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*Research Article*

**A multilevel event history analysis of the  
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(Krummhörn, Ostfriesland, 1720-1874)**

**Jan Beise**

**Eckart Voland**

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## **A multilevel event history analysis of the effects of grandmothers on child mortality in a historical German population (Krummhörn, Ostfriesland, 1720-1874)**

**Jan Beise**<sup>1 2</sup>  
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### **Abstract**

We analyzed data from the historic population of the Krummhörn (Ostfriesland, Germany, 1720-1874) to determine the effects of grandparents in general and grandmothers in particular on child mortality. Multilevel event-history models were used to test how the survival of grandparents in general influenced the survival of the children. Random effects were included in some models in order to take the potentially influential effect of unobserved heterogeneity into account. It could be shown that while maternal grandmothers indeed improved the child's survival, paternal grandmothers worsened it. Both grandfathers had no effect. These findings are not only in accordance with the assumptions of the “grandmother hypothesis” but also may be interpreted as hints for differential grandparental investment strategies.

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## 1. Introduction

Human life span and especially the long post-reproductive life span in women has drawn more and more attention during the last years, both by demographers interested in longevity (Wachter and Finch 1997 and there within Austad) and by evolutionary anthropologists interested in the evolution of human life-history traits (Hawkes et al. 1998; Hill and Kaplan 1999; Kaplan et al. 2000). Demographers are mainly interested in the causes that have lead and are still leading to an extension of the average human life span over the course of the last few hundred years and its consequences for the society both in terms of political and social changes (Oeppen and Vaupel 2002). In contrast, evolutionary anthropologists consider human longevity as a specific life history trait (Stearns 1992) and are interested in its evolutionary history (Hill 1993; Hawkes et al. 1998). Besides the comparatively long life span in general, it is the extraordinary long postreproductive part in women which demands an evolutionary explanation (Kaplan 1997; Hawkes et al. 1998). Recent theoretical development in this field see kin support - especially provided by elder women – as one key factor for the explanation of one or even both of these human life history traits (Hill 1993; Hawkes et al. 1998; O’Connell, Hawkes, and Blurton Jones 1999).

Menopause divides female life span into a fertile and an infertile period. Evolutionary-thinking biologists were initially puzzled about the very existence of female menopause since it occurs relatively early in life and leaves women with decades – not merely years - of postreproductive life span. Even women living in traditional hunter-gatherer conditions have on average a remaining life span of about twenty years after they have experienced menopause (Hill and Hurtado 1996:427). Furthermore, in general, most of those remaining years may be spent in a relatively healthy state. As Hill and Hurtado (1991:Fig. 1) showed, the decline of fertility occurs long before and at a much higher rate than any other physiological trait. Although recent studies show that humans are not the only species that show a substantial remaining life-span after the cessation of reproductive capabilities (Packer, Tatar, and Collins 1998; see also Austad 1997) they are one of the few higher order taxa and they are probably the only primates who show this feature regularly and in “non-provisioned” conditions (Pavelka and Fedigan 1991; Caro et al. 1995; Judge and Carey 2000).

The paradox about human female menopause is that evolutionary theory predicts that there should be no selection for any post-reproductive life-span. The reason is that sterility is – in principle - the selective equivalent of death (Williams 1957). Traits which are expressed only after the end of reproduction are said to be in a “selection shadow”. Post-reproductive life-span may be seen as such a trait in itself since it requires lasting efforts to keep the body functioning in a proper way – efforts which could have been invested instead in reproductive events.

A possible solution to this paradox was first proposed by George Williams in his seminal paper about the evolution of senescence (1957), which was later labeled the “grandmother hypothesis” (by Hill and Hurtado 1991; for recent reviews concerning the grandmother hypothesis see: Hawkes et al. 1998; Peccei 2001a,b; Shanley & Kirkwood 2001). The paradox is solved if one recognizes that being sterile does not necessarily mean being non-reproductive in a broader sense. After menopause, women are definitely sterile but – in a biological fitness sense – they do not have to be post-reproductive. Being reproductive need not only mean giving birth to a child but may also include rearing and supporting them. Thus, sterile women may still achieve fitness benefits by increasing survival and fertility of their offspring. Taken into account the implications of the inclusive fitness concept (Hamilton 1964) the recipients of these benefits may not only include the immediate first generation offspring but also any relatives (though the expected benefits decrease with increasing distance of relatedness).

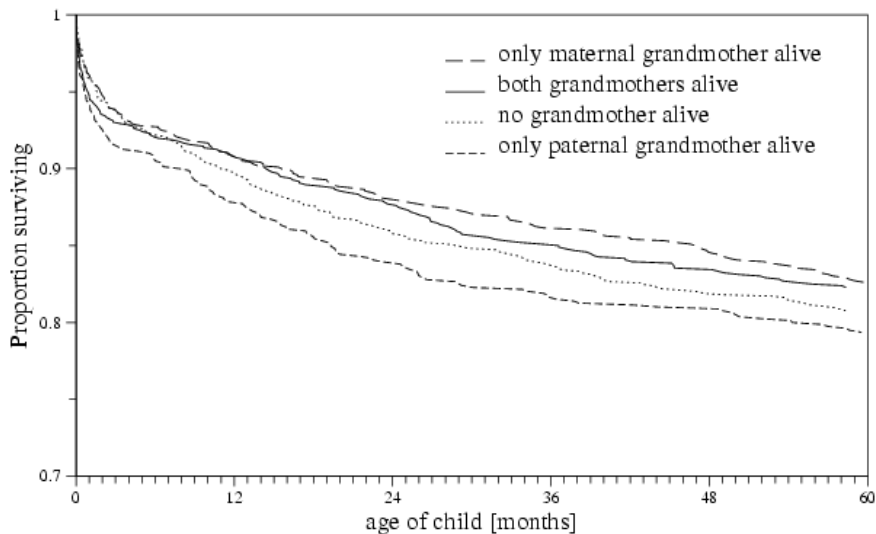
Empirical studies analyzing the helping behavior of grandmothers are not very numerous. Hill and Hurtado (1996: Chapter 13) found a positive though not significant association for the Ache population of Paraguay between the presence of both grandmothers and grandfathers and the survival of their grandchildren. They found no relationship concerning the female fertility rate. Hawkes and colleagues (1997) studied the time allocation and offspring provisioning of the Hadzas in Tanzania and found a positive correlation between grandmaternal foraging time and grandchildren’s weight changes. This last effect was especially strong for the youngest weaned children of nursing mothers.

