Fast Life Histories in Response to Death Clustering, Antwerp 1846–1910

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Fast Life Histories in Response to Death Clustering, Antwerp 1846–1910

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ABSTRACT

Evolutionary biology predicts that when confronted with conditions of high mortality humans, like other species, will respond to these circumstances with a fast life history strategy with early sexual maturation, riskier courtship and earlier reproduction. Such responses in the form of an earlier start of menarche for women and lower ages at first sex and birth have already been found in contemporary populations, but there is far fewer research on historical populations, particularly for men. In this study we examine whether high mortality in the sibset leads to earlier marriage by performing an event history analysis on the historical population of Antwerp in the 19th and beginning of the 20th century. We find that this does indeed significantly speed up the transition to marriage (which is very closely linked to the start of reproduction, particularly in this historical population). By stratifying on family membership, we demonstrate that the mechanism works at the familial level and that individual experiences with sibling mortality have an opposite effect.

Keywords: Life history theory, Evolutionary demography, Mortality clustering, Antwerp, Age at marriage

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1 INTRODUCTION[1]

Evolutionary biology and historical demography are no longer ships that pass in the night. Although the dialogue could still be intensified (Van Bavel, 2016), many historical studies now appear testing one evolutionary hypothesis or another (Burger et al., 2024). Among the most influential are hypotheses derived from "Life History Theory". This theory states that all organisms allocate resources, mostly energy derived from nutrition, to four bodily functions: maintenance (health), growth, reproduction and defence (e.g. the immune function). Between these functions, there are trade-offs, but evolution has prioritized reproduction over other functions. Thus, species adapt to conditions of scarce resources or high mortality by reproducing earlier and more frequently at the cost of growth and maintenance (Wells et al., 2022). This theory has successfully explained differences between species in the timing and pace of reproduction. But it has also been applied to differences between individuals of the same species, including humans. Thus, the theory predicts that humans when confronted with an unpredictable environment will start reproducing earlier, even at costs to the well-being of themselves and their offspring.

The theory has stimulated physical anthropologists and other scientists to look for correlations between mortality levels and reproductive traits such as age at menarche, growth, start of sexual activities, number of sexual partners, timing and number of offspring and so on (e.g. Chisholm et al., 2005). The expectations have been confirmed to some extent, but, overall, research has not resulted in a coherent clustering of physical traits, sexual risk-taking and faster reproduction (Sear, 2020). This brings us to the question of what the supposed mechanisms are that result in "fast" life histories. Evolutionary biology suggests physiological adaptations, such as early menarche at the cost of growth in response to cues from the environment, in particular at an age when the course of one's physical development can still be changed. This window of "developmental plasticity" occurs at an early age, or even at the fetal stage of life. Evolutionary psychologists, however, focus more on behavioural adaptations resulting from evolved, unconscious "strategies" that are employed when children perceive that the environment is unsafe. This leads them to "future discounting" or a trade-off between investing in a future for themselves and their children in favour of early reproduction. However, it has also been suggested that the mechanism runs through the parents. Parents can respond to the death of their children by less (emotional) investment in their surviving children, who then suffer from attachment problems resulting in a "fast life history strategy" (Störmer & Lummaa, 2014, p. 6).

As mentioned, fast life histories are triggered by signals that suggest scarce resources and a high risk of early death. In most research designs, people are compared by levels of poverty and, in particular, "extrinsic" mortality, or mortality beyond one's control (Sear et al., 2024, pp. 312–313). Researchers testing life history theory with historical data have used as their prime cue the mortality of siblings (Pink et al., 2020b; Störmer & Lummaa, 2014) or mortality rates in the local community during childhood (Pink et al., 2020a). They looked for the effects of these cues on the ages at first birth and first marriage. By analysing extramarital births, Pink et al. (2020b) also tested the idea that high extrinsic mortality stimulates sexual risk-taking. The results of these studies are in line with the theory, but inconclusive as to the mechanisms. Störmer and Lummaa (2014) found no effects of *individual* witnessing of sibling deaths, but they did find *family-level* effects. This was confirmed by Pink et al. (2020b) regarding the age at first birth. However, they also found that individual rather than family-level mortality experiences increased the risk of an extramarital birth.

