Intergenerational Transfers in Infant Mortality in Southern Sweden, 1740-1968

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

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Intergenerational Transfers in Infant Mortality in Southern Sweden, 1740-1968

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ABSTRACT

Studies conducted in historical populations and developing countries have evidenced the existence of clustering in infant deaths, which could be related to genetic inheritance, early life exposures, and/or to social and cultural factors such as education, socioeconomic status or parental care. A transmission of death clustering has also been found across generations. This paper is one of five studies that analyses intergenerational transmissions in infant mortality by using a common program to create the dataset for analysis and run the statistical models with data stored in the Intermediate Data Structure. The results of this study show that in five rural parishes in Scania, the southernmost province of Sweden, during the years 1740-1968 infant mortality was transmitted across generations. Children whose maternal grandmothers experienced two or more infant deaths had higher risks of dying in infancy. The results remained consistent when restricting the sample only to cases where the grandmother had been observed for her entire reproductive history or when controlling for socioeconomic status. When running sex specific models, significant effects of the number of infant deaths of the grandmother were observed for girls but not for boys.

Keywords: Infant mortality, Intergenerational transfers, Survival analysis, Intermediate Data Structure, Sweden

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1 INTRODUCTION

The aim of this study is to analyse intergenerational transfers in infant mortality along the maternal line in five rural parishes in Scania, the southernmost province of Sweden, for the period 1740-1968. More specifically, this work looks at whether the likelihood that a woman's children died in infancy was affected by whether any of her siblings had died within their first year of life. The study is part of a larger project looking at intergenerational transfers in infant mortality across five different historical populations in Europe – Belgium (Donrovich, Puschmann, & Matthijs, 2018), the Netherlands (Van Dijk & Mandemakers, 2018), Norway (Sommerseth, 2018), Northern Sweden (Broström, Edvinsson, & Engberg, 2018) and Southern Sweden (current article). The five studies are based on databases which have been formatted to follow the Intermediate Data Structure (IDS) (Alter & Mandemakers, 2014; Alter, Mandemakers, & Gutmann, 2009). These works are all conducted using the same methods and theoretical framework and the same programs to create the dataset for analysis and run the statistical models (Quaranta, 2018). A comparison between the regions was also made (Quaranta et al., 2017). The aim of the project is not only to contribute to the literature on intergenerational transmissions but also to produce fully comparable and reproducible studies in order to show the advantages of adopting the IDS for research.

The existence of clustering of infant mortality within certain families was shown in previous research (Das Gupta, 1990; Edvinsson, Brändström, Rogers, & Broström, 2005; Janssens, Messelink, & Need, 2010; Vandezande, 2012). Studies conducted in historical populations and in developing countries have in fact evidenced that there are large proportions of families that do not experience any infant deaths while a more limited group of families experience multiple infant deaths. In the nineteenth-century parish of Nedertorneå (northern Sweden), for example, nearly 45% of mothers did not experience any infant deaths, while a large number of children dying in their first year of life were born to around 19% of mothers (Brändström, 1984). These types of studies have identified the importance of considering the family instead of a single child as the unit of analysis (Edvinsson et al., 2005). The observed clustering in infant mortality could be possibly due to genetic inheritance, social and cultural factors related to education, socioeconomic status (SES), biological characteristics, and shared disease environment (Janssens et al., 2010).

Clustering of infant deaths within families has also been studied from an intergenerational perspective in the doctoral thesis of Vandezande (2012), who looked at transfers across the maternal and paternal line in the Belgian district of Antwerp during the second half of the nineteenth century. The results showed that mothers born in families which experienced high mortality were more likely to lose their own infants. By categorising by age at death the variable measuring the number of a mother's siblings that died in infancy, a strong correlation was found. The strongest impact on the likelihood of death of a woman's offspring was found if her siblings died in the late neonatal period (days 8-30 of life). Intergenerational transmissions were also found across the paternal line: fathers whose siblings died in the postneonatal period (one month to one year of age) were more likely to experience death of their infants, particularly during the late neonatal and postneonatal period, while those whose siblings died in the early neonatal period (from birth to day 7 of life) had reduced risks of losing their infants.

Although there is no existing research focusing on intergenerational transfers in infant mortality for Scania, various works have looked at some of the determinants of the risk of dying in the first year of life in this region. For example, a study aimed at quantifying the family frailty effect in infant and child mortality between 1766 and 1898 found that the effects of unmeasured differences between families were much stronger than the impact of socioeconomic status or gender (Bengtsson & Dribe, 2010). Infants from high-risk families experienced between 60 and 105% higher risk of dying than infants from low risk families. A strong effect of family-level factors in the first year of life, possibly due to observable characteristics such as the presence of parents or unobservable factors such as habits of breastfeeding and care, was also found in other works (Bengtsson, 2004; Oris, Derosas, & Breschi, 2004). These unobserved effects may be in part related to biological or socioeconomic characteristics that are transferred across generations.

Identifying possible causes of intergenerational transfers of infant mortality requires an understanding of the factors which affect infant health and their likelihood of dying. The framework developed by Mosley and Chen (1984) tried to combine social and medical approaches to study child survival in developing countries and, in doing so, they identified five groups of proximate determinants of child survival: maternal factors (age, parity and birth interval), environmental contamination, nutrient deficiency, injury and personal illness control. These authors identified a mixture of socioeconomic and biological factors at the individual, family and community level, which influence these proximate determinants, thus in turn affecting child health and mortality. Das Gupta (1990) argued that in addition to socioeconomic determinants, the innate

abilities of the mother, independently of her education, occupation, income and wealth, have strong impacts on child health and mortality and can partly explain the clustering of deaths in certain families. Johansson (2004) developed the model by Mosley and Chen further to study child mortality in a historical setting. He placed larger emphasis on the role of nutrition and disease as intermediate variables. In his work, individual level factors which influence nutrition and disease exposure include sex and maternal characteristics; family level factors include income, wealth, knowledge, values and preferences, and genetics; community level factors include environmental conditions, disease load and food supply. Many of the individual, family and community level factors that determine the levels of nutrition and disease among children and in turn their risk of dying, can be transmitted across generations.

