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The Inheritance of Longevity in a Flemish Village (18th–20th Century)

KOEN MATTHIJS[1]*, BART VAN DE PUTTE[1] and ROBERT VLIETINCK[2]
[1]*Katholieke Universiteit Leuven, Department of Sociology, Section of Sociology of the Family, Population and Health Care, Van Evenstraat 2B, 3000 Leuven, Belgium;* [2]*Katholieke Universiteit Leuven, Centre of Human Genetics, Genetic Epidemiology, Kapucijnenvoer 33 blok J, 3000 Leuven, Belgium (*author for correspondence, e-mail: Koen.Matthijs@soc.kuleuven.ac.be)*

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Abstract. The transmission of longevity or post-reproductive survival from parents to offspring is investigated using data on demographic and socio-economic characteristics of the inhabitants of a small Flemish village (Moerzeke) over a period of three hundred years. This research confirms the possible existence of biological mechanisms in transmitting longevity from parent to offspring. However, this finding can only be observed for men and women belonging to specific birth cohorts (mainly those men born between 1821 and 1860 and those women born between 1791 and 1830). Furthermore, the sex-specificity of the transmission, which was present in other studies, is not found in this research. Both factors indicate that the importance of one's biological potential for longevity can only be realized in specific societal conditions.

Key words: environment, historical population database, human longevity, inheritance, longitudinal

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Résumé. À partir de données sur les caractéristiques démographiques et socio-économiques des habitants d'un petit village flamand (Moerzeke) couvrant une période de 300 ans, cet article traite de l'héritabilité de la longévité ou de la survie après la reproduction. L'analyse confirme l'existence possible de mécanismes biologiques dans la transmission de la longévité d'un parent à son enfant. Cependant, ce résultat n'est observé que pour certaines générations particulières (principalement les hommes nés entre 1821 et 1860 et les femmes nées entre 1791 et 1830). De plus, on ne retrouve pas dans cette étude une transmission spécifique selon le sexe, comme cela avait déjà été trouvé dans d'autres travaux. Ces deux éléments montrent bien que le potentiel biologique de longévité, propre à chacun, ne peut se réaliser que dans des environnements sociaux particuliers.

Mots clés: environnement, base de données historiques, longévité, humaine, héritabilité, longitudinal

Introduction

This article deals with the problem of the inheritance of longevity which is analysed in a longitudinal (18th–20th century) and interdisciplinary (biological and sociological) perspective. The study of the population of some Jura villages (18th–20th century) by Cournil and others (Cournil et al., 2000) was the starting point. They concluded that longevity was transmitted from parents to their offspring, especially to daughters. Using the same methodological approach on the population of a village in Flanders (Moerzeke), we come to slightly different conclusions. We find a comparable familial aspect of longevity, which is, however, not sex-specific and which we can observe in a limited period only. Both findings suggest environmental influences on the realization of the biological potential for longevity.

1. Biological Determinants versus Environment: Theoretical Issues

The inheritance of longevity, in the sense of post-reproductive survival, has been analysed for several reasons. The potential bias due to shared environment (Cournil et al., 2000), the influence of contagious diseases, accidents and war, and environmental maternal effects during early childhood (Korpelainen, 1999) is reduced when limiting the study to parents and offspring dying beyond a certain age limit. Furthermore, genetic variability for survival is expected to increase with age following the evolutionary theory of aging and the mutation accumulation hypothesis in particular (Gavrilova et al., 1998).

The assumption of the heredity of post-reproductive survival is based on the fact that some families have biological characteristics that make them (less) susceptible to certain illnesses and diseases and thus to some causes of death. In other words, according to this view differences in age-at-death in a population are (partly) determined by its members' biological characteristics[1] because these give them a relative susceptibility to or resistance against some causes of death. Of course, there are also other determinants of age-at-death, such as wealth, health behaviour (personal and food hygiene), health beliefs and attitudes, living conditions, etc.,[2] which are often called "environmental factors".

Several authors have long suggested that longevity indeed has a genetic component. Recently, Gavrilova et al. (1998) found that life spans of offspring and parents are correlated. Also Korpelainen (1999), Cournil et al. (2000) and Gudmundsson et al. (2000) strongly suggest that there is a genetic component to longevity. There is currently much debate on the biological mechanism which could underlie the inheritance of longevity. There is, for instance, the question whether or not the inheritance of longevity is a linear process. Recently, there are several studies which suggest the non-linearity of the relation between parents' and offspring's longevity (Cournil et al., 2000; Gavrilova et al., 2000). Another topic is the sex-specificity of the inheritance of longevity. Korpelainen (1999) finds for instance that "the maternally inherited genetic component in human life span is

greater than the paternal component". Cournil et al. (2000) found "that the heritable component was substantially larger for daughters compared to sons".

However, there are some important problems in measuring the inheritance of longevity. In an overview of studies on the role of genetics in longevity, Gudmundsson et al. (2000) conclude that "many of these studies are based on selected pedigrees or a relatively small segment of the population which may lead to selection or socio-economic bias". Furthermore, it is difficult to distinguish between the genetic component and other familial components. This is the problem of the shared environment, which can cause a correlation in longevity between parents and offspring. Several authors have pointed out this important problem. For instance Perls and others (Perls et al., 1998) say that certain research "... suggests that genes play an increasingly important role in achieving an age much beyond average life expectancy", however, "... further work is needed to elucidate the contribution of genes to the familial component of extreme longevity". Poulain and colleagues come to the same conclusion (Poulain et al., 1998). For them, there is scientific agreement that longevity has a – rather weak – familial component, but that the problem of distinguishing between genes and shared environment between parents and offspring remains unsolved.

Two studies in particular try to solve these problems, by using population-based data of rather isolated and socially homogenous (in so far as this is possible, of course) populations. Cournil et al. (2000) analysed longevity in a farmers' society in the Jura (18–20th century). Gudmunsson et al. (2000, p. 744) studied longevity in Iceland, using a database reaching "all living Icelanders and the majority of their ancestors". The use of population-based data decreases sampling bias, which has often been the case as many studies used the records of selected families of the aristocracy (Gudmundsson et al., 2000). The isolation and the homogeneity of the population must help to avoid the problem of the shared environment of parents and offspring. The crucial element in for instance the study by Cournil et al. was the control of environmental factors by selecting a homogeneous population with regard to lifestyle and socio-economic status (90% of the inhabitants were farmers). This design more or less guarantees the control of the contribution of the shared environment of parents and offspring in measuring the correlation of their age-at-death. Gudmundsson et al. use a similar argumentation (2000, pp. 744–748).

