Unequal pathways to the grave?

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Time lags and inequalities in the Dutch health transition, the case of Maastricht, 1864-1955

Mayra Murkens

Propositions

1. Socioeconomic inequalities in cause-specific mortality in Maastricht emerged during the initial phase of the health transition, which is why they should be treated as a time-dependent phenomenon instead of a static phenomenon.
2. Men were in a disadvantageous position prior to the onset of the health transition in Maastricht, higher male mortality rates only diminished once the transition was well underway.
3. The characterisation of Maastricht as an extremely unhealthy location towards the end of the nineteenth century only upholds for a part of the population; women and young children were not lagging behind the Dutch mortality trends.
4. The reliability of the registration of maternal mortality is questionable, maternal mortality in Maastricht is likely to be partially registered as a different cause of death.
5. The quest for finding a linear social gradient in mortality in historical demography can be unproductive, for existing, more complex socioeconomic inequalities do not fit the linear gradient narrative.
6. Historical demographers should do their utmost to find sufficient data for women, even when this may be an arduous task; because the experience of men cannot represent the experience of the population as a whole.
7. The specialisation in historical sub-disciplines has created the two separate fields of historical demography and the history of medicine, a cooperation between the two fields would create more comprehensive research in the history of mortality and morbidity.
8. The general public values large historical demographical databases primarily for their genealogical opportunities; it is the task of the historian to emphasize the value of these datasets beyond the realm of private and local histories.
9. An n=1 study has observed a clear correlation, and perhaps causation, between studying historical infectious diseases and avoiding the contraction of modern infectious diseases, such as Covid-19.

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Mayra Murkens

Supervisors
Prof. dr. A.A.P.O Janssens
Dr. W.J.M.J. Rutten

Assessment Committee
Prof. dr. E.S. Houwaart (chair)
Prof. dr I. Devos, Universiteit Gent, Belgium
Dr. J. Roosen
Dr. E.C. Walhout, Universiteit Leiden

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CHAPTER 1

Introduction

1.1 Motivation and research aims

By the year 2000, average global life expectancy had reached the impressive number of sixty-seven years, and it has continued to rise in the subsequent years. Historian James C. Riley regarded this particular development as the *'crowning achievement'* of the modern era.[1] Over the past century and a half, modernisation has changed societies drastically. One of the most apparent examples of this change is the improvement of the standard of living, which constituted for example improvements in housing and nutrition. Yet, these changes did not occur overnight and modernisation is not known for its uniform and steady pace of occurrence. Inherently connected to modernisation therefore are emerging inequalities in two key features of life: Health and wealth, asserts Angus Deaton.[2] Despite the remarkable improvements in wealth and health, this great leap has been unequal. Not all classes have improved equally, hence the improvements are also 'one of the great injustices of the world today'.[3]

The particular modernising process central to these improvements in living standards and health is known as the health transition. This study takes the concept of the health transition as the overarching concept, as it describes the changes in health in terms of age at death, causes of death, and morbidity. These changes were not only a result of medical innovations and increasing wealth, but were also a consequence of cultural changes.[4] Other useful theories that offer valuable frameworks for understanding the profound changes in fertility and mortality are the epidemiological transition theory and the demographic transition theory. Yet the concept of the health transition offers more specificity on health than the demographic transition theory, and is broader than the concept of the epidemiological transition, which exclusively focusses on shifts in the nature of the dominant disease pattern in a population. This does not mean that the epidemiological transition theory or the demographic transition theory are ignored in this study. Both theories are also employed in this study as they draw attention to important features of demographic change.

Underlying these vast demographic processes related to health are many smaller changes of diverse origin, which all together comprise the health transition. In order to have a profound notion of these societal transitions, we therefore need a deep understanding of all the smaller and more obscure processes at the root of the health transition. That is exactly the goal of this study: It aims to illuminate the intricate process of the health transition at a micro level. A crucial element of studying this process is studying emerging and/or receding inequalities during the transition. Studying socioeconomic, age or gender inequalities is not only a goal in and of itself to learn more about existing and historical inequalities, but it also enriches our understanding of the determinants responsible for the health transition. After all, when inequalities in health

1 James C. Riley, *Rising Life Expectancy: A Global History* (Cambridge: Cambridge University Press, 2001), 1.

2 Angus Deaton, *The Great Escape: Health, Wealth and the Origins of Inequality* (Princeton: Princeton University Press, 2013).

3 Ibid., 7.

4 J. Cleland, 'The Idea of the Health Transition,' in *What We Know About the Health Transition: The Cultural, Social and Behavioural Determinants of Health. The Proceedings of an International Workshop*, ed. J.C. Caldwell et al., 2 vols (Canberra: The Australian National University, 1990), I, xviii-xix.

emerge, the resources to battle certain diseases or causes of death must be unequally distributed.[5]

Another key component of this study on the health transition and its determinants is the availability of information on not merely mortality, but also on specific causes of death. Specific causes of death are very informative of the determinants leading to the health transition. When certain diseases recede, the aetiology of the disease can offer a clearer indication of the related factors changing health. Up until now, many studies that have been able to include information on causes of death have been forced to rely on aggregated data, which often lack key information at an individual level. These studies are only able to provide a highly generalised understanding of the features of the health transition and its determinants. In contrast, studies conducted at the micro level, with individual level cause-of-death data and individual characteristics, have the great potential to reveal what remains hidden behind the aggregations. Unfortunately, these types of datasets are truly scarce in an international perspective. However, this is beginning to change: In the past years, the construction of large datasets containing individual characteristics and cause-of-death data has been undertaken by several scholars.[6]

This study partakes in the endeavour to use this new type of datasets, by deploying a unique dataset containing individual-level causes of death, alongside individual information on the deceased, such as age, gender, occupation, place of birth and place of death.[7] The dataset contains information on all the individuals who had died or died as an official resident in/of the Dutch city of Maastricht for the entire period between 1864 and 1955. The result is a very rich dataset, not only in terms of the included individual characteristics, but also spanning almost a century. The research will therefore be a case study of the health transition in the city of Maastricht. Case studies do not only serve as exploratory tools, but are *'powerful sources of inferences'* themselves, and form the basis for falsification.[8] Moreover, Wolleswinkel-van den Bosch, who studied the epidemiological transition in the Netherlands, stressed the necessity of further research on cause-specific mortality at a micro level.[9] Since different determinants of the epidemiological transition were important in different periods of the mortality decline, a detailed notion of cause-specific mortality decline enables a more sophisticated view of this transition.[10] This study will thus be able to contribute significantly to both the national and international debate on the health transition and its determinants. The central research question for this study is threefold: First, how did the health transition in the industrialising city

5 Sean A. P. Clouston et al., 'A Social History of Disease: Contextualizing the Rise and Fall of Social Inequalities in Cause-Specific Mortality,' *Demography* 53, no. 5 (2016).

6 Angélique Janssens, 'Constructing Ship and an International Historical Coding System for Causes of Death,' *Historical Life Course Studies* 10 (2021).

7 Willibrord Rutten, *The Maastricht Death and Disease Database*, Sociaal Historisch Centrum Limburg (unpublished dataset).

8 A. Palloni, 'Methodological Problems in the Study of the Health Transition,' in *What We Know About the Health Transition. The Cultural, Social and Behavioural Determinants of Health. The Proceedings of an International Workshop*, ed. J.C. Caldwell et al., 2 vols (Canberra: The Australian National University, 1990), I, 896-99.

9 Judith Wolleswinkel-van den Bosch, 'The Epidemiological Transition in the Netherlands' (1998).

10 Ibid., 1-6 and 191-204.

of Maastricht take shape; second, did the transition lead to new inequalities in cause-specific mortality in terms of socioeconomic status, age and gender; and, third, which determinants were at the root of these changes in health?

Different methods will be used to answer these questions. A common denominator of these methods, however, is the focus on changing epidemiological patterns, i.e. the study of informative changes in the dominant pattern of circulating diseases. The unique data available for Maastricht allow for a focus on cause-specific mortality, despite a population at risk not being available according to all the specific individual characteristics, such as social group.[11] By analysing changing epidemiological patterns for these particular groups instead of the changes in cause-specific mortality *rates* for these groups, it becomes possible to approximate emerging inequalities and health advantages and disadvantages. The epidemiological patterns thus help to locate changes in mortality regimes for distinct groups in society.

The reasoning behind the methodological approach followed in this study is derived from Omran's epidemiological transition theory.[12] To understand this particular approach in the analysis of emerging or receding inequalities in changing epidemiological patterns, two main characteristics of the epidemiological transition theory need to be discussed here. First, the epidemiological transition theory postulates that during the transition the dominant causes of death changed from infectious diseases to man-made and degenerative causes of death.[13] Second, when infectious diseases are rampant, there is a high mortality regime in which many young children and adults find an early death. A low mortality regime is reached when not only the dominant causes of death have changed towards non-infectious diseases, but death also predominantly occurs in old age. When cause-specific mortality rates according to socioeconomic group are lacking, it may seem impossible to establish whether the specific group was experiencing a high or low mortality regime. Yet, identifying mortality differentials across socioeconomic groups is key to understanding historical, socioeconomic health inequalities. Fortunately, the epidemiological patterns by a specific group can provide an indication of the reigning mortality regimes. When the majority of causes of death is due to infectious diseases, a high mortality regime is most likely to have been in place. A comparison of the changes in epidemiological patterns by specific groups within society over time, could therefore indicate whether one or more groups have gained an advantage or were disadvantaged compared to the other groups. An advantageous position would be one in which the proportion of infectious diseases in that group had decreased earlier compared to the rest of society. The expectation would be that mortality had declined at that moment as well. On the other hand, a disadvantageous position would mean the lingering of infectious diseases compared to other groups in society, and the continuation of a high mortality regime.

