Mortality Clustering in the Family. Fast Life History Trajectories and the Intergenerational Transfer of Infant Death in Late 19th- and Early 20th-Century Antwerp, Belgium

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Intergenerational transmissions of infant mortality using the Intermediate Data Structure (IDS)

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Mortality Clustering in the Family

Fast Life History Trajectories and the Intergenerational Transfer of Infant Death in Late 19th- and Early 20th-Century Antwerp, Belgium

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ABSTRACT

In this article, we investigate to what degree infant mortality risk was transferred from grandmothers to mothers in the Antwerp district, Belgium, during the late nineteenth and early twentieth century. We also investigate some of the determinants of infant mortality and explore the role of the family - paternal factors (presence, age, and social class), mother's childcare experience, and infant household location - in the survival of infants. The data for this research were retrieved from the Antwerp COR*-database and were transferred into the Intermediate Data Structure (IDS). The results of the survival models show that women whose mother experienced three or more infant deaths had a 77% higher risk of experiencing the loss of an infant themselves, compared to women whose mother experienced zero infant deaths in the past. These results remained robust after controlling for potential mediating and moderating factors. The results on the age of the mother at birth, her marital status, as well as the living environment suggest that at least part of the intergenerational transfer in infant mortality can be explained on the basis of life history theory: women who grew up in a high-risk family tended to reproduce earlier and faster, and often raised their children without a partner. In this way they unconsciously created riskier conditions for the raising of their own infants: the mothers had little life experience, limited resources, and often no assistance from a partner. As a result, their own children were also at an increased risk of dying in infancy.

Keywords: Infant mortality clustering, Family factors, Intergenerational transfer, Life history theory, Antwerp

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1 INTRODUCTION AND OBJECTIVES

By the middle of the 19th century, infant mortality in Western countries was much higher than it is today. Although death in high mortality regimes is often coupled with a fatalistic attitude of the population under study (cf. Courbage & Puschmann, 2015), research both on contemporary developing countries and on Western societies in the past show that infant mortality is not randomly distributed, but rather is clustered among certain families (Das Gupta, 1990; Edvinsson, Brändström, Rogers & Broström, 2005; Vandezande, 2012). In practice, this means that a considerable proportion of families in the past did not experience a single infant death, while other families – so called high risk families – experienced multiple infant deaths. Brändström (1984) found, for example, that in the 19th-century Swedish parish of Nedertorneå, characterized by high fertility and high infant mortality, approximately 45% of mothers did not experience any infant death, while 19% of the mothers experienced a disproportionately high share of infant deaths.

The causes of infant death clustering in families are diverse and complex. One cause is related to the fact that infant mortality risks can be transmitted from one generation to the next, along both the paternal and maternal family lines (Vandezande, 2012). This finding gave rise to a typical nature-nurture debate, which can be summarized as 'faulty genes versus faulty parents' (Janssens, Messelink, & Need, 2010). According to the current state of research, the intergenerational transmission of infant mortality is related to both. On the one hand, high risks seem to be transmitted from one generation to the next through genetic inheritance – concerning mainly neonatal deaths – (Reid, 2001); on the other hand, behavioral practices and resources (e.g. economic, social and cultural capital) are transmitted from parents to their children (Edvinsson & Janssens, 2012; Reid, 2001). Children who are being born in families with limited transferrable capital have lower life chances and face higher mortality risks (e.g. Arntzen & Andersen, 2004). Not only do we mean tangible resources, such as social status, economic capital and level of education of the parents, but also parental abilities to care for their children (cf. Das Gupta, 1990; Janssens & Pelzer, 2012). A knowledgeable parent is more likely to promote and practice health-protective behaviors, and to create a safe environment for an infant. Moreover, a healthy parent is more likely to produce healthy offspring. In all cases, the mother is particularly vital in ensuring the infant's chance of survival, and thus many studies of the past and present have focused solely on maternal factors, such as age at birth, maternal health/nutrition and level of education. The role of fathers in infant survival is less explored and usually limited to the impact of their socioeconomic position, although in some cases the impact of his death on the survival chances of the infant has been also explored (e.g. Beekink, Van Poppel, & Liefbroer, 1999). In a limited number of studies, the influence of the father on infant mortality risks is measured indirectly by-way-of the mother's marital status (e.g. illegitimacy) or by father's marital status. However, even in case of the latter, the focus in historical studies is directed towards the female caretaker by way of testing the 'Cinderella effect' (e.g Willführ & Gagnon, 2012).

On a broader scale, intergenerational transfers of infant mortality can be viewed within the scope of life history theory, which suggests that individuals adapt their reproductive strategies to the environmental conditions in which they grew up (Roff, 2002; Stearns, 1992). Accordingly, individuals who were exposed to harsh environments early in life – e.g. high mortality in the neighborhood and/or in the family – reach sexual maturity earlier, marry at a younger age, begin reproduction earlier and have shorter birth intervals compared to individuals who grew up in a safe environment, characterized by low mortality risks (Pink, 2017). To ensure that their genes will be passed on to the next generation, individuals who were raised in a high mortality environment unconsciously choose offspring quantity over quality, and fast over slow reproductive trajectories (i.e. a rapid and riskier reproductive life course). In this way, families with high infant mortality pass on increased mortality risks from one generation to the next through their surviving children's reproductive behavior.

Infant mortality has shown heterogeneity in terms of time and place. Even within national borders, high levels of internal variation have been measured for countries both large and small (Klüsener et al., 2014). There is also evidence of significant regional differences in the strength of the intergenerational transmission of infant mortality. While in certain regions the mortality history of infants is strongly correlated with the survival of infants in the previous generation, in other regions the effect is rather weak or completely absent (Edvinsson & Janssens, 2012; Reid, 2001). Vandezande (2012) suggests that this is related to differences in local culture and family systems, but he hypothesizes that it might be also related to strong local variants in gene defects. In practice, regional differences might also

be related to the fact that most studies focus on a very limited number of rather small regions, and that different studies are hard to compare because they have applied a different methodology, both in terms of data handling and in terms of statistical analysis. Therefore, for this analysis we try to overcome the last problem by applying the same methodology (and statistical programming) on data which have been transferred into a standardized data format developed for large historical databases, the Intermediate Data Structure (IDS) version 4 (Alter & Mandemakers, 2014). In earlier research, an international joint study on the transfer of infant mortality on the maternal line was conducted in five historical European populations: the Antwerp district (Flanders, Belgium), the Skellefteå region (northern Sweden), the Scanian region (southern Sweden), the province of Zeeland (the southwestern coast of the Netherlands), and Troms (northern Norway) (Quaranta et al., 2017). For all regions, though to varying degrees, the risk of an infant dying was associated with the infant mortality level of the grandmother. In further research, all participants from the joint project further investigate infant mortality in their respective regions (Van Dijk & Mandemakers, 2018; Broström, Edvinsson, & Engberg, 2018; Quaranta, 2018a; Sommerseth, 2018). In this analysis, we expand on this research by providing an in-depth case study of our region: the Antwerp district in the 19th century.

The overall objective of this research is to deepen our understanding of the historical (and critical) role that the family context played in the survival of individual family members. Using the Antwerp COR*-database covering the Belgian district of Antwerp during the 19th and early 20th centuries, we test to what degree infant mortality risk can be explained through a transmission along the maternal line (i.e. from grandmother to mother) using the common programming code developed to enable cross-regional comparisons with other historical datasets (Quaranta, 2016; 2018b). Expanding on the joint analysis (Quaranta et al., 2017), the second objective is to further investigate the role of parental factors during the infant's first year of life, given the dependency of infants on parental resources and caretaking ability for their survival. To this end, we include additional proxy variables which are largely unaccounted for in other infant mortality studies, in an attempt to capture the effect of maternal ability (i.e. previous child care experience, number of younger siblings). We also introduce paternal factors into our statistical models (presence, social class, parental age) to study the impact of fathers (if any) on infant death risk. In doing so, we are able to not only test whether there was an intergenerational transfer of infant mortality risk, but also the proposed mechanisms behind such a transfer. Finally, we aim to complement the work previously conducted by Vandezande (2012) that studied infant mortality in depth in the same region. In this way, we can comprehensively investigate infant mortality by way of intergenerational transmission, including mediating and moderating factors.