In this research, we aim to replicate existing research, as, clearly, more empirical studies are needed. As in other studies, we use stratified and non-stratified models to distinguish between individual and family-level experiences of mortality. However, we differ from previous research designs in two respects. First, we focus on a large city to see whether the effects of extrinsic mortality as predicted by evolutionary biology also occur in modern settings. Second, we use a relative measure of sibling mortality. Angélique Janssens and Sören Edvinsson, among others, have put the phenomenon of death clustering on the agenda, that is the fact that deaths were not randomly distributed, but often clustered in families (Edvinsson & Janssens, 2012). We expect that when a large share of the children

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[1] In this paper I reworked an unpublished manuscript largely prepared by Ward Neyrinck, whose untimely death in 2021 prevented revision of the text towards publication. Section 2, in particular the statistical analysis, is primarily based on this manuscript (Neyrinck & Kok, 2021). Regrettably, we also had no opportunity to discuss the outcomes and evaluate them in the light of more recent literature. My personal reflections are presented in an Afterthought.

have died, the effect on reproductive timing is stronger than the experience of mortality as such. This approach is in line with Donrovich et al. (2018) who have linked death clustering in "high risk" families to higher mortality in the third generation (see also Vandezande, 2012). They suggest that women having experienced high sibling mortality, had children (too) early, at the detriment of these children. We only study age at first marriage rather than age at the first birth. Arguably, in a period when birth control was still limited, marriage was often rapidly followed by the arrival of the first child. In fact, more than half of all (Dutch) brides marrying before age 23 (in 1811–1915) were already pregnant at the wedding (Kok & Mandemakers, 2021, p. 199).

In the next section, we briefly introduce our data and perform the statistical analyses, which we then discuss. We end with an afterthought on the applicability of life history theory in historical demography.

2 DOES SIBLING MORTALITY AFFECT AGE AT MARRIAGE?

For our analysis we use the Antwerp COR*-database, a historical demographic kin-linked microlevel dataset containing information from the population registers and the vital registration records (1846–1910) for all the communities of the Antwerp district (Matthijs & Moreels, 2010; Puschmann et al., 2022). The COR*-database is a letter sample of people in the Antwerp district whose surname started with the letters COR (e.g. Cornelis, Corsi, Coreynen). This combination of letters was chosen because it was most representative geographically and socioeconomically and led to an acceptable sample size (Van Baelen, 2007).

For our research, individuals had to have both of their parents identified (so that the sibling structure could be reconstructed) and in case they had immigrated into the district, had to have been accompanied by their mother, as an indicator of family migration rather than individual migration. In the latter case, the set of siblings may have been incomplete due to some siblings staying behind in the municipality of origin. Individuals also needed to have their birth year known. This led to a subsample of 1,438 individuals containing 693 women and 745 men. Mean age at marriage was 24.2 for the women exposed to sibling mortality and 24.9 for those not exposed. For men, these respective differential ages are 25.3 and 26.9. Thus, initial inspection suggests that those who were exposed to sibling mortality married younger than the group not exposed.