The importance of nutrition and infection as two of the main determinants of mortality in historical populations has been widely emphasized in the literature. It has also been claimed that changes in such factors have led to the historical mortality decline. Nutrition and infection are strongly linked. Inadequate nutrition debilitates individuals and makes them more susceptible to disease (McKeown, 1976). Nutritional status, or net nutrition, is the balance between the quantity of food consumed (diet) and the claims made on such energy and nutrients for basic bodily maintenance, fighting diseases, and performing work and other activities (Floud, Fogel, Harris, & Hong, 2011; Fogel, 2004). Infectious diseases worsen the nutritional status of an individual not only because energy is required to activate the immune system in order to fight the diseases, but also because during periods of illness there is a loss of appetite, a reduced capacity to absorb ingested nutrients and an increased metabolic loss of nutrients (Dasgupta, 1993; The Journal of Interdisciplinary History, 1983). The role of nutrition and disease for mortality among infants is more complex, since such factors can also operate through the health and capacities of the mother during pregnancy and lactation. Maternal nutrition during pregnancy can affect birthweight, and during lactation it can influence the quantity of breastmilk and its nutritional quality. Causes and mechanisms of infant mortality could also vary depending on the time when the death occurs. Early neonatal mortality is often related to maternal health and nutrition during gestation, pregnancy complications, obstetric problems during delivery, premature birth, low birth weight, malformations, the new-born child's lack of adaptation to the extra uterine environment and poor partum or post-birth hygiene, while late neonatal deaths are more frequently linked to infections and poor after-birth care (World Health Organization, 2006).

Inadequate nutrition and exposure to disease not only affect individuals at the time when such exposures occur, but they also have long-term consequences. It has been shown in the literature that infectious disease outbreaks or nutritional deprivation in early life influence health in young- and old-age as well as cognitive ability, education, and income and socioeconomic status in working ages (e.g. Almond & Currie, 2011; Barker, 1994; Bengtsson & Lindström, 2000; Case & Paxson, 2010; Gluckman, Hanson, & Buklijas, 2010; Lindeboom, Portrait, & van den Berg, 2010; Quaranta, 2014). Evidence of transfers of these effects across generations was also observed (Quaranta, 2013). Intergenerational transfers in infant mortality could therefore partly be explained by adverse early life exposures shared by the mother and her siblings. Such exposures could have caused the early death of one or more of the mother's siblings and could have scarred the mother, negatively affecting her health and wellbeing later in life, including her reproductive health, the viability of her offspring and her capacity to care for them. Research conducted in Scania for similar periods as the current work found that individuals exposed to adverse conditions in early life, most importantly to a high disease load in their year of birth, had increased mortality and worse wellbeing later in life (Bengtsson & Lindström, 2000; 2003; Johansson, 2004; Quaranta, 2013; 2014). It was also seen that offspring born to mothers who were exposed to a high disease load in infancy experienced higher neonatal mortality, and women who were exposed in infancy to whooping cough gave birth to a lower proportion of boys, probably as a result of a higher incidence of spontaneous abortions of male foetuses (Quaranta, 2013).

Other possible explanations of intergenerational transmissions in infant mortality are intergenerational transmissions of SES. SES determines house quality, crowding and the capacity of families to put food on the table. It is also associated with parental education, values and preferences, which affect breastfeeding and other aspects of the care given to infants and children. All of these factors have an impact on infant and child mortality by influencing the nutritional level of children and their risk of being infected by disease (Johansson, 2004). SES also affects weight and other indicators of health at birth through the mother's nutrition, health and behaviour during pregnancy. Even if, over the past century, living standards have risen, advancements were made in medicine, sanitation and housing, and infant mortality has declined, the inverse relationship between social class and infant mortality has not narrowed (Antonovsky & Bernstein, 1977). Evidence of transmittance of SES across two and three generations was in fact found and infant and child mortality can therefore be associated also with the SES of grandparents. It was shown, for example, that children born in high-SES families are more likely to attain high SES in adulthood (e.g. Solon, 1999). In a study that used

data from Scania for the period 1815-2011, an association was also found between the class or occupational status of grandfathers and grandsons' SES, net of the association between fathers and sons, although no grandparental effect was found for earnings (Dribe & Helgertz, 2016). In pre-industrial populations, there was a strong degree of social homogamy in marriages, a pattern also observed in nineteenth-century Scania (Dribe & Lundh, 2009). This means that the SES of paternal grandparents was highly correlated with the SES of maternal grandparents. Given that SES is transmitted across generations and that there is a strong degree of SES-homogamy in marriages, mothers born into low SES families are more likely to be of low-SES in adulthood, and since SES influences infant mortality, low SES families are likely to have experienced more infant deaths in both the grandmothers' and the mothers' generations.

Intergenerational transfers in infant mortality could also be related to common reproductive characteristics of mothers and grandmothers. Mother's age, birth order and previous birth interval, all of which are determined by the reproductive behaviour and the reproductive health of women, have a strong impact on the health and survival of infants and young children (e.g. Hobcraft, McDonald, & Rutstein, 1985; Nault, Desjardins, & Légaré, 1990). Breastfeeding plays a large role in some of these patterns. Other factors shown to be related to increased risks of early death in certain families include family size (Zaba & David, 1996), maternal ability (Das Gupta, 1990; 1997), maternal death (Pavard, Gagnon, Desjardins, & Heyer, 2005), remarriage of the mother and earlier stillbirths (Edvinsson et al., 2005). Studies have shown, to various degree, intergenerational transmissions of fecundity, fertility intentions, parental behaviour and other indicators of reproduction (e.g. Anderton, Tsuya, Bean, & Mineau, 1987; Bras, Van Bavel, & Mandemakers, 2013; Jennings, Sullivan, & Hacker, 2012; Kotte & Volker, 2011; Reher, Ortega, & Sanz-Gimeno, 2008). Associations between the ages at marriage of parents and of their children have also been found, which can partly be attributed to social class (e.g. van Poppel, Monden, & Mandemakers, 2008).

Transmissions in infant mortality could also be related to genetics. The likelihood of death of foetuses during pregnancy and of babies during infancy is influenced by their genetic components. For example, earlier research has suggested that Rh disease could explain part of the clustering of perinatal deaths found in some families (Häggström, Lundevaller, & Edvinsson, 2012). This type of disease leads to stillbirths of Rh-positive foetuses from an Rh-negative mother and similar patterns could occur in the next generation if a surviving Rh-negative daughter conceives Rh-positive children.

The current work is structured into five sections. The first section presents the area and data sources. In section 2 a description of the steps that were carried out to transfer the database into the IDS is described. Section 3 presents the results in two parts. The first part consists of the basic analysis, which follows the same methods and models as the other four studies conducted within the common project. The second part extends this analysis by estimating models that also control for SES, and that measure the effects separately for boys and girls and for children born in different time periods. Section 5 discusses the findings and concludes the work.