2 HISTORICAL CONTEXT

The research setting is the Antwerp district during the period 1839-1915. The district consisted of more than fifty rural municipalities, and the port city of Antwerp turned into the largest Belgian city during the nineteenth century. Its population grew from a little less than 80,000 in 1840 to 315,000 by the end of the research period. Mortality decline and urban in-migration drove population growth (Puschmann, Donrovich, Grönberg, Dekeyser, & Matthijs, 2016). Due to its function as a port and with a wide range of commercial activities, the city attracted migrants from all social strata. International migrants who comprised about 10% of the city's population by 1910 were mainly drawn from the higher levels of society and experienced considerable upward mobility. Internal migrants were usually former peasants and agricultural laborers who upon arrival entered the ranks of the laboring poor, but who nevertheless enjoyed considerable opportunities for upward mobility (Puschmann, 2015). While the men were mainly engaged in heavy port labor, female internal migrants moved to Antwerp for domestic service or for marriage (Winter, 2009).

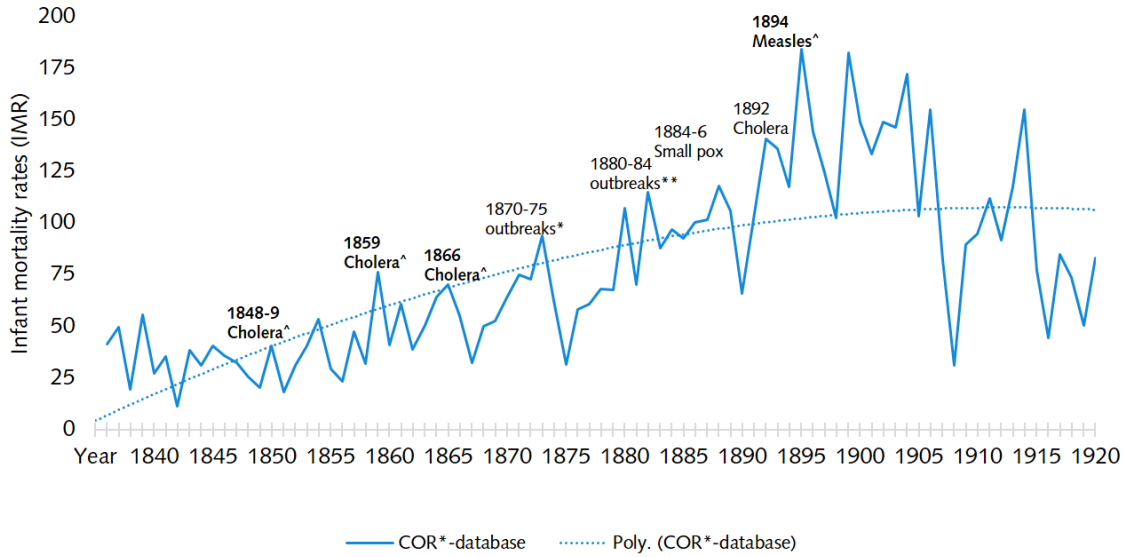
By the middle of the nineteenth century both in Antwerp city and the neighboring suburban and rural communities, mortality was relatively low compared to other regions in Belgium and Western Europe (Devos, 2006). By 1850 Antwerp city had a crude mortality rate of about 21 per 1,000, while the figure was 19 per 1,000 for the rural municipalities (Puschmann et al., 2016). The relatively low mortality in the urban area was most likely related to the absence of large scale industry, which caused heavy air and water pollution in European cities of the time and therefore posed a major health threat, in addition to work-related accidents. However, the absence of the cottage industry might have been

even more important, since the absence of common safety regulations made it even more deadly than heavy industry (Van Rossem, Deboosere, & Devos, 2017). Thus, in terms of health it might have been a blessing for Antwerp's population that textile manufacturing in the city collapsed earlier in the nineteenth century (Winter, 2009).

The absence of industry explains also why the rural-urban divide within the Antwerp district was rather minimal. However, due to strong urban in-migration, population density increased considerably during the nineteenth century, leading to a higher chance of epidemics to spread. It was indeed not until after the wrecking of ramparts and the re-organization of the urban living space by the start of the twentieth century that major epidemics disappeared, with the exception of the Spanish flu following World War I. Although internal and international migrants contributed significantly to the increase in population density, they enjoyed – mainly due to positive selection effects in the region of origin – lower mortality risks as adults (ages 30+) compared to the native Antwerp population (Puschmann et al., 2016). There is good reason to believe that this was not due to selective return migration of the weak, sick and elderly, or related to measurement errors (Puschmann, Donrovich, & Matthijs, 2017).

In order to investigate intergenerational transfers in infant mortality, it is important to view how mortality developed over the period under analysis. In Figure 1 the infant mortality rates in the Antwerp district as recorded in the Antwerp COR*-database are presented.

Figure 1 *Infant mortality rates in Antwerp and noted epidemic outbreaks (1840-1920)*



Notes: a) ^ Denotes major epidemics with approximately 1,000 or more cases reported in the district
*b) * Denotes multiple small-scale outbreaks of scarlet fever, typhus, small pox, measles and cholera*
*c) ** Denotes multiple small-scale outbreaks of puerperal fever, small pox, and measles*

The infant mortality rates (IMR) are calculated as total number of recorded infant deaths divided by births for each year in the Antwerp COR*-database. In this figure, we have also noted years of major cholera and measles outbreaks (classified by 1,000 or more cases recorded in the city) in the Antwerp district; other years with smaller-scale multiple epidemics (less than 500 recorded cases) are also noted in the graph (cf. Kruithof, 1964). Major epidemic diseases may explain some of the mortality peaks present in the graph (e.g. the 1859 and 1862 cholera outbreaks which led to spikes in the infant mortality rate, and the high mortality peak during the 1894 measles outbreak). However, if we look at the graph in Figure 1 from a general perspective, the level of infant mortality around the middle of the nineteenth century in Antwerp (below 60) was very low according to European standards. Around that time IMR was still around 150 in England (Woods, Williams, & Galley, 1993) and Sweden (Brändström, 1993), about 180 in the Netherlands (Walhout, 2010) and fluctuated somewhat between 275 and 300 in Austria (Kytir & Münz, 1993). As mentioned, the relatively low mortality in Antwerp could be

partially explained by the absence of a large-scale industry, which led to air and water pollution, and poorer living conditions among many European cities of the time. Further, it is important to reiterate that the IMRs displayed in the graph are calculated using the data stored in the COR*-sample database and may not perfectly represent the trend in all infant births and deaths within the city, due to the methodology of how the database was constructed (i.e. a letter sample of the population). See Section 3.1 for more information about the database construction.

Nevertheless, the (polynomial) trendline which best fits the shape of the data suggests that infant mortality increased over time in the Antwerp district, whereas in most other areas in Europe it started to decline (Corsini & Viazzo, 1993). The increase in infant mortality in Antwerp was most likely related to the heavy increases in population density, caused by strong urban in-migration (Puschmann, 2015; Winter, 2009). However, the neighbouring sourthern part of the Netherlands also witnessed a rise in infant mortality during the same period of time (Van den Boomen & Ekamper, 2015).