In our next analysis we use a relative indicator of sibling mortality. We construct a variable named "low survivorship", a dummy set to one when a family is at the bottom 30 percentile in terms of the survival ratio of its offspring (thus the siblings of ego). This can be considered a rough indicator of death clustering as it also keeps into account family size. We analyze the data using Cox event history analysis (Stata SE 12.1). We can visualize effects on age at marriage over time through a graph showing the hazard of marriage for a woman who has a low survivorship in her family of origin. We plot the hazard against analysis time and keep all the other covariates either equal to the reference categories or in such a combination that their effects cancel each other out. Although the baseline hazard in a Cox event history model is not directly estimated, it can be retrieved later by plotting the smoothed weighted (according to the found covariate effects) hazard contributions. By then using the formula:

$h(t) = h_0(t) * \exp(\beta_1 * \text{low survivorship} + \ldots + \beta_{1t} * \text{low survivorship} * \text{analysis time})$ we can calculate the hazard of those women who have low survivorship in their families of origin. Using the found covariate effects and filling them in the hazard then becomes:

$h(t) = h_0(t) * \exp(0.7259123 * \text{low survivorship} + \ldots -0.0621372 * \text{low survivorship} * \text{analysis time})$. We do the same thing for men. In that case the general formula is the following:

$h(t) = h_0(t) * \exp(\beta_1 * \text{low survivorship} + \beta_2 * \text{male} + \beta_3 * \text{male} * \text{low survivorship} + \ldots + \beta_{1t} * \text{low survivorship} * \text{analysis time} + \beta_{2t} * \text{male} * \text{analysis time} \ldots)$ and the found covariate effects just need to be filled in again. The results are shown in Figure 1 and 2.

Figure 1 *Women from low survivorship families marry sooner*

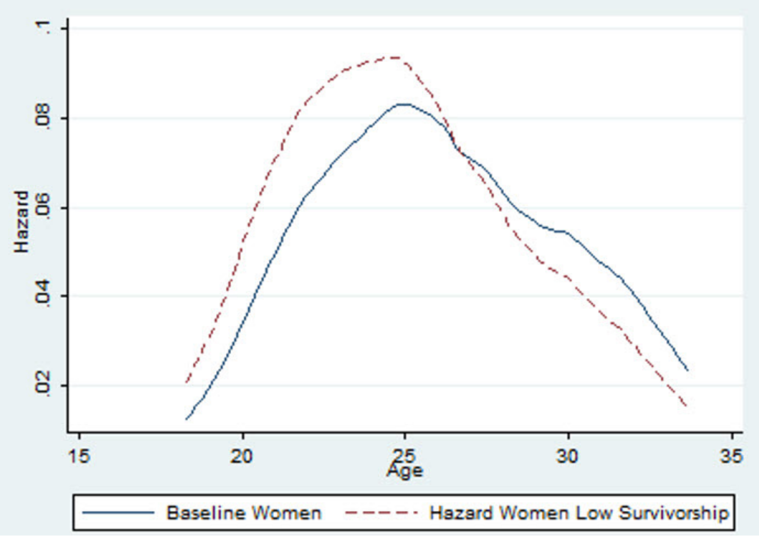
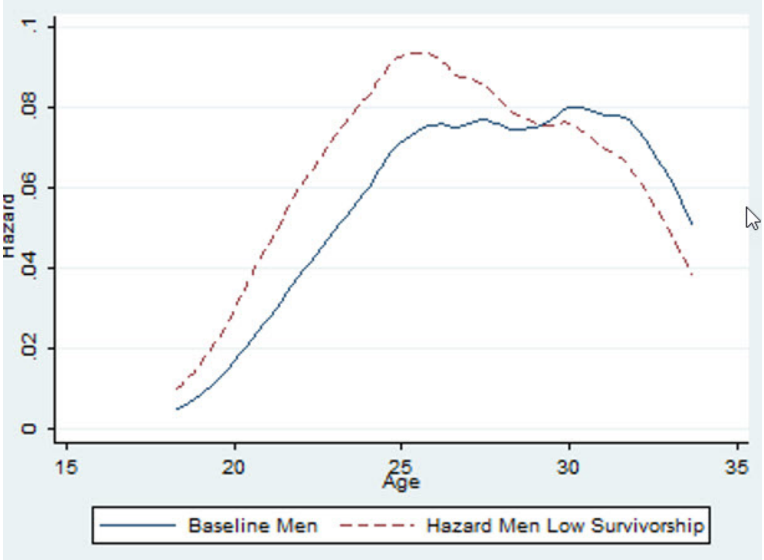


