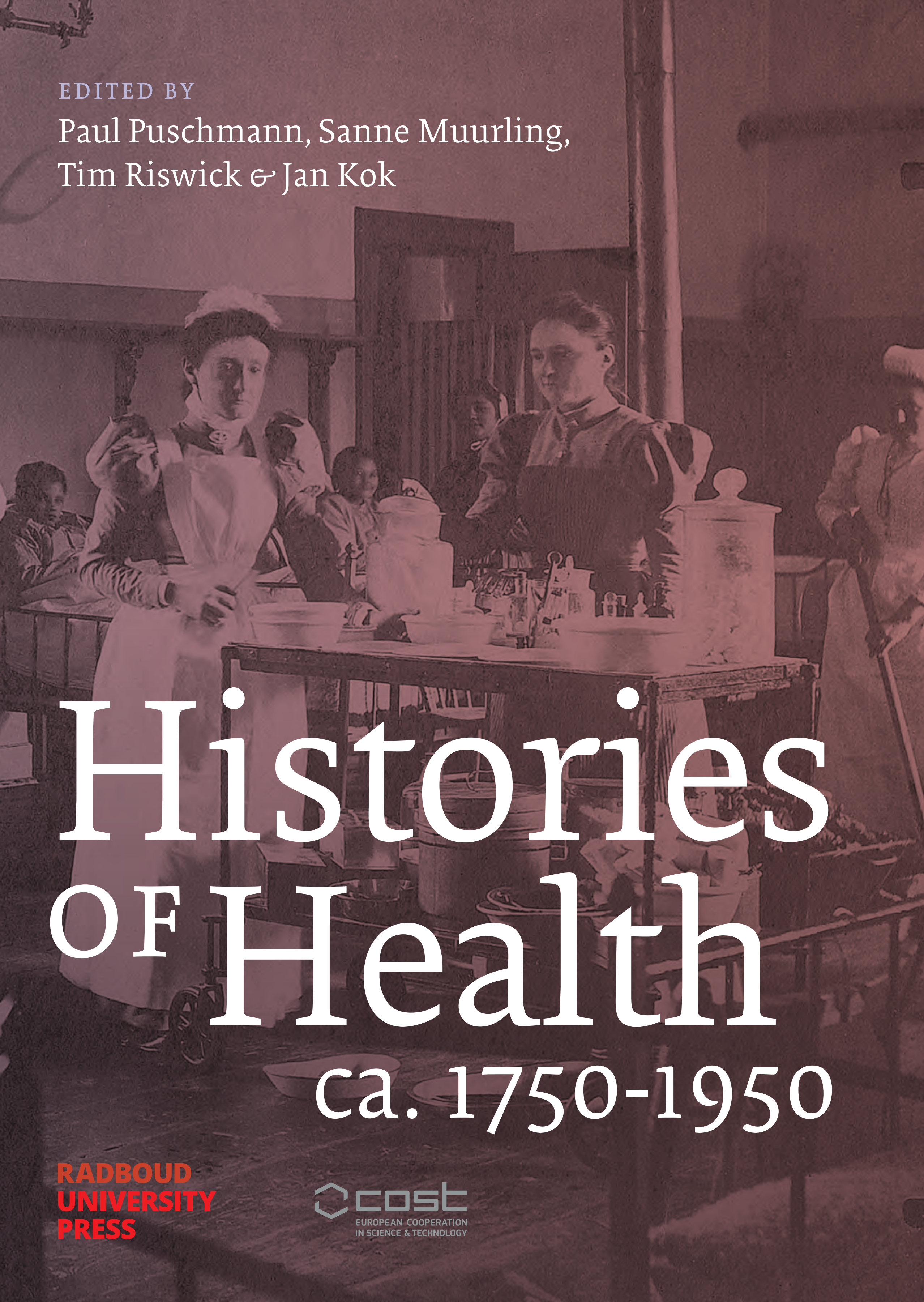

EDITED BY
Paul Puschmann, Sanne Muurling,
Tim Riswick & Jan Kok

Histories of Health ca. 1750-1950

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HISTORICAL LIFE COURSE STUDIES
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Introduction

Histories of Health

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ABSTRACT

Over the past two centuries, global health has undergone a revolutionary transformation, with life expectancy more than doubling. While the decline in the burden of infant and childhood mortality has contributed significantly to this improvement, all age groups have experienced remarkable improvements in health and longevity. Yet this health revolution has been uneven and remains incomplete. This introductory article discusses key theoretical frameworks — demographic, epidemiological, and health transition models; fundamental cause theory; and the life course approach — while also critically assessing (some of) their limitations. We highlight how health improvements have coincided with persistent and emerging inequalities, both between and within societies, shaped by such factors as socio-economic status, gender, race, and intersecting forms of disadvantages. The article explores enduring debates, such as the relative importance of nutrition, public health, and medical interventions in driving mortality decline, and identifies key knowledge gaps, including the social origins of morbidity, early-life determinants of adult health, and the historical timing of the rise in health disparities. Recent advances in historical demography, particularly life course and family-based analyses using individual-level cause-of-death data with a standardized international coding and classification system, have opened up new avenues for research. By situating contemporary inequalities in their historical context and linking individual health and disease trajectories to broader social processes, this article provides a foundation for the contributions in this issue and underscores the need for interdisciplinary, longitudinal approaches to the history of health.

Keywords: Health, History of health, Mortality decline, Health transition, Causes of death

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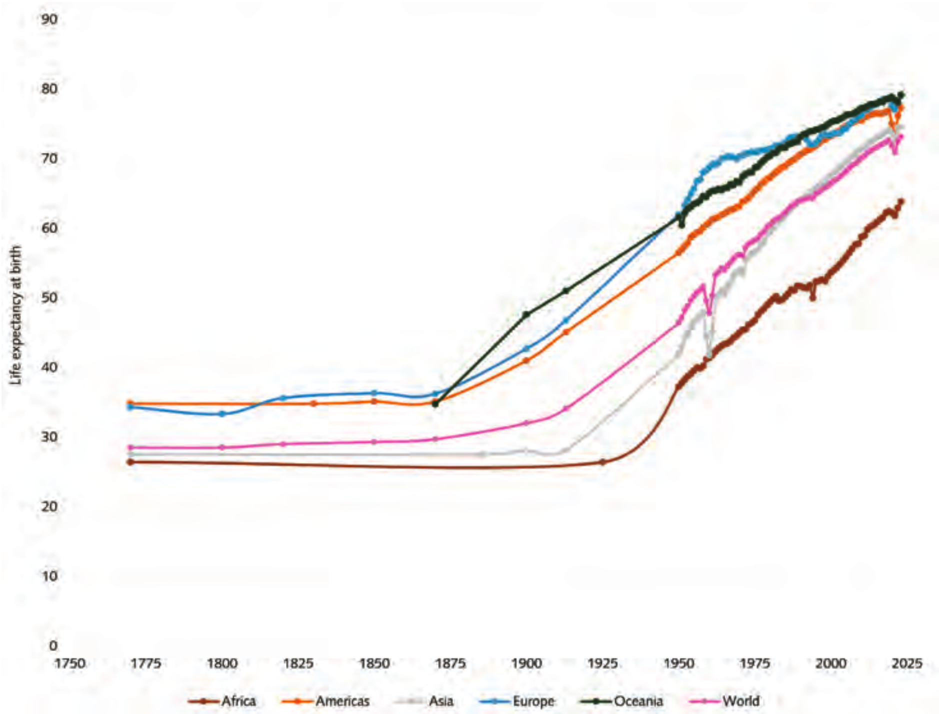
This issue is dedicated to **Professor Angélique Janssens**, who recently retired from her chair in historical demography at the Faculty of Arts and Social Sciences at Maastricht University and the Department of Economic, Social and Demographic History at Radboud University. Her pioneering research explores the history of health, labor, and living standards in the 19th and 20th centuries, with a particular focus on mortality and causes of death. Through her many publications and research projects — such as *Genes, Germs and Resources*, *Lifting the Burden of Disease*, and *Death in Amsterdam* — as well as her leadership of the SHiP (Study of Health in Port Cities) network and her role as co-editor-in-chief of *The History of the Family*, she has made a lasting contribution to the historical study of health. The contributions in this issue are all, in one way or another, inspired by her work.

1 A GLOBAL HEALTH REVOLUTION WITH UNEVEN OUTCOMES

Over the past two centuries, the world population has witnessed remarkable improvements in health and longevity. In 1800, no region in the world had a life expectancy at birth exceeding 40 years. At that time, the global average life expectancy at birth was just 28.5 years. Since then, it has more than doubled, reaching 73.2 years. While the dramatic reductions in infant and child mortality played a major role in this progress, life expectancy has increased across all age groups. Although significant disparities persist between and within countries, virtually all populations around the world have experienced substantial improvements in health. These gains began in Western countries, initially resulting in wide gaps in life expectancy between world regions. However, during the 20th century, other continents followed a similar trajectory, leading to a trend of global convergence in health outcomes. Today, life expectancy at birth exceeds 70 years in Oceania, Europe, the Americas, and Asia. Africa, while still lagging behind with a life expectancy of around 63.8 years in 2023, is showing a similar upward trend (see Figure 1). This suggests the continent is following the same long-term path of improvement observed elsewhere (Roser, 2018).

While global life expectancy has shown a broadly similar upward trend, large variation between countries continue to exist. By 2023, life expectancy at birth in affluent city-states such as Monaco, San Marino, and Hong Kong had exceeded 85 years. In stark contrast, it remained below 55 years in Chad and Nigeria (HMD, 2024; Riley, 2005; UN WPP, 2024; Zijdeman & Ribeira da Silva, 2015). This represents a gap of over 30 years, highlighting profound health disparities driven by differences in living standards, working and housing conditions, and the quality, quantity, and accessibility of food, clean drinking water, and healthcare services.

Figure 1 *Development of life expectancy at birth in the world and by world region*



Sources: UN WPP (2024); HMD (2024); Zijdeman and Ribeira da Silva (2015); Riley (2005) — with minor processing by Our World in Data. "Life expectancy at birth — Various sources — period tables" [Data set]. Human Mortality Database, "Human Mortality Database"; United Nations, "World Population Prospects"; Zijdeman and Ribeira da Silva., "Life Expectancy at birth 2"; James C. Riley, "Estimates of Regional and Global Life Expectancy, 1800-2001" [original data].

Inequalities in life expectancy are not only stark between countries but also within them — driven by socio-economic status, education, race, geography, and access to healthcare. In the U.S., Chetty et al. (2016) found that between 2001 and 2014, life expectancy at age 40 differed by over 10 years for women and nearly 15 years for men between the richest and poorest 1%. These internal disparities grew over time, particularly among low-income groups, with smoking and regional differences playing a key role. In cases where life expectancy was higher in poorer areas, it was associated with greater public spending, higher levels of education, and larger migrant populations. These findings highlight a critical point: despite overall progress, deep inequalities in health persist between and within societies.

2 FRAMING THE HEALTH REVOLUTION AND PERSISTENT HEALTH INEQUALITIES

Scholars have sought to explain both the dramatic improvements in health over the past two centuries and the persistence of health inequalities by placing them within broader theoretical frameworks. One of the earliest and most influential is the demographic transition model, developed by Thompson (1929) and Notestein (1945). This model describes changes in mortality and fertility and their relationship to population growth. It posits that all populations pass through four or five stages, transitioning from high mortality and high fertility to low mortality and low fertility. Because mortality decline typically precedes fertility decline, this leads to a period of rapid population growth: many children are still being born, but unlike in earlier times, most now survive into adulthood. As fertility begins to decline, population growth gradually slows again.

The driving forces behind the demographic transition are modernization processes, including industrialization, urbanization, as well as the linked improvements in food supply, living and working conditions, sanitation, public health policy, and availability and access to contraception. To be sure, important local variations in the timing and speed of the transition have been observed, leading several scholars to doubt the existence of a single process (Szołtysek, 2007). For instance, although industrialization started in England, fertility decline began in France, even before the fall in mortality. This requires the elaboration of new hypotheses (e.g., Cummins, 2009). Nevertheless, by and large the transition model shows a good fit with demographic developments in the world during the past two centuries. It is likely no coincidence that the rise and fall of health disparities — shaped by the uneven timing of the demographic transition — mirror the patterns of the Great Divergence (Pomeranz, 2000) and the Great Convergence (Baldwin, 2019). As economic inequality between countries declines, health disparities also narrow, pointing toward the anticipated *grand convergence* in mortality (e.g., Norheim, 2014).

The *epidemiological transition model* — developed by Abdel Omran (1971; 2005) — builds on and complements the demographic transition model. The model describes and explains in stages the transition from high mortality and low life expectancy to low mortality and high life-expectancy by adding a cause-specific explanation to the model and by including morbidity as an important factor. The model focuses on shifts in causes of death resulting from changing disease prevalence. Omran originally included three stages. The first one he called "The Age of Pestilence and Famine" and was marked by high mortality due to infectious diseases. Epidemics were the main cause of death in this period in which life expectancy at birth was low (between 20 and 40 years). Epidemics spread easily due to bad sanitation and hygiene and due to high susceptibility as a result of malnutrition, famine and war. In the second stage "The age of Receding Pandemics", that coincides with Industrialization, epidemic spikes become less frequent or even disappear. As a result, life expectancy at birth raises to 30 to 50 years and population starts to grow exponentially. In the third stage "The Age of Degenerative and Man-Made diseases" mortality decline continues further and degenerative diseases, cardiovascular disease (CVD), cancer, violence, accidents, and substance abuse supersede infectious diseases as the main causes of death, partially because of lifestyle changes. Life expectancy now starts to exceed 50 years of age. According to Omran, all countries in the world go through these stages, but the timing and pace differs. In practice he distinguished between a classic western model of the epidemiological transition, an accelerated version (most notably for Japan) and a delayed model for developing countries.

Barrett et al. (1998) extended Omran's Epidemiological model with two more stages: (1) The Age of Declining CVD Mortality, Aging and Emerging Diseases, and (2) The Age of Aspired Quality of Life with Persistent Inequalities. In the former stage deaths from cardiovascular diseases decrease further thanks to medical intervention and lifestyle changes (e.g., less and less people smoke and drink alcohol, more people exercise, etc.). However, in the long run mortality levels level off as technological advances in medicine stabilize, while new diseases emerge due to new pathogens, like Ebola or Zika, and old diseases threaten to return as they can overcome human immunity due to mutations. In the latter stage, health inequalities continue to exist, mainly along socio-economic, ethnic and gender lines.[1]

The most important framework that tries to explain (persistent) social differences in mortality is *fundamental cause theory*. Link and Phelan (1995) argue that persistent inequalities in health are the result of inequalities in terms of socio-economic status and other markers of differences, that allow some to avoid risks and to apply protective health strategies, thanks to their resources (money, power, prestige, beneficial social connections, etc.), while others (the poor, and those disadvantaged due to racial or other hierarchies) lack these resources and are therefore less able to protect their health and die at younger ages. In this context, socio-economic status is therefore defined as a fundamental cause of health inequalities (Clouston & Link, 2021). In addition to socio-economic factors, however, health inequalities also arise through other, intersecting forms of social injustice and inequality — such as race, ethnicity, gender, sexual orientation, migration status, age, and so forth. This brings in intersectionality, a concept originating from Black feminist studies. Intersectional approaches to health highlight that different systems of oppression (and privilege) — like racism, sexism, classism, and heterosexism — do not simply add up separately. Instead, they interact and reinforce each other, together shaping and deepening health inequalities (Collins, 2022). Intersectionality in health and care has received ample attention from scholars over the past decades and is arguably one of the most influential concepts in health research.

While epidemiological transition theory remains influential in (historical) health studies, the broader concept of the health transition is increasingly used to explore social, cultural, behavioral, and political factors in greater depth. Whereas the original theory provided a general outline of potential determinants, more recent work builds on and extends it by examining the complex interplay of behavior, lifestyle, and broader societal influences (Caldwell, 1993). The health transition framework puts inequality at the center of analyses. Moreover, it does not consist of fixed stages and is less linear than the epidemiological transition theory. This linearity also has been criticized by Mackenbach (2020) who shows on the basis of historical observations since the 18th century that diseases (he studied forty of them) have a rise and fall pattern and that the changes and variations in diseases are mostly man-made. When the prevalence of certain diseases decreases, others emerge or re-emerge — challenging the idea of inevitable progress. In Mackenbach's view, there is therefore no single epidemiological or health transition; rather, each disease follows its own trajectory, shaped by historically contingent factors such as changes in the virulence of infectious agents, advances in food preservation techniques, and behavioral shifts related to 'social distinction' (e.g., smoking).

The life course approach offers a way to frame historical developments in health. Building, amongst others, on the seminal work "Children of the Great Depression" by Elder (1974), epidemiologists have studied since the 1980s how behavioral, psychological and biological processes operate across an individual's life course and influence health outcomes. Especially influential has been the work by David Barker. The so-called Barker (1990) hypothesis — also known as the Developmental Origins of Health and Disease (DOHaD) or fetal programming hypothesis — states that disease in adulthood result from adverse *in utero* conditions, linking later life-outcomes in health to fetal development (i.e., programming), and thereby fundamentally changing the way how scholars perceive the relationship between early-life conditions and later life health outcomes. Barker and colleagues found for instance an association between low birth weight and the risk of dying from heart diseases, type 2 diabetes, stroke and hypertension (Barker et al., 2002). Ever since, historians and social scientists have increasingly adopted life course approaches in the field of (historical) health studies (e.g., Alter & Oris, 2010; Donrovich et al., 2014; Quaranta, 2013; Riswick, 2018).

1 Omran (1998) included these two extra stages in his later work.

3 DEBATES AND GAPS IN OUR KNOWLEDGE

3.1 REVISITING THE DRIVERS OF THE HEALTH TRANSITION

Although our understanding of the history of health has expanded significantly in recent decades, important gaps remain, and key debates continue. Central to ongoing inquiry is the need to identify the main drivers of the health transition — especially the forces responsible for the decline of specific diseases and causes of death. In this context, the longstanding debate surrounding the so-called *McKeown thesis* remains highly relevant. Thomas McKeown (1976, 1979) — a British physician, epidemiologist, and medical historian — provoked considerable controversy by arguing that the sustained decline in mortality in Western countries from the 18th to the early 20th century was primarily due to rising living standards, particularly improved nutrition. According to McKeown, advances in hygiene, public health interventions, and medical treatments played only a minor role. He claimed that infectious diseases receded largely because better nourishment strengthened resistance, casting doubt on the efficacy of quarantine, vaccination, sanitary reforms, and curative medicine.

McKeown's thesis has been widely debated and critiqued. Simon Szreter (1988), for example, challenged his conclusions by highlighting the pivotal role of public health measures — such as the introduction of sewage systems, piped water, waste removal, vaccination, and quarantine — in reducing mortality, especially in urban areas. Similarly, Anne Hardy (1993) emphasized the impact of preventive medicine and local public health efforts in mitigating the effects of the eight major diseases that afflicted England in the late 19th century. She noted a crucial distinction between diseases primarily affecting children (scarlet fever, diphtheria, measles, and whooping cough) and those that struck mostly adults (smallpox, typhoid, typhus, and tuberculosis). While preventive medicine had limited impact on the former, it was instrumental in controlling the latter. For the 20th century, Preston (1975) had already suggested that factors other than income growth, such as public health programs, maternal and child health services, and antibiotics, accounted for a sizable part of the rise in life expectancy between 1930 and 1960. Despite similar studies elsewhere, the focus of this research remains heavily weighted toward Western Europe — particularly England — while non-Western countries remain significantly understudied, although the field of colonial demography is clearly on the rise (Coghe & Widmer, 2015). Moreover, while it is widely acknowledged that rising living standards, better diets, and public health initiatives all contributed to declining mortality, their relative importance in addressing specific diseases remains unclear. Understanding how these factors varied across regions and over time remains an essential task for future research.

3.2 UNEQUAL FROM THE START?

The timing and causes of health inequalities remain, in many respects, also unclear. While *fundamental cause theory* emphasizes social inequality as a persistent driver of health disparities, this relationship is most evident in contemporary societies. Today, individuals in higher socio-economic strata enjoy longer life expectancy (easily up to 10 years more than the poor) and lower disability rates — largely due to greater material resources, higher levels of control, and stronger social participation (Marmot, 2003). But when did this social gradient in health first emerge? Some scholars argue that significant SES-related health differences only became apparent in the mid-20th century — the so-called divergence hypothesis — coinciding with the rise of modern welfare states (Bengtsson et al., 2020). Bengtsson and Dribe (2011) found little to no evidence of a consistent social gradient in Sweden before 1950. Based on this study, it would seem that the rich are better able to protect themselves from cardiovascular diseases, cancers, and accidents, than they were from infectious diseases.

However, other studies challenge the view that the social gradient in health is a recent phenomenon. Among infants, Derosas (2025) shows that, compared to mothers from the middle class and the elite, day laborer mothers experienced much higher infant mortality due to significantly earlier weaning. For children, Jaadla et al. (2017) also found clear evidence of a social gradient in health in pre-20th-century Estonia, indicating that socio-economic inequalities in child mortality already existed well before the modern welfare state. Moreover, there is increasing evidence that epidemics exacerbated existing inequalities. For the last outbreak of smallpox in the Netherlands in 1871, Muurling et al. (2023) found significant social disparities at the neighborhood level in Amsterdam. These disparities were at least partly linked to crowding and poor housing conditions among the laboring classes, as well as socially differentiated vaccination uptake. Research on cholera outbreaks observed similar tendencies (Liczbińska, 2021). This does not imply that patterns of social inequality always shaped mortality in the same way. While Schumacher and Oris (2011) identified disparities across all ages in 19th-century Switzerland, Van Poppel et al. (2005) emphasized that health inequalities may vary across age groups, being present in some but not others. Moreover, Thompson and van Ophem (2023) observed no socio-economic gradient in mortality in the Netherlands during the latter half of the 19th century. However, they did identify a clear gradient in adult height, indicating that significant health disparities may exist below the surface and remain invisible when relying solely on mortality data. Such findings underscore the importance of context — both geographic and temporal — as well as the need to consider age group and the choice of health indicator. They also highlight the importance of broadening our methodological toolkit to better capture historical health inequalities.

Looking beyond socio-economic status (SES), there is strong evidence that significant health inequalities existed long before the 20th century. One clear example is the phenomenon of infant and child death clustering. In both historical and present-day high-mortality settings — especially in many developing countries — the burden of disease and death has not been randomly distributed across families. In fact, under high mortality regimes, most families experienced no child deaths at all, while others suffered repeated losses (Edvinsson & Janssens, 2012). This pattern appears largely independent of SES. The causes remain debated: were these outcomes the result of genetic vulnerability, parental behavior, or greater exposure to infectious disease due to environmental factors or parental occupations? What is clear is that health inequality existed not only between individuals, but also between families, that it was transmitted across generations, and that SES alone cannot explain these disparities (Donrovich et al., 2018).

Sex and gender represent another important axis of inequality. Until recently, it was widely believed that (Western) Europe had largely escaped the gender discrimination seen in parts of South and East Asia, where it has contributed to excess female mortality (Lynch, 2011). However, recent research has revealed mounting evidence of "missing women" in European history as well, suggesting that discriminatory practices did occur (Beltrán Tapia & Szołtysek, 2022). Still, much remains unknown about when and why excess female mortality emerged and declined, and to what extent it was driven by overt discrimination, gendered patterns of disease exposure (e.g., through caregiving), or biological vulnerability to certain epidemics — for instance, tuberculosis — an idea occasionally proposed, especially in earlier literature (Berrut et al., 2018; Henry, 1989). More recently, it has also become clear that women have suffered from the medical tendency to treat the male body as the norm. Women have often been excluded from clinical trials and diagnostic studies, with findings based on men generalized to the entire population. Contemporary research shows that this has led to inaccurate diagnoses, delayed or ineffective treatments, and even harmful outcomes for women (Merone et al., 2022). However, historical research on this topic is still in its infancy and should aim to understand both female and male excess mortality for specific diseases.

3.3 MORBIDITY PATTERNS AND THEIR IMPLICATIONS REMAIN UNDEREXPLORED

Most historical research on health has relied on mortality data, largely due to the wide availability of sources such as vital registration, parish registers, burial records, and population registers (Edvinsson et al., 2023). In contrast, pre-20th-century data on frailty and illness remain sparse, often indirect, and typically limited to specific subgroups of the population. Commonly used sources — such as hospital records, insurance documents, military files, poor relief data, autopsy reports, medical casebooks, physicians' notes, and personal writings (letters, diaries, autobiographies) — can offer rich, detailed insights into the progression and treatment of specific diseases at the individual and family level. However, these sources pose significant challenges for generalization due to inherent selectivity. For example, individuals who are hospitalized differ in important ways from those with similar illnesses who recover at home. This selection may reflect disease severity, but it is also influenced by factors such as the availability of caregiving support, socioeconomic status (e.g., the ability to afford medical care), and geographic location (e.g., proximity to hospitals). Additionally, access to professional healthcare, and the profile of those who used it, evolved significantly over time — complicating direct comparisons across periods (Diepgrond & Riswick, forthcoming; Riswick et al., 2024).

A further challenge in historical morbidity research is the prevalence of comorbidity and multimorbidity — especially among older individuals — where multiple health conditions coexist and interact (Devos et al., 2024). Historical studies have only begun to grapple with the implications of such complexities. In general, much remains to be explored in the field of historical health research concerning frailty, illness, and disease. Key questions include: Who contracted which diseases, for how long, and with what severity? Who recovered, who died, and who lived with chronic aftereffects? How did these experiences vary by gender, age, sexual orientation, and social class? To what extent were such differences shaped by unequal access to care or disparities in the quality of healthcare?

Beyond individual outcomes, historical research must also consider broader social, economic, and demographic implications. What were the immediate and long-term effects on families when a breadwinner became ill? How did this shape the lives of spouses and children, both socially and economically? Conversely, how did chronic illness in children affect the trajectories of parents and siblings? While historical studies on the consequences of family member deaths are increasingly available (e.g., Oris & Derosas, 2002; Rosenbaum-Feldbrügge, 2020), the long-term implications of disease and illness remain understudied. Addressing these questions will require the systematic collection of longitudinal life course data on morbidity and the development of databases to support such research.

3.4 HEALTHY AGEING

While we often assume that morbidity and mortality move in tandem — an idea central to the *Compression of Morbidity* hypothesis, which posits that longer lives are accompanied by shorter periods of illness (Fries, 1980) — this is not necessarily the case. Alter and Riley (1989) used data from British Friendly Societies to demonstrate that declining mortality in the latter half of the 19th century coincided with a marked increase in age-specific morbidity. Drawing on insurance records that tracked the frequency and duration of sick leave among employees, they revealed the complex and sometimes counterintuitive relationship between mortality, health, and disease. This complexity arises in part from demographic selection. As mortality declines, more people survive into older age — many of whom, in earlier periods, would not have. While survival improves, these individuals may live longer in poor health. Moreover, ageing itself increases vulnerability to particular age-specific diseases. This explains the rising incidence of degenerative diseases like Alzheimer's and Parkinson's in ageing societies. Additionally, it has become clear in recent years that life expectancy and healthy life expectancy do not always increase in parallel. Prolonged survival may entail prolonged morbidity, which is why the WHO (2015) increasingly stresses that added years of life should be healthy years. However, healthy ageing is not guaranteed. Crimmins and Beltrán-Sánchez (2011), for example, showed that between 1998 and 2008 the number of years lived with disease and disability increased, and that progress in addressing age-related diseases had been limited. From 1960 to 1990, the incidence of heart attacks remained stable, while the likelihood of contracting cancers and diabetes actually increased.

Historical research on longevity has offered crucial insights into the phenomenon of healthy ageing. First, it is evident that genetics play a role: longevity and low morbidity often cluster within families and are transmissible across generations. Yet identifying the specific genetic markers linked to long life and health remains a difficult task (van den Berg et al., 2019). Here, historical data are especially valuable. Multigenerational datasets — whether built from population registers, genealogical databases like Geni.com, or other family-based mortality and morbidity data — can be used to identify long-lived families. These families, in turn, can be targeted for genetic sampling, providing a rare and valuable link between historical demography and genetic research. Such interdisciplinary efforts are already underway. Projects like *Genes, Germs and Resources*, funded by the Dutch Research Council (2014) and led by Angélique Janssens, exemplify successful collaboration between historical demographers and geneticists. But more such initiatives are needed to fully exploit the potential of historical data in understanding the roots of healthy ageing.

That said, genetics account for only 20–30% of the variation in longevity. The remainder is explained by behavioral, environmental, and social factors — domains in which historical research can also make substantial contributions. One promising area of investigation is the study of Blue Zones: regions where people live significantly longer and remain relatively free of chronic disease. The original Blue Zones include Okinawa (Japan), Ogliastra (Sardinia, Italy), the Nicoya Peninsula (Costa Rica), Ikaria (Greece), and Loma Linda (California, USA). Shared traits among these populations include daily low-intensity physical activity, plant-based diets, strong social networks, a sense of purpose, moderate wine consumption, and regular routines (Buettner & Skemp, 2016). While several characteristics of Blue Zone lifestyles — the so-called Power 9 — have been identified, the causal mechanisms behind their exceptional longevity remain only partially understood. Historians have contributed to some studies of Blue Zones, but systematic historical research on the phenomenon remains largely absent. Given the field's tools and methodologies, it is well positioned to deepen our understanding of how social, cultural, and environmental factors interact over time to promote healthy ageing.

4 RECENT ADVANCES IN HISTORICAL DEMOGRAPHY AND THE HISTORY OF HEALTH

Much of the pioneering research on health transitions has relied on aggregate data, linking changes in life expectancy and cause-of-death patterns to broad shifts in income, nutrition, and medical knowledge. While these studies laid essential groundwork, they offer limited insight into the underlying causal mechanisms of health change. A more nuanced understanding emerges when individual life histories can be linked to disease onset and timing of death. Even more promising is the possibility of situating these life histories within familial and social contexts, allowing researchers to explore disease clustering, mortality patterns, and intergenerational transmission.

In recent decades, substantial efforts have been made to develop longitudinal databases of individual life courses (Mandemakers et al., 2023). This life course data enables researchers to examine health within the broader context of individuals' lives, linking it to multiple, interconnected domains. One such domain is the family of orientation. For example, growing up with many siblings might dilute parental resources and attention, potentially leading to poorer health outcomes; conversely, siblings can become critical sources of social and economic support in adulthood (Donrovich et al., 2014). Similarly, early parental death has been shown to have long-term negative consequences, particularly when experienced during early childhood (Quanjer et al., 2023). Such early-life events may even shape biological and behavioral responses, such as accelerated reproductive timing, as explored by Kok and Neyrinck (2025) or more risky sexual behavior, i.e., out of wedlock fertility (Pink et al., 2020). Geographic and environmental factors represent another key domain. Health is shaped not only by local living conditions but also by migration histories. For instance, Sesma Carlos et al. (2024) highlight the role of migration trajectories in shaping health outcomes, while Puschmann (2025) demonstrates that the so-called "healthy migrant effect" may persist across generations. New lines of research are also beginning to investigate the long-term health effects of forced migration, such as the mass expulsions and deportations following the Second World War (Bauer et al., 2019).

Despite these advances, the integration of life course data to morbidity outcomes remains a relatively recent outcome. Promising early studies include cohort analyses of hospitalized populations — such as foundlings (Schneider, 2022) — as well as efforts to correlate individual life histories with specific causes of death (e.g., Thompson et al., 2020; Yeung et al., 2014). The expansion of large-scale record linkage techniques is likely to accelerate this work. Increasingly, researchers have access to rich individual-level cause-of-death data and the tools required for their systematic analysis. A particularly notable initiative in this context is the SHiP network (Studying the History of Health in Port Cities), founded by Angélique Janssens (2021). This international collaboration brings together historians and social scientists working with individual-level cause-of-death records. For a long time, scholars were constrained by highly aggregated national statistics and outdated 19th-century disease classifications. The SHiP network has addressed this gap by developing the ICD10h coding scheme, a novel classification system that aligns historical causes of death with modern disease frameworks (Reid et al., 2024). The focus on port cities is strategic: these urban hubs, historically characterized by high mobility and dense populations, were critical entry points for the transmission of disease — analogous to the role played by modern airports in the global spread of illnesses like COVID-19, Ebola, and Zika (Janssens, 2021). One important output of this initiative is the special issue "What was Killing Babies? European Comparative Research on Infant Mortality Using Individual-Level Causes of Death", published in *Historical Life Course Studies* (Janssens & Reid, forthcoming). This collection offers comparative insights into infant mortality using harmonized individual-level cause-of-death-data.

Most recently, efforts to bring together individual-level cause-of-death data across countries have been supported in SHiP+, extending the network beyond port cities. This initiative was followed by the COST Action network *The Great Leap: Multidisciplinary Approaches to Health Inequalities, 1800–2022*, coordinated by Tim Riswick. This network fosters comparative and collaborative research on specific diseases, child mortality, and broader patterns of health inequality. It also promotes the development of new standards and analytical tools for harmonizing cause-of-death data across different national and historical contexts (Mourits et al., 2024).

5 CONTRIBUTIONS IN THIS ISSUE

Several contributions in this issue build directly on the historiographical developments outlined above and most of the authors are active within the SHiP+ and GREATLEAP research networks. This is also evident in the structure of the issue, which is organized into four thematic clusters. The first, *The Administration of Death*, focuses on the historical recording of causes of death and the actors, practices, and institutions that shaped how mortality was documented and understood. These contributions shed light on the evolving authority of medical professionals, the underreporting of maternal mortality, and the professionalization of death registration systems. The second cluster, *Mortal Beginnings*, turns to infant mortality, exploring how survival in early life was shaped by social inequality, medical practices, breastfeeding behaviors, and migration status. The third cluster, *Engines of Disparity*, examines health inequalities among adults, emphasizing how socioeconomic status, religion, occupational exposure, and urban environments structured health and vulnerability to disease. The fourth and final cluster, *Lives Under Pressure*, brings together studies that analyze how epidemiological, environmental, and social stressors — ranging from seasonality and epidemics to long-term disability and demographic pressures — shaped mortality and life trajectories over time. Together, these clusters offer a rich, comparative perspective on historical health and mortality, grounded in rigorous empirical research and attentive to both continuity and change.

5.1 THE ADMINISTRATION OF DEATH

The first article in this section, "Agents of Change. The Evolution of Cause of Death Reporting in Sweden (1749–1950)" by Maria Hiltunen Maltesdotter and Sören Edvinsson (2025), offers a detailed exploration of Sweden's long-standing tradition of mortality reporting, initiated with the founding of *Tabellverket* in 1749. The authors trace how various actors — clergy, physicians, officials, and local communities — shaped the recording and interpretation of causes of death in Sweden. They present the reporting system as a dynamic information network shaped by uneven medical knowledge, shifting authority, and cultural views on disease. While clergy initially dominated, physicians gradually gained influence as medical expertise expanded. Despite limitations, the archive offers valuable insights into Sweden's changing patterns of health and mortality from the mid-18th to mid-20th century.

Alice Reid and Eilidh Garrett (2025) examine a set of linked birth and death registers from the town of Kilmarnock, Scotland, covering the period 1855–1901. Using a combination of demographic and prosopographical methods, the authors investigate why medical practitioners often assigned causes of death that obscured fatalities related to childbirth — thus masking cases of maternal mortality. By triangulating various categories of death, they conclude that Scottish doctors did not intentionally conceal maternal deaths. Rather, they often failed to recognize that these women's deaths were linked to recent childbirth.

Mayra Murkens and Wieke Metzlar (2025) also address the registration of maternal mortality, focusing on late 19th- and early 20th-century Maastricht in the Netherlands. Drawing on three sources — municipal reports, individual-level cause-of-death data from the Maastricht Death and Disease Database, and mortality rates for women linked to births within a year of their death — the authors examine whether the Netherlands' relatively low maternal mortality rates at the time reflected reality or resulted from under-registration. Their findings reveal clear evidence of under-registration: in half of the cases where women died within 42 days postpartum, the recorded causes of death were not directly attributed to childbirth.

Joana Maria Pujadas-Mora and Enrique Perdiguero-Gil (2025) assess the accuracy of causes of death for the port city of Palma, Mallorca, Spain for the period 1836–1930, using a novel lexicographical approach. They analyze processes of standardization and more precision evolved over this time period. They find that diagnostic qualifiers were more often used over time, while lengthy description of the causes of death nearly disappeared. Moreover, for teething, fever and diarrhea diagnostic discrepancies between burial and parish records are analyzed. As it turns out the latter more often lacked a cause of death, as, contrary to the burial records, these were not required to carry the name of a physician.

Kees Mandemakers (2025) examines the individuals who reported deaths on official certificates in the Netherlands between 1812 and 1939, focusing on their relationship to the deceased. He distinguishes between non-professional declarants — typically relatives or acquaintances — and professional declarants, such as undertakers and employees in the medical or health care sectors. Mandemakers concludes that the process of death reporting became increasingly professionalized over time. His analysis shows that the share of professional declarants rose steadily, from 22% in the early 19th century to 60% by the end of the study period.

5.2 MORTAL BEGINNINGS

Theo Engelen (2025) compares infant mortality in Lugang, Taiwan (1895–1945), and Nijmegen, the Netherlands (1840–1900), to test whether the demographic regimes of East Asia and Western Europe were as fundamentally different as Thomas Malthus had proposed. In particular, he engages with the interpretation put forward by James Lee and colleagues, who argue that high infant mortality in East Asia — especially among girls — functioned as a preventive check on population growth. According to this view, practices such as female infanticide were not merely responses to poverty or crisis, but deliberate strategies of family planning within a broader demographic regime. If this interpretation were accurate, one would expect significantly higher infant mortality in Lugang compared to Nijmegen, particularly among girls. However, Engelen finds only modest differences in overall infant mortality between the two cities: approximately 85% of infants survived in Nijmegen and 83% in Lugang during the respective study periods. Neonatal mortality, however, was twice as high in Lugang. Importantly, Engelen finds no evidence of excess female mortality, suggesting that female infanticide or willful neglect, if it occurred, was not a widespread or systematic form of population control.

In their pilot study, Bárbara Revuelta-Eugercios and Anne Løkke (2025) compare birth weight and preterm birth rates among infants born in 1927 in Copenhagen, Denmark, distinguishing between those delivered at home and those born in the Royal Maternity Hospital. Their analysis is based on two samples of midwives' birth records, which are almost fully preserved for the period 1861–1978 in the Danish National Archives. The study reveals that infants born in the hospital had, on average, a birth weight 300 grams lower than those born at home and were more than four times as likely to be born preterm. Revuelta-Eugercios and Løkke attribute these differences primarily to the selective nature of hospital admissions. However, they also identify considerable variation in how birth weight was measured and recorded, highlighting the need for critical source analysis — even when working with unusually rich historical datasets.

Renzo Derosas (2025) investigates the relationship between breastfeeding practices and infant mortality using individual-level longitudinal data from the Venetian population registers. He employs Cox proportional hazards models and Aalen additive regression models, the latter specifically to estimate the timing of weaning. Derosas finds a pronounced social gradient in infant mortality. The poorest mothers typically breastfed for no more than one month, whereas mothers from higher socioeconomic backgrounds continued breastfeeding for six to eight months or longer. These differences in breastfeeding duration are closely mirrored in disparities in infant mortality: infants of the poorest mothers experienced significantly higher mortality risks, which can be partly attributed to shorter breastfeeding periods.

Paul Puschmann (2025) investigates whether the well-documented healthy migrant effect — previously observed among both domestic and international migrants — extended to the next generation in 19th- and early-20th-century Antwerp. To explore this question, he analyzes infant mortality risks by parental migration status using Cox proportional hazards models on longitudinal data from the Antwerp COR*-database. His analysis reveals that infants born to domestic migrant mothers faced a 17–19% lower hazard of dying compared to those born to native-born Antwerp mothers. This effect remained statistically significant even after controlling for various infant and parental characteristics. However, no such advantage was found for infants of international migrant mothers, which Puschmann attributes to their lower level of social integration. Additionally, the migration status of the father showed no significant effect on infant mortality.

5.3 ENGINES OF DISPARITY

Ingrid van Dijk (2025) investigates the social gradient in maternal mortality in the Dutch province of Zeeland between 1812 and 1913, drawing on the LINKS dataset — a large-scale historical demographic resource for family reconstruction. Maternal mortality is defined as the death of women within 42 days of childbirth. Van Dijk uncovers a reversed social gradient in the 19th century: women from elite backgrounds, as well as those from the lower middle class and skilled working class in the early part of the century, faced higher maternal mortality than unskilled workers. This pattern, however, disappears by the early 20th century. The author discusses possible explanations for the 19th-century reversal, including the risks associated with medical intervention and differences in fertility patterns.

Elena Crinela Holom and Mihaela Hărăguş (2025) investigate social disparities in tuberculosis mortality in Transylvania between 1850 and 1914, when the region formed part of the Hungarian Kingdom. Drawing on the Historical Population Database of Transylvania, they use binary logistic regression models to compare deaths from tuberculosis with those from other causes. The study examines the influence of environmental context, occupation, gender, age, and population mobility on tuberculosis outcomes. The authors find that industrialization and migration accelerated the spread of the disease. Mortality was higher among Greek and Roman Catholics in open-type settlements (i.e., locations that were more open to interaction, mobility, and exchange with the outside world) while Calvinists and Orthodox populations experienced slightly lower rates. Notably, tuberculosis mortality showed no significant variation by gender or socio-economic status.

Joris Kok and Sanne Muurling (2025) examine the role of occupational patterns in the decline of pulmonary tuberculosis in Amsterdam during the second half of the 19th and early 20th centuries, with a focus on differences between Jewish and non-Jewish neighborhoods. The authors seek to explain the initially low tuberculosis mortality in the Jewish quarter — despite widespread poverty and overcrowding — as well as the relatively slow decline in mortality rates that followed. They argue that the early advantage stemmed from the fact that many Jews worked outdoors, reducing exposure to the airborne disease. However, with industrialization, the Jewish population became increasingly concentrated in the diamond industry, working indoors in crowded and poorly ventilated workshops. This shift in working conditions, they suggest, contributed to the slower decline in tuberculosis mortality among Amsterdam's Jewish population.

Isabelle Devos (2025) analyzes cause-specific mortality patterns in early 20th-century Antwerp, comparing them with those of Brussels, Ghent, and Liège. Despite its status as a major port city — typically associated with elevated infectious disease risks — Antwerp exhibited a health advantage over the other Belgian cities. Using a decomposition approach, Devos reveals that this overall advantage masked important vulnerabilities by age and sex. Men faced higher mortality from accidents due to the physically hazardous nature of port labor. Children bore a greater burden of infectious diseases, while women, despite having higher overall life expectancy, experienced elevated mortality during their childbearing years.

Ken Smith, Huong Meeks, Silvia Rizzi, and Rune Lindahl-Jacobsen (2025) examine how cause-specific mortality shaped sex differences in life expectancy in Utah and Denmark during the 20th and early 21st centuries. Their analysis draws on two rich data sources: the Danish Register of Causes of Death, based on death certificates, and the Utah Population Database, which links demographic, medical, and genealogical data at the individual and family level. The authors find that elevated cardiovascular mortality among men is a key driver of the female life expectancy advantage in both populations. Additional contributors to the sex gap include higher rates of suicide, homicide, and motor vehicle accidents among men. However, the female advantage is partially offset by higher cancer mortality among women during their early reproductive years.

5.4 LIVES UNDER PRESSURE

Katalin Buzasi and Tim Riswick (2025) examine seasonal mortality patterns in Amsterdam between 1812 and 1931, using data from the Amsterdam Cause-of-Death Database and monthly death counts from municipal yearbooks. For the period 1856–1891, when continuous cause-specific data are available, their analysis reveals that while airborne infectious diseases were the main contributors to elevated winter mortality, a notable winter excess remained even after these causes were excluded. This suggests that non-infectious conditions — particularly cardiovascular diseases influenced by environmental stressors rather than viral transmission — also played a key role. The authors highlight the importance of considering broader seasonal risk factors, including cold temperatures, reduced sunlight, and social inequality, in future research on mortality patterns.

Hilde Sommerseth and Evelien Walhout (2025) investigate the interaction between whooping cough and measles in Christiania (present-day Oslo) from 1826 to 1927, using annual data on morbidity, mortality, and fertility. Adopting the ecological interference framework proposed by Rohani et al. (2003), the authors examine whether epidemic dynamics shifted from out-of-phase (i.e., epidemics of the two diseases occurred at different times) to in-phase patterns (i.e., epidemics of both diseases peak at the same time) as fertility declined and the pool of susceptible individuals shrank. The analysis is divided into two periods based on crude birth rates. During the high-fertility era, the two diseases generally followed out-of-phase cycles. Contrary to theoretical predictions, this pattern persisted even during the low-fertility period. The authors explore possible explanations for this unexpected continuity, including population size thresholds necessary for transmission and data limitations.

Alphonse MacDonald, Matthias Rosenbaum-Feldbrügge, and Björn Quanjer (2025) reassess the impact of the 1918–1919 influenza pandemic in Suriname and the Dutch Caribbean islands. Despite shared colonial governance, differences in local conditions and record-keeping practices complicate direct comparison. Using data from *Koloniale Verslagen* and newly digitized civil registration records, the authors estimate approximately 2,200 influenza-related deaths in Suriname and 210 in the Dutch Caribbean. In Suriname, mortality was significantly higher among contract laborers from the Dutch East Indies and British India than among the Creole population of African descent — likely due to the latter's prior exposure and partial immunity to endemic influenza strains.

Jan Kok and Ward Neyrinck (2025) test predictions from life history theory using data from the Antwerp COR* database, covering the period 1646–1910. According to the theory, high extrinsic childhood mortality should promote earlier and riskier reproductive strategies. The authors examine whether elevated sibling mortality led individuals to marry earlier — a proxy for the onset of reproduction in this context — using Cox proportional hazard models. By stratifying their models by family, they show that the effect operates primarily at the familial level. Interestingly, individual exposure to sibling mortality is associated with delayed marriage, contradicting theoretical expectations. This finding prompts a critical re-evaluation of life history theory and its application to historical populations.

The last contribution in this issue is by Johan Junkka, Erling Häggström Gunfridsson, and Lotta Vikström (2025). These authors investigate shifts in disability prevalence during Sweden's demographic transition, focusing on the roles of population aging and changing age-specific disability rates. Drawing on longitudinal parish register data covering 194,500 individuals in Västerbotten County between 1900 and 1950, the analysis tracks trends across four disability types: sensory, physical, mental, and intellectual. Using demographic decomposition methods, the authors disentangle the influence of age structure from underlying disability rates. The results show that rising disability prevalence was driven primarily by increases in disability rates — especially mental disabilities, which grew from 0.8% to 2.5% — rather than by population aging. Notably, the increase was most marked among adults aged 25–54, challenging the assumption that aging was the dominant factor. The findings highlight the importance of social and environmental influences in shaping historical patterns of disability.

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I

The Administration of Death

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Agents of Change

The Evolution of Cause of Death Reporting in Sweden (1749–1950)

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ABSTRACT

Sweden has a long-standing tradition of recording causes of death, dating back to the establishment of *Tabellverket*, in 1749. At that time, parish ministers were responsible for documenting causes of death in parish registers and compiling related statistics, which were then used for national population data. This article explores the various agents involved in the development and maintenance of this reporting system. These include government officials (including medical scientists), practising physicians, clergy and the close social networks of the deceased. Each group played a vital role in shaping how causes of death were recorded and understood. The reporting system functioned as an information network, influenced by the differing levels of knowledge and perspectives on disease and mortality held by these agents. While the clergy initially occupied a central position in this system, the role of physicians — limited at first — gradually expanded as medical expertise and authority grew over time. Despite the limitations of the early reporting system, the preserved cause-of-death data offers invaluable insights into the changing landscape of public health, disease patterns and mortality in Sweden from the mid-18th to the mid-20th century.

Keywords: Public health statistics, Cause of death reporting, Death and burial registers, Sweden 1749–1950

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1 INTRODUCTION

One of the most significant achievements in human health over the past few centuries has been the substantial increase in life expectancy, accompanied by improved control of disease. Although the broad patterns of this transformation — including the extension of lifespan and the epidemiological shift from infectious to non-communicable diseases — are well established (Omran, 1971), the specific determinants and mechanisms underlying these changes remain only partially understood. Longitudinal data on causes of death represent an essential resource for elucidating the processes that have shaped these long-term trends in mortality and morbidity. In this contribution, we explore the historical conditions and social structures that made such developments in mortality reporting possible.

This study takes Sweden (1749–1950) as a historical case through which to examine how cause of death reporting systems emerged and evolved in practice. We focus on the roles of four key groups — government officials (including medical scientists), practising physicians, clergy, and the deceased's immediate social network — in shaping these records. These actors contributed in distinct but interrelated ways to the recording and classification of causes of death. Government officials and medical scientists established the reporting framework, which physicians applied in their diagnostic work. Clergy and members of the community also played essential roles — not only by observing symptoms, interpreting them, and relaying information to the authorities, but also by fulfilling key administrative responsibilities in the registration and transmission of cause of death information. By illuminating their contributions, we show how administrative systems, medical practice, and community knowledge converged to produce the mortality data historians rely on today. Rather than tracing the broader development of medical knowledge or diagnostic theory — though these remain important contextual factors (Janssens & Devos, 2022) — this study centers on the reporting process itself and the main actors who shaped it. Our aim is not to evaluate their practices by contemporary medical standards, but to understand them within the frameworks and expectations of their time. The sections that follow assess how these roles influenced the development and functioning of the Swedish cause of death reporting system. To understand how these roles emerged and evolved, we begin by examining the historical context in which Sweden's system of cause of death reporting developed. Section 3 analyses government officials, Section 4 analyses physicians, Section 5 analyses the central administrative role of the clergy, and Section 6 analyses how the deceased's immediate social network contributed to the broader system of cause of death reporting.

1.2 SOURCES AND METHODS

As part of our methodological approach, we draw on a diverse array of sources — including official regulations[1] (state mandates), medical journals (professional reasoning), and parish registers[2] (local reporting practices) — alongside prior research. These materials allow us to triangulate and trace how each actor group influenced the development and operation of Sweden's cause of death reporting system.

The Swedish historical databases with causes of death that we have used are the Tabverk database (see Demographic Data Base, CEDAR online resource Tabverk) which contains population statistics obtained from Sweden's parishes during the period 1749 to 1859, and the databases with individual causes of death from parish records and death certificates hosted by the Swedish research infrastructure SwedPop.

2 HISTORICAL BACKGROUND

For a long time, Sweden was a predominantly rural society, with agriculture being the primary livelihood. From 1750 to 1850, approximately 90% of the population lived in the countryside. Although urbanisation began in the mid-19th century, it progressed slowly. It was not until the 20th century that urbanisation in Sweden really accelerated. As urban growth increased, the dominant role of agriculture in the economy diminished, giving way to industrial production, the service sector, and trade, which grew in importance.

1 See Swedish National Archive regarding Tabellverket (n.d.), Church Law 1686 (1885) and Svensk Författningssamling (1860, 1911)

2 Accessible through the Demographic Data Base at Umeå University and digitised archives at the Swedish National Archives.

In parallel with these social and economic changes, significant developments occurred in the area of population records. According to the Church Law of 1686, all parish ministers were required to maintain records of births (baptisms), deaths (burials), and marriages within their parishes. They were also obligated to keep registers of all residents in their parishes. Since membership in the Swedish Lutheran Church was compulsory, this effectively meant that the entire population was included in the registers. However, until the mid-18th century, detailed reporting on the causes of death was rare. A more systematic, annual reporting of causes of death began in connection with the creation of national population statistics, which started in 1749 under the name *Tabellverket* (for more on the reporting of causes of death, see Rogers 1999; Sköld 2001, 2004). In comparison to other European countries, Sweden was notably early in implementing comprehensive national statistics, encompassing both mortality rates and cause of death statistics. Distinctively, the Swedish state employed the clergy as agents in the collection of population and health data.

To facilitate these national statistics, parish ministers were tasked with submitting population data from their parishes, based on the parish registers (see Table 1). In the preprinted forms used for this purpose, reporting causes of death took up a substantial section. The parish ministers were responsible for categorising deaths according to predefined causes. However, between 1831 and 1859, the list of pre-printed causes was limited to just two options, resulting in a significant decrease in the national reporting of causes of death during this period.

Table 1 *Overview of forms and nosologies in Sweden 1749–1951, major milestones in bold*

Year	*Number of causes of death/ Cause of death groups (No.)*	*Specific characteristics & changes*
1749	**Diseases/disease groups (23)** **Violent causes of death (10)**	**Tabellverket — The Swedish National Population Statistics Agency was introduced and included all parishes in Sweden.** **Aggregated cause of death statistics reported in preprinted forms.** **Causes of death by age and sex.**
1774	Diseases/disease groups (41) Violent causes of death (16)	Aggregated cause of death statistics. Causes of death by age and sex.
1802	Diseases/disease groups (35) Violent causes of death (15)	Aggregated cause of death statistics. Causes of death by age, sex and month.
1811	Diseases/disease groups (33) Violent causes of death (15)	Aggregated cause of death statistics. Causes of death by age, sex and month.
1821	Diseases/disease groups (34) Violent causes of death (15)	Aggregated cause of death statistics. Causes of death by age, sex and month.
1831	Diseases/disease groups (2) Violent causes of death (14)	Aggregated cause of death statistics. Causes of death by age, sex and month.
1860	**Chapters (7)** **Causes of death-diseases (92)** **Violent causes of death (23)**	**Statistics Sweden — Nosology for causes of death was introduced with disease names in both Swedish and Latin.** **Instructions and regulations:** **All deaths in cities and other places with a physician must be reported with the cause of death, based on death certificates issued by physician or midwife.** **Death certificate stated primary and secondary cause of death.**
1874	Chapters (19) Causes of death-diseases (150) Violent causes of death (21)	New nosology for causes of death. Naming in Swedish and Latin. Death certificate stated primary and secondary cause of death.
1891	Chapters (19) Causes of death-diseases (105) Violent causes of death (13)	New nosology for causes of death in Swedish and Latin. Death certificate stating main cause and contributing causes of death.
1911	**Chapters (18)** **Causes of death-diseases (87)** **Violent causes of death (14)**	**Statistics Sweden — New nosology for causes of death.** **Instructions and regulations:** **All deaths in Sweden should be reported with the cause of death, based on death certificates or verified by physician.** **Death certificate stating main cause and contributing causes of death.**
1931	Chapters (18) Causes of death-diseases (170) Violent causes of death (22)	New nosology for diseases and violent causes listed in Latin.
1951	Sweden joins ICD-6	

A further shift occurred in 1860 when the collection of population data was reorganised with the establishment of Statistics Sweden (Svensk författningssamling, 1860). Instead of the locally produced statistics, excerpts from the parish registers were now sent directly to Statistics Sweden in Stockholm. At this time, a new requirement was introduced: deaths in towns and cities where a practising physician or midwife was available had to include a reported cause of death, and a signed death certificate became mandatory.

As public health awareness grew in the late 19th and early 20th centuries, the demand for reliable health statistics intensified — driven in part by the high morbidity and mortality of tuberculosis. In response, Sweden implemented mandatory reporting of causes of death in 1911 for all parts of the country. Under this system, parish registers documented the reported causes, which were then formally certified through collaboration between parish ministers and attending physicians (Svensk författningssamling, 1910, 1911).

Thus, three key milestones mark the evolution of cause of death reporting in Sweden between 1749 and 1950. The first, in 1749, introduced cause of death reporting nationwide. The second, in 1860, saw the shift to mandatory reporting in towns and cities. The final milestone, in 1911, required complete nationwide reporting. These developments reflect the growing importance of standardised health data, which would later become integral to the work of medical scientists and governmental officials.

3 OFFICIALS IN GOVERNMENTAL AGENCIES (INCLUDING MEDICAL SCIENTISTS)

The development of a systematic approach to health data in Sweden coincided with broader advancements in medical research and governance. However, the integration of cause of death reporting into public health policy was not without challenges. Despite Sweden's slower progress in formal medical education and research infrastructure compared to much of Europe, medical scientists and officials within governmental agencies played a crucial role in shaping the healthcare system.

In 1613, the establishment of a Faculty of Medicine at Uppsala University marked the beginning of medical research and education in Sweden. To enhance their expertise, Swedish medical students often studied abroad at leading European institutions such as those in Leiden and Paris. This international exposure ensured that Swedish physicians were well-versed in the latest medical knowledge, directly benefiting from the research of prominent figures like Boerhaave and Sydenham. As a result, Swedish medical professionals were early participants in a larger European research network, where new medical discoveries were shared and applied in practice (see Lindroth 1975, 1978).

In the 18th century, medical education and scientific progress took significant steps forward in Sweden, particularly at Uppsala and Lund. The era's leading scientist, Carl Linnaeus, became a professor of medicine after studying at European institutions like Leiden. Linnaeus' work in biological classification had a profound influence, and he also developed a disease classification system (Nyström, 1988; Uddenberg, 2015). His contributions extended beyond the academic realm; he was approached to help categorise causes of death for the "Tabellverket system", though he was sceptical about the competence of the clergy to accurately report causes of death. While Linnaeus did not take on this task, another prominent physician, Abraham Bäck, took on the challenge, assuming that the clergy were familiar with the diseases listed in the reports (Nyström, 1988). These early efforts led to varying lists of causes of death, used from 1749 to 1830, which ranged from 30 to 45 categories.

During the 18th century, the spread of new medical knowledge was accelerated through the publications from the Swedish Royal Academy of Science and the earliest medical journals such as *Veckoskrift för Läkaren och Naturforskaren* (Weekly magazine for doctors and natural scientists). For example, Jenner's findings on smallpox vaccination were quickly introduced to Swedish physicians within a few years of their publication (Sköld, 1996).

The establishment of governmental agencies responsible for medicine and healthcare, for example the *Collegium Medicum*, assumed a significant role in integrating medical science into public policy.

Several physicians became involved in government work, further strengthening the connection between medical science and governance.

The role of medical science in the development of cause of death reporting became increasingly important. In the early stages, there were debates about whether the clergy, tasked with reporting causes of death, had the necessary expertise. Some suggested that a more general, popularly understood terminology might suffice. In an effort to address this, the government published books to improve the medical knowledge of clergy (Brändström, 1984; Hjelt, 1892). For instance, Hedin's (1796) publication aimed to enhance the therapeutic and diagnostic abilities of the clergy. Similarly, Nils Rosén von Rosenstein's (1764) work on childcare, a pioneering text in paediatrics, was first published as chapters in the widely distributed annual almanacs of the Academy of Sciences.

By the 19th century, Swedish medical education had become more established, with most physicians receiving their training in Sweden. Despite this, many physicians continued to travel to prominent European medical faculties to stay abreast of new developments. These insights were subsequently disseminated in prominent Swedish medical journals, such as *Hygiea* and *Svenska Läkare-Sällskapets Handlingar* (Acts from the Swedish Medical Society). Particular attention was paid to emerging threats, notably the cholera epidemics, which prompted extensive debate over competing medical theories, including miasmatic versus contagious origins. As the century progressed, significant breakthroughs — most notably in bacteriology through the pioneering work of Pasteur and Koch — were rapidly communicated within the Swedish medical community. This ongoing exchange of knowledge illustrates the continued integration of Swedish medical practice into the broader European medical community.

A major shift occurred in 1860, when a standardised nosology (disease classification) was introduced in Sweden following discussions within medical societies. This new system was more structured than the previous lists used in the *Tabellverket* forms and was influenced by international developments, particularly the work of William Farr in England (Janssens & Devos, 2022). The aim of this new nosology was to bring uniformity to cause of death reporting, ensuring consistency in terminology. Despite having aligned itself with international nosological developments — introducing new classification systems in 1874, 1891, 1911, and 1931 — Sweden refrained from adopting the International Classification of Diseases (ICD) upon its initial publication in 1893 and did not formally accede to the ICD until 1951.

As Sweden's medical sciences matured under governmental and academic guidance, the role of physicians in public health initiatives and cause of death reporting steadily gained prominence. In the following section, we examine how physicians in both urban centres and outlying districts shaped the evolution and maturation of Sweden's death reporting system between 1749 and 1950.

4 PHYSICIANS

Physicians played a crucial role in advancing healthcare accessibility and medical practice throughout Sweden, particularly as the country transitioned from limited medical access in the 18th century to a more robust healthcare system in the 20th century. Their education, professional expertise, and their growing involvement in governmental duties significantly contributed to the evolution of cause of death reporting. This section explores how physicians were integral in the development of medical records and death certificates, while also examining the challenges posed by their limited presence, especially in rural areas.

Throughout the 18th century, and to a significant extent well into the 19th century, physicians remained scarce in Sweden. In 1805, the country had fewer than 300 practising physicians, although this number had risen to over 1,300 by 1900 (Edvinsson, 1992). Accessibility to medical care in Sweden was largely confined to urban centres, with most physicians residing in towns and cities, while the majority of the population lived in rural areas. As a result, many people had limited contact with doctors for health issues.

To address the gap in healthcare provision, the government established the position of *provinsialläkare* i.e. district medical officers, in 1773 (Hjelt, 1892). These officers combined governmental duties with private practice, aiming to make medical services more accessible across Sweden.

Due to the scarcity of physicians, they were seldom involved in diagnosing causes of death, particularly in rural areas. However, with the reorganization of Swedish population statistics in 1860, it became mandatory for every death in towns and cities to be accompanied by a death certificate, which was sent to the parish minister for recording in the Death and Burial book. This practice was largely adhered to in urban areas, such as Sundsvall and Linköping. In contrast, rural parishes saw only a small proportion of deaths diagnosed by physicians. In three rural parishes outside Sundsvall, for instance, only 0.8% of the deaths were officially recorded by a physician between 1860 and 1894 (Demographic Data Base, 2025).

By 1911, Sweden had implemented nationwide reporting of causes of death (Svensk författningssamling, 1910), though this did not necessarily mean physicians were physically present at every death. In rural areas, district medical officers were tasked with reviewing the death records in the Death and Burial books monthly and requesting clarifications when needed. However, the proportion of deaths certified directly by physicians remained low. For example, in four rural parishes outside Skellefteå, only 7% of deaths between 1911 and 1950 were certified by a physician (Demographic Data Base, 2025).

Despite this, physicians were still indirectly involved in many cases. If a person had been under a physician's care in the year leading up to their death, the parish minister would often consult the physician for an assessment of the cause of death. This was not always recorded, but in some parishes these physician consultations were noted in the death records, indicating that physicians were, in many instances, consulted posthumously.

In Sweden, licensed midwives, who were more numerous than physicians, played a significant role in diagnosing perinatal and neonatal deaths. They had the closest contact with local populations, particularly in rural areas. While their role in other health matters is harder to quantify, midwives' medical expertise contributed significantly to the local medical knowledge within their communities.

To summarise, while physicians were rarely involved in cause of death reporting during the 18th century, their role in this area grew substantially in the 19th and 20th centuries, as their numbers and responsibilities expanded, particularly in urban and later rural settings.

5 THE CLERGY

The clergy in Sweden played an integral role in the administration and operation of the cause of death reporting system, making them one of the most significant agents in ensuring the machinery of death reporting functioned effectively. Their close relationships with parishioners and their frequent interactions in religious and administrative capacities positioned them as the key individuals responsible for recording and reporting causes of death throughout the studied period.

Throughout the entire period from 1749 to 1950, the clergy maintained a central role in documenting and reporting causes of death. As the primary point of contact for parishioners, ministers were involved in many aspects of their lives — meeting them during sermons, catechetical examinations, and other church-related activities. Through these interactions, ministers kept track of the population in each household, making them uniquely positioned to carry out government tasks, such as contributing to national statistics beginning in 1749. One of their primary responsibilities in this context was to record causes of death in the Death and Burial book, a task that was essential for compiling vital statistics on mortality.

However, the task of compiling cause of death statistics was not without its challenges. Rogers (1999) compared cause of death entries in the Death and Burial Books with the figures reported to *Tabellverket*. Although the discrepancies were generally small, the comparison reveals that parish ministers at times struggled to align locally reported causes of death with the standardised categories prescribed in the official statistical forms (see also Hedenborg, 2004).

These classification challenges were not the only source of difficulty for the clergy. Many ministers complained about the burden of this duty, and in 1831, they were no longer required to report the cause of death for every individual. Despite this change, some ministers continued the practice, though coverage significantly declined. With the introduction of new regulations in 1911, the requirement for complete nationwide cause of death reporting was reinstated. At this point, clergy once again had the responsibility to document the cause of death, though this information was often reviewed by district medical officers. In cases where no death certificate was presented, causes were either determined through consultation with a physician or based on information from relatives.

The clergy's influence on cause of death reporting remained strong for a significant portion of this period, but the quality and accuracy of their reports were often a point of contention. In the beginning of *Tabellverket*, they even lacked instructions on how to fill in the forms (Rogers, 1999). While some physicians doubted the clergy's ability to provide accurate diagnoses, others recognised the valuable contributions the clergy made to local health knowledge, particularly in rural areas where medical professionals were scarce.

Although parish ministers lacked formal medical training, their knowledge was not without merit. Many 18th-century theological students were deeply interested in science, and medical literature on health and diseases was distributed to all parishes to support the clergy in their reporting duties (Hjelt, 1892). This resource aimed to enhance their ability to accurately record causes of death, even though it did not equip them with the depth of medical expertise. Additionally, due to the shortage of physicians across large parts of Sweden, prominent figures in the medical field, such as Carl Trafvenfelt, advocated for a greater role for the clergy in disseminating medical knowledge (Hjelt, 1892).

From 1811 onward, grants were offered to theological students who wished to take medical courses, a program known as "prästmedicin" (clerical medicine), though interest among students was low. Nevertheless, the clergy's involvement in health-related matters extended beyond death reporting. Ministers were responsible for the "sockenapotek" (parish pharmacy), which provided basic medicines to parishioners in need, further embedding medical knowledge into their duties (Darelius, 1760).

Some parish ministers, such as Gustaf Hjortberg (1724–1776), were particularly passionate about health and medicine. Hjortberg, influenced by Carl Linnaeus, collected natural history specimens during his voyages with the Swedish East India Company and actively promoted medical knowledge in his parish. His contributions to the spread of health information demonstrate how some clergy members went beyond the basic requirements of their roles to engage with contemporary medical practices (Lindroth, 1975). Despite his expertise, however, even Hjortberg's family was not immune to the health challenges of the time, as evidenced by the early deaths of many of his children.

In summary, the clergy played a crucial role in the reporting of causes of death throughout the entire study period. Their involvement in identifying causes of death was most prominent in the 18th and early 19th centuries, but even as their direct role in cause identification diminished, they remained central to the reporting system well into the 20th century.

While the clergy's role in the reporting system was indispensable, their work often relied on the information provided by those closest to the deceased. In many instances, the immediate family or local community members were the first to identify the cause of death, especially in situations where physicians or clergy were unavailable. Their observations and knowledge were instrumental in forming the basis of the death records. The following section will explore the role of this "closest network" in the reporting and identification of causes of death, illustrating how their input contributed to the broader system of cause of death reporting.

6 THE CLOSEST NETWORK OF THE DECEASED

The immediate family and close community members of the deceased played a significant role in identifying and reporting the cause of death. Before the rise of institutionalised healthcare, deaths typically occurred at home, where the family became the initial source of information on the circumstances leading to the death. This information was then reported to the local parish minister, who would record it in the Death and Burial book.

While most individuals had limited medical knowledge, it is important to note that by the 18th century, many Swedes were literate. However, medical literature was rarely found in private homes as presented in estate inventories (Carlsson, 1972), which suggests that the family's understanding of medical conditions was rudimentary. That said, information about health and diseases was occasionally distributed through literature sent to parishes.

Many families held traditional beliefs about diseases and their causes, often using local or colloquial terms to describe conditions. In the absence of physicians, alternative medicine was frequently sought, with people relying on home remedies and local healers (Tillhagen, 1958). When reporting a death to the parish minister, families could only describe the symptoms leading up to the death as they understood them, and the final cause.

By the late 19th and early 20th centuries, however, the increasing publication of books on popular medicine (a preliminary categorisation from book titles in Swedish Book catalog (*Svensk Bok-katalog*) of medical literature directed to the public shows a fourfold increase 1865–1874 to 1886–1894) began to reach a wider audience, even in private homes (Kristenson, 1987). This trend helped broaden the medical knowledge of the general population, gradually diminishing the reliance on traditional and alternative methods.

The challenges of reconciling local understandings of diseases with the official medical terms used in the death reporting system can be seen in death records from two parishes in northern Sweden — Nora and Skog. In these parishes, the statistical records reveal a sharp rise in the reporting of *engelska sjukan* (rickets) in the 1820s, which coincided with a decrease in the reporting of diarrhoea cases. This shift aligned with the introduction of a new statistical form, which included some changes to the preprinted causes of death, as noted in Table 1 (see also Demographic Data Base, CEDAR, online database TABVERK). A comment from the local district medical officer suggested that what the locals referred to as *ris* (a popular term for rickets) actually encompassed a broader range of childhood ailments, including symptoms of weakness and diarrhoea (Medén, 1804). This example highlights a specific instance in which the local population's terminology diverged from official medical language, illustrating the potential for challenges in accurate reporting.

