KU LEUVEN

FACULTEIT SOCIALE WETENSCHAPPEN

Born to die

Death clustering and the intergenerational transmission of infant mortality, the Antwerp district, 1846-1905

Promotor: Prof. Dr. Koen Matthijs
Co-promotor: Prof. Dr. Jan Kok
Onderzoekseenheid:
Centrum voor Sociologisch Onderzoek [CeSO]

Proefschrift tot het verkrijgen
van de graad van
Doctor in de Sociale Wetenschappen
aangeboden door
Mattijs VANDEZANDE

2012

KU LEUVEN

FACULTEIT SOCIALE WETENSCHAPPEN

Born to die

Death clustering and the intergenerational transmission of infant mortality, the Antwerp district, 1846-1905

Mattijs VANDEZANDE

Proefschrift tot het verkrijgen van de graad van
Doctor in de Sociale Wetenschappen

Nr. 230

Samenstelling van de examencommissie:

Prof. Dr. Jan Van den Bulck (voorzitter)
Prof. Dr. Koen Matthijs (promotor)
Prof. Dr. Jan Kok (co-promotor)
Prof. Dr. Isabelle Devos [Universiteit Gent]
Prof. Dr. Jan Van Bavel
Prof. Dr. Frans Van Poppel [Nederlands Interdisciplinair Demografisch Instituut (NIDI), NL]
Prof. Dr. Bart Van de Putte [Universiteit Gent]

2012

Gepubliceerd door:
Faculteit Sociale Wetenschappen - Onderzoekseenheid: Centrum voor Sociologisch Onderzoek [CeSO], KU Leuven, Parkstraat 45 bus 3601 - 3000 Leuven, België.

D/2012/8978/35

A WORD OF THANKS

It seems like yesterday that I embarked on a journey to complete a PhD, but since then an entire life course has passed. The journey was intense, tough, but rewarding. The hills were steep, but the summits were worth it. The road was never straightforward, but overall it was pleasurable. Today, four years later, the only thing that is left is the satisfactory task of genuinely expressing my thanks to all who have supported, encouraged, or endured me.

First of all, I would like to thank my supervisor, Koen Matthijs. You gave me the freedom to pursue my own research interests, never failed to support my divergent research plans and ambitions, and stimulated me to taste from a wide array of aspects of life in academia.

I also thank my co-supervisor, Jan Kok. You challenged me to always go one little step further, stimulating my thoughts with a never-ending stream of new ideas. I very much enjoyed your stays in Leuven, which were always intense and refreshing. In-between visits you promptly answered every possible question, supplemented with well-timed encouragements, be it from the train, the ferryboat, or any airport in one of the far corners of the world.

I am much indebted to many for their constructive comments, methodological advices and inspiring suggestions. First of all this means the discussants and participants to the sessions of the many conferences, workshops and seminars where I had the opportunity to present my work. I would like to mention some of these scholars more specifically, who generously devoted their precious time and attention to discuss my research and provided it with invaluable comments: George Alter, Anders Brandström, Göran Broström, Glenn Deane, Sören Edvinsson, Theo Engelen, Hill Gates, Kate Lynch, Jim Oeppen, Ken Smith, Gray Swicegood, Frans Van Poppel, Lotta Vikström, Arthur Wolf. Also thanks to Hideko, Jorre and Koen, who constructively guided me through the doctoral seminars, and the members of the PhD guidance and examination committee: Isabelle Devos, Jan Van Bavel, Jan Van den Bulck, Bart Van de Putte and Frans van Poppel, for their thoughtful and inspiring remarks. Thank you to Kristien Hermans for carefully watching over ‘your’ pupils, for your encouragements, and for meticulously mapping out the bureaucratic maze. Most especially, I would like to thank the co-authors with whom I had the honour to collaborate with: George, Jan, Kees, Koen, Maaike, Sarah, Sören, Sylvie, Yunsun. You taught me the meaning of the

‘search’ in ‘research’, and together we enjoyed that unique pleasure and excitement found when treading upon the roads less taken.

My journey took me along many stimulating places, both for shorter (conferences) or longer (research visit) stays. I am grateful to Academic Foundation Leuven (ASL), Flanders’ Research Foundation (FWO), and the SSHA committee of the Tilly Student Travel Awards to provide me with the necessary financial backup.

Research plans are at risk of vanishing into thin air without good data to crystallize them. The construction of the COR*-database, which made this PhD project possible, was realized with the help, hard work – and even selfless sacrifices – of many: Herman Van Isterdael, and his staff at the Leuven Provincial Archives, the staff at the Beveren provincial and Antwerp provincial and municipal archives, the staff at the ‘Dienst Bevolking’ of the municipalities in the Antwerp district, Jos Jonckheer who volunteered to collect and code many Antwerp certificates, the over 60 students who spend their summers transcripting data, and my predecessors at the research team, Hanne Van Baelen and Sarah Moreels.

I thank Anders Brandström from the *Demografiska Data base* at Umeå University for granting me access to the Swedish data and inviting me at his institute. I also thank system developers Jimmy Ljungberg, Lars-Göran Carlsson and Päivi Jänkälä, investigator Carin Hedlund and project leader Erik Törnlund for translating my never-ending demands in a ready-to-use data-retrieval.

A special place is reserved here for the members of the Centre for Population Studies at Umeå University. A national stereotype goes that people from the high North are silent and closed, but you gave me and Sarah a very warm welcome, and always set your doors wide open for us. We instantaneously felt at home. Thank you to Anders for making this stay possible. Thank you Sören and Lotta for your stimulating thoughts, enthusiastic energy, and offering us tastes of Swedish culture (although we liked Jämtlands finest better than surströmming), and making our stay so pleasurable and memorable. Thanks to Henrik, for taking up the stairs to the fourth so many times to discuss the ins and outs of probability distributions and ice hockey divisions. Thanks to Peter and Gabriela for the insights on the Sámi, and Pär for the comments on database design. Thanks to Anna, Annika, Carin, Erik, Glenn, Maria, Mattias, Per-Olof, Roger and many others for sharing your infectious love for Norrland. You started

spring after a long winter in my journey, and I will always look back on the Swedish chapter as one of the best moments in my PhD project.

During summer courses, conferences and similar events where academics gather, I have touched upon many members of next generation's historical demographers. Aggie, Alessandra, Aliaksandr (Sacha), Anna, Antonio, Bárbara, Christa, Christine, Dwight, Elitsa, Emily, Evelien, Ferenc, Georgios (George), Joana Maria, Juan, Luciana, Maaike, Maja, Martin, Nina, Ning, Reto, Richard, Rik, Shuang, Sofie, Sophie, Stefan, Susan, Sylvie, Wiebke, Xingchen, Yulin, Yunsun: over the past four years I have shared my joys and sorrows with you, and some of you became true friends. Special thanks to Reto, for the long conversations and vivid discussions in our shared office on the twists and angles of regression, multi-language federalism, and other less pertinent topics. Thank you for reminding me to stay on track.

Thank you to the colleagues at CeSO for the many lunches, coffee breaks or just quick hello's en route to the printing room, always available to offer a pleasant distraction and helping me to forget about the dead infants for a while. Special thanks to all members at the FaPOS research team, former and current, for the special moments both during and after office hours: An-Katrien, Gray, Graziela, Hans, Jan, Jan, Jeroen, Jozefien, Maira, Nele, Paul, Robyn, Sara, Sarah, Sarah, Sarah, Sofie, and Ward.

To my parents, family and friends: thank you for the never-ending patience and support. While most only saw me standing on the summits of my journey, you were the ones that endured my complaints, and you were always there when I lost track underway. I apologize for the long silences, rudely ignoring your mails and calls, and postponing the meetings we've arranged during the busy last months. A PhD project is a long and all-absorbing undertaking indeed, but I promise I will never do it again.

Last but foremost… Sarah, my fellow traveler. You joined me during all the episodes of my journey, the most beautiful, but also the very ugly ones, taking up all possible roles one could need, both colleague and mentor, partner and closest friend. I cannot describe how much I am indebted to you, and if this project has succeeded, it is because of you on my side. Thank you for being my dearest companion.

TABLE OF CONTENTS

TABLE OF CONTENTS - EXTENDED

LIST OF FIGURES

LIST OF TABLES

PROLOGUE

In the recent literature on young-age mortality, quite some attention has been devoted to the spread of deaths between families. Most infant deaths seem to cluster in a rather limited number of families, an observation which has been named 'death clustering'. This finding has both methodological and theoretical consequences, since it requires to think of mortality from the level of the family instead of from the level of the individual.

Ever since Monica Das Gupta's (1990) call to put death clustering on the research agenda, many studies have aimed to describe and explain clustering, both in contemporary populations, as well as in the European past. Despite the attention it received, the clustering phenomenon has not yet been explained, nor do we know to what extent it played a role. However, there seems to be a consensus on two aspects: (1) death clustering can be found in any population; and (2) the study of death clustering will impact our understanding of the causal mechanisms underlying infant mortality. Some scholars argue that clustering "points to the need to re-evaluate our interpretations of the causes of infant mortality" (Edvinsson et al. 2005:321). This re-evaluation is made possible due to a conceptual shift in focus: while previous research studied mortality averages, clustering is about the variance of mortality from family to family.

It is the general aim of this doctoral dissertation to contribute to the study of clustering by exploring an entirely new element, a family's genealogical history of infant mortality. This study intends to assess if, and explore why, infant deaths are transmitted from one generation to the next. By adding an intergenerational element, the study of clustering is moved away from temporal or contextual effects. Meanwhile, the focus is redirected to the true nature of clustering: so-called family effects, i.e. effects that play on a family level.

This doctoral dissertation is intended as a collection of research chapters. Therefore it is structured in four main parts. The *first part* provides a theoretical background on infant mortality, and on death clustering. From a literature description the main research question is distilled, and hypotheses are constructed. The *second part* sets the stage of this research: it describes the data and contextualizes the study population: the infants and their families living in the Antwerp district ('arrondissement') in the second half of the nineteenth century (1846-1905). The *third part* forms the main body of this dissertation: in four chapters the main research question is studied from different angles, using different research methods. Each chapter stands on its own, starting from a different perspective (individual, family or population), highlighting a different aspect of the theoretical framework and explaining the applied methodology. The results, their meaning, and the technical consequences and efficiency of the used methodologies are discussed in a concluding *fourth part*.

PART ONE

YOUNG-AGE MORTALITY AND THE FAMILY

1.1

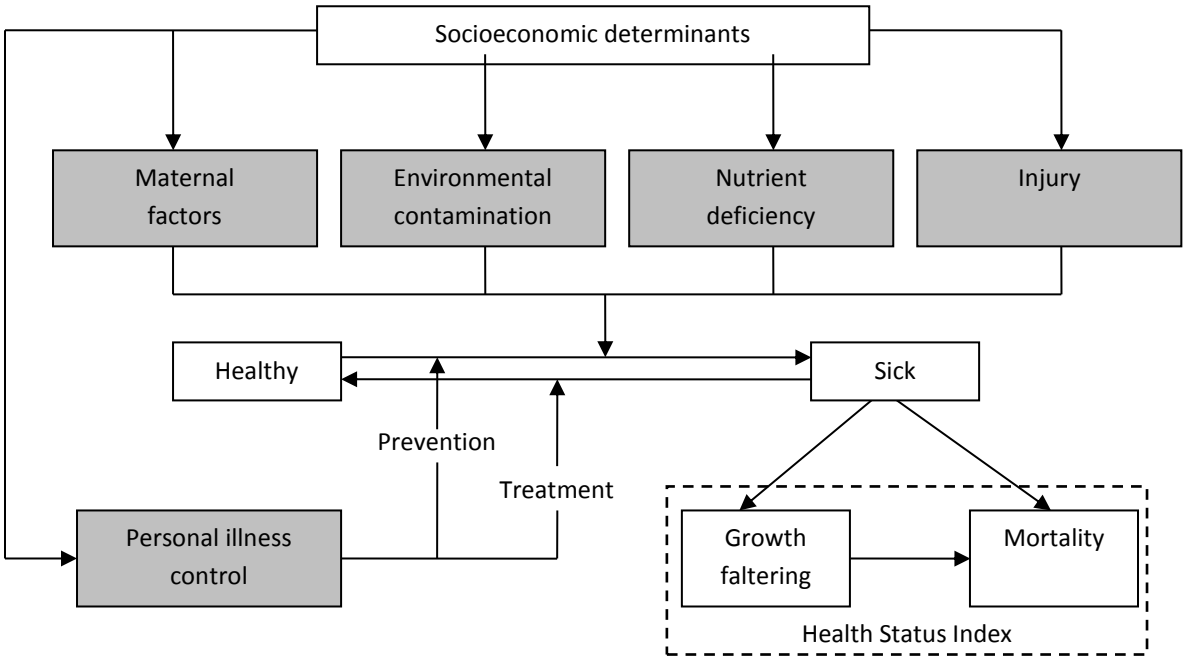
BACKGROUND

STATE OF THE ART

1.1.1. Framing studies on young-age mortality: the Mosley and Chen framework

In 1984 Henry Mosley and Lincoln Chen proposed an analytical framework for studying the determinants of child survival. Their paper (reproduced and commented on in the WHO Public Health Classic Series, Hill 2003) integrates both medical and social science research, resulting in a unified causal model (see figure 1.1.1). Up until then, medical researchers and social scientists concerned with child mortality differed in their approach, their assumptions and their goals. Roughly spoken, while the former focused primarily on treating disease processes with medical interventions, the latter were more interested in socioeconomic gradients in mortality, and in adjusting social policy accordingly. The framework proposed by Mosley and Chen turned out to be very influential. Since its publication, the paper has shaped and directed both medical and social science research on child morbidity, mortality and public health measures (Hill 2003).

Figure 1.1.1. Mosley & Chen's integrative framework (1984) of the proximate determinants on the health dynamics of a population



The causal model was put forward in response to recent renewed interest into the causes of infant deaths in high mortality developing regions. Despite the literature increase, no real common theoretical framework was available, like there is the Davis and Blake model in the related field of fertility (1956). In analogy with Davis and Blake, Mosley and Chen proposed five groups of proximate determinants, or intermediate variables. However, unlike with

fertility, the outcome of the 'child survival' model was not so simple. As the dependent variable, Mosley and Chen developed a "health status index". The index is a measure that combines information on the expected weight of the child, its actual weight, and death, thereby integrating both the concepts of morbidity and mortality. The proximate determinants by definition regulate the relation between health and disease, or the 'health dynamics' of a population. If the balance between the two is flipped too much towards 'disease', it ultimately results in death. The advantage of the model is that mortality studies, both medical and social, can be guided towards factors that influence the proximate determinants, and thus rebalance health.

The groups of proximate determinants affecting child health, as discerned by Mosley and Chen (see figure. 1.1.1), are:

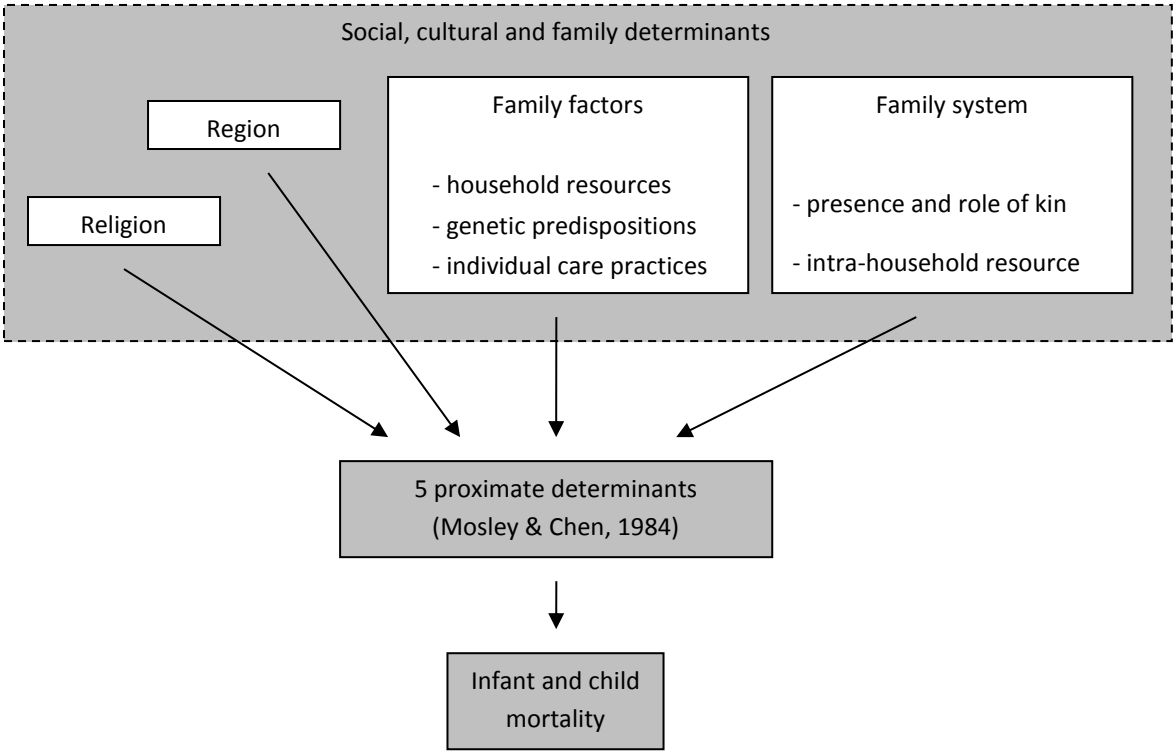
- *maternal factors*: including her age, how many times she gave birth, and the time interval between those births;
- *environmental contamination*: the level of contamination is related to the occurrence and spread of diseases. It can be assessed by microbiological examination, or measured by the incidence of acute infectious diseases. Mosley and Chen therefore suggest using a composed indicator, combining microbiology (presence of micro-organisms) and epidemiology (spread of diseases in the population);
- *nutrient deficiency*: the nutritional diet of children, but also that of their mothers during pregnancy and lactation;
- *injury*: not only referring to instantaneous health shocks, but also to long-term effects of disabilities, related to earlier injury;
- *personal illness control*: both preventive care and medical treatment, undertaken by the individual or offered by care providers.

While all of the factors in these groups influence the process from being healthy to getting sick, the treatment factors in the last group also affect the rate of recovery. The proximate determinants are limited in number, but they are meant to be exhaustive. They are chosen in such a way that the health status of children is dependent on change in one of the factors of the proximate determinants.

More appealing to sociologists are the 'socioeconomic determinants' of child mortality. These determinants include all possible social, economic, cultural and health

system background variables. They operate only through one or more of the five proximate determinants. Quantifying this relationship, or even accurately measuring change in the proximate determinants itself, is not straightforward, if even possible. Although the model of Mosley and Chen is very clear, and claims to be exhaustive, it is not very practical to use. This is especially true for research on mortality in the past, where information is scarce, and the researcher has to work with those elements that survived history.

Figure 1.1.2. Conceptual model for the relation between social, cultural and family determinants and infant and child mortality (adapted from Kok et al. 2011)



A more practical approach is to focus on differentials in the background variables, and how these affect infant and child mortality outcomes. Therefore the researcher breaks the 'socioeconomic determinants' from Mosley & Chen down in a number of measurable variables, sometimes including aspects of the five proximate determinants (see e.g. Lalou 1991). A recent example of this practice for historical research is proposed in Kok, Vandezande & Mandemakers (2011), see figure 1.1.2. It is an example of a very broad interpretation of the background variables, breaking them down into four major (groups of) variables: religion, region, family factors and family system. While religion is a cultural variable, and family system is mainly culturally determined, region is a combination of

cultural determinants and elements of the proximate determinant 'environmental contamination', which makes it an ambiguous yet practical and measurable variable. Family factors is a combination of a socio-economic aspect (the resources a household has at its disposal), a biological component (genetic frailties) and the socio-cultural care practices of each individual parent.

1.1.2. The clustering of infant and child deaths in families

According to Monica Das Gupta, the influential framework of Mosley and Chen determined future literature, in that it, as she formulated it, "crystallizes [an] implicit assumption in the literature" (Das Gupta 1990:489). This assumption deals with the interpretation of differences in mortality levels on the second socioeconomic level, that of the household. Child mortality differences between households are explained by socioeconomic factors on the level of the household (income, wealth) and on the level of the individuals that form the household (the literature often focuses on their education and occupation). The residual variation is often seen as 'nuisance', or 'unmeasurable heterogeneity' (Trussell and Rodriguez 1990). Implicitly it is assumed to be either an unsystematic nuisance variation in a nevertheless perfectly predictable environment, or otherwise a side effect from using inaccurate measures. Das Gupta argues that the residual variation in mortality on a household level is far from unsystematic: it is clearly patterned. For Das Gupta, there is more to it than a mere bad or overly crude measurement.

Das Gupta found that in rural Punjab, India, child deaths are clustered in a limited number of families. This disproportionate spread remains after controlling for Mosley and Chen's (socioeconomic) determinants. She argues that this clustering is caused by an entirely new determinant, not just an inadequate measurement of the old determinants. The unequal distribution of child deaths – or 'death clustering', as Das Gupta called it – might be explained by the "basic abilities and personal characteristics" of the mother (p. 490), independent from her educational level or occupation, or the 'maternal factors' from Mosley and Chen's framework (figure 1.1.1). Das Gupta argues that 'personal abilities' to take care of children differ very much from individual to individual, even after controlling for education. Although education or other socioeconomic determinants can improve an individual's abilities, they do not fully explain why some mothers are 'better' at childcare than others. According to Das Gupta mothers have different 'innate' abilities, something which has been neglected in the literature. Das Gupta therefore argues that a mother's 'personal abilities' should be added as a

new determinant to Mosley and Chen's framework, at the position now occupied solely by 'socioeconomic determinants'.

In her 1990 paper Das Gupta points to a number of previous studies, mainly on mortality in developing countries, in which the authors expressed their concerns about an interfamilial correlation in child mortality, or in which the data seem to suggest the presence of death clustering. In addition, she refers to personal communications with a number of other researchers in the field, who confirmed similar family-level heterogeneities. In every mentioned study group or population, deaths are clustered in a limited number of families. In the field of historical demography some additional studies have pointed in the same direction. As early as 1984, in his (Swedish) dissertation "The careless mothers" on 19th century infant mortality, Anders Brändström mentioned an observation he found remarkable. The area he observed, the parish of Nedertorneå, was characterized by high fertility (the average amount of children ever born was 4,75) and a notoriously high infant mortality (due to the lack of breastfeeding mortality was as high as 400 infant deaths per 1.000 live births). Nonetheless a large proportion of the mothers (nearly 45 per cent) did not experience a single infant loss. Meanwhile, a substantial proportion (19 per cent) of the mothers ("högdödlighetsfamiljer", or "high mortality families") lost a disproportionately large number of children. Nault et al (1990) state that in seventeenth and eighteenth century French-Canada certain mothers had a higher propensity to lose their children than others. Some of these precedents mention the methodological consequences of an intra-familial heterogeneity, or "intraclass correlation" (e.g. Pebley & Stupp 1987). Most studies however stay on the descriptive level.

Das Gupta was the first to put clustering explicitly on the research agenda, predicting that "this finding has major implications for our understanding of the determinants of child survival" (p. 490). A number of contributions followed. Many of these papers demonstrate the existence of clustering in a population different from the one studied by Das Gupta. Since Das Gupta's paper on rural Punjab, India, so far clustering has been found to exist in present-day Bangladesh (Zenger 1993; Saha & van Soest 2011), Brazil (Curtis, Diamond & McDonald 1993; Sastry 1997), Guatemala (Guo 1993), Senegal (Ronsmans 1995), Kenya (Zaba & David 1996; Omariba, Rajulton and Beaujot 2008), several other Indian regions such as West-Bengal (Makepeace & Pal 2008), Uttar Pradesh (Bhalotra & van Soest 2008), and Kerala (Arulampalam & Bhalotra 2006), Ghana (Van Bodegom 2010), and even Denmark (Øyen et al. 2009). Also in populations in the (19th century) past deaths are found to be clustered. So far this has been confirmed for several regions in Northern Sweden (Brändström 1984, Edvinsson et al. 2005, Holmberg & Broström 2012), the Derbyshire region in England (Reid 2001;

2002), the village of Sart in Belgium (Alter, Oris & Broström 2001), the village of Lonneker in the Netherlands (Janssens, Messelink & Need 2010), the city of Venice in Italy (Derosas and Broström 2010), some parishes in Styria, Austria (Hohenwarter 2011), and the island of Tasmania (Kippen, 2011). Silently, a shift in the interpretation of 'clustering' took place: while Das Gupta originally seemed to use the term only for an unexplained *residual* variation of child mortality on a family level, later publications – including her own paper from 1997 – use the term to describe the unequal distribution, or *overall* variation, of child mortality on a family level. Clustering is almost exclusively used in the latter, wider definition. This wider definition, in which clustering is the results of all kinds of factors – socioeconomic situation of families, maternal factors and innate abilities, genetic elements – is therefore preferred throughout the rest of this text.

When reading through the literature on child mortality, one could get the idea that death clustering is omnipresent. In all populations, even the ones that are very homogeneous with regard to common mortality determinants (e.g. the women in Niakhar, Senegal, in Ronsmans 1995), there seems to be a considerable variation in child mortality on a family level. Conversely, I could not find a single publication which shows a population where child mortality is distributed equally across all families.

Despite the efforts made in describing clustering in different contexts, little progress has been made on explaining it. So far, no study was able to fully explain the inter-familial differences, although many studies in death clustering had clear opinions on what possibly caused clustering. Some authors suggest a biological explanation (Guo 1993), others favour the role of individual mothers concerning health care, child care practices, and their personal abilities (Das Gupta 1990; Millward & Bell 2001), yet others point to specific fertility patterns regarding birth spacing (Arulampalam & Bhalotra 2006), or a mixture of these (Ronsmans 1995; Edvinsson et al. 2005). The death clustering literature is characterized by a large diversity in opinions, but at the same time there is little consensus on the causes and mechanisms of clustering.

1.1.3. Why do we care about clustering?

Das Gupta's call to focus on the family came with a strong belief that mortality research would not only benefit from the study of clustering, but that the discovery of clustering will alter our very understanding of mortality and its determinants. Although Das Gupta refers to 'major implications', it is not clear what she means by that. In the long run, this might be problematic: despite the large response to Das Gupta's call there is few reflection on the

scientific relevance of clustering. Few arguments have been made to convince that clustering is a topic worth devoting so much attention to. In this section I try to illuminate why we should nevertheless study clustering, and how it can prove meaningful.

The clustering of deaths on the family level has a methodological consequence. Death clustering implies that children born in the same family have linked death risks. Acknowledging the existence of death clustering means acknowledging the interdependence of mortality risks from children that belong to the same household. Whether this interdependence is direct (e.g. a common cause of death) or indirect (shared background variables) is irrelevant at this – methodological – point: the main issue is that siblings are not independent, contrary to what is often implicitly assumed in mortality research. In a field dominated by studies on either the aggregate (community) level or the individual level, the level of the family has been overlooked. According to Guo (1993), ignoring sibling interdependence has been standard practice. The fact that twins and triplets are often excluded in demographic research is for him an indication that researchers are not totally unaware of this problem, but it is an insufficient countermeasure. Ignoring strong dependence on a certain level, such as the family, has an important statistical consequence. Most regression techniques assume observations to be independent from each other. If this assumption is violated, the standard errors of parameter estimates can be underestimated, resulting in the statistical significance of effects being overestimated. In a field where focus of the interpretation is on relationships that are statistically 'significant', and hence considered 'proven', one wants to make absolutely certain that results are not falsely labelled as 'significant', as can be the case when ignoring dependency between the observations. Possibly even more important, seemingly relevant relationships – such as the one between longer birth intervals and lower infant mortality – can be spurious due to the effect of unobserved family traits. The hypothesized effect of a long birth interval might just as well be an inaccurate measurement of a third, unobserved family-level trait, resulting in both longer birth intervals and in lower infant mortality. When using sibling data, which is often the case in many demographic surveys or historical databases, appropriate modelling techniques should be used to overcome this shortcoming. 'Appropriate' here is to be interpreted as 'taking into account clustering'. According to the methodological argument, familial clustering of deaths is seen as a statistical nuisance, but one that potentially affects our final outcomes. Therefore, we should not ignore the family-level clustering of deaths, but account for it in the models we build.

Aside from methodological fine-tuning, the study of clustering itself can also be meaningful. In the literature there are several examples of where clustering might lead us.

Death clustering has been described as "an expression of heterogeneity in the risk of child mortality between sub-groups of a population", in other words: an expression of the existence of mortality differentials (Ronsmans 1995: 443). In Ronsmans' view, 'death clusters' can occur in many dimensions. A comparison of mortality between periods is a study into clustering in the time dimension; regional differences in mortality refer to a spatial clustering. In Das Gupta's research – and also in this doctoral dissertation – the main focus is on differences between families. The study of familial differences, according to Ronsmans, should be conducted similarly as that into regional or temporal differences. Child death risks differ between families, just like they do between regions or birth years. In the case of families these differences can partly be explained by socio-economic differentials (income, occupation, education, etcetera) or context differentials (region, birth cohort). Still, some important differences remain. An in-depth study of the residual differences on a family level can point in the direction of determinants which are difficult to measure or observe, or that are potentially missing in Mosley and Chen's framework. One can then search for alternative measures for these determinants, and test them specifically. Das Gupta illustrated the latter by suggesting an extension of the Mosley and Chen framework with an additional determinant, which she called 'parental abilities'. She supported this assumption with the collection of additional data, including questions focusing on individual childcare practices related to child health, and on the households' sanitary and environmental conditions. After her interpretation of social class effects was questioned in Guo (1993), she later admitted that there is a socio-economic gradient in importance of parental competence: the majority of the diversity in competence is to be found among the lower social classes (Das Gupta 1997). Guo himself suggested that the genetic make-up of the family was of greater importance than assumed before. Ronsmans (1995) in addition pointed to the different ecological settings in which children from different families grew up, and the different strategies to cope with food shortages. Clustering is more than a statistical nuisance, it can act as a tool to trace and validate both measurable (e.g. social class), formerly unknown (e.g. ecological settings) or unmeasurable (e.g. genetic loadings) determinants.

Clustering has a conceptual message as well. Young-age mortality, and infant mortality in particular, is an important measure of inequality (for example in the Human Development Index). Inequalities at the aggregate level of the country or the region are often explained by inequalities at the level of the individual child. The study of the clustering of young-age mortality in families puts the focus on the family as a main unit of analysis. It

explains the need to study inequality at the level of the family, and to look for explanations not only at the level of the individual, but also on the level of the family.

Because clustering explicitly puts the focus on the family, often impersonated by the mother, it also facilitates related research, most especially studies on the interplay between fertility and mortality. The clustering of child deaths is an important observation for our understanding of fertility. There is a close relationship between the number of children dying at young ages on the one hand, and the total number of children a mother gives birth to, and the interval between these births, on the other hand (Preston 1978). The death of the previous child in particular is an important determinant in the timing of the next birth (see for example Van Bavel 2004). Birth intervals are heavily influenced by breastfeeding. Intensive breastfeeding can postpone pregnancy, and thus the birth of the next child. The death of a weaned child interrupts breastfeeding, and therefore often results in a much shorter birth interval until the next child (Preston 1978; Santow 1987). The higher incentive of giving birth when the previous child has died can also be explained by a 'replacement' mechanism. (Knodel 1982; Kemkes 2006). This hypothesis states that parents decide more quickly to have a next child, in order to replace a deceased one, a theory which can be applied to non-succeeding births as well. Given these mechanisms, and the length of the reproductive period, the only way for a women to have a certain very high number of births is the shortening of the birth intervals due to child loss. High fertility would thus be correlated to high mortality; fertility heterogeneity by death clustering. Fertility and mortality are related in another way as well: very small families can be the result of 'stopping' behaviour (i.e. ending reproduction 'early'). In some cases, women are infertile because of difficult deliveries. Also maternal mortality limits the number of children ever born. Both are positively correlated with the death of the last child. Families with many deaths are the only way to reach very high fertility levels, but they because of difficult deliveries or maternal mortality they are also at risk of 'early stopping'. Some scholars have used clustering to show the existence of a scarring effect (Arulampalam & Bhalotra 2006; Saha & van Soest 2011). A (too) short birth interval resulting from the death of the previous child 'scars' the mother, and its long-term health effect is that the succeeding children are lower in birth weight, and have higher death risks. According to Ronsmans (1995) this feeds the assertion that at least a part of clustering is a necessary side-effect of fertility mechanisms. Ronsmans found this to be true in a simulated environment, but only to a limited extent. Moreover, fertility theory may be explaining the existence of very high mortality families, but it does not explain why some families have a much lower mortality than expected.

While contemporary researchers focus on clustering either as a statistical nuisance, or as a meaningful tool in explaining mortality or understanding fertility, historical demographers have used the same base observation to focus on the nature and the extent of clustering itself. Clustering is not seen as a window, a channel through which research is conducted, but as a dependent variable, a goal towards which research is aimed. Also, most of their attention has gone to infant deaths, or deaths within the first twelve months of life. One characteristic of clustering that has caught their attention in particular is the unexplained dependency between siblings. For example, in a practical adaptation of the Mosley and Chen framework for historical neonatal mortality, Lalou (1991) mentioned 'the fate of brothers and sisters' as a separate 'demographic' variable. A common way to investigate this is digging deeper into the effect of the death of the previous sibling, which illustrates the dependency between siblings that succeed each other in birth order (see e.g. Lynch & Greenhouse 1994; Janssens, Messelink & Need 2010).

A suggestion for a more holistic approach was done in Edvinsson et al. (2005). Following the work of Brändström (1984; 1988), who co-authored the paper, they focused on families with higher-than-normal mortality, Brändströms 'high mortality families' ("högdödlighetsfamiljer") or 'high-risk families', as they called them. Avoiding very complicated analyses, they developed a simple but elegant method to label single families as 'high risk'. According to their definition, in order to be 'high-risk family' the proportion of infant deaths needs to double the population infant mortality rate. For example, in a population with an IMR of 250 (250 infant deaths per 1.000 live births) all families which lose at least half of their infants are considered 'high risk'. Despite the weak theoretical basis for this decision, and the lack of robustness regarding family size in low mortality populations, labelling families as 'high risk' is a very practical and intuitive approach. According to Edvinsson et al. an in-depth study aimed at recognizing high-risk families is a favourable approach to unravel "the fundamental reasons for spatial differences or temporal changes in infant mortality". They were able to unravel a biological (experiencing stillbirths) and a social component (remarried mothers). Following this approach, the preliminary results of Walhout (2011) show that there are little differences between social status and the occurrence of high risk families. Individual analysis on causes of death show that infant deaths in high-risk families are more often due to diarrhoea and contractions than deaths in non-high-risk families, suggesting the importance of behavioural practices (in this case: breastfeeding) for clustering. Not only historical demographers, but also contemporary policy makers in developing regions could benefit greatly from such a perspective. Locating families

with excess young-age mortality can focus and intensify campaigns directed toward lowering child mortality, rendering them more cost-efficient (Zaba & David 1996).

The findings of Edvinsson and his colleagues led them to invite a group of historical demographers in order to get a better grasp of the causes and extent of death clustering ("International workshop on death clustering, 22-23 October 2010, Umeå, Sweden). The goal of the workshop was threefold: (1) to explore to what extent clustering is present in several historical populations, (2) to bring forward and evaluate different ways of measuring clustering, and (3) to offer (additional) explanations for clustering. The focus was thus on clustering as such, and not on the theoretical consequences of clustering. The latter, it was argued, can only be started after we have a better idea of what clustering is. It quickly became clear that (the study of) clustering is far more complicated, and its causes much more diverse than expected. This is partly due to the nature of the question, and the unfamiliarity with the different way of thinking on mortality. Or, as one of the participants phrased it, "death clustering is not about the mean mean, but about the variability of the variance". It was concluded that an interdisciplinary, international comparative perspective is necessary. Although the palette of possible causes remained diverse, there seemed to be a general awareness that part of the answer can be found by a deeper study on the family and its kin.

Clustering has been studied for some time. However, both its extent and its consequences for mortality theory remain vague. There is a general agreement that the family as unit of analysis is more optimal than the individual. Also, there is a shared presumption that our understanding of the determinants of mortality, as represented in the Mosley & Chen framework, will be altered due to this conceptual and methodological shift in focus. Both geographical differences (clustering in space) and the mortality transition (clustering in time) need to be rethought and reanalysed from the family instead of the individual. As such, the study of clustering can provide an alternative approach, or even alternative theory, to the further study of mortality. As for today, however, the study of clustering is still in its infancy: it lacks good, comparable measures, and many of its aspects are not yet unravelled.

1.2

RESEARCH QUESTIONS

HYPOTHESES

1.2.1. Aim of this study

1.2.1.1. Main research questions

We still have no clear idea of what constitutes family-level heterogeneity mortality at young ages. The aim of this study is to make a contribution to a better understanding of the mechanisms behind clustering. One of the main questions in clustering research is:

> "Why is it that in some families more children die than in others?"

The existing literature proves that this is a very broad question. The possible causes for clustering are very diverse and not always measurable. Therefore the above central question will be redirected to Edvinsson's *high-risk families*: can we predict if a two newly wed partners will later lose a large proportion of their infants, given only the genealogical background of bride and groom? If someone is born in such a high mortality family, survives childhood, reaches adulthood and wants to start his or her own family, is that family more likely to have a high infant mortality as well? Does a higher propensity of young-age mortality carry over to the next generation? The focus in this dissertation will thus be narrowed down to the study of one particular aspect: the family's genealogical history of young-age mortality. The central question of this study will be:

> *"Is the propensity of losing a large proportion of infants transmitted through generations?"*

Or, from the perspective of a parent:

> *"Is the propensity of losing infants carried over from parent to child?"*

As such, intergenerational transmission of infant death risks will be put forward as one of the explanations of clustering. Doing so, the individual infant will not only be seen in the context of his or her present family, but also the family's past will be considered. The central focus of this study is thus twofold: (1) on intergenerational patterns in infant mortality, and (2) on the meaning of these patterns for the clustering of infant deaths in families.

Research with such an intergenerational focus, channelled through clustering, will provide answers not only to the clustering phenomenon, but also to whether and how the transmission of death risks between generations took place. Ultimately, researching mortality

in an intergenerational family context might allow us to unveil the mechanisms of some of the behavioural and genetic determinants of mortality at a young age, mechanisms which would remain hidden otherwise.

1.2.1.2. Mechanisms of intergenerational transmission

The transmission of mortality risks from one generation to the other might work in multiple ways. Following the analytic framework of Kok et al. (cfr supra, figure 1.1.2), all proposed social, socioeconomic, cultural or family determinants can be transmitted from one generation to the other. An intergenerational transmission of infant mortality can therefore be understood as the persistence of infant mortality determinants from one generation to the next. For example, if a parent sticks to the same *religious denomination* or religious ideals as the ones he or she grew up with, and his or her partner shares this religion and ideals, this determinant, through socialization within the family, can affect infant survival chances in both generations in a similar way. As such, existing religious differentials persist through generations (for the Netherlands, see Van Poppel, Schellekens and Liefbroer 2002). Regions with a long history of endemic malaria, not uncommon in places with brackish water in 19th century Western Europe (Van Poppel, Jonker & Mandemakers 2005; Devos 2006) affect multiple generations. This *environmental effect* can hide a direct intergenerational transmission of death risks of families that reside in the same area for multiple generations. A similar blurring effect can be caused by household resources, especially in a population with low intergenerational social mobility. If social statuses are often transmitted from father to son, a seemingly intergenerational correlation between mortality risks can effectively be masking a social class differential in infant mortality, persisting from one generation to the other through *status inheritance*.

A direct intergenerational transmission is mainly caused by family factors (cfr supra, figure 1.1.2). These aspects can be social or biological in nature.

A first component which can be directly transmitted across generations is a social one. Families have different norms and attitudes towards traditions in care practices, the role of kin, the resource allocation within the household or gender and family size preferences. In part this is related to group membership, for example as far as these norms and attitudes are related to a certain religious denomination. Nevertheless, norms and attitudes vary from individual to individual, and thus from parent to parent. Moreover, individual behaviour does not always reflect what is dictated by societal or individual norms. For example, care

practices, such as the artificial feeding instead of breastfeeding of infants, are known to create important differences in young-age mortality on the family level, even if these families belong to the same social or denominational group (Walhout 2010). Traditions can include practices that, at first sight, are not directly related to childcare. An example is the tradition of making fires from dried corpses of seabirds on the Icelandic Westman Icelands (Vestmannaeyjar), typically a women's task. Water shortage led women to wash their hands with unclear water from shallow ponds, or not wash them at all, before they took care of their babies. This lead to many cases of neonatal tetanus, which was the main factor responsible for the extremely high infant mortality levels of over 700 per 1.000 live births (Garðarsdóttir & Guttormson 2009). Traditions and knowledge on practices, norms, attitudes, etcetera, can all be passed on from one generation to the next. Children, when learning from their parents, might be continuing the same practices and traditions, whether they are beneficial to or worsening the health condition of infants. The continuation in both attitudes and behaviour from one generation to the next can be interpreted as an intergenerational form of *socialization*. Socialization can also improve infant survival chances from generation to generation. Parents might adapt 'better' childcare practices after the experience of infant loss, and their children might socialize these new practices, especially if they do not lead to recurrent infant loss.

Not all children follow in the footsteps of their parents. As adults, children might adapt deliberately different or even opposed childcare practices as compared to their parents. A logical cause for such *reversed socialization* might be the awareness that certain behaviour (e.g. nursing children with undiluted cow milk) leads to an increase in child loss.

Next to a social component, there can be a biological component as well. Specific genetic loadings can falter the health of children, increasing their risks of dying. These *'faulty' genes*, which have been suggested as a cause for clustering (Janssens, Messelink & Need 2010), can be transmitted from one generation to the other. This genetic makeup should not only be understood as that of the child, but also that of the mother. A mother's risk of miscarriages or difficult deliveries is partly genetically determined, and as such can be transferred from mother to daughter. Counter to this mechanism, the biological component might also run the other way around. According to *biological selection* theory, individuals with weak genes are more prone to die (often very soon after birth), than individuals without any genetic malfunctions. In this case there either is no next generation (which cannot be measured with an intergenerational approach, so the story ends here), or it selects the strongest ones to start the next generation. Infant mortality in the next generation can thus be expected to be lower.

The above mechanisms through which infant mortality risks can be inherited from one generation to the next, are summarized in table 1.2.1. Because no previous research into the intergenerational dimensions of infant mortality is available, these mechanisms are yet referred to as 'hypotheses'. For each hypothesis, it is indicated whether one can expect a positive (+) correlation of infant deaths between generations, or a negative (-) one.

Table 1.2.1. Possible mechanisms of intergenerational infant mortality transmission

Hypothesized mechanism	Nature of correlation
Denomination hypothesis	+
Environmental hypothesis	+
Status inheritance hypothesis	+
Socialization hypothesis	+/-
Reversed socialization hypothesis	-
Faulty genes hypothesis	+
Biological selection hypothesis	-

Of all these mechanisms of intergenerational transmission, social and biological effects, although very distinct in theory, are the most difficult to disentangle in practice. One possible way to go is to look at the age at which young children die. Biological causes of death *tend* to manifest themselves very soon after birth, in the very first days or weeks. Other, external or 'exogenous' causes of death start to become visible later on, after a couple of weeks. This is not an absolute rule. Some genetic causes typically become apparent after a couple of months, such as a genetic propensity for the sudden infant death syndrome. On the other hand, some deaths right after birth can be attributed to social or cultural traditions related to the process of giving birth (Lalou 1991). Despite the ambiguities in interpretation in terms of causal mechanisms, the age at death can give a rough, albeit vague, indication.

1.2.1.3. An additional explanation for clustering?

The second focus of this study is on the explanatory power of a family's genealogical history of infant mortality for the clustering phenomenon. How much clustering is explained by the intergenerational component? The answer of this question is related to the nature of the population which is studied. If the social, cultural or family determinants of young-age mortality are very heterogeneous in a population, there will be a lot of clustering. Clustering is an expression of the variance in a population on the family level. The more homogeneous families in a population are, the less clustering there will be (Ronsmans 1995). Likewise, in a

population with a mix of traditions, where both breastfeeding practices and artificial feeding are both common practice, clustering will be higher than in a population with mainly breastfeeding practices (and reduced mortality risks), or even a population that relies solely on artificial feeding (and hence has increased mortality risks). The *absolute* explanatory power of an intergenerational element for this diversity will be at its strongest in a population with a mixture of families, with large variation in how deaths are transmitted between generations. An example of such a population is a population during modernisation processes, where some families are loyal to the traditions, while others are forerunners of a new, modernized society. Conversely, in homogenous societies, with very little social stratification, where all families have an equal religion and share the same health care practices, etcetera, clustering will be lower, but the remaining differences will be easier to relate to intergenerational effects, so the *relative* explanatory power of an intergenerational element will be larger.

1.2.2. Focus on infancy

Studies on young-age mortality make some very clear distinctions in the first periods of a human life. Generally, a division is made between infants and children, with twelve months of age as an arbitrary, commonly agreed cut-off point. Although the original paper of Das Gupta studied the period from birth up until the fifth birthday, many of the studies on clustering following her paper are limited to infants. This has not only practical reasons: 'infant' in its meaning of the first twelve months of life is a very common measure in surveys and other demographic data sources, and is already present in 19th century registers. There are theoretical arguments as well: infancy is the part of childhood where children are weaned, where they are at their weakest, and where mortality is the highest. During this period of infancy, the mutual importance of the proximate determinants of mortality is different from the rest of childhood (Mosley & Chen 1984). Therefore this study will be focusing on the first year of life. Measuring the variability of mortality across families accurately requires an accurate observation of the number of deaths, including stillbirths. Without stillbirths we would have a biased view on the mortality spread. Stillbirths are also related to (a biological component in) clustering: high-risk families not only have more infant deaths, but more stillbirths as well (Edvinsson et al. 2005). In this dissertation both infant deaths and stillbirths are studied. Any further notions of 'young-age mortality' therefore refer to 'infant mortality', and 'infants' or 'births' implicitly include stillbirths.

1.2.3. Relevance of this research

An intergenerational view is new in the research on mortality at young ages. Aside from an isolated Swedish conference paper (Lindkvist & Broström 2006), this perspective has never been taken, nor in historical studies, nor in contemporary research. In the scope of a single doctoral dissertation it is nigh impossible to unravel all mechanisms and aspects of the intergenerational transmission of death risks with the limited measures available. Therefore this study will be confined to an empirical exploration of intergenerational patterns in various settings. Also, a prototype method to measure the impact of a possible intergenerational transmission of death on clustering will be tested.

Despite this narrow focus, the intergenerational approach nevertheless has several strengths and opportunities:

(+) The innovative character of this new perspective can lead to a refreshing view on a topic that has been on the research agenda for over two decades, but still remains largely unknown;

(+) By comparing mortality risks in two generations the impact of context is largely controlled for. Especially temporal effects, such as epidemics or historical events, are placed in the background. In contrast, the role of kin is illuminated;

(+) Where clustering focuses on inequalities between families at a particular moment, an intergenerational perspective focuses on the transmission of these inequalities over time. By studying how mortality risks are transmitted over time (through generations), this perspective can provide a deeper insight in the persistence of inequalities between families.

However, there are also important weaknesses and shortcomings:

(-) The intergenerational transmission of high infant mortality families holds a paradox. In the ultimate high mortality family, where all children die at young ages, no one survives to form the next generation. The problem lies with the composition of the family in the first generation. Family lines in which all children decease at young ages die out. In these families there is no next generation, and therefore they cannot be studied. In order to avoid this paradox, the study of intergenerational transmission of young-age mortality necessarily focuses on families where at least one child survives.

For this study, a child in the first generation not only needs to survive until adulthood, it needs to have at least one sibling, with a certain risk of dying. Single child families cannot be studied either;

(-) The need for two successive, linked generations places high demands on data quality. Only few databases span a large enough time, with sufficient information on multiple generations. Also, kin related families need to be linked, so family genealogies can be reconstituted. This is not often the case. Also in terms of methodology the main research question can be demanding.

Of these two treats, the latter is the most straightforward to avoid, simply by using apt data and methodologies. This dissertation makes use of a multi-directional approach, resulting in different methodologies allowing to study the same phenomenon from different angles. These methods are explained wherever they are applied: in the respective chapters in the third part of this dissertation. The data that is mainly used in these chapters is introduced and discussed in the second part of this dissertation.

PART TWO

SETTING THE STAGE

2.1

THE ANTWERP COR*-DATABASE

GENESIS AND STRUCTURE

The data that is used in this study stems from the 2010 release of the so-called 'Antwerp COR*-database', or 'COR*-database' (Matthijs & Moreels 2010), with the exception of the Swedish data in chapter 3.4, which is extracted from the Swedish *Demografiska Database* (housed at Umeå University). The Antwerp COR*-database is a data warehouse containing a sample of life courses from the Antwerp district ('arrondissement') in the second half of the nineteenth century (1846-1910). It contains information on births, marriages, deaths, migrations, housing and occupations of over 30.000 individuals, who are followed throughout their lives in their household contexts and network of (co-residing) relatives. In the realization of this study, a substantial effort went to aiding in the construction of this database. This chapter is therefore devoted to the description, the construction, the structure and the characteristics of the COR*-database. The Swedish data is used as comparative material only, and the database from which it is retrieved is introduced and discussed in chapter 3.4.

The information on the COR*-database in this chapter is based on Van Baelen (2007) and Matthijs & Moreels (2010), enriched with information from numerous (unpublished) files and documents created during the database construction phases, and with personal experience based on close involvement in the construction process, from coding pieces of life courses as a job student, to assisting in all major phases of the construction of the COR* data warehouse as a junior researcher collaborating to the project. All tables, figures and schemes are based on the latest release (August 2010).

2.1.1. The Antwerp COR*-database – a brief history

Belgium is known to possess a high-quality registration of its 19th century population. Up to a decade ago this benefit has mainly been derived for the Walloon part of the country, while for Flanders the available sources have long been underused. The existing historical demographic literature on 19th century Flanders focused mainly on small, rural areas (Neven & Devos 2001). In response to this one-sided focus in Flemish demographic history, efforts have been made to collect vast numbers of (individual-level) historical records, and compile them in large and complex historical databases. Doing so, Flemish historians and social scientists are following a recent trend in the field of historical demography, a development towards hoarding up information on individuals, linking bits and pieces of demographic events to individual life courses, connecting life courses of kin, and making those linked life courses (publicly) available in rich, easy-to-access data warehouses (Dillon & Roberts 2002; Kok 2007).

In 2003, a Leuven team of family sociologists from the research group of the Family and Population (now: Family and Population Studies) from the Centre of Sociological Research at KU Leuven took up the initiative to put Flanders on the map of historical demographic databases, enabling a detailed look into the lives of Flanders' inhabitants in the 19th century using population register sources and large collections of vital registration records. The original goal was to draw a sample for the whole of Flanders, a goal which resonates in its former name 'Demographica Flandria Selecta', Latin for 'Demographic Sample of Flanders' (a reference to a Dutch initiative, the Historical Sample of the Netherlands, which was founded in 1987). In 2004, a pilot project started covering the Antwerp city (see Van Baelen 2007 for the early stages of the database process). This municipality was chosen because it contained the largest and fastest growing city in the second half of the nineteenth century. If the data collection strategy and record linkage procedures receive a positive evaluation in this difficult and challenging context, they probably will work in other municipalities as well. Moreover, *urban* historical demography was and still is underrepresented in the field, making the choice for a city, as opposed to (a group of) villages, a logical one. For Flanders, most notably Antwerp was lacking in studies on its demographic past, so the choice of Antwerp responded to a literature gap in Belgian historiography as well. After evaluating and tweaking the procedures of data collection and record linkage, large groups of job students were sent out to the State Archives to collect and digitize historical records. Gradually, the regional scope expanded. At the public release of the database in 2010, the whole of the Antwerp district was covered, consisting of the Antwerp city and a total of 62 surrounding towns and villages.

2.1.2. The source materials

The data in the COR*-database mainly stems from two series of historical source: the population registers, and certificates of birth, marriage and death (Belgium's "vital registration records"). This section will give a brief description of both.

