are many sister groups among mothers in the data. This fact induces dependencies in the data set, which may either be a problem (using methods assuming independence), bau it may also be possible to turn this fact into an advantage (using mixed effects models and think of intergenerational transfer as similarity between siblings). In the latter case the explanatory variable *grandmother's IMR* is replaced by clustering on grandmother. This scenario is investigated in the section about *Extended results*.

The distribution of the sizes of sibling groups is shown in Figure 3.

How many grandmothers are also mothers (and vice versa)? The answer is 23, or 1.66 per cent of the grandmothers.
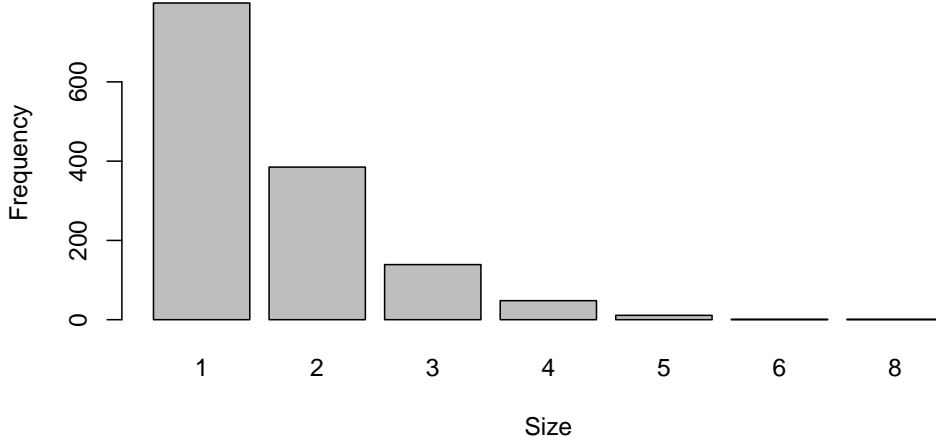
Figure 3: Distribution of sister group sizes.

6.2   Standard results

Poisson regression

The expected value of the number of infant deaths D_i for mother No. i, $i = 1, \ldots, n$, is modeled by a Poisson distribution as

$$E(D_i) = R_i e^{\boldsymbol{\beta} \mathbf{x}_i},$$

where R_i is total risktime for mother No. i, $\mathbf{x}_i$ a vector of her explanatory variables, and $\boldsymbol{\beta}_i$ is the vector of regression coefficients. (For a mother with no infant deaths, the risktime is equal to her number of births.) Formally, R_i is entered into the model as an *offset* after taking logs.

The results are presented in two steps: First, *the statistical significance* is calculated and shown, in Figure 4. The solid horizontal red line at 5% is our (conventionally) chosen nominal limit for statistical significance. The dashed line is the limit that should be respected in honor of the *multiple comparisons* situation (Holm, 1979). The first (leftmost) covariate, *G0InfD_ cat*, is clearly statistically significant, while *G0_Births* is just barely significant. *M_birthdate* is clearly out. Second, the *effect sizes* are graphically evaluated in Figure 5.

So, the likelihood ratio test (LRT) shows that `gmDeaths` and *gmBirths* are highly *statistically* significant in the model.
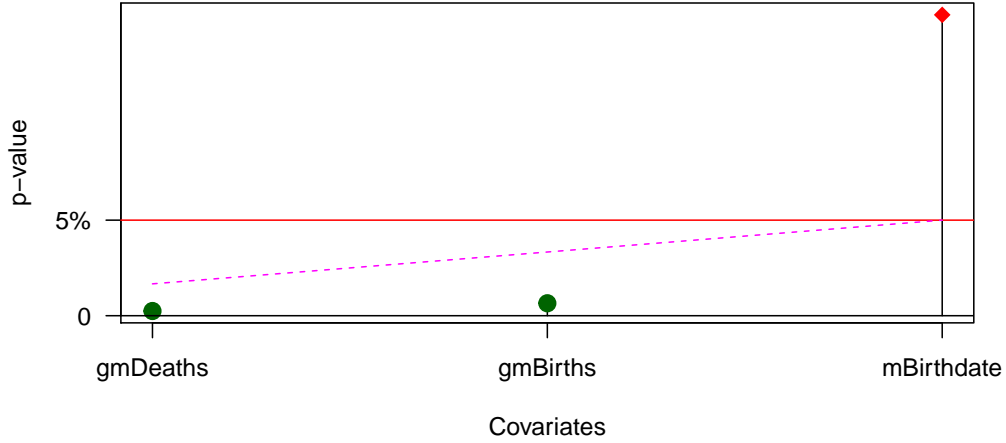
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Figure 4: LRT p-values for the covariates, Poisson regression. Dashed line is significance limit with "multiple comparisons" correction.

