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„Life History Theory: How the childhood environment affects humans' later life outcomes such as reproductive and marriage behavior, educational attainment and income. "

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The last five years of my professional and private life very much circled around fertility behaviour and fertility decisions. In 2014, when I started to work on my PhD, I was very consumed by questions such as 'When is the best time to get pregnant?' or 'How and to what extend will a woman's professional career suffer from becoming a mother?'. The more I focused on women's lives in my study populations, the impact of their fertility decisions on their lives and their children's lives, the more I realized that there will never be an optimal timing and children always come with costs. Regardless, close to the finalisation of my PhD the wonder of life struck me. I gave birth to this amazing little girl and all the questions regarding fertility decisions faded. My journey of becoming a researcher and a mother would not have been possible without several people that accompanied me.

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Abstract

Human fertility behaviour and reproductive decision-making is highly influenced by social and economic factors and is expected to be driven also by evolutionary processes. The present thesis is looking at human fertility behaviour through the evolutionary lens and therefore provides novel insights to what extent biological, ecological and socio-economic factors shape fertility patterns and reproductive decision-making in different stages of the demographic transition and how they interfere with each other.

The first study tests if exposure to high mortality within the natal family in early childhood leads to faster and riskier reproductive strategies in pre-industrial European society. The results reveal that women who were exposed to high mortality cues within the natal family were at a greater risk to reproduce earlier and outside a stable union. Giving birth to an illegitimate child served as a proxy for risky sexual behaviour. Further, the study shows that the risk of giving birth out of wedlock is linked to individual mortality experience rather than to family-level effects. In contrast, adjustments in marital reproductive timing are influenced more by family-level effects than by individual mortality experience.

The second study therefore investigates the impact of famine-related high mortality and social factors on union formation in a pretransitional/transitional European population. The results show that individuals accelerate their transition to marriage when they were exposed to high mortality cues during early childhood. These results further stress the importance of individual's early life conditions on their life-history trajectory.

The third study considers the findings that fertility behaviour and reproductive decision-making varies across social classes and sheds some light on sex-biased parental investment in a post-transitional Western population. The study reveals that parents bias their parental investment/support depending on their social class towards the sex with the higher expected reproductive success. Low status parents invest more in their daughters' higher education, whereas high status parents invest more in their sons' higher education.

Chapter 1

General Introduction

Explaining fertility patterns and declining birth rates below replacement level is of great interest for demographers, sociologists, economists and behavioural ecologists. Most developed countries completed the first demographic transition in the previous century. During the second stage of the demographic transition mortality rates decline due to improvements in nutrition, hygiene and medical treatment. In response to both declining morality and the increase of population growth, human fertility behaviour changed, hence the fertility rates start to decline in the third stage of the demographic transition (Coale and Coots Watkins, 1986).

Human fertility behaviour and reproductive decision-making is highly influenced by an individual's personal life circumstances (*e.g.* genetic, ecological, economic and social factors – Barban et al., 2016; Perry et al., 2014; Voland, 1998). In the view of demographers and sociologists, declining mortality rates was due to social and economic changes such as industrialisation, urbanisation, as well as a greater access to education (Bongaarts and Watkins, 1996; Notestein, 1953). Social class affiliation has a strong influence on human fertility patterns and its impact has changed over the course of the demographic transition. Furthermore, individuals from different social classes sex-bias their parental investment. Parents from high social classes invest more in their sons' status attainment than in their daughters. In contrast, parents from a low social class favour their parental investment towards daughters rather than towards sons (Hopcroft, 2005; Hopcroft and Martin, 2016). In the historical context high socioeconomic status was associated with high fertility (Betzig, 1986) as well as in modern societies, male social status is associated with an increase in fertility mostly due to a lower percentage of childless individuals among men of high social status (Fieder et al., 2011; Fieder et al., 2005; Hopcroft, 2018; Hopcroft, 2015; Nettle and Pollet, 2008).

However, while previous demographic and sociological studies have pointed to the importance of social and economic factors that are influencing human fertility behaviour, they have overlooked the importance of biology in human fertility processes. This omission of the evolutionary perspective hampers our understanding of reproductive processes and fertility behaviour (Hopcroft, 2018; Sear et al., 2016; Stulp et al., 2016). To get a better understanding of human fertility behaviour and reproductive decision-making, it is crucial to integrate the evolutionary approach into sociological, demographic and economic research.

From a purely biological point of view, reproduction is the single most important trait to maximize/optimize the individual's own fitness. Reproduction can therefore be expected to be driven also by evolutionary processes, and not merely by social and economic factors. Individuals adapt their fertility behaviour according to the ecological and social context they are living in. Hence, these individual adaptation processes are the reason for variation within human fertility behaviour. Life-history theory predicts that individuals accelerate their reproductive strategy under harsh environmental conditions: earlier maturation, earlier marriage, earlier reproduction, shorter interbirth intervals and less investment in each offspring (Roff, 2002; Stearns, 1992). For instance, high mortality is one of these harsh environmental conditions that has a strong influence on human fertility behaviour. A high mortality rate minimizes the number of reproductive events and hence fitness is reduced. Therefore, individuals will benefit from an early reproductive onset and lower parental investment (Belsky et al., 2010; Chisholm et al., 2005; Quinlan, 2007).

By acknowledging the importance of both biological and socioeconomic factors in shaping human fertility patterns new insights can be gained. By viewing human fertility behaviour through the evolutionary lens, this thesis pursues the question up to what extent biological, ecological and socio-economic factors shape fertility patterns and reproductive decision-making in different stages of the demographic transition and how they interfere with each other.

This thesis is structured in five chapters and is based on three publications (either published or submitted) that are presented in chapter two to four.

Chapter one introduces the field of fertility behaviour research, considering the influence of biological, economic and social factors. Firstly, one of the basic biological theories (life-history theory) that is applied to explain the variation of fertility behaviour among many species will be introduced. Secondly an overview of biological conditions – with a focus on mortality – that induce early reproduction is given. This is followed by an introduction to different social factors (reproduction within a stable union versus giving birth out of wedlock) that are associated with divergent reproductive trajectories. The last section of chapter one focuses on parent's reproductive decision-making in terms of sex-biased parental investment. This part introduces the Trivers-Williard hypothesis and explains how social class influences parental investment.

Chapter two focuses on the impact of high mortality within the natal family and that of social class on women's reproductive behaviour. The aim is to test if exposure to sibling death in early childhood (0-5) increases women's likelihood of giving birth out of wedlock and therefore outside a stable union in pre-industrialized European population.

Based on the findings that high mortality within the natal family led to reproduction outside a stable union **chapter three** investigates the impact of famine-related mortality and social factors on union formation (marriage) in the 19th century Belgium. In this period reproduction was mostly limited to married people and therefore marriage could be seen as the social maturation to enter the reproductive phase in life.

Chapter four sheds light on the findings that fertility behaviour varies across social classes. Previous research had shown that men's social status is positively influencing the total number of offspring. Chapter four investigates how parents bias their investment depending on their social class towards the sex with the higher expected reproductive success.

Chapter five summarizes the contents of the three publications and the general conclusion.

1.1. Life-history theory

1.1.1. General background

In evolutionary biology, life-history theory (LHT) is a concept that has been developed to explain the variation in age-specific life function such as growth, sexual maturation, reproduction, and death. The LHT concept is based on thermodynamic laws: energy that is used for one purpose cannot be used for another one (Hill, 1993). The energy that an individual is harvesting from the environment during its life cycle is invested in different age-specific life functions such as growth, maintenance, maturation and reproduction (*e.g.* Hill, 1993; Roff, 2002; Stearns, 1992).

Since the available energy is limited, individuals are facing certain trade-off decisions between somatic and reproductive efforts during their life span. These trade-off decisions are not taken consciously and rationally; they are rather a result of physiological regulation (Störmer and Voland, 2014). By investing the allocated resources in growth – including height, weight and overall mass – an individual increases its body size. Good environmental conditions such as sufficient nutrition, clean water, and access to health care increase adult body size (Ellis, 2004; Hill and Hurtado, 1996; Walker et al., 2006). By investing a certain amount of energy into maintenance the organism is able to perform metabolic processes, to repair/replace somatic tissue and to defend itself against pathogens and parasites by immune action (Hill, 1993; Worthman, 2003, p. 293). After reaching a certain body size, harvested energy is channelled into the development of body functions required for reproduction and reproduction per se.

Once energy is dedicated to reproduction length growth is inhibited. In humans the growth phase is completed within the first two decades of life followed by the reproduction phase (Kaplan et al., 2001). Via this strategy humans minimized the following trade-off decisions: On the one hand, growth and the development of body functions increase the likelihood of the individual's survival. On the other hand, via reproduction an individual replicates its genes and therefore maximizes its long-term fitness. Entering puberty is the starting point of the reproductive phase. For females, age at menarche is a marker of puberty timing that is genetic heritable ($h^2=0.49$) and varies between individuals (Perry et al., 2014; Towne et al., 2005). In order to reproduce an individual uses energy for gametes, gestation and lactation. In humans, as in most species, female reproduction comes at higher energy costs than male reproduction (Barrett et al., 2002, p. 94; Störmer and Voland, 2014, p. 7).

Human reproduction in general depends heavily on the disposability of resources. Three major trade-offs in terms of reproduction are defined: those between the quality and the quantity of offspring, those between the current and the future reproduction, and those between mating and parenting effort (DelGuidice et al., 2015). All of the three trade-off decisions are affected by ecological circumstances. Accordingly, natural selection should favour life-history traits that lead to the highest fitness outcome under certain ecological circumstances (Hill, 1993).

Although life-history theory research covers a variety of interesting fields the following introduction chapter will mainly pay attention towards human reproduction since the focus of this thesis lies on individual's trade-off decisions between current vs. future reproduction and sex-biased parental investment.

1.1.2. Human reproductive behaviour

Human reproduction is at the slow spectrum of the fast-slow continuum. This implies that we have a large body size, late onset of sexual maturation and reproduction, long gestation period, high intrauterine mortality, mostly singleton births, prolonged parental investment, and long interbirth intervals (Bielby et al., 2007; Placek and Quinlan, 2012; Wood, 1994). Due to various limiting factors, i.e. the time interval between menarche and marriage, the time interval between marriage and first birth, lactational amenorrhea and consecutive births, the maximal biological capacity is not achieved in any human societies (see Figure 1).

Human reproductive behaviour and trade-off decisions are generally influenced by numerous biological and social factors. The reproductive costs can be divided into three different categories: *i)* mating effort (mate choice and courtship), *ii)* parental investment (gestation, birth, lactation and child care) and, *iii)* nepotism (see Störmer and Voland, 2014, pp. 4-5). Bateman's principle provides the biological explanation for the first category (human mate choice). Bateman (1948) proposed that mean lifetime reproductive success (LRS) of both sexes has to be equal but the sex-differences are reflected in its variance. In most species males have a higher variance in their LRS and invest less energy in their reproduction in comparison to females, which have lower variance in their LRS, but their reproduction is more energetically costly (larger gametes, long gestation and lactation periods).

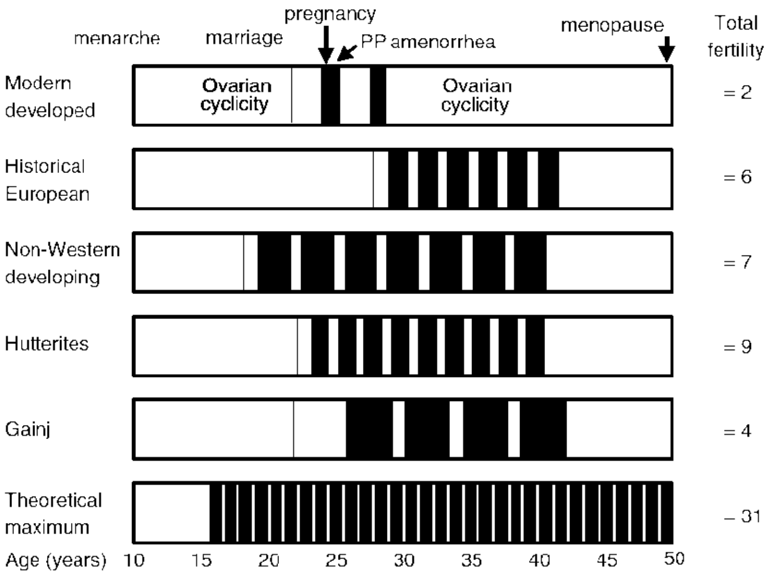

Figure 1. Life-history trajectories in different human populations (Worthman, 2003, p.292, modified from Wood 1994, which was based on Bongaarts and Potter 1983)

Based on these facts females benefit from being more selective in terms of mate choice, and thus, tend to select a partner based on his genetic quality but also on his ability to contribute to childcare (reviewed in Barrett et al., 2002, p. 94). Since human offspring have a long period of dependency, one fundamental trade-off decision is between the number of offspring (quantity) and the fitness of offspring (quality) (Hill, 1993; Hill and Kaplan, 1999). Parental investment (*e.g.* parental care – breastfeeding, provisioning of resources, protection) per offspring decreases with increasing number of offspring (Gillespie et al., 2008). By increasing the investment of the available resources per offspring parents can increase offspring's survival chances and offspring's fitness, hence offspring's quality (Hill, 1993; Kaplan and Bock, 2001; Trivers, 1972). By extending the interbirth interval (IBI) parents increase offspring's survival chances and fertility (Hill and Hurtado, 1996). By investing in offspring-embodied capital (*e.g.* strength, knowledge, skills and other abilities) parents ensure that their offspring is well-adapted to the environment and competitive (Hill and Kaplan, 1999; Kaplan and Bock, 2001). Limiting parental investment per offspring has a negative consequence on parental fitness due to the higher likelihood of low-quality offspring with reduced life span and lowered fertility (Hill, 1993). Waiting for a reliable partner and establishing a stable union before starting to reproduce will increase offspring's quality due to the higher amount of parental investment per offspring but waiting for a reliable partner, however, might have an impact on reproduction timing.

This leads to the second major trade-off decision in terms of reproduction: current versus future reproduction (Stearns, 1992). Postponing reproduction in a growing population theoretically reduces parental fitness due to the smaller contribution of the offspring to the gene pool of the future population (Hill, 1993). During times of resource scarcity delayed reproduction in favour of energy investment in increasing body size or storing extra fat, might be beneficial for future reproduction and increases the probability of survival of mother and child (Hill, 1993; Störmer and Voland, 2014).

1.2. Do early harsh environmental conditions induce early reproduction?

Stressful early life conditions such as harsh and unpredictable environmental circumstances can accelerate an individual's life-history. It has been suggested that humans are most sensitive to such stressful life conditions between zero and seven years of life (*e.g.* Belsky et al., 1991; Quinlan, 2010). During this so-called "sensitive period" individuals are more likely to adapt unconsciously to these predominant environmental conditions. This adaptation process is known as phenotypic plasticity and includes behavioural, physiological and morphological changes (Chisholm, 1993; Kuzawa and Bragg, 2012). Stressful early life conditions that can accelerate an individual's life-history are for instance high mortality/morbidity, insecure neighbourhoods, resource insecurity, low parental investment and a father's absence (*e.g.* Nettle et al., 2011; Quinlan, 2003; Sheppard and Sear, 2011; Störmer, 2011). The present thesis focuses on mortality as proxy for harsh environmental conditions. In general, mortality can be divided into *i)* intrinsic and *ii)* extrinsic mortality.

Intrinsic mortality causes in general include age-specific physical and functional degradation as well as diseases caused by genetic defects (Koopman et al., 2015; Störmer and Voland, 2014). Furthermore cancer, heart diseases and other degenerative diseases are defined as intrinsic mortality causes (Gurven and Fenelon, 2009).

Extrinsic mortality is considered as a result of environmental hazards such as infectious diseases, predation, war and famine (Carnes and Olshansky, 1997). Individuals cannot control these environmental hazards and escaping them by adapting their behaviour is mostly not feasible or very difficult for the majority of people (Gurven and Fenelon, 2009; Quinlan, 2007). Extrinsic mortality has an age-specific risk as well: certain types of infectious disease, such as measles, are more common among infants and children than adults. Famine related mortality has the strongest effect on infants, children and elder people (Störmer and Voland, 2014). Promislow and Harvey (1990) have shown that extrinsic mortality is one of the key driving factors behind accelerating an individual's life-history. Individuals adapt to a high extrinsic mortality by maturing and reproducing earlier in order to increase the likelihood of passing on their genes to the next generation (Nettle, 2010; Quinlan, 2010). Furthermore, extrinsic mortality has an influence on the trade-off decision between current/future offspring and quality/quantity of offspring. In an environment with high mortality it might be advantageous to favour current reproduction over future reproduction. The trade-off decision between current and future offspring in the light of high mortality is addressed in the present thesis (chapter 2).

Extrinsic mortality can be subdivided into *i)* local mortality (*e.g.* municipality level) and *ii)* family-level mortality (*e.g.* parental death and sibling death). Local famine related mortality as well as family-level mortality – measured as exposure to sibling death – was of particular interest in the present thesis (chapter 2 and 3).

1.2.1. Local level

As mentioned above, growing up in an environment with high mortality cues leads to an adaptive response in an individual's /or individuals' life-history strategy. If the total mortality at the local municipality level is high, natural selection should favour a faster life-history strategy, reflected in early maturation and reproduction (Ellis et al., 2009). Quinlan (2010) showed that women who were born when the local infant mortality rate was high tended to have a lower age at first birth. This was even more the case when local infant mortality was high at the age of maturation. Störmer (2011) investigated the impact of epidemic-related infant and child mortality (smallpox epidemics) on male and female life strategies in the 18th century Krummhörn population (East-Frisia, Germany). She found that exposure to high infant and child mortality during the sensitive time period in early childhood accelerated male's life histories – meaning that they started to reproduce earlier and fathered offspring with a lower probability of survival due to lower effort on mate selection.

Most of the research that investigates the impact of local mortality on individuals' reproductive behaviour focuses on local age-specific mortality, in particular infant mortality, as a sensitive measurement for environmental conditions (*e.g.* Ellis et al., 2009; Low et al., 2008; Quinlan, 2010). An attempt to take adult mortality (probability of dying between 15 and 60 years – WHO, 2017) into account and to differentiate between the effect of infant mortality and adult mortality on an individual's reproductive behaviour was made by Placek and Quinlan (2012). They found that both infant mortality and adult mortality rates during the sensitive period in early childhood (0-7 years) affect an individual's reproductive behaviour stronger than the infant and adult mortality rates during an individual's reproductive period. These findings support the conclusion that exposure to high mortality cues, whether it is infant or adult mortality, during the first seven years of life significantly influences human reproductive behaviour.

Chapter 3 of this thesis focuses on famine-related adult-juvenile mortality rates among a historical Flemish Brabant population (Belgium) and investigates the influence of high mortality cues within the first five years of life on an individuals' marriage behaviour. In the historical setting of the study (chapter 3) marriage and reproduction were strongly linked to each other (see chapter 1.3).

1.2.2. Family level

Family members not only share genetic factors with each other but also social and economic conditions (*e.g.* household resources, parental investment, household composition). Due to these common bio-demographic characteristics some families might be more vulnerable to high mortality than others (Mosley and Chen, 1984).

The majority of the literature focuses on the proximate determinants of death clustering within families. Proximate determinants that influence children's exposure to risk are: mother's age at birth, parity, interbirth intervals, marital status, mother's health, parental socioeconomic status and education (*e.g.* Das Gupta, 1990; Janssens and Pelzer, 2012; Sastry, 1997; Zaba and David, 1996). From the life-history point of view, the family environment a child is reared in is a good point of reference for its future environment. Störmer and Lummaa (2014) investigated whether siblings adapt their life-histories differently in terms of the number of deaths they experienced at the family level during the sensitive period in early childhood. They argued that family membership in terms of genetic predisposition and shared environment has a stronger influence on the adaption of an individual's life-history strategy than the individual-level mortality experience (*i.e.* number of siblings' death and its timing). Donrovich et al. (2018) found that women, in historical Antwerp (Belgium), who were exposed to sibling's death during childhood had a higher risk of infant mortality among their own offspring. They assumed that growing up in harsh environmental conditions such as a high mortality environment had a negative consequence on the individual's later health outcome. Poor general health conditions during woman's fertile years were associated with higher infant mortality. Individuals who were exposed to the death of a sibling do not only face a higher mortality risk but also respond to these circumstances by adapting their life-history strategies (Arulampalam and Bhalotra, 2006; Störmer and Lummaa, 2014). Exposure to high mortality within the natal family (parental death and/or siblings' death) promotes alternative behavioural strategies and accelerates individual's life histories (Störmer and Lummaa, 2014; Voland and Willführ, 2017).

Studies that investigated the influence of father absence due to death on female reproduction-related outcomes are more ambiguous. Shenk and Scelza (2012) found that paternal death during the sensitive period (0-7 years) has no influence on age at first birth but accelerates the progress to first marriage as well as the transition from marriage to giving birth. Exposure to paternal death during this sensitive period has been also found to accelerate sexual maturity (earlier age at menarche, early sexual debut) and to increase the likelihood of teen pregnancies, (*e.g.* Belsky et al., 2010; Nettle et al., 2011; Quinlan, 2010; Romans et al., 2003). Another study of a transitional population Shenk et al. (2013) found the opposite effect of father's death on the daughter's marriage and fertility behaviour: paternal death among Bangladeshi women was associated with older age at first marriage and first birth. Bereczkei and Csanaky (1996) revealed similar findings among Hungarian girls. In summary, the influence of mortality exposure in the natal family on a female's reproductive behaviour has been gaining scientific interest for several decades. Previous studies, however, reported rather contradictory results. To shed more light into the importance of mortality exposure within the natal family, chapter 2 of the present thesis uses a novel approach to investigate the effect of exposure to sibling death during early childhood (0-5 years) on female's reproductive behaviour outside a stable union in a pre-industrialized population in Germany.

1.3. Divergent reproductive trajectories: childbearing within a marriage versus illegitimate offspring

Among humans there are two common mating strategies *i)* a long-term mating strategy (*e.g.* marriage and common-law marriages), and *ii)* a short-term mating strategy (*e.g.* one-night stands, affairs and extra pair copulation).

In terms of resources, certainty and parental investment women will benefit more from investing in a long-term mating strategy (Buss and Schmitt, 1993). Resource certainty allows women to invest more in their offspring, therefore increasing the child's survival chances and maximizing their own fitness (Voland, 1988). Within the mainly historical context of the present thesis the long-term mating strategy is the most applicable strategy for the populations under observation. Nevertheless, even though short-term mating strategies were associated with social stigma and exclusion, some people favoured them over the safer long-term mating strategy.

The present thesis aims to address the impact of exposure to high mortality (at the local and family level) on an individual's mating strategy (chapter 2 and 3).

1.3.1. Marriage

In most human societies a formal form of a long-term mating strategy (*e.g.* marriage and common-law marriages) exists and most individuals will enter this stage at one point in their lives (Buss and Schmitt, 1993). A long-term relationship assures *i)* semi-permanent or permanent access to sex, *ii)* support during pregnancy, lactation and child rearing, and *iii)* legitimate status of offspring (Buss and Schmitt, 1993; Daly and Wilson, 1988, p. 187).