Belgium was among the very first countries in the world to continuously monitor its population on such a detailed level (Poulain 2010). The earliest known example in the western world are the Swedish *husförhörslängd* (Nilsdotter Jeub 1993), household registers administered on a parish level (1665, officialized in 1686)[1]. From 1749 onwards, parish

[1] These parish registers are the main source of the *Demographic Database*, providing the data for some the Swedish regions analyzed in chapter 3.4 of this doctoral dissertation.

figures, aggregated by the local parish priests, were collected and centralized in order to create national figures, the *Tabellverket*, Swedish population censuses (Sköld 2004). A central, national administration compared new aggregates with aggregates of previous years, and local parish priests were requested to provide feedback on inconsistencies (and, if necessary, instructed to travel to Stockholm to comment on them). This system of ecclesiastical or parish registers was temporarily extended to Finland and Estonia, following the Swedish occupation of these countries in the second half of the 18th century. Similar initiatives were found in the Austrian Empire – by parish – by Maria Theresa in 1753, and in the French Republic – by municipality – in 1795 (Poulain 2010). The value of a continuously updated source such as the population register, allowing an accurate overview of the population at any point in time, was recognized in the First International Statistical Congress in 1853. This congress, aimed at bringing uniformity in the national statistics, recommended the installation of a population register in every country, an advice that was taken in several – but, unfortunately, far from all – countries (Gutmann & van de Walle 1978).

The Belgian *population register* is a highly detailed, longitudinal source of individuals in their household context. The population register is preceded by various local initiatives: from the beginning of the 19th century onwards many communities started to experiment with household registers of some kind. These initiatives were based on an arrest of the French occupying government from 1808, which decreed mayors to generate yearly lists of the *de jure* population, recording their arrival and departure from the municipality (Leboutte & Obotela 1988). On 15 October 1846, following the young country's first national population census, the Belgian government ordered the installation of a population register in all Belgian municipalities. Local authorities based themselves on the returns of the census, which they copied in large registers, devoting a double page to each household (Gutmann & van de Walle 1978).

Every household was described as follows (Bracke 2008, also see Piron 1957 for an edition of the regulations in 1901): per household, all officially domiciled inhabitants were listed: first the head of the household, then his wife, children and other relatives, followed by other, non-related members of the household such as servants, farm-hands lodgers, etc. Each inhabitant was described by first and last name, place and date of birth, marital status, occupation, nationality and date of entry in that house(hold). Later, mostly from the 1900s onwards but with examples as early as the 1880s, each individual's (kin) relationship to the head of the household was specified. The registers were continuously updated with new entries (births and immigrations), changes in marital status (marriage, divorce) when

individuals moved out of the household (crossed out in normal ink) or following deaths (crossed out in red ink). Migrations were described more in detail with the place of origin or destination, the dates of departure from the old residence and the date of registration in the new residence. Registration in a new municipality typically took place up to three days after being removed from the register of the old municipality. Migration within a municipality was recorded similarly, with references to the pages where old and new household were written down. A separate column on the right hand page left room for remarks per individual, such as date and place of death, or other notifications such as references to the military register ("militieregister") in case of military service or conscription. This setup allows to literally follow individuals from household to household, linking different pages of multiple population registers to reconstruct residential histories, and individual life courses.

At regular times, by and large every ten years and often following a population census, the existing registers were closed, and new ones were opened. It prevented the registers from becoming too messy (especially registers where updates were plenty, for example in some Antwerp districts with frequent migrations), and established a correct, updated view of the population (anticipating inevitable underregistration of migratory movements, see Gutman & van de Walle 1978).

A second series of sources are the *vital registration records*, namely certificates of births, marriages and deaths (Bruneel & Bruwier 1989; Bracke 2008). For the Belgian municipalities, a (civil, as opposed to parish records) registration of vital events was created by the French occupiers in 1797, and systematized in 1804. The certificates of birth, marriage and death were created by the local officials of the municipality where the event took place. In case of a death outside of the community where one was domiciled (for example if one died abroad), a transcript of the certificate was made and sent to the municipality of residence. Every year, these transcripts were compiled and added as appendices to the register of death certificates of that year.

2.1.3. The COR*-sample

Creating a longitudinal, individual-level database is a meticulous but tedious project. In order to make such a project feasible within certain financial and time constraints, many database limit themselves to a reasonably small, yet sufficiently large number of life courses. In order to cover an area and period as wide as possible, the study population is sampled.

The COR*-database is based on a name sample. This means that individuals are selected based on their family name. The sample focuses on family names starting with the

letter combination C-O-R. The most common examples of such family names are Cornelis, Cornelissen, Corremans, Cornelissens and Corluy (see table 2.1.1), but the whole sample counts over 700 different COR* family names. Over 270 of them relate to more than one individual. The choice for 'C-O-R' was not made arbitrarily. After ample evaluation of combinations of two and three begin letters, this particular three-letter combination was valued the most representative in terms of geographical spread across Flanders and according to a number of socio-demographic characteristics, including migratory status, civil status, sex and structure of the labour market (Van Baelen 2007). This combination of C, O and R as

Table 2.1.1. List of 20 most common COR* family names in the COR* database

Family name	N° of individuals in database
Cornelis	1132
Cornelissen	831
Corremans	699
Cornelissens	618
Corluy	558
Cornet	486
Coremans	306
Corbet	268
Corthout	219
Corten	211
Corijnen	175
Corthals	167
Corrijnen	158
Correwijn	138
Cordon	152
Corewijn	138
Corens	122
Cortebeek	104
Cormo	92
Cordemans	92

starting letters for family names is present in other languages as well. Although the combination's representativeness is studied for Dutch (Flemish) family names only, a wide variety of international immigrants are sampled as well. Common examples, aside from many immigrants from the Netherlands, are France (Cornemuse), Germany (Cornelsen), Prussia (Cornesse), Sweden (Corneliusson), Italy (Corradini), etcetera. The international appeal of the Antwerp harbor is reflected in a small number of more 'exotic' – i.e. non-European – nationalities, including families from, among others, the United States (Coronel, a typical Jewish name, from New York), French Quebec (Corriveau from Beaumont), the Ottoman

Empire (Cori from Smyrna, now Izmir, Cornaro from Constantinople), Brazil (Corvalho from Rio de Janeiro), Argentina (Cormon from Buenos Aires), and the Philippine Islands (Corpuz from Subic). It is probably no coincidence that many of these places are major international port cities. Although not overlooked by the sample strategy, these non-'Flemish' families cannot be claimed to constitute a representative sample from Antwerp's international community.

Family names in Belgium are carried over from a father to his children (with the exception of children from an unmarried mother, who take on the family name of their mother until they are legitimized by marriage of their mother). If only those individuals are sampled who have a family name starting with COR*, only male lineages would be selected, which would create difficulties for certain studies, such as fertility (and related) research. Therefore not only 'CORs' were sampled, but also their co-residing kin members. While entering the data from the population registers, coders were instructed to scan the registers for CORs, and act depending on to the situation they were confronted with.

1. The COR-person is the head of the household, or related to the head of the household. In this case, all kin members of the household were selected and entered in the database. Unfortunately, non-related household members, such as servants and lodgers, were not selected;
2. The COR-person is not related to the head of the household, but is nonetheless domiciled at that address, for example servants, farmhands, lodgers, but also friars and nuns in abbeys and cloisters, or even soldiers at army barracks. In this case, only the COR-person is entered, as well as the head of the household;
3. The nature of the relation between the COR-person and the head of the household is uncertain. In that case, both the COR-person(s), the head of the household, as well as all kin members of the household are selected, as in n° 1.

As for the vital registration, all information on a certificate was entered under the following conditions:

1. Birth certificates: either the mother's or the father's name starts with COR*. Illegitimate children who were later legitimized were entered if either the mother's, or the legitimizing father's name starts with COR*;

2. Marriage certificates: either the bride's or the groom's name starts with COR*;
3. Death certificates: whenever one of the following persons has a family name starting with COR*: the deceased individual, or the parents, or the (deceased or divorced) partners of the deceased individual.

Sampling on the basis of a surname starting with a certain letter combination is not unique for this database, but has several precedents, of which the best known examples are the French TRA-database (Dupâquier & Kessler 1992), and the sample of B's in French-speaking populations such as Geneva (Ornstein & Darroch 1978; Schumacher 2010). This is because a name sampling strategy offers several advantages. Some of them are related to the process of data collection. A name sample requires but a simple selection criterion. Since indices to the (Belgian) written sources are not always reliable, collecting name sampled data becomes a very straightforward process: every physical volume of every vital registration or population register for every municipality in the Antwerp district between 1846 and 1910 was read from cover to cover, scanning for 'CORs'. All bits and pieces of one's life course were retrieved first, and linked together only afterwards. This strategy ensures that individuals who migrate within the district are almost always retrieved, in both the register of origin and the register of destination, even when the migration movement is not recorded and the link between these registers is broken. A name sampling strategy thus maximizes the yield of complete life courses. Moreover, the specific combination of C, O and R only leads to very few spelling ambiguities (a detailed analysis is given in Van Baelen 2007: 8-12), ensuring sampling of the correct individuals and reducing the amount of selected 'false positives' and neglected 'false negatives'. A name sample also has major consequences for the research questions that can be asked. A sample of certain surnames results in an overrepresentation of people sharing the same surname, and consequently an overrepresentation of kin ties, and of complete families. Individuals in the COR* database are more likely to be related to one another than in other databases. This makes a database constructed using a name sampling strategy an ideal starting base for studying family networks and kin relations, allowing both intergenerational and intragenerational approaches. A name sampling setup is thus well fit to answer research questions such as the ones in this dissertation. Name sampling unfortunately also leads to additional complexities for research. This is related to the fact that a name sample is not a random sample: the individuals in the COR*-database are not chosen at random, but selected on the basis of their name. Individuals with the same family name are therefore not

independently selected. Given that this independence is a strict requirement for many statistical techniques, studying the CORs on an individual level requires the use of more complicated, advanced statistical modelling. Fortunately, recent advances in (social science) methodology, for example the introduction of and growing familiarity with multilevel models in demographic research, lower the methodological threshold for correct analyses with this database.

2.1.4. Collecting the data

In the first phase of the database construction, the data needs to be traced in the sources and digitized. Both population registers and vital registration records were consulted. While the population registers provide information one a piece of one's life course, the vital registration records allows to control and correct inaccurate or incomplete data, such as dates, and also allows to trace individuals who were never entered in the population registers, such as stillbirths.

The original population registers were found in the various municipal archival depots of the Antwerp district, ranging from modern, well-equipped archives to remote lockers in a city hall. In many cases the registers were microfilmed by members of the Church of Jesus Christ of Latter Day Saints (Mormon church), who gifted a copy to the State Archives. These copies reside at the Provincial State Archives of Antwerp and Beveren. To facilitate an easy co-ordination of the Antwerp data collection process from Leuven, single-use copies of the microfilms were ordered directly from the Mormon repository in Salt Lake City (Utah). If no microfilms were available, the original sources were searched for in the local municipalities, and digital photographs were taken of all relevant pages in the documents. The population registers are a rarely consulted, well preserved source, and the condition and overall quality of the physical sources was good to excellent in almost all communities of the Antwerp district. One major exception is the town of Merksem, whose collection of population registers was burnt by bombing raids on Antwerp near the end of the Second World War. This gap can partly be covered using the vital registration records, which was saved. As for the vital registration, all certificates of the municipalities in the Antwerp district within the temporal scope of the database are already digitized by members of the Mormon Church. They were consulted on microfilms.

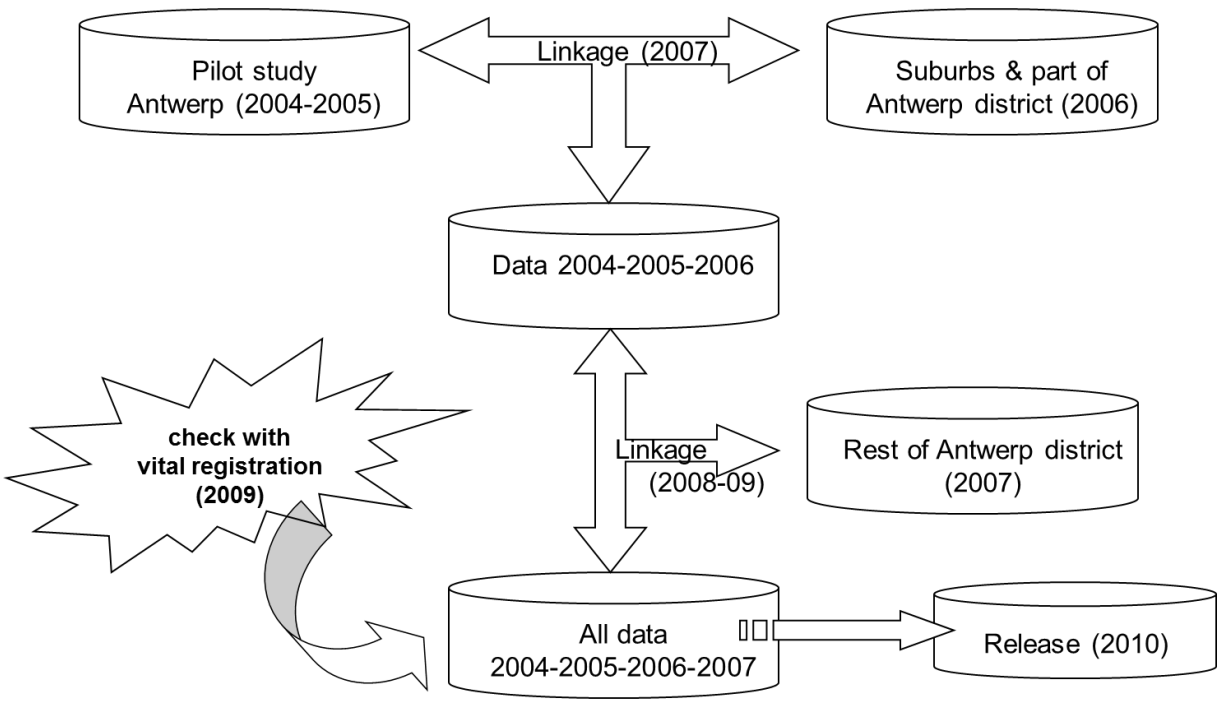
Digitization of the selected data was done using Microsoft Access: handwritten data was entered in Access forms. These forms contained strict rules on the information that could be entered. This streamlined the information inflow, reducing the risk of errors. In total,

56.386 pieces of individuals' life courses were entered from the population registers, supplemented with 6.982 birth certificates, 2.118 marriage certificates and 5.597 death certificates (see Matthijs & Moreels 2010 for more detailed figures).

2.1.5. From data entries to linked life courses: constructing a longitudinal database

After a large number of parts of individual life courses were collected, these pieces needed to be ordered, linked together and supplemented with vital registration data to form reconstructed life courses. This was done in three major steps. In a first step all pieces were matched in pairs, and those pairs that potentially referred to the same life course were retained. Potential pairs were selected based on four main parameters: first name, last name, place and date of birth. These four parameters are available in every population register, and on every vital registration record. In a second step these 'potential' pairs were evaluated on a semi-automated basis. In a third step unique individual identification numbers were assigned, so that each piece that refers to the life course of the same individual received the same identification number.

Figure 2.1.1. Phased record linkage of the Antwerp COR*-database



The nominal record linkage (Desama 1991) was done in several phases, see figure 2.1.1. First the population registers were linked together, in a first phase for the city of Antwerp, in a second for its contemporary suburbs, and in a third for the remaining municipalities in the district. In a fourth and final phase all vital registration data from the Antwerp district was linked to the life courses reconstructed in the first three phases. After every phase, starting from the second, the newly reconstructed life courses were compared to those reconstructed in earlier phases, and supplemented if necessary. With every new phase a new wealth of information became available, allowing to control and, if applicable, correct links that were established or denied before. Step by step life courses were constructed, first within each part of the district, then between the different parts of the district. After a number of beta releases in the summer of 2009 and in February 2010, the end product was released and made available for research in August 2010.

2.1.5.1. Data cleaning

In order to maximize the number of potential pairs, three of the four main variables on which pairs were selected, first and last name an place of birth, were standardized. The reasoning behind this step is that these textual variables are prone to variation in spelling. By standardizing them we aim to eliminate changes in the way names were written, or variations due to reading or typing errors while entering the data. In data warehousing this step is known as 'data cleaning'.

First names in 19th century Flanders are often derived from the Latin names of Catholic saints. After testing different strategies, first names were 'cleaned' by replacing them with their Latin variant. This operation eliminated spelling inconsistencies (Jacob versus Jakob, Joanna versus Johanna), differences between formal and every-day life names (Guilielmus versus Willem, Elisabeth versus Liesbeth) and language variants (Petrus versus Pierre, Julia versus Giulia).

Last names were standardized using a set of 23 simple rules (see also Van Bavel 2002), taking into account common Dutch pronunciation and spelling variations, so 'ph' became 'f', 'y' and 'ey' became 'ij', etcetera.

Places of birth were converted to numerical codes. Belgian municipalities received a 5-digit standard municipality code provided by the National Institute of Statistics. Foreign municipalities were given 8-digit codes, where the first four digits refer to the country, and the last four digits to the municipality within that country.

To facilitate research with the database, all other variables were cleaned as well. Dates were standardized, and conflicting dates corrected. All occupational titles were supplemented with a set of codes referring to the Historical International Standard for the Classification of Occupations (HISCO, see Van Leeuwen, Maas & Miles 2002), allowing for an easy transformation to HISCO-based classification schemes for social classes.

2.1.5.2. Nominal record linkage

After cleaning the main variables for nominal record linkage, potential pairs of life course pieces were selected. All pairs showing large resemblance in first and last name and place and date of birth were selected as potential pairs. Resemblance in dates was translated into a difference of maximum two full years between dates, or one different digit in the year. Resemblance in names was measured using a Dutch adaptation of the Guth algorithm (Guth 1976), which gives a numerical score to the difference between two names based on similarities in the presence of certain letters. Potential pairs were selected on the basis of a set of 13 rules, gradually allowing differences in one or more of the four parameters. Some of these rules specifically focus on legitimized children, who have completely different family names at birth as compared to after their legitimization, while others are afterwards tightened by ignoring the extremely common first name 'Maria' (which is part of the first name in 44 per cent of all sampled women), or when the place of birth was the city of Antwerp (which is the case for 38 per cent of all sampled individuals).

The potential pairs are evaluated using semi-automated record linkage, i.e. a combination of computerized and manual decisions. First, a 'matching score' is calculated for each pair. The score measures 'resemblance' in the four parameters, weighing each parameter (a match between rare family names gets a higher score than a match between a very common family name). The higher the score, the larger the resemblance. Very low scores and very high scores are automatically evaluated as negative matches (do not link) and positive matches (link), respectively. The remaining pairs from the 'grey zone', the pairs with match scores between the cut-off points, are evaluated manually. To facilitate manual evaluation additional information is involved, such as the household context, occupation, place of residence, etcetera. The more records are linked already, the easier decisions in the manual evaluation process can be taken. Afterwards, many control operations are run, specifically aiming at detecting potential mistakes in both the manual and the automated link decisions. For example, one of these operations checks for twins, which score identical matches on three of the four parameters, except first name.

Once all pairs are evaluated, identification numbers are assigned to identify every piece as a part of an individual life course. These identification numbers are the key element in the database, allowing to link all possible information to the right individual.

2.1.5.3. Check with vital registration

The vital registration records were linked to the population register data in a similar way, supplementing the reconstructed life courses with information from the birth, marriage and death certificates. A reliability check on the life courses was done using the registers of birth and death. Where necessary, dates were corrected, faulty links were broken, and new links were established.

The vital registration records pointed to several individuals who were never recorded in the population registers. In most cases they are children who died at, or very soon after birth, and never made it into the population register. However, both for mortality and fertility studies it is crucial that they are sampled. For this reason these children were also assigned unique individual identification numbers, and treated as any other sampled individual in the database.

2.1.6. Structure of the database

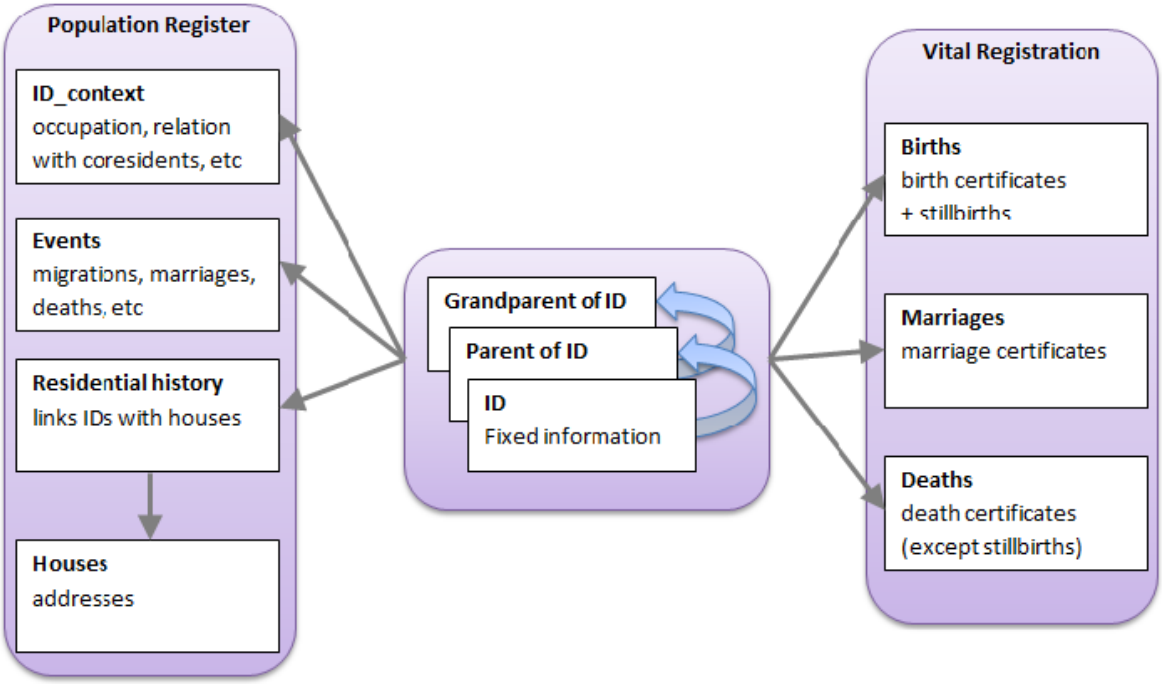
After all sampled life courses were reconstructed and labelled using identification numbers, all information was structured in the database. The final database structure consists of several main tables, reflecting the original source material (figure 2.1.2).

Four tables refer to the population register: three tables for the individuals' context, the houses and the demographic events, and one residential history table linking individuals with the houses. Three tables refer to the vital registration records: one for the birth certificates, one for the marriage certificates, and one for the death certificates. Stillbirths, which are registered in the death certificates, but do not have a birth certificate, were separated from the death certificate table and added to the birth certificate table to facilitate fertility analyses.

Central in the database structure is a table containing 'fixed information'. It gathers the most vital, invariable information of all individuals (hence 'fixed'), and therefore is the starting point for almost all research questions. This table functions as a vital backbone defining the life course, while the other tables supplement it with all kinds of other information. Every individual in the COR*-database has its own line in the 'fixed information' table. Next to the identification number this table holds information on the name of the individual, sex, date and place of birth, date and place of death, whether or not the individual

was born as a twin, whether or not the individual was stillborn. Illegitimate children, whose family name changes when legitimized, have a combination of two family names separated with GEW, a Dutch abbreviation for legitimized (e.g.: PeetersGEWCornelissen, meaning at birth the mother, named Peeters, was unmarried, but later married someone named Cornelissen).

Figure 2.1.2. Structure of the COR*-database (2010 release)



A key element in the table 'fixed information' are variables for both father and mother. Whenever a parent of an individual is sampled as well, the identification number of the parent is entered here, on that individual's line. These variables allow to link someone with his or her parents, who have a line of their own somewhere down the table. The table 'fixed information' is not only the vital backbone of the database, because it holds the vital information of all individuals. It is also the spine for intergenerational research, linking sampled individuals between generations.

The above general structure allows to extract data for a wide variety of research questions. For this doctoral dissertation, three different data sets were retrieved: (1) intergenerationally traceable families along the maternal line, used in chapter 3.1 and 3.4; (2) children with traceable infancy of aunts and uncles on the maternal side (chapter 3.2); and (3) children with traceable infancy of aunts and uncles on the paternal side (chapter 3.3). More detailed information is given in the respective chapters.

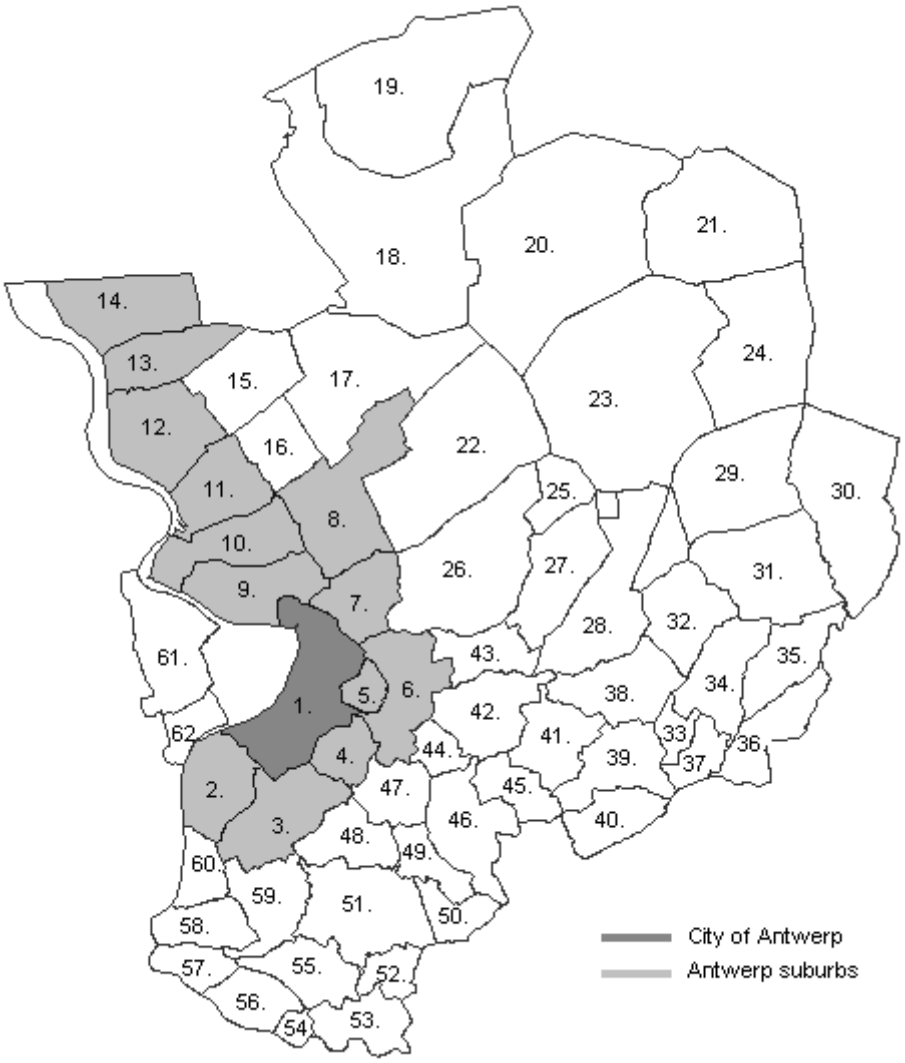
2.2

A BRIEF HISTORICAL DESCRIPTION OF THE 19TH CENTURY ANTWERP DISTRICT

2.2.1. The sample area

The data that is used in this study not only covers the city of Antwerp, but also some suburb municipalities and the surrounding countryside area, together forming the Antwerp district ('arrondissement'). Three districts, the Antwerp one, the district of Turnhout to the east and the district of Mechlin (Mechelen) to the south, together form the province of Antwerp. Wherever referring to 'Antwerp' in this dissertation, the district of Antwerp is meant, unless specified otherwise.

Figure 2.2.1. Municipalities in the Antwerp district around 1900[2]



[2] Numbers 61 and 62, referring to Zwijndrecht and Burcht respectively, were not part of the Antwerp district until 1923. Since the COR*-database samples the area of the present-day district, these communities on the left bank of the Scheldt river are included as well.

Legend of figure 2.2.1 (all municipality names in Dutch)

1. Antwerpen
2. Hoboken**
3. Wilrijk**
4. Berchem**
5. Borgerhout**
6. Deurne**
7. Merksem**
8. Ekeren**
9. Oosterweel**
10. Wilmarsdonk**
11. Oorderen**
12. Lillo**
13. Berendrecht**
14. Zandvliet**
15. Stabroek
16. Hoevenen
17. Kapellen
18. Kalmthout
19. Essen
20. Wuustwezel
21. Loenhout
22. Brasschaat
23. Brecht
24. Sint-Lenaarts
25. Sint-Job-in-'t-Goor
26. Schoten
27. 's-Gravenwezel
28. Schilde
29. Westmalle
30. Oostmalle
31. Zoersel
32. Halle
33. Massenhoven
34. Zandhoven
35. Pulderbos
36. Pulle
37. Viersel
38. Oelegem
39. Broechem
40. Emblem
41. Ranst
42. Wommelgem
43. Wijnegem
44. Borsbeek
45. Vremde
46. Boechout
47. Mortsel
48. Edegem
49. Hove
50. Lint
51. Kontich
52. Waarloos
53. Rumst
54. Terhagen
55. Reet
56. Boom
57. Niel
58. Schelle
59. Aartselaar
60. Hemiksem
61. Zwijndrecht
62. Burcht

** Municipality's area currently belongs to the Antwerp city, and data was therefore collected in the Antwerp city archives during the phase 'Antwerp suburbs (& polder villages)'.

In the 19th century the sampled district consisted of 60 (+ 2) municipalities (see figure 2.2.1), not counting Zwijndrecht and Burcht. Some of these municipalities are founded during the 19th century, for example Sint-Lenaarts (1846), Lint (1869), Hoevenen (re-founded in 1865) or Terhagen (1874). In these cases the communities already existed before, but their population was counted together with that of a neighbouring municipality. Other communities, such as the polder villages (examples are Oosterweel, Wilmarsdonk, Oorderen, Lillo) have been absorbed by the city of Antwerp or its expanding port area. In some cases the local population was even forced to move to make space for the harbour activities, although this did not happen until after the period studied in this dissertation. The whole district area is not larger than 1.000 square kilometres, but with over 500.000 inhabitants in 1900 it is a very densely populated one (Hasquin & Van Uytven & Duvosquel 1980; Vrielinck 2000).

2.2.2. The city of Antwerp – a socio-economic transformation

During the period under investigation Antwerp is a city that underwent dramatic transitions. By the turn of the 18th into the 19th century Antwerp has lost much of its former glory, and is probably best described as a regional textile centre with a moderate port activity. The local textile industry was at danger of collapsing due to warfare, tense international relations and lack of investments. Antwerp's textile activities were no match for competitive, industrializing textile centres such as the nearby Ghent or the English regions. Replacement by the relatively new cotton industry, followed by the production of linen, both failed to revive the local economy and were granted but a short life. Only luxury goods (lace, embroidery) were moderately successful in their struggle for survival (Thijs 1987; Winter 2009).

During the French occupation, Napoleon realized the important (military) strategic location of the city and invested heavily in the expansion and modernisation of the port infrastructure. His investments paid off later on, when the successive Dutch (1815-1830) and Belgian governments (1830 onwards) profited from the modernized infrastructure and stimulated the expansion of Antwerp's commercial activities. A good business climate and competition from neighbouring ports, along with a continuous industrialization in Belgium, the rapid transformation of the Rhine district (Germany) and the invasion of European markets with cheap foreign grain indirectly stimulated the port activities. The expansion of the railway system promoted these market opportunities even more. From the second third of the nineteenth century onwards, the city underwent a rapid economic transformation, and its harbour developed into a dynamic, international port. The concentration of economic activity, together with rising employment in the port and a strong population growth created a favourable environment for the establishment of various industrial activities. In 1896, more than 35 % of industrial employment in the province of Antwerp was located in the port city itself, and more than two thirds was established in the vicinity of the city of Antwerp (Jeuninckx 1964; Veraghtert 1986; Loyen 2003; Loyen, Buyst & Devos 2003; Van Klink 2003; Asaert 2007a; Winter 2009).

The transformation of Antwerp as a port and service centre thoroughly changed the labour market. Irregular employment in the port, and the demand for workers with good physical strength and endurance made that unemployed textile workers often could not (because of an inappropriate stature) or would not (because of pride for its own textile industry) work in the Antwerp port. This turned Antwerp into an attraction pole for a steady

stream of (mainly male) immigrants, a majority of which stemmed from the surrounding countryside area (the Antwerp district). These immigrants easily found their way towards jobs as day labourer, loading and unloading ships. They grouped themselves by common origin, and were organized in various protective 'naties' (labour unions of some kind), further denying local unskilled textile workers' access to the port's economic activities. The typically female work opportunities as cotton spinner or, to a lesser degree, lace maker became increasingly scarce as well. An increasing number of women practiced a job in the informal service sector, often linked to one of the port activities (e.g. fish seller, hawker, etc.), creating opportunities for a new generation of lower middle class (Van Isacker 1966; Lis 1986; Veraghtert 1986; Vanfraechem 2005; Asaert 2007b; Winter 2009).

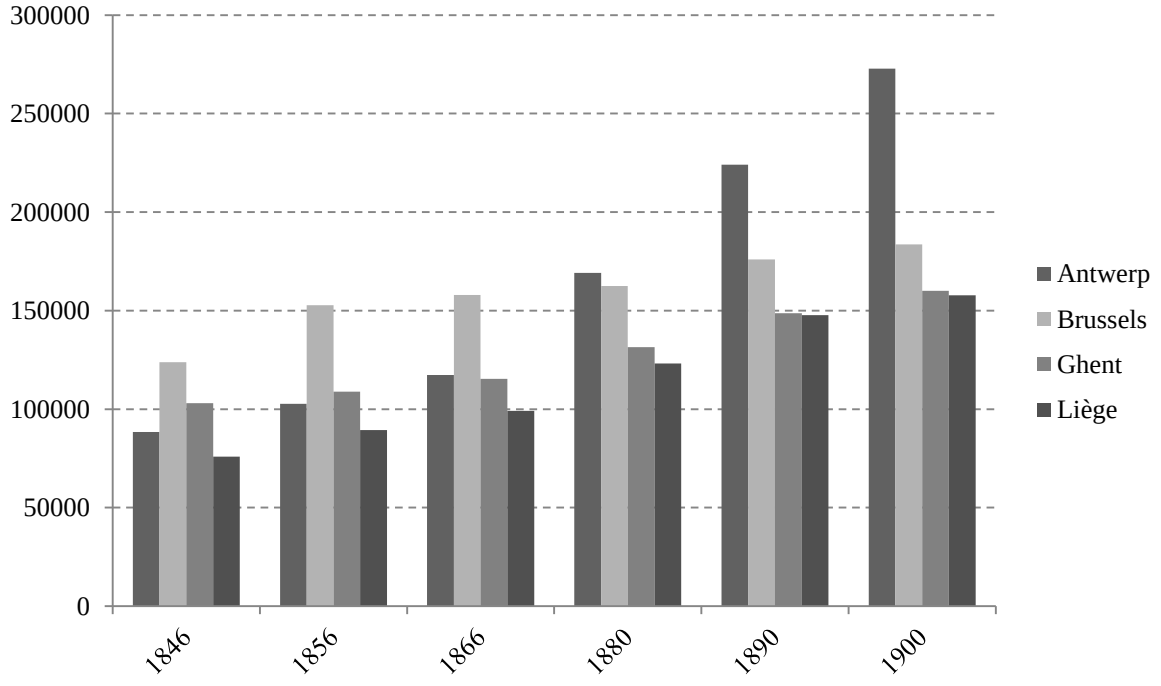
The large and continuous migrant influx resulted in an enormous boost of the population. This explosive population growth had a strong impact on the social life of the Antwerp inhabitants. Fluctuations in rents and the shortage on the local housing market forced the poor into the overcrowded streets and slums and increased the appeals for public charity (Hannes & Lis 1969; Lis 1969; 1986; Vercauteren 2001; Winter 2008; 2009). Thousands of immigrants who were looking for new employment opportunities in Antwerp were often confronted with housing, hygiene and integration problems (Lampo 2002; Asaert 2007b; Van Houtven 2008). During the last decades of the 19th century, these problems were even strengthened by the transit of more than one million European Red Star Line customers, who used Antwerp as a gateway for their one-way ticket to the 'New World' (Veraghtert 1986; Vervoort 2005; Asaert 2007c).

As a result of these economic and social changes, the city of Antwerp faced a chronic lack of space for both the port and urban development during the 19th century. The expansion of the port (with the construction of new docks), the straightening of the Scheldt river banks, and the gradual demolition of the 16th century city walls (1859-1881) offered a solution to the increasing population pressure in Antwerp (De Kesel 1964; Veraghtert 1986; De Caigny 2000; Blomme 2003; Devos 2003; Asaert 2007b). Following this urban expansion, from the mid-19th century onwards efforts in public infrastructure were intensified, leading to the development of transport, water, electricity and gas supplies, and sewers. The city ordered the construction of schools, a second hospital (the Stuyvenberg hospital in 1872-1873), an orphanage (1876), but also police and fire stations (Bertels 2011).

2.2.3. Socio-demographic developments in the Antwerp district

Antwerp's economic transformation from an industrial decline to a commercial revival went hand in hand with a strong demographic expansion. From about 55.000 inhabitants at the start of the 19th century, the Antwerp population steadily increased during the following decades. At the time of the 1846 census, more than 88.000 people were *de facto* (i.e. presence-based) living in the city. During the period 1846-1900, the demographic context changed even more dramatically. According the 1900 census, its *de jure* (i.e. residence-based) population amounted almost 273.000 inhabitants. The annual average growth rate rose from 0,7 per cent in 1801-1810, over 1,6 per cent in 1847-1856, to 2,1 per cent in 1891-1900. Between 1867 and 1890 the annual growth rate even rose beyond 3 per cent (Kruithof 1964). Soon, Antwerp surpassed Brussels and became the biggest, and fastest growing city of Belgium (see figure 2.2.2).

Figure 2.2.2. Evolution of the total population in Belgium's four largest cities, 1846-1900[3]



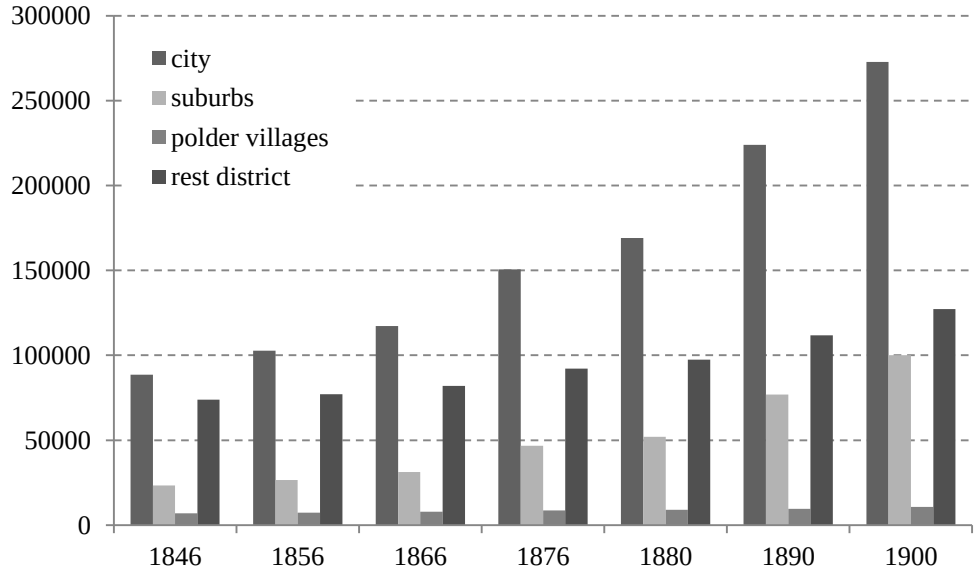
Source: *Recensement général de la population Belgique*, 1846, 1856, 1866, 1880, 1890, 1900; Vrielinck 2000

Antwerp's expansion stimulated the urbanization process in the communities in its immediate surroundings, most notably Berchem, Borgerhout, Deurne, Hoboken and Merksem (see figure

[3] The figures from the 1846 and 1856 census are based on the *de facto* population, and are not fully comparable with the figures from the censuses of 1866 onwards, which represent the *de jure* population.

2.2.1 for their geographical localization). The strongest transformation took place in Borgerhout, which evolved into a flourishing commercial centre, profiting from its location along a vital trade axis with Antwerp's hinterland (the 'Turnhoutsebaan'). Between 1846 and 1900 its population increased sevenfold (5.347 to 37.693 inhabitants). The demolition of the Antwerp city walls expanded the local housing market to the nearby Berchem and Deurne. After 1880 these communities became increasingly attractive for immigrants heading for Antwerp. In only three decades (1880-1910) the population in Deurne more than doubled (5.252 to 12.318) and the population in Berchem more than tripled (9.348 to 30.274). Merksem, due to its favourable location directly east of the most important dock areas, provided housing for Antwerp's dockworkers: its population almost tripled between 1876 and 1900 (4.307 to 11.648). Hoboken's population growth (3.410 in 1876 to 10.202 in 1900) was boosted by an individual act. Unable to meet the demands for increasingly large vessels because of space limitations in the northern dock area, in 1873 John Cockerill decided to move his famous Cockerill Ship Yard company south of the Antwerp city, to Hoboken. In one blow the rural village was led into industrialization, attracting many labour migrants. Also Wilrijk and Ekeren provided housing for Antwerp's port labourers, but only to a lesser extent. The suburbanization process in these municipalities was so distinct that in 1910 visiting British commissioners, studying the 'cost of living' in Antwerp, wrote that "it seems problable that [these suburb villages], to which the street tramways run, will be incorporated with the main city at some near date" (Cost of Living 2010).

Figure 2.2.3. Evolution of the total population in the Antwerp district, 1846-1900



Source: *Recensement général de la population Belgique*, 1846, 1856, 1866, 1880, 1890, 1900

The other municipalities in the Antwerp immediate area – Berendrecht, Oosterweel, Oorderen, Wilmarsdonk, Zandvliet and Lillo – preserved their rural character. These polder villages, flooded plains and marshes on the banks of the Scheldt, housed an endemic form of malaria. They never attracted many immigrants, despite the efforts to balance bad living conditions with increased wages and lower rents. The 19th century economic and demographic transformations in the immediate Antwerp area passed over these villages. In the second quarter of the nineteenth century these villages were absorbed by the expanding harbour infrastructure, and ceased to exist (Dierickx 1954; Hannes & Soetewey-Campers 1969; Hasquin, Van Uytven & Duvosquel 1980; Veraghtert 1986; Vrielinck 2000; Devos 2006).

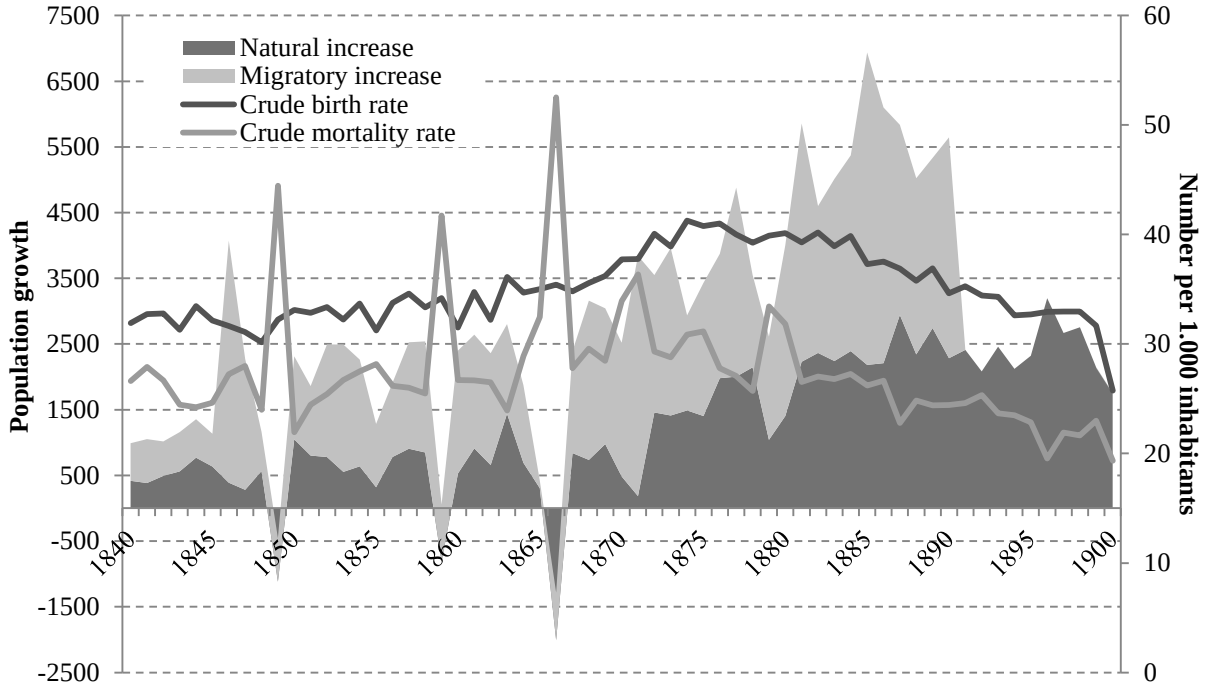
Population growth was less strong in the other villages in the Antwerp district. The majority of them kept their rural character. In some villages, such as Niel or Boom, the industrial activity in the local brickyards boomed as supplier of building materials for Belgium's factories, and these rural centres developed into small towns. In the second half of the nineteenth century, the population of the rural district increased from 84.858 to 143.225 inhabitants. The relative importance of the population in these villages in the totality of the whole district reduced from about 37 per cent in 1846 to less than 25 per cent (Hasquin, Van Uytven & Devosquel 1980; Vrielinck 2000).

2.2.4. Antwerp – a population in transition

A closer look at the migratory and natural increase for the Antwerp city provides detailed information on the mechanisms of its population growth (see figure 2.2.4). The demographic expansion in the city of Antwerp was strongly influenced by immigration. The migration balance was positive and indicates a continuous, mostly male immigration to the city. On average, the net migration rate (immigration minus emigration) increased from roughly 7 to 15 per 1.000 during the 19th century. At the start of the century, about 22 per cent of the migrant stock-population was not born in the city of Antwerp. In the course of the 19th century this proportion amounted 43 per cent. In 1900, half of the foreign migrant population originated from the Netherlands, mostly from the nearby provinces of Zealand, North-Brabant and Limburg, and a fourth were Germans (Kruithof 1964; Lis 1986; Winter 2009; Sanderson & Eggerickx 2010).

The numbers of births and deaths indicate an upward trend in the rate of natural increase. The crude *birth* rates are quite high during the first half of the 19th century (average of 35). After increasing during 1871-1880 (birth rate of 40), the birth rate declined around 1885, and fell back to its former level or 35 in 1890, a decline which is continued towards the

Figure 2.2.4. Population growth in the city of Antwerp, 1840-1900[4]



Source: Antwerp City Reports; vital registration records.

turn of the century (Lesthaeghe 1977). This temporary increase here in birth rates, right before the decline of fertility – in Antwerp increased birth rates in the period 1870-1885 – is a sign of the so-called 'ski jump' fertility (Van de Walle 1974; Dyson and Murphy 1985; a detailed elaboration in the Antwerp context is found in Moreels & Vandezande 2012). The crude *death* rates remained high throughout most of the nineteenth century, fluctuating on average between 27 and 30 between 1800 and 1880, but showing some peaks of extremely high crisis mortality. As visible in the figure, epidemics hit the Antwerp population on a regular basis, most notably in 1849 (cholera), 1859 (cholera and 'marsh fever', very likely an uprise of endemic malaria) and an all-time high in 1866 (severe cholera outbreak). There are some smaller peaks in 1870-71, 1874 and 1880-81 (smallpox). Measles (1862, 1864, 1871, 1879-1880, 1890, 1892, 1893) and to a lesser extent also scarlet fever (1862-1864, 1870) make frequent victims under Antwerp's young children (an overview in Kruithof 1864:524-525). Despite these epidemic outbreaks, the city's crude death rates start to decline around 1875.

Contrary to the decline in overall mortality rates, infant mortality on a national level remained high for another two-and-a-half decades. Unfortunately, there is no easy access to

[4] Figures on immigration and emigration are absent in this figure for the period 1890-1900.

detailed figures of infant mortality for the city of Antwerp, or even the district of Antwerp, for several consecutive periods.

Throughout the 19th century Belgian infant mortality remained somewhat constant on the fairly high level of about 165 infant deaths per 1.000 live births During the 1870's there was even a temporary increase to about 180 infant deaths per 1.000 live births. Belgian infant mortality in the 19th century was absent from any major evolution, and the irreversible decline did not start until after 1900 (Lesthaeghe 1977; Poulain & Tabutin 1977; Hofstee 1983; Lesthaeghe 1983; Masuy-Stroobant 1983; Vallin 1991; Chesnais 2000; Debuisson 2001). In the Antwerp district this increase seems to be even higher, given the level of 197 in 1890, and 186 in 1900 (Lesthaeghe 1977). Contemporaries expressed their concerns on the high mortality among Belgian infants. A systematic study by Jacquart (1907, summarized in Lesthaeghe 1977) related the high figures to a number of factors: (1) the decrease, or lack, of lactation, due to the high labour participation of women, which led mothers to leave their children with neighbours, grandparents or other mothers; (2) the widespread use of poppy heads or opiate syrup to keep crying infants and children quiet; and (3) the pollution of drinking water in urban centres by industrial (e.g. retting of flax) or organic waste, and in the alluvial polders by pollution of the local artificial wells. These explanations may hold in the areas studied by Jacquart, the provinces of Western Flanders and Eastern Flanders, but it is not clear to what extent they are valid explanations for Antwerp.

PART THREE

RESEARCH CHAPTERS

3.1

INHERITED DIMENSIONS OF INFANT MORTALITY

Detecting signs of disproportionate mortality risks in successive generations

Mattijs Vandezande & Prof. dr. Koenraad Matthijs

This paper has been accepted (29th Nov. 2012) in
the History of the Family. An International Quarterly

3.1.0. Abstract

In quest to unravel the diffusion and mechanisms of long-term fertility change, there is a growing body of literature on the intergenerational transmission of reproductive behaviour. 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 stems from the COR*-database, a longitudinal multigenerational life course dataset covering the larger Antwerp area in the second half of the 19th century. We use the family as main unit of analysis, a perspective that proves a worthy alternative to that of the individual child.

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

3.1.1. Introduction

One of the current aims of the study of human reproduction is to unravel mechanisms of an intergenerational transmission of reproductive behaviour (Bittles, Murphy & Reher, 2008; Murphy, 1999). A better understanding of these mechanisms will help 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 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 behaviour. 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 both women to target similar family sizes (Reher et al. 2008). However, the number of surviving children in both generations is also influenced 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.

3.1.2. Infant mortality from a familial perspective

In the recent literature on young age mortality, quite some 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 is known as 'death clustering' (Das Gupta 1990).

There is no consensus on the underlying causes for 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, quite some studies have emphasized the role of a mother in the loss of her infants. Brändström (1988) has suggested that female labour 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 labour participation was reaffirmed by Janssens and Pelzer (2012) for some 19th century Dutch textile towns. She 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 19th 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 amount of their infants, or so-called 'high risk families' (Edvinsson, Brändström, Rogers & Broström 2005).

In this paper 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.

3.1.3. 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, socio-economic 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, etcetera (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 socio-economic 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, Monden & Mandemakers, 2008). Similarly, values and norms related to childcare (intensive weaning, washing hands, diluting cow milk with unboiled, contaminated water, etcetera) 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, socio-economic differences, and norms and attitudes towards fertility behaviour.

3.1.4. 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 ten years a new register is started.

Using these sources a sample of life courses was reconstructed and digitized in the so-called Antwerp COR*-database (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, etcetera) proved to be the most representative in terms of geographical spread and a number of socio-demographic 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 until 1920), a restriction set because of privacy regulations censoring the last 100 years. The vital registration records were only consulted up until 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 1.000 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 1.000 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 1.000 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 6.967 infants and stillbirths, born in 1949 different 'index' families. Of these, 1.080 died before they reached their first birthday, which

.

Table 3.1.1. Distribution of families according the number of births and the number of infant deaths, Antwerp, 1846-1905

	Infant deaths in family													
Births in family	0	1	2	3	4	5	6	7	8	9	Total families	Percentage of families	Percentage of total births	Percentage of total deaths
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	1.285	391	153	64	21	21	8	2	3	1	1.949	100,0	100,0	100,0
Percentage of families	65,93	20,06	7,85	3,29	1,08	1,08	0,41	0,10	0,15	0,05				

equals a rate of 155 deaths for every 1.000 births.[5] 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 1.949 families (34 per cent). Table 3.1.1 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 1.949 mothers remained for analysis (table 3.1.2).