However, also this strategy is not sufficient to deal with the problem of the influence of the environmental conditions on the measurement of the inheritance of longevity. Firstly, the measurement of the influence of environmental conditions can be extended. Section 2.1 below deals with this methodological issue. Secondly, limiting the study to a specific population means that the results are only valid for that population. The findings of Cournil et al. are for instance restricted to a specific macro-environment, namely a farmers' society in the French Jura region. By macro-environment we mean all economic, cultural, social, political and epidemiological forces which create the context in which biological and micro-environmental characteristics (personal characteristics such as wealth,

health practices, etc.) operate. This specific macro-environment may (for whatever reason) obscure some correlations and increase others. An example may clarify this point. It is possible that in a specific macro-environment men work harder than women and that this hard work makes them more susceptible to some diseases. For this reason the importance of some causes of death may be sex-specific, which makes it likely that the importance of biological characteristics is also sex-specific.[3] In this example it is the difference in working and living conditions and not the difference in biological characteristics which creates the difference between men and women.

In other words, environmental factors cannot be reduced to factors on the micro-level. It is our view that environmental factors on the macro-level should also be included in the debate on the heredity of longevity. Which determinants of mortality (biological characteristics versus micro-environmental factors) are the most important depends on these broader environmental conditions. Thus, whether someone "gets" a specific disease or not, whether someone dies from a disease or not, is to some extent also determined by factors such as the epidemiological environment, the health-care system, general socio-economic circumstances (for instance the increase in wealth in the 20th century) and cultural changes (which may for instance result in changing health practices, such as smoking).

It is obvious that these macro-environmental conditions have not remained stable over recent centuries and that societies differ in the prevalence of these factors. First of all, the epidemiological environment changed dramatically in the period observed. Crisis mortality started to decline in the course of the 18th century (plague, yellow fever, malaria, typhus and smallpox declined or disappeared before the middle of the 19th century) and endemic infectious diseases became relatively more important (Schofield and Reher, 1991). The decrease in infectious processes and the rise of degenerative diseases (cancer, diseases of the circulatory system) are the main components of the health transition which took place at the end of the 19th century (Caselli, 1991; Neven, 1997; Elman and Myers, 1999). This transition may have been especially important for the younger age groups, but it was also of importance to the older age groups (people over 50). This process could imply a change in importance of determinants of mortality differentials. In a pre-transition community, differential resistance (due to malnutrition) and differential exposure (due to overcrowding, poor hygiene, polluted water) to diseases determine mortality differentials (Schellekens, 1989). In a post-transition community, other factors which can affect resistance and exposure may also become important (such as health behaviour). Moreover, even in the same time period there are important regional differences in disease environment (Johansson and Kasakoff, 2000; Woods and Shelton, 2000).

Society also changed with regard to the treatment of diseases (and in the availability of health care). The importance of the biological potential to be less susceptible to a certain disease may become irrelevant in the light of the medical treatment of that disease. Also new lifestyles and a general increase in wealth may

prevent the development of diseases (e.g. better food quality) or, on the contrary, may enhance the development of diseases (e.g. smoking).

For this reason, it is unlikely that someone's biological potential is equally important in each and every society or time period. Due to societal conditions, other determinants may (at some place and/or in some time period) suppress the importance of the biological potential. It is therefore improbable that the influence of biological characteristics has a universal validity. Consequently, it is uncertain whether the realization of the potential for longevity and its sex-specificity as observed in for instance the Jura will also be present in other regions.

The first objective of this study is to test whether the results of Cournil et al. are also found in Moerzeke. We want to compare the results of the Jura villages with the Flemish village because of the relative similarity of the society and the period under scrutiny, that is a farmers' society in Western Europe in the 18th–20th century. If these results are not the same – and they are not – than the question is what factors cause the differences between the two regions. It is not easy to provide an answer to this question as the knowledge of the interaction between the realization of the potential for longevity and environmental characteristics is currently still limited. However, we will try to provide some new elements in this debate.

Before the results of our analysis are presented (section 3), information is given about the methodology, the database and Moerzeke.

2. Material and Methods

2.1. THE PROBLEM OF THE SHARED ENVIRONMENT

Do we really measure "biological" heredity when we measure the correlation between age-at-death of parents and offspring, or are there other mechanisms which are responsible for this correlation? That is the key methodological question in analysing the biological aspects of longevity. The source of the problems in measuring the relation between parents' and offspring's age-at-death is to be found in their shared "environment". Parents and offspring often belong to the same socio-economic and even occupational category, and often share the bulk of their daily practices, habits, attitudes and material environment. For this reason, an observed relationship between parents' and offspring's mortality may not always be caused by their biological relation. Three broad categories can be taken into account to explain the observed correlation between parents' and offspring's age-at-death:

- parents and offspring have the same biological characteristics
- parents and offspring have the same socio-economic characteristics (wealth, occupation, family size and other material life conditions)
- parents and offspring have the same cultural characteristics (customs and practices)

The third possibility can be controlled for by restricting the analysis to a homogeneous community (for instance the Jura or Moerzeke). However, the process of increasing individualisation during the second half of the 20th century could limit the power of this strategy for this period. For instance, 20th-century smoking habits cannot be controlled for, while 18th-century breastfeeding practices can (or at least, with more certainty).

In this study socio-economic characteristics are controlled for by a combination of four strategies. The first is the same strategy as used by Cournil et al. The study is restricted to a small village where (it can be expected that) socio-economic characteristics and material life conditions are more or less the same for most inhabitants. Nevertheless, this strategy is probably not sufficient, at least for Moerzeke, to control for all socio-economic factors. Differences in things such as climate, access to clean water and sewage systems may be non-existent or rather small but differences in wealth, socio-economic status and working environment are surely present. The second strategy is the use of occupational category as an indicator of wealth, socio-economic status and working environment. However, the problem here is the lack of information on, for instance, the possession of land. To tackle this problem, two supplementary strategies are followed. The offspring's socio-economic position and material life conditions are controlled for by comparing the age-of-death of the offspring with the age-of-death of their partners, who are supposed to share this socio-economic position and the material life conditions (for a similar argumentation, see Desjardins and Charbonneau, 1990, pp. 612–613). Furthermore, the evolution of the effect of parental longevity on offspring's longevity will be explored. If this effect changes over time, this indicates its contingency on environmental conditions.

These four strategies will help us controlling for the effect of the shared environment between parents and offspring. Even if these strategies would be perfect, another 'shared environment' problem still exists. Indeed, there are two kinds of shared environment: the shared environment of parents and offspring, and the shared environment of siblings which is not shared with the parents. These siblings have for instance experienced the same intra-uterine conditions (see Madise and Diamond, 1995). Technically spoken, this causes a problem of non-independency of the observations. This forces us to add a random family factor in our statistical model (see section 2.3 for details). With this procedure, we estimate the average effect – over all families – of parents' longevity on offspring's longevity.