2 AREA AND DATA SOURCES

The area considered for this study comprises five rural parishes, Hög, Kävlinge, Halmstad, Sireköpinge and Kågeröd, located approximately 10 km from the coast in the western part of Scania, the southernmost province of Sweden. The most closely situated towns (10 to 30 kilometres away) are Lund, Landskrona and Helsingborg. Kågeröd, Halmstad and Sireköpinge formed a continuous district, while Hög and Kävlinge were located approximately 10 kilometres to the south.

The five parishes studied are not representative of Sweden nor of Scania. However, they are compact in their geographical location and presented differences in terms of size, topography and socioeconomic conditions that were common to peasant societies (Bengtsson & Dribe, 1997). Consequently, the life courses of people who lived and worked in these communities were similar to those of most families in rural settings (Bengtsson, 2004). Furthermore, the geographical heterogeneity of these localities was limited.

Life expectancy at birth and fertility were somewhat higher in the parishes than for Sweden as a whole during the nineteenth century, but closely followed the same development over time (Bengtsson & Dribe, 2010; Quaranta, 2013). Mortality started to decline in the late eighteenth century, initially through a fall of infant mortality, closely followed by child mortality. In the parishes, infant mortality fell from about 250 per thousand in the 1760s to about 10 per thousand in 1900 (Johansson, 2004), which is similar to the development for Sweden as a whole (Hofsten & Lundström, 1976). Adult mortality also started to fall from

the mid-nineteenth century. Life expectancy at birth increased from about 40 years in the beginning of the nineteenth century to around 50 years in 1900 (Quaranta, 2013). In the period that preceded the fertility decline, age-specific fertility was somewhat higher in Scania than in Sweden as a whole (Bengtsson & Dribe, 2010). The decline in fertility in Sweden began in the 1880s and lasted for approximately 50 years (Dribe, 2009). In Scania the decline began slightly later, in the 1890s, and it continued without interruption until the 1930s.

The source material used for this work comes from the Scanian Economic Demographic Database (SEDD - Bengtsson, Dribe, Quaranta, & Svensson, 2014), administered by the Centre for Economic Demography, Lund University, Sweden. The SEDD contains data on births, deaths and marriages occurring in the years 1630-1968 in the five rural parishes, obtained from church books maintained by the clergy. After 1813 register type data obtained from catechetical examination registers (*husförhörslängder*) is also available,[1] which, in addition to vital events, also contains information on migration. The family reconstitutions were performed automatically using a computer program, and the results have been checked manually. The methods used were described and evaluated thoroughly in a previous work by Bengtsson and Lundh (1991).

The quality of the data is very high, as has been shown in previous works. The percentage of stillbirths and the rates of infant and neonatal mortality give no indication of serious recording problems for the years 1766 to 1865 (Bengtsson & Dribe, 2010). Calculations for the years 1813 to 1898 of the proportion of infant deaths taking place during the first month of life and of the sex ratio at birth show no indications of problems with the data either (Quaranta, 2013).

In addition to data from parish and catechetical examination registers, the SEDD contains information from poll-tax records and land registers (*mantalslängder*). These registers were used annually for the collection of taxes and, among other things, they provide for each year information on the size of the landholding, therefore evidencing the productive potential of the farms (Dribe, 2000). The quality and representativeness of the poll-tax records improved starting from 1766, when a much greater proportion of landless families were included in the registers.

Data on occupations of individuals and of household heads, obtained from church books, poll-tax and income registers is also available in the SEDD. As this area was characterized by very high migration rates, when constructing the SEDD, married individuals were traced back to their birth parish where possible to include information regarding the occupations of their fathers at the time of birth. Occupations in SEDD have been coded into HISCO and later classified according to HISCLASS (van Leeuwen, Maas, & Miles, 2002), which is a categorical social class scheme. In this work occupations were also coded into the HISCAM scheme (Lambert, Zijdeman, Van Leeuwen, Maas, & Prandy, 2013).[2] Alternative to HISCLASS, which links occupations with positions in a theoretical model of the social class structure, HISCAM uses an empirical model to place occupations within a continuous dimension of social stratification.

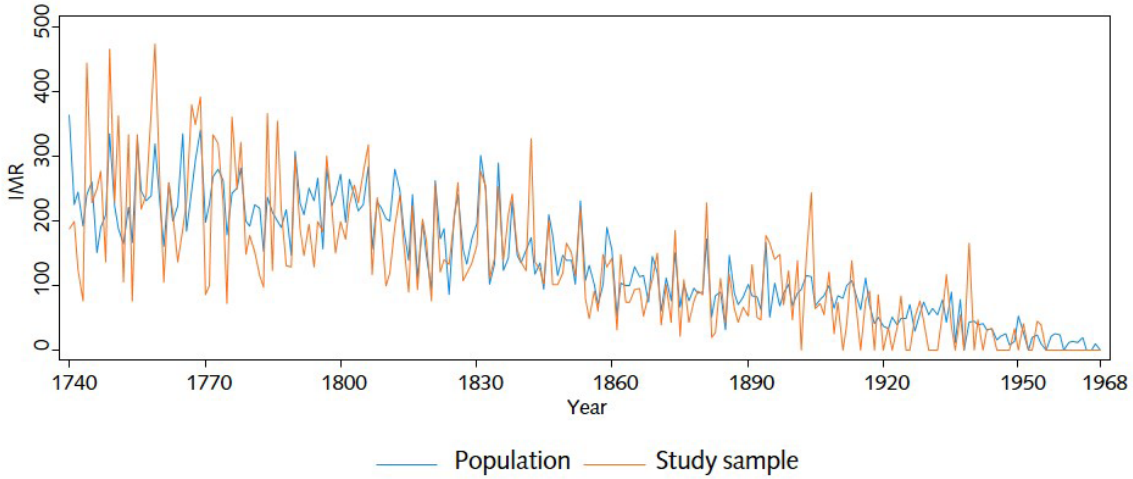
The sample selected for analysis in the current work consists of women for whom we have information about their births and infant mortality/survival of their offspring and for whom we have information about the births and infant mortality/survival of their mothers (the 'grandmothers'). In addition, grandmothers must have given birth to at least two children. The sample includes 1874 grandmothers born between 1664 and 1929, 2451 mothers born between 1700 and 1949 and 8770 children born between 1740 and 1968, of whom 1162 died in infancy. Mothers born before 1700 and children born before 1740 were excluded due to small numbers.

Figure 1 shows the development of the intant mortality rate (IMR) in Scania for the period 1740 to 1968, for the full population including in SEDD and for the selected study sample. Large year to year fluctuations can be observed and, with the exception of the initial years, a declining trend is also seen. A steeper decline in the trend is observed from around 1810 to around 1870. IMR fell to values below 100 per 1000 during the second decade of the 20th century. The patterns displayed in Scania are similar to those observed for Sweden as a whole, where infant mortality declined rapidly and continiously starting from the 18th century.