Among human societies marriage behaviour is strongly driven by social and cultural norms. In many societies one of these norms is that reproduction was/is restricted to married people. In all societies mentioned there is a varying time gap between sexual maturity, marriage and the onset of reproduction (see Figure 1 – Wood, 1994). Among historical European populations this time between sexual maturity and marriage is known as the "north-western European marriage pattern" (Hajnal, 1965). Due to a high average age at first marriage (female average age at first marriage: 25 to 26 years; male average age at first marriage: 27 to 28 years – Hubbard, 1983, p. 18) the gap between sexual maturity and marriage was the largest among historical north-western European societies. It was also not uncommon in these societies that a high number of individuals remained unmarried throughout their lives (Hajnal, 1965, p. 135). There are various explanations for the high average age at first marriage. Individuals who wanted to enter marriage had to meet specific criteria such as a certain age, consent of the parents, ability to establish an independent household (Kraus, 1979). Furthermore, a man had to establish an independent livelihood before marriage to ensure that he could support his future family (Hajnal, 1965, p. 133) to enter marriage. To afford a wedding and establish an independent own household (referred to in the literature as "neolocal household" – Hajnal, 1982; Puschmann and Solli, 2014) individuals who were not designated heirs had to work in domestic service to earn and save money.

In pre-industrialized European societies individuals lived together in family-like domestic groups. Any potential marriage had to meet the social and economic needs of the domestic group to which the individual was part of. These groups were heavily dependent on marital fertility, since children were an active part of the labour force, especially during old age, due to the lack of a social security system (Mitterauer and Sieder, 1982, p. 122). To ensure the fertility of a marriage so called "trial marriages" were common during that time period. This marriage-like union allowed men and women to start courtship and sexual intercourse to test the fruitfulness of the union. In case of the occurrence of a prenuptial pregnancy during the "trial marriage" it was the common understanding that the man had to marry the woman. If the union was not fruitful and the woman did not get pregnant the man could break up the courtship (Gray and Anderson, 2010, p. 57; Mitterauer and Sieder, 1982, pp. 121-122). This common practice reflects in the high premarital conception rate for first born children in pre-industrial Europe (Wood, 1994). Another phenomenon during that time period was a so-called "shotgun wedding". A "shotgun wedding" occurred when pregnancy as well as marriage were not intended but the involved parties were forced to get married to prevent the social stigma of an illegitimate child (Laslett et al., 1980, p. 8).

The populations under observation in the present thesis (chapter 2 and 3) were among those countries in the "north-western European marriage pattern" and therefore had a high average age at first marriage (Lesthaeghe, 2015; Willführ and Störmer, 2015). European historical populations are a good model for investigating life-history theory since cultural norms for late age at marriage existed and reproduction was strongly linked to marriage. Not only social and cultural norms influenced individuals' marriage behaviour and timing, but ecological circumstances also had an effect on it. Most of the previous research that investigated the impact of harsh ecological conditions such as high mortality on marriage behaviour focused either on family mortality within historical context or on contemporary data, where marriage restrictions were not that strict or not present at all. The present thesis therefore put on the task to investigate the impact of famine-related high mortality on individuals' marriage behaviour at a time when marriage had to meet certain social and cultural norms, and therefore was not possible for everyone.

1.3.2. Illegitimate births

As mentioned before, giving birth to an illegitimate child was associated with social stigma and exclusion. Nevertheless, there was a steep increase of illegitimate births in most of the central and north-western European countries between the end of the 18th century and the middle of the 19th century (Hopf, 1994 pp. 8-9; Knodel, 2002, p. 193; Kok, 1990). By definition an illegitimate child is a child that was born to parents who were not married at the time of the child's birth. From a biological point of view, giving birth to an illegitimate child was a costly and risky reproductive strategy for a woman within historical context. The mother was mostly facing resource uncertainty since there was no guarantee that the father would provide resources and support in childrearing. Furthermore, illegitimate children were 40% more likely to die before their first birthday in comparison to legitimate children due to social stigmatization and resource uncertainty (Brändström, 1996; Gardarsdóttir, 2000; Kok, van Poppel, & Kruse, 1997). Since the 1970s researchers try to find a possible explanation for the rise of illegitimacy during the 18th and 19th centuries. Mitterauer (1983) and Kok (2005) reported a positive relation between the prevalence of high illegitimacy and the north-western European marriage pattern. In Mitterauer's (1983) point of view these similarities are due to the high proportion of domestic servants and the associated marriage restrictions among this group that might further lead to a social acceptance of premarital intercourse. The illegitimacy rate within the group of domestic servants, day labourers, journeymen and people from the low socioeconomic classes was high (Matthys, 2016; Reid et al., 2006; Schumacher et al., 2007). As abovementioned (chapter 1.3.1), working as a domestic servant in a stranger's house was a way of earning and saving money to establish an independent household often led to a marriage delay (Szołtysek, 2009). Working as a domestic servant gave women the opportunity to gain experience in household tasks and childcare. Men could enlarge their professional experience through their working experience gained from the households of strangers (Szołtysek, 2009). By law individuals who were working in the domestic sector were prohibited from getting married, but they did not necessarily abandon sexual intercourse that might lead to an illegitimate child (Mitterauer, 1983, p. 50; Mitterauer and Sieder, 1982, pp. 123-127).

A heavily criticised explanation for the increase of the illegitimacy rate was given by Shorter (1975). In his opinion the rise of illegitimate births was due to an ongoing "sexual revolution". He argued that changes in the labour market and access to education promoted women's emancipation. He further argued that women broke with the traditional norms regarding premarital intercourses and changed their mating behaviour accordingly. He was strongly criticised by fellow researchers (*e.g.* Lee, 1977; Mitterauer, 1983, pp. 86-90; Tilly et al., 1976) who attributed the rise of illegitimate births to economic changes due to urbanization and industrialization rather than to an ongoing "sexual revolution".

Laslett (1980) postulated that most of the illegitimate children were mothered by a few bastard-producing women, who were related to each other by kinship and marriage. His concept is known as "bastardy prone sub-society". He assumed that this group of women passed on their sexual behaviour to the next generation. Furthermore he subdivided the so-called "bastard-bearers" into three different types: *i)* "repeaters" – mothers who gave birth to more than one illegitimate child, *ii)* "singletons" – mothers who only gave birth to one illegitimate child and were related to a "bastard-bearer", and *iii)* "sparrows" – mothers who only gave birth to one illegitimate child and had no connection to any other "bastard-bearer" (see Laslett, 1980).

While the topic of illegitimacy is well studied in historical demography and family history, the present thesis (chapter 2) takes the novel approach to apply life-history theory as an explanatory paradigm to this phenomenon. As mentioned in chapter 1.2 individuals adjust their life-history strategy according to their rearing environment. If the rearing environment is a harsh one, with for instance high mortality cues, individuals might devaluate the future and therefore invest in more risky sexual behaviour to complete at least one reproductive event before they die (Hill et al., 1997). Within the historical context of chapter 2, giving birth out of wedlock can be used as a proxy for risky reproductive behaviour because it is associated with social stigmatization and higher infant mortality. To further investigate if more promiscuous behaviour was clustered within certain families, as argued by Laslett (1980), a family fixed effect Cox regression (comparing sisters with each other) was used.

1.4. Sex-biased parental investment and socioeconomic status

As mentioned before (see chapter 1.1.2), there is a fundamental trade-off decision in terms of reproduction between quantity and quality of offspring. By investing in offspring's quality parents increase the probability of survival to adulthood and successful reproduction of their offspring (Kaplan and Bock, 2001). Parental investment in offspring-embodied capital (knowledge, skills, education and other abilities) does not only affect offspring's survival chances but also offspring's social status and, as a result of that, offspring's LRS (Kaplan and Bock, 2001). Status in contemporary societies is often associated with educational and occupational attainment, occupational prestige and income. Parental status and/or wealth does not only influence the number of offspring but also the offspring's condition which in turn influences the parents' fitness through the number and quality of grandchildren (Barrett et al., 2002, p. 138). Biasing parental investment towards the sex with a higher LRS therefore should be beneficial in terms of fitness.

According to Fisher (1930) parents should favour their investment towards the sex of offspring that in return are more likely to produce more offspring itself. Trivers and Willard (1973) further elaborated on Fisher's concept and predicted that parents' sex-bias their parental investment depending on their position within the social and economic structure. Their concept is known as the Trivers-Willard (T-W) hypothesis (Trivers and Willard, 1973). Among humans, socioeconomic status is a proxy for parental position within a society. Socioeconomic status is often measured by income, educational attainment and occupational prestige (Dutton and Levine, 1989). Other research used the Duncan socioeconomic index (SEI) as a measure of socioeconomic status (Blau and Duncan, 1967; Hopcroft, 2005). Trivers and Willard (1973) assumed that the parental position within the society and the offspring's position within society should be positively correlated with each other. This assumption holds for human societies, where the intergenerational social mobility is quite stable (*e.g.* Luo et al., 2016; Van Bavel, 2006). In the course of human evolution, the ways in which men indirectly invested – mostly via provisioning – in childcare changed. In modern industrialized and post-industrialized societies, men mostly contribute to childcare indirectly through their wealth and/or status. By contrast, in hunter-gatherer societies and in agricultural societies, men contributed indirectly through their hunting skills or landownership (reviewed in Barrett et al., 2002, p. 94). Among ancient as well as contemporary societies high status and/or wealth of men was associated with higher access to resources and therefore with a higher LRS (Betzig, 1986; Fieder and Huber, 2012; Morris and Scheidel, 2009; Nettle and Pollet, 2008). But in women high status and/or wealth often has a null or negative relation with LRS (Fieder and Huber, 2007; Fieder et al., 2005).

Regarding sex-biased parental investment, many studies in different species tested the Trivers-Willard hypothesis. The results among human societies are ambiguous. Voland et al. (1997) found in the Krummhörn population of the 18th and 19th century (population under investigation in chapter 2 of the present thesis) that low class families channelled their parental investment towards daughters. Studies that investigated the T-W hypothesis in contemporary industrial societies focused more on breastfeeding, time spent per week, and educational attainment, than on infant survival. These studies found that contemporary US low status families invest more in the educational attainment of their daughters than of their sons. It is the opposite among high-class families where parental investment favours sons (Hopcroft, 2005; Hopcroft and Martin, 2014, 2016). In modern western societies educational attainment can be seen as prerequisite for status attainment (Kaplan, 1996) and often influences the later income of the offspring. Numerous studies have shown that educational attainment and income are transmitted from one generation to another (*e.g.* Hauser et al., 1973; Solon, 1992). The transmission of income from one generation to the next is known as intergenerational income mobility. Previous studies have shown that income elasticity was higher among sons than among daughters in Western industrialized societies (Chadwick and Solon, 2002; Shea, 1997; Solon, 1992). This sex difference might be due to the fact that women had limited access to the labour force than men. Most of the studies have focused on the intergenerational transmission of income and educational attainment from fathers to sons. The intergenerational transmission of income and educational attainment from father to daughters and from mothers to both, sons and daughters, is widely neglected. Chapter 4 of the present thesis aims to close this gap in the current literature by studying the effect of sex-biased parental investment on offspring's status attainment measured by income and educational attainment in the contemporary US.

Chapter 2

Effects of individual mortality experience on out of wedlock fertility in 18th and 19th century Krummhörn, Germany

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Effects of individual mortality experience on out of wedlock fertility in 18th and 19th century Krummhörn, Germany

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Abstract

Life history theory predicts that exposure to high mortality in early childhood leads to faster and riskier reproductive strategies. Individuals who grew up in a high mortality regime will not overly wait until they find a suitable partner and form a stable union as premature death might prevent them from reproducing at all. Cox proportional hazard models were used to determine whether women who experienced sibling death during early childhood (0-5 years) reproduced earlier and were at an increased risk of giving birth to an illegitimate child, whereby illegitimacy serves as a proxy for risky sexual behavior. Furthermore, we investigate if giving birth out of wedlock is influenced by individual mortality experience or by more promiscuous sexual behavior that is clustered in certain families. Models are fitted on pedigree data from the 18th and 19th century Krummhörn population in Germany. The results show that there is a relationship between sibling death in early childhood and the risk of reproducing out of wedlock, and the reproductive timing. The risk of giving birth out of wedlock is linked to individual mortality experience rather than to family-level effects. In contrast, adjustments in connubial reproductive timing are influenced more by family-level effects than by the individual mortality experience.

Keyword: life history theory, reproductive timing, illegitimacy, mortality, Krummhörn

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1. Introduction

Until the middle of the 20th century, childbearing in most European countries was strongly linked with marriage, which marked the onset of sexual reproduction. In Western Europe, marriage occurred well after biological maturation due to culturally imposed norms such as the economic self-sufficiency of newlyweds (Engelen and Wolf, 2005; Hajnal, 1965). Although there was a considerable time-lag between physical maturation and age at first marriage, out of wedlock fertility was rather low in pre-modern times, as giving birth to an illegitimate child was associated with negative consequences for both the mother and the child (Laslett et al., 1980; Mitterauer, 1983). Besides the social stigma, fathers of illegitimate children were often absent, mostly invested few or no resources in their children's upbringing and infant mortality was higher than among legitimate children (Gardarsdóttir, 2000). Hence, in the historical context giving birth out of wedlock was a risky reproductive strategy.

However, roughly between the end of the 18th to the middle of the 19th century, European countries experienced a considerable and, in some cases, even a dramatic increase of illegitimate births. The phenomenon was mainly found among the working classes, and although certain cities, such as Stockholm and Vienna, reached very high levels of out of wedlock fertility, many rural areas were also affected. Several potential explanations have been put forward in the literature for the rise in illegitimacy. While Shorter (1975) interpreted it as the result of an early sexual revolution, Tilly et al. (1976) argued that men more often broke marriage promises due to economic challenges posed by early-industrialization and urbanization. Laslett (1980) found that many of the women who gave birth to children out of wedlock were linked to each other and were so-called 'repeaters', i.e. they gave birth to multiple illegitimate children, who were themselves at an increased risk of becoming the parents of illegitimate children. He put forward that these women and men were part of a bastardy-prone sub-society and stated that within this sub-population social norms regarding sexuality and marriage deviated from larger society. Kok (2005) by contrast, saw the rise in out of wedlock fertility as a consequence of the breakdown of social control systems in the wake of urbanization and industrialization.

While numerous scholars have attempted to explain (the rise in) out of wedlock fertility from a historical perspective, taking the agency of historical actors as well as changing contexts into account, socio-biological perspectives are missing. In this regard, little is known about the impact of exposure to high mortality during earlier childhood on the risk of giving birth out of wedlock. Life history theory predicts that mortality experiences in early childhood lead to faster and riskier life strategies and such mortality experiences have indeed been shown to lower the age at first birth, both in preindustrial and contemporary societies (Nettle et al., 2011; Placek and Quinlan, 2012; Quinlan, 2010; Störmer, 2011). There are, accordingly, good reasons to test whether life history theory predictions also hold in the case of culturally imposed restrictions of reproduction to marriage. Does exposure to high mortality lead to a higher risk of engaging in premarital intercourse, resulting in an increased risk of giving birth to an illegitimate child? Can peaks in exposure to high mortality in early childhood, in other words, add a further explanation to rises in illegitimate births in eighteenth- and nineteenth-century Europe?

2. A Life History Approach to Out of Wedlock Fertility

During its life cycle an organism harvests energy from the environment and makes certain trade-off decisions regarding the investment of resources in terms of age-specific functions such as growth, maintenance and reproduction (Roff, 2002; Stearns, 1992). Life history theory posits that these trade-off decisions are influenced by local ecological circumstances (Schaffer, 1983). The level of extrinsic mortality is one important ecological factor that influences the timing of certain life events (Promislow and Harvey, 1990). Extrinsic mortality is a result of environmental hazards such as infectious diseases, predation, war, famine or accidents and individuals cannot escape or control it by behavioral change (Gurven and Fenelon, 2009; Quinlan, 2007). It acts as environmental harshness or as environmental unpredictability (Ellis et al., 2009). Environmental harshness varies spatio-temporally and is largely inevitable. Environmental unpredictability is the random variation of environmental harshness under which the success of behavioral adaptation is arbitrary (Kavanagh and Kahl, 2016). In unpredictable environments the future is uncertain, devaluated and long-term investment might not pay off due to premature death. Hence individuals might shift their time perspective from future-orientated to present-orientated (Brumbach et al., 2009) and adapt their behavior accordingly (Adams and Nettle, 2009): Life is here and now. Present-orientated individuals devaluate the future and pursue high-risk strategies in favor of short-term gains. They invest less in growth and maintenance and shift their resources towards an early maturation and reproduction. A faster life history strategy with an early onset of reproduction to obtain an optimal fitness maximization strategy could be seen as an adaptive strategy to beat the odds of not reproducing at all in a harsh and unpredictable environment where life expectancy is reduced (Anderson, 2010; Griskevicius et al., 2011; Low et al., 2008; Wilson and Daly, 1997). Previous studies in contemporary societies have shown that exposure to high mortality during early childhood (0-7 years) can trigger fast life history strategies characterized by earlier age of menarche, first pregnancy and birth (Belsky et al., 2010; Chisholm et al., 2005; Nettle, 2010; Nettle et al., 2011; Quinlan, 2010). Furthermore, it is well documented that earlier age of menarche is strongly associated with earlier age at first date, first kiss and risky sexual behavior such as earlier age at first sexual intercourse (Hoier, 2003; Lam et al., 2002) and a higher prevalence of teenage pregnancies (Nettle et al., 2011; Romans, Martin, Gendall, and Herbison, 2003). The early onset of reproduction in turn entails risks such as higher neonatal and post-neonatal mortality, and a higher likelihood of giving birth to a low birth weight, premature, and small-for-gestational-age infants (Fraser et al., 1995; Olausson et al.,1999). Despite these risks, in high mortality environments a faster life strategy (faster maturation and early reproduction) reduces the risk of death prior to producing offspring (Brumbach et al., 2009).

3. Objectives

The present study uses pre-industrial historical longitudinal data from the Krummhörn region in North-West Germany from the 18th and 19th centuries to study the effects of high mortality as measured by exposure to sibling death on young women's risk of bearing an illegitimate child. The rearing environment is an important point of reference for future environmental conditions an individual might have to face. Focusing on exposure to sibling death instead of measuring mortality on a higher level of aggregation allows us to investigate the influence of the family environment on the development of an individual's life history strategy. Siblings are not exposed to the same environmental cues due to the timing of certain events and their birth rank. Störmer and Lummaa (2014) pointed out the importance of family environment rather than individual mortality experience. They hypothesize that some kind of 'family mentality' is operating: parental behavior is stress sensitive and loss of a child will have an impact on it. In line with Chisholm (1993) they argue that the surviving offspring is confronted with an unpredictable environment and therefore adapt their life history strategies accordingly. Furthermore, we address whether shared family environment (genetic predisposition (Barban et al., 2016) and heritability of fertility traits, such as age at first reproduction (Bolund et al., 2013; Milot et al., 2011)) or individual mortality experience within the natal family, trigger riskier and faster life histories of women.

4. Data & Methods

4.1 Study Population and Period

Our data are derived from a family reconstitution dataset based on Protestant church registers and tax rolls of the Krummhörn region in East Frisia (Germany) from the 18th and 19th centuries (for a comprehensive description of the database and the methodology see Voland, 2000). The historical Krummhörn was divided into 33 neighboring parishes, all of which are included in the dataset. The dataset contains 118,778 individuals who were in 34,708 marriages. It is archived at the GESIS-Institute (Cologne) with the label *ZA8630*. Many of the records dated before 1720 are incomplete, and families from the social and economic elite tend to be overrepresented in these early records. After 1874, the church was no longer responsible for maintaining records of births, deaths, and marriages, as this task had been transferred to the civil registry offices ("Standesämter"). Due to the bias in the early records and the censoring after 1874, we limit our analysis to females who were born between 1720 and 1850 (N= 25,487) and do not include events after 31th December 1874 in our analyses. From this sample, 11,874 women are censored before possible childbirth, since either they migrated out of the study area as bachelors or they did not become mothers before 1874. We initially included women who were born out of wedlock and who were never legitimized. The information about their context of origin was included via a dummy variable in the models (results not shown). Since only few women who were born out of wedlock reproduced and results only differed marginally, we do not include those women in our final analyses. Our final sample contains 8,339 women who gave birth to their first children within marriage and 379 women who gave birth to their first child out of wedlock and never married the father of this child.

Geographically, the peripheral rural region of the Krummhörn is bordered to the north and west by the North Sea; to the south by the River Ems; and to the east by sandy soil and moorlands, which were impenetrable at that time. The Krummhörn region itself had very fertile marsh soil that was suitable for raising both crops and livestock. In the late medieval period, the settlement of the Krummhörn region had been completed (Ohling, 1963), and there was no significant population growth during the study period (Klöpper, 1949). As the region was a saturated habitat with a finite amount of arable land, the population faced local resource competition (Voland and Dunbar, 1995). Due to the limited access to land, a stratified social structure arose among the Krummhörn population. The large-scale farmers with capital and status were at the top of this social hierarchy, while the small-scale farmers, craftsmen, and landless workers occupied the lower end of this social structure. In the 18th century about 70 percent of the Krummhörn's families had either no land at all or their farms were too small to ensure subsistence, and thus were forced to supplement their income by working for the large-scale farmers (Willführ and Störmer, 2015). Although there are no records indicating that the region was affected by famine or war during the study period, as in other parts of Europe, smallpox and other infectious diseases took a significant toll on the people of the region over the course of the 18th century (Omran, 2005). The average family size was about four children (Voland and Dunbar, 1995; Willführ and Störmer, 2015). The families of the region practiced a form of ultimogeniture in which the youngest son inherited the undivided farm from the father and the other offspring had to be compensated, often with cash (Ohling, 1963). A daughter could expect to receive half of the amount of compensation each son received. Due to these inheritance practices, families in the Krummhörn region tended to be relatively small and the average age at first marriage was high (female average age at first marriage: 26.285 (± 5.406) – Willführ and Störmer, 2015). Thus, late reproduction and low birth rates were the norm.

4.2 Modeling the risk to give birth out of wedlock

We use Cox regression (Allison, 2014; Cox, 1972) to model the life course of reproductive females, starting from birth up to the age of first childbirth. Reproductive women in this context means that these women gave birth at least once in their lifetime regardless whether the children were born within marriages or out of wedlock. As mentioned above, we use the event of giving birth out of wedlock as a proxy for increased risk-taking behavior. The traditional definition of giving birth out of wedlock refers to all non-marital births (Laslett et al., 1980). This definition, however, might fall too short in the case of the Krummhörn region, because it was not uncommon that couples married after conception or shortly after birth of the first child. Such a reproductive behavior does not necessarily reflect increased risk-taking, but structural factors such as marriage bans during the harvest season or during religious holidays. We therefore stick to a strict definition: Born out of wedlock means that the woman never married the biological father.

