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Inherited dimensions of infant mortality. Detecting signs of disproportionate mortality risks in successive generations

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In an effort to unravel the diffusion and mechanisms of long-term fertility change, there is a growing body of literature on the intergenerational transmission of reproductive behavior. Several reproductive traits have already been studied in an intergenerational dimension, but the impact of infant deaths and their clustering within families on these intergenerational mechanisms of reproduction has been overlooked. This paper explores the continuity of infant deaths between successive generations. Data are from the COR*-database, a longitudinal multigenerational life course dataset covering the larger Antwerp area in the second half of the nineteenth century. We use the family as main unit of analysis, a perspective that proves a useful alternative to that of the individual child.

Keywords: infant mortality; death clustering; intergenerational transmission; family effects

1. Introduction

One of the current aims of the study of human reproduction is to unravel mechanisms of an intergenerational transmission of reproductive behavior (Bittles, Murphy, & Reher, 2008; Murphy, 1999). A better understanding of these mechanisms will help us to grasp how long-term change in fertility took place. Several aspects of reproduction have already received attention, such as age at entry to marriage (Van Bavel & Kok, 2009; Van Poppel, Monden, & Mandemakers, 2008), the interval between marriage and first birth (Desjardins, Bideau, Heyer, & Brunet, 1991), family size (Preston, 1976), the number of surviving children, and the length of the reproductive period (Reher, Ortega, & Sanz-Gimeno, 2008), and a combination of these, summarized as ‘patterns of fertility timing’ (Anderton, Tsuya, Bean, & Mineau, 1987). An element as yet barely touched upon is the intergenerational transmission of young age mortality. However, just as young age mortality is key to understanding fertility patterns—e.g. in crude versus net parity, or in the timing of subsequent births (see e.g. Preston, 1978)—the transmission of mortality risks from one generation to the next likewise is essential for the study of the intergenerational transmission of reproductive behavior. The effect of a potential transmission of mortality risks can be thought of in many ways. For example, a woman’s number of surviving children might reflect that of her mother because of an intergenerational transmission of fertility decisions, leading the two women to target similar family sizes (Reher et al., 2008). However, the number of surviving children in both generations is also influenced

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by the number of children lost in these two generations. Also, both mothers and their daughters might give birth to their respective children at fast paced rates, leading to short birth intervals, because neither of them breastfeeds intensively, a practice that hence could be assumed to be an intergenerational continuity. However, short birth intervals can equally likely result from a shared tendency of both mother and daughter to lose their infants soon after birth, instantly ceasing breastfeeding and leading to a shorter interval to the next birth. Intergenerational mechanisms of fertility are influenced—and in some occasions perhaps even caused—by an intergenerational transmission of infant (and child mortality). In this paper we aim towards filling this gap in the literature by describing the continuity of infant deaths between successive generations.

2. Infant mortality from a familial perspective

In the recent literature on young age mortality, much attention has been devoted to the perspective of the family. Deaths at young ages, and infant deaths in particular, seem to cluster in a rather limited number of families. This unequal spread of infant and child deaths is known as ‘death clustering’ (Das Gupta, 1990).

There is no consensus on the underlying causes of death clustering, and a diverse set of explanations has been put forward. Das Gupta, who studied the phenomenon in a contemporary context in Punjab, argued that clustering is caused by the basic abilities and personal characteristics of the mother, independently from her education or the household income. According to Guo (1993), who studied infant mortality in Guatemala, the effect of parental competence as it is described by Das Gupta must not be overstated. Genetic factors, he argues, probably play a role as well, although they prove difficult to measure. Ronsmans (1995) extended the discussion with a family’s strategy and ability to cope with crises, for example food shortages in present-day Kenya.

Several historical studies have found evidence of clustering as well. While searching for explanations on the level of the family, many studies have emphasized the role of a mother in the loss of her infants. Brändström (1988) has suggested that female labor conditions, combined with inadequate breastfeeding patterns, can explain strong patterns of clustering in Nedertorneå and Jokkmokk, northern Swedish parishes with very high levels of infant mortality. Recently the importance of female labor participation was reaffirmed by Janssens and Pelzer (2012) for some nineteenth century Dutch textile towns. They found this effect to be of greater importance than the social position of the father. Millward and Bell (2001) investigated the role of the mother’s health in both the unequal spread and long-term decline of infant mortality in nineteenth century Britain. Yet others, for example Reid (2001) in England, have pointed out a mother’s birth history, reasoning that multiple (difficult) deliveries wear down a mother’s health, and the survival chances of the subsequent births. In many of these studies it has been suggested that our understanding of young age mortality might improve if it was studied from the perspective of the mother (or the family), instead of that of the individual child. One possible way of doing this is focusing on mothers that lose a disproportionate number of their infants, or so-called ‘high-risk families’ (Edvinsson, Brändström, Rogers, & Broström, 2005).