Just recently, Sear and colleagues studied the effects of kin on child nutritional status (2000) and on mortality (2002) in a rural population in Gambia. They found that from all grandparents, only the maternal grandmother had a positive influence on the child’s situation concerning both nutritional status and survival (there was actually an increased risk of the child dying when the maternal grandmother died). The survival effect was only apparent during the ages of toddlerhood (12-23 months), but not during infancy (0-11 months) and not in later childhood (22-59 months).

This study analyzes the effect of grandmothers on child mortality in the historic population of Krummhörn in northern Germany (1720-1874). In a previous study Voland and Beise (2002) found a slight positive effect on the parity progression probability of woman if both their own mother and their mother-in-law were alive compared to woman for whom these two were dead. Furthermore, using a simple transition rate model, they found significant effects of the grandmothers on the survival of the children. Surprisingly, the effects differed in direction conditional on whether the grandmother was related maternally or paternally: maternal grandmothers increased the survival of the children, paternal grandmothers decreased it. Figure 1 displays Kaplan-Meier survivor functions for children according to the survival status of their grandmothers. Those children of whom only the maternal grandmother was alive (at the

time of their birth) had the highest survival. Children whose grandmothers were both still alive had a slightly lower survival. The children who had only a paternal grandmother had the lowest survival – their survival was even lower than those without a living grandmother.

**Figure 1:** *Kaplan-Meier survival curves classified by the constellation of the grandmother's survival status (at the time of birth of the child), modified from Voland and Beise 2002.*



In the following analysis the pattern of the grandmaternal effect on child mortality will be studied using more appropriate event-history models. First, multi level transition rate models will be applied in order to account for the fact that many mothers have contributed several children to the analysis (which may violate assumptions about the independency of observations which usual regression models assume). Furthermore, data from historical populations are usually quite limited concerning the information that is available. Therefore, it can be assumed that unobserved heterogeneity may play an important role in analysis using those data. In order to have some control for this kind of heterogeneity, random effects will be included in some of the models.

## **2. Data**

The data derive from a family reconstitution study based on church registers, as well as on tax rolls and other records of the Krummhörn region (Ostfriesland [East Frisia], Germany, from the 18<sup>th</sup> and 19<sup>th</sup> centuries). Methods of family reconstitution studies and their usage in historical demography are discussed in Voland (2000). At present, data collection for 19 of the 32 parishes in this region is completed. Although a few parish registers had been kept since the 17<sup>th</sup> century, data could not be considered reliable until the 18<sup>th</sup> century, when partial under-registration (especially of stillbirths and children who died young) could be ruled out. At present the vital and some social data from slightly more than 23,000 families are available. A summary of some of the main results of the Krummhörn study is given in Voland (1990, 1995).

The Krummhörn region is characterized by a very fertile marsh soil. In contrast to the neighboring heathland and moor regions, large and medium-sized businesses dominated the farming economy. A capital and market-oriented agriculture developed and then was replaced by a pure subsistence economy earlier than elsewhere in Germany. Accumulation of returns was possible and indeed led to remarkable wealth concentration in some lineages. Consequently, a “two-class society” developed with big farmers who owned both the land and the capital on the one hand, and a large mass of landless workers and propertyless rural craftsmen on the other. This division of society is also reflected in reproductive strategies. While the relatively prosperous farmers manipulated the fates of their children in accordance with the logic of a resource competition scenario (Voland and Dunbar 1995; Beise 2001), the mass of workers dealt with fertility “opportunistically”, i.e. the sex of the children and the family size already attained played practically no role in their investment decisions at any time. As a result infant mortality did not show any remarkable variance with sex or birth order for the workers in contrast to the farmers. This is why the farmer families were excluded from the sample of this study. Doing this we avoid any possible grandmother effects that could be overshadowed by economically motivated investment decisions.

For further details about the socio-cultural background of the Krummhörn see Engel (1990) and Beise (2001) and the related references therein.

### **Sample preparation and selection**

Historical data sets like the one on which the following analyses are based always suffer from some kind of limitations. Since the information was collected historically for quite different reasons it is necessary to take a great deal of care over data preparation and selection (see also Voland 2000).

The basic sample of this study was restricted to families fulfilling the following criteria:

- The family reproductive history is completely known. That is, included families are those for whom the start of the marriage is exactly known (wedding was recorded) as well as the date of death of the spouse who died first (for this criterion see Voland and Dunbar 1995).
- The family was non-prosperous, i.e. it owned not more than 75 grasen of land (1 gras = approx. 0.37 ha). The relatively prosperous farmer families owning 75 grasen and above were excluded since, as we mentioned above, the farmers were the only socio-economic group which manipulated the fates of their children to a substantial extent. The borderline of 75 grasen is in principle arbitrary but fits well the historic sources concerning the definition of the social and economic upper-class (“Vollbauern”; for references see Beise 2001:53).
- We only include first marriages for both husband and wife.

Furthermore, from these families only those children were selected

- who were born between 1720 and 1870,
- who were born alive,
- whose date of birth is precisely known,
- whose mothers survived the first five years of life of the child (in order to avoid interfering effects by loss of the mother at an early age).

Naturally, this sample does not represent the general population - particularly because one economic segment of the population was excluded (although for good reasons). Besides this, the most prominent bias is that the more mobile segment of the population was excluded, too. Since the precondition of being recorded in the data base is that any specific life-history event had to take place in a geographically restricted area, this means that only children of families could be considered that lived in this area for at least three generations. But since the reduced mobility concerns children with and without living grandmothers, this selection should not bias the analysis.



### 3. Methods

#### 3.1 Event history models

Event history models were used to model the probability of death for the children over time. These models are useful for analyses like this since first they account for the time dependency of a process and second, they can easily cope with censored data. A transition rate model can be represented mathematically by:

$$\ln \mu_i(t) = y(t) + \sum_k \beta_k x_{ik} + \sum_l \lambda_l z_{il}(t) \quad (1)$$

where  $\ln \mu_i(t)$  is the log-hazard of occurrence of the event at time  $t$  for the  $i$ th child,  $y(t)$  captures the baseline hazard,  $x_k$  is the  $k$ th time constant covariate and  $z_l$  is the  $l$ th time varying covariate with  $\beta$  and  $\lambda$  as the respective regression parameters.