Another important aspect to consider is the lived experience of individuals in a high-mortality society. People were constantly exposed to illness, which led to a more direct, empirical knowledge of disease symptoms and progression. Epidemic diseases were frequent, and the population was often well-versed in identifying them, even if their terms differed from those used by medical professionals. As contact with professional medicine increased, however, the language of disease began to converge, reducing the discrepancies between popular and medical terminology.

The relationship between local knowledge and official medical classifications highlights the importance of understanding the broader context in which cause of death reporting evolved. As discussed, while the clergy and medical professionals played central roles, the immediate network surrounding the deceased — typically family and community members — was essential in shaping the data recorded.

The final section will discuss the implications of this relationship, focusing on how the evolving medical landscape and public health systems gradually improved the accuracy and reliability of cause of death reporting in Sweden.

7 CONCLUDING REFLECTIONS

In this final section we reflect on how local observers — family, community members, and clergy — interacted with official medical authorities to shape Sweden's cause of death records and how the subsequent developments in medical practice and public-health infrastructure progressively enhanced the precision and dependability of these statistics.

Over the course of the 18th and 19th century, institutional efforts to standardise medical terminology laid the groundwork for more uniform reporting. Central authorities promulgated officially sanctioned nosologies, which the clergy adopted through formal training and circulars. Consequently, local descriptions of disease events were increasingly "translated" into a coherent, shared lexicon — thereby reducing terminological variability across parishes.

Moreover, from 1860 in urban areas, the introduction of death certificates assigned by physicians and the structured lines of communication between physicians and parish ministers introduced a crucial layer of professional validation.

A further milestone arrived in 1911, when legislation mandated reporting of causes of death for all deaths nationwide. When a minister sought clarification, a formally trained physician could confirm — or correct — the provisional cause supplied by lay informants, thereby verifying the cause of death. This reform improved geographic coverage, representativeness and accuracy. Simultaneously, the 20th-century expansion of the physician workforce amplified this effect: as more medically qualified practitioners became available, their direct involvement in death certification grew, further reinforcing data quality.

Taken together, these iterative reforms did more than harmonise local and official medical lexicons — they established a robust methodological framework that underpins modern Swedish mortality statistics. By combining standardised nosologies, professional validation, and comprehensive coverage, the system delivered increasingly accurate and reliable data. Such high-quality cause of death statistics have been indispensable for evidence-based public-health planning — informing interventions against smallpox, tuberculosis, measles, and other historically significant diseases.

In conclusion, the ambition to establish a national cause of death reporting system in Sweden involved a range of agents, each with their own knowledge and perspectives. Understanding these roles and the context in which they operated is essential. Despite the limitations of the early reporting system, the preserved cause of death data offers invaluable insights into the profound changes in public health, disease, and mortality over the last few centuries.

Tracing the interplay between lay observers and medical authorities illuminates how Sweden's cause of death reporting evolved from a locally driven practice towards a nationally consistent, scientifically grounded enterprise. The preserved data not only chart centuries of demographic and epidemiological change but also continue to guide health decision-making, demonstrating the enduring value of meticulously collected mortality statistics.

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How Can a Combination of Historical Demography and Prosopographical Methods Aid the Understanding of Causes of Death?

An Illustration Using Maternal Mortality as an Example

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ABSTRACT

The growing availability of individual-level historic cause of death data is allowing increased insight into the construction of official mortality statistics, the role of changing medical provision and knowledge, and the practices of individual medical practitioners. Even the most detailed demographic data can shed little light on the particular choices that doctors made, however. We argue that a mixed methods approach, combining demographic and prosopographical approaches, can help to resolve such questions. We illustrate this using a particular conundrum relating to cause of death recording: why doctors "hid" deaths in childbirth by allocating them to causes which cannot be assumed to be maternal mortality. Triangulating different types of evidence from different sources for a particular, but fairly typical, Scottish doctor in the mid-19th century, we argue that doctors were unlikely to have deliberately obscured the maternal nature of deaths. The evidence suggests that they were more likely to have failed to realise that although they knew a woman had recently delivered, this fact was not indicated in the death register and thus the causes of death they offered could often not be identified as maternal mortality.

Keywords: Maternal mortality, Doctors, Cause of death, Civil registration, Prosopography, Scotland

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1 INTRODUCTION

This contribution brings together two of Angelique Janssens's academic interests — the understanding of causes of deaths — in particular the factors influencing what is written on death certificates (Janssens & Devos, 2022), and maternal mortality — the deaths of mothers in childbirth (Janssens & van Dongen, 2017).

Questions about how the characteristics of mortality changed as it declined (in the epidemiologic and health transitions) and why it declined (as in the McKeown hypothesis) — all rely on recorded causes of death (Colgrove, 2002; Frenk et al., 1991; Omran, 1971). To date, most research on these topics has been based on the numbers of deaths in different cause of death categories summarised in official reports, but these are ultimately derived from the decisions that individual doctors (or other informants) made and recorded on cause of death certificates as part of the death registration process. The characteristics of doctors and the influences on their choices are therefore among the many aspects of cause of death recording which complicate the analysis of changes over time. As medical knowledge and access to medical care grew, and more guidance was given to doctors on how cause of death should be certified, there was a shift in recording from vague and symptomatic causes to precise disease descriptions and an increasing propensity to list more than one cause on a certificate. Over time, these changes contributed to shifts in the way diseases or conditions were grouped into categorisations or nosologies. It has also been suggested, however, that in an era when doctors needed to maintain a private clientele the semi-public nature of the death registration process encouraged them to avoid certain causes of death which might be embarrassing for the family (such as a sexually transmitted disease) or hint at incompetence on their part (such as death during childbirth).

The growing availability of individual level cause of death data allows a variety of ways to check the accuracy of certain causes of death recorded in tables of official statistics. Historical demographers have linked the deaths of women to their recent births, finding that many of the causes of death on the certificates of women who died shortly after childbirth did not allow the death to be placed in a maternal mortality category (Kippen, 2005; Reid & Garrett, 2018). Furthermore, this was a particular problem when causes of death were certified by doctors as opposed to reported to the registrar by a relative of the deceased, something not uncommon in populations who had limited access to medical help (Reid & Garrett, 2018). Loudon (1992, pp. 34–38) has suggested that doctors may have deliberately "hid" maternal deaths, for example reporting a death as due to 'haemorrhage' rather than 'ante-partum haemorrhage', or writing 'peritonitis' or 'fever' rather than 'puerperal fever'. How might we be able to tell whether this was actually the case?

One possibility is to check against other sources. "Triangulation" refers to the process of comparing different sources to verify a finding or interpretation, and can be used when comparing the same types of data (e.g. two quantitative sources, such as the birth and death registers of a community) or comparing quantitative with qualitative (e.g. comparing statistical rates with those suggested in a narrative account such as a diary or newspaper report and the way these were discussed). The use of mixed methods involves using insights from both qualitative and quantitative data to deepen the understanding of a phenomenon (Fielding, 2012).

The historical techniques of prosopography (narrative writings about individuals, or groups of individuals) and factoid prosopography, in which systematic information about a group of people is gathered into a database (Bradley & Short, 2005), can be used to provide both qualitative and quantitative data about doctors which can be combined with historical demographic data to shed light on the doctors' interests in childbirth and maternal mortality, opinions on different techniques, and even the maternal mortality rates in their own practices. On its own such information is unlikely to reveal any motivations for 'hiding' maternal deaths as other causes. When such factoid prosopographical databases are used in combination with demographic sources, however, it becomes possible to compare the recording of maternal mortality in both sets of data; a comparison which can shed light on why doctors might not have indicated a maternal cause for women dying as a result of childbirth. This paper illustrates such a comparison and argues that more use of mixed methods such as this has significant potential to further the understanding of causes of death.

2 DATA

The demographic data we use consists of a set of linked birth and death registers for the town of Kilmarnock, Scotland, between 1855–1901 (Reid et al., 2002). Maternal deaths were identified in the death register by a clear maternal cause, or though linkage to a birth up to six weeks previously (Reid & Garrett, 2018).

Each death in the Kilmarnock death register included the name of the doctor who certified the death, and these doctors were identified in the London and Provincial Medical Directories, which also covered Scotland, for the years 1855–1901 to generate our prosopographical database. Unlike the Medical Register (the official list of qualified and registered doctors), the Medical Directory relied on doctors returning an annual set of information.[1] In addition to their address, qualifications and positions held, doctors often included their contributions to journals, and we identified and downloaded those contributions by the doctors practicing in Kilmarnock that related to pregnancy, childbirth and delivery. Most of these relate to curious case studies, including two descriptions of introversion of the uterus (Arbuckle, 1885; Macleod, 1857), a possible case of superfoetation — the situation where a second conception takes place after a first has been established (Paxton, 1866), and contributions to a debate about whether reluctance to use forceps had allowed Queen Charlotte, the wife of King George III, to die in childbirth (McLeod, 1889). Most of the journal articles do not shed any light on maternal mortality or its recording, although they can be informative about doctors' obstetric practices and the type of details they recorded. They range from long and detailed contributions spanning tens of pages, to shorter notes such as the following communication entitled "Triplets":

> Dr David MacDonald (Walmer, Kilmarnock) writes: The following case may be of some interest as showing heredity. Mrs M., aged 38, gave birth to three girls on January 27th 1893. Labour began on the preceding evening about 8 p.m., and the first girl was born with the discharge of *liquor amnii*, with little pain, about 1.30 a.m. Pains were absent for about three hours. After the membranes were ruptured the second was born, about 5.30 a.m. Pains did not recur until about 6.15, when the membranes were ruptured and the third child was born at 6.30. The placenta was removed by hand at 7 a.m., after which the uterus was firmly contracted and small. The presentations were: First, head; second, cross, shoulder, changed to breech; third, breech. Placentae, three, partially joined. Large amount of *liquor amnii* from each sac. The children weighed 8lbs, 7.5lbs, and 7.5lbs respectively, and look healthy and fairly well developed and likely enough to live. The mother's history is that it is her ninth pregnancy (four alive and four dead); that she is herself a triplet; her sister died six years ago; her brother visited her a few days ago; (her mother died at the confinement); she was brought up by a foster mother. She did not expect her confinement until a month hence. (MacDonald, 1893)

Parts of this account can be double checked by cross-referencing the linked demographic records. Births for the three triplets were indeed registered, although with a birth date of January 26th rather than 27th. The doctor had been overly optimistic about their prospects for survival, as they were registered as dying on the 3rd, 8th and 12th of February respectively. He must have sent his letter to the *British Medical Journal* (BMJ) before the 3rd of February, as he certified the infants' causes of death himself: the first from 'asthenia' and the others from 'weakness from birth'.[2]

Other contributions allow a deeper insight, including two articles by Kilmarnock doctor John Thomson which presented statistics about complications and birth outcomes in his midwifery cases, illustrated with extensive commentary and quotes from his case notes. The first of these, published in 1855, was a statistical report about his 3,300 deliveries in the previous 15 years (Thomson, 1855). He followed this up with a further report in 1864, by which time his tally had risen to 5,000 cases (Thomson, 1864).[3]

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1 If no return was received from a doctor, the information from the previous year's directory would be carried forward and marked with an asterisk.

2 The mother's history is less easy to verify. We can find the parents' marriage in December 1883 and one previous birth, as well as a possible birth to the mother prior to her marriage (October 1883), and a birth to the husband and his former wife in 1876. It is possible that some the mother's other pregnancies resulted in stillbirths or miscarriages and were therefore not recorded in the registers, or that they took place before marriage and/or outside Kilmarnock.

3 The titles of his articles refer to 3,300 and 5,000 cases respectively, but it is a little confusing as the texts also mentions "upwards of four thousand cases" and "six thousand" respectively. However it seems that the latter refer to the total cases he had ever witnessed, while statistics (and the titles) exclude those where he attended a case to aid another doctor (and possibly a period at the start of his career when he was training), and so does not have complete case notes.

Table 1 *Number of births with "complications" and maternal deaths out of Dr John Thomson's 5,000 maternal deliveries, by type of complication.*

Presentations	No. of cases	Maternal deaths	Stillborn
Breech	31		10
Face to pubes	51		
Face	9		
Shoulder	9	1	7
Placental	10	3	10
Forceps	104	2	3
Complicated with convulsions	8		6
Funis umbilicalis	7		4
Ovarian tumour	2		1
Footling	10		1
Contracted pelvis	3		2
Flooding	11	1	2
Puerperal fever	23	12	
Twin cases	41		
Stillborn*	102		102
Triplets	1		
Puerperal mania	3	2	
Perforator and crotchet	2		2
TOTAL		21	150
Number per 1,000 cases		4.2	30

Source: Thomson, 1864

*Notes: * We assume that stillbirths listed against other complications are not also listed in the stillbirth row. If they are then the stillbirth rate would be 20.4 per 1,000.*

In his first contribution, Thomson discussed the management of long labours, his approach to breech deliveries, placenta praevia and eclampsia, with detailed accounts including both happy and unhappy outcomes. In his second contribution he engaged in an extensive discussion of the relative merits and dangers of the long and short forceps. In both pieces he provided a table listing the number of different complications, together with the numbers of maternal deaths and stillbirths associated with each complication, he had encountered. The table from his second publication is reproduced as Table 1, with the addition of rows for the total numbers of maternal deaths and stillbirths, and their rates per 1,000 cases (assuming a denominator of 5,000).

3 DR THOMSON'S OBSTETRIC PRACTICE

What can we learn from Dr Thomson's statistics and discussions? First, we should consider his obstetric practice and experience. Midwifery was clearly a major part of John Thomson's general practice: his published papers suggest that he averaged 220 deliveries a year between 1840 and 1855, and 190 per year between 1855 and 1864 (an overall average of 208 per year), amounting to around four deliveries per week. This number would have been very high for a midwife in the late 19th or early 20th century — in 1915 Janet Lane-Claypon considered that 150 cases a year was 'as many as one midwife can reasonably undertake' (Lane-Claypon, 1915). Thomson's level of midwifery was clearly not unusual; in his contribution to the *BMJ* Donald McLeod, another Kilmarnock doctor, indicated that his annual average was around 175 deliveries a year over a 40 year career (McLeod, 1889). However, such doctors were unlikely to have been present for the whole of each labour. Dr Thomson's case reports indicate that he was called at various stages during the labour to assess progress, and a monthly nurse or handy-woman may have provided continuity of care, possibly even delivering some of the uncomplicated births herself (Leap & Hunter, 1993). Nevertheless, doctors most likely attended all the complicated cases for which they were booked. Dr Thomson was also sometimes called in to assist births for which a midwife or another doctor had been booked, but his publications indicate that his statistics are "exclusive of all [he saw] in the practice of others" (Thomson, 1864, p. 27). The cases reported in Dr Thomson's publications were therefore probably a reasonably representative sample of confinements in Kilmarnock.

Thomson's articles indicate that his maternal mortality ratio was around 4.2 per thousand deliveries, a rate which was only a little under the average ratio for Scotland between 1855 and 1864; and his stillbirth rate was a bit lower than the estimated rate for England and Wales at a similar time.[4] Neither suggests he was likely to be systematically hiding or under-reporting results. In fact he might have had better outcomes than other birth attendants: his case reports suggest that he was a thoughtful practitioner, arguing against the sort of "meddlesome midwifery" which reportedly characterised some "male midwives" (Thomson, 1855, pp. 129, 135). While he congratulates himself on his success, this does not prevent him from admitting to mistakes: "In one or two cases, on looking back, I have to regret that the forceps were not used, instead of leaving them to the unaided efforts of nature" (Thomson, 1855, p. 135).

4 COMPARISON OF DR THOMSON'S MATERNAL MORTALITY BASED ON HIS ARTICLES AND HIS CERTIFIED CAUSES OF DEATH

Thomson's publications allow us not only to assess his self-reported outcomes in obstetric practice, but also to compare them against the way he certified maternal deaths, as recorded in the civil registers of death in Kilmarnock. Because our dataset starts in 1861, there is only a short overlap between Thomson's reported statistics and our own. We do not know how many of the births and deaths which contribute to the table in Thomson's *BMJ* article occurred between 1861 and 1865, but if his delivery rate and maternal mortality ratios were similar to his average rate between 1855 and 1864, we would expect to see him certify around seven maternal deaths in the registers. Thompson certified his last death in Kilmarnock in 1865, and although he certified 273 causes of death during the 1861–1865 period, none of them referred directly to maternal causes. However, linkage to the Kilmarnock birth registers shows that six of the deaths he certified were of women who had given birth in the six weeks before their demise, as detailed in Table 2. The causes that Dr Thomson assigned in these six cases all strongly suggest that the women died as a direct consequence of having given birth. The 'peritonitis' and 'fever' that killed four of them were almost certainly puerperal and the 'repeated haemorrhage' was also likely to have been linked to delivery. Dr Thomson could therefore be accused of having "hidden" these six deaths by ascribing them to causes which would not allow them to be classed as maternal deaths in the official statistics. The obvious question is why would he do this? Was it, as suggested by Loudon (1992, pp. 34–38), fear of losing business or professional prestige? The Scottish death registration system and the way he talks about his midwifery cases in print offer some answers.

Table 2 *Dr John Thomson's maternal deaths, 1861–1865*

Days since birth of child	Length of last illness	Original cause of death given by Thomson
3	5 & 4 days	diarrhoea, peritonitis
4	3 days	inflammation of bowels
5	3 days	peritonitis
8	7 days	peritonitis
9	2 days	fever
31	4 weeks	repeated haemorrhage

Source: Kilmarnock linked database

4 The maternal mortality ratio for 1855–1864 in Scotland was 4.8 per 1,000 births (Loudon, 1992, p. 546), and the stillbirth rate for England and Wales 1840–1859 was 41 per 1,000 (Woods, 2009, p. 96).

Was it likely that when certifying the deaths he was suppressing information in order to shield his reputation among potential clients? If this was the case, he would probably have been most anxious to conceal information about deaths from puerperal fever. Thomson's 1855 article discusses a local outbreak of cases of puerperal fever in 1848–1849, following an epidemic of erysipelas, a closely related infection also caused by the streptococcus bacterium, making it clear that doctors were aware of the possibility that they might transfer the infection between patients, probably influenced from Semmelweis's seminal publication on the matter in the previous year. Thomson writes "The boldest and best of our practitioners were panic-struck, and, afraid lest they themselves might be the means of carrying the contagion from patient to patient, in some cases sought to escape from the responsibility by refusing attendance altogether". Despite this, by far the most common cause of maternal death in Thomson's own statistical summary of his cases was puerperal fever — he clearly was not worried about admitting to it in print.

Thomson's reports were, however, published in the *British Medical Journal* (BMJ), aimed at a professional medical audience. It is possible that doctors happy to admit to puerperal fever deaths to an audience of their peers were still concerned about revealing such mortality to their existing or potential clientele. This was certainly considered to be a problem in England and Wales, where the process of death certification and registration involved the doctor filling in a cause of death certificate and handing it to a relative of the deceased, who was then responsible for handing it to the registrar when registering the death. A parliamentary enquiry in 1893 heard evidence that this meant that doctors south of the Scottish border were often reluctant to certify causes such as delirium tremens, syphilis, cancer, and suicide (Select Committee on Death Certification, 1893, p. xlii).

In Scotland, however, the system was different: the doctor independently delivered the medical certificate of cause of death direct to the registrar, who then copied the information into the register alongside the details of the deceased supplied by the next-of-kin or another informant. The next-of-kin did not see the official cause of death, leaving the possibility open for the doctor to verbally offer them a vaguer cause, while supplying the accurate cause on the cause of death certificate. Various witnesses to the parliamentary enquiry certainly felt that a move to this system in England and Wales was likely to improve the accuracy of cause of death recording (Select Committee on Death Certification, 1893, p. xlii). As Dr Thomson practiced in Scotland, he was therefore unlikely to have been concerned that he might incur reputational damage through his certification of a maternal death.

Table 3 *Certification of maternal deaths by individual doctors, Kilmarnock 1861–1901*

					Direct maternal deaths		Puerperal fever	
Forenames	**Surname**	**Year of 1st qualification**	**First seen in K**	**Last seen in K**	**Number**	**% Allocated a maternal cause**	**Number**	**% Allocated a puerperal fever cause**
Donald	Macleod	1850	1861	1901	27	74.1	20	75.0
Alexander	Marshall	1851	1861	1894	74	85.1	55	92.7
James	Rankin	1857*	1869	1901	24	66.7	12	41.7
James	McAlister	1858*	1866	1900	23	95.7	12	100.0
William	Frew	1872	1888	1901	15	86.7	12	83.3
John Christie	McVail	1873	1874	1891	14	100.0	9	100.0
Wm. Aitken	MacLeod	1881	1882	1897	26	34.6	24	33.3

Source: Kilmarnock linked database, Medical Directory

*Notes: * indicates a Licentiate of Midwifery qualification.*

Direct maternal deaths include deaths from puerperal fever.

Only doctors with at least 10 direct maternal deaths are shown.

Another possibility is that Dr Thomson wrote 'puerperal peritonitis' or some other cause which clearly specified the maternal nature of the death on the certificate, but the registrar did not transfer the full certified cause into the register. While this is a possibility, we would then expect clear patterns by registrar or by year (as there was only one registrar at any one time in Kilmarnock), but these are not visible in the data. Instead, the patterns by doctor are far clearer, as shown in Table 3 which provides the statistics for each of the seven doctors who certified at least 10 *direct* maternal deaths in Kilmarnock between 1861 and 1901, listed in order of the year of their first qualification.[5]

In contrast to John Thomson, some doctors made the maternal nature of the deaths they certified very clear — and this is reflected in the relevant entries in the death register. This was certainly the case for all the maternal deaths certified by John Christie McVail, and for all but one of those certified by James McAlister. Although he has too few deaths to appear in this table, John Thomson (present in Kilmarnock only at the start of the period) seems to have been the worst offender, but William Aitken MacLeod (present only at the end) was a close second, obscuring the nature of two-thirds of the maternal deaths he certified. With the variation seen, it is likely that the registrars were faithfully copying the information received on the medical certificates supplied by each doctor. There seems to have been little pattern in terms of cohort of doctor, whether or not they had a qualification in midwifery[6], or the probable size of their practice (based on the number of maternal deaths per year in Kilmarnock). It is likely that instead, how doctors recorded a death was an individual and personal matter, whether a death was maternal or due to another cause (for an analysis of deaths in old age, see Reid et al., 2015).

It seems probable then, that it was sloppy certification rather than deliberate obfuscation which prevented Thomson's maternal deaths from being classified as such. We suggest that he knew that the deaths from peritonitis and fever were puerperal, and that he was simply not being specific enough in the type of peritonitis or fever. Perhaps he failed to realise that there was no reason for the coding clerks to know that the death was maternal unless he made that clear as part of the cause of death (at that time there was no systematic way in the death register to indicate a recent delivery — this would be introduced later). Similarly, he probably did not realise that identifying a death as 'repeated haemorrhage' was not specific enough to allow maternal haemorrhage to be assumed. Thomson was undoubtedly not alone. The booklets of medical certificates of cause of death, issued to medical practitioners from the earliest days of civil registration in Scotland, contained "Suggestions for Medical Practitioners on death certification" which even in the first decades of the 20th century were still exhorting doctors to ensure that if "parturition or miscarriage [had] occurred in the month before the death of the patient, the fact should be certified…" and stating that deaths from "puerperal eclampsia or puerperal septicaemia should be so described, and not merely described as eclampsia or septicaemia", suggesting that the accuracy of some doctors' certificates was less than optimal.[7]

Of course the fact that doctors were careless in their cause of death recording does not mean they did not care deeply and carefully for their patients — but the process of inspecting the body and producing a certificate was regarded by some as "burdensome and unreasonable" and some doctors may have preferred to concentrate on caring for patients before death (Select Committee on Death Certification, 1893, p. xlii).

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5 Direct maternal deaths arise as an immediate result of pregnancy or childbirth (for example pre- or post-partum haemorrhage, puerperal eclampsia, and puerperal fever). Indirect maternal deaths are due to non-pregnancy-related causes which become aggravated by the pregnancy (for example an expectant mother may be more likely to catch and die from influenza). Indirect maternal deaths were much less likely to be allocated a maternal cause, but we do not show these here as there was less consensus at the time over what should be attributed to maternal mortality. See Reid and Garrett (2018) for details of the underreporting of indirect maternal mortality in Kilmarnock; Appendix A indicates levels for individual doctors.

6 It was not until the Medical Act of 1886 that all medical training had to include obstetrics, although the Medical Act of 1858 had made it possible and some medical schools had instituted compulsory obstetric training from even earlier dates, such as Edinburgh in 1833 (Reid, 2012).

7 The Suggestions were "prepared and issued by the Registrar General for Scotland"; they changed over time. Those quoted date from the 1910s. See National Records of Scotland GRO5/814 Registration Branch Files 1855–1944, pp. 46 and 50.

5 CONCLUSIONS

In this paper we have illustrated the ways in which a combination of historical demography and prosopographical sources and approaches can shed more light on aspects of the recording of maternal death, in ways that each source on its own cannot do.

As individual level cause of death data have become increasingly available to researchers much progress has been made in our understanding of cause of death statistics. In particular they have revealed the variations and changes in terminology within the apparently uniform and stable categories used in official statistics, and allowed changes in ways that the balance of causes shifted over time to be detailed (Janssens & Devos, 2022; Reid & Garrett, 2018; Reid et al., 2015; Revuelta-Eugercios et al., 2022). Working with individual level causes forces us to confront the messy and highly variable ways that causes of death were recorded by doctors — and lay people — and to consider the influences on the choice of words they made. Although comparisons with official statistics can point to ways in which one phrase became superseded by another as medical knowledge developed or fashions changed, some of the choices made by doctors remain clouded in mystery. Better understanding of registration practices and how doctors interacted with them to choose what cause to write on the certificate is necessary if we are to achieve greater understanding of the factors influencing the manifestation of causes of death in the official record and how we should interpret them.

In this paper, we advocate for a mixed method approach combining qualitative and quantitative elements. On their own, our demographic sources (cause of death records linked to births records) reveal that many doctors managed to obscure the maternal nature of deaths to women who had recently given birth, but cannot tell us why, so we are forced to speculate about whether or not this was deliberate.

Similarly, reading the opinions of doctors and examples of their case notes is fascinating, but can only take us so far, even when organised into a systematic factoid prosopographical database. In the case of John Thomson we would know that he was interested in obstetrics and difficult births, provided extensive details of particular cases, held strong opinions on a number of related matters, and was not unusual in these matters among his peers.

It is only combining the demographic and prosopographical sources — following Thomson's career and triangulating the cases he wrote up with the demographic records — which allows the mismatch between his writings and his recording to be revealed. The incorporation of qualitative evidence from the way he wrote about his cases and mistakes gives us insight into his recording practices, and offers plausible reasons for the mismatch. Given our findings, we argue that Thomson, and Scottish doctors in general, were unlikely to have been deliberately "hiding" maternal deaths within other cause categories.

Individual level causes of death are highly detailed but deeply complex. Trends over time and differences between places are affected not only by real differences in mortality from different conditions, but by medical provision, organisation and knowledge, and by registration and recording practices. Many cause of death registers include the names of the certifying doctor. Gathering these into a database and enriching with more qualitative sources such as the writings of medical practitioners can allow researchers to compare the recording practices of individual doctors with their views on particular diseases or conditions which may, in turn, suggest why they came to write the causes of death they reported on death certificates. We feel that the mixture of approaches in this methodology has considerable potential to enhance understanding of other causes of death, particularly those which may be under-recorded such as syphilis, cancer and suicide, and how they change over time and vary between places. In this way it offers a fruitful avenue towards a deeper understanding of historical cause of death statistics.

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Hidden Maternal Mortality?

Under-Registration of Maternal Mortality in Maastricht, 1870–1910

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ABSTRACT

Maternal mortality rates in the 19th-century Netherlands were low compared to other European countries. But was this due to high-quality midwifery care or under-registration? This study examines maternal mortality registration in Maastricht by comparing data from 1870–1879 and 1900–1909 across three sources: municipal reports, individual-level causes of death from the Maastricht Death and Disease Database, and mortality rates based on women who were linked to births up to one year prior to their death. The results indicate under-registration of maternal mortality in the municipal reports. Half of the women who died within 42 days postpartum were recorded with causes unrelated to childbirth. In Maastricht, suspected cover-up causes like peritonitis and fever were not the most commonly used. Instead, tuberculosis, heart disease, and pneumonia were frequently recorded when pregnancy-related factors were omitted from the cause of death.

Keywords: Maternal mortality, Historical causes of death, 19th century

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1 INTRODUCTION

Today, the Netherlands are well-known for their exceptionally low maternal mortality rates compared to other high-income countries. With an average maternal death rate of 4 per 100,000 live births in the Netherlands in 2020, this is substantially lower than the average rate of 12 in other high-income countries (Kallianidis et al., 2022; World Bank Group, 2025). This modern-day phenomenon is also observed in the past, as the maternal mortality rates in the Netherlands were among the lowest in European countries in the late 19th century (Loudon, 1992, p. 449–452; Ory & van Poppel, 2013). The Dutch advantageous position has been linked to the widespread practise of giving birth at home and the high standard of midwifery care (Loudon, 1992; Ory & van Poppel, 2013; Shepherd et al., 2011). However, the estimation of historical maternal mortality is tricky, due to narrow definitions of maternal mortality and/or the intentionally or unintentionally obscuring of maternal deaths by contemporaries (Breathnach & Gurring, 2017; De Brouwere, 2007; Kippen, 2005; Loudon, 1992). The question remains if maternal mortality was registered with similar vigour across 19th-century Europe. Was Dutch maternal mortality truly low compared to other European countries or was the Dutch registration plagued disproportionally by accidental or even deliberate distortions?

Nowadays, maternal mortality is defined by the World Health Organization (WHO) as "The annual number of female deaths from any cause related to or aggravated by pregnancy or its management (excluding accidental or incidental causes) during pregnancy and childbirth or within 42 days of termination of pregnancy, irrespective of the duration and site of the pregnancy" (World Health Organization, 2025). These deaths can be divided into direct and indirect obstetric causes, where indirect maternal mortality is defined as "resulting from previous existing disease or disease that developed during pregnancy and not due to direct obstetric causes but were aggravated by the physiologic effects of pregnancy" (World Health Organization, 2025). Moreover, these indirect maternal deaths can occur, according to modern day definitions, up to one year after giving birth or experiencing pregnancy (World Health Organization, 2025).

In the past, no such standardized definition of maternal mortality existed. Loudon distinguished three categories of maternal mortality in historical registration. The first consists of the direct causes. These causes of death described maternal mortality rather straightforwardly, often directly mentioning the relation to pregnancy, childbirth, or the postpartum phase (Loudon, 1992, pp. 27–28, p. 35). However, other maternal mortality could remain unregistered because of the lack of a standardized definition. Thus, the other two categories Loudon identified refer to this under-registration of maternal mortality: hidden maternal deaths and associated maternal deaths.

Loudon suggests hidden maternal deaths are the causes where the recorded cause of death provides what actually led to death, but does not mention the childbirth factor, and thus uses the non-maternal equivalent of the cause of death (Loudon, 1992, p. 34). Common complications such as puerperal fever or peritonitis may have been registered without the specification of its occurrence after a birth or miscarriage, as simply fever or peritonitis, which could distort the estimations of total maternal mortality rates (Loudon, 1992, p. 35). These practices may have transpired due to an incentive for medical practitioners to hide maternal mortality. When their expertise was at stake and they were at risk of receiving the blame for maternal deaths, they may have felt the need to omit the childbirth factor from the registered cause of death.

The category of associated deaths contains causes that are not directly related to pregnancy and includes chronic illnesses such as heart disease, tuberculosis and nephritis (Kallianidis et al., 2022; Loudon, 1992, p. 28). These causes of death can be associated with childbirth due to the fact that women may have been weakened by the pregnancy or childbirth. As a result they no longer had the strength to fight the already existing disease. This category closely aligns with what is now referred to as indirect maternal mortality by the WHO (World Health Organization, 2025). In England and Wales, a separate category existed in the public reports for diseases as "not classified to pregnancy but returned as associated therewith" (Loudon, 1992, p. 30). The category of associated maternal mortality could have been quite extensive, as it has been estimated that one third of maternal deaths in Sweden was the result of a cause from this category (Högberg, 2004). In other countries, these associated deaths were often included in the total maternal mortality category, but the question still remains if *all* of these associated deaths indeed ended up to be counted as maternal deaths (Loudon, 1992, p. 30). Scalone (2014) contends that it is preferable to use time after birth to estimate maternal mortality, instead of causes of death, because these associated deaths remain unidentified when using the cause of death descriptions.

In this study, we aim to delve into the registration of maternal mortality in the late 19th and early 20th century with a case study on Maastricht. Individual-level data on this Dutch city allow for a close scrutinization of the registration practices concerning maternal deaths. Using these data, Murkens (2023) observed relatively few causes of death among adult women pertaining to childbirth, compared to other causes of death in Maastricht between 1864 and 1955. However, a closer examination showed that some women died shortly after giving birth, while their cause of death did not refer to maternal mortality at all. This indicates the presence of at least some associated maternal mortality in the Maastricht cause-of-death registration. In this study, different measures of maternal mortality in Maastricht will be examined during two sample periods (1870–1879 and 1900–1909) to shed light on the question if and to what extent associated and hidden maternal mortality could have distorted the estimations of maternal mortality rates in the Netherlands. We compare these two periods, as in the first period the existence of puerperal fever had not yet been fully accepted by the medical profession, but the professionalisation of obstetric care had taken off as per the newly enforced medical legislation in 1865. By the second period, the existence and causes of puerperal fever had been accepted by medical professionals, and professionalisation had been on its way for quite some time. Therefore, we expect less under-registration of maternal mortality in the second period, compared to the first period. We do not yet expect actual decline in maternal mortality itself, since this decline is observed in later periods, starting in the 1930s (Loudon, 2000).

In order to find out whether these numbers provided a valid estimation of maternal mortality, or if under-registration of maternal mortality indeed occurred, this study will conduct a three-step examination for the two sample periods (1870–1879 and 1900–1909). First, the municipal reports of Maastricht will be examined to create an overview of the rate of direct maternal mortality. Second, the maternal mortality rates resulting from the official reports will be compared to the mortality rates based on individual-level causes of death and on the number of women dying within a year after giving birth. Third, we will look closer into which causes of death were used to describe the mother's death and whether there were registration differences as time passed by after giving birth. This third step may provide more insight into whether the under-registered deaths may have been hidden or associated deaths.

2 IDENTIFYING MATERNAL MORTALITY IN MAASTRICHT

The first step of our study examines the death statistics of the Maastricht municipal reports. In the Netherlands, municipal reports included aggregated cause-of-death statistics form 1865 onwards, and from 1875 onwards, these statistics were compiled at the national level.[1] These national statistics have been used by Loudon (1992) to estimate the Dutch maternal mortality rates. However, the standardization of the causes of death in the Dutch registration allowed for the registration of only one cause of death. In case of multiple causes of death, for instance childbirth and tuberculosis, a doctor could only assign one cause of death. Consequently, the registration of maternal mortality in public reports is likely to have under-registered maternal mortality (Loudon, 1992, p. 28). To calculate maternal mortality rates, we used the total number of births plus the total number of stillbirths reported in the municipal reports as the denominator of maternal mortality.

To examine maternal mortality rates based on individual-level cause-of-death data we used the Maastricht Death and Disease Database (MDDD; Sociaal Historisch Centrum Limburg). This dataset includes individual-level data of 76,264 individuals that died in Maastricht, or were registered as a citizen of Maastricht, between 1864 and 1955. Not only the non-standardized, individual-level cause of death has been preserved; the dataset also includes information on the name, address, occupation, age, sex and religion of the deceased. These data were derived from a combination of death registers and death certificates and are believed to be complete (Murkens, 2023). The individual-level causes of death provide more detailed information about death than municipal reports, as they include more elaborate descriptions and often contain second, third or even more causes of death. As such, these sources can shed more light on the amount of hidden maternal deaths in Maastricht. All women that died between age 16 and 50 (i.e. the reproductive age) in the periods 1870–1879 and 1900–1909 have been selected to be included in our analysis, which resulted in an initial dataset of 1,262 female deaths.

1 See Appendix Table A1 for the categories used in the municipal reports over time.

Our other estimation of maternal mortality examines women that died up to one year after giving birth. In order to gain information on the time passed between giving birth and dying, we first linked the deaths of women between the ages 16 and 50 from the MDDD to the Maastricht birth registers. We manually assigned links based on the first name(s) and last name of the mother in the birth register and the names of the deceased women in the MDDD. If the interval between a birth date and the death of a mother was more than one year, no link was assigned. 757 (60%) of the selected women in the MDDD were registered with a partner. The names of the father (birth certificate) and partner have been used to check these assigned links. The linkage of women without a registered partner in the MDDD might be less precise compared to those with a partner. The first names in the MDDD and birth registers often included second and third names, thus creating more unique names and therefore reducing the chances of false links. Moreover, in cases where there was any doubt no links were assigned, to minimize the number of false positive links. The data sample is therefore more likely to contain an under-representation than an overrepresentation of women that died after giving birth.

Next, we manually searched for all women in the online search engine for civil registry records WieWasWie. This search allowed the inclusion of other links that were missed in the linkage to the birth registered due to spelling variations. Moreover, WieWasWie includes death certificates which allowed the linkage of mothers' deaths not only to live births but also to stillbirths. In this period, stillbirths were not recorded in the birth certificates in the Netherlands, but they were registered in death certificates. We could therefore also link women who died after stillbirths. Ideally, we would also have information on which women died after a miscarriage where the fetus was not old enough to be registered as a stillbirth. Unfortunately, that information is not available. However, including the registered stillbirths already improves our dataset compared to using only maternal mortality linked to live births. In total, 236 deceased women have been identified as to have died up to one year after giving birth (see Table 1).

Table 1 *Number and share of number of deceased women linked to births*

Period	**Total female deaths in selected period**	**Linked mothers N (%)**
1870–1879	620	134 (*21,6*)
1900–1909	642	*102* (*15,9*)

The causes of death of the women aged 16–50 were classified into three categories. Category one includes all causes of death that belong to the O category of the ICD10h, which is the designated category for all pregnancy- and childbirth-related mortality (Reid et al., 2024a, 2024b) and which includes by default terminology referring to pregnancy or childbirth (words used such as puerperal, gravidity, pregnancy, partum). There were 18 cases where the woman had been assigned a cause of death clearly related to childbirth that had not been linked to a birth or stillbirth. In some instances the cause of death itself specified that the death was a result of a miscarriage or abortion. Of the other women, it is not clear whether they suffered a miscarriage or died after having given birth. Yet this means we should keep in mind that the women we could link to a birth or stillbirth may still be an under-representation of the actual numbers.

The second category comprises the common complications of pregnancy or giving birth, such as peritonitis, endometritis, phlebitis, or thrombosis (Shorter, 1982). These are the causes of death that could contain hidden maternal death. The final category contains all other deaths which could not be connected directly to pregnancy. Tuberculosis, heart disease and nephritis all belong to this category. This last category is most likely to include associated maternal mortality, if there was any. Five women that were linked to a birth or stillbirth had no specification of the cause of death (i.e. either a blank or stated to be unknown). We included these women in category three as well. There were no causes of death referring to accidents, which should be excluded from maternal deaths in case of using the WHO definition of deaths occurring within 42 days after birth.

3 MATERNAL MORTALITY IN MAASTRICHT

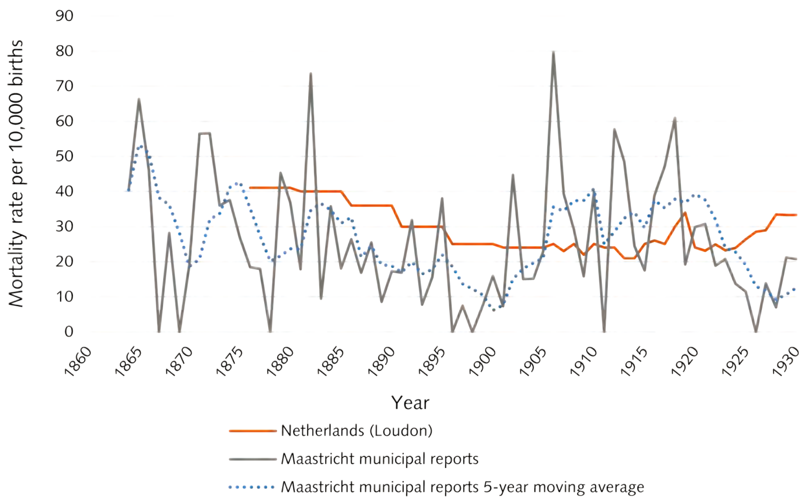
3.1 TOTAL MATERNAL MORTALITY RATES

Compared to the rates of the Netherlands, maternal mortality in Maastricht was fairly low in the final decades of the 19th century (see Figure 1). Although a decline in maternal mortality could be expected in the later 19th century as a result of antisepsis and improved birth care, such a trend is difficult to identify for Maastricht. There is a slight decline in official maternal mortality after the mid-1860s, but this is followed by an increase in the early 20th century, which even exceeds the national average. The early-20th century increase could be a statistical artefact, resulting from improved diagnostic practices. In 1913, a school for midwives was founded in Heerlen, near Maastricht, which could have improved the registration of maternal deaths. However, the increase in maternal mortality already appears from 1905 onwards. The effects of the midwifery school should be expected to show only a couple of years after 1913, when midwives had completed their training and started practicing in for example Maastricht. The foundation of the school itself may also indicate another transformation; perhaps maternal deaths had gained more attention over the years, resulting both in the establishment of the school and increased registration.

Next, the maternal mortality rates of Maastricht are compared to the other estimations based on the individual-level data. Figure 2 shows the annual maternal mortality rates per 10,000 births (including stillbirths) based on four different calculations and sources. It compares the maternal mortality rate in (1) the municipal reports, (2) the causes of death of all women that were clearly related to childbirth from the MDDD (category 1), (3) the number of linked deaths occurring within 42 days after birth (WHO definition) regardless of the cause of death, and (4) the number of linked deaths that occurred within a year after birth regardless of the cause of death.[2]

When comparing the official maternal mortality statistics to the other rates, it is clear that the statistics in the municipal reports likely under-reported maternal mortality. However, the differences between the municipal reports and the MDDD-causes-of-death are limited. In some years, the rates are similar, and in other the maternal mortality rate calculated from the individual-level causes of death is even lower. This would mean that some causes that we would not identify as direct maternal mortality, were actually viewed as such by contemporary physicians and/or clerks.

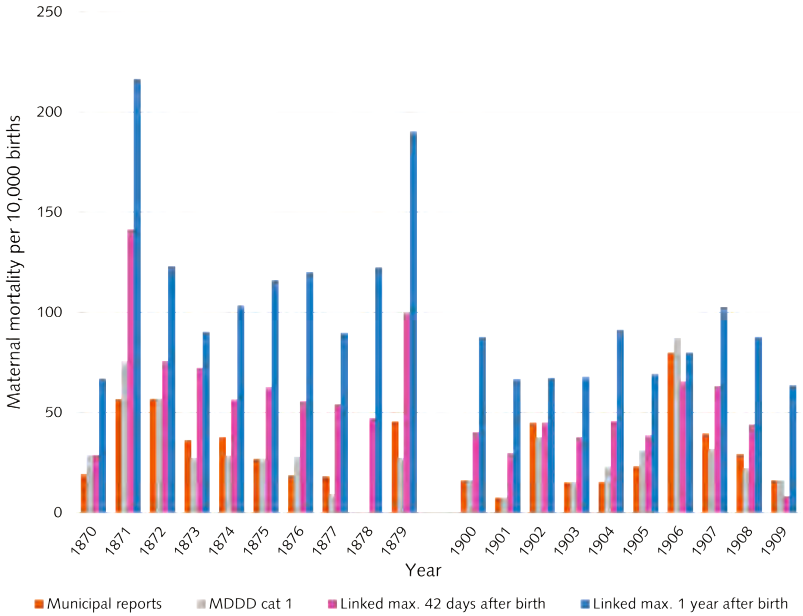
Figure 1 *Maternal mortality rate per 10,000 births, the Netherlands and Maastricht, 1864–1930*



Source: Loudon, 1992, p.561–563; Verslag van de toestand der gemeente Maastricht over het jaar 1864–1930

2 See Appendix Table A2 for the annual number of deaths and death rates per estimation method.

Figure 2 *Maternal mortality per 10,000 births, Maastricht 1870–1879 and 1900–1909*



Sources: Verslag van de toestand der gemeente Maastricht over het jaar 1870–1879 and 1900–1909; Maastricht Death and Disease Database; Maastricht birth registers years 1869–1879 and 1899–1909.

The differences between the official statistics and deaths based on the time after giving birth are more striking. In 1870–1879, the maternal mortality rate based on the 42-day period after birth exceeds the rates of both the municipal reports and the individual-level causes of death. This suggests the presence of associated maternal mortality: women who died from other causes that might have become fatal due to a recent pregnancy or childbirth. In 1906, this rate is lower than the one based on the individual-level causes of death. In fact, the deaths of six women in this year have been attributed to a cause of death related to pregnancy or childbirth in the MDDD, while they were not linked to a birth. This could concern women who died following a miscarriage or a failed pregnancy, when the fetus had not been old enough to be registered as a stillbirth. However, since 1906 stands out in the missing links, it may be the case that the registration of 1906 was not complete. In the 1900s, the number of deaths within the 42-day mark still exceeded those officially related to childbirth. Apparently, diagnostic practices still did not include all maternal mortality, even when these deaths occurred relatively soon after giving birth.

Maternal mortality rates were the highest in the final category containing the linked deaths up to one year after birth, indicating potential higher associated maternal mortality rates in a longer birth-death interval. Maternal mortality was highest in the first researched period. Since the mortality decline for adult women was in progress at that moment in time (Murkens, 2023), it is likely that some of these deaths were not related to maternity. However, it should still be considered that a combination of circumstances, including pregnancy and childbirth, contributed to the eventual death of these women. After all, several scholars have argued that multiple births took a toll on women's bodies. The resulting maternal depletion constituted an important factor in tuberculosis mortality among women (Janssens & van Dongen, 2017; Manfredini et al., 2020).

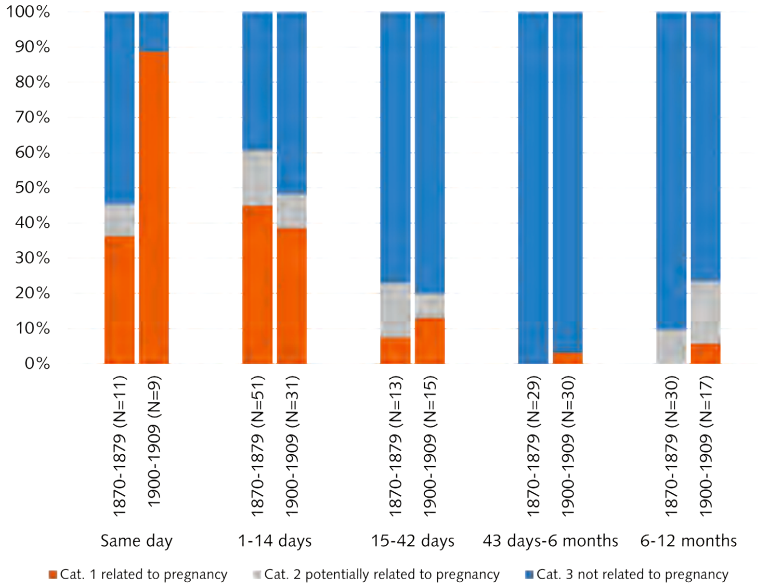
3.2 HIDING MATERNAL MORTALITY

Higher mortality rates among women linked to a birth or stillbirth suggest the presence of hidden maternal mortality in the Maastricht registration. To shed more light on the registration practices, the share of each of the causal categories is shown across different interval periods between the birth of a child and death of the mother.[3] Figure 3 demonstrates the deaths occurring on the same day, within 14 days (i.e. the period in which puerperal fever generally develops), between fifteen and 42 days (WHO definition), and the remaining period up to one year.

Figure 3 reveals that the vast majority of deaths characterized as being related to childbirth (category 1) occurred within the 14-days interval in both periods. After the first 14 days the chances of a death being registered as a maternal death had declined substantially. In general, limited differences are observed in the relative registration of maternal deaths between the two time periods; only the recognition of maternal deaths occurring on the same day as the birth had increased by the early 20th century. However, the total number of women dying on that same day is exceptionally small. We should therefore refrain from drawing conclusions based on the changes in registration over the two periods that we observe for the first day.

The share of deaths potentially related to pregnancy (category 2), such as peritonitis or fever, is low, as the small discrepancy between the maternal mortality rates of the MDDD and the linked mothers in Figure 2 already suggested. This indicates that the share of hidden maternal mortality in Maastricht was probably low. There are, however, many causes of death that could have been associated with pregnancy or childbirth, even for deaths occurring within the first 14 days after birth. This suggests that the factor of associated deaths may have been more prevalent than hidden deaths. However, a death occurring on the same day as the birth can hardly be recognized as an associated death. Perhaps attending physicians tried to hide the death not by writing down the non-maternal equivalent of the cause of death, as Loudon explained, i.e. peritonitis instead of puerperal peritonitis or fever instead of puerperal fever. Instead, they recorded something entirely different, unrelated to childbirth.

Figure 3 *Share of deaths per category, by time interval and period, Maastricht 1870–1879 and 1900–1909*



Sources: Verslag van de toestand der gemeente Maastricht over het jaar 1870–1879 and 1900–1909; Maastricht Death and Disease Database; Maastricht birth registers years 1869–1879 and 1899–1909.

3 We checked whether these patterns differed by age of the women dying, but no clear differences were found.

A closer inspection of the registration of specific causes of death shows that there was some hidden maternal mortality among 'usual suspects' such as peritonitis. However, the term 'fever' without further specification did not occur. In itself, fever was not a widely used term either, as only two women out of all the women dying in these two research periods (n = 1,262) died of fever, albeit not as a consequence of childbirth. Peritonitis, on the other hand, was used in total in nine instances. It was used five times for a death occurring within the first 42 days, but the birth had occurred longer ago in the other four cases. Whether the latter actually concerned maternal mortality is therefore difficult to establish. It seems possible that something else had happened to these women to cause peritonitis. Another 'usual suspect', septicaemia, was only used twice, for deaths occurring before the 42-day mark. In sum, although the 'non-maternal equivalent' causes reveal there may have been some hidden maternal mortality, this does not appear to have been a common practice.

Causes of death that were not directly associated with childbirth were more prevalent. The three most common causes of death across the entire period for the whole 365 day interval were tuberculosis of the lung (n = 49), heart disease (different specific terminology used, total n = 17) and pneumonia (n = 16). Janssens and van Dongen (2017) already suggested that tuberculosis in women could have been related to maternal depletion due to many pregnancies, yet this relation might have been even stronger. Not only were these causes of death registered after the 42-day mark, which would make sense as they might be considered to be associated deaths, but they were also used before the 42-day mark. Tuberculosis of the lung even occurred twice as a cause of death in a woman dying on the same day as giving birth, and six more times before the 14-day mark. The same applies to heart disease and pneumonia; both were used for deaths occurring within 14 days and even on the same day.

It is striking how common these causes of death were, even though the total number of deaths was low (only 236 in total). Tuberculosis of the lung was the second most frequently used cause of death in the deaths occurring between day 1 and 14 after birth, coming second to puerperal fever. Heart disease came fourth, after eclampsia. A considerable amount of mothers were not registered as having died of causes related to childbirth, even though some of them had died only a few days after giving birth. If women had suffered from tuberculosis, a heart disease, or pneumonia, it is likely that pregnancy and delivery took a substantial toll on women's health, and therefore contributed to their death.

4 CONCLUSION

Maternal mortality in the Netherlands was among the lowest across Western Europe. Were the circumstances indeed so much better in the Netherlands as a result of the high standard midwifery care? Or was low maternal mortality a consequence of considerable under-registration? Although it is beyond the scope of this paper to compare potential under-registration of maternal mortality in the Netherlands to other Western countries, the highly detailed dataset created here, allows for a close scrutinization of maternal mortality in the Dutch city of Maastricht. Based on the existing literature, under-registration of maternal mortality was expected for two reasons. First, Loudon (1992) stated certain incentives existed for medical practitioners in the late 19th century to cover up maternal deaths, resulting in hidden maternal mortality. Second, an exploration of total female adult mortality in Maastricht ran into some odd deaths, that were registered as non-pregnancy related, even though the death had occurred shortly after giving birth (Murkens, 2023). In this paper, we aimed to shed light on the extent of under-registration of maternal mortality and asked whether this could be related to more deliberate practices or resulted from associated deaths not being considered as maternal mortality. Additionally, we asked whether registration practices may have improved over time, making hidden maternal mortality less common.

The registration of maternal mortality by contemporary officials and medical practitioners was most likely flawed. Municipal reports account for considerably lower maternal mortality than the maternal mortality rates we calculated based on individual-level causes of death and the number of mothers dying within 42 or 365 days after giving birth. The differences could be very substantial. One year after birth maternal mortality rates were four times higher than what the official statistics reported. Similarly, rates estimated for the 42-day mark were at times double the size of what was stated in the official statistics. The deaths of mothers were not only registered as unrelated to childbirth after the 42-day mark, but also before. In total, 50% of deaths occurring within 42 days of giving birth were supposedly induced by causes unrelated to childbirth. Only 11.5% of the deaths could be attributed to pregnancy while leaving out the childbirth qualifier, based on the individual cause-of-death information. As Loudon (1992) suggested, this concerned causes that medical practitioners could most easily, and with the fewest consequences, use to obscure maternal mortality, if they felt the need to do so. However, these causes of death, such as peritonitis, fever or phlebitis, do not seem to have been the most prevailing causes to cover up a maternal death. Instead, more general causes of death were used for that purpose, such as tuberculosis of the lung, heart disease and pneumonia.

This means that the estimations of maternal mortality were most likely distorted because associated maternal mortality was under-registered in Maastricht. Other categories of death are therefore likely to include some maternal mortality as well. Although the share of total maternal mortality, defined as every woman dying within a year after giving birth, was not extremely large in total female adult mortality (19%), historical demographers should be aware that when studying for example female tuberculosis mortality, this could include some associated maternal deaths as well. Moreover, the diagnostic practices did not seem to have improved much by the early 20th century. Studying mortality after the turn of the century does by no means exempt one from the issues of under-registration.

Although we revealed considerable under-registration, this study does suffer from small numbers. We should therefore not only interpret some of our results with caution, it also remains to be seen whether the results are representative for the Netherlands as a whole. How these results relate to registration practices in other countries requires further scrutiny. Moreover, this study does not examine the motives of medical practitioners to register maternal deaths differently. Potential ways of doing so are to analyse whether maternal deaths occurred in hospitals, or if they were the results of home births, attended by midwives. Which profession needed to protect their professional reputation the most? A turning point analysis may provide additional clues as to whether professionalisation among medical doctors or midwives could have played a role in improving registration. Whatever the reasons may have been, it is clear that women in Maastricht suffered considerably more from maternal mortality than has up until now been assumed.

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APPENDIX

Table A1 *Categorization of maternal mortality in the Maastricht municipal reports*

Year	Category	Disease
1864–1870	Peritonitis puerperalis	-
1871–1877	Puerperal disease (*Ziekte van het kraambed*)	Metrorrhagia, Dystocia, Abortion, Sudden death
		Eclampsia, Mania puerperalis
		Febris puerperalis
1878–1886	Puerperal disease *(Ziekte van het kraambed)*	Febris puerperalis
1887–1900	Diseases of the urinary and genital organs	Puerperal diseases
1901–1930	Puerperal fever	-
	Other diseases of pregnancy and labour	

Source: Verslag van de toestand der gemeente Maastricht over het jaar 1864–1930.

Table A2 *Number of deaths and mortality rate per 10,000 births per estimation method, Maastricht 1870–1879 and 1900–1909*

Year	Municipal reports		MDDD cat. 1		Linked max 42 days		Linked max 1 year	
	N	Rate	N	Rate	N	Rate	N	Rate
1870	2	19,1	3	28,6	3	28,6	7	66,8
1871	6	56,4	8	75,3	15	141,1	23	216,4
1872	6	56,7	6	56,7	8	75,5	13	122,8
1873	4	36,0	3	27,0	8	72,1	10	90,1
1874	4	37,5	3	28,1	6	56,3	11	103,2
1875	3	26,7	3	26,7	7	62,4	13	115,9
1876	2	18,5	3	27,7	6	55,4	13	119,9
1877	2	17,9	1	9,0	6	53,8	10	89,7
1878	0	0,0	0	0,0	5	47,0	13	122,2
1879	5	45,2	3	27,1	11	99,5	21	190,0
1900	2	15,9	2	15,9	5	39,8	11	87,6
1901	1	7,4	1	7,4	4	29,5	9	66,5
1902	6	44,7	5	37,3	6	44,7	8	59,7
1903	2	15,0	2	15,0	5	37,5	9	67,6
1904	2	15,2	3	22,8	6	45,5	12	91,0
1905	3	23,1	4	30,7	5	38,4	9	69,2
1906	11	79,8	12	87,0	9	65,3	11	79,8
1907	5	39,4	4	31,5	8	63,0	13	102,4
1908	4	29,2	3	21,9	6	43,8	12	87,7
1909	2	15,9	2	15,9	1	7,9	8	63,6

Sources: Verslag van de toestand der gemeente Maastricht over het jaar 1870–1879 and 1900–1909; Maastricht Death and Disease Database; Maastricht birth registers years 1869–1879 and 1899–1909.

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Advancing Precision in Childhood Causes of Death

Wording and Source Discrepancies in Palma (Spain), 1836–1930

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ABSTRACT

Assessing the precision of causes of death is essential for gaining a clearer understanding of past disease incidence and its evolution. This study introduces a novel lexicographical approach to examining childhood mortality in the port city of Palma between 1836 and 1930, drawing on three sources that recorded individual causes of death: burial registers, parish books, and the civil register. In this sense, we estimate the number of words used in diagnoses to trace how efforts toward greater precision and standardization evolved over time. These are reflected in the increasing use of diagnostic qualifiers and the near disappearance of lengthy, undetermined diagnostic descriptions — particularly in cases of congenital diseases, which are a significant group within ICD10h related to infant mortality. To further explore the meaning of diagnoses, we use medical and general dictionaries, focusing on the labels teething, fever, and diarrhoea to better understand diagnostic discrepancies between burial and parish records. These discrepancies appear to stem largely from the higher incidence of death certificates without a stated cause in parish books — likely due to the requirement in burial registers to include the name of the certifying physician. In the case of teething, we observe a notable association with digestive system diseases, as well as with fever itself. Finally, it is worth noting that diarrhoea came to be understood more as a symptom than as an independent disease as a result of new ways of conceptualising disease that developed during the 19th century.

Keywords: Infant mortality, Child mortality, Causes of death, Diagnostic precision, Lexicographical analysis

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1 INTRODUCTION

The epidemiological profiles of past societies in the 19th and 20th centuries can be studied by collecting and analysing individual causes of death recorded in parish and civil registers. This process requires a significant investment of time and resources to build representative samples, yet it yields rich details that allow for an in-depth evaluation of health changes over time. This richness, however, contrasts with the highly aggregated epidemiological data published in state statistical bulletins, which usually use the International Classification of Diseases (ICD) to present the data (Moriyama et al., 2011). While the ICD has been periodically updated, these revisions hinder long-term comparisons — an essential aspect of understanding mortality decline within the framework of the first demographic transition. In this regard, the ICD10h coding scheme, developed by the SHiP network team led by Professor Janssens[1] provides an ideal tool for comparing mortality trends across both time and space. It enables the processing of large numbers of historical disease descriptions from various European linguistic regions, while ensuring compatibility with modern disease patterns as it is built upon the contemporary ICD10 system. Another key feature of the scheme is its focus on coding words rather than diseases, minimizing interpretative bias in historical records (Janssens, 2021; Janssens & Devos, 2022).

The reconstruction of epidemiological patterns from individual-level cause-of-death data relies on the accuracy of recorded causes, the dissemination and social adaptation of medical-scientific knowledge, and the influence of disease classifications and nomenclatures (Anderton & Leonard, 2004; Bernabeu Mestre, 1992). Assessing the precision of these records is essential for gaining a clearer picture of past disease incidence and change, ultimately advancing in the understanding of current epidemiological patterns (Mackenbach, 2021; Reid et al., 2015). However, research on the reliability of cause-of-death records requires a lexicographical approach to understand their coetaneous meaning (Barona, 1993). In this regard, few studies on epidemiological patterns in infant mortality have partially drawn on medical dictionaries (*e.g.*, Hiltunen Maltesdotter & Edvinsson, 2025; Janssens & Devos, 2022; Raftakis, 2021).

In this context, the paper aims to analyse how causes of death were diagnosed, focusing on their wording and variations in registration practices. It offers a novel perspective on the evolution of medical knowledge and the interpretation of disease over time by drawing on both medical and general dictionaries, alongside a wide range of sources that report causes of death simultaneously — an approach not previously explored for this region and rarely applied to other geographical contexts. This approach is theoretically situated within the study of the determinants of the demographic and epidemiological transitions, as well as the shifting patterns of disease from past to present (Mackenbach, 2021; van de Walle, 1986). The study is grounded in a case study of infant and child mortality in the port city of Palma (Spain) between 1836 and 1930 — a crucial period in the demographic transition, as these age groups were directly responsible for the significant gains in life expectancy. Furthermore, this process coincided with a shift in the way childhood was perceived by both society and medicine (Bernabeu-Mestre et al., 2007).

2 THE CITY OF PALMA AS AN ECONOMIC AND SANITARY HUB

Port cities constitute essential objects of epidemiological research, as they historically functioned as primary nodes for the introduction and dissemination of diseases, as the SHiP network has prominently highlighted[2], while simultaneously serving as hubs of economic dynamism. In the case of Palma, its commercial relevance was particularly pronounced, facilitating both mainland and international trade. This economic activity was underpinned by commercial agriculture and industrial production — principally textiles and footwear — operating within a putting-out system, small workshops and factories attached to mercantile capital (Escartín, 2001; Manera, 2001). From a demographic perspective, Palma had over 40,000 inhabitants in 1842, making it the tenth most populous provincial capital in Spain out of the 50 existing at the time. By 1900, its population had grown to over 60,000,

1 May this article serve as our tribute to Professor Janssens for her outstanding contributions to historical demography, particularly in her works on the study of health history. Her dedication and scholarship have profoundly shaped the field, leaving a lasting legacy that will continue to inspire researchers for years to come. We extend our deepest gratitude and admiration.

2 For more information on the SHiP network, visit https://www.ru.nl/en/departments/radboud-institute-for-culture-and-history/ship.

placing it twelfth nationally — a position it retained in 1930 with more than 88,000 residents. Despite this marked demographic growth, Palma exhibited the highest life expectancy in Spain, reaching 42.5 years in 1900 and rising to 53.2 years by 1930, a trend largely attributable to persistently low infant and child mortality rates (Pujadas-Mora, 2009, 2024). These figures make Palma a valuable case study and a natural laboratory for understanding the determinants of the demographic and epidemiological transitions, allowing us to observe the distinctive factors that set the region apart and positioned it as a pioneer in these processes.

In terms of healthcare provision, the Balearic Islands — of which Palma is the capital — had a higher density of qualified physicians than the national average: slightly more than one doctor per 12 km², compared to one per 16 km² in the rest of Spain (Gallego, 2009). Despite this, the province had lacked a university since 1829, and higher education would not be re-established on the islands until 1978. As a result, aspiring doctors were required to pursue their training on the mainland, typically at leading institutions such as the University of Barcelona, known for its longstanding reputation, expertise, and international standing in medical education (Canaleta, 2013).

In contrast, the number of medical associations in the Balearic Islands was remarkably high compared to other Spanish regions. These associations, largely concentrated in Palma, played a key role in the dissemination of medical knowledge, serving as a substitute intellectual infrastructure in a province that lacked a university, as referred to before (Granjel, 2006). Throughout the period under study, different medical institutions were active in Palma — though not always simultaneously — including the Mallorcan Phrenological Society, the Medical-Military Academy of Mallorca, and the Mallorcan Surgical Academy. From the second half of the 19th century onward, two major institutions emerged as the primary authorities setting medical and professional standards well into the 20th century: the Royal Academy of Medicine and Surgery of Palma and the Medical-Pharmaceutical College (the precursor to the current Medical College). Both institutions were grounded in the exchange and dissemination of medical knowledge, whether through regular meetings for the discussion of clinical cases or through publications such as the *Revista Balear de Ciencias Médicas* (Balearic Journal of Medical Sciences), the official journal of the Medical-Pharmaceutical College. This journal not only published the scientific vanguard of the region but also served as a conduit for introducing medical innovations from abroad. It was widely exchanged with other medical journals across Spain, as well as with counterparts in Europe and the Americas (Rodríguez Tejerina, 1981). Its pages also reported on medical papers presented at national and international congresses (Prats & Pujadas-Mora, 2008). All of this suggests that the medical professionals of Palma were well attuned to contemporary scientific developments, thanks to the dynamic circulation of knowledge fostered by the city's professional associations, as outlined above. Moreover, the participation of Mallorcan physicians in international circuits of medical knowledge is also evidenced by documented commissions to study emerging therapies firsthand — such as the case of doctors sent to the Pasteur Institute in Paris to learn about the newly developed antidiphtheric serum (Pujadas-Mora, 2009).

In 19th-century Spain, within the framework of emerging liberal states, the provision of medical services was predominantly considered an individual responsibility. In contrast, public health — an area increasingly regulated and promoted by the state — sought to prevent threats to collective well-being, particularly epidemics. It also encompassed charitable health care aimed at those unable to afford private medical attention. In Palma, such care was provided by the Hospital Provincial (Muñoz Machado, 1975, pp. 32–33). Moreover, institutions delegated by the state — namely, municipal and provincial councils — were also only responsible for enforcing sanitary measures in times of epidemic risk, but also for providing ongoing health-related and charitable services to their populations. In the case of Mallorca, this translated into the widespread presence of municipal medical doctors across the island from the second half of the 19th century, including in the capital, Palma. This early and comprehensive implementation placed Mallorca ahead of many other Spanish regions in the development of local medical services (Pujadas-Mora & Salas Vives, 2014). These public services were further complemented by the charitable care offered in religious convents run by religious orders. From the early decades of the 19th century, these institutions demonstrated a pronounced commitment to community health (Moll, 2005). From the 20th century onwards, there was also a notable proliferation of private clinics closely associated with certain medical dynasties (Canaleta, 2013).

3 SOURCES AND METHODS

To address our research objectives, we will use two individual-level death record sources — parish and burial registers — for the period 1836 to 1881, during which they were compiled concurrently, along with a third source, the civil register, for the years 1881 to 1930. Burial registers began in 1821 with the opening of a new cemetery outside the town centre, following the hygienic/public health principles established in Spain since the late 18th century. From that point onward, burials were no longer permitted within the city, let alone inside churches. These records were intended to include the deceased's name, age, parents, parish of residence, time and cause of death certified by a medical doctor, and, additionally, whether a will was made, along with its date and the executor's name[3]. Notably, in the case of parish registers — which date back to the *Rituale Romanum* (1614) or even before — the inclusion of the cause of death was not mandatory until the Royal Order of December 1, 1837 (Bernabeu Mestre, 1992).

While earlier parish records occasionally noted causes of death, it was the Royal Order that established their mandatory inclusion in death certificates "The illness that caused the death, according to the doctor's certification, without which the corpse cannot be buried". It was one of the state administration's attempts to rationalize the parish register of vital events, following the repeated failures to secularize it (Muro et al., 1996). However, unlike burial registers, it remains unclear whether the medical certification requirement was consistently enforced, as parish death certificates did not include the certifying doctor's name, unlike burial records (Pujadas-Mora, 2009).

The comparison of both sources reveals that burial registers contain more death entries than religious registers for the period 1836–1881, with an average difference of 5%. For individuals who died at ages three or four, however, the discrepancy can reach up to 10% at certain points (Pujadas-Mora, 2009). This can be attributed to the fact that, during epidemics, cemetery records were maintained with greater diligence due to the strict control over burials as part of epidemic containment efforts. Furthermore, the centralized nature of burial registers — where different books were overseen by a single individual — contrasted with the decentralized parish system, in which each parish maintained its own register and had its own registrar. Likewise, we begin using data from the civil register in 1882, as the earliest years show limited reliability — a pattern also observed in other Spanish regions (Brel Chacón, 1998). From that point onward, the civil register became the most comprehensive source, leading most studies on epidemiological patterns to prioritize its use (Ramiro Fariñas, 1998; Robles González, 2002; Sanz Gimeno, 1997).

In this sense, our analysed data contains 14,917 deaths between birth and age one and 23,354 deaths between ages one and four. Each cause of death was coded using the ICD10h coding system, resulting in 358 codes for infant deaths and 450 codes for child deaths. These codes were then grouped into causal categories, according to the system referenced (Janssens, 2021). For infant mortality, these groups are as follows: air-borne diseases (5,306 deaths), congenital and birth disorders (1,866), convulsions (32), external causes (30), ill-defined and unknown (1,004), other infectious (307), other non-infectious (2,839), stated to be 'unknown' (517), teething (2,221), and water-food borne diseases and weakness (339). For child mortality (ages 1–4), the classification is: air-borne diseases (5,642 deaths), convulsions (307), external causes (109), ill-defined and unknown (1,299), other (4,586), other air-borne diseases (935), other infectious (510), respiratory (3,606), stated to be 'unknown' (593), tuberculosis (547), water-food borne diseases (5,095), and weakness (125).