In this study we investigated whether or not a mother is prone to lose her children during infancy. We predicted a mother’s risk to lose infants using the proportion of infants lost in the previous generation; i.e., the family in which a mother was raised as a child. If this proportion is high, we wanted to know if that affects the survival chances of a mother’s own children. Conversely, if this proportion is low, we wanted to assess whether a mother succeeds more often in keeping her infants alive.

3. Infant mortality risks inherited through successive generations

Differences in infant mortality result from a mix of determinants (Mosley & Chen, 1984). Some children are biologically weaker at birth, others have genetic disorders or weaknesses and yet others suffer from complications during delivery. These endogenous factors can exist aside from, or are at interplay with, exogenous determinants such as environmental, social, socioeconomic, and cultural differences (Lalou, 1997; Smith, 1991; Van Poppel & Mandemakers, 1997). Environmental differences result from, among others, the availability of and access to clean water; crowding and hygiene in the direct area of the residence; disease environment; availability of public health infrastructure such as sewers, hospitals, nursing homes; etc. (Van Poppel & Van der Heijden, 1997). Environmental differences interact with social differences, for example by means of residential segregation by wealth and social class (Smith, 1991). Within regions mortality levels differ according to differences in socio-economic positions, income, wealth, education and reading ability of the parents, but also depend on culture, in the definition of a complex set of values and norms shaping childcare practices (breastfeeding, hygiene practices) and fertility decisions (such as when to start reproduction, how many children to give birth to, and how much to spread out these births), either as a set of societal norms (e.g., influenced by religion) or as part of a familial tradition (Van Poppel & Mandemakers, 1997).

If mortality differences persist from one generation to the next, it is because the determinants that cause these differences (see above) persist through generations as well. Environmental effects, for example, can persist through time, causing differentials in infant mortality due to these effects to persist from one generation into the next. In that case, families residing in the same location for multiple generations are prone to the same environment, leading to a continuity in mortality risks from one generation to the next. Also, the socioeconomic context is often transmitted over successive generations, for example when intergenerational social mobility is limited (Grusky, 1983). Values and norms might be socialized in a family context; e.g., from parents to their children. The age at first marriage, for example, which is an indicator for the onset of the reproductive period, seems to be transmitted from mother to daughter, especially in groups with strong social control mechanisms (Van Bavel & Kok, 2009; Van Poppel et al., 2008). Similarly, values and norms related to childcare (intensive weaning; washing hands; diluting cow milk with unboiled, contaminated water, etc.) are socialized from mother to daughter, especially in pre-transitional contexts. Lastly, children also inherit genes and biological weaknesses from their parents. In this paper, we try to get an insight into the mechanisms of intergenerational continuities in infant mortality risks by separating out environmental influences, socioeconomic differences, and norms and attitudes towards fertility behavior.

4. Data and context: intergenerationally traceable CORs

This study is based on information from the Belgian birth and death records (*Akten der Burgerlijke Stand*) and from the Belgian population registers (*Bevolkingsregisters*). The latter are a municipal source in which all inhabitants of the community are registered by household. Along with the address, the members of the household, and their mutual kin relationship, the registers compile information on individual characteristics, such as birth place and date, civil status, occupation, migration to or out of the household, and place and date of decease (Gutmann & Van de Walle, 1978; Leboutte & Obotela, 1988). Although the registers are continuously updated, about every 10 years a new register is started.

Using these sources a sample of life courses was reconstructed and digitized in the so-called Antwerp COR*-database¹ (Matthijs & Moreels, 2010). All individuals with a

certain family name were selected. After ample evaluation family names starting with a combination of C, O, and R (e.g., Cornelissen, Corluy, Coremans) proved to be the most representative in terms of geographical spread and a number of sociodemographic characteristics (Van Baelen, 2007). The sample is drawn from the Antwerp district, an area containing the expanding port city of Antwerp as well as the surrounding rural area, consisting of 61 municipalities. It is restricted in time due to the availability of, and public access to, the source materials. The sample starts in 1846, the year in which all Belgian municipalities were obliged to start maintaining a uniform population register, and ends with the information from the 1910 register (containing information up to 1920), a restriction set because of privacy regulations censoring the past 100 years. The vital registration records were only consulted up to 1906–1910, depending on municipality and year of data collection. Selected individuals, as well as their co-resident kin members, were followed throughout the population registers, at least for as long as they stayed within the geographical and temporal frame. The vital registration supplemented information in the case of unregistered information (such as full dates) or missing individuals (mostly stillbirths and infants who died soon after birth, and consequently were never recorded in the population register, but who are vital for mortality research). The sample yielded about 30,000 life courses, either individuals sharing COR* family names or their direct, co-residing relatives, which allows for easy family reconstitution.