Table 3.1.2. Number of births, infant deaths, and families in the analysis, Antwerp, 1846-1905

	All families	Intergenerationally traceable families
Births	6.967	1.826
Infant deaths	1.080	300
Mortality rate	155	164
Total number of families	1.949	376
Families with infant deaths	664 (34 %)	161 (43 %)
Families without infant deaths	1.285 (66 %)	215 (57 %)

The resulting 376 mothers gave birth to a total of 1.826 children, 300 of which (= 164 per 1.000) died within the year. Although every sixth child born in the Antwerp district died

[5] 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 certain an over-registration due to national regulations in the 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)

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 19th 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).

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.

3.1.5. 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., 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 proportion of 0,078 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 either 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 1.949 Antwerp families, observed in the period 1846-1905, are shown in Table 3.1.3.

Table 3.1.3. Operationalization of high, medium and low mortality families, Antwerp

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

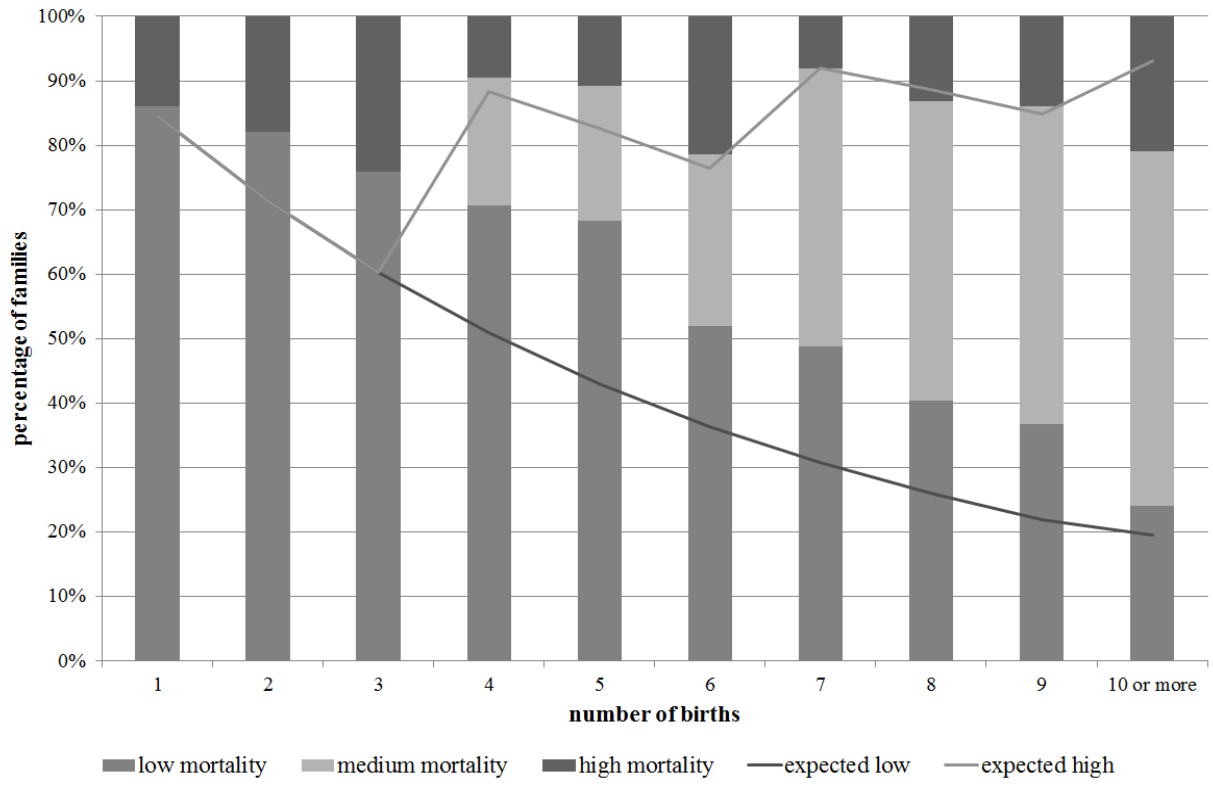
The strength of the above definition is that it is not only based on the population average of infant mortality, but also on the number of births in a family, taking into account a population's distribution by family size, and thus also its underlying fertility pattern. Table 3.1.4 shows how the families are distributed by the proportion of infants lost.

Table 3.1.4. Distribution of families according to proportion of infants lost, Antwerp, 1846-1905

Sample	Low mortality	Medium mortality	High mortality	Total
All families	1.291 (66 %)	346 (18 %)	312 (16 %)	1.949
Intergenerationally traceable families	217 (58 %)	80 (21 %)	79 (21 %)	376

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 more strict in large families. This blurs our clear vision on the real impact of family sizes. The operationalization effect is clearly visible in Figure 3.1.1, 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 3.1.3, but the general trend seems to be fairly constant over all family sizes.

Figure 3.1.1. Distribution of families according to mortality level, by number of births, Antwerp, 1846-1905 (N = 1.949)



The proportion of children lost not only differs across family size, but also from family to family given a certain size. For families which 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. 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 to keep an 'operationalization effect' under control. Based on the overall mortality rate, we used a binomial distribution to assess how much families we expect to lose a disproportionately high (or, respectively, low) number of infants, and this for every family size.[6] The resulting expected proportions are shown as lines on top of the bars in Figure 3.1.1. 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 3.1.1. 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 3.1.3).

[6] 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 3.1.3) allows to calculate the proportion of families we expect to lose either a high, a medium or a low proportion of their infants.

Figure 3.1.1 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 (6 up until 9 births) the family size does not affect whether or not a family has a high level of mortality. Very large families (10 birth 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 until 5 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.

3.1.6. 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.

The following variables were included:

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 3.1.1). 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 2011). 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 which existed until well into the 20th 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.

3.1.7. Results

Table 3.1.5 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 visualized in Figure 3.1.2. There is a highly significant correlation between the mortality level of the index family, and the mortality level in the previous generation ($Chi^2(4) = 20{,}8465$; $p < 0{,}0001$).

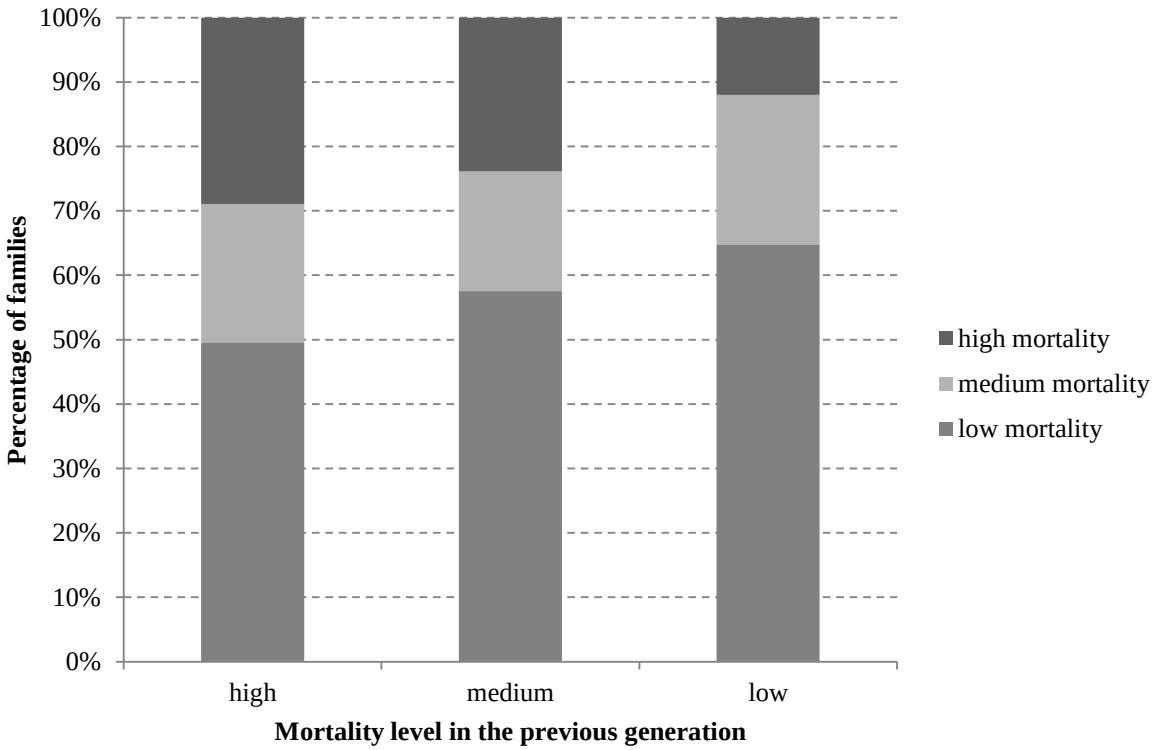
High mortality families are more common when the previous generation also had a high level of mortality, and less common when the previous generation had a low level of mortality. Conversely, low level mortality families are more common among women who were born in a low level mortality family themselves,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

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).

Table 3.1.5. Summary statistics on the variables included in the analysis, by mortality level of the index family, Antwerp, 1846-1905

	Mortality level			
	High	Medium	Low	Total
Mortality level in the previous generation				
high	35 29 %	26 21 %	60 50 %	121
medium	27 24 %	21 19 %	65 57 %	113
low	17 12 %	33 23 %	92 65 %	142
Age of the mother mean & standard error	22,48 se=0,484	23,41 se=0,507	24,87 se=0,392	24,05 se=0,275
Children ever born				
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
Occupation of the father				
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
Region				
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
Total	79 21 %	80 21 %	217 58 %	376

Figure 3.1.2. Distribution of families according to mortality level, by mortality level of the previous generation, Antwerp, 1846-1905 (N = 376)



The results are shown in Tables 3.1.6 for high level mortality (models 1, 2 and 3) and 3.1.7 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 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 3.1.2 are thus affirmed

Table 3.1.6. Logistic regression on the odds that a family has a 'high level' of infant mortality, Antwerp, 1846-1905

	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
mortality level in the previous generation												
high	1,952	0,010	1,973	0,014					1,296	0,384	1,407	0,280
medium									(ref)		(ref)	
low					0,377	0,001	0,405	0,004	0,433	0,014	0,485	0,044
age of the mother			0,935	0,049			0,943	0,095			0,945	0,101
children ever born												
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
occupation of the father												
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)	
region												
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
constant	0,209		0,051		0,360		0,092		0,314		0,074	
N	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 3.1.7. Logistic regression on the odds that a family has a 'low level' of infant mortality, Antwerp, 1846-1905

	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
mortality level in the previous generation												
high	0,614	0,029	0,446	0,003					0,726	0,224	0,522	0,039
medium									(ref)		(ref)	
low					1,604	0,031	1,884	0,018	1,359	0,237	1,360	0,323
age of the mother			1,034	0,236			1,029	0,313			1,029	0,321
children ever born												
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
occupation of the father												
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)	
region												
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	

in a multivariate environment. We tried to further explore on 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 little (significant) differences between different social classes, nor 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, which 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^2s, which stand for the variance that is explained by the model, learns 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 for this difference is the variable 'number of births in a family'. We don't believe this supports a potentially very important effect of reproductive behaviour 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 3.1.1. 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 upon 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 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, disabling us to detect differences in the mechanisms of the intergenerational continuities between families with high or low infant mortality levels.

3.1.8. 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 into account 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 behaviour (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 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 exploring further upon 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 behaviour, 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.

3.2

INTERGENERATIONAL PATTERNS OF INFANT MORTALITY

The Antwerp district, Belgium, 1846-1905*

Mattijs Vandezande, Sarah Moreels & Prof. dr. Koenraad Matthijs

Previous versions of this paper have been presented at:

- European Social Science History Conference, Ghent, 13-16 April 2010
- Workshop on death clustering, Umeå (Sweden), 22-23 October 2010
- 35th Annual Meeting of the Social Science History Conference, Chicago, 18-21 Nov. 2010
  awarded with Charles & Louise Tilly award for Best Graduate Paper

This paper is in preparation for *Population Studies: a Journal of Demography*

*The authors thank the discussants and participants at the European Social Science History Conference, Ghent, 2009, the annual meeting of the Social Science History Association in Chicago, 2010, the participants of the workshop on death clustering (Umeå, October 2010), and four anonymous reviewers for their constructive advice and comments.

3.2.0. Abstract

During the last two decades, scholars found infant mortality to be clustered within families, both for historical (western European) populations and in contemporary less developed regions. Research on this clustering phenomenon mostly focuses on single households. This study looks beyond single households, and adds an intergenerational element to infant mortality clustering, this for a historical context. Using multilevel event history techniques, this study elaborates a quantitative measure for infant mortality clustering. Data is obtained from the Antwerp COR*-database, a kinship based, individual level longitudinal database (the Antwerp district, Belgium, 1846-1905). Our findings are that a considerable amount of infant mortality clustering can be explained by the family history of (late neonatal and postneonatal) infant mortality across generations.

Keywords

historical demography; nineteenth century; Belgium; infant mortality; death clustering; intergenerational transmission

3.2.1. Introduction

In the European past, infant deaths were unevenly distributed among families: most infant deaths occurred in a relatively small number of families (Lynch & Greenhouse 1994; Edvinsson et al. 2005). Additionally a large proportion of families did not experience a single infant death, regardless of the number of children born in that family. This disproportionate mortality spread, named ‘clustering’, has not yet been explained. Nor do we have a clear view on the extent of the phenomenon.

The impact of this death clustering on our research is clear: it serves as a basic assumption to argue that our understanding of infant mortality needs to be reassessed. Also, since most of the determinants for child survival are given shape on the family level, families might be a better unit for analysis than the individual child. Some scholars suggested that our interpretations of the causes of infant mortality may have to be re-evaluated (Edvinsson et al. 2005).

The existence of clustering of infant mortality within families in contemporary societies has been confirmed in several less developed regions, such as Punjab in India (Das Gupta 1990; 1997), Mali (Hill & Aguirre 1990), Guatemala (Guo 1993), Brazil (Curtis et al. 1993), Bangladesh (Zenger 1993), and Kenya (Zaba & David 1996; Omariba et al. 2008). Each of these studies specifically points to the observation that the survival chances of

children in the same family are more alike than those of children born in different families. Various explanatory factors have been put forward, yet no commonly agreed explanation of clustering was found.

An ever returning approach in infant mortality studies is the role of the mother in causing the death of her children. Her education, her income (or more general 'resources'), her socio-economic position, and her competence of childcare practices are all part of the social mechanisms that affect infant survival chances and cross-family mortality variations. Next to these exogenous factors, some also suggest endogenous, bio-medical factors, like genetic frailty or other biological weaknesses, both on the level of the individual child and on a maternal level. In this last case infant mortality is linked to the mother's age, parity of the family (meaning the number of children born), and survival of preceding children. Although they are measured at the family level, these biological determinants lose some power in explaining infant mortality clustering. In many populations clustering tends to be bound to postneonatal infant deaths (above the age of one month), an age at which endogenous factors are generally less important.

The same focus on the mother as a "medium of infant mortality" is also present in death clustering studies on historical populations. One of their primary concerns is the local existence of a breastfeeding-tradition as a major explanatory factor of infant mortality (see for example Millward & Bell 2001). Lack of breastfeeding makes infants more affected by diseases with symptoms such as diarrhoea, causing their death. There is also a link with fertility: mothers who do not breastfeed their children tend to have more children, and shorter birth intervals (Knodel & van de Walle 1967; Bongaarts & Potter 1983). These in turn are believed to induce prematurity and lower birth weight, both proximate causes of infant deaths (Gribble 1993). In addition, for high parity families, this brings up the concept of maternal depletion. Depletion of the mother leads to lower weight at birth of the child, and a lowered overall health of both mother and child (Conde-Agudelo & Belizan 2000; Dewey & Cohen 2007). These studies therefore do not confine themselves to postneonatal mortality, but investigate the survival chances of the infant from birth onwards. Moreover, they devote a major part of their attention to possible biological determinants of infant mortality, typically seen as most important in the perinatal and late neonatal period. Indeed, as opposed to contemporary less developed regions, in the European past not only postneonatal deaths are found to cluster, but also neonatal deaths and even stillbirths are shown to share this unexplained, disproportionate spread between families (Edvinsson et al. 2005).

In both approaches, focusing on either contemporary or historical populations, the role of the mother appears to be a useful concept to study clustering. Yet the precise mechanisms explaining why children of certain mothers have increased death risks are not well understood. Looking beyond the 'mother'-concept, for example by taking her own childhood into account, might give us further insights in the complex mixture of social and biological mechanisms causing infant deaths to cluster.

The aim of this empirical study is twofold: to further develop on the family aspect of clustering in the past, and to elucidate the role of the mother therein. In order to do so with the limited information available in historical sources, we added an intergenerational dimension to the clustering framework. Among the possible explanations for mortality clustering on a family level, biological (e.g. genetic) and social (socialization, social learning) causes seem to be the most obvious ones. In some families 'weaker' genes may lead to an excess in infant mortality. In other families high infant mortality can be explained by specific nursing practices (e.g. breastfeeding), potentially passed on from mother to daughter. If these explanations are to some extent important to the death clustering phenomenon, then the family history might be an important factor in explaining death clustering. This study therefore links deaths among a mother's children to infant deaths in an earlier generation, namely the mother's siblings. A relation in deaths between two generations points to a common cause, transferred across time along the maternal bloodline. To be able to gain knowledge on how the mother transfers mortality risks, special attention is devoted to the ages at which infants die.

3.2.2. Data and context: the COR*-sample and the Antwerp case

For this research, use will be made of the Antwerp COR*-database, release August 2010 (see Matthijs & Moreels 2010). This database contains longitudinal and intergenerational data at the individual level for the whole district of Antwerp, Belgium. The two main sources for the database are the population registers and the vital registration records. In Belgium, the overall quality of both sources is very good, and they are usually well preserved and stored in state or municipal archives. The Belgian population registers (Piron 1957; Gutmann & van de Walle 1978) are a continuously updated source that stores its information on individuals per housing unit. Within each household a "head" is appointed, and for every individual living in that household its (kin) relationship with the head is recorded. This allows for an easy family reconstitution. The vital registration records (Bruneel & Bruwier 1989) refer to birth, death and marriage events. The linkage of individuals in the population registers with those in the

vital registration records provides an extra check on the recording of vital events in the population register, and points to infants who died before they could be registered in the population register, e.g. stillbirths. The database sets off in 1846, when all Belgian municipalities started to maintain the obligatory population register, and goes as far as 1920 (a mark set because of Belgian privacy regulations).

The Antwerp COR*-database is a sample (N ≈ 30.000) of individuals with a surname beginning with the letters C, O, and R, as well as their co-residing relatives, living or experiencing vital events within the Antwerp district during the period 1846-1920. Extensive evaluation showed that this sample strategy yielded an adequate representation of overall Belgian population, both in geographical spread as for a number of socio-demographic characteristics (Van Baelen 2007). Opting for a letter sample has several advantages, one of them being the oversampling of kin relationships (see for example the TRA*-database in France, Dupâquier & Kessler 1992). The Antwerp database went through several processes of internal data linkage to connect both several pieces of one's life course, and the many kinship ties that were present amongst the individuals selected in the sample. As a result, the Antwerp COR*-database contains 737 different COR-surnames, which stand for 2.610 two-generation families (parents and 9.961 children), of which 1.119 three-generation families (grandparents, referring to 4.691 grandchildren). All this characteristics make the Antwerp COR*-database an almost unique tool, fit for the research questions under examination here.

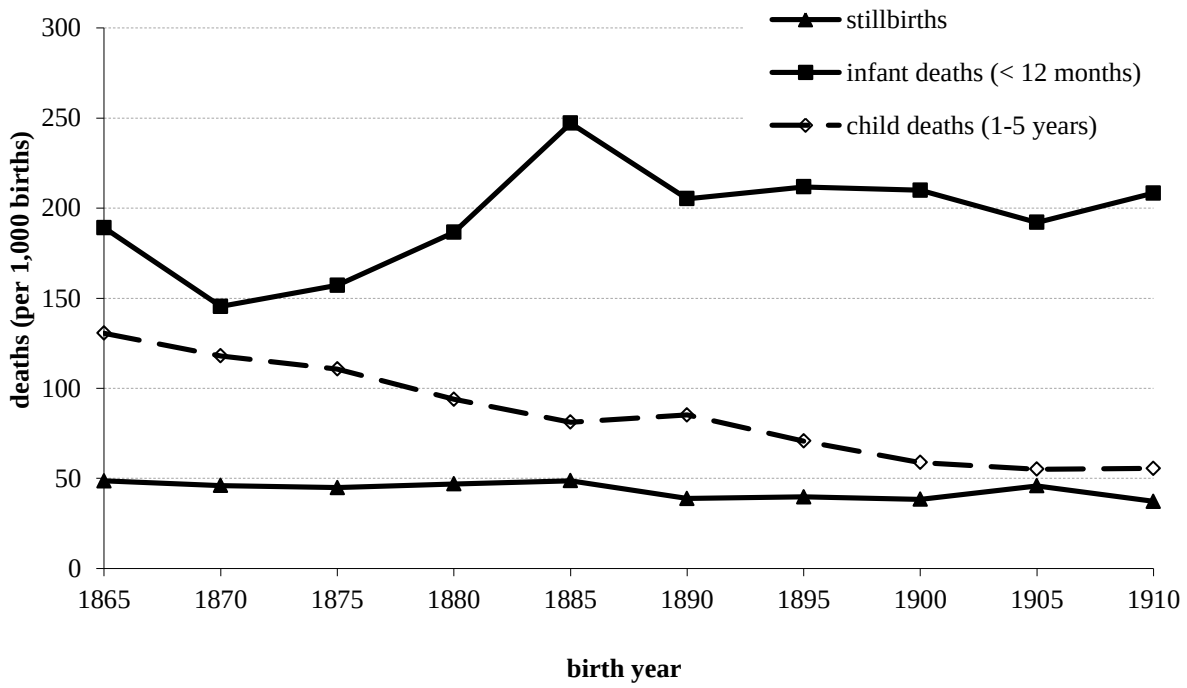
For this study, we selected all legitimate singletons born within the Antwerp district between 1846 and 1905, and we followed them during infancy. Illegitimate children represent a special category, of whom we don't know if their deaths can connected to those of their aunts and uncles, so they were dropped from the analysis. Twins were dropped as well. The remaining children were linked with the infant life courses of their aunts and uncles on the maternal side. Children whose mother had no siblings were dropped as well. Of the remaining 1.347 infants, born into 332 different families, 222 (164,8 per thousand) died within twelve months after birth.

The Antwerp district consists of the port city of Antwerp, which gave its name to both the district and the larger province, and 61 smaller municipalities. Antwerp was the biggest and fastest growing city of nineteenth century Belgium. Its population of 56.000 in 1800 (88.000 in 1846) grew to 273.000 inhabitants in 1900, mainly because of immigration from the district's countryside. This immigration filled the needs of an exploding labour market, fuelled by a revival of the port activities since the first decades of the nineteenth century (Winter 2009). Followed by a rapid port expansion, a dense transport network was created

connecting the Antwerp port with its eastern and north-eastern hinterland (Veraghtert 1986; Bertels 2011). The resulting massive migration is recorded in the Antwerp COR*-sample, which covers the whole district area of Antwerp. For this study, the whole district was used, and any further references to 'Antwerp' should be interpreted accordingly.

On a more aggregate level, for the whole of Belgium, a continuous series of young age mortality rates can be made from 1865 onwards. Figure 3.2.1 shows the evolution of the infant mortality rate (number of deceased infants per 1.000 live births) in the period covered by the COR*-database. The graph shows a rise of infant mortality from 1870 onwards, which peaks in the 1880s, to remain constant at 200 for the two following decades. A similar measure for stillbirths (number of stillbirths per 1.000 live births) remains remarkably constant, with only a slight decrease after 1885. The decline of mortality at young ages is only visible in the age category 'one up to five years', an evolution which is shown here for reasons of comparison. Infant mortality decline in Belgium only started after 1900 (Debuisson 2001).

Figure 3.2.1. Evolution of infant (IMR) and child mortality, Belgium, 1865-1910



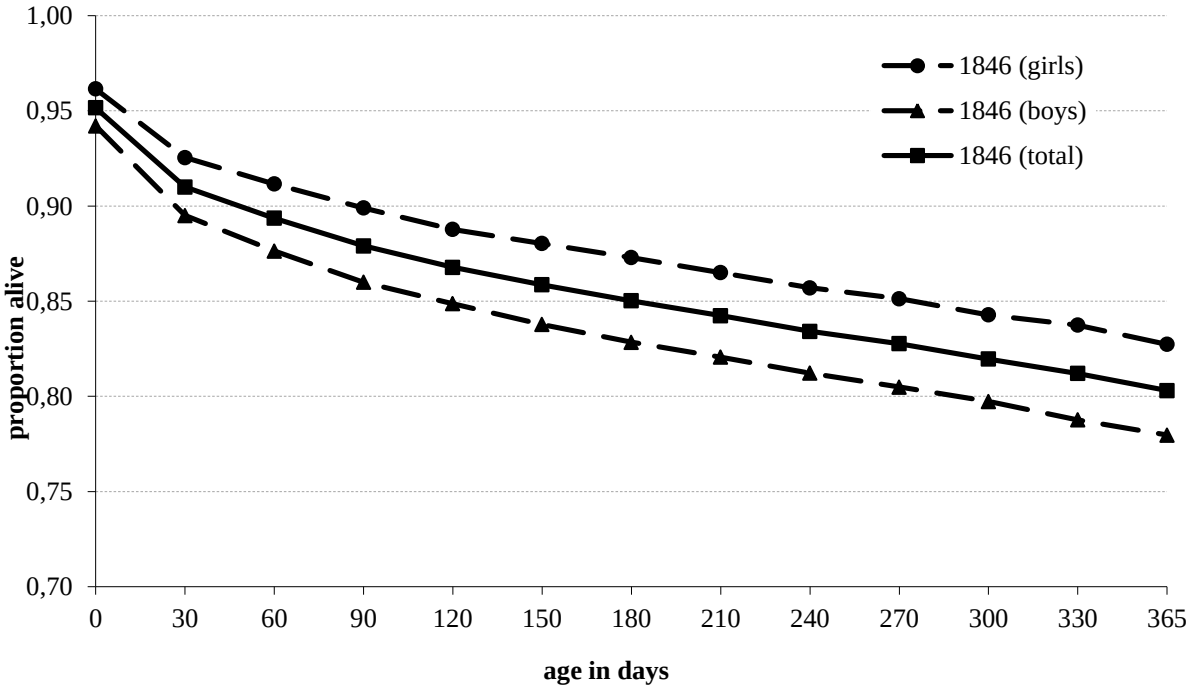
Source: NIS, Annuaire Statistique, 1865-1910.

Comparing the infant mortality rate to the rate of stillbirths learns us that during the second half of the nineteenth century one fifth of all deaths before the age of 12 months were due to stillbirths. Many studies on infant mortality do not take stillbirths into account. Since we are

not only interested in environmental circumstances affecting living children, but also in genetic aspects predetermining death, we opted to include stillbirths in this infant mortality study.

Figure 3.2.2 shows the age-specific survival rates for the whole province of Antwerp in 1846. Only in this year figures with such detailed age categories on the level of a province were available. Stillbirths were also included here, both as deaths as well as in the denominator (they are counted together with the live births). Stillbirths constitute 5 per cent of all births in the province of Antwerp, mid-nineteenth century. After 365 days, 200 per thousand of the children have died. In the province of Antwerp, stillbirths account for one quarter of all infant deaths, so they have an even bigger impact on mortality than in the country average. The overall infant mortality however is lower than the country average (which was already 200 per thousand without including the stillbirths). Sex specific figures show better survival chances for girls than for boys, for all ages up to 365 days.

Figure 3.2.2. Cumulative probability of infant survival (stillbirths included), province of Antwerp, 1846



Source: Récensement générale de la population, 1846.

Table 3.2.1. Clustering of infant deaths between families, district of Antwerp, 1846-1905

	Infant deaths in family													
Births in family	0	1	2	3	4	5	6	7	8	9	Total families	Percentage of families	Percentage of total births	Percentage of total deaths
1	30	2									32	9,6	1,9	0,7
2	41	8	3								52	15,7	6,1	5,1
3	30	10	4	1							45	13,6	7,9	7,6
4	33	6	4	0	0						43	13,0	10,0	5,1
5	11	7	3	2	1	0					24	7,2	7,0	8,4
6	12	15	9	0	0	0	0				36	10,8	12,6	12,0
7	9	7	3	1	1	0	0	0			21	6,3	8,6	7,3
8	6	4	2	4	0	0	0	0	0		16	4,8	7,5	7,3
9	9	8	2	0	0	1	0	0	0	0	20	6,0	10,5	6,2
10	3	7	0	2	0	1	2	0	0	0	15	4,5	8,7	11,0
11	1	4	1	3	2	3	0	0	0	0	14	4,2	9,0	13,8
12	2	0	1	1	3	0	0	0	1	1	9	2,7	6,3	12,4
13	0	2	0	0	0	0	0	0	0	0	2	0,6	1,5	0,7
14	0	0	1	1	0	0	0	0	0	0	2	0,6	1,6	1,8
15	0	0	0	0	0	0	0	0	0	0	0	0,0	0,0	0,0
16	0	0	0	0	0	0	0	0	0	0	0	0,0	0,0	0,0
17	0	0	0	0	0	0	0	0	0	0	0	0,0	0,0	0,0
18	0	0	1	0	0	0	0	0	0	0	1	0,3	1,0	0,7
Total families	187	80	34	15	7	5	2	0	1	1	332	100,0	100,0	100,0
Percentage of families	56,3	24,1	10,2	4,5	2,1	1,5	0,6	0,0	0,3	0,3	100,0			

It should be noted, however, that these are aggregated totals. When comparing with the individual-level COR*-sample, one should be aware of the fact that the Antwerp district, covered by the sample, is the most urbanized district in the Antwerp province. The mortality rate for the Antwerp district can therefore be expected to be slightly higher than the ones for the province, as given in Figure 3.2.1 or 3.2.2.

Table 3.2.1 gives a general overview of the distribution of infant deaths among families in the Antwerp district, 1846-1905. It shows the number of families, according size (number of children ever born), and number of infant deaths. The table is based on the actual family size, consisting of both the sampled children, and their siblings which might have been dropped from further analysis (because they were born either illegitimate or as twins, or because they were born outside the temporal study frame). Table 3.2.1 is based on 1.718 births and 275 infant deaths (=160 per thousand). The births occur in 332 families; 187 families (56,3 per cent) remain 'mortality-free'. Using the observed mortality rate, and the number of families per size, an expected number of families without infant deaths can be calculated. The expected number of 'mortality-free' families is 155 (46,7 per cent), which is lower than observed. Infant deaths happen in a smaller proportion of the families than expected, meaning infant mortality is clustered in the Antwerp sample.

Table 3.2.2. Clustering of infant deaths over generations, district of Antwerp, 1846-1905

	Siblings of mother who died as infant								
Infant deaths in family	0	1	2	3	4	5	6 or more	Total families	Percentage of families
0	104	47	17	10	6	3	0	187	56,3
1	45	16	12	2	2	2	1	80	24,1
2	13	11	5	4	1	0	0	34	10,2
3	9	2	3	0	0	1	0	15	4,5
4	5	0	0	1	0	1	0	7	2,1
5	3	0	1	0	0	0	1	5	1,5
6	2	0	0	0	0	0	0	2	0,6
7	0	0	0	0	0	0	0	0	0,0
8	0	0	0	1	0	0	0	1	0,3
9	0	1	0	0	0	0	0	1	0,3
Total families	181	77	38	18	9	7	2	332	100,0
Percentage of families	54,5	23,2	11,4	5,4	2,7	2,1	0,6	100,0	

Table 3.2.2 presents the numbers of families, according to the infant deaths in those families, and the number of aunts and uncles of the children in those families that died during infancy. Although it is difficult to draw straightforward conclusions from table 3.2.2, there is a slight

tendency towards more infant deaths if there were more siblings of the mother who died as an infant. In the next section this claim will be confirmed using regression analyses.

3.2.3. Measures and methods – problems and solutions

One problem of investigating familial clustering of infant mortality lies with the classical assumption of many regression techniques, namely that all observations should be statistically independent. Observations are always to some (lesser) degree dependent, without causing too much troubles for the analysis. When researching infant mortality clustering within families this assumption cannot be ignored. The idea of mortality clustering implies that the deaths of children from the same family are predicted by identical determinants when measured on the family level. For example, the number of siblings of the mother that died as infants has the same effect on every single child from that mother. Hence, the children's mortality hazards from the same family are dependent. This is a brutal violation of the independency assumption, which almost always means an underestimation of the standard errors of the regression coefficients, which in turn leads to an overstatement of the statistical significance. When violating the assumption, results seem significant, while in reality they are not. A possible solution is to take only one child per family, but that is an awkward workaround, which implies a whole set of extra assumptions, and a much larger data sample. The problem can only adequately be solved by the use of multilevel techniques (Luke 2004). Those techniques use the concept of multiple hierarchical levels of observations, in which data are clustered together to form independent groups. Our observations were analyzed on both the level of the child, and the hierarchically higher level of the mother at which several children are clustered or 'nested'.

Using multilevel techniques does not solve all problems. The age-specific survival rates, as shown in Figure 3.2.2, clearly demonstrate that a child's death risk heavily depends on the age of the child. The hazard of death is not constant, but diminishes over time (i.e. as the child grows older), particularly during the first 12 months of life. Because of this time dependency we used event history models. Those type of models are fit to address questions on the risk of a certain event at any given point in time, conditioned on the absence of the event up until that given point in time. For example, we investigate the hazard or probability of an infant death, at any given age of the infant, on the condition that the infant survived until that given age. An added value of the models is that they can handle censored events with ease (Allison 1984).

We combined the multilevel and the event history approach by opting for a piecewise constant exponential (PCE) event history model with gamma frailty (Holford 1980; Laird and Olivier 1981). In a PCE-model, continuous time (age of the child, in days) is split into discrete intervals ('pieces') or age categories. Within these intervals we assume the hazard of dying, also called incidence rate, to be constant. Based on Figure 3.2.2, these age categories are constructed as follows: a perinatal one (age 0 to 7 days, including the stillbirths), a late neonatal one (age 8 to 30 days), and a postneonatal one (age 31 to 365 days). To this model, a random intercept term was added, which is specified to have a normal distribution with expectation 0. This model is applied to the data in Stata 10, using the `xtpoisson` command with the `i` and `exposure` options (Rabe-Hesketh & Skrondal 2008). For every infant *i* of mother *j*, the logarithm of the hazard rate in age category *s*, λ_{sij}, or the logarithm of the expected rate of death in age category *s*, can be expressed as:

$$\ln(\lambda_{sij}) \equiv \ln\left(\frac{\mu_{sij}}{t_{sij}}\right) = \alpha_s + \sum_{a=1}^{p} \beta_a x_{aij} + z_j \quad , \quad \text{s} = 1,2,3$$

where μ_{sij} stands for the expected number of deaths, and t_{sij}, or exposure time (in days) spent in age category *s*. Once a child dies, exposure time stops, and the child is removed from the analysis. The first term in the right hand member of the equation stands for the age-dependent baseline hazard, the hazard of dying for an individual in age category *s* when all other covariates are zero. We chose three different age categories: perinatal, late neonatal, and postneonatal (coded 1, 2 and 3 respectively); each of them has its own α. The second term is a set of explanatory variables β (*p* in total), be it on the level of the child, or on the level of the mother. The third term is a random intercept, which models between-mother differences in infant survival. This term stands for the non-explained variation shared by children of the same mother, and can be interpreted as a set of unobserved characteristics of the mother, such as her ability to take care of children, her affection or emotional involvement, hygiene etc (Wienke 2011).

We mainly apply multilevel techniques here because of our interest in intergenerational relations. This approach has the additional advantage of presenting a measure for the amount of clustering. The random intercept z_j is used to estimate non-measured differences between mothers. It is called 'random' because it is an intercept that is allowed to vary from mother to mother. We interpret the intercept through its exponentiated form, exp(z_j), usually called 'frailty'. Exp(z_j) is assumed to be gamma distributed, with a

mean of 1 and a variance σ. This variance expresses the differences between mothers, and is a measure of clustering. When the variance is large, there are larger differences between mothers, or more clustering of infant deaths between mothers. If variables are added to a model, a lowered variance of the gamma frailty means that those variables explain differences between mothers in infant death risks. A variance of zero would mean that there are no differences left between mothers, or that all infant death clustering is explained.

The dependent variable in the following analyses is the risk of dying for infants at a given point in time. The models will include variables on both the child and the mother level. Variables on the first level, the level of the individual child, include:

male: dummy variable denoting the sex of the child, 1 if male, 0 if female. Boys are known to have a higher incidence of dying during the first year, due to genetic disadvantages (Tabutin & Willems 1998).

age of the mother: the age of the mother at birth of the child affects the child's hazard of dying in a more complex, non-linear way. If the mother was very young when giving birth, the child is more likely to die than average. But if the mother was rather old when giving birth, due to various factors the child is also more likely to die (Knodel & Hermalin 1984; Nault et al. 1990). A categorical variable was created, differentiating between the ages 'younger than 20', '20-34' and '35 and older'.

the number of older siblings: the higher the birth order of a child, the higher the chances of dying as an infant, a relationship which proved to be fairly linear in our sample. This is due to a combination of two effects. The first one is the maternal depletion effect, which means that having a lot of babies deteriorates a women's health, and thus that of her offspring, resulting in lower birth weights and higher infant death rates (Conde-Agudelo & Belizan 2000). The second is the competition-for-resources hypothesis: the more children present, the less resources available for each child, whether these resources are physical or emotional (Knodel & Hermalin 1984). Both effects are most important early on in life. During a child's infancy, no younger siblings are born yet. Therefore the number of older siblings resembles the total family size, minus the index child.

birth interval with previous child: a shorter birth interval between two children leads to a higher likelihood of the second child to die early on. Part of this effect can be attributed to the maternal depletion thesis, when the mother did not totally recover from the preceding birth (Rutstein 2005). In historical studies birth spacing is often used as a crude measure (proxy) to

control for (the length of) breastfeeding on an individual level (for example Pebley et al. 1991). A dummy was constructed, denoting short birth intervals (short being 24 months or less).

socio-economic status (SES): some variation between infant deaths from different families should be explained when controlling for the SES of the mother. The most common indicator of SES in historical research is the occupation. However, in historical sources female occupations tend to be badly or under-registered (Moreels 2010). Therefore, studies concerned with historical populations often opt for the occupation of a male person who is related closely enough to predict the socio-economic status of the mother and her children, such as the father. We obtained the father's occupational title from the birth certificate of each child. The variable is constructed by first standardizing occupations in HISCO codes (Van Leeuwen et al. 2002), and consequently recoding these codes into four classes (unknown occupation, worker class, farmer class, and elite and middle class).

Variables on a hierarchical higher level, the level of the mother, include:

number of siblings of the mother who died as infants: several variables were constructed to assess the number of aunts and uncles (on the mother's side) who died at an early age.[7] A first variable counts all siblings of the mother that died during infancy. Four other variables split this total in four age categories: the number of siblings of the mother who died (1) as stillbirths, (2) in the perinatal period, 0 to 7 days old, (3) in the late neonatal period, 8 to 30 days old, and (4) in the postneonatal period, 31 to 365 days old. This division along age of the siblings of the mother allows to make distinctions between determinants of a child's death. High effects associated with the early ages of the mother's siblings point in the direction of intergenerational biological (genetic) factors, while effects of higher age groups suggest an intergenerational passing through of social factors, such as breastfeeding practices.

[7] We experimented with the proportion of infant deaths among a mother's siblings, and with a count of the dead siblings. Due to small family sizes there is not much variation in the proportion (one sibling can mean either 0 or 100 per cent dead siblings). Also a proportion does not take size into account (a mortality of 100 per cent for one sibling is much more common than a 100 per cent mortality for seven siblings). Although a count does not take into account the surviving siblings, we found it to be the least imprecise of both alternatives.

3.2.4. Results

Table 3.2.3 presents the results of six multilevel piecewise constant exponential models. A division is made between variables on the first level, the level of the individual child, and variables on the second level, the level of the parents. In the table, the values next to the variables stands for incidence rate ratios (IRR), which are the exponentiated β's from the equation mentioned above. Compared to those β's the IRR's are interpreted in terms of a ratio of risks: when larger than 1, they denote an increased incidence (risk) to die, between 0 and 1 stands for a decrease in risk. The gamma frailty variance σ denotes the amount of clustering, p whether this variance significantly differs from zero[8].

The first model only include the three base hazards and the gamma frailty. The base hazard is highest in the first age category (perinatal), and lowest in the third age category (postneonatal). Not only in the first, but in all models the hazard of dying is much higher in the first age category compared to the other two. The highest risk of dying is thus shortly after birth, in the perinatal period. This confirms what we expected, namely that the hazard of dying for an infant declines as age increases, which encouraged us to analyse the infancy period in three separate pieces, and not in its entirety.

In model 2 the controlling variables are introduced. The controlling variables 'age of the mother younger than 20', 'older siblings' and 'birth interval' have the expected values, in contradiction to 'male' and 'age of the mother 35 and older' (although neither 'age of the mother' nor 'male' are significant). Children with a very young mother, or a higher birth order (children with older siblings) are more likely to die. Short birth intervals with the previous child result in a strongly increased incidence of death. The occupation of the father is not significant, but farmers' children seem to be the best off in terms of survival chances. This is due to the high variety in urbanization within the Antwerp district. Children from farmers mostly live on the countryside, while children from workers mostly live in the very dense urban environment of the Antwerp harbour. The significant higher chance of death for children of 'unknown occupation' compared to farmer class children refers to families where the father is unemployed, and thus (often urban) families with a less stable income. In total,

[8] A variance component cannot take on values below zero. Testing whether the variance differs from zero therefore means testing on the boundary of the parameter space. This is handled by cutting the distribution of the maximum-likelihood estimate of the variance, a normal distribution, at the boundary value. The likelihood ratio test statistic is a mixture of a chi-square with one degree of freedom, and a chi-square with zero degrees of freedom. This one-tailed normal distribution procedure is standard in Stata, see Gutierrez et al. 2001.

Table 3.2.3. Multilevel piecewise constant exponential event history models of the incidence rate of dying, district of Antwerp, 1846-1905

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Constant base hazards						
α_1 (0-7days)	0,0050 ***	0,0019 ***	0,0045 ***	0,0043 ***	0,0018 ***	0,0017 ***
α_2 (8-30 days)	0,0008 ***	0,0003 ***	0,0007 ***	0,0007 ***	0,0003 ***	0,0003 ***
α_3 (31-365 days)	0,0004 ***	0,0002 ***	0,0004 ***	0,0004 ***	0,0002 ***	0,0002 ***
First-level variables	IRR	IRR	IRR	IRR	IRR	IRR
Male (ref. = female)		0,941			0,940	0,950
Age of the mother						
- younger than 20 (20-34 = ref.)		1,173			1,182	1,191
- 35 and older		0,813			0,834	0,832
Number of older siblings		1,115 ***			1,115 ***	1,116 ***
Short birth interval (24 months or less)		1,355 **			1,355 **	1,376 **
Second-level variables	IRR	IRR	IRR	IRR	IRR	IRR
Occupation of the father						
- unknown (ref. = farmers)		2,360 *			2,222 *	2,218 *
- higher and middle class		1,513			1,453	1,455
- workers		1,743			1,636	1,653
# siblings of mother died						
- as infants			1,138 *		1,132 *	
- in perinatal period (0-7 days)				0,995		0,950
- in neonatal period (8-30 days)				1,824 **		1,895 **
- in postneonatal period (31-365 days)				1,141		1,144 *
Frailty term						
frailty variance	0,9213	0,7098	0,8951	0,8579	0,6962	0,6441
p-value	<0,001	<0,001	<0,001	<0,001	<0,001	<0,001
Model statistics						
Wald chi² (p < 0.0001)	6.335,53	6.583,94	6.419,72	6.520,63	6.639,36	6.808,01
degrees of freedom	3	11	4	6	12	14
N (observations)	3.788	3.787	3.788	3.788	3.787	3.787
N (mothers)	332	332	332	332	332	332

*(p < 0,10) **(p<0,05) ***(p<0,01)

the above variables in model 2 explain almost a quarter (23 per cent) of all variance in infant mortality between families.

Models 3 and 4 only use the variables of interest, the variables counting the siblings of the mother who died as an infant. In model 5 and 6, the controlling variables are introduced as well. When a mother lost siblings during the first twelve months of their life, her own children are about 14 per cent more likely to die per deceased sibling of her. The size of the effect is a little lower when controlling for variables important to the clustering phenomenon (13,2 per cent, model 6), but it is still (weakly) significant. This means that part of an infant's survival chances can be predicted by his or her family history of infant mortality. This effect cannot be fully explained by the controlling variables. It makes the intergenerational effect an important determinant for infant mortality clustering. This determinant could be explained due to both social and biological causes. Splitting the deceased siblings of the mother in age categories reveals some remarkable results (model 4). When a mother had siblings that were stillborn, or who died in the first week (both are in the category 'perinatal period', 0-7 days old), it has very little or no effect on the incidence of death among her own children. However, after the first week the death of a sibling of a mother has a strongly significant, very predictive effect on the survival of her infants. Children with an aunt or uncle that died in the late neonatal period saw their mortality risks almost double (89,5 per cent increase in model 6). The effect is much lower, but still significantly present when these aunts and uncles died after the first month. It should be noted that perinatal deaths are known to be mostly attributed to genetic or biological characteristics of mother or child (miscarriages, being born as a twin or other causes of lower birth weights, congenital defects, etc.). The results presented in table 3.2.3 (and most notably in model 4 and 6) do not reveal a strongly visible intergenerational transmission of these genetic causes.

A clearer view can be obtained by running separate models for infants in the three age categories: the perinatal period (model 7), the late neonatal period (model 8) and the postneonatal period (model 9), results are to be found in table 3.2.4. Fitting good models was not always possible: model 7, in which the survival chances of infants in their perinatal period are modelled, is not significant. Model 8, a similar configuration for the late neonatal period, is only weakly significant. Although no further conclusions can be made for the perinatal period, we see that mortality in the late neonatal period is correlated between generations, as is the case with mortality in the postneonatal period. The late neonatal deaths of siblings of the mother have a huge impact – more than seven times as much chance of dying – on her infants in the same age period (model 8). The effect of late neonatal deaths among a mother's

siblings seems to reverberate with her postneonatal infants (model 9), although it is not significant. A mother's siblings dying in the postneonatal period increase mortality risks for postneonatal children (model 9); with late neonatal children the effect is reversed, although it is not significant (model 8). A mother's siblings who die right after birth, in the perinatal period, have no significant effect on the mortality risks of her own children, in neither of all three age categories.

Table 3.2.4. Multilevel poisson models of the incidence rate of dying, per age group, district of Antwerp, 1846-1905

	Model 7 perinatal	Model 8 neonatal	Model 9 postneonatal
First-level variables	IRR	IRR	IRR
Male (ref. = female)	0,748	0,545	1,100
Age of the mother			
- younger than 20 (20-34 = ref.)	1,226	1,874	1,029
- 35 and older	1,648	1,104	0,628*
Older siblings	1,117	1,030	1,124***
Short birth interval (24 months or less)	1,206	1,038	1,532**
Second-level variables	IRR	IRR	IRR
Occupation of the father			
- unknown (ref. = farmers)	1,253	1,131	3,382**
- higher and middle class	1,556	1,303	1,653
- workers	1,004	1,796	2,107
# siblings of mother died			
- in perinatal period (0-7 days)	1,286	0,475	0,928
- in neonatal period (8-30 days)	0,834	7,482***	1,693
- in postneonatal period (31-365 days)	1,156	0,695	1,179*
Frailty term			
frailty variance	4,0751	0,3799	0,9189
p-value	<0,001	0,321	<0,001
Model statistics			
Wald chi²	11,49	18,03	30,20
p	0,4035	0,0809	0,0015
degrees of freedom	11	11	11
N (observations)	1.333	1.232	1.222
N (mothers)	330	321	322

*(p <0,.10) **(p<0,05) ***(p<0,01)

After presenting the results in terms of explaining individual chances of infant mortality, it is equally instructive to look at the explanatory power of the models in terms of infant mortality clustering within families. Therefore we return to the models in table 3.2.3, and focus on the

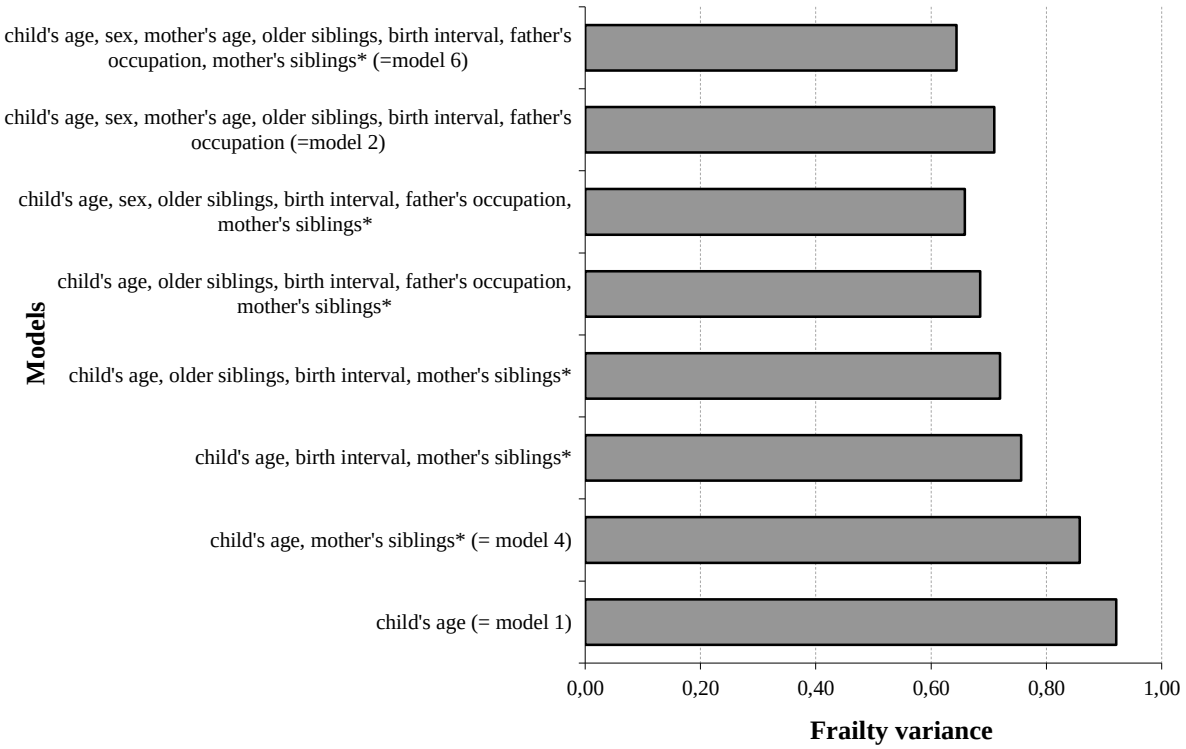
gamma frailty variances of model 3 and 4 on the one hand, and model 5 and 6 on the other hand. The variances in model 3 and 4, as compared to the null model (model 1), show that both models 3 and 4 explain some infant mortality clustering. Model 5 and 6 allow us to assess whether these explained clustering overlaps with the clustering explained by the controlling variables. As expected, this is the case. Model 5 explains less clustering than the sum of the explained clustering by model 2 and by model 3; model 6 explains less than the sum of model 2 and 4 together. The drops in variance are not fully cumulative because the variables of interest explain partly the same clustering as the controlling variables. Moreover, model 5 barely explains more clustering than model 2, meaning that the variable 'number of siblings of the mother dying during infancy' does not explain much additional clustering than the controlling variables. When this variable is disaggregated into age categories (model 6), it does become meaningful for the clustering phenomenon. The age-specific deaths of a mother's siblings explain differences between families that were not explained before. The family history of infant mortality is thus not only an important determinant of infant mortality, but it has additional explanatory power to the clustering phenomenon when compared to the controlling variables.