2.2. DATA

To realize these strategies we chose the option of performing a historical population study of a small village, namely Moerzeke. All parochial and civil registers of Moerzeke were used to build a database containing all individuals who were born, married or died in Moerzeke in the period 1700–1976. This database was reorganised in a family database, consisting of the first generation (every individual and

Table I. Percentage of inhabitants who were born and died in Moerzeke (by period, sex and occupation)

Period of birth	Men		Women	
	Percentage of inhabitants who are non-migrant*	Percentage of farmers who are non-migrant	Percentage of inhabitants who are non-migrant	Percentage of farmers who are non-migrant
1701–1800	72.2	80.2	75.9	78.8
1801–1850	75.8	80.1	77.1	79.5
1851–1876	70.7	75.5	68.7	72.3

*By non-migrant we mean inhabitants who were born and died in Moerzeke.

his partner) and the second generation (the children of the first generation). There are 5,107 families in the database.

Compared to genealogical databases a database based on family reconstruction has the advantage of recording information on all family members,[4] not just on the ancestors or the male descendants (family size, birth intervals, etc. can be measured). Also the community characteristics, which are unrecorded in historical sources can be held constant (which is impossible with the use of genealogies that gather together family members scattered over a larger area).

The main problem is the loss of emigrants (see also below). People who were born in Moerzeke but who died elsewhere are not included in the analysis simply because their age-at-death is not known. In demographic research this can be a problem for two reasons. First, migration is not an aselect phenomenon. Sexes, occupational and wealth categories, age categories, etc. are often not evenly represented. Therefore, limiting the study to the group of non-migrants makes the sample selective.[5] For Moerzeke, this problem is also present, but it is not of major importance. Firstly, the research question at hand can be studied without using an aselect sample. We are not interested in drawing a total picture of the heredity of longevity in Moerzeke, but we want to analyse this mechanism in well-controlled circumstances. Secondly, the selection effects do not seem too important. In Table I we observe only minor differences. Farmers for instance emigrate less than average, women emigrate less than men (except for the last period).

The second reason why migration is a problem in demographic research is that emigrants almost by definition have a higher life expectancy at birth than non-migrants. People who live longer have more opportunities to emigrate. By contrast, infants who die before their first birthday have little chance of emigrating. But all this is of less importance when the analysis is limited to life expectancy at age 50, because emigration is more common in early life (migration on marriage, migration in search of a job – although emigration of the elderly in relation to care for a family member must not be underestimated). We will perform some checks on this problem in the next section.

2.3. SELECTION OF PARENTS AND OFFSPRING, STATISTICAL ANALYSES

In order to compare the results of Moerzeke with those of the Jura, we take the method developed by Cournil et al. as a starting point (see Cournil et al., 2000, pp. 1021–1022). Their (and our) analysis of longevity is based on the mean conditional life expectancy at a minimum age of 50 years for parents and 55 years for children.[6] Typical of Cournil et al.'s method is the variance and contrast analysis of differences in mean age-at-death of the offspring of four parental groups (parents grouped by their age-at-death). Thus the relationship between the longevity of parents and offspring is not seen as linear (see for instance Poulain and Debuisson, 1990). On the contrary, they think of a threshold-effect. This threshold-effect is not only found by Cournil et al. but also by others (see for instance: Gavrilova et al., 1999, 2000).[7]

The *sampling of parental couples* was carried out in two stages. In the first stage parents were selected if the following criteria were fulfilled: their date of birth and death is known, they were born between 1700 and 1850, and for at least one child the dates of birth and death are known and the place of birth and death of this child is Moerzeke.[8] In the second stage couples were dropped if one of the parents belonged to the first quartile of the age-at-death distribution.[9] This procedure was performed separately for each sex and ten-year birth cohort.

796 couples fulfil these criteria. The mean lifespan of the fathers was 73.7 years (se = 0.29), which is slightly higher than the mean lifespan of the mothers (72.4 years, se = 0.3). The minimum ages at death are 53.3 years for the fathers and 51 years for the mothers.

This group of parents was split into four groups, according to their age-at-death. Fathers and mothers were defined as long-lived if they exceeded the percentile 30 of the age-at-death distribution. This procedure was performed separately for three birth cohorts (and for each sex)

- Parental group 1 (pg 1) = short-lived father and mother
- Parental group 2 (pg 2) = short-lived father, long-lived mother
- Parental group 3 (pg 3) = long-lived father, short-lived mother
- Parental group 4 (pg 4) = long-lived father, long-lived mother

We will focus on the differences between the first and the fourth parental groups. The differences between other combinations of the parental groups are also useful, for instance in determining the relative importance of each parent in the inheritance of longevity. However, discussing the biological mechanism in detail is not the aim of this article.

The four parental groups are homogeneously distributed over the time period (year of birth is not significantly different over the four groups – non-parametric Kruskall-Wallis test in a one-way anova procedure). Also, the age-at-marriage of both parents did not differ significantly over the four groups (for the importance of this factor, see Cornell, 1989). However, the first and fourth parental group differed from the second and third in terms of the age gap between the parents.

Table II. Percentage of emigrants per parental group, men and women born before 1877 (N = 2510 for men, N = 2460 for women)

	Men		Women	
	% of emigrants	N of emigrants	% of emigrants	N of emigrants
Parental group 1	18.8	228	20.3	243
Parental group 2	18.8	90	19.6	97
Parental group 3	18.1	95	17.6	88
Parental group 4	13.6	38	17.4	44

These 796 couples had 4,378 children for whom exact dates of birth and death are known. The problem here is how to test whether missing values are homogeneously distributed over sex and parental group. Most missing values on date of death are accompanied by missing values on place of death. This means we cannot separate those who died in Moerzeke on an unknown date from those who died elsewhere on an unknown date. Our opinion is that almost all of them are emigrants (see Table II).[10] However, the missing data (or: the number of emigrants) are homogeneously distributed over parental group and sex (χ^2-tests).[11]

To avoid censoring problems, we have limited the analysis to children born before 1877 (as the observation of deaths was limited to 1976). The dates of birth spanned the period of 1727–1876, and the dates of death the period of 1795–1974. 1,533 of the children of the group with known dates of birth and death died after their 55th birthday. The mean lifespan of the sons was 73.6 years (se = 0.3 and n = 808), and 73.3 years for daughters (se = 0.4 and n = 725).

As a result of this procedure, the number of observations was restricted to 639 parents and 1,533 children, which were distributed in this way:

- pg 1: 312 parents with 722 children
- pg 2: 130 parents with 297 children
- pg 3: 126 parents with 297 children
- pg 4: 71 parents with 217 children

Compared to Cournil et al. we perform some additional analyses in order to evaluate the impact of environmental factors. Firstly, for the anova-analysis we use a mixed model. We add a random factor, namely family, which is nested within parental group, as each family can only belong to one parental group. This nested variable is indicated as “family (parental group)”. The effect of parental group on offspring’s age-at-death is tested using an additional error term, namely family (parental group). Also for the other variables the appropriate error term is used (for technical details on the use of random factors in anova, see Jackson and Brashers, 1994). Secondly, we extend the anova-analysis with more control variables. After that, we replace age-at-death of the offspring (as the dependent variable) by age-

at-death of the offspring's partner. Finally, the evolution of the age-at-death is examined by plotting it by parental group and sex.