Are they also *practically* significant? Figure 5 shows the *expected risks* of infant death by the number of grandmother's deaths and the number of her births.

Survival analysis

The **R** package `eha` (Broström, 2015, 2012) is used, and the explanatory variables are almost the same as in the Poisson regression analysis. The difference is that instead of mother's birthdate, the infant's birthdate is used (no big difference), and a new variable, *mother's age at child birth*, is introduced. It is centered around age 32. Other covariates, *parity*, *infant's birthdate*, etc. are omitted, since they do not contribute much to the model.

[Table 1 about here.]

The standard form of results is shown in Table 1. The statistical significance of involved covariates is found in Figure 6.

The effects are shown in Figure 7

The estimated relative risks are best shown by plotting the *cumulative hazards* for the three groups (Figure 8).

There is an evident deviation from the assumption of *proportional hazards*: It is cases with exactly one grandmother infant death that deviates.

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Figure 5: Infant mortality by grandmother's number of infant deaths (left) and grandmother's number of births(right). Comparisons made at reference value of other covariates.
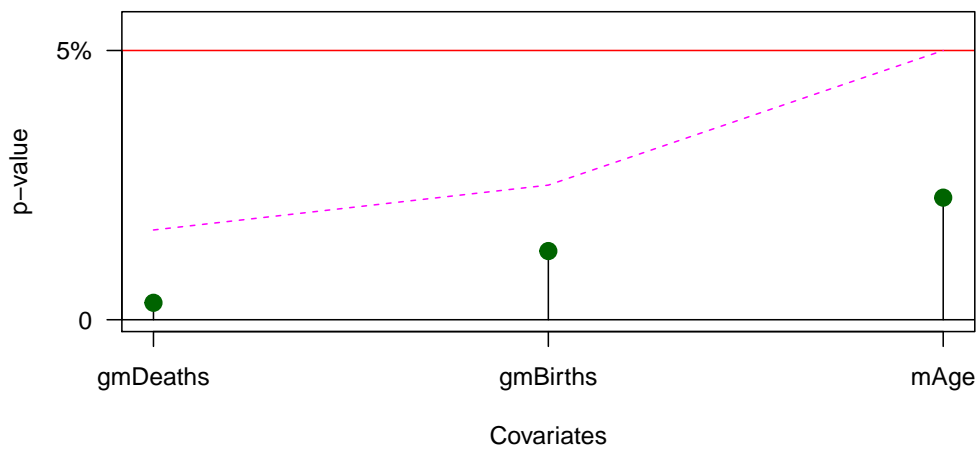