1 Catechetical examination registers were started in 1813 in Kågeröd, 1821 in Halmstad and Sireköpinge and 1829 in Hög and Kävlinge.

2 Version U2, male only, 1800-1938 was used.

Figure 1 *Infant mortality rate in Scania, 1740-1968*



3 IMPLEMENTATION OF THE IDS IN THE SEDD

The transfer of SEDD into IDS was made in two steps, producing two different databases. The initial implementation, which was conducted in 2009, included data for the periods when catechetical examination records were available: 1813-1968.[3] In the SEDD population register IDS database, individuals can be followed across the five parishes at all times. Besides storing information found in the sources into the five IDS tables, many variables were constructed for research purposes and were stored in extended IDS tables (EIDS - see Quaranta, 2015).

The initial implementation of SEDD into IDS was done in Microsoft Access in a modular basis, grouping common sets of queries into separate files. Additional variables and programs created after 2009 were made using Microsoft SQL Server Management Studio. The source database is currently stored in SQL server. Various files of queries produced in Access and Microsoft SQL Server Management Studio are connected to the source database to produce the IDS and EIDS tables, which are also stored as an SQL server database. Researchers using the SEDD are given a Chronicle and a Variable Setup file (see Quaranta, 2015) containing all available variables. The IDS and EIDS tables will also be made available for researchers in the future.

The SEDD had been used for research over several decades before transferring the data into the IDS, which meant that many important decisions such as how to make links between individuals and between individuals and the context in which they lived in, had already been taken before the transfer to the IDS. Some of the difficulties that are commonly encountered when transferring newer databases into the IDS were not encountered for SEDD. Nevertheless, given that SEDD was the first database to adopt the IDS, many ad-hoc solutions were used. Some changes are therefore needed in order to comply fully with the specifications of the latest version of IDS and prior to making the IDS tables directly available to researchers.

One of the main difficulties encountered when transferring the SEDD into the IDS was the implementation of the context. In the Swedish case, the family often represents the consumption unit. When the SEDD was originally created individuals were linked to a family ID and to a household ID. Different families could live in the same household. Members of the same family could also live in different households if, for example, a married woman was a servant or a lodger in a different house during a certain period. In addition to information about families and households, the SEDD also contains data from poll-tax registers. Each entry in the poll-tax register is linked to a family head, and if a family head owned more than one property, he/she would be linked to more than one poll-tax register the same year. To allow storing these different types of information in the IDS tables, three

3 As stated earlier, in some parishes catechetical examination records were not available until 1821 or 1829.

types of contexts were created in the CONTEXT table: family, household and poll-tax. In the SEDD IDS tables, at each point in time individuals are linked to a family context and to a household context in the INDIV_CONTEXT table. Poll-tax registers are linked to families in the CONTEXT_CONTEXT table. Start and end dates in the INDIV_CONTEXT table were assigned using event and migration dates from the catechetical examination registers.

The data for the period preceding 1813 was also transferred into the IDS recently, creating the SEDD family reconstitution IDS database. The population register and family reconstitution databases were stored as two separate databases, given that they differ in their characteristics and in the methods that need to be employed when using them for research. For the current study, however, the two databases were combined to allow following individuals from 1740 to 1968. It was possible to do so, since the common software used in this project can be run with databases created from church books, civil registers, or population registers. The SEDD family reconstitution IDS database contains data on births, deaths and marriages from parish records and yearly information from poll-tax registers for the periods preceding the start of the catechetical examination registers of each parish. Besides storing relationships between individuals in the INDIV_INDIV table, links between individuals and their family identifier were also included in the INDIV_CONTEXT table to facilitate also in this database the linkage of information from poll-tax registers. The start date in the time stamp of such row of the INDIV_CONTEXT table was set to be the birth or marriage date if such events were observed for the individual, and was left blank otherwise. The end date was set to be the date of death if such event was observed. An end date was also set in the links between individuals and their family of birth when a marriage event was observed. Family identifiers and information from poll-tax registers were stored in the CONTEXT table. Families were linked to their corresponding poll-tax registers using the CONTEXT_CONTEXT table.

Besides providing important information about the land, poll-tax registers were also used as additional measures of exposure for the pre-1813 period, therefore using a-so-called mixture of family and farm reconstitutions. For example, we could have a woman with an observed marriage event on May 13 of 1780 and poll-tax declarations for her family of birth for the years 1766-1779 and for her family of marriage for 1780-1810. Using pure family reconstitution such woman could not be included in the sample of grandmothers in the current study, since her death was not observed. Instead, by using a mixture of family and farm reconstitution, we can consider such woman in the sample of grandmothers from 1780 to 1810. Since her family paid taxes until 1810, we can assume that this was the last year she was present in the study area and thus under exposure. In order to be able to use information from poll-tax registers to define exposure when using the common software produced for the current project, the event "Poll_tax_declaration" was added to the INDIVIDUAL table for all individuals who belonged to each family on each date of declaration of the poll-tax.

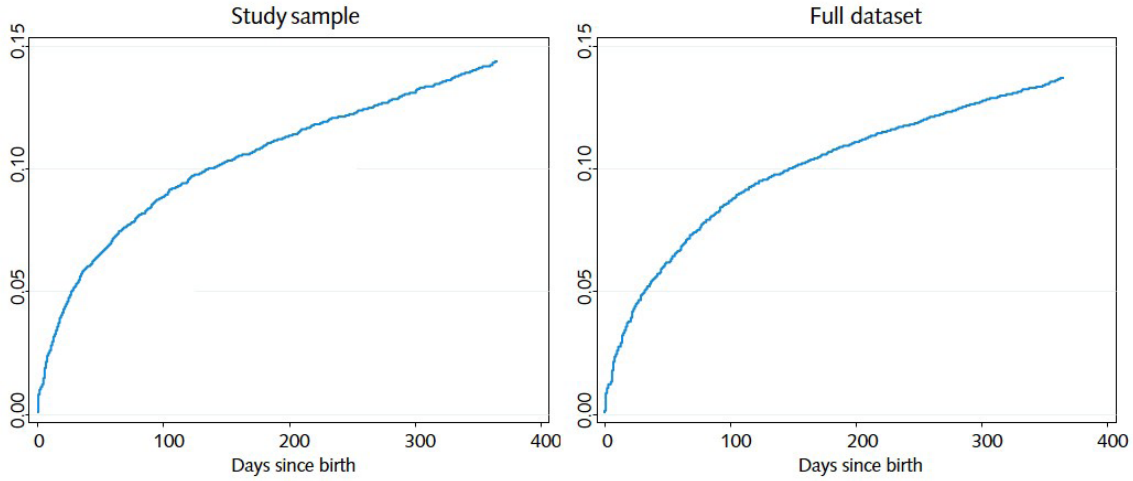
4 METHODS

This work is one of five studies that shares the same theoretical framework and methodology. The code developed by Quaranta (2016; 2018) was used to construct the dataset for analysis and run the basic statistical models. Figure 2 shows that the cumulative hazard of death in the first year of life for the study sample was rather similar to the cumulative hazard for the full dataset. Therefore, there should not be large biases that are caused by the way the sample for analysis was selected.