The following sections of this introduction will first offer a discussion of the most important transition theories that together function as the theoretical framework for

11 In particular a population at risk according to socioeconomic status is not available.

12 Abdel R. Omran, 'The Epidemiologic Transition: A Theory of the Epidemiology of Population Change,' *Milbank Memorial Fund Quarterly* 49, no. 4 (1971).

13 Ibid.

this study; the demographic transition theory, the epidemiological transition theory, and the here preferred overarching concept of the health transition. Thereafter, I discuss the determinants causing the mortality decline in the course of the epidemiological and health transition, as these have been researched by many scholars in the past decades. These determinants had the potential of creating disparities in health in society, which is why factors potentially causing major inequalities, mainly socioeconomic status and gender, will also be discussed. Finally, an overview of the individual chapters of this dissertation will be provided.

1.2 Theoretical considerations

1.2.1 Transition theories

The mother of all transition theories that emerged during the twentieth century is the demographic transition theory, which was formulated to theorise the massive population growth from the mid-eighteenth century until the mid-twentieth century. The origins of the demographic transition theory can be traced back to the first decades of the twentieth century and several authors are considered as the founders of the theory, while the formal formulation of the theory is dated to 1953.[14] The theory deals with the interaction between two main demographic phenomena: Fertility and mortality. In pre-transitional societies, the demographic regime consists of both high mortality and high fertility, while post-transitional societies have low mortality and low fertility. The transition sets in when mortality declines. As a consequence, the remaining high fertility sparks an increased natural growth, until also fertility declines. Once fertility has declined, a new equilibrium between the mortality and fertility rates once more comes into place. In this final stage, the population will not grow as explosively anymore, as was the case in the intermediate stage of the transition.

Inherent in demographic transition theory is the idea of modernisation, as it identifies the process of modernisation as having caused both the decline of mortality and the decline in fertility. Scholars have tried to explain which factors specifically caused these declines. The most important factors for the decline in mortality are thought to have been medical discoveries, health improvements and higher standards of living.[15] The decline in fertility has proven more complicated to explain. The main arguments conclude that fertility declined as a result of structural changes in economy and society, although the impact of modernisation on the fertility decline was more gradual as opposed to its impact on mortality.[16] A prerequisite for the decline in fertility was often found in economic development.[17] Since the demographic transition theory includes both mortality and fertility, it functions as an important yet distant framework for this

14 J. Chesnais, *The Demographic Transition: Stages, Patterns and Economic Implications: A Longitudinal Study of Sixty-Seven Countries Covering the Period 1720-1984* (Oxford: Clarendon Press, 1992), 1-10.

15 Ibid.

16 Simon Szreter, 'The Idea of Demographic Transition and the Study of Fertility Change: A Critical Intellectual History,' *Population and Development Review* 19, no. 4 (1993).

17 Chesnais, *The Demographic Transition*, 1-10.

research. The transition theories that aim to explain the decline in mortality in particular are more directly relevant.

The epidemiological transition theory provides a crucial methodological framework for this study, as it formulates the disease-specific characteristics of the mortality decline. The epidemiological transition theory was formulated in 1971 by the epidemiologist Abdel R. Omran. According to his theory, among the key features of the secular decline in mortality was a change in the dominant causes of death; from infectious diseases towards non-infectious, degenerative and man-made diseases.[18] The epidemiological transition theory consists of five main propositions about population dynamics and mortality. The first proposition concerns the fundamental role of mortality in population dynamics; Omran asserts that mortality is a decisive factor in population growth. Omran believes that even if there are very high fertility rates, mortality determines whether the population grows or not.

The most well-known proposition of the epidemiological transition theory is the second proposition, which asserts that central to the decline in mortality was a decline in communicable diseases and pandemics of infectious diseases. Over time, these were replaced by degenerative and man-made diseases. Omran distinguishes three successive stages, the first being the age of pestilence and famine. Specific to this period were high mortality rates and an average life expectancy at birth between twenty and forty years. The second stage was the age of receding pandemics, when mortality declined progressively and the life expectancy at birth increased to an average between thirty and fifty years. The final stage was the age of degenerative and man-made diseases, when diseases such as cancer and cardiovascular diseases became the prevailing causes of death. In this stage, mortality declines until it reaches a certain stability, whereafter fertility becomes the determining factor in population growth. Eventually, life expectancy at birth exceeds fifty years.[19]

Mortality affects distinct groups in the population differently, however, which is considered in the third proposition. According to Omran, children and young women are the two groups in a population mostly affected by changes in health and disease patterns. They are deemed to have had a high susceptibility to infectious and deficiency diseases, such as gastroenteritis and malnutrition. Once such diseases receded, children and young women benefitted the most. If the average life expectancy was low, especially women in the adolescent and reproductive age periods would have had a higher risk of dying than men, Omran assumes. However, this disadvantage for women disappeared during the transitional period with the rise in life expectancy.[20] This third proposition feeds into one of the key features of this research: the inequalities emerging and/or receding in the transitional process. The case study on Maastricht may contest the applicability of Omran's third proposition, at least in this particular city, where adult women appear to not have been in such a disadvantageous position.

Central to the fourth proposition is the process of modernisation. A strong association between the epidemiological transition on one side and the demographic

18 Omran, 'The Epidemiologic Transition'.

19 Ibid., 516-22.

20 Ibid., 522.

and socioeconomic transitions on the other side, appears to have existed. Socioeconomic improvements such as increasing wealth could effectuate improved nutritional status, which is beneficial to survival chances from infectious diseases.[21] The fourth proposition is especially relevant concerning the specific determinants of the transition, which will be discussed more elaborately in the following section of this chapter.

Finally, the epidemiological transition did not occur in a similar fashion or at a similar pace around the globe. Omran distinguishes between three distinct models, in order to account for the differences in the transition for certain countries or regions. He formulates them in his fifth and last proposition: (1) The classical or western model, (2) the accelerated model, and (3) the contemporary or delayed model.[22] For each model, Omran indicates the specific paces, patterns and determinants. For the first, the classical or western model, Omran puts forth mainly socioeconomic factors, in particular improvements in standard of living, health habits, hygiene and nutrition, as the primary determinant. Sanitary, medical and public health measures then enhanced the transition in the late nineteenth and early twentieth century.[23] The accelerated model takes a similar path as the classical model, although the transition takes place more rapidly. Modernisation has already started in the countries following this model. Here, the decline in mortality is due to a combination of general social improvements and advances in sanitation and medicine. The social component loses its importance in the contemporary or delayed model, the model specific to developing countries. These countries can build on the already obtained medical knowledge in the developed countries, which, according to Omran, makes the most important determinant in developing countries public health measures.[24]

Although Omran's theory provides a major methodological framework for this study, exercising a modicum of caution is appropriate. The theory has been widely criticised, even for not being a theory at all, but rather a description of what has occurred in several countries.[25] Moreover, after the publication of the theory, new changes in epidemiological patterns occurred. Other scholars have therefore proposed a fourth phase of delayed degenerative diseases, or even a fifth phase of the re-emergence of infectious and parasitic diseases.[26] After the past two decades in which the world population has had to deal with SARS, Ebola, the swine flu and finally two years of lockdowns due to COVID-19, the existence of this fifth phase is difficult to deny. Nevertheless, despite many adaptations and revisions, even from Omran himself, the theory has not been discarded completely.[27] What seems to be fruitful, is to use the epidemiological transition theory, as quoted by

21 Ibid., 511-22.

22 Ibid., 530-31.

23 Ibid., 530 and 34.

24 Ibid., 535-36.

25 See Bernard Harris, 'Public Health, Nutrition, and the Decline of Mortality: The Mckeown Thesis Revisited,' *Social History of Medicine* 17, no. 3 (2004).

26 S. Jay Olshansky et al., 'Emerging Infectious Diseases: The Fifth Stage of the Epidemiologic Transition?,' *World Health Statistics Quarterly* 51, no. 2, 3, 4 (1998); S. Jay Olshansky and A. Brian Ault, 'The Fourth Stage of the Epidemiologic Transition: The Age of Delayed Degenerative Diseases,' *The Milbank Quarterly* 64, no. 3 (1986).

27 A. R. Omran, 'The Epidemiological Transition Theory: A Preliminary Update,' *Journal of Tropical Pediatrics* 29, no. 6 (1983).

Robert McKeown, as *'a way of looking at and understanding the relation among disease, mortality patterns, and population dynamics rather than as a definitive explanation or prediction.'*[28] This approach seems only natural to historians, for whom using a theory as a definitive explanation or prediction feels unnatural. Using the theory is thus still appropriate for a historical study into epidemiological change between the mid-nineteenth and the mid-twentieth century.