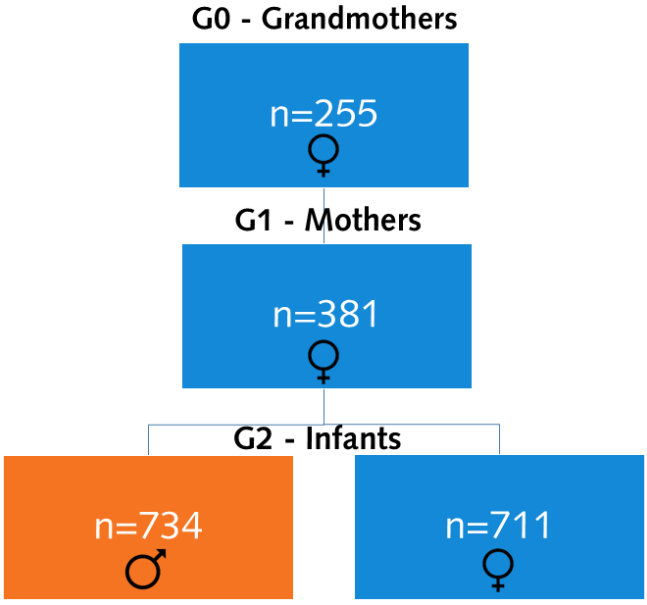
3 DATA AND METHODOLOGY

3.1 DATA AND SUBSAMPLE

The data for this study originate from the Antwerp COR*-database, a large historical demographic database based on a letter sample from the population registers and the birth, marriage, and death certificates from the Antwerp district in the period 1846-1920. The database covers life course information – e.g. birth, marriage, death, migration, and occupational changes – on more than 30,000 individuals and spans up to three generations. All persons who lived at some time in their life in the Antwerp district between 1846 and 1920 and whose family name started with the letters 'C-O-R' were selected, and their life courses were 'reconstructed' from the primary sources. Due to the sampling method, the database is not only suited for the study of family relations from within the household, but also extends to outside of the household. Family relations are clearly defined on the basis of the source material, and it is possible to track individuals and families from one address to the other within the Antwerp district during the period covered by the database. For more detailed information on the database, see Matthijs and Moreels (2010).

For our research purposes, we retrieved a sub-sample of mothers from the Antwerp COR*-database, for which we could follow their own reproductive life course and that of their own mothers. We also included information on their husbands (the fathers of the infants under analysis). This resulted in a dataset consisting of 381 mothers (G1), who gave birth to 734 boys and 711 girls (G2) (see Figure 2).

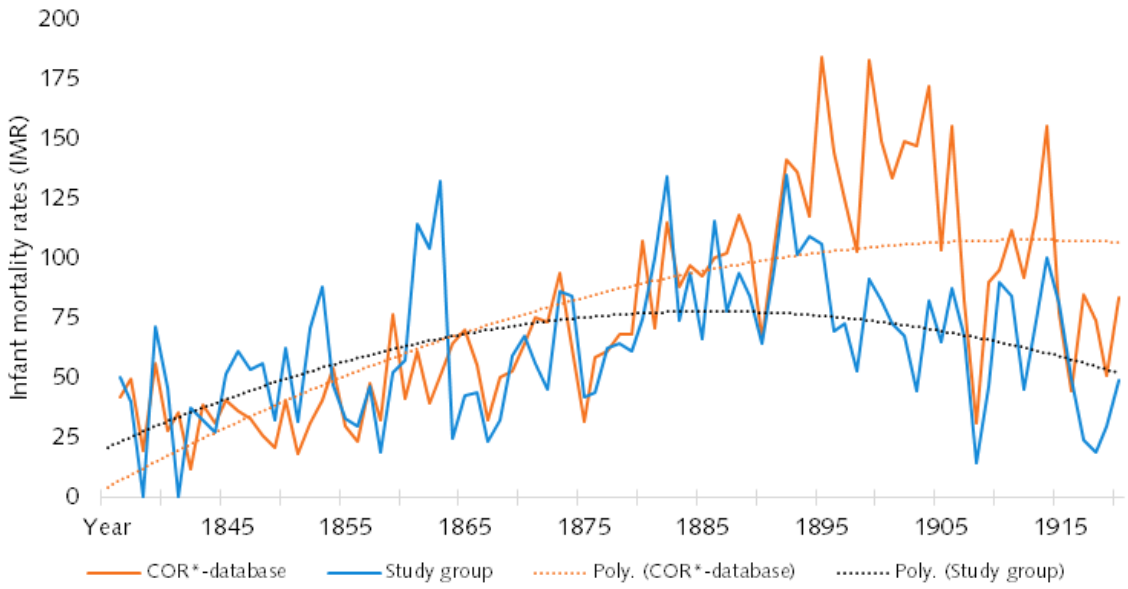
Figure 2 *Generations included in the study sample from the Antwerp COR*-database*



The dataset contains reproductive life course information on 255 women (G0), who were the mothers of G1, and the grandmothers of G2. The infants, G2, are the research persons whose survival history will be analyzed in this paper. A limitation of the data is related to the fact that the reproductive life courses of the mothers and grandmothers are not always complete, due to migration and the time boundaries of the database. Since our sample is relatively small, we decided to not work exclusively with full life courses, as this would further limit the statistical power of the analysis.

Turning back to the development of infant mortality over the period in Antwerp, in Figure 3 we display the IMRs for both the database as a whole, and for our sub-sample used in this analysis in order to compare the development of infant motality over the period. In this graph, it is apparent that the IMR of the study group generally follows the development of the IMR as calcuated for the COR*-database as a whole, which makes it assumable that the selection of the study group did not lead to major biases in the analysis. Additionally, the database in its design was constructed based on a letter sample, and deemed representive of the district of Antwerp (Matthijs & Moreels, 2010). As such, one can expect that the entire city itself experienced a similar pattern in terms of infant mortality. One exception is visualized, however, which points to an important 'under-rating' of the IMR in the 10-year period from about 1895 to 1904 in our study group. Given that a three-generational family structure is needed to study transmissions of intergenerational infant mortality risk, migrants and their infants are excluded from this analysis. Accordingly, the under-reporting of the IMR during this period is most likely related to the omission of migrant infant deaths. After all the period of under-reporting happened when urban net-migration peaked in our subsample (cf. Puschmann, Van den Driessche, Grönberg, Van de Putte, & Matthijs, 2015). See Section 5 for a further discussion on our selection criteria and limitations of the study.

Figure 3 *Infant mortality rates recorded in the COR*-database as a whole and in the study sample*



Source: Antwerp COR-database*

3.2 THE IDS TRANSFORMATION PROCESS

In order to expand upon previous research on the transmission of infant mortality (Quaranta et al., 2017), we first followed the methodological approach developed by Quaranta (2018b) which makes use of the IDS format version 4 (Alter & Mandemakers, 2014) for database management, and subsequent analysis. Since the Antwerp COR*-database is not stored in such a format, we first needed to transform our study sample into the IDS format to allow for cross-database standardization, so that we defined and measured variables in the same way. The IDS allow for comparability across different regions and countries, i.e. for the purposes of this research, Belgium, the Netherlands, Norway and northern and southern Sweden (all using unique databases). All relevant variables needed for the common approach were selected from the raw data in the COR*-database Microsoft Access tables.

Next, this information was cleaned and tested for internal consistency. After internal validation, it was subsequently moved into IDS. In practice, all family relations - between husbands and wives, and between mothers and their children - are being defined in the INDIV_INDIV table. The characteristics and events of these individuals - sex, birth date, start and end of observation, marriage date and death date - were being stored in the INDIVIDUAL table. These events were then being used to compute additional variables, such as the number of infant deaths the grandmother experienced, the mother's age at birth, the birth order of the children, which were used in the common analysis.

3.3 ANALYTICAL STRATEGY

Part I of the analyses was conducted using the programming code developed by Luciana Quaranta (2016; 2018b) which was provided as a series of STATA 14 do files including coding syntax for study selection criteria, descriptive statistics, preparing the data as time-to-event, the survival models, post-estimations and robustness checks. The primary goal of using the common code is to enable analyses across regions for models using variables (relatively) consistent across regions and databases. In a second stage of analysis, we perform an extended model using additional explanatory variables unique to the Antwerp COR*-database, which allow to dig deeper into the influence of parental factors on infant mortality risk.