Throughout the second half of the nineteenth century, Belgian infant mortality is characterized by an ‘absence of evolution’ (Masuy-Stroobant, 1983). Infant mortality remained at a steady high level of about 165 deaths per 1000 live births. A decline in infant mortality in Belgium did not start until after 1900 (Chesnais, 2000: 69–78). In the sampled area, the Antwerp district, the infant mortality rate stayed within range of 150–199, but slightly above the Belgian average, mainly due to the city of Antwerp, where infant mortality was higher than average with 200 infant deaths per 1000 live births (Kruithof, 1964; Lesthaeghe, 1977; Masuy-Stroobant, 1983). During the period 1870–1890, when Antwerp was urbanizing at full speed, infant mortality slightly increased to levels of 220–230 infant deaths per 1000 live births (Kruithof, 1964).

For the purpose of this analysis, we selected all individuals born within the Antwerp district in 1846 or in one of the six following decades (1846–1905), and followed them for the first year of their life. This resulted in a total of 6967 infants and stillbirths, born in 1949 different ‘index’ families. Of these, 1080 died before they reached their first birthday, which equals a rate of 155 deaths for every 1000 births.² This rate, which is based on a sample in the whole Antwerp district, is a little lower than the average infant mortality rate for the whole population in the Antwerp city. These deaths occurred in 664 of the 1949 families (34 per cent). Table 1 shows how these deaths are distributed across families according the total number of children born in those families.

Next we identified the mothers of all these children (the mothers in the index families), and tried to reconstruct the families in which these mothers were born and raised as infants. We only selected those cases in which the mothers had at least one sibling, and we assessed whether those siblings survived infancy. A total of 376 out of 1949 mothers remained for analysis (Table 2).

The resulting 376 mothers gave birth to a total of 1826 children, 300 of whom (= 164 per 1000) died within the year. Although every sixth child born in the Antwerp district died during infancy, about 57 per cent of the Antwerp families do not experience a single infant death. Other historical studies have reported similar figures. For example, in nineteenth century Northern Sweden, the infant mortality rate was on average 150, yet 67 per cent of the mothers did not lose any infants (Edvinsson et al., 2005).

Table 1. Distribution of families according the number of births and the number of infant deaths, Antwerp, 1846–1905.

Births in family	Infant deaths in family										Total families	Percentage of families	Percentage of total births	Percentage of total deaths
	0	1	2	3	4	5	6	7	8	9				
1	294	48									342	17.55	3.95	4.09
2	260	43	14								317	16.26	7.31	6.05
3	214	51	15	2							282	14.47	9.76	7.42
4	157	44	16	5	0						222	11.39	10.24	7.76
5	121	37	13	5	1	0					177	9.08	10.21	6.99
6	78	40	23	6	0	2	1				150	7.70	10.38	10.23
7	60	39	14	4	4	2	0	0			123	6.31	9.93	8.95
8	40	30	16	10	1	2	0	0	0		99	5.08	9.14	9.04
9	29	26	13	2	4	4	1	0	0	0	79	4.05	8.20	8.53
10	19	15	7	11	1	3	3	0	1	0	60	3.08	6.92	9.12
11	8	7	8	6	3	4	0	0	0	0	36	1.85	4.57	6.22
12	5	5	7	6	6	0	1	0	1	1	32	1.64	4.43	7.16
13	0	4	3	3	1	1	0	0	1	0	13	0.67	1.95	3.07
14	0	0	1	2	0	2	1	1	0	0	7	0.36	1.13	2.64
15	0	0	2	1	0	1	0	0	0	0	4	0.21	0.69	1.02
16	0	2	0	0	0	0	0	1	0	0	3	0.15	0.55	0.77
17	0	0	0	1	0	0	0	0	0	0	1	0.05	0.20	0.26
18	0	0	1	0	0	0	0	0	0	0	1	0.05	0.21	0.17
19	0	0	0	0	0	0	1	0	0	0	1	0.05	0.22	0.51
Total families	1285	391	153	64	21	21	8	2	3	1	1949	100.00	100.00	100.00
Percentage of families	65.93	20.06	7.85	3.29	1.08	1.08	0.41	0.10	0.15	0.05				

Table 2. Number of births, infant deaths, and families in the analysis.