Figure 6: LRT p-values for the covariates, Cox regression. Dashed line is significance limit with "multiple comparisons" correction.
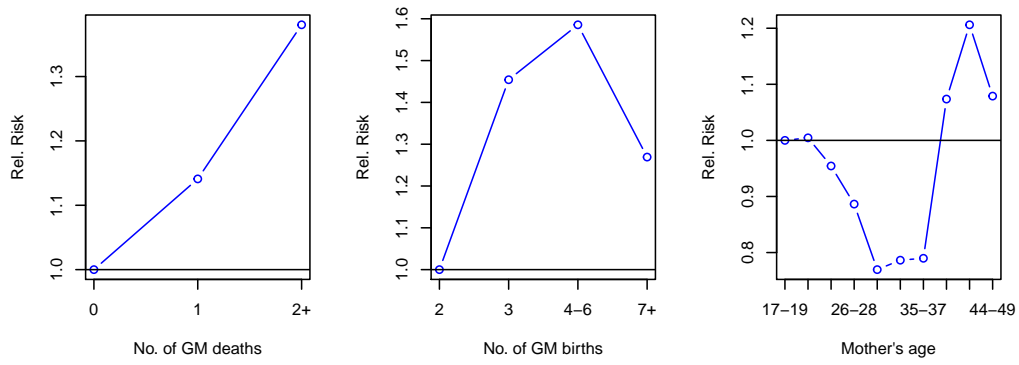
Figure 7: Effects of included covariates, Cox regression. The leftmost value is the reference in all panels.
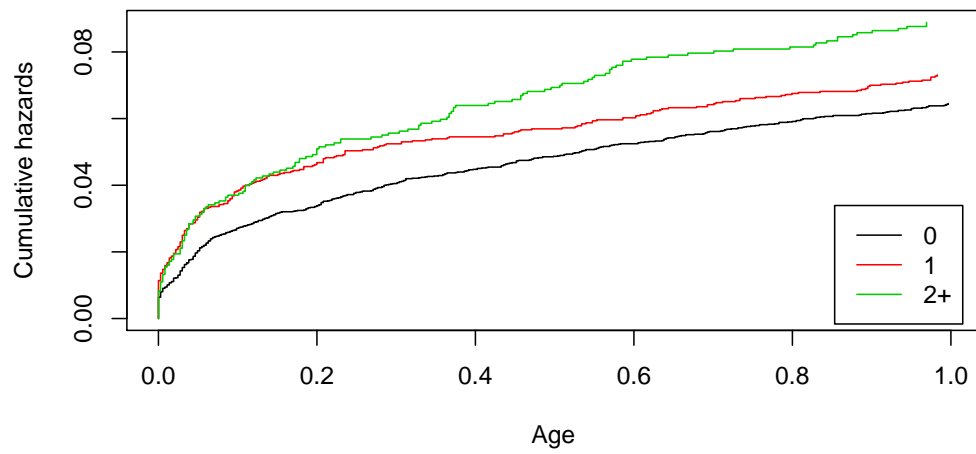

Figure 8: Cumulative hazards by the number of grandmother's infant deaths.
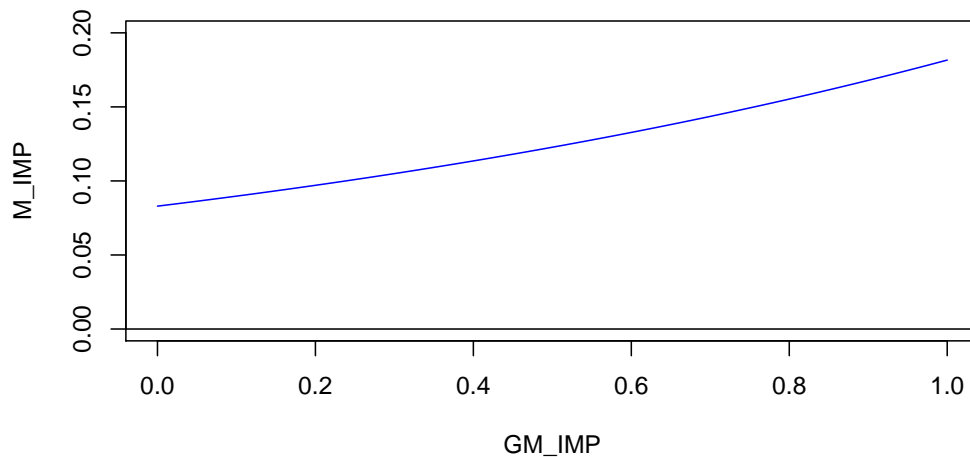
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Figure 9: Probability of infant death for mother (M IMP) by probability of infant death for grandmother (GM IMP). Poisson regression.