Table 1 *Descriptive statistics, common analysis, Antwerp 1839-1915 (N=1,445)*

Variable	n	Percent	Mean	SD	Min	Max
Infant sex						
Female	707	49%				
Male	738	51%				
Number of Grandmother's infant deaths						
0	901	62%	0.649	0.967	0	5
1	254	18%				
2+	290	20%				
Number births of the Grandmother			5.587	2.344	2	13
2	113	8%				
3	230	16%				
4 to 6	597	41%				
7+	504	35%				
Maternal age			29.06	6.38	16	47.93
15-24	447	31%				
25-34	710	49%				
35+	289	20%				
Infant birth order			3.921	2.829	1	18
1	310	21%				
2	265	18%				
3	212	15%				
4	400	28%				
7	258	18%				
Infant birth year	-		1887.55	13.55	1839	1915
Infant birthdate, centered	-		9.05	13.58	-39.07	36.44

To account for time-dependent factors, we monitor all births of mothers that are captured in the data using survival analysis. Each infant is followed from birth until 12 months (365 days), wherein the failure event is specified at the date of infant death occurring at or before age 1. The failure event did not occur if the infant is still alive at the end of this interval. We measured the timing of infant death, using child's age as the baseline hazard which was defined as:

$$h(t) = h_0(t)e^{(b_1X_1+b_2X_2+\ldots+b_nX_n)}$$

where, *h(t)* is the hazard rate and h_0 *(t)e* is the baseline hazard (Cox, 1972). In the common analysis we fitted two additional survival models: 1) Weibull with shared frailty on the mother and 2) Weibull with shared frailty on the grandmother. In our results we focus on results from the Cox model in both Part I and Part II of the analyses.

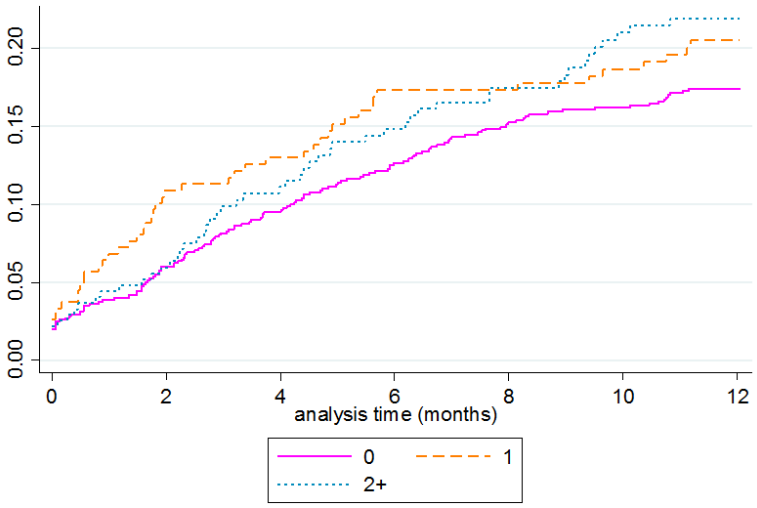
In the common approach, our main variable of interest is the number of infant deaths of the grandmother, categorized as 0, 1, or 2 or more. The models are also adjusted for other variables that have an association with infant mortality: child's sex, total fertility of the grandmothers (measured as number of grandmother births), mother's age at birth, child's birth date (centered), and infant birth order. See Table 1 for the descriptive statistics of the covariates for the study group. In Table 1 descriptive statistics for the common model are displayed for the 1,445 infants included in the analysis.

4 RESULTS

4.1 RESULTS PART I – BASIC MODEL

First, we interpret the cumulative hazard graph (Figure 4) for our main study variable to get an initial impression of how different levels of infant mortality of the grandmother relate to infant death among the mothers in the study. The associations between categories of infant mortality of the grandmother are displayed as cumulative hazard estimates over the 12-month period of analysis time (from infant birth to age 1). At the start of the analysis time the hazard of grandmothers with one infant death was higher than those with no infant deaths and two or more infant deaths. However, as study time increases, toward the end of the period those with two or more infant deaths had higher hazard rates, suggesting that the proportionality hazards assumption was violated for this variable.[1] Yet, during the largest portion of the analysis time infants with grandmothers who lost at least one infant to early death had the highest cumulative risk of death, suggesting that there was a certain degree of intergenerational transmission. More specifically, it points in the direction that infant mortality risk was clustered within certain households. After all, the increased risk of losing an infant is passed on from one generation to the other through the mother's line.

Figure 4 *The Nelson-Aalen cumulative hazard estimates by infant mortality of the grandmother (categorized as 0, 1, or 2+) presented in months*



1 In part II of the analysis we tested this violation for all variables included in the survival models and adjusted the model by including a time interaction for any statistically significant violations.

In Table 2, we present results from the full survival models used in the common analysis (fitting a Cox model, and Weibull with shared frailty on the mother or grandmother). Our results suggest an intergenerational transfer of mortality risk in our study group, since compared to grandmothers who had no infant deaths, having one or having two or more was related to elevated relative risk of infant death of the mother. There was approximately 19% and 29% higher mortality risk compared to the reference category in the Cox Model, and slightly higher hazard ratios were obtained in the Weibull shared frailty models (though the frailty variance was not statistically significant). There was a minimal impact from higher numbers of grandmothers' births, with slightly lower relative risk compared to grandmothers who had only one child, consistent across the different models.

In terms of the influence of the mother's fertility characteristics there was higher risk depending on the infant's birth rank: compared to firstborns, being a higher birth ordered infant relates to a significant 76% and 180% higher death for infants born 4th-6th in the birth order and those born 7th or higher, respectively. Importantly, we found that maternal age had a strong association with infant fate, and that both younger and older age categories related to opposite outcomes. Compared to the reference category of mothers between ages 25-34 at the time of birth of the child, infants with older mothers were associated with much lower risk of infant death (HR=0.626), while infants born to younger mothers experienced rather high elevated infant mortality risk (HR=1.691).

Table 2 *Intergenerational tranmission of infant mortality, event history models, Antwerp, 1839-1915*

	Cox model		Weibull model, mother shared frailty		Weibull model, grandmother shared frailty	
	HR	**p-value**	**HR**	**p-value**	**HR**	**p-value**
N. of infant deaths of the grandmother, (ref: 0)	1.000	ref.	1.000	ref.	1.000	ref.
1 infant death	1.185	0.327	1.268	0.280	1.238	0.355
2+ infant death	1.286	0.193	1.368	0.202	1.252	0.414
N. of births of the grandmother (ref:2)	1.000	ref.	1.000	ref.	1.000	ref.
3 births	0.891	0.679	0.976	0.943	0.970	0.929
4-6 births	0.967	0.894	1.027	0.930	1.078	0.808
7+ births	0.797	0.422	0.835	0.602	0.867	0.690
Female	1.000	ref.	1.000	ref.	1.000	ref.
Male	0.845	0.189	0.806	0.104	0.818	0.126
Birth order (ref:1)	1.000	ref.	1.000	ref.	1.000	ref.
2	1.106	0.641	1.125	0.590	1.133	0.568
3	1.357	0.182	1.355	0.196	1.396	0.154
4-6	1.761	0.009	1.703	0.021	1.770	0.012
7+	2.768	0.000	2.534	0.001	2.699	0.000
Child birth date centered	0.995	0.308	0.996	0.464	0.994	0.321
Mother age 15-24	1.691	0.002	1.639	0.008	1.674	0.005
25-34 (ref.)	1.000	ref.	1.000	ref.	1.000	ref.
35-50	0.626	0.021	0.672	0.063	0.670	0.061
Intercept			0.130	0.000	0.128	0.000
Frailty variance			0.539	0.122	0.484	0.098
N of children	1445		1445		1445	
N of infant deaths	246		246		246	

4.2 RESULTS PART II – EXTENDED MODEL

In an extension of the results found in the common models for Part I of the analysis, in the next stage of analysis we had four main goals: (1) re-measuring two key variables: the infant mortality transmission variable and the maternal age variable; (2) further explore detailed parental factors; (3) take into account household location within Antwerp, and (4) to test (and if necessary, adjust for) covariates' violation of the proportionality assumption imposed by the Cox proportional hazards model.