	All families	Intergenerationally traceable families
Births	6967	1826
Infant deaths	1080	300
Mortality rate	155	164
Total number of families	1949	376
Families with infant deaths	664 (34%)	161 (43%)
Families without infant deaths	1285 (66%)	215 (57%)

The nature of this kind of intergenerational research makes it difficult for the sample to be fully representative for the whole population, especially in the case of intergenerationally traceable families. For example, the requirement of information on two successive generations in a certain region leads to migrant families being underrepresented. This bias can be reduced by sampling over a larger geographical area. For example, our sample covers the larger Antwerp area, which contains the main attraction pool for the city's migrants: the Antwerp countryside (Winter, 2009). Even so, long-distance immigrants, originating from outside the larger Antwerp area, are systematically excluded from the sample. Also the earlier generation must contain a surviving girl, who later becomes the mother of the next generation. Since she always survives infancy (it is a requirement), additionally we need at least one sibling who is 'at risk' to die during infancy. The previous generations must therefore produce at least two children. This means that families are underrepresented if the previous generation was a (very) small family. In our study the effect of small families does not amount to a large bias, since the number of small families in the previous generation is limited. Fertility levels in the whole district remained high until 1880, to gradually decline only afterwards (Lesthaeghe, 1977). Since our study is limited to children born no later than 1905, their previous generation was almost exclusively formed before 1880, in a pre-transitional, high-fertility regime. Nevertheless, the following first section of our study, where the risk status of families in the population is described, is based on all families. In the subsequent sections, when information from the previous generation is introduced, the study is limited to those families we can trace intergenerationally.

5. The distribution of infant deaths on a family level

In order to differentiate between families by the proportion of children lost during infancy, we first need to identify families with a disproportionate number of infant losses. Edvinsson et al. (2005) propose to start from the overall infant mortality rate, and define 'disproportionate' as a family 'that experienced an infant mortality rate twice as high as the average' (p. 328). They call such families 'high risk families'. We find this term misleading, since it does not cover families with an increased *risk* to lose infants, but only those families in which the mortality risk actually resulted in a high number of deaths. In order to avoid a confusing interpretation of the (dis)proportionate spread of infant mortality (see below), we prefer the term 'high-mortality families'. Following the method of Edvinsson et al. (2005), our study departs from the mortality rate among all infants in our sample (0.155), and defines a high-mortality family as a family where a proportion of at least 0.310 of the infants did not survive their first birthday. Conversely, we determine low-mortality families as families that experienced an infant mortality rate of no more than half the average, which in the Antwerp case are families that lost no more than a

Table 3. Operationalization of high, medium and low mortality families for Antwerp, 1846–1905.

Number of births	Number of deaths		
	High mortality family	Medium mortality family	Low mortality family
1–3	≥ 1	-	0
4–6	≥ 2	1	0
7–9	≥ 3	1–2	0
10–12	≥ 4	1–3	0
13–16	≥ 5	2–4	0 or 1
17–19	≥ 6	2–5	0 or 1

proportion of 0.0775 of their children. For most families this means that all children must survive. The remaining families—i.e. those with a proportion of infant losses that is closer to normal—are defined as medium-mortality families. The number of (infant) deaths that must occur for a family to be called a high-, a medium-, or a low-mortality family is defined by the infant mortality rate, and thus differs by place and time. The requirements for the 1949 Antwerp families, observed in the period 1846–1905, are shown in Table 3.

The strength of the above definition is that it is based not only on the population average of infant mortality, but also on the number of births in a family, taking account of a population's distribution by family size, and thus also its underlying fertility pattern. Table 4 shows how the families are distributed by the proportion of infants lost.

However, this dependency on family size is also a weakness: it introduces a disturbing 'operationalization effect'. By 'operationalization effect' we mean that, for example, low-mortality families are less common among large families, only because the definition for 'low mortality' is stricter in large families. This blurs our clear vision on the real impact of family sizes. The operationalization effect is clearly visible in Figure 1, in which the occurrence of three types of families ('high-mortality', 'low-mortality', and the intermediate 'medium-mortality') is shown by number of births in the family. In the case of few births, most families have a low level of mortality. As the number of births in a family grows larger, low-mortality families become decreasingly common, while an ever increasing proportion of the families can be defined as 'medium-mortality' families. The small fluctuations in the proportion of high-mortality families result from the criteria in Table 3, but the general trend seems to be fairly constant over all family sizes.

The proportion of children lost differs not only across family size, but also from family to family given a certain size. For families that are equally large, some lose all their infants, while others lose none. This, however, is far from a sign that infant deaths are unequally (in the meaning of 'not randomly') distributed. In the terminology of Das Gupta (1990): the existence of 'high-mortality' families does not prove that deaths are clustered. The finding that, if a number of families are at equal *risk* of losing infants, some of them will lose infants, while in other families all infants will survive, is not particularly strange.

Table 4. Distribution of families according to proportion of infants lost, Antwerp, 1846–1905.

Sample	Low mortality	Medium mortality	High mortality	Total
All families	1291 (66%)	346 (18%)	312 (16%)	1949
Intergenerationally traceable families	217 (58%)	80 (21%)	79 (21%)	376

Figure 1. Distribution of families according to mortality level, by number of births, Antwerp, 1846–1905 ($N = 1949$).