To clarify the contemporary meanings of expressions such as teething, fever, and diarrhoea — commonly found as causes of death in burial books and parish registers — we consulted the principal medical dictionaries published in Spain from the early 19th century to the 1930s as a direct approach to understanding the circulation of medical knowledge at the time. Although it is unclear whether certifying physicians directly used medical dictionaries, they nonetheless provide an authoritative and contemporaneous account of how diseases were understood and described at the time. Therefore, they remain valid and valuable references for interpreting the causes of death recorded in historical documents, as they reflect the medical framework within which such terms were likely conceptualized. The dictionaries used in this study are compiled in the resource *Tesoro Lexicográfico Médico* (TELEME, Medical Lexicographical Treasury), which brings together eight dictionaries of medical terminology — one from the 18th century and seven from the 19th century (Gutiérrez Rodilla & Pascual, 2022).

3 Article 10º of the proclamation of the constitutional town council of Palma, dated 29th of March, 1821; AMP, FP851: *sobre la habilitación de Cementerio Rural*.

Additionally, to capture a broader, less specialized interpretation of disease-related terms, we have used the dictionaries of the *Real Academia Española* (Royal Spanish Academy) gathered in the *Nuevo Tesoro Lexicográfico de la Lengua Española* (New Lexicographical Treasure of the Spanish Language).

4 EVALUATING THE PRECISION OF CAUSES OF DEATH IN CHILDHOOD

To assess the precision of recorded causes on children's death certificates, as noted above, we propose two analytical strategies: examining the wording and identifying discrepancies in the diagnoses of causes of death. The wording of diseases is a relevant factor in the development of modern medical language as a result of the evolution of medical knowledge and practice (WHO, 2022). Thus, we take as a reference the number of words used in diagnoses to trace how the quest for greater precision and standardisation has evolved along with the historical shift from symptom-based causes to signs of injury, dysfunction or aetiology, especially of an infectious nature (Beemer, 2009). This approach focuses on the linguistic formulation of diagnoses, rather than their contemporary meaning. To explore the meaning of diagnoses, we employ a lexicographic analysis of three causes of death, two of which — teething and fever — are classified by the ICD10h as ill-defined diseases. Between 1836 and 1930, teething accounted for approximately 800 deaths, while fever was recorded as the cause of death in around 1,500 cases. Additionally, we examine diarrhoea, one of the most significant causes of mortality among water-borne diseases, given its importance within the Mediterranean epidemiological pattern, which accounted for more than 600 deaths during the same period.

4.1 WORDING OF CAUSES OF DEATH BY COUNTING WORDS

In Palma, between 1836 and 1930, an average of 60% of recorded causes of death in infant mortality were described with a single word, compared to nearly 70% in child mortality (see Figure 1a and 1b). However, the proportion of causes of death recorded with two words increased over time. In the case of infant mortality, this trend shows a continuous rise throughout the study period, becoming particularly evident after 1900, when it grew from 20% to 40% of all recorded causes. For child mortality, the share of two-word diagnoses reached 30% by the end of the series. A significant factor in this shift was the increasing use of qualifiers, which provided additional details about the progression of diseases, their anatomical location, or descriptions of pathological processes, etc. This was especially notable in the registration of airborne and non-infectious diseases, as we will discuss later.

In the case of infant mortality, three-word causes of death held little significance, primarily concentrated in the early years of the study. This is largely due to their frequent association with congenital and birth-related disorders, often encapsulated in the recurring diagnosis of *no ser viable* (not being viable) (see Figure 1a). However, within this same category, four-word causes had greater prominence, driven by the recurrent use of expressions such as *no ser del tiempo* (not being of time) and the persistence of ambiguous descriptions like 'irritation/inflammation in the womb', which would lose significance after 1870. For child mortality, also from the 1870s onward, there was a significant rise in three-word causes of death, peaking around 1910. This trend may, in part, be attributed to the increasing use of qualifiers (e.g., chronic pulmonary tuberculosis, acute capillary bronchitis). However, the continued presence of vague terms — such as stomach disease, heart disease, chest complaint, gastric intestinal irritation, or serous stroke — also contributed to this pattern, particularly until the late 19th century (see Figure 1b). In the case of child mortality, multiple causes of death were slightly more frequent than in infant mortality, though their overall proportion remained relatively low. Many of these cases fell into the category of three-word causes, as they often involved two causes of death linked by a preposition (e.g., *diarrea por enteritis* (diarrhoea due to enteritis)) or a conjunction (e.g., measles and diphtheria).

Diagnoses related to water-food borne or airborne causes tend to be the most concise, with over 60% of infant mortality cases and just over 50% of child mortality cases described in a single word. Additionally, 40% and 35% of these cases, respectively, are summarized in just two words (see Figure 2a and 2b). Among these two-word diagnoses, qualifiers such as acute/chronic, intestinal/gastric, cerebral, serous, or capillary are common. In contrast, congenital and birth disorders in infant mortality typically require three to eight words, as previously noted. It is also noteworthy that under the 'other non-infectious' group of causes of death we will find, especially during the 19th century, expressions such as: disease/irritation/inflammation in a particular region or organ. Specific examples would be 'disease of the head' (*enfermedad de la cabeza*), 'disease of the lung' (*enfermedad en el pulmón*), 'inflammation of the left arm' (*inflamación en el brazo izquierdo*), 'inflammation of the right thigh' (*inflamación del muslo derecho*), etc. These formulations are vague as they only present the symptomatology, which would also indicate a lack of precision in diagnosing the disease that led to death. For infant mortality, these long diagnostics account for 59% of four-word diagnoses, 26% of five-word diagnoses, and 70% of six-word diagnoses (see Figure 2a). In child mortality, they represent 47% of three-word diagnoses, 81% of four-word diagnoses, and 62% of five-word diagnoses (see Figure 2b). In the same vein, it is worth highlighting ill-defined causes in five-word diagnoses, such as the phrase 'common class disease' — a vague and non-specific diagnosis found in infant mortality cases.

Figure 1a *Number of words by diagnostic label in Palma's infant deaths, 1836–1930*

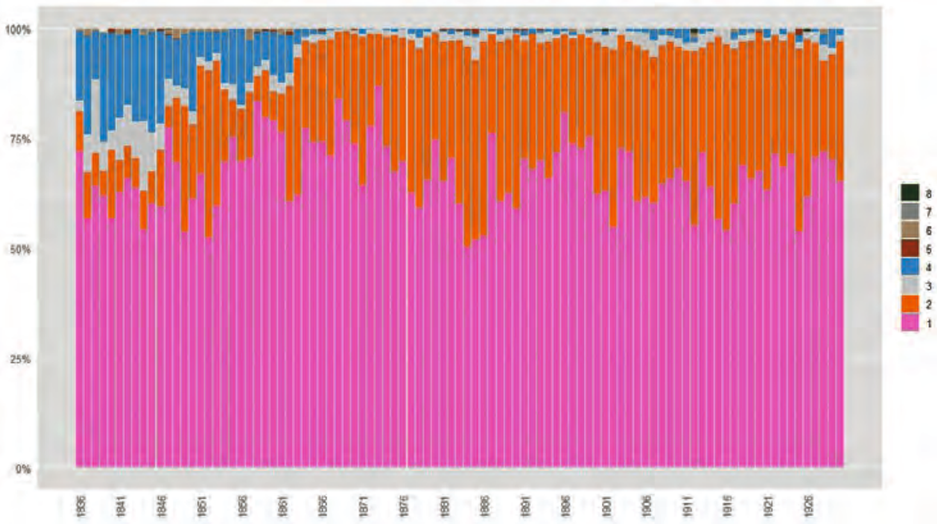


Figure 1b *Number of words by diagnostic label in Palma's child deaths, 1836–1930*

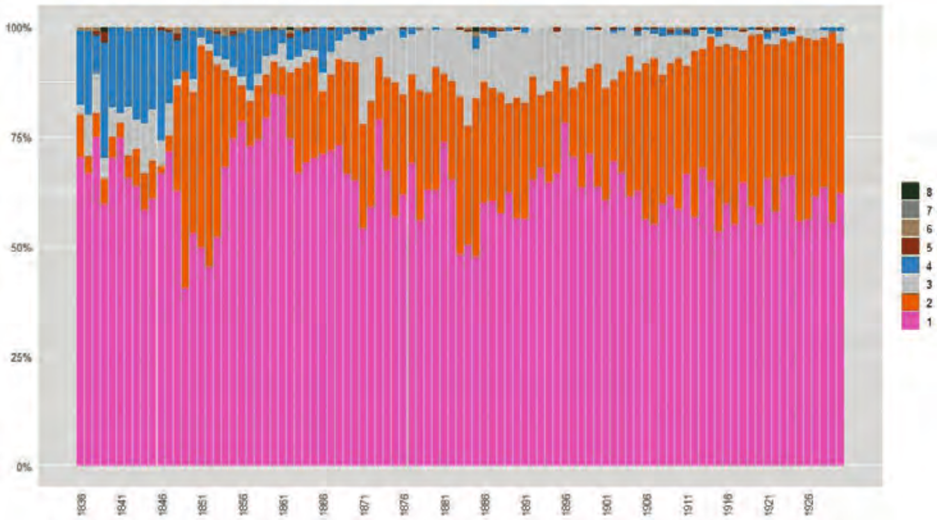


Figure 2a *Number of words for causal group in Palma's infant deaths, 1836–1930*

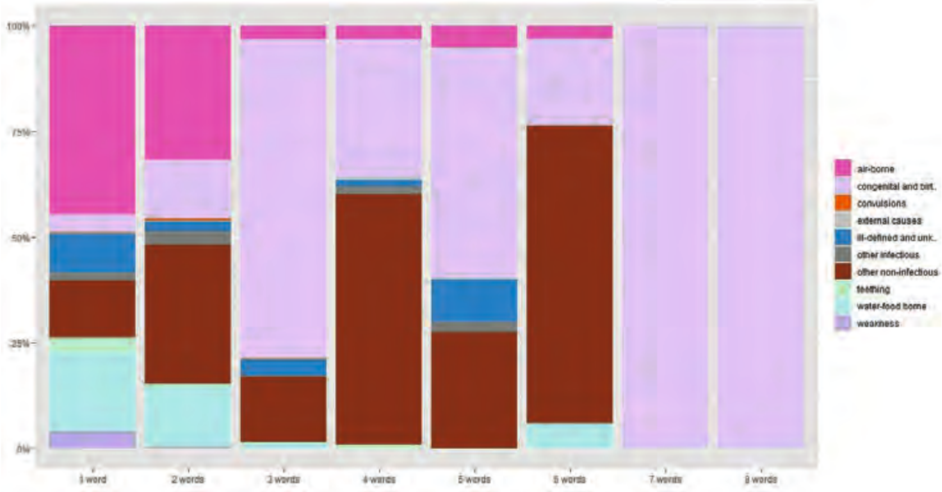
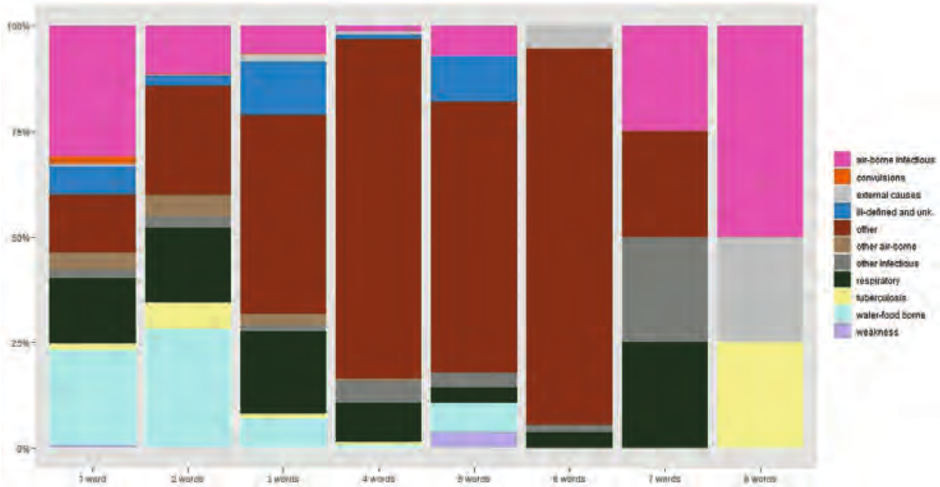


Figure 2b *Number of words for causal group in Palma's child deaths, 1836–1930*



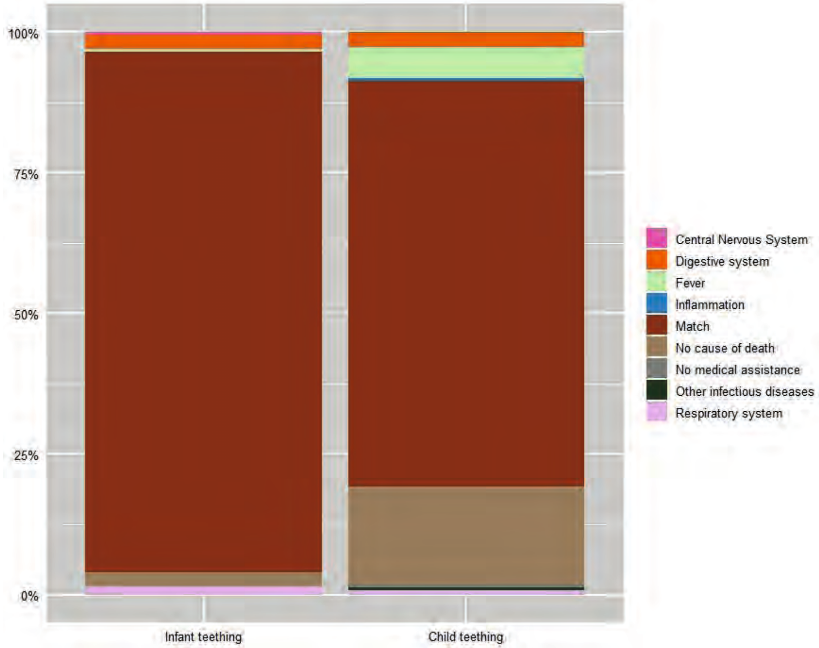
4.2 DISCREPANCIES IN RECORDING PRACTICES

The study of discrepancies in causes of death from different sources has a long-standing tradition in medicine, aimed at improving treatments and ensuring the quality of mortality statistics (Smith Sehdev & Hutchins, 2001). In this regard, we propose a historical perspective on this methodology by comparing burial registers and parish registers — two sources that record individual deaths but differ in nature and purpose — allowing us to examine the evolution of cause-of-death descriptions over time, specifically from 1836 to 1881, using three specific diseases: two ill-defined (teething and fever) and one infectious of gastric nature (diarrhoea), as mentioned before.

4.2.1 TEETHING

In the early 19th century, Ballano's medical dictionary (1805–1807), a highly influential reference, explicitly stated that teething could lead to death. However, later medical dictionaries consistently defined teething as a physiological process and the period during which teeth emerge — aligning with both general dictionaries and those of the Royal Spanish Academy. Yet, by the end of the century, Larra y Cerezo's dictionary (1894) once again emphasized the potential dangers of teething, although it does not explicitly state that it can result in death. This may help explain why it remains a persistent cause of death.

Figure 3 *Percentage of teething records in burial books that match or do not match the same cause of death in parish registers for childhood deaths in Palma, 1836–1881*



Note: We have chosen to classify the diagnoses of causes of death by organ systems in order to unpack their possible meanings, given the low specificity of many recorded causes.

When comparing deaths recorded as teething (*dentición*) during the first year of life in burial books, we find that in more than 90% of cases, this same diagnosis appeared in parish registers (see Figure 3). However, in the case of child mortality, this percentage dropped to just over 70%, as children in this age group were likely already being diagnosed with the specific diseases that caused their deaths, rather than being recorded with a cause of death that merely referred to the period in which it occurred. These correspondences also reflect notable synonymy, including expressions such as *dentadura* (which is also translated as teething), *enfermedad de los dientes* (disease of the teeth), *de las muelas* (of the molars), *difícil dentición* (difficult teething), or *dentición laboriosa (*laborious teething), among others.

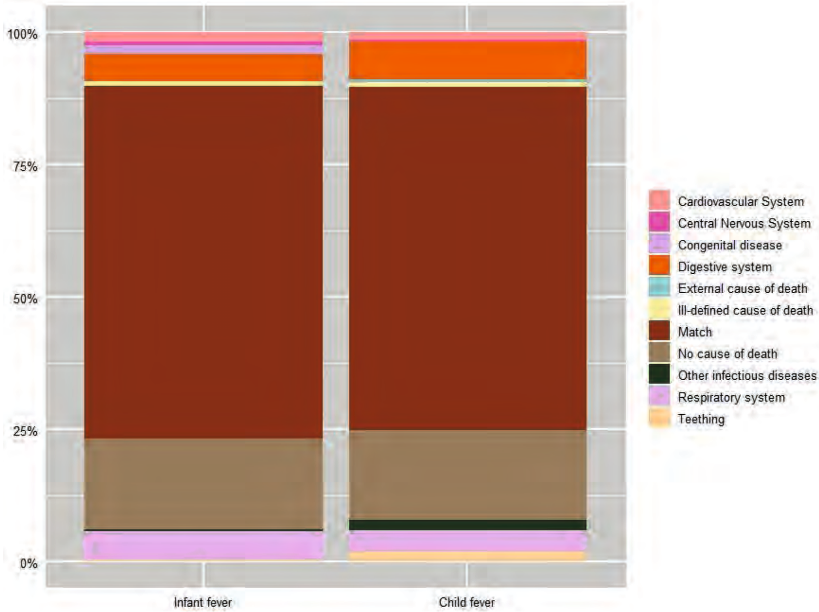
The discrepancy between both sources is due to the higher proportion of entries without a recorded cause of death in parish registers — over 17% for child mortality compared to just over 2% for infant mortality. As previously mentioned, the mandatory medical certification of the cause of death from the outset in burial records had a positive impact on reducing the number of deaths recorded without an apparent cause (Pujadas-Mora, 2024). This may also have led to a more frequent use of diagnostic labels that did not necessarily correspond to well-defined diseases but served to avoid leaving the cause of death unspecified. Furthermore, it is also important to consider that this diagnostic label was consistently used by doctors themselves. Perhaps, doctors and the responsible persons of parish registers found it practical for communicating with parents of the deceased, given their acculturation of this diagnostic label to adapt it to their own experience and knowledge, as many fatal diseases coincided with the teething period (Perdiguero, 1993). The fact that teething was an ill-defined cause of death helps explain why other similarly imprecise diagnoses, such as *calentura* (fever), also appear in parish registers. In the case of infant mortality, fever accounted for just over 5% of recorded deaths. Moreover, the strong seasonality of teething — concentrated mainly in the summer months (calculations not shown) — suggests that in more than 2% of cases, both for infant and child mortality, deaths were recorded in parish registers under a diagnosis related to the digestive system, whether infectious or not. Moreover, there was a belief that the diarrhoeal processes that occurred during the teething months should not be treated, as the expulsion of the drool was considered healthy. Purgatives were even used to force it out. It is important to note that this behaviour was guided by the recommendations of academic medicine in previous decades (Perdiguero, 1993). While some cases were also described under respiratory or other infectious diseases in parish registers, their overall representation remained minimal.

4.2.2 FEVER

The diagnosis of fever (*calentura*) in burial records matched that in sacramental registers in more than 60% of cases, both in infant and child mortality. However, in both groups, a significant percentage of deaths — 17% — had no assigned cause (see Figure 4). In this regard, the physician Fernando Weyler (1808–1879), author of a medical topography of the Balearic Islands (1854), highlighted the challenges of diagnosing fever "they do not present themselves with such decisive characteristics as to permit their prompt and exact classification" (p. 236). He further noted that "the only way to understand what happens in the fevers of Majorca is to analyze them individually" (p. 237). Weyler classified fevers into five types: inflammatory, gastric, intermittent, pernicious, and a final group that included ataxic, adynamic, nervous, and typhoid fevers. This classification likely stemmed from the nosography of Philippe Pinel (1745–1826), which had a significant influence across Europe. In Pinel's system, fever formed one of the most important disease categories (López Piñero, 1961, 1983). The use of these terms, which appear in a more concise form in the consulted medical dictionaries, reflects the evolving meaning of the term fever over time. Pinel structured his nosography around lesional signs, moving away from symptoms as a classificatory criterion.

These considerations help explain the discrepancies in causes of death between the two sources, particularly for diseases related to the digestive system — both infectious and non-infectious — which account for more than 5% of cases. They are comparable in magnitude to those found in respiratory diseases for infant mortality and slightly lower for child mortality. It is also worth noting that for both infant and child mortality — though more prominently in the latter — there is a correspondence between teething (recorded in parish books) and fever (noted in burial records). However, both terms are ill-defined, suggesting that the disease responsible for death was not known. Furthermore, the correspondence between fever and congenital diseases in cases of infant mortality highlights the challenges of accurate diagnosis at this early stage of life.

Figure 4 *Percentage of fever records in burial books that match or do not match the same cause of death in parish registers for childhood deaths in Palma, 1836–1881*



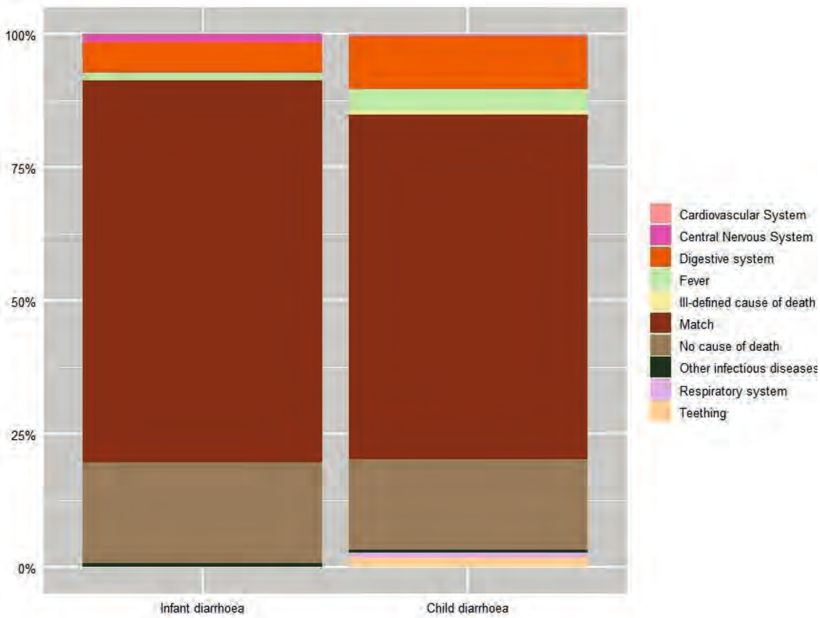
Note: We have chosen to classify the diagnoses of causes of death by organ systems in order to unpack their possible meanings, given the low specificity of many recorded causes.

4.2.3 DIARRHOEA

In the case of diarrhoea, the coincidence between the two sources was over 70% for infant mortality and 65% for child mortality (see Figure 5). In fact, diarrhoea is a condition that is less likely to be misinterpreted. Medical dictionaries from the analysed period consistently define it, logically, as the frequent passage of fluid stools from the anus. However, it is important to note that diarrhoea, often with stools of varying appearances, was frequently considered the direct cause of death. As the 19th century progressed, medical dictionary authors, after defining diarrhoea, clarified that it was, indeed, a symptom of various underlying diseases. Some dictionaries even recognized diarrhoea as indicative of conditions such as hepatic disorders, typhus, cancer, intestinal tuberculosis, enteritis, cholera, or intestinal parasitosis (Cuesta, 1884, pp. 51–52; Larra, 1894, pp. 311–312). This shift in understanding, considering diarrhoea as a symptom rather than an independent disease, may offer a possible explanation for some of the discrepancies observed, which we will address further. Rather than listing diarrhoea as the cause of death, it would have been attributed to the underlying disease responsible for it.

In the parish registers, the most significant discrepancies came from cases with no assigned cause (18.98% and 17.03%, respectively). The other most relevant group of discrepancies is concentrated in diseases of the digestive system with diarrhoea which appear in parish registers with very vague expressions such as disease of the stomach, inflammation of the belly or irritation of the belly. In the case of child mortality, the presence of 'fever' is also significant (4.05%), a poorly defined disease that could be explained by the ease of recording this generic cause instead of diarrhoea. For this reason, Weyler (1854) stated that, although dysentery "offers a very distinctive symptom, which does not exist in diarrhoea, which is the presence of blood in the evacuations, nevertheless, both diseases are often confused under the single concept of dysentery" (p. 231). In this sense, we have found in the parish books expressions of causes of death registered as 'courses of blood and vomiting'. However, the percentage of convergence between diarrhoea and dysentery as a cause of death was very low. Moreover, Weyler added that "diarrhoea is more common than dysentery, causes little havoc and is usually of short duration" (p. 231). Additionally, it can be observed that diarrhoea was linked to pulmonary tuberculosis (Caballero, 1886, p. 252; Larra, 1894, p. 312). Furthermore, as noted earlier in the discussion of wording, expressions related to chronology (e.g., chronic/acute) begin to emerge to describe diarrhoea, signalling the growing emphasis on pathochrony. This marks a shift away from the more localized perspectives on the disease.

Figure 5 *Percentage of diarrhoea records in burial books that match or do not match the same cause of death in parish registers for childhood deaths in Palma, 1836–1881*



Note: We have chosen to classify the diagnoses of causes of death by organ systems in order to unpack their possible meanings, given the low specificity of many recorded causes.

As previously documented in the diseases analysed above, we observe again how this condition is linked to teething. Notably, Cuesta (1884, p. 52) distinguishes a 'diarrhoea of teething', attributing it to an excess of blood flow to the intestine resulting from inflammation (though not explicitly termed as such) in the oral cavity. However, he does not suggest that this could be the cause of death. By the end of the century, Larra (1894, p. 312) similarly notes that diarrhoea can be caused by teething. Thus, several medical authors in the dictionaries consulted associated diarrhoea with teething — a form of diarrhoea that, as noted earlier, was considered beneficial by some segments of the population.

5 CONCLUSION

The shift towards shorter cause-of-death descriptions reflects a broader trend away from vague terms and ill-defined diseases. This transition coincided with a growing emphasis on using specific qualifiers to refine diagnoses, especially in the context of infectious diseases. As medical knowledge advanced, the need for more precise and accurate descriptions became increasingly apparent, contributing to a more standardized approach in documenting causes of death, as demonstrated by the case of Palma (Spain) during the early stages of the demographic transition. In fact, our analysis of the wording and variations in the registration of causes of death reveals that these shifts were the product of a reconceptualization of pathology. At the same time, significant changes in the disease environment — driven by the demolition of city walls, the expansion of municipal sanitation services, the hiring of public physicians, the development of water and sewage infrastructure, and even shifts in the virulence of certain diseases — contributed to a transforming epidemiological pattern. However, the accuracy in determining causes of death — understood as medical judgments — was primarily due to a deeper understanding of disease made possible by those advances in medicine, as well as the fact that recording these causes eventually became the sole responsibility of physicians.

An alternative approach to tracing the evolution in the accuracy of recorded causes of death involves analyzing discrepancies between burial records and parish registers. Most of them can be attributed to the fact that a large number of deaths in the parish records were not assigned any cause. This likely reflects stricter adherence to the requirement of recording medically certified causes of death in the burial books, where such documentation was mandatory from the outset. In contrast, parish registers — having operated for centuries prior — were only later subjected to this obligation. Other discrepancies arise from different spellings or terms that can be considered equivalent according to the terminology of the time. In some cases, though relatively few, parish records began to include terminology for causes of death that reflects an evolving understanding of disease. These are causes that are no longer based solely on symptoms — or at least are informed by nosographies that seek to incorporate other classificatory criteria, such as those derived from pathological anatomy. However, there remains some uncertainty about who was responsible for recording the causes of death in these records, since, unlike the burial books, the name of the certifying physician was not included — something that might have prevented causes being recorded without a diagnosis, even if it meant resorting to ill-defined categories. Likewise, the association of stroke with descriptions such as diarrhoea or fever is difficult to explain, although such cases are relatively rare.

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And What About the Declarants?

Professional and Relational Background of the Declarants in the Death Certificates of the Netherlands, 1812–1939

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ABSTRACT

The Historical Sample of the population of the Netherlands (HSN) is a database containing life histories from the 19th and 20th centuries. In total, around 85,600 individuals have been included, starting with the birth certificate. A distinctive feature of the civil registry is the role of declarants and witnesses in official records. In this article, I aim to provide greater insight into the nature and status of declarants in death certificates.

Keywords: Death certificates, Declarants death certificates, Civil registry, Historical Sample of the Netherlands

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1 INTRODUCTION

The Historical Sample of the population of the Netherlands (HSN) is a database containing life histories from the 19th and 20th centuries. The sample is based on birth certificates from the period 1812–1922, during which approximately 14.5 million births were recorded. The sampling fraction is 0.75% for the period 1812–1872 and 0.5% for 1873–1922. In total, around 85,600 individuals have been included, nearly all of whom (n = 85,568) have now been entered into the database (Mandemakers, 2000; Mandemakers & Mourits, 2025).

At the time of the start of the HSN, there was an ongoing debate about whether data input should be process-oriented or source-oriented. A process-oriented approach involves entering only the data deemed necessary to test a specific hypothesis or answer a particular research question. In contrast, a source-oriented approach fully incorporates an entire source (Boonstra et al., 2006). Given the infrastructural nature of the HSN, the source-oriented method was chosen as the preferred approach (Mandemakers & Dillon, 2004). This implies that from the certificates all data about witnesses and declarants were entered as well.

The HSN began in 1991 with a pilot project in the province of Utrecht, initially focusing solely on entering civil certificates. The results of this pilot were published in *De levensloop van de Utrechtse bevolking in de 19e eeuw* [The life course of the Utrecht population in the 19th century] (Mandemakers & Boonstra, 1995). Many of the research directions later pursued by the HSN (Mandemakers & Kok, 2020) were already present in this volume. A distinctive feature of the civil registry is the role of declarants and witnesses in official records. While these have been extensively studied in marriage certificates (Bras, 2011) and birth certificates (Mandemakers, 2017), they have not yet been examined in detail for death certificates. In this article, I aim to provide greater insight into the nature and status of declarants in death certificates. A distinction will be made between professional and non-professional declarants, and the rise of the professionals will be described within the context of the modernization processes in the 19th- and early 20th-century Netherlands.

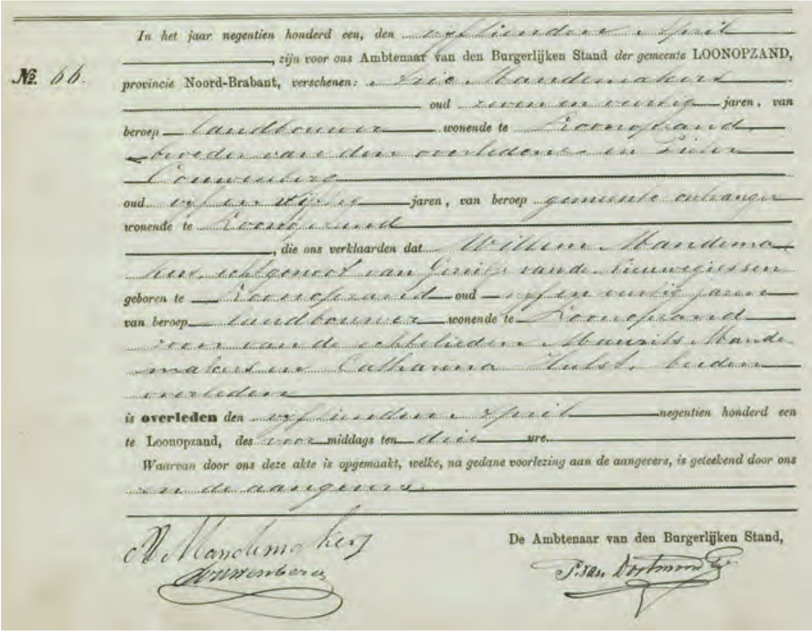
2 THE DEATH CERTIFICATE

Figure 1 provides an example of a death certificate. In addition to the name of the civil registrar, this includes the names, professions, ages, and places of residence of the declarants as well as whether or not they have signed the certificate. Until 1 January 1935, the relationship between the informants and the deceased was also recorded. The certificate also lists the names of the deceased's parents, along with their occupations, ages, and places of residence — if they were still alive. As for the deceased, the certificate includes their name, profession, age, sex, date and place of birth, date and place of death, and place of residence. Up to 1 January 1935, if the deceased was widowed, the name of their last spouse was mentioned; after that date, all spouses were recorded (Vulsma, 2002).

After death, death had to be determined. In the early 19th century, this was often done by a family member or an undertaker. When registering at the civil registry, one could also specify a cause of death. The municipality made overviews of this, but at the time, the usability was already considered questionable. With the introduction of the Burial Act of 1869, the matter was better regulated. Someone could only be buried with the permission of the civil registrar, who in turn had to receive a *doodsbriefje* (death note) from a doctor or another authorized person (until 1900, about 95% came from doctors). A cause of death was also stated on this form. Unfortunately, the cause of death was never included on Dutch death certificates, unlike in many other countries. In 1927, a distinction was made between two forms: A and B. Form A contained a concise description of the cause of death, while form B was more detailed and made a distinction between primary and secondary causes of death. This envelope remained closed and was used for the national statistics on causes of death. When the personal family card (PK) was introduced, the cause of death was recorded on the card according to form A. A practice that continued until 1 April 1956, after which a new burial law removed the cause of death from form A (van Drie, 2011; van Poppel & van Dijk, 1997).

Until 1 January 1935, the law required two declarants, after that only one. Until 1 January 1955, an extract of the death certificate was included in the death register of the place of residence if the resident died in another municipality. This extract is almost identical to the original certificate except that the declarants are missing (Elenbaas, 1936; Vulsma, 2002).

Figure 1 *Example of a death certificate*



№ 66.

In het jaar negentien honderd een, den vijftienden April, zijn voor ons Ambtenaar van den Burgerlijken Stand der gemeente LOONOPZAND, provincie Noord-Brabant, verschenen: [illegible] Vandemakers, oud twee en veertig jaren, van beroep landbouwer, wonende te Loonopzand, broeder van den overledene en Pieter Couwenberg, oud vijftig jaren, van beroep gemeente ontvanger, wonende te Loonopzand, die ons verklaarden dat Willem Vandemakers, [illegible] van [illegible] vande [illegible], geboren te Loonopzand, oud vijf en vijftig jaren, van beroep landbouwer, wonende te Loonopzand, zoon van de echtelieden [illegible] Vandemakers en Catharina Hulst, beiden overleden, is overleden den vijftienden April negentien honderd een te Loonopzand, des voormiddags ten drie ure.

Waarvan door ons deze akte is opgemaakt, welke, na gedane voorlezing aan de aangevers, is geteekend door ons en de aangevers.

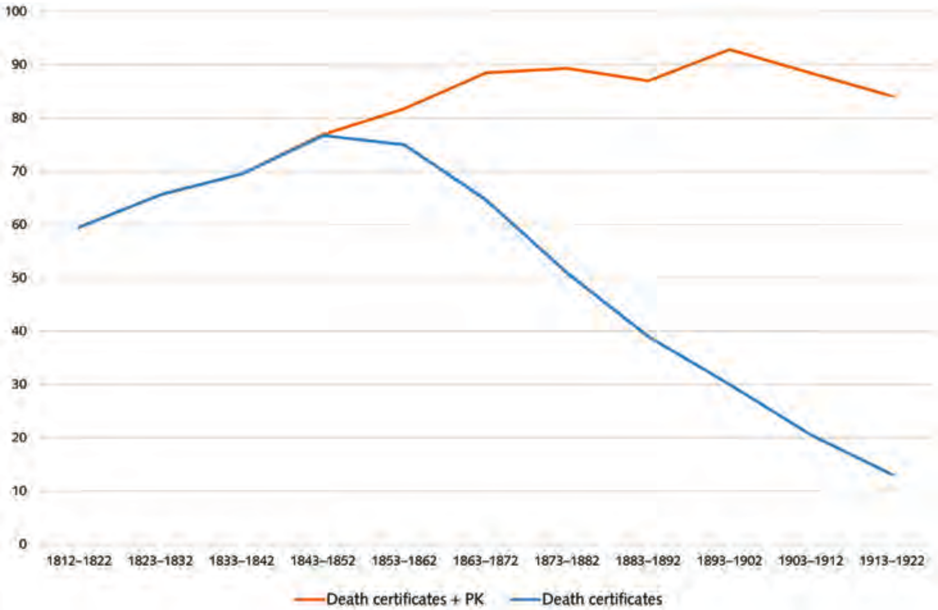
A. Vandemakers
Couwenberg

De Ambtenaar van den Burgerlijken Stand,
P. van Dortmond

Source: Overlijdensregister 1901, archiefnummer 911, aktenummer 66, gemeente Loon op Zand.

At the HSN, the death certificates were systematically collected until the date of death 1 January 1940. From that moment on, every deceased person in the Netherlands has a PK, which includes much more information than the death certificate. But on the PK there are no declarants. So, in our analysis of the declarants, we limit ourselves to the period 1 January 1812–1 January 1940 (Mandemakers, 2000).

Figure 2 *Relative number of death certificates and personal cards, HSN births, 1812–1922*



Remark: The total figures are without overlap (n = 1,128); number of births = 85,568 (100%).

The HSN database consists of 85,568 birth certificates (Mandemakers & Mourits, 2025). In 43,232 cases the death certificates of these persons were found manually through indexes and entered. In 27,265 cases the date of death was found manually through the PK and entered. Figure 2 presents the percentage of death certificates and the total percentage of death dates per birth cohort. The latter is the percentage including the PKs, which is relevant from the birth cohort 1843–1852 onwards.

The percentage of death certificates entered for individuals starts to decline from the cohort 1853–1862. Due to their birth years, more and more persons passed away after 1939 and the death date was entered through the personal card (PK). In contrast, the 1903–1922 cohort with death certificates primarily consists of individuals who died before the age of five. The percentages of records found for the period before 1853 range between 60% and 75%. From the cohort 1853–1862 till the turn of the century the percentages rose from 75% to over 90%. It is expected that with further data entry 90% will be reached for all later cohorts as well. The primary reason for missing records will be emigration abroad.

For the period before 1850, however, the main reason for the lack of death certificates remains the lack of indexes for these certificates. Especially, Amsterdam represents a significant gap. Since in first instance the data entry concentrated on deaths before the 10th birth year (based on the local 10-yearly indexes; Mandemakers, 2000), young deaths will be quite complete and therefore overrepresented before 1850.

Based on the number of 42,203 death certificates (before 1940) and regulations a number of 82,629 declarants could be expected.[1] The actual number of declarants is 82,597, resulting in a discrepancy of 32. This shortfall may be due to some certificates listing only a single declarant, though minor errors during data entry cannot be ruled out entirely.

3 WHO WERE THE DECLARANTS?

3.1 RELATIONSHIP TO THE DECEASED

Although there was a legal obligation to record the declarant's relationship to the deceased, not all declarants had such a relationship. Perhaps this value was left out when the registrar considered that there did not exist a serious relationship. Table 1 gives a first impression of the relationship of the declarants to the deceased person.

Table 1 *Presence and validity of the relationship of the declarants to the deceased, HSN death certificates, 1812–1939*

	N	%
Valid value	25,726	31.1
Valid value (father = declarant)	10,312	12.5
No valid value	381	0.5
No relationship in certificate	42,221	51.1
Relationship not entered	3,948	4.8
Total	82,588	100

In about half of the cases, no relationship is specified between the declarant and the deceased. The lack is so common that it was not noticed during the construction of the first versions of the HSN data entry program. This means that for 3,948 cases, no relationship has been entered at all. These are left out of the analysis.[2] Of the 82,588 declarants, 10,312 cases (12.5%) concerned the father of the deceased, mostly young. In cases with no relationship the surname of the declarants matched the surname of the father or mother of the deceased in 1.7% of the cases, suggesting that not all possible relationships were recorded. For the other (valid) values, this was 14.4%.

1 Death certificates registered in the form of extracts (n=214) were excluded because declarants were not extracted.

2 It concerns the input programs up to and including version 3.31. Nevertheless, in about 300 cases a relationship has been entered (taken afterwards from the comments that an importer could make). Proportionally, about 600 records (out of 3,948) with a statement of a relationship will still be missing.

Table 2 *Nature of the relationships of declarants to deceased, HSN death certificates, 1812–1939*

	N	N	%
Neighbor	6,390		17.7
Family	15,927		44.2
Father		10,312	
First degree		1,903	
Second degree		1,903	
Third degree or higher		1,809	
Witness	282		0.8
Acquaintance or friend	7,978		22.1
Not related	5,461		15.2
Total	36,038		100.0

The number of declarants with a valid value on the relationship field is 36,038. In Table 2 this group is divided according to the nature of the relationship. Neighbors mainly appear as *Nabuur*, *Gebuur*, *Buur* and *Buurman*. Apart from the father, family relationships are divided according to the distance from the deceased. The first degree mainly concerns a son or husband. For the second degree we can think of grandfathers, grandchildren, brothers, sisters (including in-laws, for example, brothers-in-law). The third degree includes all cases that are not first or second degree. The category 'Witness' includes persons who are explicitly referred to by terms such as relation 'Unknown', or 'Strange', or literally as witnesses such as *Getuige van de dood* (Witness of death). The category 'Acquaintance or friend' is in 90% of all cases 'Acquaintance' or 'Good acquaintance'. The category 'Not related' stands for values which explicitly state that there is no blood relationship; the most common values are *Geen bloedverwant* (No blood relation), *Niet verwant* (Not related) and *Geen nabestaande* (No surviving relative), plus a large number of variations on these terms. Female declarants were not common but not ruled out by law (Elenbaas, 1936). Judging by the relationship (sister, wife, maid, etc.), female declarants occur only 84 times.

3.2 FATHER AS DECLARANT

In about 24% of cases, the father is one of the declarants on the death certificate. Due to the nature of the relationship, we can expect that this will mainly involve deceased children. Table 3 shows a breakdown by age and by period. For the deceased up to four years old the percentage is above 40%, after which it drops to almost 2% for the 20-year-olds and older. This is of course partly a result of the extinction of the fathers (the oldest deceased with a father as declarant is 47 years old). Looking at the percentages per period it shows that the percentages in the period 1812–1849 are considerably higher than those in the 20th century.

Table 3 *Relative share of fathers acting as declarants, according to age of the deceased and period, HSN death certificates, 1812–1939*

	1812–1849	1850–1899	1900–1939	Total %	Total N
0	53.2	45.7	36.8	46.4	13,137
1	54.2	40.3	30.0	43.0	3,404
2	49.4	40.7	26.5	41.8	1,621
3	49.8	37.6	28.4	40.5	968
4	45.2	33.9	33.0	37.0	689
5–9	39.8	33.2	18.0	33.1	1,814
10–19	30.2	25.8	16.7	24.3	1,969
>19	10.9	3.6	0.8	1.9	18,601
Total %	48.9	29.1	7.8	24.4	
Total N	6,939	19,524	15,740	42,203	42,203

Table 4 *Relative share of fathers of 0-3 years deceased, acting as declarants, according to period and region and urbanization, HSN death certificates, 1812–1939*

	1812–1849	1850–1899	1900–1939	Total %	Total N
North-West	34,2	31,2	26,9	31.7	2,840
East	25,3	21,7	11,0	20.6	2,629
West	63,7	47,2	29,4	50.1	9,480
Middle & South	56,6	60,2	57,6	58.7	4,142
Large towns	52,5	32,4	18,2	35.7	7,753
Other	53,1	51,9	47,2	51.6	11,338
Total	52,8	43,9	34,6	45.1	19,091

One may ask if the relative disappearance of the fathers as declarants is in line with the modernization process as it developed in the Netherlands during the 19th and early 20th century. There is no room here to delve deeply into the concept of modernization, but in short, it comes down to the change in a society from more traditional patterns of life to modern ones belonging to the industrial society. Occupational differentiation and professionalization is one of the characteristics of modernization. The 'replacement' of traditional declarants such as fathers and other family members and neighbors by undertakers and official witnesses such as constables, is an example of this. In the Netherlands, modernization (as expressed in demographic developments, among other things) took place broadly from the northwestern to the southeastern part of the country (Boonstra & van der Woude, 1884; Engelen & Hillebrand, 1990; Hofstee, 1972). In this article, this has been operationalized by dividing the Netherlands into four areas: North-West, East, West, and Middle & South.[3]

For the group with the largest share of fathers, the 0 to 3-year-old deaths, this has been investigated in more detail per region and for the division in urban and rural municipalities (see Table 4 for this breakdown). In the first half of the century, the West and the Middle & South accounted for the largest share of fathers acting as declarants of their young children's deaths. While the Middle & South maintained a share of on average 59%, the West declined from 63% to 29% by the 20th century. It is the East that showed by far the lowest level in the first half of the 19th century and the relatively fastest drop to the 20th century. But the North-West also has a relatively low percentage of father-declarants. Regarding urbanization levels, the decline was most pronounced in large cities, whereas the countryside remained relatively stable at around 50% throughout the entire period. Overall, we can conclude that the disappearance of the father is a phenomenon that fits in the modernization pattern. But it is also especially something of the North-East. When we take the North-West without Noord-Holland the percentages for the North drop to levels that are even lower (13.2% for the whole period) than those of the East.[4]

3.3 OCCUPATIONS

Table 5 provides an overview of the occupations of declarants, excluding fathers. The standardization of occupations follows Mandemakers et al. (2020). Based on these standardized classifications, the occupations were further categorized according to their nature. The first distinction was whether declarants could be classified as "professional declarants" in any capacity. This group was then subdivided into occupations related to burials, such as undertakers; those employed in municipal services; and those associated with medical professions or institutions. One-third of the declarants' occupations can be classified as professional, while 61% cannot, with farmers and laborers making up by far the largest occupational groups. Values for occupational titles that are not valid primarily consist of entries such as 'no occupation'.

3 The North-West includes the provinces of Groningen, Friesland and Noord-Holland above the river IJ; the East Drenthe, Overijssel and the northern and eastern part of Gelderland; the West the provinces of Zuid-Holland and Zeeland and the western part of Utrecht; the Middle & South the eastern part of Utrecht, the western and southern part (river area) of Gelderland and the provinces Noord-Brabant and Limburg, according to Map 1 of Boonstra and Van der Woude (I984) which was based on the work of Hofstee (1981).

4 This is in line with Boonstra and Van der Woude (1984) who argue that there are reasons to split the North-West in two areas.

Table 5 *Occupational titles of declarants, HSN death certificates, 1812–1939*

	N	N	%	%
Professionals	24,095		33.3	
Funeral services		12,396		51.4
Municipal services		10,957		45.5
Medical services		742		3.1
Non professionals	43,938		60.9	
Workers		8,953		20.3
Farmers		8,747		19.9
Others		26,238		59.8
Value not valid	4,243		5.8	
Total	72,276		100	

Table 6 presents the frequency of the main professional occupations. Among those working in funeral services, the most prominent are undertakers and their assistants. The municipal service group can be divided into three main categories: constables, municipal messengers, and civil servants working in or alongside the municipal secretariat. This classification is similar to the group of 'municipal witnesses' identified as professional witnesses in birth certificates (Mandemakers, 2017). Compared to these witnesses, constables and municipal messengers played a significantly more prominent role as declarants. In the case of constables, this may suggest that they personally visited the home of the deceased. The third and smallest group of professional declarants mainly includes janitors, overseers, and occasionally a doctor or nurse.

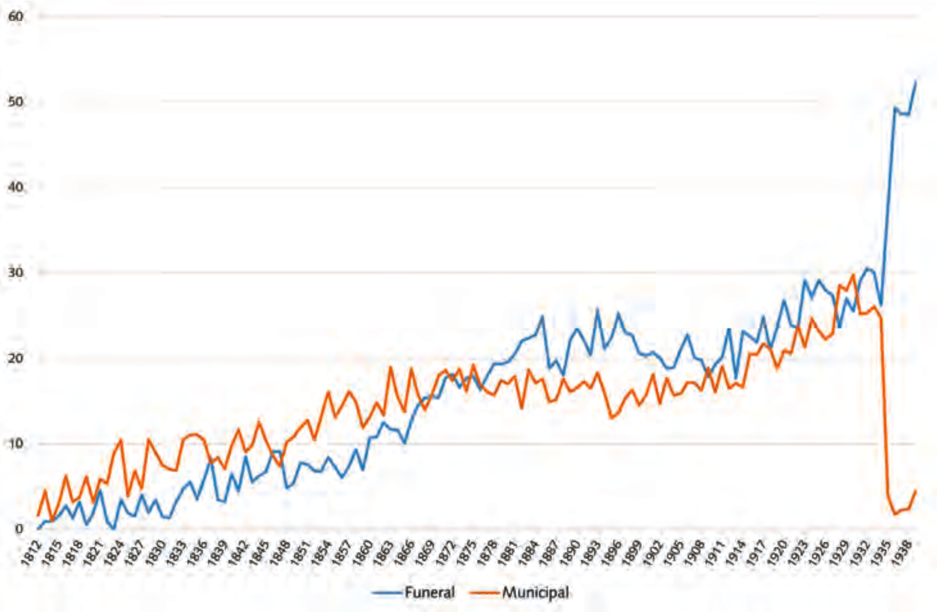
Looking at the professional titles of the declarants and their relationship to the deceased, we see that within the professional witness subgroup of funeral services, 1,499 were still recorded as acquaintances or friends, 67 as family members, and 20 as neighbors. Among municipal witnesses, the numbers were 1,332, 107, and 234, respectively. In total, this amounts to 3,231 related individuals, representing 13.6% of all professional declarants.

Table 6 *Frequency distribution of the most common occupational groups within the main categories of professional declarants, HSN death certificates, 1812–1939*

Funeral services	**12,396**
Aanspreker [undertaker's man]	7,289
Bedienaar [undertaker]	1,692
Lijkbezorger [funeral attendant]	1,677
Begrafenisondernemer [undertaker]	554
Lijkbidder [funeral prayer]	539
Koster [sexton]	430
Bode begrafenisfonds [collector funeral fund]	215
Municipal services	**10,957**
Gemeentebode [municipal messenger]	4,268
Ambtenaar secretarie [secretarial civil servant]	3,593
Veldwachter, politieagent [constable, police officer]	3,096

Figure 3 illustrates a steady increase in the proportion of professional declarants, rising from 10% in 1820 to around 40% between 1880 and 1920, and further to approximately 60% by the late 1930s. A particularly striking — if not dramatic — shift occurred following the enactment of the 1935 law, which appears to have led funeral professionals to almost entirely replace municipal witnesses. The main reason for this change was the reduction in the number of required declarants to just one, making a single individual carrying the obituary sufficient. This development, till 1935, parallels the relative rise of municipal witnesses in birth certificates, which increased from below 10% to around 40% between 1812 and 1922 (Mandemakers, 2017).

Figure 3 *Relative share of the professional declarants, HSN death certificates, 1812–1939 (n = 24,095)*



The next question is whether this trend toward professionalization followed the broader pattern of modernization in the Netherlands. Table 7 presents the percentages for funeral service professionals alone, revealing that this development was mostly characteristic of urban areas, where their share grew from 11% to nearly 50% over the course of the period 1812–1939. From a regional perspective, the western part of the country — with its concentration of major cities — took the lead, while the southern region lagged behind. We conclude that funeral services only partially followed the expected modernization pattern, with the northern and eastern regions falling short of expectations.

Table 7 *Relative share funeral services as declarant, per region and cohort, HSN death certificates, 1812–1939*

	1812–1849	1850–1899	1900–1939	Total %	Total N
North-West	2.3	6.7	18.2	10.2	12,942
East	4.4	11.1	18.4	13.3	12,919
West	6.2	30.8	42.0	30.9	27,021
Middle & South	2.6	5.3	8.9	6.5	15,042
Large towns	10.8	40.3	47.1	39.3	26,765
Other	0.8	3.0	8.1	4.5	41,159
Total	4.4	17.0	24.8	18.2	67,924

Rising professional groups replaced more traditional ones: acquaintances and neighbors. These groups were typical of the 19th century. After 1900, they accounted for no more than 1% of all declarants, whereas between 1812 and 1850, neighbors made up 30% and acquaintances 19%. Neighbors were especially concentrated in the Middle & South (49%), as well as in the North-West and East, with 38% and 40% respectively. If there ever was a typical *naoberplicht* (neighborly duty), it was not particularly characteristic of the eastern part of the country. Acquaintances, by contrast, were more often found in urban areas.

3.4 CHARACTERISTICS NON-PROFESSIONAL DECLARANT

One might expect that a person who comes forward as a declarant would also be able to sign the document. However, this was not the case for 3,603 non-professional declarants (8.1%), including 55 individuals who abstained from signing due to Sabbath observance (Orthodox Jews were permitted to declare but not to sign), and 90 others for reasons unrelated to illiteracy. Failure to sign was most common among neighbors and family members in the first half of the 19th century. Among fathers acting as declarants, 20.8% did not sign. This aligns with the pattern seen in the birth registrations, where only 2.5% of the same fathers displayed incongruent signing behavior.

Declarants had to be at least 21 years old and before 1901 even 23 years old (Vulsma, 2002). The average age of all declarants was 45.4 years. The professional group of the funeral services was clearly older than the average at 48.6 years, the secretarial part was close to the average at 44.4 years. If the relationships with the deceased are considered, the first and third degree family is the youngest (40.6 years) and the second degree is the oldest (48.4 years). The neighbors (43.3) and acquaintances and friends (44.7) are below the average age. Given the uneven distribution of the age at death (relatively many young and older deceased), it makes little sense to compare the average age of the father-declarant and deceased.

Finally, we compare the social background of the non-professional declarants and deceased where the deceased was 18 years or older at the time of death. For the social background, we use the HISCLASS classification of social groups (van Leeuwen & Maas, 2011). HISCLASS is based on the HISCO coding (van Leeuwen et al., 2002). In practice, the standardizations and codes of Mandemakers et al. (2020) were used. Of the 42,417 deceased, 19,185 were at least 18 years old. Because of the large number of older deceased, the professional title 'without profession' predominates. In addition, 'without profession' was usually recorded for married women as well. All told, 7,212 cases remained with a corresponding number of 8,682 non-professional declarants with valid occupations.

The distribution by social background is shown in Table 8. In half of the cases (49.6%), the declarant and the deceased come from the same social background. This is especially true for farmers where 66.7% share the background. Also striking is the broad distribution by social background of the declarants among the deceased skilled workers; a distribution that also corresponds quite well with the total distribution.

The HISCLASS classification is a classification by social class. Another way to classify people is by social position. An increasingly used classification for this is the HISCAM classification, in which each profession is scaled to a continuum from 1 to 100 (Lambert et al., 2013). The advantage of such a scale is, among other things, that professions can be compared across the different classes. The question then becomes to what extent the average HISCAM score of the declarants differs from that of the deceased. Table 9 shows the average social status on the HISCAM ladder per HISCLASS category. Even though we see relatively large differences among the deceased (except between the skilled workers and the farmers), among the declarants the averages are close to each other, although there is still a social gradient.

Table 8 *Social background deaths 18 years and older and non-professional declarants, HSN death certificates, 1812–1939*

Social background deceased	**Social background declarants**					
	1	2	3	4	5	Total
1 Elite (higher managers and professionals)	12.6	27.4	37.7	13.5	8.8	215
2 Lower middle class	4.1	31.7	33.7	8.8	21.7	978
3 Skilled workers	2.8	16.7	44.0	14.0	22.5	1,954
4 Self-employed farmers and fishermen	1.5	6.2	13.6	66.7	11.9	2,451
5 Unskilled workers and farm workers	2.2	13.2	21.4	15.3	47.8	3,084
Total (N)	225	1,257	2,266	2,497	2,437	8,682
Total (%)	2.6	14.5	26.1	28.8	28.1	100.0

Remark: In 1,470 cases there were two (non-professional) declarants for one deceased person (these cases are included twice in the table). The distribution of the 12 HISCLASS categories over five new assembled ones was done according to Mandemakers et al. (2020).

Table 9 *Social background in terms of social status (HISCAM), deceased and declarants, HSN birth and death certificates, 1812–1939 (n = 8.682)*

Social background deceased	Average HISCAM	
	Deceased	Declarants
1 Elite (higher managers and professionals)	80.2	59.9
2 Lower middle class	66.1	56.9
3 Skilled workers	52.1	54.4
4 Self-employed farmers and fishermen	54.1	54.0
5 Unskilled workers and farm workers	48.0	52.8

Remark: In 1,470 cases there were two (non-professional) declarants for one deceased person (these cases are counted twice in the average). The distribution of the twelve HISCLASS categories over five new assembled ones and the attribution of the HISCAM levels was done according to Mandemakers et al. (2020).

4 SUMMARY AND CONCLUSION

Until 1 January 1935, every death in the Netherlands had to be reported to the municipality by two declarants. From 1869 onward, one of these declarants was required to present a notification of death issued by a doctor.

In analyzing the declarants of death certificates, their relationship to the deceased was a key focus. A total of 42,417 individuals from the HSN sample had a death certificate dating from before 1940. Among the declarants, a distinction was made between professional declarants — such as those involved in funeral services, municipal employees, or individuals working in the medical or healthcare sector — and non-professional declarants. The share of professional declarants steadily increased from 22% in the first half of the 19th century to 50% by around 1930. When the requirement was reduced to just one declarant, the proportion of professional declarants rose to 60%.

Fathers often acted as declarants, particularly for the deaths of young children. This practice was most common in rural areas and remained prevalent in the Middle & South well into the 20th century, where more than 60% of declarants were fathers. Among non-professional declarants, there was strong social homogeneity between the deceased and the declarant among the skilled workers and the farmers.

Overall, the professionalization of declarants aligned with the broader modernization trends in the Netherlands. Notably, the eastern region played a prominent role in this development, taking the lead in the professionalization of funeral services. This pattern mirrors the growing involvement of municipal services as witnesses in birth certificates. These trends contrast with those observed in marriage certificates. Hilde Bras (2011) identified a reverse trend after 1880, attributing it to the growing ceremonial and cultural importance of marriage. This shift increasingly assigned the role of witness to family members, at the expense of friends and unrelated individuals.

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II

Mortal Beginnings

HISTORICAL LIFE COURSE STUDIES
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East is East, and West is West?

Comparing Historical Infant Mortality Rates in Taiwan and the Netherlands

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ABSTRACT

This article compares infant mortality rates in two historical populations: one in Europe (the city of Nijmegen) and the other in China (the city of Lugang). The author investigates whether the Malthusian divide — between a preventive demographic system in Western Europe and a positive system in Asia — can be substantiated. The conclusion is that the differences in infant mortality were not as large or as structural as often assumed. Furthermore, the expected negative impact of the patrilineal family system on girls in China could not be confirmed.

Keywords: Historical demography, Infant mortality patterns, Comparative research, China, the Netherlands

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1 INTRODUCTION

Conclusions of scientific research are most intriguing when they challenge existing views. The reason for this is simple: whenever theories can be falsified, partly or fully, this opens up avenues for new insights. It is the eternal dialectical process that drives scholarly progress. I experienced this myself when studying the differences between Chinese and European demography in the past two centuries. According to traditional Malthusian ideas we were expected to find in China until the 1950s a system of young and almost universal marriage, and in Europe, that is west of the line Triëste–St. Petersburg, up to the Second World War a restrictive marriage pattern. John Hajnal and others found this to be true for western Europe, be it with some variations. The Chinese population, again in Malthus' view, had to pay a high price for universal marriage. Since this resulted in a large number of births, he predicted, nature would correct this reckless behavior with a red pen by way of positive checks, excess mortality (Malthus, 1798).

In the comparative project *Population and Society in Taiwan and the Netherlands* we took these notions as our point of departure. Since we had elaborate data on the individual level at our disposal we could test Malthusian predictions. To those who wondered whether these countries can be taken as representative examples of European and Chinese societies, we explained that, like in an analysis of variance, the differences within Europe and within Asia are much smaller than between Europe and China. There are case studies galore to show this.

Within the larger project, Hsieh Ying-hui and I chose to compare two cities in more detail, Nijmegen in the Netherlands and Lugang in Taiwan (Engelen & Hsieh, 2007). For Taiwan, the most reliable data are gathered by the Japanese authorities. Between 1895 and 1945 Japan occupied the island and kept extraordinary precise and elaborate registers of the population. Our period for comparison in Nijmegen was chosen carefully in such a way that the decor against which the demographic processes played was similar. We looked at the number of inhabitants, the economic structure, the phase in the demographic transition, and the shape of the age-distribution. By choosing for the years 1840 to 1890 in Nijmegen, we had two cities with comparable characteristics.

In this paper we focus on a striking characteristic of pre-industrial demography, infant mortality. By modern standards the chances of survival for newly born children in pre-modern societies were astonishingly low. Almost one quarter of infants did not reach their first birthday, and mortality remained high throughout childhood.

The high death rates of the very young highlight the economic conditions of life in the societies involved, the social differentiation within these societies, and the "deliberate" choices made by the historical actors. Since infant mortality is a reflection of general mortality, more precisely the mortality of the most vulnerable members of society, its level also permits us to assess whether Malthus' prediction on the prevalence of positive checks in Chinese society is valid. According to the classic division of the world in two parts, mortality should be significantly higher in our Taiwanese city than in Nijmegen.

Positive checks thus may show in a high level of mortality, but can also have a very deliberate form. Malthus already mentioned the "custom of exposing children". James Lee and his collaborators also emphasized infanticide as one of the ways in which Chinese couples consciously regulated the number of their offspring. In their view, the gender-differentiated character of infant mortality provides us with a strong indication of "proactive" behavior rather than of positive checks (Lee and Wang, 1999). For that reason, the comparison between Nijmegen and Lugang will deal explicitly with possible differences in infant mortality between girls and boys. Is there indeed a higher mortality among Lugang female babies than among male babies? And — since this custom does not have to be restricted to Chinese parents only — do we find evidence of gender differences in infant mortality in Nijmegen as well?

These then are the two questions we want to answer in this paper:

1. Was infant mortality in Lugang indeed significantly and structurally higher than in Nijmegen?
2. Were the chances of survival for baby girls in Lugang (and Nijmegen) significantly lower than for boys?

2 THE GENERAL LEVEL OF INFANT MORTALITY

In 19th century Europe, adult mortality stabilized or even slightly declined, but the chances of survival for the very young declined in the third quarter of the century. In Germany, France and Spain we find evidence of rising infant mortality between roughly 1840 and 1870. In his 1913 presidential address for the American Association for the Study and Prevention of Infant Mortality, L. Emmett Holt attributed the mid-19th century increase of infant mortality to the process of urbanization and industrialization. Densely populated cities and, especially, mothers working in factories proved fatal for many babies (Emmeth Holt, 1913). The link between women's work and infant mortality is breastfeeding, because all authors agree on the fact that the extent and duration of breastfeeding is the best predictor of infant mortality (Knodel, 1988). The Netherlands were no exception. Van Poppel and Mandemakers observed that for the country as a whole infant mortality increased markedly between 1840 and 1875 (van Poppel & Mandemakers, 1997).

What about Taiwan then? The Japanese colonial government not only compiled a large amount of high quality data between 1895 and 1945, but also heavily influenced mortality rates. The Governor-General soon found that more Japanese soldiers died in Taiwan of diseases than as a result of hostilities. In order to eliminate major epidemics and indigenous diseases on the island he launched large-scale programs to control major epidemics, to improve public health conditions, and to increase medical resources. The effects of these efforts show in a declining crude mortality rate of Taiwan from 30 per 1,000 inhabitants in 1906 to 16 per 1,000 in 1942 (Shi-yung, 2004).

Whether or not this general decline affected infant mortality has been recently assessed on the basis of data for 14 field sites across the island. Male infant mortality went from 223 in 1908 via 195 in 1925 to 130 in 1945, and the respective rates for women were 207, 136 and 130; an impressive decline. On the whole, the colonial period thus witnessed a general decline in infant mortality and a closing of the distance between male and female infant mortality (Yang & Hsieh, 2004).

The findings mentioned above guide our expectations for infant mortality in Lugang and Nijmegen. If the Dutch town lives up to the national average, the mortality among the very young will probably show a rise, whereas the probability of survival for babies in the Taiwanese town is expected to increase. We also wanted to know whether the rates developed in time. Therefore, we calculated in Nijmegen infant mortality by using the information on two periods (1850–1869 and 1870–1889). The Lugang infant mortality rates in the table start in 1920. This is because Barclay established an underregistration of births and deaths for the period before 1915. The same author also refers to three exceptional years of epidemic diseases. In 1915 the country suffered from an unusual high number of malaria deaths. The worldwide influenza outbreaks following World War I hit Taiwan in 1918 and 1920 (Barclay, 1954). Since we are interested in the long term development, we used information only from 1920 onwards, and divided the rest of the colonial period into 1922–1933 and 1934–1945. In the comparison both of level and development of IMR we refer in both cases to an earlier and a later period.

Table 1 *Infant Mortality Rates (IMR) in Nijmegen and Lugang*

	Nijmegen IMR	Lugang IMR
Early period	139	206
Later period	167	144
N births	*4,191*	*3,898*

Sources: Nijmegen civil registers and censuses; Lugang population database of the Program for Historical Demography, Academia Sinica.

The change we find in Nijmegen follows the general direction we expected from existing research. In other words, the general rise of European IMR in the second half of the century is visible for Nijmegen residents too. By contrast, Lugang IMR declines dramatically from well above the Nijmegen level (206 versus 139) at the beginning of the periods compared here to a level below the Nijmegen value (144 versus 167). This allows us to deal with the prediction made by Thomas Malthus. He expected positive checks to be more active in China than in Europe. Since we have only two observations for Lugang, we have to be careful with our conclusions. Still, Malthusian penalties for unlimited female nuptiality seem indeed to have been the fate of Lugang in pre-Japanese times. The Japanese health measures started before 1920, so the IMR must have been higher at the start of the century, leaving us with a marked difference when compared to Nijmegen. We have to put this observation in proportion, however. First, the sharp decline between the two periods shows that a Chinese population could very quickly move to an IMR markedly below the average European level. On the other hand, a European population could reach a "Chinese" level of IMR at the same pace. The difference in other words, was not as structural and as marked as we expected.

3 DIFFERENT MORTALITY AMONG MALE AND FEMALE INFANTS?

In order to trace the existence of positive checks we also have to look for sex differences in infant mortality in our two towns, because both Malthus, in 1798, and Lee and Wang, in 1999, referred to infanticide as one of the options for Chinese parents for controlling the number of their offspring. Given the importance of sons under patriliny this method would be applied more often to girls than to boys. The topic is very complicated however. First of all, biology favors survival chances for girls. When mortality rates for boy and girl infants are the same, this implies that sons get preferential treatment. Only a higher female infant mortality rate points almost certainly at gender specific treatment of infants. Also, from the demographic measures one cannot learn what exactly happened. A society could use direct infanticide, preferential neglect, differences in age of weaning, or a combination of all these measures.

The literature on this subject is biased in suggesting that direct or indirect infanticide is a Chinese or Asian predilection. Still, when comparing a European and a Chinese population, one has to be aware of son preference in European societies too. Isabelle Devos, for instance, found that mortality of girls in 19th century Belgium was 15 to 20% higher than mortality of boys, especially for childhood and adolescent mortality. Devos claimed that her findings were representative of most Western European countries (Devos, 2000). There is evidence, however, that a higher probability of dying for girls is not a universal European phenomenon. Knodel, for instance, did not find evidence for preferential treatment of sons in his 14 German villages (Knodel, 1988). More recently, a comparative survey concluded that Europe indeed had its "missing girls" too, be it especially in East and South Europe (Beltrán Tapia & Szołtysek, 2022).

Table 2a *IMR, neonatal, and postneonatal mortality Nijmegen*

	IMR		neonatal		postneonatal	
	M	F	M	F	M	F
Early period	145	133	38	29	111	107
Later period	190	142	44	27	153	118

Table 2b *IMR, neonatal, and postneonatal mortality Lugang*

	IMR		neonatal		postneonatal	
	M	F	M	F	M	F
Early period	222	188	137	105	99	92
Later period	155	133	83	57	78	80

Sources: Nijmegen civil registers and censuses; Lugang population database of the Program for Historical Demography, Academia Sinica.

In both cities average infant mortality among boys is higher than among girls. More precisely, male infant mortality exceeds female infant mortality in Nijmegen by 13% and in Lugang by 18%. When we divide infant mortality into neonatal and post-neonatal mortality the conclusion for Nijmegen remains the same. Also, the development between our two periods points in the same direction, although less unequivocally. The Lugang data tell a different story. Assuming that neonatal mortality is caused mainly by endogenous factors, we find the expected result. Male neonatal mortality is higher and even rises between the two periods. In post-neonatal mortality, on the other hand, we find evidence for preferential treatment of sons. Even in the first period the differences between male and female mortality are only marginal. More significant is the finding that between 1933 and 1945 female post-neonatal mortality was 5% higher, which runs contrary to the biological advantage girls had over boys. The interpretation could be that at this point son preference shows itself. Interestingly, though, this is not the case for neonatal mortality in the two periods, and it also does not appear for post-neonatal mortality in the first period. These calculations therefore provide no evidence for a specific Chinese form of "proactive" behavior, namely infanticide, the less so since we also find relatively high female infant mortality in Nijmegen during the earlier period.