With respect to the first goal, we re-categorized the grandmother infant mortality variable to include the additional category of 3 or more infant deaths. The purpose of this additional category was to see whether this could better capture the effect of a 'high mortality household', since we expect that a higher level of infant mortality (more infant deaths) would be associated with an increase in risk transmitted from grandmother to the mother. Given that infants born to young mothers were worse off and those born to older mothers were better off in the results from Part I of the analysis, our second goal is to include additional parental measures that may help explain this finding. We followed the line of thinking posed by Das Gupta (1990) regarding maternal abilities and how they may be associated with age. Since advanced maternal age was beneficial for infant survival (and younger aged mothers were disadvantaged), we hypothesize that caretaking ability increases with age and older mothers may be more prepared in terms of knowledge of infant health and safe health practices. We re-categorize maternal age to allow for more sensitivity between age categories. As in the common analysis there were only three age categories defined: 15-24, 25-34, and 35+, we include five categories for the extended analysis in order to separate the effect of being born to teen mothers (ages <20), specifically. Additionally, we include two new relevant measures. The first is a measure for whether the mother ever had previous childcare experience (i.e. a dummy variable which is coded as 0 or 1 based on whether she had a registered occupation in the database in the domestic field). Since we also expect that if a mother had younger siblings, she had learned to care for an infant at an early age, potentially gaining hands-on experience by caring for her younger sibling(s) (i.e. co-breeding), we include a variable for whether the mother had any number of younger siblings, categorized as no younger siblings, younger siblings between 5 and 7 years younger, and younger siblings between 7-15 years younger.

Next, since the role of fathers had been completely absent from the story in the common analysis, we aimed to include relevant paternal variables into our extended model: father's age at the birth of the child, occupation, and whether he was unknown or absent. Father's age at birth was measured in as ages <30, 30-39, and 40+. Occupation was categorized using HISCO coding (Historical International Standard Classification of Occupations) to create HISCLASS categorizations (Van Leeuwen, Maas & Miles, 2002). Based on the 12 HISCLASS classifications, due to small numbers we combined them into three levels and a missing category, if the father's occupation was not recorded in the database. Finally, we created an indicator for whether the father was unknown, in that the infant was not linked to any father in the COR*-data.

Given that Antwerp experienced rapidly increasing population during the period and being born within or outside of the urbanizing city could have different outcomes for the health of the infant (and the mother), we adjusted for birthplace (household location), distinguishing between an infant born in Antwerp city, its suburbs, or in a rural region in the outskirts of Antwerp. Since epidemics were more severe and occurred more frequently in densely populated urban areas, we expect that the survival chances of infants were lower in Antwerp city compared to the adjacent suburban and rural areas. See Table 3 for variable construction and descriptive statistics of all covariates that were re-measured (grandmother infant mortality, maternal age) and newly added in the model (paternal characteristics, birthplace, mother caretaking proxies) in the extended analysis.

And to our fourth goal, after post estimation testing, using the *estat phtest, detail* command in Stata, which tests the proportionality hazards (PH) assumption for all included covariates individually, as well as for the entire global model, we fitted our extended Cox model with time interactions for two variables using the *tvc* option in the stcox model syntax. The two variables (birth order and number of grandmother births) that were shown to be statistically significant in violating our PH assumption, meaning that the effect was not proportional over time. We interacted these variables with time to account for the proportionality violations.

Table 3 *Descriptive statistics for additional variables in the extended analysis*

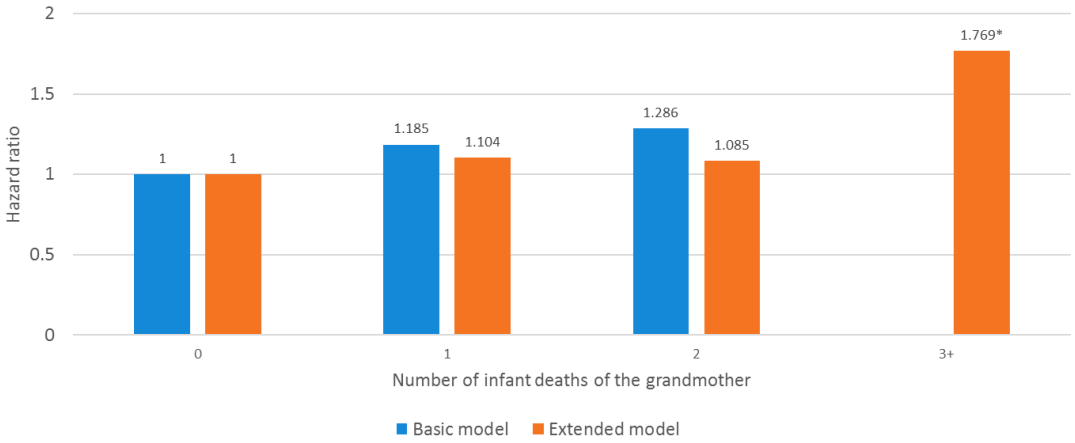
Variable	n	Percent	Mean	SD	Min	Max
Number of Grandmother's infant deaths (updated)						
0	901	62%				
1	254	18%				
2	199	14%				
3+	92	6%				
Mother's age at birth (updated)						
<20	80	6%				
20-24	367	25%				
25-29	394	27%				
30-34	315	22%				
35+	289	20%				
Mother childcare experience						
No	1313	91%				
Yes	132	9%				
Mother with younger siblings						
No younger siblings	457	32%				
5-7 years younger	331	23%				
8-15 years younger	657	45%				
Unknown father						
Known	1354	94%				
Unknown	91	6%				
Father's occupation						
Non-manual and professionals	187	13%				
Medium skilled manual	371	26%				
Low and unskilled	735	51%				
Missing/Unknown[a]	152	10%				
Father's age			32.55	7.817	16	67.997
<30	587	41%				
30-39	533	37%				
40+	234	16%				
Unknown[b]	91	6%				
Birthplace						
City	541	37%				
Suburban	307	21%				
Rural/other	597	41%				

Notes: a) Includes missing information due to fathers being unknown and for fathers with no recorded occupation in the database
b) Only unknown for infants with unknown fathers

4.2.1 RE-ESTIMATING INFANT MORTALITY TRANSMISSION

In the extended model we re-categorized the infant mortality level of the grandmother to include a fourth category, 3 or more infant deaths. Figure 5 displays the association between grandmother's infant mortality from the basic model (in which it was categorized as 0, 1, and 2+) and in the extended model which included the fourth category of 3+. Results are presented from the separate, fully adjusted models, including all covariates.