Variables used in the statistical analyses are the following:

- parental group of the index person (index person = offspring) (see above)
- family: identification variable per family (parents and offspring)
- age-at-death of the offspring and of the offspring's partner
- ten-year birth cohort of the index person
- sex of the index person
- occupational category of the husband in the family of the index person (occupational category of the index person if the index person is a man, occupational category of the husband of the index person if it is a woman). Three categories: farmers; elite – white collar workers – craftsmen – merchants; and a residual group of mainly unspecialised and unskilled helpers.
- family size of the index person: three groups (1–3, 4–6, more than 6 children)

2.4. MOERZEKE

Moerzeke is a small village in the centre of Flanders (Belgium), in the province of East Flanders (*Oost-Vlaanderen*). It can be described as geographically isolated as it is almost completely surrounded by the river Scheldt. Moerzeke was mainly populated by farmers until well into the 20th century (De Ridder, 1984). During the second half of the 19th century the rural textile industry became gradually more important. Before the First World War almost every inhabitant worked in agriculture in Moerzeke (De Beule, 1962). After the War some (modest) industrial activity came to the village, but in 1947 60% of the employed males were still involved in farming (De Beule, 1962).

The population of Moerzeke rose from approximately 2,000 in 1761 to 4,706 in 1950 (De Beule, 1962). Not surprisingly for a farmers' community, the age at first marriage was rather high. It rose from 1760 onwards (De Ridder, 1984) and peaked in the mid-19th century (own figures). Also, fertility was traditionally high (De Ridder, 1984) and dropped at the beginning of the 20th century (De Beule, 1962). In the 18th century major mortality crises (mainly dysentery) occurred, but these became less grave as the 18th century advanced (De Ridder, 1984). Striking were the heavy infant and child mortality (De Ridder, 1984). The infant mortality did not drop until the first decades of the 20th century (own figures). The life expectancy at age 50 rose steadily for those born in the 19th century, reaching a peak at the end of the observation period (those born after 1850).

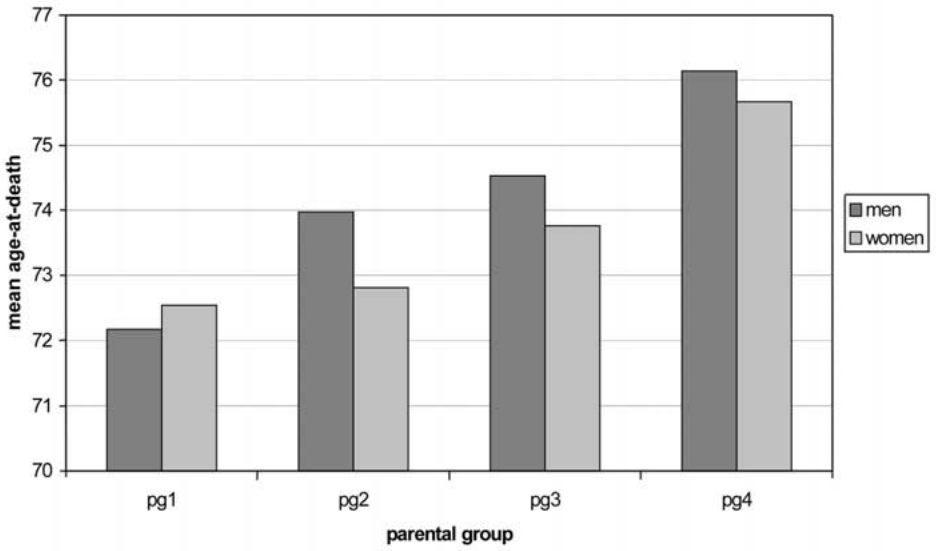

Figure 1. Moerzeke: mean age-at-death at age 55 of offspring of four parental groups (pg1, pg2, pg3, pg4) (men and women separate).

3. Results: Moerzeke versus the Jura

3.1. THE HERITABILITY OF LONGEVITY IN MOERZEKE

In Moerzeke, there is a considerable difference in mean age-at-death of the offspring of the four parental groups. Figure 1 shows these means. Contrast analysis shows for example that the difference between pg1 and pg4 is significant (Table III – other contrasts are also significant). The general effect of parental group is however not significant (Table III). If we would not have used the random family factor – like Cournil et al. – then the general effect of parental group would have produced highly significant results.[12] Neither sex (Table III), nor the interaction between sex and parental group is significant. Also, the contrast analysis of male versus female, controlling for parental group shows no significant results. It may be that the most straightforward measure of the heredity of longevity is the difference between the two extreme parental groups (pg1 and pg4), henceforth called pg-differences. In Moerzeke, this difference is 4.0 years for men and 3.1 years for women.

3.2. COMPARISON BETWEEN MOERZEKE AND THE JURA

In both Moerzeke and the Jura there is an important difference in mean age-at-death between the offspring of the fourth and the first parental group (sons and daughters not separated). These differences are 3.7 years in the Jura and 3.6 years in Moerzeke (Figure 2).

Table III. Effect of parental group, cohort and sex on offspring's age-at-death at age 55

Type III, random factor anova	DF	F Value	Pr > F
Parental group	3	1.96	0.1188
Sex of index person (offspring)	1	0.22	0.6374
Parental group* sex of index person	3	0.53	0.6606
Family (parental group)	601	1.32	**0.0015**
Birth cohort index person	15	1.07	0.3859
MODEL	1133	1.33	**0.0004**
ERROR	387		
Contrast analysis			
Parental group 1 versus parental group 4	1	6.25	**0.0126**
Parental group 2 versus parental group 3	1	0.20	0.6531
Parental group 1 versus parental group 3	1	1.48	0.2248
Parental group 1 versus parental group 2	1	3.03	**0.0822**
Parental group 2 versus parental group 4	1	0.80	0.3725
Parental group 3 versus parental group 4	1	1.77	0.1831
Contrast analysis: male versus female			
Parental group 1	1	0.33	0.5634
Parental group 2	1	0.61	0.6171
Parental group 3	1	0.00	0.9634
Parental group 4	1	0.12	0.7256

There are however also important differences. The difference between the two extreme parental groups has a different sex composition in the two regions. In Moerzeke the sons (Figure 3) show the largest difference (4.0 years–2.1 years in the Jura); in the Jura the daughters show a difference of 5.6 years whereas in Moerzeke the difference is 3.1 years (Figure 4). Also, the significant difference between sons and daughters of the fourth parental group does not appear in Moerzeke.