#### 3.2 Single level vs. multilevel

In this study multilevel models will be applied (Goldstein 1997). The necessity of using multilevel models is caused by the nested or hierarchical structure of the data. The data are nested because the same family (or the same mother, which in this case is the same because only one marriage per spouse was considered) could contribute several children to the analysis. We assume that children from the same mother or the same family share many traits which have an impact on their survival probability. This can be due to shared genetic effects, social practices, and parental competence in basic childcare abilities (e.g. Das Gupta 1990; Bolstad and Manda 2001). This dependence violates the assumption of independence of observations which is required by traditional (single-level) statistical models. Intra-class correlation can lead to misspecifications, because - for example - standard errors of coefficients will be underestimated (for further details see Kreft and de Leeuw 1998:10 and the literature cited therein). Multilevel models – by using the clustering information – provides correct standard errors, confidence intervals and significance tests.

#### 3.3 Unobserved heterogeneity

A related but still different topic concerns the assumption of additional unobserved heterogeneity in the data set. Family or woman specific characteristics other than those

considered as covariates may have an important influence on the mortality of the children. These characteristics may contribute to individual knowledge or the ability to care for and raise children, to aspects of the household which can result in a favorable or harmful environment (like attitudes toward hygiene, commitment to children, etc.) or even to the genetic make-up of parents and children. These characteristics are unobserved – either because data about them are missing or because they are not measurable in principle. In order to take these unobserved characteristics into account and control for their potential impact on the outcome, a normally distributed random effect on the mother-level (with zero mean and variance of sigma-square) was included in some of the models. In these models the parameter sigma is estimated as a measure for heterogeneity. An estimated standard deviation of sigma, which differs significantly from 0, would indicate that there are unmeasured mother- or marriage-specific characteristics which affect the survival of all children of this family.

The software package aML 1.04 (Lillard and Panis 2000) was used to run all multilevel models, the software package TDA 6.4a (Rohwer and Pötter 1999) was used to run a non-parametric transition rate model.

### **3.4 Variables**

This study focuses on the influence of grandmothers on the well-being or fitness of the grandchildren. Since subtle fitness consequences for the children (physical or psychological health, speed of development, etc.) are difficult or impossible to measure, the survival of the children will serve as ultimate proxy for their fitness.

The probability of death was analyzed for the first 5 years of a child's life. Children surviving the first 60 months got censored at this age. This borderline is arbitrary in principle but was chosen for a number of reasons: First, since the death of a child is the event under observation, there should be enough events in order to get reasonable estimates. Human mortality starts from a relatively high level just around birth (especially in historical populations, in developing countries or non-modern populations) but then drops sharply just afterwards and reaches a minimum during teenage time before it slowly increases again (Hill and Hurtado 1996:Fig. 6.2; Mace 2000:Fig. 1b; Weeks 2002:120). Second, human children are in the first years of life extremely dependent on provisioning by helpers (such as the mother or others; Kaplan 1997). Depending on the specific socio-ecological setting of the population, this dependency will decrease more or less rapidly with increasing maturation. In this time window of high dependency, help by grandmothers would have the greatest effect on the child's well-being. And third, especially in data bases of historical populations, many dates are not exactly known, thus dates of death may actually have the form of a

closed or even open interval based on comparisons with other family-related dates. As a consequence, the longer the observation time, the more cases that have to be excluded.

### **The grandmothers**

The explanatory variables can be distinguished into explanatory variables in a literal sense and those covariates which are included merely as a control function. The main focus in this study is the potential grandmaternal impact.

Since we have no information about the quantity of support an individual grandparent contributes to a child or its mother, the grandparent's survival itself is used instead as a proxy. The logic is that only a living grandparent is able to give support.

Grandmaternal survival entered different models both as a time-constant and as a time-varying covariate. In the time-constant models, the survival status of the grandmother was measured at the time of the birth of the child. In these cases no exact date of death (of the grandmother) was necessary; instead a right censored situation was sufficient. Exact dates of death for the grandmothers are needed when their survival status entered as time-varying covariates. This decreased the sample size for those models slightly.

### **The grandfathers**

In principle, the same conditions apply for the grandfathers as for the grandmothers. Although the role of grandfathers is not explicitly taken into account in most of the theoretical considerations so far (see for example Hawkes et al. 1998, or Gurven and Hill 1997), the grandfathers are included here for at least two reasons: First, as a kind of general control variable in order to make sure that a potential finding of grandmaternal support is not the result of grandpaternal influence. Second, the grandfather variable is included as a kind of control variable in a more specific way: Since children have the same degree of relatedness to their grandfathers as to their grandmothers (for the moment assuming that social relatedness corresponds completely to biological relatedness) any difference in effects between the survival of grandmothers and grandfathers on the effect of survival of their grandchildren should reflect true behavioral causes and not potential genetic ones (ignoring here any potential sex-linked effects in heritability, e.g. Cournil, Legay, and Schächter 2000).

### **Age of mother**

Age of mother is the most important control variable. It is well known that infant mortality is tightly connected to the age of mother (McNamara 1982). When plotted as a graph, infant death rates usually have a J-shape indicating a higher child mortality rate for very young mothers and again a higher rate for advanced maternal ages of approximately 35 and older (see for example Bolstad and Manda 2001:Fig. 10). In our context it is particularly important to control for the age of mothers because there is a direct dependency between our primary covariate (grandmaternal survival) and the age of the mother: Younger mothers tend to have younger grandmothers – or to put it another way: Children with living grandmothers have younger mothers on average. Thus, children with living grandmothers should have on average a higher probability of survival simply because their mothers are younger.

### **Cohort**

This study covers an observation time for birth cohorts of children from 1720 to 1870. In order to control for the uneven distribution of cases throughout this period and for differences in mortality rates, 20 year cohorts are included as covariates. The cohorts enter as dummy variables in order to account for non-linear dependencies.

### **Sex**

The mortality of a child is effected by its sex in two ways. First, infant mortality is in general higher among males than among females for physiological reasons (see references in McNamara 1982). Second, some populations show a differential infant mortality rate according to sex due to socio-cultural reasons. This is also true for the Krummhörn region where the extent of differential mortality depends on membership in a socio-economic class (Volland 1990).

### **Number of living siblings**

Birth order effects on infant mortality are well-known in historical demography (e.g. Wrigley and Schofield 1989; Lynch and Greenhouse 1994). In general, birth order and mortality are positively correlated. The main reason may be a maternal heterogeneity in the ability to keep children alive (e.g. Lynch and Greenhouse 1994). But family

strategic reasons may also play a role here since families may control their investment into a child's well-being according to the number of older siblings (e.g. Volland and Dunbar 1995). The number of living siblings is used here instead of birth order since it can be assumed that the motivation of grandmothers to help is more closely related to the actual family size than to the simple birth order.