Figure 5 *Infant mortality risk by the number of infant deaths of the grandmother*



Source: Antwerp COR-database*

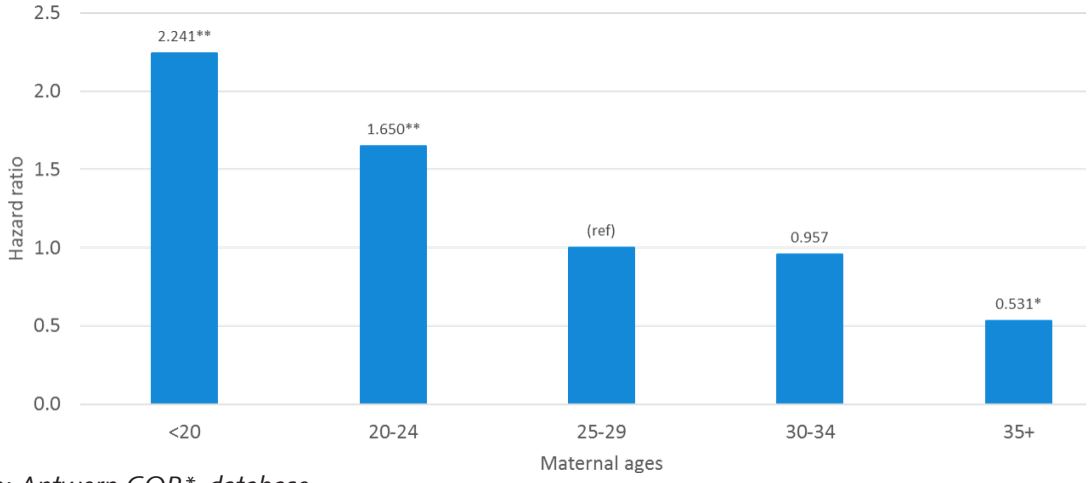
Notes: a) Basic model adjusts for all covariates included in the basic model from Part I; Extended model adjusts for all additional variables in Part II of the analysis
*b) * p < 0.05, ** p < 0.01, *** p < 0.001*

These results show that by extending the categorization to a higher level of infant mortality a stronger intergenerational link was established. It also tells us that if a grandmother had one or two infant deaths there was only a slight increase in infant mortality risk of her daughter in the new model, as the hazard ratios are slighly lower for these two categories in the extended analysis. However, if the grandmother had three or more infant deaths in the past, more than 75% higher infant mortality risk was found in the next generation, and this effect is significant in the extended model.

4.2.2 PARENTAL EFFECTS

Next, a primary focus of the extended analysis was to disentangle maternal effects. In order to dig deeper into our finding in the common model (that infants born to younger mothers had higher mortality risk, and infants born to older mothers had lower relative risk), we re-categorized the variable to allow for more sensitivity between age categories. In Figure 6 the results for the new maternal age variable are shown.

Figure 6 *Infant mortality risk by maternal ages at birth*



Source: Antwerp COR-database*

Notes: a) Results from the full extended model (all control variables)
*b) * p < 0.05, ** p < 0.01, *** p < 0.001*

In the figure we can see that mortality risk is highest for infants born to the youngest mothers, and lowest for infants with the oldest mothers, with the hazard ratios decreasing with each higher age

category. The highest risk group is infants born to teen moms (less than 20 years old at the time of the child's birth). This category relates to more than twice the mortality risk compared to the reference category of infants born to mothers aged 25-29. For infants born to mothers aged 20-24 there is approximately 66% higher mortality risk. While for those whose mothers were in their early 30s at time of birth differed minimally from the reference category, infants born to mothers aged 35 and older have the lowest mortality risk (HR=0.532).

Next we included two additional variables to try to capture 'maternal quality or maternal experience'. Given that the role of fathers was absent in the common analysis, we also wanted to explore paternal measures that we believe are relevant to the study. Results for the main parental variables are displayed in Table 4 (the extended model includes all covariates from the basic model, as well as the additional measures described in Table 3).

Table 4 *Influence of parental factors on infant mortality risk, Cox proportional hazard model, Antwerp, 1839-1915*

	Hazard Ratio	P-value
Mother age (ref: 25-29)		
<20	2.241	0.005
20-24	1.659	0.009
30-34	0.957	0.833
35+	0.532	0.016
Father age (ref: 30-39)		
<30	0.916	0.640
40+	1.509	0.053
Mother younger siblings (ref: no)		
Yes: 5-7 years younger	0.674	0.044
Yes: 8-15 years younger	0.957	0.776
Mother childcare experience (ref: no)		
Yes	1.781	0.004
Unknown father (ref: no)		
Yes	2.150	0.008
Birthplace (ref: city)		
Suburban	0.755	0.145
Rural/other	0.719	0.034
TVC (time interactions)		
Birth order=category 4-6	3.489	0.016
G0 births=category 3	0.267	0.064
N=1445, failures=246		

Note: Adjusted for all variables included in the common model from Part I, and father's occupation class

Building upon the survival model used in the common analysis we now included additional variables for the extended analysis: re-categorized maternal age, father's age, maternal caretaking experience proxies (having younger siblings, previous domestic occupation), a dummy indicator for whether the father was unknown, household location, and socioeconomic status (father occupation – not presented). The full table is shown in Appendix A.

In both the common model and extended model, we find that being born to older mothers was associated with lower mortality risks and being born to younger mothers related to higher risks, as shown in Figure 6. Given that maternal age was a strong indicator of infant death we tested also for paternal age, finding that the age effect worked in the opposite direction for men: there was slightly lower infant mortality risk for younger fathers, and higher risk for older fathers, with 50% higher infant mortality for children born to fathers over age 40.

To further disentangle the finding that older mothers had better chances of having their infant survive to age one, we included the two variables used as proxies for maternal ability: if the mother had younger siblings, or if the mother had previous work experience in the child caretaking or domestic sphere. We found a positive effect of mothers who had siblings between 5-7 years younger, with a hazard ratio of approximately 0.67. Being born to a mother who had younger siblings with the larger age gap (8-15 years younger) also yielded slightly lower risk than being born to mothers who had no younger siblings, though this effect was minimal (HR=0.958) and not statistically significant. The dummy variable, childcare experience, revealed a rather surprising finding: mothers who had previously worked outside of the home as a servant or in the childcare sphere had a significantly higher hazard of losing a child in infancy than those who did not (HR=1.781). A dummy indicator which noted whether the infant's father was unknown had a strong association with the infant's risk of death: children whose father was unknown (i.e. not identified or traced in the COR*-data) had more than twice the infant death risk (HR=2.15). Finally, we included a variable for birthplace of the infant (i.e. household location in Antwerp) given that the environment in the urban city center would be different than in the countryside or the suburban parts of Antwerp (and accordingly, the health of the mother and fetus may be affected by negative externalities associated with urbanized areas, such as crowding, pollution, the availability of clean water, or the ability for diseases to spread more easily) (Van Poppel, & Van der Heijden, 1997). We found that relative to those born in Antwerp city center, those born in households located outside of the city had lower mortality risks, and the category of rural households yielded the lowest risk with a hazard ratio of 0.719. Only the latter of these two categories was statistically significant.

5 LIMITATIONS

Some limitations in this study are worth noting, mainly relating to data selection restrictions and the methodological approach. The relatively small sample size for three generations within the COR*-database (due to the time boundaries of the database) as well as the highly mobile society (with a high level of in- and out-migration) means we may have lacked full fertility histories of grandmothers during their reproductive years. This may have led to an underreporting of births and infant deaths of the grandmothers and may have somewhat weakened the intergenerational effect in this analysis. We did find that grandmothers who were observed continuously in the Antwerp district between ages 20-50 was a rather small proportion of the total 256 grandmothers included in the analysis (sensitivity analysis, not reported). For the grandchildren of these women, the mortality risk before age 1 did not differ substantially from the total cohort, and still remained highest for infants whose grandmother experienced the largest number of infant deaths in the past. In our extended analysis, we also chose to include all infants from the basic models, even those who had missing information on paternal variables (e.g. an unknown occupation), in order to not exclude any research persons from the extended analysis.

Another limitation of the study was highlighted in Figure 3 which displayed the IMRs for the study sample and for the database as a whole. Given that Antwerp experienced vast migration in the port city, we were not able to account for disparities in infant mortality among natives and migrants in our analysis based on the study criteria (a three-generational timespan, meaning that we needed at least some information on fertility histories of the grandmother). This can partly explain why the IMR differs at some points between the database and the restricted study sample, particularly in the later periods of analysis when migration was at its peak. Accordingly, in this analysis we are cautious to generalize the results from our subsample to the entire Antwerp population during the study period, as migrants comprised a sizeable subgroup of the total population in the district (especially in the later period of the analysis), and it is well established that natives and migrants have experienced differences in mortality risk both in the past and present.