In estimating the effects of sibling mortality experience, we rely on a combination of models adjusted for clustering at the family level, and models stratified at the maternal level (family fixed effects see Allison, 2009). The former models investigate the general association between mortality experience and the risk of giving birth out of wedlock. The latter models estimate likelihood functions with separate terms for each of the families in the dataset, and thus allow each family to have their own individual baseline hazard function. The key difference between the stratified and the clustered Cox regression models is that the stratified models identify the effect of mortality experience using the variation within families, but not between families. These stratified models control for unobserved heterogeneity, *e.g.* genetic and environmental factors if these were shared by sisters. By comparing the results of the clustered with the results of the stratified models we try to assess the research question whether the risk of giving birth out of wedlock is affected by individual mortality exposure or whether mortality and more promiscuous sexual behavior is clustered in certain families. The flaw of models which are stratified on the family level is that singlets (women without sisters) are excluded from the analysis. Therefore, if the results of a clustered model version are not in line with the results of the corresponding stratified version, one needs to check if the different results are due to difference in the sample size or due to differences in the hazard function estimation. A similar approach was used by Fox et al. (2017) to study whether having siblings affects mortality and reproductive success and by Willführ et al. (2018) to study kin effects on the mortality of reproductive women.

The time-varying information about early sibling death experience are included as dummy variables in the models. It should be emphasized that we only consider deaths of siblings which occurred during childhood of the individuals of interest. Sibling deaths before birth or after the 15th birthday are not considered in the analysis. To investigate whether an alleged sibling mortality experience effect is age-specific, we varied the age range in which we consider sibling deaths. Life History Theory and the theory of evolutionary socialization predicts that mortality experience is not equally effective over the juvenile period. We expect to find "sensitive windows" where the loss of siblings is most effective. Therefore, we decided to employ a model in which sibling death experience before the age of 15 is coded in three variables I) sibling death experience between birth and the age of five, II) sibling death experience between the age of five and ten, and III) sibling death experience between the age of ten and 15. These age categories are more or less arbitrary and we test also other coding for sibling death experience. For instance, we use other age categories and we limit sibling deaths experience until the age of seven. The results of these models are attached to appendix of this paper. In order to control for potential confounders, we include time-varying information about the number of siblings alive (as a measurement for current family size) and whether the father or mother of the individual had deceased. Each change in one of these covariates is an event, which brings a new episode of observation to the model.

We further include birth order, birth cohort and the parental socioeconomic status (SES) as time-invariant control variables in the models. The possibility to experience sibling death is dependent on the individual's birth order and on sibling size, since, for example, firstborns are not exposed to sibling mortality until the parents have a second child. Females birth cohort is coded in decades and is included as control for time trends. All cases are categorized into five groups based on their parents' land ownership status. Families who owned more than 75 *grasen* are classified as "large-scale farmers", families who owned between 10 and 75 *grasen* are assigned to the "mid-scale farmers" group, while families who owned less than 10 *grasen* are categorized as "small-scale farmers." Families who had no land property are classified as "landless," and families for whom the level of land ownership was unknown are placed in the "unknown" group. The borders between these categories are more or less arbitrary, but fit well into the historical context (Beise, 2001; Willführ and Störmer, 2015). All analyses were performed in R-3.5.3 with the help of the following packages: data.table, reshape, and Hmisc (includes survival), broom, and ggplot2.

4.3. Limitations

Due to substantial migration of young adults out of the study area, the death dates of many individuals are missing. This might pose a problem for the current study design. We can assume that children survived to adulthood if the parents' marriage was under observation. This criterion is fulfilled if the start and end of the marriage are recorded, and we only include individuals who derived from such families. We therefore can assume survival of siblings up to the age of 15 if their date of death is missing. This procedure might result in a systematic underestimation of sibling death. However, infant and child survival estimates which are based on this selection criterion are in line with estimates that *e.g.*. are based on census data. We believe that this potential underestimation is not interfering with our research questions, as we might only face the problem of false negative results, but not of false positive.

5. Results

Consistent with previous literature (*e.g.* Laslett et al., 1980; Mitterauer, 1983; Shorter, 1975) we find that the proportion of illegitimate births started to increase at end of the 18th century (Figure 1). This trend was constantly increasing until the end of the study period in 1874.

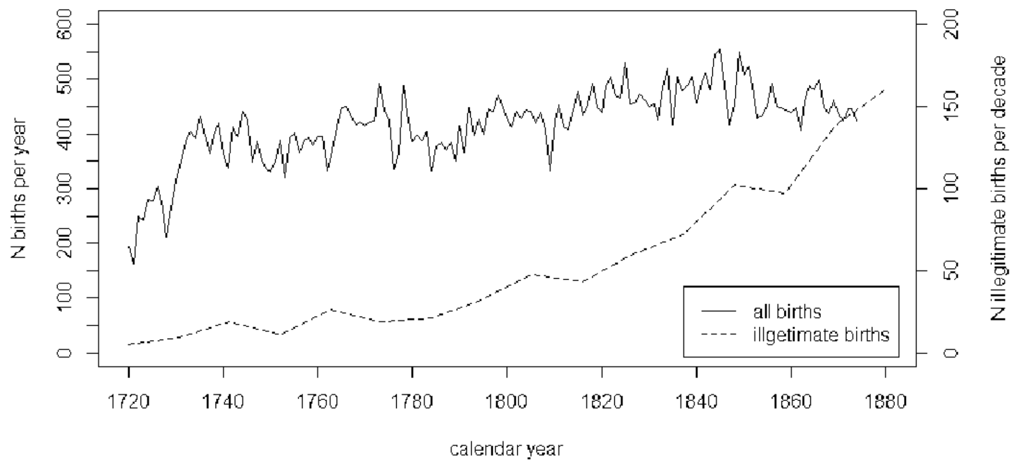

Figure 1 – Number of births and proportion of illegitimate births per calendar year for the Krummhörn population (Ostfriesland, Germany).

The results of the Cox regression, which estimate the effect of sibling mortality experience on the risk of giving birth out of wedlock, are summarized in Figure 2. The full models including test for proportional hazard assumption are given in section A1 in the appendix. We find that sibling death experience before the age of five is statistically significant associated with an increased risk to give birth out of wedlock. The impact of sibling death experience before the age of five is detectable in the clustered as well as in the fixed-effect model versions, which indicates that this association is driven by individual experience and is not due to unobserved family shared characteristics, such as genes and environmental factors. Sibling death experience between the age of five and ten as well as between ten and 15 does statistically not significantly affect the risk to give birth out of wedlock.

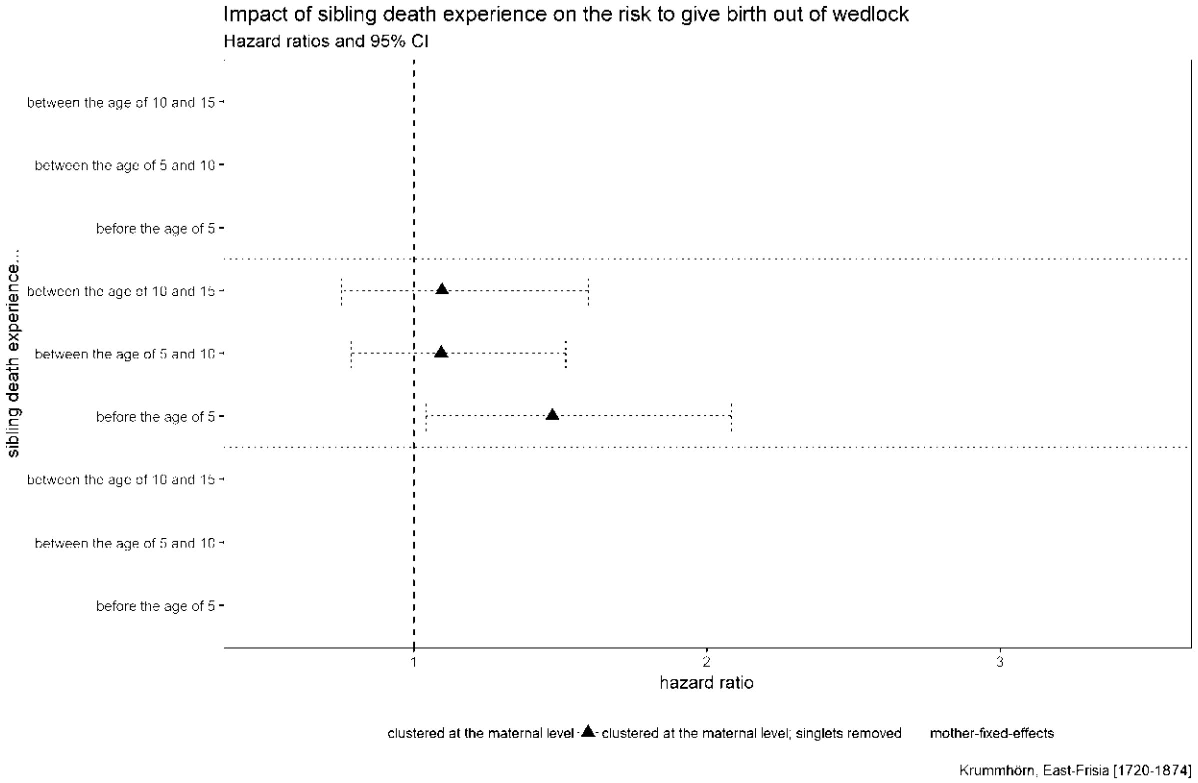

Figure 2 – Results of the Cox regression modeling the risk of giving birth out of wedlock. Models control for number of siblings alive, whether father or mother has died, child's birth cohort (in decades), child's birth order, and parent's socioeconomic status (omitted in the mother-fixed-effect versions). Results of the full models, including tests for proportional hazard assumption, are given in the section A1.1 in the appendix.

The results of the Cox regression, which estimate the effect of sibling mortality experience on timing of first childbirth, are given in Figure 3. The full models, including tests for proportional hazard assumption, are given in section A2 in the appendix. Like the model estimating the risk to give birth out of wedlock, the clustered model version estimating the time to first childbirth suggests that sibling death experience before the age of five statistically is significantly associated with earlier childbirth. The association, however, is not suggested by fixed-effect and by the clustered model version which excludes singlets (reproductive females without a reproductive sister in the data). This is an indication that the association is explained by unobserved family shared characteristics or by factors which are absent in single daughter families.

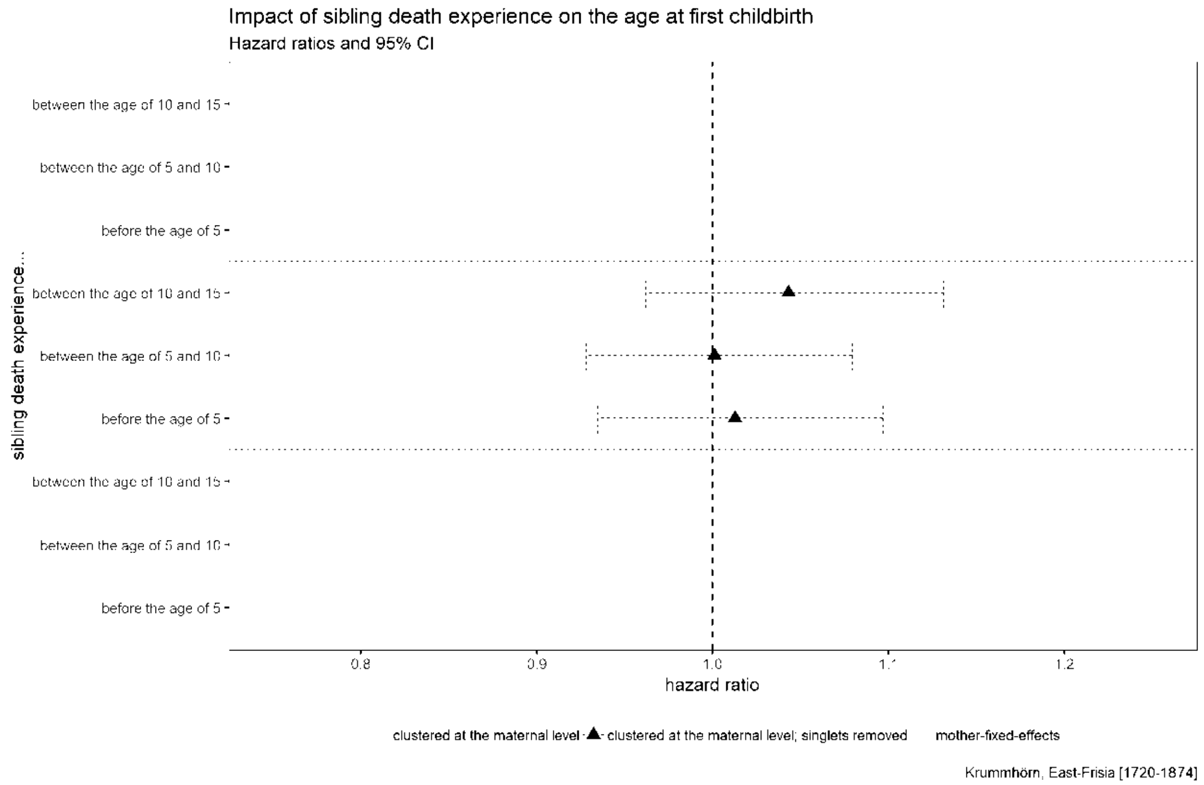

Figure 3 – Results of the Cox regression modeling time to first childbirth. Models control for number of siblings alive, whether father or mother has died, child's birth cohort (in decades), child's birth order, and parent's socioeconomic status (omitted in the mother-fixed-effect versions). Results of the full models, including tests for proportional hazard assumption, are given in the section A1.1 in the appendix.

To test, whether the association between sibling death experience in childhood and the risk of giving birth out of wedlock may be driven by confounding factors, we ran several robustness checks which all are presented in the appendix. Firstly, we rerun our models with different categories of sibling death experience. We test the effect of sibling loss before the age of seven (A3) and 15 (A4). We also run models where we include the categories sibling death before the age of three, between the age of three and seven, and between the age of seven and 15 (A6). The results only differ marginally.

Secondly, we investigate whether the effects are explained by the intensity of sibling death experience or by the incidence that there is any sibling death experience. In section A5 we (re-)transform the dichotomous variables coding for count variables which codes the numbers of deceased siblings. The results of the models with these modified variables are comparable with the models in Figure 2. Thirdly, we are interested in the raw effects of the variable of interests (estimated in models without controls, see A7).

6. Discussion

Most studies put great emphasis on the importance of individual and historical context – industrialization, urbanization and its economic and social effects – in explaining the rise of illegitimate births in Europe in the 18th and 19th centuries (Mitterauer, 1983; Schumacher et al., 2007). Our study incorporates life history theory as additional explanatory factor. We assume that individuals experiencing high mortality (experiencing sibling death) pursue risky reproductive strategies. Risky in this context means, that they give birth at an earlier age and have more illegitimate children, as they start having sex before a stable relationship is established and a marriage tie is knot. In line with previous research (Störmer and Lummaa, 2014; Voland and Willführ, 2017) we distinguish between the impact of individual mortality experiences within the natal family and family-level effects (shared environment/genetic predisposition). The results obtained in this study show indeed that women who witnessed the death of one or more of their siblings in early childhood (0-5 years) were more likely to give birth out of wedlock. Our results stress the importance of the individual early life mortality experience (*i.e.*, the number of siblings that died is related with the tendency to give birth out of wedlock) rather than family membership in pursuing a riskier reproductive behavior. Changing one's life history strategy might be due to a shift in an individual's time perspective. Under harsh environmental circumstances, such as high mortality, individuals might benefit from a present-orientated perspective. A present-orientated individual is devaluating the future and therefore is assumed to invest more in riskier behavior resulting in short-term rewards (*e.g.* sexual intercourse and sexual pleasure) than in future-orientated behaviors that require long-term planning and investments (*e.g.* courtship, establish a stable union and an independent household - Boyd and Zimbardo, 2005; Schechter and Francis, 2010). Even when individuals have to cope with resource uncertainty, high child mortality, and the risk of being stigmatized and socially excluded (Brändström, 1996; Gardarsdóttir, 2000; Kok et al., 1997; Laslett et al., 1980), the short-term reward of a reduced risk aversion and therefore diminished self-control might outweigh such drawbacks. In the face of adversities and unpredictability completing one reproductive event might be a successful life history strategy, as it leads to reproduction before death, while long-term strategies might fail due to early death, resulting in no offspring at all.

Apart from the tendency to reproduce out of wedlock, our results also indicate that women who experienced the deaths of siblings during early childhood (*i.e.*, 0-5 years) started reproduction earlier. In contrast to the results on out of wedlock fertility, family membership is revealed to be more important than the individual mortality experience, with respect to timing of first birth. This is in line with previous research of Störmer and Lummaa (2014). Using data from three different historical populations (Finland, Quebec and Krummhörn) they were able to demonstrate that the family environment is more important for modifying reproductive timing than the individual mortality experience within the natal family. Assumedly, parents respond to the loss of a child in a high mortality environment by changing their parenting behavior and thereby creating an uncertain environment for the surviving offspring who themselves are responding by adapting their life histories (Belsky et al., 1991; Chisholm, 1993).

We also examine the time-dependent effect of exposure to sibling deaths with interactions on the likelihood of giving birth to an illegitimate child and age at first birth. Our results show that mortality exposure is interacting with time, more precisely with the stage of the ontogenetic development, in the Krummhörn population. Thus, mortality exposure in early life affects reproductive timing and decision making to a greater degree than in later phases of childhood. It has already been demonstrated that children who experience high mortality tend to develop insecure attachment styles (Chisholm, 1993). Insecure attachment is associated with faster development, loose bonds, present-oriented and riskier behavior, low parental effort and low mating effort (Ivan and Bereczkei, 2006; O'Connor et al., 1999; Schechter and Francis, 2010).

In sum, a growing body of research suggests an important role of mortality in shaping human reproductive strategies. The current study indicates that mortality in the form of exposure to sibling death during early childhood is significantly associated with the risk of giving birth out of wedlock in the eighteenth- and nineteenth-century Krummhörn population. It underlines the importance of life history theory in explaining reproductive behavior of individuals in general and the timing of the onset of reproduction as well as the likelihood of giving birth out of wedlock in particular.

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APPENDIX

A1-Full models for tabel 2

```
Call:
coxph(formula = Surv(start, end, dummy) ~ sibdead5 + sibdead5_10 +
    sibdead10_15 + famsize + dadD + momD + cohort + birth rank +
    socrank + cluster(idf), data = sample, method = "efron")

  n= 75902, number of events= 379

                      coef exp(coef) se(coef) robust se     z Pr(>|z|)
sibdead5          0.461497  1.586447 0.125305  0.131416 3.512 0.000445
***
sibdead5_10       0.167975  1.182907 0.126961  0.126837 1.324 0.185391
sibdead10_15      0.162196  1.176091 0.137046  0.142559 1.138 0.255228
famsize           0.084818  1.088519 0.025807  0.026671 3.180 0.001472
**
dadD              0.187404  1.206114 0.110842  0.120033 1.561 0.118460
momD              0.106220  1.112067 0.121478  0.129631 0.819 0.412556
cohort            0.019654  1.019848 0.002240  0.002481 7.922 2.34e-15
***
birth rank        0.014512  1.014618 0.026060  0.026972 0.538 0.590550
socrank2          0.393138  1.481623 0.605929  0.653053 0.602 0.547174
socrank3          0.487240  1.627817 0.585943  0.608657 0.801 0.423412
socrank4          1.629369  5.100653 0.460996  0.469211 3.473 0.000516
***
socrank5          1.545115  4.688509 0.454658  0.457490 3.377 0.000732
***
---
Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1
```

```
                 exp(coef) exp(-coef) lower .95 upper .95
sibdead5             1.586     0.6303    1.2262     2.053
sibdead5_10          1.183     0.8454    0.9225     1.517
sibdead10_15         1.176     0.8503    0.8894     1.555
famsize              1.089     0.9187    1.0331     1.147
dadD                 1.206     0.8291    0.9533     1.526
momD                 1.112     0.8992    0.8626     1.434
cohort               1.020     0.9805    1.0149     1.025
birth rank           1.015     0.9856    0.9624     1.070
socrank2             1.482     0.6749    0.4120     5.329
socrank3             1.628     0.6143    0.4938     5.367
socrank4             5.101     0.1961    2.0334    12.794
socrank5             4.689     0.2133    1.9126    11.493

Concordance= 0.717  (se = 0.015 )
Rsquare= 0.003   (max possible= 0.077 )
Likelihood ratio test= 226.6  on 12 df,   p=<2e-16
Wald test            = 166.7  on 12 df,   p=<2e-16
Score (logrank) test = 203  on 12 df,   p=<2e-16,   Robust = 157.5
p=<2e-16

  (Note: the likelihood ratio and score tests assume independence of
     observations within a cluster, the Wald and robust score tests do
not).
                      rho  chisq     p
sibdead5         -0.01682 0.1363 0.712
sibdead5_10      -0.00677 0.0191 0.890
sibdead10_15      0.02886 0.4308 0.512
famsize           0.01909 0.1483 0.700
dadD              0.05179 1.4164 0.234
momD              0.01947 0.2035 0.652
cohort            0.00521 0.0170 0.896
birth rank       -0.07031 2.3104 0.129
socrank2          0.01419 0.1030 0.748
socrank3          0.06065 1.6290 0.202
socrank4          0.01176 0.0567 0.812
socrank5          0.02670 0.2846 0.594
GLOBAL                 NA 7.5054 0.822
```

```
Call:
coxph(formula = Surv(start, end, dummy) ~ sibdead5 + sibdead5_10 +
    sibdead10_15 + famsize + dadD + momD + cohort + birth rank +
    socrank + cluster(idf), data = sample2, method = "efron")

  n= 41102, number of events= 203 (singlets removed)

                       coef exp(coef)  se(coef) robust se      z
Pr(>|z|)
sibdead5           0.386772  1.472221  0.165635  0.176855  2.187
0.02875 *
sibdead5_10        0.088385  1.092408  0.168763  0.168093  0.526
0.59902
sibdead10_15       0.091466  1.095780  0.184185  0.191509  0.478
0.63293
famsize           -0.009013  0.991027  0.039107  0.042707 -0.211
0.83285
dadD              -0.072287  0.930264  0.152190  0.164362 -0.440
0.66008
momD              -0.049084  0.952101  0.172919  0.189518 -0.259
0.79564
cohort             0.017781  1.017940  0.003420  0.003807  4.671    3e-
06 ***
birth rank         0.039015  1.039786  0.032872  0.033575  1.162
0.24522
socrank2           0.639981  1.896444  0.913661  1.033326  0.619
0.53569
socrank3           1.246246  3.477263  0.817154  0.830875  1.500
0.13364
socrank4           2.126302  8.383808  0.721219  0.723216  2.940
0.00328 **
socrank5           1.990532  7.319429  0.716892  0.721912  2.757
0.00583 **
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1
```

```
                  exp(coef) exp(-coef) lower .95 upper .95
sibdead5             1.4722     0.6792    1.0410     2.082
sibdead5_10          1.0924     0.9154    0.7858     1.519
sibdead10_15         1.0958     0.9126    0.7529     1.595
famsize              0.9910     1.0091    0.9115     1.078
dadD                 0.9303     1.0750    0.6741     1.284
momD                 0.9521     1.0503    0.6567     1.380
cohort               1.0179     0.9824    1.0104     1.026
birth rank           1.0398     0.9617    0.9736     1.111
socrank2             1.8964     0.5273    0.2502    14.372
socrank3             3.4773     0.2876    0.6823    17.721
socrank4             8.3838     0.1193    2.0316    34.597
socrank5             7.3194     0.1366    1.7782    30.128

Concordance= 0.701  (se = 0.022 )
Rsquare= 0.002   (max possible= 0.07 )
Likelihood ratio test= 93.06  on 12 df,   p=1e-14
Wald test            = 56.11  on 12 df,   p=1e-07
Score (logrank) test = 75.94  on 12 df,   p=2e-11,   Robust = 74.84
p=4e-11

  (Note: the likelihood ratio and score tests assume independence of
     observations within a cluster, the Wald and robust score tests do
not).
                      rho    chisq       p
sibdead5         -0.09023  2.70573 0.09999
sibdead5_10       0.03836  0.37777 0.53880
sibdead10_15      0.00435  0.00636 0.93643
famsize           0.08094  1.87082 0.17138
dadD              0.02449  0.16503 0.68457
momD              0.01300  0.05315 0.81767
cohort            0.03405  0.41779 0.51804
birth rank       -0.16950  7.67653 0.00559
socrank2         -0.06930  1.80571 0.17902
socrank3          0.06326  0.98406 0.32120
socrank4         -0.01989  0.08633 0.76890
socrank5         -0.00669  0.00989 0.92078
GLOBAL                 NA 19.26901 0.08224
```

```
Call:
coxph(formula = Surv(start, end, dummy) ~ sibdead5 + sibdead5_10 +
    sibdead10_15 + famsize + dadD + momD + cohort + birth rank +
    strata(idf), data = sample2, method = "efron")

  n= 41102, number of events= 203

                      coef exp(coef) se(coef)      z Pr(>|z|)
sibdead5           1.13874   3.12284  0.41584  2.738  0.00617 **
sibdead5_10        0.53830   1.71308  0.41107  1.309  0.19037
sibdead10_15       0.47774   1.61242  0.41175  1.160  0.24595
famsize           -0.14558   0.86452  0.37109 -0.392  0.69483
dadD              -0.27087   0.76272  0.53261 -0.509  0.61105
momD              -0.07831   0.92468  0.59176 -0.132  0.89472
cohort             0.01862   1.01880  0.04134  0.450  0.65235
birth rank         0.13694   1.14676  0.13490  1.015  0.31005
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

                  exp(coef) exp(-coef) lower .95 upper .95
sibdead5             3.1228     0.3202    1.3822     7.055
sibdead5_10          1.7131     0.5837    0.7654     3.834
sibdead10_15         1.6124     0.6202    0.7194     3.614
famsize              0.8645     1.1567    0.4177     1.789
dadD                 0.7627     1.3111    0.2685     2.166
momD                 0.9247     1.0815    0.2899     2.949
cohort               1.0188     0.9815    0.9395     1.105
birth rank           1.1468     0.8720    0.8803     1.494

Concordance= 0.62  (se = 0.054 )
Rsquare= 0   (max possible= 0.005 )
Likelihood ratio test= 13.98  on 8 df,   p=0.08
Wald test            = 12.11  on 8 df,   p=0.1
Score (logrank) test = 13.27  on 8 df,   p=0.1
```

```
                       rho   chisq      p
sibdead5          -0.03333 0.21379 0.644
sibdead5_10        0.06261 0.83710 0.360
sibdead10_15       0.10205 2.01881 0.155
famsize            0.00711 0.00905 0.924
dadD               0.01667 0.06997 0.791
momD               0.05111 0.50809 0.476
cohort             0.03415 0.27989 0.597
birth rank        -0.06339 1.08026 0.299
GLOBAL                  NA 7.80950 0.452
```