### **Place of residence**

The Krummhörn population showed mostly a patrilocal pattern (in relation to the parish of residence) – although this pattern was by far not the only prevailing appearance (Beise 2001). This variable entered as two dummy covariates indicating a shared parish of residence of the children's family with the maternal grandparents and the paternal ones, respectively. A further dummy variable indicated missing information of the residence pattern. Note that the children's family can live in the same parish with one, both or neither member of the grandparental pair. Furthermore, this variable indicates the residence irrespective of the survival status of the grandparents.

Table 1 gives a short numerical overview of the explanatory variables and the outcome variable.

**Table 1:** *Statistical description of the sample and the variables on which the following analyses are based.*

<b>Baseline</b>	<b>Number of cases at risk</b>	<b>Number of events</b>
<b>Total</b>	3550	659
<b>Age intervals</b> <sup>1</sup>		
0-1 month	3530	170
1-6 months	3360	120
6-12 months	3240	69
12-24 months	3171	121
24-36 months	3050	77
36-60 months	2973	77
<b>Covariates</b>	<b>Number of cases</b>	<b>Number of events</b>
<b>maternal grandmother</b> <sup>2</sup>		
alive	1777	313
dead	1753	346
<b>paternal grandmother</b> <sup>2</sup>		
alive	1613	315
dead	1917	344
<b>maternal grandfather</b> <sup>2</sup>		
alive	1440	260
dead	1822	352
missing	268	47
<b>paternal grandfather</b> <sup>2</sup>		
alive	1159	229
dead	2131	385
missing	240	45
<b>sex</b>		
male	1783	339
female	1747	320
<b>age of mother</b>		
15-25 yrs.	504	100
25-35 yrs.	1921	354
35-50 yrs.	992	176
missing	113	29
<b>birth cohort</b>		
1720-1749	102	19
1750-1779	526	128
1780-1809	840	187
1810-1839	1407	242
1840-69	655	83
<b>number of siblings</b>		
0	868	159
1-2	1506	301
3+	1156	199
<b>place of residence</b> <sup>3</sup>		
matrilocal	1609	342
patrilocal	2362	431
missing	161	25

## Notes

<sup>1</sup> Age intervals inclusive the left border and exclusive the right border.<sup>2</sup> Survival status at the time of birth of the child.<sup>3</sup> Note that the residence indication refers to the parish level, i.e. following this definition a family can be at the same time matrilocal and patrilocal (see text).

## 4. Results

In the following models the nested structure of the data will be taken into account. This will be done by applying a multilevel approach to the hazard models. Furthermore, since it can be assumed that unobserved characteristics of mothers or families have an influence on child mortality, unobserved heterogeneity will be added in some of the models. The analyses were carried out using the special purpose software aML (Lillard and Panis 2000). This software captures the baseline hazard  $y(t)$  in Equation (1) as a piecewise-linear spline (or generalized Gompertz or piecewise linear Gompertz). The resulting parameter estimates of the baseline can be understood as “slope parameters”. Knowing the starting point of the function (which in this case is the intercept) and the nodes it is possible to graph the baseline spline using this slope information. Such a graph is in fact the only sensible way of displaying the results for interpretation. In the following section the results will be shown next to the tabled model estimates. The estimates of the categorical covariates will be given as risks relative to the baseline level (i.e. the anti-log of the estimated coefficients).

### 4.1 Grandparents’ survival at time of child’s birth

The first round of multilevel models will treat grandparental death as a set of time constant covariates indicating the survival status at the time of birth of the child. In order to overcome the proportionality assumption of the piecewise linear spline hazard model, an interaction is run between the baseline and the grandmother covariates. The interaction gives the force of mortality the form:

$$\ln \mu_{ij}(t) = z_i y_1(t) + (1 - z_i) y_2(t) + \sum_k \beta_k x_{ijk} \quad (2)$$

where  $z_i$  is a (binary) covariate representing the grandmaternal survival status at the time of the child’s birth ( $z=1$  if dead and  $z=0$  if alive). The remaining terms are the same as in Equation 1. Such a model effectively estimates two different baselines:  $y_1(t)$  captures the baseline hazard for children with grandmothers alive at the time of their birth and  $y_2(t)$  captures the baseline hazard for those whose grandmothers were already dead.

Table 2 summarizes the results of this model. The interaction of the survival status of the maternal and paternal grandmothers with the baseline hazard are estimated in separate models. The respective non interacting grandmother variable enters as a simple time constant variable in the model. For every interaction two models are estimated, namely (i) one stripped down model which beside the interacting grandmother indicator

**Table 2:** Interaction with the baseline of the grandmother's survival status as a time constant variable (in parentheses reference category of covariates; note: gm = grandmother, gf = grandfather).

<b>Maternal grandmother</b>				
<b>Baseline (Age of Child)</b>	<b>Model 1</b>		<b>Model 2</b>	
	<b>b</b>	<b>b</b>	<b>b</b>	<b>b</b>
	<b>alive</b>	<b>dead</b>	<b>alive</b>	<b>dead</b>
Intercept	-2.0510 **	-2.0729 **	-2.2676 **	-2.3538 **
1 month	-2.1797 **	-1.9819 **	-2.4463 **	-1.9642 **
6 months	-0.3502 **	-0.3162 **	-0.3060 **	-0.3304 **
12 months	0.0297	0.0715	0.0116	0.0711
24 months	0.0007	-0.0609 *	0.0058	-0.0643 *
36 months	-0.0557 *	-0.0335	-0.0512 +	-0.0292
60 months	0.0020	-0.0133	-0.0028	-0.0054
<b>Covariates</b>				
	<b>relative risk</b>		<b>relative risk</b>	
<b>maternal gm (alive)</b>				
dead				
<b>paternal gm (alive)</b>				
dead	0.90		0.83 *	
<b>maternal gf (alive)</b>				
dead			1.08	
<b>paternal gf (alive)</b>				
dead			0.97	
<b>sex (male)</b>				
female			0.94	
<b>age of mother (25-35 yrs.)</b>				
15-25 yrs.			1.02	
35-50 yrs.			0.99	
missing information			1.95 **	
<b>birth cohort (1840-69)</b>				
1720-1749			1.44	
1750-1779			1.82 **	
1780-1809			1.75 **	
1810-1839			1.30 +	
<b>number of siblings (0)</b>				
1-2			1.15	
3+			0.92	
<b>place of residence<sup>1</sup></b>				
matrilocal			1.20 *	
patrilocal			0.88	
missing information			0.84	
<b>N</b>	3530		3043	



Table 2: (cont.)