We tried to avoid this confusion by using ‘high-/medium-/low-mortality’ instead of ‘high-/medium-/low-risk’ terminology. Death clustering is only meaningful when the number of families with a certain number of infant deaths is beyond proportion. In order to discuss the (dis)proportionate spread of infant deaths across families, we need to look at the actual versus the mathematically expected distribution by mortality level. When death clustering is present, the number of families that lose a certain proportion of infants is different from what we would expect when all deaths were distributed randomly across families (i.e., when deaths were not clustered), given the definition. This operation therefore also allows one to keep an ‘operationalization effect’ under control. Based on the overall mortality rate, we used a binomial distribution to assess how many families we expect to lose a disproportionately high (or, respectively, low) number of infants, and this for every family size.³ The resulting expected proportions are shown as lines on top of the bars in Figure 1. In order to facilitate the interpretation, we subtracted the expected proportion of ‘high mortality’ families from ‘1’, so the line appears on top of Figure 1. Since the categorization of families into ‘high’, ‘medium’, and ‘low’ mortality levels depends on the number of births in those families, this line is disrupted, reflecting change in the required number of deaths according to our operationalization of ‘high mortality’ (Table 3).

Figure 1 indicates that, although a fair number of families lose a large proportion of infants, small and average-sized ‘high-mortality’ families are less frequent than we would expect if deaths were distributed randomly. In large families (six to nine births) the family size does not affect whether or not a family has a high level of mortality. Very large families (10 births or more), on the other hand, more often lose a large proportion of their infants. These results suggest a negative effect of crowding, resulting in decreased survival chances for infants born in large families. This effect is especially visible in very large families (10 births or more), but in most family sizes (up to five births) it seems the other

way around: fewer families lose most of their infants. ‘Low-mortality’ families, on the other hand, are between 10 and 20 per cent more frequent than expected, and this for all family sizes, while ‘medium-mortality’ families are always less frequent than expected. When addressing the issue of clustering, the question is thus not (exclusively) why some families lose so many infants, but possibly more pressing, why other families succeed in keeping all their infants alive.

6. Variables used in the analysis

In the next section we test whether the proportion of infants lost can be predicted using information on infant mortality in the previous generation. The following analyses are carried out on the smaller sample, containing only those families that are traceable for at least two successive generations. We included the following variables:

Previous generation. The main variable is whether the previous generation was a high-, medium-, or low-mortality family. We used the same definition as with the index families, but altered it to adjust for the survival of the mother by only counting the mother’s siblings while assessing the number of births in the previous generation.

Age of the mother at birth of first child. This mean-centered variable indicates the start of a mother’s reproductive period.

Number of children ever born. The number of births in a family heavily affects the definition of whether a family has a high, medium, or low mortality level (see Figure 1). At the same time this variable is a measure of the level of fertility.

Social class. The occupational title of the father is recorded at or close to the birth of the first child, either on the birth certificate or in the population registers. It is classified using the HISCO classification scheme (Van Leeuwen, Maas, & Miles, 2002), and recoded into classes using HISCLASS (Van Leeuwen & Maas, 2011). The resulting social classes were further merged into ‘elite or middle class’, ‘worker class’, ‘farmers’, or ‘unknown occupation’.

Region. The sampled area is diverse in population density, level of urbanization, and environmental quality. The ‘city of Antwerp’ itself is densely populated, and its fast urbanization led to poor housing quality and lack of access to clean water, an often mentioned factor in infant mortality (see literature review in Van Poppel & Van der Heijden, 1997). The city government responded by developing the urban infrastructure, including an extensive sewer system (Bertels, 2012). North of the city, close to the river, lays a group of communities known as the Antwerp ‘Polders’. As their name suggests, these villages lie below sea level, creating an ecological environment known for an endemic form of malaria that existed until well into the twentieth century (Devos, 2006). The rest of the district consists of a mix of rural villages and small towns. Empirical evidence from a small town in East Belgium suggests that origin (rural versus small town) is not an important factor in infant mortality (Neven, 2000). The remaining villages and towns are therefore grouped in one ‘countryside’ category.

7. Results

Table 5 provides summary statistics on the independent variables, as well as how the categories are distributed according to the level of infant mortality (‘high’, ‘medium’, or ‘low’) in the index family. The results for the variable ‘previous generation’ are depicted in Figure 2. There is a highly significant correlation between the mortality level of the index family and the mortality level in the previous generation, $\chi^2(4) = 20.8465$;

Table 5. Summary statistics on the variables included in the analysis, by mortality level of the index family.