The process of the epidemiological transition in the Netherlands has been researched in detail by Judith Wolleswinkel-van den Bosch. The objective of her dissertation is to re-evaluate the validity of the epidemiological transition theory based on an analysis of the process of mortality decline in the Netherlands. Since the theory has been useful for contemporary research in the field of public health and for projections of future health, a critical re-evaluation of the theory was believed desirable, especially because of the critique the theory has received.[29] Wolleswinkel-van den Bosch concludes, on the basis of aggregated Dutch mortality data, that the epidemiological transition theory definitely should not be discarded, yet some adjustments should be made.[30] As different determinants were important in different periods of the mortality decline, Wolleswinkel-van den Bosch argued that a more sophisticated view of the epidemiological transition based on cause-specific mortality data was needed to account for these differences.[31]

The framework of this study is extended further by using the health transition theory and its implications. Omran's framework of the epidemiological transition is complementary to the concept of the health transition. Yet, the health transition is the preferred concept for this study because it adds a broader dimension to the transition phenomenon by emphasising the importance of specifically social, cultural and behavioural determinants of health.[32] Not only environmental or economic determinants influenced the decline in mortality, cultural factors such as religion or culturally-induced hygienic behavioural practices have been significant as well. Furthermore, the idea of a health transition refers to the interaction between mortality and morbidity.[33] A decline in mortality does not immediately imply a healthier population, because morbidity probably increases or is prolonged when mortality decreases. It became easier during the health transition to postpone death, but not always disease, even though the diseases one suffered from could be different. Nevertheless, morbidity in a historical sense is difficult to measure accurately. As is the case in the majority of performed historical studies, I will have to rely on mortality data.

1.2.2 Determinants of mortality decline

Different determinants for the decline in mortality in the course of the health transition have been put forward. Before Thomas McKeown's theory 1976 theory on the causes of

28 Robert E. McKeown, 'The Epidemiologic Transition: Changing Patterns of Mortality and Population Dynamics,' *American Journal of Lifestyle Medicine* 3, no. 1 suppl. (2009), 20s.

29 Wolleswinkel-Van Den Bosch, 'The Epidemiological Transition in the Netherlands,' 1-6.

30 Ibid., 191-204.

31 Ibid.

32 Cleland, 'The Idea of the Health Transition,' xviii-xix.

33 For an overview see Graham Mooney, 'Historical Demography and Epidemiology: The Meta-Narrative Challenge,' in *The Oxford Handbook of the History of Medicine, ed.* M. Jackson (Oxford: Oxford University Press, 2011).

the rise of population in the modern era, it was generally thought that medical innovations and improved living conditions were primarily responsible for the decline in mortality.[34] McKeown presented a different approach, in which he offered a structural explanation for the decrease in mortality and the subsequent increase in population. First, he refuted the idea that medical innovations were responsible for the decline in infectious diseases in the eighteenth, nineteenth and early twentieth century. His main argument was that, apart from some exceptions like the smallpox vaccine, effective therapies against infectious diseases were only discovered at the beginning of the twentieth century. The most effective therapies, like antibiotics, came into use even later than that, just after the Second World War. McKeown concludes that before 1935, *'immunization and treatment contributed little to the reduction of deaths from infectious diseases'*.[35] A change in virulence of the micro-organisms causing the infections and a reduction in exposure (at least for airborne diseases) were also rejected as main determinants for the decline in mortality.[36]

According to McKeown, the most important determinant for the reduction in mortality was improved nutrition in the eighteenth and nineteenth century, as a result of increased food production.[37] McKeown's work has been highly influential and has sparked heated debate. Several historians did not agree with McKeown's derivations and conclusions.[38] McKeown focused primarily on airborne infectious diseases. In doing so, he was criticized for ignoring the high infant mortality rates experienced throughout Western countries, which were mainly caused by water and foodborne infectious diseases. McKeown, however, did acknowledge the importance of reduced exposure for water and foodborne diseases, even though he acknowledged this determinant as being of major importance only after the second half of the nineteenth century. By only allocating a small role to water and foodborne infectious diseases in the mortality decline, McKeown perhaps insufficiently acknowledges the importance of infant mortality as part of total mortality and its decline.[39]

The relationship between the decline in airborne infectious diseases and the improved nutritional intake is in itself also too simplistic. Mackenbach argued there must have been other factors which contributed to the decline in mortality, as a unilateral explanation like McKeown's does not account for the overall decline in mortality.[40] Furthermore, McKeown seems to have taken the ascribed causes of death for granted, whilst Reid argued that especially the airborne infectious diseases could have been severely over-registered. Pneumonia, for example, can be a complication of many other diseases,

34 Thomas McKeown, *The Modern Rise of Population* (New York: Academic Press, 1976), 3.

35 Ibid., 108.

36 Ibid., 89-99, 117.

37 Ibid., 159.

38 Simon Szreter, 'The Importance of Social Intervention in Britain's Mortality Decline C. 1850-1914: A Re-Interpretation of the Role of Public Health,' *Social History of Medicine* 1, no. 1 (1988); R. I. Woods, P. A. Watterson, and J. H. Woodward, 'The Causes of Rapid Infant Mortality Decline in England and Wales, 1861–1921 Part I,' *Population Studies* 42, no. 3 (1988).

39 Woods, Watterson, and Woodward, 'The Causes of Rapid Infant Mortality Decline in England and Wales, 1861–1921 Part I,' 345.

40 Johan P. Mackenbach, *De Veren van Icarus. Over de Achtergronden van Twee Eeuwen Epidemiologische Transities in Nederland* (Utrecht: Bunge, 1992): 41.

which may not be of an airborne origin.[41] Although it is impossible to know whether some airborne infections were primary or secondary diseases when this was not explicitly reported, it should cause at least some restraint in embracing a general all-encompassing theory.

One of the most fervent critics of the McKeown thesis was Simon Szreter. Over a decade after McKeown had published his theory, Szreter refuted the idea of improved nutrition as the key determinant for the decline in mortality. Instead of believing in what Szreter called McKeown's structural approach of an 'invisible hand', Szreter preferred the concept of human agency. Human interventions in the form of public health measures were instead accountable for the mortality decline, at least in Britain.[42] Szreter's argumentation is related to the standard-of-living debate, which deals with the alleged improvements gained as a consequence of industrialisation. Industrialisation can on the one hand improve the economic position of the working population, which could lead to improved nutritional status in the reasoning of McKeown. On the other hand, working and living circumstances can deteriorate significantly at the start of industrialisation, which leads to a worsened standard of living.[43] Thus, although economic growth from industrialisation could facilitate improvements in health in the long run, it would more immediately cause what Szreter called the 'four D's': Disruption, deprivation, disease and, ultimately, death. When institutions did not respond appropriately to reverse the disruption, it could be followed by deprivation, disease and death.[44] Countervailing measures to mitigate disruption, and hence allowing mortality to decline, consisted of interventions in the field of sanitation and public health by local governments and voluntary associations.[45] However, by focusing solely on the aspect of human agency, Szreter loses sight of the structural aspects. It is more productive to use a multi-causal approach, whereby multiple causes may influence the health of the population successively or simultaneously.

Indeed, Riley asserted that different countries' and/or regions' strategies to decrease mortality rates can vary, ranging from strategies in public health, medicine, wealth and income, nutrition, behaviour, to education.[46] Since the 1980s, scholars have paid more attention to the multiplicity of causes of the health transition instead of focusing on only one or two leading determinants.[47] In all the strategies that could be deployed, there is,

41 Alice Reid et al., "A Confession of Ignorance': Deaths from Old Age and Deciphering Cause-of-Death Statistics in Scotland, 1855-1949,' *The History of the Family* 20, no. 3 (2015): 342.

42 Szreter, 'The Importance of Social Intervention in Britain's Mortality Decline,' 34-35. For the ongoing discussion, see also the replies by Sumit Guha, 'The Importance of Social Intervention in England's Mortality Decline. The Evidence Reviewed,' *Social History of Medicine 7* (1994); Simon Szreter, 'Mortality in England in the Eighteenth and the Nineteenth Centuries. A Reply to Sumit Guha,' *Social History of Medicine 7*, no. 2 (1994).

43 For an overview see: Roderick Floud et al., *The Changing Body: Health, Nutrition, and Human Development in the Western World since 1700* (Cambridge: Cambridge University Press, 2011): 6-14.

44 Simon Szreter, 'The Population Health Approach in Historical Perspective,' in *Health and Wealth. Studies in History and Policy,* ed. Simon Szreter (Rochester: University of Rochester Press, 2005); Simon Szreter 'Economic Growth, Disruption, Deprivation, Disease, and Death: On the Importance of the Politics of Public Health for Development,' *Population and Development Review* 23, no. 4 (1997).

45 Szreter, 'Economic Growth, Disruption, Deprivation, Disease, and Death.'

46 Riley, *Rising Life Expectancy.*

47 Ibid., xi, 11-16.

however, one other aspect, which should not be overlooked. Whatever intervention was put in place or whatever changes took place, acceptance by the population was a crucial factor. People needed to be willing to accept changes and incorporate them into their personal lives. First and foremost, a population had to become receptive to cultural and behavioural determinants, such as modern insights and attitudes, to manage health and illness.