A2- Full models for table 3

```
Call:
coxph(formula = Surv(start, end, dummy) ~ sibdead5 + sibdead5_10 +
    sibdead10_15 + famsize + dadD + momD + cohort + birth rank +
    socrank + cluster(idf), data = sample, method = "efron")

  n= 75902, number of events= 8304

                        coef  exp(coef)   se(coef)  robust se       z
Pr(>|z|)
sibdead5           0.0889959  1.0930762  0.0296907  0.0306592   2.903
0.00370 **
sibdead5_10        0.0556656  1.0572441  0.0281799  0.0291235   1.911
0.05596 .
sibdead10_15       0.0460261  1.0471017  0.0309632  0.0312132   1.475
0.14033
famsize           -0.0025546  0.9974486  0.0059498  0.0063271  -0.404
0.68639
dadD               0.0756681  1.0786045  0.0241470  0.0261074   2.898
0.00375 **
momD               0.1431891  1.1539479  0.0255973  0.0270556   5.292
1.21e-07 ***
cohort             0.0012841  1.0012849  0.0003609  0.0003860   3.326
0.00088 ***
```

```
birth rank        -0.0122299  0.9878446  0.0060599  0.0062139 -1.968
0.04905 *
socrank2          -0.0433941  0.9575339  0.0563588  0.0687967 -0.631
0.52820
socrank3          -0.2166046  0.8052483  0.0587142  0.0702607 -3.083
0.00205 **
socrank4          -0.2636986  0.7682051  0.0454434  0.0543188 -4.855
1.21e-06 ***
socrank5          -0.3539909  0.7018813  0.0435255  0.0529750 -6.682
2.35e-11 ***
---
Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

                  exp(coef) exp(-coef) lower .95 upper .95
sibdead5             1.0931     0.9148    1.0293    1.1608
sibdead5_10          1.0572     0.9459    0.9986    1.1193
sibdead10_15         1.0471     0.9550    0.9850    1.1132
famsize              0.9974     1.0026    0.9852    1.0099
dadD                 1.0786     0.9271    1.0248    1.1352
momD                 1.1539     0.8666    1.0944    1.2168
cohort               1.0013     0.9987    1.0005    1.0020
birth rank           0.9878     1.0123    0.9759    0.9999
socrank2             0.9575     1.0443    0.8367    1.0958
socrank3             0.8052     1.2419    0.7017    0.9241
socrank4             0.7682     1.3017    0.6906    0.8545
socrank5             0.7019     1.4247    0.6327    0.7787

Concordance= 0.549  (se = 0.004 )
Rsquare= 0.002   (max possible= 0.828 )
Likelihood ratio test= 178.6  on 12 df,   p=<2e-16
Wald test            = 141.9  on 12 df,   p=<2e-16
Score (logrank) test = 186.5  on 12 df,   p=<2e-16,
Robust = 152.5  p=<2e-16

  (Note: the likelihood ratio and score tests assume independence of
     observations within a cluster, the Wald and robust score tests do
not).
```

```
                        rho     chisq         p
sibdead5           -0.01521    2.0795 1.49e-01
sibdead5_10        -0.01895    3.2629 7.09e-02
sibdead10_15        0.01041    0.9409 3.32e-01
famsize             0.03065    9.3269 2.26e-03
dadD               -0.01974    3.8730 4.91e-02
momD               -0.01072    1.0771 2.99e-01
cohort              0.03853   15.0333 1.06e-04
birth rank          0.00277    0.0695 7.92e-01
socrank2            0.01348    2.2572 1.33e-01
socrank3            0.02968   10.5500 1.16e-03
socrank4            0.06767   55.3887 9.89e-14
socrank5            0.06311   49.2998 2.20e-12
GLOBAL                   NA  140.4404 4.88e-24

Call:
coxph(formula = Surv(start, end, dummy) ~ sibdead5 + sibdead5_10 +
    sibdead10_15 + famsize + dadD + momD + cohort + birth rank +
    socrank + cluster(idf), data = sample2, method = "efron")

  n= 41102, number of events= 4104

                         coef  exp(coef)   se(coef)  robust se       z
Pr(>|z|)
sibdead5            0.0125044  1.0125829  0.0400346  0.0407968  0.307
0.759221
sibdead5_10         0.0009663  1.0009667  0.0373203  0.0385534  0.025
0.980005
sibdead10_15        0.0422626  1.0431684  0.0405057  0.0413892  1.021
0.307206
famsize            -0.0157184  0.9844045  0.0088202  0.0100250 -1.568
0.116899
dadD                0.0330060  1.0335567  0.0335372  0.0376126  0.878
0.380201
momD                0.1210104  1.1286367  0.0362946  0.0399192  3.031
0.002434 **
cohort             -0.0002603  0.9997398  0.0005774  0.0006428 -0.405
0.685554
```

```
birth rank        -0.0124831  0.9875945  0.0075979  0.0076423 -1.633
0.102379
socrank2          -0.1758523  0.8387418  0.0747080  0.0875493 -2.009
0.044579 *
socrank3          -0.2417456  0.7852559  0.0765612  0.0915048 -2.642
0.008244 **
socrank4          -0.2483411  0.7800938  0.0585654  0.0677307 -3.667
0.000246 ***
socrank5          -0.3040157  0.7378492  0.0568414  0.0672259 -4.522
6.12e-06 ***
---
Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

                  exp(coef) exp(-coef) lower .95 upper .95
sibdead5             1.0126     0.9876    0.9348    1.0969
sibdead5_10          1.0010     0.9990    0.9281    1.0795
sibdead10_15         1.0432     0.9586    0.9619    1.1313
famsize              0.9844     1.0158    0.9653    1.0039
dadD                 1.0336     0.9675    0.9601    1.1126
momD                 1.1286     0.8860    1.0437    1.2205
cohort               0.9997     1.0003    0.9985    1.0010
birth rank           0.9876     1.0126    0.9729    1.0025
socrank2             0.8387     1.1923    0.7065    0.9958
socrank3             0.7853     1.2735    0.6563    0.9395
socrank4             0.7801     1.2819    0.6831    0.8908
socrank5             0.7378     1.3553    0.6468    0.8418

Concordance= 0.537  (se = 0.006 )
Rsquare= 0.001   (max possible= 0.768 )
Likelihood ratio test= 53.52  on 12 df,   p=3e-07
Wald test            = 45.24  on 12 df,   p=9e-06
Score (logrank) test = 55.68  on 12 df,   p=1e-07,
Robust = 45.06  p=1e-05

  (Note: the likelihood ratio and score tests assume independence of
     observations within a cluster, the Wald and robust score tests do
not).
```

```
                    rho   chisq        p
sibdead5       -0.01794  1.4115 2.35e-01
sibdead5_10    -0.01997  1.7767 1.83e-01
sibdead10_15   -0.00465  0.0950 7.58e-01
famsize         0.03295  5.9884 1.44e-02
dadD           -0.00636  0.2125 6.45e-01
momD           -0.01347  0.9009 3.43e-01
cohort          0.04309 10.0745 1.50e-03
birth rank      0.00198  0.0169 8.96e-01
socrank2        0.02099  2.4881 1.15e-01
socrank3        0.01524  1.3659 2.43e-01
socrank4        0.06363 22.4516 2.16e-06
socrank5        0.04415 10.9334 9.44e-04
GLOBAL               NA 57.0893 7.62e-08

Call:
coxph(formula = Surv(start, end, dummy) ~ sibdead5 + sibdead5_10 +
    sibdead10_15 + famsize + dadD + momD + cohort + birth rank +
    strata(idf), data = sample2, method = "efron")

  n= 41102, number of events= 4104

                    coef exp(coef)  se(coef)      z Pr(>|z|)
sibdead5        0.043200  1.044146  0.078863  0.548    0.584
sibdead5_10    -0.057072  0.944526  0.081598 -0.699    0.484
sibdead10_15   -0.047344  0.953759  0.084859 -0.558    0.577
famsize        -0.012963  0.987121  0.081260 -0.160    0.873
dadD            0.231750  1.260804  0.112274  2.064    0.039 *
momD            0.120991  1.128615  0.130524  0.927    0.354
cohort          0.007920  1.007951  0.008112  0.976    0.329
birth rank     -0.039983  0.960806  0.026156 -1.529    0.126
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

               exp(coef) exp(-coef) lower .95 upper .95
sibdead5          1.0441     0.9577    0.8946     1.219
sibdead5_10       0.9445     1.0587    0.8049     1.108
sibdead10_15      0.9538     1.0485    0.8076     1.126
```

```
famsize             0.9871     1.0130    0.8418     1.158
dadD                1.2608     0.7931    1.0118     1.571
momD                1.1286     0.8860    0.8739     1.458
cohort              1.0080     0.9921    0.9921     1.024
birth rank          0.9608     1.0408    0.9128     1.011

Concordance= 0.511  (se = 0.013 )
Rsquare= 0   (max possible= 0.091 )
Likelihood ratio test= 7.94  on 8 df,   p=0.4
Wald test            = 7.9  on 8 df,   p=0.4
Score (logrank) test = 7.93  on 8 df,   p=0.4

                      rho  chisq     p
sibdead5         -0.00653 0.1762 0.675
sibdead5_10       0.01189 0.5888 0.443
sibdead10_15      0.01067 0.4755 0.490
famsize          -0.02297 2.2319 0.135
dadD             -0.01760 1.2895 0.256
momD             -0.01527 0.9607 0.327
cohort            0.00628 0.1635 0.686
birth rank        0.00183 0.0142 0.905
GLOBAL                 NA 6.8566 0.552
```

A3- Experiencing sibling's death before age 7

```
Call:
coxph(formula = Surv(start, end, dummy) ~ sibdead7 + famsize +
    dadD + momD + cohort + birth rank + socrank + cluster(idf), data =
sample,
    method = "efron")

  n= 75902, number of events= 379

                 coef exp(coef) se(coef) robust se     z Pr(>|z|)
sibdead7     0.465231  1.592382 0.113156  0.116511 3.993 6.52e-05 ***
famsize      0.086435  1.090281 0.025593  0.026436 3.270 0.001077 **
dadD         0.192721  1.212544 0.110686  0.119225 1.616 0.105999
```

```
momD          0.099410  1.104519 0.121623  0.129338 0.769 0.442128
cohort        0.019580  1.019772 0.002238  0.002477 7.904 2.70e-15 ***
birth rank    0.015007  1.015121 0.025876  0.026707 0.562 0.574156
socrank2      0.400041  1.491886 0.605888  0.654263 0.611 0.540910
socrank3      0.468340  1.597340 0.585859  0.608341 0.770 0.441380
socrank4      1.624355  5.075144 0.460901  0.468149 3.470 0.000521 ***
socrank5      1.536179  4.646800 0.454663  0.456835 3.363 0.000772 ***
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

              exp(coef) exp(-coef) lower .95 upper .95
sibdead7          1.592     0.6280    1.2673     2.001
famsize           1.090     0.9172    1.0352     1.148
dadD              1.213     0.8247    0.9599     1.532
momD              1.105     0.9054    0.8572     1.423
cohort            1.020     0.9806    1.0148     1.025
birth rank        1.015     0.9851    0.9634     1.070
socrank2          1.492     0.6703    0.4138     5.378
socrank3          1.597     0.6260    0.4848     5.263
socrank4          5.075     0.1970    2.0275    12.704
socrank5          4.647     0.2152    1.8980    11.377

Concordance= 0.719  (se = 0.015 )
Rsquare= 0.003   (max possible= 0.077 )
Likelihood ratio test= 225.2  on 10 df,   p=<2e-16
Wald test            = 162.1  on 10 df,   p=<2e-16
Score (logrank) test = 201.4  on 10 df,   p=<2e-16,
Robust = 155.6  p=<2e-16

  (Note: the likelihood ratio and score tests assume independence of
     observations within a cluster, the Wald and robust score tests do
not).
                   rho  chisq      p
sibdead7 -0.06338 1.7761 0.1826
famsize        0.03131 0.4082 0.5229
dadD           0.06476 2.0887 0.1484
momD           0.02218 0.2538 0.6144
cohort         0.00870 0.0463 0.8297
```

```
birth rank    -0.07993 2.8310 0.0925
socrank2       0.00574 0.0157 0.9002
socrank3       0.05795 1.4342 0.2311
socrank4       0.00875 0.0305 0.8612
socrank5       0.02174 0.1840 0.6680
GLOBAL              NA 8.9280 0.5390

Call:
coxph(formula = Surv(start, end, dummy) ~ sibdead7 + famsize +
    dadD + momD + cohort + birth rank + socrank + cluster(idf), data =
sample2,
    method = "efron")

  n= 41102, number of events= 203

                    coef exp(coef)  se(coef) robust se      z Pr(>|z|)
sibdead7        0.317840  1.374156  0.151994  0.157586  2.017  0.04370 *
famsize        -0.010735  0.989323  0.039072  0.042944 -0.250  0.80261
dadD           -0.072987  0.929613  0.152070  0.163685 -0.446  0.65567
momD           -0.048866  0.952309  0.172861  0.188584 -0.259  0.79554
cohort          0.017686  1.017843  0.003414  0.003820  4.630 3.65e-06
***
birth rank      0.041123  1.041980  0.032659  0.033339  1.233  0.21739
socrank2        0.636399  1.889663  0.913644  1.032705  0.616  0.53773
socrank3        1.227898  3.414045  0.816898  0.828106  1.483  0.13813
socrank4        2.118561  8.319156  0.720982  0.721174  2.938  0.00331
**
socrank5        1.982052  7.257623  0.716803  0.720344  2.752  0.00593
**
---
Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

              exp(coef) exp(-coef) lower .95 upper .95
sibdead7         1.3742     0.7277    1.0090     1.871
famsize          0.9893     1.0108    0.9095     1.076
dadD             0.9296     1.0757    0.6745     1.281
momD             0.9523     1.0501    0.6580     1.378
cohort           1.0178     0.9825    1.0103     1.025
```

```
birth rank        1.0420     0.9597     0.9761      1.112
socrank2          1.8897     0.5292     0.2497     14.303
socrank3          3.4140     0.2929     0.6736     17.304
socrank4          8.3192     0.1202     2.0240     34.194
socrank5          7.2576     0.1378     1.7686     29.782

Concordance= 0.7  (se = 0.022 )
Rsquare= 0.002   (max possible= 0.07 )
Likelihood ratio test= 91.24  on 10 df,   p=3e-15
Wald test            = 54.1  on 10 df,   p=5e-08
Score (logrank) test = 73.82  on 10 df,   p=8e-12,
Robust = 73.89  p=8e-12

  (Note: the likelihood ratio and score tests assume independence of
     observations within a cluster, the Wald and robust score tests do
not).
                  rho   chisq       p
sibdead7      -0.1083  3.2820 0.07004
famsize        0.0824  1.9680 0.16066
dadD           0.0306  0.2389 0.62502
momD           0.0156  0.0736 0.78612
cohort         0.0458  0.7412 0.38927
birth rank    -0.1810  8.2932 0.00398
socrank2      -0.0784  2.0171 0.15554
socrank3       0.0606  0.8202 0.36513
socrank4      -0.0270  0.1560 0.69287
socrank5      -0.0139  0.0417 0.83811
GLOBAL             NA 19.5901 0.03338


Call:
coxph(formula = Surv(start, end, dummy) ~ sibdead7 + famsize +
    dadD + momD + cohort + birth rank + strata(idf), data = sample2,
    method = "efron")

  n= 41102, number of events= 203
```

```
                  coef exp(coef) se(coef)      z Pr(>|z|)
sibdead7       0.70269   2.01918  0.35474  1.981   0.0476 *
famsize       -0.19442   0.82331  0.36045 -0.539   0.5896
dadD          -0.19932   0.81929  0.52705 -0.378   0.7053
momD          -0.11762   0.88904  0.58155 -0.202   0.8397
cohort         0.02569   1.02602  0.04045  0.635   0.5254
birth rank     0.05415   1.05565  0.12368  0.438   0.6615
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

              exp(coef) exp(-coef) lower .95 upper .95
sibdead7         2.0192     0.4952    1.0074     4.047
famsize          0.8233     1.2146    0.4062     1.669
dadD             0.8193     1.2206    0.2916     2.302
momD             0.8890     1.1248    0.2844     2.779
cohort           1.0260     0.9746    0.9478     1.111
birth rank             1.0556     0.9473    0.8284     1.345

Concordance= 0.595  (se = 0.056 )
Rsquare= 0   (max possible= 0.005 )
Likelihood ratio test= 8.53  on 6 df,   p=0.2
Wald test            = 7.88  on 6 df,   p=0.2
Score (logrank) test = 8.37  on 6 df,   p=0.2

                   rho    chisq      p
sibdead7      -0.05323 0.551604 0.4577
famsize        0.00137 0.000357 0.9849
dadD           0.03227 0.246498 0.6196
momD           0.04279 0.375226 0.5402
cohort         0.05140 0.610269 0.4347
birth rank    -0.11654 3.413590 0.0647
GLOBAL              NA 5.538400 0.4768
```