	Mortality level			
	High	Medium	Low	Total
<i>Mortality level in the previous generation</i>				
High	35 29%	26 21%	60 50%	121
Medium	27 24%	21 19%	65 57%	113
Low	17 12%	33 23%	92 65%	142
<i>Age of the mother</i>	22.48	23.41	24.87	24.05
Mean & standard error	SE = 0.484	SE = 0.507	SE = 0.392	SE = 0.275
<i>Children ever born</i>				
1	5 10%	0 0%	44 90%	49
2	15 23%	0 0%	49 77%	64
3	18 35%	0 0%	33 65%	51
4	4 8%	8 17%	36 75%	48
5	7 28%	7 28%	11 44%	25
6 or more	30 21%	65 47%	44 32%	139
<i>Occupation of the father</i>				
Unknown	28 29%	18 18%	52 53%	98
Elite or middle class	10 17%	8 14%	41 69%	59
Farmer	2 8%	7 29%	15 63%	24
Worker	39 20%	47 24%	109 56%	195
<i>Region</i>				
City	38 21%	35 19%	108 60%	181
Countryside	37 22%	41 24%	93 54%	171
Polders	3 17%	4 22%	11 61%	18
Unknown	1 17%	0 0%	5 83%	6
<i>Total</i>	79 21%	80 21%	217 58%	376

$p < 0.0001$). High mortality families are more common when the previous generation also had a high level of mortality, and less common when the previous generation had a low level of mortality. Conversely, low-level mortality families are more common among women who were born in a low-level mortality family themselves, and less common when they were born in a high-level mortality family.

Figure 2. Distribution of families according to mortality level, by mortality level of the previous generation, Antwerp, 1846–1905 ($N = 376$).

Apparently some of the characteristics affecting the (infant) mortality level of a family persist through generations, making the family in the next generation more likely to lose a similar proportion of infants. This correlation is tested in multivariate analyses, in order to get a better grasp of the intergenerational mechanisms at work. We performed a series of logistic regression analyses on the odds for a family to have a high (respectively low) level of infant mortality, using the logit command from the Stata package (Hosmer & Lemeshow, 2000; Long & Freese, 2006). The results are shown in Table 6 for high-level mortality (models 1, 2, and 3) and Table 7 for low-level mortality (models 4, 5, and 6). For every dependent variable (high or low mortality level) we explored the effect of the family mortality level in the previous generation in three ways: (1) high versus medium and low mortality in models 1 and 4; (2) low versus medium and high mortality in models 2 and 5; and (3) either high or low versus medium mortality in models 3 and 6. The effect of the previous generation was tested both without (a-models) and with controlling variables (b-models).

All models show significant effects for the level of mortality in the previous generations. If the family in the previous generation had a high level of infant mortality, the next generation was almost twice as likely to have a high level of infant mortality as well (model 1a), and at the same time 40 per cent less likely to have a low level of infant mortality (model 4a). Conversely, if the previous generation had a low level of infant mortality, the next generation was almost three times less likely to lose a disproportionately high number of infants (model 2a), and 60 per cent more likely to

Table 6. Logistic regression on the odds that a family has a ‘high level’ of infant mortality.

	Model 1a		Model 1b		Model 2a		Model 2b		Model 3a		Model 3b	
	Odd ratio	p	Odd ratio	p	Odd ratio	p	Odd ratio	p	Odd ratio	p	Odd ratio	p
<i>Mortality level in the previous generation</i>												
High	1.952	0.010	1.973	0.014					1.296	0.384	1.407	0.280
Medium									(ref)		(ref)	
Low			0.935	0.049	0.377	0.001	0.405	0.004	0.433	0.014	0.485	0.044
<i>Age of the mother</i>												
<i>Children ever born</i>												
1			(ref)				(ref)				(ref)	
2			2.879	0.069			3.053	0.055			3.067	0.055
3			4.606	0.009			4.413	0.011			4.554	0.009
4			0.780	0.731			0.784	0.737			0.784	0.737
5			4.944	0.021			4.733	0.026			4.980	0.022
6 or more			2.640	0.078			2.669	0.076			2.751	0.068
<i>Occupation of the father</i>												
Unknown			1.924	0.041			1.873	0.051			1.926	0.043
Elite or middle class			1.162	0.721			1.094	0.831			1.127	0.777
Farmer			0.515	0.409			0.533	0.435			0.547	0.455
Worker			(ref)				(ref)				(ref)	
<i>Region</i>												
City			(ref)				(ref)				(ref)	
Countryside			1.273	0.403			1.247	0.445			1.243	0.454
Polders			0.861	0.827			0.646	0.530			0.698	0.606
Unknown			1.286	0.838			1.456	0.758			1.407	0.782
<i>Constant</i>												
N	0.209		0.051		0.360		0.092		0.314		0.074	
	376		376		376		376		376		376	
Log likelihood (LL)	−190.06		−175.01		−187.32		−173.53		−186.94		−172.95	
p (LL = 0)	0.0108		0.0005		0.0005		0.0002		0.0017		0.0002	
Pseudo R ²	0.0168		0.0946		0.0310		0.1023		0.0329		0.1053	

Table 7. Logistic regression on the odds that a family has a 'low level' of infant mortality.