The case of late nineteenth-century Amsterdam can illustrate the importance of behavioural adaptation.[48] According to Verdoorn, the decline in mortality in Amsterdam could not sufficiently be explained in terms of improvements in medicine or an increase in public health measures only. Cultural acceptance of modern medicine and public health were equally important as the changes these fields themselves. In order to interpret the decline in mortality rates in Amsterdam, Verdoorn used the concept of *'gezondheidswezen',* which can best be translated by the term *health system.* The health system is defined as the health condition of a certain place within a certain period, while taking into account cultural circumstances. The health system therefore resembles a dynamic process, instead of a state of being. Furthermore, the health system consists of two sides of the same coin, which can be understood in terms of production and consumption, or supply and demand. On the one hand, production or supply regulate the availability of, in this case, medical facilities and knowledge of hygienic practices. On the other hand, consumption or demand determine the acceptance and utilisation of these medical facilities and the adoption of hygienic practices. In order to improve the health system and to ultimately achieve a decline in mortality rates, improvements in both elements are required and need to be balanced. If medical facilities are improving, whilst the 'consumption' of these facilities is not incorporated in the prevailing cultural behavioural pattern, the health condition of a community will not improve and mortality rates will not decline.[49] Verdoorn's argumentation is predominantly based on the work of the sociologist Hofstee, who stressed the importance of the receptivity of a society to cultural changes and adaptation during modernisation.[50] This approach is particularly relevant to the situation in Maastricht: it has been used when analysing the Dutch province of Limburg's health system, with its capital city Maastricht.[51] Both Hofstee and Philips have argued that the health lag in the province of Limburg in relation to the rest of the Netherlands was at least partially the result of a slower acceptance of modern hygienic behaviour.[52]

The acceptance of modern medical practices could be achieved in several ways. First, the trickling down of new ideas from higher social strata to lower social strata

48 J. A. Verdoorn, *Het Gezondheidswezen te Amsterdam in de 19e Eeuw* (Nijmegen: SUN, 1981). This is a reprint of the original published in 1965: J.A. Verdoorn, *Volksgezondheid en Sociale Ontwikkeling. Beschouwingen over het Gezondheidswezen te Amsterdam in de 19e Eeuw* (Utrecht: Het Spectrum, 1965).

49 Ibid., 428-31.

50 E. W. Hofstee, 'De Groei van de Nederlandse Bevolking,' in *Drift en Koers. Een Halve Eeuw Sociale Verandering in Nederland*, ed. A. N. J. Hollander et al. (Assen: Van Gorcum, 1962).

51 Rudolf Philips, *Gezondheidszorg in Limburg. Groei en Acceptatie van de Gezondheidsvoorzieningen 1850-1940* (Assen: Van Gorcum, 1980).

52 E. W. Hofstee, *Korte Demografische Geschiedenis van Nederland van 1800 tot Heden* (Haarlem: Fibula-Van Dishoeck, 1981); Philips, *Gezondheidszorg in Limburg.*

was important. According to Rutten, this was also facilitated by medical practitioners. Nineteenth-century medical doctors benefitted population health not in their provision of curative care, but by functioning as intermediaries between the upper class and the working class in disseminating new hygienic and medical beliefs and practices.[53] In this fashion, ideas on hygiene and child care could reach the lower classes when they were already accepted by the upper classes.[54] Even more effective in improving health throughout the country, and especially in the more peripheral areas, was the 'community approach'. Organisations embedded in local communities were more likely to reach their goals than state officials who tried to improve health by a top-down approach.[55] For example, the Cross organisations (*Kruisverenigingen*) employed a bottom-up approach by visiting families at home and organising educational meetings for mothers. Cross organisations were private organisations with either a neutral or a religious background. In the pillarised Dutch society of the nineteenth century and the first half of the twentieth century all social structures were arranged around ideological or religious communities.[56] Here, a health organisation founded on common ideals was more likely to be accepted by the community than was the case with a paternalistic state official. The Limburg branch of the Green Cross was the organisation which aided the improvement of health in Limburg because of their community approach.[57]

1.2.3 Unequal distribution of determinants of mortality decline

Hygiene was not the only new ideas that was perhaps unequally distributed across different groups within society. Many resources required to combat disease and early death were potentially restrictively available, causing inequality in survival outcomes during the health transition. More distant and indirect phenomena operating through the direct determinants of mortality decline, could, therefore, effect inequal paths towards a lower mortality regime. In the widely used Mosley and Chen framework for the analysis of infant and early childhood mortality, socioeconomic status functions as the indirect determinant, which operates through proximate determinants such as nutritional factors and disease environment.[58] The mechanism of indirect and proximate determinants is a key factor in understanding the inequalities in mortality decline. This model will be discussed more elaborately in chapter 4 on infant mortality.

Socioeconomic status therefore can be viewed as an indirect yet fundamental determinant of mortality decline, which was proposed by Link and Phelan in their

53 W.J.M.J. Rutten, 'An Outline of Socio-Medical Care in the Netherlands, 19th and Early 20th Centuries,' in *Death at the Opposite Ends of the Eurasian Continent. Mortality Trends in Taiwan and the Netherlands 1850-1945*, ed. Theo Engelen, John R. Shepherd, and Yang Wen-shan (Amsterdam: Aksant, 2011), 157.

54 Ibid.

55 M. TH. Wijnen-Sponselee, *Het Wit-Gele Kruis in Noord Brabant: Intermediair Tussen Medische Verworvenheid en Sociale Acceptatie* (Tilburg: Stichting Zuidelijk Historisch Contact, 1997).

56 In the Dutch pillarised society, the four common pillars to which one could belong were the Protestant, Catholic, Liberal and Socialist pillars. The pillars provided an ideological ground for both institutions and organisations. Organised social interaction was predominantly contained to the pillar, and cross-overs between the pillars were rare. The ideological footing of the pillars was therefore highly important to the community.

57 Ibid., 161-63.

58 W. H. Mosley and L. C. Chen, 'An Analytical Framework for the Study of Child Survival in Developing Countries,' *Population and Development Review* 10 (1984).

fundamental causes theory.[59] They asserted that socioeconomic disparities in health will continue to exist, even when the dominant diseases, which at first expressed the differences in socioeconomic status, have diminished. The fundamental causes theory consists of four premises about the role of socioeconomic status as a fundamental health determinant. First, socioeconomic status must have an impact on more than just one disease outcome. Second, those outcomes are influenced by multiple risk factors. Third, the fundamental cause encompasses access to certain flexible resources, such as income and knowledge, which can influence health and risk exposure in various ways. Fourth, the association between socioeconomic status, as a fundamental cause, and health will not vanish over time, but will be replaced by other risks and diseases.[60] While the increased availability of resources, such as knowledge, money or power, will therefore help to combat certain diseases, other, new diseases will replace the old hazards. The new diseases will again cause inequalities based upon socioeconomic status which operate through the availability of resources.

However, the role of socioeconomic status in mortality decline is not straightforward to assess. Indeed, there has been decades of debate between scholars on the historical role of socioeconomic status in mortality decline. Before Link and Phelan identified socioeconomic status as a fundamental cause, Antonovsky observed a divergence and subsequent convergence in mortality patterns as a result of differences in socioeconomic status.[61] Although Antonovsky did not view socioeconomic status as a fundamental, interminable health discriminator, he found increasing socioeconomic inequalities in mortality after 1650. Before this period, diseases were simply too communicable to be effectively shielded from, even for particular resourceful social groups. Yet between 1650 and 1850, two distinct processes caused a divergence in health among social groups. The first process encompassed the slow development of an industrial lower class, while simultaneously upper classes experienced the benefits of economic improvements. The second process was of a biological nature, which caused highly virulent diseases to become less prevalent, and more nutrition-based diseases to emerge. Both processes benefitted the health of higher social groups, while the nascent industrial lower classes did not yet benefit health-wise. After 1850, when the phase of convergence was entered, class differentials started to diminish again during the mortality decline at the end of the nineteenth century, and they had almost disappeared by the middle of the twentieth century.[62]

Since recent research has shown again emerging class differences in health, Bengtsson and Van Poppel argued a divergence-convergence-divergence would be more applicable to the past two hundred years, instead of Antonovsky's divergence-convergence model.[63]

59 Bruce G. Link and Jo Phelan, 'Social Conditions as Fundamental Causes of Disease,' *Journal of Health and Social Behavior* (1995).

60 Ibid., 80-94.

61 A. Antonovsky, *Social Class, Life Expectancy and Overall Mortality* (New York: Milbank Memorial Fund, 1967).

62 Ibid.

63 Johan P. Mackenbach et al., 'Widening Socioeconomic Inequalities in Mortality in Six Western European Countries,' *International Journal of Epidemiology* 32, no. 5 (2003); Michael Marmot, *The Status Syndrome: How Social Standing Affects Our Health and Longevity* (New York: Henry Holt, 2004); Tommy Bengtsson and Frans van Poppel, 'Socioeconomic Inequalities in Death from Past to Present: An Introduction,' *Explorations in Economic History* 48, no. 3 (2011).

Moreover, Bengtsson and Van Poppel stated that some class differentials started to appear much earlier and were in fact no product of industrialisation. They also disagreed with the fundamental causes theory, since they found many historical studies did not observe such class differentials in mortality at all. Bengtsson and Van Poppel concluded that 'a consistent causal link between socioeconomic status and mortality is open to serious doubt'.[64]

However, the debate over a consistent causal link between socioeconomic status and mortality does not necessarily have to imply that the fundamental causes framework can be fully discarded. Again, the need for a cause-specific approach may be informative. Whereas inequalities in all-cause mortality may level each other out, certain inequalities may still exist at a cause-specific level. A recent, more specialised addition to the fundamental causes theory actually accommodates this issue. Clouston et al. extended the fundamental causes theory by the inclusion of a four-stage model, which applies to each individual cause of death and which distinguishes levels of inequality that are inherent in each stage.[65] Consequently, this urges a consideration of the particular context when analysing socioeconomic inequalities in health. The model departs from the idea that the prevention or cure of all causes of death is related to the availability and accessibility of *flexible resources*. These flexible resources include knowledge about a disease, power, money, prestige and beneficial social conditions.[66] The spread of availability and accessibility dictates the stage a specific disease goes through, which has implications for the differences in socioeconomic status among the deceased. A specific cause of death can pass through the stages of *natural mortality, producing inequality, reducing inequality* and *reduced mortality*.[67] The first stage of natural mortality is characterised by little knowledge about the disease and the disease is non-preventable. Inequalities can exist, but are more based upon either unknown factors or coincidence. It is not unlikely in this stage that higher socioeconomic strata actually experience higher mortality levels.