The gamma frailty variance not only captures the intra-familial dependency, but also has a direct interpretation in terms of the hazard ratio (for an application, see Guo 1993). A variance of 0,92 (model 1, table 3.2.3) means that if there is an infant death in the family, the index infant experiences a 92 per cent increase in risk of dying. These family effects as seen in model 1 are quite large, but diminish to 0,71 after inclusion of the controlling variables (model 2), because the controlling variables partly explain the intra-familial dependency. By extending the model with intergenerational effects (model 6), the residual familial effects further decrease to 0,64. Keeping all other factors constant, infants still have a 64 per cent increase in risk of dying when one of their siblings died. This residual familial effect remains unexplained.

Figure 3.2.3 visualises the explanation of clustering by assessing the gamma frailty variance for different variable configurations. The variance in the bottom bar, model 1, is the total amount of clustering present. We started with the full model (model 6), and dropped all variables from this model in a stepwise manner. Leaving out a variable from the model results in an increased gamma frailty variance, indicating the loss in explanatory power of a model without this variable for the clustering phenomenon. Leaving out all variables results in the total amount of clustering present. The less complete a model is, the more the increase in variance will be. Figure 3.2.3 shows that every variable – at least to some degree – explains

between-family differences in infant mortality. Among them, the age-specific deaths of a ‘mother’s siblings’ seems a relative strong component in explaining clustering.

Figure 3.2.3. Explanatory strength of some variables for the clustering phenomenon by stepwise reduction of model 6



* ‘mother’s siblings’ refers to the age-specific deaths of the siblings of the mother

3.2.5. Conclusion

The aim of this study was to re-examine the role of mothers in the historical clustering of infant mortality in certain families. Most scholars focus on determinants measured at the level of the single household, mostly on the level of the mother. This study tried to look beyond that single household by comparing mortality in different generations of the same family. More specifically, death risks of the siblings of a mother during infancy were compared with death risks of her own infants.

Our results show, firstly, that mortality risks are correlated between generations along the maternal line. Age-specific analyses revealed that the age at death is determining for the intergenerational transmission of mortality risks. When deaths occurred more than one week after birth, the mortality risk was transferred to the next generation. Especially late neonatal

deaths (second up until fourth week after birth) are very predictive. Infant death risks are thus affected by the family history of infant mortality.

Secondly, our findings also demonstrate that this intergenerational mortality pattern is an important determinant for infant mortality differences between families, or the clustering of infant death. The intergenerational effects on infant mortality clustering are not explained by the age of the mother, the gender of the child, the birth interval with the previous birth, the birth order, or the socio-economic status of the father; factors which are put forward in the literature in order to explain clustering.

Our figures on the intergenerational effects by age at death reveal some information on the mechanisms that transfer deaths risks across generations. The intergenerational transfer of infant deaths seems to be caused mainly by determinants that emerge only after more than a week after birth, and are at their strongest during the second up until the fourth week after birth. After the first week of age biological causes and genetic predispositions are starting to loose their impact on death. Although the risk of dying as an infant is both socially and biologically determined, it is not dominantly transferred through the genes, but possibly more through behaviour. This is a statement that is very hard to prove, but it seems to be suggested by our age-specific analyses.

Lack of confirmation of this hypothesis is mostly caused by the correlation between postneonatal deaths in successive generations, which is weaker than expected. It is hard to point to behavioural explanations exclusively, as the strongest correlation is found with late neonatal deaths, a period in which other factors, such as specific genetic loadings or biological weaknesses, are still important. Therefore, and because of the absence of a clear distinction between what caused death, our study does not succeed in providing a conclusive answer on how mortality is transferred from one generation to the other. Nor do our results give a definite explanation of the clustering phenomenon. However, it does prove that an intergenerational approach is worthwhile, in that it provides an additionally, previously unstudied determinant of both infant mortality and death clustering, namely the family history of infant mortality.

Family and the family's mortality history are important for an infant's survival. Infant mortality cannot be understood properly when one does not take family into account. Family refers to more than the mother or even the mere household. It also includes intergenerational kin. Infant mortality is found to be transmitted through different generations, at least to some extent. Studying infant mortality in an intergenerational view can significantly improve our knowledge of the relative importance of the factors causing infants to die.

The meaning of this newly-gained knowledge on clustering for our general understanding of infant mortality is not clear yet. Death clustering, and especially the intergenerational component in it, can help us to understand the mortality transition. This evolution from high to low mortality figures for both children and adults has been found in many countries as part of a range of processes, which are often called 'modernization'. The decline of infant mortality has been explained from above: medical progress, better education of both doctors and midwives, increase in understanding of public health and a deliberate effort to improve it, etcetera led to an improved health awareness of both medical personnel and mothers. This in turn, combined with social progress of these mothers, led to an increase in infant health and a reduction in their mortality. Before the transition, there is evidence of strong familial differences in infant mortality, which – as we were able to show with this study – persisted from one generation to the other. A better understanding of how these risks are transmitted through generations would be very helpful in understanding how the mortality transition took shape, and how it diffused to different families. Besides only factors outside the family, clustering may point to the transmission of risks within families as well.

3.3

USING DEATH CLUSTERING IN AN ATTEMPT TO REASSESS THE IMPACT OF MEN ON INFANT MORTALITY

An intergenerational approach

Mattijs Vandezande

3.3.0. Abstract

This paper shows that death clustering, the unequal distribution of young age mortality between families, can be used to better our understanding of the mechanisms causing infant deaths. Applying concepts (high-risk families) and methodology (multilevel approach) of death clustering, patterns of infant mortality were studied between generations along the paternal line. Using a rich multigenerational database sampling 19th century Antwerp (Belgium), mortality in the postneonatal period (one month of age onwards) was found to be transmitted across generations. However, deaths very soon after birth were found to enhance survival chances of newborns in the next generation. Fathers, who were a child in one generation and parent in the next, must have played a key role, linking deaths between generations. We believe the possible mechanisms at play are intergenerational transmitted paternal care, as well as biological selection close after birth.

Keywords

death clustering – infant mortality – intergenerational transmission – fathers – parental care

3.3.1. Introduction

The goal of this paper is to explore an alternative way in which a father can affect infant mortality. The focus is on the caretaking responsibilities of the father. Parents' abilities to take care of children, the parental (in)competence (or ignorance), is mentioned as a likely cause of existing differences in young age mortality between families. Qualitative evidence by Das Gupta (1990), for example, confirms the importance of this factor in contemporary societies. For historical populations Edvinsson et al. (2005) argue that care-giving capabilities probably explain more of these differences than genetic heredity, although they find no link with the (mother's) educational level. Remarkably, Das Gupta as well focuses her research on the competence of the mother instead of on both parents, along with many other researchers concerned with parental care and young age mortality. Indeed, little is known on how fathers interact with the survival or death of their children, especially for historical populations.

That does not mean that the father is always neglected in studies on (historical) young age mortality. Characteristics of the father are commonly used to obtain an indirect measure of an aspect of the mother-child relationship. For example, many historical populations lack the sources to value the level of the education, the relative social status or economic power of the mother (Hill 1993; Szelenyi 2001). Therefore, it has become common practice to turn to the father, and to use his occupational title to incorporate the whole family into a social class

(Britten & Heath 1983). The real impact of the father on infant mortality, according to the attention given to it in the literature on historical demography, seems to be connected with his mere presence. There is a whole branch of studies devoted to the absence of the father in the family, and the distress it causes, such as an increased risk for infants to die (an overview in Sear and Coall 2011). Still, there is relatively few attention for the role of fathers in young age mortality.

One of the main causes for this lack of attention is that it is commonly agreed upon that in the past taking care of the children was mainly a mother's task. While the mother took care of the household and raised the children, the father had a breadwinning role (Shorter 1975; Simonton 1998). This strict gender division is often perceived as absolute. However, it is hard to believe that fathers had no direct impact on the survival of their children. This study therefore tries to assess the effect of paternal care on infant survival.

3.3.2. A gendered parental care

Evolutionary anthropologists studying hunter-gatherer societies consider biparental investments in offspring a peculiarity specific to the human species (Kaplan 1994). In contrast to other species, women have to raise several dependent children at the same time. It takes additional adults to make all the investments necessary to raise these children worthwhile. A dominant view is that the husband assists his wife, resulting in an interdependence of men and women in the human reproductive strategy (Lovejoy 1981). However, it proves to be difficult to confirm this empirically. In general women do not seem to be as dependent on men as we think (Hawkes, O'Connell and Blurton Jones 1997). Men's investment is only supporting, and not very necessary per se. Their main role is therefore theorized as to allow women to reduce their own parental investments, in favour of increasing those in fertility (Winking 2006). Men's auxiliary role might as well be played by another (kin-related) adult. This alternative to paternal care is sometimes even more efficient, e.g. in the case of grandmothers (Hawkes et al. 1998).

Similar considerations are found in literature on populations from the 19th century past, where the role of the father is often studied through the impact of his death. According to Beekink, Van Poppel and Liefbroer (2002) the loss of a father can affect the health of his children in multiple ways. Fathers, it is argued, are the main breadwinners, and their deaths deprive the surviving family members from a major share – if not all – of a family's income. This is the most direct and most visible impact. Also, if fathers were the primary maintainer of a social network, the loss of this social safety net may isolate the family in times where

help might come most useful. Another mechanism explains the health impact on the child through the spouse. The emotional and psychological consequences of the death of a partner might negatively affect the functioning of a mother, and thus the health of her children. As with hunter-gatherer societies, these mechanisms are not often found to have an empirical historical basis. The majority of studies do not find any (significant) relation between the loss of a father and child survival, especially not when only very young children are studied (see literature reviews in Sear and Mace 2008; Sear and Coall 2011). Besides, women in historical populations might be less dependent than we think, even on the economic level. Although often underregistered in historical sources, women from the past did have a clearly defined economic role (Simonton 1998; Tilly and Scott 1978).

Parental care is thus mostly considered as a mother's concern, where fathers seem to play a supporting role of minor importance. The loss of a father, and the termination of his contribution, does not always result in noticeably altered child survival chances. In the past families could probably cope with this kind of distress by increasing the maternal investment, or by receiving a helping hand from outside the household. Kin members, such as grandparents, aunts, etcetera, came to aid. In some cases institutions turned the tide, by providing (financial) support to widows. Parental care, at least in some cases, was replaceable. This makes it very difficult to study the role of fathers on child survival, when solely looking at the effect of his death. It is necessary to find another way to assess the effect of fathers on infant survival. The problem hereby lies in disentangling parental care into a maternal and a paternal component, since the latter tends to be compensated by the former in order to maintain the total level of care.

3.3.3. Infant mortality in an intergenerational perspective

When solving the problem of the intertwined role of mother and father in parental care, we need to be able to measure aspects of this care that can only be attributed to one of either parents. Especially for the father this is almost impossible. It is very hard to find characteristics of the father that solely affect his impact on child survival, and at the same time do not affect the impact of the mother. This is where the intergenerational transmission of deaths from a familial perspective becomes useful.

Recent studies have revealed the existence of a disproportionate spread of (young age) mortality within families, called 'death clustering', both in contemporary (Das Gupta 1990, 1997) and in historical populations (Lynch and Greenhouse 1994; Edvinsson et al. 2005). Much has yet to be discovered about this phenomenon, and its meaning for our general

understanding of young age mortality is unknown to date. Although many aspects still remain uncovered, there seems to be some general awareness that clustering might alter our interpretations of mortality, at least for historical populations (Edvinsson et al. 2005; Janssens, Messelink & Need 2010). Until now the description and causes of this phenomenon received a lot of attention, but the complete picture of its determinants and impact on our current knowledge remains unclear.

In an earlier study we explored intergenerational patterns of infant mortality as a possible cause for clustering (see chapter 3.2). Infants were found to be more likely to die if they had aunts or uncles (along the maternal line) who also died at young ages. Infant death risks were thus found to be inherited between generations along the same (maternal) pedigree. Some families have a genealogical history of high infant mortality, while others do not, which was found to be one of the causes for differences in infant mortality between those families. A similar scope can be applied to study infant deaths along the father's pedigree. If deaths in the father's family of origin are connected with the deaths of his own children, it points to a role of the father in infant mortality, aside from the role of the mother.

This paper uses the concept of death clustering to answer some questions on the mechanisms of infant mortality in the past. This is done by constructing a theoretical framework around the family in which the child grew up, instead of around the individual child. Within this framework we focus on the father, and link his parental role to all children simultaneously. His role is separated out from that of the mother by involving his family of origin in the survival chances of his children. We will thus analyse infant survival chances while taking familial clustering into account, hereby making use of the correlation of infant deaths between generations.

The unequal spread of child deaths between families creates a group of families with an increased child mortality, so called 'high-risk families' or 'high-mortality families' (for a discussion: see chapter 3.1). Consequently, there is also a group of families with reduced infant mortality. Not all children in high-mortality families die young. Some of them survive, and start families of their own. Their experience of growing up in a high-mortality family might affect the survival chances of their own children. Their siblings might have died as a consequence of specific parental care (or the lack of it), and (in)competence of the parents. Although this does not often result in an immediate death, it wears down the fitness of the child, ultimately, after several weeks or months, resulting in the death of the child. An important component of parental care is the amount of breastfeeding a mother gives to her children. If this is only limited, or is replaced by unpasteurized cow milk too soon, it can

negatively affect child health. Breastfeeding, along with other childcare practices such as simple hygiene measures like washing hands, is something that parents learn from their own parents. If a tradition of insufficient parental care is passed on from parent to child, the resulting child deaths might be found between generations as well. This is already confirmed for mothers and their daughters in a previous study (see chapter 3.2). If a similar correlation is found between children deaths, and deaths at young ages in the father's pedigree, this can be caused by the means of similar traditions of insufficient childcare practices.

Care practices are usually associated with the mother-daughter relationship, but an intergenerational transmission of death risks along the paternal lineage linked with bad parental care suggests that the fathers also play a role in how childcare takes place, e.g. by imposing his will on when and how frequent a mother gives breast. If present, this role of the father is hypothesised to be weaker than the role of the mother. Unfortunately, with our data it is not possible to investigate the impact of deaths in the previous generation along the maternal and the paternal lineage simultaneously, so we cannot directly compare the strength of intergenerational transmission along both maternal and paternal lineages.

Aside from a behavioural component, biology might also play a role. Deaths of children can be transmitted between generations because of shared biological weaknesses that make the child vulnerable. If genetic they can be inherited from one generation to the other. In other words, genes transferred from fathers to their children can invoke an increased death risk. A genetic transmission between generations can affect infant survival chances both in a positive and in a negative way. If a father's siblings died early on, it can mean he and his siblings share weaker genes, which in turn can be inherited by his children. On the other hand, since the father himself survived his early childhood, it can also mean that there is a selection effect in his family whereby the ones with weaker genes die early on, leaving the biologically stronger kids alive. This might result in children having higher survival chances at young ages than the siblings of their father had, because of the natural selection in the father's early childhood. Deaths that have pure genetic or biological causes tend to manifestate themselves very early on, in the first weeks or even first days after birth. If there is a visible (i.e. significant) biological correlation in survival chances between generations, it would show up mostly among perinatal (first week) and late neonatal (second up until fourth week) infants.

This study is restricted to infants, whereby infancy is defined as the first twelve months of life. The central question is whether mortality in this group can be predicted by infant mortality in the previous generation from the father's side. Survival chances are expected to be lower among children from fathers who had siblings that died during infancy.

To further elaborate on the causes of this relation, the results will differentiate between infants in the perinatal (first week after birth), late neonatal (8 up until 30 days after birth) and postneonatal period (first month onwards), both for the observed children as for their aunts and uncles: the siblings of the father. If biological causes, which tend to show up right after birth, are of importance in the intergenerational transmission of infant mortality risk, a correlation can be expected among perinatal survival chances between the two generations. If infant deaths are caused by the quality of the parental care, this correlation is expected to be found mainly in the postneonatal groups.

3.3.4. Data and sample selection

This study uses data from the Antwerp COR*-database, release August 2010 (Matthijs and Moreels 2010). The database is built with individual, longitudinal data from the Belgian population registers and Civil Registration (birth, marriage and death certificates). The temporal scope is 1846-1920, marked by the availability of the sources and nowadays' privacy regulations. The geographical focus is the district of Antwerp, a densely populated 1.000 square kilometre area in the north of Belgium, consisting of the port city of Antwerp and the surrounding countryside, in total another 61 municipalities. For this area a name sample was taken: all individuals with a family name starting with the letters COR* were selected (such as Cornelis, Corthals, Correwyn, etcetera), as well as their cohabiting relatives. The sampled letter combination was tested to be representative as for geographical spread and a number of sociodemographic characteristics, such as sex, marital status, social status and concentration of migrants (Van Baelen 2007). As long as these people were present within the temporal and regional frame, individual life courses were reconstructed, ideally from birth until death, including information on occupation, marriage, migration and reproduction. This yields a total of over 33.000 individuals, roughly one third of them having a COR* family name (resulting in nearly 800 different COR* family names). The choice for a name sample leads to the inclusion of many kin relations within the database. Combined with the longitudinal approach it makes the data most suitable for intergenerational research.

This study restricts itself to the period 1846-1906. The Belgian population register starts in 1846, marking the start of this study. The choice of truncating the study at 1906 is based on the availability and reliability of the collected death certificates: not all death

certificates later than 1906 were (publicly) available at the moment of data collection.[9] All individuals born between 1846 and 1905 were selected, and followed until they reached their first birthday, or until they were censored due to death or outmigration during infancy. Stillbirths were selected as well, and they were treated as perinatal deaths. If individuals were born outside of the sample area, they were followed from their immigration into the sample area onwards. Then for all these individuals the sibling composition was reconstructed as accurately as possible. Siblings born outside of the study's time frame, although not included as observations, were accounted for in the family reconstitution. Furthermore the amount and birth order of siblings of the father were reconstructed as well. Only children with a surviving father were selected. Where confusion can arise they will be called 'reference children', as compared to the childhood periods of siblings of the father, which are the aunts and uncles of the reference children. For illegitimate children (i.e. children from an unmarried mother) it is often hard to trace the father, therefore they were not included in the study. Since the focus is on the survival chances of the siblings of the father, children from fathers without siblings were dropped from the study.

Table 3.3.1. Mortality by age interval, Antwerp sample, 1846-1905

age interval	# of deaths (proportion)	# of children*	mortality rate (per 1000 births)	per diem risk of dying (per 1000 births)
perinatal (0-7 days)	59 (0,17)	1.756	34	4,80
late neonatal (8-30 days)	41 (0,11)	1.674	24	1,06
postneonatal (31-365 days)	257 (0,72)	1.634	157	0,47
total	367 (1,00)	1.767	208	0,55

* #of children = (number of births + number of immigrations) – (number of deaths + number of emigrations)

These operations yield a total of 1.767 infants, and 408 different fathers. From this selection, 367 infants, or 208 per 1.000 births, spread across 183 different fathers, died before they reached age one (stillbirths included), which is a little more than the national population average during 1846-1905, a constant 175 per 1.000 (Masuy-Stroobant, 1983; own

[9] The collection of death certificates was done in the summer of 2007. The Belgian privacy law states that civil registration is not publicly accessible for non-kin members for a period of 100 years. Therefore death certificates were collected up to 1906.

calculations).[10] Table 3.3.1 shows the distribution of deaths and infant risks in three different age intervals: the perinatal period (0-7 days since birth, stillbirths included), the late neonatal period (8-30 days since birth), and the postneonatal period (from 31 days since birth onwards). Despite the (per day) risk of death being the highest right after birth, and declining as the child grows older, both the absolute number of deaths and the mortality rate are the highest in the postneonatal period, since this is by far the longest period. The number of children is not declining in function of the number of deaths, since the sample of infants is not a stable population, but one with both immigration and emigration. Migration was accounted for by calculating the population at risk based on the actual number of observed infants in every age category. Some descriptive results on the amount of infant deaths per family according to infant deaths in the paternal pedigree can be found in table 3.3.2.

Table 3.3.2. Infant deaths related between generations, Antwerp sample, 1846-1905

	Siblings of father who died as an infant								
Infant deaths in family	0	1	2	3	4	5	6 or more	Total families	Percentage of families
0	129	52	9	11	6	3	3	213	52,2
1	37	14	8	3	0	2	0	64	15,7
2	32	12	7	5	3	1	0	60	14,7
3	25	3	4	2	0	0	0	34	8,3
4	13	2	0	1	0	0	0	16	3,9
5	4	1	0	0	1	0	0	6	1,5
6 or more	6	3	1	2	2	1	0	15	3,7
Total	246	87	29	24	12	7	3	408	100,0
Percentage of families	60,3	21,3	7,1	5,9	2,9	1,7	0,7	100,0	

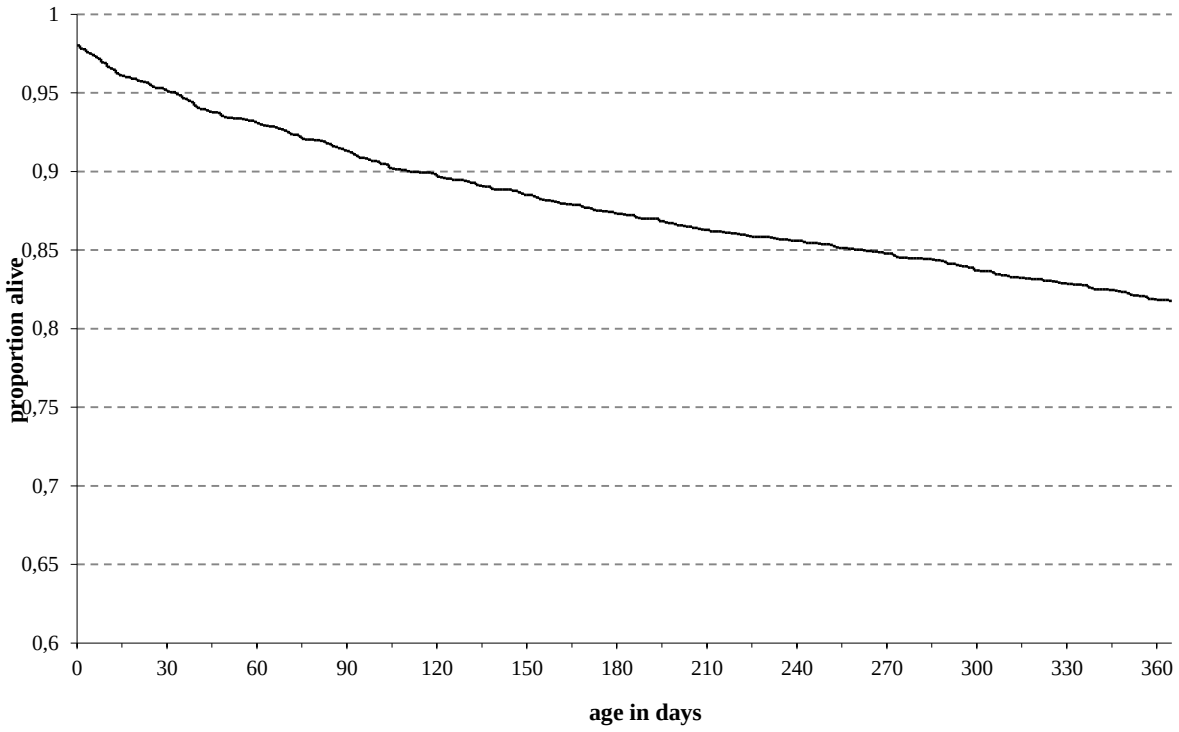
3.3.5. Methodology

An well-known aspect of childhood mortality is that mortality risks typically decrease with age. The highest risk for an infant to die is right after birth. This risk decreases at a declining rate: mortality chances drop very quickly in the first weeks, but are close to stable at the end of infancy. This known evolution of the 'hazard' of infant death has methodological consequences which should not be ignored. When studying the timing of death, it is necessary to account for the fact that death risks are not constant, but decreasing as time since birth has elapsed. Therefore a survival analysis approach is chosen: the piece-wise constant exponential (PCE) model (Yamaguchi 1991). In this model the time during which a process is observed is

[10] Belgian infant mortality only starts to decline as late as 1900, while the second half the nineteenth century is characterized by what is called 'the absence of an evolution' (Masuy-Stroobant 1983).

split up into several intervals, or 'pieces', each having a different constant. Aside from this constant, covariates are assumed to have the same influence in each interval. Their prediction of the dependent variable, here the death of an infant, is estimated using Poisson regression. In PCE models the Poisson approach allows intervals to have variable lengths, making this approach superior to the more common logistic regression. The PCE model is especially useful when the outcome of the dependent variable, the risk of an infant death, is known to be varying across intervals, as is the case with infant mortality, where death risks gradually decrease across intervals. The cut-off points that separate the pieces can be chosen freely. This study splits every child's infancy in three intervals: from birth onwards including the first week ("perinatal"), from the eighth day after birth until the end of the first month ("late neonatal"), and the remaining eleven months until the end of infancy ("postneonatal"). Stillbirths are included as observations in the perinatal interval.

Figure 3.3.1. Kaplan-Meier graph of infant survival, Antwerp sample, 1846-1905



A second peculiarity of this study, also one with important methodological consequences, is the nested structure of the data. Due to the sampling technique there are several observed children who share the same father. Characteristics such as his occupation or the survival of his siblings affect all his children at the same time, each of them being individually analyzed. In statistical terminology, several observations (children) are nested within the same group

(offspring sharing a father). Therefore a multilevel approach of the PCE model is used (Rabe-Hesketh & Skrondal 2008). Multilevel techniques allow for two error terms: one on the individual level and one on the level of the group (Luke 2004). Also the model intercept is allowed on both the individual level and on the group level. Error terms and intercepts on the group level ('random' errors and intercepts) take on the same value for observations in the same group (children from the same father), but are different for observations from different groups (children with different fathers). On the individual level both error terms and random intercepts ('fixed' errors and intercept) are different for every observation, independent from their group membership. Usually the nested structure of data is seen as a nuisance, and by using multilevel techniques we try to get rid of this nuisance. Here the multilevel approach has an additional advantage. Since the random intercept varies across groups (or fathers), its variance is a measure of the differences across these groups. This variance can be used as a direct measure for the spread of infant mortality across families, or death clustering. The higher the variance, the more clustering in the data. Adding variables that explain inter-familial differences in mortality leads to a decrease in variance, or a decrease in clustering. When the variance is zero, there is no unexplained clustering left. Therefore a random intercept will be included in the PCE model.

A major downside of this approach, a PCE model with a random intercept, is that effects are estimated to play an equal role across all ages, after taking into account an age-specific constant. When a father lost his infant siblings, it is assumed that this has an equal effect on his children in the perinatal, the late neonatal and the postneonatal interval. This assumption might not be fully correct. Children might be affected more in certain intervals than in others. Therefore, for every 'piece' or age interval a separate model is needed, in which is checked whether covariates do have similar effects across ages.

In all models the dependent variable is whether or not a child dies within a given age interval. The explanatory variable of interest denotes how many siblings of the father die during infancy. Additional variables count the number of siblings of the father that die during a specific period of infancy. As to periods the perinatal, the late neonatal, the postneonatal categories are discerned, analogous to the age intervals of the observed children in the dependent variable. Their effect on infant survival is controlled for several indicators known to affect infant mortality. They are family-specific, and might be transferred through generations, causing spurious relationships between infant deaths in the father's family of origin, and among his own children. The sex of the child and whether or not the child is a twin birth are such indicators. Infant boys tend to have slightly higher mortality risks than infant

girls, because of a biological disadvantage (Tabutin and Willems 1998). Twins are known to have on average lower birth weights, which raises mortality risks, especially in a low-quality medical care environment such as the 19th century past. A third indicator is the family size, which is measured through the amount of older siblings a child has, which is the family size at birth of the reference child, as well as a measure of its birth order. Children with higher birth orders are more likely to die, not only because of preferences for the firstborns, but mostly because the increased family size creates a problem of resource dilution (Knodel and Hermalin 1984). Resources, both material and emotional, diminish (or 'dilute') if they have to be spread across more individuals, turning a family into a stressful environment of resource competition (Blake 1981). A last but very important controlling variable is the socio-economic status of the father. Its importance lies with the amount of attention the literature has devoted to socio-economic differences in infant mortality. Intergenerational social mobility is low in the 19th century (Van Bavel et al 2011), so a father's status is likely to be correlated across generations. Social classes are constructed based on the occupational titles, which are standardized using the HISCO format (Van Leeuwen, Maas and Miles 2002), and transferred to a reduced form of HISCLASS, a social class scheme for historical populations (Van Leeuwen and Maas 2011). Also the age of the father was considered, but adding this variable did not significantly improve the model fit, nor did it have significant effects, so it was dropped from further analysis.

3.3.6. On death clustering in Antwerp

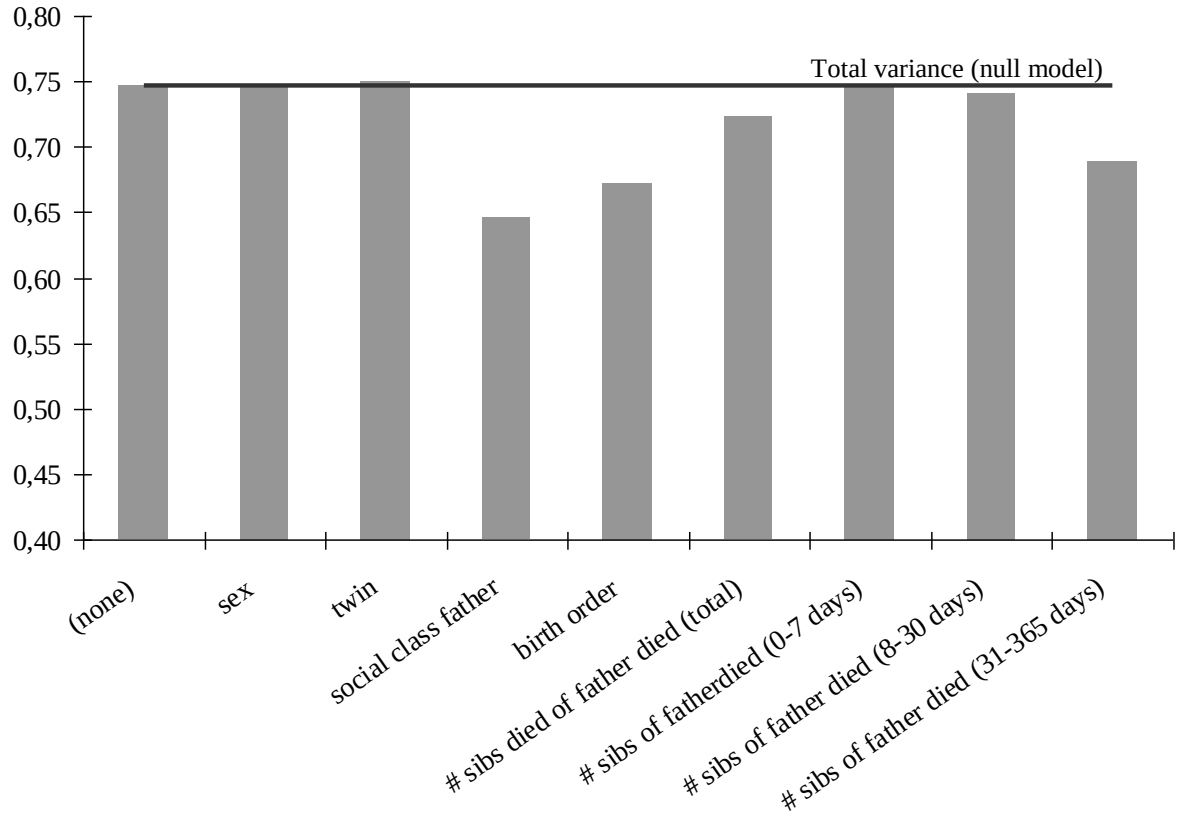
Table 3.3.3. Clustering of age-specific infant deaths, Antwerp sample, 1846-1905

age interval	# of observations	# of fathers	random intercept variance	p
infancy (0-365 days)	5.064	408	0,689	<0,001
perinatal (0-7 days)	1.756	405	4,561	<0,001
late neonatal (8-30 days)	1.674	400	0,582	0,191
postneonatal (31-365 days)	1.634	399	0,829	<0,001

Before turning to the multivariate analyses, first the role of death clustering within the Antwerp district is assessed. Therefore mortality risks were modelled in the multilevel PCE

framework, but without covariates, which yields the results shown in table 3.3.3. The number of observations refers to the number of age intervals in the analysis. A surviving, non-migrating infant typically has three observations, referring to the three age intervals during which the infant was observed. The random intercept variance gives an idea of the amount of death clustering. If this variance is very likely to differ from zero ($p < 0,05$), then there are significant differences between families in infant mortality. The biggest infant mortality differences between families are found in the perinatal group, which means that perinatal deaths are more clustered in families than late neonatal or postneonatal deaths. First-week mortality is very unequally distributed across families. The value for the late neonatals is not significant, which means that it cannot be concluded that there actually are differences between families for mortality within this age interval. Late neonatal deaths are probably distributed quite randomly. This is not the case with the postneonatal deaths, where there is significant death clustering present, but postneonatal deaths are more equally distributed than perinatal deaths.

Figure 3.3.2. Residual variance in infant mortality between families, given covariates



The random intercept variance can not only be used to confirm the existence of family-level infant mortality differences. It is also a useful tool to directly calculate the relative importance of explanatory variables for the clustering phenomenon. Therefore the random intercept variance of a model with a single explanatory variable is compared to the same variance in the null model, i.e. a model without covariates (which, for all infants, can be found in table 3.3.3). The results are presented in figure 3.3.2. The horizontal line represents the variance of the random intercept from the null model. The bars show the residual variance, after controlling for a single variable. The lower the bar, the more death clustering is explained by the corresponding variable. From this graph it is clear that the explanatory factors reducing most differences between families are 'social class of the father', 'birth order', and 'the amount of siblings of the father that died in the postneonatal period'. The other factors, although important in explaining infant mortality, do not have a tremendous impact on the unequal spread of mortality between families. Figure 3.3.2 suggests that the intergenerational transmission of postneonatal deaths is a strong explanatory factor for the death clustering phenomenon. To understand the nature of this relationship, it is necessary to control for all variables simultaneously in order to rule out a spurious relationship caused by a third, intergenerationally transmitted factor.

3.3.7. The intergenerational transmission of infant mortality risks

Table 3.3.4 shows the results for the models where all age intervals are combined. Four models are presented, which differ in their different combinations of covariates. Models 1 and 3 only include the variables of interest, models 2 and 4 also include controlling variables. In every model the age-specific constants are declining as the child is growing older, which corresponds to our knowledge of the infant mortality curve. For every variable in every model an incidence rate ratio (IRR) are estimated. They stand for an increased risk of dying when larger than one, or a decreased risk of dying when lower than one. In both model 2 and 4 there is a significantly increased mortality risk if the infant is born as a twin, or has a higher birth order. As to the occupation of the father, children from farmers have decreased risks of dying as compared to children from unskilled workers, although this effect is only weakly significant.

As for the variables of interest, if a father had siblings who died during infancy, his own children have a 10 per cent increase in mortality risk, although also this effect is weakly significant. When adding controlling variables, the effect gets weaker, and loses even more significance. To further elaborate on this intergenerational effect, the siblings of the father are

counted in different age intervals. A very significant effect is found with siblings who died postneonatally, while no effect is found with the perinatal or late neonatal siblings. The weak transmission of infant mortality risk appears to be mostly due to a much stronger effect of the siblings of the father that die in the postneonatal period. Per sibling of the father dying after one month of age, the father's own children have a 22 per cent increased mortality risk. Most of this effect stays after adding the controlling variables. This combination of variables, as shown in model 4, explains differences between families the best.

Table 3.3.4. Multivariate results of the effect of father's siblings' survival on infant mortality (model 1-4), Antwerp sample, 1846-1905

	Model 1		Model 2		Model 3		Model 4	
$constant_{0\text{-}7days}$	0,0045		0,0040		0,0045		0,0040	
$constant_{8\text{-}30days}$	0,0010		0,0009		0,0010		0,0009	
$constant_{31\text{-}365days}$	0,0005		0,0005		0,0005		0,0005	
	IRR	p	IRR	p	IRR	p	IRR	p
male (ref.: female)			1,058	0,611			1,055	0,625
twin			2,356	<0,001			2,336	<0,001
birth order			1,074	<0,001			1,074	<0,001
occupation of the father								
- unknown (ref.: unskilled)			0,817	0,303			0,833	0,349
- elite			0,352	0,123			0,354	0,120
- middle class			0,673	0,100			0,707	0,146
- skilled labourers			0,667	0,155			0,699	0,203
- farmers			0,659	0,093			0,649	0,082
- low skilled labourers			0,727	0,213			0,778	0,324
# of siblings of father that died								
- during infancy	1,104	0,067	1,097	0,079				
- perinatal period (0-7 days)					0,930	0,613	0,917	0,544
- late neonatal period (8-30 days)					0,687	0,176	0,766	0,331
- postneonatal period (31-365 days)					1,227	0,005	1,211	0,007
group level variance	0,657		0,508		0,602		0,464	
Log Likelihood	-1.851,54		-1.829,24		-1.848,93		-1.827,17	
p_{model}	<0,001		<0,001		<0,001		<0,001	
N observations	5.064		5.064		5.064		5.064	
N fathers	408		408		408		408	

Infant death risks thus seem to be transmitted across generations along the father's pedigree, but only if those deaths occurred to infants older than one month. As a consequence of the PCE approach this affects children at all age intervals equally. This assumption can be too strong, and the fact that the effects were small and not always significant can be an indicator of this. Therefore the next step is to check whether postneonatal deaths among a father's siblings affects all his children, or only those that survive their first month as well. Therefore

separate models were run for every age interval the observed infants are in. Tables 3.3.5, 3.3.6 and 3.3.7 show the results of the models for both the perinatal group (models 5- 8), the late neonatal group (models 9-12) and the postneonatal group (models 13-16), respectively. To make the results comparable to the ones in table 3.3.4, the same covariates were kept for all age intervals.

Table 3.3.5. Multivariate results of the effect of father's siblings' survival on perinatal mortality, Antwerp sample, 1846-1905

	perinatal interval (0-7 days)							
	Model 5		Model 6		Model 7		Model 8	
	IRR	p	IRR	p	IRR	p	IRR	p
male (ref.: female)			1,039	0,891			1,032	0,909
twin			2,987	0,004			3,088	0,004
birth order			1,093	0,061			1,093	0,063
occupation of the father								
- unknown (ref.: unskilled)			0,444	0,163			0,452	0,181
- elite								
- middle class			0,899	0,853			0,879	0,826
- skilled labourers			0,289	0,152			0,284	0,156
- farmers			0,397	0,228			0,394	0,224
- low skilled labourers			0,866	0,809			0,838	0,775
# of siblings of father that died								
- during infancy	0,838	0,282	0,819	0,242				
- perinatal period (0-7 days)					1,154	0,716	1,184	0,693
- late neonatal period (8-30 days)					0,575	0,533	0,754	0,760
- postneonatal period (31-365 days)					0,775	0,267	0,723	0,182
group level variance	4,409		3,637		4,423		3,907	
Log likelihood	-542,11		-532,45		-541,66		-532,01	
p (model)	0,282		0,040		0,589		0,087	
N observations	1.756		1.756		1.756		1.756	
N fathers	405		405		405		405	

Postneonatal mortality risks (results are found in table 3.3.7) are very clearly aggravated by being born as a twin or having a higher birth order. The sex of the child and the occupational status of the father do not yield significant results, besides a weakly significant positive effect of middle class children as compared to unskilled worker's children in model 14. As to the intergenerational transmission of infant risks, postneonatal mortality is found to be positively correlated with infant mortality in the father's family of origin (model 13 and 14). As expected, this is caused by siblings of the father who die after the age of one month (model 15 and 16). Postneonatal mortality in the family of origin is thus transmitted to the next

generation, increasing postneonatal mortality risks among the children of a father. Postneonatal mortality is connected between generations.

Table 3.3.6. Multivariate results of the effect of father's siblings' survival on late neonatal mortality, Antwerp sample, 1846-1905

	late neonatal interval (8-31 days)							
	Model 9		Model 10		Model 11		Model 12	
	IRR	p	IRR	p	IRR	p	IRR	p
male (ref.: female)			0,932	0,824			0,941	0,847
twin			1,328	0,613			1,360	0,579
birth order			1,031	0,566			1,034	0,528
occupation of the father								
- unknown (ref.: unskilled)			0,285	0,043			0,283	0,041
- elite			1,129	0,908			1,099	0,927
- middle class			0,660	0,454			0,803	0,691
- skilled labourers			0,965	0,948			1,168	0,780
- farmers			0,610	0,373			0,509	0,218
- low skilled labourers			0,377	0,192			0,422	0,247
# of siblings of father that died								
- during infancy	1,208	0,09	1,192	0,105				
- perinatal period (0-7 days)					0,453	0,105	0,401	0,065
- late neonatal period (8-30 d)					0,783	0,680	0,936	0,911
- postneonatal period (31-365 days)					1,557	0,001	1,562	0,001
group level variance	0,516		0,229		0,146		< 0,001	
Log likelihood	-262,33		-258,34		-258,45		-253,97	
p (model)	0,090		0,459		0,007		0,066	
N observations	1.674		1.674		1.674		1.674	
N fathers	400		400		400		400	

The same approach is applied to the other age intervals of the reference children. Perinatal and late neonatal mortality among the siblings of the father were not found to influence infant mortality among his children. Are perinatal and late neonatal mortality maybe only transmitted to the perinatal, respectively the late neonatal age interval? These results are found in tables 3.3.5 and 3.3.6. According to the p-value of the models, not every combination of variables is a good fit for this data, especially not when studying only perinatal or late neonatal mortality. For example in the case of the perinatal age interval model 5 and 7 are not considered a good representation of the effects at play in the data. This can be caused by the lower number of observations, but also because it is, using the described advanced methodology, not mathematically possible to find a clear relationship between the infant mortality risks when the reference child is in the perinatal period. There is no significant

relation between infant deaths among the father's siblings, and perinatal mortality risks of the reference children. Counting the father's siblings according to their age intervals does not produce clearer results, nor does it make model 8 a significant improvement over model 6. This additional observation suggests the latter explanation, namely the absence of an intergenerational correlation in the perinatal period.

Table 3.3.7. Multivariate results of the effect of father's siblings' survival on postneonatal mortality, Antwerp sample, 1846-1905

	postneonatal interval (31-365 days)							
	Model 13		Model 14		Model 15		Model 16	
	IRR	p	IRR	p	IRR	p	IRR	p
male (ref.: female)			1,095	0,487			1,090	0,510
twin			2,513	< 0,001			2,487	< 0,001
birth order			1,081	0,001			1,080	0,001
occupation of the father								
- unknown (ref.: unskilled)			1,027	0,904			1,054	0,809
- elite			0,304	0,142			0,309	0,144
- middle class			0,615	0,090			0,647	0,129
- skilled labourers			0,715	0,310			0,742	0,363
- farmers			0,717	0,238			0,712	0,232
- low skilled labourers			0,688	0,215			0,747	0,335
# of siblings of father that died								
- during infancy	1,153	0,018	1,144	0,023				
- perinatal period (0-7 days)					0,985	0,927	0,995	0,975
- late neonatal period (8-30 d)					0,686	0,229	0,742	0,345
- postneonatal period (31-365 days)					1,284	0,002	1,257	0,005
group level variance	0,778		0,617		0,721		0,581	
Log likelihood	-1.037,29		-1.019,37		-1.035,03		-1.017,72	
p (model)	0,018		< 0,001		0,016		< 0,001	
N observations	1.634		1.634		1.634		1.634	
N fathers	399		399		399		399	

As to late neonatal mortality, a similar model fit problem arises as with perinatal mortality. Since model 10, containing infant deaths among the father's siblings and control variables, is not a good fit, the focus goes directly to the age-specific deaths in the father's family of origin. A very significant and very strong correlation is found between postneotal deaths in the previous generation and late neonatal mortality. Siblings of the father dying after one month increase the mortality risk of all children of the father from the age of one week onwards. An unexpected effect is found with perinatal deaths: if the siblings of the father die very soon after birth, his own children have a decreased risk of dying in the late neonatal

period. Although the effect is only weakly significant, it is remarkable nonetheless. Perinatal mortality risks do seem to be intergenerationally transmitted, although only to late neonatal (and not perinatal) mortality in the second generation, and in a contrary manner to what was expected: they increase late neonatal survival chances. There seems to be some positive selection in the perinatal age category. A possible explanation is that there is some kind of biological selection in the family of origin. If children survive the perinatal period in a family with an increased risk of deaths in that age category, they might be biologically stronger than their deceased siblings. When they become adult fathers with children of their own, their children might therefore have higher chances to survive their first month. While postneonatal deaths in a paternal pedigree increase a child's postneonatal mortality risks, perinatal deaths in these previous generations on the other hand decrease early childhood mortality. This however does not cause any visible differences between families in late neonatal mortality (extremely low variance of the random intercept), not in model 10, nor in the total sample (see above, table 3.3.3). Perhaps interfamilial mortality differences are neutralized in the light of the opposite effects of perinatal and postneonatal deaths in the father's family of origin.

3.3.8. Conclusion and discussion

The starting point of this study was fathers in the past and their role in the survival of their offspring. The literature is rather one-sided when discussing fathers and young age mortality. The focus is either on the distress caused by the loss of a father, or on the use of his occupation to position the whole family in a certain social class. Their actual role in mediating mortality risks is difficult to measure, especially for historical populations, since we lack clear indicators, and it is often entwined with the more important impact of the mother.

This study suggests a new, alternative approach to the study of the father-child relationship. This approach focuses on a specific paternal input that can be differentiated from the investments of the mother: his pedigree. Using concepts (high-risk families) and methodology (multilevel approach) of death clustering, patterns of infant mortality were studied between generations, and this along the paternal line. If mortality patterns persist from one generation to the other, this was perceived as an intergenerational transmission of mortality risk. The father is thus seen as a potential mortality risk transmitter.

This research found mortality to be transmitted through generations along the paternal line. Deaths in a family were unprecedentedly shown to affect the mortality risks of offspring of the surviving (male) children. In-depth, age-specific analyses revealed that this is mainly caused by an intergenerational correlation of deaths in the postneonatal period (one month

after birth and older). When a father had siblings who died during infancy, but after having survived their first month, his own offspring had higher death risks in the same age period. If his siblings died right after birth however, his children had strongly increased chances of surviving their first month.

Questions can be raised on how this transmission took place. No definite answers can be given, at least not with this research. However, our knowledge of age-specific mortality can point out some possible directions. Although there is no clear-cut distinction, purely biological causes of death (e.g. genetic weaknesses affecting birth weight and overall health, malformations, etcetera) tend to affect children very soon after birth, mostly in the very first weeks. Other, external causes such as parental neglect, insufficient care, lack of intensive breastfeeding, nutrition based on undiluted cow milk, etcetera, degrade health more slowly, which in some cases leads to death, often after the infant struggled several weeks for its survival. The main complication with these determinants of young age mortality is that they are not exclusively, yet dominantly, bound to certain age categories. Parental neglect can cause infants to die after several days already, while biological weaknesses can result in death only after several months (such as the sudden infant death syndrome). Moreover, the 'nature' and the 'nurture' aspects of mortality can interact, until they are indistinguishable from each other. Genes can cause infants to be less resilient to health shocks, caused by the lack or absence of good childcare. Nonetheless, given some caution, mortality patterns which are persistent in one age category, but are nonexistent in another, can be attributed to aspects of either 'nature' or 'nurture'. These aspects, when transmitted across generations, form the basis of a transmission of infant deaths between generations.

Infant deaths were found to be transmitted across generations through the father. A father who sees his siblings die very soon after birth, has children which are strong enough to have an above average chance to survive their first month, which points to a biological positive selection. While something is causing his siblings to die early on, the father transmits a certain resistance to this risk, which makes his own children stronger. This resistance is not transmitted by fathers who saw their siblings survive the first month. The observation of the correlated postneonatal deaths points to a role of the father in transmitting the factors causing postneonatal deaths. The most common factor of postneonatal deaths is the lack of sufficient breastfeeding. Obviously the father has no direct role in this. It might however be possible that he took part in the decision on how to raise the child, or when and where the child could be breastfed. In the context of 19th century Catholic Antwerp, where the church had a growing impact on private life, women were discouraged to breastfeed their children in public

(Meurkens 1984). Did the father take part in this normative discussion on a family level? This paper's results cannot confirm this. However, his role in transmitting postneonatal infant mortality opposes the proposition that gender division was so strong that fathers had no affinity with childcare.

Although it remains difficult to shed light on how fathers affected the survival chances of their infants, this paper's quantitative evidence suggests that they indeed had a role of their own, aside from the mother: fathers were mediating infant mortality risks between one generation and the next. A deeper study of this intergenerational transmission is required to disentangle possible pathways of how this transmission might work. We hope this study provides the basic fundaments for a whole set of new questions. Is the intergenerational transmission of behaviour stronger in some social strata than in others? Does the age of the father at the birth of his children play a role in the intergenerational transmission of genes? And are some children more likely to mediate intergenerational transmission than others? Does sex and birth order play a role in this?

3.4

MEASURING DEATH CLUSTERING

Comparing and explaining the spread of infant mortality between families in nineteenth century Belgium and Sweden*

Mattijs Vandezande & Prof. dr. Sören Edvinsson**

A previous version of this paper has been presented at:
36th Meeting of the Social Science History Conference, Boston (MA), 17-20 November 2011

*The authors thank Henrik Holmberg and Göran Broström for advice and comments, Henrik Holmberg for providing a copy of his dissertation, and the researchers and programmers at the Demographic Data Base for extracting and interpreting the data retrieval.

**Centre of Population Studies, Umeå University, Sweden

3.4.1. Death clustering, parental competence and the intergenerational transmission of death risks

In 1990 Monica Das Gupta published her now often quoted paper on child mortality in rural Punjab, India. What made the paper so noticed, is her observation on the spread of child deaths across families. Deaths have a strong tendency to occur in a limited number of families. Later, these families have become known as "high-risk families" (Edvinsson et al. 2005). In her 1990 paper Das Gupta already cited examples of several other developing regions where the existence of clustering was suggested. Her paper was followed by many others, who found evidence of death clustering in several regions of the developing world, such as – among others – Mali (Hill & Aguirre 1990), Guatemala (Guo 1993), Brazil (Curtis et al. 1993), Bangladesh (Saha & van Soest 2011; Zenger 1993), and Kenya (Zaba & David 1996; Omariba et al. 2008), as well as in populations of the past, such as Sweden (Edvinsson et al. 2005; Lynch & Greenhouse 1994), Belgium (Alter, Oris & Broström 2001) or the Netherlands (Janssens, Messelink & Need 2010). All of these studies found a similar disproportionate spread of child or infant deaths among families, and several of them have put forward possible causes. So far, no full explanation of the phenomenon is agreed upon. However, there seems to be some kind of general awareness that clustering is very important for the study of child mortality, since it might alter our understandings of the factors determining child deaths.

Much of the effort so far has gone towards explaining what makes families with many infant deaths so different than others. As Das Gupta already suggested in 1990, and confirmed in her later study of 1997, the diverse degrees of individual parental (in)competence probably lies at the basis of clustering. "'Incompetent' parents […]", according to Das Gupta, "[…] give their children poorer care, are slower to recognize and respond effectively to their needs, and consequently lose children" (p. 505). While education and income might improve personal parental abilities, a considerable variation in parental skills remains, especially in the lower social strata (Das Gupta 1997). While seemingly plausible, a hypothesis like this is difficult to prove, especially in historical populations.

In order to shed light on this issue, we argue that an intergenerational view might be very useful, especially for populations where little background information is known. The risk of losing many infants might be inherited through socialization. Hygiene-related behaviour, such as washing hands, and traditions and practices of childcare, such as the amount of breastfeeding, are learned from one generation to the other. If parents lose their infants because of their lack of childcare competence, the surviving offspring might acquire such

'faulty' behaviour through socialization, affecting the survival chances of their own children. Such an intergenerational view might give clues about unobserved behaviours, although the interpretation is difficult because of the possible simultaneous effect of specific genetic setups, weakening the resilience of infants to health shocks.

Our previous findings for 19th century Antwerp show that infant death risks are transmitted from one generation to the next. If the proportion of infant losses in a family is high, the proportion of infant losses in the following generation is likely to be high as well. Conversely, if all infants in a family survive, infants in the next generation have higher chances of survival as well (chapter 3.1). On an individual level, infants had increased mortality risks if there was as least one family member in the previous generation who did not survive infancy. This observation between successive generations was still valid after keeping different kinds of socioeconomic factors constant, both for the maternal (chapter 3.2) and paternal lineage (chapter 3.3). Moreover, age-specific analyses showed that an intergenerational transmission of death risks was focused towards postneonatal deaths (between one and twelve months of age), as caused by, for example, lack of breastfeeding. This made a biological hypothesis, the intergenerational transmission of faulty genes, less likely. If parental competence really lies at the basis of clustering, it might be revealed through its intergenerational transmission.