4 CONCLUSION

When we measure positive checks through infant mortality, the difference between Nijmegen and Lugang was not that great with an 85% survival chance for Nijmegen babies against 83% in Lugang. During the period covered here, infant mortality in Nijmegen increased from 139 to 167 — an increase found in many European countries — whereas the medical policy of the Japanese colonial government in Taiwan resulted in a declining infant mortality, from 206 to 144. This shows that the difference between our Chinese and European populations was neither as structural nor as marked as one would expect from the Malthusian model.

We do find a marked difference between Nijmegen and Lugang when we compare the levels of neonatal and post-neonatal mortality. Neonatal mortality in the Dutch town was stable or slightly declining — again, as was the case in most European countries — while neonatal mortality in Lugang was double the Nijmegen level. The sharp increase in Nijmegen infant mortality was therefore completely driven by post-neonatal mortality. We deduce from these findings that delivery and taking care of newly born babies in the Netherlands was arranged better than in Taiwan. The increase in post-neonatal mortality on the other hand proves that the economic conditions in Nijmegen deteriorated in the 19th century. In the colonial period, the opposite was true for Taiwan.

The most surprising result from our multivariate analysis is the predominance of biological factors when explaining infant mortality (Engelen & Hsieh, 2007). Although social class in a descriptive presentation seems to influence the level of infant mortality, this association disappears when we control for other variables. The only class effect that remains is found for neonatal mortality in Nijmegen. Obviously, the richer the parents, the better medical care they could hire for the delivery and the immediate aftermath. For the remainder we only find that in both cities chances of dying for infants are influenced by being a twin and by small birth intervals. Interestingly, the sex differences in infant mortality do not follow the path shown by Malthus or Lee. Male babies died more often than female babies, and we therefore have no evidence of female infanticide.

One final remark: the Taiwan case cannot simply be taken as exemplary for China. Although many characteristics of the Taiwanese society were typically Chinese, the Japanese occupation transformed Taiwan into a more modern version of the regions on the mainland.

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Birth Weight and Prematurity in Copenhagen 1927

Health at Birth Explored in Midwife Records From Home Births and Births at the Royal Maternity Hospital

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ABSTRACT

This study examines two key indicators on perinatal health — birth weight and prematurity — based on 1,151 home births in Copenhagen in 1927, attended by trained, examined, authorized midwives, who had to call in a physician to assist in cases of complicated births. This is a 17% sample of all home births in the city, which comprises two thirds of all births in Copenhagen for that year. To compare, we also examine 398 hospital births, equal to a 10% sample of all births at the Royal Maternity Hospital for the same year. The findings reveal significant differences between home and hospital births in infant health at birth, attributable to the selective nature of hospital admissions. Hospital births revealed an average birth weight of 300 grams lower than home births and a preterm rate of more than four times as high (33% vs. 7%). The study is based on midwife birth registers in which Danish midwives registered details of every birth they attended. A nearly complete collection of these birth registers 1861–1978 is preserved at the Danish National Archives. This is a pilot study meant to explore the possibilities for further studies in this extensive source for individual birth information, of which our sample constitutes only a tiny part. But already we can see not only differences between hospital and home births but also different individual profiles in the clientele and the practices of the midwives. This shows the need for careful analysis before interpreting differentials in historical health data registered in different settings.

Keywords: Birth weight, Prematurity, Home birth, Midwives, Birth attendance, Perinatal health, Early 20th century, Denmark

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1 INTRODUCTION

Internationally, comprehensive collections of historical birth weight information for home births are rare. Much rarer than recordings of birth weights from lying-in hospitals. Many hospitals began systematically collecting data on birth outcomes in the late 18th century and started to include birth weight in the mid-19th century. Therefore, historical studies on birth weight have often had to rely on records from lying-in hospitals. However, until the mid-20th century, such institutions only catered for a minority of women giving birth, most often vulnerable sub-populations, such as unwed women, married women living in severe poverty and women with a history of very complicated births. This represents a selection bias which makes information about birth weight for more average parts of the population very much in demand for studying historical perinatal health for whole populations.

Denmark offers a unique case of study as the Danish National Archives holds a large, population wide collection of midwife-birth-registers for home births 1861 to 1978. Maturity information is systematically included since 1861 and birth weight information since the 1920s.

This article is a pilot study focusing on birth weight and prematurity information from a sample of midwife-birth-registers for home births and births at the Royal Maternity Hospital in Copenhagen in 1927. The aim is twofold: 1) to explore whether birth weight and prematurity among home births in Copenhagen differs from births taking place at the Royal Maternity Hospital; 2) to gain domain expertise about the Danish midwife-birth-registers as a source for historical perinatal health by identifying and addressing methodological challenges in studying health from these records. We will focus on differentials in the clientele of the individual midwives and challenges in assessing the accuracy of the weighing both according to the individual midwives and the weighing technology of the time.

2 FORMER STUDIES OF HISTORICAL BIRTH WEIGHT

Birth weight and prematurity are often used as key measures of newborn infants' health, reflecting the interplay of maternal health, environmental influences and socio-demographic factors, and thereby offering insights into patterns of health and well-being in populations. Since Barker et al. (1993, 1997) published their now classic studies on later-life health effects of birth weight, there has been an increasing interest in life course epidemiology, prompting an international demand for trustworthy information about historical birth weight. Most of these studies have been based on lying-in hospital records. For instance, Koupil (2007) and Schneider (2017) provide comprehensive presentations of a range of such studies and the selections bias of the women who gave birth at hospitals, before the large-scale shift in birth place from home to hospital during the mid-20th century in the western world.

We have only been able to identify three studies that had access to birth weight data for the majority of historical births taking place at home. Andersson et al. (2000) is based on both home and hospital deliveries in Gothenburg: full-term singleton, female births with known gestational age, born into five birth cohorts (1908, 1914, 1918, 1922 and 1930). They found that infants born at home had a higher birth weight across the cohorts (p. 270). Schneider (2017) used records from maternity hospitals, but included records from the Boston Lying-In Hospital's outpatient clinic, which consisted of home births. He finds the average birth weight was 150 grams higher at the outpatient clinic than at the Hospital (Table B1). The Uppsala Longitudinal Study of Adult Men (ULSAM) is an ongoing study which includes both hospital and home births of males born 1920 to 1924. To that we can add Quaranta et al. (2022), who use Swedish hospital-birth weight data from the 1930s and 1940s. In Sweden, however, the majority of births had already moved to hospitals by then, so the selection bias should be less.

3 BIRTH ATTENDANCE AND PLACE OF DELIVERY IN DENMARK

The Danish birth care system has been midwife-based for centuries. What is different to many other countries is that physicians never competed with midwives to attend normal births, although midwives were obliged to call in a physician in case of a complicated birth. Midwives were included in the Danish authorized healthcare personnel in 1672, which was the first occasion such a category became meaningful. Since 1714 midwives have had to be formally examined and sworn in. In 1787 the National School of Midwifery opened at the Royal Lying-In Hospital in Copenhagen and the Midwifery Law (1810) decreed that every provincial town and rural parish was to have a district midwife trained at the school. A district midwife had to build up a private practice and charge patient fees, but she also received a basic wage, including a free house with a garden, firewood and fodder for a cow, paid for by taxes. In return, she had to attend free of charge births of destitute women. All midwifes trained at the School of Midwifery were free to compete with the district midwives as private practising midwives without the basal financial support. This way, thinly populated areas were provided with professional birth attendance, while districts with many births could attract more midwives without increasing public expenses. Since 1830, virtually all Danish births have been attended by professionally trained, examined, authorized midwives (Løkke, 1997, 2002, 2007, 2012). In the early 20th century, about three quarters of the midwives outside Copenhagen were subsidized district midwives (Nellemose, 1935).

In the city of Copenhagen, subsidized district midwives were never introduced. Instead, the authorities determined how many privately practising midwives to authorize, to make sure all of them could earn a decent livelihood. In the late 1920s, the number of midwives was officially fixed at 108 for the administrative unit of Copenhagen and at 136 for the greater Copenhagen area including Frederiksberg and Gentofte (Nellemose, 1935). Professional birth attendance for all was guaranteed in Copenhagen by the Poor Relief Board, who paid the midwives' fees on behalf of patients too poor to pay themselves (The Midwifery Law, 1914).

Until the early 20th century, nearly all births in Denmark were home births. The one and only lying-in hospital was the Royal Maternity Hospital, which had been founded in 1762. The primary aim of the Royal Maternity Hospital was to provide free and anonymous professional birth attendance for unwed expecting mothers, who might otherwise have given birth unattended and killed the infant afterwards (Løkke, 2013). In 1910, the Royal Maternity Hospital was integrated into the newly-built *Rigshospitalet* (The National Hospital) and expanded into two wards, A and B. In 1927, wards A and B admitted primarily unwed women (two thirds). The other third were married women who were either living in such poor conditions that home birth was deemed dangerous or they were expecting severe birth complications. Ward C was established in 1924 because of an acute housing shortage in Copenhagen. The ward was paid for by the poor relief authorities (*Københavns fattigvæsen*) and catered for married women living in Copenhagen expecting a normal birth, but living in housing conditions so bad, that home birth was considered unsafe for mother and child. Of the married women in wards A and B, approximately 10% lived outside the Copenhagen area and were admitted because of complicated births. As the majority of unwed mothers were admitted anonymously, it is not known how many of them were living in Copenhagen. The hospital estimated that at least half of the unwed in-patients were living outside the Copenhagen area in 1927 and in this way, their numbers have contributed to the inflation of both the number of births and the illegitimate rate in Copenhagen (Beretning om Rigshospitalet, 1928). Thus, the population giving birth at the Maternity Hospital in 1927 was a vulnerable subpopulation admitted precisely because of their marital, social or medical vulnerabilities.

Like in most of Western Europe, the place of birth in Denmark shifted from home to hospital in the mid-20th century. However, while Sweden was among the first to do so, Denmark was among the last (Vallgårda, 1996). In 1927, more than 80% of all births in Denmark still took place at home. However, in the administrative unit of Copenhagen, two thirds of all births were home births this year. The total number births (including stillbirths) was 9,950. Of these, 3,101 took place at the Royal Maternity Hospital, while 321 births took place at newly-established, small private maternity clinics (Statistisk Aarbog for København, 1928, Table 19 and 37).

4 MIDWIFE-BIRTH-REGISTERS

The collection of midwife-birth-registers (*fødselsprotokoller*) for all of Denmark for the years 1861 to 1978, held at the Danish National Archives (*Rigsarkivet*), represents a real treasure trove of information about the individual women giving birth, the birth itself and the new-born infants.

The midwives recorded details of every birth they attended in a bound volume of pre-printed forms provided by the health authorities. When a midwife had completed a register, she submitted it for approval and then obtained a new one from the health authorities. Right from the start in 1861, the midwife-birth-registers recorded a wide range of information about the woman giving birth, the progress of the birth, if the infant was born prematurely or at full term and whether the mother and infant survived the birth. Some midwives voluntarily included birth weight during the late 19th century; however, the Midwifery Law of 1914 and the Midwifery Instruction of 1920 made the recording of information about a newborn infant's weight and length mandatory. Whenever a midwife obtained a new preprinted birth-register after she had completed her old one, she was obliged to fill out the now included new columns for length and weight.

By examining the midwife-birth-registers from Copenhagen, we found that 1927 was the first year when all midwives, who had records for the full year (97 midwives), had also all systematically registered birth weight. That is why we choose 1927 for this study. The midwife-home-birth-registers for Copenhagen consist of 469 archival boxes and are archived are archived under the *arkivskaber* (archival creator) *Stadslægeembedet i København*. The older ones are on-line readable (Stadslægeembedet i København, 1862–1977).

The midwife-birth-registers (*journalprotokoller*) from the Royal Maternity Hospital are also kept at the National Archives. The oldest date from 1763, but not all years are preserved. In 1927, the midwives who attended births at the Royal Maternity Hospital recorded them using the same pre-printed forms in bound volumes as the privately-practising midwifes used for the home births.

5 OUR SAMPLE OF COPENHAGEN BIRTH RECORDS FOR 1927

This study is based on midwife-birth-records from 1,549 deliveries resulting in 1,508 live births in Copenhagen in 1927. Of these, 1,151 deliveries (1,133 live births) were home births and 398 deliveries (375 live births) took place at the Royal Maternity Hospital. This represents approximately 17% of all home births in Copenhagen and 10% of all births at the Maternity Hospital in 1927 (Statistisk Aarbog for København, 1928, Table 19 and 37). We designed the sample as follows:

For the home births, we identified 111 bound volumes of personal midwives-birth-registers from Copenhagen which included records for the year 1927. Ninety-seven midwives had records for the full year and systematically registered birth weight. We reviewed each of these 97 bound volumes to collect information about their general birth-recording. Of the 97 volumes, we fully transcribed all records for 1927 from 13 midwives. We chose 13 midwives from those who had attended a minimum of 50 births in 1927, and who systematically used the metric system, and we ensured geographic spread across city districts. The area of Frederiksberg is not included, as it is an independent administrative unit.

For the hospital births, we fully transcribed every tenth midwife-birth-record for 1927 from the three maternity wards A, B and C.

Birth weight in our sample was registered in kilos (e.g. 4, 3½, 3¼) or in grams (e.g. 2,500, 4,000, 3,780). Prematurity was reported as yes/no answers in two columns headers: *skønnes fuldbaarent* (estimated full term) and *skønnes ufuldbaaren* (estimated premature).

6 THE MIDWIVES, THE MATERNITY WARDS AND THE MOTHERS

The socio-demographic profile of the mothers in our sample differs, as expected, very much between the home birth clientele of the midwives and those in the hospital wards (see Table 1). Those in wards A and B were younger, two thirds of them were unwed and a little more than 60% were primiparous women, experiencing their first delivery. Of the midwife patients, far fewer were under 26 years of age, the rate of primiparous women was lower and only a small minority were unwed (maximum was 10%). Ward C only admitted married women from poor housing conditions and these were on average younger and more often giving birth for the first time than the married women who gave birth at home.

Table 1 *Main socio-demographic characteristics of the mothers – all deliveries in the 1927 midwife and hospital samples*

					Birthing mothers			
Midwife/ward	**Deliveries (n)**	**Live Births (n)**	**Born without life (n)**	**District**	**% < 26 years**	**Mean age (years)**	**% Unwed**	**% First birth**
Astrid Brücker	96	95	1	Amager	31.3	**28.3**	**9.4**	29.2
Emilie B. Isaksen	63	62	1	Amager	20.6	30.0	3.2	**57.1**
Dagmar M. J. Løsecke	121	119	2	Amager	29.8	29.5	2.5	28.1
Julie C. Nielsen	138	138	0	Inner City	**42.3**	**27.7**	6.5	23.2
Betty Nielsen	154	152	2	Nørrebro	**37.9**	**27.9**	**9.7**	**57.8**
Clara B. Olesen	60	58	2	Nørrebro	**41.7**	28.7	**10.0**	**41.7**
Bertha Petersen	74	71	3	Nørrebro	**43.1**	**27.4**	**9.5**	33.8
Juliane M. Peitersen	121	119	2	Vanløse	24.8	29.3	3.3	24.0
Helga Emilie Sørensen	51	50	1	Vesterbro	25.5	29.0	3.9	31.4
Ane C. Petersen	86	85	1	Vesterbro	32.6	28.7	4.7	16.3
Caroline Chr. Boserup	53	51	2	Østerbro	17.0	29.9	1.9	37.7
Petra Holt	82	81	1	Østerbro	22.0	28.8	6.1	30.5
Petra Sørensen	52	52	0	Østerbro	11.5	30.6	1.9	**44.2**
Royal Hospital A	162	150	12	Hospital	**65.0**	**24.3**	**64.2**	**62.3**
Royal Hospital B	163	152	11	Hospital	**70.4**	**24.1**	**70.6**	**66.9**
Royal Hospital C	73	73	0	Hospital	**68.5**	**24.9**	0.0	**49.3**

Note: The bold numbers are those referred to in the text as signifiers for specific social-demographic profiles of the midwives' clientele.

Unexpected, however, are the rather marked differences between the demographic profiles of the mothers who had chosen the individual midwives, even within our relatively small sample. Four midwives had more than a third of their clientele being less than 26 years of age (Bertha Petersen, Julie Nielsen, Clara Olesen and Betty Nilsen). Three of these, living in the Nørrebro district, also had the highest rate of unwed mothers, although none in the ranges of the hospital. Two of the Nørrebro midwifes (Nielsen and Olesen) also had a high rate of primiparous women. However, a high primiparous rate was also found, as with Isaksen at Amager and Sørensen at Østerbro, who had very low unwed rates and a rather high mean age for their clientele.

Nørrebro and the inner city districts had many small, older apartments and a high share of working class in the population. Østerbro was a more affluent part of the city with a higher share of bigger apartments. However, the districts in Table 1 are where midwives lived, and the expecting mothers were free to choose any midwife in the city. The midwife-birth-registers do include addresses of the women giving birth, so in the future this can be explored, but we have not done that in this pilot study.

However, the different profiles of the midwives' clientele do suggest that there were factors influencing the women's choice of midwife at the individual level. These may include availability, loyalty, cost, reputation and geographic proximity, as suggested by Reid (2012) and Curtis (2005). The selective nature of these midwife practices requires careful consideration when interpreting aggregated health outcomes from a small sample, as ours is. But it also suggests that the Danish midwife-birth-registers will be a tremendously rich source for studying factors influencing health at birth over time and in different settings, once the archives make them all accessible in transcribed formats for the whole of Denmark during the entire period 1861 to 1978.[1]

1 The midwife-birth-registers for 1926–1978 are under transcription and integration with other sources in the project *Historisk Medicinsk Fødselsregister* led by associate professor Jennifer Lyn Baker (Center for Clinical Research and Prevention, Copenhagen University Hospital — Bispebjerg and Frederiksberg) and chief advisor Jeppe Klok Due (Danish National Archives). The project was awarded by the Novo Nordisk Foundation in 2024.

7 BIRTH WEIGHT AND PREMATURITY — HOME BIRTHS AND HOSPITAL BIRTHS COMPARED

The average birth weight was higher in the live home birth sample (3,520 grams) than for the hospital wards A and B (3,160 grams). Ward C was in between these two (3,390 grams) (see Table 2). We round up the figures in the text so as not to indicate too high an accuracy in the original measurements.

The babies born in wards A and B, primarily to young, unwed women, many of whom were primiparous (see Table 1), were on average 360 grams lighter than those born at home, while babies born in ward C (to married women from poor housing conditions) were only 200 grams lighter.

Our home birth average weight is very close to the findings from Southern Sweden in the 1930s and 1940s, shown in a dataset designed to capture two full-coverage, locally-based populations. The birth weights for full-term singletons were 3,530 grams and 3,520 grams (Quaranta, 2022, Table 1). Schneider's (2017) dataset from Boston (1884–1900) found a slightly lower average in outpatient home births (3,480 grams). The average birth weight in our home birth sample is also very close to the average for singletons in contemporary Denmark which, in every year from 1997 to 2018, was in the range of 3,500 to 3,550 grams (Danmarks Statistik, 2025). This may support Schneider's suggestion of relative stability in average birth weights over the last century (Schneider, 2017).

However, the same average birth weight may mask very different combinations of full-term birth weights and preterm birth weights. Low Birth Weight (LBW; weights of less than 2,500 grams) occurs both in preterm and full-term births. When a full-term infant is born with low birth weight it is called "small for date" and is often caused by factors other than preterm birth. For infants born preterm, their birth weight is of course lower, the lower the gestational age of the fetus, but also preterm fetuses can be small for their gestational age. It is likely that, in historical populations, the relative impact of prematurity and full-term infants born "small for date" may vary over time and place. In this paper we have not explored all the possible combinations in our sample, but below we share some preliminary observations.

Quaranta (2022, p. 609) included only full-term, live born singletons in their two populations. They found a LBW of 1.1 % and 1.6%, so actually they have delineated small for date rates for a historical population. In Denmark in the period 1953–1955, LBW was 4.8% for boys and 5.4% for girls (among live births, including twins and preterm births) (Spædbørnsdødelighed og fødselsvægt, 1960, p. 206). During the period 1997 to 2018, the Danish LBW was between 3.4% and 3.5% for live singletons inclusive of preterm births (Danmarks Statistik, 2025).

Our Copenhagen home birth sample had a LBW rate of 2.9% and a preterm rate of 7% (see Table 2). Substantial differences emerge when we compare LBW rates in home and hospital births. The LBW rate at the wards A and B was three time as high as in the home birth sample (12.2% versus 2.9%) (see Table 2).

Table 2 *Birth weight and births before term for live births Copenhagen — the 1927-sample (n = 1508) distributed after place of birth (home/Royal Maternity Hospital)*

	Home births	Ward A	Ward B	Ward C	All
Births live (n)	1,133	150	152	73	1,508
Birth weight					
Information missing (n)	6	9	7	0	22
Low birth weight* (n)	33	19	18	5	75
Low birth weight rate**	2.9	12.7	11.8	6.8	5.0
Average weight (grams)	3,516	3,139	3,184	3,318	3,438
Minimum weight (grams)	900	1,100	1,400	1,800	900
Maximum weight (grams)	6,000	4,700	4,900	4,700	6,000
Births before term					
Information missing	16	8	5	2	31
Before term (n)	79	50	49	8	186
Before term birth rate	7.0	33.3	32.2	11.0	12.3

*Notes: *Low birth weight < 2500 grams. ** Rate = number per 100 live births in the column.*

These differences in LBW are very likely primarily explained by differences in prematurity. However, we cannot calculate premature birth rates precisely as they are defined today, as the midwife-birth-registers from 1927 do not provide the precise information as to whether an infant is "born before the end of the 37th week of gestation", as it is defined today. However, the Hospital's wards recorded some information about how many weeks before term a birth occurred: most of their premature births were estimated to have been born two to four weeks before term. That means that the hospital counted an infant as preterm until the (estimated) end of the 38th week of gestation.

If we keep this definition in mind, it makes sense to calculate the percentage of records marked as "estimated premature". The proportion of estimated premature live births was 33% in wards A and B, more than four times higher than the 7% observed in the home births. These huge preterm rates heavily affect the average birth weight for all births on wards A and B. The differences in average birth weight between hospital and home deliveries for full-term births only, narrows to less than 100 grams (A and B — primarily young and unwed mothers) and 180 grams (C young, married women living in poor housing).

Our hospital sample is very small. However, we can control the representivity of the sample data from ward A, because this ward (and only this ward) reported in the Annual Report of the Hospital for 1927, that 9% of all their births (including stillbirths and miscarriages) had a birth weight of under 2,000 grams. We have done exactly the same calculations on our ward A sample and get a reassuring 9.3% births with a birth weight of less than 2,000 grams. Ward A's report on the preterm rate is also in line with our sample: the annual report shows that 1,001 out of 3,001 births occurred before term. This aligns perfectly with the 33.3% pre term rate in our ward A sample (see Table 2) (Beretning om Rigshospitalet, 1928).

The large differences between home and hospital births are most likely explained by the selective nature of hospital delivery. The demographic characteristic of the mothers of our hospital sample wards A and B (see Table 1) are known to be associated with high risk of LBW and prematurity (Morgen et al., 2017; Mortensen et al., 2009; Poulsen et al., 2015; Schneider, 2017).

8 SOME THOUGHTS ABOUT MEASUREMENT UNCERTAINTY AND MEASUREMENT ERRORS IN HISTORICAL BIRTH WEIGHT INFORMATION

How accurate are historical birth weight measurements? That is a question which needs to be addressed, but is impossible to adequately answer in this paper. The following are some initial thoughts about the scales used and the degree of heterogeneity of birth weights reported in our home birth sample.

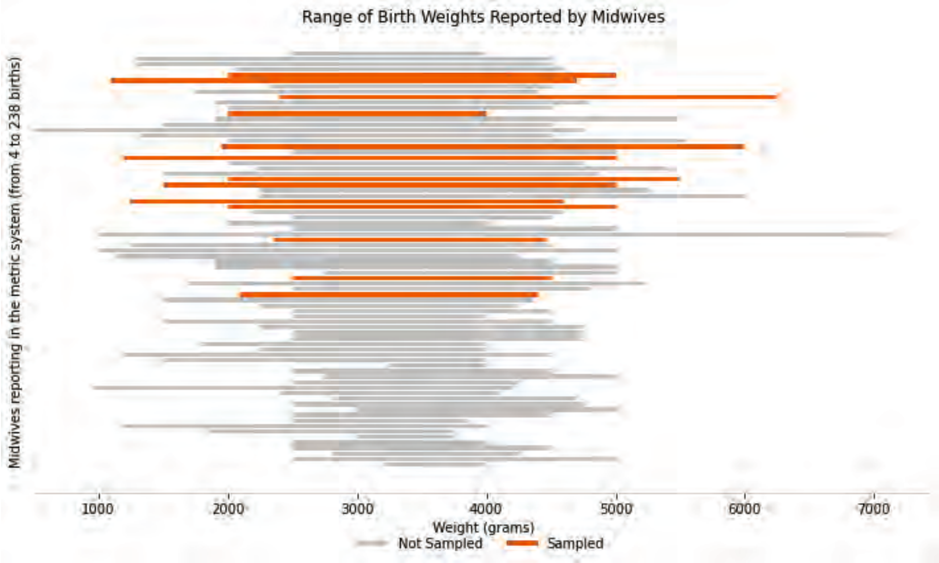
8.1 THE SCALES

The head midwife of the School of Midwifery and of ward B in 1927, Johanne Rødtness, wrote in her professional memoirs (Nellemose, 1935, p. 155), that the scale preferred by the practicing midwives was the spring scale (*fjedervægt*), because it was small and easy to carry. She writes that the chief obstetrician of ward B, head of the School of Midwifery, Prof. Erik Hauch, demonstrated the inaccuracy of spring scales by weighing a number of infants both with a spring scale used by a practising midwife for years and a *bismervægt* (Scandinavian steelyard balance). The spring scale showed about 500 grams more than the steelyard balance. She also explained that the weight and length of a newborn was used for assessing whether the infant was full-term or not.

8.2 THE REGISTRATION PRACTICES OF THE MIDWIVES

What can we deduce from the registered weight measurements themselves? First we compared our home births sample to all the midwives operating in the city, according to the maximum and minimum birth weights reported by the 77 midwives that both measured in either grams or kilos and also weighed preterm births (see Figure 1).

Figure 1 *Maximum and minimum reported birth weight (preterm infants included) for all midwives-birth-registers with records done in kilos or grams for the whole year 1927 in Copenhagen (77 birth registers)*



Our sample is reasonable within the maximum and minimum weight pattern of all midwives in Copenhagen in 1927. At least our sample is not so strikingly different. Thus we have no reason to think that the reporting behavior of our sample is a complete outlier. The sample seems just to be part of the overall heterogeneity (see Figure 1).

However some midwives, both in our sample and among the not sampled, have registered maximum weights of more than 5,000 grams. That is not impossible, but it does suggest that some of the scales these midwives used may have had measuring errors.

We then examined differences in how midwives recorded birth weight, within our home births sample (see Table 3). The last columns shows the share of a midwife's births, which were in the rather high range of 4,000 to 4,499 grams. Here we see that Ane Petersen is an outlier with 39.5 % heavy births, while three other midwives had more than 25% heavy births. That suggests that these four may have had scales showing values that were too high.

When examining the number of unique values registered as birth weight by the individual midwives (see Table 3), we see a large spread from very few unique weights up to 50. Løsecke recorded weights in only six distinct categories for her 119 births and Ane Petersen, who attended 86 births, only used nine. The average number of grams between any two measurements for these two midwives is around 500. This may indicate that they did not even use a scale, but just sized the infants by hand. That suggests that Ane Petersen may have generally estimated weight higher instead of lower.

At the other end of the precision scale are three midwives with a high number of unique measurements and smaller differences in average distance in grams between the unique measurements. Isaksen attended 63 births and recorded 45 unique weights, Julie Nielsen had the second highest number of unique weighs (50 for her 140 births) and Boserup had 30 measurements for 52 births. Between these two clearly distinguishable groups the rest of the midwives measured in grams but with average distances between measurements of around 100–200 grams.

These findings rely on a small number of cases and only for a year. However, they do underline the fact that there were substantial differences in how midwives measured and recorded the information, which needs to be taken into consideration during analysis, as these differences very likely have effects on the results, when the birth weight information is used.

Table 3 *Number of births (stillbirth and miscarriages inclusive) with birth weight registered, distributed after midwife — the 1927 midwife sample*

Midwife	Number of births weighted	Number of births not weighted	Measure	Unique values measured (n)	Average measurement distance	Mean grams	Min grams	Max grams	% Range 3000–3499	% Range 3500–3999	% Range 4000–4499
Dagmar M. J. Løsecke	119	2	kilos	6	500,0	3,399	2,000	4,500	42.0	26.9	25.2
Ane C. Petersen	86	0	kilos	9	437,5	3,747	2,000	5,500	19.8	17.4	39.5
Bertha Petersen	71	3	grams	10	333,3	3,596	2,000	5,000	15.5	32.4	26.8
Clara B. Olesen	59	1	kilos	14	189,7	3,402	2,200	4,666	28.8	25.4	22.0
Betty Nielsen	152	2	kilos	15	178,6	3,448	2,000	4,500	22.4	47.4	15.8
Petra Holt	82	0	grams	22	166,7	3,532	1,500	5,000	19.5	37.8	22.0
Juliane M. Peitersen	120	1	grams	26	154,0	3,660	2,400	6,250	26.7	32.5	26.7
Helga E. Sørensen	51	0	grams	27	136,5	3,436	1,950	5,500	29.4	35.3	11.8
Petra Sørensen	52	0	grams	27	196,2	3,453	900	6,000	21.2	38.5	13.5
Caroline Chr. Boserup	51	2	grams	30	79,3	3,549	2,100	4,400	19.6	51.0	17.6
Astrid Brücker	96	0	grams	32	122,6	3,511	1,200	5,000	19.8	31.2	17.7
Emilie B. Isaksen	63	0	grams	45	71,4	3,493	2,360	5,500	39.7	25.4	14.3
Julie C. Nielsen	138	0	grams	50	73,5	3,441	1,100	4,700	29.0	39.1	17.4

9 CONCLUSION

There is relatively little knowledge about the evolution of perinatal health. For birth weight and prematurity during the 19th and the first half of the 20th centuries, most of what we know is based on hospital births, due to the scarcity of historical records of home births. This article has contributed to the literature by providing a pilot study on midwife-birth-registers from Copenhagen for the year 1927, comparing home births with births at the Royal Maternity Hospital.

We found significant differences in birth weights and prematurity between home and hospital births, attributable to the selective nature of the hospital admissions. Hospital births at ward A and B showed an average birth weight of 360 grams less than in home births (3,160 grams versus 3,530 grams for home births). The Low Birth Weight Rate (LBR < 2,500 grams) at the wards A and B was three times as high as in the home birth sample (12.2% versus 2.9%) and the preterm rate was more than four times higher (33% versus 7%).

We also found huge differences in the socio-demographic characteristics between the mothers giving birth at home and at the Hospital (see Table 1). These differences were caused by the selective admittance to the Royal Maternity Hospital admitting mostly unwed, young, primiparous women to wards A and B, and very poor married women to ward C, while the home births included mostly married women throughout the fertile ages. These social demographic differences go a long way to explain the differences in birth output. Additionally, the existence of these differences shows that the Danish collection of midwife-home-birth- registers from 1861 to 1978 is a tremendously rich historical source well worth exploring further to understand historical health at birth for the majority of births taking place at home until the mid-20th century.

However, we have also found indications of substantial variations in the practices of birth weight measurement and reporting standards by the midwives, which complicates the use of historical birth weight information. This emphasizes the need for careful analysis before interpreting differentials in historical birth weight data registered in different settings.

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The Weanling's Dilemma

Breastfeeding and Socioeconomic Status in 19th-Century Venice

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ABSTRACT

Weaning is one of the most crucial steps in an infant's life. This study investigates how families of different socioeconomic conditions coped with weaning and its consequences for infant mortality, using individual-level longitudinal data drawn from the Venetian population register. As in previous studies, breastfeeding patterns are inferred from infant mortality features. However, the approach adopted differs in part from similar research, combining Cox proportional hazards and Aalen additive regression models. Aalen models allow coefficients to vary over time, showing discontinuities that can be interpreted as signs of the start of weaning. The Venetian case reveals a pronounced social gradient: the poorest mothers breastfed for no more than one month, whereas others continued for 6–8 months or longer. This disparity contributed substantially to socioeconomic inequalities in infant mortality.

Keywords: Breastfeeding, Infant mortality, Aalen's additive regression model, Cox proportional hazards regression model, 19th-Century Venice, Infant mortality seasonality

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1 INTRODUCTION

The "weanling's dilemma" refers to the choice to which infants are subjected, between loss of immunity and exposure to potentially contaminated food at weaning on the one hand, and malnutrition due to prolonged breastfeeding on the other. The term was coined by Gordon and colleagues (1963) to define the best feeding practices for children in poor countries, and was thereafter widely adopted in scholarly literature (Kendall et al., 2021). This study focuses on the weanling's dilemma in mid-19th-century Venice. In particular, it investigates the duration of exclusive breastfeeding and how it differed between social classes.

Contemporary guidelines from the World Health Organization (WHO, 2023) recommend six months of exclusive breastfeeding, followed by 18 months of complementary feeding. Nevertheless, fewer than half of infants under six months are currently breastfed. Breastfeeding rates are lowest in high-income countries (UNICEF & WHO, 2023), notwithstanding evidence linking formula feeding to a 49% increase in post-perinatal mortality (Ware et al., 2023) and to elevated risks of metabolic disorders across the life course (Munblit et al., 2020). However, it is in low- and middle-income countries, where 98% of infant deaths occur, that the consequences of suboptimal infant feeding are more severe (North et al., 2022). According to Victora and colleagues (2016), universal breastfeeding might annually avert 823,000 under-five deaths.

In the past, artificial feeding was even more harmful than it is today, as evidence from European countries in the late 19th and early 20th centuries suggests. According to Vögele and colleagues (2013), in Germany the death rate of bottle-fed infants was seven times higher than that of breastfed infants. In Paris, during summer time it was ten times higher (Rollet, 1997). In Düsseldorf, the mortality rate of bottle-fed infants from well-off families was twice as high as that of breastfed infants from poor families (Vögele, 2010), while among Bavarian working-class families breastfeeding reduced infant mortality by 75% (Brown & Guinnane, 2018). In Derbyshire, the post-neonatal mortality of hand-fed infants was 50% higher than that of breastfed infants (Reid, 2002).

Unfortunately, information on the rate and duration of breastfeeding in the past is rare, and only covers the early 20th century. Consequently, scholars of breastfeeding in earlier periods have to use indirect evidence to make inferences on feeding practices. This study aims to contribute to both these issues. From a substantive point of view, I ask whether in 19th-century Venice insufficient breastfeeding was pre-eminently a social problem, as contemporaries strongly denounced. From a methodological point of view, I suggest an approach that allows making inferences on the age at weaning. I use summer mortality as a litmus test of feeding practice, and show that summer hazards varied significantly by both age and social status. I argue that such variations are suggestive of the end of exclusive breastfeeding.

The study is organized as follows. In Section 2, I briefly review the historical literature on breastfeeding, focusing on the available sources and the methods used by scholars to infer information on feeding practices. Section 3 presents the historical context and the source material used for my analysis. In Section 4, I carry out a survival analysis of infant and child mortality, using Cox proportional hazards and Aalen's regression models. Results are discussed in Section 5. Finally, in Section 6 I draw some conclusions, stressing the relevance of early nutrition in mortality regimes, past and present.

2 HISTORY OF BREASTFEEDING: SOURCES AND METHODS

The literature on the history of infant feeding is extremely rich, spanning from the prehistoric past to classical antiquity and early modern times (see e.g. Cocozza et al., 2025; Fildes, 1986; Fulminante, 2015; Matthews Grieco, 1991; Obladen, 2014; Rebay-Salisbury, 2017; Wickes, 1953). Scholars reconstructed the evolutionary, biological, historical, cultural, and socioeconomic contexts of feeding habits, highlighting the stunning variety of the methods employed, the differences between geographical areas, cultures, and social conditions, and their evolution over time. However, these studies mostly rely on qualitative evidence. A notable exception is bioarchaeologists and osteoarchaeologists, who obtain information on breastfeeding and weaning behaviour from the isotope analysis of skeletal remains (Jay, 2013). Interestingly, this approach was successfully applied also to early-modern populations (see e.g. Herring et al., 1998; Moggi-Cecchi et al., 1994; Newman & Goland, 2017; Nitsch et al., 2011).

For instance, Waters-Rist and colleagues (2022) found that, against expectations, in a protestant, dairy farming Dutch community in the 19th-century breastfeeding was either absent or very short.

The earliest statistics on breastfeeding were published in the late 19th century, when governments and social reformers, driven by concerns over high infant mortality, promoted surveys on infant rearing conditions. Aggregate statistics at the district level were produced for Germany (Kintner, 1985), England (Fildes, 1998), Sweden (Brändström et al., 2002), Iceland (Guttormsson & Garðarsdóttir, 2002), and France (Rollet, 1990). Such materials have been widely studied (see e.g. Kintner, 1988a, 1988b; Knodel & van de Walle, 1967; Woods et al., 1988, 1989). However, the analysis of aggregate data suffers from several limitations, and poses the risk of ecological fallacy (Shih et al., 2023). On the other hand, historical sources with information on the rate and duration of breastfeeding at the micro level, like those used by Woodbury (1925) for his study in eight American cities, Fildes (1992) for London, Reid (2002, 2017) for Derbyshire, and Guttormsson and Garðarsdóttir (2002) for Iceland, are unique of their kind, and all concern the early 20th century.

In the absence of direct evidence on breastfeeding, researchers carried out demographic analyses from which to infer the infant feeding habits prevailing in a population or social group. Given the effect of lactation on postpartum amenorrhea, some studies used birth intervals as proxies of breastfeeding habits (Davenport, 2019; Jaadla et al., 2020; Janssens & Pelzer, 2014; Newton, 2011; Pebley et al., 1991). Other scholars focused on infant mortality patterns that could suggest the absence or the inadequacy of breastfeeding. For instance, Knodel and Kintner (1977) argued that deviations of the age pattern of infant mortality from the standard linear biometric model reveal the deficiency or early termination of breastfeeding. Similarly, summer mortality peaks and the burden of deaths from diarrhoea and gastrointestinal diseases have been considered as suggestive of poor or unfit feeding practice (see e.g. Cheney, 1984; Huck, 1997; Mühlichen & Doblhammer, 2025; Murkens et al., 2023; Thornton & Olson, 2011; van den Boomen & Ekamper, 2015; van Poppel et al., 2002, 2018; Walhout, 2010).

Both the "birth-interval approach" and the "mortality approach" allow comparisons between groups or populations. Differences in breastfeeding related to socioeconomic status and religious affiliation have been given special attention.

Contemporary reformers typically addressed the poorest strata of population, attributing low rates of breastfeeding to ignorance and material deprivation and prompting interventions like maternal education and welfare programs (Brown & Guinnane, 2018; Fildes, 1992, 1998; Kintner, 1985; Vögele et al., 2013). However, the social gradient in breastfeeding was far from linear. As a rule, mothers in the upper class were less likely to breastfeed. In London, the attitude of the elites towards maternal breastfeeding began to change in the late 18th century, significantly contributing to child survival (Davenport, 2019; Trumbach, 2013). Breastfeeding was instead more common among the families of day labourers, whose newborns had surprisingly low mortality rates (Jaadla et al., 2020). Still in the early 20th century, breastfeeding was more widespread in the poorest boroughs in London, though it was early terminated or integrated with solid food (Fildes, 1992).

The impact of culture, namely religious culture, on infant feeding is a particularly controversial topic. There is a widespread agreement that breastfeeding, infant care, and infant wellbeing largely depended on religious affiliation: Jews always enjoyed the most favourable conditions, far outpacing Protestants and Catholics (see e.g. Connor, 2017; Gráda, 2006; Preston et al., 1994; Thornton & Olson, 2011; van Poppel et al., 2002). Thorvaldsen (2008) suggested that Europe has been long divided into two broad areas: in northern Protestant Europe, breastfeeding was widely practiced, while in southern Catholic countries recourse to wet-nurses and artificial feeding was common. However, when moving from such a broad picture to more detailed analyses, things become problematic. For instance, in a forthcoming paper I show that among Venetian Jews breastfeeding was neither as widespread nor as protracted as one would expect (Derosas, forthcoming).

Dutch historians lively discussed the role of Catholicism in the upsurge of infant mortality that afflicted some regions of the Netherlands in the late 19th century. While Van Poppel (1992) blamed the reluctance towards breastfeeding induced by the conservative involution of Dutch Catholicism, Walhout (2010), Janssens and Pelzer (2014), and Van den Boomen and Ekamper (2015) argued instead that the differences in breastfeeding between Catholics and Protestants were much smaller than supposed, and that locality influenced individual behaviour more than religion.

3 HISTORICAL CONTEXT AND SOURCE MATERIAL

Around mid-19th century, Venice was a large but impoverished city. After the end of the aristocratic regime in 1797, the economy had collapsed and the population shrunk by one third, from around 180,000 to less than 120,000 inhabitants. The 1850s were one of the worst periods in Venetian history. The city had not yet recovered from the defeat in the 1848–1849 revolution, a harsh siege by the Austrian army, and a devastating cholera epidemic. The agrarian crisis of 1854–1855 tripled the cost of food, while the presences in the poor workhouse soared from 114,000 to 315,000. The registered poor entitled to receive public support were 35,000, one fourth of the total population, though only 3,000 received some daily relief (Derosas & Munno, 2022). American writer W.D. Howells (1878) described Venice as "a gloomy and dejected city". It was also one of the deadliest European cities to live in: crude death rate was around 30 per 1,000, life expectancy at birth was around 33 years, and infant mortality rate around 240 per 1,000 (Derosas, forthcoming).

In 1876, Cesare Musatti (Venice, 1846–1930), a Jewish doctor and one of the first paediatricians of his time, published an interesting book of instructions and recommendations on child rearing. He claimed that a few simple attentions could avoid the countless infant deaths that afflicted Venetian families. Breastfeeding was by far the most important remedy to adopt. Musatti urged mothers to breastfeed for at least six months, but few followed his recommendations. Many were too poor to afford breastfeeding, and often abandoned their children out of despair of being unable to raise them. Others turned to artificial feeding after two or three months. Infants were fed cow, goat or donkey milk. Solid food included paps of sago, tapioca, arrowroot, breadcrumbs, wheat flour, or semolina, which mothers often pre-chewed and salivated to make them digestible. Musatti was also aware that his middle-class readers preferred resorting to wet-nurses, and devoted a large part of the chapter on infant nutrition to advices about properly choosing and dealing with them (Musatti, 1876).

This study tests hypotheses derived from Musatti's observations. The main source is the city population register, established in 1850 and updated until 1869. Population registers are a sort of dynamic census, in which information on individuals and families is systematically updated as events occur, for instance when a new entry or exit changes the composition of a household. To improve the data quality, the information from the population register has been verified and integrated with data from the parish registers, the municipal registers of deaths, and the city census of 1869. Furthermore, local newspapers provided daily data on wheat price and outdoor temperature.

The dataset includes four parishes representing Venice's socioeconomic spectrum. San Luca was among the richest parishes of the city centre, mainly inhabited by members of the elites. San Geremia and Santa Eufemia were working-class parishes, with many employed in glass factories, at the railway station, or in hemp and leather workshops. Finally, San Raffaele Arcangelo was the poorest parish, inhabited by fishermen, boatmen, porters, and day labourers. The quality of dwellings and overall hygienic conditions were rather poor. The majority of houses had no running water. Poor sewage arrangements were common and most houses in the poorest areas had no toilet facilities.

According to the 1869 census, the four parishes had 15,825 inhabitants. The dataset includes 26,894 individuals, observed for spells of different length between 1850 and 1869. The total person-years adds up to about 272,000, with 8,802 births and 7,102 deaths. I categorised socioeconomic status (SES) according to the social position and the presumptive level and continuity of income of the household head. SES includes day labourers (fishermen, porters, boatmen); wage workers; artisans and shopkeepers; members of the middle and upper class (employees, civil servants, professionals, bankers).

4 SURVIVAL ANALYSIS

Table 1 presents infant and child mortality rates by SES, calculated from population register data. The results reveal a consistent social gradient across all age groups, with one notable exception: neonatal mortality (0–28 days), where elite rate nearly equalled that of day labourers (137 vs. 124 per 1,000).

Table 1 *Infant and child mortality rates by SES. Venice 1850–1869*

SES	Neonatal (0–28)	Post-neonatal (29–365)	Infant (0–1)	Child (1–4)
Day labourers	137	201	275	96
Wage workers	110	157	226	72
Artisans, shopkeepers	97	140	202	70
Middle, upper class	124	100	193	39
Unknown	67	213	218	73
Total	117	168	238	79

Source: My elaboration on population register data.

Table 2 presents the results of three multilevel Cox proportional hazards models, analysing mortality across neonatal (days 1–28), post-neonatal (days 29–365), and early childhood (ages 1–4 years) periods. To avoid potential misclassification of stillbirths (as parents occasionally baptized stillborn infants), the analysis excludes perinatal mortality, commencing observation from the second day of life.

The primary exposure variable is socioeconomic status (reference category: day labourers), with models adjusted for:

1. Sex (reference: male).
2. Seasonal weather patterns, based on daily distributions of outdoor temperature (reference: mild, mean 12°C, min -2, max 24):
   - Hot season: June to mid–September (mean daily temperature 19°C, min 12, max 39);
   - Cold season: December to March (mean daily temperature 2°C, min -10, max 12).
3. Economic stress periods (reference: normal/low prices):
   - Defined as days when wheat price reached the top quartile of the daily distribution.
4. Cholera outbreaks (reference: non-epidemic periods):
   - Four major epidemics occurring August–October 1854, May–September 1855, September–October 1866, and July–October 1867.

The models incorporate household-level random effects to account for intra-cluster correlations in mortality outcomes.

The analysis reveals striking socioeconomic disparities in infant and child mortality, though with an important exception during the neonatal period. From infancy through early childhood, children of day labourers faced mortality risks approximately twice as high as those of elite families, with artisans' and skilled workers' children experiencing intermediate risks (20–25% lower than day labourers but still significantly higher than the upper classes). This social gradient, however, breaks down in the first month of life, where neonatal mortality shows no significant class differences — a finding that aligns with the patterns visible in Table 1. The elite's neonatal rates nearly matched those of the poorest families.

Surprisingly, cholera outbreaks did not have a significant influence on infant and child survival.

Economic crises showed delayed effects. While food price shocks left infant mortality unaffected, they increased childhood mortality risks by 29%, possibly through gradual malnutrition or compounded health stresses. It should be noticed, however, that the estimate violates the proportionality assumption.

Table 2 *Cox regressions of infant and child mortality. Venice 1850–1869 (Fixed coefficients only)*

	Model 1: Neonatal			Model 2: Post-neonatal			Model 3: Child		
	Obs.	Events	Schoenfeld residuals	Obs.	Events	Schoenfeld residuals	Obs.	Events	Schoenfeld residuals
	11,779	688		43,133	1,128		125,049	1,696	
Fixed coefficients	exp(coef)	p	p	exp(coef)	p	p	exp(coef)	p	p
Sex: female (ref: male)	0.86	0.054	0.342	0.87	0.022	0.078	0.84	0.001	0.862
Head's SES (ref: day-labourer)			0.588			0.123			0.105
Wage worker	0.79	0.023		0.82	0.010		0.80	0.000	
Artisan, shopkeeper	0.72	0.010		0.75	0.002		0.76	0.000	
Middle, upper class	0.89	0.650		0.43	0.002		0.57	0.002	
Unknown	0.66	0.320		0.68	0.210		1.12	0.580	
Weather (ref: mild)			0.049			0.000			0.000
Cold	1.83	0.000		1.15	0.054		0.96	0.530	
Hot	0.40	0.000		1.39	0.000		1.35	0.000	
Food price: high (ref: low)	0.89	0.210	0.864	1.05	0.460	0.834	1.29	0.000	0.000
Cholera epidemic (ref: absent)	0.88	0.620	0.063	0.93	0.640	0.103	1.22	0.059	0.882

Source: As in Table 1.

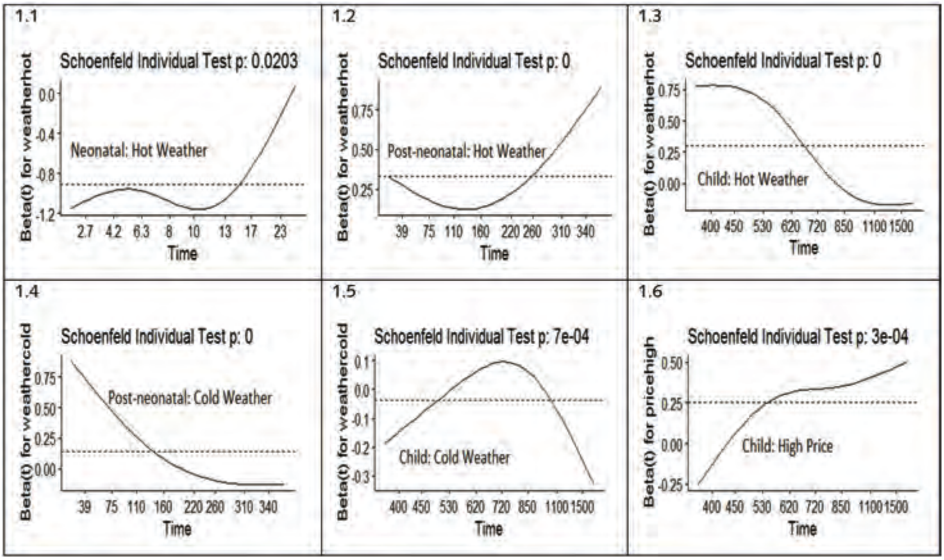
The same is true for the effect of weather in all of the three models estimated. On the one hand, they show that the impact of outdoor temperature shifted dramatically over time. Cold weather proved particularly deadly for newborns, nearly doubling their mortality risk compared to mild periods. Heat, by contrast, offered strong protection in the neonatal phase — likely by reducing respiratory infections — only to reverse into a significant hazard as infants grew older, probably reflecting weaning-related exposure to summer diarrhoeal diseases. On the other hand, the Schoenfeld's tests warn that there is some further time-dependency that needs to be addressed. In other words, these risk factors do not operate uniformly over time but depend on children's developmental stages in ways that demand more nuanced modelling.

Figure 1 illustrates these temporal patterns through smoothed plots of scaled Schoenfeld residuals for the variables violating the proportional hazards assumptions. The dotted lines trace the time-invariant coefficients estimated by the Cox regressions, revealing where actual risks diverge from model assumptions.

The plots in Figure 1 demonstrate clear violations of the proportional hazards assumption, which would require $\beta(t)$ to approximate a horizontal line. Instead, we observe significant temporal variation in all panels. P-values indicate that the correlation between the Schoenfeld residuals and time is statistically significant. For instance, the time-invariant coefficient estimated for the newborns in the hot season is -0.91 (model 1: exp. coeff. 0.40, p-value < 0.001). Panel 1.1 shows that in the first two weeks of life the protective effect of heat — as compared to mild weather — is slightly larger than estimated, and progressively vanishes afterwards. In the post-neonatal period (panel 1.2) the hot season appears moderately dangerous until around the fifth month, and starts a noticeable upward trend thereafter, turning increasingly dangerous. It is only around the third birthday that heat stops being harmful (panel 1.3). The impact of cold follows an inverse trend, with high risks at birth (model 1 above), declining until they disappear around the sixth month (panels 1.4-1.5). Finally, panel 1.6 suggests that children become more sensitive to economic crises as they grow up.

When the proportional hazards assumption fails — as in our case — researchers typically employ several solutions: partitioning the observation period into distinct phases, stratifying the analysis, or incorporating time-dependent coefficients (Therneau & Grambsch, 2000, pp. 142–147). While these approaches can yield statistically unbiased estimates, our focus lies elsewhere. Rather than "fixing" the time-varying effects, we aim to understand what drives them — to interpret these violations of proportionality not as statistical nuisances, but as meaningful bio-social phenomena manifesting across a child's development.

Figure 1 *Schoenfeld residuals from Cox regressions*



Source: My elaboration from Cox regressions in Table 2.

All of these temporal patterns point to nutrition as a critical underlying factor. The counterintuitive seasonal effects — where heat becomes dangerous while cold proves protective after infancy — likely mirror the epidemiology of diarrhoeal diseases, which thrive in summer months when weaned infants face greater exposure to contaminated foods. Meanwhile, the delayed but growing impact of food prices reflects nutritional stress accumulating through early childhood. This raises a pivotal question: did socioeconomic groups show distinct seasonal mortality patterns that might reveal variations in infant feeding practices?

Aalen's additive models offer distinct advantages for our analysis by permitting time-varying coefficients. They estimate how departures from reference conditions (e.g. cold vs. mild weather) contribute additively to mortality hazards through linear hazard modelling (Aalen & Scheike, 2005) — crucial when risks evolve across developmental stages.

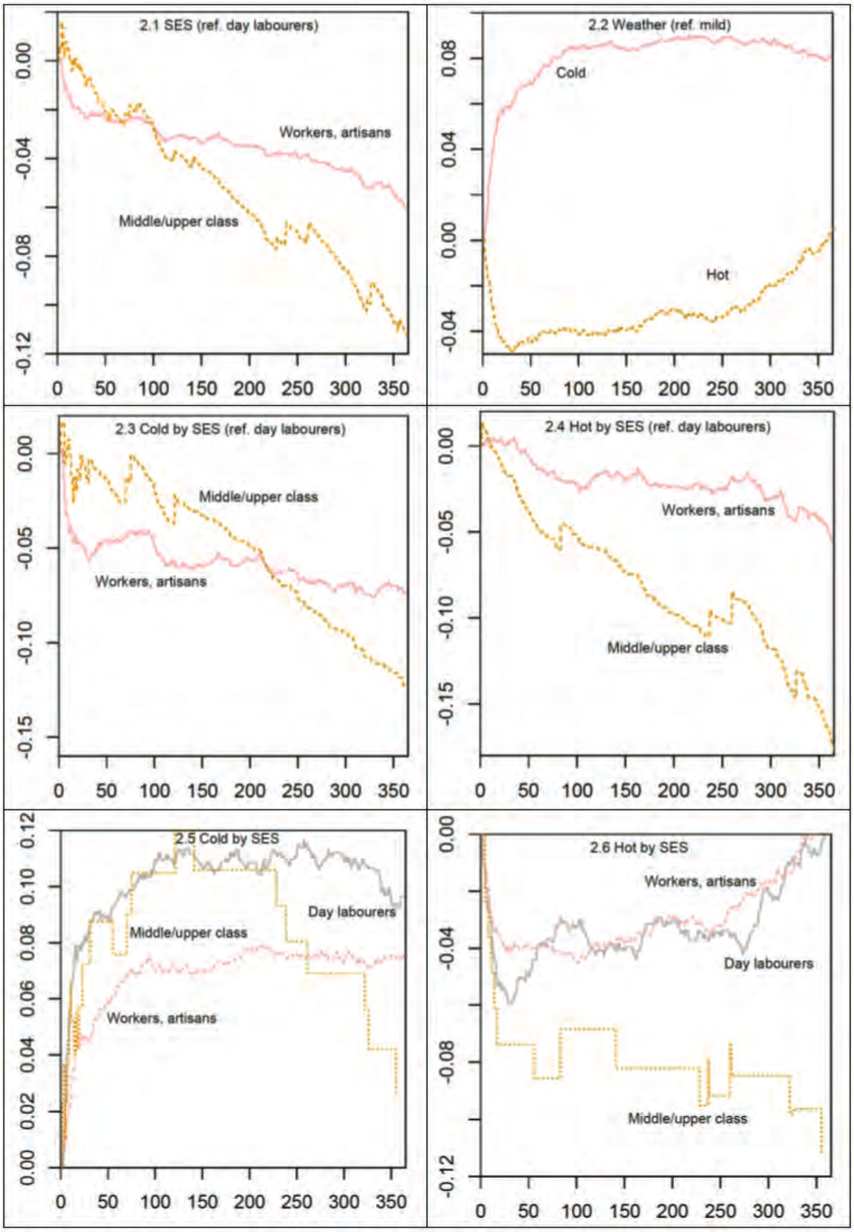
Several simplified models were fitted, incorporating only socioeconomic status and weather as covariates. Given the similar mortality patterns observed across wage workers, artisans and shopkeepers, these groups were consolidated into a single category. Selected results from Aalen's regressions are presented in Figure 2. The plots illustrate the additive contributions of covariate deviations from reference values. To enhance clarity of the central patterns, baseline hazards and confidence intervals were omitted from the visual representation.

The panels collectively reveal consistent mortality patterns through complementary analytical approaches. Panels 2.1–2.2 present global estimates using the complete dataset with both weather and SES as covariates. Subsequent panels employ stratified analyses: panels 2.3–2.4 examine SES effects within weather categories (reference: day labourers), while panels 2.5–2.6 assess weather's effects within SES groups (reference: mild weather).

Panel 2.1 contrasts the cumulative estimated coefficients by SES. It confirms the social gradient already shown in Cox models 1 and 2. This also includes the elite's odd trend: in the first three months of life, their relative advantage over the day labourers is lower than that enjoyed by workers and artisans. In the very first days, they fare even worse than the day labourers themselves. In late infancy, however, the gap in their favour increasingly widens.

Panel 2.2 confirms the specular trends of the effects of hot and cold weather on survival. The hazards of the cold season rocket steeply in the first three months and level off henceforth. Hot temperatures exert instead a strong protective effect in the first month, but become increasingly harmful afterwards.

Figure 2 *Aalen's cumulative hazards plots*



Source: My elaboration from Aalen's regressions.

Panels 2.3 and 2.4 show how the impact of weather varies by SES. Both with cold and hot, the elite and the working class enjoy a clear advantage over the day labourers. However, while cold makes no significant difference between the elite and the working class, in the hot period the advantage of the former is large and consistent.

Interpreting panels 2.5 and 2.6 is less straightforward. The plots compare the relative impact of cold and hot weather for each social group, combining the estimates of three distinct models. For instance, the curve of the day labourers in panel 2.5 represents the cumulative hazards run by day labourers in cold weather, relative to the hazards run by day labourers in mild weather.

Interestingly, cold is relatively as dangerous for the wealthy as it is for the poor, at least until seven months of age. Afterwards, the harmful effect of cold persists for the poor, and almost disappears for the rich. The hazards of cold for workers and artisans grow until the third month of life and then level off.

Panel 2.6 confirms that the trends outlined in panels 2.2 and 2.4 significantly differ by SES. The day labourers follow closely the global trend of panel 2.2, with hot weather turning abruptly from protective to harmful after the first month of life. The small number of events makes it difficult to detect a smooth trend for the wealthy. Nevertheless, the protective effect of hot weather persists throughout infancy, and no evidence of a trend inversion can be detected. In the case of workers and artisans, an upward trend seems instead to start around the eighth month of life.

5 DISCUSSION

These results paint a compelling picture of infant and child mortality in Venice. First, both SES and weather conditions exerted a strong influence on infant and child mortality. Second, social groups exhibited marked disparities in their ability to cope with cold and hot weather. Third, these disparities manifested not only in the magnitude of weather's impact but also in its timing. While the poor faced higher mortality risks under all weather conditions, the social divide was most pronounced during the hot season.

Hot weather initially acted as a protective factor against neonatal mortality (whereas cold weather was extremely harmful), but this effect quickly reversed as infants aged. Panel 2.2 indicates that the shift from protection to risk began around 30 days of age — a pattern driven almost entirely by the day labourer class (panel 2.6). For the working class, the turning point occurred much later (around eight months), while the wealthy showed no clear inversion.

I argue that these turning points signal the onset of weaning and the transition to mixed or artificial feeding. Breastfeeding serves as a critical immune shield for newborns, and its interruption in unhygienic environments exposes infants to pathogens — especially during the hot season, when contaminated food/water and pathogen proliferation are most likely. The timing of hot weather's shift from protective to harmful thus serves as an indirect marker of breastfeeding cessation. In other words, they are a flag that the "weanling's dilemma" is occurring. This does not imply that all mothers abruptly stopped breastfeeding, but rather that a sufficient proportion shifted to riskier feeding practices to alter population-level trends. If this interpretation holds, this analysis suggests that the mothers of the lowest social strata seldom exclusively breastfed beyond one month. The working-class and petty bourgeois mothers typically breastfed for significantly longer durations than the poorest families. Regarding elite families, the interpretation remains more complex. Quite certainly, the excess mortality displayed in the first days of life is not a fictitious result. Similarly, the effect of an environmental disadvantage can be safely ruled out, let alone an economic disadvantage. On the other hand, identifying a behavioural cause is not an easy task. Musatti (1876) reported that some mothers delayed feeding their newborns for three or four days, but he did not suggest that such an unsafe practice was particularly widespread among the wealthy. Possibly, the search for suitable wet-nurses created dangerous delays in establishing suitable feeding. Even if this was the case, the absence of clear early-weaning signals in the elite mortality patterns indicates that, once arranged, feeding practices among wealthy families provided sustained nutritional and hygienic protection.

6 CONCLUSIONS

This paper pursued two objectives: methodological and substantive.

From a methodological point of view, I suggest an approach that allows making some inference on the duration of exclusive breastfeeding. Neither the "birth-interval approach" nor the "mortality approach" approximate breastfeeding rate or duration. At most, they reflect some greater or lesser predisposition of different social groups towards breastfeeding. My approach tries to estimate the age at weaning, the most critical moment in infant survival after birth. I use a combination of Cox proportional hazards models and Aalen's additive regression models. While Cox models provide precise (though potentially misleading) estimates of variables effects, Aalen's models reveal how these effects evolve over time, offering a more nuanced understanding of the underlying processes. When time-dependency is a critical feature — as in the case of feeding habits — Aalen's models prove to be a valuable addition to the analytical toolkit. Substantively, my analysis corroborated Cesare Musatti's claim that inappropriate nutrition — due to either premature weaning or a complete absence of breastfeeding — was the primary cause of the high infant mortality that plagued contemporary Venice. Musatti (1876) blamed the widespread poverty that forced many mothers to abandon breastfeeding out of economic necessity. Although ignorance of appropriate feeding methods also played a role, the "weanling's dilemma" that the poor had to face arose from the conflicting needs and constrains of mothers and children. On the other hand, maternal ignorance, malnutrition, and poor health, which also hindered breastfeeding, resulted from socioeconomic deprivation as well.

Inadequate feeding had dramatic consequences: in the population under study, over 41% of infant deaths were due to nutrition-related diseases (diarrhoea, gastroenteritis, marasmus), which spiked during the hot season. Spasms and airborne diseases represented 20% each, but many of them likely affected malnourished children with severe immunodeficiency deriving from previous gastrointestinal infections (Ginsburg et al., 2015; Kirolos et al., 2021). Crucially, the poor bore a disproportionate burden: a cause-specific analysis of mortality in this same population shows that the children of day labourers were three times as likely to die from nutritional diseases as middle/upper-class children, and twice as likely as the children of artisans and wage workers (Derosas, forthcoming).

Although mortality in adult and old age is beyond the purpose of this study, we should also consider that the impact of inadequate nutrition did not only concern infancy but encompassed the whole life-course. The Developmental Origin of Health and Disease (DOHaD) paradigm stresses that maternal nutrition during pregnancy and breastfeeding determine health condition in later life (Bianco-Miotto et al., 2017; Gialeli et al., 2023; Lacagnina, 2019). A growing body of literature shows that early nutrition has an impact on a wide range of outcomes, such as height, functionality, impaired cognitive function, mental health, reproduction, obesity, metabolic syndrome, diabetes, heart diseases, and cancer (Alves & Alves, 2024; Hildreth et al., 2023; McEniry, 2013; Orià et al., 2016).

The Exposomics paradigm stresses that early-life conditions determine the biological capital accumulated in an individual's lifetime, influencing the capability to access to other forms of capital — cultural, economic, and social — with dramatic consequences on life achievements (Vineis & Barouki, 2022; Vineis et al., 2016). Empirical research shows that chronic exposure to stressful environments causes a biological embedding of disadvantage, perpetuating social and racial health disparities (Meloni et al., 2022). Several historical studies confirmed the correlation between exposure in infancy and poor health and social outcome in adulthood and old age (see e.g. Bengtsson & Broström, 2009; Bengtsson & Lindström, 2000, 2003; Cormack et al., 2024; Quaranta, 2014; Schellekens & van Poppel, 2016; van Dijk et al., 2018).

Finally, we should also consider that sick and malnourished children are themselves sources of sanitary hazards and psychosocial stress, contributing significantly to the pathogenic load to which the other members of the household and neighbours are exposed. Individuals are not isolated, and diseases do not come alone. This is the major assumption of the Syndemics paradigm, which emphasizes the connection between interacting, co-present, or sequential diseases, and the social and environmental factors that foster and amplify the negative effects of disease interaction (Singer et al., 2017).

Current social and environmental epidemiology stresses that the consequences of feeding habits go beyond individual survival and well-being, to inform large sections of society and its mortality regime. Recent research on 19th-century Maastricht (Murkens et al., 2023) and Rostock (Mühlichen & Doblhammer, 2025) showed that food- and waterborne diseases were the major component of infant mortality and the key factor of health and social inequalities. Only their dramatic decline made a drastic decrease of mortality rate possible. Qualitative evidence supports the thesis that feeding practices, childcare, and hygiene played a crucial role. A more accurate knowledge of how societies and social groups coped with the "weanling's dilemma" may provide further evidence for this interpretation.

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Does the Healthy Migrant Effect Extend to the Next Generation?

Infant Mortality and Parental Migration Status in the Antwerp District, 1846–1906 cohorts

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ABSTRACT

This study examines whether the healthy migrant effect extends to the next generation by analyzing infant mortality by parental migration status in the Antwerp district for cohorts born between1846–1906. During this period, the region experienced large-scale migration from within Belgium and neighboring countries. Using longitudinal data from the Antwerp COR*-database, I conduct survival analysis to examine whether and how parental migration status impacted infant mortality. Cox proportional hazard models reveal that infants born to domestic migrant mothers had significantly lower mortality risks compared to those of native mothers — a 17 to 19% lower risk of dying. This effect remained robust after adjusting for the infant's sex, birth year, legitimacy status, maternal age at birth, and paternal socioeconomic status. No such advantage was observed for infants of international migrant mothers, likely due to their mothers' lower social integration. While infants of both domestic and international migrant fathers also exhibited lower mortality risks than those of native-born fathers, these effects were considerably smaller than those of domestic migrant mothers and not statistically significant.

Keywords: Healthy migrant effect, Infant mortality, Antwerp COR*-database, Antwerp, Survival analysis

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1 INTRODUCTION

The healthy migrant effect has been observed in various historical and contemporary contexts. Migrants are, on average, healthier and outlive natives, even if they originate from areas with higher mortality and have lower education and socio-economic status than non-migrants (Markides & Rote, 2019; Vang et al., 2016). Although the healthy migrant effect is not universal — occasionally, excess mortality among migrants has also been observed (Bakhtiari, 2018; Potarca & Bernardi, 2018) — it has been documented across a multitude of migrant groups in various societies and time periods (Alter & Oris, 2005; Lu & Qin, 2014; Puschmann et al., 2016a). Several explanations have been proposed — including selective return migration, differences in early life conditions, and variations in lifestyle — but most scholars attribute the phenomenon to positive selection in the area of origin: only the healthiest individuals move away from the communities in which they grew up, and the healthier they are, the more likely they are to migrate (multiple times) and to move further. Consequently, migrants form a selective group of particularly healthy and robust individuals (Lu, 2008; Puschmann et al., 2017).

While non-migrating siblings do not seem to have a health advantage — suggesting that the healthy migrant effect results from individual rather than family selection — it remains unclear whether the children of migrants benefit from their parents' health advantage (Mourits & Puschmann, 2023). The key question is whether migrants pass the healthy migrant effect on to their offspring — under the idea that healthy parents produce healthy children — or whether the advantage diminishes over time, disappears in the next generation, or even turns into a disadvantage, potentially because of adverse circumstances, such as discrimination and social exclusion. For the first generation, it has been observed — both in historical and contemporary contexts — that the healthy migrant effect diminishes or completely disappears over time, the longer migrants reside in their new destination. This pattern suggests a convergence in health between migrants and non-migrants over the life course (Bakhtiari, 2018; Kesztenbaum & Rosenthal, 2011; Loi & Hale, 2019). Thus, the question remains: do the offspring of migrants benefit from their parents' health advantage?