Within the basic analysis that is common across the five works included in the project, survival models are estimated, which consider as dependent variable the likelihood that a woman's child died in infancy and as explanatory variables the number of infant deaths of the grandmother (categorical variable: 0, 1, 2+), the number of births of the grandmother (categorical variable: 2, 3, 4-6, 7+), the sex of the child (categorical variable), the birth order of the child (categorical variable: 1, 2, 3, 4-6, 7+), the date of birth of the child (centred) and the age of the mother at the birth of the child (categorical variable: 15-24, 25-34, 35-49). A Cox model is first estimated, followed by two Weibull models that include a shared frailty component based on the mother and grandmother respectively. Shared frailty allows to control for shared characteristics of the mother in the likelihood of death of all her children. As a sensitivity analysis additional Weibull models with shared frailty on the mother are estimated, making different restrictions to the data to select only cases where the maternal grandmother was observed

during her entire reproductive period (using various definitions of such observation period), and/or which censor on their date of birth children that had no additional observations for themselves or their mothers after such date.

Figure 2 *Cumulative hazard of death in the first year*



Using the same dataset an extended analysis is also conducted specifically for Scania. The first part takes into consideration the occupation of the father at the time of the child's birth, operationalized using the HISCAM score. The study next considers whether there are differences in the effects for boys and girls. Models that consider the likelihood of death in infancy according to the number and sex composition of deaths of the maternal grandmother's offspring are estimated as well. The last part of the extended analysis focuses on different time periods.

5 RESULTS

Basic models

Descriptive statistics are shown in Table 1. As can be seen in the table, 27% of children had grandmothers who experienced the death in infancy of one of their offspring, and 22% of children had grandmothers who experienced 2 or more infant deaths. The grandmothers of more than 80% of children gave birth to 4 or more children. About 50% of children were born to mothers who were 25-34 years old, and the mean year of birth for children in the sample is 1851.

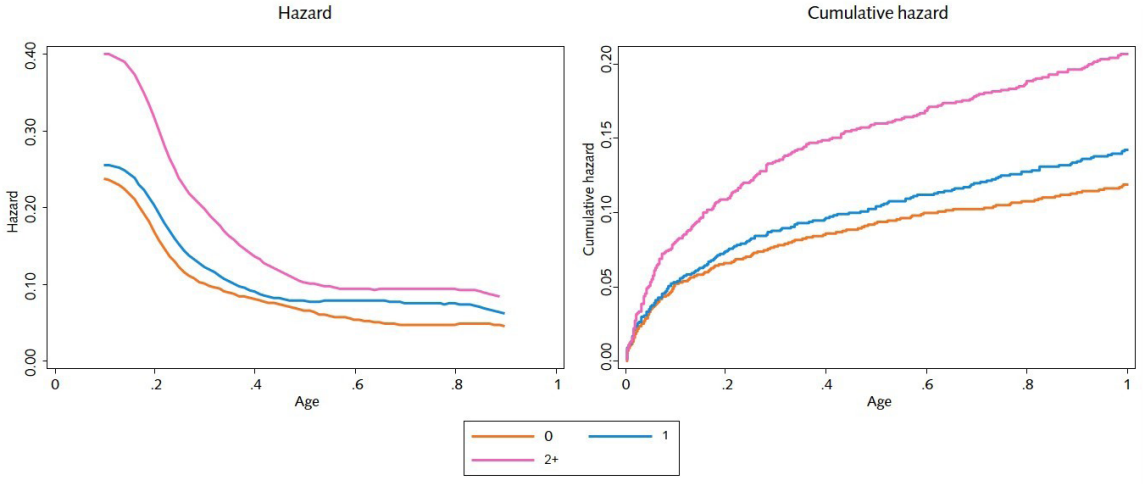
The hazard and cumulative hazard curves (Figure 3) show that, during the period 1740-1968, children whose maternal grandmothers had experienced infant deaths were at higher risk of dying in infancy. These patterns are confirmed by the results of the survival models shown in Table 2. Children whose maternal grandmothers had experienced two or more infant deaths were between 21% and 24% more likely to die in infancy. Such result were statistically significant at the 5% level in all three model specifications. Children whose maternal grandmothers experienced one infant death were between 3% and 6% more likely to die in infancy, although such results were not statistically significant.

The effects of control variables were similar across the three models estimated. The risks of death in infancy were higher for boys relative to girls and for first born children relative to higher parities. Reductions in mortality across time are also seen, while there were no significant differences in the risk of death in infancy by mother's age. The frailty variance components were high and statistically significant in the model that considered shared frailty on the mother as well as in the model that considered shared frailty on the grandmother, with similar values for the two models. This shows that there were unobserved shared characteristics within each family and that it is important to account for them in the estimations.

Table 1 *Descriptive statistics, Scania 1740-1968*

	1740-1864		1865-1968	
	HR	p-value	HR	p-value
N. of infant deaths of the grandmother, (ref: 0)	1,000	ref.	1,000	ref.
1 infant death	0,996	0,970	0,971	0,873
2+ infant death	1,281	0,011	0,671	0,172
N. of births of the grandmother (ref:2)	1,000	ref.	1,000	ref.
3 births	1,302	0,191	0,708	0,355
4-6 births	1,144	0,420	0,773	0,387
7+ births	1,113	0,535	1,067	0,828
Child's sex, (ref: girl)	1,000	ref.	1,000	ref.
Boy	1,129	0,068	1,280	0,081
Birth order (ref:1)	1,000	ref.	1,000	ref.
2	0,704	0,001	1,151	0,516
3	0,740	0,009	1,213	0,427
4-6	0,731	0,003	1,109	0,658
7+	0,893	0,416	1,302	0,399
Child birth date centered	0,993	0,000	0,981	0,000
Mother age 15-24	1,088	0,410	0,953	0,818
25-34 (ref.)	1,000	ref.	1,000	ref.
35-50	1,126	0,187	0,766	0,187
Intercept	0,150	0,000	0,213	0,000
Frailty variance	0,235	0,000	0,596	0,000
N of children	5434		3336	
N of infant deaths	949		213	

Figure 3 *Hazard and cumulative hazard of death in infancy by the number of infant deaths experienced by the grandmother, Scania 1740-1968*



Tests to check whether there were any violations of the proportional hazards assumptions were conducted after the Cox models. No violations of the assumption were observed for the main explanatory variable. For the control variables only the child's birth order shows strong violations of the assumption (p-value <0.001 for the last two categories). Child sex also shows slight violations (p-value 0.09). However, since these variables are only used as controls in the models such violations in the proportional hazards assumption are not a major concern for the study as they should not alter the effect of the intergenerational transmission variable.