Thus, in both regions there is indeed a difference in the mean age-at-death of the children of the different parental groups. But every region shows for one sex that larger differences are possible. Why do women in Moerzeke not realize their hereditary potential for longevity? Why do men in the Jura not realize their hereditary potential for longevity? A possible reason could be the composition of the sample. In the Jura, 90% of the population consisted of farmers. This is not the case in Moerzeke. In order to tackle this problem we split the sample according to the occupational category of the selected individuals.

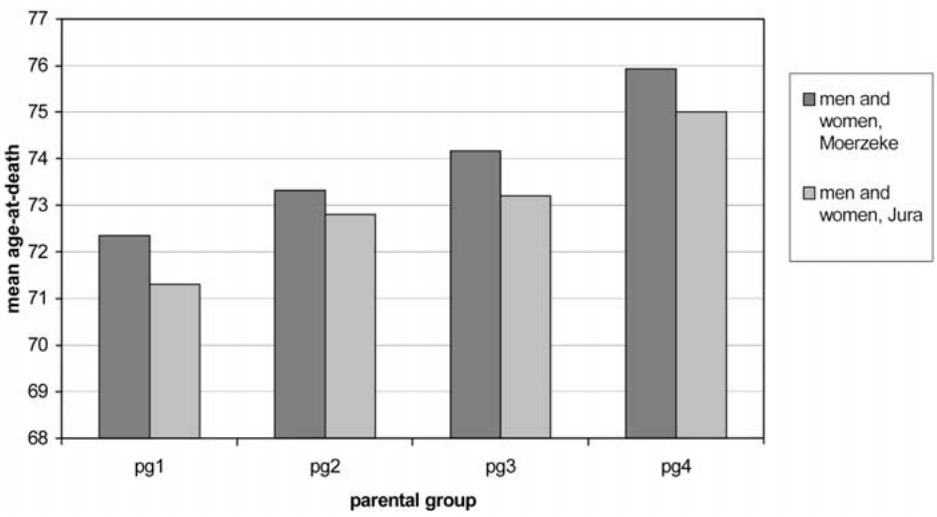

Figure 2. Moerzeke versus the Jura: mean age-at-death per parental group (men and women together) (Source for the Jura: Cournil et al., 2000).

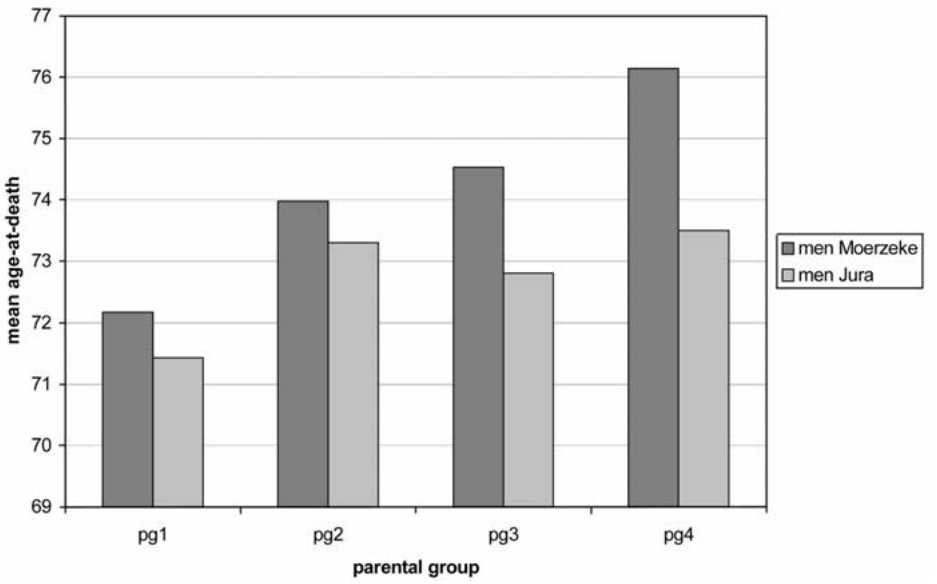

Figure 3. Mean age-at-death at age 55 of offspring of four parental groups (pg1, pg2, pg3, pg4), men, Moerzeke versus Jura (Source for the Jura: Cournil et al., 2000).

3.3. LONGEVITY BY OCCUPATION

By limiting our analysis to farmers, we make our sample more homogeneous and more comparable to the sample of Cournil et al.[13] From Table IV we can conclude that – for men – the above difference between parental groups remains high, even if

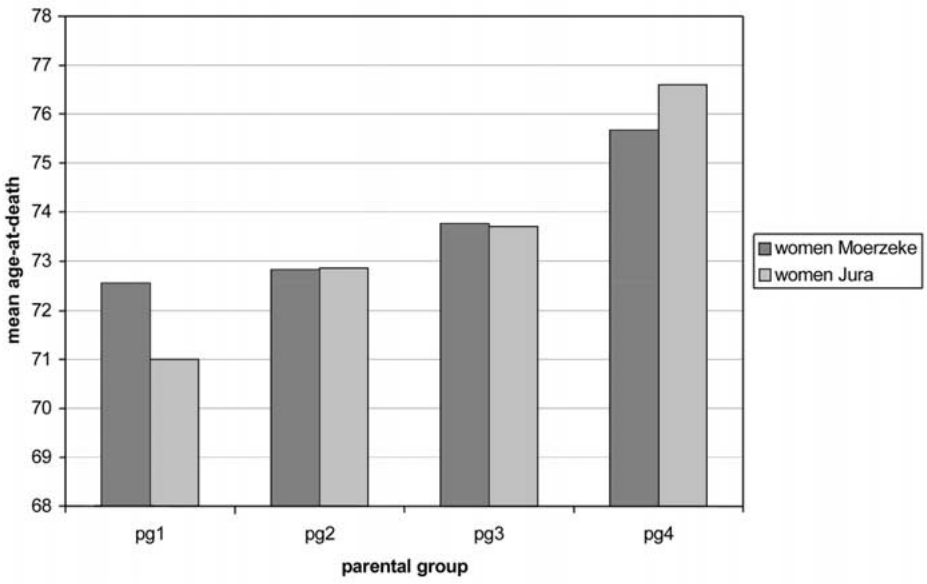

Figure 4. Mean age-at-death at age 55 of offspring of four parental groups (pg1, pg2, pg3, pg4), women, Moerzeke versus Jura (Source for the Jura: Cournil et al., 2000).