A4- Experiencing sibling's death before age 15

```
Call:
coxph(formula = Surv(start, end, dummy) ~ sibdead15 + famsize +
    dadD + momD + cohort + birth rank + socrank + cluster(idf), data =
sample,
    method = "efron")

  n= 75902, number of events= 379

                     coef exp(coef) se(coef) robust se     z Pr(>|z|)
sibdead15      0.366998  1.443395 0.105457  0.111644 3.287 0.001012 **
famsize        0.080732  1.084080 0.025928  0.027089 2.980 0.002880 **
dadD           0.182011  1.199628 0.110943  0.119811 1.519 0.128724
momD           0.110678  1.117035 0.121312  0.129240 0.856 0.391789
cohort         0.019565  1.019758 0.002236  0.002466 7.933 2.14e-15 ***
birth rank     0.021155  1.021380 0.025812  0.026692 0.793 0.428044
socrank2       0.386278  1.471494 0.605951  0.653514 0.591 0.554468
socrank3       0.475357  1.608588 0.585941  0.609095 0.780 0.435137
socrank4       1.644037  5.176025 0.460949  0.468884 3.506 0.000454 ***
socrank5       1.542854  4.677924 0.454520  0.457670 3.371 0.000749 ***
---
Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

               exp(coef) exp(-coef) lower .95 upper .95
sibdead15          1.443     0.6928    1.1597     1.796
famsize            1.084     0.9224    1.0280     1.143
dadD               1.200     0.8336    0.9486     1.517
momD               1.117     0.8952    0.8671     1.439
cohort             1.020     0.9806    1.0148     1.025
birth rank         1.021     0.9791    0.9693     1.076
socrank2           1.471     0.6796    0.4088     5.297
socrank3           1.609     0.6217    0.4875     5.308
socrank4           5.176     0.1932    2.0648    12.975
socrank5           4.678     0.2138    1.9076    11.472

Concordance= 0.716  (se = 0.015 )
Rsquare= 0.003   (max possible= 0.077 )
```

```
Likelihood ratio test= 221.2  on 10 df,   p=<2e-16
Wald test            = 162.7  on 10 df,   p=<2e-16
Score (logrank) test = 196.3  on 10 df,   p=<2e-16,
Robust = 157.5  p=<2e-16

  (Note: the likelihood ratio and score tests assume independence of
     observations within a cluster, the Wald and robust score tests do
not).
                    rho   chisq      p
sibdead15 -0.00290 0.00411 0.9489
famsize         0.02638 0.30016 0.5838
dadD            0.05199 1.36210 0.2432
momD            0.02487 0.31798 0.5728
cohort          0.00671 0.02756 0.8681
birth rank     -0.07991 2.84338 0.0918
socrank2        0.00945 0.04246 0.8368
socrank3        0.05723 1.43224 0.2314
socrank4        0.00774 0.02400 0.8769
socrank5        0.02320 0.21153 0.6456
GLOBAL               NA 6.74510 0.7493


Call:
coxph(formula = Surv(start, end, dummy) ~ sibdead15 + famsize +
    dadD + momD + cohort + birth rank + socrank + cluster(idf), data =
sample2,
    method = "efron")

  n= 41102, number of events= 203

                    coef exp(coef)  se(coef) robust se      z Pr(>|z|)
sibdead15  0.194126  1.214249  0.141185  0.149534  1.298  0.19422
famsize        -0.011272  0.988791  0.039157  0.043059 -0.262  0.79349
dadD           -0.073321  0.929303  0.152160  0.164137 -0.447  0.65509
momD           -0.045750  0.955281  0.172743  0.188530 -0.243  0.80826
cohort          0.017725  1.017883  0.003419  0.003794  4.672 2.99e-06
***
birth rank      0.045727  1.046789  0.032614  0.033469  1.366  0.17185
```

```
socrank2        0.641557  1.899436  0.913763  1.033511  0.621  0.53476
socrank3        1.237124  3.445691  0.816969  0.829731  1.491  0.13596
socrank4        2.147177  8.560659  0.720953  0.721933  2.974  0.00294
**
socrank5        1.984022  7.271931  0.716600  0.720501  2.754  0.00589
**
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

               exp(coef) exp(-coef) lower .95 upper .95
sibdead15         1.2142     0.8236    0.9058     1.628
famsize           0.9888     1.0113    0.9088     1.076
dadD              0.9293     1.0761    0.6737     1.282
momD              0.9553     1.0468    0.6602     1.382
cohort            1.0179     0.9824    1.0103     1.025
birth rank        1.0468     0.9553    0.9803     1.118
socrank2          1.8994     0.5265    0.2506    14.400
socrank3          3.4457     0.2902    0.6777    17.520
socrank4          8.5607     0.1168    2.0797    35.239
socrank5          7.2719     0.1375    1.7716    29.850

Concordance= 0.693  (se = 0.022 )
Rsquare= 0.002   (max possible= 0.07 )
Likelihood ratio test= 88.92  on 10 df,   p=9e-15
Wald test            = 51.74  on 10 df,   p=1e-07
Score (logrank) test = 71.49  on 10 df,   p=2e-11,   Robust = 73.39
p=1e-11

  (Note: the likelihood ratio and score tests assume independence of
     observations within a cluster, the Wald and robust score tests do
not).

                     rho    chisq       p
sibdead15       -0.01115  0.03408 0.85353
famsize          0.07976  1.81200 0.17827
dadD             0.01522  0.06000 0.80649
momD             0.01620  0.07933 0.77821
cohort           0.04100  0.58771 0.44331
```

```
birth rank     -0.17776  8.13500 0.00434
socrank2       -0.06349  1.30139 0.25396
socrank3        0.06415  0.97940 0.32235
socrank4       -0.02248  0.10961 0.74059
socrank5       -0.00346  0.00263 0.95911
GLOBAL               NA 16.02912 0.09880


Call:
coxph(formula = Surv(start, end, dummy) ~ sibdead15 + famsize +
    dadD + momD + cohort + birth rank + strata(idf), data = sample2,
    method = "efron")

  n= 41102, number of events= 203

                   coef exp(coef) se(coef)      z Pr(>|z|)
sibdead15       0.72747   2.06984  0.38951  1.868   0.0618 .
famsize        -0.14990   0.86079  0.36209 -0.414   0.6789
dadD           -0.23772   0.78842  0.52494 -0.453   0.6507
momD           -0.06580   0.93632  0.58112 -0.113   0.9098
cohort          0.03006   1.03052  0.04035  0.745   0.4563
birth rank            0.06907   1.07151  0.12467  0.554   0.5796
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

               exp(coef) exp(-coef) lower .95 upper .95
sibdead15         2.0698     0.4831    0.9647     4.441
famsize           0.8608     1.1617    0.4233     1.750
dadD              0.7884     1.2684    0.2818     2.206
momD              0.9363     1.0680    0.2998     2.925
cohort            1.0305     0.9704    0.9522     1.115
birth rank              1.0715      0.9333    0.8392      1.368

Concordance= 0.589  (se = 0.057 )
Rsquare= 0   (max possible= 0.005 )
Likelihood ratio test= 8.13  on 6 df,   p=0.2
Wald test            = 7.41  on 6 df,   p=0.3
Score (logrank) test = 7.99  on 6 df,   p=0.2
```

```
                  rho    chisq     p
sibdead15      0.00642 0.007326 0.932
famsize       -0.00224 0.000905 0.976
dadD           0.02849 0.184952 0.667
momD           0.04247 0.358049 0.550
cohort         0.04292 0.413571 0.520
birth rank    -0.10335 2.573838 0.109
GLOBAL              NA 4.666107 0.587
```

A5- Number of experienced sibling's deaths

```
Call:
coxph(formula = Surv(start, end, dummy) ~ sibdead5 + sibdead5_10 +
    sibdead10_15 + famsize + dadD + momD + cohort + birth rank +
    socrank + cluster(idf), data = sample, method = "efron")

  n= 75902, number of events= 379

                     coef exp(coef) se(coef) robust se     z Pr(>|z|)
sibdead5         0.374332  1.454019 0.098797  0.101825 3.676 0.000237
***
sibdead5_10      0.191595  1.211180 0.092567  0.097244 1.970 0.048810
*
sibdead10_15     0.144705  1.155699 0.105869  0.110882 1.305 0.191879
famsize          0.086169  1.089990 0.025769  0.026374 3.267 0.001086
**
dadD             0.193350  1.213307 0.110598  0.119221 1.622 0.104852
momD             0.110195  1.116496 0.121324  0.129165 0.853 0.393587
cohort           0.019950  1.020150 0.002249  0.002489 8.014 1.11e-15
***
birth rank       0.013428  1.013518 0.026133  0.026811 0.501 0.616486
socrank2         0.397187  1.487634 0.605988  0.653648 0.608 0.543422
socrank3         0.513969  1.671913 0.586173  0.611074 0.841 0.400298
socrank4         1.649980  5.206874 0.461126  0.470090 3.510 0.000448
***
socrank5         1.564472  4.780150 0.454905  0.458573 3.412 0.000646
***---
```

```
Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

                exp(coef) exp(-coef) lower .95 upper .95
sibdead5            1.454     0.6877    1.1910     1.775
sibdead5_10         1.211     0.8256    1.0010     1.465
sibdead10_15        1.156     0.8653    0.9300     1.436
famsize             1.090     0.9174    1.0351     1.148
dadD                1.213     0.8242    0.9605     1.533
momD                1.116     0.8957    0.8668     1.438
cohort              1.020     0.9802    1.0152     1.025
birth rank          1.014     0.9867    0.9616     1.068
socrank2            1.488     0.6722    0.4132     5.357
socrank3            1.672     0.5981    0.5047     5.538
socrank4            5.207     0.1921    2.0722    13.083
socrank5            4.780     0.2092    1.9458    11.743

Concordance= 0.718  (se = 0.015 )
Rsquare= 0.003   (max possible= 0.077 )
Likelihood ratio test= 230.4  on 12 df,   p=<2e-16
Wald test            = 167.6  on 12 df,   p=<2e-16
Score (logrank) test = 205.8  on 12 df,   p=<2e-16,   Robust = 157.8
p=<2e-16

  (Note: the likelihood ratio and score tests assume independence of
     observations within a cluster, the Wald and robust score tests do
not).
```

```
                     rho   chisq     p
sibdead5        -0.01376 0.08660 0.769
sibdead5_10     -0.02371 0.28482 0.594
sibdead10_15     0.04026 0.83459 0.361
famsize          0.02252 0.20222 0.653
dadD             0.04927 1.25520 0.263
momD             0.01870 0.18751 0.665
cohort           0.00262 0.00437 0.947
birth rank      -0.06950 2.18185 0.140
socrank2         0.01652 0.13614 0.712
```

```
socrank3           0.06397 1.83391 0.176
socrank4           0.01524 0.09627 0.756
socrank5           0.03062 0.37963 0.538
GLOBAL                  NA 8.02705 0.783


Call:
coxph(formula = Surv(start, end, dummy) ~ sibdead5 + sibdead5_10 +
    sibdead10_15 + famsize + dadD + momD + cohort + birth rank +
    socrank + cluster(idf), data = sample2, method = "efron")

  n= 41102, number of events= 203

                      coef exp(coef)  se(coef) robust se      z
Pr(>|z|)
sibdead5           0.362837  1.437401  0.131007  0.140118  2.590
0.00961 **
sibdead5_10        0.058686  1.060443  0.127769  0.125155  0.469
0.63914
sibdead10_15       0.148106  1.159636  0.136879  0.150795  0.982
0.32601
famsize           -0.007887  0.992144  0.039147  0.042603 -0.185
0.85313
dadD              -0.064310  0.937714  0.152036  0.163183 -0.394
0.69351
momD              -0.047870  0.953257  0.172806  0.189060 -0.253
0.80011
cohort             0.018122  1.018287  0.003440  0.003804  4.764  1.9e-
06 ***
birth rank         0.037362  1.038068  0.033006  0.033297  1.122
0.26184
socrank2           0.663808  1.942175  0.913735  1.036904  0.640
0.52205
socrank3           1.295453  3.652650  0.817756  0.836097  1.549
0.12128
socrank4           2.163487  8.701431  0.721371  0.725961  2.980
0.00288 **
```

```
socrank5           2.017942  7.522830  0.717140  0.725185  2.783
0.00539 **
---
Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

                 exp(coef) exp(-coef) lower .95 upper .95
sibdead5            1.4374     0.6957    1.0922     1.892
sibdead5_10         1.0604     0.9430    0.8298     1.355
sibdead10_15        1.1596     0.8623    0.8629     1.558
famsize             0.9921     1.0079    0.9127     1.079
dadD                0.9377     1.0664    0.6810     1.291
momD                0.9533     1.0490    0.6581     1.381
cohort              1.0183     0.9820    1.0107     1.026
birth rank          1.0381     0.9633    0.9725     1.108
socrank2            1.9422     0.5149    0.2545    14.822
socrank3            3.6526     0.2738    0.7094    18.806
socrank4            8.7014     0.1149    2.0973    36.102
socrank5            7.5228     0.1329    1.8159    31.164

Concordance= 0.701  (se = 0.022 )
Rsquare= 0.002   (max possible= 0.07 )
Likelihood ratio test= 96.02  on 12 df,   p=3e-15
Wald test            = 57.9  on 12 df,   p=5e-08
Score (logrank) test = 78.71  on 12 df,   p=7e-12,
Robust = 74.98  p=4e-11

  (Note: the likelihood ratio and score tests assume independence of
     observations within a cluster, the Wald and robust score tests do
not).
                      rho    chisq       p
sibdead5         -0.08570  2.39766 0.12152
sibdead5_10       0.02804  0.19550 0.65838
sibdead10_15      0.04174  0.64665 0.42131
famsize           0.07833  1.79347 0.18050
dadD              0.01855  0.09250 0.76103
momD              0.00961  0.02950 0.86363
cohort            0.03305  0.39893 0.52764
birth rank       -0.16572  7.33011 0.00678
```

```
socrank2          -0.06083  1.30063 0.25410
socrank3           0.06766  1.14243 0.28514
socrank4          -0.01594  0.05670 0.81178
socrank5          -0.00266  0.00161 0.96802
GLOBAL                  NA 18.95540 0.08961


Call:
coxph(formula = Surv(start, end, dummy) ~ sibdead5 + sibdead5_10 +
    sibdead10_15 + famsize + dadD + momD + cohort + birth rank +
    strata(idf), data = sample2, method = "efron")

  n= 41102, number of events= 203

                      coef exp(coef) se(coef)      z Pr(>|z|)
sibdead5           0.80389   2.23421  0.32548  2.470   0.0135 *
sibdead5_10        0.42510   1.52975  0.33785  1.258   0.2083
sibdead10_15       0.24077   1.27222  0.29634  0.812   0.4165
famsize           -0.16405   0.84870  0.37388 -0.439   0.6608
dadD              -0.18343   0.83241  0.52527 -0.349   0.7269
momD              -0.04375   0.95719  0.58832 -0.074   0.9407
cohort             0.02774   1.02812  0.04128  0.672   0.5017
birth rank         0.08889   1.09296  0.13654  0.651   0.5150
---
Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

                  exp(coef) exp(-coef) lower .95 upper .95
sibdead5             2.2342     0.4476    1.1805     4.228
sibdead5_10          1.5297     0.6537    0.7889     2.966
sibdead10_15         1.2722     0.7860    0.7117     2.274
famsize              0.8487     1.1783    0.4079     1.766
dadD                 0.8324     1.2013    0.2973     2.330
momD                 0.9572     1.0447    0.3021     3.032
cohort               1.0281     0.9726    0.9482     1.115
birth rank           1.0930     0.9149    0.8363     1.428

Concordance= 0.607  (se = 0.055 )
Rsquare= 0   (max possible= 0.005 )
```

```
Likelihood ratio test= 11.71  on 8 df,   p=0.2
Wald test            = 10.37  on 8 df,   p=0.2
Score (logrank) test = 11.31  on 8 df,   p=0.2

                      rho   chisq     p
sibdead5          0.0046 0.00457 0.946
sibdead5_10       0.0474 0.48954 0.484
sibdead10_15      0.1115 2.41305 0.120
famsize           0.0185 0.06210 0.803
dadD              0.0150 0.05513 0.814
momD              0.0397 0.30034 0.584
cohort            0.0268 0.17171 0.679
birth rank       -0.0450 0.56395 0.453
GLOBAL                NA 6.85994 0.552
```

A6- Raw effect of experiencing sibling's death

```
Call:
coxph(formula = Surv(start, end, dummy) ~ sibdead5 + sibdead5_10 +
    sibdead10_15 + cluster(idf), data = sample, method = "efron")

  n= 75902, number of events= 379

                    coef exp(coef) se(coef) robust se     z Pr(>|z|)
sibdead5          0.4603    1.5846   0.1229    0.1268 3.630 0.000283
***
sibdead5_10       0.1338    1.1432   0.1270    0.1274 1.051 0.293470
sibdead10_15      0.1686    1.1836   0.1367    0.1398 1.206 0.227856
---
Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

                  exp(coef) exp(-coef) lower .95 upper .95
sibdead5              1.585     0.6311    1.2359     2.032
sibdead5_10           1.143     0.8747    0.8906     1.467
sibdead10_15          1.184     0.8449    0.9000     1.557
```

```
Concordance= 0.551  (se = 0.017 )
Rsquare= 0   (max possible= 0.077 )
Likelihood ratio test= 17.6  on 3 df,   p=5e-04
Wald test            = 17.81  on 3 df,   p=5e-04
Score (logrank) test = 19.53  on 3 df,   p=2e-04,
Robust = 13.31  p=0.004

  (Note: the likelihood ratio and score tests assume independence of
     observations within a cluster, the Wald and robust score tests do
not).
                        rho    chisq     p
sibdead5          -0.021933 0.196701 0.657
sibdead5_10        0.000843 0.000275 0.987
sibdead10_15       0.067639 1.826316 0.177
GLOBAL                   NA 1.948200 0.583



Call:
coxph(formula = Surv(start, end, dummy) ~ sibdead5 + sibdead5_10 +
    sibdead10_15 + cluster(idf), data = sample2, method = "efron")

  n= 41102, number of events= 203

                      coef exp(coef) se(coef) robust se      z Pr(>|z|)
sibdead5           0.34847   1.41689  0.16264   0.17099  2.038   0.0416
*
sibdead5_10       -0.02379   0.97649  0.16837   0.16938 -0.140   0.8883
sibdead10_15      -0.02462   0.97568  0.18333   0.19030 -0.129   0.8971
---
Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

                  exp(coef) exp(-coef) lower .95 upper .95
sibdead5             1.4169     0.7058    1.0134     1.981
sibdead5_10          0.9765     1.0241    0.7006     1.361
sibdead10_15         0.9757     1.0249    0.6719     1.417

Concordance= 0.567  (se = 0.023 )
Rsquare= 0   (max possible= 0.07 )
```

```
Likelihood ratio test= 4.34  on 3 df,   p=0.2
Wald test            = 4.23  on 3 df,   p=0.2
Score (logrank) test = 4.65  on 3 df,   p=0.2,
Robust = 3.46  p=0.3

  (Note: the likelihood ratio and score tests assume independence of
     observations within a cluster, the Wald and robust score tests do
not).
                      rho  chisq      p
sibdead5          -0.1280 3.7795 0.0519
sibdead5_10        0.0129 0.0353 0.8509
sibdead10_15       0.0463 0.4891 0.4843
GLOBAL                 NA 4.4095 0.2205



Call:
coxph(formula = Surv(start, end, dummy) ~ sibdead5 + sibdead5_10 +
    sibdead10_15 + strata(idf), data = sample2, method = "efron")

  n= 41102, number of events= 203

                    coef exp(coef) se(coef)     z Pr(>|z|)
sibdead5          0.8829    2.4179   0.3903 2.262   0.0237 *
sibdead5_10       0.1439    1.1548   0.3538 0.407   0.6841
sibdead10_15      0.1466    1.1579   0.3637 0.403   0.6869
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

                  exp(coef) exp(-coef) lower .95 upper .95
sibdead5              2.418     0.4136    1.1252     5.196
sibdead5_10           1.155     0.8660    0.5773     2.310
sibdead10_15          1.158     0.8636    0.5677     2.362

Concordance= 0.577  (se = 0.04 )
Rsquare= 0   (max possible= 0.005 )
Likelihood ratio test= 5.81  on 3 df,   p=0.1
Wald test            = 5.4  on 3 df,   p=0.1
Score (logrank) test = 5.68  on 3 df,   p=0.1
```

```
                    rho  chisq      p
sibdead5        -0.0108 0.0271 0.8693
sibdead5_10      0.0793 1.1882 0.2757
sibdead10_15     0.1255 3.2130 0.0731
GLOBAL               NA 4.4826 0.2139
```