Table 2 *Intergenerational transmissions in infant mortality, survival models, Scania, 1740-1968*

	Cox model		**Weibull model**		**Weibull model**	
			mother shared frailty		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	1,033	0,665	1,044	0,605	1,047	0,592
2+ infant death	1,209	0,018	1,225	0,025	1,240	0,023
N. of births of the grandmother (ref:2)	1,000	ref.	1,000	ref.	1,000	ref.
3 births	1,228	0,195	1,217	0,266	1,227	0,252
4-6 births	1,140	0,311	1,126	0,414	1,121	0,440
7+ births	1,193	0,183	1,195	0,232	1,188	0,255
Child's sex, (ref: girl)	1,000	ref.	1,000	ref.	1,000	ref.
Boy	1,160	0,012	1,161	0,013	1,162	0,013
Birth order (ref:1)	1,000	ref.	1,000	ref.	1,000	ref.
2	0,797	0,018	0,782	0,011	0,785	0,012
3	0,846	0,107	0,827	0,069	0,833	0,079
4-6	0,850	0,082	0,813	0,031	0,817	0,035
7+	1,104	0,401	0,992	0,950	0,997	0,979
Child's birth date centered	0,990	0,000	0,990	0,000	0,990	0,000
Mother's age 15-24	1,039	0,668	1,039	0,680	1,051	0,585
25-34 (ref.)	1,000	ref.	1,000	ref.	1,000	ref.
35-50	1,009	0,914	1,047	0,570	1,054	0,519
Intercept			0,124	0,000	0,123	0,000
Frailty variance			0,265	0,000	0,256	0,000
N. of children	8770		8770		8770	
N. of infant deaths	1162		1162		1162	

The patterns of results remained consistent in the survival models estimated as sensitivity analysis (Table 3). Children whose maternal grandmothers had experienced 2 or more infant deaths had between 22% and 37% higher risks of dying in infancy. Such results were statistically significant in Models 1-4 and 6, and just above the threshold for statistical significance in Model 5 (grandmother observed at least from age 20 to 50 and none of the children of the grandmother had an unknown birth date). No statistically significant differences in the risk of death in infancy for children whose maternal grandmother experienced only one infant death was observed in any of the models estimated in the sensitivity analysis.

Table 3 *Sensitivity analysis for intergenerational transmissions in infant mortality, Weibull models with mother shared frailty, Scania, 1740-1968*

	Model 1		Model 2		Model 3		Model 4		Model 5		Model 6	
	HR	p-value	HR	p-value	HR	p-value	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.	1,000	ref.	1,000	ref.	1,000	ref.
1 infant death	1,044	0,605	1,070	0,554	1,007	0,951	1,057	0,646	0,961	0,746	1,073	0,538
2+ infant death	1,220	0,028	1,357	0,011	1,302	0,017	1,374	0,012	1,232	0,108	1,354	0,011
N. of births of the grandmother (ref:2)	1,000	ref.	1,000	ref.	1,000	ref.	1,000	ref.	1,000	ref.	1,000	ref.
3 births	1,216	0,267	0,895	0,643	0,843	0,451	0,869	0,588	0,938	0,794	0,895	0,642
4-6 births	1,122	0,429	0,639	0,026	0,742	0,117	0,610	0,019	0,684	0,073	0,639	0,026
7+ births	1,192	0,239	0,769	0,191	0,836	0,351	0,728	0,132	0,901	0,627	0,770	0,195
Child's sex, (ref: girl)	1,000	ref.	1,000	ref.	1,000	ref.	1,000	ref.	1,000	ref.	1,000	ref.
Boy	1,159	0,014	1,149	0,090	1,130	0,109	1,163	0,087	1,119	0,197	1,149	0,090
Birth order (ref:1)	1,000	ref.	1,000	ref.	1,000	ref.	1,000	ref.	1,000	ref.	1,000	ref.
2	0,779	0,010	0,707	0,008	0,679	0,002	0,654	0,003	0,717	0,015	0,706	0,007
3	0,823	0,062	0,645	0,002	0,649	0,001	0,669	0,009	0,646	0,004	0,644	0,002
4-6	0,811	0,029	0,680	0,003	0,715	0,005	0,686	0,006	0,686	0,006	0,681	0,003
7+	0,989	0,931	0,794	0,177	0,828	0,235	0,802	0,227	0,730	0,085	0,795	0,179
Child birth date centered	0,990	0,000	0,991	0,000	0,991	0,000	0,991	0,000	0,990	0,000	0,991	0,000
Mother age 15-24	1,040	0,668	0,935	0,590	0,967	0,778	0,926	0,569	0,923	0,543	0,936	0,595
25-34 (ref.)	1,000	ref.	1,000	ref.	1,000	ref.	1,000	ref.	1,000	ref.	1,000	ref.
35-50	1,046	0,585	1,119	0,316	1,095	0,381	1,080	0,527	1,133	0,290	1,116	0,327
Intercept	0,125	0,000	0,210	0,000	0,197	0,000	0,221	0,000	0,209	0,000	0,210	0,000
Frailty variance	0,265	0,000	0,180	0,000	0,200	0,000	0,167	0,000	0,196	0,000	0,180	0,000
N of children	8770		4642		5302		4035		4058		4642	
N of infant deaths	1162		621		717		538		554		621	

Note: Model 1 – Children under observation; Model 2 – grandmother observed at least from age 20 to 50; Model 3 – grandmother observed at least from age 20 to age 50 or death; Model 4 – grandmother observed at least from age 20 to 50 and first husband of the grandmother observed at least until the grandmother's 50th birthday; Model 5 – grandmother observed at least from age 20 to 50 and no children with unknown birthdates for the grandmother; Model 6 – grandmother observed at least from age 20 to 50 and children under observation.