Table IV. Difference (in years) between the first and the fourth parental group by occupational category (men and women, Moerzeke)

	Men		Women	
	Difference between first and fourth parental group in years)	N	Difference between first and fourth parental group (in years)	N
Occupation: farmer	4.8	396	1.3	269
Occupation of partner: farmer	2.9	225	2.7	216
Occupation: not farmer	0.9	331	3.7	319
Occupation of partner: not farmer	3.3	320	1.1	281
Occupation of father: farmer	3.4	259	3.0	254
Occupation of father and occupation of son/daughter: farmer	4.1	169	0.2	146
Occupation of father and occupation of partner of son/daughter: farmer	4.1	92	–0.3	102
Total	4.0	808	3.1	727

*There are a lot of missing values for the occupational category of women, which makes interpretation difficult.

we limit our sample to farmers. The group of non-farmers is very heterogeneous, so the low level of difference between the parental groups could perhaps be ascribed to this. For women whose husbands are farmers, the differences between parental groups remain more or less the same.

From these figures it can be concluded that the above differences between Moerzeke and the Jura cannot be ascribed to the difference in sample composition. It is not the occupational structure of Moerzeke that makes the observed realization of the potential for longevity different from the Jura. Our conclusion is that the realization of the potential for longevity as such is different in Moerzeke and in the Jura. Although in both regions we can observe a significant difference between the mean age-at-death of the offspring of the extreme parental groups, these regions show different figures for both sexes.

4. The Influence of Environmental Conditions

The results of the previous section lead to the proposition that environmental conditions are at stake. We cannot take Cournil et al.'s results as "universal facts" (which they do not claim). There are three possible ways in which environmental conditions can influence the correlation between the longevity of parents and offspring:

- there is no correlation between the longevity of parents and offspring. Other determinants cause the correlation between parents' and offspring's age-at-death, for instance the shared environment of parents and offspring;
- there is a correlation, but it is not (wholly) visible because other determinants obscure it;
- the correlation is not consistent over time.

To test this we incorporated new variables in the anova-analysis (see 4.1.). Secondly, we performed further analyses to explore the role of the shared environment (see 4.2.). Finally, we controlled for environmental changes by comparing different periods (see 4.3.).

4.1. CONTROL VARIABLES

We can control for some environmental conditions during adulthood by introducing the occupational category of the index person (or his partner) and the number of children in the family of the index person into the analysis. The number of children is important as "For women, the lower mortality at post-reproductive ages ... could be associated with lower fertility ..." (Caselli and Capocaccia, 1989, p. 153).

The above variables were introduced in a new anova-analysis (Table V). From the anova-results, we can conclude that controlling for these additional variables makes the general effect of parental group more significant (compare with Table III). This shows that these variables obscured the observation of the effect of parental group on offspring's longevity. The difference between pg1 and

Table V. Effect of parental group, cohort, sex and control variables on offspring's age-at-death at age 55 (men and women, Moerzeke)

Type III, random factor anova	DF	F Value	Pr > F
Parental group	3	3.20	**0.0229**
Sex of index person (offspring)	1	0.03	0.8580
Parental group* sex of index person	3	0.40	0.7500
Family (parental group)	601	1.41	**0.0141**
Birth cohort index person	12	1.92	0.0412
Occupational category**	2	0.06	0.9448
Sex of index person∗Family size of index person	6	1.24	0.2913
MODEL	1410	1.38	**0.0155**
Error	110		
Contrast analysis			
pg1 versus pg4	1	6.43	**0.0114**

**For women: occupational category of the partner.

pg4 remains significant, the interaction term (sex and parental group) remains insignificant.

4.2. NON-BIOLOGICAL HEREDITY OF POST-REPRODUCTIVE MORTALITY

Some authors suggest that the origin of the heredity of longevity is not biological but social (see above) and point to the shared environment of parents and offspring as the reason for this misunderstanding. This doubt is justified since the method of controlling for other causes of heredity with the present data is not wholly satisfactory (we only have information on the occupational category) or simply impossible (we have no data on health practices such as smoking habits). We can only partly tackle this doubt (see below).

But the examination of the mortality of the index person's partner is an important check. Suppose the correlation between age-at-death of parents and offspring is caused by their shared socio-economic position. The underlying argumentation could be that parents live longer because their wealth allows them to avoid hard work, buy better food, have adequate medical care, etc. and that they pass on this wealth and the ability and/or opportunity to live longer to their offspring. If this is true, then the mean age-at-death of the index child's partner should also benefit from the wealth of the parental family of the index person, because this wealth is not given to the individual child but to the family of that child.[14]

Figure 5 shows the mean age-at-death of the index persons' partners, grouped by the parental group of the parents of the index persons. The difference between pg4

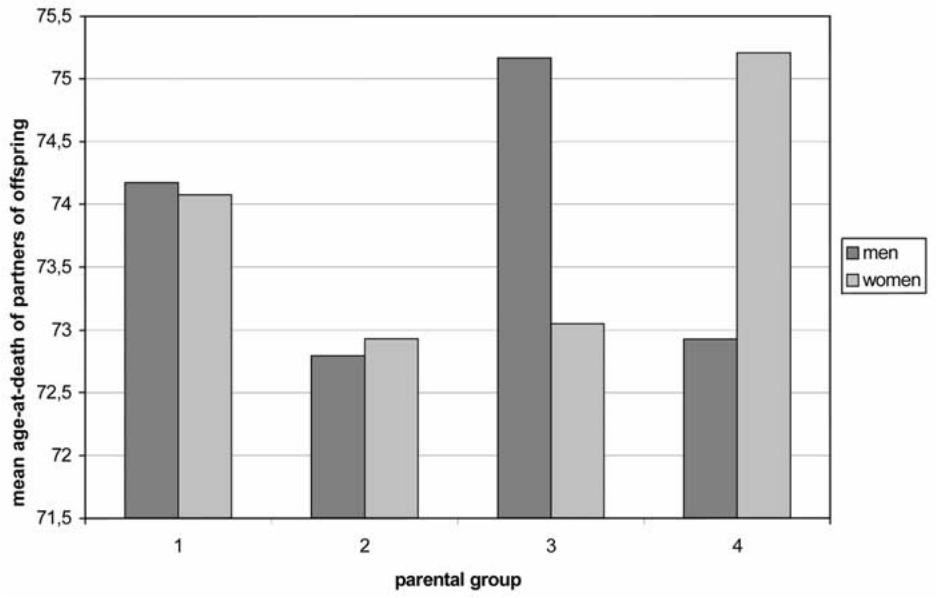

Figure 5. Mean age-at-death at age 55 of partners of the offspring by parental group (men and women, Moerzeke).

and pg1 is 1.2 years for men and 1.1 years for women. Clearly, the index persons' partners do not benefit from their parents-in-law's wealth. It would be strange if the parental wealth only helped their own children and not these children's partners. The only acceptable socio-economic conclusion in explanation of this observation might be that parental wealth at an early age is beneficial throughout the index person's life.