<b>Paternal grandmother</b>				
<b>Baseline (Age of Child)</b>	<b>Model 3</b>		<b>Model 4</b>	
	<b>b</b>	<b>b</b>	<b>b</b>	<b>b</b>
	<b>alive</b>	<b>dead</b>	<b>alive</b>	<b>dead</b>
Intercept	-1.8878 **	-2.5011 **	-2.1599 **	-2.8618 **
1 month	-2.4594 **	-1.7060 **	-2.5258 **	-1.8155 **
6 months	-0.3267 **	-0.3350 **	-0.3137 **	-0.3271 **
12 months	0.0476	0.0589	0.0278	0.0616
24 months	-0.0005	-0.0639 *	0.0052	-0.0696 *
36 months	-0.0733 **	-0.0157	-0.0758 **	-0.0030
60 months	-0.0054	-0.0055	-0.0078	-0.0024
<b>Covariates</b>	<b>relative risk</b>		<b>relative risk</b>	
<b>maternal gm (alive)</b>				
dead	1.14 +		1.18 +	
<b>paternal gm (alive)</b>				
dead				
<b>maternal gf (alive)</b>				
dead			1.08	
<b>paternal gf (alive)</b>				
dead			0.97	
<b>sex (male)</b>				
female			0.94	
<b>age of mother (25-35 yrs.)</b>				
15-25 yrs.			1.02	
35-50 yrs.			0.99	
missing information			1.96 **	
<b>birth cohort (1840-69)</b>				
1720-1749			1.44	
1750-1779			1.81 **	
1780-1809			1.75 **	
1810-1839			1.31 +	
<b>number of siblings (0)</b>				
1-2			1.15	
3+			0.92	
<b>place of residence<sup>1</sup></b>				
matrilocal			1.20 *	
patrilocal			0.88	
missing information			0.84	
<b>N</b>	3530		3043	

Note: + p<0.1; \* p<0.05; \*\* p<0.01

<sup>1</sup> Note that the residence indication refers to the parish level, i.e. following this definition a family can be at the same time matrilocal and patrilocal (see text).

only includes one other covariate, namely the remaining grandmother and (ii) a second – full – model with all the covariates included. The baseline of the models are graphed in Figure 2.

The graphs in Figure 2 show the typical shape of the mortality hazard for children, i.e. a very high mortality right after birth and a very fast decrease followed by a stability at a relatively low level after approximately the age of three. Since the full models hardly differ from the stripped-down models the following description refers to both kinds of models.

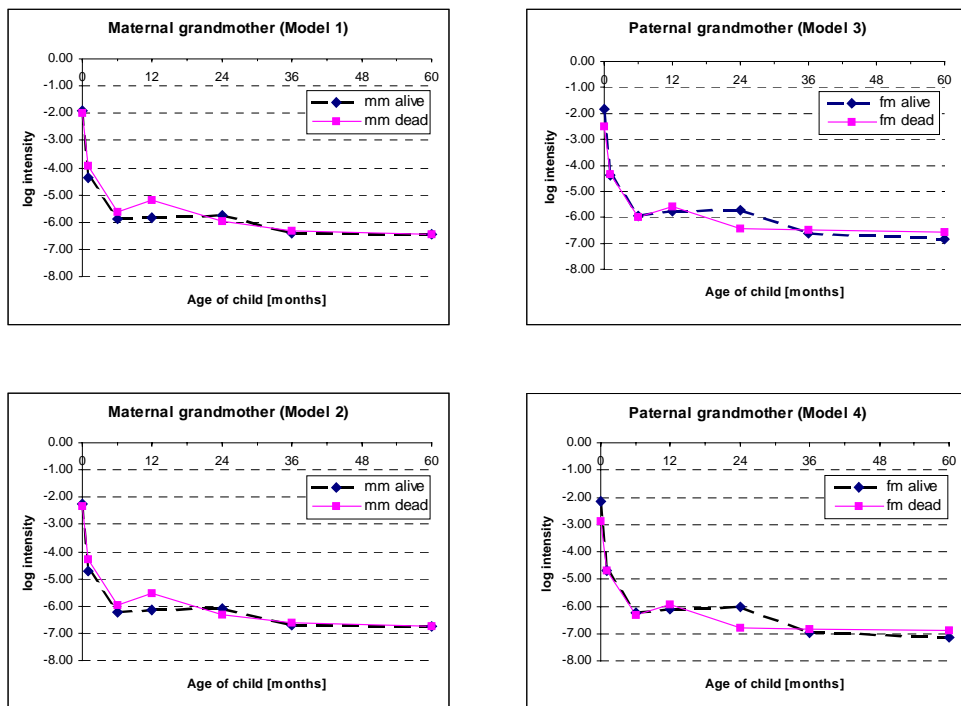
Comparing the baseline hazards for children whose grandmothers were alive at their birth and children whose grandmothers were dead, two distinct differences can be noticed: First, while a dead *maternal* grandmother increased the grandchild's hazard of dying, a dead *paternal* grandmother decreased the hazard. Or to put it in relation to a positive survival status of the grandmother: If the maternal grandmother was alive, a child was less likely to die than if she was dead. The opposite was true for a paternal grandmother: If she was alive the probability of the child dying was higher. Second, the differences in the baseline hazards between living and dead grandmothers became evident at different ages for the two grandmothers. For maternal grandmothers, there was no difference in the hazards right after birth. The hazards start to diverge around one month after birth and are especially pronounced between the ages of 6 months and 2 years. For paternal grandmothers the biggest difference appears right after birth. There is almost no difference in the hazards between the ages of 1 month and 12 months, then there is again a difference around the age of 2 years.

The contrasting effects of the grandmaternal impacts are also obvious from the relative risks in Table 2, although the effects are not always significant. While a dead paternal grandmother decreased the relative risk for her grandchild to die by 10% or 17% (for models 1 and 2, respectively), a dead maternal grandmother increased the relative risk by 14% or 18% (models 3 and 4, respectively). Furthermore, it should be noted that both maternal and paternal grandfathers have virtually no effect on the mortality of their grandchildren.

Also, there is no effect from the number of living siblings, the age of the mother or from the sex of the child (though girls seem to fare somewhat better than boys, as expected). For missing values of the age of the mother (due to missing maternal date of birth), a separate dummy variable was included in the model. The effect of this covariate is highly significant, though it is not clear in which way this selection for higher mortality worked.

The birth cohort also had a significant influence on the estimated mortality hazards. Cohorts born before 1840 had a higher mortality (even increasing with increasing temporal distance) than the subsequent cohort, born between 1840 and 1869.