The seemingly random occurrence of a disease becomes more foreseeable in the second stage of *producing inequality*, in which medical discoveries have led to increased knowledge regarding the disease and the preventive possibilities. Provided that someone possesses the flexible resources, the disease becomes preventable or curable. Hence, a widening gap between the social strata arises, where the higher socioeconomic strata are in a more advantageous position and mortality rates in this group decrease. The more disadvantageous groups with lower socioeconomic status have to await the next stage of *reducing inequalities*, when either the flexible resources important for the specific disease have become more widely disseminated, or measures that affect the entire population have been taken. Only thereafter can the final stage of *reduced mortality* be reached, where either a disease is eradicated or a disease can still occur, however only in small numbers and indiscriminate of socioeconomic status.[68]

64 Bengtsson and Van Poppel, 'Socioeconomic Inequalities in Death from Past to Present,' 355.

65 Clouston et al., 'A Social History of Disease.'

66 Ibid., 1632.

67 Ibid., 1635-38.

68 Ibid.

For this research the extended fundamental causes theory proposed by Clouston et al. will provide an additional framework in the analysis of mortality inequalities emerging during the health transition. In particular, the availability of individual cause-of-death data, instead of only total mortality or aggregated cause-of-death data, enables the utilisation of the extended fundamental causes theory. This will allow for an assessment of individual cause-of-death categories. In addition to socioeconomic health inequalities, distinct health inequalities could arise from gender differences. Cause-specific mortality may vary between the sexes as a result of both biological factors related to sex, or cultural factors related to gender. Biologically, it has been shown that, especially in infancy, boys have lower resistance to infectious diseases. Moreover, women tend to outlive men in many societies nowadays.[69] Whereas women appear to have a biological health advantage over men, gender inequalities encompass more than just biological differences. Socially mediated behaviour, for example, causes men to engage in more risky behaviour in almost every society, while women tend to express more health-seeking behaviour.[70]

However, women may have historically been in a disadvantageous position compared to men for several reasons. When resources were limited, women were most likely to put their own health in last place or were put in last place, on account of gendered preferences. In the male-breadwinner family, resources were first and foremost allocated to the ones providing the income, the male breadwinner, and young adult sons present in the household. Since the entire family was dependent on the capacity of the male head of the household, nutrition and medical care were more available to him compared to younger or female family members.[71] Women were also more exposed to diseases, since they were traditionally expected to care for sick family members.[72] Finally, repeated pregnancies did not only put women at the risk of complications during childbirth, but the many births wore the women's bodies down. As a result, it has been shown that a female mortality disadvantage existed in the Netherlands as well, at least in rural areas.[73] In urban and industrialised areas, relative inequalities between men and women could be reversed; working class men were disproportionally affected by the harsh working conditions and work-related risks of urban and industrial labour markets, leading to substantially higher tuberculosis mortality.[74] For those reasons, gender is an important

69 Ingrid Waldron, 'Sex Differences in Human Mortality: The Role of Genetic Factors,' *Social Science & Medicine* 17, no. 6 (1983).

70 Mark R. Cullen et al., 'The Weaker Sex? Vulnerable Men and Women's Resilience to Socio-Economic Disadvantage,' *SSM - Population Health* 2 (2016).

71 Jane Humphries, "Bread and a Pennyworth of Treacle'. Excess Female Mortality in England in the 1840s,' *Cambridge Journal of Economics* 15 (1991).

72 George Alter, Matteo Manfredini, and Paul Nystedt, 'Gender Differences in Mortality,' in *Life under Pressure: Mortality and Living Standards in Europe and Asia, 1700-1900*, ed. Tommy Begtsson, Cameron Campbell, and James Z. Lee (Cambridge, Mass.: The MIT Press, 2004): 331; W.J.M.J. Rutten, 'Ongelijke Behandeling Binnen het Gezin. Een Onderzoek naar de Leeftijdsverschillen in de Kans op Geneeskundige Hulp in Nederland (Ca. 1870-1900),' *A.A.G. Bijdragen* 28 (1986): 246.

73 Angélique Janssens and Elien van Dongen, 'A Natural Female Disadvantage? Maternal Mortality and the Role of Nutrition Related Causes of Death in the Netherlands, 1875-1899,' *Tijdschrift voor Sociale en Economische Geschiedenis* 14, no. 4 (2017).

74 Andrew Hinde, 'Sex Differentials in Phthisis Mortality in England and Wales, 1861-1870,' *The History of the Family*

health discriminator that will be taken into account when looking into mortality inequalities in this research.

1.3 Structure of the thesis

The effects of socioeconomic status and gender on health outcomes varied throughout the life course. Both the extent of exposure to health risks and the nature of these risks changed when one grew older. Infancy and early childhood in particular were dangerous stages of life in the nineteenth and early twentieth centuries. Mortality rates among young children were exceptionally high across the Western world before the health transition. After the first five years of life, mortality risks decreased and the odds of survival were fairly good until early adulthood, while during adolescence and adulthood risks started to increase again. To account for health risks changing throughout the life course, the analyses in this dissertation will be conducted for different age categories. The three most important age categories with the most pronounced changes in mortality were infants (age 0 to 1), young children (ages 1 through 4) and adults (ages 20 through 49). The empirical chapters of this dissertation are structured accordingly. The age category of 50 years and older is not included in this study for two reasons. First, once one had survived until the age of 50, the chances of dying of an acute infectious disease decreased. The extent to which the epidemiological transition occurred in this age category is open for discussion, since many elderly already succumbed to degenerative diseases in the pre-transitional phase. Second, as a more practical consideration, the causes of death become more vaguely recorded for individuals over the age of 50, as old age and debility become more commonly used to describe the causes of death among the elderly. The information on socioeconomic status is also not as rich as for the older age categories, since this is derived from occupations, and many elderly did not have a registered occupation anymore.

Before turning to the analyses of each age group, chapter 2 will first discuss the dataset and the methods that have been used. The dataset used in this study is extremely rich and is a goldmine for historians with an interest in demography or the history of medicine. Moreover, the preservation of this type of individual-level cause of death for the duration of almost a century is unique in the Netherlands. The assembly of the database and the origin of the underlying historical sources will be further contextualised in chapter 2. The internal validity of the data, especially of the causes of death, will be scrutinised as well. Nineteenth-century causes of death can never be taken at face value because of their sometimes-symptomatic nature, as well as the changing medical knowledge and practices during this period. Finally, how the data was dealt with in terms of standardising, coding and classifying, and how the derivations of other population data were constructed, will be discussed.

Chapter 3 presents a discussion of the most important characteristics of historical Maastricht. This southern Dutch city, situated close to the borders with both Belgium and Germany, was special in several respects compared to the rest of the Netherlands as

20, no. 3 (2015).

a whole. Its proximity to the borders contributed to the unique characteristic of being one of the few truly industrialising cities the Netherlands has known. The industrial sector expanded from the 1830s onwards, bringing forth new challenges in terms of population health and living circumstances. This particular historical context is essential in understanding the following analyses on health inequalities in the period. Chapter 3 therefore provides an overview of the socioeconomic setting of the city, the demographic developments throughout the nineteenth and early twentieth centuries, and the state of public health organisations and medicine.

Chapter 4, the first analytical chapter, examines the emerging and receding inequalities in infant mortality. Maastricht, and the province of Limburg overall, was known for the extremely high infant mortality rates in contrast to the rest of the Netherlands. Whereas infant mortality started to decline in the north-western provinces from the 1860s onwards, it actually increased in Limburg in the late nineteenth century.[75] This chapter looks into the potentially emergent inequalities in epidemiological patterns before, during and after infant mortality finally started to decline from the twentieth century onwards.

The next chapter on early childhood mortality takes a slightly different approach. While mortality was high among young children, it was not as high as it was among infants. We therefore lack the numbers to perform the same analyses as for the other age categories. However, the nature of the disease environment among young children actually makes a different approach more desirable. Young children did not succumb to just a single dominant disease: In this stage of life children had to fight a whole range of different diseases. This particular disease environment is the subject of interest in chapter 5, which presents an analysis of the individual role of particular diseases. Even so, some socioeconomic and gender differences seem to have played a distinct role in the type of diseases children of this age died of.

The final analytical chapter, chapter 6, turns to an analysis of adult mortality. Among adults, the interaction between gender and socioeconomic status becomes even more relevant. The analysis concentrates on emerging inequalities according to socioeconomic status along gender lines. Although airborne infectious diseases were rampant among both men and women, the importance of other factors differed between the genders. Nonetheless, socioeconomic status proves to influence epidemiological patterns for both genders during mortality decline.

Finally, in the conclusion, we will evaluate the main emerging and receding inequalities in the course of the health transition in the Maastricht population. Which groups gained an early advantage, and which groups lagged behind? And what can we say about the most likely determinants for the pattern of receding mortality hazards and the emerging or disappearing inequalities that have been found in this study?

75 Peter Ekamper and Frans van Poppel, 'Zuigelingensterfte Per Gemeente in Nederland, 1841-1939,' *Bevolkingstrends*, no. 1 (2008).