However, this does not disturb the main conclusion: Two or more grandmother infant deaths is harmful, quite in line with the results from the Poisson regression.

6.3   Extended models

The Poisson model

It turns out that the model fit to data is somewhat better with *number of births* as the offset (log scale). Also showing a slight improvement is to use *grandmother's IMR* as explanatory variable rather than her *absolute number of deaths*. The IMR is defined as the number of infant deaths divided by the number of births. These changes also implies that the comparison *mother vs. grandmother* happens on a probability scale rather than on the intensity one.

[Table 2 about here.]

The general conclusion is not changed: Grandmother's IMR has a strong influence on her daughter's IMR.

The size of the effect is shown in Figure 9.

Survival analysis

The same modification as in the Poisson case is introduced here.

[Table 3 about here.]

The regression coefficient for `gmIMR` is 0.748, highly statistically significant, as seen in Table 3. The other covariates are of less importance.

Dependency structures There are a couple circumstances that introduce dependence structures in the data: Infants being siblings share genetic and environmental unmeasurable properties, some mothers have sisters that themselves are prresent as mothers, thus sharing grandmother. Possible ways of handling the situation are *shared frailty models* (Aalen et al., 2008) and the implementation of *robust variances* (Therneau and Grambsch, 2000). We have tried both, but neither do change the results in any noticeable way.

A radical way to eliminate the sibling effect among infants is to include the firstborn for each mother. We get the results shown in Table 4.

[Table 4 about here.]

The effect of *grandmother's IMR* is even stronger for firstborn than generally (estimated to 0.897), however, the statistical significance is weak, a logical consequence of the much lower number of infant deaths (compare Tables 3 and 4).

Parametric proportional hazards The Weibull model usually fits infant mortality data well. Let us check this (Table 5).

[Table 5 about here.]

A graphical comparison of the Weibull and nonparametric baseline cumulative hazards (Figure 10) shows an exceptionally good fit.