**Figure 2:** *Baseline hazard for models 1 to 4 (models estimating an interaction with the baseline of grandmaternal survival status as a time constant covariate). The upper panels show the baselines of the stripped-down models while the lower panels show the baselines of the full models (note: mm = maternal grandmother [mother’s mother], fm = paternal grandmother [father’s mother]).*



Only the very first birth cohort showed a mortality opposing this trend. This could be a hint for an under-registration of death in this early period.

And finally, another effect is puzzling: A matrilocal residence pattern increased significantly the children’s mortality while a patrilocal decreased it (though this effect is not significant). This means (controlling for the grandpaternal survival status) that a matrilocal residence was inferior for the survival of the children – especially compared to patrilocal residence.

## 4.2 Grandparents' survival as time varying covariates

In the next models, grandparental deaths enter as time-varying covariates. This takes into account that grandparents may have survived the birth of their grandchild but died sometime later during the observation period of the first five years. This model design is therefore probably the most accurate one, although the sample size decreased slightly compared to the previous models, since now only cases with exact information about the grandparental death could be considered (while in the previous models it was sufficient to know if the grandparent died sometime *before* the date of birth of the child – in historical data sets, this kind of information is not rare). Again the model was specified in a way that the time-varying covariate ‘grandmaternal death’ interacts with the baseline. This was solved by defining in addition to the baseline hazard a second time dependent linear spline term which enters conditionally on the event that the grandmother dies. This term then additively changes the baseline hazard. The mathematical representation can be written as follows:

$$\ln \mu_{ij}(t) = y(t) + z_i c(t - d_i) + \sum_k \beta_k x_{ijk} + \sum_l \lambda_l z_{il}(t) + U_j \quad (3)$$

where  $z_i$  is a similar binary covariate like in equation 2 (indicating if a specific grandparent is dead or not at a specific point in time) and  $c(t-d_i)$  is a time dependent linear spline term which enters the model only if the grandparent is dead. This “correction” term represents the effect of the dead grandparent on the mortality hazard with  $d_i$  indicating the time of death of the grandmother concerned (relative to the age of the child). In addition, this equation includes a random effect term  $U_j$  which captures potential unobserved heterogeneity at the mother level. Again, this model is estimated for every grandmother separately. For each grandmother, two full models are estimated, differing only in the random effect term which is included just once. The results are listed in Table 3 and the estimated baselines are graphed in Figure 3.

Due to the modified specification of the model presented above, the plotting of the baseline (Figure 3) differs from that of the previous models. The basic baseline represents the hazards for children with a living grandmother. The effect of having a dead grandmother enters the model as an extra term at the time point (in the child’s life) when she dies. This means, in order to get a curve for the hazards for children with a dead grandmother it is necessary to add a graph of the additional effect (of a dead grandmother) to the graph for children with living grandmothers. Thus, three curves are depicted in each diagram. The upper row of panels shows the baselines for the models without unobserved heterogeneity, the lower row the baselines for the models including unobserved heterogeneity.

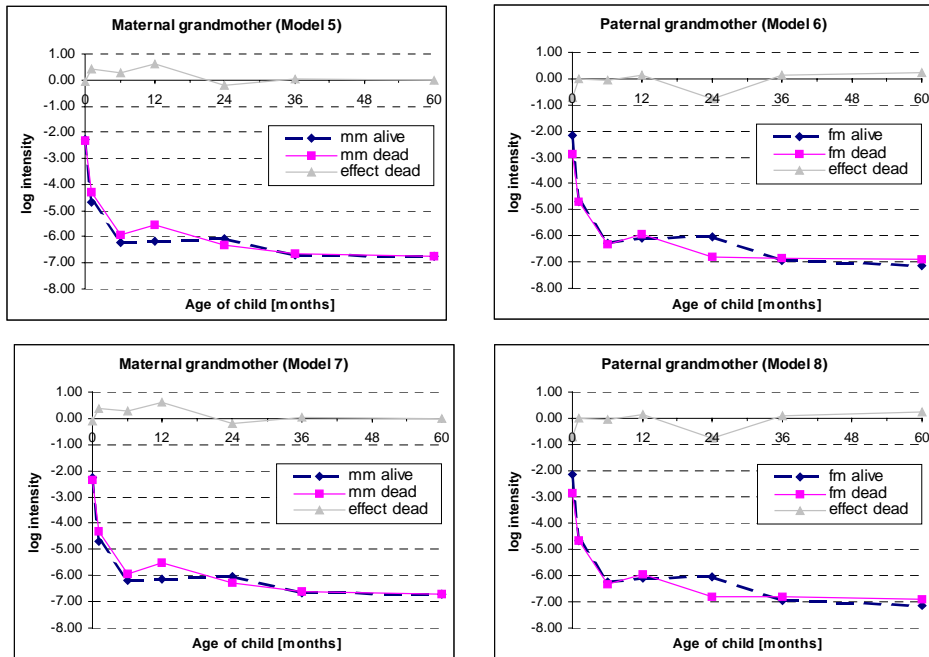
**Table 3:** Interaction with the baseline of grandmother's survival status as a time varying variable (in parentheses reference category of covariates; note: gm = grandmother, gf = grandfather).

Baseline (Age of child)	Maternal grandmother		Paternal grandmother	
	Model 5	Model 6	Model 7	Model 8
<b>Grandmother alive</b>				
Intercept	-2.2577 **	-2.2407 **	-2.1598 **	-2.1520 **
1 month	-2.4423 **	-2.4343 **	-2.5273 **	-2.5151 **
6 months	-0.3073 **	-0.3044 **	-0.3138 **	-0.3124 **
12 months	0.0137	0.0123	0.0276	0.0275
24 months	0.0048	0.0061	0.0046	0.0056
36 months	-0.0501 +	-0.0503 +	-0.0760 **	-0.0763 **
60 months	-0.0021	-0.0018	-0.0082	-0.0076
<b>Difference of dead grandmother</b>				
Intercept	-0.0806	-0.0920	-0.7093 **	-0.7226
1 month	0.4792	0.4755	0.7154 *	0.7042
6 months	-0.0231	-0.0236	-0.0142	-0.0130
12 months	0.0580	0.0602	0.0345	0.0354
24 months	-0.0690	-0.0692	-0.0751 +	-0.0753 +
36 months	0.0215	0.0216	0.0728 +	0.0731 +
60 months	-0.0032	-0.0033	0.0056	0.0052
<b>Covariates</b>	<b>relative risk</b>	<b>relative risk</b>	<b>relative risk</b>	<b>relative risk</b>
<b>maternal gm (alive)</b>				
dead			1.23 *	1.22 *
<b>paternal gm (alive)</b>				
dead	0.83 *	0.81 *		
<b>maternal gf (alive)</b>				
dead	1.00	1.00	1.00	1.00
<b>paternal gf (alive)</b>				
dead	1.01	1.01	1.01	1.01
<b>sex (male)</b>				
female	0.93	0.93	0.94	0.93
<b>age of mother (25-35 yrs.)</b>				
15-25 yrs.	1.02	1.02	1.03	1.02
35-50 yrs.	1.00	0.97	0.99	0.96
missing information	1.94 **	2.00 **	1.94 **	1.99 *
<b>birth cohort (1840-69)</b>				
1720-1749	1.42	1.47	1.43	1.47
1750-1779	1.81 **	1.83 **	1.81 **	1.82 **
1780-1809	1.76 **	1.78 **	1.75 **	1.77 **
1810-1839	1.30 +	1.30 +	1.29 +	1.30 +
<b>number of siblings (0)</b>				
1-2	1.15	1.13	1.15	1.14
3+	0.92	0.84	0.92	0.85
<b>place of residence <sup>1</sup></b>				
matrilocal	1.20 *	1.20 *	1.20 *	1.20 *
patrilocal	0.89	0.89	0.89	0.89
missing information	0.85	0.83	0.85	0.83
<b>Unobserv. Heterogeneity</b>				
Sigma		0.4213 **		0.4165 **
<b>N</b>	3043	3043	3043	3043