Existing studies on this issue show mixed results. I will first discuss findings from studies on contemporary societies, followed by a discussion of historical studies on the topic. Hummer et al. (2007) found that infants born to Mexican immigrants in the United States between 1995 and 2000 had about a 10% lower mortality risk during the first hour, first day, and first week of life compared to infants born to American-born women. DeCamp et al. (2015) confirmed that infants of Mexican-born women in Los Angeles County had lower mortality risks than those of U.S.-born women. Vidiella-Martin & Been (2023) documented lower mortality risks among infants of immigrant mothers in the Netherlands who were born extremely preterm, compared to equally early born infants of native-born mothers. Tang et al. (2019) found that domestic migrant mothers in contemporary Shanghai had a lower likelihood of pregnancy complications and gestational diabetes but that their infants faced increased health risks compared to those of native-born Shanghai women. Choi et al. (2019) observed that, in Australia, infants of mothers born in Africa and various Asian and Middle Eastern countries had elevated risks of stillbirth and preterm delivery and were more often in need of immediate care after birth compared to infants of Australian-born mothers. Finally, Wallace et al. (2023) detected that children of immigrants in various European countries today face increased risks of stillbirth as well as heightened perinatal, neonatal, and infant mortality risks. The authors point out, among other things, the importance of addressing racism, xenophobia, and discrimination in the healthcare system as well as in broader society. They recommend reevaluating integration policies, promoting full participation, and combating inequalities in education, income, social protection, and other areas.

Several historical studies have also investigated the health of the infants and children of migrants (Bakhtiari, 2018; Dribe et al., 2020; Eriksson & Niemesh, 2016; Oris et al., 2023; Olson & Thornton, 2011; Preston et al., 1994). Eriksson and Niemesh (2016) examined infant mortality among African Americans who migrated from the rural South to the urban North of the United States during the first half of the 20th century. They found that infants born in the North to parents originating from the South faced a higher mortality risk compared to those born in the South. This was largely attributed to the generally higher infant mortality rates in urban areas and the tendency of migrants to settle in unhealthy neighborhoods. Bakhtiari (2018) analyzed childhood mortality in the U.S. in 1910 by migration status and found that children of European immigrants faced higher mortality risks compared to children born to white, U.S.-born parents. However, their mortality risks remained lower than those of African American children. Dribe et al. (2020) reported similar findings for migrants in general. However, by disaggregating the data by nationality, they observed significant differences.

While children of Mexican, Canadian, Irish, and Hungarian parents had higher mortality risks, those of Russian, Scandinavian, German, and Dutch parents actually experienced lower mortality risks than children of native-born white American parents. The authors attributed these differences to varying levels of societal integration — a view supported by their finding that children of immigrant parents who had married native-born white Americans enjoyed a mortality advantage.

The study most similar to mine was conducted by Oris et al. (2023). They examined the survival chances of infants and children born to native and domestic migrant mothers in Madrid between 1916 and 1926. Their findings showed that children of mothers from Castilla-La Mancha had slightly better survival chances than those of Madrid-born mothers, while those from Castilla y León fared slightly worse. Overall, they concluded that maternal migration status had a limited effect. However, children born to mothers originating from more distant regions of Spain enjoyed significantly better survival chances after the first three months of life. The authors explained this by their higher socio-economic status, which enabled them to avoid the poor and unhygienic neighborhoods that contributed to high infant and child mortality at the time.

This study examines the survival chances of infants by parental migration status in the Antwerp district for cohorts born between 1846 and 1906. Previous research on the city of Antwerp identified a healthy migrant effect among both domestic and international migrants aged 30 and over during the period 1850–1930 (Puschmann et al., 2016a). Building on this, the current study explores whether this effect extended to the next generation by assessing the mortality risks of infants born to migrant mothers and fathers, and whether these infants experienced a survival advantage compared to those of native-born parents. To do this, I use life course data from vital registration records and population registers, retrieved from the Antwerp COR*-database (Matthijs & Moreels, 2010; Puschmann et al., 2022). I generate Kaplan-Meier survival curves and fit survival models — i.e., Cox proportional hazard models — initially including only the variables of interest, i.e., the mother's and father's migration status. In subsequent models, I add controls for the newborn's sex, birth year, and whether the child was legitimate or illegitimate, the mother's age at birth and the father's socio-economic status.

2 DATA AND METHODS

The data for this study were retrieved from the 2010 release of the Antwerp COR*-database (Matthijs & Moreels, 2010). The database consists of a letter sample, including all individuals whose last name started with "COR", as well as their spouses and other household members, from the population of the Antwerp district in the 19th and early 20th centuries. It is based on data from population registers and vital registration records of births, marriages, and deaths. The sample is representative of the larger population in terms of several key characteristics, including socio-economic status and migration status. The database enables life course reconstructions and family reconstitution and has been used for longitudinal studies on topics such as mortality, partner choice, marriage, fertility, migration, and social inclusion (Puschmann et al., 2022).

For the present study, all live births in the larger Antwerp district were selected from the birth certificates for the period 1846–1906. This period represents the part of the database that fully covers birth and death certificates, as well as the population register, ensuring that all infant births and deaths in the study are comprehensively documented. The study population consists of a total of 5,622 live births, of which 926 (16.5%) died within the first year of life in the study area. After selecting the live births from the database, I computed the variables for the survival analysis: survival time in days and failure status, i.e., whether the infant died within the first year of life (n failures = 926). Additionally, 24 infants were right-censored due to leaving the study area before their first birthday, and their survival time was adjusted accordingly.

Next, the main variables of interest were constructed: 1) migration status of the mother and 2) migration status of the father, based on their birthplace information. Parents born in the Antwerp district are considered natives (coded as 0); parents born elsewhere in Belgium are treated as domestic migrants (coded as 1); and parents born abroad are treated as international migrants (coded as 2). Parents whose birthplace was unknown were placed in a separate category (coded as 3).

Subsequently, I created the control variables: sex (0 = female; 1 = male), birth year (continuous), (il)legitimacy (0 = legitimate; 1 = illegitimate), age of the mother at birth (0 = < 20; 1 = 20–29; 2 = 30–39; 3 = 40+), and SES of the father. The latter is based on the father's occupational title at birth, which is coded in the database using the Historical International Standard Classification of Occupations (HISCO) (van Leeuwen et al., 2002). The HISCO code for each occupation in the database was recoded into HISCAM, a continuous occupational stratification scale ranging from 0 (very low SES) to 100 (very high SES) (Lambert et al., 2013). The variable was then recoded into a categorical variable consisting of: 1) high class (HISCAM ≥ 90), 2) middle class (HISCAM ≥ 50 and < 90), 3) working class (HISCAM < 50), and 4) unknown (no occupation present). Among the high class, we find occupations such as professor, doctor, lawyer, and high-ranking officers in the army, such as lieutenants and captains. The middle class consists mostly of clerks and craftsmen, ranging from bakers, tailors, and shoemakers to diamond workers, as well as farmers. The lower classes consist of various groups of unskilled and lower-skilled laborers and farm workers. Summary statistics for all variables are provided in Appendix 1. What stands out from these, is the relatively high share of mothers (15,8%) and especially fathers (19,53) with an unknown migration status, which is a weakness of the data that might potentially bias some of the results in the analysis.

When the dataset was ready, I conducted survival analysis using Stata 11.1. I produced Kaplan-Meier survival estimates by the migration status of the mother (Graph 1) and separately for the father (Graph 2). Subsequently, I fitted Cox (1972) proportional hazard models, with all-cause mortality (ages 0–1) as the failure event. All individuals were censored either after death or after leaving the study area. For those who did not die or leave the study area before their first birthday, they were censored after 365 days. In the first models, I included only the variables of interest, i.e., migration status of the mother and migration status of the father, separately. The results are displayed in the form of forest plots (Graph 3). Then I combined migration status of the father and the mother in one model and stepwise added control variables on the child and the parents in the next models (Table 1): sex of the infant, (il)legitimacy and birth year, age of the mother, and subsequently also socio-economic status of the mother. To check whether the proportionality assumption was violated, I examined the Schoenfeld residuals. No evidence of a violation of the assumption was found, as p-values for all variables were larger than 0.05, suggesting that the variables were not correlated with time.

3 HISTORICAL CONTEXT

During the 19th century, Antwerp grew into the largest city in Belgium, attracting large numbers of urban in-migrants, mainly from the Flemish countryside (especially from the larger Antwerp province) and to a lesser extent from French-speaking Wallonia and the neighboring countries: Germany, the Netherlands, France and England. Demographic pressure, agricultural crises, and innovations and scaling up in farming pushed migrants toward Antwerp city. The city's evolving port, handling an ever-growing volume of passengers and goods, acted as a major pull factor. Initially, Antwerp attracted mainly male migrants, as the port offered jobs primarily suited to men. However, in the last decades of the 19th century, the number of female migrants — some accompanying their families, others arriving as singles — began to rise as well, including those who migrated from greater distances (Greefs & Winter, 2016; Winter, 2009).

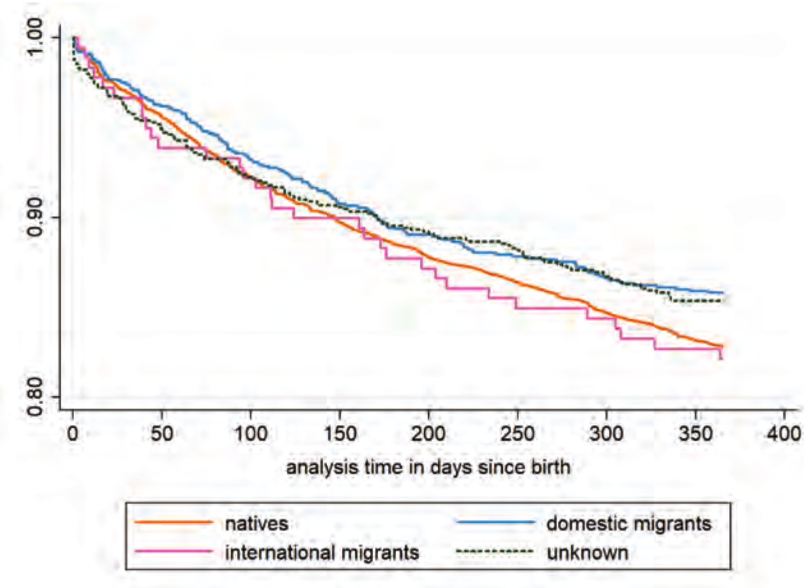
The rise of the port went hand in hand with the decline of Antwerp's textile industry, which could no longer compete with cheap linen from England. The transition from an industrial to a port city negatively affected the working and living conditions of the working class. It led to a decrease in real wages, which translated into heightened economic insecurity. Women, who had previously been heavily involved in the textile industry, were particularly affected, as the port offered few employment opportunities for them. As a result, the percentage of women active in the labor market declined, while others were forced to work as waitresses, domestic servants, or prostitutes (Lis, 1986). The growing insecurity and vulnerability in the lives of women is reflected in the rise of bridal pregnancies and out-of-wedlock fertility, as well as the increasing infant mortality in Antwerp from the mid- to late 19th century, with significant peaks during epidemic outbreaks, which were linked to rising population pressure (Donrovich et al., 2018; Vries & Puschmann, 2023).

The social inclusion of migrants was not always a smooth process, due to cultural differences and limited human capital (Puschmann et al., 2016b). The former was mainly true for international migrants and those from the Walloon provinces, while the latter was typical of Flemish rural-to-urban migrants. Overall, the process of social inclusion was easier in Antwerp than in other major port cities, such as Rotterdam and Stockholm. Migrants who arrived as children fared much better than those who arrived after their 30th birthday. International migrants performed very well in the labor market, holding higher positions on average than natives. Internal migrants initially had to content themselves with lower positions, but the longer they stayed, the better integrated they became in the labor market. Those who stayed long enough ultimately outperformed natives. However, migrants' access to marriage and reproduction was severely hampered, especially for international migrants and French-speaking Walloon migrants. Migrants who did marry often did so with fellow migrants, and mixed marriages were much less common than would be expected if partner selection were random, suggesting that many migrants and natives lived segregated lives and that cultural barriers prevented long-distance migrants from becoming socially integrated (Puschmann, 2015; Puschmann et al., 2013, 2016b).

4 RESULTS

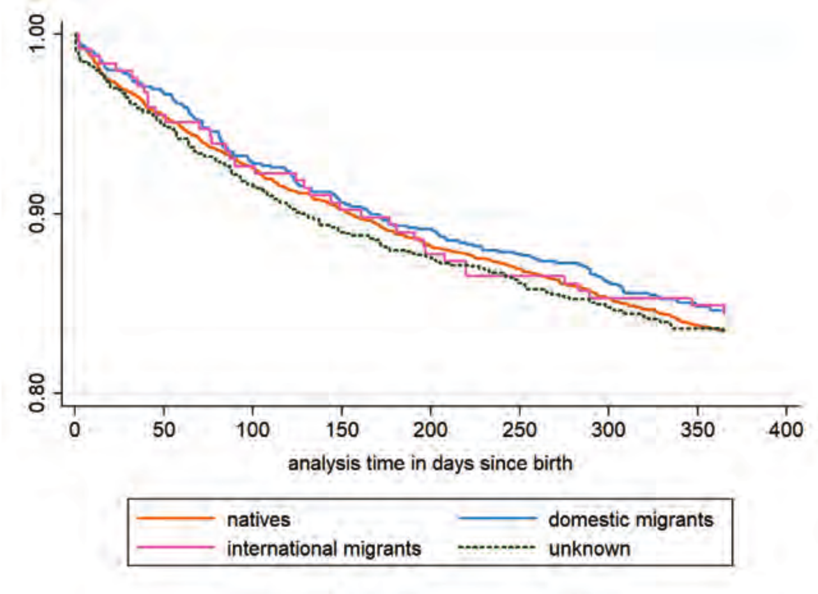
Graph 1 displays the Kaplan-Meier survival curves of infants in the study population by the migration status of the mother. There are few, if any, differences in survival chances by maternal migration status during the neonatal phase (0–27 days). Only the survival rate of infants with mothers of unknown birthplace seems to have been somewhat lower. However, in the post-neonatal phase (28 days to 1 year), differences begin to appear and grow gradually over time. The survival chances of infants born to domestic migrant mothers and mothers with an unknown migration status were somewhat better compared to those of native mothers, and even more so compared to infants of international migrant mothers. However, given the jagged line for the latter category, these observations are less reliable due to small sample sizes. Indeed, there are only 179 (= 3.18%) international migrant mothers in the study population.

Graph 1 *Kaplan-Meier survival curves by migration status of the mother (n = 5,622)*



Source: Antwerp COR-database.*

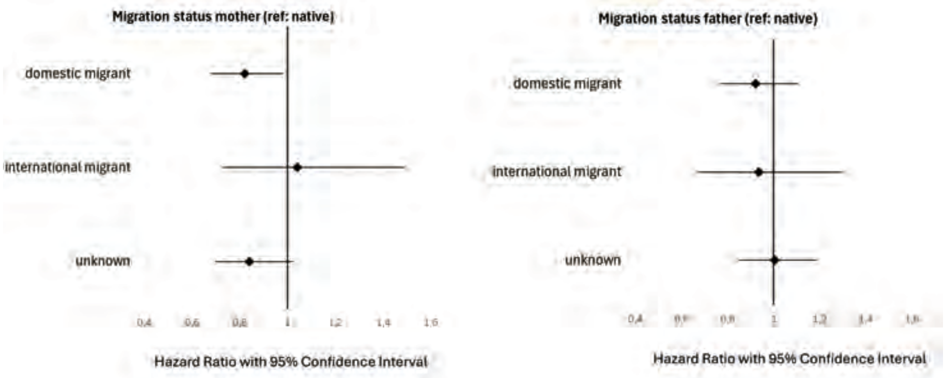
Graph 2 *Kaplan-Meier survival curves by migration status of the father (n = 5,622)*



Source: Antwerp COR-database.*

Graph 2 shows the survival curves of infants in the study population by the migration status of the father. Again, there are few differences in the survival curves during the neonatal phase, although infants with fathers of unknown migration status fared slightly worse. In the post-neonatal phase, small differences in the survival curves emerge. Infants of migrant fathers generally had somewhat higher survival chances than those of native fathers for most of the period, but the differences were smaller than those observed for maternal migration status. Over the entire period, infants with fathers of unknown migration status fared worse. Infants with international migrant fathers initially did somewhat better than those with native fathers, until about 180 days, after which they fared worse, and then better again toward the first birthday. However, as with the previous analysis, it is clear that the estimates for international migrant fathers (n = 246; 4.35% of the total study population) are less reliable due to the small sample size.

Graph 3 *Cox proportional hazard models for infant mortality (Unadjusted; n = 5,622)*



Source: Antwerp COR-database.*

Graph 3 displays the results of the Cox regression on infant mortality for the two main variables of interest in separate unadjusted models by ways of forest plots: migration status of the mother and migration status of the father. The only significant effect is found for domestic migrant mothers. Infants born to these mothers have a lower hazard ratio (HR: 0.82) of mortality in the first year of life compared to infants born to native Antwerp mothers. The results for children born to migrant fathers — both domestic and international — point in the same direction, although the effects are smaller (HR for domestic migrants: 0.92; HR for international migrants: 0.93) and not significant.

Table 1 *Cox proportional hazard models for infant mortality (n = 5,622)*

	Model 1	**Model 2**	**Model 3**
	HR	HR	HR
Migration status mother			
Native	Ref	Ref	Ref
Domestic migrant	0,82**	0,81**	0,83*
International migrant	1,04	1,04	1,08
Unknown	0,69**	0,75	0,75
Migration status father			
Native	Ref	Ref	Ref
Domestic migrant	0,98	0,94	0,95
International migrant	0,96	0,96	0,96
Unknown	1,28**	1,18	1,22
Sex			
Female		Ref	Ref
Male		1,04	1,03
Legitimacy			
Legitimate		Ref	Ref
Illegitimate		1,08	1,13
Birth year		1,00***	1,01***
Age mother			
< 20			Ref
20–29			0,75*
30–39			0,71*
40+			0,63**
SES Father			
Elite			Ref
Middle class			1,41
Laborers			1,60
Unknown			1,21

*Note: *p < 0.10, ** p < 0.05, *** p < 0.01.*

Source: Antwerp COR-database.*

In the final part of the analysis, I fit Cox proportional hazard models with both variables of interest included simultaneously (Model 1). Subsequently, I add control variables. In Model 2, I add characteristics of the child: sex, year of birth, and whether the child was legitimate. In Model 3, I add characteristics of the parents: age of the mother at birth and SES of the father at birth. The results from the multivariate analysis largely confirm the descriptive results as well as the unadjusted models. I find a survival advantage only for infants born to domestic migrant mothers. In Model 1, there is an 18% lower risk of mortality for infants born to domestic migrant mothers. This effect is significant and remains constant and significant in Models 2 and 3 when I add control variables for the infant and the parents. In Model 1, there is also a significant effect for mothers and fathers with an unknown migration status. However, after controlling for other factors, these effects are no longer significant. The hazard rates indicate a minor survival advantage for infants of domestic (HR: 0.98) and international migrant fathers (HR: 0.96). However, both results are not significant. In the case of the latter category, this is likely due to a lack of statistical power. Infants born to fathers with an unknown migration status have a significantly higher mortality risk (HR: 1,28) in model 1, but when the controls are added the effect becomes insignificant.

5 CONCLUSION AND DISCUSSION

This research showed that infants of domestic migrant mothers in late 19th- and early 20th-century Antwerp had better survival chances than those born to native mothers. This effect persisted even after controlling for infant and parental characteristics. These findings suggest that the healthy migrant effect may be transmitted from mother to child, with healthier mothers giving birth to healthier infants. However, the Kaplan-Meier survival curves showed that the health advantage only clearly emerged in the post-neonatal phase, suggesting it is less likely to be biologically driven (e.g., by genetic disorders, prematurity, birth complications, or low birth weight) and more likely linked to social and environmental factors (e.g., lower risks of infectious diseases, malnutrition, weaning problems, or environmental pollution). This finding, of course, raises new questions: Is the survival advantage among infants of domestic migrant mothers due to the mothers simply being healthier? Or is it because these mothers were better able to protect their children from health hazards — for instance, by breastfeeding more frequently or for longer durations — compared to native-born mothers? This may itself reflect better health, but could also be shaped by different living and working conditions, or a combination of both.

No survival advantage was found for infants of international migrant mothers. Although not significant, their hazard rates were even slightly above 1. I believe that the absence of a health advantage among this group may have been due to the lower social integration of their mothers. Previous research highlighted that international migrant women who lost their spouses had a much higher mortality risk compared to domestic migrants in similar situations (Donrovich et al., 2014). This suggests that international migrant women in Antwerp had fewer family members and friends to rely on for social and financial support. Due to cultural differences, they may have also found it harder to build such a network (Puschmann et al., 2016b). Moreover, international migrant women in Antwerp had a higher risk of remaining single and were more likely to give birth to one or more illegitimate children, underlining their lack of social support and vulnerability. By contrast, domestic migrant women had an even lower risk of giving birth to an illegitimate child than native-born Antwerp women (Vries & Puschmann, 2023). We might therefore tentatively conclude that the healthy migrant effect is only transmitted from mothers to children if the mothers are well integrated into the destination society. This could explain the mixed results in the literature, where in some cases, the offspring of migrants enjoy a survival advantage, while in others, they face a disadvantage or no difference.

We found no evidence that the migration status of the father mattered. The hazard ratios of infants of migrant fathers — both domestic and international — pointed to a small survival advantage, but the effect was not significant. Most likely, the health of mothers is more important for infant survival, given that children spend approximately nine months in the womb and are often breastfed by their mothers. Moreover, existing research on the Netherlands finds that the death of a mother during infancy (0–1) or early childhood (1–5) was associated with a considerably higher risk of child mortality compared to when the father died (Quanjer et al., 2023). These results should not surprise as women acted as the primary caretakers of infants and the role of fathers was mostly confined to that of (financial) provider.

One limitation of this study is the categorization of parental migration status into only three broad groups: natives, domestic migrants, and international migrants. It would be valuable to further distinguish within the domestic migrant group between Flemish and Walloon migrants — especially since this study, along with previous research (e.g., Wallace et al., 2023), highlights the importance of societal integration. Walloon migrants, due to differences in language and culture, may have faced greater challenges integrating into Antwerp society compared to Dutch-speaking Flemish migrants, and this might have resulted in higher mortality among their offspring. Similarly, within the group of international migrants, it would be interesting to examine survival differences among infants by the parents' national origin — for example, comparing Dutch migrants, who share a language with the local population, to migrants from France. However, the current data sample does not support such detailed analysis due to the small number of cases in these specific subcategories. Another limitation is the relatively large proportion of parents with unknown migration status — 16% of mothers and 19.5% of fathers.

Future research could look also into whether the health advantage of children of domestic migrant women in Antwerp persisted into childhood, adolescence, and adulthood, or whether it diminished over time. Additionally, more replication studies are needed to determine whether the results from Antwerp hold in other historical contexts. Is it indeed only the best-integrated migrants who pass on the healthy migrant effect to their offspring? Is maternal migration status always more critical for child survival than the migration status of the father?

To better understand the healthy migrant effect in historical contexts, future research should move beyond all-cause mortality and focus on cause-specific mortality analyses. Which causes of death were migrants and their children less likely to experience compared to natives? By systematically comparing causes of death across various contexts — between migrant and non-migrant populations, as well as between offspring of migrant parents who did and did not exhibit a health advantage — we can better identify the causal mechanisms underlying the healthy migrant effect and its intergenerational transmission or lack thereof. This approach could potentially also shed a light on which health-protective characteristics and behaviors are actually transferred from migrant parents to their children, and which factors contribute to non-transmission or even a reversal of health advantages.

In recent years, an increasing number of cause-of-death registers have been digitized — especially within the SHIP network — and a historical cause-of-death coding and classification scheme for individual-level causes of death, the ICD10h, has been developed (Janssens, 2021; Janssens & Devos, 2022; Reid et al., 2024). The S.O.S. Antwerp (2025) project will provide cause-of-death data for the city of Antwerp covering the period 1820–1946. With these advancements, the necessary data and tools to conduct the proposed analyses are now within reach — not only for Antwerp but also for many other European cities — opening up new avenues for comparative research on the health of migrants and their offspring.

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APPENDIX

Appendix 1 *Summary Statistics*

	Percentage	Mean	SD	Min	Max
Migration status mother					
Native	63.3				
Domestic migrant	17.6				
International migrant	3.1				
Unknown	15.8				
Migration status father					
Native	59.1				
Domestic migrant	17.0				
International migrant	4.4				
Unknown	19.5				
Sex					
Female	49.0				
Male	51.0				
Legitimacy					
Legitimate	93.4				
Illegitimate	6.6				
Birth year		1880.1	16.8	1846	1906
Age mother		30.0	6.6	15	50
< 20	3.0				
20–29	46.1				
30–39	39.2				
40+	11.7				
SES father					
Elite	1				
Middle class	58.2				
Laborers	31.6				
Unknown	9.3				
N of infants	5,622				

III

Engines of Disparity

HISTORICAL LIFE COURSE STUDIES
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Social Differences in Maternal Mortality in Zeeland 1812–1913

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ABSTRACT

Using population reconstructions from linked civil certificates for the province of Zeeland, the Netherlands, for the period 1812–1913, I study the social gradient in maternal mortality. Maternal mortality is defined as deaths in the first 42 days after the birth of a child. Among the women — mother to at least one child and followed between age 20 and 45 — maternal mortality constitutes about one third of the total number of observed deaths. Maternal mortality is higher for upper class women in early 19th century Zeeland than for unskilled laborers. By the early 20th century, maternal mortality had become an uncommon event and social differences in its likelihood negligible. A comparison of the social gradient in maternal mortality to the social gradient in all mortality in the reproductive ages (age 20-45) in this period shows that the reverse social gradient in mortality is limited to maternal mortality — it is not found for all women's deaths in this period of life.

Keywords: Maternal mortality, Social differences in mortality, Mortality decline, Cause of death statistics, Social gradient

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1 INTRODUCTION

In the mid-19th century Netherlands maternal mortality rates — mortality of mothers to causes related to childbirth — rapidly fell (Ory & van Poppel, 2013; Woods, 2009). Unhygienic practices around childbirth, and the absence of trained midwives contributed to high maternal mortality earlier in the 19th century (Woods, 2009). Moreover, high fertility and short interbirth intervals in combination with a low general standard of living and nutritional deficiencies increased the risk of maternal mortality (Janssens & van Dongen, 2018). In the mid-19th century, living conditions in the Netherlands started to improve. From the 1880s adult mortality started to fall (Wolleswinkel-van den Bosch et al., 2001), spearheaded by declining maternal mortality rates due to similar improvements that, around the same period, also started to benefit survival of infants and young children: better hygiene and a higher standard of living and fewer nutritional deficiencies (van Dijk et al., 2024). A higher overall standard of living could have contributed to women's general health before pregnancy and childbirth, making them less prone to infection and disease after giving birth.

The possible contribution of resource-related factors suggests that social class differences may have existed in maternal mortality. Ory and Van Poppel (2013) show that in several regions of the Netherlands maternal mortality was higher for women from the class of skilled workers and even more so for farmers. In England, in the late 19th century and early 20th century, elite women had a higher risk of maternal mortality (Loudon, 1986), possibly because of medical care of a poor standard that was detrimental to women's health and survival after childbirth in the absence of proper hygienic practices. In the late 19th century, midwifery went through a transformation leading to increased professionalization and increased implementation of antiseptic techniques (Løkke, 2012; Woods, 2009) which greatly contributed to the survival of mothers and children. From a modern perspective, a social gradient could be expected in such mortality improvements, with the upper class benefiting most from such developments. Previous work (Ory & van Poppel, 2013) has shown a higher maternal mortality among skilled workers and farmers in the period 1846–1902, next to other risk factors, both biological and social.

However, little is known about changes over time in the contribution of social class to the maternal mortality risk. With time, risk factors for maternal mortality in the higher classes could have changed into a comparative advantage. For example, in the presence of improved hygienic measures of doctors and midwives their help at birth could have started to contribute more to survival chances of women giving birth (Loudon, 1986). Previous studies of maternal mortality have not accounted for such possible time trends in social differences in maternal mortality in the Netherlands. In this paper, I address long-term change in maternal mortality using family reconstructions, addressing the level and decline of maternal mortality and developments in social differences in maternal mortality in Zeeland, the Netherlands, 1812–1913.

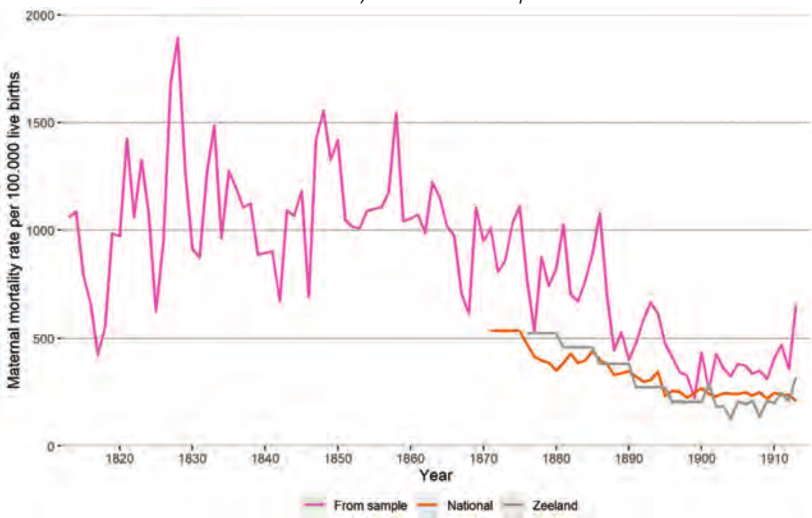
2 MATERNAL MORTALITY IN ZEELAND AND THE NETHERLANDS IN THE RESEARCH PERIOD

For the period 1875–1899, maternal mortality was reported in five-yearly cause of death statistics at the municipal, provincial and national level. Maternal mortality was reported in two categories: puerperal fever and other pregnancy and childbirth related causes of death. National cause of death registration was introduced in the Netherlands in 1865 by the introduction of the Public Health Inspectorate Act and the Medical Practitioners Act, resulting from efforts of the Dutch hygienist movement (van den Boomen & Ekamper, 2015; Walhout, 2010). Doctors were obliged to record the cause of death before burial. Slips on which causes of death were registered were send to the local administration and aggregated for country-wide statistics. Not all patients had been seen by a doctor and the cause of death was sometimes attributed after death or even burial (van den Boomen & Ekamper, 2015). These official statistics on puerperal fever and other pregnancy and childbirth related causes of death may therefore be incomplete for maternal mortality.

Reconstructing the maternal mortality level for the 19th century is possible using population reconstitution data. Linked indexes of civil records can be used to estimate the level of maternal mortality, defined as deaths in the first 42 days after childbirth. A death in the postpartum period is the contemporary WHO definition of maternal mortality if a specific cause of death is unknown. The linked data used in this paper contain observations on women's marriages, death of their partner, births of their own children, and the date of their own death, so that a mother's death in the first 42 days after childbirth can be identified. The maternal mortality rate based on linked civil records is significantly higher than the nationwide reports indicate. Compared to the number of births of these women, this results in an estimate of the maternal mortality rate — maternal deaths per 100,000 births — of over 1,000 per 100,000 childbirths in the early and mid-19th century. By 1875, the maternal mortality rate was rapidly falling, to around 750 mothers per 100,000 births. In this year, official national maternal mortality statistics were introduced. These result in estimations of the maternal mortality rate of slightly over 500 per 100,000 births for the province of Zeeland (526 over the years 1876–1880) and somewhat under 500 per 100,000 for all the Netherlands (476 in the year 1876).

Why do these estimates from official statistics and based on reconstituted family data for maternal mortality in Zeeland differ? Some maternal deaths after childbirth may incorrectly be included in maternal mortality estimates, and the degree to which that happens could differ between the two sources. The annual fluctuations observed in Figure 1 suggest that in some years part of the mortality attributed to obstetric causes may have had their primary cause in epidemic conditions. Yet, women could be more vulnerable to infectious disease in the postpartum period, so that this mortality is at least in part related to their status as new mothers. The same might be the case for mortality that is included with other causes of death in the official statistics: women may have been at greater risk because of their recent birth, and some mortality in relation to the childbirth may have incorrectly been included as due to another cause of death. Estimates of maternal mortality based on family reconstructions could therefore be more complete than official statistics. However, Ory and Van Poppel (2013) suggest that the true maternal mortality rate is probably lower than suggested by family reconstructions but also higher than what official statistics maintain, as official statistics may have omitted some maternal mortality by accident, to avoid culpability of doctors, or because deaths that would not have occurred without the pregnancy but that had a different primary reason (such as tuberculosis) were not included.

Figure 1 *Decline in maternal mortality in the research period*



Maternal mortality from the sample is based on mothers' deaths in the first 42 days after giving birth, divided by the number of births in the sampled group, based on population reconstitutions from linked civil certificates in LINKS, Zeeland. The national annual number of births nationwide and in Zeeland are from Statistics of Population Change in the Netherlands ("Statistiek van den loop der bevolking van Nederland"). Maternal mortality statistics are from 5-year reports on causes of death for regions and the nation "Vijfjarig overzicht van de sterfte naar den leeftijd en de oorzaken van den dood in elke gemeente van Nederland" for the years 1880 (1875–1879), 1885 (1880–1884), 1890 (1885–1889), 1895 (1890–1894), 1900 (1895–1899) (van den Boomen, 2021). Annual maternal deaths nationally 1875–1900 are taken from the "Statistics of Mortality by the causes of death over the year 1903" (Statistieken van den sterfte naar den oorzaken van den dood over het jaar 1903). Annual deaths after 1900 nationally and Zeeland from theS Statistics of Mortality by the causes of ddeath over the year...(Statistieken van den sterfte naar den oorzaken van den dood over het jaar…) 1901–1913. Data on maternal mortality and replication code is available from Zenodo: https://doi.org/10.5281/zenodo.15487217.

In this paper, I use maternal mortality rates estimated from family reconstructions. First, I estimate the share of maternal mortality in all mortality in the linked sample. A strikingly large number of deaths among Zeeland mothers aged 20–45 occurs in the first 42 days postpartum. In the period 1812–1839 around one in three deaths at this age range is in the postpartum period, falling to one in four in the period 1890–1913 (see Table 1).

3 DATA AND METHODOLOGICAL APPROACH

Analyses employ LINKS — LINKing System for historical family reconstruction — a large-scale historical demographic dataset for the province of Zeeland, Netherlands (Mourits et al., 2022). The data and code for this paper are available (https://doi.org/10.5281/zenodo.15487217). The province of Zeeland is situated in the southwestern corner of the Netherlands and consists largely of islands. The population had high fertility throughout the study period, with a mean number of children of around 8 for married couples (van den Berg et al., 2019). Infant and child mortality were high and life expectancy was low, with child mortality reaching 50% before the fifth birthday in some municipalities and years, with high levels of epidemic disease in particularly affected communities (Hoogerhuis, 2003; van Poppel & Mandemakers, 2002).

The LINKS database uses linked indexed civil certificates that originate from obligatory vital event registration, which was introduced in the Netherlands in 1812. The certificates include information on births, marriages and deaths, including the names of the individual and his or her parents, the municipality of the vital event, the individuals' place of birth, age and occupational titles of the index person and parents. Life course events of the same individuals have been linked together through name-based linkage of certificates pertaining to the same individuals, using first and last names of the ego and his or her parents, spouses, and children. The full database contains information on 1,930,189 individuals from Zeeland who experienced a vital event in that province between 1812–1913 for births, 1812–1938 for marriages, and 1812–1963 for deaths. LINKS has been used extensively for historical demographic research on the Netherlands and has been subject to thorough checks of quality and reliability (van den Berg et al., 2021). A unique advantage of the data for our purpose here is that also stillbirths were registered so that mortality in the postpartum period after any birth is included.

For the analysis, women in the reproductive ages 20–45 are included, if they married in Zeeland and at least one childbirth is observed. Descriptive statistics may be found in Table 1. Children are born between 1812 and 1913. 96,661 mothers are included, and 515,632 childbirths and the postpartum period are observed. For a full overview of selection criteria, see Figure A1. A full death date is available for 72,329 of the mothers (74,8%) included in the analyses. A death date or observation after age 45 (a civil record linked after age 45 which is not the death certificate, but for example a (re)marriage) is available for 73,171 women or 75.7% of all women. In our main analysis, it is presumed that women without follow-up survived to age 45 as mortality is generally low in this period of life and linkage between married women's life course observations with their own deaths are generally of high quality. However, some of the women for whom no death certificate was available may have out-migrated from the province of Zeeland. In a robustness check, women with no information about their death date are dropped from the analysis, and the differences between social classes in the period 1812–1839 are now smaller and only marginally significant (see Section 4).

It is important to note that contrary to previous studies, here, all women for whom at least one birth is observed are included in the analyses. In the analyses of maternal mortality, mothers' mortality in the 42 days postpartum is analyzed, the contemporary WHO definition of maternal mortality in absence of a clear cause of death (Ronsmans & Graham, 2006). The postpartum period after a stillbirth is also included. Stillbirths were registered separately in the Dutch municipal administration and to study maternal mortality these cases are of particular importance, as a difficult childbirth could result in the death of both mother and child.

Next to studying mothers' deaths in the 42-day postpartum window, the hazard ratio of all mortality of mothers between age 20 and 45 is also studied. All women for whom at least one childbirth is observed in the province of Zeeland, are included, i.e. the same sample of 96,661 mothers as used for the analysis of the first 42 days after childbirth. Here, follow up is from first observation of the woman in the research area (the year 1812, age 20 for Zeeland-born women, or her marriage or first observed childbirth in the province) to her death, age 45 or the year 1913 when the observations end. A fair amount of mortality between age 20–45 occurs in the post-partum period, as mortality rates due to all causes generally bottom out in this age group. It should be noted that a significant downside of the approach taken here is that the reproductive histories of women cannot be included in the analysis, as I analyze *any* maternal death that happened in Zeeland in the period 1812–1913. The reason I choose this approach is that social differences in maternal mortality can be analyzed further back in time as no full record of mothers' marital and fertility history is necessary. Sociodemographic patterns can differ between socioeconomic groups so that not controlling for women's marital histories makes it impossible to pinpoint some of the mechanisms that contribute to social differences in maternal mortality.

The key predictor is the socioeconomic status (SES) of women's households. In the research period, men were usually the primary breadwinner. Female labor market participation was severely under registered (Janssens, 1997). In line with previous research on this region and period, analyses therefore rely on the occupation of the husband. SES was measured using the highest known occupational title with HISCLASS scores across all indexed civil certificates linked to one person. HISCLASS is a system that groups historical occupational titles into broad occupational classes. This broad status indicator is recoded into 5 occupational groups: elite (HISCLASS 1–2), lower middle class (HISCLASS 3–6), skilled workers (HISCLASS 7 and 9), unskilled workers (the reference group for the analysis, HISCLASS 10–13), farmers and fishermen (HISCLASS 8), and a separate, sixth category for missing observations (van Leeuwen & Maas, 2011). Occupational titles from the Netherlands have been standardized in previous work (Mandemakers et al., 2020).

Table 1 *Descriptive statistics by period*

Sample of births	Any year	1812–1839 Birth cohort	1840–1869 Birth cohort	1870–1889 Birth cohort	1890–1913 Birth cohort
SES of the mothers (HISCLASS)					
Elite	6,7 (34,436)	10,9 (9,987)	8,0 (12,669)	5,1 (6,320)	3,9 (5,460)
Lower middle class	15,5 (80,109)	11,9 (10,904)	13,5 (21,429)	17,4 (21,696)	18,5 (26,080)
Skilled workers	18,6 (95,633)	17,0 (15,616)	18,5 (29,317)	17,7 (22,046)	20,4 (28,654)
Unskilled workers	28,6(147,346)	31,3 (28,816)	31,4 (49,772)	28,2 (35,177)	23,9 (33,581)
Farmers	14,8 (76,133)	13,4 (12,322)	13,3 (20,993)	15,3 (19,083)	16,9 (23,735)
Missing SES	15,9 (81,865)	15,5 (14,279)	15,2 (24,132)	16,3 (20,351)	16,4 (23,103)
Children/births	515,522	91,924	158,312	124,673	140,613
Maternal deaths (first 42 days)	4,139	992	1,612	976	559
Mothers	96,565	21,463	36,498	30,574	35,710
Sample of mothers	Any year	1812–1839 Period	1840–1869 Period	1870–1889 Period	1890–1913 Period
SES (HISCLASS)					
Elite	6.7	9,8	7,8	5,5	4,5
Lower middle class	15,2	11,7	13,3	16,5	18,5
Skilled workers	18,9	17,7	18,5	18,3	20,7
Unskilled workers	29,7	33,4	33,4	29,0	23,9
Farmers	13,4	11,5	11,5	14,5	15,8
Missing SES	16,1	16,0	15,5	16,3	16,6
Mothers	96,565	27,237	49,779	44,598	44,491
Maternal deaths (first 42 days)	4,139	992	1,612	976	559
Deaths age 20–45	13,408	2,779	5,443	3,020	2,166
Person-years	2,065,277	367,743	649,047	470,613	577,875
Children/births	515,522	91,924	158,312	124,673	140,613

Source: LINKS, Zeeland, 1812–1913 (Mourits et al., 2022).

Notes: Descriptive statistics for child sample refer to the percentage of children from a SES background based on father's occupation. Descriptive statistics for the mother sample are in person years and refer to SES associated with the husband's occupation.

4 RESULTS

Results are shown in Figure 2–4. Figure 2 presents the risk of mortality in the first 42 days after childbirth for every birth observed in the province of Zeeland, by social class and period. In Figure 3, the overall mortality risk of women between age 20 and 45, by social class and period, is reported. In the period 1812–1839, the risk of mortality in the first 42 days after the birth of a child is significantly higher for skilled workers and elite women and, marginally significantly, for the lower middle class. In the final period reported, 1890–1913, women in the farming class have a marginally significantly higher risk of mortality in the first 42 days postpartum than unskilled workers, but other social differences in mortality are no longer detected. Moreover, in the period in between no significant social differences in maternal mortality are found.

Over 30% of the mortality among women aged 20–45 with at least one child occurs in the first 42 days after a childbirth. The share of maternal mortality in all mortality declines from over 35% in the period 1812–1839 to about one quarter of observed deaths among women with at least one child in the period 1890–1913. Not only does the contribution of maternal mortality to all deaths decline, but the adult mortality rate also falls strongly in this period.

The results for women's mortality in the postpartum period contrast with overall mortality between age 20 and 45 for mothers (Figure 3). For all deaths among mothers, regardless of the timing of childbirth, mortality is only somewhat raised in elite groups and among skilled workers and the lower middle class in the period 1812–1839 — and these results are entirely driven by mortality in the postpartum period (Figure 4). Without maternal mortality, the hazard rate of mortality is significantly lower among the elite and middle class in the period 1840–1869 than among unskilled workers.

Figure 2 *Hazard Ratio of maternal mortality in the first 42 days after childbirth, Zeeland*

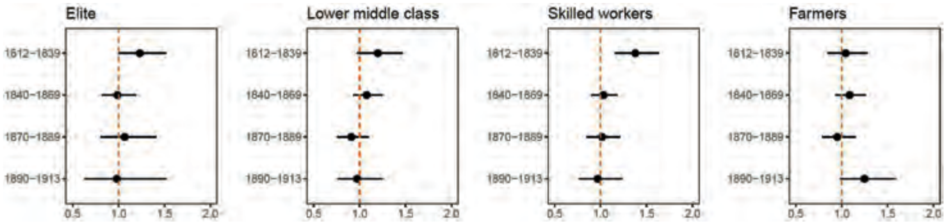


Figure 3 *Hazard Ratio of mothers' mortality age 20–45 by social class, Zeeland*

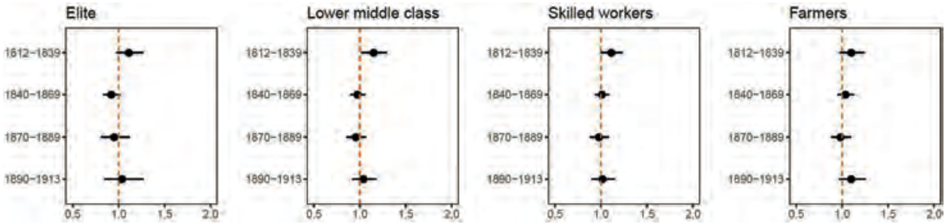
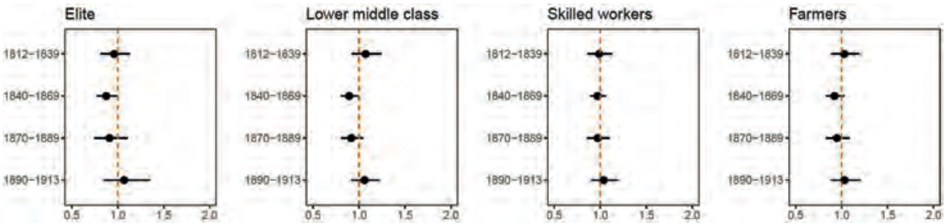


Figure 4 *Hazard Ratio of mothers' mortality age 20–45 excluding deaths in the postpartum period by social class, Zeeland*



Source: LINKS, Zeeland, 1812–1913.

Note: Reference group: Unskilled workers. Analyses are separately by period.

In a robustness check, only women for whom there is an observation after age 45 or whose death is observed are included. That is, survival is not assumed for women whose death is not observed before age 45, but they are removed from the sample, dropping about one quarter of the women (see Section 3). Results are shown in Appendix Figure 2. In this version of the analyses some of the previously shown differences between social groups are less pronounced for the period 1812–1839, indicating a possible underestimation of mortality among women from the reference group, the class of unskilled workers, in the primary models. The higher mortality of women from the class of skilled workers and lower-middle-class workers than in the reference group of unskilled workers is here marginally significant ($p < .10$) instead of significant at the more common $p < .05$ level in the first period. The largest change is that there are no significant differences found between elite women and unskilled workers. Finally, in the 1840 to 1869 and 1870 to 1889 periods, farmers have significantly lower mortality than unskilled workers. These findings make clear that the results presented in Figure 3 should be interpreted with caution. At the same time, as out-migration should be less of an issue shortly after the birth of a child than over the entire life course of women, results in the models presented in Figure 2 should be less affected by the absence of follow-up observation than those over the entire 20–45 age window.

5 CONCLUDING DISCUSION

Using population reconstructions from linked civil certificates for the province of Zeeland for the period 1812–1913, I compared social differences in maternal mortality with social differences in women's overall survival between age 20–45. I showed that in the province of Zeeland, maternal mortality was higher among elite, lower middle class and skilled worker class-women in the early 19th century, compared to unskilled workers. Their comparative disadvantage disappeared by the middle of the 19th century. By the early 20th century maternal mortality had become an uncommon event and social differences in its likelihood negligible. In comparison to all women's mortality between age 20–45, substantially larger socioeconomic differences in maternal mortality are found. The differences in mortality between social groups for women aged 20–45 in the period 1812–1839 are driven by mortality in the postpartum period and when maternal mortality is excluded, lower mortality for women from elite and lower middle-class households than for women from unskilled worker's households is found.

Higher mortality among elite women has been found previously, for example, for England for the late 19th and early 20th century (Loudon, 1986). In this paper, evidence for such a phenomenon is only found for the early 20th century. It has previously been hypothesized that increased mortality for the elite groups may have been related to dangerous medical help in the absence of good antiseptic and aseptic procedures. Although elite women in Zeeland may have had access to medical care more than women from unskilled laborer's households, it is unclear if that may also have been true for skilled workers' households. Other explanations for the patterns found here may relate to, for example, fertility differences between social groups. With the chosen approach, addressing any death in the first 42 days following a (linked) childbirth, social inequalities in maternal mortality can be addressed somewhat further back in time than in previous work (Ory & van Poppel, 2013), using data from LINKS Zeeland from 1812 to 1913. However, by not addressing women's fertility histories nor following them through their marital life and reproductive career, it was not possible to address fertility differences as a mechanism linking class and maternal mortality. The exact mechanisms related to these social differences therefore remain elusive. In the future, more comprehensive data sources for the period preceding the nationwide implementation of civil records would help illuminate the mechanisms contributing to higher maternal mortality among the upper class in the first part of the 20th century, and the later disappearance of such social class differences in maternal mortality.

To investigate social differences in maternal mortality, the socioeconomic status of the household was used, based on the occupation of women's husbands. In historical data, women's work is under-registered, even if most women worked, at home, in the family business, and in paid labor (Janssens, 1997). Differences by women's own social status to the extent that it related to their own occupation were not included in this work. Neither does it account for social inequalities that may exist beyond socioeconomic status alone. Spatial or familial clustering of particularly poor conditions, putting women at risk of infection and death after childbirth, or a local shortage of medical help and help for women in labor, may have resulted in familial and spatial concentration of maternal mortality. Finally, the death of a mother had a large, negative effect on her children — reducing the survival chances and health of her children in historical Netherlands (Quanjer et al., 2023). The decline of maternal mortality therefore likely contributed to the increasingly good standards of living in which children in the Netherlands grew up, and played a crucial role in the transition to longer lives in better health witnessed over the 19th and 20th century.

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The linked indexes of the civil records are available from the Institute of Social History, Amsterdam, Netherlands: https://hdl.handle.net/10622/QUJNCD. Data on maternal mortality and replication code is available from Zenodo (https://doi.org/10.5281/zenodo.15487217. Code for replication of figures and tables is also available at ingridvandijk.com/papers/maternal-mortality. This research was supported by the ERC StG "Relative Health: Long-Run Inequalities in Health and Survival Between Families and Across Generations" (grant agreement 101163144). I thank Nynke van den Boomen and Evelien Walhout for assistance with the data sources on maternal mortality levels in the Netherlands. All mistakes are, of course, mine. A final acknowledgement is for Angelique Janssens. As my PhD supervisor, she has been a source of inspiration and support, and I am indebted to her curiosity and broad scientific interest. Her passion for historical demography and the history of women's work, of households, improvements in mortality and health has been a great source of inspiration. Angelique, thank you for your guidance, encouragement and support over the years.

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APPENDIX

Figure A1 *Data selection*

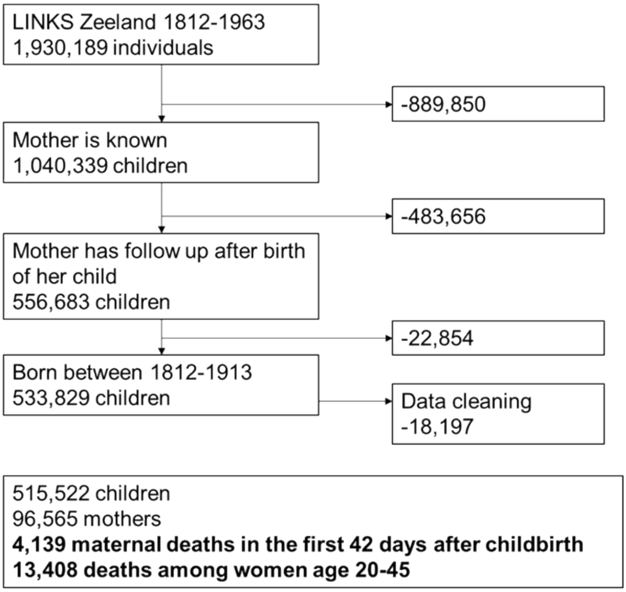
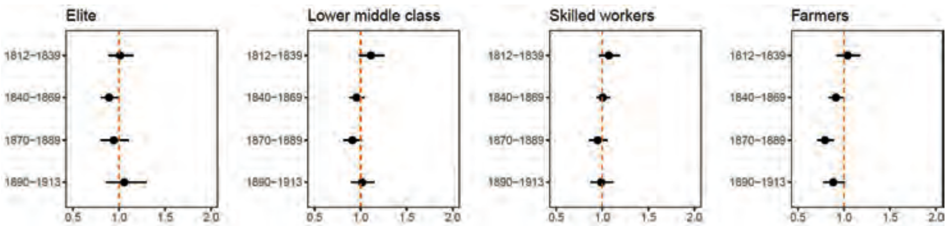


Figure A2 *Women's mortality between age 20 and 45, women with follow up observation, Zeeland*



Source: LINKS Zeeland. Reference group: Unskilled workers. Analyses are separately by period.

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A Detailed Individual-Level Analysis of Tuberculosis-Related Deaths Among Adults From Transylvania, 1850–1914

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ABSTRACT

In the late 19th and early 20th centuries, tuberculosis (TB) was a major public health issue across Eastern Europe, including Transylvania, then part of the Hungarian Kingdom. Nearly one million TB deaths were recorded in Hungary between 1901 and 1915, mostly among adults. This study examines TB mortality in Transylvania from 1850 to 1914 using data from the Historical Population Database of Transylvania, focusing on adults from the Greek-Catholic, Orthodox, Roman-Catholic, and Reformed (Calvinist) denominations. It explores how factors like environment, occupation, gender, age, and population movement influenced TB outcomes. Industrialization and population mobility, especially after 1881, increased the spread of TB across all denominations. Greek- and Roman-Catholics in open-type settlements had higher mortality, while Calvinists and Orthodox fared slightly better. Higher socio-economic status did not consistently protect against TB, revealing the central role of occupation and working conditions. Unlike many other studies, this analysis found that gender had minimal impact on TB deaths — likely because of women's active participation in agricultural labour in addition to their indoor responsibilities. The research shows that TB became more virulent approaching the 20th century and highlights the need for future studies incorporating urban areas and variables such as housing, nutrition, and healthcare to better understand the dynamics of this complex disease.

Keywords: Tuberculosis, Adults, Individual-level data, Transylvania, 19th Century

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1 INTRODUCTION

Tuberculosis (TB) remains a major global health challenge, closely linked to social and economic inequalities. While its burden is disproportionately felt in developing nations, wealthier and industrialized regions are by no means immune. This persistent presence of TB in modern public health echoes its long and complex history. Archaeological findings trace its origins to as early as the Neolithic period in both Eurasia and Africa. By the Middle Ages, TB had already left a visible mark on European populations. However, it was during the 18th and 19th centuries that TB evolved into a widespread epidemic. The movement of people, crowded living conditions, poor public health infrastructure, and growing industrial activity in many communities contributed to the rapid spread of the disease in countries such as England, France, Italy, and the United States, where it became endemic and had important demographic consequences. In England and Wales alone, TB accounted for about 13% of all deaths between 1851 and 1910. In 1871, one in every seven deaths in England was attributed to TB, while in cities like Stockholm, the disease claimed one in five lives between 1750 and 1830 (Johnston, 1993; Livi Bacci, 2003; Reeves, 2008). As such, the disease became a significant health issue, and the 19th century came to be regarded as "the age of tuberculosis" (Woods & Shelton, 1997).

In line with the complex nature of TB, many studies, particularly focused on the 19th century realities, have sought to address various aspects of the disease, including its demographic impact, dynamics of and influences on the living environment, gender differences, and the consequences of human mobility. While TB mostly affected young adults, the prevalence of the disease in urban settlements was often questioned, as both rural and urban living contexts faced their own distinct challenges (McNay et al., 2005; Woods & Shelton, 1997). When gender was considered as a variable, other studies indicated that the rural environment was, in many cases, detrimental to women, who were more susceptible to respiratory diseases, including TB, due to unequal access to resources and differences in work environments, with men generally working outdoors in the fields, and women predominantly indoors at home (Alter et al., 2004). For many women, particularly in rural areas, disparities in access to food and medical care increased their vulnerability to TB, especially during and after pregnancy, with this disease playing a significant role in maternal mortality (Janssens & van Dongen, 2018). Other studies show that mortality differences from TB were influenced by social and economic factors such as employment and migration patterns. As such, many women in urban areas contracted the disease while working in manufacturing factories, whereas men in rural areas were often exposed to TB through labour migration (Reid & Garrett, 2018). Other investigations indicated that gender differences in TB mortality were significant and varied across regions, highlighting the complex dynamics of the disease and underscoring the need for a deeper examination of underlying factors (Hinde, 2015), as well as an expanded focus on lesser-investigated regions.

2 THE IMPACT OF TB IN HUNGARY AND TRANSYLVANIA: A DEADLY EPIDEMIC AND PUBLIC HEALTH RESPONSE (19TH-20TH CENTURIES)

Beginning in the second half of the 19th century, tuberculosis spread more rapidly across many regions of Eastern Europe, developing into a serious epidemic by the end of the century (Johnston, 1993). This growing threat was also echoed in the Transylvanian press, where reports on TB often carried an alarming tone, emphasizing the vast number of lives it claimed. TB was described as "a cruel disease, which claims hundreds of thousands of souls every year, more merciless than cholera, typhus, and other illnesses" (Gazeta Transilvaniei, 1893). The disease was viewed as "the plague of the era, sparing no one, with each person mourning the loss of a loved one who has fallen victim to it" (Dopp, 1906).

Published statistics indicated a high mortality from tuberculosis in Hungary, of which Transylvania was a part until 1918. The capital, Budapest, was severely affected, and the newspapers reported that "year after year, thousands upon thousands of lives are extinguished by consumption in Hungary. In 1903 alone, 65,724 people died from this horrible disease. In July, 6,742 people perished from tuberculosis nationwide, with 313 deaths in Budapest" (Revaşul, 1905).

Tuberculosis (TB) is primarily transmitted through the air when an infected person coughs, sneezes, or speaks, with common symptoms including a persistent cough, chest pain, coughing up blood, fatigue, weight loss, fever, night sweats, and loss of appetite. Many of these transmission routes and symptoms were already recognized by doctors in Transylvania at the time, who also warned of the risks posed by contact with objects contaminated by the faeces, sweat, saliva, or sputum of infected individuals. Press reports offered detailed accounts of the disease's symptoms and progression, noting that TB "begins with small boils and night sweats, coughing, often accompanied by blood, and in some cases diarrhoea, as the disease and its microbes penetrate almost every organ" (Chitul, 1914).

As a result, newspapers across Transylvania recommended preventive measures such as disinfecting rooms by painting, ventilating living spaces, and washing hands before meals. One of the most frequently criticized habits was spitting on the ground, and many publications advised the use of spittoons or handkerchiefs, which were to be disinfected or treated with lye to curb the spread of the numerous microbes present in the sputum of infected individuals (Gazeta Transilvaniei, 1911).

Nevertheless, TB continued to pose a major problem, and during World War I, the Hungarian Central Statistical Office began publishing a report on the devastating impact of the disease, which was seen as a serious threat to population growth in Hungary. Covering the period from 1901 to 1915, the study revealed that 967,738 people had died from TB. Over these 15 years, tuberculosis accounted for 14.4% of all deaths nationwide, with rates reaching 20.3% in Budapest and 12.7% in Transylvania (Magyar Statisztikai Közlemények, 1925).

The report also highlighted significant regional differences in TB mortality, with notable variations between counties and settlements. In terms of gender differences, the report mentioned that TB was a significantly more common cause of death among women in Hungary. However, these differences varied considerably according to residence. Men in urban areas, especially those employed in the industrial sector, experienced a higher increase in mortality due to TB. In contrast, urban women benefited more from improvements in living conditions and health care, while rural women continued to be affected by poor socio-economic conditions. The report also drew attention to the high number of deaths among Roman-Catholics, Greek-Catholics, Orthodox, and Reformed communities, noting "a great fatality in tuberculosis mortality across the four denominations". Lastly, the report emphasized that TB primarily affected adults in "the most valuable age groups from the point of view of the economy and the maintenance of the species" (Magyar Statisztikai Közlemények, 1925).

Given these factors, this study aims to analyse the patterns and determinants of TB deaths among adults in Transylvania between 1850 and 1914, focusing specifically on differences across religious denominations (Greek-Catholic, Orthodox, Roman-Catholic, and Reformed). Situating Transylvania within the broader European context of the "age of tuberculosis" and engaging with existing debates in the literature, the analysis seeks to understand how social, economic, gendered, and environmental factors influenced the spread and impact of TB. Additionally, this research will expand our understanding not only of TB, but also of the Transylvanian context, which, in the 19th century was marked by confessional and ethnic heterogeneity, with a population that grew from 2,073,737 in 1850 to 2,098,507 by the 1910 census. Historically part of the Kingdom of Hungary and later incorporated into the Habsburg Empire, the region underwent gradual economic transitions from feudalism to market-oriented agriculture and early industrialization, particularly after 1881. Urbanization increased, with the rural population declining from 93.5% in 1850 to 87.6% by 1910 (Bolovan, 2000). This complex interplay of ethnic diversity, historical governance, and evolving economic conditions significantly influenced societal and demographic behaviours, including mortality patterns of TB during this period.

3 SOURCES, VARIABLES, AND METHODS

We used the Historical Population Database of Transylvania (HPDT), which contains information from the parish death registers of Orthodox, Greek-Catholic, Calvinist (Reformed), and Roman-Catholic communities across 23 localities in six counties (for details about this historical population database, see Dumănescu et al., 2022). In many instances, TB was inadequately recorded in death registers, particularly within the Orthodox community (Rus, 2023). Additionally, it appears that TB cases among children were frequently omitted, which justifies our focus on adult populations in this analysis.

The causes of death were standardized and coded according to the International Statistical Classification of Diseases and Related Health Problems, 10th Revision (ICD-10). Tuberculosis (TB) was recorded in Romanian, Hungarian, and Latin, and was often noted using colloquial expressions, archaisms, and regionalisms. As a result, it appeared in a variety of forms, such as *tuberculosa*, *gümőkór*, *phthisis universalis*, *hectica*, *oftica*, *morbu de plămâni*, and *boala uscată*. In most cases, these terms referred to respiratory tuberculosis, although in some instances they described tuberculosis affecting other organs, including *béltuberkulózis* (tuberculosis of the intestines), *gége tuberkulózis* (tuberculosis of the larynx), *tuberculosis visceris* (tuberculosis of internal organs), and *meningitis basilaris tuberculosa* (tuberculous meningitis).

We employed binary logistic regression models, aiming to capture the impact of various factors on the likelihood for a death during 1850–1914 to be a death caused by TB. The dependent variable of our logit model was 'cause of death', having value 1 for a 'TB death' and 0 for a 'death from any other cause'. Given the observed omissions in assigning TB as cause of death for children, we focused exclusively on deaths above age 14.

We differentiated the analysis by denomination, selecting the four predominant religions in Transylvania at that time. This approach also considered the unique characteristics of each death register and its specific way of recording information. Consequently, we employed four binary logistic regression models, with a working sample that included 15,655 deaths (cases with valid information for all the variables used). The independent variables used in the models are presented in Table 1, along with their manner of construction.

Table 1 *Independent variables and their construction*

Variable	**Categories**	**Notes**
Socio-economic status	1. Upper/middle class 2. Agriculturists 3. Skilled workers 4. Semi-skilled workers 5. Unskilled workers 6. Unknown	Based on occupations coded using HISCO and SOCPO. If not available, spouse's occupation used (for women).
Denomination	1. Greek-Catholic 2. Orthodox 3. Calvinist (Reformed) 4. Roman-Catholic	Based on entries by priests or cover of parish registers.
Marital status	1. Unmarried 2. Married 3. Previously married (divorced/widowed) 4. Missing/unknown	-
Gender	1. Male 2. Female	-
Age	Six 10-year groups	A decadal grouping (14–24, 25–34, 35–44 etc.).
Locality type	1. Open (7 localities) 2. Peripheral (16 localities)	Based on economic features, market access, and infrastructure. Open includes industrialized or well-connected localities; peripheral includes isolated/agricultural localities.
Time period	1. 1850–1880 2. 1881–1914	Divided by the 1881 Law 44 industrial development milestone.

Notes: For information regarding the HISCO and SOCPO schemes, see Van De Putte & Miles (2005). For more information on Law 44/1881, see Nagy (2011). The localities included in the analysis were: Ocna Mureş, Ocna Dej, Gurghiu, Glăjărie, Caşva, Uioara de Jos, Războieni, Hodac, Orşova, Solovăstru, Spini, Bucium Cerbu, Muntele Rece, Nima, Călăraşi, Decea, Lunca Mureşului, Chileni, Voşlăbeni, Ibăneşti, Moldoveneşti, Rusu Bârgăului, and Liviu Rebreanu.

4 RESULTS

The descriptive results of the analysis indicated that between 1850 and 1914, TB caused the deaths of 10% of adults in Transylvania. The disease primarily affected young and middle-aged adults, with a lesser impact on older individuals. In the later period, 1881–1914, there was a slight increase in deaths due to TB. These were lower in peripheral localities, suggesting that environmental factors and working conditions played a role. The disease was more prevalent among semi-skilled and skilled workers, as well as members of the upper-middle class. Interestingly, TB deaths were less common among Orthodox communities, which might suggest some level of protection, or differences in the accuracy of registering causes of death. Men and women appear to have been affected in the same way (see Table 2).

Subsequently, the results of the multivariate analysis indicate that the period between 1881 and 1914, marked by accelerated industrialization in Transylvania, was unfavourable in terms of the risk of dying from TB. The sharpest effects were found in the case of Roman-Catholics, who were most likely to lose their lives to this disease (Table 3).

Table 2 *Distribution of deaths by different characteristics*

	Tuberculosis		Other	
Male	770	10%	7,050	90%
Female	803	10%	7,030	90%
Greek-Catholic	642	9%	6,440	91%
Orthodox	145	3%	4,024	97%
Roman-Catholic	179	13%	1,239	87%
Reformed	596	20%	2,329	80%
Other	11	18%	50	82%
Open	1,006	13%	6,493	87%
Peripheral	567	7%	7,589	93%
Unskilled workers	109	10%	1,110	90%
Semi-skilled workers	193	16%	1,201	84%
Skilled workers	108	21%	523	79%
Agriculturists	300	10%	3,119	90%
Upper-middle	91	15%	622	85%
Unknown	772	9%	9,080	91%
1850–1880	646	8%	7,328	92%
1881–1914	927	12%	6,754	88%
14–24 years	316	15%	1,851	85%
25–34 years	305	16%	1,617	84%
35–44 years	269	13%	1,764	87%
45–54 years	304	13%	2,126	87%
55–64 years	231	9%	2,394	91%
65+	148	3%	4,330	97%
Total	1,573		14,082	

Source: HPDT (authors' calculation).

Table 3 *Results of the multivariate analysis, adults*

Adults (14+)		Greek-Catholic			Orthodox			Roman-Catholic			Reformed		
		Cases	Exp(B)	Sig.	Cases	Exp(B)	Sig.	Cases	Exp(B)	Sig.	Cases	Exp(B)	Sig.
Social status	Unskilled workers	243	1		254	1		330	1		275	1	
	Semi-skilled workers	682	2.352	***	88	1.497		69	1.660		349	1.540	**
	Skilled workers	89	2.676	**	22	2.811		169	1.402		234	1.670	**
	Agriculturists	1,564	1.839	**	897	1.986	*	284	1.086		373	1.613	**
	Upper-middle	180	2.437	**	46	4.598	***	154	0.876		237	1.299	
	Unknown	4,323	1.760	*	2,861	0.644		412	1.226		1,457	1.222	
Time period	1850–1880	3,830	1		1,954	1		683	1		1,494	1	
	1881–1914	3,251	1.763	***	2,214	1.635	**	735	7.457	***	1,431	1.271	**
Age group	14–24 years	1,013	1		568	1		172	1		406	1	
	25–34 years	856	1.361	**	507	0.916		164	0.639		390	1.310	
	35–44 years	964	1.134		515	0.668		187	0.743		361	0.930	
	45–54 years	1,142	0.993		596	0.674		231	0.644		437	0.984	
	55–64 years	1,217	0.763		688	0.321	***	251	0.359	***	463	0.575	***
	65+	1,889	0.282	***	1,294	0.053	***	413	0.073	***	868	0.206	***
Locality type	Open	3,904	2.197	***	509	0.055	***	1,015	2.947	***	2,017	0.798	**
	Peripheral	3,177	1		3,659	1		403	1		908	1	
Gender	Male	3,521	1		2,109	1		717	1		1,436	1	
	Female	3,560	1.147		2,059	0.748		701	0.856		1,489	1.162	
Marital status	Never married	1,062	1		583	1		166	1		291	1	
	Previously married (divorced, widowed)	1,545	0.727	*	751	0.877		289	1.330		651	0.937	
	Married	3,917	0.753	**	2,738	1.078		823	1.331		1,269	0.882	
	Missing, unknown	557	0.736		96	2.623	**	140	1.206		714	0.685	*
Nagelkerke R Square			0.086			0.166			0.249			0.110	

Source: HPDT (authors' calculation).

*Note: * for $p < 0.1$, ** for $p < 0.05$, *** for $p < 0.01$.*

After 1881, Transylvania entered into a more accelerated phase of industrialization, during which industrial development and related occupations were regarded as major factors in the spread of tuberculosis (Magyar Statisztikai Közlemények, 1925). Besides the impact of industrialization, after 1881, the population in Transylvania underwent an increased process of mobility, which also contributed to the spread of TB. The periodical *Telegraful român* highlighted this issue in 1900, quoting the chief county physician's observations "tuberculosis spares neither palaces nor huts, whether they are in the mountains or the lowlands. As soon as commerce and communication lead to the growth of a locality, and people congregate there, all the ills of humanity inevitably follow" (Telegraful român, 1900).