The advantage of the parametric (Weibull) model vs. the Cox regression model is that we can effortlessly estimate the baseline hazard function (no kernel estimation with ad hoc bandwidth selection). It is further possible to *formally test* the hypothesis of proportional hazards for a categorical covariate through stratification and the LRT test. If proportionality is rejected it is still possible to test for nonproportional effects. (Logically, if proportionality is rejected, then there cannot be equality, but statistical hypothesis testing is not always logical in a common sense. Best to make sure.)
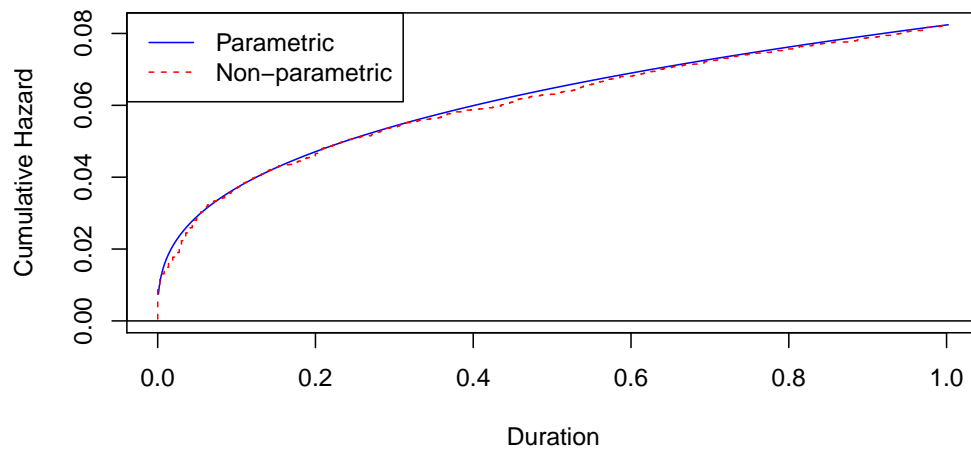
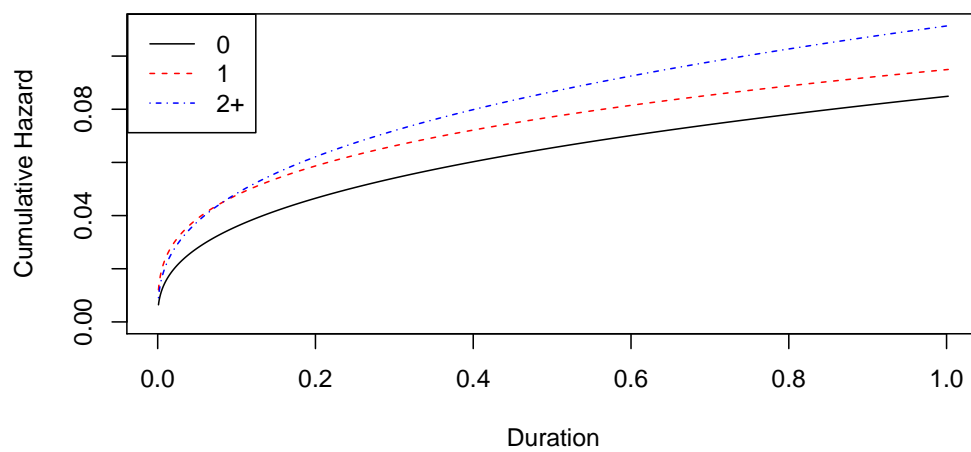Figure 10: Weibull cumulative hazard vs. Nelson-Aalen estimate.

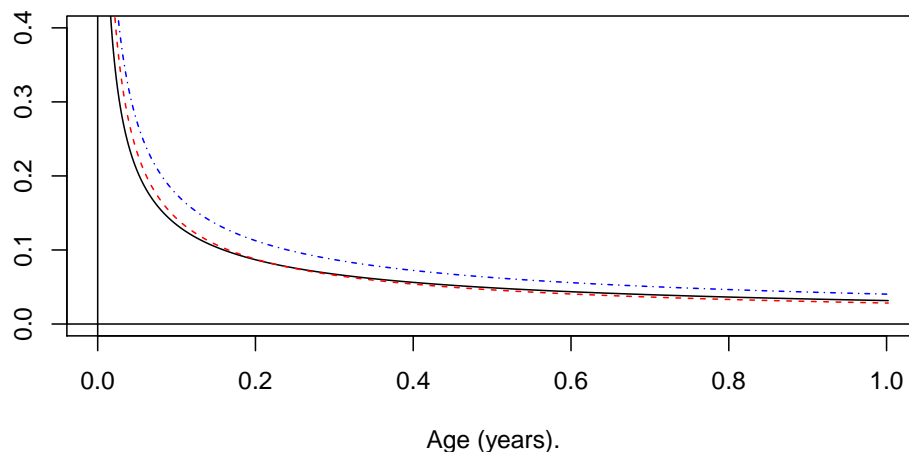Figure 11: Weibull regression, stratified by gmDeaths.

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Figure 12: The Weibull baseline hazard functions.

Test of proportionality   We modify the model by *stratifying on gmDeaths* (with the categories *"0", "1", "2+"*) and plot the result, see Figure 11.

A formal test rejects the hypothesis of proportional hazards (no surprise), and a further test of equality of the curves also rejects (ditto). This is in exact agreement with our conclusion regarding the Cox regression model (Figure 8).

The corresponding Weibull hazard functions are shown in Figure 12. Their values at zero are infinite.

