Introduction: Histories of Health

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To cite this article: Puschmann, P., Kok, J., Muurling, S., & Riswick, T. (2025). Introduction: Histories of Health. *Historical Life Course Studies, 15*, 47–64. https://doi.org/10.51964/hlcs23635

HISTORICAL LIFE COURSE STUDIES

Histories of Health

VOLUME 15, SPECIAL ISSUE 7

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EHPS NETWORK

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HISTORICAL LIFE COURSE STUDIES

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HISTORICAL LIFE COURSE STUDIES
VOLUME 15 (2025), 47–64, published 29-08-2025

Introduction

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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

e-ISSN: 2352-6343
DOI article: https://doi.org/10.51964/hlcs23635

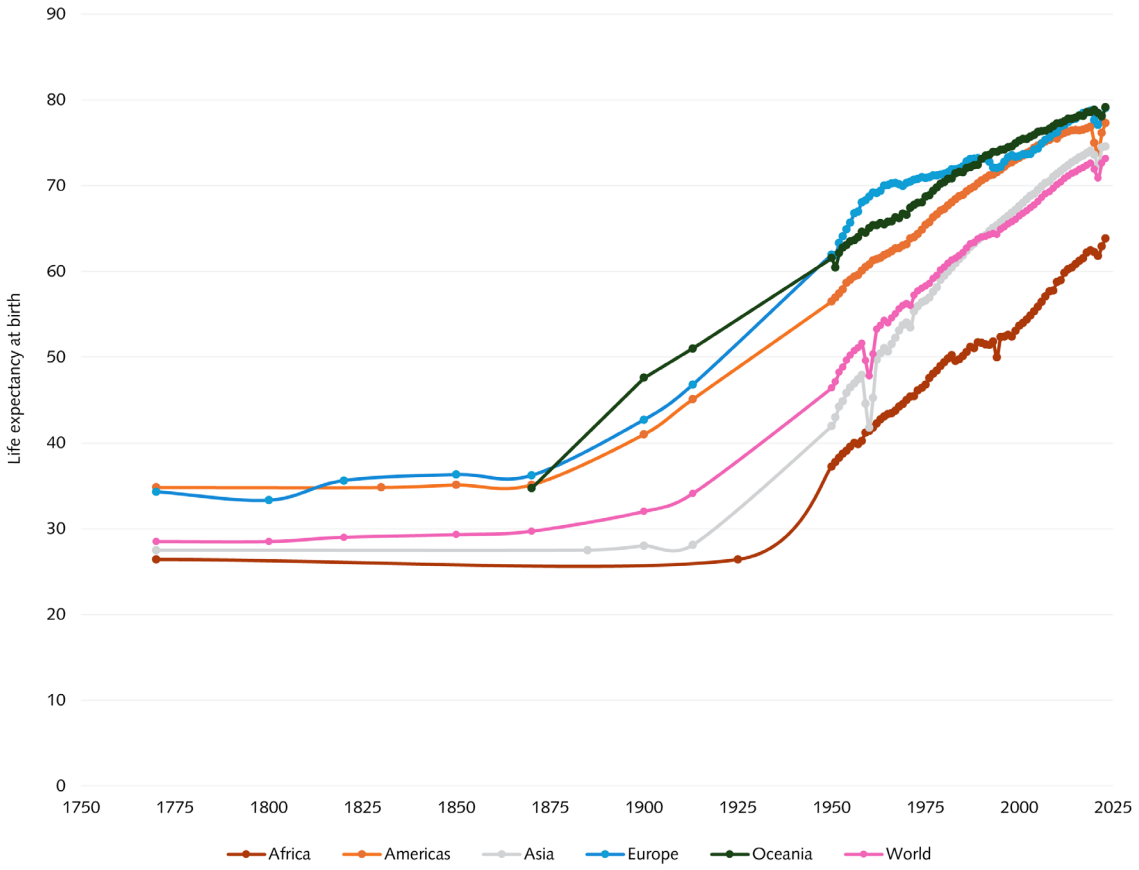
This issue is dedicated to **Professor Angélique Janssens**, who recently retired from her chair in historical demography at the Faculty of Arts and Social Sciences at Maastricht University and the Department of Economic, Social and Demographic History at Radboud University. Her pioneering research explores 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 Hărăguș (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.

ACKNOWLEDGEMENTS

This issue has been made possible with the generous financial support of HiDO — Scientific Network for Historical Demography (Research Foundation Flanders, Application number [W001821N]) and is based upon work from COST Action GREATLEAP [CA22116], supported by COST (European Cooperation in Science and Technology). We are also grateful to Linda Robertus for her meticulous copy-editing, and to Marja Koster for overseeing the layout and design. Our sincere thanks go to all the external reviewers of the individual contributions, whose thoughtful feedback greatly enhanced the quality of this issue: Nicola Barban (University of Bologna, Italy), Onno Boonstra (Radboud University, the Netherlands), Gabriel Brea Martinez (Lund University, Sweden), Xingchen ChiaChi Lin (Tamkang University, Taiwan), Nadeche Diepgrond (Radboud University, the Netherlands), Ingrid van Dijk (Lund University, Sweden), Theo Engelen (Radboud University, the Netherlands), Joris Kok (International Institute of Social History & Radboud University, the Netherlands), Koen Matthijs (KU Leuven, Belgium), Stanislao Mazzoni (Spanish National Research Council), Saverio Minardi (University of Bologna, Italy), Wieke Elien Metzlar (Radboud University, the Netherlands), Michel Oris (Spanish Research Council), Joana-Maria Pujadas-Mora (Autonomous University of Barcelona & Universitat Oberta de Catalunya, Spain), Matthias Rosenbaum-Feldbrügge (Radboud University, the Netherlands), Jessie van Straaten (Leiden University, the Netherlands), Jan Van Bavel (KU Leuven, Belgium) and Coen van Galen (Radboud University, the Netherlands).

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