In searching for the reasons behind the greater impact of TB on Roman-Catholic adherents, the mobility of people should certainly be taken into account. Additionally, the settlements of Gurghiu and Ocna Dej, classified as open localities and hosting a significant number of Roman-Catholics, experienced major developments in transportation during this period.

Gurghiu, a key economic hub in the Gurghiu Valley, was notable for hosting an important weekly fair (Irimescu-Andruș, 1982–1983). In 1898, the construction of a narrow-gauge railway line between Reghin and Lăpușna started. This 37 km stretch of railway, completed in 1905, was primarily used for forestry operations along the Reghin-Gurghiu-Lăpușna route (Rus, 2023). Between 1881 and 1914, Gurghiu attracted many Roman-Catholic settlers, including individuals from distant Transylvanian localities such as Remetea (Harghita), Acățari, and Iernut (Mureș), as well as from cities in the former Austro-Hungarian Monarchy, such as Tompa (Hungary) and Detva (Slovakia).

A significant number of Roman-Catholics also lived in Ocna Dej, an old salt-mining locality. Modernization here began in 1910, but starting in 1882 some improvements were made in salt transport with the inauguration of the Cluj-Apahida-Dej-Jibou railway line, which was later extended to the salt mine. As such, the improvements in transportation and the movement of people created opportunities for the spread of TB and the subsequent deaths of many, a significant proportion of whom were Roman-Catholics. These patterns observed among Roman-Catholics in the Gurghiu and Ocna Dej localities were similar to situations in other regions around the world, where trade and migration facilitated the spread of TB. Movement plays a significant role in the transmission of infectious diseases like TB, as people arriving from areas with high infection rates can introduce new cases into their new communities. Indeed, the chronic nature of tuberculosis often allows individuals to travel and relocate before the disease becomes debilitating (Johnston, 1993).

Living in open localities doubled the odds for a TB death in case of Greek- and Roman-Catholics, while the effect was lower in case of Orthodox and (Calvinist) Reformed individuals. The report on tuberculosis mortality in Hungary from 1901 to 1915 noted that individuals in peripheral, predominantly rural areas were more protected from this pathogen (Magyar Statisztikai Közlemények, 1925). The same study highlighted that social conditions in these rural areas were generally more favourable, providing greater security for individuals' physical health. Additionally, the natural environment in rural areas contributed to lower tuberculosis incidence, explaining why the disease was less prevalent in these settings (Magyar Statisztikai Közlemények, 1925).

On the other hand, living in open localities had negative effects only for Greek-Catholics and Roman-Catholics, but not for Orthodox and Calvinist adults. A key factor contributing to the lower number of deaths due to TB among the latter two groups was the social benefits provided to workers and their families in the railway sector, where many Orthodox and Calvinist residents of Războieni were employed. These benefits, including healthcare and housing, were offered by the Hungarian state, likely helping reduce TB incidence in these denominational groups. This trend was also observed across Hungary, as noted in the state report on TB mortality from 1901 to 1915. The report highlighted that railway workers experienced lower TB mortality due to the medical and social advantages provided by the state (Magyar Statisztikai Közlemények, 1925). While studies generally suggest that employment in certain sectors increases the risk of exposure and dying due to the TB bacteria (Reid & Garrett, 2018), the case of the Orthodox and Calvinist denominations in Războieni show that, in this instance, employment came with benefits that helped reduce TB risk.

The results show that higher socio-economic status did not offer protection against TB deaths, as deaths caused by this disease were more likely among individuals from the upper-middle class, agriculturists, and skilled and semi-skilled workers than among labourers. This situation was observed for Greek-Catholic, Orthodox, and Reformed individuals, but not for Roman-Catholics. Given that the first three groups included large numbers of persons with unknown occupations, the results should be interpreted with caution.

TB in Transylvania did not discriminate according to socio-economic status, and being situated at the top of the social ladder did not offer protection against it. This was also confirmed by the survey on tuberculosis deaths in Hungary from 1901 to 1915, which highlighted the prevalence of deaths among clerks, including accountants, tax collectors, office clerks, and weighing clerks. Despite being regarded as "the middle class of the country and the backbone of society", and possessing significant intellectual resources, this group still experienced considerable losses. The survey emphasized that while intelligence, education, and enlightenment were crucial in combating tuberculosis, and this occupational group was well equipped with all these attributes, the widespread impact of the disease among them indicated serious deficiencies in their social conditions and living standards, particularly for public service employees (Magyar Statisztikai Közlemények, 1925). An elevated mortality rate due to TB was also observed among clerks and white-collar workers in Scotland, who mainly performed sedentary office jobs in crowded and poorly ventilated spaces (Reid & Garrett, 2018).

The upper-middle-class Greek-Catholics and Orthodox in Transylvania were primarily composed of teachers and priests, who were especially vulnerable due to their frequent interactions with others and their work in crowded, poorly ventilated spaces during teaching activities and religious services. Press reports during that time noted that members of the teaching profession were especially vulnerable to respiratory illnesses such as "hoarseness, angina, weakness of the lungs, and even TB" (Elefterescu, 1894). Similarly, priests were at risk of "pulmonary inflammations and other conditions that could rapidly lead to death" (Cionca, 1905).

In the case of the Greek-Catholic adults, town accountants, tax collectors, clerks, shoemakers, hairdressers, barbers, painters, and salt mine workers, classified as in the upper-middle class, agriculturist, skilled, and semi-skilled categories, faced a higher risk of dying from tuberculosis. This reinforced the idea that in many instances tuberculosis spared no one, regardless of social position, power, or wealth (Telegraful român, 1910).

On the other hand, the category of unskilled workers mainly included day labourers and factory workers. Day labourers typically depended on agriculture for their livelihood and resided in areas with an agro-industrial profile, such as Ocna Dej. In Hungary, there were industrial localities where people combined industrial work with agriculture. Many day labourers, who maintained an agrarian background, benefited from working outdoors, as it allowed for more exposure to fresh air. Furthermore, in various parts of Hungary, factory working conditions — regarding space, lighting, and health facilities — were often superior to those in small workshops (Magyar Statisztikai Közlemények, 1925). A notable example is the soda factory in Ocna Mureş, which, funded by Belgian and Austrian capital, began operations in 1896 and employed 148 workers by 1900 (Holom et al., 2018). While many factories, particularly those involved in textiles and mining, were overcrowded and exposed workers to harmful dust and irritants that compromised respiratory health and increased susceptibility to infections, including TB (Hinde, 2015; Reid & Garrett, 2018), there were cases where improvements in factory working conditions had positive effects in reducing TB risk.

In terms of age, the results show that older age groups (55+ and 65+) were more likely to experience a TB death than younger ones across all denominations, with the exception of Greek-Catholics, where age group 25–34 was also more likely to experience a TB death. Our results are consistent with patterns observed in other populations, where young adults up to the age of 34 were the most impacted (Reid & Garrett, 2018; Woods & Shelton, 1997). Advancing age reduced the odds of death from TB across all religious denominations analysed in Transylvania, a trend similar to that observed across the entire Hungarian Kingdom. Between 1901 and 1915, it was noted that after the age of 60 the impact of disease began to weaken, as numerous other natural causes of death emerged, reducing the significance of TB (Magyar Statisztikai Közlemények, 1925).

The results on marital status show that being involved in a marital relationship, even a previous one, seems to have mattered only in the case of Greek-Catholics. For other denominations, such differences were not observed. The Hungarian authorities who conducted research on TB stated that it was not marital status that mattered, but rather youth, vitality, and a body and mind unaffected by work and other fatigues, which were important factors influencing TB mortality (Magyar Statisztikai Közlemények, 1925).

Regarding gender, some research shows that TB generally caused more deaths among women, particularly in rural settings (Hinde, 2015; Johnston, 1993; Woods & Shelton, 1997, p. 97), due to unequal access to food, resources, and healthcare, reduced resistance to infection during childbearing years, and greater exposure to indoor pathogens through performed domestic activities (Alter et al., 2004; Janssens & van Dongen, 2018). However, our results show no significant difference by gender. We believe that the socio-professional structure of Transylvania, and particularly of the localities in our sample, that can be described as a transitional (agro-industrial), wherein many people continued to earn a living from agriculture (including many females), contributed to this similarity between genders. Although Transylvania was undergoing a process of industrialization and urbanization, these developments were still in their early stages. In 1910, 73.3% of the population continued to work in agriculture, and 87.6% lived in rural areas.

4 CONCLUSIONS

Just as the 19th century came to be known as "the age of tuberculosis" in many parts of the world, this characterization was equally applicable to Eastern Europe, particularly during its latter half. In Hungary, of which Transylvania was a part until 1918, TB became a serious issue, prompting authorities to intensify their scrutiny of the disease's impact. A report on tuberculosis-related deaths from 1901 to 1915 documented nearly one million fatalities, emphasizing that adults were particularly vulnerable, with notable variations across regions and socio-demographic groups.

Our analysis was focused on the lesser-known realities of adult TB deaths in Transylvania between 1850 and 1914, while engaging with existing debates in the literature on the complex nature of the disease and emphasizing the importance of factors such as environment, age, gender, occupation, and population movement, alongside the specific characteristics of the province and localities studied.

Using data from the Historical Population Database of Transylvania, this investigation focused on the four major denominations in the province: Greek-Catholic, Orthodox, Roman-Catholic, and Calvinist (Reformed). It revealed both similarities and differences among these groups, shaped by the environments in which they lived as well as by the social and economic factors characteristic of the period under study. During the phase of accelerated industrialization between 1881 and 1914, all denominational groups experienced an increased risk of tuberculosis-related death. Population mobility and industrial development further facilitated the spread of *Mycobacterium tuberculosis*, significantly impacting community health. This situation was also seen in other European contexts, such as in Hungary, England, and Scotland, suggesting that industrial growth universally heightened TB risks, however TB risk also operated through locally specific socio-economic and demographic pathways.

In open-type localities, Greek-Catholics and Roman-Catholics had a higher likelihood of dying from tuberculosis, while Calvinists (Reformed) and Orthodox individuals in the same areas faced lower risks. This reconfirms the crucial role played by occupations in understanding the historical geography of TB (Hinde, 2015; Reid & Garrett, 2018). Similar to realities in Hungary and Scotland, higher socio-economic status did not offer consistent protection against TB, underscoring again the complex interplay between occupation, working conditions, and disease vulnerability (Magyar Statisztikai Közlemények, 1925; Reid & Garrett, 2018).

An interesting finding that contrasts with much of the existing literature (Hinde, 2015; Johnston, 1993; Woods & Shelton, 1997, p. 97) is that gender did not have a significant impact on TB deaths in this sample. This is likely due to the agro-industrial structure of the province and localities studied, where women continued to play an active role in agricultural labour in addition to their typical indoor activities.

Our data confirmed that tuberculosis became progressively more virulent in Transylvania as the 20th century approached, affecting people across all denominations. While the factors we analysed help explain some of the dynamics of this complex disease, there is still a need to incorporate data from other localities, particularly urban areas, and to extend the observation period to include the interwar years to better understand when we can identify a decline in TB-related deaths. Additionally, variables such as housing conditions, nutrition, and access to healthcare for people from Transylvania's past should also be considered in future research endeavours.

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The Role of Occupations in the Decline of Pulmonary Tuberculosis

Insights From Amsterdam's Jewish Neighbourhoods, 1856–1909

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ABSTRACT

The decline of pulmonary tuberculosis in the second half of the 19th century was instrumental in shaping long-term shifts in historical mortality patterns and life expectancy. Despite its significance, the underlying determinants of this decline remain a subject of ongoing debate. This study contributes to the discussions about the role of standards of living by examining the impact of occupation on the decline of pulmonary tuberculosis mortality in Amsterdam between 1856 and 1909. It does so through the lens of the city's Jewish neighbourhoods, that, despite facing poverty and overcrowding, exhibited substantially lower tuberculosis mortality rates than the rest of the city, but also experienced a slower decline over time. Using individual-level mortality data from the Amsterdam Causes-of-Death Database and occupational data from marriage certificates, we analyse how shifts in occupational structures following industrialization influenced these trends. Our findings highlight the significant role of labour conditions in shaping historical health disparities and suggest that work environments, alongside nutrition and public health measures, played a pivotal role in shaping and exacerbating intra-urban health disparities.

Keywords: Tuberculosis, Amsterdam, Jewish, Mortality decline, Historical occupation data

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1 INTRODUCTION

Amsterdam underwent a remarkable transformation during the second half of the 19th century. The capital's population doubled, its stagnating economy rebounded, industrialisation went full steam ahead, and mortality dropped dramatically. Economic changes caused overall wellbeing to rise dramatically, reflected in indicators such as stature (Tassenaar, 2019) and real wages (de Zwart, 2023; van Zanden & van Riel, 2004), which rose more sharply in Amsterdam than in the rest of the country. Concurrently, the disease burden of pulmonary tuberculosis and other nutrition-related diseases started falling (Jansen & de Meere, 1982; Lammertink, 2023). Since pulmonary tuberculosis constituted the main cause of death among the adult population (Hardy, 1993), its decline had a significant impact on overall mortality patterns, not only in the Netherlands but across the Western hemisphere (Condran & Cheney, 1982; Mercer, 2014; Puranen, 1991; Rothman, 1994; Zürcher et al., 2016). This trend is particularly striking given that extensive public health interventions — let alone the discovery of truly effective treatments — would not emerge until after the turn of the century, by which time the decline in tuberculosis was already well underway (Hueting & Dressing, 1993).

While this "early decline" in pulmonary tuberculosis mortality has sparked considerable debate, its determinants remain poorly understood. Since the introduction of McKeown's controversial nutritional thesis in 1976, discussions have primarily focused on whether living standards, particularly in terms of diet, played a more significant role in the reduction (or absence thereof) of tuberculosis mortality, or whether medical and public health interventions were the key factors instead (Mackenbach, 2020; Szreter, 1988). More recently, attention has been given to the influence of a combination of factors, including better nutrition, as well as other aspects relating to standards of living such as occupational conditions (Devos, 1996; Greenlees, 2005; Reid & Garrett, 2018). Workplace environments provided plausible settings for increased exposure and maximised opportunities for transmission, particularly if they were crowded and when high levels of dust and humidity provided additional risk factors (Murkens, 2023; van Rossem et al., 2017).

In this study, we shed further light on the possible contribution of occupational composition and labour conditions to patterns of mortality from pulmonary tuberculosis through the lens of Amsterdam's Jewish neighbourhoods between 1856 and 1909. Amsterdam witnessed a substantial reduction in tuberculosis mortality during this period, but this decline did not occur uniformly across the population and was instead marked by significant socioeconomic differences between poorer and wealthier neighbourhoods (Muurling et al., 2026). Moreover, notable variation was observed between the small but important Jewish enclave, comprising roughly 10% of the city's population, and Gentiles: (i) pulmonary tuberculosis rates in Jewish neighbourhoods were much lower but also (ii) saw a slower decline when tuberculosis mortality dropped rapidly among other social groups in Amsterdam. To what extent can the divergent occupational structures of Amsterdam's Jews help explain these peculiar characteristics of their pulmonary tuberculosis mortality? And what are the implications for our understanding of declines in tuberculosis mortality more broadly speaking?

To scrutinise the roles of occupational exposure in shaping observed pulmonary tuberculosis mortality patterns, we will first discuss how, from a historiographical perspective, the case-study of the Amsterdam Jews complicates any attempts to establish a singular explanation for the decline in tuberculosis mortality and a need to consider factors beyond nutrition. Following a section about the sources and methods that we employ, we examine the developments in tuberculosis mortality rates and explore its possible connections with the occupational structures of Jews and Gentiles during the 19th and early 20th centuries. By doing so, we provide further evidence for the role of work environments in shaping the course of the historical decline of pulmonary tuberculosis.

2 TUBERCULOSIS MORTALITY IN AMSTERDAM

In the 19th century, tuberculosis was the main cause of death for the European workforce. Nearly half of all young adult deaths were attributable to tuberculosis. Despite this numeric importance, the historical decline in tuberculosis mortality from the second half of the 19th century onwards remains poorly understood. Most notedly, McKeown (1976) used the process of elimination to contend that falling tuberculosis mortality was explained by increasing wellbeing and improved nutrition. He claimed that the drop occurred prior to the introduction of vaccinations and other preventive measures. Others have argued for a greater importance of investments in public infrastructure (Mitchell, 1990; Szreter, 1988), the isolation of sick individuals (Fairchild & Oppenheimer, 1998; Wilson, 1990), or a conscious effort to reduce overcrowding (McFarlane, 1989; Zürcher et al., 2016).

In Amsterdam tuberculosis mortality started falling in the final three decades of the 19th century. In line with McKeown's reasoning, tuberculosis declined prior to key medical advancements such as the BCG-vaccination, preventative institutions like sanitoriums, or societies aimed at fighting tuberculosis established after the turn of the century (van der Korst, 1988). In fact, contemporary medical professionals in the Netherlands were still unsure whether tuberculosis was communicable or not in the 1870s (Betz, 1878). At the time tuberculosis mortality started falling, population densities in Amsterdam were on the rise. Since 1870, Amsterdam was characterised by a rapid inflow of domestic immigrants attracted by economic recovery. With delays in the expansion of the city's housing stock, the number of families and persons per house increased significantlyin Amsterdam's inner city between 1869 and 1899 (Laloli, 2007). Declines in overcrowding can therefore hardly explain the fall in tuberculosis mortality in Amsterdam. Meanwhile, the decline of tuberculosis occurred concurrently with significant improvements in wages and male stature (de Zwart, 2023; Tassenaar, 2019), common proxies for nutrition. However, tuberculosis mortality started falling earlier and faster for women, raising doubts about the universality of improving nutrition (Muurling et al., 2026).

An additional counterpoint against nutrition is offered by Amsterdam's Jewish community. As was the case in other cities, Amsterdam Jews had lower mortality rates than non-Jewish Amsterdam residents. This was true for virtually all age groups and for a wide range of diseases (Riswick et al., 2022) including tuberculosis (Boekman, 1936; Sanders, 1918), and was already observed by 19th-century contemporaries (Coronel, 1864a). Although it has been clear that fewer Jews died from tuberculosis — as well as from other causes — throughout 19th-century Europe and the United States (Sawchuk & Herring, 1984), a clear consensus regarding the determinants remains absent. It also appears paradoxical given that Jews in mid-19th-century Amsterdam were generally poorer than the average Amsterdam resident (Wallet, 2017), experienced higher population densities, and worse housing conditions — all believed to worsen tuberculosis mortality. Jews' below-average economic standing problematises attributing their health advantages related to tuberculosis to better nutrition (see also Muurling & Ekamper, 2024, p. 20). Several other commonly cited potential explanations for the overall lower mortality rates among Jews, such as differences in breastfeeding patterns (Israëls, 1862), specific hygienic rituals (Pinkhof, 1908), and more trust and use of medical professionals (Blom & Cahen, 2017; Zaidman-Mauer, 2022), do not seem plausible in the context of pulmonary tuberculosis. Meanwhile, less alcohol abuse (Stephan, 1904) and particularly an occupational structure with less health-affecting careers (Sawchuk et al., 2013) are more worthy of consideration.

Most of the aforementioned factors, including nutritional intake, also fall short when we try to explain the much slower reduction in tuberculosis mortality among Jews. Between 1870 and 1920, the time during which tuberculosis mortality fell the hardest, Jews' gains in socioeconomic resources were larger than among other social groups in the city (Kok, 2025a). They also experienced the starkest improvements in their living conditions, moving from the cramped Jewish Quarter, with the highest population densities in the Dutch capital, to more sanitary newly-built neighbourhoods in Amsterdam East and South (Tammes, 2011). We therefore need to find another factor to explain Jewish-Gentile differences in tuberculosis mortality. To identify this reason, we will focus on conditions of and developments in occupational structures.

3 DATA AND METHODS

To investigate whether occupational structures might have impacted tuberculosis mortality rates we use detailed information on occupations and deaths in Amsterdam. Mortality data for the years 1856–1909 is derived from the Amsterdam Causes-of-death Database (ACD) (Janssens et al., 2023) based on the cause-of-death registers initiated by the city authorities in the early 1850s (Neurdenburg, 1929). In addition to the cause of death, the registers record the residential address of the deceased, the date of death, age, sex, marital status and occupation. The causes of death are coded according to the ICD10h; a joint coding scheme constructed by the SHiP+ network to study mortality dynamics using individual cause-of-death-data across Europe between 1850 and 1950 in a systematic way (Janssens, 2021; Reid et al. 2024). We focus on respiratory tuberculosis (all codes starting with A16 in the ICD10h). Since the ACD has no direct measure to identify Jews and Gentiles, we use a neighbourhood proxy instead. All deaths occurring to individuals who lived in the Jewish Quarter — comprising the old Jewish Quarter with districts C, P, Q, R, and S, as well as newer districts V and W — are considered Jewish (Bregstein & Bloemgarten, 1978; Kok, 2025a).[1] To standardise comparisons across time, we limit the comparison group to the remaining 43 districts of inner-city Amsterdam, that is, all districts that existed in Amsterdam in 1850 and excluding the city's expansions since 1896. While the mortality rates are calculated based on the total male neighbourhood populations, our further analyses are based on a sample limited to adult men. Roughly 80% of men between the ages of 20 and 60 were recorded with an occupation in the ACD. The high frequency of listed occupations and constancy across age groups and time suggests that the undercounting of male occupations was negligible.

To measure changes in the male occupational structure in Amsterdam, we turn to the LINKS database containing all marriage certificates in Amsterdam from 1811 up to 1932 (Mandemakers et al., 2023). Following Kok (2025b), we adopt a name-based approach to identify Jewish and non-Jewish families on the certificates. This enables us to compare the differential trends in occupational changes between Jewish and Gentile men. To limit second-order marriages, we limit the sample to all men who married between the ages of 18 and 40. We also filter out marriages before 1830 due to greater frequencies of misleading occupational titles. Note that the marriage certificates do not directly tell us the relative size of an industry, but offer a proxy for the relative attractiveness of sectors for young entrants.

In both the ACD and the marriage certificates, each occupational title is given a HISCO-code (van Leeuwen et al., 2002). Additionally, we code each occupation with whether it was performed indoors or outdoors following Rijpma and colleagues (2022). We make this distinction between indoor and outdoor occupations due to assumptions in scholarly literature about its potential effects on exposure and transmission, as well as the mentions of the breathing in of high levels of dust particles in enclosed environments as an important additional risk factor (Greenlees, 2005; Murkens, 2023; Reid & Garrett, 2018).

4 THE LOW BURDEN OF TUBERCULOSIS MORTALITY

For a long time, the endemic nature of tuberculosis did not cause vast, occasional mortality spikes, but instead resulted in a steady death toll that for a long time seemingly raised little public health concern. The development of pulmonary tuberculosis mortality in Amsterdam in census years is presented in Figure 1. It demonstrates the high mortality rates discussed earlier, as well as its decline over time for Amsterdam's entire population. Moreover, the figure reveals the stark differences between the mortality rates from pulmonary tuberculosis in the Jewish Quarter relative to the rest of Amsterdam's historical inner city.[2] First, it shows the lower levels of tuberculosis mortality rates in the Jewish Quarter throughout the second half of 19th century. Especially prior to the 1870s, mortality rates for men in Jewish neighbourhoods were substantially reduced compared to their Gentile counterparts. The dip in 1869 can likely be explained by excess mortality during the 1866 cholera epidemic. Second, it makes apparent that the Jewish neighbourhoods experienced a less steep decline during and following industrialisation. Thus, while Jews died less frequently from tuberculosis in mid-19th-century Amsterdam, their advantage dissipated in the final decades of the century.

There are good reasons to believe that tuberculosis mortality is associated with a locality's occupational distribution. Both industrial production and agricultural work are believed to be strongly correlated to tuberculosis mortality (Devos, 1996; Janssens, 2016; Janssens & van Dongen, 2018; Murkens, 2023; Reid & Garrett, 2018). For men, several occupational groups have been linked to high rates of tuberculosis mortality (Greenlees, 2005). These occupations required workers to spend prolonged periods of time

1 This is the area that in the 19th century was considered 'Jewish' (Bregstein & Bloemgarten, 1978). A discussion of changing Jewish settlement patterns in the late 19th and early 20th century can be found in Chapter 7 of Kok's dissertation (Kok, 2025a).

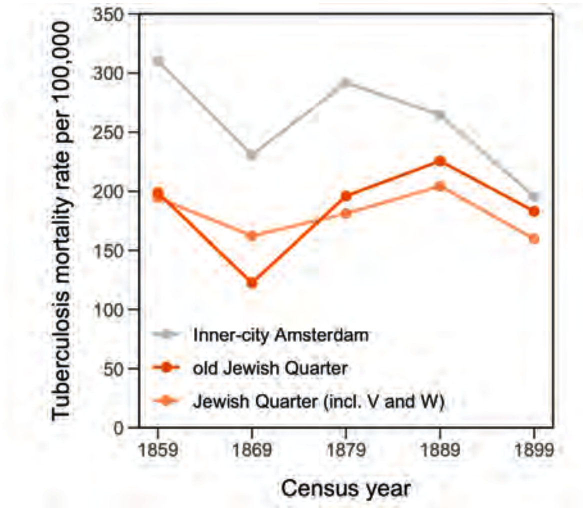
2 Since neighbourhoods V and W had a distinct age structure owing to the presence of multiple *oude-liedenhuizen* (old people's homes), we have chosen to show the Jewish Quarter with and without these neighbourhoods separately.

in areas with increased particle densities in the air and poor ventilation. Think, for instance, of textile workers, but also miners, those involved in leather and tobacco production, ceramic workers, stone masons, metal workers, diamond workers, and typesetters (Bavinck, 1897). In short, industrial labour performed indoors with high densities and lacking air quality. The workplace environment may have also been important among men working in clerical occupations, probably as a result of a combination of increased exposure and greater opportunities for transmission (McIvor, 2012).

Jews in Amsterdam were characterised by an occupational profile that was distinctly different from their Gentile peers. Eighteenth-century guild interference excluded Jews from the guild system and, in turn, closed possibilities to learn skilled trades (Lucassen, 1994). Consequently, few Jews worked in industry even after the guild system was abolished at the end of the 18th century. Important exceptions included the occupational niches in the diamond industry, where both employers and employees were virtually all Jewish by the mid-19th century (Heertje 1936), and a strong Jewish presence in the tobacco industry (Knotter, 1991). In contrast, Jews were heavily overrepresented in all strata of trade, including as peddlers, and frequently employed as unskilled labourers (Tammes, 2012), primarily in the transportation of goods (Kok, 2025a). Until Amsterdam started industrialising in the 1870s, causing the occupational structure of Jews to transform dramatically, Jewish men's occupations can be characterised as generally favourable compared to the rest of the Amsterdam population in terms of tuberculosis mortality hazards.

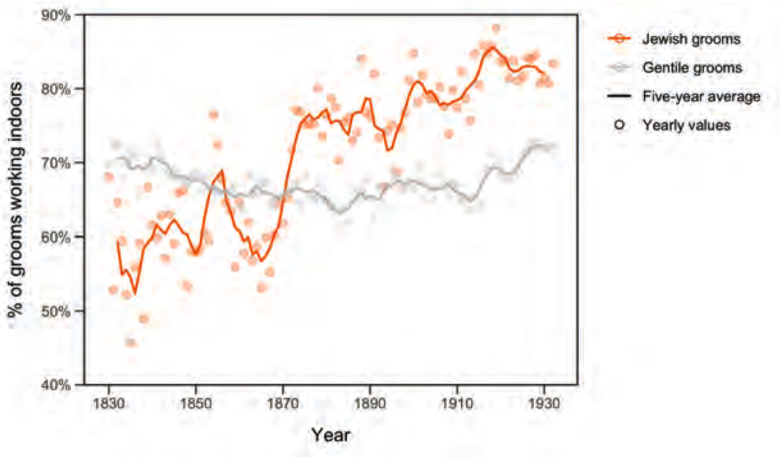
The marital certificates of men who married between the ages of 18 and 40 support this characterisation of the Jewish occupational structure as comparably favourable. As Figure 2 shows, Jews more frequently worked outdoors compared to their non-Jewish counterparts up to 1870. This is explained by much higher rates of unskilled labour, the primary group that worked outside. Moreover, within the category of unskilled labourers, Jews were also more likely to work outside. Unskilled Jews most frequently worked as peddlers, porters, and carters, all occupations that took place in the open air. Between 1850 and 1870, less than 4% of Jewish grooms with unskilled occupations worked in occupational groups that generally laboured inside, compared with 36% of Gentile workers. While these percentages grew more similar during industrialisation, it showcases the potential for better health in the Jewish area due to working in the open air (Stokvis, 1869).

Figure 1 *Total male tuberculosis mortality in the Jewish Quarter and the rest of Amsterdam, 1859–1899*



Note: Since ACD data is incomplete for 1899, deaths are taken from 1898. Mortality rates are lower in 1869 presumably due to an aftershock from excess cholera mortality in 1866. The mortality rates for inner-city Amsterdam exclude the Jewish Quarters.

Figure 2 *Share of Jewish and Gentile grooms working in 'Indoor' occupational groups, Amsterdam 1830–1932*



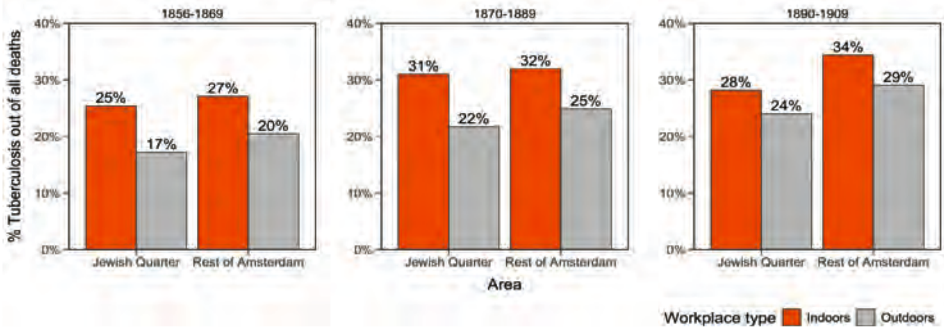
5 THE SLOWER DECLINE OF TUBERCULOSIS MORTALITY

Jews' occupational structure changed completely during Amsterdam's industrialisation. Key in this transformation is the rapid expansion of the diamond industry. Between 1870 and 1890, the diamond industry expanded from 1,500 to 10,000 workers, nearly three-quarters of whom were Jewish (van Tijn, 1976). By the early 20th century, roughly 30% of all working Jewish men were employed in the diamond industry, compared with 2% of non-Jewish men (van Zanten, 1926). Jews who would a decade earlier have started their working lives as carters or porters in the open air now started as diamond polishers and setters in congested factories with subpar ventilation and filled with coal vapour and dust particles from polishing. Tuberculosis was rampant in this industry and the trade was commonly mentioned as one of the worst offenders (e.g. Bavinck, 1897). Louis Heijermans, who spent time as a health inspector in the diamond workers' union and published one of the first books on occupational diseases in the Netherlands (Heijermans, 1908), pointed to the sedentary nature of the work in an enclosed space as particularly fruitful for tuberculosis (Heertje, 1936, pp. 251–252).

During industrialisation, the expansion of unhealthy industries, such as the diamond industry (Coronel, 1864b), became more common, especially among Jews. The transition from unskilled labour in the open air to skilled work in factories and offices likely hampered the decline of Jewish men's tuberculosis mortality relative to that of Gentile men. This is reflected in Figure 2, which shows that Jews underwent more drastic changes in their occupational structure than the non-Jewish population at the same time as industrialisation accelerated. From 1870 onwards, Jews were much more likely to work inside. High wages paid to diamond workers in an industry where Jews had the upper hand was a massive pull for the sons and daughters of unskilled workers (Kok, 2025a). The contents of the labour created adverse conditions for respiratory diseases such as tuberculosis.

The impact of working indoors can clearly be seen in the ACD. Figure 3 contrasts the share of all deaths attributable to respiratory tuberculosis by workplace type for three periods — roughly comprising before, during, and after industrialisation. In each period, and in both the Jewish Quarter and the rest of Amsterdam, individuals working indoors had higher percentages of tuberculosis mortality. During the period of industrialisation — when mortality rates decreased across the entire population but comparatively less so for the Jewish neighbourhoods — the percentage of deaths attributable to pulmonary tuberculosis rose among Jews with "indoor" occupations by 5%. The rest of Amsterdam's population experienced a roughly similar increase in pulmonary tuberculosis deaths, but due to the growing proportion of Jews employed in factories and offices, the impact of this rise in the second period (1870–1889) may have been more pronounced for the Jewish population. It seems plausible that at least part of the slower relative decline of pulmonary tuberculosis mortality during the last decades of the 19th century can be attributed to deteriorating working environments for Jewish men.

Figure 3 *Tuberculosis as percentage of all deaths among men aged 20–59 in- and outside of the Jewish Quarter, Amsterdam 1856–1909*



Note: 1892–1894 and 1899 are excluded due to incomplete data in the ACD.

The relationship between labour conditions and tuberculosis mortality is further underscored by developments in the third period, i.e. between 1890 and 1909. Looking at the development of tuberculosis death shares across the three periods, its overall gradual increase among both workplace types might be construed as indicative of a general change in the epidemiological profile of the city rather than a change in work environments. After all, if fewer people died from now treatable infectious diseases, chances of dying from degenerative and (at that time) non-curable diseases like tuberculosis would theoretically rise. However, the data provides little support for any straightforward "exchange" with pulmonary tuberculosis. As expected based on existing literature (e.g. Lammertink, 2023), a comparison of the compositions of different groups of causes of death in Amsterdam over time (not shown here) reveals a consistent decline in the share of infectious diseases and a corresponding rise in the share of degenerative diseases, with little distinction between indoor and outdoor workers. The pattern for tuberculosis, on the other hand, is more complex. While its share among causes of death increased during the period of industrialisation, it declined among male Jewish indoor workers in the subsequent period (see Panel 3). As a result, mortality differences between indoor and outdoor workers in the Jewish Quarters largely disappeared. The timing of these changes aligns with the introduction of new labour regulation and collective action, such as the Labour Law of 1889 (Swuste et al., 2019), which introduced the Labour Inspection in 1890, and the formation of the diamond workers' union in 1894 (Hofmeester, 2020). Conditions in the factories, especially in the diamond industry, would soon improve through lowered densities, improved ventilation, and the reduction of work hours.

6 CONCLUSION

In this paper we investigated the differential trends in pulmonary tuberculosis mortality in the Jewish and non-Jewish communities of Amsterdam during the mortality transition: (i) the low overall pulmonary tuberculosis mortality rates of the Jewish neighbourhoods compared to the rest of Amsterdam and (ii) the slower reduction in tuberculosis mortality among Jews. Explaining these differences is helpful in solving the larger tuberculosis puzzle, in particular in regard to the larger debates about the impact of different facets of standards of living on the historical tuberculosis decline.

Given the initially backward socioeconomic position of Amsterdam Jews, generally poorer and living in more deprived and dense areas of the city, we focused on their distinct occupational profile and its development over time. Using detailed spatial mortality information on Amsterdam, our results potentially support the hypothesis that differences in tuberculosis mortality between Jews and Gentiles were driven by (a) initial differences in occupations and (b) changing occupational structures during the period of industrialisation. Prior to industrialisation, Jews were much more prone to working outside. When the diamond industry — the premier occupational niche of Amsterdam Jews — expanded during industrialisation, thousands of Jewish workers instead started working in congested and unhealthy factories. Consequently, this transition slowed down their decline in tuberculosis mortality. The fact that Jewish women did not see such a comparative slowdown (not reported here) supports this finding.

The results of this short paper have implications for more general studies of tuberculosis and overall mortality. For one, distinguishing relevant characteristics of occupations — such as working in- or outdoors — can provide key new interpretations regarding mortality differences by social classes. While occupational differences have often been used to argue for mortality differences between urban and rural areas, newly-digitised individual-level mortality data enable further study of these differences within important, large urban localities.

Moreover, differences between Jews and Gentiles merit further studies too. So far, the main focus has been on infant and child mortality and emphasis has been put on Jewish mothers' greater propensities to breastfeed as a key determinant in Jewish children's health advantages. Studying ethno-religious differences in adult mortality, however, can additionally provide a better understanding of which determinants impacted the survival chances of the majority of the (European) workforce. While we by no means contest that nutrition constituted a key driver in the historical decline of tuberculosis mortality, we argue that the modifying effects of other socioeconomic factors, such as labour conditions, should not be overlooked. By advancing the positions of some and hampering the conditions of others, work environments and labour conditions played a pivotal role in shaping and exacerbating intra-urban health disparities.

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A Decomposition Approach to Cause-Specific Mortality in the Port City of Antwerp in the Early 20th Century

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ABSTRACT

Building on Janssens' work, which highlights the distinct epidemiological profiles of port cities, this study explores cause-specific mortality in early 20th-century Antwerp, Belgium's largest city and a major international port. Using Arriaga's decomposition method, we compare life expectancy and mortality by cause of death in Antwerp with those in Brussels, Ghent, and Liège, the country's next three largest cities. Despite its status as a bustling port city, Antwerp showed a relative health advantage. However, this advantage masked gender- and age-specific risks. Young adult men experienced elevated mortality from accidents, largely due to hazardous port labor, while women faced excess mortality from childbirth, likely linked to socioeconomic vulnerabilities among working-class and immigrant women. Notably, child mortality from infectious diseases was higher in Antwerp than in the other three cities, reflecting particular public health challenges. These findings highlight the importance of individual-level data to better understand localized mortality and cause-of-death patterns. They also underscore the need for further comparative research within the frameworks of the SHiP and Great Leap networks.

Keywords: Mortality, Historical causes of death, Decomposition method, Port cities, SHiP network, Aggregated data, 20th Century, Belgium

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1 INTRODUCTION

This article builds upon the foundational reflections presented in *Gateways of Disease?* by Angélique Janssens and Evelien Walhout, which explored the distinctive mortality and epidemiological patterns of port cities within the broader context of the SHiP (Studying the History of Health in Port Cities) network (Janssens & Walhout, 2018). Their chapter highlighted how 19th- and early 20th-century European port cities served as dynamic laboratories for demographic and health transitions. These urban centers, characterised by high levels of mobility, global commercial ties, and intense socio-economic transformations, were not only hubs of trade and migration but also centers of disease transmission (Hein & Schubert, 2021; Vögele & Umehara, 2015). The study of Janssens and Walhout showed that in the late 19th century, the two major seaport cities in the Netherlands, Amsterdam and Rotterdam, had the highest rates of infectious diseases among children, but not among adolescents and adults, despite being large and rapidly growing port cities. While adult men in these cities faced a high risk of death from infectious diseases, other cities like Maastricht and 's-Hertogenbosch had even more lethal conditions. Port cities facilitated the spread of disease through high traffic, but nearby inland cities also suffered from similar conditions due to crowding and migration. The western Netherlands, where these two port cities were located, saw earlier declines in mortality rates, linked to better living conditions and the spread of medical knowledge.

Inspired by this conceptual framework and the Dutch results, the present study explores mortality patterns and causes of death in the port city of Antwerp in Belgium in the early 20th century. Antwerp's rapid expansion as a major international seaport and commercial gateway during this period makes it an ideal case to further interrogate the assumptions proposed by Janssens and the SHiP network. By examining aggregated cause-of-death series and employing Arriaga's decomposition method, we aim to delineate the disease environment of Antwerp, Belgium's largest city, and assess the extent to which its mortality and cause-of-death patterns aligned with or diverged from those of the next three largest cities: Brussels, Ghent, and Liège. We pay special attention to gender differences, which is a key focus in Janssens' work (Janssens, 2014; Janssens & Pelzer, 2012). Through the Antwerp case study, we also aim to critically reflect on the methodological challenges and potential of historical individual-level cause-of-death data, a concern raised in the SHiP network's research agenda (Janssens, 2021) and now a central element of the ongoing COST Action Program, The Great Leap.

The article starts with a brief overview of the socioeconomic context of Antwerp and the other three cities. In Section 2, we address our sources, while Section 3 delves into the urban health penalty, analysing life expectancies and age-specific mortality risks for men and women across these cities. We then present, based on Arriaga's decomposition, the findings on the causes of death for men and women, with a particular focus on the patterns unique to the port city of Antwerp. We conclude with some final remarks and suggestions for future research.

2 ANTWERP AT THE TURN OF THE 20TH CENTURY

Antwerp's trajectory during the 19th and early 20th centuries was closely linked to its port. From the 1830s, Antwerp evolved from a modest textile hub to a major international seaport and commercial center, driven by Belgium's industrialisation, advances in shipping technology, and improved transport links. The city's transformation was striking, with its population rising from 50,000 in the early 1800s to 301,766 by 1910, making it Belgium's largest city, largely surpassing Brussels (177,078), Ghent (166,445), and Liège (167,521) (Ghent University Quetelet Center, n.d.). Roughly 10% consisted of international migrants (Puschmann, 2015). By 1850, it was one of north-western Europe's most connected inland ports, strategically serving Belgium, the German Rhineland, the southern Netherlands, and northern France. Regular shipping routes to Rotterdam, London, Hull, and Le Havre supported trade with Britain, France, and the Netherlands, while intercontinental links extended to Rio de Janeiro, New York, Singapore, and other ports. This expansion fostered export industries and local shipbuilding such as the Cockerill shipyard, while the city became a key departure point for emigrants, particularly with the rise of the Red Star Line company which ferried nearly two million emigrants from Antwerp to the United States (Winter, 2009).

The transport and trade sectors thrived in Antwerp, supported by shipbuilding companies, commercial agencies, import and export firms, banks, insurance companies, and transport enterprises, along with cafés and restaurants. The 1910 industrial and trade census reveals that ca. 30% of the male workforce was employed in transport-related jobs (see Table 1). Many men also worked in sales (ca. 16%), while women, though facing more limited employment opportunities, were active in sales and catering (ca. 58%) (see Table 2). Clothing was another large sector for women. Similar patterns were observed in Liège and Brussels, while in Ghent women predominantly worked in the mechanised cotton industry (Van Rossem, 2018). In Liège, men found work in coal mining, steel, and machine-building, while Brussels, where technical innovation was largely absent and industry was smaller-scale, offered jobs in construction, furniture, printing, and the production of luxury and consumer goods. Many also worked in finance and government due to the city's capital status (Ghent University Quetelet Center, n.d.).

This economic boom brought significant social challenges. Antwerp's labour market was polarised, with skilled merchants, international merchants, and specialised artisans alongside unskilled dockers and manual labourers (Greefs & Winter, 2016). Living conditions for the working class had deteriorated throughout the 19th century, characterised by widespread poverty, overcrowding, and inadequate housing (Lis, 1986). According to the special census of habitations in 1910, 10% of households in Antwerp had more than two people per room. Similarly, the percentages were high in Brussels (12%) and Liège (9%), while a much lower proportion was observed in Ghent (4%) (Van Rossem, 2018). In some Antwerp neighbourhoods, particularly near the port, households averaged between 9 and 12 occupants per house (Bertels et al., 2010). Sanitation infrastructure (sewage) lagged. Piped water was introduced in 1881, but the distribution of the water network across the city did not occur until the 1920s (Van Craenenbroeck, 1998).

Table 1 *Occupational structure (%) of men in Brussels, Antwerp, Ghent, and Liège (1910)*

	Antwerp	Brussels	Ghent	Liège
Industry				
Construction	6.6	10.1	10.5	7.9
Metals	6.2	8.6	11.5	30.2
Clothing	2.6	8.1	2.6	3.2
Timber and furniture	5.7	7.9	9.5	6.4
Transport	30.4	6.8	11.2	8.2
Food	5.4	5.4	5.6	4.6
Leather	1.9	5.1	2.3	2.9
Printing	2.1	4.6	2.5	2.0
Art	9.2	3.7	1.4	1.1
Textiles	0.3	1.0	21.7	0.6
Tobacco	1.5	0.4	1.2	0.8
Mining	0.0	0.1	0.1	11.1
Other	1.6	2.1	2.9	2.2
Trade				
Sales	15.9	20.0	12.4	13.6
Catering	4.3	9.4	2.8	3.1
Banking	1.4	3.0	0.5	0.8
Insurance	0.9	1.1	0.4	0.6
Intermediary	3.4	1.0	0.5	0.3
Leisure	0.2	0.3	0.3	0.2
Unclassified				
	0.5	1.4	0.1	0.3

Table 2 *Occupational structure (%) of women in Brussels, Antwerp, Ghent, and Liège (1910)*

	Antwerp	Brussels	Ghent	Liège
Industry				
Clothing	28.5	39.5	21.9	35.1
Textiles	2.5	3.0	43.9	3.6
Leather	0.4	2.6	0.4	0.7
Printing	0.4	2.1	0.3	1.6
Art	2.0	1.1	0.2	0.2
Food	2.5	1.1	0.6	2.0
Timber and furniture	0.3	1.0	0.5	1.0
Metals	0.2	0.9	0.2	4.7
Tobacco	2.3	0.8	0.1	1.8
Transport	1.6	0.4	0.4	0.7
Construction	0.1	0.2	0.1	0.1
Mining	0.0	0.0	0.0	1.4
Other	1.1	2.1	2.1	2.3
Trade				
Sales	35.2	31.4	17.3	35.0
Catering	22.1	11.9	11.7	9.2
Intermediary	0.4	0.3	0.1	0.1
Leisure	0.1	0.2	0.3	0.1
Banking	0.1	0.1	0.0	0.1
Insurance	0.1	0.1	0.0	0.1
Unclassified				
	0.1	1.4	0.0	0.3

Sources: Statistique de la Belgique, 1910, Recensement de l'industrie et du commerce, 31 décembre 1910. *Bruxelles, Ministère de l'Industrie et du Travail. Accessed through Van Rossem (2018) and LOKSTAT (n.d.).*

The changing labour market brought about changing working conditions. Unlike the industrial centers of Liège and Ghent, where labour unions had a strong presence in large factories and helped improve working conditions, Antwerp's port laborers experienced limited unionization before 1914 (Deferme, 2007; Van Rossem, 2018). Although working conditions were harsh, and wages and job security were low (reflecting loose labour), actions remained limited, apart from the general strike in 1907 (Vanfraechem, 2005). This is remarkable, considering the many occupational health hazards associated with working in the port. Dockers, in particular, were vulnerable during the loading and unloading of ships, facing risks such as broken bones, suffocation, drowning, and burns (Van Elsen, 2003).

3 SOURCES, DATA AND METHODS

The reconstruction of mortality risks and life expectancies at the city level for the early 20th century in this article are based on the population census of 1910 and *Le Mouvement de la Population et de l'Etat Civil* (State Archives Belgium, 1908–1912). The decennial population census delivers population numbers by age and sex, while *Le Mouvement* summarises yearly vital events per municipality. Since 1886, cause-of-death data have also been included. They are based on the individual-level cause-of-death registers that Belgian municipalities have been obliged to maintain since 1851. Besides the cause of death, specific data had to be collected with the name of the deceased, surname, sex, occupation, age, marital status, and date and place of death (Velle, 1985). Unfortunately, most of these individual-level municipal registers were destroyed for privacy reasons (Antwerp is a notable exception). The quality of the cause-of-death registers, however, depended on the medical knowledge of the person completing them. In many municipalities, it was common practice for civil servants to fill out the forms, relying on family members or neighbours to provide the cause. In some places, however, a doctor was appointed for the task, and in large cities such as Antwerp, Brussels and Liège, specific physicians were appointed as *médecins vérificateur* (Hacha, forthcoming).

Le Mouvement provides at census years an age-specific classification of causes of death, using six age categories: <1 year, 1–6, 7–14, 15–20, 21–49, 50+. It employs a classification system based on 40 diseases and 10 headings. This system was introduced in 1903, coinciding with the introduction of the first International Classification of Diseases (ICD-1) at the municipal level. Compared to earlier classifications, it was more detailed and paid more attention to degenerative diseases. In other words, while the causes of death in municipal registers were classified according to ICD-1, a separate and less detailed classification scheme was used in *Le Mouvement* for the aggregated municipal data at the national level. In this article, we use the data for 1910 from *Le Mouvement*, as it is the first year for which the cause-specific mortality data based on the new extended nomenclature in 1903 is distinguished by age.

We reclassified the 40 causes of death in *Le Mouvement* into 14 categories. This reclassification is primarily based on the 10 headings in *Le Mouvement*, but we reassigned some diseases for historical purposes. The following disease categories remained unchanged: (1) cardiovascular diseases; (2) diseases related to perinatal conditions (lack of viability of the infant); (3) diseases related to childbirth (mother); (4) respiratory diseases; and (5) urogenital diseases. We separated out several diseases from the original category of general diseases because of their historical importance: (6) pulmonary tuberculosis and (7) cancer were classified as separate categories (see also Reid et al., 2015). Pulmonary tuberculosis was a typical disease of the industrial era, while cancer as a degenerative disease became increasingly important during the epidemiological transition. Tuberculosis of the meninges was reassigned from the category of general diseases to the category of (8) neurological diseases because of its strong link to meningitis. A similar procedure was taken for acute and chronic alcoholism that was placed with the (9) gastrointestinal diseases, due to its relation with liver cirrhosis which was probably also caused by alcohol abuse. The remaining general diseases were classified under the new heading of (10) infectious diseases. We furthermore separated out (11) accidents from the category of (12) violent deaths, because we suppose a relationship with working conditions. This was also the case for (13) enteritis from the gastrointestinal diseases, as it was a prime cause of childhood deaths. Finally, because the category old age was not a well-defined category we included it among the group of (14) other causes of mortality. The so-called doubtful cases were also reassigned from the category of violent deaths to the category other causes. This was also the case for sudden death and unspecified or poorly defined causes of death (previously the separate category of unspecified or poorly defined causes).

4 MORTALITY

4.1 URBAN HEALTH PENALTIES IN THE 19TH CENTURY

In a previous study, we set the stage for a more detailed analysis of mortality in Antwerp by cause of death (Devos & Van Rossem, 2015). In an article in the *Journal of Belgian History*, we showed that in 19th-century Belgium the urban penalty was evident, but not in every city to the same extent. Throughout the 19th century, life expectancies in the four largest cities were consistently lower than the national average, illustrating the common burden of urban living conditions. Yet, Antwerp often fared better than its peers, especially Brussels. In 1846, life expectancy at birth in Antwerp stood at 37.3 years, close to the Belgian average (37.8 years). After a significant drop in life expectancy due to a cholera epidemic in 1866, Antwerp rebounded quickly, surpassing cities like Ghent and Brussels in life expectancy. By 1900, life expectancy in Antwerp had risen to nearly 43 years (Belgium), exceeding that of Ghent (41 years) and Brussels (39 years), but below Liège (45 years). These numbers reflect not just the general decline in mortality in Belgium (Devos, 2006), but also Antwerp's comparatively favourable health outcomes (Devos & Van Rossem, 2015).

4.2 MALE AND FEMALE LIFE EXPECTANCY IN ANTWERP IN THE EARLY 20TH CENTURY

To get better insight into the determinants of the Antwerp health advantage and the particularity of the cause-of-death profile of the port city, we here examine men and women separately. We calculated life tables over five years for the period 1908–1912 to smooth out possible short-term fluctuations. Comparing the four cities in 1908–1912, we see in Table 3 that the male life expectancy was highest in Liège (45.7 years), closely followed by Antwerp (45.5 years) and then Ghent (43.6 years). In the capital however it was over seven years lower, with only 38.1 years. For the Antwerp women the figures were the highest with 52.2 years, compared to 50.6 years in Liège, 48.4 in Ghent and only 43.8 years in Brussels.

Table 3 clearly shows that urban penalties affected men and women in each city differently. Although life expectancy in Antwerp was comparatively high, gender differences were also the highest here. In 1910 we observe a gap of no less than 6.7 years between the life span of men and women, compared to 4 years in the rest of Belgium, nearly 5 years in Ghent and Liège, and ca. 5.6 years in Brussels. The age-specific mortality probabilities of dying for males and females (see Table A1 in the Appendix) highlight that Antwerp outperforms the other cities at most ages, except for children below age 5 in Liège. At working ages, however, Ghent exhibits the lowest risks of dying.

Table 3 *Male and female life expectancy at birth in Antwerp compared to Brussels, Ghent and Liège (1908–1912)*

	Men	Women
e0 Antwerp	45.5 years	52.2 years
Difference with Brussels	+7.4	+8.5
Difference with Ghent	+1.9	+3.8
Difference with Liège	-0.2	+1.5
Difference with Belgium	-3.4	-0.5

Figure 1 *Sex ratio (male to female) of probabilities of dying (nQx) in Antwerp, Brussels, Ghent and Liège (1908–1912)*

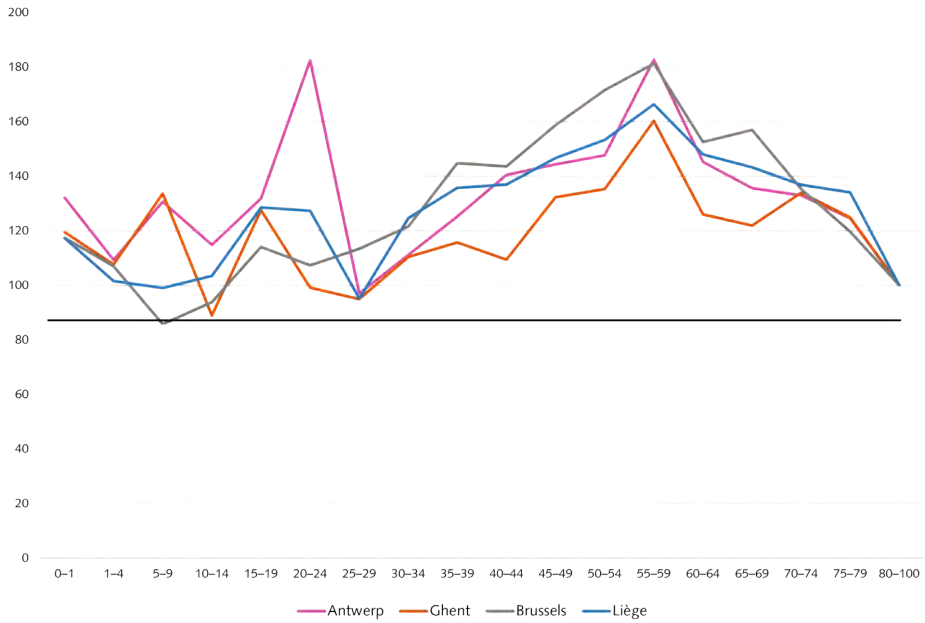


Figure 1 with the sex ratio of the age-specific mortality risks (male to female) shows in fact that urban hazards in Antwerp were particularly severe for adult males, especially at ages 20–24. In an earlier study, we suggested that employment conditions are to blame and pointed to the specificity of the urban labour market and its associated health hazards (Devos & Van Rossem, 2015). We suggested that men's disadvantages in Antwerp can be linked to the working conditions in the port. Research has shown that the transformation of Antwerp into a booming international port center had fundamental implications for employment opportunities. Antwerp's labour market was heavily segmented according to gender, age, and origin. Most of the emerging employment in the port and trading sectors was taken up by single men. The men found their way to hazardous occupations, such as loading and unloading ships, which demanded physical strength and endurance (Greefs & Winter, 2020; Van Elsen, 2003; Winter, 2009). It is therefore necessary to examine whether these workplace hazards are reflected in the cause-of-death pattern.

5 CAUSES OF DEATH

To unravel the determinants of the Antwerp health advantage, we first examine the underlying causes of death, and then compare its cause-of-death profile with that of the other three cities, using Arriaga's decomposition method. We focus on the year 1910, as *Le Mouvement* only provides age-specific cause-of-death data at census years.

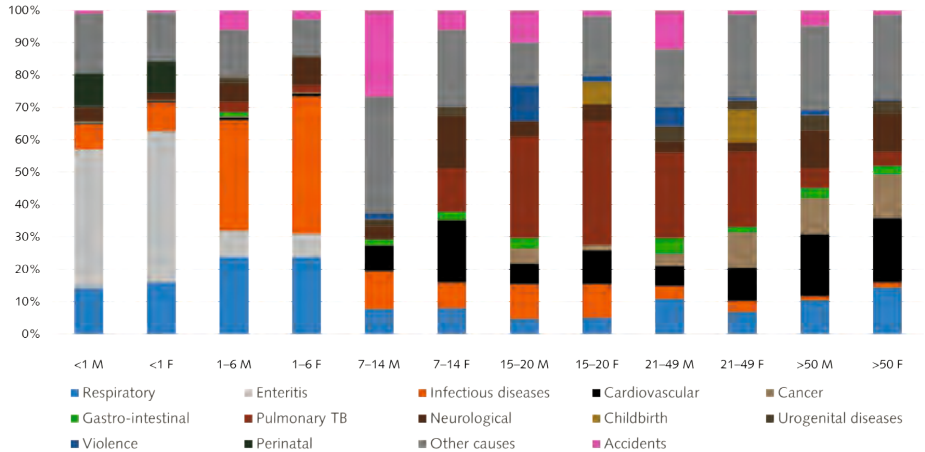
5.1 CAUSE-OF-DEATH PATTERN OF MEN AND WOMEN IN ANTWERP

Figure 2 shows the deaths of men and women by age group and cause of death category. Although a significant portion of deaths, approximately 11% to 35%, lacks a clearly assigned medical cause, several important conclusions can be drawn. Enteritis (diarrhoeal disease) is the leading cause of death for infants, accounting for more than 40% of infant deaths. Excluding other causes of death, the second most important category is respiratory diseases, which account for 15%. For young children, infectious diseases are the most predominant cause of death, explaining 34% and 42% of male and female deaths, respectively. The second most important category for this group is respiratory diseases, which account for about 24%. For children aged 7 and older, the cause of death profile becomes more gendered. Boys are notably more vulnerable to accidents, which account for 27% of their deaths, while girls are more likely to die from heart diseases (18%), neurological diseases (e.g. meningitis) (16%), or pulmonary tuberculosis (13%). Among adolescents and young adults, pulmonary tuberculosis is the leading cause of death, with approximately one in three adolescents and one in four young adults dying from it. Additionally, maternal mortality for women and violent or accidental deaths for men are significant causes of death in these age groups. The high proportion of young adult females dying from degenerative diseases, specifically cardiovascular diseases (10%) and cancer (10%), is striking in comparison to men, where these causes account for 6% and 4%, respectively. For adults aged 50 years and older, cardiovascular diseases occupy the top spot, responsible for 19% of deaths in both men and women. Cancer (12%), neurological diseases (11%), and respiratory diseases, particularly among women (14%), are the other most significant causes of death in Antwerp. For adults aged 50 years or older, cardiovascular diseases occupy the top spot, responsible for 19% of deaths in both men and women. Cancer (12%), neurological diseases (11%), and respiratory diseases — particularly among women (14%) — are the other most significant causes of death in Antwerp.

5.2 CAUSE-SPECIFIC CONTRIBUTIONS TO MALE AND FEMALE LIFE EXPECTANCY

Now that we have established the main causes of death for men, women, and children in Antwerp, it is important to compare these results with those of Brussels, Ghent, and Liège in order to detect any particularities in the cause-of-death patterns of a port city like Antwerp. This comparison will provide insights into which (lethal) diseases were relatively less common in Antwerp and may help explain the city's health advantage or, conversely, reveal conditions that were masked by this advantage.

Figure 2 *Causes of death (% of total deaths) by sex and age group, Antwerp (1910)*



We use Arriaga's decomposition method to break down the male and female life expectancy difference between Antwerp and the three selected cities into cause-of-death-specific components (Arriaga, 1984). While the more analytically precise decomposition method by Pollard generally yields similar results, Arriaga's method is conceptually simpler and easier to apply using traditional life table data (Auger et al., 2014). The method generates components that reflect how much of the difference in life expectancy, by age group, can be attributed to specific causes of death. The age-specific calculation considers that part of the difference is due to age-specific mortality rates in the compared populations (direct effect), while another part can be attributed to variations in survivor proportions arising from different mortality rates (indirect effect). The direct and indirect effects focus on mortality differences within a single age group. However, the calculation also considers that a small part of the difference is due to interaction effects, as a distinct number of survivors experience changed death rates at older ages. The proportions of deaths due to certain causes within specific age groups are used to calculate the contributions of these causes to the life expectancy gap. In our analysis, a positive component indicates lower mortality from a particular cause of death in Antwerp compared to the other cities. A negative component reflects higher cause-specific mortality in Antwerp. The method allows for the distinct consideration of contributions from competing causes of death within the same age group, which may cancel each other out.

Figures 3 and 4 (details in the Appendix Table A2) show the contributions of various causes of death to the life expectancy gap in Antwerp for both men and women. To avoid overburdening the text with graphs, we opt to compare Antwerp with the three cities combined (unweighted average). The figures indicate positive contributions from most causes of death, suggesting lower survival rates in Antwerp. However, there are negative contributions from accidents for adult men (-0.15 years for ages 21–49 and -0.11 years for ages 50+), and from childbirth for women (0.11 years for ages 21–49). The excess mortality from accidents among adult males in Antwerp is likely due to the concentration of male workers in hazardous port-related occupations. Unlike in the industrial city of Ghent, where labor organizations had succeeded in advocating for safer working conditions. The higher mortality rate among Antwerp women due to childbirth may reflect the vulnerability of the increasing number of single immigrant women in the port city (Greefs & Winter, 2016). Infectious diseases contributed significantly to mortality among infants and young children (-0.25 to -0.28 years for male and female infants, and -0.59 to -0.89 years for male and female children aged 1–6). While diseases such as enteritis and perinatal conditions in infants, and respiratory and neurological disorders affected children in Antwerp less than in other cities, it is crucial to note that infectious diseases show the opposite trend: more children in Antwerp die from infectious diseases compared to those in the other three cities. This may suggest a differential impact of certain determinants (e.g., water infrastructure, childcare, crowding) in these cities. However, it is important to highlight the contribution of 'other diseases', which were notably much higher in the other cities. This could be related to the more cautious registration practices by the Antwerp health service compared to those in the other cities (Hacha, forthcoming), where some (infectious) causes may have been misclassified under the wrong heading or left unidentified during the registration and the disease categorisation process (Alter & Carmichael, 1999; Janssens & Devos, 2022; Reid et al., 2015). Unfortunately, the aggregated cause-of-death categories in *Le Mouvement* do not allow for much reclassification and a more detailed analysis. To improve this, individual-level data are needed.

Figure 3 *Cause-specific contributions by age group to the difference in life expectancy between Antwerp and the other cities (1910): men*

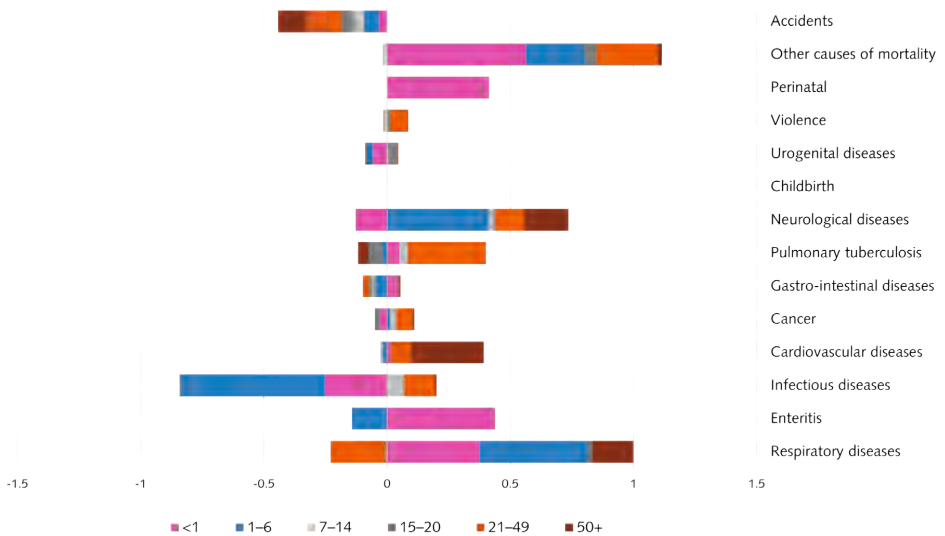
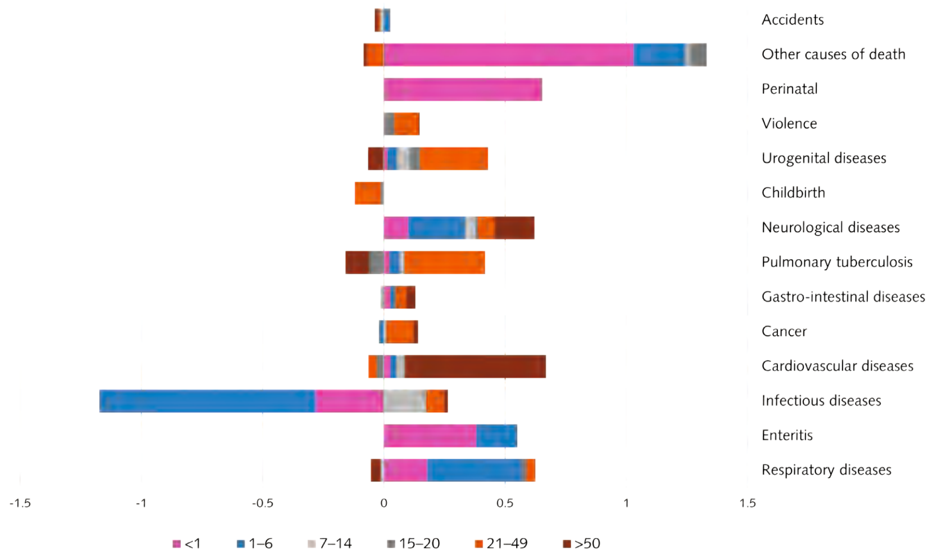


Figure 4 *Cause-specific contributions by age group to the difference in life expectancy between Antwerp and the other cities (1910): women*



6 CONCLUSIONS

In this article, we have explored cause-specific mortality patterns in early 20th-century Antwerp, focusing on how they compare to those of Brussels, Ghent, and Liège, and how these findings fit within the conceptual framework proposed by Janssens and the SHiP network. The results provide several important insights into the distinct health environment of Antwerp as a major international port city.

Contrary to the common assumption that port cities are characterised by higher mortality risks due to high mobility, trade, and dense populations, Antwerp exhibited a comparative health advantage. Life expectancy in early 20th-century Antwerp was the second highest among Belgium's four largest cities for men (45.5 years) and the highest for women (52.2 years). However, our decomposition approach revealed that this health advantage masks important gendered and age-specific vulnerabilities. Specifically, men in Antwerp experienced higher mortality from accidents in working-age groups, which can be directly linked to the city's occupational structure, particularly its reliance on physically hazardous port labour. While women had higher overall life expectancy, they faced some excess mortality during childbirth, likely reflecting the socioeconomic vulnerabilities of working-class women in Antwerp, many of whom were unmarried migrants. Another key finding relates to childhood mortality. Despite Antwerp's relatively high life expectancy, the city saw significant excess mortality in children due to infectious diseases, a trend consistent with the findings in the Dutch port cities Amsterdam and Rotterdam examined by Janssens and Walhout. However, we should be wary of incorrect disease categorisations, considering the limited medical knowledge at the time. The category of 'other diseases' was notably higher in the three other cities, which may have encompassed a range of (infectious) diseases that were either misclassified or unidentified.

Overall, these results reinforce Janssens' argument that port cities were rather unique socio-spatial contexts in which specific risks and protections coexisted. This aligns with SHiP's focus on the social ecology of port cities, where mobility and labour dynamics intersect with gender in shaping health risks. Antwerp serves as an example of a booming port city navigating the epidemiological transition, yet still constrained by the occupational and gendered hazards embedded in its urban and economic structure.

Still, in interpreting the findings, it is important to acknowledge the limitations in our data. First, the cause-of-death data are based on a snapshot from 1910. While this offers useful information on mortality patterns, a more comprehensive analysis over a longer period is essential to draw more definitive conclusions. Second, the aggregated nature of the cause-of-death records in this study may obscure the episodic, often seasonal, nature of specific infectious diseases.

To better understand how port cities like Antwerp managed, or failed to manage, such diseases, future research should use individual-level cause-of-death data. Antwerp is unique in Belgium for having preserved a long cause-of-death register (1820–1946), presenting a promising opportunity for more granular investigations. The ongoing large-scale citizen science project SOS Antwerpen (www.sosantwerpen.be) is working to make these individual-level records more accessible. Inspired by earlier work on Amsterdam led by Angélique Janssens, this initiative holds great potential for improving our understanding of how port cities adapted to the burden of infectious diseases over time (Devos & Janssens, 2017; Devos et al., 2023). Further research into different time periods and additional cities will be crucial in determining whether the epidemiological trajectories observed in Antwerp, Amsterdam, and Rotterdam are representative of 19th- and early 20th-century port cities more broadly. Ultimately, detailed and comparative individual-level studies like these are essential for validating and deepening the conclusions drawn from aggregated analyses such as this one.