Finally, one of the aims of this research was to participate in a cross-comparative international study of the intergenerational transmission of infant mortality risk. Applying the IDS on the Antwerp data allowed our results to be compared with other datasets from several other socio-historical contexts, using identical programming in Part I of the analysis. In doing so, this approach provided benefits to the analysis, such as the validation of key findings in other study regions, and given the transparent

methodology, the ability to further replicate or expand on the study design in other datasets stored in the IDS. In a sense, the first part of analysis serves as a pilot study to provide proof of the concept that data stored in, or easily transferred to, the IDS could be used to address research questions across multiple regions and datasets. While performing the analysis in such a way that the methodology and statistical analysis were uniform across regions and, accordingly, allowed for cross-comparisons, our results did however highlight one limitation to this approach. Had we not performed the extended analysis we may have missed some of the effects deemed important in the historical context of Antwerp. What worked fine for the others, was not ideal for us in terms of some of the categorizations of key infant mortality measures. Accordingly, a common approach plus an extended analysis per region or dataset may be the best study design to allow for both comparability (and to validate key findings), and the flexibility to further explore the region-specific contexts based on the strengths of the individual datasets or preliminary results from the common model.

6 DISCUSSION AND CONCLUSIONS

In Part I of the analysis, in the common approach, we found some evidence of an intergenerational transmission of infant mortality risk between grandmothers and mothers, as infants with grandmothers that had one or two or more deaths had slightly higher risk of death before age one. In our extended analysis, we re-coded the infant mortality level variable to include a higher level of categorization (3+ infant deaths). This category related to more than 75% higher mortality risk for the infant of the mother, suggesting that intergenerational transmissions are strongest in more extreme cases, i.e. when maternal grandmothers had a higher number of infant deaths in the past. In fact, other research using the COR*-data found a similar effect in previous studies on infant mortality (Vandezande, 2012), and in terms of adult mortality where adults who grew up in a high-risk household (one with a high proportion of infant deaths) had higher post-reproductive mortality risk in later life (Donrovich & Matthijs, 2016). In this sense, we can interpret our finding from another point of view: the mothers who grew up in a high- risk environment and who were thus exposed to a household that was associated with many sibling infant deaths had poorer (total and/or reproductive) health. In turn, during her own fertility years this impact on health was associated with higher infant mortality risk.

We also dug deeper into the mechanisms behind the relationship between parental factors and infant mortality (or survival), as our preliminary findings in the common model suggested a strong association with maternal age. The maternal age variable remained strong and significant in the extended model which included additional controls variables, such as socioeconomic status and birthplace. In our analysis, we found increased risk for young mothers, apparent in the common model for mothers aged 15-24, but particularly noticeable once we included additional maternal age categories in the extended model with more than double the mortality risks for infants born to teen moms. High infant mortality may be related to physical immaturity of young mothers (Alam, 2000). One explanation can relate to lower birth weight which is often related to poorer health of the fetus and increased risk of early death or childhood morbidity (cf. McCormick 1985), and young mothers are at increased risk of post-neonatal infant death, in particular (i.e. after 28 days) (Phipps, Blume & DeMonner, 2002). In our analysis, post neonatal deaths amounted to nearly 75% of all 246 infant deaths included in our sub-sample (see Appendix B). In his analysis, Vandezande (2012) subdivided infant death risk into three periods: perinatal, late neonatal, and post neonatal periods. He found that early infant deaths, which are most affected by biological causes, were not transmitted to successive generations. He proposed that the explanation of 'faulty genes' seemed not to be the main driver of transmitted infant mortality in our study region, Antwerp. In fact, our cumulative hazard graph (Figure 4) also pointed in this direction, as the increased risk for death during the first year of life among infants whose maternal grandmother experienced two or more infant deaths is displayed only towards the last months of the analysis time (approximately from month 9). Given that genetic causes of infant mortality are foremost associated with neonatal mortality, i.e. mortality during the first month of life (Reid, 2001) and unlikely to explain the increased infant mortality risk in post neonatal ages, we believed that this finding may have been related to the inexperience of young mothers, and accordingly, the ability to care for their infants. We also expected that having been raised in a household with younger siblings (infants and small children) as a 'co-breeder' might have been an important experience gained in early life. Not only women who had lived with considerably younger siblings in the household, but also

women who had worked as a servant in someone else's household, had experiences other women lacked, as such living arrangements offered a unique opportunity in the life course to learn how to feed and take care of infants before becoming a mother (cf. Reynolds, 2016). It seems likely that women who became mothers at later ages more often had had such experiences. To further explore this, we included two variables which could account for this maternal experience and that had not been tested in the previous work of Vandezande (2012): whether the mother had younger siblings and whether she had a previous occupation in the domestic sphere. Our maternal age effect remained robust even though the model was adjusted for these caretaking proxies, and when we adjusted for socioeconomic status (father's occupation). Infant's birth order was also adjusted for (finding that higher ordered infants fared far worse than firstborns). This suggests that the increased mortality risk for infants of young mothers cannot be fully explained by purely economic factors (e.g. low social class), by lack of caretaking experience or knowledge, by transferred genetic effects that affect mostly early perinatal deaths, or by birth rank of the infant (which is also associated with maternal age). If this is not due to educational differences, genetic effects, or economic resources[2], our findings may point to a health effect, in that older mothers may have had better health and nutrition compared to younger mothers (and therefore, *in utero* conditions) which related to increased health and survival of the infant.

One unexpected finding in our results was that if the mother had a previous occupation in domestic service or in the child care sphere, the infant had a higher mortality risk than those born to mothers without these occupational experiences. We in fact expected that such work experience better prepared the woman for motherhood and would have led to an increased knowledge of child care and health practices. One explanation for this finding can be that a woman who cared for children in other households meant that she was at increased risk to be infected by diseases contracted within the household in which she worked and spent a lot of her time, for example the epidemics that struck the region during the period of study (see Figure 1). One other disease which poses a risk and is carried by small children, in particular, is cytomegalovirus (CMV). CMV is spread through fecal matter, urine, and saliva, which the woman would have been in contact with while feeding and changing an infant or child during her servant or caretaking work. Once infected, the virus can lay dormant during a latency period and, even during viral episodes, is largely asymptomatic. However, the infection can cause major complications down the line during pregnancy and permanent issues for the fetus (Gardella, 2008).

Another reason why women who worked in dome[stic occupations may have had higher risk of their infant dying before age 1 can also be related to working conditions during pregnancy and physical labor, which may have taken a toll on her health, e.g. due to poor working conditions, injury, or exhaustion. Given that this variable in our analysis only captures whether the women *ever* worked in a domestic occupation (and not the timing of employment) we can only speculate that some women may have worked until close to the time of birth. Further, if a woman continued employment during the first year after birth, this could have an implication on breastfeeding practices and, in turn, quality of care of her infant, as breastfeeding is strongly associated with infant and child survival (cf. the systematic literature review conducted by Sankar et al. (2015)).

When we turn to the influence of fathers, the opposite finding in terms of ages at birth was found. One would expect that, using the same line of thinking for the mothers, the youngest fathers were most likely just as inexperienced as their female counterparts and were also less financially established than their older peers. However, the influence of paternal age on infant mortality risk has been studied less extensively than that of maternal age. This is not surprising since the mothers have a direct role in the health and survival of the fetus, after all she carries the baby to term. However, recently in the literature there has been a heightened interest in paternal effects on infant and child health outcomes. For example, given the positive link between birth weight and infant health, Reichmann and Teitler (2006) found that young fathers were less likely (and older fathers more likely) to have low birth weight babies. On the other hand, advanced paternal age has been shown to relate to higher risk of fetal death (cf. Andersen, Hansen, Andersen & Smith, 2004). Sperm quality decreases with age, and women with male partners over age 45 have a higher chance of miscarriage[3]. We found evidence that

2 The extended model adjusts for occupation class. We also tested the association of maternal literacy (as a proxy for education, i.e. that the mother could sign her own marriage certificate) but this variable did not distinguish any differences between literate and illiterate in terms of infant mortality risk (not presented).