Note: + p<0.1; \* p<0.05; \*\* p<0.01

<sup>1</sup> Note that the residence indication refers to the parish level, i.e. following this definition a family can be at the same time matrilocal and patrilocal (see text).

**Figure 3:** Baseline hazards for models 5 to 8 (models with an interaction with the baseline of the grandmaternal survival status as a time varying covariate). The upper panels show the baseline for the full models without heterogeneity, the lower panels the baselines for models including unobserved heterogeneity (Note: mm = maternal grandmother, fm = paternal grandmother).



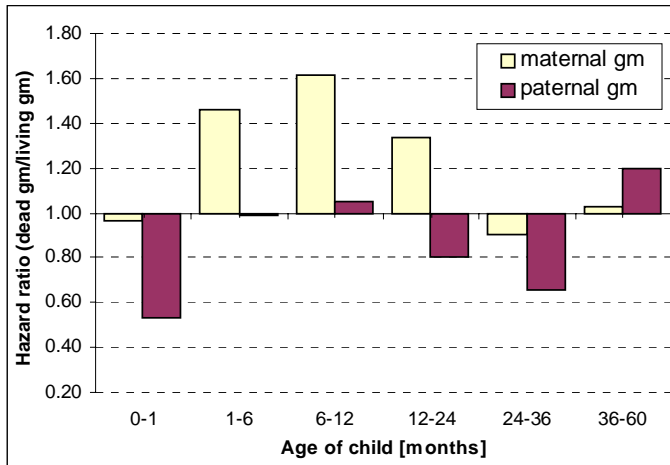
The baseline hazards look almost identical to the ones we found when we treated grandmaternal survival as time constant covariates. Also, the relative risks of the control variables (Table 3) are very similar to those from the previous full model, but especially the effect of the maternal grandmother’s survival sharpened further and is now significant at the  $\alpha=0.05$  level. According to these new models the risk of dying is now over 20% higher for children without a living maternal grandmother than for children with a living grandmother.

But since we are less interested in the shape of the mortality curves itself but in the relative difference in the hazards between the two groups of children, it is actually more useful to display the ratio of the hazards. Figure 4 shows the ratio of the mortality

hazard for children without a living grandmother to the hazard of children with a living grandmother. The ratios are calculated for age intervals by using the anti-log of the log-hazards and after conversion of the linear splines into average hazards for these age intervals. The graph is scaled to a balanced ratio. Every box rising above the baseline (of a ratio of 1) indicates an increased mortality for children without the relevant grandmother; every box below this line indicates a lower mortality. The ratios for maternal and paternal grandmothers are combined into one plot.

Figure 4 shows very clearly that the decrease in the force of mortality of children without a living paternal grandmother (which is the same as an *increase* in the force of mortality in association with the *survival* of this grandmother) is especially strong in the very first month of life. A smaller but concurrent effect can be seen in the second and third year of life. The slight mortality increasing effect of a missing paternal grandmother for the age interval 36 to 60 months could be the result of a selection effect by the increased mortality before this age (which means that the more frail individuals already fell victim to the surplus mortality of the first three years and the survivors are therefore more robust compared to the average child at this age). The mortality increase associated with a dead maternal grandmother is highest in the second half of the child's first year and in the two surrounding age classes.

**Figure 4:** *Mortality hazard of children without a living grandmother relative to those with a living grandmother (age intervals inclusive the left border and exclusive the right border).*



The results in Table 3 show a large standard deviation of the random effect, which is highly significant. This indicates a large amount of unobserved heterogeneity among the mothers of this sample which can be interpreted in a way that mothers varied systematically in their ability to keep their children alive – no matter whether this variability was based on genetic, behavioral, socioeconomic or any other differences. But this heterogeneity did not influence either the effect of the main explanatory variables (the grandmother's survival status) or the effects of the other control variables. None of these variables changed substantially their estimated coefficients when unobserved heterogeneity was taken into account.

## 5. Discussion

Two main results emerge from this study. First, in accordance with the expectations of the “grandmother hypothesis”, grandmothers in the Krummhörn of the 18<sup>th</sup> and 19<sup>th</sup> century may indeed have improved the survival of their grandchildren. Referring to the maternal grandmother, the odds increased by up to 23% over the first 5 years of the child's life (Table 3). Second, this effect was limited to the maternal grandmothers while having a living paternal grandmother was even more harmful for a child than having none: When the paternal grandmother was alive, the odds of surviving *decreased* by up to 19% (Table 3).

But the effects of the two grandmothers differed not only concerning the direction of the impact but also concerning the timing of these effects (Figure 4). Children without a living maternal grandmother had a higher mortality risk especially between the ages of 6 and 12 months (higher by 60% !), a risk which started already after one month of life and was elevated still during the second year. By contrast, the substantial – and opposite – effect of the paternal grandmother was almost exclusively evident in the very first month of life: In this month the average hazard for children with a *dead* paternal grandmother was over 40% lower than the hazard of children with this grandmother alive.