The question remains whether the intergenerational transmission of infant death risks is an important factor in death clustering. This is addressed in the underlying paper.

3.4.2. Quantifying clustering

While quite some studies, including our own, have suggested possible explanations of clustering, it is impossible to evaluate their impact on clustering without being able to measure clustering. While many studies are successful in proving that deaths are spread disproportionately between families, making statements on the amount of clustering proves more difficult. A popular approach to clustering is to predict an infant's survival chances by the effect of one deceased sibling (e.g. Janssens, Messelink & Need 2010; Lynch & Greenhouse 1994), or even the complete maternity history (Reid 2001). Such an effect indicates a mutual dependency between siblings in survival chances, and therefore suggests the existence of a 'family' effect. However, it is not possible to quantify the family effect with this approach.

A statistically more advanced alternative is to test whether there are differences in infant death risks between families using hierarchical modelling (e.g. Janssens & Pelzer,

2012; Ronsmans 1995; see also chapters 3.2 and 3.3). These multilevel regression models can be used to statistically assess whether significant clustering is present, potentially taking into account significant predictors. In hierarchical modelling the standard deviation of a so-called random (i.e. family) effect is estimated. This yields a quantifiable measure of clustering, interpretable as the heterogeneity between families. However, its size depends on the model setup: whether one applies logistic, Poisson or Cox regression (for a theoretical description: see Goldstein 1995). Therefore results from one study cannot easily be compared with results from another study, which impedes comparing the extent of clustering in two different (regional or temporal) settings. Also, it remains difficult to interpret the results in terms of 'large' or 'little' clustering.

In order to overcome the lack of tools to measure clustering, Holmberg & Broström (2012) propose a method to quantify clustering in terms of the distance between the expected (under the hypothesis of no clustering) and observed number of families with a given number of deaths. Their method (from here on called the 'H&B method') allows to precisely measure clustering before and after inclusion of covariates, independent from sample size, allowing to quantify the impact of covariates on clustering. Moreover, they claim that their method, which they tested on a remote parish in northern Sweden, can be applied to any region or period, and the results can be compared directly. The main strength of their method therefore lies in its ability to describe regional differences or temporal change in clustering.

In the underlying paper we apply the new H&B method to assess the impact of an intergenerational transmission of death risks on clustering, controlling for other covariates such as socio-economic status, age of the mother, and family size. We do this for three different regions at once: (1) the Antwerp district (Belgium); (2) some industrializing parishes around the town of Sundsvall (Sweden); and (3) a group of rural parishes in the northernmost part of Sweden, populated by a mix of Swedish settlers and the Sámi, a group of indigenous semi-nomads. While (1) and (2) are examples of on-going urbanization and industrialization processes, (3) is a society with two strong (intergenerational) traditions: one of native semi-nomads, and one of colonist Swedes.

3.4.3. Introduction to the data

The first dataset is retrieved from the Belgian "Antwerp COR*-database" (Matthijs & Moreels 2010), a database built and maintained by the research group Family and Population from the Centre of Population Studies, Catholic University of Leuven. This database is based on information of population registers ("Bevolkingsregisters") and vital registration records

("akten der Burgerlijke Stand"), including certificates of birth, marriage and death. The sources accessed for data construction span nearly seven decades (1846-1920). The database comprises the Antwerp district: the city of Antwerp, an industrializing and modernising world port, its suburbs, and the surrounding countryside. The whole area is no larger than 1.000 square kilometres (600 square miles), but with over 500.000 inhabitants in 1900 it is a very densely populated one. After the collapse of the local textile industry, Antwerp focused on developing its regional harbour into a world port, invigorating both commercial activity and demographic expansion. As a result, Antwerp's population grew explosively, mainly due to massive immigration (Kruithof 1964; Loyen, Buyst & Devos 2003; Winter 2009).

From this area a 0,4 per cent name sample was taken of all people living within the area and the research period, having a surname beginning with C-O-R, or being directly kin-related to a person with such a name (Matthijs & Moreels 2010; Van Baelen 2007). This yielded about 30,000 individuals, whose life course was followed for as long as they stayed within the regional (Antwerp district) and temporal (1846-1920) frame of the study.

The dataset extracted from the database for the purpose of this paper limits itself to infants, born in the 1846-1905 period, of whom we have the full reproductive life course of the mother. Additionally, in order to be able to conduct intergenerational analyses, we did not allow children whose mother had no siblings. An important bias in the retrieved data therefore is that only those families were selected of which two successive generations lived within the larger Antwerp area. The resulting dataset consists of 1.826 children, born in 376 families.

The Antwerp data is compared to digitized data from the Demografiska Databasen at Umeå University (DDB, http://www.ddb.umu.se/), a huge historical repository of Swedish life courses, the largest database of its kind in Europe, and one of the oldest in the world. More importantly, the quality of their source material is similar to the 19th-century Belgian sources: both are very detailed, and very rich. The database is based on church records – which are basically catechetical registers on a household level ("husförhörlängder") – enriched with registers of baptisms, marriages and burials (for a description of these sources, see Nilsdotter Jeub 1993). Whereas in Belgium the sources were maintained by state (or city government) officials, and from 1846 onwards, the Swedish sources were maintained by parish ministers, and often date much further back in time. No sample was taken, but for several parishes all people were collected. For this paper data was extracted from the so-called POPUM3 database. The datasets used here cover a number of parishes from two different regions. First, we have chosen three out of eighteen parishes in the Sundsvall region (SUL), because of good data quality in these localities and because these parishes industrialized, presenting an

interesting population mix. A fourth parish in the region (Timrå) also becomes heavily industrialized, but was excluded because of problems with the source material in the middle of the 19th century. Secondly, we chose some parishes in the most extreme north of the country, so-called Northern Inland parishes (NIL). Maps are found in appendix.

Sundsvall is a region named after a small town, mainly known for its sawmill economy, located in the central east of Sweden (approx. 400 km north of Stockholm). The selected parishes lie in (the isle of Alnö) or around (Njurunda, Skön) the estuaries of the Indal river (*Indalälven*) and the Ljungan river (*Ljunganälven*), together forming a natural harbour in the Gulf of Bothnia. These parishes have the perfect location for a sawmill industry: logs which are cut deep in the inland forest and, floating eastwards on the river, are processed at the coast in the vicinity of a natural sea harbour. From the middle of the nineteenth century onwards the first industrial (steam-driven) sawmills are installed, first in Skön (1849), later many other places around the Indal and Ljungan estuaries. The region rapidly transforms from a couple of small agricultural villages into an industrializing migrant attraction pole, leading to an enormous population boost its population from about 12.000 in 1860 to 50.000 in 1900, only comparable to some very expansive areas in the nineteenth century United States (Alm Stenflo 1994; Tedebrand 1993). The Sundsvall area became "most dynamic and fastest growing industrial area in northern Europe" (Brändström & Ericsson 1995: 254-255). The large sudden migrant influx resulted in overcrowding, and the local doctors constantly reported a lack of hygiene, endangering healthy living conditions (Edvinsson & Nilsson 2000).

In contrast to this thriving coastal centre of economic activity, the Northern Inland parishes are characterized by their vastness but also their remoteness. These parishes (from north to south: Karesuando, Jukkasjärvi, Gällivare and Jokkmokk) cover the whole western part of Sweden's extreme north ("Norra Norrland"). The region is separated from Norway in the west by a chain of mountains, but the landscape from west to east gradually transforms in lowlands. Lying almost completely within the Arctic Circle, the climate is unforgiving, with very short summers, and long and harsh winters. Population is extremely scarce, and is located in a small number of villages in the east of each parish. Also registered at these parishes are the indigenous Sámi nomads, reindeer hunters who follow their herds in a seasonal cycle from the mountains in the west in summer, to the eastern coast in winter. Some of them settle down, mostly during wintertime, while others turn into fishermen for major parts of the year. Although the Sámi originally differ from the Swedes in economic lifestyle, cultural practices, language and religion, 'colonisation' already took place for more than two

centuries, resulting among others in the conversion of all inhabitants of Sweden to the Swedish Protestant Church. Partly due to the enforcement and closure of the national borders (especially with Norway in the northwest), a growing number of Sámi gave up their traditional lifestyles to choose a permanent residence, sometimes intermarrying with the Swedish colonists (Axelsson & Sköld 2006; Lantto 2010). At the end of the nineteenth century, the discovery of iron ore and the subsequent opening of large iron mines – first in Gällivare, later in the more northern Kiruna (in Jukkasjärvi) – started a constant flow of immigrants coming from more southern regions. This population flow was encouraged and sustained by a rapidly constructed railroad connecting this region to the port of Luleå at the east coast, the *Iron Ore Line* or *Malmbanan* (Sköld & Axelsson 2008; Warg 2002)

To this Swedish data, similar restrictions as in Antwerp were applied in order to retrieve a dataset usable for analysis (full reproductive history of two successive generations, mother should have at least one sibling). Overall, data was obtained for 7 parishes belonging to one of the two regions (see table 3.4.1). The infants were born roughly in the period 1790-1894, although the years differ from one region to the other (e.g. data from Sundsvall started more or less one generation later, mainly because the registration focuses on the 19th century, and because of issues with data quality in earlier periods). The operation yielded a total of 6.370 infants, born in 1.534 families. A quarter of them were born in the Sundsvall region (1.689), the rest was born in the Northern Inland region (4.681).

Table 3.4.1. Parishes retrieved from the DDB

Region/Parish	**Birth years**	**Infants**	**Families**
Antwerp district (ANTW)	1846 – 1905	1.826	376
Sundsvall region (SUL)			
Alnö	1835 – 1893	442	119
Njurunda	1845 – 1891	999	297
Skön/Skönsmo	1839 – 1892	248	74
Norrlands Inlandregion (NIL)			
Gällivare	1790 – 1903	1.118	271
Jokkmokk	1804 – 1895	1.276	320
Jukkasjärvi	1817 – 1900	1.985	373
Karesuando	1816 – 1894	302	80

Two out of three regions, one Swedish (Sundsvall) and the Belgian one (Antwerp), are named after their largest and most influential parish or community (although in the case of Sundsvall the town of Sundsvall, a commercial trade centre, did not meet the data quality standards and

therefore is not included in the SUL-dataset). In the following text the name of that parish or community will be used for all parishes or communities selected from those regions.

3.4.4. The spread of infant mortality in Belgium and Sweden

An overview of the infant mortality in all regions is given in table 3.4.2. In order to overcome possible differences in the (under)registration of Antwerp stillbirths, the definition of infant mortality used in this paper includes stillborn children as well. The infant mortality rate mentioned here is therefore calculated as the number of deaths before the age of twelve months (stillbirths included), divided by the number of all births (both live and stillbirths). This registration issue is mainly acute in Antwerp, in the Swedish parishes the distinction between stillbirths and early infant mortality is less of a problem. To enhance comparability, we used the same above definition to all regions.

Infant mortality differs only little between the selected Swedish regions and Antwerp, but it is highest in the Swedish regions. Both have an infant mortality rate of about 175 deaths per 1000 live births. Especially for the less developed Northern Inland this figure is relatively low. An explanation that has been put forward in the Swedish literature is the relatively low impact of smallpox, due to a combination of the geographical isolation and the nomadic lifestyle of the Sami, allowing them to literally flee the disease (Sköld 1997).

In order to get some idea of the spread of these infant deaths over families, we followed a common strategy. We assessed the proportion of families that did not experience a single infant death, given a certain level of mortality. These numbers of given in the second part of table 3.4.2. A closer, comparative look reveals some interesting patterns. Although the mortality levels in two Swedish regions are similar, in the Sundsvall region infant mortality occurs in a much smaller proportion of the families (36 versus 48 per cent). This means that the Sundsvall region has a larger proportion of families that are (infant) mortality-free, suggesting deaths are more clustered in the Sundsvall area than in the Northern Inlands. Also, the proportion of mortality-free families in Antwerp holds the middle between that of the Northern Inlands and the Sundsvall region. However, the levels of infant mortality in Antwerp are the lowest of all regions. Moreover, one would expect that if the overall mortality rate increases, more and more families experience infant losses. The Sundsvall region disproves this expectation, having almost the highest mortality rate, but infant losses occur in the lowest proportion of families. Again, this suggests that deaths are clustered the most in Sundsvall.

Although this kind of suggestive figures is almost a constant in many papers on the spread of mortality within families, they are essentially meaningless for the study of death clustering, at least without information on the underlying distribution of family sizes. The more children there are in a family, the higher the risk that there will at least be one infant death. If fertility is high in a population, and family sizes are large, families without deaths will be more rare than in a low-fertility population, where family sizes are small. To be able to make statements on the level of clustering in a population, information on both the infant mortality rate and the distribution of family sizes is necessary. This reasoning is explicitly described in Derosas and Broström (2010), and is also present in Holmberg and Broström (2012). They argue that clear statements on clustering can only be made if the spread of infant mortality between families in a population is compared with what is mathematically expected for that given population, if there would be no clustering. The mathematical expectations are based on a series of so-called Bernoulli trials, where the number of trials stands for the number of children in a family. As such, underlying fertility patterns are taken into account. The larger the difference between the actual infant death spread, and the expected spread in an ideal world without clustering, the more important clustering is. Basically that means they created an indirect measure, not putting a value on clustering itself, but on how important clustering is.

Table 3.4.2. The spread of mortality in Antwerp and two Swedish regions

	Antwerp	Northern Inlands	Sundsvall
births	1.826	4.681	1.689
infant deaths*	300	828	295
mortality rate**	164	176	175
families	376	1.044	490
- with deaths	161	497	174
% of total	43 %	48 %	36 %
- without deaths	215	547	316
% of total	57 %	52 %	64 %

* stillbirths included
** calculated as the number of deaths (stillbirths included) per 1.000 births (live births *and* stillbirths)

3.4.5. Measuring clustering

3.4.5.1. The Binomial density function

Before it can be calculated how many mortality-free families can be expected, we need to assume an ideal world in which there is no clustering present, where infant deaths are distributed randomly across families. In such a world, infant deaths in a certain family follow a binomial distribution, with parameters *n* (number of trials) and *p* (overall chance of success). Using the binomial density function, the chance of a given number of successes can be calculated.[11]

In order to make this clear, we can illustrate this using the parallel of a dice game. Imagine an experiment where one needs to throw five dices, and try to throw as much sixes as possible. The number of trials, in stochastic theory, here stands for the number of dices that are thrown: five. The overall chance of throwing a six is, usually, one out of six. With this information it is possible to calculate the chance of, let's say, no sixes at all, which is 0,40. This means that if you would try throwing all five dices 100 times, on average 40 throws would contain no sixes at all.

The same stochastic theory applies to infant deaths in families. The chance of having no infant deaths in a family of five children, given an overall mortality rate in the population of one out of six (16 per cent), is 0,40. This means that if such a population would contain 100 families with five children, on average 40 families would have no infant deaths. At least, this would be the case under the assumption of no clustering. Similarly, an expected number of families can be calculated with families of all possible sizes, every time using the same overall mortality rate. This would result in an expected proportion of all families which is mortality-free, a number which varies depending on the distribution of family sizes in the population.

In reality, infant mortality is not comparable to a dice game, because some families are more at risk to lose children during infancy then others. They are so because of their environment, their childcare practices, their hygienic behaviour, even their genetic predispositions. Therefore there is a difference between the (observed) proportion of

[11] The number of infant deaths *K* 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 0,164 in the Antwerp case) is given by the probability mass function *f*:

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

mortality-free families in a population, and the proportion one would expect, given there is no clustering. The difference between this expected and the observed proportion of mortality-free families is an indicator of clustering. The bigger the difference between observed and expected mortality-free families, the more clustering in the population.

This methodology is put into practice here with the Swedish and Belgian regions. Firstly, information on the distribution of number of births in those regions is necessary. This information is shown in figure 3.4.1. The figure shows large regional differences in the distribution of the population according to the number of births in a family. Sundsvall contains relatively many small families, with almost half of the families (26 per cent + 18 per cent) giving birth to only one or two children. Antwerp has a large proportion of families with four births or less, but also a substantial number of families which are very large (10+ children). The number of births per family in the Northern Inland parishes are moderately large as well, with quite some families gave birth to 5, 6, 7 or 8 children. A more detailed description of the differences in reproductive behaviour in the three regions would lead us too far. Although the 'number of children (ever) born in a family' is definitely inadequate to grasp the differences in fertility regimes between the three regions, figure 3.4.1 provides the necessary information for the calculations on how infant deaths can be spread across families.

Figure 3.4.1. Distribution of families by number of births, by region

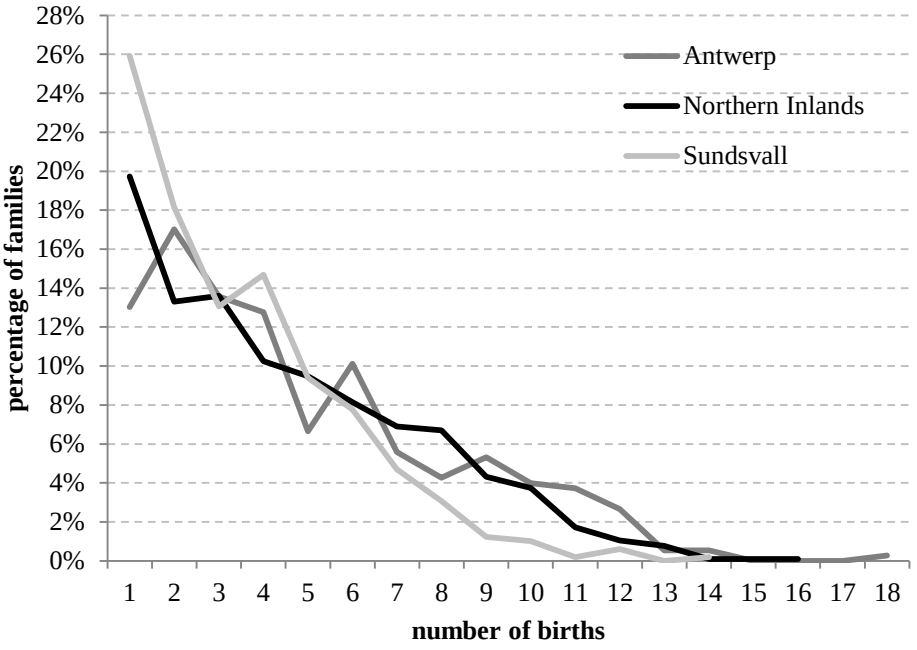


Table 3.4.3. Observed versus expected percentage of mortality-free families, for three regions

	Antwerp	Northern Inlands	Sundsvall
births	1.826	4.681	1.689
infant deaths*	300	828	295
mortality rate**	164	176	175
% mortality-free families (obs)	57 %	52 %	64 %
% mortality-free families (exp)	48 %	48 %	56 %
difference	9	4	8

* stillbirths included;
** calculated as the number of deaths (stillbirths included) per 1.000 births (live births *and* stillbirths).

The information from figure 3.4.1 is used to calculate an expected proportion of families that is without infant deaths (table 3.4.3). If there is an excess of mortality-free families (compared to what can be expected), there must be less families *with* infant deaths than expected to make up for this deficit. In other words: infant deaths are clustered in a (too) small number of families. We knew already that all three regions contain a large proportion of mortality-free families. Table 3.4.3 shows that these families are more common than one could expect, and this for all three regions. Contrary to what was suggested in table 3.4.2, death clustering is most distinct in Antwerp, closely followed by Sundsvall clustering. The 57 per cent of Antwerp families that is mortality-free, is 9 per cent points more than expected. In Sundsvall there are 8 per cent points more mortality-free families than expected (64 per cent). While the Northern Inlands have the highest mortality rate and Antwerp has the lowest, we can expect an equal proportion of mortality-free families in both regions. The actual proportion of mortality-free families in the Northern Inlands follows our expectations more closely than in Antwerp, with a difference of only 4 per cent points. Our intuitive interpretations of clustering based on table 3.4.2 were wrong. This illustrates the importance of the underlying fertility patterns, or more specifically, the distribution of family sizes (size meaning: number of births), in these regions.

3.4.5.2. The H&B method

Holmberg and Broström's method (2012) builds further on this concept of a no-clustering assumption. When deaths are assumed to be spread 'equally' across families (as in: they are not clustered), not only the proportion of mortality-free families can be calculated, but also the proportion of families with one, two or more infant deaths, using the same calculation as in 3.4.5.1. We did this for all three regions: the observed (1) and expected (2) percentage of

families with a specific number of deaths for Antwerp, the Northern Inlands and Sundsvall are shown in table 3.4.4, 3.4.5 and 3.4.6 respectively. When the difference (3) between observed and expected is positive, there is an excess of such families; when the difference is negative such families are less frequent than expected. There are indeed less families with one, two and three infant deaths than expected. However, in both the cases of 'no deaths' and 'many deaths', i.e. 4 and more (and in the Sundsvall region even 3 or more), they are much more frequent than expected. Tables 3.4.4 (Antwerp), 3.4.5 (Northern Inlands) and 3.4.6 (Sundsvall) thus show that in all regions infant deaths are distributed very unequally across families. Clustering is caused by an excess of two kinds of families, namely (a) families with no infant deaths, and (b) families with many infant deaths. In the Sundsvall region the latter type of family is even over four times more observed than expected (see (4) in table 3.4.6). In general, and as a consequence, if parents did lose one of their infants, they were very likely to lose some more.

Table 3.4.4. Clustering of infant deaths in families in Antwerp

deaths	observed (1) *(in %)*	expected (2) *(in %)*	difference (3) = (1) - (2)	proportional diff. (4) = (3) / (2)
0	57,18	48,30	8,88	+ 18 %
1	24,20	32,21	-8,01	- 25 %
2	10,11	13,14	-3,03	- 23 %
3	3,99	4,56	-0,57	- 13 %
4+	4,52	1,78	2,74	+ 154 %
(absolute) total	100,00	100,00%	**23,23**	

Table 3.4.5. Clustering of infant deaths in families in the Northern Inlands

deaths	observed (1) *(in %)*	expected (2) *(in %)*	difference (3) = (1) - (2)	proportional diff. (4) = (3) / (2)
0	52,39	48,50	3,89	+ 8 %
1	28,54	32,07	-3,53	- 11 %
2	11,59	13,19	-1,60	- 12 %
3	4,41	4,58	-0,17	- 4 %
4+	3,07	1,67	1,40	+ 84 %
(absolute) total	100,00	100,00%	**10,59**	

Table 3.4.6. Clustering of infant deaths in families in the Sundsvall region

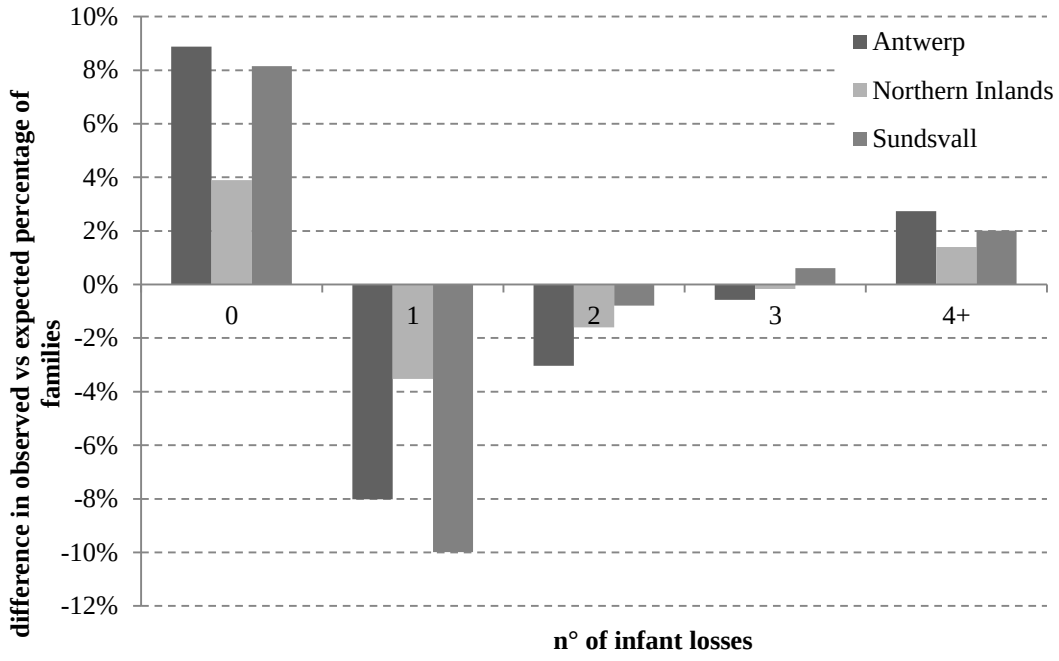
deaths	observed (1) *(in %)*	expected (2) *(in %)*	difference (3) = (1) - (2)	proportional diff. (4) = (3) / (2)
0	64,49	56,33	8,16	+ 14 %
1	21,02	31,01	-9,99	- 32 %
2	8,78	9,56	-0,78	- 8 %
3	3,06	2,45	0,61	+ 25 %
4+	2,65	0,64	2,01	+ 315 %
(absolute) total	100,00	100,00%	**21,55**	

The sum of the absolute values of the differences (3), bold in tables 3.4.4, 3.4.5 and 3.4.6, totals all deviations of the expected proportions from the observed proportions of families. It is a measure of clustering that can be compared across regions or periods. Antwerp appears to have the largest clustering (23,23), closely followed by the Sundsvall region (21,55). In the Northern Inlands there is only half as much clustering (10,59) than in the other two regions.

An interregional comparison of the differences (3) between observed and expected from tables 3.4.4, 3.4.5 and 3.4.6 is easier interpreted when visualized, see figure 3.4.2. The x-axis stands for what is expected. The interpretation stays, so a positive value means there are more such families than expected, a negative value means these families are underrepresented.

Figure 3.4.2 shows that clustering has a very similar shape in all three regions. There are always more families without infant deaths than expected, and there are always more families with 4 or more deaths than expected. This excess of families is mostly compensated by an underrepresentation of the families with one (and to a lesser extent, two infant deaths). Families with three infant deaths are about as frequent as expected, especially as compared to families with another number of infant deaths. Remind that, due to the used methodology, these graphs take into account the different distribution of family sizes in the three regions. It is very remarkable that the nature and shape of clustering is almost identical in all regions, despite the huge geographical and cultural distance between them (e.g. between modernizing Antwerp city dwellers, and a traditional semi-nomadic population like the Sámi). This observation strengthens the presumption that clustering is a universal phenomenon.

Figure 3.4.2. The amount of clustering in three different regions



Only the extent of clustering differ across populations, as is shown in tables 3.4.4, 3.4.5 and 3.4.6. The origins of this difference can be easily derived from figure 3.4.2. The differences in clustering between the three regions are represented in every category, except for families with 'one' or 'two' infant deaths. In those categories Sundsvall has a larger deficit of families with one infant death, at the cost of its share of families with two infant deaths. The excess of families with none and with 4 or more infant deaths in Sundsvall is not compensated by a deficit of families with two infant deaths, as this proportion is very close to what is expected This means that in the Sundsvall region the gap between families with 'few' infant deaths, and families with 'many' infant deaths is less wide than in the other regions. Again, this is not caused by a lower average of family sizes in Sundsvall, since this is taken into account.

3.4.6. Clustering and the intergenerational transmission of infant mortality risks

3.4.6.1. Predicting family-specific mortality rates

The added value of the H&B method extends beyond the comparability of clustering between regions. It is also a tool to test the impact of determinants of mortality on clustering, or – stated otherwise – a way to explain clustering. Holmberg & Broström (2012) tested for birth order, in order to invalidate the proposition that clustering results from the strong relationship between fertility and mortality. Firstly, the death of an infant facilitates the birth of the next

child, due to the hypothesis of a replacement effect, or simply the cessation of lactation (Preston 1978; Reher & Sanz-Gimeno 2007; Van Poppel et al. 2012). Since the reproductive period is limited in time, in the light of clustering this would mean that a very large family size cannot be reached without a certain number of infant deaths shortening the birth intervals. Typically, according to this hypothesis, in very large families there almost always would be some infant deaths. Secondly, the more children in the household, the more competition for resources, especially in families with limited resources available (the poorest families). The larger the household, the lower the survival chances during infancy due to an effect of crowding (e.g. Knodel & Hermalin 1984). Thirdly, some authors suggest the existence of a 'scarring effect'. The loss of several infants can hurt, or 'scar' the mother, resulting in an increased mortality risk for subsequent children (Arulampalam & Bhalotra 2006; Saha & van Soest 2011). Also here, this hypothesis based on the birth history of (older) mothers focuses on very large families, where the survival chances are diminished for the later-born children. By recalculating the expected proportion of families with a number of infant deaths, controlling for birth order, Holmberg & Broström showed that these hypotheses indeed play a role in clustering, but as a whole it is of minor importance.

Here we apply the same method to test for the impact of the intergenerational transmission of deaths. It starts from the approach used in 3.4.5.2, except for the value of the overall mortality rate *p*. Instead a new value is calculated for every single family, based on how the overall risk of infant mortality for that particular family is altered, given certain characteristics, for example its genealogical history of infant deaths. Variables measuring these characteristics were used in standard (Poisson) regression techniques to predict the specific mortality rate for every individual family[12]. The next step, calculating the expected proportion of families with a given number of infant deaths, gets computationally very intensive, especially in the case of families with two or more deaths. Therefore, as Holmberg and Broström suggest, simulation techniques were used instead of the analytically complex elaboration of the binomial density function. Under a binomial distribution $Bin(n_i,p_i)$, in every family i with a certain family size n_i and a family-specific mortality rate p_i, a 'random' number of deaths was simulated. This was done 10.000 times, and for every family the resulting averages were taken, creating an expected distribution of infant deaths in all

[12] A rate can be predicted using Poisson regression to predict a count (e.g. the number of infant deaths), divided by the number of trials (e.g. the number of births in the family) or exposed time (Agresti 2002). This was done in Stata 12 using the `Poisson` command to predict 'number of infant deaths' in a family, with the `exposure` option for 'number of births' in that family (Long & Freese 2006).

families. Using such a large number of replications yielded results very close to the ones obtained analytically (i.e. using the formula of the binomial density function). The result is a renewed version of tables 3.4.4, 3.4.5 and 3.4.6 with new expected values. In this paper we apply this step to get an idea of the impact of the intergenerational transmission of infant deaths on clustering, controlling for other possible candidates of explaining clustering. Therefore this step is divided in two phases: a first phase in which we try to explain clustering with only some controlling variables, and a second phase in which we extend the models from the first phase with the variables of interest: the count of infant deaths in the previous generation. The following two sections discuss the variables and the model setup, and display the results, of the two phases respectively. These results will be compared and discussed in a third section.

3.4.6.2. Phase one: a first attempt to explain clustering

In this phase Poisson models are run containing the following controlling variables (analogous to chapter 3.1 of this dissertation):

- *Age of the mother*. Calculated once per family, at the birth of the first child. This variable indicates the start of the reproductive phase. The variable is centered around the mean.
- *Occupation of the father*. Occupational titles for Antwerp are obtained from the marriage certificate, or if missing, from the birth certificates of the children. For the Swedish parishes they are taken from the birth and catechetical registers. They are coded into three social classes: 'higher and middle class', 'workers', and 'farmers'. A fourth category 'unknown' contains those families in which either no occupation was registered, not on the marriage certificate, and not on one of birth certificates of the children, or in case there was no known father recognizing the children.
- *Number of births in the family*. Despite the fact that the distribution of family sizes is taken into account in calculating the total amount of clustering, it still might have an impact. Not because the more births in a family, the more infant might die, because this is exactly what is taken into account. Larger families might also lose more children than smaller families due to the existence of a scarring effect, effects of crowding, or shortened birth intervals (see 3.4.5.2.), which is why this variable needs to be included in the model. The variable contains the categories '0', '1', '2', '3', '4', '5', and '6 or more' births in the family.

To enhance the comparability of the variable effects in the three regions, no regional differentiation is made within each region, as was the case with the Antwerp district in chapter 3.1. The results from the regression analyses are found in table 3.4.14 (see appendix). The regression analyses lead to new expected proportions of families with a given number of infant deaths, which are given in tables 3.4.7 (Antwerp), 3.4.8 (the Northern Inlands) and 3.4.9 (the Sundsvall region).

Table 3.4.7. Remaining clustering of infant deaths in families in Antwerp

deaths	observed (1) *(in %)*	expected (2) *(in %)*	difference (3) = (1) - (2)	proportional diff. (4) = (3) / (2)
0	57,18	51,94	5,24	+ 10 %
1	24,20	28,35	-4,15	- 15 %
2	10,11	11,97	-1,86	- 16 %
3	3,99	4,90	-0,91	- 19 %
4+	4,52	2,84	1,68	+ 59 %
(absolute) total	100,00	100,00%	**13,84**	

Table 3.4.8. Remaining clustering of infant deaths in families in the Northern Inlands

deaths	observed (1) *(in %)*	expected (2) *(in %)*	difference (3) = (1) - (2)	proportional diff. (4) = (3) / (2)
0	52,39	48,47	3,92	+ 8 %
1	28,54	32,26	-3,72	- 12 %
2	11,59	13,03	-1,44	- 11 %
3	4,41	4,50	-0,09	- 2 %
4+	3,07	1,73	1,34	+ 77 %
(absolute) total	100,00	100,00%	**10,51**	

Table 3.4.9. Remaining clustering of infant deaths in families in the Sundsvall region

deaths	observed (1) *(in %)*	expected (2) *(in %)*	difference (3) = (1) - (2)	proportional diff. (4) = (3) / (2)
0	64,49	59,28	5,21	+ 9 %
1	21,02	27,37	-6,35	- 23 %
2	8,78	9,03	-0,25	- 3 %
3	3,06	3,04	0,02	+ 1 %
4+	2,65	1,28	1,37	+ 107 %
(absolute) total	100,00	100,00%	**13,20**	

3.4.6.3. Phase two: introducing variables on the genealogical history of infant mortality

This phase is similar to phase one, we counted the number of infant deaths in the previous generation (along the maternal line) in three variables: the number of perinatal deaths (between 0 and 7 days of age), the number of late neonatal deaths (between 8 and 30 days of age) and the number of postneonatal deaths (between 31 and 365 days of age). The results of this regression are found in table 3.4.15 (see appendix). The recalculated expected proportions of families, resulting from phase two, are given in tables 3.4.10 (Antwerp), 3.4.11 (the Northern Inlands) and 3.4.12 (the Sundsvall region).

Table 3.4.10. Remaining clustering of infant deaths in families in Antwerp

deaths	observed (1) *(in %)*	expected (2) *(in %)*	difference (3) = (1) - (2)	proportional diff. (4) = (3) / (2)
0	57,18	52,31	4,87	+ 9 %
1	24,20	28,27	-4,07	- 14 %
2	10,11	11,70	-1,59	- 14 %
3	3,99	4,71	-0,72	- 15 %
4+	4,52	3,02	1,50	+ 50 %
(absolute) total	100,00	100,00%	**12,75**	

Table 3.4.11. Remaining clustering of infant deaths in families in the Northern Inlands

deaths	observed (1) *(in %)*	expected (2) *(in %)*	difference (3) = (1) - (2)	proportional diff. (4) = (3) / (2)
0	52,39	48,75	3,64	+ 7 %
1	28,54	31,99	-3,45	- 11 %
2	11,59	12,90	-1,31	- 10 %
3	4,41	4,53	-0,12	- 3 %
4+	3,07	1,83	1,24	+ 67 %
(absolute) total	100,00	100,00%	**9,76**	

Table 3.4.12. Remaining clustering of infant deaths in families in the Sundsvall region

deaths	observed (1) *(in %)*	expected (2) *(in %)*	difference (3) = (1) - (2)	proportional diff. (4) = (3) / (2)
0	64,49	59,33	5,16	+ 9 %
1	21,02	27,33	-6,31	- 23 %
2	8,78	9,01	-0,23	- 3 %
3	3,06	3,01	0,05	+ 2 %
4+	2,65	1,32	1,34	+ 101 %
(absolute) total	100,00	100,00%	**13,09**	

3.4.6.4. Explaining clustering: summarizing results

In the previous sections we three times calculated the expected proportion of families with a given number of infant deaths: (1) using the overall mortality rate; (2) using a family-specific mortality rate based on the variables 'age of the mother', 'number of births' and 'occupation of the father'; and (3) using a family-specific mortality rate based on the three previous variables and three variables measuring the genealogical history of infant mortality. This whole operation was aimed towards reducing the differences (3) between what we observe and what can be expected. The results from tables 3.4.4 to 3.4.12 are summarized in table 3.4.13, and figures 3.4.3 (Antwerp), 3.4.4 (the Northern Inlands) and 3.4.5 (the Sundsvall region).

Table 3.4.13. Total and explained clustering in three regions

	Total clustering	Unexplained clustering (1)	Unexplained clustering (2)
Antwerp	23,23	13,84	12,75
	100,0 %	*59,6 %*	*54,9 %*
the Northern Inlands	10,59	10,51	9,76
	100,0 %	*99,2 %*	*92,2 %*
the Sundsvall region	21,55	13,20	13,09
	100,0 %	*61,3 %*	*60,7 %*

(1) Clustering remaining after controlling for the variables 'mother's age', 'number of births' and 'occupation of the father';
(2) Clustering remaining after controlling for the variables 'mother's age', 'number of births', 'occupation of the father', and the intergenerational variables 'number of perinatal/late neonatal/postneonatal deaths in the previous generation.

The mentioned variables were quite successful in explaining clustering in both Antwerp and the Sundsvall region. In both regions about 40 per cent of all clustering was explained after

including the variables ‘mother’s age’, ‘number of births’ and ‘occupation of the father’ (from 23,23 to 13,84 and from 21,55 to 13,20 respectively). For the Sundsvall region this was sufficient to explain the deficit of families with exactly two infant deaths, and the excess of families with exactly three infant deaths (figure 3.4.5). The amount of clustering in the Northern Inlands is hardly reduced (from 10,59 to 10,51). While in Antwerp the intergenerational transmission of infant deaths explained an additional 5 per cent of clustering (from 13,84 to 12,75), and in the Northern Inlands even 7 per cent (from 10,51 to 9,76), in Sundsvall it barely makes a difference (an additional 0,6 per cent is explained). Even after including all these variables, the remaining unexplained clustering in Antwerp and the Sundsvall region (12,75 and 13,09, respectively) is close to, but still larger than the total amount of clustering present in the Northern Inlands (10,59), indicating that familial differences in infant mortality are still bigger in Antwerp and the Sundsvall region than in the Northern Inlands.

Figure 3.4.3. Explained clustering in Antwerp

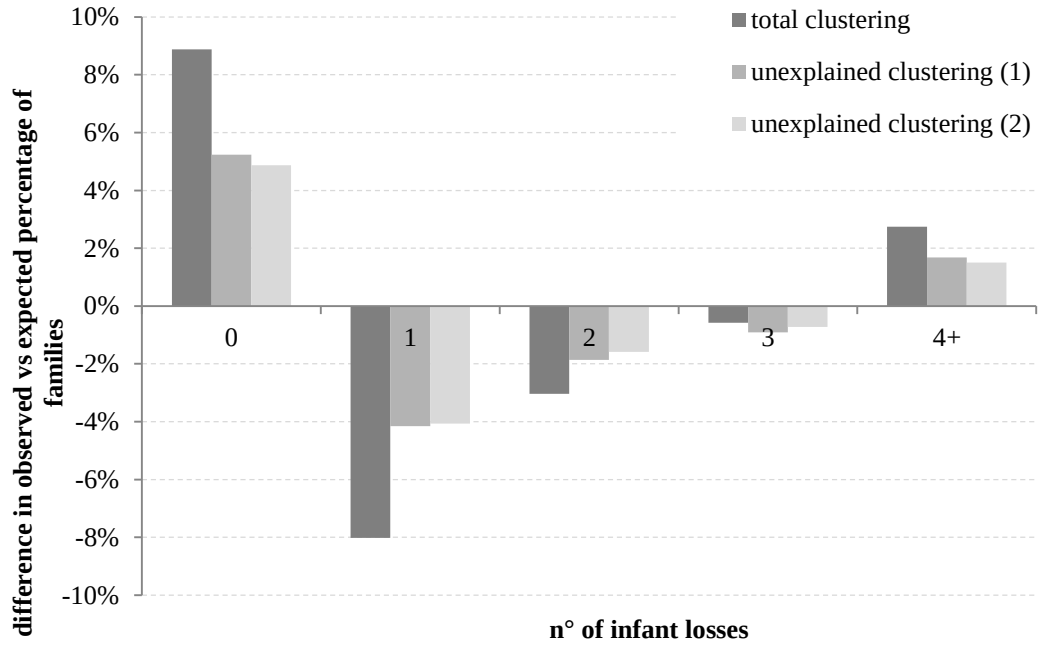


Figure 3.4.4. Explained clustering in the Northern Inlands

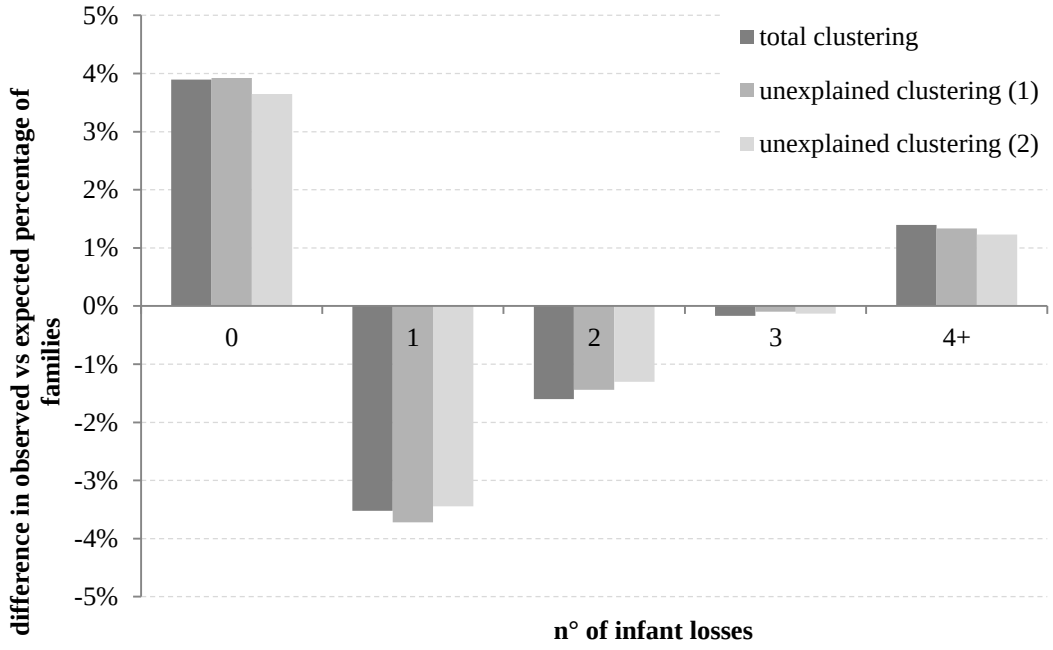
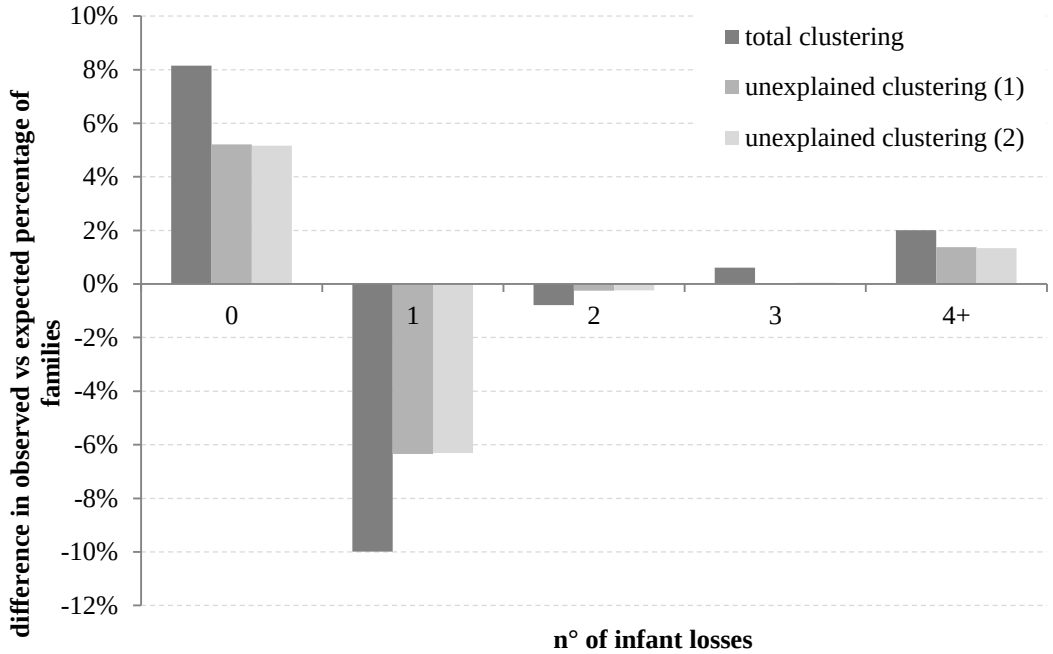


Figure 3.4.5. Explaining clustering in the Sundsvall region



In all regions there is still a considerable amount of unexplained clustering left. While there are large interregional differences in the total amount of clustering, we succeeded in explaining most of these differences. In all three regions the remaining clustering is almost equally large.

3.4.7. Conclusion

One of the most recent advances in the study of death clustering is the development of a method which allows to quantify clustering, independent from time or place, and to test the role of mortality predictors in causing an unequal spread of infant deaths across families. Holmberg & Broström first proposed this method (dubbed the 'H&B method') in 2010, and illustrated it using the effect of birth order on infant death clustering in a northern-Swedish town. We applied this prototype method for the first time on several regions simultaneously: the Belgian district of Antwerp, the Swedish Northern Inlands and the Swedish region of Sundsvall. All three investigated regions are under observation in roughly the same time period (second half of the nineteenth century), a period in which they differ from one another in many aspects, including processes of urbanization, industrialization and economic development, demographic regime, cultural and religious practices, and environmental and epidemiological conditions. In these regions, we apply the H&B method to test for the effect of a genealogical history of infant mortality on death clustering, controlling for several other variables.

As for clustering, we found infant deaths in Antwerp and in the Sundsvall region to be more unequally spread across families than in the Northern Inlands. We also found this regional difference to be independent from underlying local aspects of reproduction, such as the diversity in family sizes due to number of births.

One of the main advantages of the H&B method is the possibility to visualize clustering, due to its breakdown of the population into subgroups of families with a given number of deaths. In a study on 19th century Skellefteå (Sweden), Holmberg and Broström illustrated that families tended to have either no infant deaths, or a lot of infant deaths. Families with one or two infant deaths are therefore underrepresented. We confirmed this overall pattern in all three examined regions. Introducing additional proportional differences revealed that especially families with 4 or more deaths were massively overrepresented in all three regions. We did find some local variation in the occurrence of families with three infant deaths, but in general this particular group of families deviated relatively little from what was expected. Combined with the findings of Holmberg and Broström, and given the enormous

diversity between the tested regions, this suggests that the visualized pattern of clustering might be universal.

Another strength of the H&B method lies in its ability to quantify the explanatory power of factors causing clustering. We tested the number of births in a family, the age of the mother at the start of reproduction, and the occupation of the father in the family. A combination of these factors explains about 40 per cent of the clustering in both the Sundsvall region and in the Antwerp district. At first, we were unsuccessful in explaining clustering in the Northern Inlands. Not surprisingly, as this region is less diverse in social structure (no real upper class, most inhabitants are either reindeer hunters, or small subsistence farmers, especially in the decades before the discovery of iron ore), as opposed to the Antwerp district (containing both a city and countryside, with both upper class and a quite substantial proportion of lower middle class), and the Sundsvall region (inhabited by freeholding farmers and sawmill workers). Apparently also the age at which women start having children and the number of children they have do not result in large differences in infant mortality between families, since these variables do not explain much clustering either. Indeed, deaths are clustered more in one region than in the other because the determinants of mortality vary more from family to family in on region than in the other.

A family's genealogical history explained some additional clustering in both Antwerp (5 per cent) and in the Northern Inlands (7 per cent), making it a distinguishing aspect in those populations, creating substantial differences in infant mortality between families. This is not the case in the Sundsvall region, nor do we find any evidence of an intergenerational transmission in this area. We believe the difference of an intergenerational transmission of death risks between Antwerp on the one hand, and in the Sundsvall region on the other hand, lies in their history of immigration. In the studied period, Antwerp is a migrant attraction pole for many generations, sufficiently long to create lineages of both natives and second- (and higher) generation migrants with distinct traditions towards childcare, hence the heterogeneity between families in the effect of their genealogical history. The population boost due to immigration in the Sundsvall region is relatively recent, and much more explosive than in Antwerp, making the bias towards a stable population (i.e. those families that are in the region since at least two generations) larger in Sundsvall than in Antwerp. The sudden economic expansion attracts many poor families from more southern areas, seeking their luck in this new area of opportunities. This creates an environment in which intergenerational family ties might be corroded, which is why we did not find any effect of infant deaths in the previous generation for both infant mortality and infant mortality clustering in the Sundsvall families.

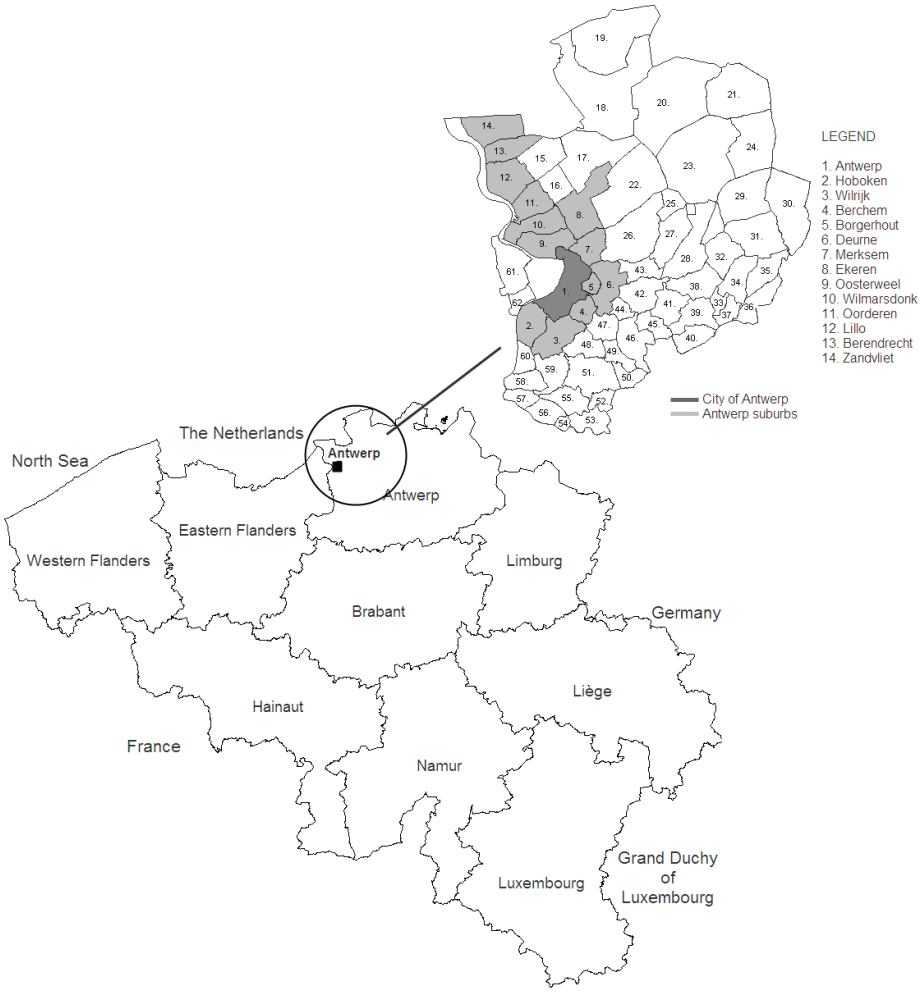
Although the Northern Inlands appear quite a homogeneous region as well, with only a small but hard to explain amount of death clustering in families, the intergenerational transmission of death risks creates substantial differences between families. We believe this difference might be caused by the coexistence of two different cultures in this region, visualized in the data by an intergenerational transmission of cultural traditions. Although Sámi are known to breastfeed their children for a longer time than Swedes, often well beyond infancy, it has been suggested that this effect is countered by the nomadic life style and harsh conditions related to the work year of reindeer herding, explaining higher mortality among Sámi infants (Brändström 1990; Sköld et al. 2011). A possible explanation for our findings is that families with a Sámi lineage could increase the mortality risks of their infants by sticking to the nomadic traditions that are related to Sámi cultural belonging.