4.3. LONGEVITY BY TIME PERIOD

The degree to which the potential for longevity is realized is not stable over time. The differences between the mean age-at-death of the offspring of the fourth and first parental group change dramatically. Figures 6 and 7 show the evolution of the mean age-at-death of pg1 and pg4. For men, the differences between pg1 and 4 rise to a maximum for men born in the period between 1821 and 1860. In this period, the mean pg-difference is 4.5 year. Thereafter, the difference decreases and almost disappears at the end of the observation period. For women this pattern is somewhat different. The pg-differences start earlier and also decrease earlier. Differences between pg1 and pg4 are at a maximum for women born between 1791 and 1830. In this period, the mean pg-difference is 5.5 year.

We can draw two conclusions from these results. Firstly, the link between age-at-death of parents and offspring changes over time; this is another indication of the influence of environmental conditions. Secondly, the potential for longevity

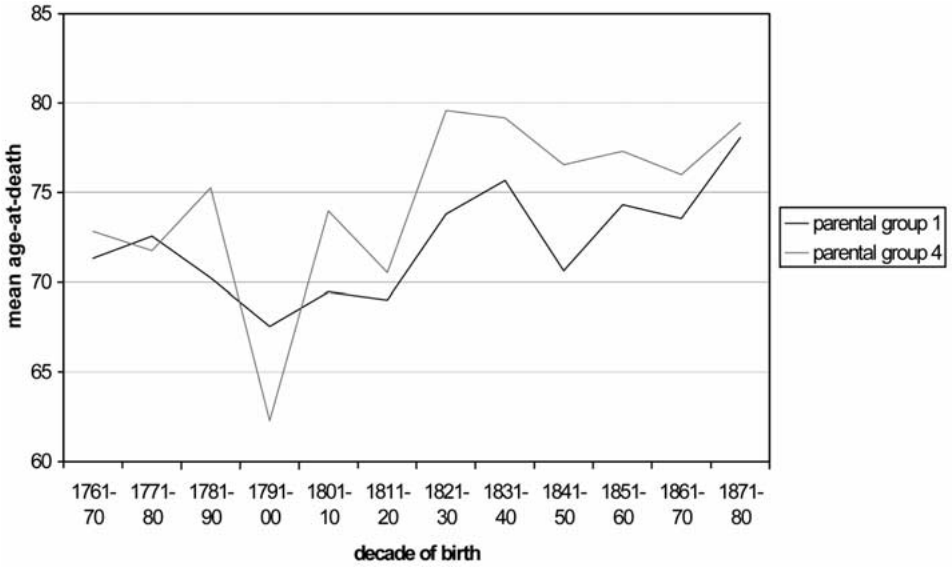

Figure 6. Mean age-at-death at age 55 of the offspring by parental group (men, Moerzeke).

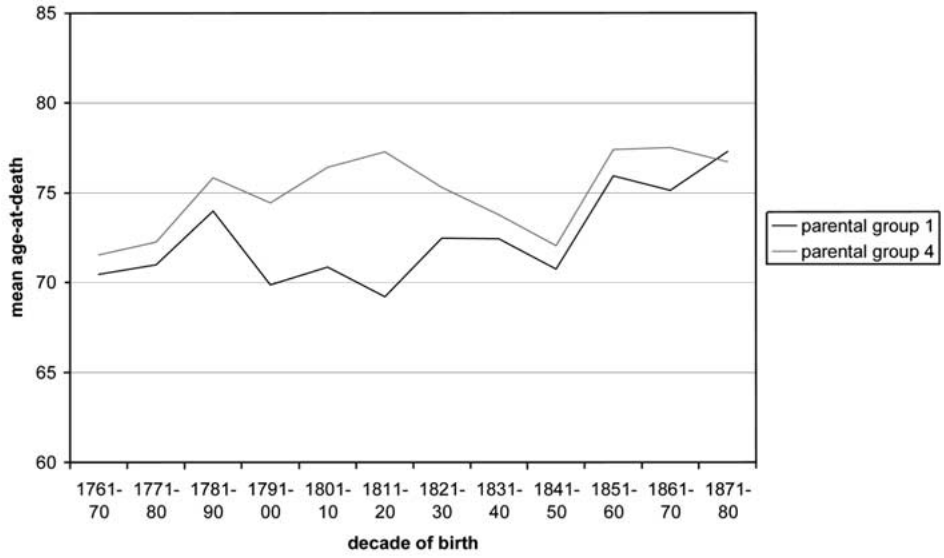

Figure 7. Mean age-at-death at age 55 of the offspring by parental group (women, Moerzeke).

was indeed "obscured", mainly for women born between 1791 and 1830. These women show a pattern similar to the women in the Jura (for the whole period).

It is only in a temporary, specific constellation of societal conditions that the parental group is a determinant of the offspring's age-at-death. The question is what environmental changes cause this changing importance of the parental group. Answering this question is difficult, as relevant information on things such as

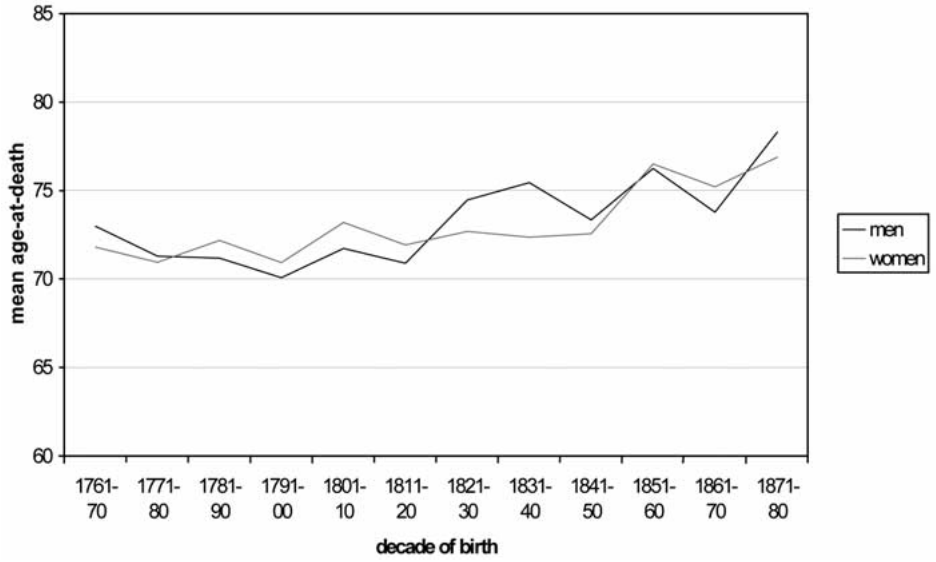

Figure 8. Mean age-at-death at age 55 of the offspring by sex (Moerzeke).

causes of death (and also on environmental characteristics) is not available for Moerzeke. However, we will try to sketch the answer in broad terms.