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APPENDIX

Table A1 *Age-specific probabilities of dying (nQx) in Antwerp and the three other cities (1908–1912): men and women*

Males (nQx)	**Antwerp**	**Ghent**	**Brussels**	**Liège**	**Ghent/ Antwerp**	**Brussels/ Antwerp**	**Liège/ Antwerp**
0–1	0.18	0.24	0.23	0.16	130	128	86
1–4	0.08	0.08	0.11	0.07	101	136	89
5–9	0.02	0.02	0.02	0.02	101	111	103
10–14	0.01	0.01	0.01	0.01	94	155	123
15–19	0.02	0.02	0.02	0.02	91	116	105
20–24	0.03	0.02	0.03	0.03	65	102	99
25–29	0.02	0.02	0.04	0.03	89	173	117
30–34	0.03	0.03	0.04	0.04	89	156	128
35–39	0.04	0.03	0.06	0.04	76	155	109
40–44	0.05	0.04	0.07	0.06	82	151	117
45–49	0.06	0.06	0.10	0.08	90	161	126
50–54	0.09	0.08	0.13	0.11	89	145	116
55–59	0.14	0.12	0.18	0.15	86	127	112
60–64	0.18	0.16	0.22	0.21	89	124	117
65–69	0.24	0.25	0.33	0.29	104	135	120
70–74	0.35	0.36	0.41	0.42	104	116	119
75–79	0.50	0.52	0.51	0.59	103	101	117
80–100	1.00	1.00	1.00	1.00	100	100	100
Females (nQx)	**Antwerp**	**Ghent**	**Brussels**	**Liège**	**Ghent/ Antwerp**	**Brussels/ Antwerp**	**Liège/ Antwerp**
0–1	0.138	0.199	0.200	0.133	143	144	96
1–5	0.076	0.077	0.105	0.073	102	139	96
5–10	0.013	0.013	0.021	0.017	99	169	136
10–15	0.008	0.010	0.015	0.011	121	189	137
15–20	0.015	0.014	0.020	0.016	94	134	108
20–25	0.016	0.019	0.027	0.022	120	173	142
25–30	0.022	0.020	0.033	0.027	91	148	120
30–35	0.026	0.023	0.037	0.029	89	143	114
35–40	0.031	0.025	0.041	0.031	82	134	101
40–45	0.034	0.035	0.050	0.040	105	147	120
45–50	0.043	0.042	0.063	0.054	98	146	124
50–55	0.062	0.060	0.077	0.069	97	125	112
55–60	0.076	0.074	0.097	0.093	98	128	123
60–65	0.125	0.128	0.147	0.143	103	118	115
65–70	0.179	0.208	0.208	0.203	116	116	113
70–75	0.264	0.272	0.301	0.304	103	114	115
75–80	0.404	0.414	0.427	0.440	102	106	109
80–100	1.000	1.000	1.000	1.000	100	100	100

Table A2 *Cause-specific contributions by age group to the difference in life expectancy between Antwerp and the three other cities (1910): men and women*

	Accidents	Cancer	CVD	Gastro.	Enteritis	Infectious	Respir.	Pulm. TB	Neuro.	Urogen.	Childbirth	Perinatal	Violence	Other
Men <1	-0.03	0.00	0.02	0.05	0.44	-0.25	0.38	0.05	-0.13	-0.06		0.41	0.00	0.56
1–6	-0.06	0.00	-0.02	-0.05	-0.14	-0.59	0.44	-0.02	0.41	-0.02		0.00	0.00	0.24
7–14	-0.05	0.00	-0.01	-0.01	0.00	0.07	0.00	0.03	0.02	0.01		0.00	-0.01	-0.02
15–20	-0.04	0.00	0.00	-0.01	0.00	0.00	0.02	-0.06	0.01	0.04		0.00	0.01	0.05
21–49	-0.15	0.00	0.08	-0.03	0.00	0.12	-0.23	0.32	0.12	0.00		0.00	0.07	0.25
50+	-0.11	0.00	0.29	0.01	0.00	0.01	0.17	-0.04	0.18	-0.01		0.00	0.00	0.01
Women <1	0.00	0.00	0.03	0.03	0.38	-0.28	0.18	0.02	0.10	0.01	0.00	0.65	0.00	1.03
1–6	0.03	-0.02	0.02	0.01	0.17	-0.89	0.39	0.04	0.23	0.04	0.00	0.00	0.00	0.21
7–14	-0.01	0.01	0.04	-0.01	0.00	0.17	-0.01	0.02	0.04	0.05	0.00	0.00	0.00	0.02
15–20	0.00	0.00	-0.03	0.01	0.00	0.00	0.01	-0.06	0.01	0.04	-0.01	0.00	0.04	0.07
21–49	-0.01	0.11	-0.04	0.05	0.00	0.08	0.04	0.33	0.07	0.28	-0.11	0.00	0.10	-0.07
50+	-0.01	0.02	0.58	0.04	0.00	0.01	-0.04	-0.10	0.17	-0.07	0.00	0.00	0.00	-0.01

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How Cause-Specific Mortality Contributes to Sex Differences in Life Expectancy over Time

Trends in Utah and Denmark

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ABSTRACT

Female life expectancy exceeds male life expectancy. To better understand the basis for this discrepancy, we use Arriaga decomposition methods to examine differences in life expectancy by exploring age-specific and cause-specific mortality differences between the sexes and how these differences arise over time and between two distinct populations, Denmark and Utah (U.S.). Our focus is on how specific causes point to shifts in secular circumstances and behavioral factors that help to explain female-male differences in mortality across these two populations. Our findings point to the prominence of cardiovascular mortality as a key contributor to the female advantage in life expectancy for both Denmark and Utah but its influence is waning. External causes of mortality, including suicide and homicides along with motor vehicle accidents, also have large effects for both locales despite differences in lifestyles and policies. Cancer mortality in younger reproductive ages slightly subtract from the female life expectancy advantage. Overall, if the objective is to reduce sex differences in life expectancy, achieving this goal can be aided by promoting policies that reduce overall mortality but also mortality from key causes of death that are the basis for these discrepancies.

Keywords: Sex differences, Life expectancy, Causes of death, Decomposition

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1 INTRODUCTION

The survival advantage of females over males is nearly universal for most Western nations and it has been growing for many decades in the 20th century. This advantage is now seen throughout most countries in the world (Barford et al., 2006; Sauerberg et al., 2023) though the pattern varies by time and place (Bergeron-Boucher et al., 2022). Females enjoy this advantage for many reasons due to differences in biology (Austad, 2006), genetics (Eskes & Haanen, 2007; Moller et al., 2009) and behavioral/social factors (Kalben, 2000).

In previous work that explored sex differences in survival between three distinct populations (Utah, Sweden and Denmark), the female advantage in cohort life expectancy was found to be similar between these populations (Lindahl-Jacobsen et al., 2013). On one hand, the authors reported that over time the sex differences in the *improvement in life expectancy* was two years smaller for those identified as members of the Church of Jesus Christ of Latter-day Saints than in Denmark and Sweden. This finding suggests that shifts in lifestyle (for Denmark and Sweden, where the *improvement* was larger) are likely a reason for the overall change seen in cohort life expectancy. On the other hand, sex differences in cohort life expectancy at the age of 50 years were similar for individuals actively affiliated with the Church of Jesus Christ of Latter-day Saints and for Denmark and Sweden. Given that those affiliated with the Utah-based church are proscribed from using tobacco and alcohol, two key behaviors affecting disease risk, these results also point to possible biological mechanisms or other unknown risk factors.

1.2 AIMS

In this paper, we examine differences in life expectancy by exploring age-specific and cause-specific mortality differences between males and females and how these differences contribute to the female survival advantage. We investigate how these differences arise over time and between two distinct populations, Denmark and Utah in the United States. Our focus is on how specific causes of death contribute to sex differences in life expectancy over time and how these causes point to shifts in secular circumstances and behavioral factors that help to explain female-male differences in mortality. By looking at sex differences in life expectancy over many decades and across populations, we are able to observe how shifting cause-specific mortality patterns drive alterations in the patterns underlying these changes in life expectancy. Identifying these cause-specific patterns also helps to identify how previous changes in policies or social trends contributed to the male disadvantage and potentially nominate targets for intervention that may serve to increase life expectancy for both sexes and minimizing differences between them.

This paper includes data on Utah and Danish mortality and survival. Utah provides a useful comparison to Denmark that should serve to reveal differences in the distribution of causes of death that may align with differences in social patterns between the two populations. Utah's population has the largest percentage of individuals and families affiliated with the Church of Jesus Christ of Latter-day Saints. Those who are active adherents to the religion not only experience overall survival benefits but may have lower risks of specific types of death given that they are discouraged from using alcohol and tobacco, encouraged to fast once a month, and engage more often in church and related social activities compared to others (Mineau et al., 2004). Among states in the contemporary U.S., Utah has the lowest rate of adult tobacco usage (6.7%) whereas Denmark has rates 2.5 times higher. With respect to alcohol, per capita annual consumption is the lowest in Utah among all states (4.65 liters) and Denmark's is 10.4 liters.

Utahns have had and continue to have some of the highest fertility rates in the U.S. (Bean et al., 1990) which is likely to alter the risks of certain causes of death such as breast cancer (Lima et al., 2021). Early and frequent childbirth reduces the risk of incident breast cancer and lowers the risk of breast cancer mortality. By comparison, Denmark has significantly lower fertility rates with a total fertility rate of 1.46 child per women in 2024 (Statistics Denmark, 2025).

2 DATA

2.1 DENMARK

Death counts by primary causes of death were obtained from the Danish Register of Causes of Death (Helweg-Larsen, 2011; Juel & Helweg-Larsen, 1999) for one-year age and time period intervals from 1952 to 2015 for all citizens with an address in Denmark. The Danish causes of death registry data are based on the death certificates completed by medical doctors for all Danish decedents since 1952. Causes of death were classified according to four-digit International Classification of Diseases versions 6, 7, 8 and 10 in 1951–1957, 1958–1968, 1969–1993, and 1994–1998, respectively and were bridge-coded (Jacobsen et al., 2006; Janssen & Kunst, 2004) into 18 causes of death and by cancer specific deaths. This classification was applied to the Utah deaths as well. Person-year time by sex and one-year age and period groups was retrieved from the human mortality database (Max Planck Institute for Demographic Research, 2015). Deaths in Denmark for this study total 3,355,720.

2.2 UTAH

The Utah Population Database (UPDB) is the source for data on survival and causes of death in Utah. UPDB is a comprehensive database comprising linked demographic, medical, and genealogical individual and family-level data spanning the Utah population for the last two centuries (Smith et al., 2022; Smith & Mineau, 2021). UPDB holds considerable data on medical outcomes, including causes of death, for all decedents in the state since 1904. UPDB comprises data on families with and without an affiliation to the Church of Jesus Christ of Latter-day Saints.

The Utah data for this analysis are based on several inclusion criteria. To be part of the analysis, individuals must be Utah born between 1847 (the arrival of church members into the Utah territory) and 2015. We observe these individuals up to their last follow-up year (including the year of death) between 1904–2015. Since we compute a series of decade-specific *period life tables* and we need to observe causes of death, individuals must have the opportunity to be followed until 90 years of age. The official Utah vital record system was established in 1904 where Utah death certificates were issued that contain primary (and all other) cause of death information. In calculating *period life expectancy* from individual life histories, person-years were calculated from birth to death (in Utah) or through the year in which the individual was last seen alive in Utah. Given the broad historic coverage of UPDB, we are aware of deaths inside and outside Utah but that lack coded causes of death. These deaths are included for the purposes of generating life tables but their causes are in the "unknown" category. Because death certification did not begin until 1904, we start with the 1940–1949 period life table since it is well past 1904 and for persons born in 1847, they could have reached age 92. With these restrictions, the number of deaths totals 365,448.

2.3 DIFFERENCES IN DATA REGISTRATION

The data are structured differently for the two populations. For Denmark, the period life tables are based on age-sex counts in a given year, and death certificates are issued regarding decedents that same year. If a Danish resident leaves the country, no longer has an address in the country, and dies outside Denmark, they are not included in the estimation.

For the Utah analysis, the data are at the individual-level over their life time. If a person is alive and then dies in a given year within Utah, this person is counted in the person-year and death calculations when estimating mortality rates. If instead they leave Utah in a given year and we observe their death outside Utah, they are included in the person-year calculation for all preceding time points but are censored at the year they leave Utah. The UPDB often records these out-of-state deaths as well as the year of out-migration but the UPDB does not collect non-Utah death certificates where causes of death are recorded. In the end, the full life history data of individuals are formatted to derive the usual period life table person-year and death counts by calendar year and sex.

3 METHODS — ARRIAGA DECOMPOSITION

The study of sex differences in causes of death permits an analysis of the differences in mortality from two different perspectives. First, there is the estimation of how each cause of death affects the *sex difference in mortality for each age group*. This is followed by a second estimation, where we determine the contribution of each cause of death to *sex differences in life expectancy* (Arriaga, 1984, 1989).

Each of these stages of estimation is summarized here. The contribution of differences in life expectancy attributable to sex differences in all-cause mortality at each age is calculated first. This means that we estimate the years added or subtracted from life expectancy attributable to differences (above or below) in the mortality rates in the period life table. Then, the influence of mortality differences by causes of death on life expectancies is calculated. This estimation requires an assumption: the contribution to *sex differences in life expectancy* by causes of death *for each age group* is proportional to the *sex differences in the total mortality rate* due to the sex differences in cause-specific mortality in the same age group. For example, if the contribution to the sex difference (female minus male) in **life expectancy** for ages 50–54 was 1.2 years, and the sex difference in cardiovascular **mortality rates** for ages 50–54 represented 30% of the sex difference in mortality rates of all causes for ages 50–54, then the contribution of cardiovascular mortality for ages 50–54 to sex differences in **life expectancy** would be (1.2 years x 0.30) = 0.36 years.

The following is a general presentation of these calculations adapted from Arriaga (1989), though other closely related methods give similar results (Beltrán-Sánchez & Preston, 2007). For each age group j, the influence on life expectancy attributable to sex differences in mortality for a given age group is Age - Specific Difference in Life Expectancy or $ASD_j({}_ie_x)$, which is a function of the mortality difference for each age group between males and females $(m_j^{male} - m_j^{female})$. We define $Dj = f(m_j^{male} - m_j^{female})$ to represent the total mortality sex difference for age j. Now, this is expanded to represent each age-specific mortality difference for cause of death c: ${}_cD_j = {}_cm_j^{Male} - {}_cm_j^{Female}$. Accordingly, if you sum all the ${}_cD_j$ across all causes you get D_j. This simply means that the difference in total mortality between groups for a given age equals the sum of all differences by cause in that age group.

The aforementioned assumption means that the influence on life expectancy attributable to a sex difference for a cause of death c is proportional: ${}_cASD_j({}_ie_x) = ASD_j({}_ie_x) \times ({}_cD_j/D_j)$. Estimation of the Arriaga decomposition was done using R (Auger et al., 2014).

4 RESULTS

Both figures for Utah and Denmark (Figures 1 and 2, respectively) have age on the x-axis and a y-axis showing the difference in years of life expectancy (LE) between males and females. Each age-specific bar is "stacked" comprising the contribution that each cause of death (the colored components or ${}_cASD_j({}_ie_x)$) provides toward the sex difference in LE at a given age. As males generally have higher mortality rates than females at all ages, the stacked bars show positive values indicating that most causes of death contribute to the female advantage in LE. Negative values occur for some ages where a specific cause of death *reduces* the sex differences in LE that generally favor females. The net height of an age-specific stacked bar (adding all positive and negative values) is the overall contribution, at that age, to the sex difference in LE.

To establish how we interpret the decompositions, we first provide an example assessment for one age- and time-specific results in a single decade (1970–1979) to setup how we interpret the decompositions of sex differences in life expectancy. For this decade at age 65 in Utah, the largest contribution to the female advantage is attributable to diseases of the circulatory system (component). Indeed, it is the differential effects of mortality by cardiovascular disease that is most responsible for the female advantage for ages 45–85.

Several key trends arise by period, age and causes of death that serve to reveal how shifts in the female life expectancy advantage have occurred for both populations. With respect to historical periods, the size of the overall female advantage is smaller in the early decades but then grows substantially in the ensuing years and then shrinks starting in the 1990s.

Figure 1 *Arriaga decomposition of period life expectancy by decade, age and cause of death for males and females in Utah*

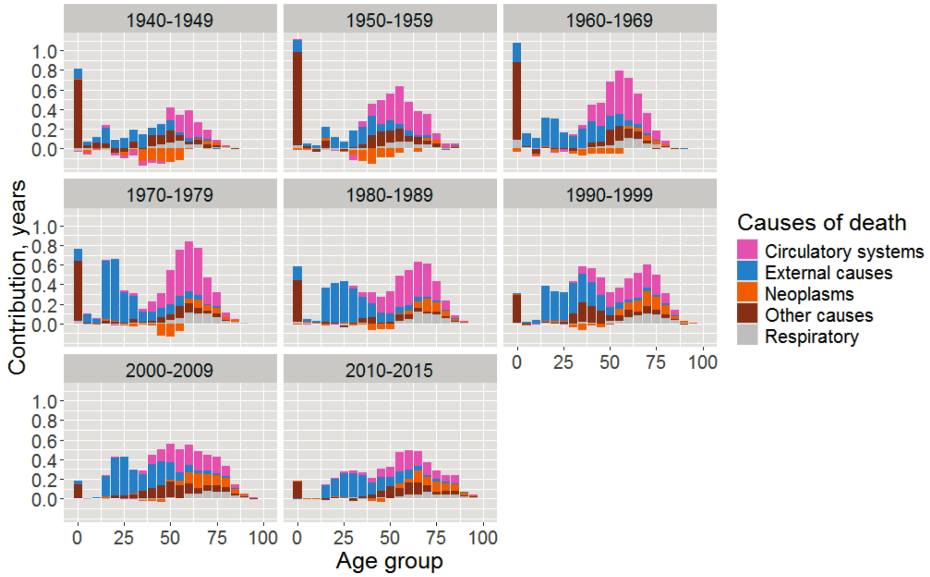
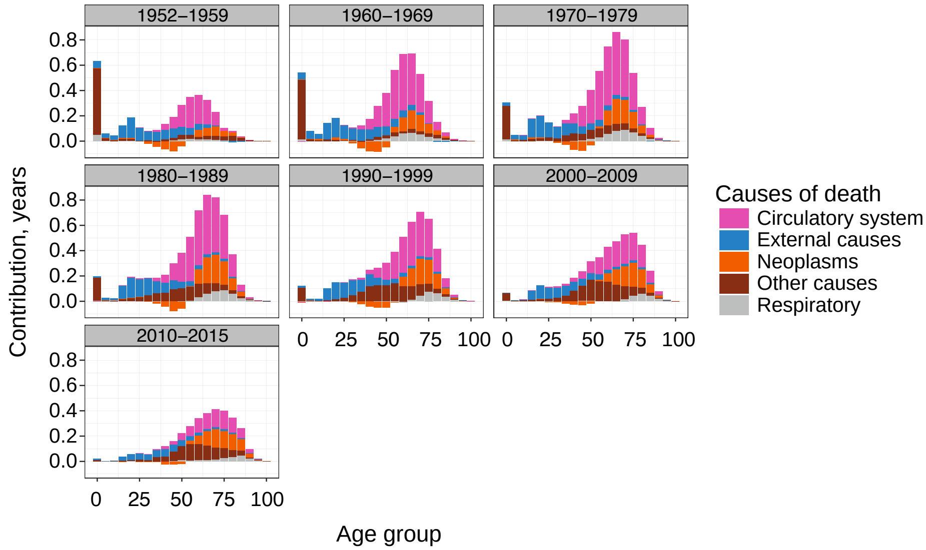


Figure 2 *Arriaga decomposition of period life expectancy by decade, age and cause of death for males and females in Denmark*



The decomposition plots peak around ages 60 to 70 where the mortality rates of males and females on an absolute scale is large but when there are still sufficient years of potential life left to live such that differences can arise. The right tail of the plots has very low values naturally as the number of years of life remaining is falling quickly for both sexes.

With respect to the effects of cardiovascular mortality on sex differences, in the 1940–1949 period for Utah, its contribution to the female advantage arises around age 50 and is the dominant cause of death for all subsequent ages except for age 90 or older. This general pattern persists for all periods though its absolute magnitude declines as the absolute difference in life expectancy between men and women declines. It is likely that the substantially larger effects of cardiovascular mortality (pink), and to a smaller extent death attributable to respiratory diseases (grey), during the years spanning 1940 to 1990 coincides with high smoking prevalence rates among men compared to women. While smoking tobacco played a role, it would be less significant in Utah where tobacco usage is the lowest in the U.S. It is likely that the rising rates of unhealthy weight from the 1960's onward contributed to more cardiovascular deaths for men than women (Parikh et al., 2007).

For Denmark, the prominence of cardiovascular mortality is also present in the first (partial) decade (1952–1959) in accounting for large sex differences in life expectancy. This pattern continues to grow in prominence though its absolute magnitude declines in the 1990s as the sex differential in life expectancy declines here as in Utah. Denmark's and Utah's patterns are also similar in terms of the age at which cardiovascular mortality first contributes to the sex difference occurs, in their forties.

The contribution of neoplasms (i.e., cancer) to sex differences in life expectancy is dissimilar between Utah and Denmark. For Utah, cancers serve to *reduce* the female longevity advantage during the early adult and middle age years. This finding first arose in the 1940's and continued until the new millennium. Around age 60, male cancer mortality, like nearly all other causes, adds to the male deficit in life expectancy.

Denmark generally reveals a similar cancer pattern except the magnitude is considerably larger. Cancer again serves to subtract from the female advantage in life expectancy in the early and middle adult years but this penalty is larger than it is for Utah. After age 60, cancer mortality adds to the male disadvantage but these additions are larger and extend further into advanced ages in comparison to Utah.

The role of external causes of death, including accidents, homicides, and suicides, has considerable effects on life expectancy generally as these deaths tend to occur earlier in life but they also contribute to sex differences since this broad class of death arises disproportionately among males. While the effects of external causes of death of sex differences in life expectancy are present for all decades, it has an outsized effect starting around 1970 with more homicides among men being a major contributor (O'Brien, 2023). This phenomenon accelerated during the 1970's through the 1990's and then declined slightly. While homicides are an important component, the 1960's marked the rise of automobile ownership and access to firearms which led to more motor vehicle accidents and suicides, all of which affected males more than females. For Danes, where miles travelled by car are lower than in Utah and access to firearms is illegal (unless you are hunter), external causes of death consistently add to the male disadvantage where it is concentrated in the younger and middle ages. For the entire period, the largest contributor of most external causes of death in Denmark are male suicides and motor vehicles accidents.

The very large spike occurring during the first years of life reflect the well-known excess risk of infant and childhood mortality for males. Large contributors to this male excess arise from death due to birth defects, prematurity, and infections. For both populations, the magnitude of these early deaths toward the male disadvantage has declined considerably as infant mortality rates overall have declined in the latter half of the 20th century and into the 21st century.

5 DISCUSSION

Sex differences in life expectancy have long favored females but this advantage is not constant over time or across populations. In this paper, we have investigated the underpinnings of this differential as reflected in the contribution that specific causes of death make that give rise to the fact that females outlive males. The populations in question, Denmark and Utah in the United States, share commonalities as both are economically large Western societies (indeed, only certain governments collect and maintain cause of death information for their citizens for many decades) but there are also differences in terms of factors such as lifestyle, family size and access to health care. The years examined in these data also span numerous decades where public health and demographic shifts arise, as reflected in the use of tobacco, access to firearms, and fertility rates.

Despite the differences in the two populations, one of the main conclusions drawn from these analyses is the undeniable influence of cardiovascular mortality. It is fundamental to contributing to the almost impervious female life expectancy advantage in a similar fashion for both Utah and Denmark. Not surprisingly, as mortality from cardiovascular diseases is the leading cause of death in both locales, this source of mortality is the largest contributor to the female advantage despite differences in the two populations and secular changes over the past 60 to 80 years (Hoyert, 2012). While rates of cardiovascular mortality have declined for both sexes, there have been persistently higher rates for men than women (Dimala et al., 2024; Haunsø et al., 2020). While heart and circulatory diseases have been responsive to public interventions (e.g. anti-tobacco ad campaigns, taxation on cigarettes) that have led to lower disease rates, our results nonetheless underline the strong biological forces that persistently increase the risk of cardiovascular mortality for men. Indeed, if this biological liability for men could be eliminated, it would likely do more to narrow the sex life expectancy gap. In a manner, this is happening already because cardiovascular mortality rates are declining for everyone, which means that the dominance of this source of female advantage is steadily diminishing.

While cardiovascular mortality is fundamental to understanding the dynamics of sex differences in the length of life, external causes of death are also central as they occur more often at younger ages where the years forfeited are greater. To the extent that males at younger ages take more risks, often starting around puberty (Owens, 2002), there are again biological bases for external causes of death as contributors to the male disadvantage. This large contribution of external causes is observed in both populations. Our analyses treat all external causes of death as a single category, a group that is quite heterogenous that includes homicides, suicides and a number of unintentional causes (e.g., falls, fires and burns, drowning, poisoning by gases and vapors, and firearms). Nonetheless, what is striking is the similar contribution that external causes make for both Denmark and Utah in adding to the male disadvantage. This is noteworthy because the socio-environmental conditions are very different between the two populations in terms of drug and alcohol use, access to firearms, family size, and government support for health care (Pedersen et al., 2005). While the two sets of decomposition plots share similarities by time and age, it is the case that a more detailed analysis of specific types of external causes of death, which is beyond the scope here, should reveal how the different social contexts yield different types of accidental mortality.

The role of cancer in generating sex differences in life expectancy is different from the other leading causes. We have shown that during the reproductive years, cancer mortality serves to reduce the female advantage, a pattern found in both populations. As cancer is generally presented as a disease closely aligned with aging (Berben et al., 2021), some cancers are more lethal at younger ages such as breast cancer (Gnerlich et al., 2008).

The remarkable decline in infant and child deaths throughout the decades considered here not only served to elevate life expectancy over all but helped to diminish a large male penalty seen in the earlier decades. Indeed, the contribution of any infant cause of death toward a greater female advantage in life span has largely disappeared by the 21st century. Certainly more work is needed to further elucidate how mortality selection (or "culling") of more males at younger ages may serve to graduate more robust males to older ages where their mortality would be lower, and hence more similar to female mortality risks for many potential causes of death (Bruckner & Catalano, 2007; Griffin et al., 2018; van Dijk et al., 2019).

In sum, based on this comparison of Denmark and Utah, the longevity differential favoring females will likely continue but diminish in size. Gains in prevention and treatment of cardiovascular (and likely respiratory) diseases and cancer will contribute to the trend of increasing parity in life expectancy between the sexes. Additional social and public health policies will be needed to address persistent sex differences in external causes of death.

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IV
Lives Under Pressure

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The Rhythm of Death

Seasonality of Mortality in Amsterdam, 1812–1931

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ABSTRACT

Between 1812 and 1931, Amsterdam experienced profound demographic, social, and epidemiological changes that reshaped how, when, and why people died. By tracing seasonal mortality patterns over this time period, trends in the rhythm of death are explored in our study. Using monthly death counts from municipal yearbooks and the Amsterdam Cause-of-Death Database, and applying wavelet power spectrum analysis, we identify both persistent winter excess mortality and key disruptions caused by epidemics. For the period 1856–1891, for which continuous cause-specific data is available, our findings reveal that, although airborne infectious diseases largely shaped the winter mortality peak, excess winter deaths remained evident even after their removal. This suggests the important role of other causes-of-death, such as cardiovascular diseases, which are caused by other factors than seasonal viruses. Beyond these findings, it is argued that environmental exposures, such as temperature and reduced sunlight, alongside social inequalities in shaping seasonal vulnerability should be taken into account in future research.

Keywords: Mortality, Seasonality, Amsterdam

DOI article: https://doi.org/10.51964/hlcs23092

1 INTRODUCTION

The diseases that people contract, and ultimately die from, reflect the conditions in which they lived. The same holds true for when, during the year, they pass away. Mortality is not distributed evenly across the calendar; instead, it follows a rhythm shaped by environmental pressures, economic hardships, cultural practices, and biological vulnerabilities. Studying the seasonality of death provides insights into how historical populations were affected by factors such as food scarcity, temperature extremes, epidemic outbreaks, and social customs related to work, migration, and caregiving. By examining mortality trends in Amsterdam in the 19th and early 20th century, our study seeks to explore these seasonal dimensions of death and their broader implications. In other words, our aim is to investigate how mortality rhythms changed over time and what they reveal about living conditions in the past. Were there consistent seasonal peaks in deaths, and if so, can we hypothesize what forces drove them?

Earlier research has demonstrated that in temperate climates such as Europe, mortality rates tend to peak during winter months, a phenomenon known as excess winter mortality (Fowler et al., 2015). Three main factors help explain why winter remains such a lethal season. First, many viruses, including influenza, follow a seasonal cycle and reach their peak during winter, accounting for a significant portion of these excess deaths. That many other excess winter deaths are from cardiovascular causes provides a second likely factor. Cold temperatures have biological effects that increase the risk of blood clots, coronary thrombosis, and respiratory infections. However, despite large temperature differences between Northern and Southern Europe, winter mortality rates are surprisingly similar. This suggests that people adapt to their local climates, meaning the relative impact of cold weather is felt similarly across regions (The Eurowinter Group, 1997). More recent comparative studies, including those involving very warm countries, suggest that temperature alone does not fully explain excess winter mortality. Reduced exposure to sunlight, and consequently lower Vitamin D production, may also play a related role. Finally, behavioral and environmental changes in winter, such as dietary shifts, decreased physical activity, air pollution, varying humidity, and even cultural events, may contribute as well to excess mortality, but are almost never studied from this perspective (Kinney et al., 2015).

Relatively few historical studies have focused specifically on these kinds of seasonality effects, despite the extensive body of studies examining mortality in the past (Ekamper et al, 2009; Kunst et al., 1991). While many adopt a descriptive approach (e.g., Lee & Son, 2012, on Korea; Williams, 1992, on Sheffield, UK), some employ statistical models, such as Cox proportional hazard models and time series techniques, to analyze seasonal mortality patterns. One key finding is that the seasonality of mortality is not necessarily stable over time. For instance, in Germany (1946–1995), winter mortality persisted but declined in intensity and sensitivity to cold (Lerchl, 1998). In Northern England, infant deaths shifted from winter peaks in the early 19th century to summer peaks by the century's end (Huck, 1997), whereas in 18th-century London, the reverse trend was observed (Landers & Mouzas, 1988). Others have therefore argued that changes in mortality seasonality can signal broader social, environmental, and public health transformations. The reduction of winter excess mortality over the 20th century is often linked to improved heating systems (Lerchl, 1998; Staddon et al., 2014), while the rise of summer infant mortality in the 19th century correlates with declining breastfeeding and increased waterborne infections (Huck, 1997). Public health measures, such as water sanitation improvements in the late 19th and early 20th centuries, likely weakened summer peaks in infant deaths (Macassa et al., 2006; Peltola & Saaritsa, 2019). Additionally, shifts in epidemic disease patterns, such as smallpox seasonality, have been associated with vaccination programs (Krylova & Earn, 2020). In short, seasonal mortality patterns are shaped by a complex interplay of biological, environmental, and societal factors, making their historical analysis crucial for understanding long-term trends in public health.

Our study contributes to our current understanding by taking a long-term perspective (covering the years between 1812 and 1931) to examine how the seasonality of all-cause mortality developed over time. This period was one of profound transformation, marked by industrialization, urbanization, medical advancements, and shifts in public health policies, all of which reshaped patterns of disease and mortality. In particular, Amsterdam underwent the epidemiological transition, shifting from a mortality regime dominated by infectious diseases to one increasingly driven by chronic conditions (Lammertink, 2023; Stalpers et al., 2021). These changes likely influenced not only how people died, but also the seasonal patterns of their vulnerability. Moreover, our study is able to take a closer look at how patterns may have changed during the period 1856–1891 by focusing on deaths due to specific causes such as respiratory diseases, diarrheal infections, or cholera and smallpox epidemics. It allows us to assess more closely whether the decline of infectious diseases altered the seasonal rhythm of death and whether certain causes of death were more sensitive to seasonal influences than others. In doing so, we build on earlier observations that the effects of the assumed decline of infectious diseases and rise in non-infectious diseases as part of the epidemiological transition may require further revisions (Wolleswinkel-van den Bosch et al., 1997).

By examining these seasonal patterns of mortality, we not only explore how environmental, medical, and societal forces might have shaped life and death in Amsterdam but also contribute to a broader historiographical tradition that seeks to contextualize health and disease of ordinary people within their social fabric. It is in this spirit that we dedicate this article to Angélique Janssens, whose pioneering research over the past decades has greatly enriched our understanding of historical mortality and health. As she retires, her legacy continues to inspire scholars in historical demography, reminding us that the study of death is, at its core, a study of life.

2 SOURCES, DATA AND METHODS

For our study we use two types of sources. The first are the Statistical Yearbooks of Amsterdam in which the total number of annual and monthly deaths are reported. While the Statistical Yearbook 1905–1909 (Bureau van Statistiek der Gemeente, 1910, pp. 108–115) covers the information for the period 1812–1908, information about later years are coming from the following individual yearbooks. The second source is the Amsterdam Cause-of-death Database (ACD) (Janssens et al., 2023), which covers the period between 1854 and 1940. However, we limit ourselves to the period 1856–1891 because the analysis depends on a complete sequence of annual and monthly data. Missing information (1855 and from January 1892 to June 1894) therefore preclude the possibility of performing the analysis before or after the study period. Still, although shorter, this period still covers the start of the mortality decline in the 1880s. After removing stillbirths (N = 22,632), observations without address (N = 4,770)[1] and cause of death (N = 4), the remaining death count is 272,344.

To explore how mortality seasonality evolved in Amsterdam, the percentage of deaths in each month and the excess winter mortality is calculated first. The latter is done by calculating the excess winter death index (EWDI) and can be interpreted as the difference between the number of deaths in winter months (December to March) and the average number of deaths in non-winter months (average from August to November of the previous year and from April to July in the year of interest) expressed as a percentage of the latter (Healy, 2003). Next, wavelet analysis is used for examining the periodicity of mortality patterns over time. It allows us to break down a time series into its spectral components, identifying dominant cycles and how they change across different periods (Cazelles et al., 2007; Rösch & Schmidbauer, 2018). In essence, the technique measures the correlation between the mortality data and a set of wavelets: mathematical functions of varying widths that correspond to different periodicities. As these wavelets are moved along the time series, their correlation with the data fluctuates, revealing moments when certain seasonal patterns were more or less pronounced. The stronger the correlation between the death counts and a particular wavelet, the greater the wavelet power, meaning that a particular cycle was a significant feature of the mortality pattern at that point in time.

We apply wavelet power analysis to monthly death counts between 1812 and 1931, focusing first on overall mortality trends before turning to cause-specific patterns in the period 1856–1891. Our aim is to determine whether mortality exhibited clear seasonal rhythms, whether these rhythms remained stable or shifted over time, and how different causes of death contributed to any observed changes. To achieve this, we begin by analyzing all-cause mortality to establish whether seasonality was a consistent feature of Amsterdam's death patterns or if it varied over time. Once we confirm the presence of periodicity, we move step by step to isolate the influence of specific disease categories.[2] First, we remove deaths attributed to cholera and smallpox, two epidemic diseases that caused dramatic but generally irregular

1 Observations without addresses were left out because further analyses of poor, rather poor, rather wealthy and wealthy neighborhoods (not included in this article) required this information.

2 Before the analysis, the data was pre-processed as suggested by Thai et al. (2015). After adjustment for month length, counts which deviated more than 10% from the official statistics were linearly interpolated and transformed by taking the square root. Trends were modelled with local polynomial regression with span parameter 0.5 ("loess" package in R). Finally, the detrended time series were normalized to restrict the range of values between zero and one. The analysis was conducted using the "WaveletComp" library in R (Rösch & Schmidbauer, 2018). The significance tests are based on simulated (N = 15,000) white noise processes.

spikes in mortality. Next, we exclude deaths related to the respiratory system, followed by diarrheal diseases, and then endemic causes, each time observing how their removal alters the seasonal patterns in mortality. This process allows us to assess the contribution of different disease groups to overall seasonality, without conflating their effects. However, the approach is not cumulative: while cholera and smallpox remain excluded throughout, other causes of death are removed one at a time, ensuring that their individual impact on mortality rhythms can still be observed. Only in the final step, when all major infectious causes have been removed, do we analyze the seasonality of non-infectious deaths, to see what patterns persist once epidemic and communicable diseases are no longer a factor.

3 SEASONALITY OF MORTALITY IN AMSTERDAM 1812–1931

Throughout the 19th century, mortality in Amsterdam exhibited significant year-to-year fluctuations, largely driven by epidemics and endemic diseases. Only from the 1880s onwards a gradual decline in mortality began, which accelerated in the 1890s. Figure 1 visualizes how death was distributed across the months per year as a percentage from 1812 to 1931, using a color gradient where earlier years are represented in dark blue and later years in yellow. The difference in distributions becomes smaller and more stable over time (i.e. lines with a yellowish color referring to later years are closer to each other than lines referring to earlier years). However, a distinct seasonal pattern remains evident: in most years, mortality was higher in winter than in summer. This pattern is further confirmed in Figure 2, which illustrates the excess winter mortality index over time. The smoothed trend (dashed line) shows that excess winter deaths were particularly pronounced at both the beginning and the end of the study period, with somewhat lower values in between.

Interestingly, prior to the 1880s, some years saw mortality peaks in the summer instead. Several factors could explain this. For example, cholera outbreaks struck the Netherlands in 1833 and 1849, and similar epidemics may have contributed to summer mortality spikes in other years. Additionally, fluctuations in infant mortality could have played a role. Previous research indicates that excess winter deaths were less common among infants (Janssens & Riswick, 2023), meaning that changes in infant mortality patterns may have influenced the overall seasonal distribution of deaths. After 1900, summer mortality peaks disappear entirely, while winter peaks persist, largely due to seasonal viruses. Influenza and other respiratory illnesses tend to thrive in colder months, though the reasons for this remain a topic of ongoing debate (Huang et al., 2023; Nichols et al., 2021). Notably, influenza epidemics swept through Amsterdam in 1890 and again between 1918 and 1922, while a severe whooping cough outbreak claimed many lives in 1891.

Figure 1 *Percentage of total deaths per month in Amsterdam, 1812–1931*

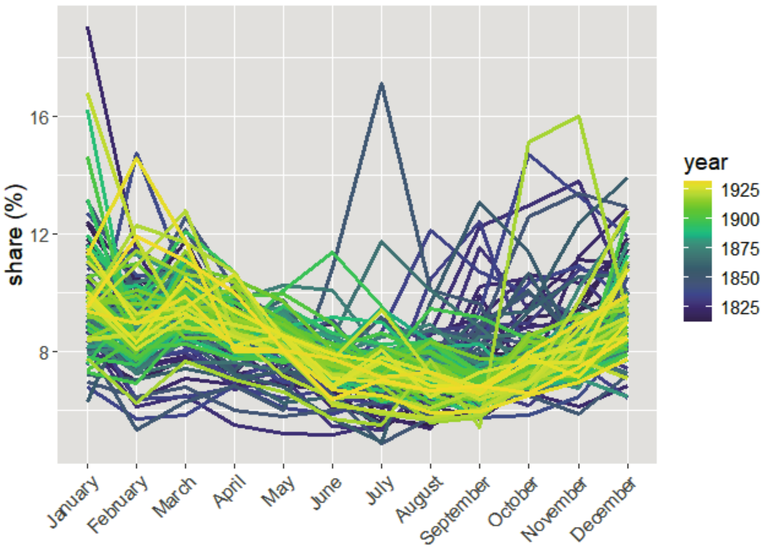
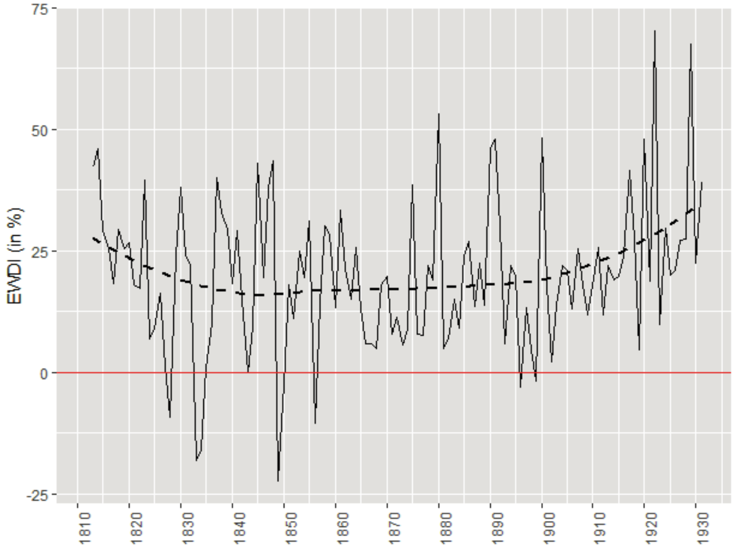
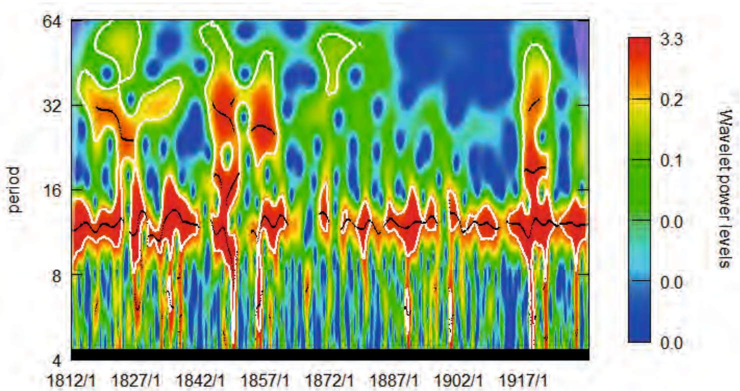


Figure 2 *Excess winter death index (EWDI) by year Amsterdam, 1812–1931*



Note: The dashed line represents smoothed values.

Figure 3 *All-cause mortality Wavelet power spectrum Amsterdam, 1812–1931*



To further explore seasonal mortality patterns in Amsterdam, Figure 3 presents a wavelet power spectrum analysis. The months of our study period (January 1812 to December 1931) are presented on the x-axis, the investigated wavelet lengths in months are on the y-axis. The color scheme reflects the power of the wavelets. Significant wavelet powers are marked with white contours. The black lines, called ridges, point out the periods with the highest power. We find the highest wavelet power (red areas) around month 12 throughout the study period. Although the wavelet power analysis is not able to show when this annual cycle of excess mortality takes place in a year, we know from the previous figures that it reflects a winter peak. However, the wavelet spectrum also reveals disruptions in this pattern. These breaks correspond to periods of lower excess winter death index (EWDI) values observed in Figure 2, suggesting that certain epidemics temporarily altered the typical seasonal mortality trends. Beyond this dominant 12-month cycle, the analysis identifies multi-year fluctuations in mortality. The presence of such multi-year cycles, visible in periodicities extending up to 32 months, might capture the presence of certain diseases that followed outbreak patterns spanning several years rather than adhering strictly to annual seasonal variation. In other words, these longer-term cycles point to the influence of endemic diseases with extended transmission patterns and/or affecting specific age groups. For instance, measles and whooping cough affected mainly children, which likely contributed to variations in mortality over time.

The same is true for intensity of seasonal mortality patterns within the 12-month cycle, which fluctuate across the study period. When patterns deviate from the typical annual cycle this indicates the presence of additional seasonal peaks. Instead of following a strict 12-month periodicity, the wavelet power spectrum reveals shorter cycles of approximately six months, suggesting that mortality peaked not only in winter but also in summer during certain years. This pattern is particularly evident during major epidemic outbreaks. For instance, cholera epidemics in 1833 and 1849 most likely introduced significant summer mortality peaks, disrupting the usual winter-dominated trend. These findings may indicate that while mortality was generally influenced by seasonal factors, severe disease outbreaks could override this structure, leading to increased deaths at different points throughout the year. Still, these seasonal mortality peaks are not really weakening after 1900.

These findings suggest that there may only be a very gradual reduction in the seasonality of mortality over time, which may partly explain why the winter excess mortality is still observed nowadays. Improvements in public health, sanitation, housing, and medical advancements, which could have mitigated the impact of seasonally driven diseases, particularly respiratory infections, seem therefore to have limited effects in our study period. The clearest exception is the influenza pandemic of 1918–1922 which led to mortality surges outside of the expected seasonal pattern, contributing to a breakdown of the standard yearly rhythm. In other words, when taking a long-term perspective we observe that Amsterdam's mortality patterns were highly seasonal, but also shaped by epidemic outbreaks and longer-term disease cycles. Winter peaks dominated throughout the 19th century, major epidemics periodically disrupted these patterns, and seasonality appeared to remain in the early 20th- century, despite advancements in disease prevention and medical care. Yet, to be able to give more detailed answers, in the next section specific diseases are taken into account.

4 SEASONALITY OF CAUSE-SPECIFIC MORTALITY IN AMSTERDAM 1856–1891

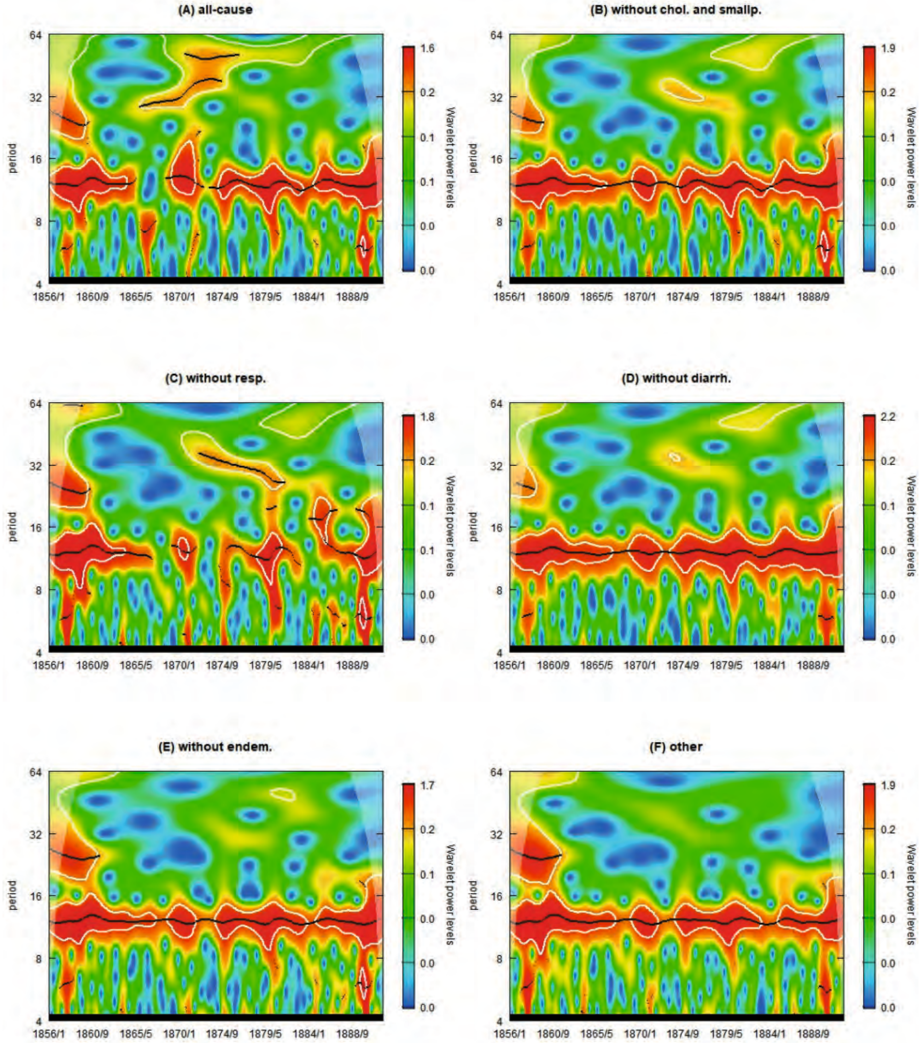
When examining all-cause mortality for the period 1856–1891 in Figure 4 (Panel A), the timeframe for which cause-specific mortality data is also available, earlier conclusions are reinforced. The analysis confirms a strong 12-month periodicity, with deaths consistently peaking in winter.[3] However, with cause-specific data, it becomes possible to identify which diseases were responsible for disrupting this annual pattern in certain years (see Figure 5 which shows the seasonality for specific causes of death). For example, significant breaks in the 12-month cycle are evident during the cholera epidemic of 1866 and the smallpox epidemic between 1870 and 1872. Since both of these outbreaks peaked in summer and had high mortality rates, their disruptive effect on the usual winter mortality pattern is unsurprising.

To further isolate the effects of individual causes of death, we progressively remove specific categories of disease from the analysis. When cholera and smallpox deaths are excluded (Panel B), the annual periodicity becomes more stable, though power remains weak around the epidemic years. This likely reflects the fact that removing individuals who died in an epidemic does not account for the competing risks of diseases: some may have died from other causes later, altering seasonal mortality distributions. These considerations highlight inherent limitations in our approach.

The removal of respiratory diseases (Panel C), a typical winter disease group, further disrupts the dominant 12-month cycle, as the proportion of deaths from non-winter-seasonal causes, such as diarrheal diseases, increases. While a seasonal ridge remains, it is weaker and frequently interrupted. Interestingly, vertical bands of high wavelet power emerge, indicating short-lived mortality spikes across multiple frequencies. These likely correspond to epidemic outbreaks, as seen in measles surges in December 1874 and early 1880, and a cluster of outbreaks in the early 1880s involving scarlet fever, diphtheria, and whooping cough. This suggests that, once respiratory deaths are removed, endemic diseases become the primary drivers of the highly variable seasonal mortality patterns.

3 This latter conclusion cannot be drawn from the wavelet power plots, we performed additional analysis to confirm the excess winter death.

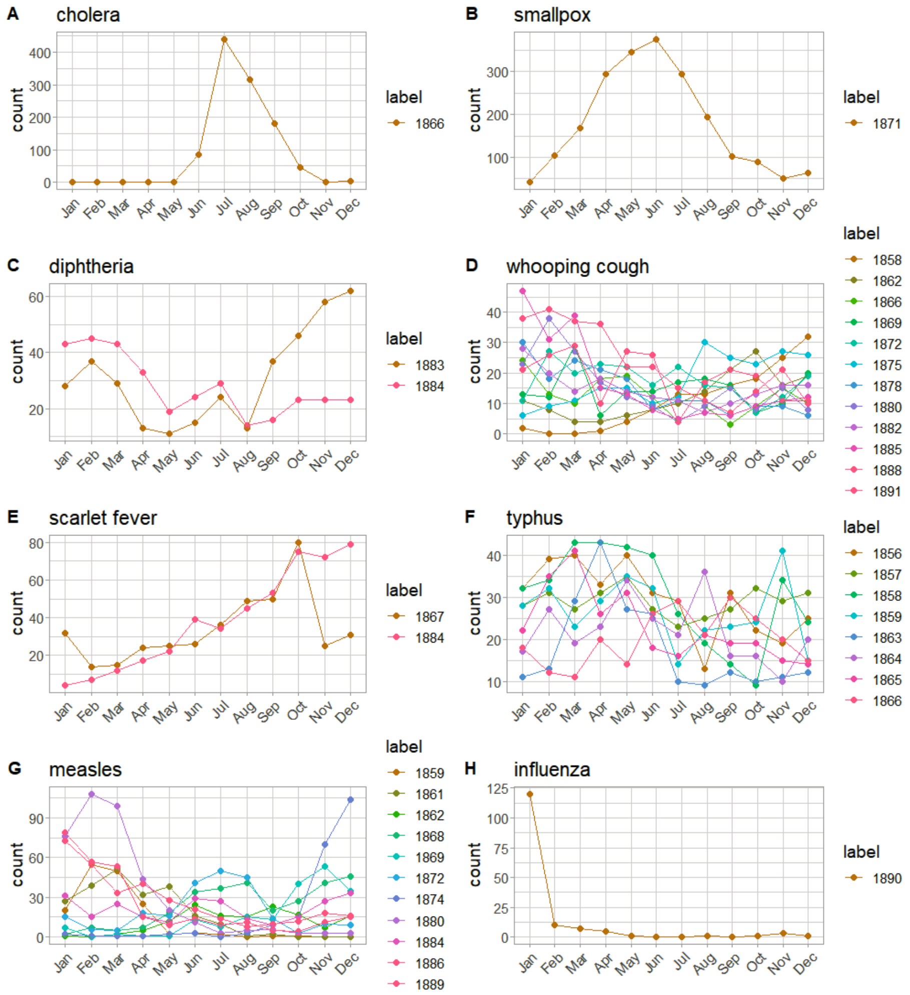
Figure 4 *Wavelet power spectrum for all-cause mortality, excluding several causes-of-death, Amsterdam, 1856–1891*



When diarrheal diseases, a typical summer disease group, are excluded (Panel D), the annual mortality cycle strengthens, as summer mortality declines, making winter peaks more pronounced. The next step, removing endemic diseases (Panel E), such as diphtheria, whooping cough, scarlet fever, typhus fever, measles and influenza, eliminates the longer-than-annual cycles, confirming that these diseases contributed to the multi-year mortality fluctuations observed earlier. The remaining pattern closely resembles that of Panel B, though winter seasonality is even stronger in the 1860s, likely due to the removal of non-winter-seasonal endemic diseases such as typhus, measles, and whooping cough, as well as a major scarlet fever outbreak in October 1867 (see Figure 5 for the seasonality of specific epidemics).

Finally, when all major infectious disease categories are removed (Panel F), the 12-month cycle remains dominant, with only a few interruptions in the late 1860s, early 1870s, and early 1880s. These periods align with disruptions seen in Panel E. These findings are consistent with earlier research on contemporary populations, which similarly identified cardiovascular causes as a major contributor to seasonal mortality and emphasized that temperature alone does not fully explain excess winter deaths. This suggests that, while infectious diseases played a significant role in shaping seasonal mortality, other factors, potentially related to environmental conditions, nutritional deficiencies, or non-epidemic causes, also contributed to the broader rhythm of death in Amsterdam.

Figure 5 *Seasonality of specific causes of death in Amsterdam, 1856–1891*



5 DISCUSSION AND CONCLUSION

This study has shown that mortality in Amsterdam during the 19th and early 20th centuries followed a strong seasonal rhythm, with deaths typically peaking in winter. This pattern was largely driven by airborne infectious diseases. However, winter excess mortality remained visible even after excluding these causes, suggesting that other factors, particularly cardiovascular diseases, also played an important role. Major epidemics disrupted this pattern, introducing additional mortality peaks in the summer months. These findings align with existing research but also point to broader and more complex drivers of excess winter mortality beyond seasonal viruses alone. In turn, a key contribution of this study is the demonstration of how a long-term perspective, combined with cause-specific mortality data, can deepen our understanding of seasonal mortality patterns.

To build on our exploratory work, future research should take a more detailed approach, focusing on specific age groups and what individual-level causes of death can reveal about how, and why, the rhythm of death was shaped. One promising direction is the integration of historical temperature data, which is available in the HISKLIM database (Brandsma et al., 2000; Ekamper et al., 2009). This would allow for an investigation into the physiological effects of cold exposure on cardiovascular mortality, particularly deaths from heart attacks and strokes. Additionally, because the database includes general weather descriptions, researchers could also explore the impact of reduced sunlight exposure, which has been linked to lower Vitamin D levels and potential health consequences. Most interesting, however, is that the effects of cold and sunlight are unlikely to have been experienced equally across socio-economic groups. The urban poor, often living in poorly insulated housing in shadowly alleyways, would have had fewer resources to protect themselves from the cold or benefit from direct sunlight. This raises the possibility that clear differences in seasonal mortality patterns existed between social classes in a city like 19th-century Amsterdam, which may add to the current debates on the development of inequalities in health during this period.

While taking a closer look at environmental factors, such as temperature and sunlight, is important, they do not act in isolation. Future research should acknowledge that behavioural changes linked to social norms and cultural practices may also have shaped seasonal health outcomes. Winter brings subtle shifts in behaviour, such as reduced physical activity, dietary changes, and increased alcohol consumption, that could have contributed to excess mortality. In addition, these effects could differ by gender, age and social economic status. Although historical studies may not always be able to account for these nuances directly, they remain vital to consider when interpreting past patterns. This highlights the broader value of our historical demographic approach in uncovering how environmental, physiological, and behavioural mechanisms behind seasonal mortality evolved over time. By combining long-term cause-of-death data with interdisciplinary insights, we can continue to refine our understanding of the history of health — an endeavour that remains as relevant today as it was when Angelique Janssens' started her pioneering work.

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Epidemics in Motion

Exploring the Interaction between Childhood Diseases in a Norwegian City, 1863–1928

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ABSTRACT

This article explores interactions between measles and whooping cough in Christiania (now Oslo) during the period 1863–1928, using annual morbidity, mortality, and fertility data. Drawing on the ecological interference model proposed by Rohani et al. (2003), we examine whether epidemic patterns shifted from so-called out-of-phase to in-phase dynamics as fertility declined and the pool of susceptibles decreased. Dividing the analysis into two periods based on crude birth rates, we find that during the high-fertility era, disease cycles were typically out of phase. Surprisingly, this dynamic persists even in a period with comparatively lower birth rates, contradicting theoretical expectations. We discuss potential explanations, including population size thresholds for transmission and limitations in the available data. Modest in scope but exploratory in spirit, the study contributes to ongoing efforts - such as those initiated by Angélique Janssens — to use historical health data to understand long-term epidemic dynamics and inter-disease relationships.

Keywords: Ecological interference, Measles, Whooping cough, Case fatality rates, Historical populations

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1 INTRODUCTION

A marked decrease in urban mortality was a key driver of the overall reduction in death rates in late 19th-century Norway. During the early stages of this transition — specifically before and around its onset, between 1860 and 1920 — Norway consistently recorded some of the lowest crude death and infant mortality rates in Europe. Airborne diseases emerged as major contributors to the decline, accounting for over one-third of the reduction observed in Christiania, the historical name of Norway's capital, now known as Oslo. Notably, nearly half of this decrease stemmed from reductions in mortality due to common childhood illnesses such as diphtheria, measles, scarlet fever, and whooping cough (Hubbard, 2002). This underscores the crucial role of declining childhood mortality in the broader urban mortality transition.

The historical interplay between infectious diseases has long shaped patterns of mortality, vulnerability, and survival. With the growing availability of digitized, individual-level cause-of-death and morbidity data, researchers are now in a position to revisit these dynamics with new tools and perspectives. This contribution explores how interactions between infectious diseases — so-called ecological interference — may have influenced epidemic trajectories in the past. We propose that historical time series on reported illness, cause-specific deaths, and births offer a compelling opportunity to explore how one pathogen can constrain or shape the trajectory of another.

This approach builds on recent insights from ecology and epidemiology, but also aligns closely with emerging directions in historical demography and the social history of medicine. It reflects a broader move away from studying diseases in isolation, toward understanding their interaction before, during and after the demographic and epidemiological transition.

This perspective also resonates with the scientific legacy of Angélique Janssens, whose work has significantly shaped the field of historical demography. Her research brought together gender, health, mortality, and the overall impact of infectious diseases (Janssens, 2016). Her leadership in major research projects such as Studying the history of health in port cities (SHiP) and Lifting the burden of disease exemplifies a dedication to interdisciplinary research. It is in this spirit that our study seeks to push methodological boundaries and examine mortality patterns through the lens of inter-disease dynamics.

2 ECOLOGICAL INTERFERENCE

We draw our inspiration from an exploratory exercise by ecology experts Rohani and colleagues, entitled Ecological interference between fatal diseases, published in *Nature* in 2003 (Rohani et al., 2003). In this letter, they present a technically elaborate analysis, but at its core lies the idea that specific infectious diseases interact or "compete". Measles, for example, typically occurs only once in a person's life, due to the combination of initial susceptibility at birth and lifelong immunity after infection. For measles to circulate, a continuous supply of susceptible children is needed. This so-called pool of susceptible children (the population at risk for catching measles) is replenished through births and depleted by measles infections or deaths prior to infection.

If some of these susceptible children are temporarily bedridden with another infection — Rohani et al. use whooping cough (*pertussis*) as an example — they are removed from active circulation. Their illness reduces their social contact, effectively shrinking the susceptible pool for measles. This, in turn, slows the spread of measles. Likewise, during a measles outbreak, whooping cough might spread more slowly for similar reasons. This phenomenon is referred to as ecological interference. Rohani et al. argue that this interaction is not purely theoretical: their modelling suggests that the effect should be detectable, and their data analysis provides empirical indications of its presence.

Building on this framework, our study investigates whether ecological interference between measles and whooping cough can be observed in a historical setting. Specifically, we test two hypotheses (this will be discussed in more detail in section 3):

Out-of-phase epidemic dynamics: During periods of high fertility, when the pool of susceptible individuals is rapidly replenished, measles outbreaks reduce the number of susceptibles available to whooping cough, resulting in alternating or negatively correlated epidemic cycles.

In-phase epidemic dynamics: During periods of low fertility, the slower replenishment of the susceptible pool allows measles and whooping cough outbreaks to occur simultaneously, resulting in positively correlated epidemic patterns.

A second, related line of inquiry draws from the more recent work of Mina and colleagues (Mina et al., 2015), who demonstrate that measles infection can suppress the immune system for up to several years, increasing vulnerability to other infections (see also Mercer, 1990). From this perspective, we might expect measles outbreaks to be followed by increased mortality from other infectious diseases, including whooping cough.

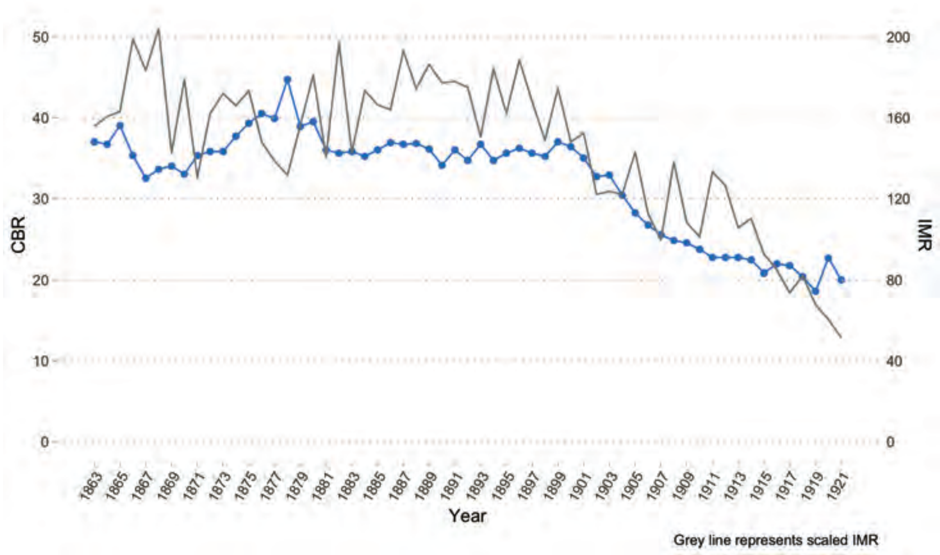
Ultimately, these types of diseases should not be studied in isolation, but in relation to each other. Like Rohani et al. and Mina et al., most authors in the medical sciences stress how rare it is to find data sources robust enough to properly test hypotheses like these. However, historical individual-level cause-of-death data, in combination with morbidity records, might offer opportunities to do so. By exploring these interactions in historical Christiania, we aim to contribute to a growing body of research that seeks to understand disease dynamics through the lens of ecological interaction.

3 CONCEPTS AND DATA

Like Rohani et al., our exploration is based on measles and whooping cough. As stated, measles provides lifelong immunity. Experiencing a measles infection also causes broader immune suppression, which can last for up to two years in those who survive the illness. People who have had whooping cough acquire some immunity to future whooping cough infections (Rohani et al., 2003). Other infectious diseases, such as scarlet fever, pose a slightly different challenge. Immunity after infection is not always lifelong, and the pathogen can circulate without clear clinical symptoms. It is plausible that children sick at home with scarlet fever might reduce measles transmission, but measles might not significantly influence scarlet fever dynamics in return.

We use data from Christiania to explore the ecological interference hypothesis. The dataset was specifically constructed for this study and includes annual counts of births, infant deaths, population figures, counts of measles and whooping cough cases, as well as deaths attributed to both diseases, covering the period 1863–1928.[1] On average, there were 4,591 births per year. The average population of Christiania during this period was approximately 175,000.

Figure 1 *Crude fertility rates and infant mortality rates, Christiania 1863–1928*



1 Data on infant mortality beyond 1921 are lacking.

Morbidity data were drawn from annual medical reports submitted by district physicians. Beginning in 1861, these medical officers were required to report cases of acute diseases with epidemic potential to the city's health commission on a monthly basis. The reporting form included details such as the patient's name, age, occupation, address, diagnosis, date of onset, suspected cause, and treatment administered.[2] This practice coincided with Norway's adoption of its first official nomenclature and cause-of-death certificates (Sommerseth, 2023).

Mortality data were derived from the same medical reports. Until 1874, death counts were based on priest burial registers; from 1875 onwards, they were drawn from doctor-issued death certificates. The medical reports also noted the coverage rate of these certificates, which ranged between 93% and 98% annually when compared to priest-reported deaths.

It is important to interpret these sources with caution. The number of reported infections is likely underestimated, which may result in an overestimation of the case fatality rate.

How can we conceptualize the pool of susceptible individuals in a historical urban setting? Measles has a very limited survival time outside the human body. It requires a continuous supply of new, non-immune individuals to sustain transmission. In populations with low birth rates and minimal immigration of non-immune individuals, the virus cannot maintain itself and will likely disappear before causing a major outbreak.

Following Rohani et al., we use crude birth rates to approximate the recruitment of new susceptibles. A key limitation of using crude rates is that infants must survive their first year of life to become part of the susceptible population: a factor that changed significantly over time. In Christiania, for example, the annual infant mortality rate (IMR) shows a clear downward trend beginning in the early 1900s. While the IMR exceeded 200 per 1,000 live births in the early 1860s and continued to fluctuate for decades, it began to decline more steadily in the twentieth century. By the early 1900s, the IMR consistently fell below 150, and after 1910 the decline became more pronounced, reaching approximately 50 per 1,000 by 1921 (see Figure 1). As a result, not all newborns entered the pool of susceptibles for infections such as measles and whooping cough.

Another crucial consideration is that even after surviving infancy, children remained at risk of dying from other infectious diseases, such as scarlet fever and diphtheria. These should be regarded as competing risks. As a result, estimating the number of children who lived long enough to become susceptible to infections like measles or whooping cough is highly challenging, particularly in contexts where a significant proportion of children succumbed to other diseases before reaching the typical age of infection. In such cases, arriving at a reliable estimate of the susceptible population may be difficult, if not impossible.

Despite relatively high infant mortality, the population of Christiania doubled from 112,000 to 221,000 between 1878 and 1898. This growth was driven by boundary expansion in 1877, a surplus of births over deaths, and the immigration of young people seeking employment. Fertility began to decline in the early 1890s (see Figure 1), leading to a noticeable slowdown in population growth toward the end of the period.

As suggested by Rohani et al., we introduce the concepts of *in-phase* and *out-of-phase* epidemic dynamics to describe the temporal relationship between two diseases competing for the same pool of susceptible individuals. In-phase refers to epidemics of two diseases occurring simultaneously, while out-of-phase indicates alternating outbreaks, presumed to be driven by ecological interference. This raises the question: how can interference be detected in our historical data, and are there signals in our data indicating trends of synchronized or staggered epidemics over time?

When birth rates were high, measles (being more contagious) tended to infect and remove susceptible individuals more quickly, thereby delaying the onset of whooping cough outbreaks. This dynamic resulted in alternating epidemic cycles, or out-of-phase patterns, typically reflected in a negative correlation between the case numbers (or case fatality rates) of the two diseases, as a function of fertility levels. In practical terms, one disease would peak while the other declined. In contrast, when birth rates were low and fewer new susceptibles entered the population, measles and whooping cough outbreaks tended to occur simultaneously (i.e., in-phase) with deaths from both diseases rising and falling together on an annual basis.

2 A pilot project at HistLab, UiT The Arctic University of Norway, is underway to digitize the sources and link them to the Historical Population Register of Norway.

4 EXPLORING ECOLOGICAL INTERFERENCE IN HISTORICAL CHRISTIANIA

Figure 2 shows the annual (squared) case fatality rates for measles and whooping cough in Christiania between 1863 and 1928. The graph illustrates how the two diseases fluctuated over time, with noticeable peaks and declines. While both exhibit epidemic cycles, their timing appears to vary, suggesting potential out-of-phase dynamics, particularly in the earlier period.

While a visual inspection of the epidemic curves suggests potential phase relationships between measles and whooping cough outbreaks, we complement this with a more systematic approach by calculating Pearson correlation coefficients. This allows us to quantify the strength and direction of the relationship between the two diseases across different fertility regimes.

To do this, the analysis is divided into two distinct periods, based on differing levels of crude birth rates: *Period 1*, marked by higher birth rates, and *Period 2*, characterized by comparatively lower birth rates. Period 1 spans from 1863 to 1901 and is characterized by relatively high and fluctuating fertility levels, averaging around 35 births per 1,000 inhabitants. Period 2, from 1902 to 1928, is marked by a sharp and sustained decline in fertility, dropping from 32 to 7.2 births per 1,000 inhabitants in less than three decades.