Using the above results, we combined the method's interregional comparability and its ability to evaluate determinants of clustering, and found that the variables used in this paper (number of births in a family, mother's start of reproduction, father's occupation, family's genealogy of infant mortality) explain most of the interregional differences. After controlling for these variables, the remaining unexplained clustering is relatively equal in all regions. Whatever the remaining cause that makes infant deaths to cluster in a limited number of families, it is a factor that plays an equal role in all three regions, how different these regions may be, be it socio-economically, demographically, culturally or on an environmental level.

Death clustering has been proven to be an interesting approach to the study of mortality at young ages. Whether it is a worthy alternative to the traditional approach of the individual infant, has yet to be discussed. Up until now, it has been difficult to evaluate the clustering approach, since we lacked the proper tools to characterize the spread of infant deaths between families, nor were we able to fully explain it. Whatever explanations were investigated, there was still some unexplained clustering left. Although a full explanation of clustering is but a distant goal, the H&B method is a major step forward, since it allows to describe and quantify clustering and hence evaluate the impact of mortality determinants on clustering. This was fully impossible with more traditional, complex inferential techniques, such as hierarchical modelling, but the results from this methods could not easily be compared, nor between populations, nor between periods. The H&B method provides a solution to this shortcoming, opening the door for further exploration of death clustering using comparative – interregional, like in this paper, or long-term – approaches.

3.4.8. Appendices

Figure 3.4.6. Map of the Antwerp district within Belgium



Reproduced from: Matthijs & Moreels 2010: 111.

Figure 3.4.7. Map showing the location of the Swedish region of Sundsvall

Reproduced from: Tedebrand 1993: 11.

Figure 3.4.8. Map showing the location of the Northern Inland parishes in the northern parts of Sweden

Source: Törnlund 2011.

Table 3.4.14. Poisson regression of a family's infant mortality rate (number of infant deaths by number of births), predicted by three variables

	Antwerp		Northern Inlands		Sundsvall region	
	β	p	β	p	β	p
constant	-2,506	<0,001	-1,542	<0,001	-2,180	<0,001
age of the mother (centered)	-0,037	0,017	-0,082	0,405	-0,594	<0,001
occupation of the father						
unknown	0,458	0,001	0,246	0,001	0,609	0,007
higher or middle class	0,044	0,819	-0,009	0,969	0,299	0,338
farmer	-0,201	0,478	-0,215	0,133	-0,021	0,900
worker	(ref)		(ref)		(ref)	
family size						
1	(ref)		(ref)		(ref)	
2	0,431	0,387	-0,462	0,028	0,071	0,817
3	0,337	0,494	-0,164	0,357	0,136	0,662
4	-0,233	0,650	-0,336	0,069	0,122	0,678
5	0,791	0,109	-0,311	0,084	0,493	0,087
6 or more	0,532	0,245	-0,257	0,085	0,570	0,030
N		376		1044		490
Log likelihood		-399,23		-1128,51		-453,15
p		<0,001		0,003		<0,001
df		9		9		9
pseudo R^2		0,042		0,011		0,034

Table 3.4.15. Poisson regression of a family's infant mortality rate (number of infant deaths by number of births), predicted by three controlling variables and three 'intergenerational' variables

	Antwerp		Northern Inlands		Sundsvall region	
	β	p	β	p	β	p
constant	-2,657	<0,001	-1,583	<0,001	-2,258	<0,001
age of the mother (centered)	-0,030	0,059	-0,072	0,465	-0,592	<0,001
occupation of the father						
unknown	0,447	0,001	0,236	0,002	0,597	0,009
higher or middle class	0,074	0,704	-0,014	0,949	0,262	0,404
farmer	-0,212	0,457	-0,224	0,121	-0,031	0,852
worker	(ref)		(ref)		(ref)	
family size						
1	(ref)		(ref)		(ref)	
2	0,450	0,368	-0,464	0,027	0,106	0,731
3	0,409	0,409	-0,171	0,337	0,155	0,620
4	-0,183	0,723	-0,351	0,058	0,154	0,606
5	0,837	0,091	-0,301	0,095	0,520	0,075
6 or more	0,618	0,180	-0,281	0,060	0,600	0,024
deaths in the previous generations						
perinatal (0-7 days)	-0,057	0,581	0,077	0,194	0,148	0,212
late neonatal (8-30 days)	0,461	0,013	-0,045	0,444	-0,039	0,790
postneonatal (31-365 days)	0,116	0,025	0,074	0,039	0,057	0,444
N		376		1044		490
Log likelihood		-394,26		-1125,22		-452,07
p		<0,001		0,002		<0,001
df		12		12		12
pseudo R^2		0,054		0,014		0,036

PART FOUR

GENERAL CONCLUSION

Death clustering in families is a relatively recent concept that has drawn a considerable amount of attention of scholars in both contemporary and historical demography. It is a new view on infant mortality, focusing on the family instead of on the individual child. Therefore, it is not about mortality averages, but instead it is all about variation in mortality. It is about heterogeneity, about variation that is normal, and variation that is systematic. Therefore it is a difficult topic to grasp intuitively. Or as a participant to a workshop on death clustering phrased it: "clustering is not about the mean mean, it is about the variability of the variance". It is about what makes infant mortality in one family so different than in the other.

Despite over two decades of research, the study of death clustering is still in its infancy. Death clustering remains insufficiently understood, both as a describable phenomenon, a measurable entity, a process of family-level inequality, and as leverage for a better understanding of, and improved theory on infant mortality. This doctoral dissertation aims to contribute to a better understanding of the unequal distribution of infant deaths between families. It explores one possible cause of infant death clustering: the impact of a family's genealogical past of infant mortality. During this exploration it became apparent that no single method allows to both detect, describe and at the same time explain clustering. As already hinted in the concluding remarks of a recent workshop on 'death clustering in the past' (Umeå, Sweden, Oct 2010), the study of clustering lacks well described appropriate tools. In the main research chapters of this dissertation, the problem of clustering was therefore approached from three different perspectives: *individual* but correlated infant mortality risks (chapters 3.2 and 3.3), the *family* as a unit with few or many infant losses (chapter 3.1) and the distribution of infant deaths between families from the perspective of the *population* (chapter 3.4). This multi-directional approach using both micro (individual), meso (family) and macro (population) levels proved fairly useful to get a better grasp of both the description and the causes of clustering.

4.1. Discussing the results – genealogical history of infant mortality and death clustering

4.1.1. Inherited dimensions of infant mortality

The first and main objective of this study, the 'if' part of the question, is to assess *whether* infant deaths in one generation affect the survival chances of infants in the next generation. This research question is tackled in chapters 3.1, 3.2 and 3.3. While in the former two chapters the previous generation was studied along the maternal lineage only, the latter chapter focused on the paternal lineage. In all three chapters a correlation in infant deaths between two generations was confirmed. Chapter 3.1 highlighted the intergenerational

correlation in both low and high mortality families. Girls who lost a high proportion of their infant siblings were up to two times less likely to see all of their own infants survive infancy. Conversely, if girls grew up in a family where all children survived infancy, as adult mothers they were up to two times less likely to lose a large proportion of infants. Chapter 3.2 allows for a more precise interpretation. Generally speaking, infant mortality risks increased significantly if the infant was born in a family with a genealogical history of infant mortality. These infants saw their mortality risks increase by almost 14 per cent per infant death in the previous generation. This correlation was less visible when considering the previous generation along the paternal lineage (chapter 3.3), with a weakly significant increase in risk of about 10 per cent ($p = 0{,}067$). Infant death risks in two successive generations, be it along the maternal lineage or the paternal lineage, are linked to each other. The first part of the main research question can be answered affirmatively. Some children are more likely to die during infancy then others, and this is related to infant deaths in their family's past. If new-borns had infant deaths in their genealogical family tree, it casted a dark shadow over their own survival chances during infancy. With a little exaggeration and a touch of drama, one could state that some of these children were predestined to die, they were dead on arrival, or even 'born to die'.

The second part of the main objective is to *explain* the relationship between infant deaths in two successive generations, the 'why' part of the question. This is a very difficult, if not impossible goal to reach. Mechanisms of transmission are plenty, often at work simultaneously, and we lack accurate measures to describe and make a distinction between these mechanisms, especially for a population in the past. For example, one's available resources or income is often but approximated by one's occupational title, and whether someone possesses certain unspecified 'faulty' genes can only be guessed at. We know an intergenerational transmission of death risks is possible when there is a persistence in mortality causing factors from one generation to the other. These factors, for example Mosley & Chen's proximate determinants, are very difficult to measure, while others, such as the determinants proposed by Kok et al., are too indirect to fully support or disprove the hypotheses. Still, the results from this study nevertheless allow to partly disentangle some of the mechanisms of intergenerational transmission. This should not be interpreted too optimistically, since the hypothesized mechanisms, as listed and discussed in the first part of this study, can still not be fully tested.

One hypothesis can be discarded: the denominational mechanism. This mechanism is described as the differences between different religious groups, not how religious a family is.

In Antwerp religious differentials in infant mortality are not the cause of the established intergenerational transmitted death risks, simply because there is very little variation in religion in the sampled families. This cannot be derived from the data, as individual religious denomination is not registered in the used historical sources. However, the vast majority of the Antwerp population is Roman Catholic. Along with immigration, some religious minorities are , for example the Jewish community in the city of Antwerp (Saerens 1999). Before 1900 however, they were not numerous, and almost exclusively originating from abroad. Only afterwards they start to build up a genealogical heritage in the Antwerp area. Due to the sample selection criteria, these families were never included in the intergenerational sample. Although the denominational hypothesis might still play a role in the intergenerational transmission of death risks in other populations, differences between religious denomination are of virtually no influence on the results found in this dissertation.

The other hypotheses are not so easily confirmed or rejected. We need to rely on proxies to capture their effect. The role of status (and resource) inheritance and that of environment, for example, is approximated by the occupational status and the region of residence, respectively. Infant mortality risks, both individually and from a family perspective, were not found to differ significantly between social classes, nor between regions. The only exception is the value 'unknown' for father's occupation, which significantly increased mortality risks. Note that this is not a reflection of the higher mortality among children where the father himself is 'unknown', since illegitimates were excluded in the analyses of both chapter 3.2 and 3.3. More likely this reflects a larger stress on families with an unemployed father, a family deprived of the main income. In general however, the results from this study do not allow to make statements on the importance, or even the presence, of either a status inheritance mechanism or a persistent environmental effect.

After controlling for several variables measuring these mechanisms, the effect of deaths in the previous generations on infants' survival chances remained significant, and still moderately strong. Controlling for region and father's social class minimizes the potential impact of intergenerational status transfer or environmental effect on intergenerational transmission, hereby hopefully clarifying the importance of the mechanisms of socialization and biology. To get a clearer view on these mechanisms, chapter 3.2 and 3.3 dig a little deeper in the nature of the transmission. This is done by differentiating infancy in a perinatal period (the first week), a late neonatal period (the remainder of the first month), and a postneonatal period (the remainder of the first year).

Age-specific analyses demonstrated that the (positive) correlation in infant mortality risks between generations consistently starts to show up after the first week of age, for the paternal lineage even only after the first month. Infants have increased risks to die after the first week, or even after the first month, which is related to having relatives in the previous generation who also died after the first week, or after the first month, respectively. Also, the risk of having stillbirths or new-born infant deaths, deaths that are often linked to biological causes, was not found to be transferred from one generation to the other. This finding clearly points in the direction of sole remaining hypothesis: the socialization hypothesis. The nature of the correlation runs counter the reversed socialization hypothesis, which can therefore be discarded. Meanwhile, the faulty genes hypothesis is moved to the background. Can it be discarded, then? The answer to that question is: no. The connection between 'early' and 'late' infant deaths on the one hand, and causes of 'nature' and 'nurture' on the other hand, is not so strong as we would like. Although genetic causes of death manifest themselves often soon after birth, genes play a role in health throughout infancy, just as non-biological factors – conveniently generalized here as 'nurture' – influence health from birth onwards. Even the distinction between 'nature' and 'nurture' is less sharp than we would like: certain genetic predispositions are slumbering until triggered by environmental or social conditions, such as (lack of) hygiene. 'Faulty' genes can work indirectly by making some individuals more susceptible to environmental conditions. The strong presence of an effect between postneonatal deaths in both generations, and the absence of a similar effect between perinatal deaths, should therefore not be overstated. This finding is worth what it is: it carefully hints towards the socialization hypothesis, it vaguely suggests the predominance of nurture over nature, but nothing more than that.

An interestingly different but difficult to interpret result was found in chapter 3.3. An excursion along the lineage of the father revealed a link between early deaths in successive generations which contrasted with all previous findings. If infants died very soon after birth, infants in the next generation were not less, but actually much *more* likely to survive the (late) neonatal period. The death of one aunt or uncle on father's side during his or her first week after birth more than doubled an infant's late neonatal survival chances. Unfortunately, the models were inconclusive regarding the effect on the first week after birth. Also, neonatal deaths in the previous generation were not found to affect survival chances in the next generation, not in any age group. Perinatal deaths were not found to affect perinatal deaths in the next generation, but only (and very significantly) *late* neonatal deaths in the next generation.. According to the theoretical framework, this finding provides some support for

the biological selection hypothesis, but the lack of consistency makes an easy interpretation of the result unlikely. The correlation is only found between one, and not all of the combinations of perinatal and late neonatal deaths in both generations. Still, one could align these findings phrasing that while some infants are born to die, others are born to stay alive, at least during the first weeks after birth.

The above negative correlation between perinatal and late neonatal deaths in two generations was only found along the paternal line, and not along the maternal line. Possibly the effect is less visible along the maternal line, but it also might be less strong or even absent. Unfortunately it is not possible to further elaborate on this: the used datasets do not allow to make statements on the difference in the effects that were found between father's and mother's genealogical history. Chapter 3.2 and chapter 3.3 are based on different (sub)samples: the 332 families in chapter 3.2 are not the same families as the 408 families in chapter 3.3. In order to qualify for selection in these samples, a parent must have a name starting with COR*, so both his or her siblings could be retraced, as well as his or her own children. This means is it not possible to study both lineages (of father and mother) at the same time, as it would require both parents to have a COR* name. Only a handful of families in the COR* database apply to this criterion; too few to form a solid basis for a comparison between the effect of the maternal genealogical history and the effect of the paternal genealogical history.

4.1.2. 'Equal' versus 'unequal' distribution of infant mortality

The second main objective of this dissertation is to explore the meaning of an intergenerational transmission of infant mortality for the clustering phenomenon. It is the next question to ask after the finding that infant deaths are linked between generations. Chapter 3.1 showed that mothers who lost a large proportion of their infant siblings more often lose a large proportion of their own infants, as compared to mothers whose siblings stayed alive. Mothers with a genealogical history of infant mortality lose more infants than mothers without. This implies that infant deaths in the previous generation (in part) cause the difference in infant losses between mothers. As a consequence, the genealogical history of infant mortality must be one of the reasons for clustering, one of the answers to its origin.

The results of the chapter 3.1 provide but indirect evidence that clustering is explained by a family's genealogical evidence. Direct evidence can only be provided by demonstrating that clustering is reduced if the genealogical history is taken into account, which in turn implies that clustering is measurable.

The problem with the approach taken in chapter 3.1 is that the existence of high mortality (and low mortality) families is related to, but not prove of, clustering. Clustering stands for an unequal distribution of infant deaths across families. But what do we understand under 'equal' distribution? 'Equal' does not mean that every family loses a fixed number of infants, but instead that every family has an 'equal' *risk* of losing infants. The more children a mother gives birth to, the more likely she is to lose some. Conversely, the fewer children she gives birth to, the more often she will lose them all during infancy. In fact, there will always be some families in which all infants die, or in which all infants survive. Depending on the overall mortality rate and the number of births in a family, these families will be more or less frequent, ranging from present in large numbers to extremely exceptional. This is a natural consequence of an equal distribution of infant mortality. In statistical terminology: infant deaths spread equally across families are best described under a binomial distribution, with parameters 'overall infant mortality rate' and 'number of births in a family'.

The setup of chapter 3.1 is therefore not the optimal way to study clustering. The weakness of the 'high versus low mortality families' approach is most visible in the sensitivity of both definition and results in this chapter to the number of births in a family. In that chapter it was called an effect of 'operationalization', but statistically it can be perceived as a 'binomial trap': the larger the diversity in the distribution's parameter 'number of births', the greater the impact of its effect (see chapter 3.1 for a more detailed discussion). This is important since it is a fundamental aspect of clustering, causing the most intuitive and tempting approach of 'high-risk' or 'high mortality families' to be unrelated to a good understanding of clustering.

4.1.3. Explaining clustering using hierarchical modelling

One way of dealing with this 'binomial trap' is to leave the level of the family, and return to the level of the individual child. This approach requires a more complex statistical modelling, involving the simultaneous use of event history models (for the probability of survival) and multilevel components (for measuring clustering). Family effects, causing clustering, can be measured in models that estimate the variance between families in individual infant mortality. This variance measures the overall differences between families in infant mortality, or 'total' clustering. Doing so, the focus is shifted from the average infant mortality in a family to the variability of infant mortality from family to family. This approach was taken in chapters 3.2 and 3.3.

Even with this approach the sensitivity of clustering to the number of births in a family is still not absent. Some of the clustering occurs because there are both large and small families. This was solved by including a covariate on the single level. In chapters 3.2 and 3.3 the effect of 'number of births' was captured using the single-level covariate 'number of older siblings'. Since these chapters are restricted to (singleton) infants, the effect of the variable 'number of older siblings' parallels the effect of 'number of births' during the infancy of the reference child.

The chapters using a multilevel event history approach confirm a considerable amount of clustering in the Antwerp families, both in the subsample from chapter 3.2 (332 families) as well as in the subsample from chapter 3.3 (408 families). The measure used in these chapters learns that, in general, one infant death in the family almost doubled the infant mortality risks for all siblings. For infants in the perinatal period this effect was much larger than for infants in the late or postneonatal period. This means there are much larger differences between families in perinatal mortality (and stillbirths), than in late and postneonatal mortality. Perinatal deaths are much more clustered than late and postneonatal deaths.

Along both maternal and paternal lineages, the genealogical history of infant mortality explains some clustering. Infant deaths in the previous generation explain a clearly noticeable, but relatively small part of the differences between families. The impact of an intergenerational transmission of infant deaths partly overlaps with the impact of other variables, such as age of the mother or socio-economic status of the family. However, the majority of its effect remains. This means that although we were able to explain some of the clustering, the intergenerational effect additionally explains a small but distinctive amount of clustering, which was not explained before. Nevertheless, even after taking into account this new cause of clustering, in both subsamples more than two thirds of all clustering remains unexplained.

4.1.4. Re-acquiring the overview through a visualization of clustering

Abandoning the family perspective as in chapters 3.2 and 3.3 provided a solution to the clumsy way of avoiding the 'binomial trap' from chapter 3.1, but while doing so the overview on the spread of infant mortality was lost. Using hierarchical event history modelling an individual is stripped from his or her most important trait in the light of clustering: the number of siblings who died during infancy. This technique predicts survival chances of individual infants, taking into account the possibility of family effects. Whether someone's siblings die

during infancy, and exactly how many of them die as infants, is not visible using this regression technique. Siblings, whose infant mortality risks correlate because of clustering, are treated as completely independent; all their commonalities are absorbed by the random intercept (of which the variance was a measure for clustering). A straightforward answer is to include the loss of the previous sibling as a covariate. However, this creates a new problem: doing so changes the interpretation of the random intercept variance, the measure of clustering. It is still a measure of differences between families, but *given the loss of at least one infant*, meaning it effectively removes part of the clustering. When the goal is to assess the amount of clustering, including information on the survival of the other siblings is a complete no-go. In the most extreme case, including a covariate how many siblings died as infants completely removes all differences between families. In this case the random intercept variance is exactly zero. It is still a measure for the (remaining) differences between families given the model setup, but it is far from a measure for clustering anymore. Information on the survival of siblings was therefore necessarily excluded from all models in chapters 3.2 and 3.3.

Regaining the overview on the number of deaths in a family requires the use of a different methodology. Chapter 3.4 applies a recently developed prototype method, the H&B method, to visualize clustering using an absolute – i.e. comparable – measure. The method is named after the scholars who developed it, Henrik Holmberg and Göran Broström. It starts from the perspective of a population without clustering, taking the number of infants in a family into account by simulating infant deaths in families with a given number of births. The (absolute sum of) differences between simulated and observed proportion of families with a given number of infant deaths creates a new measure of clustering. Gradually allowing covariates to vary in the simulated population leads to a fictional population that increasingly resembles the study population.

This method led to a deeper insight into death clustering among the Antwerp families. Families without infant deaths, and families with many (four or more) infant deaths were largely overrepresented. Families with only one infant death, and to a lesser extent families with two infant deaths, were underrepresented. Broadly put: Antwerp families either had no infant deaths, or a lot of infant deaths. Over 40 per cent of this clustering was explained by (a combination of) the age of the mother (at the start of reproduction), the occupation of the father and the number of births in the family. Adding the genealogical history of the family explains an additional 5 per cent of all clustering.

In disentangling clustering by number of deaths in a family, the above factors mainly make clear why so many families experience no infant deaths, and why so few families only experience one infant death. The excess of families with many infant deaths is relatively less well explained by these factors. This suggests that answers for clustering are easiest to find when not focusing on why some families experience so many infant deaths, but instead, why others succeed in keeping their infants alive. For example, as for the genealogical history of infant mortality in the light of the socialization hypothesis, it is not only 'bad' habits in infant care that are transmitted from mother to daughter, but – maybe more importantly – the good practices. A similar suggestion was found in chapter 3.1, where low mortality families were explained almost twice as easily as high mortality families. It calls for a renewed interest in infant *survival*, as opposed to the mainstream focus in the literature on infant *mortality*.

4.2. Generalizing the results – the Antwerp COR* families as an example?

The above results are based on a select group of families: fathers and mothers, living and giving birth in and around Antwerp. These parents were born in the wider Antwerp area, had at least one sibling, and stayed within the region during the reproductive phase of their life course, a phase completed before 1906. The sample(s) used in this dissertation therefore have a number of characteristics atypical for a completely random sample drawn from the Antwerp population, hampering an easy generalisation of the results. These characteristics are either structural or data-specific.

Structural characteristics are characteristics resulting from the nature of the research question. The main research question, the effect of sibling infant deaths on infant deaths in the next generation, requires a sample containing only families with at least two children in one generation: one needs to be a girl (or a boy for male lineages), survive childhood and give birth to an unspecified number of children, the other is her (or his) sibling influencing infant mortality risks among those children. This means that families without (surviving) children and one-child families will never qualify as research subjects for this research question. The results of the underlying study, as well as of any other future one of this kind, are thus by nature biased towards families with less than extremely high infant mortality (not every infant dies), and a sample's mean number of births that is a little higher than average (since one-child families are excluded). The former issue can sometimes be dealt with, as was aimed for in chapter 3.1 using an adaptation to the definition of what constitutes a 'high infant mortality' in the previous generation. The impact of the latter issue in the case of Antwerp

fortunately is soothed due to the majority of the families stemming from a clearly pre-transitional fertility regime, as is discussed in chapter 3.1.

Data-specific characteristics reduce sample representativeness because of restrictions in the sources or availability of the data. In this study the COR*-database was used. This database is built with a sample of individuals within a certain area (the 62 municipalities forming the Antwerp district). Therefore life courses of immigrants prior to their arrival in the region are unknown, as are life courses of emigrants after their departure. This study furthermore required two successive generations to be present in the region, hereby excluding even more (first-generation) immigrants and families who leave the region. Ideally these families should be backtracked to their origin (in the case of immigrants), or followed to their destination (in the case of emigrants). Unfortunately, this strategy is too time-consuming to be feasible within the context of this study. Moreover, the additional benefit for the sample representativeness would be limited. The COR*-sample not only covers the Antwerp city and its suburbs, localities characterized by strong immigration, but also the wider area of surrounding communities, localities where many of Antwerp's immigrants originate from. The sample thus covers the lives of many migrants, at both origin and destination. Still, the dataset used in this study does not represent international or long-distance migrants. This makes the sample more homogenous (less migrants) than the actual 19th century population, and decreases the diversity in care traditions. If long-distance migrants increase the diversity in childcare traditions, both clustering and the impact of an intergenerational transmission of infant death risks on clustering can be expected to be higher than what is found here. The impact of a family's genealogical history on death clustering is thus probably slightly underestimated because of the bias in the sample towards a more stable population.

Also, because of the (public) availability of the Belgian data sources, only the second half of the nineteenth century is sampled in the COR*-database. Given the requirement of two successive generations, this means that the infants whose lives are at risk in this study are born towards the latter decades of the nineteenth century, decreasing temporal diversity between different families. Since infant mortality remains high throughout the period under investigation, the impact of this temporal bias is less clear.

The results in this dissertation therefore apply to infants born in the latter third of the nineteenth century, with parents and grandparent(s) originating from the wider Antwerp area. They are yet to be confirmed for infants from other periods, or infants from families originating outside the Antwerp district. Although clustering can be expected to be larger in the total population, as well as a family's genealogical history possibly having a larger impact

on clustering, there are no reasons to believe that the results of this study would be fundamentally different, or that its conclusions would change if a different, more representative sample was used.

Aside from widening the sample to other, yet underrepresented groups of the population (e.g. long-distance migrants), further research can also head into other directions. This study is limited to infants. This has the advantage that infants from the same family rarely experience their infancy at the same time, which reduces the effect of instantaneous events, such as accidents, epidemics, etcetera. The results on the postneonatal infants suggest that intergenerational effects extend beyond the age of twelve months as well. However, beyond this age the effect of other, context-bound factors becomes increasingly important, and the theoretical framework requires adaptation. Further research could thus focus on older children as well, but will face additional complexities.

The research questions in this study are investigated with a strict, and limited number of variables. Future, more in-depth research could focus on adding additional factors. A few are suggested here. In chapter 3.3 the potential impact of grandparents was mentioned, and more specifically that of the role of the grandmother on father's side. The COR*-database allows to assess whether that grandmother is still alive while her grandchildren are in their infancy, whether she is present in the household during that period, or even whether she resides in the immediate vicinity of her son and his grandchildren. Other factors are possible as well. Future studies could, for example, take into account individual causes of death. When unavailable, as in Antwerp, more aggregate figures on the occurrence of certain types of causes of death could be used. This study is meant to be exploratory, and discovered but the tip of the proverbial iceberg. Much more studies are needed, exploring additional factors, seeking confirmation in many other populations, both past and present.

4.3. Confirming the results – the Antwerp COR*-families from a comparative perspective

Despite the sample's shortcomings, it is possible to put the Antwerp results in context by comparing them to the results in other populations. Until recently the study of clustering lacked the proper tools to easily compare different populations. Due to the recent development of the H&B method, a new way of analysing and visualizing clustering based on simulation techniques, comparing clustering across regions becomes an intriguing possibility.

In chapter 3.4 clustering in Antwerp is compared with clustering in two regions in Sweden: some industrializing parishes around the town of Sundsvall, and some parishes in the far north of Sweden, the Northern Inlands. Data for these regions stems from the

Demografiska database (DDB), one of the few historical data warehouses with a sufficient number of intergenerationally linked individuals.

In all three regions, Antwerp as well as the two Swedish ones, infant deaths were clustered: there was an excess of families with a lot of infant deaths, and an excess of families without deaths, while families with one or two infant deaths were underrepresented. However, in some regions (Antwerp, Sundsvall) infant deaths were clustered almost twice as much than in others (Northern Inlands). Moreover, the extent of clustering is not directly related to the level of infant mortality: while the Swedish regions have a similar infant mortality rate, infant deaths are much more clustered in the parishes around Sundsvall than in the Northern Inlands. This is because the infant mortality rate is a measure of the average mortality, while clustering reflects the spread of mortality. More clustering in a region means that this region is more diverse in terms of determinants for infant mortality. Less clustering means a region is more homogenous. An unanticipated finding was that the coexistence of two cultures in the Swedish part of the arctic circle, roughly spoken one of Swedish sedentary smallholders and one of Sámi reindeer nomads, as a whole is less diverse than an area in Belgium containing both a fast growing, urbanizing city and the surrounding countryside. One of the possible explanations lies in the different socio-economic stratification of the population, which is much more homogenous in the Northern Inlands than in Antwerp, causing families to differ more from one another in Antwerp than in the Northern Inlands.

Explaining clustering with a limited number of explanatory variables, including a measure for social class, proved to be most fruitful when clustering was large. In fact, the amount of clustering that remained unexplained was very alike in all three regions. This suggests that a larger diversity in mortality determinants, for example larger socio-economic differences, are the very reason for clustering being higher in Antwerp and the Sundsvall region than in the Northern Inlands. It also suggests that there is a ‘fixed’ amount of clustering that is difficult to explain.

Adding an intergenerational component nowhere fully accounts for the remaining, unexplained differences between families. In fact, an intergenerational component is not even found everywhere. In the Sundsvall region no evidence was found of an effect of infant deaths in the previous generation on infant mortality in the next generation, at least not after controlling for covariates. This does not necessarily mean it is absent, only that it is not clearly visible. For the Sundsvall parishes it might be a data-related issue, resulting from the bias towards a stable population: lack of linkage between parishes leads migrants to be much more underrepresented in the Sundsvall region than in Antwerp, where all individuals in all

municipalities are fully linked. There are considerably less non-natives to contrast with in the Sundsvall parishes, but it is unclear if this leads to a decreased visibility of an intergenerational effect. In my opinion it might also be related to the specific Sundsvall historical background. The rapid development of the industrialized parishes in this region, followed by an explosive population boost, might have led to a corrosion of intergenerational family ties, weakening the correlation between infant deaths in two successive generations. It is difficult to point out a definitive explanation, since it remains hard to confirm the mechanisms behind an intergenerational transmission of death risks. Bluntly put: since we don't have a full understanding of what caused an intergenerational transmission, it is difficult to explain why no evidence of such a transmission was found in the Sundsvall parishes.

Contrary to the findings for the Sundsvall parishes, evidence was found in the Northern Inlands, where the effect of infant deaths in the previous generation on clustering was more important than in Antwerp. This might be related to the specific nomadic life style of the Sámi, exacerbating the survival chances of their infants.

Apparently, the previous generation's infant mortality does not matter for every population. When it does, it only explains a relatively small, but additional part of clustering. This means that the genealogical history of infant mortality is not the sole remaining cause for clustering: there are still many other, yet unidentified causes. Infant deaths in the previous generation is just one of them, but one with large regional differences in importance. In some regions it was more important than in Antwerp (Northern Inlands), but in other regions there is no prove that it played any role whatsoever (Sundsvall). What was found for Antwerp, is thus confirmed to exist elsewhere, but not everywhere.

The lack of a consistent, universal effect of infant deaths in the previous generation in all regions suggests that the presence of an intergenerational effect is locally bound. Can this interregional perspective be used to further elucidate upon the mechanisms of an intergenerational transmission, or more specifically, the importance of the socialization hypotheses as opposed to the biology-related hypotheses? The regional diversity, as well as the findings for the Northern Inlands region, suggest mechanisms related to the diversity in local cultures. Family ties can be stronger in one region than in another. However, also gene defects can have strong local variants. If inheritable gene defects are present in a very limited number of families in one locality, it does not only lead to differences in intergenerational transmission between regions, but it also causes deaths to be more clustered in that locality, with an increased importance of the intergenerational component in explaining clustering.

Although an interregional perspective is fascinating, it raises more questions to the nature of an intergenerational transmission than it can solve.

4.4. Learning from the results – death clustering as a new approach for studying infant mortality

The genealogical history of infant mortality is thus one of the causes for the clustering of infant deaths in families. It is not the only cause, nor is it the main one. Nonetheless it plays a limited, yet not unimportant role in the unequal spread of infant deaths between families. For the major part this role is new, and as such has not been considered or tested before. It points to the existence of a number of family traits that are transmitted across generations, including childcare traditions, gene environments, hygiene practices, etcetera. Unfortunately, the methods and measures applied in this dissertation do not allow to fully understand what traits are important, and what traits are not. Consequently, at this point it remains unclear how exactly the correlation of infant deaths and infant death risks between generations should be understood.

Aside from the main research question in this doctoral dissertation, which is the *leitmotiv* in all four chapters in part 3, every one of those chapters has its own starting point. Indirectly, this dissertation is intended as an example of how death clustering, and a focus on the family in general, might be beneficial to our understanding of demographic processes.

Chapter 3.1 starts from the study of reproductive behaviour, and, more specifically, how it is transmitted between generations. In this corner of the field of demography scholars are interested in how reproductive behaviour is determined and diffused, and how that relates to long-term fertility change. Infant (and child) mortality is strongly related to fertility, through the interval between successive births, and in connecting the number of births to an achieved family size. Just like infant mortality is key to get a proper understanding of fertility outcomes, an intergenerational transmission of infant deaths is key to grasping the mechanisms of an intergenerational transmission of reproductive behaviour. Chapter 3.1 therefore points to an additional element that needs to be taken into account when studying inherited dimensions of fertility, using clustering and the family perspective to ease the link between the study of infant mortality and the study of reproduction.

Chapter 3.2 directly connects to the literature on death clustering, in which the mother is often put forward as a cause for familial differences. Adding information on her familial background, her genealogical history of infant mortality, further elucidates on the many

aspects of her role. By involving information measured one generation earlier, the mother is the sole remaining link between that previous generation and her own infants at risk of dying.

Chapter 3.3 starts from a very similar, but mirrored approach, focusing on the role of the father. A father's impact on infant mortality is often reduced to his role as main breadwinner, or even his presence. This chapter reveals that his role in the survival of his infants goes beyond the economic aspect. The family effects on infant mortality are thus not only attributable to the mother or the disposable resources, but also to the father, either directly or indirectly, but independent from the mother. The findings suggest, among others, that there is a biological selection at work, increasing survival chances for children whose father lost siblings close after birth. At the same time postneonatal deaths are transmitted along the paternal lineage. This raises the question whether the socialization hypothesis is also valid in this setup. Was the father influencing his partner's childcare practices with knowledge and experience he socialized through his own mother? Or was his mother directly involved in raising the children of her son? Controlling for her presence, either in the household, or in the neighbourhood of the household, belongs to the future possibilities.

Chapter 3.4 has a methodological aim, in response to the lack of a valid tool to unambiguously measure clustering. It briefly discusses the shortcomings of the most frequently used methods, and applies a recently proposed prototype method, the H&B method, to try out its claim that the method can be used for (regional) comparisons. Since many aspects of clustering are immeasurable, a comparative perspective reveals what aspects can be common, and what aspects are specific to certain regions. Additionally, clustering is made more 'visual' by differentiating between families with a fixed number of deaths. This led to the insight that in all regions – both in the ones studied in this dissertation as in the one which was originally used to test and illustrate the H&B method – clustering had the same appearance. Most families lost either none, or many of their infants.

4.5. Added value of this research – this study as a point of departure for future research

The finding that infant deaths in previous generations are relevant and predictive grants us new angles to study infant mortality. Also, this study lays the foundation for a whole set of future, demographically interesting and sociologically fascinating questions.

An important dimension that is not yet discussed so far is 'time'. 'Time' here stands for a long-term perspective of both an intergenerational infant death risk transmission and of clustering; yet also the relation of these with the overall decline in infant mortality. Throughout this dissertation, the aspect of 'time' was deliberately not incorporated, mainly

because of scope and feasibility arguments. The focus was on the clustering in infant deaths in a limited number of families, and on the role of a family's genealogical history in this clustering. In doing so, the connection between the study of clustering on the one hand, and its meaning for general demographic transition theory on the other hand, remains unclear. This is not only the case in this study. Ever since Monica Das Gupta put death clustering on the research agenda, scholars referred to a 'large potential impact', even 'major implications' of death clustering on our understanding of infant mortality. Despite some careful attempts (for example Das Gupta's suggestions on innate parental capabilities, or Guo's references to genetic elements), there remains a gap between the empirical observation of clustering, and the theoretical link between the family perspective and demographic theory and processes. So far, death clustering remains too ill understood to properly claim revisions of our current theoretical frameworks – such as the one from Mosley and Chen – or to improve our understanding of the mortality decline.

The most promising added value of this study lies in the unique intergenerational perspective in infant mortality. In taking an intergenerational perspective, this study lays the foundation to bridge the gap between the observation of clustering and demographic theory. The next step in bridging this gap is to focus on change over time: change of clustering over time, and long-term changes in the intergenerational transmission of death risks. So far, infant mortality is too often studied on an individual level, while the traditional explanations for the mortality decline – improved knowledge and training of medical practitioners and midwives, increased attention for hygiene, better nourishment, diffusion of breastfeeding – are situated on a higher level, that of the family. Moreover, the decline of infant mortality is not a constant process, but an evolution with accelerations, delays, and constant fluctuations. This is not explained so far, but could very well be related to (changing) family characteristics. An increased attention to the family in a long-term perspective could be an important aspect in understanding the timing and pace of the decline in infant mortality.

The results from this study show that infant mortality is related to infant deaths in the previous generation. The results also suggest that practices and traditions of infant care play a key role in this intergenerational transmission of death risks. It has been argued that infant mortality started to decline when 'bad' care practices and lack of hygiene were improved under the influence of newly acquired medical knowledge and professionalized medical care. Doctors, midwives and other medical practitioners are believed to be important actors in reducing infant mortality. Doing so, they are able to relieve the burden of a genealogical history of infant mortality. As their role increases, and infant mortality starts to decline, the

effect of infant deaths in the previous generation is expected to weaken. Conversely, strong intergenerational ties can impede a family's openness to discard the childcare practices and hygiene customs they are used to, following medical advice. A long-term perspective on changes in this intergenerational component will help us to understand the characteristics of the long term decline of infant mortality, in all its wavering and geographical diversity.

REFERENCES

Agresti, A. (2002). *Categorical data analysis*. Hoboken (NY): Wiley.

Allison, P.D. (1984). *Event history analysis. Regression for longitudinal data.* Quantitative applications in the social sciences, 46. Beverly Hills: Sage.

Alm Stenflo, G. (1994). *Demographic description of the Skellefteå and Sundsvall regions during the 19th century*. Umeå: Umeå University.

Alter, G., M. Oris and G. Broström (2001). The family and mortality: a case study from rural Belgium, *Annales de Démographie Historique* 101(1): 11-31.

Anderton, D.L., N.O. Tsuya, L.L. Bean, and G.P. Mineau (1987). Intergenerational transmission of relative fertility and life course patterns, *Demography* 24: 467-480.

Arulampalam, W. and S. Bhalotra (2006). Sibling death clustering in India: state dependence versus unobserved heterogeneity, *Journal of the Royal Statistical Society Series A* 169(4): 829-848.

Asaert, G. (2007a). *De 25 dagen van Antwerpen: stadsgeschiedenis in 25 delen. Deel 16. 3 mei 1836. De eerste trein naar Brussel. [The 25 days of Antwerp: urban history in 25 volumes. Volume 16. 3th of May 1836. The first train to Brussels]*. Zwolle: Waanders.

Asaert, G. (2007b). *De 25 dagen van Antwerpen: stadsgeschiedenis in 25 delen. Deel 17. 13 mei 1866. Cholera maakt 3.000 doden [The 25 days of Antwerp: urban history in 25 volumes. Volume 17. 13th of May 1866. Cholera kills 3000]*. Zwolle: Waanders.

Asaert, G. (2007c). *De 25 dagen van Antwerpen: stadsgeschiedenis in 25 delen. Deel 18. 19 januari 1873. Red Star Line steekt van wal. [The 25 days of Antwerp: urban history in 25 volumes. Volume 18. 19th of January 1873. The Red Star Line sets sail]*. Zwolle: Waanders.

Axelsson, P. and P. Sköld (2006). Indigenous populations and vulnerability. Characterizing vulnerability in a Sámi context, *Annales de démographie historique* 111(1): 115-132.

Beekink, E., F. van Poppel and A.C. Liefbroer (2002). Parental death and death of the child: common causes of direct effects? In: R. Derosas and M. Oris (eds.), *When dad died. Individuals and families coping with distress in past societies*. Bern: Peter Lang, 233-260.

Bertels, I. (2011). *Building the city, Antwerp, 1819-1880*. Brussels: Academic and Scientific Publishers.

Bhalotra, S. and A. van Soest (2004). Birth spacing, fertility and neonatal mortality in India: dynamics, frailty and fecundity, *Journal of Econometrics* 143(2): 274-290.

Bittles, A., M. Murphy and D. Reher (2008). Inherited dimensions of human populations in the past. *Human Nature* 19: 1-6.

Blake, J. (1981). Family size and the quality of children. *Demography,* 18: 421-442.

Blomme, J. (2003). The Antwerp port: elements of spatial planning. In: R. Loyen and E. Buyst (eds.). *Struggling for Leadership: Antwerp - Rotterdam port competition between 1870-2000*. Heidelberg: Physica-Verlag, 162-168.

Bongaarts, J. and R.G. Potter (1983). *Fertility, biology and behavior. An analysis of the proximate determinants*. New York/London: Academic Press.

Bracke, N. (2008). *Een monument voor het land. Overheidsstatistieken in België, 1795-1870.* Gent: Academia Press.

Brändström, A. (1984). *"De kärlekslösa mödrarna". Spädbarnsdödligheten i Sverige under 1800-talet med särskild hänsyn till Nedertorneå* [The careless mothers. Infant mortality in Sweden in the 1800s with special reference to Nedertorneå]. PhD thesis. Umeå: Umeå Universitet.

Brändström, A. (1988). The impact of female labour conditions on infant mortality: a case study of the parishes of Nedertorneå and Jokkmokk, 1800-96. *Social History of Medicine* 1: 329-358.

Brändström, A. (1990). Från förebild till motbild. Spädbarnsvård och spädbarnsdödlighet i Jokkmokk [From model to counterpart. Infant care and infant mortality in Jokkmokk]. In: S. Åkerman and K. Lundholm (eds.), *Älvdal i Norr. Människor och resurser i Luledalen 1300-1800 [River valley in the North. People and resources in the Luleå valley 1300-1800]*. Umeå studies in the humanities n° 91. Umeå: Umeå University, 307-351.

Brändström, A. and T. Ericsson (1995). Social mobility and social networks. The lower middle class in late nineteenth century Sundsvall. In: A. Brändström and L.-G. Tedebrand (eds.), *Swedish urban demography during industrialization*. Report n° 10 from the Demographic Data Base. Umeå: Umeå University, 251-284.

Britten, N. and A. Heath (1983). Women, men, and social class. In: E. Gamarnikow, D. Morgan, J. Purvis, and D. Taylorson (eds.), *Gender, class, and work.* London: Gower,

46-60.

Bruneel, C. and M. Bruwier (1989). De burgerlijke stand [The vital registration records]. In: E. Van Driessche (ed), *De erfenis van de Franse Revolutie [The heritage of the French Revolution]*. Brussels: ASLK, 107–115.

Chesnais, J.-C. (2000). *The demographic transition: Stages, patterns, and economic implications. A longitudinal study of sixty-seven countries covering the period 1720-1984*. Oxford: Clarendon Press.

Conde-Agudelo, A. and J.M. Belizan (2000). Maternal morbidity and mortality associated with interpregnancy interval: cross sectional study, *British Medical Journal* 321(5): 1255–1259.

(1910). *Cost of living in Belgian towns. Report of an enquiry by the Board of trade into working class rents, housing and retail prices, together with the rates of wages in certain occupations in the principal industrial towns of Belgium, with an introductory memorandum and a comparison of conditions in Belgium and the United Kingdom*. London: Darling & Son.

Curtis, S.L., I. Diamond and J.W. McDonald (1993). Birth interval and family effects on postneonatal mortality in Brazil, *Demography* 30(1): 33–43.

Das Gupta, M. (1990). Death clustering, mother's education and the determinants of child mortality in rural Punjab, India, *Population Studies* 44(3): 489–505.

Das Gupta, M. (1997). Socio-economic status and clustering of child deaths in rural Punjab, *Population Studies* 51(2): 191–202.

Davis, K. and J. Blake (1956). Social structure and fertility: an analytical framework, *Economic Development and Cultural Change* 4, 211-235.

De Caigny, S. (2000). *Het Eilandje: snijpunt van stad en haven. Een studie naar het urbanisatieproces en de sociaal-economische betekenis van de Antwerpse havenwijk, 1830-1914. [The Island: intersection of city and port. A study of the urbanization process and the socio-economic significance of the Antwerp port district]*. KU Leuven: History department (Licentiate's thesis).

Debuisson, M. (2001). The decline of infant mortality in the Belgian districts at the turn of the 20th century, *Belgisch Tijdschrift voor Nieuwste Geschiedenis [Belgian Journal of Contemporary History]*, 31(3-4): 497-527.

De Kesel, J. (1964). Structurele ontwikkeling van de haven (1803-1914) [Structural development of the harbour (1803-1914)]. In: Genootschap voor Antwerpse Geschiedenis [Antwerp History Society] (Ed.), *Bouwstoffen voor de geschiedenis van Antwerpen in de XIXde eeuw [Materials for the history of Antwerp in the 19th century]*. Antwerp: Lloyd Anversois, 124-168.

Derosas, R. and G. Broström (2010). Infant death clustering in nineteenth-century Venice, *Paper presented at the international workshop for death clustering*, Umeå University, Sweden, 22-23 October 2010.

Desama, C. (1991). Le couplage automatique des données nominatives: nouvelles perspectives. In : Société Belge de Démographie (ed.), *Historiens et populations. Liber Amicorum Etienne Hélin*. Louvain-la-Neuve : Academia, 119-126.

Desjardins, B., A. Bideau, E. Heyer and G. Brunet (1991). Intervals between marriage and first birth in mothers and daughters, *Journal of Biosocial Science* 23: 49-54.

Devos, G. (2003). Land-use policy in the port of Antwerp (1870-1994). In: R. Loyen and E. Buyst (eds.). *Struggling for Leadership: Antwerp - Rotterdam port competition between 1870-2000*. Heidelberg: Physica-Verlag, 199-219.

Devos, I. (2001). Selective bibliography of Belgian historical demography, 1981-2001 (19th and early 20th centuries), *Belgisch Tijdschrift voor Nieuwste Geschiedenis* 31(3-4): 347-369.

Devos, I. (2006). *Allemaal beestjes. Mortaliteit en morbiditeit in Vlaanderen, 18de-20ste eeuw. [Little creatures. Mortality and morbidity in Flanders, 18th-20th century]*. Ghent: Academia Press.

Dewey, K. and R. Cohen (2007). Does birth spacing affect maternal or child nutritional status? A systematic literature review, *Maternal and Child Nutrition* 3: 151–173.

Dierickx, H. (1954). *Geschiedenis van Hoboken [History of Hoboken]*. Antwerp: De Sikkel.

Dillon, L. and E. Roberts (2002). Introduction to the special issue: Longitudinal and cross-sectional historical data: intersections and opportunities, *History and Computing* 14(1-2): 1-7.

Dupâquier, J. and D. Kessler (1992). L'enquête des 3000 familles [The investigation of 3000 families]. In: J. Dupâquier and D. Kessler (eds), *La société française au XIXe siècle. Tradition, transition, transformations* [The French society in the nineteenth century.

Tradition, transition, transformations]. Paris: Fayard, 23–61.

Dyson, T. and M. Murphy (1985). The onset of fertility transition, *Population and Development Review* 11(3): 399-440.

Edvinsson, S. and H. Nilsson (2000). Urban mortality in Sweden during the 19th century. In: A. Brändström and L.-G. Tedebrand (eds.). *Population dynamics during industrialization*. Report n° 13 from the Demographic Data Base. Umeå: Umeå University, 39-82.

Edvinsson, S., A. Brändström, J. Rogers and G. Broström (2005). High-risk families: the unequal distribution of infant mortality in nineteenth-century Sweden, *Population Studies* 59(3): 321-337.

Garðarsdóttir, Ó. and L. Guttormson (2009). Public health measures against neonatal tetanus on the island of Vestmannaeyjar (Iceland) during the 19th century, *History of the Family* 14(2): 266-279.

Goldstein, H. (1995). *Multilevel Statistical Models. Second edition*. Kendall's Library of Statistics, 3. New York: John Wiley and Sons.

Gribble, J. (1993). Birth intervals, gestational age, and low birth weight: are the relationships confounded?, *Population Studies* 47(1): 133–146.

Grusky, D.B. (1983). Industrialization and the status attainment process: the thesis of industrialism reconsidered, *American Sociological Review* 48: 494-506.

Guo, G. (1993). Use of sibling data to estimate family mortality effects in Guatemala, *Demography* 30(1): 15–31.

Gutierrez, R.G., S.L. Carter and D.M. Drukker (2001). On boundary-value likelihood-ratio tests, *Stata Technical Bulletin* 60: 15-18.

Guth, G.J.A. (1976). Surname spellings and computerized record linkage, *Historical Methods Newsletter* 11(1): 10-19.

Gutmann, M. and E. Van de Walle (1978). New sources for social and demographic history: the Belgian population registers, *Social Science History* 2(2): 121-143.

Hannes, J. and C. Lis (1969). De sociale hiërarchie in de woningbouw. Antwerpen omstreeks 1834. [The social hierarchy in the construction of housing. Antwerp around 1834], *Belgisch Tijdschrift voor Nieuwste Geschiedenis [Belgian Journal of Contemporary*

History] 1(1): 86-92.

Hannes, J. and D. Soetewey-Campers (1969). De verstedelijking van de gemeenten Deurne en Borgerhout volgens het kadaster (1810-1914) [The urbanization of the municipalities of Deurne and Borgerhout according to the cadastre (1810-1914)], *Tijdschrift van de Belgische Vereniging voor Aardrijkskundige Studies [Journal of the Belgian Society for Geographical Studies]* 38: 129-138.

Hasquin, H., R. Van Uytven and J.-M. Devosquel (1980). Gemeenten van België. Geschiedkundig en Administratief Geografisch Woordenboek [Belgian municipalities. Historical and administrative geographical dictionary]. Brussel: La Renaissance du Livre.

Hawkes, K., J.F. O'Connell and N.G. Blurton Jones (1997). Hazda women's time allocation, offspring provisioning and the evolution of long postmenopausal life spans, *Current Anthropology* 38: 551-578.

Hawkes, K., J.F. O'Connell, N.G. Blurton Jones, H. Alvarez and E. Charnov (1998). Grandmothering, menopause and the evolution of human life histories, *Proceedings of the National Academy of Sciences of the United States of America* 95: 1336-1339.

Hill, A.G. and A. Aguirre (1990). Childhood mortality estimates using the preceding birth technique: some applications and extensions, *Population Studies* 44(2): 317–340.

Hill, B. (1993). Women, work and the census: a problem for historians of women, *History Journal Workshop* 35: 78-94.

Hill, K. (2003). Frameworks for studying the determinants of child survival, *Bulletin of the World Health Organization* 81(2), 138-139.

Hohenwarter, M. (2011). The clustering of infant death in Austrian families during the first demographic transition: a regional comparison of two parishes in Styria, *Romanian Journal of Population Studies* 5(2): 91-113.

Hofstee, E.W. (1983). Geboorten, zuigelingenvoeding en zuigelingensterfte in hun regionale verscheidenheid in de 19de eeuw [Births, infant nourishment and infant mortality in their regional diversity in the 19th century], *Bevolking en Gezin [Population and family]* Suppl. 2: 7-60.

Holford, T.R. (1980). The analysis of rates and survivorship using log-linear models, *Biometrics* 65: 159-165.

Holmberg, H. and G. Broström (2012). Paper 3. On statistical methods for clustering; a case study on infant mortality, northern Sweden 1831-1890. In: H. Holmberg. *Generalized linear models with clustered data*. Doctoral thesis in Statistics (defended on 30th March 2012, Handelshögskolan, Umeå University). Umeå: Umeå University.
Forthcoming in *Biodemography and Social Biology* 58(2).

Hosmer, D.W. and S. Lemeshow (2000). *Applied logistic regression. Second edition*. New York: John Wiley and Sons.

Jacquart, C. (1907). *La mortalité infantile dans les Flandres: étude de démographie belge*. Brussels : Editions A. Dewit.

Janssens, A., M. Messelink and A. Need (2010). Faulty genes or faulty parents? Gender, family, and survival in early and late childhood in the Netherlands, 1860-1900, *The History of the Family* 15(1): 91-108.