A7- Experiencing sibling's death before the age of three, between the age of three and seven, and between the age of seven and 15

```
Call:
coxph(formula = Surv(start, end, dummy) ~ sibdead3 + sibdead3_7 +
    sibdead7_15 + famsize + dadD + momD + cohort + birth rank +
    socrank + cluster(idf), data = sample, method = "efron")

  n= 75902, number of events= 379

                   coef exp(coef) se(coef) robust se     z Pr(>|z|)
sibdead3       0.385898  1.470935 0.163506  0.163394 2.362 0.018188 *
sibdead3_7     0.460612  1.585044 0.122941  0.125355 3.674 0.000238
***
sibdead7_15    0.139163  1.149311 0.118467  0.122021 1.140 0.254084
famsize        0.083755  1.087363 0.025822  0.026728 3.134 0.001727
**
dadD           0.187776  1.206564 0.110809  0.119679 1.569 0.116650
momD           0.100879  1.106143 0.121516  0.129107 0.781 0.434591
cohort         0.019706  1.019901 0.002244  0.002491 7.912 2.53e-15
***
birth rank     0.015152  1.015267 0.026065  0.026724 0.567 0.570724
socrank2       0.403467  1.497005 0.606035  0.652589 0.618 0.536407
socrank3       0.486401  1.626453 0.585973  0.610019 0.797 0.425245
socrank4       1.643002  5.170667 0.460935  0.468542 3.507 0.000454
***
socrank5       1.557077  4.744929 0.454735  0.456643 3.410 0.000650
***
---
Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1
```

```
                    exp(coef) exp(-coef) lower .95 upper .95
sibdead3                1.471     0.6798    1.0679     2.026
sibdead3_7              1.585     0.6309    1.2398     2.026
sibdead7_15             1.149     0.8701    0.9048     1.460
famsize                 1.087     0.9197    1.0319     1.146
dadD                    1.207     0.8288    0.9543     1.526
momD                    1.106     0.9040    0.8588     1.425
cohort                  1.020     0.9805    1.0149     1.025
birth rank              1.015     0.9850    0.9635     1.070
socrank2                1.497     0.6680    0.4166     5.379
socrank3                1.626     0.6148    0.4920     5.376
socrank4                5.171     0.1934    2.0641    12.953
socrank5                4.745     0.2108    1.9388    11.612

Concordance= 0.719  (se = 0.015 )
Rsquare= 0.003   (max possible= 0.077 )
Likelihood ratio test= 230  on 12 df,   p=<2e-16
Wald test            = 172.1  on 12 df,   p=<2e-16
Score (logrank) test = 206.2  on 12 df,   p=<2e-16,   Robust = 158.9
p=<2e-16

  (Note: the likelihood ratio and score tests assume independence of
     observations within a cluster, the Wald and robust score tests do
not).
                         rho    chisq     p
sibdead3           -5.43e-05 1.33e-06 0.999
sibdead3_7         -7.17e-02 2.21e+00 0.137
sibdead7_15         2.65e-02 3.17e-01 0.573
famsize             2.57e-02 2.73e-01 0.601
dadD                5.77e-02 1.71e+00 0.191
momD                2.23e-02 2.57e-01 0.612
cohort              1.11e-02 7.79e-02 0.780
birth rank         -7.77e-02 2.68e+00 0.102
socrank2            1.12e-02 6.09e-02 0.805
socrank3            6.39e-02 1.81e+00 0.179
socrank4            1.15e-02 5.50e-02 0.815
socrank5            2.51e-02 2.49e-01 0.618
GLOBAL                    NA 9.60e+00 0.651
```

```
Call:
coxph(formula = Surv(start, end, dummy) ~ sibdead3 + sibdead3_7 +
    sibdead7_15 + famsize + dadD + momD + cohort + birth rank +
    socrank + cluster(idf), data = sample2, method = "efron")

  n= 41102, number of events= 203

                      coef exp(coef)  se(coef) robust se      z
Pr(>|z|)
sibdead3          0.379133  1.461018  0.206586  0.207617  1.826
0.06783 .
sibdead3_7        0.276275  1.318210  0.169331  0.174993  1.579
0.11439
sibdead7_15       0.064521  1.066648  0.157606  0.162466  0.397
0.69127
famsize          -0.009295  0.990748  0.039126  0.042772 -0.217
0.82796
dadD             -0.071820  0.930698  0.152089  0.163543 -0.439
0.66055
momD             -0.047146  0.953948  0.172964  0.188795 -0.250
0.80280
cohort            0.017891  1.018052  0.003423  0.003820  4.684 2.81e-
06 ***
birth rank        0.039254  1.040035  0.032883  0.033069  1.187
0.23521
socrank2          0.651894  1.919173  0.913734  1.033500  0.631
0.52819
socrank3          1.248966  3.486735  0.817158  0.831808  1.502
0.13322
socrank4          2.133262  8.442362  0.721120  0.723762  2.947
0.00320 **
socrank5          1.998418  7.377378  0.716892  0.722626  2.765
0.00568 **
---
Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1
```

```
                 exp(coef) exp(-coef) lower .95 upper .95
sibdead3              1.4610     0.6845    0.9726     2.195
sibdead3_7            1.3182     0.7586    0.9355     1.858
sibdead7_15           1.0666     0.9375    0.7758     1.467
famsize               0.9907     1.0093    0.9111     1.077
dadD                  0.9307     1.0745    0.6755     1.282
momD                  0.9539     1.0483    0.6589     1.381
cohort                1.0181     0.9823    1.0105     1.026
birth rank            1.0400     0.9615    0.9748     1.110
socrank2              1.9192     0.5211    0.2532    14.549
socrank3              3.4867     0.2868    0.6829    17.801
socrank4              8.4424     0.1185    2.0436    34.876
socrank5              7.3774     0.1355    1.7898    30.409

Concordance= 0.7  (se = 0.022 )
Rsquare= 0.002   (max possible= 0.07 )
Likelihood ratio test= 93  on 12 df,   p=1e-14
Wald test            = 55.2  on 12 df,   p=2e-07
Score (logrank) test = 75.51  on 12 df,   p=3e-11,
Robust = 74.52  p=5e-11

  (Note: the likelihood ratio and score tests assume independence of
     observations within a cluster, the Wald and robust score tests do
not).
                      rho   chisq       p
sibdead3         -0.05299  0.7789 0.37747
sibdead3_7       -0.09494  2.3813 0.12280
sibdead7_15       0.03544  0.3585 0.54937
famsize           0.08077  1.9365 0.16405
dadD              0.02210  0.1290 0.71949
momD              0.01621  0.0798 0.77752
cohort            0.04500  0.7511 0.38614
birth rank       -0.17601  8.1161 0.00439
socrank2         -0.06420  1.4277 0.23214
socrank3          0.06915  1.1480 0.28397
socrank4         -0.02095  0.0961 0.75657
socrank5         -0.00915  0.0187 0.89127
GLOBAL                 NA 19.9098 0.06881
```

```
Call:
coxph(formula = Surv(start, end, dummy) ~ sibdead3 + sibdead3_7 +
    sibdead7_15 + famsize + dadD + momD + cohort + birth rank +
    strata(idf), data = sample2, method = "efron")

  n= 41102, number of events= 203

                    coef exp(coef) se(coef)      z Pr(>|z|)
sibdead3         0.47655   1.61051  0.41623  1.145   0.2522
sibdead3_7       1.00821   2.74070  0.40918  2.464   0.0137 *
sibdead7_15      0.45389   1.57442  0.40814  1.112   0.2661
famsize         -0.19255   0.82485  0.36933 -0.521   0.6021
dadD            -0.37637   0.68635  0.54427 -0.692   0.4892
momD            -0.12373   0.88362  0.59134 -0.209   0.8343
cohort           0.02354   1.02382  0.04095  0.575   0.5653
birth rank       0.10956   1.11578  0.13121  0.835   0.4037
---
Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

                exp(coef) exp(-coef) lower .95 upper .95
sibdead3           1.6105     0.6209    0.7123     3.641
sibdead3_7         2.7407     0.3649    1.2290     6.112
sibdead7_15        1.5744     0.6352    0.7075     3.504
famsize            0.8248     1.2123    0.3999     1.701
dadD               0.6864     1.4570    0.2362     1.994
momD               0.8836     1.1317    0.2773     2.816
cohort             1.0238     0.9767    0.9449     1.109
birth rank         1.1158     0.8962    0.8628     1.443

Concordance= 0.571  (se = 0.056 )
Rsquare= 0   (max possible= 0.005 )
Likelihood ratio test= 11.25  on 8 df,   p=0.2
Wald test            = 10.08  on 8 df,   p=0.3
Score (logrank) test = 10.94  on 8 df,   p=0.2
```

```
                  rho    chisq     p
sibdead3     -0.021243 0.098871 0.753
sibdead3_7    0.000264 0.000014 0.997
sibdead7_15   0.096976 1.775006 0.183
famsize       0.008090 0.012511 0.911
dadD          0.015755 0.061397 0.804
momD          0.043667 0.391964 0.531
cohort        0.047515 0.535013 0.465
birth rank   -0.083484 1.763200 0.184
GLOBAL              NA 6.927525 0.544
```

Chapter 3

Famine related mortality in early life and accelerated life histories in 19th Century Belgium

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Famine related mortality in early life and accelerated life histories in 19th Century Belgium

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Abstract

Density-dependent and extrinsic mortality are predicted to accelerate reproductive maturation. The first 5 years of life is a proposed sensitive period for life-history regulation. This study examines the effect of local mortality during the sensitive period on subsequent marriage timing in 19th Century Belgium (N women= 13,179; N men= 16,688). Local mortality during the sensitive period was inversely associated with age at first marriage for men and women controlling for literacy, occupational status, population growth, and migration. Cox regression indicated decreased time to marriage for women (HR=1.627, 95% CI: 1.521-1.740) and men (HR=1.328, 95%CI: 1.247-1.413) from high mortality municipalities. Rising population growth rates were associated with earlier marriage for men, but not for women. Migration (rural-rural and urban-rural) was associated with later marriage for men and women. Consistent with life history predictions, harsh ecological conditions such as famine influenced marriage timing.

Keywords: life-history theory, mortality, marriage behaviour, Belgium

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Introduction

Recent studies indicate effects of high mortality environments in early childhood on marriage behaviour (1, 2). However, little is known about life-history (LH) effects on marriage timing when marriage is largely decoupled from biological maturation. European historical populations provide a good testing ground, because the link between marriage and childbearing was very strong: marriage was an indicator of social maturity and marked the onset of sexual reproduction. In Belgium, the onset of the fertility transition, which further decoupled marriage from reproduction, began around 1880 in the French-speaking parts (3). Prior to that, lack of access to effective means to limit fertility, as well as a lack of willingness to use them, meant that fertility was not limited to a desired family size, and reproduction began soon after marriage. Marriages, however, were comparatively late: mean age at first marriage was 30.5 years for men and 28.6 for women in 1846-56 (4), and thus occurred well after physical maturity. Likewise, births outside of marriage were relatively rare (4). Hence, timing of marriage and first birth were less dependent on physical maturation, similar to marriage patterns in contemporary high-income populations. Do LH predictions hold in the case of a substantial culturally imposed lag between physical maturation and age at marriage?

Life-history theory (LHT) predicts that harsh and uncertain living conditions accelerate maturation to compensate for the probability of fitness failure due to extrinsic risk (5) and density-dependent mortality (*e.g.*6, 7, 8). Mortality risk is predicted to accelerate reproductive maturation and interbirth intervals (9) in what is sometimes referred to as "psychosocial acceleration". In psychosocial acceleration the first 5 to 7 years of life is a sensitive period shaping reproductive development. Previous work has shown that even in utero exposure to harsh environmental conditions such as epidemiological stress can affect life expectancy negatively and leads to reduced fertility (number of children) as well as an earlier onset of childbearing (10, 11). Effects of mortality exposure during childhood, however, appear to be complicated by multiple factors (12): Extrinsic (or unpredictable) and density-dependent mortality can influence biological maturation and reproductive decision-making throughout the life course (13, 14), and energy availability and environmental predictability show substantial collinearity across human populations (15). The current study investigates the impact of high mortality cues on male and female marriage behaviour. We aim to improve the understanding of LH decisions around a cultural norm for relatively late marriage among historical and pretransitional human populations. Based on LHT, we predict that local famine related mortality (7, 16) influences marriage timing when marriage and physical maturation are largely decoupled. Following psychosocial acceleration theory (17, 18) we hypothesize that local mortality rates in the first 5 years of life influence timing of marriage even when marriage tends to follow puberty by many years. Marriage is predicted to occur earlier in communities experiencing high mortality, compared with communities that were little affected by famine and experienced comparatively low mortality.

Data and methods

Study population and data collection

We examined associations between mortality and marriage timing in the context of an ongoing food crisis due to potato blight in 19th century Flanders. Data were collected from population censuses, statistical yearbooks and civil registers. The study population (N women= 13,179; N men= 16,688) was selected based on a database of marriage certificates from the province of Flemish Brabant (then including the capital of Brussels) from people who were born throughout Belgium between 1841-1850. Mortality data were collected at the municipal level from population censuses and statistical yearbooks. We obtained the morality data from the Historical Databases of Local and Cadastral Statistics (LOKSTAT-POPPKAD), Ghent University, Quetelet Centre. We included women between 15-40 years and men between 15-50 years.

Early-life mortality pressure and population growth

In the 1840s and 1850s, the mean crude death rate in Belgium was 23.4 (4). However, local and regional variation in crude death rates was high – ranging, *e.g.*, in 1841 from 3.7 to 57.1 and in 1847 from 0.0 to 100.2 per 1,000 among communities in our dataset. Such high local variability is identified in LHT as potentially salient for understanding individual differences in mating strategies (7). Two dimensions of mortality are thought to shape this process: overall mortality rates, and the ratio of juvenile to adult mortality (7, 16). In this paper, we focus on overall mortality rates that could be determined at the local level for all 2,538 Belgian communities for the period 1841-1850. Adult-juvenile mortality rates were only available at higher levels of aggregation.

We selected individuals born between 1841 and 1845 because it was a period of local food crisis. Harvests in 1846 to 1850 were only 40 to 60% of what they had been before, and potato blight destroyed around 87% of the 1845 harvest of Belgium's staple food. This led to subsistence crisis in a period of rapid population growth - a crisis that was characterized by local variation and affected some areas (*e.g.* West Flanders – see Figure 1) much more deeply than others (19). We included the community level crude death rates (CDRs) pertaining to the first five years of each individual's life, which are considered key developmental years in LHT (9, 20). Because our key interest is with the impact of exposure to extreme mortality conditions, we categorized CDRs in three different categories that led to approximately equal exposure group sizes (1=low (CDR <20); 2=middle (CDR between 20 and 32); 3=high (CDR >32)).

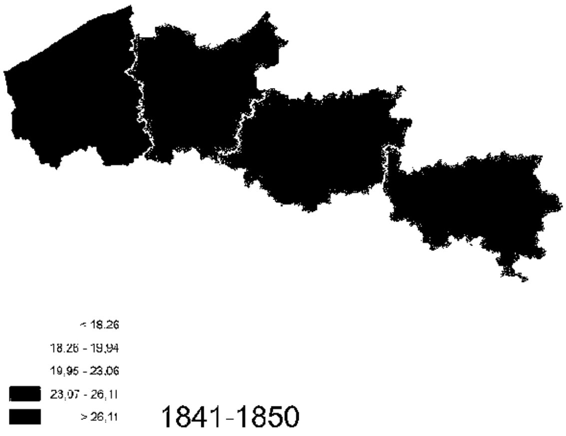

Figure 1. Number of deaths per 1000 inhabitants in 1841-1850

In rapidly growing populations, future reproduction is discounted as a function of population growth (21). The population of Belgium grew between 0.7 and 1% per year from 1810 to 1850. The mortality peaks in the crisis years 1845-1847 led to a decrease in the population growth in 1847 (19). We included the local average crude population growth during an individual's first five years of life to evaluate its effect on the timing of first marriage.

Marriage timing

We examined timing of marriage over three levels of local mortality in the first five years of life for men and women separately. Men and women were analysed separately due to substantial differences in their marriage behaviour that could obscure important gender interactions with other predictor variables.

In Belgium the proportion of unmarried women was decreasing between 1846 and 1910 (see Figure 2). Marriage was a state that individuals desired and generally entered at one point in their lives. The proportion of unmarried 25-29 year old women fell from 60% to 37% and for 40-45 year old women from 22% to 17% in the abovementioned period (22).

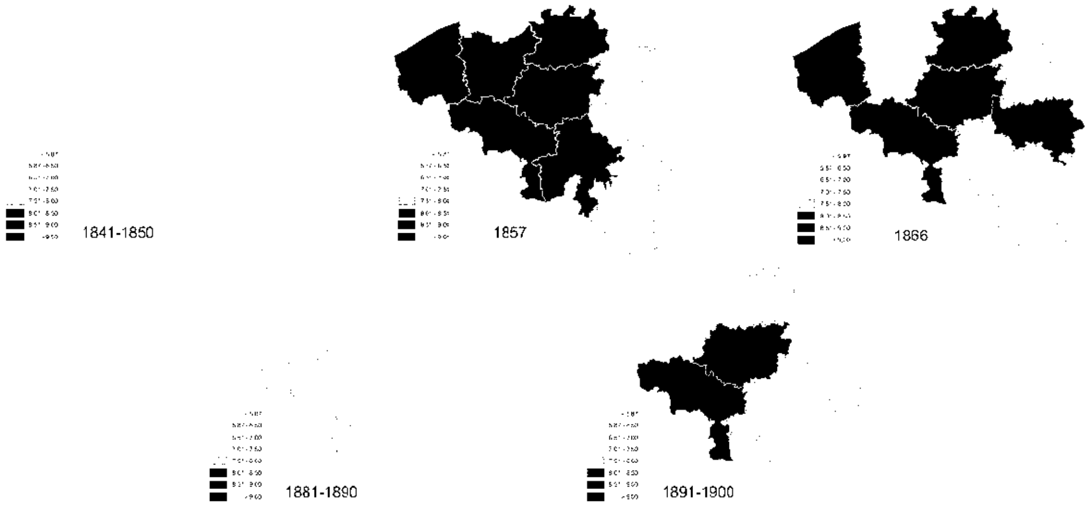

Figure 2. Number of marriages per 1000 inhabitants in 1841-1850, 1857, 1866, 1881-1890 and 1891-1900

Because higher occupational class is associated with later age at marriage (23, 24), we control for the effect of occupational class (HISCLASS) by including the profession of the groom in the model for men, and the occupation of the father of the bride for women. In the study period, brides' occupations were not systematically recorded, and therefore we use the father of the brides' occupation as a proxy for her socio-economic status. We categorized the HISCLASS variable into four categories (1= low; 2=farmer; 3=middle; 4=high). We also controlled for the ability of groom and bride to sign the marriage certificate. Literacy is used as a proxy for education in historical-demographic studies (25, 26). People who were literate married later and therefore had a lower overall fertility (27).

Marriage timing can be affected by migration. This has been observed for Belgium in the 19th century context in studies on Antwerp, where migrants married later than locals, and the impact of migration on marriage timing depended on the nature of the migration, with mean age at first marriage lower for domestic migrants than for international migrants (28). To control for the effect on migration, we included a variable in all models specifying whether an individual had migrated between birth and marriage, by comparing birthplace and marriage place with each other. We categorized the migration variable in four categories (0= non-migration; 1= rural-rural migration; 2= rural-urban migration; 3= urban-rural migration).

Of the 19 municipalities of the current Brussels-Capital Region only eight were urbanized at the time period under investigation: City of Brussels, Sint-Joost, Sint-Gilles, Elsene/Ixelles, Koekelberg, Anderlecht, Sint-Jans-Molenbeek and Schaarbeek. These eight municipalities were coded as urban, whereas the remaining 11 municipalities were coded as rural.

Life-history behaviours also may be influenced by population growth rates. In a rapidly growing population, delaying reproduction results in relatively reduce reproductive value because future reproduction is discounted as a function of population growth, expressed as e-r(y-x) in R.A. Fisher where r is the rate of increase and (y-x) is the delay in reproduction (21).

Statistical Analysis

Cox regression analyses were used to model time to first marriage. To account for the mix of individual and community-level data, we included a theta parameter for random effects between provinces. This parameter controls for unobserved heterogeneity between provinces (29).

Results

The crude death rate (CDR) varied between the years 1841-1850 with a peak in 1847 and 1849. The mean CDR in the first five years among the communities in the brides' sample was 26.14 (SD: ± 5.62), and 25.55 (SD: ± 5.40) among the communities in the grooms' sample. The mean age at first marriage among brides was 26.14 years (SD: ± 4.78 years) and among 29.35 years among grooms (SD: ± 6.20 years). This is in line with the marriage pattern during this time period (4). 55.9% of brides were capable of signing the marriage certificate, and 70,2% of grooms. 65.7% of the brides were so called 'stayers' (no migration) and 47.26% of grooms. Individuals who migrated from rural to urban settings (eight municipalities of Brussels-Capital Region) covered around 1/3 in the brides' and grooms' sample. The CDR was strongly associated with local population density (groom: r=0.64; brides: r=0.69). When CDR and population density entered models together, they caused substantial collinearity. Hence, mortality effects examined here include density-dependent and extrinsic mortality, both of which predict accelerated maturation in LH models (8, 30).

Descriptive Kaplan-Meier curves suggest that there is a general positive association between high community level mortality in early childhood and age at first marriage among both men and women (see Figures 3 and 4).

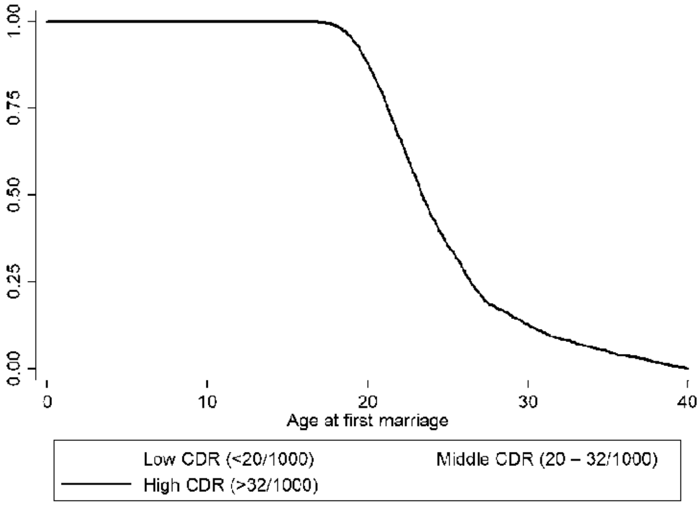

Figure 3. Kaplan-Meier curve of transition to marriage among women by CDR exposure (low, medium, high) during the first five years of life

This association was confirmed by the results of Cox regression (see Table 1 and Table 2), which estimated the effect of community level mortality in early childhood (0-5 years) on age at first marriage, are summarized in Table 1 and 2.

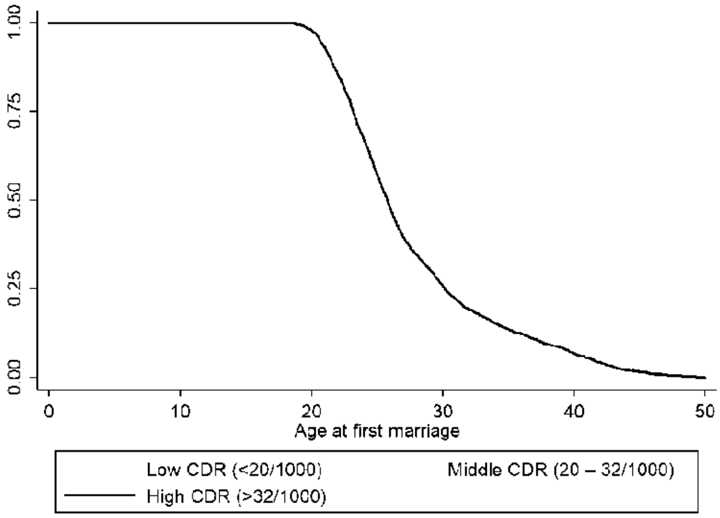

Figure 4. Kaplan-Meier curve of transition to marriage among men by CDR exposure (low, medium, high) during the first five years of life

Women who were exposed to high mortality during the sensitive period married significantly earlier than women who were not exposed to high mortality (hazard ratio: 1.627***; Table 1). Men who were exposed to high mortality during early childhood were significantly younger when they married (hazard ratio: 1.328***; Table 2). Furthermore, our results indicate that the average community-level population growth rate during the first five years had a significant effect on men's age at first marriage, although the effect size was rather small (hazard ratio: 1.028***; Table 2,). We observed a similar trend among women, although this trend was not significant.

Variable	HR	St.Er.	z	p-value	95% Conf. Interval	
Total CDR						
(Ref.Cat.: Low <20/1000)						
Middle (20 – 32/1000)	1.103	0.031	3.530	0.000	1.044	1.165
High (>32/1000)	1.627	0.056	14.180	0.000	1.521	1.740
HISCLASS father bride						
(Ref.Cat.: High)						
Low	0.764	0.067	-3.090	0.002	0.644	0.906
Farmer	0.828	0.074	-2.110	0.034	0.696	0.986
Middle	1.029	0.093	0.320	0.749	0.863	1.228
Bride literate	0.814	0.015	-11.340	0.000	0.786	0.844
Total population growth	1.003	0.005	0.620	0.538	0.994	1.012
Bride migration status						
(Ref.Cat.: Not migrated)						
Rural-rural migration	0.916	0.022	-3.610	0.000	0.873	0.961
Rural-urban migration	1.025	0.033	0.760	0.446	0.962	1.091
Urban-rural migration	0.613	0.042	-7.080	0.000	0.535	0.702
N						13,179
Theta	0.016	0.011				
Likelihood ratio test	26.460					
p-value likelihood	0.000					

Table 1. Effects of community level mortality on women's age at first marriage

The effect of migration on women's and men's marriage behaviour varied between categories. Both women and men who migrated from rural settings to large cities showed no significant effect in terms of their age at first marriage. Individuals who migrated from rural-rural and urban-rural were significantly older at their first marriage than individuals who married in their natal community (women: hazard ratio: 0.916***, hazard ratio: 0.613***; Table 1; men: hazard ratio: 0.903***, hazard ratio: 0.743***; Table 2). For the rural-rural migrants the effect size was very small (see Table 1 and Table 2). For urban-rural migrants by contrast the transition to marriage was substantially delayed. These groups, however, were very small in numbers (1.71% of brides and 1.98% of grooms).