Extended models

The first part of the extended analysis takes into account occupation of the father. Survival models were estimated controlling for the HISCAM score[4] of the father's occupation at the time of the child's birth (Table 4). It was not possible to determine an occupation, either because of unknown data or because it was not possible to link the occupation to a HISCAM score, for 1209 children, who were excluded from the models. For other children, HISCAM scores ranged between 39.9 and 99. When controlling for occupation, the results remained consistent: children whose maternal grandmothers had experienced infant deaths were more likely to die in infancy. Statistical significance was, also in this case, only observed among children whose grandmothers had experienced 2 or more infant deaths. These children were 32% more likely to die than children whose grandmothers did not experience any deaths. A hazard ratio slightly below 1 was obtained for the HISCAM score, evidencing that infant mortality declined as socioeconomic status improved. When introducing an interaction between the intergenerational transmission variable and the occupational score, children whose grandmother

4 The models incorporated a centred value of the HISCAM score. The variable is centred by subtracting from each HISCAM score 51.22, the mean score among children in this area and study period. Centring this variable does not affect the estimations, but it facilitates the interpretation of the results.

experienced 2 or more infant deaths showed higher risk of dying in infancy, with hazards that declined with increasing occupational score.

Table 4 *Intergenerational transmissions in infant mortality, simple and interaction survival models considering SES, Scania, 1740-1968*

	Simple model		Interaction model	
	HR	p-value	HR	p-value
N. of infant deaths of the grandmother (ref: 0)	1,000	ref.	1,000	ref.
1 infant death	1,119	0,233	1,099	0,330
2+ infant death	1,321	0,008	1,282	0,021
Hiscam score	0,980	0,014	1,000	0,995
G0 1 infant death * Hiscam score			0,962	0,118
G0 2+ infant death * Hiscam score			0,952	0,026
N. of births of the grandmother (ref: 2)	1,000	ref.	1,000	ref.
3 births	1,189	0,411	1,217	0,353
4-6 births	1,100	0,594	1,118	0,534
7+ births	1,279	0,174	1,305	0,142
Sex of child (ref: girl)	1,000	ref.	1,000	ref.
Boy	1,224	0,004	1,225	0,004
Birth order (ref: 1)	1,000	ref.	1,000	ref.
2	0,754	0,012	0,756	0,013
3	0,784	0,046	0,788	0,051
4-6	0,777	0,024	0,779	0,026
7+	0,990	0,945	0,997	0,982
Child birth date centered	0,992	0,000	0,992	0,000
Mother age 15-24	0,985	0,885	0,980	0,855
25-34 (ref.)	1,000	ref.	1,000	ref.
35-50	1,050	0,608	1,042	0,666
Intercept	0,106	0,000	0,104	0,000
Frailty variance	0,311	0,000	0,312	0,000
N of children	7561		7561	
N of infant deaths	859		859	

Table 5 shows the results of separate models estimated for boys and girls. Among girls, those whose maternal grandmothers had experienced two or more infant deaths had a 42% higher risk of dying in infancy, a result which was highly statistically significant. The effect for girls whose maternal grandmother had experienced only one infant death was small and statistically insignificant. For boys negligible and statistically insignificant effects were obtained for those whose maternal grandmothers had experienced 1 or 2 or more infant deaths. A model for all children that included an interaction between sex and the intergenerational transmission variable was also estimated. The difference in the hazard of death between boys and girls whose grandmothers had experienced 2 or more infant deaths was statistically significant. One possible explanation for this pattern could be that boys whose grandmothers had higher infant mortality of their offspring may be more strongly selected in-utero, something which would result in a lower proportion of male births for this group. No evidence of such pattern is, however, found in the data.

Table 6 shows the results of models that measure the likelihood of death in infancy according to the number and sex composition of deaths of the maternal grandmother's offspring. The reference category in the models are children whose grandmother did not experience the death of any of their offspring. In the model considering boys and girls, the risk of death was 38% higher if the grandmother had experienced two or more deaths of female offspring (statistically significant at the 5% level), 28% higher if the grandmother had experienced two or more deaths of male offspring (marginally statistically significant), and 17% higher if grandmother experienced two or more deaths of offspring of mixed sex (slightly above the threshold for statistical significance). It should be noted, though, that

the first two results are limited by small numbers since such groups of children only represented 3.5% and 4.5% of the sample respectively. Patterns in the same direction but with stronger magnitude and statistical significance were obtained when only focusing on girls, while no statistically significant effects were obtained in the model that considered only boys.

Table 5 *Intergenerational transmissions in infant mortality, sex specific models and interactions with sex, Scania, 1740-1968*

	Girls		**Boys**		**Interaction**	
	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	1,041	0,734	1,047	0,666	1,037	0,758
2+ infant death	1,416	0,005	1,078	0,527	1,431	0,002
Sex of the child (ref: girl)					1,000	ref.
Boy					1,264	0,011
G0 1 infant death * Boy					1,010	0,946
G0 2+ infant death * Boy					0,747	0,042
N. of births of the grandmother (ref:2)	1,000	ref.	1,000	ref.	1,000	ref.
3 births	1,042	0,870	1,397	0,139	1,221	0,258
4-6 births	1,038	0,856	1,222	0,286	1,128	0,407
7+ births	1,100	0,651	1,295	0,180	1,199	0,223
Birth order (ref:1)	1,000	ref.	1,000	ref.	1,000	ref.
2	0,804	0,138	0,774	0,048	0,782	0,011
3	0,874	0,382	0,811	0,145	0,830	0,075
4-6	0,801	0,122	0,847	0,200	0,808	0,027
7+	0,928	0,684	1,161	0,375	0,994	0,962
Child birth date centered	0,989	0,000	0,990	0,000	0,990	0,000
Mother age 15-24	1,012	0,933	1,070	0,577	1,038	0,686
25-34 (ref.)	1,000	ref.	1,000	ref.	1,000	ref.
35-50	1,208	0,112	0,890	0,294	1,044	0,599
Intercept	0,123	0,000	0,141	0,000	0,118	0,000
Frailty variance	0,240	0,000	0,248	0,000	0,266	0,000
N. of children	4250		4520		8770	
N. of infant deaths	527		635		1162	

Table 6 *Intergenerational transmissions in infant mortality by sex composition of the deaths of the maternal grandmother's offspring*

	All children			**Girls**			**Boys**		
	%	HR	p-value	%	HR	p-value	%	HR	p-value
N. of infant deaths of the grandmother (ref: 0)	51,736	1,000	ref.	52,351	1,000	ref.	51,159	1,000	ref.
1 female infant death	13,646	1,124	0,249	13,785	1,083	0,584	13,515	1,154	0,273
1 male infant death	13,199	0,967	0,755	12,725	0,984	0,918	13,645	0,959	0,764
2+ female infant deaths	3,562	1,376	0,049	3,573	1,833	0,004	3,552	1,038	0,866
2+ male infant deaths	4,467	1,282	0,096	4,057	1,666	0,011	4,852	1,022	0,912
2+ mixed infant deaths	13,389	1,171	0,124	13,509	1,225	0,159	13,277	1,126	0,376
N. of children		8770			4250			4520	
N. of infant deaths		1162			527			635	

Note: results estimated through Weibull regression models with shared frailty on the mother that also control for the number of births of the grandmother, the birth order and date of birth of the child, the age of the mother and, in the model for all children, also sex.