CHAPTER 2

2

Data & methods

2.1 Introduction

At first glance death is the one event in life which seems to be devoid of constructive factors; eventually everybody dies. The analysis of death is nonetheless far from straightforward and, especially in historical analyses, death should be treated with caution. Not only is the event of death embedded in many constructed social practices, but death in itself could even be a contested event, as the issuing of the Burial Law in 1869 in the Netherlands demonstrates. The Burial Law required a certificate of death issued by a physician in order to prevent, accidently, being buried alive.[76] The analysis of death becomes even more complex when the causes of death are taken into account. Not only time-dependent medical knowledge influenced the reliability of the registration of causes of death, a range of cultural and institutional factors dictated the accuracy of cause-of-death registration. And while death in itself may seem to be devoid of many constructive factors, *causes of* death or diseases are regarded as social constructs, consisting of more than just biological phenomena.[77]

Thus, while historical cause-of-death statistics over longer periods represent on the one hand epidemiological changes, they represent historical changes in scientific and cultural views on diseases on the other hand.[78] Despite reliability issues stemming from this bilateral nature of historical cause-of-death data, individual cause-of-death data are a goldmine for historians when treated with caution. Individual level cause-of-death data add great analytical power to historical mortality studies compared to aggregated cause-of-death data, because of the higher degrees of detail and the greater analytical possibilities.[79] This chapter will discuss the reliability and use of historical cause-of-death data, in particular the data in the *Maastricht Death and Disease Database*, and how the data was dealt with and used in this dissertation. In order to deal with the reliability issues, a discussion of the most common reliability issues and the development of cause-of-death registration with the particular Dutch limitations will follow first. The focus thereafter shifts to the *Maastricht Death and Disease Database* and how the variables, in particular the causes of death, were coded and classified, and which analytical methods are used in the dissertation.

2.2 Interpretating historical causes of death

Despite the valuable insights historical causes of death can provide us in our study of historical populations, we do need to evaluate the limitations of such data. In the 1990s, Charles Rosenberg put forth his seminal ideas on the complexity of the entity of disease,

76 Frans Van Poppel and Jitse P. Van Dijk, 'The Development of Cause-of-Death Registration in the Netherlands, 1865-1955,' *Continuity and Change* 12, no. 2 (1997): 267.

77 Charles E. Rosenberg, 'Introduction. Framing Disease: Illness, Society and History,' in *Framing Disease: Studies in Cultural History*, ed. Charles E. Rosenberg and Janet Golden (New Brunswick: Rutgers University Press, 1992), xiii.

78 Nynke van den Boomen, 'Born Close to Death: Region, Roman Catholicism and Infant Mortality in the Netherlands, 1875-1899' (Radboud University, 2021), 56.

79 Angélique Janssens and Isabelle Devos, 'Introduction to the Special Issue the Limits and Possibilities of Cause of Death Categorization for Understanding Late Nineteenth Century Mortality,' *The Social History of Medicine* (2022).

which is more than 'a less than optimum physiological state'.[80] Instead, many constructive processes are involved in establishing a disease, as 'disease is at once a biological event, a generation-specific repertoire of verbal constructs reflecting medicine's intellectual and institutional history, an occasion of and potential legitimation for public policy, an aspect of social role and individual – intrapsychic – identity, a sanction for cultural values, and a structuring element in doctor and patient interactions'.[81] Many of these attributions are also valid for causes of death, although a cause of death may not necessarily be similar to a disease.[82] Causes of death are just as socially and culturally mediated, are verbal constructs dependent on the specific historical context and can be used politically, as disease entities. Using historical cause-of-death data without any consideration of what the data actually represent and taking the written causes of death at face value would therefore have the potential to provide an exceedingly positivist interpretation of what caused the deaths of historical populations.

A vast body of literature exists that deals with the complexity of historical causes of death.[83] Risse provided an insightful summary of the most common issues, in which he identified five major problems when using historical causes of death, constituting five shifts. These shifts are first a shift in the ecology of disease, second in the definitions of health and illness, third in the definition of death, fourth in the construction of death records and finally in the use of death records and statistics.[84] The first problem concerns the particular disease environment which determines the ecology of diseases. When certain highly deadly diseases are rampant, less people will suffer from other diseases. Once the highly deadly diseases recede, the occurrence of other diseases will most likely increase.[85] Diseases do therefore not occur in a vacuum, which is why the particular historical context and disease environment is highly important when studying epidemiological patterns. The second problem concerns the constructive processes in which illness and health are defined. Nosologies started to be created from the seventeenth century onwards in Europe, yet were often based on competing premises. Whereas earlier nosologies were mainly based on symptoms, the shifting ideas on the origin of illnesses caused the emergence of aetiological nosologies in the mid-nineteenth century. In earlier periods, a disease could be caused by a multitude of factors, but diseases came to be seen as being caused by a single prerequisite in later nosologies.[86] Third, not only

80 Rosenberg, 'Introduction. Framing Disease,' xiii.

81 Ibid., xiii.

82 K. Codell Carter, 'Causes of Disease and Causes of Death,' *Continuity and Change* 12, no. 2 (1997).

83 George Alter and Ann Carmichael, 'Studying Causes of Death in the Past. Problems and Models, an Introduction,' *Historical Methods* 29, no. 2 (1996); Douglas L. Anderton and Susan Hautaniemi Leonard, 'Grammars of Death. An Analysis of Nineteenth-Century Literal Causes of Death from the Age of Miasmas to Germ Theory,' *Social Science History* 28, no. 1 (2004); Stephen J. Kunitz, 'Premises, Premises. Comments on the Comparability of Classifications,' *Journal of the History of Medicine* 54 (1999); Reid et al., "A Confession of Ignorance.'; Günter B. Risse, 'Cause of Death as a Historical Problem,' *Continuity and Change* 12, no. 2 (1997); K. Codell Carter, 'Causes of Disease and Causes of Death.'

84 Günter B. Risse, 'Cause of Death as a Historical Problem.'

85 Mirko D. Grmek, *Pathological Realities. Essays on Disease, Experiments, and History*, ed. Pierre-Olivier Méthot , 1st ed. (New York: Fordham University Press, 2019); Risse, 'Cause of Death as a Historical Problem.'

86 K. Codell Carter, 'Causes of Disease and Causes of Death.'

the definition of diseases shifted, the definition of death itself also shifted. The assigned cause of a death can depend on more than just the observed disease; political or personal values, environmental influences and belief systems can affect what cause of death is reported.

The fourth problem concerns the actual practice of cause-of-death registration. Across Europe, cause-of-death registration already emerged in some cities from the seventeenth century onwards.[87] Yet the institutions in charge of the registration differed. In Sweden and in Britain, the clergy were tasked with the registration of causes of death. During the nineteenth century the responsibility was transferred to state bodies. Moreover, the attendance, or lack thereof, of a physician at the event of death and its registration influences the reliability of the prescribed death. Even when a physician was in charge of assigning a cause of death, it mattered whether the death certificate was handed directly to the officials, or to the relatives. In the latter case, stigmatised causes of death such as tuberculosis and syphilis are more likely to have been avoided.[88] Finally, the fifth problem Risse identified deals with the shifting use of death records and statistics. The records of cause-of-death statistics can be strong tools in setting public policy, which may have influenced the reporting of causes of deaths.[89] The strong emphasis on infectious diseases for the control of environmental factors and/or public health may cause differences in accuracy of reporting infectious versus chronic and degenerative diseases.[90]

2.3 Cause-of-death registration in the Netherlands

From 1811 onwards, the registration of death became mandatory in the Netherlands. The municipalities were required to keep track of the death certificates that had been issued, yet not from which cause of death someone had died.[91] However, local initiatives to register the specific cause of death had already emerged in the second half of the eighteenth century. The city of The Hague was the first to enact a cause-of-death registration in 1755, followed by the cities of Alkmaar, Rotterdam and Amsterdam in the 1770s.[92] The issuing of national regulations on the registration of causes of death took longer. Only when medical reforms had taken place in 1865, did the registration of causes of death became a nationwide mandatory practice.

The need for a thorough analysis of mortality became increasingly felt by physicians and 'hygienists' during the mid-nineteenth century, in order to improve the poor state of health of the majority of the Dutch population.[93] The private organisation

87 Reid et al., "A Confession of Ignorance."; Risse, 'Cause of Death as a Historical Problem.'; J.P. Van Dijk, *Doodsoorzakenclassificaties 1750-1950. Schets Van De Ontwikkeling Van Plaatselijke Sterftelijsten Tot Een Internationale Classificatie Van Doodsoorzaken* (Groningen: Rijksuniversiteit Groningen, 1981).