An important fact is that in the period in which female pg-differences start to rise (women born after 1791), their mean age-at-death is also higher (see Figure 8). This could indicate a lower impact of infectious diseases on women in that period.[15] There are two (not mutually exclusive) possible explanations for the different timing in the increase in pg-differences for men and women. The first possibility is that men suffer more from bad or dangerous working and living conditions than women. This is not implausible. The fact that men more often fall victim to (some) infectious diseases is sometimes ascribed to their higher exposure to these diseases because of their more frequent contacts with "the outside world" (see Sköld, 1997 for an illustration of this in the case of the decline of smallpox). Another possibility, however, is that men and women have a different susceptibility to some diseases and causes of death. It is not impossible that infectious diseases are less important for women than for men. This would result in the dominance of other causes of death, and, along with this, in the dominance of other determinants of mortality.

If we accept that biological characteristics are more important for non-communicable diseases (see below) and if women suffer less from infectious diseases (for whatever reason), then it is not surprising that pg-differences start to increase at an earlier stage for women. These conclusions are, however, speculative. We shall return to this matter in the discussion section.

Another peculiarity is the difference between men and women in the timing of the decline of the pg-differences. Figure 8 shows that for those born in the period 1821–1850, men show a higher mean age-at-death and show also a continuation

of the high pg-difference. Also in this case the sex with the highest pg-difference has the highest mean age-at-death. For what reason women in this period have a lower mean age-at-death? The history of Moerzeke teaches us that in this period infant mortality was high (see section 2.4 above). This high level of infant mortality coincides with an agricultural crisis. However, we have no particular reason to state that men suffered less from these heavy living conditions. So, whatever the reason for the lower female age-at-death, it shows that something constrains their life span, and this hinders the realisation of the differences in mean age-at-death by parental group.

5. Discussion

The main conclusion of this article is that it is premature to ascribe the observed sex-specific differences in heredity of longevity in the Jura to purely biological and universal differences between men and women. Yet we do not exclude the existence of biological determinants of longevity. In our study on Moerzeke, as in the study on the Jura, important and significant differences in longevity of the offspring can be linked with longevity characteristics of the parents. Two important qualifying remarks have to be made. Firstly, our results show the importance of control variables. It is, of course, not unthinkable that omitted (unmeasured) control variables could influence the statistical results. In general we could say that the knowledge on the relation between the biological and environmental characteristics is still too limited to permit a clear-cut interpretation. Secondly, it is our opinion that the biological mechanisms which possibly underlie the heredity of longevity can only be observed in appropriate macro-environmental conditions. This is illustrated by the fact that pg-differences only occur in specific periods (for men born between 1821 and 1860 and for women born between 1791 and 1830). These changes in the importance of pg-differences over time are, of course, caused by a complex interplay of several variables. We lack the direct information on Moerzeke needed to elucidate this problem in all its aspects. This means that we can only try to give a broad (and rather vague) explanation.

A few hypotheses can be stated. The increase in pg-differences during the 19th century might be linked with the epidemiological transition. In the first phase (18th century) crisis mortality was prominent, but declining in importance (De Ridder, 1984). In this phase, malnutrition and exposure may be the main determinants of death. Nevertheless, resistance to the infectious diseases which cause crises may have familial roots. An infectious disease such as tuberculosis may have a familial component, whereby some children inherit a lower resistance to the disease (Puranen, 1991). In the second phase crisis mortality decreased (the stabilisation of death) but endemic infectious diseases became relatively more important (Schofield and Reher, 1991). Some suggest that resistance may at this moment be partly caused by familial resistance, although its impact on the mortality decline seems relative (Perrenoud, 1991). However, it is more probable

that non-communicable diseases are more closely linked with biological potential than communicable diseases simply because only it is in the case of the latter that the degree of "exposure" to the disease is an important – and not biologically determined – factor. The combination of the earlier rise in the pg-differences for women and their higher life expectancy in that early period of high pg-difference suggests this. We can conclude that at this moment it is not wholly clear whether resistance to infectious diseases or resistance to degenerative diseases lies at the origin of this increase in pg-differences. It is impossible to solve this problem without more information on causes of death in Moerzeke. But we can state with more certainty that the increase in the pg-differences during the 19th century shows that it is only after the decline of crisis mortality that biological characteristics become important.

The decrease in the pg-differences in the 20th century could be linked to the general increase in wealth and the increasing availability of health care, thus making biological disadvantages less important. Another possible explanation can perhaps be found in the growing importance of lifestyle and health practices in the 20th century. A puzzling result is the different timing of the decrease in pg-difference between men and women. This could be due to harder living and working conditions for women as they have a lower mean age-at-death in this period.

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Notes

[1] By biological characteristics or biological potential we mean genetic characteristics.

[2] Important material life conditions are for instance: family characteristics (size, crowding), family wealth (nutrition, type of housing, access to clean water, good sanitation, adequate food and health care), occupation (wealth, working conditions, contact with other communities).

[3] Of course, not all causes of death are linked with biological characteristics to the same degree.

[4] On the condition that their demographic events occur in Moerzeke.

[5] However, this does not necessarily mean that (all) demographic characteristics of migrants are different from non-migrants (Cloet and Vandenbroeke, 1989).

[6] The border of age 55 is chosen for reasons of consistency. We use the same methods as Cournil et al. to have an optimal comparison between Moerzeke and the Jura.

[7] Apart from this, we also have a pragmatic reason for this choice. Using a method measuring linear correlation would make it impossible to estimate the role of environmental conditions as Cournil et al. and we would measure different phenomena.

[8] Using this procedure, parents are likely to have lived most part of their lives in Moerzeke (they

were present at least at the birth of their children). The same goes for the offspring (the possibility exists however that between their birth and death in Moerzeke they lived elsewhere).

[9] The 75% oldest parents are selected. We used the same strategy as Cournil et al.

[10] We compared the size of this group with migration figures. We also estimate death registration as (relatively) exact.

[11] There may be some problems for the last period (women born after 1876). Women emigrated more in that period, or – what is more probable – lived longer and were still alive at the moment the compilation of the dataset was closed.

[12] The effect of parental group in the Jura is highly significant (Cournil et al., 2000). The difference in significance of the general effect of parental group between Cournil et al.'s analysis and ours, is probably caused by our use of a random family factor. The Figures 1, 2, 3 and 4 give a better perspective on the similarities and the differences between the Jura and Moerzeke.

[13] Also the time period is somewhat different. We do not think that this explains the difference between Moerzeke and the Jura, although the relation between time and pg-differences is important (see section 4.2.). Limiting the difference in time periods to a minimum does not make our results more comparable to Cournil et al.'s results.

[14] One might argue that this is only valid for women as men are primarily dependent on their own socio-economic status and less on the socio-economic status of their parents-in-law.

[15] "Deaths from acute conditions are more prevalent in high mortality groups" (Himes, 1994, p. 642).

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