Figure 2 *Men from low survivorship families marry sooner*



In the multivariate models (see Table 1) we control for period, sex, and type of environment (city, polders, and countryside). We also control for age at death of father or mother as this may impact on the resources needed to marry, as well as for ages of the parents which indicate the family life cycle. Older parents may, for instance, exert pressure on children to delay marriage in order to care for them. Furthermore, by including the age of the parents at birth of ego we control for a possible effect of intergenerational transmission of age at marriage (Van Bavel & Kok, 2009). The occupational position of the father serves as proxy for the availability of resources in the family. Family size is included because the presence of many siblings can delay marriage through "resource dilution" (Bras & Kok, 2016). Birth order has also been shown to have a (modest) effect on marriage timing (Suanet & Bras, 2013). The main independent variable is survivorship of siblings, as defined above. Following Störmer and Lummaa (2014, p. 3) we use stratified event history analyses: "Stratifying on families (full siblings) means that for each family (sibship) a separate baseline is allocated (but not estimated). Such a model compares individuals from the same family and therefore controls for shared family factors". Our second model is stratified, thus gives individual responses to mortality. The hazard ratio is always between 0 (effect is so negative that it is impossible that the event/failure of interest occurs) and +∞ (the effect is so strong and stimulating the event happens immediately). A hazard ratio of 1 means there is no effect (Cleves et al, 2010).

The table shows that in the family model, low survivorship or death clustering resulted in earlier marriage (the hazard ratio is 2.067). As expected, men married later than women (hazard ratio is 0.289). But the effect of sibling mortality does not differ by gender (hazard ratio is 1.170), although life history theory predicts that men (with higher mortality themselves and lower investment in offspring) would respond with earlier reproduction more than women (Störmer & Lummaa, 2014). Interesting, but also unexpected, is that the stratified model giving individual effects shows an effect of low survivorship in the opposite direction: thus a higher age at marriage. We ran the same models using "exposure to sibling mortality" as the main variable (not shown here). The outcomes are much weaker: respectively 1.187 (not significant) for the family model and 0.323 (borderline significance) for the individual model. Thus, the share of deceased siblings is indeed a more powerful indicator. To some extent this was also suggested by Störmer and Lumma (2014) who found that "mortality experience" in small families had a larger impact than in large families (p. 4). Their models also show an interaction of this effect (of mortality experience) with analysis time, in the sense that the effect is stronger at the young ages. A similar exercise (models not shown here) confirms this for the effect of low sibling survivorship in the family model, whereas no significant interaction was found in the individual model.

Table 1 *Age at marriage in Antwerp (1846–1910) regressed on family death clustering, Cox regressions (hazard ratios)*

Variables	**Family model**	**Individual model**
Low survivorship family (time varying) ref = not low	2.1**	0.4*
Male (ref. = female)	0.3***	0.3**
Male* low survivorship	1.2	1.0
Period 1856–1865 (ref. = 1865–1889)	0.5***	0.8
Period 1889–1910	1.3**	0.7
Living in city and immigrated (ref. = born in city)	1.2	2.6*
Living in the polders	0.8	3.3
Living in countryside	0.8*	1.0
Family size	1.0	0.9
Birth rank	1.1*	1.1+
Age of ego at death of father (ref = over 15)		
0–5	1.2	0.2
5–10	0.4*	0.1*
10–15	0.8	0.4*
Age unknown	1.0	0.1
Age of ego at death of mother (ref = over 15)		
0–5	0.8	0.9
5–10	0.7	2.1
10–15	0.8+	0.0
Age unknown	0.7**	0.0
Paternal age at birth ego (ref. = over 40)		
0–40	1.1	1.3
Age unknown	0.7+	1.00
Maternal age at birth ego (ref.= 30–40)		
0–20	1.3	1.7
20–30	1.4**	1.1
40–55	0.8+	1.0
Age unknown	1.0	0.5
Father is high and middle class (ref. = skilled worker)	1.1	1.3
Low skilled worker	1.1	1.3
Farmer	1.3	1.9
Unknown	1.1	1.3
N Persons	1,438	1,438

Note: * $p < 0.05$, + $p < 0.1$, ** $p < 0.01$, *** $p < 0.001$.