Janssens, A. and B. Pelzer (2012). *De rol van moeders in de overlevingskansen van kinderen in het verleden. Fabrieksmeisjes versus dienstboden, 1880-1940. [The role of mothers in the survival chances of children in the past. Factory girls versus servants, 1880-1940]*. WOG working papers, 2. Retrieved from www.historicaldemography.be/ publications_working_papers_wog.html.

Jeuninckx, K. (1964). De havenbeweging in de Franse en Hollandse periode [The port movement in the French and Dutch period]. In: Genootschap voor Antwerpse Geschiedenis [Antwerp History Society] (Ed.), *Bouwstoffen voor de geschiedenis van Antwerpen in de XIXde eeuw [Materials for the history of Antwerp in the 19th century]*. Antwerp: Lloyd Anversois, 94-123.

Kaplan, H. (1994). Evolutionary and wealth flow theories on fertility: empirical tests and new models, *Population and Development Review* 20: 753-791.

Kemkes, A. (2006). Does the sex of firstborn children influence subsequent fertility behaviour? Evidence from family reconstitution, *Journal of Family History* 31(2):144-162.

Kippen, R. (2011). 'A pestilence stalks abroad': familial clustering of deaths during the Tasmanian scarlet fever, measles and influenza epidemics of 1852-1854, *Genus. Journal of Population Sciences* 67(2): 57-75.

Knodel, J. and E. van de Walle (1967). Breastfeeding, fertility and infant mortality: an

analysis of some early German data, *Population Studies* 21(2): 109–131.

Knodel, J. (1982). Child mortality and reproductive behaviour in German village populations in the past: a micro-level analysis of the replacement effect, *Population Studies* 36(2): 177-200.

Knodel, J. and A.I. Hermalin (1984). Effects of birth rank, maternal age, birth interval, and sibship size on infant and child mortality: evidence from 18th and 19th century reproductive histories, *American Journal of Public Health* 74(10): 1098-1106.

Kok, J. (2007). Principles and prospects of the life course paradigm, *Annales de Démographie Historique* 113(1): 203-230.

Kok, J., M. Vandezande and K. Mandemakers (2011). Household structure, resource allocation and child well-being. A comparison across family systems, *Tijdschrift voor Sociale en Economische Geschiedenis/The Low Countries Journal of Social and Economic History* 8(4): 76-101.

Kruithof, J. (1964). De demografische ontwikkeling in de XIXde eeuw [The demographic evolution in the 19th century]. In: Genootschap voor Antwerpse Geschiedenis [Antwerp History Society] (Ed.), *Bouwstoffen voor de geschiedenis van Antwerpen in de XIXde eeuw [Materials for the history of Antwerp in the 19th century]*. Antwerp: Lloyd Anversois, 508-543.

Laird, N. and D. Olivier (1981). Covariance analysis of censored survival data using log-linear analysis techniques, *Journal of the American Statistical Association* 76: 231-240.

Lalou, R. (1991). *Des enfants pour le paradis: la mortalité des nouveau-nés en Nouvelle-France*. PhD thesis. Montréal: University of Montréal.

Lalou, R. (1997). Endogenous mortality in New France: at the crossroads of natural and social selection. In: A. Bideau, B. Desjardins and H. Pérez-Brignoli (eds.). *Infant and child mortality in the past*. Oxford: Clarendon Press, 203-215.

Lampo, J. (2002). *Tussen kaai en schip. De Antwerpse havenbuurt voor 1885 [Between quay and ship. The Antwerp port area before 1885]*. Leuven: Davidsfonds.

Lantto, P. (2010). Borders, citizenship and change. The case of the Sámi people, 1751-2008, *Citizenship Studies* 14: 543-556.

Leboutte, R. and R. Obotela (1988). Les registres de population en Belgique. Genèse d'une

technique administrative et d'une source de démographie historique, *Bulletin de la Commission royale d'Histoire* 154: 285-305.

Lesthaeghe, R.J. (1977). *The decline of Belgian fertility, 1800-1970*. Princeton (N.J.): Princeton University Press.

Lesthaeghe, R.J. (1983). De borstvoeding als verklaring voor regionale verschillen in vruchtbaarheid en zuigelingensterfte: Nederland en België in het midden van de XIXe eeuw [Breastfeeding as an explanation for regional differences in fertility and infant mortality], *Bevolking en Gezin [Population and Family]* Suppl. 2: 61-84.

Lindkvist, M. and G. Broström (2006). Clustered infant mortality in an intergenerational perspective, Skellefteå 1831-1890, *paper presented at the Social Science History Conference*, Minneapolis, 1-5 November 2006.

Lis, C. (1969). Woontoestanden en gangensaneringen te Antwerpen in het midden der 19de eeuw als maat der klassentegenstellingen [Living conditions and interior yard sanitations in Antwerp in the middle of the 19th century as a measure of class differences]. *Belgisch Tijdschrift voor Nieuwste Geschiedenis [Belgian Journal of Contemporary History]* 1(1): 93-131.

Lis, C. (1986). *Social change and the labouring poor. Antwerp, 1770 - 1860*. London: Yale University Press.

Long, J.S. (1997). *Regression models for categorical and limited dependent variables*. Advanced Quantitative Techniques in the Social Sciences, vol. 7. Thousand Oaks (CA): Sage.

Long, J.S. and J. Freese (2006). *Regression models for categorical dependent variables using Stata*. College Station (TX): Stata Press.

Loyen, R. (2003). Throughput in the port of Antwerp (1901-2000): an integrated functional approach. In: R. Loyen and E. Buyst (eds.). *Struggling for Leadership: Antwerp - Rotterdam port competition between 1870-2000*. Heidelberg: Physica-Verlag, 29-61.

Loyen, R., E. Buyst and G. Devos. (2003). *Struggling for leadership: Antwerp – Rotterdam port competition between 1870-2000*. Heidelberg: Physica-Verlag.

Lovejoy, C.O. (1981). The origin of man, *Science* 211: 341-350.

Luke, D.A. (2004). *Multilevel modelling*. Quantitative applications in the social sciences, 143.

London: Sage.

Lynch, K.A. and J.B. Greenhouse (1994). Risk factors for infant mortality in nineteenth-century Sweden, *Population Studies* 48(1): 117–133.

Makepeace, G. and S. Pal (2008). Understanding the effects of siblings on child mortality: evidence from India, *Journal of Population Economics* 21(4): 877-902.

Masuy-Stroobant, G. (1983). *Les determinants de la mortalité infantile. La Belgique d'hier et d'aujourd'hui [The determinants of infant mortality. Belgium in the past and nowadays]*. Leuven: Ciaco.

Matthijs, K. and S. Moreels (2010). The Antwerp COR*-database: a unique source for historical-demographic research, *The History of the Family* 15(1): 109-115.

Meurkens, P. (1984). *Sociale verandering in het oude Kempenland 1840-1910: demografie, economie en cultuur van een preïndustriële samenleving [Social change in the former Campine area 1840-1910: demography, economy and culture of a preindustrial society]*. Nijmegen: Katholieke Universiteit Nijmegen.

Millward, R. and F. Bell (2001). Infant mortality in Victorian Britain. The mother as medium, *Economic history review* 54(4): 699-733.

Moreels, S. (2010). Classifying historical occupational titles of women in a social class scheme. The career mobility of fertile women living in Antwerp during 1846-1906, *Belgisch Tijdschrift voor Nieuwste Geschiedenis/Revue Belge d'Histoire Contemporaine [Belgian Journal of Contemporary History]*, 40(1-2): 143-175.

Moreels, S. and M. Vandezande (2012). Migration and reproduction in transitional times. Stopping behaviour of immigrants and natives in the Belgian city of Antwerp (1810-1925), *Historical Social Research – Historische Sozialforschung* 37(3): 321-350.

Mosley, W.H. and L.C. Chen (1984). An analytic framework for the study of child survival in developing countries, *Population and Development Review* 10: 25-45.

Murphy, M. (1999). Is the relationship between fertility of parents and children really weak?, *Social Biology* 46: 122-145.

Nault, F., B. Desjardins and J. Légaré (1990). Effects of reproductive behaviour on infant mortality of French-Canadians during the seventeenth and eighteenth centuries, *Population Studies* 44(2): 273-285.

Neven, M. (2000). Mortality differentials and the peculiarities of mortality in an urban industrial population: a case-study of Tilleur, Belgium, *Continuity and Change* 15: 297-329.

Neven, M. and I. Devos (2001). Breaking stereotypes. Historical demography in Belgium since 1981 (19th and early 20th centuries), *Belgisch Tijdschrift voor Nieuwste Geschiedenis* 31(3-4): 311-346.

Nilsdotter Jeub, U. (1993). *Parish records. 19th century ecclesiastical registers*. Haparanda and Umeå: Demographic Data Base.

Omariba, D.W.R., F. Rajulton and R. Beaujot (2008). Correlated mortality of siblings in Kenya: the role of state dependence, *Demographic Research* 18: 311–336.

Ornstein, M.D. and G.A. Darroch (1978). National mobility studies in past time: a sampling strategy, *Historical Methods* 11(4): 59-87.

Øyen, N., H.A. Boyd, G. Poulsen, J. Wohlfahrt and M. Melbye (2009). The clustering of premature deaths in families, *Epidemiology* 20(5): 757-765.

Pebley, A.R. and P.W. Stupp (1987). Reproductive patterns and child mortality in Guatemala, *Demography* 24(1): 43-60.

Pebley, A.R, A.I. Hermalin and J. Knodel (1991). Birth spacing and infant mortality: evidence for eighteenth and nineteenth century German villages, *Journal of Biosocial Science* 23(4): 445–459.

Piron, T. (1957). *Des registres de population en Belgique: un manuel pratique*. Lier: Van In.

Poulain, M. and D. Tabutin (1977). Mortalité aux jeunes âges en Belgique de 1840 à 1970, *Population et Famille* 42: 49-86.

Poulain, M. (2010). Le registre de population belge. In: T. Eggerickx and J.-P. Sanderson (eds.) *Histoire de la population de la Belgique et de des territoires. Actes de la Chaire Quetelet 2005*. Louvain-la-Neuve : Presses Universitaires de Louvain, 83-116.

Preston, S.H. (1976). Family sizes of children and family sizes of women, *Demography* 13: 105-114.

Preston, S.H. (ed.) (1978). *The effects of infant and child mortality on fertility*. New York: Academic Press.

Rabe-Hesketh, S. and A. Skrondal (2008). *Multilevel and longitudinal modelling using Stata*.

College Station, TX: Stata Press.

Reid, A. (2001). Neonatal mortality and stillbirths in early twentieth century Derbyshire, England, *Population Studies* 55(2): 213-232.

Reid, A. (2002). Infant feeding and post-neonatal mortality in Derbyshire, England, in the early twentieth century, *Population Studies* 56(1): 151-166.

Reher, D. and A. Sanz-Gimeno (2007). Rethinking historical reproductive change: insights from longitudinal data for a Spanish town, *Population and Development Review* 33(4): 703-727.

Reher, D.S., J.A. Ortega and A. Sanz-Gimeno (2008). Intergenerational transmission of reproductive traits in Spain during the demographic transition, *Human Nature* 19: 23-43.

Ronsmans, C. (1995). Patterns of clustering of child mortality in a rural area of Senegal, *Population Studies* 49: 443-461.

Rutstein, S.O. (2005). Effects of preceding birth intervals on neonatal, infant and under-five year mortality and nutritional status in developing countries: evidence from the Demographic and Health Surveys, *International Journal of Gynaecology and Obstetrics* 89(4) Suppl 1: 7–24.

Saerens, L. (1999). *Vreemdelingen in een 'wereldstad'. Een geschiedenis van Antwerpen en haar Joodse gemeenschap 1880-1944 [Strangers in a 'world city'. A history of Antwerp and her Jewish community 1880-1944]*. Doctoral thesis in History, KU Leuven. Leuven: KU Leuven.

Saha, U.R. and A. van Soest (2011). Infant death clustering in families: magnitude, causes, and the influence of better health services, Bangladesh 1982-2005, *Population Studies* 65(3): 273-287.

Sanderson, J.-P. and T. Eggerickx (2010). Des sources de données méconnues pour l'histoire de l'immigration international: les Registres des étrangers et les « recensements des étrangers » de 1938 et 1939. *Histoire de la population de la Belgique et de des territoires. Actes de la Chaire Quetelet 2005*. Louvain-la-Neuve : Presses Universitaires de Louvain, 117-141.

Santow, G. (1987). Reassessing the contraceptive effect of breastfeeding, *Population Studies* 41(1): 147-160.

Sastry, N. (1997). Family-level clustering of childhood mortality risk in Northeast Brazil, *Population Studies* 51(2): 245-261.

Schofield, R., D. Reher and A. Bideau (eds.) (1991). *The decline of mortality in Europe*. Oxford: Clarendon Press.

Schumacher, R. (2010). *Structures et comportements en transition. La reproduction démographique à Genève au 19e siècle*. Bern: Peter Lang.

Sear, R. and R. Mace (2008). Who keeps children alive? A review of the effects of kin on child survival, *Evolution and Human Behavior* 29: 1-18.

Sear, R. and D. Coall (2011). How much does family matter? Cooperative breeding and the demographic transition, *Population and Development Review* 37: 81-112.

Shorter, E. (1975). *The making of the modern family*. New York: Basic Books.

Simonton, D. (1998). *The history of European women's work. 1700 to the present.* London and New York: Routledge.

Sköld, P. (1997). Espace from catastrophe: the Saami's experience with smallpox in eighteenth- and nineteenth-century Sweden, *Social Science History* 21(1): 1-25.

Sköld, P. (2004). The birth of population statistics in Sweden, *The History of the Family* 9(1): 5-21.

Sköld, P. and P. Axelsson (2008). The northern population development. Colonization and mortality in Swedish Sápmi, *International Journal of Circumpolar Health* 67(1): 29-44.

Sköld, P., P. Axelsson, L. Karlsson and L. Smith (2011). Infant mortality of Sami and settlers in Northern Sweden: the era of colonization 1750-1900, *Global Health Action* 4, published online Oct. 27th 2011, doi: 10.3402/gha.v4i0.8441.

Smith, D.S. (1991). Mortality differentials before the Health Transition, *Health Transition Review* 1: 235-237.

Szelenyi, S. (2001). The 'woman problem' in stratification theory and research. In: D.B. Grusky (ed.). *Social stratificiation: class, race, and gender in sociological perspective*. Boulder (CO): Westview Press, 681-688.

Tabutin, D. and M. Willems (1998). Differential mortality by sex from birth to adolescence: the historical experience of the West (1750-1930). In: United Nations, Dept. of Economic and Social Affairs, Population Division. *Too young to die: genes or sex?*

New York: United Nations, 17–52.

Tedebrand, L.-G. (1993). Introduction. In: Brändström, A. and L.-G. Tedebrand (eds.). *Health and social change. Disease, health and public care in the Sundsvall district 1750-1950*. Report n° 9 from the Demographic Data Base. Umeå: Umeå University, 7-14.

Thijs, A.K.L. (1987). *Van 'werkwinkel' tot 'fabriek': de textielnijverheid te Antwerpen (einde 15de – begin 19de eeuw) [From sweatshop to factory: textile industry in Antwerp (end 15th – start of 19th century)]*. Brussels: Gemeentekrediet.

Tilly, L.A. and J.W. Scott (1978). *Women, work and family*. New York: Holt, Rinehart and Winston.

Törnlund, E. (2011). Kyrkböker – Församlingsinformation [Church records – Parish information]. In: *Demographic Data Base Manual.* Umeå: Umeå University.

Trussell, J. and G. Rodriguez (1990). Heterogeneity in demographic research, in Adams, J., D.A. Lam, A.I. Hermalin and P.E. Smouse (eds.), *Convergent issues in genetics and demography*. Oxford: Oxford University Press, 111-132.

Vallin, J. (1991). Mortality in Europe from 1720 to 1914. Long-term trends and changes in patterns by age and sex. In: R. Schofield, D. Reher and A. Bideau (eds.) *The decline of mortality in Europe*. Oxford: Clarendon Press, 38-67.

Van Baelen, H. (2007). *Constructie van een historisch-demografisch longitudinale database: Methodologie van de Demographica Flandria Selecta [Construction of a historical demographic longitudinal database. Methodology of the Demographica Flandria Selecta]*. Leuven: Centre for Sociological Research.

Van Bavel, J. (2002). *Van natuurlijke naar gecontroleerde vruchtbaarheid? Geboortebeperking in Leuven, 1846-1910 [From natural to controlled fertility? Birth control in Leuven, 1846-1910]*, Leuven: Leuven University Press.

Van Bavel, J. (2004). Detecting stopping and spacing behaviour in historical demography: a critical review of methods, *Population – English edition* 59(1): 117-128.

Van Bavel, J. and J. Kok (2009). Social control and the intergenerational transmission of age at first marriage in rural Holland, 1850-1940, *Population* 64: 343-360.

Van Bavel, J., S. Moreels, B. Van de Putte and K. Matthijs (2011). Family size and intergenerational social mobility during the demographic transition. A case study in 19th

century Antwerp, Belgium, *Demographic Research* 24: 313-344.

Van Bodegom, D. (2010). Clustering of child mortality in a contemporary polygynous African population, *Paper presented at the international workshop for death clustering*, Umeå University, Sweden, 22-23 October 2010.

Van de Walle, E. (1974). *The female population of France in the nineteenth century: a reconstruction of 82 départements*. Princeton (NJ): Princeton University Press.

Vanfraechem, S. (2005). *Een sfeer om haring te braden? Arbeidsverhoudingen aan de Antwerpse haven, 1880-1972 [A brooding athmosphere? Labour relations in the Antwerp port, 1880-1972]*. Gent: Academia Press.

Van Houtven, S. (2008). Invisible boundaries: domesticating the Antwerp Seefhoek neighbourhood and its 'cités ouvrières' during the final quarter of the nineteenth century. *Paper presented at the IXth International Conference on Urban History*, Lyon, 27-30 August 2008.

Van Isacker, K. (1966). *De Antwerpse dokwerker 1830-1940 [The Antwerp dockworker 1830-1940]*. Antwerp: De Nederlandsche Boekhandel N.V.

Van Klink, A. (2003). The Kempen Nexus. The spatial-economic development of Rotterdam and Antwerp. In: R. Loyen and E. Buyst (eds.). *Struggling for Leadership: Antwerp - Rotterdam port competition between 1870-2000*. Heidelberg: Physica-Verlag, 143-159.

Van Leeuwen, M.H.D., I. Maas and A. Miles (2002). *HISCO: Historical International Standard Classification of Occupations*. Leuven: Leuven University Press.

Van Leeuwen, M.H.D. and I. Maas (2011). *HISCLASS: A Historical International Social Class Scheme*. Leuven: Leuven University Press.

Van Poppel, F.W.A., J. Kok and E. Kruse (1995). La cause principale des différences c'est le crime: essai de explication de la surmortalité des enfants illégitimes à la Haye au milieu du XIXe siècle, *Annales de Démographie Historique* 95: 241-275.

Van Poppel, F.W.A. and K. Mandemakers (1997). Differential infant and child mortality in the Netherlands, 1812-1912: first results of the Historical Sample of the Population of the Netherlands. In: A. Bideau, B. Desjardins and H. Pérez-Brignoli (eds.). *Infant and child mortality in the past*. Oxford: Clarendon Press, 276-300.

Van Poppel, F.W.A. and C. Van der Heijden (1997). The effects of water supply on infant and

childhood mortality: a review of historical evidence, *Health Transition Review* 7: 113-148.

Van Poppel, F.W.A., J. Schellekens and A.C. Liefbroer (2002). Religious differentials in infant and child mortality in Holland, 1955-1912, *Population Studies* 56(2): 277-289.

Van Poppel, F.W.A., M. Jonker and K. Mandemakers (2005). Differential infant and child mortality in three Dutch regions, 1812-1909, *Economic History Review* 58(2): 272-309.

Van Poppel, F.W.A., C. Monden and K. Mandemakers (2008). Marriage timing over the generations, *Human Nature* 19: 7-22.

Van Poppel, F.W.A., D. Reher, A. Sanz-Gimeno, M. Sanchez-Dominguez, and E. Beekink (2012). Mortality decline and reproductive change during the Dutch demographic transition: revisiting a traditional debate with new data, *Demographic Research* 27(11): 299-338.

Veraghtert, K. (1986). Part III: From inland port to international port 1790-1914. In F. Suykens, G. Asaert, A. De Vos, A. Thijs and K. Veraghtert (eds.). *Antwerp. A port for all seasons*. Antwerp: Ortelius Series, 277–452.

Vercauteren, G. (2001). De zorg voor de behoeftige ouderen te Antwerpen in de negentiende eeuw [The care of the needy elderly in Antwerp in the 19th century]. *Belgisch Tijdschrift voor Nieuwste Geschiedenis [Belgian Journal of Contemporary History]* 31(1-2): 253-281.

Vervoort, R. (ed.) (2005). *Red Star Line: Antwerpen = Amerika: een nieuwe toekomst tegemoet [Red Star Line: Antwerp = America: on towards a new future]*. Antwerp: Petraco-Pandora.

Vrielinck, S. (2000). *De territoriale indeling van België. 1795-1963* [*The territorial division of Belgium. 1795-1963]*. Leuven: Leuven University Press.

Walhout, E.C. (2010). Is breast best? Evaluating breastfeeding patterns and causes of infant death in a Dutch province in the period 1875-1900, *History of the Family* 15(1): 76-90.

Walhout, E.C. and J. Mandemakers (2011). Can social class differences in infant mortality be explained by breastfeeding practices? *Paper presented at the 4th Historical Demography Day*, Nijmegen, 8th December 2011.

Warg, S. (2002). *Familjen I gruvmiljö. Migration, giftermålsmönster och fertilitet I*

norrbottnisk gruvindustri 1890-1930 [The family in the mining context. Migration, marriage patterns and fertility in Norbottens mining industry 1890-1930]. Report n° 20 from the Demographic Data Base. Umeå: Umeå University.

Wienke, A. (2011). *Frailty models in survival analysis*. Boca Raton (FL): Chapman and Hall/CRC.

Winking, J. (2006). Are men really that bad as fathers? The role of men's investments, *Biodemography and Social Biology* 53: 100-115.

Winter, A. (2008). Caught between law and practice. Migrants and settlement legislation in the Southern Low Countries in a comparative perspective, ca. 1700-1900, *Rural History* 19(2): 137-162.

Winter, A. (2009). *Migrants and urban change: newcomers to Antwerp, 1760-1914*. London: Pickering and Chatto.

Yamaguchi, K. (1991). *Event history analysis*. Applied Social Research Methods Series, 28. Newbury Park, CA: Sage.

Zaba, B. and P.H. David (1996). Fertility and the distribution of child mortality risk among women: an illustrative analysis, *Population Studies* 50(2): 263–278.

Zenger, E. (1993). Siblings' neonatal mortality risks and birth spacing in Bangladesh, *Demography* 30(3): 477–488.

SUMMARY

SAMENVATTING

RÉSUMÉ

ENGLISH SUMMARY

Born to die. Death clustering and the intergenerational transmission of infant mortality, the Antwerp district, 1846-1905

In the recent literature on young age mortality a considerable attention has been devoted to the phenomenon of death clustering: the unequal distribution of (infant) mortality between families. Infant deaths seem to occur in a limited number of families. So far death clustering has been observed many populations, both historical and contemporary. The causes of this death clustering are still very unclear. This is partly due to the fact that there are no good tools available to unravel the characteristics, causes, significance and consequences of mortality clustering. However there is a consensus that this perspective on mortality might yield new and relevant information, not only in describing and explaining the historical mortality transition, but also for our understanding of the fertility transition.

This study examines death clustering in the district of Antwerp in the second half of the 19th century. The study has a double set-up. Firstly, attention is paid to the methodological aspects of clustering: the development and application of methods and techniques to measure and visualize clustering. Therefore infant mortality is first approached from the perspective of the loss of children within the family, then from the mortality of the individual child, and finally from the unequal distribution of mortality between populations. For the latter, the Antwerp analyses are compared with data from northern Sweden. Furthermore this allows to introduce a new dimension adding to the debate on the causes of death clustering, i.e. the effect of infant mortality from previous generation to the survival of children of the next generation.

One of the main results is that mortality rates of infants in successive generations are strongly correlated, both along the maternal and the paternal line. Those who lose their infant brothers and sisters, later on have a significantly greater risk to lose their own infants. This correlation is caused by a complex interplay of factors, such as socialization of educational practices and breastfeeding traditions, intergenerational inheritance of social status, regional influences, but also genetic defects. In practice it proves very difficult to detect and separate the combined contributions of these factors.

A genealogical history of infant mortality is one of the reasons for the unequal distribution of infant deaths on families: the mortality of infants is clustered in a small number of families, in part because the parents of those families descended from families with high infant mortality.

These findings have several implications, both methodological and theoretical. For the empirical reconstruction and the theoretical interpretation of mortality, death clustering, and the decline of infant and child mortality it is statistically necessary and theoretically useful to consider family characteristics. In turn this is crucial for explaining the demographic transition, the transition from high to low fertility and mortality.

NEDERLANDSE SAMENVATTING

Geboren om te sterven. Sterfteclustering en de intergenerationele transmissie van zuigelingensterfte, arrondissement Antwerpen, 1846-1905

In de recente literatuur over sterfte op jonge leeftijd is ruime aandacht besteed aan het fenomeen sterfteclustering: de ongelijke verdeling van (zuigelingen)sterfte tussen gezinnen. Die sterfte is geclusterd in een kleine minderheid van de gezinnen. Dat werd vastgesteld in veel populaties, zowel historische als hedendaagse. De oorzaken van die sterfteclustering zijn nog erg onduidelijk. Dat heeft onder meer te maken met het feit dat er nog geen goede instrumenten beschikbaar zijn om de kenmerken, de oorzaken, de betekenis en de gevolgen van sterfteclustering te ontrafelen. Er is wel een consensus dat dit perspectief op sterfte nieuwe en relevante informatie kan opleveren, niet alleen voor het inzicht in de historische sterftetransitie, maar ook in de vruchtbaarheidstransitie.

Dit proefschrift bestudeert sterfteclustering in het arrondissement Antwerpen in de tweede helft van de 19de eeuw. Er is een dubbel opzet. Eerst gaat de aandacht naar de methodologische aspecten van clustering: het ontwikkelen en toepassen van methoden en technieken om clustering te meten en te visualiseren. Daarbij wordt zuigelingensterfte eerst benaderd vanuit het perspectief van het verlies van kinderen binnen het gezin, vervolgens vanuit de sterftekansen van het kind en ten slotte vanuit de ongelijke verdeling van sterfte tussen populaties. Voor dat laatste worden de Antwerpse analyses vergeleken met data van het noorden van Zweden. Dat laat vervolgens toe om een nieuwe dimensie toe te voegen aan de discussie over de oorzaken van sterfteclustering, namelijk de invloed van zuigelingensterfte van vorige generatie op de overlevingskansen van kinderen van de volgende generatie.

Een van de voornaamste resultaten is dat sterftekansen van zuigelingen in opeenvolgende generaties sterk gecorreleerd zijn, zowel langs moederlijke als langs vaderlijke lijn. Wie als kind broers en zussen als zuigeling ziet sterven, heeft zelf ook een beduidend grotere kans om later zijn of haar eigen kinderen te verliezen gedurende het eerste levensjaar. Dat wordt veroorzaakt door een complex samenspel van factoren, zoals socialisatie van opvoedingspraktijken en borstvoedingstradities, intergenerationele overdracht van status, regionale invloeden, maar ook genetische defecten. Het is in de praktijk erg moeilijk om het aparte en het gecombineerde gewicht van deze factoren te detecteren.

Een genealogische voorgeschiedenis van zuigelingensterfte is zelf één van de oorzaken voor de ongelijke verdeling van zuigelingensterfte over gezinnen. De sterfte van

zuigelingen is geclusterd in een klein aantal gezinnen, mede omdat de ouders van die gezinnen zelf ook uit gezinnen met grote zuigelingensterfte afstammen.

Deze bevindingen hebben allerlei implicaties, zowel methodologische als theoretische. Voor de empirische reconstructie en de theoretische duiding van sterfte(clustering) en van de daling van de zuigelingen- en kindersterfte is het statistisch noodzakelijk en theoretisch nuttig om gezinskenmerken in de analyses op te nemen. Dat is op zijn beurt cruciaal voor het verklaren van de demografische transitie, de overgang van hoge naar lage vruchtbaarheid en sterfte.

RÉSUMÉ FRANÇAIS

Né pour mourir. Le ‘clustering’ de la mortalité et la transmission intergénérationnelle de la mortalité infantile, l’arrondissement d’Anvers, 1846-1905

La littérature récente relative à la mortalité infantile a prêté une grande attention au phénomène du ‘clustering’ de la mortalité, c’est-à-dire la répartition inégale de la mortalité (infantile) entre les familles. Le fait que cette mortalité soit effectivement nettement plus élevée au sein de certaines familles a été constaté dans de nombreuses populations, à la fois historiques et contemporaines. Mais les causes de ce ‘clustering’ ne sont pas encore déterminées. C’est une conséquence directe du manque des bons outils permettant de mesurer et de démêler les caractéristiques, les causes, l'importance et les conséquences du ‘clustering’ de mortalité. Néanmoins il existe consensus sur le fait que cette nouvelle perspective de la mortalité est susceptible d’apporter des informations nouvelles et pertinentes, non seulement pour la compréhension de la diminution de la mortalité historique, mais aussi de la diminution de la fécondité.

Cette étude examine le ‘clustering’ de la mortalité infantile dans l'arrondissement d'Anvers au cours de la seconde moitié du 19e siècle. Le but de cette étude est double. D’une part, l’analyse des aspects méthodologiques de l’étude du ‘clustering’: le développement et l'application des méthodes et techniques qui permettent la mesure et la visualisation du phénomène. La mortalité infantile est d'abord approchée sous l'angle de la perte d'enfants dans la famille, puis de la mortalité d’un enfant en tant qu’individu, et enfin de la répartition inégale de la mortalité infantile entre les populations. Ce dernier aspect est analysé en comparant les données d'Anvers avec celles du nord de la Suède. D’autre part, cela permet d’ajouter une nouvelle dimension au débat sur les causes de la mort de ‘clustering’, à savoir l'effet de la mortalité infantile à la génération précédente à la survie des enfants de la génération suivante.

L'un des résultats principaux est que les taux de la mortalité infantile dans des générations successives sont fortement corrélés, à la fois sur la ligne maternelle et paternelle. Ceux qui perdent des frères et sœurs à la mort infantile, eux-mêmes ont un risque significativement plus élevée de perdre ses propres enfants. Cette corrélation intergénérationnelle résulte d’une interaction complexe des causes, tels que la socialisation des pratiques éducatives et des traditions d‘allaitement, la transmission intergénérationnelle de la position sociale, des influences régionales, aussi que des défauts génétiques. En pratique c’est très difficile de séparer les contributions individuelles et combinées de ces déterminants.

Des antécédents généalogiques de la mortalité infantile expliquent en même temps la répartition inégale de la mortalité infantile entre les familles. La mortalité infantile est concentrée dans un nombre limité de familles entre autres parce que les parents de ces familles eux-mêmes descendent en droite ligne de familles avec une mortalité infantile élevée.

Les conclusions de cette étude ont plusieurs implications, à la fois théoriques et méthodologiques. Pour la reconstruction empirique et l'interprétation théorique de la mortalité, du ‘clustering’, et de la diminution de la mortalité infantile, c’est une nécessité statistique et une opportunité théorique d’inclure les caractéristiques familiaux dans les analyses. Par conséquent les conclusions de cette étude sont cruciales pour expliquer la transition démographique, c’est-à-dire la transition de la fécondité et de la mortalité infantile historique.

DOCTORATEN IN DE SOCIALE WETENSCHAPPEN
EN
DOCTORATEN IN DE SOCIALE EN CULTURELE ANTROPOLOGIE

I. REEKS VAN DOCTORATEN IN DE SOCIALE WETENSCHAPPEN[(1)]

1. CLAEYS, U., *De sociale mobiliteit van de universitair afgestudeerden te Leuven. Het universitair onderwijs als mobiliteitskanaal*, 1971, 2 delen 398 blz.

2. VANHESTE, G., *Literatuur en revolutie*, 1971, 2 delen, 500 blz.

3. DELANGHE, L., *Differentiële sterfte in België. Een sociaal-demografische analyse*, 1971, 3 delen, 773 blz.

4. BEGHIN, P., *Geleide verandering in een Afrikaanse samenleving. De Bushi in de koloniale periode*, 1971, 316 blz.

5. BENOIT, A., *Changing the education system. A Colombian case-study*, 1972, 382 blz.

6. DEFEVER, M., *De huisartssituatie in België*, 1972, 374 blz.

7. LAUWERS, J., *Kritische studie van de secularisatietheorieën in de sociologie*, 1972, 364 blz.

8. GHOOS, A., *Sociologisch onderzoek naar de gevolgen van industrialisering in een rekonversiegebied*, 1972, 256 blz. + bijlagen.

9. SLEDSENS, G., *Mariage et vie conjugale du moniteur rwandais. Enquête sociologique par interview dirigée parmi les moniteurs mariés rwandais*, 1972, 2 delen, 549 blz.

10. TSAI, C., *La chambre de commerce internationale. Un groupe de pression international. Son action et son rôle dans l'élaboration, la conclusion et l'application des conventions internationales établies au sein des organisations intergouvernementales à vocation mondiale (1945-1969)*, 1972, 442 blz.

11. DEPRE, R., *De topambtenaren van de ministeries in België. Een bestuurssociologisch onderzoek*, 1973, 2 delen, 423 blz. + bijlagen.

12. VAN DER BIESEN, W., *De verkiezingspropaganda in de democratische maatschappij. Een literatuurkritische studie en een inhoudsanalyse van de verkiezingscampagne van 1958 in de katholieke pers en in de propagandapublikaties van de C.V.P.*, 1973, 434 blz.

13. BANGO, J., *Changements dans les communautés villageoises de l'Europe de l'Est. Exemple : la Hongarie*, 1973, 434 blz.

14. VAN PELT, H., *De omroep in revisie. Structurering en ontwikkelingsmogelijkheden van het radio- en televisiebestel in Nederland en België. Een vergelijkende studie*, Leuven, Acco, 1973, 398 blz.

15. MARTENS, A., *25 jaar wegwerparbeiders. Het Belgisch immigratiebeleid na 1945*, 1973, 319 blz.

16. BILLET, M., *Het verenigingsleven in Vlaanderen. Een sociologische typologieformulering en hypothesetoetsing*, 1973, 695 blz. + bijlagen.

17. BRUYNOOGHE, R., *De sociale structurering van de gezinsverplegingssituatie vanuit kostgezinnen en patiënten*, 1973, 205 blz. + bijlagen.

18. BUNDERVOET, J., *Het doorstromingsprobleem in de hedendaagse vakbeweging. Kritische literatuurstudie en verkennend onderzoek in de Belgische vakbonden*, 1973, 420 blz. + bijlagen.

19. GEVERS, P., *Ondernemingsraden, randverschijnselen in de Belgische industriële democratiseringsbeweging. Een sociologische studie*, 1973, 314 blz.

---

(1) EEN EERSTE SERIE DOCTORATEN VORMT DE REEKS VAN DE SCHOOL VOOR POLITIEKE EN SOCIALE WETENSCHAPPEN (NRS. 1 TOT EN MET 185). DE INTEGRALE LIJST KAN WORDEN GEVONDEN IN NADIEN GEPUBLICEERDE DOCTORATEN, ZOALS G. DOOGHE, "DE STRUCTUUR VAN HET GEZIN EN DE SOCIALE RELATIES VAN DE BEJAARDEN". ANTWERPEN, DE NEDERLANDSE BOEKHANDEL, 1970, 290 BLZ.
EEN TWEEDE SERIE DOCTORATEN IS VERMELD IN DE "NIEUWE REEKS VAN DE FACULTEIT DER ECONOMISCHE EN SOCIALE WETENSCHAPPEN". DE INTEGRALE LIJST KAN WORDEN GEVONDEN IN O.M. M. PEETERS, "GODSDIENST EN TOLERANTIE IN HET SOCIALISTISCH DENKEN". EEN HISTORISCH-DOCTRINAIRE STUDIE, 1970, 2 DELEN, 568 BLZ.

20. MBELA, H., *L'intégration de l'éducation permanente dans les objectifs socio-économiques de développement. Analyse de quelques politiques éducationnelles en vue du développement du milieu rural traditionnel en Afrique noire francophone*, 1974, 250 blz.

21. CROLLEN, L., *Small powers in international systems*, 1974, 250 blz.

22. VAN HASSEL, H., *Het ministrieel kabinet. Peilen naar een sociologische duiding*, 1974, 460 blz. + bijlagen.

23. MARCK, P., *Public relations voor de landbouw in de Europese Economische Gemeenschap*, 1974, 384 blz.

24. LAMBRECHTS, E., *Vrouwenarbeid in België. Een analyse van het tewerkstellingsbeleid inzake vrouwelijke arbeidskrachten sinds 1930*, 1975, 260 blz.

25. LEMMEN, M.H.W., *Rationaliteit bij Max Weber. Een godsdienstsociologische studie*, 1975, 2 delen, 354 blz.

26. BOON, G., *Ontstaan, ontwikkeling en werking van de radio-omroep in Zaïre tijdens het Belgisch Koloniale Bewind (1937-1960)*, 1975, 2 delen, 617 blz.

27. WUYTS, H., *De participatie van de burgers in de besluitvorming op het gebied van de gemeentelijke plannen van aanleg. Analyse toegespitst op het Nederlandstalige deel van België*, 1975, 200 blz. + bijlage.

28. VERRIEST, F., *Joris Helleputte en het corporatisme*, 1975, 2 delen, 404 blz.

29. DELMARTINO, F., *Schaalvergroting en bestuurskracht. Een beleidsanalystische benadering van de herstrukturering van de lokale besturen*, 1975, 3 delen, 433 blz. + bijlagen.

30. BILLIET, J., *Secularisering en verzuiling in het Belgisch onderwijs*, 1975, 3 delen, 433 blz. + bijlagen.

31. DEVISCH, R., *L'institution rituelle Khita chez les Yaka au Kwaango du Nord. Une analyse séméiologique*, 1976, 3 volumes.

32. LAMMERTYN, F., *Arbeidsbemiddeling en werkloosheid. Een sociologische verkenning van het optreden van de diensten voor openbare arbeidsbemiddeling van de R.V.A.*, 1976, 406 blz.

33. GOVAERTS, F., *Zwitserland en de E.E.G. Een case-study inzake Europese integratie*, 1976, 337 blz.

34. JACOBS, T., *Het uit de echt scheiden. Een typologiserend onderzoek, aan de hand van de analyse van rechtsplegingsdossiers in echtscheiding.* 1976, 333 blz. + bijlage.

35. KIM DAI WON, *Au delà de l'institutionalisation des rapports professionnels. Analyse du mouvement spontané ouvrier belge.* 1977, 282 blz.

36. COLSON, F., *Sociale indicatoren van enkele aspecten van bevolkingsgroei.* 1977, 341 blz. + bijlagen.

37. BAECK, A., *Het professionaliseringsproces van de Nederlandse huisarts.* 1978, 721 blz. + bibliografie.

38. VLOEBERGHS, D., *Feedback, communicatie en organisatie. Onderzoek naar de betekenis en de toepassing van het begrip "feedback" in de communicatiewetenschap en de organisatietheorieën.* 1978, 326 blz.

39. DIERICKX, G., *De ideologische factor in de Belgische politieke besluitvorming.* 1978, 609 blz. + bijvoegsels.

40. VAN DE KERCKHOVE, J., *Sociologie. Maatschappelijke relevantie en arbeidersemancipatie.* 1978, 551 blz.

41. DE MEYER A., *De populaire muziekindustrie. Een terreinverkennende studie.* 1979, 578 blz.

42. UDDIN, M., *Some Social Factors influencing Age at Death in the situation of Bangladesh.* 1979, 316 blz. + bijlagen.

43. MEULEMANS, E., *De ethische problematiek van het lijden aan het leven en aan het samen-leven in het oeuvre van Albert Camus. De mogelijke levensstijlen van luciditeit, menselijkheid en solidariteit.* 1979, 413 blz.

44. HUYPENS, J., *De plaatselijke nieuwsfabriek. Regionaal nieuws. Analyse van inhoud en structuur in de krant.* 494 blz.

45. CEULEMANS, M.J., *Women and Mass Media: a feminist perpective. A review of the research to date the image and status of women in American mass media.* 1980, 541 blz. + bijlagen.

46. VANDEKERCKHOVE, L., *Gemaakt van asse. Een sociologische studie van de westerse somatische kultuur.* 1980, 383 blz.

47. MIN, J.K., *Political Development in Korea, 1945-1972.* 1980, 2 delen, 466 blz.

48. MASUI, M., *Ongehuwd moeder. Sociologische analyse van een wordingsproces.* 1980, 257 blz.

49. LEDOUX, M., *Op zoek naar de rest ...; Genealogische lezing van het psychiatrisch discours.* 1981, 511 blz.

50. VEYS, D., *De generatie-sterftetafels in België.* 1981, 3 delen, 326 blz. + bijlagen.

51. TACQ, J., *Kausaliteit in sociologisch onderzoek. Een beoordeling van de zgn. 'causal modeling'-technieken in het licht van verschillende wijsgerige opvattingen over kausaliteit.* 1981, 337 blz.

52. NKUNDABAGENZI, F., *Le système politique et son environnement. Contribution à l'étude de leur interaction à partir du cas des pays est-africains : le Kenya et la Tanzanie.* 1981, 348 blz.

53. GOOSSENS, L., *Het sociaal huisvestingsbeleid in België. Een historisch-sociologische analyse van de maatschappelijke probleembehandeling op het gebied van het wonen.* 1982, 3 delen.

54. SCHEPERS, R., *De opkomst van het Belgisch medisch beroep. De evolutie van de wetgeving en de beroepsorganisatie in de 19de eeuw.* 1983, 553 blz.

55. VANSTEENKISTE, J., *Bejaardzijn als maatschappelijk gebeuren.* 1983, 166 blz.

56. MATTHIJS, K., *Zelfmoord en zelfmoordpoging.* 1983, 3 delen, 464 blz.

57. CHUNG-WON, Choue, *Peaceful Unification of Korea. Towards Korean Integration.* 1984, 338 blz.

58. PEETERS, R., *Ziekte en gezondheid bij Marokkaanse immigranten.* 1983, 349 blz.

59. HESLING, W., *Retorica en film. Een onderzoek naar de structuur en functie van klassieke overtuigingsstrategieën in fictionele, audiovisuele teksten.* 1985, 515 blz.

60. WELLEN, J., *Van probleem tot hulpverlening. Een exploratie van de betrekkingen tussen huisartsen en ambulante geestelijke gezondheidszorg in Vlaanderen.* 1984, 476 blz.

61. LOOSVELDT, G., *De effecten van een interviewtraining op de kwaliteit van gegevens bekomen via het survey-interview.* 1985, 311 blz. + bijlagen.

62. FOETS, M., *Ziekte en gezondheidsgedrag : de ontwikkeling van de sociologische theorievorming en van het sociologisch onderzoek.* 1985, 339 blz.

63. BRANCKAERTS, J., *Zelfhulporganisaties. Literatuuranalyse en explorerend onderzoek in Vlaanderen.* 1985.

64. DE GROOFF, D., *De elektronische krant. Een onderzoek naar de mogelijkheden van nieuwsverspreiding via elektronische tekstmedia en naar de mogelijke gevolgen daarvan voor de krant als bedrijf en als massamedium.* 1986, 568 blz.

65. VERMEULEN, D., *De maatschappelijke beheersingsprocessen inzake de sociaal-culturele sector in Vlaanderen. Een sociologische studie van de "verzuiling", de professionalisering en het overheidsbeleid.* 1983, 447 blz.

66. OTSHOMANPITA, Aloki, *Administration locale et développement au Zaïre. Critiques et perspectives de l'organisation politico-administrative à partir du cas de la zone de Lodja.* 1988, 507 blz.

67. SERVAES, J., *Communicatie en ontwikkeling. Een verkennende literatuurstudie naar de mogelijkheden van een communicatiebeleid voor ontwikkelingslanden.* 1987, 364 blz.

68. HELLEMANS, G., *Verzuiling. Een historische en vergelijkende analyse.* 1989, 302 blz.

II. NIEUWE REEKS VAN DOCTORATEN IN DE SOCIALE WETENSCHAPPEN EN IN DE SOCIALE EN CULTURELE ANTROPOLOGIE

1. LIU BOLONG, *Western Europe - China. A comparative analysis of the foreign policies of the European Community, Great Britain and Belgium towards China (1970-1986).* Leuven, Departement Politieke Wetenschappen, 1988, 335 blz.

2. EERDEKENS, J., *Chronische ziekte en rolverandering. Een sociologisch onderzoek bij M.S.-patiënten.* Leuven, Acco, 1989, 164 blz. + bijlagen.

3. HOUBEN, P., *Formele beslissingsmodellen en speltheorie met toepassingen en onderzoek naar activiteiten en uitgaven van locale welzijnsinstellingen en coalities.* Leuven, Departement Sociologie, 1988, 631 blz. (5 delen).

4. HOOGHE, L., *Separatisme. Conflict tussen twee projecten voor natievorming. Een onderzoek op basis van drie succesvolle separatismen.* Leuven, Departement Politieke Wetenschappen, 1989, 451 blz. + bijlagen.

5. SWYNGEDOUW, M., *De keuze van de kiezer. Naar een verbetering van de schattingen van verschuivingen en partijvoorkeur bij opeenvolgende verkiezingen en peilingen.* Leuven, Sociologisch Onderzoeksinstituut, 1989, 333 blz.

6. BOUCKAERT, G., *Productiviteit in de overheid.* Leuven, Vervolmakingscentrum voor Overheidsbeleid en Bestuur, 1990, 394 blz.

7. RUEBENS, M., *Sociologie van het alledaagse leven.* Leuven, Acco, 1990, 266 blz.

8. HONDEGHEM, A., *De loopbaan van de ambtenaar. Tussen droom en werkelijkheid.* Leuven, Vervolmakingscentrum voor Overheidsbeleid en Bestuur, 1990, 498 blz. + bijlage.

9. WINNUBST, M., *Wetenschapspopularisering in Vlaanderen. Profiel, zelfbeeld en werkwijze van de Vlaamse wetenschapsjournalist.* Leuven, Departement Communicatiewetenschap, 1990.

10. LAERMANS, R., *In de greep van de "moderne tijd". Modernisering en verzuiling, individualisering en het naoorlogse publieke discours van de ACW-vormingsorganisaties : een proeve tot cultuursociologische duiding*. Leuven, Garant, 1992.

11. LUYTEN, D., *OCMW en Armenzorg. Een sociologische studie van de sociale grenzen van het recht op bijstand.* Leuven, S.O.I. Departement Sociologie, 1993, 487 blz.

12. VAN DONINCK, B., *De landbouwcoöperatie in Zimbabwe. Bouwsteen van een nieuwe samenleving ?* Grimbergen, vzw Belgium-Zimbabwe Friendship Association, 1993. 331 blz.

13. OPDEBEECK, S., *Afhankelijkheid en het beëindigen van partnergeweld*. Leuven, Garant, 1993. 299 blz. + bijlagen.

14. DELHAYE, C., *Mode geleefd en gedragen*. Leuven, Acco, 1993, 228 blz.

15. MADDENS, B., *Kiesgedrag en partijstrategie*. Leuven, Departement Politieke Wetenschappen, Afdeling Politologie, K.U.Leuven, 1994, 453 blz.

16. DE WIT, H., *Cijfers en hun achterliggende realiteit*. *De MTMM-kwaliteitsparameters op hun kwaliteit onderzocht*. Leuven, Departement Sociologie, K.U.Leuven, 1994, 241 blz.

17. DEVELTERE, P., *Co-operation and development with special reference to the experience of the Commonwealth Carribean*. Leuven, Acco, 1994, 241 blz.

18. WALGRAVE, S., *Tussen loyauteit en selectiviteit. Een sociologisch onderzoek naar de ambivalente verhouding tussen nieuwe sociale bewegingen en groene partij in Vlaanderen*. Leuven, Garant, 1994, 361 blz.

19. CASIER, T., *Over oude en nieuwe mythen. Ideologische achtergronden en repercussies van de politieke omwentelingen in Centraal- en Oost-Europa sinds 1985*. Leuven, Departement Politieke Wetenschappen, K.U.Leuven, 1994, 365 blz.

20. DE RYNCK, F., *Streekontwikkeling in Vlaanderen. Besturingsverhoudingen en beleidsnetwerken in bovenlokale ruimtes*. Leuven, Departement Politieke Wetenschappen, Afdeling Bestuurswetenschap, K.U.Leuven, 1995, 432 blz.

21. DEVOS, G., *De flexibilisering van het secundair onderwijs in Vlaanderen. Een organisatie-sociologische studie van macht en institutionalisering*. Leuven, Acco, 1995, 447 blz.

22. VAN TRIER, W., *Everyone A King? An investigation into the meaning and significance of the debate on basic incomes with special references to three episodes from the British inter-War experience*. Leuven, Departement Sociologie, K.U.Leuven, 1995, vi+501 blz.

23. SELS, L., *De overheid viert de teugels. De effecten op organisatie en personeelsbeleid in de autonome overheidsbedrijven*. Leuven, Acco, 1995, 454 blz.

24. HONG, K.J., *The C.S.C.E. Security Regime Formation: From Helsinky to Budapest*. Leuven, Acco, 1996, 350 blz.

25. RAMEZANZADEH, A., *Internal and international dynamics of ethnic conflict. The Case of Iran*. Leuven, Acco, 1996, 273 blz.

26. HUYSMANS, J., *Making/Unmaking European Disorder. Meta-Theoretical, Theoretical and Empirical Questions of Military Stability after the Cold War*. Leuven, Acco, 1996, 250 blz.

27. VAN DEN BULCK J., *Kijkbuiskennis. De rol van televisie in de sociale en cognitieve constructie van de realiteit*. Leuven, Acco, 1996, 242 blz.

28. JEMADU Aleksius, *Sustainable Forest Management in the Context of Multi-level and Multi-actor Policy Processes*. Leuven, Departement Politieke Wetenschappen, Afdeling Bestuur en Overheidsmanagement, K.U.Leuven, 1996, 310 blz.

29. HENDRAWAN Sanerya, *Reform and Modernization of State Enterprises. The Case of Indonesia*. Leuven, Departement Politieke Wetenschappen, Afdeling Bestuur en Overheidsmanagement, K.U.Leuven, 1996, 372 blz.

30. MUIJS Roland Daniël, *Self, School and Media: A Longitudinal Study of Media Use, Self-Concept, School Achievement and Peer Relations among Primary School Children*. Leuven, Departement Communicatiewetenschap, K.U.Leuven, 1997, 316 blz.

31. WAEGE Hans, *Vertogen over de relatie tussen individu en gemeenschap.* Leuven, Acco, 1997, 382 blz.

32. FIERS Stefaan, *Partijvoorzitters in België of 'Le parti, c'est moi'?* Leuven, Acco, 1998, 419 blz.

33. SAMOY Erik, *Ongeschikt of ongewenst? Een halve eeuw arbeidsmarktbeleid voor gehandicapten*. Leuven, Departement Sociologie, K.U.Leuven, 1998, 640 blz.

34. KEUKELEIRE Stephan, *Het Gemeenschappelijk Buitenlands en Veiligheidsbeleid (GBVB): het buitenlands beleid van de Europese Unie op een dwaalspoor*. Leuven, Departement Politieke Wetenschappen, Afdeling Internationale Betrekkingen, K.U.Leuven, 1998, 452 blz.

35. VERLINDEN Ann, *Het ongewone alledaagse: over zwarte katten, horoscopen, miraculeuze genezingen en andere geloofselementen en praktijken. Een sociologie van het zogenaamde bijgeloof*. Leuven, Departement Sociologie, K.U.Leuven, 1999, 387 blz. + bijlagen.

36. CARTON Ann, *Een interviewernetwerk: uitwerking van een evaluatieprocedure voor interviewers*. Leuven, Departement Sociologie, 1999, 379 blz. + bijlagen.

37. WANG Wan-Li, *Undestanding Taiwan-EU Relations: An Analysis of the Years from 1958 to 1998*. Leuven, Departement Politieke Wetenschappen, Afdeling Internationale Betrekkingen, K.U.Leuven, 1999, 326 blz. + bijlagen.

38. WALRAVE Michel, *Direct Marketing en Privacy. De verhouding tussen direct marketingscommunicatie en de bescherming van de informationele en de relationele privacy van consumenten*. Leuven, Departement Communicatiewetenschap, K.U.Leuven, 1999, 480 blz. + bijlagen.