Variable	HR	St.Er.	z	p-value	95% Conf. Interval	
Total CDR						
(Ref.Cat.: Low <20/1000**)**						
Middle (20 – 32/1000)	1.096	0.026	3.800	0.000	1.045	1.149
High (>32/1000)	1.328	0.042	8.870	0.000	1.247	1.413
HISCLASS groom (Ref.Cat.: High)						
Low	1.295	0.073	4.560	0.000	1.159	1.447
Farmer	0.998	0.058	-0.040	0.969	0.890	1.118
Middle	1.295	0.073	4.570	0.000	1.159	1.447
Groom literate	0.879	0.016	-7.230	0.000	0.849	0.910
Total population growth	1.028	0.004	6.420	0.000	1.019	1.036
Groom migration status						
(Ref.Cat.: Not migrated)						
Rural-rural migration	0.903	0.017	-5.420	0.000	0.871	0.937
Rural-urban migration	0.976	0.027	-0.850	0.393	0.924	1.031
Urban-rural migration	0.743	0.043	-5.130	0.000	0.663	0.832
N						16,688
Theta	0.009	0.006				
Likelihood ratio test	50.050					
p-value likelihood	0.000					

Table 2. Effects of community level mortality on men's age at first marriage

Similar to previous research, we found that men from a low and middle occupational class married significantly earlier (hazard ratio: 1.295 ***; hazard ratio: 1.295***; Table 2) compared with high occupational classes and farmers. For women the results instead show that daughters of lower-class fathers and farmer's daughters married later than their counterparts in the middle and upper class (see Table 1).

Using literacy as a proxy for education revealed that both men and women who were literate married significantly later (hazard ratio grooms: 0.879***; hazard ratio brides: 0.814***; see Table 1 and 2) than their illiterate counterparts.

Finally, we note that in both models, the theta-parameter controlling for unobserved heterogeneity is significant, indicating that first marriage ages differed significantly between provinces within Belgium. This is consistent with the literature on Belgium, where linguistic divides and economic heterogeneity across provinces coincided with differences in reproductive behaviour (4).

Discussion

The results indicate that exposure to harsh environmental conditions such as famine and crop failure in first five years of life lead to accelerated life-history, in this case earlier marriage. A link between exposure to high mortality and faster life history has been demonstrated for preindustrial and industrial populations (2, 11, 14). Recent studies have also shown that earlier marriage is associated with exposure to harsh early childhood conditions (1). Earlier work however, did not address the effects of famine and crop failure on marriage behaviour in men and women. Furthermore, previous research paid little attention to differences between men and women in their responses to environmental stress. We used marriage records from Flemish Brabant Province, Belgium in a period of crisis (1841-1850) to address this gap in the literature. Our results suggest that age at marriage, which was in part regulated by social conventions, responded to crisis mortality cues during a sensitive period in infancy and early childhood. This finding is consistent with psychosocial acceleration as predicted by life history theory. Life-history responses to crises may be complicated by conflicting psychological and physiological mechanisms associated with causes of death and related effects on fertility (8, 30, 31). Conflicts between energy availability and psychosocial motivation for fast life history require more attention across the life span in a variety of populations.

The nature of historical demographic data does not allow us to identify precise mechanisms by which local environments influence long-term reproductive decision-making. Presumably harsh environments cause less responsive parenting and reduced parental effort (32), which in turn induce fast life-history strategies including earlier maturation (age at menarche), sexual debut, and first birth (9, 17). For this historical Belgium population, even relatively early marriages occurred well after any plausible onset of menarche; hence, marriage timing is likely under some conscious control influenced by psychological factors linked to an accelerated life-history trajectory. Increased impulsivity and delay discounting are two psychological factors association with fast life history that appear to be relevant for mating and family formation behaviours (5).

Occupational status showed different effects for timing of marriage for men and women. Among men, the general pattern requiring more education or training for relatively high-status occupations may have delayed marriage for higher status men. Furthermore, men often needed to wait for an inheritance from the parental generation to establish a socially viable marriage. This leads to higher hazard ratios for low occupational status men. Among lower class women marriage was delayed compared to middle- and upper-class women. Many lower-class women worked as domestic servants which was seen as an important preparation for marriage and a life stage to accomplish before marriage (33). Likewise, farmer's daughters married later than their middle- and upper-class counterparts, possibly because they were an important labour resource for family farms.

Literacy was associated with delays in marriage for both men and women, a pattern that is commonly seen in studies of fertility in low-income and developing populations (23).

Rural-rural migration and urban-rural migration showed a significant effect for women and men but differences in timing of marriage were large only among urban-rural migrants. In general migration is often associated with social exclusion (28). In the 18th century the choice of a marriage partner was mostly controlled by the natal family (34). Since social control by the natal family were quite strong in rural settings, rural-rural and urban-rural migration might have induced later marriage among migrated women and men in our sample.

The impact of population growth rates is an understudied aspect of life-history strategies. In rapidly growing populations, future reproduction is discounted as a function of population growth. The significant effects of population growth on men's age at marriage might be understood in the light of growing competition in the marriage market. In both of our models the effect size of total population growth is relatively small.

One important limitation of this study is that people who never married are excluded from the analysis due to limitations of the available dataset, which consists exclusively of marriage records. Inclusion of never-married individuals as censored cases could reduce hazard ratios reported here. This however, would require nationwide collection of linked birth and death records, which was not feasible in the context of this study. It is possible that the decision to never marry was also influence by exposure to high mortality in early life during the crisis years.

A second limitation is that the crude death rate does not allow us to assess effects of age-specific mortality on age at first marriage. LH models sometimes specify adult vs. juvenile mortality as important influences on reproductive behaviour (30). We assume that variation in mortality related to crop failure likely had the strongest effect on children and elderly people. Furthermore, though formal life-history models identify age-specific mortality patterns, we wonder whether human environmental perception is fine-tuned enough to respond to age-specific rather than general mortality trends such as CDR.

In sum, a growing body of research on historical populations suggests an important role of mortality on shaping human reproductive strategies. The current study indicates that famine or crop loss induced mortality is significantly associated with earlier marriage in contexts were marriage timing was decoupled from physiological maturity.

Acknowledgements

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Chapter 4

Sex Differences in Intergenerational Income Transmission and Educational Attainment: Testing the Trivers-Willard Hypothesis

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ORIGINAL RESEARCH

Sex Differences in Intergenerational Income Transmission and Educational Attainment: Testing the Trivers-Willard Hypothesis

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From an evolutionary point of view, sex differences in intergenerational transmission of income may be influenced by the Trivers-Willard (T-W) effect: Low status parents should invest more in daughters, whereas high status parents are expected to invest more in sons. This bias in parental investment may result in status-dependent sex biased parental support for higher education and educational attainment and should therefore affect the level of intergenerational income transmission for the sons and daughters. We used the data from the Wisconsin Longitudinal Study (WLS) to model the effect of parental financial investment on the child's income and educational attainment controlling for the number of siblings. The observed sex differences in intergenerational income transmission demonstrate that sons profited more from parental income and education in terms of their own income than daughters. Furthermore, we showed that fathers with a high socioeconomic index (SEI) invest more in their sons' education in terms of completed years of education and financial support during college. In contrast daughters of low SEI fathers completed more years of education and received more financial support than sons of low SEI fathers. However, the pattern in intergenerational income transmission might be better explained as a product of sociological factors and reproductive trade-offs in later life rather than as a consequence of the T-W effect.

Keywords: socioeconomic status, parental resources, income, educational attainment, Trivers-Willard hypothesis, sex differences

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1. INTRODUCTION

In modern western societies, access to resources is mainly determined by wealth that in turn is generated by income and to some degree by inheritance. Parental resources contribute to children's wealth in a variety of ways: Not only do parents leave substantial sums to their offspring upon their death, but they actively invest in their children's education and career opportunities which in turn may increase their offspring's future income. However, daughters and sons may not profit equally from their parents' resources ([illegible]).

From an evolutionary point of view, men's striving for wealth and status can be explained by the association between their access to resources and higher reproductive success. The effect of status on male reproductive success has been demonstrated both in modern and historical populations. Men with high social status father more children in modern western societies ([illegible]) and in hunter-gatherer societies samples ([illegible]). By contrast, women's income and occupational attainment negatively affect reproductive success ([illegible]). Parents' incomes not only influence the number of children, but also their children's quality in terms of status, which in turn affects the number and quality of their grandchildren. Trivers and Willard ([illegible]) predicted that parental investment should be directed in a larger proportion to the subset of their offspring that are likely to produce more offspring in turn. Low status males are less likely to reproduce than low status females, while high status males are expected to produce more offspring than high status females ([illegible]). Therefore, it can be expected that low status parents direct their resources toward their female children, whereas high status parents should favor male children. While the Trivers-Willard (T-W) effect has been demonstrated in many species (for a review see [illegible]), evidence in human societies is inconclusive ([illegible]). [illegible] found such effects in data from historical India, China and Europe: Low class families tended to favor daughters more than upper class families. [illegible] showed among the low status Mukogodo in East Africa that parents biased their investment in favor of their daughters. The results are less consistent in contemporary industrial societies: [illegible] showed that daughters of high status Americans attain a lower educational status than sons, whereas daughters of low status Americans attain a higher educational status than sons. [illegible] showed in a Polish sample that sons with higher educated fathers were breastfed for a longer time than girls. [illegible] found no differential parental investment in the time per week spent with sons and daughters respectively, nor in measures of breastfeeding and self-perceived relationship quality. [illegible] argues that these conflicting findings may be the results of self-reported proximate measures of paternal investment, since parents would have to admit to favoring one child. She further argues that wealthy societies may not have large sex differences in these certain kinds of investment at the early stage of childhood, therefore parental support for educational attainment may be a more appropriate measure to investigate a status-dependent sex bias in parental investment.

Many studies in sociology have demonstrated the importance of parental investment on offspring's status attainment (e.g. [illegible]). Few of them have focused on the effect sizes of these factors (e.g. [illegible]). All of these studies have focused on the interaction between a father's education and occupational attainment on the son's education, occupational attainment and income, but disregarded the effect of the mother's socioeconomic status and the influence of parental status on daughters. Therefore, it is difficult to judge to which extent parental status can be considered a relevant influence on the offspring's position in society and whether a T-W effect exists in a population. In order to compare the benefit from parental education vs. parental income for both sexes, this study focuses on quantifying the effects in easily interpretable units.

The association between parental income and children's incomes is often expressed as an intergenerational income elasticity ([illegible]). Intergenerational income elasticity describes the percentage of the parental income deviation from the mean that is transmitted to their children. For instance, at an intergenerational income elasticity of 50% children whose parents earn $1,000 more than the average for their country are expected to earn $500 more than average ([illegible]). For modern western societies, this measure varies between 0.115 and 0.662 for sons ([illegible]). Because of women's traditionally limited access to the labor market, the effects of parental income on daughters' income are often disregarded with the notable exceptions of [illegible] and [illegible]. These studies reported an intergenerational income elasticity between 0.39 and 0.41 ([illegible]) for daughters. In general, parental income is a weaker predictor of future annual income for daughters than for sons. [illegible] used a path model to estimate the effects of the father's income and education on the son's income for the Wisconsin Longitudinal Study (WLS) dataset. He concludes that the effects of the father's education, occupational attainment and other socioeconomic background variables are negligible when correcting for their correlation with paternal income.

In addition to parental income and education, the number of siblings may affect the amount of resources that parents invest in each child ([illegible]). [illegible] showed that a larger number of children lead to a lower frequency of interactions between each parent and child. Low parental involvement is associated with high rates of high school dropout and low educational attainment (e.g. [illegible]). A higher number of siblings may therefore negatively affect a child's income.

In contrast to other studies this study aimed to test the T-W hypothesis in a sample where respondents had already completed their reproductive lives. Furthermore, we compared children's yearly income with their parents' yearly income at a point in time where both of them had similar ages. We tested the T-W hypothesis by using children's yearly income, their educational attainment and their parents' ability to financially support them in college as a proxy for non-biological parental investment. We aimed to quantify the effects of parental income on the next generation's income for the WLS data set. Both parents' education and their pooled income were used to predict their children's income. We controlled for the number of siblings and compared the magnitude of the above-mentioned effects for sons and daughters.

Some scholars argue that the socioeconomic index (SEI) is a more appropriate measure for testing the T-W hypothesis than parental income and parental education ([illegible]). Therefore we tried to replicate the findings of [illegible] with the WLS. We used SEI of fathers to predict their children's educational attainment. As an additional measure of parental investment, we compared the extent to which parents supported their sons and daughters to go to college.

We expected both parental education and parental income to augment children's income. Based on the T-W hypothesis we predicted that sons profit more from parental resources in terms of their income than daughters. We expected the sex difference in educational attainment to differ for sons and daughters of fathers with different SEI. We predict daughters of low SEI fathers to have a higher educational attainment relative to sons in comparison to daughters of high SEI fathers. In addition, we expected daughters of low SEI fathers to be more supported in terms of their college education relative to sons when compared to daughters of high SEI fathers.

2. MATERIALS AND METHODS

WLS (http://www.ssc.wisc.edu/wlsresearch/) goes back to the state-sponsored questionnaire administered in 1957 to all students in all Wisconsin high schools in their final high school year. One third of this original cohort was randomly selected for further data collection. This random sample contains data from 10,317 Wisconsin high school graduates (5,326 women and 4,991 men) born between 1937 and 1940.

In 1957 and in 1964 information about the respondents' parents was collected such as *parental income* in $100 and the *parental education*, which comprises the *father's education* in years and *the mother's education* in years. *Parental income* was collected from the Wisconsin state income tax records for 1957–1960 and includes income generated by the mother and the father. The father's mean age at that time was 50 years (SD = 7 years) and mother's mean age was 46 years (SD ± 6 years). At that time, respondents were between 19 and 21 years old (mean age: 18 years, SD = 0.5 years). To measure familys social status we used *father's SEI* when respondents were in their final high school year coded using Duncan SEI scores ([illegible]). The value from the Duncan SEI score ranged from 1.00 to 96.00 in the WLS. Respondents were asked to state their sex coded male = 1, female = 0. In addition, respondents were asked to state their *total number of siblings ever born*. In the 1992/1993 follow-up, the respondents were asked to report their most recent *income* in US dollars before deductions. Income was measured when the respondents were between 52 and 55 years old (mean age 53 years, SD ± 0.5 years). We selected this time period instead of an earlier one where respondents were between 35 and 36 years (mean age 36 years, SD = 0.5 years) because at this point women's reproductive periods are over, allowing them to return to the labor market. In addition, at this point in live, the income has already reached a stable level ([illegible]) and the age of respondents (mean age 53 years, SD ± 0.5 years) is comparable to the mean age of their parents at the time the parental income was measured. In our analysis, we included only respondents who reported a non-zero income. We did not differentiate between part-time and full-time workers. As an additional proxy for parental investment we used respondent's reported *education* in years. Furthermore, we used the variable *parents are able to support college* measured in three categories (cannot support college = 1, can support college with scarifies = 2 and can support college easily = 3).

2.1. Statistical Analyses

Since the distributions of *respondents' income* and *parental income* are skewed, we performed a square-root transformation of both parameters before computing the regression models. Based on the descriptive analysis of the dataset, we designed a model woman and a model man for whom we then could compare the effects of parental status predicted by our regression model.

To model income, we started with a linear regression model predicting the square-root of the *respondent's income* from the *total number of siblings*, the square-root of the *parental income*, *mother's education*, *father's education* and the *respondent's sex* as well as all interactions between sex and the remaining independent variables. Because of multicollinearity, we could not include birth order in our models as it is highly correlated with the number of siblings ($r = 0.525$, $N = 9{,}524$, $p < 0.001$). For the same reasons we could not use the number of brothers ($r = 0.756$, $N = 9{,}526$, $p < 0.001$) and the number of sisters ($r = 0.744$, $N = 9{,}526$, $p < 0.001$). In a stepwise algorithm with the AIC as a criterion, we eliminated all irrelevant interactions. To evaluate the robustness of our model's coefficients, we repeated our analysis omitting one of the independent variables (and, if applicable, the interaction between the variable and the respondent's sex) at a time. We calculated the derivatives of the square of the income model to be able to quantify the effects of the independent variables in terms of dollars earned per year. Due to the fact that intergenerational income transmission favors sons over daughters we used educational attainment and parents' ability to support college as a proxy of parental investment. We started with a linear regression model predicting the *educational attainment* from *total number of siblings*, the *father's SEI* and the *respondent's sex* as well as all interactions between sex and the remaining independent variables. We used a stepwise algorithm with the AIC as a criterion to eliminate all irrelevant interactions. To model parental ability to support college we used a ordered logistic regression model predicting *parents' ability to financially support child in college* from *total number of siblings*, the *father's SEI*, and the *respondent's sex*. In the two models where we used *father's SEI* as a proxy for parental status we didn't include *father's education* and *mother's education* due to the fact that *father's education* is incorporated in the variable *father's SEI*. Statistical analyses were carried out with R version 3.2.2 ([illegible]) and SPSS 24.

3. RESULTS

3.1. Descriptives

Measures of central tendency and variance of distribution for all variables used in the empirical analysis are reported in **Table 1**. For the comparison of the effects of early life factors on women and men, respectively, we examined a model woman and a model man: Each has three siblings (the rounded mean number of siblings), parents who earned the median income for this sample, a father with ten years of schooling (rounded mean) and a mother with eleven years of schooling (rounded mean).

TABLE 1 | Descriptive statistics: model women and model men.

Variable	Women	Men	All
Median parental income in $	5,400	5,400	5,400
(Q1)	(3,400)	(3,400)	(3,400)
(Q3)	(7,400)	(7,400)	(7,400)
Mean father's SEI	30.3	30.4	30.4
(S.D.)	(22.1)	(22.2)	(22.1)
Median respondent's income in $	18,000	41,000	30,000
(Q1)	(10,000)	(20,000)	(16,000)
(Q3)	(30,000)	(60,000)	(45,000)
Mean mother's education (yrs)	10.6	10.7	10.6
(S.D.)	(2.3)	(2.3)	(2.3)
Mean father's education (yrs)	10.7	10.4	10.5
(S.D.)	(3.1)	(3.0)	(3.1)
Mean respondent's education (yrs)	13.0	13.8	13.5
(S.D.)	(1.2)	(2.0)	(2.1)
Mean number of siblings	3.2	3.0	3.2
(S.D.)	(2.3)	(2.3)	(2.3)

TABLE 2 | Effects of parental income[a] on respondent's income[a].

Variable	Est. of Coefficient	Std. Error	*t*-value	*p*-value
Intercept	103.783	6.134	17.033	<0.001***
Parental income[a]	1.006	0.503	1.993	0.047*
Father's education (yrs)	0.576	0.490	1.175	0.240
Mother's education (yrs)	1.346	0.494	2.723	0.006**
No. of siblings	0.116	0.097	1.162	0.245
Sex (male = 1)	28.037	8.113	3.463	<0.001***
Sex*Father's education (yrs)	1.763	0.694	2.526	0.011*
Sex*Mother's education (yrs)	1.332	0.713	1.860	0.064
Sex*Parental income[a]	2.0167	0.700	3.091	<0.001***

[a]Variable is square root transformed. *p < 0.05, **p < 0.01, ***p < 0.001.

3.2. Effects of Parents' Status on Their Children's Income

3.2.1. Model Coefficients

As expected, both the father's and the mother's education contribute significantly to their child's income (**Table 2**). However, the size of the effect depends on the child's sex. An additional year of education to the mean duration of the father's education benefits a son by additional $873.73 per year. Compared to this, a daughter gains only $156.62 to her yearly income. In contrast, an additional year of the mother's education (12 instead of the mean number of 11 years of schooling) benefits a son's income by additional $1,008.04 per year, whereas a daughter's yearly income increases by $368.98 (**Table 2**).

In accordance with our prediction, the child's sex not only affects his or her income, but also the extent to which this income is determined by parental status (**Figure 1**). Not only is women's income lower in this sample, women also benefit less from their parents' income in terms of their own income. If only respondents with a regular income are considered, women's median income is $18,000 per year compared to men's median income of $41,000. Taking into account the effects of parental income and of the interactions with sex, for each extra 100 dollars parents of a median income earn per year, a model daughter gains $19.72 of additional income per year. In contrast, a comparable model son benefits by additional $94.82 per year.

The number of siblings does not affect the respondent's income significantly in our main model. However, if either the parental income or the mother's education is omitted as a predictor, the number of siblings has a significant, yet only a small negative effect on the respondent's income.

3.2.2. Fit and Robustness of Coefficients

The income model explains 29% of the observed variance in respondent's income. As there is some degree of collinearity in our predictors *father's education* and *mother's education* are correlated with *r* = 0.491 (Spearman correlation, *N* = 9,228, *p* < 0.001). *parental income* is correlated with r = 0.347 to *father's*

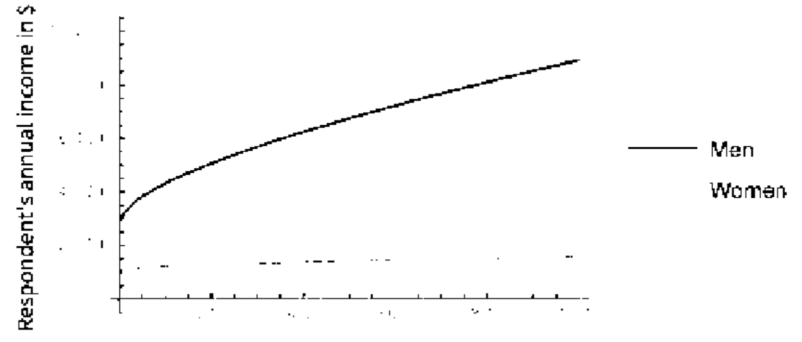

FIGURE 1 | Effect of parental income on respondent's income for men and women.

education (Spearman correlation, $N = 8,296$, $p < 0.001$), we repeated the analysis omitting one of our independent variables at a time to observe the change in the estimate of coefficients (see **Table 3**).

3.3. Model Educational Attainment

Father's SEI, an alternative proxy for parental status, significantly influences children's educational attainment. Similar to our income model, the effect size is strongly influenced by child's sex. The interaction effect of sex and *father's SEI* shows that on average sons of high SEI fathers achieve a higher educational attainment than the daughters. In contrast to our income model the *number of siblings* has a significant effect on the respondent's educational attainment. Respondents with a higher number of siblings are less likely to obtain more education on average than their counterparts. This model explains 12% of the observed variance of respondent's educational attainment (see **Table 4**).

3.4. Model Parental Ability to Support College

The negative effect of *sex* shows that sons of low SEI fathers with no siblings receive less financial support in college than daughters of low SEI fathers with no siblings. The positive coefficient of father's SEI shows that daughters of high SEI fathers with no siblings receive more financial support in college than daughters of low SEI fathers with no siblings. The positive interaction between *sex* and *father's SEI* shows that on average sons of high SEI fathers with no siblings receive more financial support in college than the daughters of high SEI fathers with no siblings (see **Table 5**).

4. DISCUSSION

In this study we have quantified the effects of parental socioeconomic status—measured by parental income and education—on the next generation's income. Using the WLS data set we have demonstrated that both parental income and parental education contribute strongly to their sons' income and

TABLE 4 | Effect of father's SEI on respondent's educational attainment.

Variable	Est. of Coefficient	Std. Error	t-value	p-value	
Intercept	12.875	0.074	173.116	<0.001	***
No. of siblings	0.106	0.011	10.186	<0.001	***
Father's SEI	0.022	0.001	13.654	<0.001	***
Sex (male = 1)	0.494	0.093	5.187	<0.001	***
Sex:Father's SEI	0.006	0.002	2.603	0.003	**

**p < 0.01; ***p < 0.001.

TABLE 5 | Effects of father's SEI on parental ability to financially support college.