It is important to note this temporal pattern since it gives hints concerning the mechanisms at work. Infant deaths can be separated into two broad categories according to causation. Death may have an exogenous cause, like infectious and parasitic diseases, accidents, or other external causes. Or death may be caused endogenously, as a result of congenital malformations, conditions of prenatal life, or the birth process itself. Exogenous causes predominate all deaths after the first month of life while deaths in the first weeks after birth are mainly the result of endogenous causes (McNamara 1982). Thus, the specific effect of the paternal grandmother just in the very first month could be a hint that her impairing influence was not directed



towards the child itself but instead worked by effecting the living conditions of the wife during pregnancy. In comparison, no effect of the maternal grandmother could be found at this age. Her beneficial effect started only after the first month and was highest during the second half of the first year, at a time when mortality is in general dominated by exogenous causes.

The effect of external help on the survival of the new-born child depends on two aspects which can be framed in the following questions: First, when is support most *needed*? And second, when *can* support effectively influence the children's survival? Support should have the greatest effect when mortality is highest, because then many lives can be saved. Since mortality is highest just after birth, this age should be the most appropriate time to give support. But due to the dominating endogenous causes there is almost no possibility to influence survival beneficially. After the first month of life, the possibilities for grandmothers to contribute to the survival of their grandchildren increase to the same extent as the importance of endogenous factors in infant mortality decreases. But still, as long as the mother is breast-feeding, the possibilities remain limited. A second mortality crisis for children occurs at the time of weaning (McNamara 1982). In the Krummhörn, this took place on average after about 10 months (Kaiser 1998:27). After weaning, mortality declines further and by then the child can be more or less fully independent of the mother *if* it gets substantial support by others.

Interestingly, the time pattern of the maternal grandmother effect reflects this interrelation between the need for and the effectiveness of support quite precisely: There is no effect of the survival of the maternal grandmother in the very first month, there is some effect for the rest of the first half year of life, and there is the strongest effect in the second half of the first year, when the child is very vulnerable due to weaning (and at the same time loses the exclusive dependence on the mother). The peak of this beneficial effect is after weaning, and this is in accordance with the study of Hawkes and colleagues (1997) who found a substantial contribution of grandmothers to the nutrition of their already weaned grandchildren. But still, the noticeable beneficial effect even before this age may point to a grandmaternal contribution to the *nursing mother* which also can be fitness enhancing (Blurton Jones, Hawkes, and O'Connell 1999).

The picture for the paternal grandmother looks very different. The higher mortality in the first month points to the fact that the paternal grandmother's influence – although it appears only after the birth of the child – actually took place during the pregnancy. This finding could reflect what is commonly known as the “evil mother-in-law” and in psychological research as the varying closeness in relationships of family members (Euler, Hoier, and Rohde 2001). It is the relationship between wives and their mothers-in-law which is supposed to be especially tension-loaded – even with potentially long

lasting effects. In a study of a Japanese village, Skinner (1997:77) found that an early death of the mother-in-law increased the wife's longevity. But what is the reason for this special relationship and the differences in investment conditional on whether the grandchild belongs to a son or a daughter?

Ultimately it may be traced back to "paternity uncertainty", a phenomenon which is responsible for a wide range of behavioral traits in humans and animals (Clutton-Brock 1991; Daly, Singh, and Wilson 1993). While women can always be sure about their biological relatedness to their children, men can not. The consequence is that the maternal grandmother is the only one among the grandparents who can be completely sure about the relatedness to her grandchildren. If investments in children are given according to the degree of certainty about relatedness, patrilineal relatives should be less willing to give support than matrilineal relatives (Alexander 1974). This insecurity about the wife's fidelity could give rise to some social conflict between the patrilineal relatives and the mother who married into the family in which the postreproductive mother of the son is especially prone to active participation. Fitting to these considerations is a study by Euler and Weitzel (1996) which found in a psychological analysis of grandparental solicitude an ordered pattern in which the maternal grandmother contributes most care and the paternal grandfather the least care. Still, it is difficult to estimate the significance of this aspect for the Krummhörn population, especially since the cultural background was predominated by a strong Calvinistic belief which made it unlikely that uncertainty about paternity was very high (although precisely this could be the result of a restrictive domestic environment). Thus, this line of argumentation remains very speculative without further knowledge about the socio-cultural setting and the contemporary mentality in the Krummhörn population.

A frequent critique related to the empirical testing of the grandmother hypothesis is that what may be interpreted as a beneficial grandmaternal effect is actually the result of genetic inheritance of something like robustness (or frailty). The idea behind this critique is that healthy and long living grandmothers also have healthy and robust grandchildren. A very similar critique is directed against explanations of a correlation with the sharing of a beneficial family environment. Neither of these critiques seems to apply here: First, beside the positive correlation between the survival of the grandmother and the grandchildren, we also found a negative correlation which contradicts the assumption of the operation of a common background variable. Second, although grandmothers and grandfathers shared almost identical environments for a large part of their life, the effects of the survival status within each grandparental couple differed considerably. A similar argument applies to the genetic influence: On average, grandmothers and grandfathers share with their grandchild to a large extent an identical amount of genetic material (though the share is not *exactly* identical due to insecurity about paternity). If genetic inheritance of robustness would be of substantial importance

in this context, similar beneficial relationships should be expected for all four grandparents. But what is seen here are positive effects, negative effects and no effects at all. It is worth to note, that these missing effects on the side of the grandfathers together with the beneficial effects of the maternal grandmother parallels the finding of Sear and colleagues (2000, 2002) for a current population in rural Gambia.

To sum up, this study – going technically beyond what was done in Voland and Beise (2002) – found a significant beneficial effect of the maternal grandmother. This effect proved to be very stable (considering the control for dependency of events between children of the same mother and the control for unobserved heterogeneity) and is unique among all the grandparents. It is in accordance with the expectations of the grandmother hypothesis and its follow-ups which emphasize the importance especially of the matrilineal kin (Hawkes et al. 1998).

Our findings give support to the idea that women can improve their inclusive fitness substantially even after cessation of their reproductive capabilities (and after their own children grew into adulthood) – and that such a trait can be observed even in a modern, though historic, European population. On the other hand, the opposite, harmful effect of the paternal grandmother – although stable as well – needs some further investigations. This effect could fit into a scenario of differing reproductive strategies according to matrilineal or patrilineal descent, but such an explanation is still very speculative. Further comparative studies on populations of differing family systems and values could offer greater clarification.

## **Changes**

On October 7<sup>th</sup> 2002, per request of the authors, the following change was made:

On page 484, the following quote "The contrasting effects of the grandmaternal impacts are also obvious from the relative risks in Table 1, although the effects are not always significant."

was changed to

"The contrasting effects of the grandmaternal impacts are also obvious from the relative risks in Table 2, although the effects are not always significant."

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