In this work intergenerational transmissions in infant mortality was studied for children born between 1740 and 1968. Given the large time horizon, the analysis was reproduced for different sub-periods: 1740-1864 and 1865-1968. The year 1865 represents an important dividing point in the history of this region. In fact, the period until 1864 includes the agricultural transformation, the early stages of industrialisation and the first phase of the demographic transition, with declining infant and child mortality (Bengtsson & Dribe, 2011). The second period was, instead, characterised by the breakthrough of industrialisation as well as by declines in adult mortality and, later, by a continued industrial expansion and the waning of the rural sector. Infant mortality rates were lower in the second period, as was shown in Figure 1, which also meant a change in the factors affecting the risk of death in infancy. As can be seen in Table 7, children born in 1740-1864, whose maternal grandmothers experienced two or more infant deaths had a 28% higher risk of dying in infancy than children whose maternal grandmothers had not experienced any infant deaths. These effects were slightly stronger than those observed in Table 2 when considering children born until 1968. Instead, for children born in 1865-1968, those whose grandmothers experienced 2 or more infant deaths showed lower risks of dying in infancy, although such effect was not statistically significant. The reversal in the effects across time could be related to underlying differences in the disease environment and composition of the study sample. The overall likelihood of dying of children was much lower during the second period (IMR of 174 per 1000 in 1740-1865 and 65 per 1000 in 1865-1968) and the proportion of children who had grandmothers that experienced two or more infant deaths was also lower (30% for children born in 1740-1864 and 8% for children born in 1865-1968).

Table 7 *Intergenerational transmissions in infant mortality over different periods, Scania*

	1740-1864		**1865-1968**	
	HR	p-value	HR	p-value
N. of infant deaths of the grandmother, (ref: 0)	1,000	ref.	1,000	ref.
1 infant death	0,996	0,970	0,971	0,873
2+ infant death	1,281	0,011	0,671	0,172
N. of births of the grandmother (ref:2)	1,000	ref.	1,000	ref.
3 births	1,302	0,191	0,708	0,355
4-6 births	1,144	0,420	0,773	0,387
7+ births	1,113	0,535	1,067	0,828
Child's sex, (ref: girl)	1,000	ref.	1,000	ref.
Boy	1,129	0,068	1,280	0,081
Birth order (ref:1)	1,000	ref.	1,000	ref.
2	0,704	0,001	1,151	0,516
3	0,740	0,009	1,213	0,427
4-6	0,731	0,003	1,109	0,658
7+	0,893	0,416	1,302	0,399
Child birth date centered	0,993	0,000	0,981	0,000
Mother age 15-24	1,088	0,410	0,953	0,818
25-34 (ref.)	1,000	ref.	1,000	ref.
35-50	1,126	0,187	0,766	0,187
Intercept	0,150	0,000	0,213	0,000
Frailty variance	0,235	0,000	0,596	0,000
N of children	5434		3336	
N of infant deaths	949		213	

6 DISCUSSIONS AND CONCLUSIONS

Using data for five rural parishes in Scania, the southernmost province of Sweden, for the period 1740-1968, the current work looked at whether the likelihood that a woman's children died in infancy was

affected by whether any of her siblings had died within their first year of life. This paper is one of five studies that analyses intergenerational transmissions in infant mortality by using a common program to create a dataset for analysis and run statistical models using databases that have been stored using the IDS. These programs, which are discussed in detail in the article by Quaranta (2018), have allowed to estimate a series of fully reproducible and comparable basic models that considered how the number of infant deaths experienced by the maternal grandmother affected the likelihood that a child would die in infancy. Using the dataset created by such programs it was also possible to estimate extended models that distinguished the possible confounding of SES and models that considered sex and cohort specific effects. The five studies conducted within the project had the aims of expanding our knowledge on intergenerational transfers and of showing the vast advantages of using the IDS for research.

The result of the current study show that in Scania there were intergenerational transmissions in infant mortality. Children whose maternal grandmothers had experienced two or more infant deaths were at a higher risk of dying in infancy, when controlling for the child's sex, birth order and birth date, the mother's age, and the number of births of the maternal grandmother. These results remained consistent also in models that only considered children whose grandmothers were observed for their entire reproductive periods or that controlled for occupation of the father at the time of the child's birth. Stronger intergenerational transmissions in infant mortality were observed for girls than for boys. Boys had, in general, higher mortality during their first year of life than girls, but their risk of dying was less dependent on whether their grandmothers' had lost any of their offspring in infancy than for girls.

The general patterns of transmissions in infant mortality observed in this work are in line with the results of the scant existing literature and of the other four studies conducted within the same project, all of which have found increased risks of deaths for children whose grandmothers had lost some of their offspring in infancy (Quaranta et al., 2017). The models estimated have established associations and at this stage they do not allow determining causal pathways, although some hypotheses of possible causes for these effects could be made. The results obtained could be partly explained by socioeconomic factors or maternal characteristics. The observed effects were strong even when controlling for basic demographic variables and occupation of the father, although further models need to be estimated to better evaluate the role of such factors. Other possible explanations include innate abilities of the mother, genetics or scarring effects triggered by adverse early life exposures shared by the mother and her siblings. Further studies are required to better understand the possible determinants of the intergenerational transfers observed and to disentangle the possible role of biological, behavioural and socioeconomic factors.

With regards to the differences found by sex, one possible explanation is that some families provided less adequate care to females than to males and that this behaviour was transferred across generations, although there are no studies for Sweden that show evidence of the existence of neglect of young girls. Another possible explanation for this pattern is that males whose grandmothers had higher infant mortality were more strongly selected in-utero. However, no differences were observed in the proportion of males according to the number of infant deaths experienced by the grandmother. More research is needed to determine whether the sex differences observed are specific to Scania or whether they are also found in other areas. One step in this direction would be to replicate the sex specific models in the four other territories considered in the project, as well as to estimate sex specific models in a pooled dataset that includes all these areas. Up to date the majority of existing studies looking into sex differences in intergenerational transmissions have only focused on psychosocial outcomes.

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