We found that both men and women from Antwerp families with relative high mortality among the children tended to marry earlier, but this effect was not found on the individual level. On the contrary, individuals experiencing sibling deaths married later. Our findings of family-level effects of mortality in the family of origin on marital timing and reproduction are in line with Störmer and Lummaa (2014) and Pink et al. (2020b). But, remarkably, *within* families, individuals reacted to sibling mortality in a way not predicted by life history theory: they tended to marry later.

3 DISCUSSION, CAVEATS AND CONCLUSION

According to the logic of life history theory, parents may have responded to the death of their children by pushing the survivors to start reproducing fast (thus ensuring a second generation). This implies that the children do not need to have witnessed the death of siblings themselves, but were affected by the parents' reaction. It has been suggested that such parental response, creating an insecure environment for the children, is itself an evolved (unconscious) strategy (Pink et al., 2020b). That is because parental investment in offspring quality does not pay off in high mortality environments (Sear et al., 2024). Still, this does not explain the individual response to the death of siblings.

An alternative explanation, outside life history theory, possibly accounts for both outcomes. First, high mortality in families may in itself point at "bad parenting", or at least at conditions unfavorable for children. Our control for occupations and, by implication, resources is probably too crude to capture the conditions in which Antwerp children in the 19th century lived. Unhealthy, cramped housing, for instance, can go far in explaining high infant and child mortality and the wish to leave home as soon as possible. Socio-economic factors play an important role in death clustering in families (van Dijk, 2019). And a bad housing situation during childhood has been shown to be related to an early age at first birth (Sheppard et al., 2016). In the past, infant and child mortality was often caused by external causes, in particular infectious diseases, but bad housing (e.g. overcrowding, lack of good water) and bad parenting (e.g. no breastfeeding, reluctance to ask for medical help, marital discord) may still result in some families having more deaths than others. This explanation, of course, is not in line with life history theory. In this scenario, high mortality is not extrinsic and — to stick with the jargon — child mortality and early marriage are "phenotypically correlated" as they are both caused by an underlying factor (Sear, 2020). Why would individuals within the same family respond differently to whether or not they experienced sibling death? Possibly it is just a psychological effect, in the sense that the sorrow surrounding the death made them wary of forming a family themselves. Or it is just a replacement effect, that is the chance that the surviving child had to stay at home and care for the parents has become larger.

Some caveats need to be mentioned. First, although the timing of marriage is indeed closely related to the arrival of a child, the actual first birth — including children to unmarried women — might have been a better test of the expected fast life history response to sibling mortality. Moreover, a more complete test of such a response would include the likelihood of marrying or having children at all, preferably using a cure model (Alter, 2019). Second, although we have found effects of interaction with time, a closer inspection of sensitive periods in line with Pink et al. (2020a, 2020b) is needed. They found that the effects were much stronger if mortality was experienced in the first 5–7 years of life, a period in which children were "primed" to learn essential lessons about parent-child relations. Third, we already mentioned the lack of proper SES indicators for childhood conditions. Some researchers suggest that (life history) reactions to mortality differ by socio-economic setting, which should be tested by adding interaction effects (Griskevicius et al., 2011).

All in all, our outcomes are mixed. We found partial support for the notion of a fast life history strategy, but alternative explanations might explain our results just as well or even better.