	Model 4a		Model 4b		Model 5a		Model 5b		Model 6a		Model 6b	
	Odd ratio	p	Odd ratio	p	Odd ratio	p	Odd ratio	p	Odd ratio	p	Odd ratio	p
<i>Mortality level in the previous generation</i>												
High	0.614	0.029										
Medium			0.446	0.003					0.726	0.224	0.522	0.039
Low					1.604	0.031	1.884	0.018	(ref)		(ref)	
<i>Age of the mother</i>												
Children ever born			1.034	0.236			1.029	0.313	1.359	0.237	1.360	0.323
1			(ref)				(ref)				(ref)	
2			0.338	0.064			0.334	0.059			0.329	0.058
3			0.198	0.006			0.211	0.008			0.198	0.006
4			0.309	0.053			0.308	0.051			0.307	0.052
5			0.052	<0.001			0.056	<0.001			0.051	<0.001
6 or more			0.033	<0.001			0.035	<0.001			0.032	<0.001
<i>Occupation of the father</i>												
Unknown			0.340	0.001			0.364	0.001			0.340	0.001
Elite or middle class			1.057	0.884			1.116	0.772			1.063	0.872
Farmer			0.841	0.756			0.857	0.781			0.813	0.713
Worker			(ref)				(ref)				(ref)	
<i>Region</i>												
City			(ref)				(ref)				(ref)	
Countryside			0.920	0.753			0.925	0.766			0.930	0.785
Polders			2.210	0.179			2.634	0.105			2.411	0.142
Unknown			5.627	0.214			4.625	0.254			5.449	0.229
Constant	1.602		21.428		1.147		11.866		1.354		18.121	
N	376		376		376		376		376		376	
Log likelihood (LL)	-253.73		-200.83		-253.77		-202.53		-253.03		-200.34	
p (LL = 0)	0.0284		<0.0001		0.0298		<0.0001		0.0450		<0.0001	
Pseudo R ²	0.0094		0.2159		0.0092		0.2093		0.0121		0.2178	

see most of its children survive infancy (model 5a). After controlling for the other variables, these effects were still significant (b-models), and even grew a little stronger when predicting the odds for a low-mortality family (models 4a vs. 4b, and 5a vs. 5b). The conclusions drawn from Figure 2 are thus affirmed in a multivariate environment. We tried to further explore this in models 3 and 6, by contrasting the effect of a high (respectively low) mortality level in the previous generation against a medium mortality level, but only found significant effects with opposite mortality levels in the two generation, where the contrasts were enlarged (low versus medium mortality in the previous generation, predicting high mortality families in model 3; high versus medium mortality in the previous generation, predicting low mortality families in model 6). If the previous generation lost disproportionately many (respectively few) infants as compared to average, the next generation has a much decreased chance of losing disproportionately few (respectively many).

A mother can partly suppress the inherited risks of losing her infants by postponing her reproduction: every additional year of age decreases the risk to become a high-mortality family by about 6 per cent (models 1b, 2b, 3b). However, we did not find any significant evidence that postponing the birth of the first child will save her from losing any infants (models 4b, 5b, 6b).

There are few (significant) differences between different social classes, or between regions. Only when the occupation of the father was unknown at the birth of the first child it strongly increased the risk of losing a large proportion of infants (models 1b, 2b, 3b), and decreased the chance that all would survive (models 4b, 5b, 6b). We have no clear explanation for this, except that those families are often families with illegitimate children, who have an increased mortality risk during infancy. This well-known effect is probably related to the lack of breastfeeding by unwed mothers, who, in absence of a male breadwinner, needed to take up work sooner in order to provide income (Van Poppel, Kok, & Kruse, 1995).

An in-depth examination of the pseudo- R^2 s, which stand for the variance that is explained by the model, shows that the overall explanatory power is relatively high (e.g., 0.21 in models 5b and 6b). Especially in the case of low-level mortality families our models explain much more variance than among the high-level mortality families. Additional tests for the contribution of every single variable (not shown here) revealed that the main cause of this difference is the variable 'number of births in a family'. We don't believe this supports a potentially very important effect of reproductive behavior for a family's mortality level, given the relatively low explanatory power (in terms of explained variance) of another indicator for fertility, the age at the mother at the start of the reproductive period. Rather, we believe that it is a reflection of the sensitivity of the operationalization of the mortality level to family size, as was already suggested in Figure 1. We therefore do not want to emphasize the effect of 'children ever born' in the family, but nevertheless stress the necessity to control for this variable.