Within each period, crude birth rates are further subdivided into defined intervals (see Figure 3). For each interval, we calculate a correlation coefficient, representing the strength and direction of the relationship between the fatality rates of the two diseases. The symbols displayed in Figure 3 indicate the number of observations (i.e., case fatality rates) per interval, offering insight into sample size and the robustness of the correlation estimates.

We expect to observe a negative correlation between measles and whooping cough fatality rates during Period 1, when birth rates were high. This would reflect out-of-phase epidemic dynamics, in which one disease suppresses the other by depleting the shared pool of susceptible individuals. In contrast, during Period 2, we anticipate a shift toward a positive linear relationship, indicating in-phase behaviour as the recruitment of susceptibles declines.

Figure 2 *Square of Case Fatality Rates for Measles and Whooping Cough, Christiania 1863–1928*

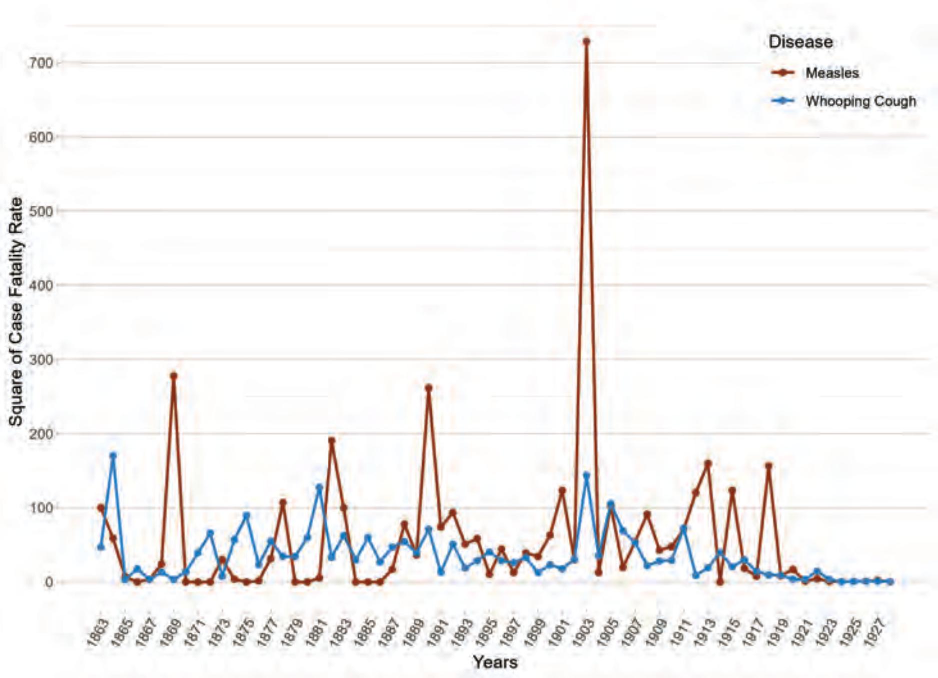
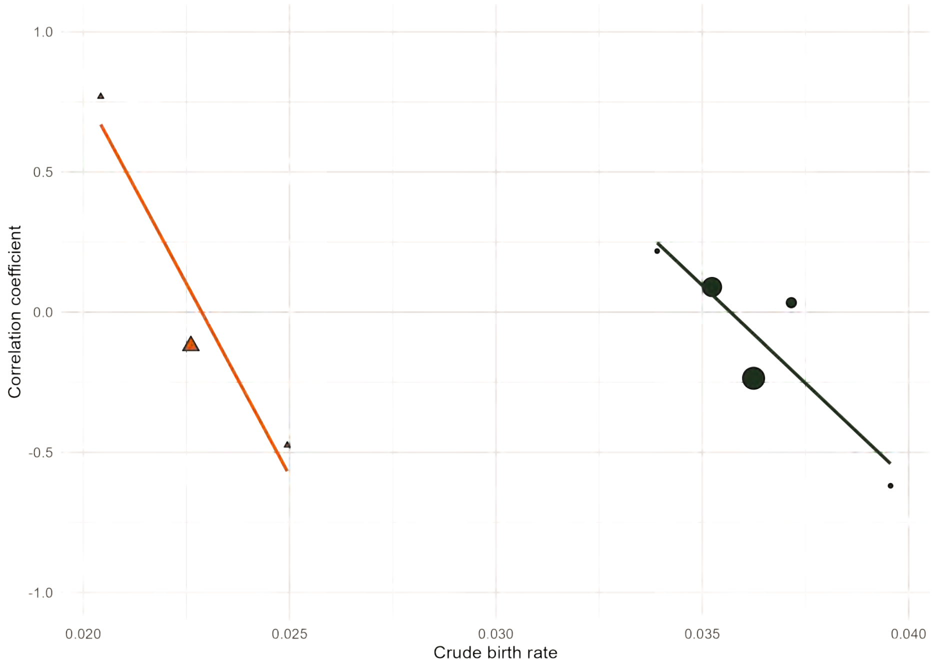


Figure 3 *Correlation coefficients between measles and whooping cough deaths as a function of the crude birth rates. Green symbols/lines refer to the 1863–1901 era; orange symbols/lines represent the period 1902–1928*



Our results show that during the first period — marked by high and fluctuating crude birth rates — the correlation between the two diseases tends to decline. This downward trend suggests that in a high-fertility context, an increase in measles mortality is often accompanied by a decrease in whooping cough mortality, and vice versa, indicating out-of-phase epidemic dynamics. In the second period, marked by a sharp and sustained decline in crude fertility, we observe a similar negative correlation. This is at odds with the findings of Rohani et al., who suggested that in low-fertility settings, epidemic patterns for measles and whooping cough tend to align annually, resulting in in-phase dynamics.

5 CONCLUSION AND DISCUSSION

This exploratory analysis set out to investigate the interaction between two major childhood infectious diseases in historical Christiania, with a particular focus on how fertility dynamics shaped their epidemic patterns. One of the central insights to emerge from this study is that examining infectious diseases in isolation may obscure the complex ecological relationships that govern epidemic behaviour.

Our findings support this view. In the first period (1863–1901), marked by relatively high and fluctuating crude birth rates, we observe a downward trend in the correlation coefficients between measles and whooping cough fatality rates. This suggests an out-of-phase dynamic, where increases in mortality from one disease tend to coincide with declines in the other. In the second period (1902–1928), characterized by a sharp and sustained decline in fertility, we observe a similar negative correlation, contrary to the expectations derived from the ecological interference model developed by Rohani et al. Their work predicts a shift toward in-phase dynamics in low-fertility settings, as both diseases would draw on the same limited pool of susceptibles at the same time.

One potential explanation for this discrepancy lies in the demographic context of Christiania. It is plausible that the city's total population was simply too small to sustain continuous endemic transmission. As Mercer (1990) has argued, measles may require a minimum population of around

7,000 individuals for persistence, and potentially 250,000 to support truly endemic transmission. Christiania's population may not have met this threshold, thereby limiting the ecological space in which a clear phase shift could emerge.

While the ecological interference model remains a powerful theoretical framework, our analysis does only represent a simplified approximation. A more nuanced understanding of epidemic dynamics would require morbidity data at a higher temporal resolution (preferably weekly or monthly), allowing for the inclusion of key epidemiological parameters such as contact rates, latency, infectious periods, and recovery times. Population structure also remains a critical factor in determining whether diseases persist or fade out.

Finally, our reliance on crude fertility rates introduces a methodological limitation. These rates encompass the entire population and do not account for age-specific fertility or infant mortality, both of which are essential to estimating the true number of children entering the susceptible pool. Future research should seek to incorporate age-specific fertility rates and survival adjustments in order to generate more realistic estimates of susceptibility. Moreover, the current dataset, while valuable, does not fully encompass a long enough time span to capture the full "arc" of fertility transition — from high to transitional to low fertility. Extending the temporal scope of the data would allow for a more nuanced analysis of how changes in population structure and birth dynamics shaped epidemic patterns over time. A refined demographic approach would contribute to a deeper and more accurate understanding of disease dynamics in historical urban populations.

This study, modest in scope and exploratory in spirit, aligns with the initiatives led by Angélique Janssens to continue the systematic collection of data on illness and causes of death.

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Estimating Mortality From the Influenza Pandemic of 1918–19 in Suriname and the Dutch Caribbean

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ABSTRACT

This paper reassesses the extent of the 1918–19 influenza pandemic in the former Dutch colonies of Suriname and the Dutch Caribbean islands. Although both colonies were under Dutch rule, local context, statistical registration practices, and available source material varied considerably. We use data derived from the Colonial Reports (*Koloniale Verslagen*) and from the recently digitised civil registration data. Whenever possible, we apply the cause-specific excess mortality approach to estimate the number of fatalities caused by the epidemic. Alternatively, all-cause excess mortality will be calculated. We estimate that the 1918–19 influenza caused about 2,200 deaths in Suriname and around 210 deaths on the Dutch Caribbean islands. In Suriname, contract labourers from the Dutch East Indies and from British India had higher influenza-related mortality rates than the creole population originating from the African continent. As influenza was endemic in Suriname, the creole population might have been better protected than the Asian contract labourers against the 1918–19 influenza.

Keywords: 1918–19 Influenza pandemic, Suriname, Curaçao, Mortality, Colonial reports, Pandemic, Spanish flu

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1 INTRODUCTION

The recent COVID-19 pandemic sparked renewed interest in the characteristics, spread, and mortality patterns of the 1918–19 influenza pandemic (Athukorala & Athukorala, 2022), which was characterized by high levels of morbidity and mortality particularly among young adults (Johnson & Mueller, 2002). It is estimated to have affected 500 million persons, about one third of the global population at that time and caused between 40 and 100 million deaths (Johnson & Mueller, 2002; Rijpma et al., 2022).

Whereas much data has been collected and much contemporary research conducted on the impact of the 1918–19 influenza in Western countries, less is known on how much non-Western countries in general and colonies in particular were affected by the pandemic. The Kingdom of the Netherlands is a good example in this regard. It is estimated that around 41,000 individuals died from the 1918–19 influenza in the Netherlands, most of them during the autumn wave of 1918 (Quanjer, 1921; Rijpma et al., 2022). Estimates for the former Dutch colonies, the Dutch East Indies (present day Indonesia), Suriname, and Curaçao and its dependencies[1], in contrast, are much less precise or even lacking. For instance, modern fatality estimates for the Dutch East Indies range from 1.5 million for the entire colony (Brown, 1987) to 4.3 million for the most populated island of Java alone (Chandra, 2013). For Suriname and Curaçao and its dependencies no modern estimates of the number of affected persons or fatalities exist at all.

In this article, we will estimate mortality from the influenza pandemic of 1918–19 in Suriname and the Dutch Caribbean. We will estimate excess mortality, mortality rates and the total number of fatalities for each colony. The quality of data collection in colonial contexts is typically lower than in the colonizing countries and is often divided by ethnic lines. Special emphasis is therefore given to the possibilities and challenges of the different types of population statistics available in each colony. We use official data from the Colonial Reports (*Koloniale Verslagen, KV*) publicly available at the time of the pandemic, and digitised civil registration data that were recently collected by the Historical Database of Suriname and the Caribbean project (HDSC).

2 BACKGROUND

2.1 THE POPULATION OF SURINAME AND THE DUTCH CARIBBEAN IN THE EARLY 20TH CENTURY

At the beginning of the 20th century the Kingdom of the Netherlands included two colonies in the Americas that were shaped by the European colonisation: Suriname, located on the North Coast of South America, and Curaçao and its dependencies (Aruba, Bonaire, Saba, Sint Eustatius, and Sint Maarten), consisting of six small islands located, as two groups of three islands, roughly 1,000 miles apart, within the Lesser Antilles in the Caribbean. In Suriname and Curaçao and dependencies up to the abolition of slavery in 1863 the main legal distinction was between the free and enslaved population. With the abolition of slavery all residents, in principle, were subject to the same legislation and had the same rights and obligations.

Since the mid-19th century, the population of Suriname had become ethnically highly diverse. In addition to the established population consisting of people of European, African, and Amerindian descent, large numbers of migrants from Madeira, China, British India, and the Dutch East Indies had been contracted to work on the Surinamese plantations in the second half of the 19th and the beginning of the 20th century. From 1906 onwards, the colonial reports presented statistical information of the population by *landaard*, i.e. by national origin or ethnic group (Europeans, people from Dutch East India, people from British India, Natives (*inboorlingen*), Others, and from 1911 onwards also Chinese). The term "Natives" incorrectly referred to the population of African descent and was restricted to the creole population that was under the authority of colonial rule. It did not cover the maroon population who were the descendants of enslaved Africans who escaped from the plantations and settled in the

1 "Curaçao and its dependencies" was the official name used for the colony including the larger Southern Caribbean islands Curaçao, Aruba, and Bonaire, and the smaller Leeward islands Saba, Sint Eustatius, and Sint Maarten. We will use this term to indicate this administrative region within this paper.

inland of Suriname, as they were not registered by the colonial government during the study period. Because of its incorrect terminology, instead of the term "Natives" in this article we use the term "Creole" to describe the largest population group of the colony.

At the end of 1917, the population of Suriname was estimated to be around 92,000, with roughly 40% living in the capital and only city Paramaribo. The largest population groups were the creoles originating from the African continent (52,000) followed by the contract labourers from British India (25,000) and from the Dutch East Indies (10,000). People belonging to the 'Other' category, which included the migrants from Madeira and the Middle East, represented 3.3% of the total population, whereas persons from European and Chinese descent formed around 1% each of the population (KV, 1918, II, Supplement A, Table b).

Unlike Suriname, Curaçao and dependencies were not plantation colonies and therefore did not experience the immigration of Asian contract workers. In the Colonial Reports, no information of the ethnic background of the population is provided, but the population groups were classified according to their place of birth and more than 97% of the population was born in the Caribbean, most of them from African descent. Less than 1% of the population was born in Europe. At the end of 1917, the population of Curaçao and its dependencies was estimated to be around 58,000, with roughly 60% living on the main island Curaçao, followed by Aruba (16%) and Bonaire (12%).

2.2 THE 1918–19 INFLUENZA PANDEMIC IN SURINAME AND THE DUTCH CARIBBEAN

2.2.1 SURINAME

Suriname had been affected by influenza epidemics in the 19th century, and influenza was endemic in the early 20th century. According to the Colonial Reports of 1919, the influenza pandemic entered the country at the Eastern border with French Guiana in mid-November 1918 and by the end of the year there were large numbers of infected persons and approximately 500 fatalities (KV, 1919, II, col. 18). The 1920 Colonial Reports stated that the "Spanish influenza which had affected the country at the end of last year and claimed many victims, especially among the lower classes of the population, ended in March" (KV, 1920, II, col. 15). No further statistics on the number of infected persons or fatalities were provided.

As Dutch Guiana, Suriname was included in a publication of the English Ministry of Health which aimed to report on the global character of the 1918–19 influenza pandemic. It reported monthly details of fatalities for 1919 with 48 fatalities in January, 116 in February, and 7 in March 1919 (Ministry of Health, 1920, p. 342).

2.2.2 CURAÇAO AND DEPENDENCIES

Neither the Colonial Reports nor *Amigoe di Curaçao*, the leading newspaper, reported comprehensively on the 1918–19 influenza. The Colonial Reports of 1919 stated that for the most populated island Curaçao, "the state of health was for most of the year not unsatisfactory. In October and November there was a severe influenza epidemic, which however was quite benign" (KV, 1919, III, col. 9). Elsewhere in the report, however, it is mentioned that in the military hospital 36% of the personnel was admitted for treatment, which was attributed to the influenza epidemic (KV, 1919, III, col. 6).

On 26 October 1918, *Amigoe di Curaçao* informed that the influenza epidemic had affected Curaçao, and probably also Aruba, and that the authorities had taken preventive measures to control the spread of the disease. On 23 November 1918, it reported "that yesterday official sources stated that the influenza epidemic had almost come to a standstill", but that there were still patients recovering and caution should be exercised. It reported that in the urban district Willemstad between 24 October and 20 November a total of 100 persons had died, probably most of them due to influenza. In its edition of 14 December 1918, it reported the deaths of two re-infected persons.

For the other islands the Colonial Reports mentioned 31 influenza deaths on Aruba and 37 deaths on Bonaire related to the influenza epidemic (KV, 1919, III, col. 10). On the smaller Leeward islands, no incidences of influenza were reported for 1918, but Sint Maarten and Sint Eustatius each reported two influenza deaths for February 1919, and Saba counted three influenza deaths in March and April of the same year (KV, 1920, III, col. 11–12).

3 DATA

In the Dutch colonies, in principle the same administrative ethos and organisational rules were applied, and several Dutch administrative structures, institutions and procedures dealing with governance, public administration, health, and education were introduced. These included a statistical service, a civil registration system, and a health service system. In practice, there was a considerable difference in the introduction of those principles and practices, affecting the availability of data that can be used to measure the incidence and fatalities of the 1918–19 influenza epidemic.

From 1848, the Dutch government had to inform parliament annually about the situation and conditions in the colonies. Over the years these annual reports had a few different official titles but were generally referred to as the Colonial Reports. They contained secondary statistical information on all aspects of relevance to the management of the colony: population and its movements, security, military and the judiciary, education, health, trade, agriculture, mining, etc., and are available for the Dutch East Indies (I), Suriname (II), and Curaçao and its dependencies (III).

3.1 SURINAME

Suriname had a well-established statistical data collection system based on the civil registration and several other registers kept by different governmental agencies. During 1913–1921 in Suriname six nationality or ethnic groups were distinguished. The main groups are the creoles, the contract labourers from British India, and the contract labourers from the Dutch East Indies, with very small numbers of Other, Europeans, and Chinese.

Mortality data were disaggregated by location, sex and age, and date of occurrence. From 1904 to 1919, sex disaggregated causes of deaths for the capital and only city Paramaribo were recorded and published in the local media and the Colonial Reports (see for instance KV, 1919, II, Appendix H). As the influenza mortality data of Paramaribo do not indicate the ethnic group of the deceased, the three smallest population groups (amounting to 5.3% of the total population), for statistical reasons, are merged with the creoles who accounted for the large majority of the population in Paramaribo (about 90%), forming a synthetic Creoles+ group. Using the influenza and related mortality for Paramaribo, and the data from the migrant groups the number of deaths and the mortality rate for the Creoles+ is obtained.

In addition, from 1909 to 1919 data on the causes of deaths of the labourers from British India and the Dutch East Indies who were under contract in that particular year were produced (see for instance KV, 1919, II, col. 26). Most of these contract labourers lived in the rural districts, which comprised of all parts of Suriname except for the capital Paramaribo, and which were dominated by plantation agriculture. Those statistics do not include the larger population of migrants from British India and the Dutch East Indies who had stayed in the colony after their contract ended after typically five years. The total number of contract workers from British India decreased rapidly in our study period because on 17 March 1917 the British Government cancelled the treaty allowing the immigration of free labourers from India. Since 1916 therefore only labourers from the Dutch East Indies were recruited and their number increased considerably. It is possible to use these causes of death data to estimate the fatality numbers and mortality rates of the 1918–19 influenza in Suriname, for the total population, the main location (Paramaribo and the districts), and the main ethnic groups for 1918 and 1919. Population information disaggregated by age is not available for Suriname, which makes it impossible to calculate age-specific mortality rates.

In addition to the information provided in the Colonial Reports, we use the recently digitised and transcribed death certificates of the capital Paramaribo for the period 1916–1920 (van Oort et al., 2024).[2] These certificates have been transcribed within the scope of the HDSC project which aims to publish all Surinamese civil certificates for the period 1828–1950. The transcription process rests on the help of hundreds of engaged citizen scientists using an online transcription platform (hetvolk.org). The death certificates do not contain causes of death, but using the age distribution at death will allow some inference to be made on the magnitude of the fatalities caused by the 1918–19 influenza.

2 The published death certificates only cover the period until 1915 so far. For the period until 1928, the death certificates have been transcribed but not yet controlled. Because we are only interested in sex and month and age of death, we are certain that the missing controls do not affect our results. The death certificates of the period until 1928 will be published with the National Archives of Suriname once the controls have been carried out.

They also allow us to identify periods of high numbers of excess deaths and to judge the quality of information provided in the Colonial Reports.

3.2 CURAÇAO AND DEPENDENCIES

For Curaçao and dependencies we use aggregated death statistics as published in the Colonial Reports. In addition, the digitised civil registration data allow us to examine the incidence of influenza in greater detail. Population information disaggregated by age is not available for Curaçao and dependencies, which makes it impossible to calculate age-specific mortality rates.

It is expected that the available data somewhat underestimate the total number of deaths because there was no legal requirement to register deaths in Curaçao and dependencies (Waterman, 1919). Furthermore, the 1921 Colonial Reports note that the so called *begrafenisverordening* (burial ordinance) was not implemented, which implied that all provided statistics in the reports were based on the assessments of doctors and civil servants rather than on reliable causes of death statistics (KV, 1921, III, col. 9). Nevertheless, the number of individual death certificates approximately matches death statistics reported in the Colonial Reports, which indicates that the use of these sources will produce similar reliable outcomes.

4 METHODS

In this article, the disease-specific excess mortality approach will be used to estimate the number of fatalities caused by the epidemic. Whenever possible, the excess mortality of the influenza-linked diseases, tuberculosis, organic heart diseases, bronchitis and pneumonia will also be considered. Ideally, the number of persons affected by the diseases during the period in which the epidemic is active will be compared with a period before and after. However, it is not always possible to have information of the period after the epidemic. In Suriname for the contract labourers no causes of death statistics were published after 1919.

For the population of Paramaribo and the two contract labourer groups from British and Dutch India, disease specific causes of death statistics for 1918 and 1919 will be compared with the period 1913–1917. To calculate influenza excess mortality rates, we use the end of year population of 1918 which is available by ethnic group in the Colonial Reports. For Curaçao and dependencies, disease-specific information is not available and the all-cause mortality for 1918 and 1919 will be compared with the averages of 1913–1917 as reference.

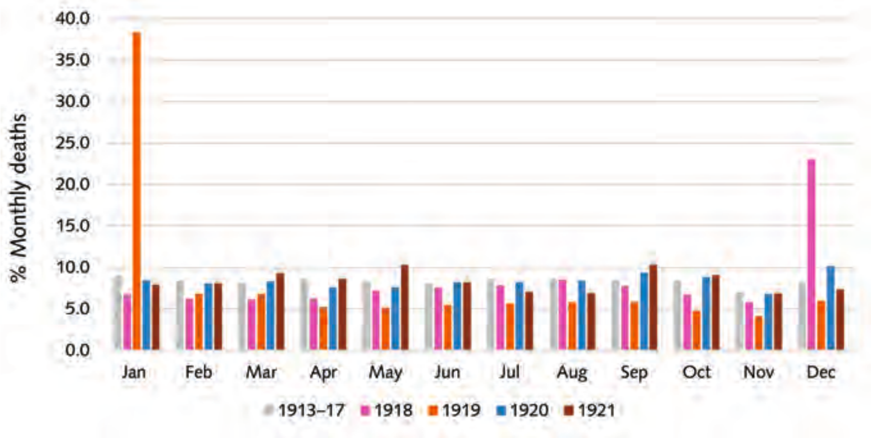
Based on the results of the first (modern) population census of Suriname in 1921 it is known that the population data used in the preparation of the statistical information for the years 1913–1919 were under-estimates. Moreover, during the period there were also British Indian contract labourers from neighbouring countries, mostly present-day Guyana and Trinidad and Tobago, in Suriname looking for employment or transport back to India. These persons were separately registered by the office of the Agent General, and for 1918 and 1919 their numbers were estimated to be around 1,900. In the reference population they were added to the number of free workers from British India.

5 RESULTS

5.1 SURINAME

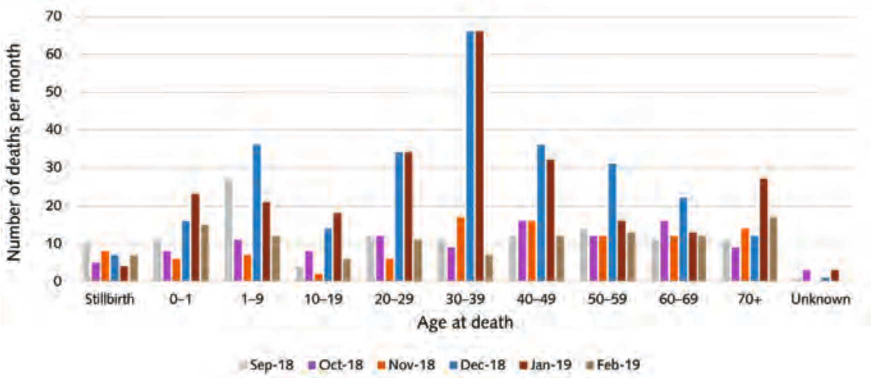
To identify the number of waves of the epidemic in Suriname, we used two different sources: The Colonial Reports of Suriname (see Figure 1) and the recently transcribed Paramaribo death certificates (see Figure 2). Both sources indicate high levels of excess deaths in December 1918 and January 1919. Figure 2 suggests that those aged 20–29, 30–39, and 40–49 were confronted with the largest mortality peaks which is typical for the 1918–19 influenza. In addition, the data indicate that excess deaths were larger among the male than among the female population of Paramaribo (not shown here).

Figure 1 *The average monthly number of deaths of the period 1913–1917 and the years 1918–1921 for Suriname*



Sources: Colonial reports of Suriname, 1913–1921.

Figure 2 *Monthly number of deaths by age group in Paramaribo, for period September 1918 to February 1919*



Source: Paramaribo death certificates.

Table 1 provides an overview of the average number of deaths caused by influenza and related diseases for the reference period 1913–1917 and for the years 1918 and 1919 for the population of Paramaribo and the contract workers from British India and the Dutch East Indies. The number of influenza-related deaths is indeed significantly larger for the periods under study. Among the population of Paramaribo, the number of influenza-related deaths increased from an average of 279 in the years 1913–1917 to 456 in 1918 and 393 in 1919. As most contract workers lived in the districts and experienced higher numbers of influenza-related deaths in 1919 than in 1918, this indicates that the pandemic spread from the (port) city to the countryside.

In Table 2, the distribution of the reference population is presented, the estimated number of deaths caused by the influenza pandemic for 1918 and 1919, and the estimated total influenza mortality rate per 1,000, by ethnic group and location. The total number of influenza fatalities in 1918 was 743 cases, nearly 50% more than the number reported in the Colonial Reports of 1919, "appearing in November it caused, till the end of the year, about 500 deaths" (KV, 1919, II, col. 14). The number of fatalities in 1919 is nearly twice (1,460) as much as in 1918; this is due to an increase of mortality in the districts (1,313 fatalities against 545 in 1918). Using the end of year population of 1918, the mortality rate of influenza and related diseases for the period November 1918–March 1919 for Suriname is 20.6 per 1,000; the corresponding rate for Paramaribo and the districts is 9.3 per 1,000 and 26.7 per 1,000, respectively. For the ethnic groups, the values are 7.6 per 1,000 for the Creoles+, 34.7 per 1,000 for the population originating from British India, and 42.7 per 1,000 for the population originating from Dutch East India.

Table 1 *The average number of deaths of the period 1913–1917 and the number of deaths in 1918–1919 caused by influenza and related diseases of residents of Paramaribo and plantation contract workers from Dutch East Indies and British India*

Data type & source	**Mortality data**								
	Paramaribo			Dutch East Indians			British Indians		
Cause of death	Average 1913–17	1918	1919	Average 1913–17	1918	1919	Average 1913–17	1918	1919
Influenza	2	48	127	1	42	145	3	25	56
Pulmonary Tuberculosis	116	97	88	2	4	10	5	2	3
Organic heart diseases	87	160	109	0	1	0	1	0	0
Acute Bronchitis	13	12	11	NA	NA	NA	NA	NA	NA
Chronic bronchitis	10	13	8	NA	NA	NA	NA	NA	NA
Bronchitis	NA	NA	NA	1	1	32	2	0	8
Pneumonia	51	126	50	0	2	27	1	2	6
Total Influenza & related deaths	279	456	393	4	50	214	12	29	73
Unknown causes	111	117	105	13	16	9	13	16	9
All other causes	679	783	936	16	20	10	38	12	36
Total deaths	1,068	1,356	1,434	37	86	233	63	57	118

Source: Colonial Reports of Suriname, 1914–1920.

Table 2 *Distribution of the reference population at the end of 1918, the estimated number of influenza deaths for 1918 and 1919, and the mortality rate by ethnic group and location*

Reference population 1918								
Ethnic group	Creoles+		British Indian origin		Netherlands Indian origin		Total	
	Number	% Grand total	Number	% Grand total	Number	% Grand total	Number	% Grand total
Paramaribo	35,322	33.06	1,671	1.56	284	0.27	37,277	34.89
Districts	23,636	22.12	34,017	31.84	11,911	11.15	69,564	65.11
Suriname	58,958	55.18	35,688	33.4	12,195	11.41	106,841	100
Estimated number of influenza deaths in 1918 and 1919								
Ethnic group	Creoles+		British Indian origin		Netherlands Indian origin		Total	
	1918	1919	1918	1919	1918	1919	1918	1919
Paramaribo	179	91	14	46	4	10	198	147
Districts	114	61	310	867	121	386	545	1,313
Suriname	293	152	325	913	125	396	743	1,460
Estimated number of total influenza deaths and total influenza mortality rate per 1,000								
	Creoles+		British Indian origin		Netherlands Indian origin		Total	
Ethnic group, Location & Category	Number	Mortality rate per 1,000	Number	Mortality rate per 1,000	Number	Mortality rate per 1,000	Number	Mortality rate per 1,000
Paramaribo	270	7.6	60	35.9	14	*	345	9.3
Districts	175	7.4	1,177	34.6	507	42.6	1,858	26.7
Suriname	445	7.6	1,237	34.7	521	42.7	2,203	20.6

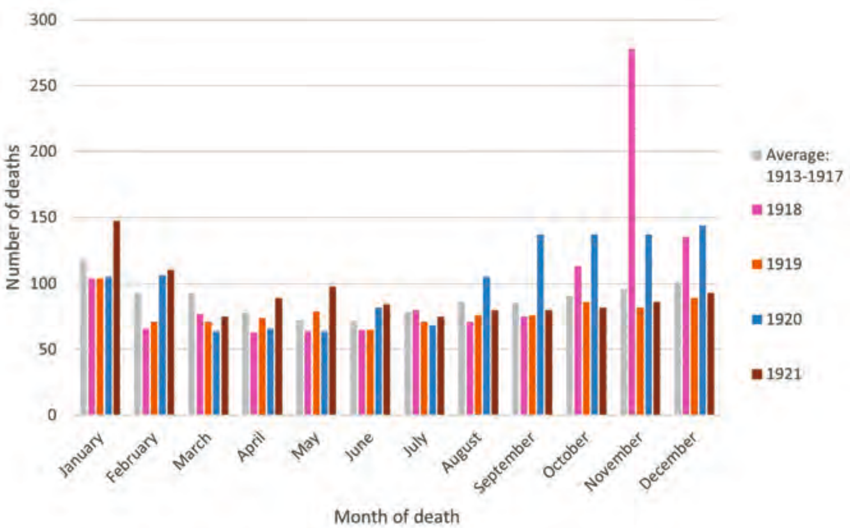
Sources: Colonial Reports of Suriname, 1918–1919; own calculations.

*Note: * Numbers too small for meaningful calculations.*

5.2 CURAÇAO AND DEPENDENCIES

There is little information on the number of waves, or the fatalities caused by the pandemic. Figure 3 shows the total number of deaths per month based on the death certificates of Curaçao, Aruba, Bonaire, Saba, and Sint Eustatius (on the certificates for Sint Maarten month of death is not available). The number of deaths started rising in October and peaked in November when it almost tripled compared to the normal rate. However, from the figure we can deduce that the number of deaths remained somewhat elevated in December as well. The elevated number of deaths a year later, in autumn 1920, is ascribed to a measles outbreak on Curaçao and Aruba which resulted in bronchopneumonia as leading cause of death according to the Colonial Reports (KV, 1921, III, col. 10).

Figure 3 *Number of deaths per month for 1913–1917 (average), 1918–1921, Curaçao and dependencies*



Source: Death certificates of Aruba, Bonaire, Curaçao, Saba, and Sint Eustatius.

Table 3 *Average number of deaths in the months October, November, and December of the period 1913–1917, and the number of deaths and excess deaths in the months October, November, and December of 1918 by age groups in Curaçao*

	Age groups								
Average number of deaths 1913–17	0	1–9	10–19	20–29	30–39	40–49	50–59	60–69	70+
October	11	3	2	6	5	5	5	5	8
November	13	4	2	5	7	5	7	5	10
December	16	5	3	8	6	5	7	6	8
Number deaths 1918	0	1–9	10–19	20–29	30–39	40–49	50–59	60–69	70+
October	13	2	5	13	10	2	6	4	6
November	27	17	18	36	25	11	15	8	14
December	21	5	3	5	3	7	6	7	14
Excess deaths 1918	0	1–9	10–19	20–29	30–39	40–49	50–59	60–69	70+
October	2	0	3	7	5	0	1	0	0
November	14	13	16	31	18	6	8	3	4
December	5	0	0	0	0	2	0	1	6

Source: Curaçao Civil Registration Dataset 1913–1918.

Table 3 deals with the situation in Curaçao only and shows that in December 1918 there were no excess deaths in the age groups 1–9 to 30–39; hence it is unlikely that the 1918–19 influenza was active during that month. The age distribution of deaths for October, and especially for November, shows high frequencies of death in the middle age groups consistent with the 1918–19 influenza epidemic. Accordingly, the influenza pandemic was most likely active in October and November 1918 only.

The available death certificates additionally allow us to construct monthly mortality trends. The timing of the 1918–19 influenza was relatively similar for Curaçao, Aruba, and Bonaire. On Aruba and Bonaire, the pandemic seemed to have peaked in November and December and was not active in October. For these islands, the number of excess deaths for November and December combined (41 and 32) are slightly different compared to the reported influenza deaths in the Colonial Reports (31 and 37). The excess mortality peak on Sint Eustatius during the same period could also reflect an outbreak of influenza. However, mainly children below age five died which would be atypical. The same holds for the elevated mortality levels during the fall of 1920. That mainly children below age five were affected indicates that this was indeed most likely an outbreak of measles.

To sum up, according to our excess-mortality calculations, the 1918–19 influenza caused 130 fatalities on Curaçao, 41 on Aruba and 32 on Bonaire in the period October–December 1918, which results in a comparatively low influenza mortality rate of around 4 per 1,000 for the Southern islands Curaçao, Aruba, and Bonaire. For the Leeward islands Saba, Sint Eustatius and Sint Maarten, the number of deaths is too low to calculate reliable estimates.

6 DISCUSSION AND CONCLUSION

The current article studied the extent of the 1918–19 influenza within Suriname and the Dutch Caribbean, with an emphasis on the possibilities and limitations posed by the availability of population statistics. Although both colonies had been dealing with the same colonising power since the late 1600s, the functioning of key administrative institutions such as population statistics and the civil registration differed considerably. This in turn resulted in colonial population statistics of different levels of completeness and diversity which affected our capabilities to adequately measure the incidence and fatalities of the 1918–19 influenza pandemic. Accordingly, different estimation methods for the incidence and fatality of the pandemic had to be used. Although our preferred method was the disease specific excess mortality approach, the lack of disease specific cause of death statistics made it necessary to use the all-cause excess method, particularly for the Dutch Caribbean islands.

The information in the Colonial Reports and the digitised civil registration data of Paramaribo indicate that Suriname experienced only one influenza wave in the months December 1918 and January 1919. The pandemic entered the country in the most Eastern district, spread to the capital (and only) city Paramaribo and to the districts where mainly workers from British and Dutch East India were located. The population of Paramaribo experienced highest excess mortality in December 1918, whereas the population in the districts experienced highest mortality levels in January 1919, after which the influenza epidemic died down quickly and never returned. Taken together, we estimate that around 2,200 people died from the 1918–19 influenza in Suriname, and as expected particularly adults aged 20–49 were heavily affected. The influenza mortality rates were considerably higher among the British and Dutch East Indian contract labourers than among the creole population. As influenza was endemic in Suriname, the creole population might have been better protected than the more recently arrived Asian contract labourers, especially those from the Dutch East Indies, against the 1918–19 influenza.

Given the low number of inhabitants of Curaçao and the dependencies, epidemics can easily affect the reference years to assess excess mortality. Despite this limitation, the number of excess deaths for Aruba and Bonaire is relatively close to the number of deaths reported in the Colonial Reports. This is quite surprising as the contemporary sources emphasize the flawed death registration on the islands. In sum, the 1918–19 influenza caused about 200 deaths (on a population of 50,912, 4 per 1,000) in the fall of 1918 on the Southern islands Curaçao, Aruba, and Bonaire and less than 10 deaths (on a population of 6,689, 1.5 per 1,000) on the Leeward islands in spring 1919. Most likely only one wave of influenza affected the islands, yet any second wave might be clouded by the outbreak of various infectious diseases during the following years.

To put our findings in perspective, in their study on the impact of the 1918–19 influenza in 12 European countries plus New Zealand and British India, Spreeuwenberg and colleagues (2018) calculated an average excess death rate of 7.9 per 1,000 in 1918 and of 1.3 per 1,000 in 1919, with British India having by far the largest excess mortality rates. Our results demonstrate that the Dutch Caribbean population was, on average, affected less heavily by the 1918–19 influenza (between 1.5 and 4 per 1,000), whereas the excess mortality rate among the total Surinamese population was considerably higher than average (20.6 per 1,000 overall). As the mortality rate of the Creoles+ population of Paramaribo was significantly lower (7.6 per 1,000) than the mortality rate of the population originating from British India (34.7 per 1,000) and the Dutch East Indies (42.7 per 1,000), it becomes obvious that the relatively large mortality rate was driven by the Asian contract labourers.

To conclude, although our overall estimates are the best possible given the nature of the data, we should be aware that a diverse population lies beneath these figures and that inequality is very much rooted within a colonial society. In both colonies reference was made to the fact that the most vulnerable members of society were most susceptible to be victims of the pandemic (KV, 1919, II, col. 15; KV, 1919, III, col. 9), such as the contract labourers from Dutch East India in Suriname. Accordingly, we consider our present article as a necessary first step in revealing health inequalities in colonial contexts. In line with Ravando (2022), we also call for more qualitative research into what preventive medical and socio-economic measures were taken by society, colonial government, and particularly cultural and religious groups to control the spread of the 1918–19 influenza among non-Western populations.

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

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ABSTRACT

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

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

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1 INTRODUCTION[1]

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

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

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

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

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

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

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

2 DOES SIBLING MORTALITY AFFECT AGE AT MARRIAGE?

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

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

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

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

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

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

Figure 1 *Women from low survivorship families marry sooner*

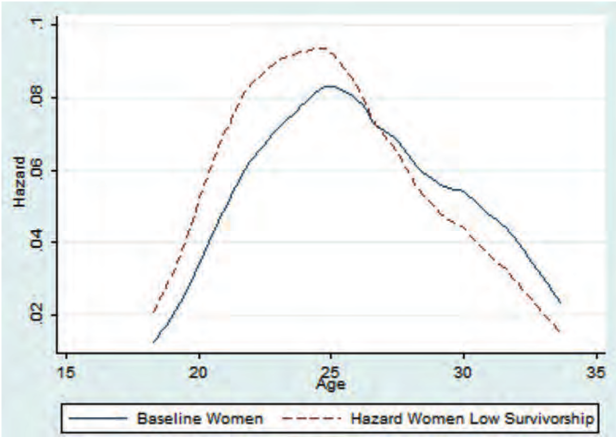
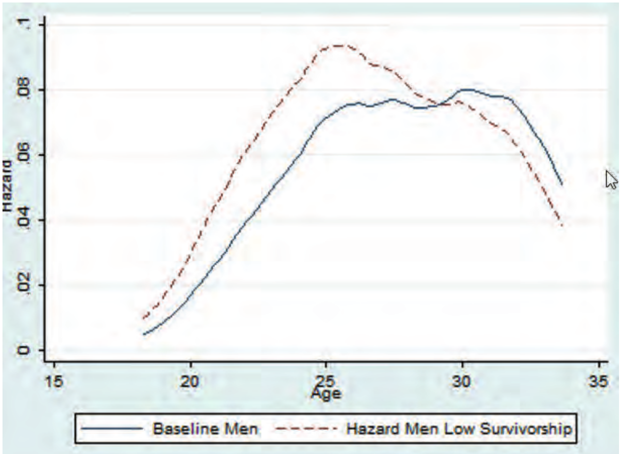


Figure 2 *Men from low survivorship families marry sooner*



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

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

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

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

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

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

3 DISCUSSION, CAVEATS AND CONCLUSION

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

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

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

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

4 AFTERTHOUGHT: DO WE NEED LIFE HISTORY THEORY

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

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

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

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

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Decomposition of Disability Prevalences

Age and Rate Effects in Northern Sweden, 1900–1950

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ABSTRACT

This study examines changes in disability prevalence during Sweden's demographic transition, focusing on the relative contributions of population aging and changing disability rates. Using unique longitudinal data from parish registers covering 194,500 individuals in Västerbotten county 1900–1950, we analyze trends in four disability categories: sensory, physical, mental and intellectual disabilities. Through demographic decomposition methods, we separate the effects of changes in population age structure from changes in age-specific disability rates. Our findings reveal that the substantial increase in disability prevalence was primarily driven by changes in disability rates during the period studied rather than population aging. Mental disabilities showed the most pronounced increase, rising from 0.8% to 2.5%, while other disability types remained stable or declined. The impact was particularly strong among middle-aged adults (25–54 years), challenging assumptions about the predominant role of population aging in historical disability trends. Our results suggest that social and environmental factors played a more significant role than demographic change in shaping disability prevalence during times of demographic transition.

Keywords: Disability, History of health, Historical population data, Sweden

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1 INTRODUCTION

The global prevalence of disabilities has increased significantly in recent decades, driven by population aging and changes in health conditions (WHO, 2022). Population aging is expected to accelerate this trend, as disability risks typically increase with age. Recent projections suggest that by 2050, more than two billion people will be aged 60 or older, compared to about one billion today (UN, 2022). This demographic shift will have profound implications for disability prevalence, healthcare systems, and social support structures.

Moving back to the 1900–1950 period, our study aims to provide historical knowledge on the dynamic relationship between population aging and disability prevalences. This relationship is complex, as longer life expectancy might lead to extended periods of disability in late life — or expansion of morbidity (Gruenberg, 1977) — while improvements in health and living conditions could also compress disability into a shorter period before death (Fries, 1980). Recent research shows varying patterns across countries and time periods, with some evidence of both compression and expansion depending on the context and type of disability (Chatterji et al., 2015; Crimmins & Beltrán-Sánchez, 2011). Historical knowledge about the relationship between population aging and disability is crucial for several reasons. First, it helps anticipate future trends as populations continue to age globally. Second, it provides insights into how social and medical progress might modify the age-disability relationship. Third, it informs policies aimed at healthy aging and disability prevention (Freedman et al., 2016; Tao et al., 2020).

The first half of the 20th century represents a key period for understanding the relationship we focus on. This era witnessed the beginning of population aging in many Western countries, with a shift from predominantly young populations toward higher proportions of middle-aged and older adults. This transformation, which we refer to as population aging, was driven by declining fertility rates and improving survival at older ages (Reher, 2004). Simultaneously, improvements in public health and medical care were transforming patterns of morbidity and mortality (Omran, 1971; Riley, 2001). Sweden's exceptional historical records and early demographic transition provide an ideal context for examining how these fundamental changes affected disability prevalences in the population.

Research on disability trends has been examined through various methodological approaches, though often constrained by data limitations. Historical studies typically rely on cross-sectional censuses or institutional records (Cohen, 2001; Hudson, 2005; Jarvis, 1855), which capture point-in-time snapshots but struggle to reveal dynamic patterns over extended periods. Contemporary disability research has increasingly employed decomposition techniques to separate the effects of multiple contributing factors. For instance, Hosseinpoor et al. (2012) and Tetteh et al. (2021) used Blinder-Oaxaca decomposition to quantify how much of gender disparities in disability prevalence could be attributed to demographic characteristics (such as age) versus differential effects of these characteristics. Similar approaches have been applied to examine socioeconomic inequalities in disability (Kim & Jeon, 2023) and geographic variations in disability program participation (Gettens et al., 2018). While these methods have enhanced our understanding of contemporary disability patterns, they have rarely been applied to historical data spanning major demographic transitions. This analytical gap between historical documentation and modern statistical techniques limits our understanding of how population aging and changing disability risks have interacted over time during periods of substantial demographic and epidemiological change, such as those experienced in early 20th century Sweden.

Our study covers some of this gap in knowledge by utilizing unique longitudinal data on Swedish populations in 1900–1950. This data provides comparatively detailed information about individuals' disability status, allowing us to analyze how disability prevalence evolved during a crucial period of demographic and epidemiological transition. We focus particularly on whether changes in disability prevalence were primarily driven by population aging or by shifts in age-specific disability risks.

Specifically, we address four research questions:

1. How did prevalence of different disability types and the age structure change in Sweden in the period 1900–1950?
2. To what extent were changes in disability prevalence driven by population aging versus changes in age-specific disability rates?
3. How did the relative contributions of population aging and disability rate changes vary over time throughout the study period?
4. Which age groups contributed most significantly to changes in disability prevalence, and did these patterns differ across disability types?

2 METHODS

Our study leverages longitudinal data from Swedish parish registers 1900–1950. This enables us to examine changes in disability prevalence during a period of significant demographic and epidemiological transition. We develop a multi-faceted analytical approach to disentangle the drivers of changing disability patterns. Drawing on Kitagawa's (1955) decomposition method, we separate changes in disability prevalence into components attributable to population aging versus changes in age-specific disability rates. Originally developed for analyzing mortality differentials, we adapt this approach to quantify the relative contributions of age structure and disability risk to changing prevalence over time. Our approach differs from cross-sectional decomposition methods used in recent disability research (Gettens et al., 2018; Hosseinpoor et al., 2012) by focusing on temporal changes rather than group differences, allowing us to track how these components evolved throughout Sweden's demographic transition.

2.1 DATA

Our data is drawn from parish registers in the Poplink database, Umeå University (2023), and covers all populations in 10 parishes across Västerbotten county, Sweden, 1900–1950. In total, the data comprises 194,500 individuals aged 15–100 years, including 4,700 with recorded disabilities. As there is not only information about when the disability was recorded but the type of it as well, we can distinguish between sensory (visual and auditory impairments), physical (bodily defects, mobility impairments), intellectual (cognitive dysfunctions from birth/childhood, e.g. 'idiocy', 'feeble-mindedness') and mental disabilities (psychiatric illnesses acquired across life, e.g. 'insanity', 'psychosis'). In these historical records, disabilities were documented when a person's sensory, physical, mental, or intellectual conditions impeded their capacity to participate fully in community life, especially their ability to maintain employment and economic independence. This conceptualization aligns with aspects of contemporary frameworks such as the WHO's, which emphasize the interaction between individual conditions and social contexts (WHO, 2022). We recognize that disability categories evolved throughout this period, reflecting changing social perceptions and institutional practices. The complex processes of disability recognition, registration practices, and individuals who receive such recognition are analyzed in detail by Wisselgren and Vikström (2023), who thoroughly examine the instructions for Swedish disability registration practices 1860–1930.

2.2 ANALYTICAL STRATEGY AND MEASURES

We employ four complementary analyses to understand the temporal dynamics of disability prevalence. First, we calculate annual prevalence rates for each disability type to establish basic temporal trends. Following standard demographic practice, we compute these rates as the number of individuals with disabilities divided by the total population at risk, expressed as percentages. To examine changes in population age structure, we calculated the percentage of the population in each age group (15–24, 25–34, ..., 75+) in 1900 and 1950, allowing us to visualize demographic aging during the study period.

Second, we implement a decomposition analysis to which changes in disability prevalence were driven by population aging versus changes in age-specific disability rates, following Kitagawa's (1955) method for rate decomposition. This approach allows us to separate changes in disability prevalence between 1900 and 1950 into two distinct components: those due to changes in population age structure and those due to changes in age-specific disability rates. Following Kitagawa's method, we express the change in disability prevalence (ΔP) between 1900 (t1) and 1950 (t + n, n = 50) as the sum of an age structure component (A) and a rate component (R):

$$\Delta P = A + R$$

The age structure component (A) measures how changes in the population's age distribution affected overall prevalence:

$$A = \sum \Delta w(x) * \frac{c(x,t) + c(x,t+n)}{2}$$

where $\Delta w(x)$ represents the change in the proportion of the population at age x, weighted by the average disability rate at that age across between 1900 (time t) and 1950 ($x + n, n = 50$). This component captures the effect of demographic shifts, such as population aging, while holding disability rates constant. The rate component (R) measures how changes in age-specific disability rates affected overall prevalence:

$$R = \sum \Delta c(x) * \frac{w(x,t) + w(x,t+n)}{2}$$

where $\Delta c(x)$ represents the change in the disability rate at age x across the two time points, weighted by the average population proportion at that age. This component captures changes in disability risks while adjusting for demographic composition.

We calculated these components for each disability type (mental, sensory, physical, and intellectual). This allows us to assess whether the relative importance of demographic versus rate effects varies across different types of disabilities. Then, we quantified the percentage contribution of each component to the total change in prevalence, providing a clear measure of their relative importance.

Third, we implement a temporal decomposition using five-year moving windows (n = 5) throughout the study period. This approach applies the same decomposition to successive years to track how the absolute magnitude and direction of demographic change versus disability risk evolved over time. This temporal analysis helps identify whether the drivers of disability prevalence remained stable or shifted during different phases of Sweden's demographic transition, and whether these patterns varied across disability types.

Fourth, we extend this decomposition to examine age-specific contributions to overall change 1900–1950, adapting Preston et al.'s (2001) methodological framework originally developed for demographic analysis. While the standard Kitagawa decomposition provides aggregate measures of age structure and rate effects, Preston's approach emphasizes that changes in population-level indicators are differentially driven by specific age groups, each with potentially distinct underlying mechanisms. Following this principle, instead of immediately summing across all ages, we maintained separate decomposition components for each age group (g) (15–24, 25–34, ..., 75+). This allowed us to identify which life stages contributed most significantly to changing disability patterns. The same equations apply, but with age groups (g) rather than individual ages (x). Importantly, these age-specific components sum up the total change in disability prevalence, allowing us to express each age group's contribution as a percentage of the total change.

This age-specific approach provides several analytical advantages over the aggregate decomposition. It identifies which age groups were the primary drivers of change, revealing whether disability trends were concentrated in particular life stages. The approach allows us to observe whether age structure and rate effects operated uniformly across age groups or exhibited different patterns at different ages. Additionally, it enables comparisons across disability types regarding which age groups contributed most to changing prevalence.

3 RESULTS

First, our analysis reveals distinct patterns in both population age structure and disability prevalence during the study period 1900–1950. Figure 1 shows the changes in age distribution of the Swedish population (age 15+) between 1900 and 1950. A clear demographic shift is evident, with a substantial decrease in the proportion of younger individuals and a corresponding increase in middle and older age groups. The proportion of the population aged 15–24 decreased markedly from 30% in 1900 to about 21.8% in 1950. Meanwhile, all age groups from 25 years and older show higher proportions in 1950 compared to 1900, indicating population aging during this period. This shift is particularly noticeable in the middle age ranges (25–64), where each group shows between 1.5–2.5 percentage points increase.

Figure 2 shows that mental disabilities exhibited the most pronounced increase, from approximately 0.8% in 1900 to 2.5% by 1950. This increase was particularly steep after 1910. In contrast, the prevalence of other disability types remained relatively stable or showed modest changes. While intellectual disabilities slightly increased from 0.5% to 0.7%, physical disabilities maintained a relatively constant prevalence around 0.2–0.3%. Notably, sensory disabilities declined gradually over time, from 0.7% to 0.4%.

Figure 1 *Age distribution in the study population 1900 and 1950*

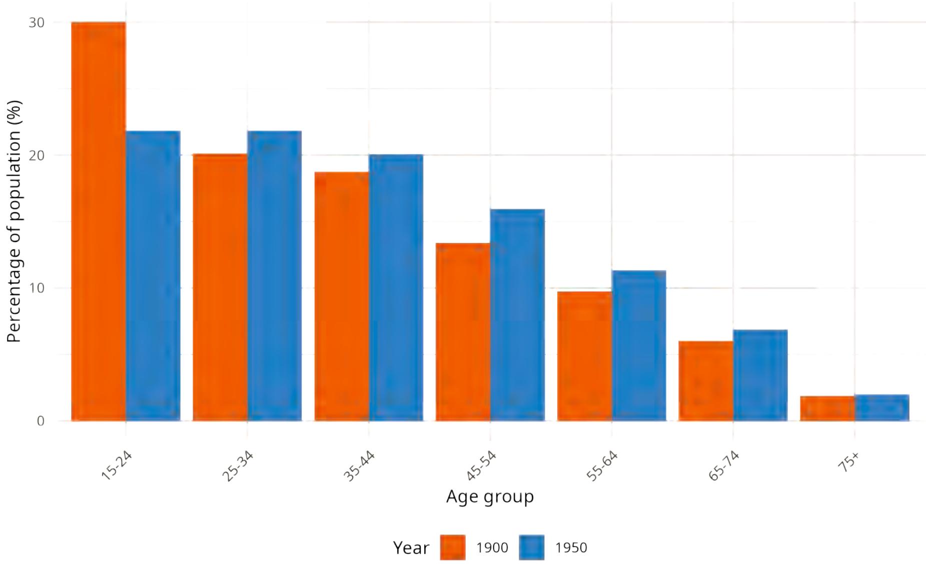
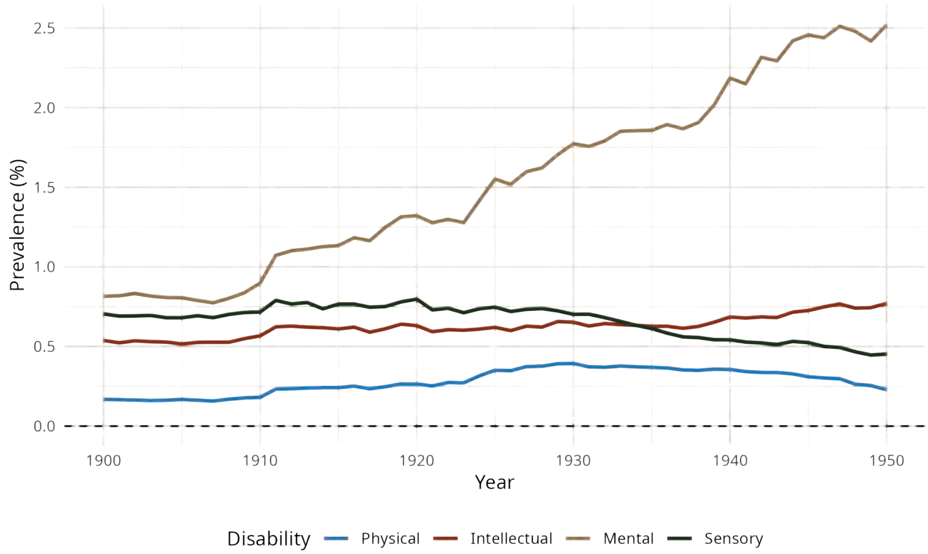


Figure 2 *Trends in prevalence by disability type in the Swedish population studied 1900–1950*



Second, our decomposition analysis reveals that the change in prevalence 1900–1950 was primarily driven by changes in disability rates rather than changes in age structure. Specifically, 91% of the total change was attributable to rate effects, while changes in age structure contributed only 9%. This pattern varied considerably across disability types (Figure 3). Mental disabilities showed the largest increase in prevalence (1.7 percentage points), with rate effects accounting for 92.8% of this change and age structure effects for 7.2%. This suggests that the rise in mental disability prevalence was predominantly due to increased rates within age groups rather than demographic aging.

Physical disabilities experienced a smaller increase (0.06 percentage points), with rate effects again dominating but to a lesser extent: 78.2% of the change was due to rate effects and 21.8% to age structure. For intellectual disabilities, which increased by 0.25 percentage points, the rate effect was even more dominant, accounting for 104% of the change, while age structure had a small negative effect (-3.5%).

Notably, sensory disabilities showed a different pattern, with prevalence decreasing by 0.24 percentage points over the period. This decline was more than fully explained by rate effects (-111% of the change), while age structure changes worked in the opposite direction, contributing to a small increase (11.4% of the total change).

Third, the temporal decomposition analysis (Figure 4) demonstrates that the magnitude and direction of rate and age structure effects varied over time. Mental disabilities show consistently positive rate effects throughout the period, with particularly strong effects during the 1910s and 1940s. The age structure effect for mental disabilities, while positive after approximately 1935, remained relatively small in absolute terms. For other disability types, both rate and age structure effects were smaller in magnitude and varied more across time. Sensory disabilities exhibited predominantly negative rate effects after 1920, while the age structure effect remained slightly positive, reflecting the ongoing aging of the population shown in Figure 1.

Figure 3 *Decomposition of relative contribution to changes in prevalence by disability type in the Swedish population studied 1900–1950*

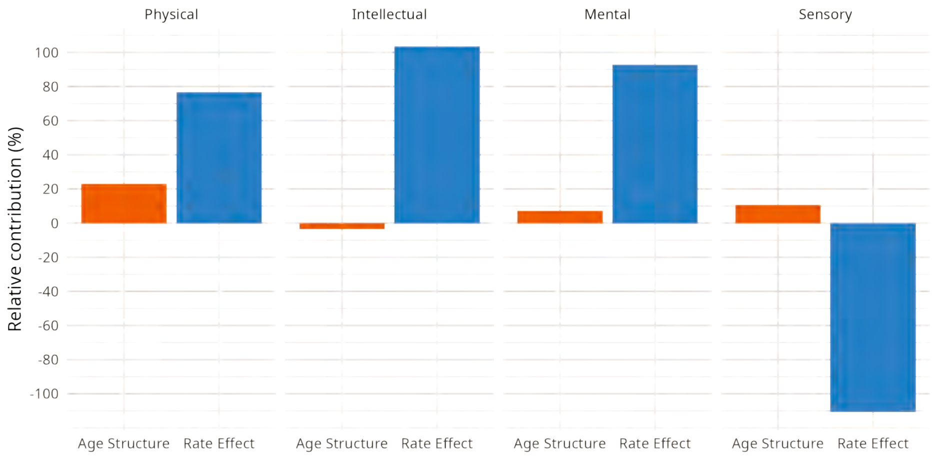


Figure 4 *Temporal patterns (five-year moving window analysis) in the decomposition of prevalence by disability type in the Swedish population studied 1900–1950*

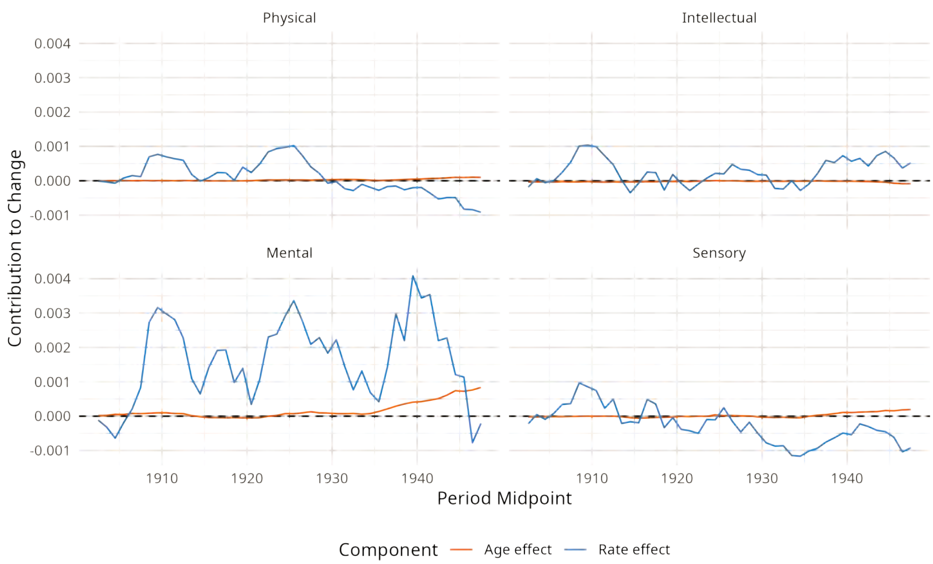
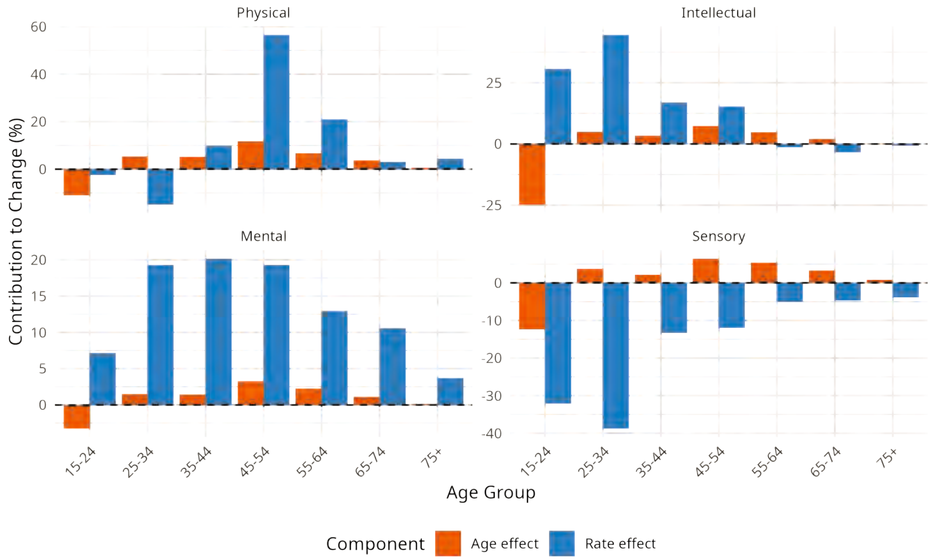


Figure 5 *Decomposition of changes in prevalence by disability type and age group (age structure vs. rate effects) in the Swedish population studied 1900–1950*



Fourth, the age-group specific decomposition (Figure 5) reveals that changes in disability rates, rather than age structure changes, were the primary drivers of prevalence trends. For mental disabilities, positive rate effects were observed across all age groups, with the largest contributions in the middle age ranges (25–54 years). The age structure effect was comparatively small and mostly positive, reflecting the aging of the population. Sensory disabilities showed negative rate effects, particularly pronounced in younger age groups (15–34 years), partially offset by positive age structure effects in older age groups.

4 DISCUSSION

Our analysis of disability patterns in northern Sweden during the first half of the 20th century contributes to ongoing debates about the relationship between population aging and disability prevalence. While contemporary research often emphasizes population aging as a key driver of increasing disability rates (Freedman et al., 2016; WHO, 2022), our decomposition analysis reveals a strikingly different pattern: changes in disability prevalence during this period were overwhelmingly driven by shifts in age-specific disability rates (91%) rather than by changes in population age structure (9%).

These findings challenge conventional assumptions about the primacy of demographic aging in driving disability trends. For mental disabilities, which showed the most dramatic increase in prevalence (from 0.8% to 2.5%), rate effects accounted for 92.8% of this change while age structure effects contributed only 7.2%. Similarly, for intellectual disabilities, rate effects explained 104% of the observed increase, with age structure having a small negative effect (-3.5%). Even for physical disabilities, with their modest increase, rate effects dominated at 78.2%. Only for sensory disabilities, which declined in prevalence, did the age structure component work against the overall trend, contributing a small positive effect (11.4%) that partially offset the strong negative rate effect (-111%).

Our temporal decomposition further illuminates this relationship, showing that rate effects for mental disabilities were particularly strong during specific periods — notably around 1910, 1925, and the 1940s — while age structure effects remained relatively small and stable throughout. This temporal pattern aligns with key institutional developments in Sweden: the expansion of the regional hospital in 1907 (Heimer, 1958), the 1920s saw expansion of welfare state institutions (Hirdman, 1989), and the 1934 opening of Umedalen asylum nearly doubled institutional capacity in the region (Eriksson et al., 2022). The timing suggests that changing institutional frameworks, rather than demographic shifts, drove the observed increases in disability prevalence.

The dominance of rate effects over age structure effects informs theoretical debates about expansion versus compression of morbidity. Gruenberg's (1977) expansion hypothesis and Fries' (1980) compression hypothesis both assume important roles for demographic aging, but our findings suggest that social and institutional factors may be more decisive than demographic change in shaping disability patterns. The threefold increase in mental disability prevalence despite relatively modest population aging could reflect what might be termed "institutional expansion of morbidity" — where the growth of institutional capacity (new facilities, more beds) led to an expansion of disability categories and the inclusion of new groups previously not classified as disabled, effectively creating supply-driven demand for institutional care (Eriksson et al., 2022). However, it is equally plausible that this increase reflects genuine deterioration in population mental health during Sweden's rapid modernization period. The disruption of traditional agricultural life, changing social structures, and new industrial work patterns created unprecedented psychological stresses (Sundin & Willner, 2007). The temporal spikes in mental disability rates around 1910 and 1925 coincide with periods of accelerated industrialization and urbanization in Sweden, lending credence to this interpretation.

The age-specific decomposition further reinforces this interpretation. Changes in mental disability rates were concentrated in middle-aged adults (25–54 years), precisely the age groups growing during this period of demographic transition. However, our decomposition shows that even within these expanding age groups, changes in disability rates — not merely the growth of these population segments — drove increased prevalence. This suggests that as Sweden's population structure shifted toward middle age, these cohorts simultaneously experienced intensified identification and classification of mental conditions.

For sensory disabilities, the declining rates despite population aging suggest differential improvement patterns across disability types during health transitions. The negative rate effect for sensory disabilities likely reflects technological adaptations (improved eyeglasses, early hearing aids) and changing work environments that reduced occupational hazards which previously caused reduced sensory impairments or their functional impacts (Johannisson, 2013). This pattern aligns with research showing that different types of disability can follow divergent trajectories during health transitions, with some impairments decreasing while others persist or increase (Cutler, 2001; Freedman & Martin, 2000). These divergent trends demonstrate that population aging does not uniformly affect all disability types.

The pronounced dominance of rate effects over age structure effects across multiple disability categories suggests broader social processes at work. While Hacking's (1999; 2013) concept of "making up people" through classification systems offers one explanatory framework — where institutions create categories that shape identification and treatment — we must also consider the possibility of genuine epidemiological change in mental health. The social disruption that accompanied Sweden's transformation from an agricultural to an industrial society likely created real psychological strain, given that this period brought radical changes in family structures, community relationships, and daily rhythms, while introducing new economic precariousness (Sundin & Willner, 2007). Similar to contemporary discussions about rising mental health problems amid social media and digital transformation (Blanchard et al., 2023; Marciano et al., 2021), the early 20th century's technological and social changes may have outpaced psychological adaptation. Our temporal analysis shows spikes in disability rate effects that coincide with both institutional developments and periods of rapid social change.

Some limitations should be considered when interpreting our results. The data comes from parish registers, where disability classification depended on ministers' observations and contemporary understanding of disabilities. It is methodologically challenging to empirically separate "actual" changes in disability prevalence from changes in registration practices, particularly because disability itself is partly constituted through classification systems. The substantial expansion of Sweden's welfare state during this period (1900–1950) likely changed the incentives and purposes for disability registration, as new social support systems developed for different categories of disability. The 1918 Poor Relief Act, for example, established new frameworks for categorizing those eligible for assistance (Rauhut, 2002). These institutional developments affected both the identification and documentation of disabilities, particularly mental conditions, which showed the most dramatic increases in our data (Wisselgren & Vikström, 2023). Additionally, our analysis concerns one region of Sweden, and patterns might differ in other areas or countries.

Our study demonstrates that focusing solely on demographic change may fundamentally misinterpret the drivers of disability prevalence. The overwhelming dominance of rate effects over age structure effects suggests that disability prevalence during Sweden's demographic transition emerged through a complex interplay of changing social responses to human variation, institutional development, and potentially genuine deterioration in population mental health amid rapid societal transformation. This parallels contemporary debates about whether increased mental health diagnosis rates reflect improved detection or actual increases in mental distress (Horwitz & Wakefield, 2007). Our findings suggest that policy responses should focus not merely on accommodating aging populations but on understanding how social institutions, labor markets, classification systems, and societal disruptions jointly shape the experience and prevalence of disability.

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Over the past two and a half centuries, the global population has witnessed significant improvements in health and longevity, with life expectancy at birth more than doubling in many regions. This transformation ranks among the greatest achievements in human history.

This volume brings together studies that reveal the complexity behind this transformation and provide a broad exploration of how these histories of health have evolved across different social and geographic contexts. The contributions explore how mortality and morbidity were recorded, understood, and experienced, focusing on the roles of social class, migration status, and sex, as well as demographic shifts and local conditions in shaping patterns of infant, childhood, and adult mortality.

Collectively, these studies demonstrate that the history of health cannot be understood as a straightforward narrative of continuous linear progress. Instead, they demonstrate it is a multifaceted process marked by societal challenges, unequal access to resources, and ongoing efforts by individuals and communities to adapt and survive in a changing world.

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