39. KOCHUYT Thierry, *Over een ondercultuur. Een cultuursociologische studie naar de relatieve deprivatie van arme gezinnen*. Leuven, Departement Sociologie, K.U.Leuven, 1999, 386 blz. + bijlagen.

40. WETS Johan, *Waarom onderweg? Een analyse van de oorzaken van grootschalige migratie- en vluchtelingenstromen.* Leuven, Departement Politieke Wetenschappen, Afdeling Internationale Betrekkingen, K.U.Leuven, 1999, 321 blz. + bijlagen.

41. VAN HOOTEGEM Geert, *De draaglijke traagheid van het management. Productie- en Personeelsbeleid in de industrie.* Leuven, Departement Sociologie, K.U.Leuven, 1999, 471 blz. + bijlagen.

42. VANDEBOSCH Heidi, *Een geboeid publiek? Het gebruik van massamedia door gedetineerden*. Leuven, Departement Communicatiewetenschap, K.U.Leuven, 1999, 375 blz. + bijlagen.

43. VAN HOVE Hildegard, *De weg naar binnen. Spiritualiteit en zelfontplooiing*. Leuven, Departement Sociologie, K.U.Leuven, 2000, 369 blz. + bijlagen.

44. HUYS Rik, *Uit de band? De structuur van arbeidsverdeling in de Belgische autoassemblagebedrijven.* Leuven, Departement Sociologie, K.U.Leuven, 2000, 464 blz. + bijlagen.

45. VAN RUYSSEVELDT Joris, *Het belang van overleg. Voorwaarden voor macroresponsieve CAO-onderhandelingen in de marktsector.* Leuven, Departement Sociologie, K.U.Leuven, 2000, 349 blz. + bijlagen.

46. DEPAUW Sam, *Cohesie in de parlementsfracties van de regeringsmeerderheid. Een vergelijkend onderzoek in België, Frankrijk en het Verenigd Koninkrijk (1987-97).* Leuven, Departement Politieke Wetenschappen, K.U.Leuven, 2000, 510 blz. + bijlagen.

47. BEYERS Jan, *Het maatschappelijk draagvlak van het Europees beleid en het einde van de permissieve consensus. Een empirisch onderzoek over politiek handelen in een meerlagig politiek stelsel.* Leuven, Departement Politieke Wetenschappen, K.U.Leuven, 2000, 269 blz. + bijlagen.

48. VAN DEN BULCK Hilde, *De rol van de publieke omroep in het project van de moderniteit. Een analyse van de bijdrage van de Vlaamse publieke televisie tot de creatie van een nationale cultuur en identiteit (1953-1973).* Leuven, Departement Communicatiewetenschap, K.U.Leuven, 2000, 329 blz. + bijlagen.

49. STEEN Trui, *Krachtlijnen voor een nieuw personeelsbeleid in de Vlaamse gemeenten. Een studie naar de sturing en implementatie van veranderingsprocessen bij de overheid.* Leuven, Departement Politieke Wetenschappen, K.U.Leuven, 2000, 340 blz. + bijlagen.

50. PICKERY Jan, *Applications of Multilevel Analysis in Survey Data Quality Research. Random Coefficient Models for Respondent and Interviewer Effects.* Leuven, Departement Sociologie, K.U.Leuven, 2000, 200 blz. + bijlagen.

51. DECLERCQ Aniana (Anja), *De complexe zoektocht tussen orde en chaos. Een sociologische studie naar de differentiatie in de institutionele zorgregimes voor dementerende ouderen.* Leuven, Departement Sociologie, K.U.Leuven, 2000, 260 blz. + bijlagen.

52. VERSCHRAEGEN Gert, *De maatschappij zonder eigenschappen. Systeemtheorie, sociale differentiatie en moraal.* Leuven, Departement Sociologie, K.U.Leuven, 2000, 256 blz. + bijlagen.

53. DWIKARDANA Sapta, *The Political Economy of Development and Industrial Relations in Indonesia under the New Order Government.* Leuven, Departement Sociologie, K.U.Leuven, 2001, 315 blz. + bijlagen.

54. SAUER Tom, *Nuclear Inertia. US Nuclear Weapons Policy after the Cold War (1990-2000).* Leuven, Departement Politieke Wetenschappen, K.U.Leuven, 2001, 358 blz. + bijlagen.

55. HAJNAL Istvan, *Classificatie in de sociale wetenschappen. Een evaluatie van de nauwkeurigheid van een aantal clusteranalysemethoden door middel van simulaties.* Leuven, Departement Sociologie, K.U.Leuven, 2001, 340 blz. + bijlagen.

56. VAN MEERBEECK Anne, *Het doopsel: een familieritueel. Een sociologische analyse van de betekenissen van dopen in Vlaanderen.* Leuven, Departement Sociologie, K.U.Leuven, 2001, 338 blz. + bijlagen.

57. DE PRINS Peggy, *Zorgen om zorg(arbeid). Een vergelijkend onderzoek naar oorzaken van stress en maatzorg in Vlaamse rusthuizen.* Leuven, Departement Sociologie, K.U.Leuven, 2001, 363 blz. + bijlagen.

58. VAN BAVEL Jan, *Demografische reproductie en sociale evolutie: geboortebeperking in Leuven 1840-1910.* Leuven, Departement Sociologie, K.U.Leuven, 2001, 362 blz. + bijlagen.

59. PRINSLOO Riana, *Subnationalism in a Cleavaged Society with Reference to the Flemish Movement since 1945.* Leuven, Departement Politieke Wetenschappen, K.U.Leuven, 2001, 265 blz. + bijlagen.

60. DE LA HAYE Jos, *Missed Opportunities in Conflict Management. The Case of Bosnia-Herzegovina (1987-1996).* Leuven, Departement Politieke Wetenschappen, K.U.Leuven, 2001, 283 blz. + bijlagen.

61. ROMMEL Ward, *Heeft de sociologie nood aan Darwin? Op zoek naar de verhouding tussen evolutiepsychologie en sociologie.* Leuven, Departement Sociologie, K.U.Leuven, 2002, 287 blz. + bijlagen.

62. VERVLIET Chris, *Vergelijking tussen Duits en Belgisch federalisme, ter toetsing van een neofunctionalistisch verklaringsmodel voor bevoegdheidsverschuivingen tussen nationale en subnationale overheden: een analyse in het economisch beleidsdomein.* Leuven, Departement Politieke Wetenschappen, K.U.Leuven, 2002, 265 blz. + bijlagen.

63. DHOEST Alexander, *De verbeelde gemeenschap: Vlaamse tv-fictie en de constructie van een nationale identiteit.* Leuven, Departement Communicatiewetenschap, K.U.Leuven, 2002, 384 blz. + bijlagen.

64. VAN REETH Wouter, *The Bearable Lightness of Budgeting. The Uneven Implementation of Performance Oriented Budget Reform Across Agencies.* Leuven, Departement Politieke Wetenschappen, K.U.Leuven, 2002, 380 blz. + bijlagen.

65. CAMBRé Bart, *De relatie tussen religiositeit en etnocentrisme. Een contextuele benadering met cross-culturele data.* Leuven, Departement Sociologie, K.U.Leuven, 2002, 257 blz. + bijlagen.

66. SCHEERS Joris, *Koffie en het aroma van de stad. Tropische (re-)productiestructuren in ruimtelijk perspectief. Casus centrale kustvlakte van Ecuador.* Leuven, Departement Sociologie, K.U.Leuven, 2002, 294 blz. + bijlagen.

67. VAN ROMPAEY Veerle, *Media on / Family off? An integrated quantitative and qualitative investigation into the implications of Information and Communication Technologies (ICT) for family life.* Leuven, Departement Communicatiewetenschap, K.U.Leuven, 2002, 232 blz. + bijlagen.

68. VERMEERSCH Peter, *Roma and the Politics of Ethnicity in Central Europe. A Comparative Study of Ethnic Minority Mobilisation in the Czech Republic, Hungary and Slovakia in the 1990s.* Leuven, Departement Politieke Wetenschappen, K.U.Leuven, 2002, 317 blz. + bijlagen.

69. GIELEN Pascal, *Pleidooi voor een symmetrische kunstsociologie. Een sociologische analyse van artistieke selectieprocessen in de sectoren van de hedendaagse dans en de beeldende kunst in Vlaanderen.* Leuven, Departement Sociologie, K.U.Leuven, 2002, 355 blz. + bijlagen.

70. VERHOEST Koen, *Resultaatgericht verzelfstandigen. Een analyse vanuit een verruimd principaal-agent perspectief.* Leuven, Departement Politieke Wetenschappen, K.U.Leuven, 2002, 352 blz. + bijlagen.

71. LEFèVRE Pascal, *Willy Vandersteens Suske en Wiske in de krant (1945-1971). Een theoretisch kader voor een vormelijke analyse van strips.* Leuven, Departement Communicatiewetenschap, K.U.Leuven, 2003, 186 blz. (A3) + bijlagen.

72. WELKENHUYSEN-GYBELS Jerry, *The Detection of Differential Item Functioning in Likert Score Items.* Leuven, Departement Sociologie, K.U.Leuven, 2003, 222 blz. + bijlagen.

73. VAN DE PUTTE Bart, *Het belang van de toegeschreven positie in een moderniserende wereld. Partnerkeuze in 19de-eeuwse Vlaamse steden (Leuven, Aalst en Gent).* Leuven, Departement Sociologie, K.U.Leuven, 2003, 425 blz. + bijlagen.

74. HUSTINX Lesley, *Reflexive modernity and styles of volunteering: The case of the Flemish Red Cross volunteers.* Leuven, Departement Sociologie, K.U.Leuven, 2003, 363 blz. + bijlagen.

75. BEKE Wouter, *De Christelijke Volkspartij tussen 1945 en 1968. Breuklijnen en pacificatiemechanismen in een catch-allpartij.* Leuven, Departement Politieke Wetenschappen, K.U.Leuven, 2004, 423 blz. + bijlagen.

76. WAYENBERG Ellen, *Vernieuwingen in de Vlaamse centrale - lokale verhoudingen: op weg naar partnerschap? Een kwalitatieve studie van de totstandkoming en uitvoering van het sociale impulsbeleid.* Leuven, Departement Politieke Wetenschappen, K.U.Leuven, 2004, 449 blz. + bijlagen.

77. MAESSCHALCK Jeroen, *Towards a Public Administration Theory on Public Servants' Ethics. A Comparative Study.* Leuven, Departement Politieke Wetenschappen, K.U.Leuven, 2004, 374 blz. + bijlagen.

78. VAN HOYWEGHEN Ine, *Making Risks. Travels in Life Insurance and Genetics*. Leuven, Departement Sociologie, K.U.Leuven, 2004, 248 blz. + bijlagen.

79. VAN DE WALLE Steven, *Perceptions of Administrative Performance: The Key to Trust in Government?* Leuven, Departement Politieke Wetenschappen, K.U.Leuven, 2004, 261 blz. + bijlagen.

80. WAUTERS Bram, *Verkiezingen in organisaties.* Leuven, Departement Politieke Wetenschappen, K.U.Leuven, 2004, 707 blz. + bijlagen.

81. VANDERLEYDEN Lieve, *Het Belgische/Vlaamse ouderenbeleid in de periode 1970-1999 gewikt en gewogen.* Leuven, Departement Sociologie, K.U.Leuven, 2004, 386 blz. + bijlagen.

82. HERMANS Koen, *De actieve welvaartsstaat in werking. Een sociologische studie naar de implementatie van het activeringsbeleid op de werkvloer van de Vlaamse OCMW's*. Leuven, Departement Sociologie, K.U.Leuven, 2005, 300 blz. + bijlagen.

83. BEVIGLIA ZAMPETTI Americo, *The Notion of 'Fairness' in International Trade Relations: the US Perspective.* Leuven, Departement Politieke Wetenschappen, K.U.Leuven, 2005, 253 blz. + bijlagen.

84. ENGELEN Leen, *De verbeelding van de Eerste Wereldoorlog in de Belgische speelfilm (1913-1939).* Leuven, Departement Communicatiewetenschap, K.U.Leuven, 2005, 290 blz. + bijlagen.

85. VANDER WEYDEN Patrick, *Effecten van kiessystemen op partijsystemen in nieuwe democratieën.* Leuven, Departement Sociologie, K.U.Leuven/K.U.Brussel, 2005, 320 blz. + bijlagen.

86. VAN HECKE Steven, *Christen-democraten en conservatieven in de Europese Volkspartij. Ideologische verschillen, nationale tegenstellingen en transnationale conflicten.* Leuven, Departement Politieke Wetenschappen, K.U.Leuven, 2005, 306 blz. + bijlagen.

87. VAN DEN VONDER Kurt, *"The Front Page" in Hollywood. Een geïntegreerde historisch-poëticale analyse.* Leuven, Departement Communicatiewetenschap, K.U.Leuven, 2005, 517 blz. + bijlagen.

88. VAN DEN TROOST Ann, *Marriage in Motion. A Study on the Social Context and Processes of Marital Satisfaction.* Leuven, Departement Sociologie, K.U.Leuven/R.U.Nijmegen, Nederland, 2005, 319 blz. + bijlagen.

89. ERTUGAL Ebru, *Prospects for regional governance in Turkey on the road to EU membership: Comparison of three regions.* Leuven, Departement Politieke Wetenschappen, K.U.Leuven, 2005, 384 blz. + bijlagen.

90. BENIJTS Tim, *De keuze van beleidsinstrumenten. Een vergelijkend onderzoek naar duurzaam sparen en beleggen in België en Nederland.* Leuven, Onderzoekseenheid: Instituut voor de Overheid [IO], K.U.Leuven, 2005, 501 blz. + bijlagen

91. MOLLICA Marcello, *The Management of Death and the Dynamics of an Ethnic Conflict: The Case of the 1980-81 Irish National Liberation Army (INLA) Hunger Strikes in Northern Ireland.* Leuven, Onderzoekseenheid: Instituut voor Internationaal en Europees Beleid [IIEB], K.U.Leuven, 2005, 168 blz. + bijlagen

92. HEERWEGH Dirk, *Web surveys. Explaining and reducing unit nonresponse, item nonresponse and partial nonresponse.* Leuven, Onderzoekseenheid: Centrum voor Sociologie [CeSO], K.U.Leuven, 2005, 350 blz. + bijlagen

93. GELDERS David (Dave), *Communicatie over nog niet aanvaard beleid: een uitdaging voor de overheid?* Leuven, Onderzoekseenheid: School voor Massacommunicatieresearch [SMC], K.U.Leuven, 2005, (Boekdeel 1 en 2) 502 blz. + bijlagenboek

94. PUT Vital, *Normen in performance audits van rekenkamers. Een casestudie bij de Algemene Rekenkamer en het National Audit Office.* Leuven, Onderzoekseenheid: Instituut voor de Overheid [IO], K.U.Leuven, 2005, 209 blz. + bijlagen

95. MINNEBO Jurgen, *Trauma recovery in victims of crime: the role of television use.* Leuven, Onderzoekseenheid: School voor Massacommunicatieresearch [SMC], K.U.Leuven, 2006, 187 blz. + bijlagen

96. VAN DOOREN Wouter, *Performance Measurement in the Flemish Public Sector: A Supply and Demand Approach*. Leuven, Onderzoekseenheid: Instituut voor de Overheid [IO], K.U.Leuven, 2006, 245 blz. + bijlagen

97. GIJSELINCKX Caroline, *Kritisch Realisme en Sociologisch Onderzoek. Een analyse aan de hand van studies naar socialisatie in multi-etnische samenlevingen.* Leuven, Onderzoekseenheid: Centrum voor Sociologie [CeSO], K.U.Leuven, 2006, 305 blz. + bijlagen

98. ACKAERT Johan, *De burgemeestersfunctie in België. Analyse van haar legitimering en van de bestaande rolpatronen en conflicten.* Leuven, Onderzoekseenheid: Instituut voor de Overheid [IO], K.U.Leuven, 2006, 289 blz. + bijlagen

99. VLEMINCKX Koen, *Towards a New Certainty: A Study into the Recalibration of the Northern-Tier Conservative Welfare States from an Active Citizens Perspective.* Leuven, Onderzoekseenheid: Centrum voor Sociologie [CeSO], K.U.Leuven, 2006, 381 blz. + bijlagen

100. VIZI Balázs, *Hungarian Minority Policy and European Union Membership. An Interpretation of Minority Protection Conditionality in EU Enlargement.* Leuven, Onderzoekseenheid: Insituut voor Internationaal en Europees Beleid [IIEB], K.U.Leuven, 2006, 227 blz. + bijlagen

101. GEERARDYN Aagje, *Het goede doel als thema in de externe communicatie. Bedrijfscommunicatie met een sociaal gezicht?* Leuven, Onderzoekseenheid: School voor Massacommunicatieresearch [SMC], K.U.Leuven, 2006, 272 blz. + bijlagen

102. VANCOPPENOLLE Diederik, *De ambtelijke beleidsvormingsrol verkend en getoetst in meervoudig vergelijkend perspectief. Een two-level analyse van de rol van Vlaamse ambtenaren in de Vlaamse beleidsvorming.* Leuven, Onderzoekseenheid: Instituut voor de Overheid [IO], K.U.Leuven, 2006, 331 blz. + bijlagenboek

103. DOM Leen, *Ouders en scholen: partnerschap of (ongelijke) strijd? Een kwalitatief onderzoek naar de relatie tussen ouders en scholen in het lager onderwijs.* Leuven, Onderzoekseenheid: Centrum voor Sociologisch Onderzoek [CeSO], K.U.Leuven, 2006, 372 blz. + bijlagen

104. NOPPE Jo, *Van kiesprogramma tot regeerakkoord. De beleidsonderhandelingen tussen de politieke partijen bij de vorming van de Belgische federale regering in 1991-1992 en in 2003.* Leuven, Onderzoekseenheid: Centrum voor Politicologie [CePO], K.U.Leuven, 2006, 364 blz. + bijlagen

105. YASUTOMI Atsushi, *Alliance Enlargement: An Analysis of the NATO Experience.* Leuven, Onderzoekseenheid: Instituut voor Internationaal en Europees Beleid [IIEB], K.U.Leuven, 2006, 294 blz. + bijlagen

106. VENTURINI Gian Lorenzo, *Poor Children in Europe. An Analytical Approach to the Study of Poverty in the European Union 1994-2000.* Dipartimento di Scienze Sociali, Università degli studi di Torino, Torino (Italië) / Onderzoekseenheid: Centrum voor Sociologisch Onderzoek [CeSO], K.U.Leuven, 2006, 192 blz. + bijlagen

107. EGGERMONT Steven, *The impact of television viewing on adolescents' sexual socialization*. Onderzoekseenheid: School voor Massacommunicatieresearch [SMC], K.U.Leuven, 2006, 244 blz. + bijlagen

108. STRUYVEN Ludovicus, *Hervormingen tussen drang en dwang. Een sociologisch onderzoek naar de komst en de gevolgen van marktwerking op het terrein van arbeidsbemiddeling.* Onderzoekseenheid: Centrum voor Sociologisch Onderzoek [CeSO], K.U.Leuven, 2006, 323 blz. + bijlagen

109. BROOS Agnetha, *De digitale kloof in de computergeneratie: ICT-exclusie bij adolescenten.* School voor Massa-communicatieresearch [SMC], K.U.Leuven, 2006, 215 blz. + bijlagen

110. PASPALANOVA Mila, *Undocumented and Legal Eastern European Immigrants in Brussels*. Onderzoekseenheid: Centrum voor Sociologisch Onderzoek [CeSO], K.U.Leuven/K.U.Brussel, 383 blz. + bijlagen

111. CHUN Kwang Ho, *Democratic Peace Building in East Asia in Post-Cold War Era. A Comparative Study*. Onderzoekseenheid: Instituut voor Internationaal en Europees Beleid [IIEB], K.U.Leuven, 2006, 297 blz. + bijlagen

112. VERSCHUERE Bram, *Autonomy & Control in Arm's Length Public Agencies: Exploring the Determinants of Policy Autonomy.* Onderzoekseenheid: Instituut voor de Overheid [IO], K.U.Leuven, 2006, 363 blz. + bijlagenboek

113. VAN MIERLO Jan, *De rol van televisie in de cultivatie van percepties en attitudes in verband met geneeskunde en gezondheid.* Onderzoekseenheid: School voor Massa-communicatieresearch [SMC], K.U.Leuven, 2007, 363 blz. + bijlagen

114. VENCATO Maria Francesca, *The Development Policy of the CEECs: the EU Political Rationale between the Fight Against Poverty and the Near Abroad.* Onderzoekseenheid: Instituut voor Internationaal en Europees Beleid [IIEB], K.U.Leuven, 2007, 276 blz. + bijlagen

115. GUTSCHOVEN Klaas, *Gezondheidsempowerment en de paradigmaverschuiving in de gezondheidszorg: de rol van het Internet.* Onderzoekseenheid: School voor Massa-communicatieresearch [SMC], K.U.Leuven, 2007, 330 blz. + bijlagen

116. OKEMWA James, *Political Leadership and Democratization in the Horn of Africa (1990-2000)* Onderzoekseenheid: Instituut voor Internationaal en Europees Beleid [IIEB], K.U.Leuven, 2007, 268 blz. + bijlagen

117. DE COCK Rozane, *Trieste Vedetten? Assisenverslaggeving in Vlaamse kranten.* Onderzoekseenheid: School voor Massa-communicatieresearch [SMC], K.U.Leuven, 2007, 257 blz. + bijlagen

118. MALLIET Steven, *The Challenge of Videogames to Media Effect Theory.* Onderzoekseenheid: Centrum voor Mediacultuur en communicatietechnologie [CMC], K.U.Leuven, 2007, 187 blz. + bijlagen

119. VANDECASTEELE Leen, *Dynamic Inequalities. The Impact of Social Stratification Determinants on Poverty Dynamics in Europe.* Onderzoekseenheid: Centrum voor Sociologisch Onderzoek [CeSO], K.U.Leuven, 246 blz. + bijlagen

120. DONOSO Veronica, *Adolescents and the Internet: Implications for Home, School and Social Life*. Onderzoekseenheid: School voor Massa-communicatieresearch [SMC], K.U.Leuven, 2007, 264 blz. + bijlagen

121. DOBRE Ana Maria, *Europeanisation From A Neo-Institutionalist Perspective: Experiencing Territorial Politics in Spain and Romania.* Onderzoekseenheid: Instituut voor Internationaal en Europees Beleid [IIEB], K.U.Leuven, 2007, 455 blz. + bijlagen

122. DE WIT Kurt, *Universiteiten in Europa in de 21e eeuw. Netwerken in een veranderende samenleving.* Onderzoekseenheid: Centrum voor Sociologisch Onderzoek [CeSO], K.U.Leuven, 2007,362 blz. + bijlagen

123. CORTVRIENDT Dieter, *The Becoming of a Global World: Technology / Networks / Power / Life*. Onderzoekseenheid: Centrum voor Sociologisch Onderzoek [CeSO], K.U.Leuven, 2008, 346 blz. + bijlagen

124. VANDER STICHELE Alexander, *De culturele alleseter? Een kwantitatief en kwalitatief onderzoek naar 'culturele omnivoriteit' in Vlaanderen.* Onderzoekseenheid: Centrum voor Sociologisch Onderzoek [CeSO], K.U.Leuven, 2008, 414 blz. + bijlagen(boek)

125. LIU HUANG Li-chuan, *A Biographical Study of Chinese Restaurant People in Belgium: Strategies for Localisation.* Onderzoekseenheid: Centrum voor Sociologisch Onderzoek [CeSO], K.U.Leuven, 2008, 365 blz. + bijlagen

126. DEVILLé Aleidis, *Schuilen in de schaduw. Een sociologisch onderzoek naar de sociale constructie van verblijfsillegaliteit*. Onderzoekseenheid: Centrum voor Sociologisch Onderzoek [CeSO], K.U.Leuven, 2008, 469 blz. + bijlagen

127. FABRE Elodie, *Party Organisation in a multi-level setting: Spain and the United Kingdom*. Onderzoekseenheid: Centrum voor Politicologie [CePO], K.U.Leuven, 2008, 282 blz. + bijlagen

128. PELGRIMS Christophe, *Politieke actoren en bestuurlijke hervormingen. Een stakeholder benadering van Beter Bestuurlijk Beleid en Copernicus.* Onderzoekseenheid: Instituut voor de Overheid [IO], K.U.Leuven, 2008, 374 blz. + bijlagen

129. DEBELS Annelies, *Flexibility and Insecurity. The Impact of European Variants of Labour Market Flexibility on Employment, Income and Poverty Dynamics.* Onderzoekseenheid: Centrum voor Sociologisch Onderzoek [CeSO], K.U.Leuven, 2008, 366 blz. + bijlagen

130. VANDENABEELE Wouter, *Towards a public administration theory of public service motivation*. Onderzoekseenheid: Instituut voor de Overheid [IO], K.U.Leuven, 2008, 306 blz. + bijlagen

131. DELREUX Tom, *The European union negotiates multilateral environmental agreements: an analysis of the internal decision-making process.* Onderzoekseenheid: Instituut voor Internationaal en Europees Beleid [IIEB], K.U.Leuven, 2008, 306 blz. + bijlagen

132. HERTOG Katrien, *Religious Peacebuilding: Resources and Obstacles in the Russian Orthodox Church for Sustainable Peacebuilding in Chechnya.* Onderzoekseenheid: Instituut voor Internationaal en Europees Beleid [IIEB], K.U.Leuven, 2008, 515 blz. + bijlagen

133. PYPE Katrien, *The Making of the Pentecostal Melodrama. Mimesis, Agency and Power in Kinshasa's Media World (DR Congo).* Onderzoekseenheid: Instituut voor Antropologie in Afrika [IARA], K.U.Leuven, 2008, 401 blz. + bijlagen + dvd

134. VERPOEST Lien, *State Isomorphism in the Slavic Core of the Commonwealth of Independent States (CIS). A Comparative Study of Postcommunist Geopolitical Pluralism in Russia, Ukraine and Belarus.* Onderzoekseenheid: Instituut voor Internationaal en Europees Beleid [IIEB], K.U.Leuven, 2008, 412 blz. + bijlagen

135. VOETS Joris, *Intergovernmental relations in multi-level arrangements: Collaborative public management in Flanders.* Onderzoekseenheid: Instituut voor de Overheid [IO], K.U.Leuven, 2008, 260 blz. + bijlagen

136. LAENEN Ria, *Russia's 'Near Abroad' Policy and Its Compatriots (1991-2001). A Former Empire In Search for a New Identity.* Onderzoekseenheid: Instituut voor Internationaal en Europees Beleid [IIEB], K.U.Leuven, 2008, 293 blz. + bijlagen

137. PEDZIWIATR Konrad Tomasz, *The New Muslim Elites in European Cities: Religion and Active Social Citizenship Amongst Young Organized Muslims in Brussels and London.* Onderzoekseenheid: Centrum voor Sociologisch Onderzoek [CeSO], K.U.Leuven, 2008, 483 blz. + bijlagen

138. DE WEERDT Yve, *Jobkenmerken en collectieve deprivatie als verklaring voor de band tussen de sociale klasse en de economische attitudes van werknemers in Vlaanderen.* Onderzoekseenheden: Centrum voor Sociologisch Onderzoek [CeSO] en Onderzoeksgroep Arbeids-, Organisatie- en Personeelspsychologie, K.U.Leuven, 2008, 155 blz. + bijlagen

139. FADIL Nadia, *Submitting to God, submitting to the Self. Secular and religious trajectories of second generation Maghrebi in Belgium.* Onderzoekseenheid: Centrum voor Sociologisch Onderzoek [CeSO], K.U.Leuven, 2008, 370 blz. + bijlagen

140. BEUSELINCK Eva, *Shifting public sector coordination and the underlying drivers of change: a neo-institutional perspective.* Onderzoekseenheid: Instituut voor de Overheid [IO], K.U.Leuven, 2008, 283 blz. + bijlagen

141. MARIS Ulrike, *Newspaper Representations of Food Safety in Flanders, The Netherlands and The United Kingdom. Conceptualizations of and Within a 'Risk Society'.* Onderzoekseenheid: School voor Massa-communicatieresearch [SMC], K.U.Leuven, 2008, 159 blz. + bijlagen

142. WEEKERS Karolien, *Het systeem van partij- en campagnefinanciering in België: een analyse vanuit vergelijkend perspectief.* Onderzoekseenheid: Centrum voor Politicologie [CePO], K.U.Leuven, 2008, 248 blz. + bijlagen

143. DRIESKENS Edith, *National or European Agents? An Exploration into the Representation Behaviour of the EU Member States at the UN Security Council.* Onderzoekseenheid: Instituut voor Internationaal en Europees Beleid [IIEB], K.U.Leuven, 2008, 221 blz. + bijlagen

144. DELARUE Anne, *Teamwerk: de stress getemd? Een multilevelonderzoek naar het effect van organisatieontwerp en teamwerk op het welbevinden bij werknemers in de metaalindustrie*. Onderzoekseenheid: Centrum voor Sociologisch Onderzoek [CeSO], K.U.Leuven, 2009, 454 blz. + bijlagen

145. MROZOWICKI Adam, *Coping with Social Change. Life strategies of workers in Poland after the end of state socialism.* Onderzoekseenheid: Centrum voor Sociologisch Onderzoek [CeSO], K.U.Leuven, 2009, 383 blz. + bijlagen

146. LIBBRECHT Liselotte, *The profile of state-wide parties in regional elections. A study of party manifestos: the case of Spain.* Onderzoekseenheid: Centrum voor Politicologie [CePO], K.U.Leuven, 2009, 293 blz. + bijlagen

147. SOENEN Ruth, *De connecties van korte contacten. Een etnografie en antropologische reflectie betreffende transacties, horizontale bewegingen, stedelijke relaties en kritische indicatoren.* Onderzoekseenheid: Interculturalism, Migration and Minorities Research Centre [IMMRC], K.U.Leuven, 2009, 231 blz. + bijlagen

148. GEERTS David, *Sociability Heuristics for Interactive TV. Supporting the Social Uses of Television.* Onderzoekseenheid: Centrum voor Mediacultuur en Communicatietechnologie [CMC], K.U.Leuven, 2009, 201 blz. + bijlagen

149. NEEFS Hans, *Between sin and disease. A historical-sociological study of the prevention of syphilis and AIDS in Belgium (1880-2000).* Onderzoekseenheid: Centrum voor Sociologisch Onderzoek [CeSO], K.U.Leuven, 2009, 398 blz. + bijlagen

150. BROUCKER Bruno, *Externe opleidingen in overheidsmanangement en de transfer van verworven kennis. Casestudie van de federale overheid.* Onderzoekseenheid: Instituut voor de Overheid [IO], K.U.Leuven, 2009, 278 blz. + bijlagen

151. KASZA Artur, *Policy Networks and the Regional Development Strategies in Poland. Comparative case studies from three regions.* Onderzoekseenheid: Instituut voor Internationaal en Europees Beleid [IIEB], K.U.Leuven, 2009, 485 blz. + bijlagen

152. BEULLENS Kathleen, *Stuurloos? Een onderzoek naar het verband tussen mediagebruik en risicogedrag in het verkeer bij jongeren.* Onderzoekseenheid: School voor Massacommunicatieresearch [SMC], K.U.Leuven, 2009, 271 blz. + bijlagen

153. OPGENHAFFEN Michaël, *Multimedia, Interactivity, and Hypertext in Online News: Effect on News Processing and Objective and Subjective Knowledge.* Onderzoekseenheid: Centrum voor Mediacultuur en Communicatietechnologie [CMC], K.U.Leuven, 2009, 233 blz. + bijlagen

154. MEULEMAN Bart, *The influence of macro-sociological factors on attitudes toward immigration in Europe. A cross-cultural and contextual approach.* Onderzoekseenheid: Centrum voor Sociologisch Onderzoek [CeSO], K.U.Leuven, 2009, 276 blz. + bijlagen

155. TRAPPERS Ann, *Relations, Reputations, Regulations: An Anthropological Study of the Integration of Romanian Immigrants in Brussels, Lisbon and Stockholm.* Onderzoekseenheid: Interculturalism, Migration and Minorities Research Centre [IMMRC], K.U.Leuven, 2009, 228 blz. + bijlagen

156. QUINTELIER Ellen, *Political participation in late adolescence. Political socialization patterns in the Belgian Political Panel Survey.* Onderzoekseenheid: Centrum voor Politicologie [CePO], K.U.Leuven, 2009, 288 blz. + bijlagen

157. REESKENS Tim, *Ethnic and Cultural Diversity, Integration Policies and Social Cohesion in Europe. A Comparative Analysis of the Relation between Cultural Diversity and Generalized Trust in Europe.* Onderzoekseenheid: Centrum voor Politicologie [CePO], K.U.Leuven, 2009, 298 blz. + bijlagen

158. DOSSCHE Dorien, *How the research method affects cultivation outcomes.* Onderzoekseenheid: School voor Massacommunicatieresearch [SMC], K.U.Leuven, 2010, 254 blz. + bijlagen

159. DEJAEGHERE Yves, *The Political Socialization of Adolescents. An Exploration of Citizenship among Sixteen to Eighteen Year Old Belgians.* Onderzoekseenheid: Centrum voor Politicologie [CePO], K.U.Leuven, 2010, 240 blz. + bijlagen

160. GRYP Stijn, *Flexibiliteit in bedrijf - Balanceren tussen contractuele en functionele flexibiliteit.* Onderzoekseenheid: Centrum voor Sociologisch Onderzoek [CeSO], K.U.Leuven, 2010, 377 blz. + bijlagen

161. SONCK Nathalie, *Opinion formation: the measurement of opinions and the impact of the media.* Onderzoekseenheid: Centrum voor Sociologisch Onderzoek [CeSO], K.U.Leuven, 2010, 420 blz. + bijlagen

162. VISSERS Sara, *Internet and Political Mobilization. The Effects of Internet on Political Participation and Political Equality.* Onderzoekseenheid: Centrum voor Politicologie [CePO], K.U.Leuven, 2010, 374 blz. + bijlagen

163. PLANCKE Carine, « J'irai avec toi » : désirs et dynamiques du maternel dans les chants et les danses punu (Congo-Brazzaville). Onderzoekseenheden: Instituut voor Antropologie in Afrika [IARA], K.U.Leuven / Laboratoire d'Anthropologie Sociale [LAS, Parijs], EHESS, 2010, 398 blz. + bijlagenboek + DVD + CD

164. CLAES Ellen, *Schools and Citizenship Education. A Comparative Investigation of Socialization Effects of Citizenship Education on Adolescents.* Onderzoekseenheid: Centrum voor Politicologie [CePO], K.U.Leuven, 2010, 331 blz. + bijlagen

165. LEMAL Marijke, *"It could happen to you." Television and health risk perception.* Onderzoekseenheid: School voor Massacommunicatieresearch [SMC], K.U.Leuven, 2010, 316 blz. + bijlagen

166. LAMLE Nankap Elias, *Laughter and conflicts. An anthropological exploration into the role of joking relationships in conflict mediation in Nigeria: A case study of Funyallang in Tarokland.* Onderzoekseenheid: Instituut voor Antropologie in Afrika [IARA], K.U.Leuven, 2010, 250 blz. + bijlagen

167. DOGRUEL Fulya, *Social Transition Across Multiple Boundaries: The Case of Antakya on The Turkish-Syrian Border.* Onderzoekseenheid: Interculturalism, Migration and Minorities Research Centre [IMMRC], K.U.Leuven, 2010, 270 blz. + bijlagen

168. JANSOVA Eva, *Minimum Income Schemes in Central and Eastern Europe.* Onderzoekseenheid: Centrum voor Sociologisch Onderzoek [CeSO], K.U.Leuven, 2010, 195 blz. + bijlagen

169. IYAKA Buntine (François-Xavier), *Les Politiques des Réformes Administratives en République Démocratique du Congo (1990-2010).* Onderzoekseenheid: Instituut voor de Overheid [IO], K.U.Leuven, 2010, 269 blz. + bijlagen

170. MAENEN Seth, *Organizations in the Offshore Movement. A Comparative Study on Cross-Border Software Development and Maintenance Projects.* Onderzoekseenheid: Centrum voor Sociologisch Onderzoek [CeSO], K.U.Leuven, 2010, 296 blz. + bijlagen

171. FERRARO Gianluca *Domestic Implementation of International Regimes in Developing Countries. The Case of Marine Fisheries in P.R. China.* Onderzoekseenheid: Instituut voor de Overheid [IO], K.U.Leuven, 2010, 252 blz. + bijlagen

172. van SCHAIK Louise, *Is the Sum More than Its Parts? A Comparative Case Study on the Relationship between EU Unity and its Effectiveness in International Negotiations.* Onderzoekseenheid: Instituut voor Internationaal en Europees Beleid [IIEB], K.U.Leuven, 2010, 219 blz. + bijlagen

173. SCHUNZ Simon, *European Union foreign policy and its effects - a longitudinal study of the EU's influence on the United Nations climate change regime (1991-2009).* Onderzoekseenheid: Instituut voor Internationaal en Europees Beleid [IIEB], K.U.Leuven, 2010, 415 blz. + bijlagen

174. KHEGAI Janna, *Shaping the institutions of presidency in the post-Soviet states of Central Asia: a comparative study of three countries..* Onderzoekseenheid: Instituut voor Internationaal en Europees Beleid [IIEB], K.U.Leuven, 2010, 193 blz. + bijlagen

175. HARTUNG Anne, *Structural Integration of Immigrants and the Second Generation in Europe: A Study of Unemployment Durations and Job Destinations in Luxembourg, Belgium and Germany.* Onderzoekseenheid: Centrum voor Sociologisch Onderzoek [CeSO], K.U.Leuven, 2010, 285 blz. + bijlagen

176. STERLING Sara, *Becoming Chinese: Ethnic Chinese-Venezuelan Education Migrants and the Construction of Chineseness.* Onderzoekseenheid: Interculturalism, Migration and Minorities Research Centre [IMMRC], K.U.Leuven, 2010, 225 blz. + bijlagen

177. CUVELIER Jeroen, *Men, mines and masculinities in Katanga: the lives and practices of artisanal miners in Lwambo (Katanga province, DR Congo).* Onderzoekseenheid: Instituut voor Antropologie in Afrika [IARA], K.U.Leuven, 2011, 302 blz. + bijlagen

178. DEWACHTER Sara, *Civil Society Participation in the Honduran Poverty Reduction Strategy: Who takes a seat at the pro-poor table?* Onderzoekseenheid: Instituut voor Internationaal en Europees Beleid [IIEB], K.U.Leuven, 2011, 360 blz. + bijlagen

179. ZAMAN Bieke, *Laddering method with preschoolers. Understanding preschoolers' user experience with digital media.* Onderzoekseenheid: Centrum voor Mediacultuur en Communicatietechnologie [CMC], K.U.Leuven, 2011, 222 blz. + bijlagen

180. SULLE Andrew, *Agencification of Public Service Management in Tanzania: The Causes and Control of Executive Agencies.* Onderzoekseenheid: Instituut voor de Overheid [IO], K.U.Leuven, 2011, 473 blz. + bijlagen

181. KOEMAN Joyce, *Tussen commercie en cultuur: Reclamepercepties van autochtone en allochtone jongeren in Vlaanderen.* Onderzoekseenheid: Centrum voor Mediacultuur en Communicatietechnologie [CMC], K.U.Leuven, 2011, 231 blz. + bijlagen

182. GONZALEZ GARIBAY Montserrat, *Turtles and teamsters at the GATT/WTO. An analysis of the developing countries' trade-labor and trade-environment policies during the 1990s.* Onderzoekseenheid: Instituut voor Internationaal en Europees Beleid [IIEB], K.U.Leuven, 2011, 403 blz. + bijlagen

183. VANDEN ABEELE Veronika, *Motives for Motion-based Play. Less flow, more fun.* Onderzoekseenheid: Centrum voor Mediacultuur en Communicatietechnologie [CMC], K.U.Leuven, 2011, 227 blz. + bijlagen

184. MARIEN Sofie, *Political Trust. An Empirical Investigation of the Causes and Consequences of Trust in Political Institutions in Europe.* Onderzoekseenheid: Centrum voor Politicologie [CePO], K.U.Leuven, 2011, 211 blz. + bijlagen

185. JANSSENS Kim, *Living in a material world: The effect of advertising on materialism.* Onderzoekseenheid: School voor Massacommunicatieresearch [SMC], K.U.Leuven, 2011, 197 blz. + bijlagen

186. DE SCHUTTER Bob, *De betekenis van digitale spellen voor een ouder publiek.* Onderzoekseenheid: Centrum voor Mediacultuur en Communicatietechnologie [CMC], K.U.Leuven, 2011, 339 blz. + bijlagen

187. MARX Axel, *Global Governance and Certification. Assessing the Impact of Non-State Market Governance.* Onderzoekseenheid: Centrum voor Sociologisch Onderzoek [CeSO], K.U.Leuven, 2011, 140 blz. + bijlagen

188. HESTERS Delphine, *Identity, culture talk & culture. Bridging cultural sociology and integration research - a study on second generation Moroccan and native Belgian residents of Brussels and Antwerp.* Onderzoekseenheid: Centrum voor Sociologisch Onderzoek [CeSO], K.U.Leuven, 2011, 440 blz. + bijlagen

189. AL-FATTAL Rouba, *Transatlantic Trends of Democracy Promotion in the Mediterranean: A Comparative Study of EU, US and Canada Electoral Assistance in the Palestinian Territories (1995-2010).* Onderzoekseenheid: Instituut voor Internationaal en Europees Beleid [IIEB], K.U.Leuven, 2011, 369 blz. + bijlagen

190. MASUY Amandine, *How does elderly family care evolve over time? An analysis of the care provided to the elderly by their spouse and children in the Panel Study of Belgian Households 1992-2002.* Onderzoekseenheden: Centrum voor Sociologisch Onderzoek [CeSO], K.U.Leuven / Institute of Analysis of Change in Contemporary and Historical Societies [IACCHOS], Université Catholique de Louvain, 2011, 421 blz. + bijlagen

191. BOUTELIGIER Sofie, *Global Cities and Networks for Global Environmental Governance.* Onderzoekseenheid: Instituut voor Internationaal en Europees Beleid [IIEB], K.U.Leuven, 2011, 263 blz. + bijlagen

192. GÖKSEL Asuman, *Domestic Change in Turkey: An Analysis of the Extent and Direction of Turkish Social Policy Adaptation to the Pressures of European Integration in the 2000s.* Onderzoekseenheid: Instituut voor Internationaal en Europees Beleid [IIEB], K.U.Leuven, 2011, 429 blz. + bijlagen

193. HAPPAERTS Sander, *Sustainable development between international and domestic forces. A comparative analysis of subnational policies.* Onderzoekseenheid: Instituut voor Internationaal en Europees Beleid [IIEB], K.U.Leuven, 2011, 334 blz. + bijlagen

194. VANHOUTTE Bram, *Social Capital and Well-Being in Belgium (Flanders). Identifying the Role of Networks and Context.* Onderzoekseenheid: Centrum voor Politicologie [CePO], K.U.Leuven, 2011, 165 blz. + bijlagen

195. VANHEE Dieter, *Bevoegdheidsoverdrachten in België: een analyse van de vijfde staatshervorming van 2001.* Onderzoekseenheid: Instituut voor de Overheid [IO], K.U.Leuven, 2011, 269 blz. + bijlagen

196. DE VUYSERE Wilfried, *Neither War nor Peace. Civil-Military Cooperation in Complex Peace Operations.* Onderzoekseenheid: Instituut voor Internationaal en Europees Beleid [IIEB], KU Leuven, 2012, 594 blz. + bijlagen

197. TOUQUET Heleen, *Escaping ethnopolis: postethnic mobilization in Bosnia-Herzegovina.* Onderzoekseenheid: Instituut voor Internationaal en Europees Beleid [IIEB], KU Leuven, 2012, 301 blz. + bijlagen

198. ABTS Koenraad, *Maatschappelijk onbehagen en etnopopulisme. Burgers, ressentiment, vreemdelingen, politiek en extreem rechts.* Onderzoekseenheid: Centrum voor Sociologisch Onderzoek [CeSO], KU Leuven, 2012, 1066 blz. + bijlagen

199. VAN DEN BRANDE Karoline, *Multi-Level Interactions for Sustainable Development. The Involvement of Flanders in Global and European Decision-Making.* Onderzoekseenheid: Instituut voor Internationaal en Europees Beleid [IIEB], KU Leuven, 2012, 427 blz. + bijlagen

200. VANDELANOITTE Pascal, *Het spectrum van het verleden. Een visie op de geschiedenis in vier Europese arthousefilms (1965-1975).* Onderzoekseenheid: Centrum voor Mediacultuur en Communicatietechnologie [CMC], KU Leuven, 2012, 341 blz. + bijlagen

201. JUSTAERT Arnout, *The European Union in the Congolese Police Reform: Governance, Coordination and Alignment?.* Onderzoekseenheid: Instituut voor Internationaal en Europees Beleid [IIEB], KU Leuven, 2012, 247 blz. + bijlagen

202. LECHKAR Iman, *Striving and Stumbling in the Name of Allah. Neo-Sunnis and Neo-Shi'ites in a Belgian Context.* Onderzoekseenheid: Interculturalism, Migration and Minorities Research Centre [IMMRC], KU Leuven, 2012, 233 blz. + bijlagen

203. CHOI Priscilla, *How do Muslims convert to Evangelical Christianity? Case studies of Moroccans and Iranians in multicultural Brussels.* Onderzoekseenheid: Interculturalism, Migration and Minorities Research Centre [IMMRC], KU Leuven, 2012, 224 blz. + bijlagen

204. BIRCAN Tuba, *Community Structure and Ethnocentrism. A Multilevel Approach: A case Study of Flanders (Belgium)*. Onderzoekseenheid: Centrum voor Politicologie [CePO], KU Leuven, 2012, 221 blz. + bijlagen

205. DESSERS Ezra, *Spatial Data Infrastructures at work. A comparative case study on the spatial enablement of public sector processes.* Onderzoekseenheid: Centrum voor Sociologisch Onderzoek [CeSO], KU Leuven, 2012, 314 blz. + bijlagen

206. PLASQUY Eddy, *La Romería del Rocío: van een lokale celebratie naar een celebratie van lokaliteit. Transformaties en betekenisverschuivingen van een lokale collectieve bedevaart in Andalusië.* Onderzoekseenheid: Institute for Anthropological Research in Africa [IARA], KU Leuven, 2012, 305 blz. + bijlagen

207. BLECKMANN Laura E., *Colonial Trajectories and Moving Memories: Performing Past and Identity in Southern Kaoko (Namibia).* Onderzoekseenheid: Institute for Anthropological Research in Africa [IARA], KU Leuven, 2012, 394 blz. + bijlagen

208. VAN CRAEN Maarten, *The impact of social-cultural integration on ethnic minority group members' attitudes towards society*. Onderzoekseenheid: Centrum voor Politicologie [CePO], KU Leuven, 2012, 248 blz. + bijlagen

209. CHANG Pei-Fei, *The European Union in the Congolese Police Reform: Governance, Coordination and Alignment?.* Onderzoekseenheid: Instituut voor Internationaal en Europees Beleid [IIEB], KU Leuven, 2012, 403 blz. + bijlagen

210. VAN DAMME Jan, *Interactief beleid. Een analyse van organisatie en resultaten van interactieve planning in twee Vlaamse 'hot spots'.* Onderzoekseenheid: Instituut voor de Overheid [IO], KU Leuven, 2012, 256 blz. + bijlagen

211. KEUNEN Gert, *Alternatieve mainstream: een cultuursociologisch onderzoek naar selectielogica's in het Vlaamse popmuziekcircuit.* Onderzoekseenheid: Centrum voor Sociologisch Onderzoek [CeSO], KU Leuven, 2012, 292 blz. + bijlagen

212. FUNK DECKARD Julianne, *'Invisible' Believers for Peace: Religion and Peacebuilding in Postwar Bosnia-Herzegovina.* Onderzoekseenheid: Instituut voor Internationaal en Europees Beleid [IIEB], KU Leuven, 2012, 210 blz. + bijlagen

213. YILDIRIM Esma, *The Triple Challenge: Becoming a Citizen and a Female Pious Muslim. Turkish Muslims and Faith Based Organizations at Work in Belgium..* Onderzoekseenheid: Interculturalism, Migration and Minorities Research Centre [IMMRC], KU Leuven, 2012, 322 blz. + bijlagen

214. ROMMEL Jan, *Organisation and Management of Regulation. Autonomy and Coordination in a Multi-Actor Setting.* Onderzoekseenheid: Instituut voor de Overheid [IO], KU Leuven, 2012, 235 blz. + bijlagen

215. TROUPIN Steve, *Professionalizing Public Administration(s)? The Cases of Performance Audit in Canada and the Netherlands.* Onderzoekseenheid: Instituut voor de Overheid [IO], KU Leuven, 2012, 528 blz. + bijlagen

216. GEENEN Kristien, *The pursuit of pleasure in a war-weary city, Butembo, North Kivu, DRC*. Onderzoekseenheid: Institute for Anthropological Research in Africa [IARA], KU Leuven, 2012, 262 blz. + bijlagen

217. DEMUZERE Sara, *Verklarende factoren van de implementatie van kwaliteitsmanagementtechnieken. Een studie binnen de Vlaamse overheid.* Onderzoekseenheid: Instituut voor de Overheid [IO], KU Leuven, 2012, 222 blz. + bijlagen

218. EL SGHIAR Hatim, *Identificatie, mediagebruik en televisienieuws. Exploratief onderzoek bij gezinnen met Marokkaanse en Turkse voorouders in Vlaanderen.* Onderzoekseenheid: Instituut voor Mediastudies [IMS], KU Leuven, 2012, 418 blz. + bijlagen

219. WEETS Katrien, *Van decreet tot praktijk? Een onderzoek naar de invoering van elementen van prestatiebegroting in Vlaamse gemeenten.* Onderzoekseenheid: Instituut voor de Overheid [IO], KU Leuven, 2012, 343 blz. + bijlagenbundel

220. MAES Guido, *Verborgen krachten in de organisatie: een politiek model van organisatieverandering*. Onderzoekseenheid: Centrum voor Sociologisch Onderzoek [CeSO], KU Leuven, 2012, 304 blz. + bijlagen

221. VANDEN ABEELE Mariek (Maria), *Me, Myself and my Mobile: Status, Identity and Belongingness in the Mobile Youth Culture.* Onderzoekseenheid: School voor Massacommunicatieresearch [SMC], KU Leuven, 2012, 235 blz. + bijlagen

222. RAMIOUL Monique, *The map is not the territory: the role of knowledge in spatial restructuring processes.* Onderzoekseenheid: Centrum voor Sociologisch Onderzoek [CeSO], KU Leuven, 2012, 210 blz. + bijlagen

223. CUSTERS Kathleen, *Television and the cultivation of fear of crime: Unravelling the black box.* Onderzoekseenheid: School voor Massacommunicatieresearch [SMC], KU Leuven, 2012, 216 blz. + bijlagen

224. PEELS Rafael, *Facing the paradigm of non-state actor involvement: the EU-Andean region negotiation process.* Onderzoekseenheid: Instituut voor Internationaal en Europees Beleid [IIEB], KU Leuven, 2012, 239 blz. + bijlagen

225. DIRIKX Astrid, *Good Cop - Bad Cop, Fair Cop - Dirty Cop. Het verband tussen mediagebruik en de houding van jongeren ten aanzien van de politie.* Onderzoekseenheid: School voor Massacommunicatieresearch [SMC], KU Leuven, 2012, 408 blz. + bijlagen

226. VANLANGENAKKER Ine, *Uitstroom in het regionale parlement en het leven na het mandaat. Een verkennend onderzoek in Catalonië, Saksen, Schotland, Vlaanderen en Wallonië.* Onderzoekseenheid: Centrum voor Politicologie [CePO], KU Leuven, 2012, 255 blz. + bijlagen

227. ZHAO Li, *New Co-operative Development in China: An Institutional Approach.* Onderzoekseenheid: Instituut voor Internationaal en Europees Beleid [IIEB], KU Leuven, 2012, 256 blz. + bijlagen

228. LAMOTE Frederik, *Small City, Global Scopes: An Ethnography of Urban Change in Techiman, Ghana.* Onderzoekseenheid: Institute for Anthropological Research in Africa [IARA], KU Leuven, 2012, 394 blz. + bijlagen

229. SEYREK Demir Murat, *Role of the NGOs in the Integration of Turkey to the European Union.* Onderzoekseenheid: Centrum voor Politicologie [CePO], KU Leuven, 2012, 313 blz. + bijlagen