3 https://yourfertility.org.au/for-men/age/

infants born to fathers over age 40 had approximately 50% higher mortality risk compared to infants born to fathers under age 30. However, even more important was whether a father was present at all, as infants born to an unknown father had twice the mortality risks of infants with an identified father. This variable was measured in the study by the dummy variable for whether the father was 'unknown' and was coded as such if a father was not identified in the COR*-database as being linked to that infant. Given our strong finding, we turned back to the raw data to get more information as to why these 91 infants had no identified father in the database. More than half (n=52) were born out of wedlock. Illegitimately born infants had a higher mortality risk, which has been associated with a lack of financial resources, low morale of the mother due to social stigma and resulting shame, social exclusion and lack of social support from a marriage partner and/or the broader family (Kok, Van Poppel, & Kruse, 1997). Further, breastfeeding patterns have shown to differ by the legitimacy status of the infant, with illegitimates being significantly less likely to have been breastfed (Reid, 2017).

Given that several new variables related to the mechanisms behind infant mortality were considered in the extended analysis, we were able to test mediating and moderating effects of their influence on our grandmother infant mortality variable. We included several factors that can help point to some of the causes behind the infant mortality risk and intergenerational transfer, such as paternal factors (presence, age, and social class), mother's childcare experience, and infant household location (birth place). When adding our new variables one by one into the model (i.e. in a nested approach, not presented) we found that the inclusion of these controls did not weaken our grandmother infant mortality variable strength in any significant way. The only exception being the inclusion of the regional variable (birth place) when the strength weakened slightly for the transmission variable (reduced to a HR of 1.59), however this effect rebounds back to 1.769 in the full model which included all control variables. Similar to the conclusions of Vandezande (2012), given that neonates comprised a large majority of our infant deaths, this can make it assumable that genetic factors likely played a limited role in the cause of increased intergenerational transmission risk in our study group. In our results, the intergenerational transfer of infant mortality remained strong in the case of a high number of infant deaths in the previous generation, even while accounting for several mediating and moderating factors, such as social class, birth place, and maternal and paternal factors.

In this analysis, we found important implications for infant survival based on maternal and paternal factors, and evidence of an intergenerational transfer of infant mortality on the maternal line in the Antwerp district in the 19th and early 20th centuries. The real strength of this finding was uncovered when re-structuring the infant mortality level variable to include an additional higher category (i.e. grandmother had more than 3 infant deaths). This suggests that in the most extreme cases, when the underlying familial factors related to high levels of infant death in the past, mortality risk is passed down to the next generation. Given the inclusion of variables in our extended analysis which are largely unaccounted for in other infant mortality studies, we were able to both establish a maternal intergenerational link and further investigate the mechanisms behind the association. Our findings suggest that the causes of the intergenerational transfer of mortality risk cannot be solely explained by economic factors (e.g. low social class), by lack of caretaking experience or knowledge, or by transferred genetic effects that affect mostly perinatal deaths. Rather we believe that the family effect in infant mortality clustering, to a great extent, traces back to a biological mechanism linking exposure to high mortality in infancy and childhood to fast life history strategies.

An important consequence of a fast life trajectory is that it leads to an increased risk of mortality among one's own offspring, due to physical immaturity, but also due to a lack of resources, and accordingly less investment in each child. Empirical studies on historical data have, for example, shown that individuals who experienced sibling death during early childhood married at younger ages (Störmer & Lummaa, 2014; Voland & Willführ, 2017). Accordingly, there is a greater risk of having to raise offspring without a partner (due to a greater risk of separation and divorce). The stress of being exposed to a high mortality environment in early life leads to a rapid and riskier reproductive life course, and increased mortality risks from one generation to the next through their surviving childrens' reproductive behavior. The importance of the living environment in our analysis (high urban mortality versus low rural mortality), and our findings that teenage and single mothers were particularly vulnerable to experiencing infant mortality, all underline this path of reasoning. Whatever the reasons behind what initially triggered high infant mortality in the family (e.g. low socio-economic status, bad hygiene, limited and poor nutrition, etc.), those increased mortality risks are being transferred to the next generation, and consequently, a fast life history strategy gives rise to a negative vicious

cycle, replicated in successive generations. This explains why infant mortality clustering is found both in historical European populations and contemporary developing countries. The fact that our results show that young fatherhood was not associated with high infant mortality, and Vandezande (2012) found that infant mortality clustering was mainly transferred through the maternal line, indicates that the fast life history strategy is gendered.

This study connects insights from two well-developed strands of research: life history theory and infant mortality clustering. We believe that combining this literature and connecting mortality experiences in the family of orientation to reproductive and offspring survival experiences in the family of procreation can lead to new insights into the phenomenon of death clustering and intergenerational transmissions of infant mortality risk in families. A way of developing this line of research further would be to explore clusters of life courses based on mortality experiences in early life and reproductive outcomes and offspring survival later in life, by ways of sequence analysis.

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APPENDIX A - EXTENDED MODEL FULL RESULTS

		Haz. Ratio	Std. Err.	P-value	[95% Conf.	Interval]
Basic Model	**Grandmother's infant deaths (ref: 0)**					
	1	1.112	0.200	0.554	0.781	1.583
	2	1.095	0.249	0.690	0.701	1.708
	3+	1.740	0.488	0.048	1.004	3.014
	Mother age (ref: 25-29)					
	<20	2.241	0.648	0.005	1.272	3.949
	20-24	1.659	0.323	0.009	1.133	2.429
	30-34	0.957	0.199	0.833	0.636	1.440
	35+	0.532	0.140	0.016	0.317	0.891
	G0 births (ref: 1)					
	2 to 3	1.480	0.504	0.250	0.759	2.885
	4 to 6	1.049	0.277	0.855	0.626	1.759
	7+	0.897	0.266	0.713	0.502	1.603
	Infant sex (ref: female)					
	Male	0.838	0.109	0.172	0.649	1.080
	Infant birth order (ref: 1)					
	2	1.238	0.277	0.340	0.799	1.919
	3	1.545	0.377	0.074	0.958	2.491
	4 to 6	1.339	0.406	0.336	0.739	2.427
	7+	3.048	0.901	0.000	1.707	5.441
	Child birthdate centered	0.997	0.005	0.507	0.986	1.007
Extended model	**Mother younger sibling (ref: no younger siblings)**					
	yes: 5-7 years younger	0.674	0.132	0.044	0.459	0.990
	yes: 8-15 years younger	0.957	0.147	0.776	0.708	1.294
	Mother childcare experience (ref: no)					
	Yes	1.781	0.359	0.004	1.200	2.642
	Unknown father (ref: no)					
	Yes	2.150	0.618	0.008	1.224	3.777
	Father occupation (ref: medium skilled)					
	Non-manual and professionals	1.374	0.329	0.185	0.859	2.195
	Low and unskilled	1.407	0.260	0.065	0.979	2.022
	Unknown	1.164	0.316	0.577	0.684	1.980
	Father age (ref: 30-39)					
	<30	0.916	0.171	0.640	0.636	1.321
	40+	1.509	0.321	0.053	0.995	2.290
	Birth place (ref: city)					
	Suburban	0.755	0.146	0.145	0.517	1.102
	Rural/other	0.719	0.112	0.034	0.530	0.975
TVC	**Time-varying covariates**					
	Birth order=4-6	3.489	1.806	0.016	1.265	9.622
	G0 births=3	0.267	0.191	0.064	0.066	1.081
	N=1445, failures=246					

APPENDIX B - DISTRIBUTION OF INFANT DEATHS BY TIMING OF DEATH (N=246)