4 AFTERTHOUGHT: DO WE NEED LIFE HISTORY THEORY

We have concluded that our individual-level effects run counter to the predictions formulated in life history theory, whereas the family-level effects seem to confirm them. Still, this does not rule out other explanations, which in fact may be even more plausible. What does this tell us about the advantages of applying theories from evolutionary biology to demographic behaviour?

The task of historical demographers is to interpret people's demographic behavior as being simultaneously a response to environmental cues and constraints produced by societal expectations, (family) laws and economic feasibilities. Dealing with cultural, economic and legal factors is already challenging, so how to fit in evolved biological and psychological mechanisms resulting in physiological changes and/or unconscious "strategies" to accelerate one's life history? To be sure, the *principle* of adding evolutionary biology to the demographer's conceptual toolbox is sound. As Smith (2013) has phrased nicely: "people make choices, taking into account their circumstances and their preferences; those preferences are generated from a complex developmental process with inputs from both genetic inheritance and social learning" (p. 111). Thus, preferences can be partly shaped by mechanisms predicted by life history theory. Indeed, life history theory has been described as an *additional* explanation for behaviour (Pink et al., 2020b). But do these mechanisms really apply to humans, in particular humans living in modern, industrialized societies?

Fast life history theory suffers from a number of shortcomings. First, causal models of the effects of extrinsic mortality ignore the role of morbidity, or the possibility that persons suffered diseases which caused "scarring" — possibly affecting reproductive capacity. Following life history theory, childhood illnesses may also trigger earlier reproduction (Dinh et al., 2022). Second, confounders are often unspecified. For example, malnutrition leads to higher mortality but also to *delayed* maturation and reproduction (Dinh et al., 2022; Sear et al., 2024). Childhood deprivation and traumas may even have an impact on gene expression, which can be transmitted to next generations. Such epigenetic mechanisms are rarely discussed in the theory (Burger et al., 2024, pp. 742–743). Third, the crucial notion of trade-offs presupposes perennial scarcity of energy and resources. But humans have by and large escaped such conditions. Wealthy humans can easily combine the functions of maintenance, growth, reproduction and defence (Sear, 2020). In what circumstances can we still expect trade-offs? Some even argue that life history strategies are in fact regulated by wealth and the logic of accumulation ("optimal control theory"), and not by mortality (Mell et al., 2021). Fourth, life history theory has ignored that humans are "cooperative breeders" and often rely on kin for rearing their offspring. Energy and resources are pooled, thus not subject to an individual trade-off. It also means that psychological mechanisms, such as attachment to parents, are less universal than supposed (Sear, 2020, p. 521). However, some have explained the importance of family-level effects of mortality by referring to cooperative breeding: children (unconsciously) realize that kin help may not be available (Störmer & Lummaa, 2014). Actually, this may serve as an example of the tendency to fit all possible outcomes to the theory. In other words, fifth, the postulates of life history theory are axiomatic. The goal of inclusive fitness (the passing on of genes) is realized through trade-offs and all human demographic behaviour somehow results from this goal. Thus, high mortality should lead to a quality-quantity trade-off: a shift towards earlier and more frequent childbirth to ensure net reproduction. But evidence as well as logic often run counter to this premise. For example, risky sexuality to ensure early procreating often leads to lower net fertility, whereas committing to a stable relation favours high fertility (Dinh et al., 2022). Inclusive fitness has also been invoked to explain, e.g., male homosexuality (Adriaens & De Block, 2006) and religious celibacy (Micheletti et al., 2022).

Recently, Sear et al. (2024) pointed out the weakness in much life history research: "many 'predictions' in life history theory in fact arise from empirical observations and/or verbally intuitive models that are rarely formalised using mathematical theory, and so are not predictions derived from theory at all" (p. 18). What is needed for a fruitful dialogue and cooperation between historical demography and evolutionary biology is a clear set of falsifiable hypotheses. This will enable historical demographers to assess in what contexts life history theory can offer better or additional explanations than more common socio-economic or cultural ones.

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