Variable	Est. of Coefficient	Std. Error	t–value	p–value	
No. of siblings	0.170	0.016	10.413	<0.001	***
Father's SEI	0.020	0.002	10.005	<0.001	***
Sex (male = 1)	0.320	0.120	2.412	<0.001	***
Sex:Father's SEI	0.025	0.002	11.207	<0.001	***
Sex:No. of siblings	0.013	0.026	0.513	<0.001	***
Intercept 1	1.025	0.036	10.400	<0.001	***
Intercept 2	1.616	0.102	12.727	<0.001	***

***p < 0.001.

TABLE 3 | Effects of omitting independent variables on our income model.

Without	Ic	Sib	Ef	Em	pI	Sex	SEf	SEm	SpI	Fit
Sib	100.104 (5.735) ***		0.019 (0.490)	1.386 (0.405) **	1.102 (0.554) *	72.040 (9.111) ***	1.774 (0.835) *	1.359 (0.716)	2.045 (0.740) ***	0.262
Ef	111.402 (5.646) ***	0.657 (0.320)		1.677 (0.406) ***	1.266 (0.614) *	62.026 (7.739) ***		2.243 (0.690) ***	2.061 (0.710) ***	0.265
Em	112.257 (5.420) ***	0.624 (0.320)	1.056 (0.453) *		1.279 (0.554) *	54.026 (7.102) ***	2.790 (0.626) ***		2.746 (0.720) ***	0.264
pI	114.235 (5.735) ***	0.756 (0.320)	0.870 (0.477)	1.456 (0.405) **		62.034 (7.070) **	2.427 (0.670) ***	1.006 (0.716) *		0.261

Ic, Intercept square root transformed for income.
Sib, Total number of siblings ever born.
Ef, Father's education; Em, Mother's education; pI, Parental income.
SEf, interaction sex and father's education.
SpI, interaction sex and parental income.
SEm, interaction sex and mother's education.
*p < 0.05; **p < 0.01; ***p < 0.001.

to a lesser degree to their daughters' income. This finding is consistent with the T-W hypothesis, but might as well arise from the difference in opportunities for men and women at this time period independent of differential parental investment. Firstly, the sample of high school students itself may be biased since parents may choose to send only one of their children to college due to financial reasons. Secondly, career opportunities differed for the women and men represented in this sample.

The observed sex differences in income transmission are consistent with [illegible] and [illegible] who showed that the intergenerational income elasticity for sons varies between 0.115 and 0.662. In comparison to these findings, the intergenerational income elasticity for daughters varies between 0.39 and 0.41 ([illegible]). However, most of these studies have either been focused on the elasticity for sons or have not taken parental education as well as the number of siblings into account. We demonstrated that parental education, namely father's education, seems to have a stronger indirect influence on son's income than on daughter's income. In contrast to the literature, we found that the number of siblings does not affect the respondent's income significantly in our sample. The intergenerational transmission of income between parents and sons in our sample is likely strong enough to affect the sons' reproductive success. This would be in line with [illegible] and [illegible] that have shown that men's income is positively related to reproductive success, whereas women's income negatively affects the number of their offspring.

The observed sex differences in educational attainment are in line with [illegible] who showed that sons of high SEI fathers had a higher educational attainment than daughters, whereas daughters of low SEI fathers had a higher educational attainment than sons. Furthermore, the result showed that high SEI father's are more likely to be able to financially support their daughters in college than low SEI fathers. Some but not all of our findings are consistent with the T-W hypothesis and with [illegible] whose study showed that sons of high SEI fathers were more likely to receive higher financial parental investment than daughters.

Previous studies tackled the T-W hypothesis with a number of measures of parental investment such as the months of breastfeeding ([illegible]), the time spent with the child ([illegible]), and how well they know their children's friends ([illegible]). Researchers agreed that parental support for educational attainment is an effective and costly form of investment and thus lends itself to investigating the T-W effect. In view of this, it is surprising that only 48% of studies report a T-W effect in modern human societies ([illegible]). Two main interpretations emerged why the T-W effect remains elusive. [illegible] argued that in order to unequivocally prove a T-W effect, all acts relying on conscientious forethought on the parts of the parents such as planning and supporting the education of offspring must be controlled for the extent to which economic prospects of the child are determined by sex, since a more basic model of cost vs. payoff may produce a better model of sex differences in attainment of social status, wealth and reproductive success. [illegible] cautions that contemporary western societies experience an abundance of resources that leads to high investment in all children. In the case of the WLS sample, both the above mentioned considerations apply: Since all parents could afford to send at least one child to college, they already represent an above average income sample in which the pressure to distribute resources unequally may be low. In addition, parental ability to support college might has been affected by different earning expectations based on the economic situation for daughters and sons of parents coming from different income and social classes.

The main limitations to our study concern the sample. The Wisconsin Longitudinal sample represents mostly white highly educated Americans born between 1937 and 1940. Since the transfer of wealth and status between generations depends on economic and cultural circumstances, our inferences cannot be extended to more modern or culturally different populations. A higher proportion of working women would likely lead to a stronger transfer of income from parents to daughters. Another difficulty lies in the interpretation of the estimate of the coefficients, as income is dependent on too many partially correlated factors to allow a model close to the true model—as evidenced by the proportion of variance explained by our model, 29%. In consequence, the estimates of the coefficients of correlated factors such as parental education and parental income are associated with a high degree of uncertainty. However, repeating the analysis while omitting one factor at a time reveals no major changes in which factors are statistically significant with the notable exception of the factor "mother's education": The estimated coefficient gains statistical significance when the mother's education is omitted as factors. In addition, variables reliably representing parental investment are difficult to find in historical datasets. While the parental support of higher education represents such an investment, it by no means covers all possible ways in which parents can invest in their offspring.

In conclusion, our results demonstrate an important heritable component of socioeconomic status between parents and sons, and to a lesser extent between parents and daughters. Both parental income and education increase a son's income and educational attainment significantly. In comparison, daughters profit little in terms of income, likely because they face a trade-off between childcare and career outcomes. The results of the present study are consistent with the T-W hypothesis. Future research is needed, however, to clarify whether the observed effects are driven by differential parental investment depending on parental status or solely the product of sociological factors and reproductive trade-offs in later life.

AUTHOR CONTRIBUTIONS

KEP contributed to the research design/conception, data analyses and writing of the manuscript. AS contributed to data analyses and writing of the manuscript. MF contributed and commented on the research design, writing of the manuscript, and contributed to data analysis.

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Chapter 5

General discussion and limitations

During our evolutionary past human fertility behaviour underwent several great transformations. The most drastic changes occurred during the first demographic transition, when populations went from high mortality/fertility rates to low mortality/fertility rates. Human reproductive behaviour is influenced by various different biological, ecological, economic and social factors. Hence, to get a better understanding of human fertility patterns, a broad and inclusive perspective of different scientific fields is required. To provide a complementary approach to topics that are well studied from a social science perspective such as giving birth out of wedlock, marriage behaviour and parental investment, this thesis applies an evolutionary approach. By incorporating the impact of biological factors alongside social ones on human fertility behaviour new insights can be gained. Chapter 2 and 3 focus on the ecological conditions that are influencing human reproductive decision-making and control for socioeconomic factors as well. Chapter 4, by contrast, focuses on parental investment strategies to ensure an individual's fitness maximisation/optimisation.

The research settings of the three chapters differ in terms of the time period covered. Chapter 2 and 3 focus on historical pre-transitional and transitioning European populations, while chapter 4 covers a contemporary post-transitional Western population. Chapter 2 investigates a dataset from the rural area of East Frisia (Germany) during the 18th and 19th centuries. The pre-modern Krummhörn population was located in a rural setting and was an agricultural society with large-scale farmers at the top of the social hierarchy. Chapter 3 uses data from the northern region of Belgium, the province of Flemish Brabant with the city of Leuven as a capital, during the 19th and 20th centuries. The industrialized region of Flemish Brabant with its small-scaled industry played a supportive role in Belgium's economic growth (Van Bavel, 2001). The selected historical datasets allow us to study the impact of high mortality cues on human reproductive decisions among two populations at different stages of demographic and industrial development, while controlling for different socioeconomic factors. Chapter 4 uses data from a post-transitional US population in Wisconsin state. This dataset allows for investigating the impact of sex-biased parental investment on the life course of individuals in terms of their educational attainment and income development.

Based on the present work three main conclusions can be drawn: First, the mortality experience of individuals in the sensitive period during early childhood has a strong influence on an individual's reproductive and marriage behaviour. Second, both local level mortality and family level mortality are shaping human fertility behaviour. Third, sex-biased parental investment has an impact on the later life outcomes of individuals. The combination of these three conclusions emphasises the importance of the rearing environment on the life trajectory of individuals. These three main conclusions are discussed in more detail in chapter 5.1.

Although this thesis advances our knowledge of the impact of biological and social factors on human fertility patterns, there are a number of limitations that deserves emphasis.

First, working with historical datasets that were collected from registers and archives bares the risk of incomplete data and the unavailability of certain data. In the case of the Belgium Flemish Brabant data (chapter 3), for instance, the observation time had to be restricted to the years 1841-1850 due to incomplete digitalization of mortality data at the municipality level in the years before 1841 and for the years 1850-1880. A comparison between individuals born during the years of the food crises and those who were born in the years before or after the crisis was therefore not feasible. To get a better understanding of human reproductive behaviour it is advisable that future studies compare cohorts before and after food crises with each other. Only then can we get a better sense of the impact of food crises on human's behaviour.

In the Krummhörn dataset (chapter 2) there was a substantial migration of young adults during the observed period due to limited access to land. However, the dataset does not cover individuals who migrated, and therefore no information either on their reproductive history and their death date. Knowing both, however, would be of interest since soon-to-be mothers of illegitimate children might have tended to migrate to bigger cities to flee into anonymity and avoid social stigma in their hometown.

Second, Western longitudinal datasets often have issues with representativeness. The Wisconsin Longitudinal study (WLS) data is not an exception to this since it only covers white highly educated individuals who were born between 1937-1940. Lower educated individuals and minorities are underrepresented in this dataset. As a result, the general conclusion of chapter 4 cannot be extended to more diverse populations.

5.1. Main conclusions

As mentioned above, three main conclusions are drawn from the studies carried out here, referring to *i)* individual mortality experience during early childhood and its impact on reproduction and marriage behaviour, *ii)* the effect of local level mortality and family level mortality on reproduction and marriage behaviour and *iii)* the impact of sex biased parental investment on offspring later life outcome. What these three conclusions have in common, is that they highlight the importance of including biological perspectives alongside sociological ones in order to enhance our understanding of human fertility behaviour.

i. Individual mortality experience during early childhood

Previous studies have shown that under harsh environmental conditions, such as high mortality, individuals accelerate their life histories and engage in riskier behaviour (*e.g.* Belsky et al., 2010; Griskevicius et al., 2011; Placek and Quinlan, 2012; Quinlan, 2007). As highlighted throughout this thesis individual mortality experience during early childhood has the strongest impact on human reproductive and marriage behaviour. During this sensitive period individuals unconsciously adapt their developmental/psychological processes according to the prevalent environmental conditions (*e.g.* Belsky et al., 1991; Kuzawa and Bragg, 2012). The environmental cues of the sensitive period in early childhood might modify an individual's time perspective. Offspring who grew up in an environment with high mortality cues might benefit from a present-oriented time perspective. For instance, individuals who have a present-oriented time perspective engage in riskier behaviour resulting in short-term rewards. Furthermore, they devaluate the future and do not engage in long-term investments or planning (Kavanagh and Kahl, 2016).

Translated to the context of chapter 2 a present-oriented individual might engage in premarital sexual intercourse with the risk of getting pregnant. The short-term rewards in this case are sexual intercourse and sexual pleasure. In an environment with high mortality cues an individual with a present-oriented time perspective and a tendency of high risk-taking behaviour might complete at least one reproductive event, pass on his/her genetic code to the next generation and thereby maximize his/her own fitness, according to the given circumstances. These benefits might outweigh the drawbacks of reproducing outside of a stable union. However, reproducing out of wedlock was rather uncommon in the period under investigation in chapter 2 (Lesthaeghe, 2015). During the 18th and 19th century there was a very strong linkage between marriage and childbearing. Marriage, an indicator of the social maturation of an individual, was strongly driven by cultural norms (see chapter 1.3.1) and occurred rather late (in Belgium between 1846-1856 the mean average age at first marriage for men was 30.5 years and for women 28.6 years – Lesthaeghe, 2015). Taking these circumstances into account, this thesis underlines the importance of mortality cues in shaping marriage behaviour (see chapter 3). Even under the circumstance of culturally imposed late age at marriage individuals adapt their life histories according to life-history theory predictions and therefore may shift their time preferences as well (devaluating future costs and benefits).

ii. *Local level mortality and family level mortality*

While it is clear that mortality cues have an influence on an individual's life trajectory, it is less clear if the local level or the family-level mortality has a stronger influence on an individual's reproductive and marriage behaviour. This thesis applies different statistical approaches to get a better understanding of the importance of local level and family-level mortality on later life outcomes. Earlier studies had shown that infant mortality clusters in families (*e.g.* Das Gupta, 1990; Edvinsson et al., 2005), and that family level factors have larger effects on individuals' life-history strategies than their individual mortality experience (Edvinsson et al., 2005; Nault et al., 1990; Störmer and Lummaa, 2014). A possible explanation for the importance of family membership in terms of reproductive timing is the strong genetic heritability of human reproductive behaviour – age at menarche, age at first birth and number of children ever born – (Barban et al., 2016; Perry et al., 2014). One-third of the variation of reproductive traits has been attributed to genetic variation (Polderman et al., 2015). Due to the genetic relatedness sisters might have a more similar reproductive timing than non-related individuals (Störmer and Lummaa, 2014). In the analyses of chapter 2 of this thesis sisters were compared with each other. Our findings show, in contrast to Störmer and Lummaa (2014), that risky reproductive strategies (giving birth out of wedlock) are influenced by the individual's mortality experience (number of siblings' deaths experienced and their timing) in early childhood rather than by family-level effects (shared environmental cues).

Notwithstanding the effects of family-level mortality on adjusting individual life-history as described above, previous research has also shown that the local level mortality has a similarly strong effect on the life trajectories of individuals (*e.g.* Quinlan, 2010). Previous studies argue that under high extrinsic mortality, parents will reduce their parental effort because they cannot increase their offspring's survival chances through behavioural changes or through more parental investment (Quinlan, 2007; Störmer and Voland, 2014; Voland, 2013). A child's response to reduced parental effort is to accelerate its life-history strategy, including earlier maturation, earlier sexual intercourse, earlier age at first birth and earlier age at first marriage (Belsky et al., 1991; Belsky et al., 2010). Chapter 3 of this thesis supports this earlier work. Its findings highlight the strong effect of local level mortality on individual's marriage behaviour and takes non-biological factors such as population growth, occupational class, literacy (as a proxy for education – Boonstra, 1998; Matsuo and Matthijs, 2016) and migration status into account. By controlling for these non-biological factors, chapter 3 underlines that non-biological factors are similar in importance to an individual's marriage behaviour as high local mortality cues. This leads to the conclusion that biological and non-biological factors play an equal role in shaping individuals' life histories. The findings of chapter 2 and chapter 3 also emphasise the importance of the individual mortality experience during early childhood regardless of whether local level mortality or family-level mortality is used.

iii. Sex biased parental investment

As mentioned above, reduced parental investment can lead to acceleration of an individual's life-history. Moreover, it has a strong influence on the survival chances of offspring, and it affects their later life outcomes (*e.g.* educational attainment, status attainment, lifetime reproductive success). Parental investment is highly influenced by environmental conditions such as high mortality. Chapter 2 and chapter 3 of this thesis argue that in high mortality environments, parents reduce their parental investment since parental effort cannot increase offspring survival chances.

In addition, parental investment differs between social groups and varies according to the sex of offspring (Trivers & Willard, 1973). Chapter 4 highlights the importance of parental investment on intergenerational income transmission by quantifying the effects of parental income on the income of their offspring in easily interpretable units. High-status parents benefit in terms of fitness maximization by investing in their male offspring rather than in their female offspring. By contrast, low-status parents benefit in terms of fitness maximization by investing in their daughters rather than in their sons. The results of chapter 4 clearly show that parental income and education contribute to sons' incomes to a stronger degree than they did to daughters' incomes. Previous research has shown that men's income is positively related to lifetime reproductive success and the number of mating partners (Fieder and Huber, 2007, 2012; Nettle and Pollet, 2008). Furthermore, high educational attainment seems to ensure competitiveness on the mating and marriage market (Lutz et al., 2007; Skirbekk, 2008). By sex biasing their investment parents could maximize their fitness. This assumption is based on two facts: *i)* low-status men have a higher likelihood to remain unmarried and childless than low-status women (Nakosteen and Zimmer, 1997); *ii)* low-status women have a higher upward mobility and therefore a higher likelihood to get married to a man with higher socioeconomic status (*e.g.* Glenn et al., 1974).

5.2. Implication for future research

The general findings and main conclusions of this thesis have important implications for future research.

First, there is an interdependence of mortality and fertility decisions. This fact should be considered in future research, especially in out of wedlock fertility research. An interesting new angle would be to investigate the interplay of mortality and out of wedlock fertility in urban settings in the 18th and 19th century. During the 19th century in areas with high population density like Paris, Stockholm, and Vienna up to 50 percent of all reported births were illegitimate ones (Fuchs and Moch, 1990; Mitterauer, 1983). Previous research has argued that high illegitimacy rates in major European cities resulted from migration accompanied by family separations and weakened social control (Laslett et al., 1980; Mitterauer, 1983). However, while most of the studies performed controlled for various sociological factors, none of them took local mortality cues into account when investigating the rise of the out of wedlock fertility during the 18th and 19th century.

Second, the findings of chapter 2 indicate that more promiscuous sexual behaviour is not clustered in certain families in the population under observation. Therefore, this thesis calls for a reassessment of Laslett's concept of a bastardy prone sub-society in which he argued that only a few women, who were related by kinship and marriage, were responsible for most of the illegitimate births (Laslett, 1980). Future research should not only study the influence of harsh environmental conditions on sisters' reproductive behaviour but expand analyses to grandmothers, mothers, and aunts. By including other female relatives, a better understanding of the family level effects (*e.g.* genetic preposition) on reproductive behaviour could be gained and Laslett's concept of a bastardy prone sub-society could be further reviewed.

Third, it would be of interest to investigate the influence of harsh environmental conditions on out of wedlock fertility in contemporary high-income populations where there is, like in several historical societies, a substantial culturally imposed lag between physical maturation, age at marriage, and age at first birth. Giving birth to an illegitimate child may have lost some of its social stigma in comparison to former days but growing up without a father has been shown to still lead to faster life histories (Ellis et al., 2003; Shenk et al., 2013). Therefore biological/environmental factors influencing out of wedlock fertility remain an important contextual force to consider in studies focusing on contemporary populations.

Fourth, future research should continue shedding light on the interaction between famine-based mortality cues and faster life histories. This thesis shows that famine-based mortality cues influence individual's marriage behaviour. Based on the findings of chapter 3 a relevant starting point for future research would be to investigate individuals' reproductive careers (age at first child, interbirth intervals, age at last birth, miscarriages and stillbirths) in the context of famine-based mortality cues.

Fifth, although the findings of chapter 4 highlight the importance of parental investment to the status attainment of their sons and suggest that parental investment in their daughters 'status attainment is weaker, further research is needed. The dataset used in chapter 4 covers a time period in which more men than women obtained a higher education degree. Since the 1990s this has changed. Nowadays more women than men are enrolled in college and obtain a higher education degree. This change could potentially have important consequences for the relationship between offspring's sex and parental investment because *i)* it has led to a higher earning potential for women (Klesment and Van Bavel, 2017); and *ii)* higher educational attainment decreases fertility (Skirbekk, 2008). Future research should take these changing sociological factors and their potential consequences into account and therefore compare more recent cohorts with each other.

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Zusammenfassung (German summary)

Die menschliche Fertilität sowie das reproduktive Verhalten werden in hohem Maße von sozioökonomischen Faktoren sowie von evolutionären Prozessen beeinflusst. Der Zeitpunkt von bestimmten Lebensereignissen wird stark von den frühkindlichen Umweltbedingungen sowie dem elterlichen Investment beeinflusst. Die vorliegende Dissertation untersucht das menschliche Reproduktionsverhalten aus der Sicht der Lebensgeschichtsevolution. Sie liefert neue Erkenntnisse darüber, inwieweit biologische, ökologische und sozioökonomische Faktoren die Fruchtbarkeitsmuster und Reproduktionsentscheidungen von Menschen in verschiedenen Phasen des demografischen Übergangs beeinflusst haben. Die erste Studie widmet sich der Frage, ob eine hohe Mortalitätsrate innerhalb der Kernfamilie zu schnelleren und riskanteren Fortpflanzungsstrategien in der vorindustriellen europäischen Gesellschaft geführt hat. Die Ergebnisse zeigen, dass Frauen, die einer hohen Sterberate innerhalb der Geburtsfamilie ausgesetzt waren, ein erhöhtes Risiko hatten, sich früher und außerhalb einer stabilen Langzeitpartnerschaft zu reproduzieren. Die Geburt eines unehelichen Kindes dient in hier als Stellvertreter für eine riskante Fortpflanzungsstrategie. Die Studie zeigt weiters, dass das Risiko einer unehelichen Geburt eher mit der individuellen Mortalitätserfahrung als mit familienspezifischen Faktoren zusammenhängt. Im Gegensatz dazu wird das Alter bei der ersten Geburt innerhalb einer Ehe eher von familiären Faktoren beeinflusst. Die zweite Studie untersucht auf Basis der ersten Studie die Auswirkungen von einer hohen Mortalitätsrate, ausgelöst durch Ernteausfälle und Hungersnot, auf das Alter bei der ersten Eheschließung am Beginn der demografischen Transformation in einer europäischen Bevölkerungsgruppe. Die Ergebnisse zeigen, dass Personen, die in ihrer frühen Kindheit einer hohen Sterblichkeit ausgesetzt waren, früher heiraten. Diese Ergebnisse unterstreichen die Bedeutung der individuellen frühkindlichen Lebensbedingungen für die Adaption der Lebensstrategien. Die dritte Studie berücksichtigt das unterschiedliche Reproduktionsverhalten der sozialen Schichten und gibt Aufschluss über das geschlechtsspezifische elterliche Investment. Die Studie zeigt, dass Eltern ihr Investment sowie ihre Führsorge je nach sozialer Schicht auf das Geschlecht mit dem höheren zu erwarteten Reproduktionserfolg ausrichten. Eltern mit niedrigem Status investieren mehr in die Bildung ihrer Töchter, während Eltern mit hohem Status mehr in die Bildung ihrer Söhne investieren.