After exploring the odds for high-mortality and for low-mortality families, we performed a third series of regression models (results not shown here, but available on request). Using the Stata `mlogit` command for multinomial logistic regression (Long, 1997; Long & Freese, 2006), we aimed to assess whether families with an average level of infant mortality ('medium') were driven towards an extreme mortality level (either disproportionately low or high), depending on the mortality level of the family in the previous generation. The added value of this modeling strategy is that it allows one to test whether or not the predictors for 'high-mortality' families are mirroring the effects for 'low-mortality' families. The results were in line with the conclusions from models 3b and

6b, showing that an average mortality family is *not* driven towards a high level of infant mortality if the previous generation lost no infants, and *not* towards a low level of infant mortality if the previous generation lost many infants. However, the results from these models were inconclusive, precluding us to detect differences in the mechanisms of the intergenerational continuities between families with high or low infant mortality levels.

8. Conclusion

In this paper we focused on explaining infant mortality on a family level as an alternative to the (more common) practice of studying the individual child. We therefore differentiated between families with a high, medium, or low proportion of deaths among their infants. We found this family approach to be a worthy alternative to the individual child approach, taking account of sibling dependency and explaining more than one fifth of all variation between families.

Our most important finding is that there are intergenerational mechanisms at work, leading infant deaths in a family to be linked with those in the preceding generation. If a mother was born in a 'high-mortality' family, she was more likely to lose a large proportion of her own infants. On the other hand, if she was born in a 'low-mortality' family she was more likely to keep her own infants alive. This intergenerational continuity of a family's infant mortality level is not likely to be caused by transmitted reproductive behavior (crude parity, mother's age at the start of reproduction), a continued social class effect, or lack of intergenerational social mobility (occupation of the father), nor did we find it to result from a persistent regional effect.

The remaining possible causes for the intergenerational correlation found in this study are still diverse, and range from a hereditary biological weakness along the maternal line to a socialization of childcare and feeding norms and practices. Although endogenous and exogenous causes of infant mortality are difficult to separate out in practice, previous studies (e.g., Das Gupta, 1990) reported a strong(er) dependence among postneonatal deaths, suggesting the importance of the latter. Unfortunately, the setup of this study does not allow further exploration of the nature of the transmission.

Furthermore, we did not find traces of regional or social class effects in predicting a family's infant mortality level. However, we did find that mothers more often keep their infants alive if they are older at the birth of their first child. This suggests that mothers can enhance the survival chances of *all* their future infants by postponing the start of the reproductive phase. Since the age of the mother is calculated at the birth of her first child, we did not observe the reversed effect of a high age at giving birth on the survival of children.

Our results are meaningful for the study of (the intergenerational transmission of) reproductive behavior, in that they explore possible causes of fertility outcomes. Intergenerational patterns of short birth intervals may result from (the lack of) intensive breastfeeding, socialized from mother to daughter, but may as well result from the death of the previous birth being frequent in both of generations. If giving birth to a large number of children is transmitted through successive generations, it may well be because the risk of losing many infants is also transmitted through generations. Future studies of intergenerational patterns in reproduction need to be aware that intergenerational continuities in infant mortality risks, as demonstrated in this study, may exist and be of influence.

Notes

1. The Antwerp COR*-database is hosted and maintained by the research group Family and Population Studies from the Centre of Sociological Research at the Catholic University of

Leuven (Parkstraat 45, post box 3601, 3000 Leuven, Belgium). For further information, please contact Email: Koen.Matthijs@soc.kuleuven.be.

2. The ratio which is calculated in this paper is not exactly the same as the more frequently used infant mortality rate (IMR). The IMR leaves out stillbirths in both nominator (deaths) and denominator (births), where we included them. According to our sample, 128 children (= 12 per cent of all infant deaths) are recorded as stillbirths, which is almost certainly an over-registration due to national regulations in vital registration. The Belgian law allows for a delay of maximum three days between the birth and the declaration of the birth in the city hall. Some of the children who died before their birth could be declared, for example during weekends, were declared as stillbirths, and recorded as such in the source material (for a discussion: see Poulain & Tabutin, 1977). Because it is impossible to differentiate real stillbirths from early perinatal deaths in the consulted Belgian sources, we avoided this registration issue by widening the definition of the IMR, including all births (instead of only live births) and all deaths (instead of only non-stillbirths).
3. Under the assumption of no clustering (no dependence between infants), the number of infant deaths K in a family can be seen as a series of independent Bernoulli trials, where every trial stands for one infant with a certain risk of dying, and is repeated for as many times as there are children in the family. K therefore follows a binomial distribution, defined by parameters n (number of trials) and k (number of successes). The proportion of families with exactly k infant deaths out of n births, in a population with an overall infant mortality rate of p (which equals 0.155 in the Antwerp case) is given by the probability mass function f :

$$f(k; n, p) = \Pr(K = k) = \frac{n!}{k!(n-k)!} p^k (1-p)^{n-k}$$

This function, in combination with the minimum required number of infant deaths for each level of mortality by family size (Table 3) allows to calculate the proportion of families we expect to lose a high, a medium, or a low proportion of their infants.

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