88 Reid et al., "A Confession of Ignorance'."; Risse, 'Cause of Death as a Historical Problem.'

89 Risse, 'Cause of Death as a Historical Problem.'

90 Alter and Carmichael, 'Studying Causes of Death in the Past.'

91 Van Poppel and Van Dijk, 'The Development of Cause-of-Death Registration in the Netherlands, 1865-1955,' 266.

92 Ibid.

93 M. G. Neurdenburg, *Doodsoorzaak en Statistiek* (Amsterdam: H. J. Paris, 1929): 26-27.

Nederlandsche Maatschappij tot Bevordering der Geneeskunst (NMG, Dutch Society for the Advancement of Medicine) was founded in 1849 by physicians and hygienists eager to do what their name stated: Advance public health and the practice of medicine. Part of their objective was to understand mortality differentials, for example by region, through statistical analysis. From 1854 onwards, they collected statistical information on mortality in the Netherlands from the Civil Registry and published their findings within their own circle. A prerequisite for their analyses was, however, a standardised format and classification of the causes of death. Deciding on such a standardised format was not an easy task, and discussions lingered on whether the causes of death should be classified on the basis of symptoms or aetiology. By the late 1850s, a preliminary model for the classification of causes of death came into use in Amsterdam, Middelburg, Leiden, Haarlem, The Hague and Schiedam. It was only in 1866, however, that the first national standardised classification of causes of death was introduced as a consequence of the issuing of new laws regulating the nation's practice of medicine and health care in 1865.[94]

This first classification contained only the following six diseases: Smallpox, scarlet fever, measles, typhus, angina diphtheria and cholera, and a final category for all other and unknown causes of death. The focus on only these diseases demonstrates the prime emphasis of physicians and health officials on diseases that were most disruptive to society: Contagious epidemic diseases.[95] The preoccupation with these diseases resulted in the issuing of a new law in 1872, regulating the monitoring of these disruptive, contagious diseases. The Contagious Diseases Act (*Wet op Besmettelijke Ziekten*) mandated the official monitoring of infectious diseases and compulsory quarantine for families who had experienced a death due to one of the contagious diseases cholera, typhus and typhoid fever, smallpox, scarlet fever, diphtheria and measles.[96]

In the following years until 1874, the format of cause-of-death registration was adjusted, resulting in a more sophisticated model including 55 causes of death, divided into 35 categories, in 1875. Contributing to the reliability of the recorded causes of death was the fact that, due to the medical laws of 1865, doctors were required to issue a medical certificate stating the cause of death of the deceased, which they had to hand over to the municipal authorities. However, as the municipal authorities were not obliged to request such a medical certificate, this resulted in a large share of deaths being reported as 'death without medical treatment', even when this may not have been the case. Once the Burial Act in 1869 was issued, the attendance of a certified physician stating the cause of death was safeguarded. A medical certificate became a prerequisite for burial, stating the deceased was indeed truly deceased and stating the specific cause of death.[97]

94 E. S. Houwaart, *De Hygiënisten: Artsen, Staat & Volksgezondheid in Nederland 1840-1890* (Groningen: Historische Uitgeverij, 1991): 127-40; Van Poppel and Van Dijk, 'The Development of Cause-of-Death Registration in the Netherlands, 1865-1955,' 267-69.

95 L. Ali Cohen, *Handboek der Openbare Gezondheidsregeling en der Geneeskundige Politie, met het Oog op de Behoeften en de Wetgeving van Nederland* (Groningen: Wolters, 1872): 191-98; Houwaart, *De Hygiënisten*, 208-09.

96 'Wet Houdende Voorzieningen Tegen Besmettelijke Ziekten,' *Staatsblad* 134 (1872).

97 Van Poppel and Van Dijk, 'The Development of Cause-of-Death Registration in the Netherlands, 1865-1955,' 271-72; Nynke van den Boomen, 'The Imperfections in Statistics: Interpretations of Causes of Infant Death in the

Despite the introduction of the 1875 formal classification of causes of death, the registration of causes of death by doctors was not flawless. A first problem was that stillborn infants were under-registered due to the lack of a definition of when a foetus became an actual child. Second, doctors continued to provide medical certificates stating the cause of death without having attended the patient. Third, the causes of death doctors registered continued to be based on a mix of aetiological, clinical and pathological/anatomical factors. Fourth, since confidentiality was not entirely ensured, doctors were reluctant to register causes of death that could harm the family's reputation, resulting in a severe under-registration of diseases such as syphilis, alcoholism and suicide in the official statistics. Finally, doctors could have specific 'pet' causes of death which they would use more often than others. These factors caused inconsistencies in cause-of-death registration across regions or over time in the Netherlands.[98]

A similar problem occurred after the introduction of the Contagious Diseases Act in 1872 (*Wet op Besmettelijke Ziekten*). The consequences for the family income when the family was obliged to quarantine and therefore not allowed to go to work could be detrimental, to which doctors were not unsympathetic. Instead, doctors could turn a blind eye to the infectious disease, and report a different cause of death. These practices particularly occurred in cases of *febris typhoidea*, since doctors may not have believed it was actually contagious. They would report the cause of death as *febris acuta, febris* remittens or *febris continua* instead.[99]

Contemporaries recognised the problematic cause-of-death registration in the late nineteenth century, which led to improvements in 1901. From 1901 onwards, all causes of death would be processed by the Netherlands Central Bureau for Statistics, which was founded in 1899, according to the first International Classification of Diseases and Causes of Death.[100] The International Classification of Diseases and Causes of Death was the result of the endeavours of a European initiative to come up with an internationally uniform classification system for the registration of causes of death. From the 1850s onwards, scientists from several European countries had tried to achieve an agreement on the kind of classification that should be used. Different nosologies had been proposed by William Farr and Marc D'Espine in the 1850s, who had been tasked with the formulation of such a classification. A suitable compromise for all participating European countries could not yet be found because of the different premises of the nosologies. While Farr proposed a nosology based on the location of the cause of death in the body, D'Espine proposed a nosology based on the acute or chronic nature of the cause of death. Only after the foundation of the International Institute for Statistics in 1887 did Jacques Bertillon propose a new classification based on the location of the disease causing death in 1893. Bertillon's classification formed the basis for the first uniform International Classification of Diseases and Causes of Death, and countries

Netherlands, 1875-1899,' *Social History of Medicine* (2021).

98 Ibid., 273.

99 Ibid., 273-74.

100 Ibid., 277.

from both the Americas and Europe started using the international classification during the late 1890s and the early twentieth century.[101]

Yet another problem remained: The issue of confidentiality. Undertakers, family members or insurance companies were still able to get hold of the alleged confidential information on the medical certificate, which was a thorn in the side of doctors. Various regulations were added to ensure confidentiality throughout the early twentieth century. In 1927, the medical certificate of death was split into two parts, form A and B. Form A was closest to a death certificate, stating the name of the doctor and the cause of death, while Form B contained detailed information on the death for statistical purposes. Reforms in 1956 finally ensured complete confidentiality, since from then onwards, only Form B should state the cause of death and the complications, and Form A only contained information about whether the death had been a natural death.[102]

2.4 The Maastricht Death and Disease Database

In the Netherlands, cause-of-death data were gathered and published for each municipality from 1875 onwards. Between 1875 and 1901, a list consisting of 55 causes of death, divided over 34 groups, was used to aggregate the causes of death for the publication of the cause-of-death tables per municipality.[103] Many cause-specific mortality studies in the Netherlands make use of these published cause-of-death data.[104] Although the study of these aggregated data have deepened our understanding of the epidemiological transition in the Netherlands, they cannot offer exhaustive answers to questions on historical mortality developments. Two characteristics of the published data restrain us from acquiring a comprehensive understanding of historical mortality in the Netherlands. First of all, the fact that the published cause-of-death data are aggregated, instead of at an individual level, causes some information to remain out of sight. While the published mortality statistics stratify according to gender and age, information on individual socioeconomic status and other personal factors is not available. Second, historical classification systems can obscure the distribution behind the aggregation and stratification. One example is age, which is often reported in categories. Producing a time series of age-specific mortality becomes even more complicated as the boundaries of the age categories differ over time in the published statistics.

101 Van Dijk, *Doodsoorzakenclassificaties 1750-1950*, 21-23.

102 Centraal Bureau voor de Statistiek, *Van de Schaduw des Doods tot een Licht ten Leven: De Historie van de Methodiek van de Doodsoorzakenstatistiek in Nederland 1865-2005* (Voorburg/Heerlen: Centraal Bureau voor de Statistiek, 2005): 58-65; Van Poppel and Van Dijk, 'The Development of Cause-of-Death Registration in the Netherlands, 1865-1955,' 279-83; Jan van Sonsbeek, 'Geschiedenis Van De Nederlandse Doodsoorzakenstatistiek,' *Bevolkingstrends*, no. 3 (2003): 36-37; Centraal Bureau voor de Statistiek, *Van de Schaduw des Doods tot een Licht ten Leven*, 58-65; Van Poppel and Van Dijk, 'The Development of Cause-of-Death Registration in the Netherlands, 1865-1955,' 279-83; Van Sonsbeek, 'Geschiedenis Van De Nederlandse Doodsoorzakenstatistiek,' 36-37.

103 Wolleswinkel-van den Bosch, 'The Epidemiological Transition in the Netherlands,' 287-92.

104 Van den Boomen, 'Born Close to Death.'; Evelien Walhout, 'An Infants' Graveyard? Region, Religion, and Infant Mortality in North Brabant, 1840-1940' (Tilburg University, 2019); Wolleswinkel-van den Bosch, 'The Epidemiological Transition in the Netherlands.'

Even more challenging is the classification of causes of death according to the 34 groups, which contain categories that we no longer find to be of the same aetiology. This classification was based partially on the location in the body of the disease causing the death, and partially on aetiology. By doing so, diseases that are different in our medical understanding because of their aetiology were put into the same group of causes of death. The diseases typhus and typhoid fever were combined into the same group, although they differ a great deal. Convulsions were combined with trismus and epilepsy, while convulsions could be used for a plethora of diseases.[105] Syphilis, rickets and scrofula were also combined into a category, thus the same group constitutes both infectious and non-infectious diseases.

These two limitations cease to be an issue when using individual-level cause-of-death data. Yet other challenges remain, both in dealing with the data in a structured manner and in dealing with reliability issues and vague causes of death. Moreover, profound knowledge of the dataset and its specific features, applying source criticism, and transparency about the data are key in using historical datasets.[106] I will therefore discuss how the data in the *Maastricht Death and Disease Database* were handled, coded and classified in the following section.