7   Conclusion

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List of Tables

Covariate		Mean	Coef	Rel.Risk	S.E.	L-R p
gmDeaths						0.0031
	0	0.5913	0	1	(reference)	
	1	0.2689	0.1319	1.1410	0.0785	
	2+	0.1398	0.3224	1.3804	0.0951	
gmBirths						0.0128
	2	0.0283	0	1	(reference)	
	3	0.0513	0.3744	1.4542	0.2832	
	4-6	0.2810	0.4610	1.5856	0.2501	
	7+	0.6394	0.2384	1.2692	0.2480	
mAge						0.0227
	17-19	0.0096	0	1	(reference)	
	20-22	0.0545	0.0046	1.0046	0.3439	
	23-25	0.1182	-0.0467	0.9544	0.3303	
	26-28	0.1547	-0.1206	0.8864	0.3278	
	29-31	0.1680	-0.2619	0.7696	0.3286	
	32-34	0.1555	-0.2403	0.7864	0.3293	
	35-37	0.1409	-0.2360	0.7898	0.3305	
	38-40	0.1044	0.0711	1.0737	0.3303	
	41-43	0.0689	0.1874	1.2061	0.3350	
	44-49	0.0253	0.0760	1.0790	0.3706	
Events		895	TTR	10180		
Max. Log Likelihood		-8262				

Table 1: Cox regression, standard model.

	Df	Deviance	AIC	LRT	Pr(>Chi)
<none>		2585.57081	4060.48666		
gmIMR	1.00000	2594.10689	4067.02274	8.53608	0.00348
mBirthdate	1.00000	2586.92086	4059.83672	1.35005	0.24527
parity	3.00000	2588.95130	4057.86716	3.38049	0.33660

Table 2: Poisson regression, grandmother's IMR. Analysis of deviance.

Covariate		Mean	Coef	Rel.Risk	S.E.	L-R p
gmIMR		0.0795	0.7484	2.1136	0.2597	0.0050
childBirthdate		30.5945	0.0054	1.0055	0.0040	0.1718
parity						0.2219
	1	0.2070	0	1	(reference)	
	2	0.1744	-0.0601	0.9416	0.1085	
	3	0.1478	-0.1235	0.8838	0.1162	
	4-6	0.3200	-0.1532	0.8579	0.0993	
	7+	0.1508	0.0593	1.0611	0.1190	
sex						0.0054
	Female	0.4865	0	1	(reference)	
	Male	0.5135	0.1870	1.2057	0.0674	
Events		895	TTR	10180		
Max. Log Likelihood		-8269				

Table 3: Cox regression, extended model.

Covariate		Mean	Coef	Rel.Risk	S.E.	L-R p
gmIMR		0.0774	0.8974	2.4531	0.5683	0.1249
childBirthdate		25.2885	0.0201	1.0203	0.0085	0.0187
sex						0.8090
	Female	0.4906	0	1	(reference)	
	Male	0.5094	-0.0349	0.9657	0.1443	
Events		192	TTR	2107		
Max. Log Likelihood		-1471				

Table 4: Cox regression, extended model with only one birth per mother.

Covariate		Mean	Coef	Risk Ratio	S.E.	L-R p
gmIMR		0.0795	0.7479	2.1125	0.2597	0.0050
childBirthdate		30.5945	0.0055	1.0055	0.0040	0.1708
parity						0.2229
	1	0.2070	0	1	(reference)	
	2	0.1744	-0.0601	0.9417	0.1085	
	3	0.1478	-0.1233	0.8840	0.1162	
	4-6	0.3200	-0.1531	0.8581	0.0993	
	7+	0.1508	0.0592	1.0610	0.1190	
sex						0.0054
	Female	0.4865	0	1	(reference)	
	Male	0.5135	0.1869	1.2055	0.0674	
Baseline parameters						
log(scale)			7.8407	2541.9366	0.4651	0.0000
log(shape)			-1.0574	0.3474	0.0330	0.0000
Events		895	TTR	10180		
Max. Log Likelihood		-2304				

Table 5: Weibull regression, extended model.