The *Maastricht Death and Disease Database*[107] was compiled at the Centre for the Social History of Limburg (*Sociaal Historisch Centrum Limburg, SHCL*), based on the registers of the municipal cemetery and death certificates. The Maastricht municipal cemetery was founded in 1811/1812 for all religions, after Napoleon had prohibited burials in churches. Every interment was meticulously registered by the cemetery, in order to keep track of the exact location of the grave at the graveyard, of the grave dues of the family members and of the duration of the grave rights.[108] From 1864 onwards, the registers of the municipal cemetery also included the cause of death of each individual that had either died within the municipality or had been a legal citizen of Maastricht.[109] Even if the individual was not buried at the municipal cemetery, it was still included in the register. The registers continue well into the twentieth century; only after 1956 do the registers cease to mention the causes of death. The termination of this practice in 1956 is no surprise as improved confidentiality legislation dictated that Form A,

105 Hermann Lebert, *Handboek der Praktische Geneeskunde*, A. Drielsma trans., 4 vols (Groningen: Wolters, 1861-1863) IV, 35; H. Pinkhof, *Vertalend en Verklarend Woordenboek van Uitheemsche Geneeskundige Termen* (Haarlem: De Erven F. Bohn, 1923).

106 Joris Roosen and Daniel R. Curtis, 'Dangers of Noncritical Use of Historical Plague Data,' *Emerging Infectious Diseases* 24, no. 1 (2018).

107 Willibrord Rutten, *Maastricht Death and Disease Database*. The SHCL initiated the project of constructing this database, which was carried out under the supervision of dr. Willibrord Rutten, with the help of data-entry clerk Hans Janssen, of Monique Kilsdonk and Michel Lemaire, and of the student assistants Celine Huits, Inge Leurs, Tom op het Veld, Lenn Gorissen and Tanyth Warat from Maastricht University. The database is not published yet, access for scientific purposes was granted for this research on the condition that personal information would be anonymised in publications using the data from the database.

108 See Introduction to the inventory, Algemene Begraafplaats Maastricht, 1840-1987, access number 20.073, Regionaal Historisch Centrum Limburg (Centre for the Regional History of Limburg).

109 Registers van overlijdens en vermissingen, 1862-1985, inventory number 19-46, Gemeentebestuur van Maastricht: stukken afkomstig van de afdeling Burgerlijke Stand, 1880-1986, access number 20.007T, Regionaal Historisch Centrum Limburg (Centre for the Regional History of Limburg).

which was given to the local register of deaths, no longer stated the cause of death of an individual. However, the start and long continuation of this practice is more complex. The registration at the municipal level of causes of death alongside personal information such as the name of the individual for such a long period of time is unique for the Netherlands. Whereas in Amsterdam the registration of causes of death of each individual was combined with some individual characteristics from 1852 onwards, the inclusion of the name of each individual was not deemed relevant from an epidemiological point of view.

There are no conclusive answers as to why the Maastricht municipal authorities registered causes of death combined with such detailed personal information, but there are some indications for the origin of the practice. In Limburg, the Provincial Committee for Medical Research and Inspection (*Provinciale Commissie voor Geneeskundig Onderzoek en Toevoorzicht*) had been installed in 1818 as a result of new national legislation.[110] Despite the centralised character of these committees, public health policy – or the lack thereof – remained a responsibility of local municipal councils. Local municipalities were mostly reluctant in taking the advice of the committees to heart. However, the committee was responsible for reports on the status of health in the province to the Maastricht city council. The request by the Ministry of Internal Affairs in 1862 to the committee to compile statistical tables of mortality,[111] appears to have been an incentive for the recording of causes of death. Whether these tables ought to represent all-cause mortality only or cause-specific mortality remains unclear.

Two years later, in 1864, the report of the committee to the city council included a detailed table containing all the deaths that had occurred in Maastricht, specifying the exact cause of death.[112] According to the report, compiling such a table was desirable in the light of science. Moreover, local doctors were said to be very cooperative in providing the information on causes of death.[113] Although these very positive notes may be taken with a grain of salt, it does enhance the reliability of the data, as the majority of doctors will probably have provided the information. The raw information was collected in the registers of the local cemetery, which appears to have been part of the responsibilities of the committee as well.[114] Thus, while the exact link between the data and the responsible organisation remains obscure, it is most likely that the recording of cause-of-death data in Maastricht was a result of the request by the Ministry of Internal Affairs in 1862.

The digitisation of the burial registers and the death certificates by the Centre for the Social History of Limburg resulted in a dataset containing 76,264 individual records.[115] The linkage of the entries in the burial registers to the death certificates was

110 R. B. M. Rigter, *Met Raad En Daad. De Geschiedenis Van De Gezondheidsraad 1902-1985* (Rotterdam1992): 18.

111 Minutes 1862, inventory number 2, Provinciale Commissie voor Geneeskundig Onderzoek en Toevoorzicht in Limburg, 1818-1866, access number 07-G04, Regionaal Historisch Centrum Limburg (Centre for the Regional History of Limburg).

112 Gemeente Maastricht, *Verslag van den toestand der Gemeente Maastricht over het jaar* 1864 (Maastricht, 1864).

113 Ibid.

114 Ibid.

115 The dataset did contain some duplicates, predominantly from stillbirths and persons who had died during the war. Cases which could be determined with certainty to be duplicates were removed. Stillbirths, however, could have been twins; duplicate stillbirths with different certificate numbers were therefore included in the dataset.

relatively straightforward; each entry in the burial register includes the corresponding administrative number on the death certificate.[116] The available individual information in the dataset is very rich; it includes information on religion, place of death, date of death, first and last name, names of the parents, marital status, last name of the spouses, occupation at death, the occupation of the parents (if they were still alive), place of birth, place of residence, gender, age and, last but not least, cause of death.[117] The general descriptive statistics of the main characteristics of the database population can be found in Table 2.1. There is a distinction made between the numbers of the entire database and the total of the subpopulations based on the age categories used in the analyses.

The main variable of interest, the cause of death, consisted of 13,684 unique entries, which comes down to 6,823 tidied individual causes of death.[118] Next, all tidied causes of death were standardised, meaning that all tidied causes of death referring to the same cause of death received the same standardised term and a numeric code according to the ICD10H, an adaptation of the *International Statistical Classification of Diseases and Related Health Problems* (ICD-10).[119] The use of the ICD10H enables the fast processing of causes of death, while historical variations in terminology were retained by providing a slightly different code for each historical variation.[120] In a few instances a cause of death was coded differently from the ICD10H when local Dutch diagnostic practices were clearly different from the ICD10h classification.[121]

116 The death certificates had already been digitised by the Centre for the Regional History of Limburg. This datafile has been made available for the linkage by the RHCL with the help of dr. Erwin Steegen.

117 An overview of all the MDDD variables and an explanation of them when needed, can be found in Annex I.

118 Tidied causes of death means the individual causes of death devoid of spelling inconsistencies. Terms were also translated into the most commonly used term, especially for causes of death in French or German. Tidying causes of death comes with choices of what should be tidied and what not, thus the 6,823 tidied causes are not a number written in stone. Terms that we believe indicate the same cause of death, but are linguistically different, were only merged in the process of standardisation, however.

119 World Health Organization, *The International Statistical Classification of Diseases and Related Health Problems, 10th revision* (Geneva: World Health Organization, 2009).

120 Janssens, 'Constructing Ship and an International Historical Coding System for Causes of Death.'

121 The ICD10H is the result of the joint research network SHiP, which consists of research groups across Europe, with English as its working language. One cause of death which had a different definition in English compared to Dutch is angina. In the Netherlands, angina was used to describe inflammation of the throat, while the British know it only as angina pectoris, a heart condition. Angina in this case therefore received a code belonging to the inflammations of the respiratory system. However, whereas angina for children is most likely inflammation of the throat, in case of people in old age it may, in fact, represent angina pectoris. Nervous fever was also coded differently in this research. Nervous fever is often used to describe typhoid fever, however, until the 1870s, it appears many doctors were not able to distinguish between typhus and typhoid fever. According to the ICD10H, nervous fever should be coded as typhus. The Dutch medical literature, though, clearly states nervous fever was used to describe typhoid fever. Since the majority of the research deals with mortality after 1870, nervous fever was coded as typhoid fever. Still, one could easily move the code for nervous fever to a different category in the phase of classifying all the causes of death. The codes do correspond to the matching ICD-10 category, and therefore some codes deviate from the ICD10H to allow for diagnostic differences between countries. By distinguishing between coding and classifying, such a multi-interpretable cause of death can be classified differently for separate elements of the analysis.

Table 2.I Database descriptives

Variable		N total database	Combined N of age categories studied*
Gender	Male	37,884	19,506
	Female	34,635	15,922
	Unknown	3,745	1
Religion	Roman-Catholic	71,923	33,855
	Dutch-Reformed (*Nederlands Hervormd*)	1,035	392
	Other protestant	758	424
	Jewish	732	270
	Other religion	344	51
	No religion	612	50
	Unknown	860	387
Place of residence	Maastricht	71,333	33,119
	Province of Limburg	3,634	1613
	Netherlands, excluding Limburg	488	268
	Belgium	635	331
	Foreign country, excluding Belgium	111	70
	Unknown/ill-specified	63	28
Place of birth	Maastricht	52,507	28,187
	Province of Limburg	12,719	3,714
	Netherlands, excluding Limburg	5,348	1,682
	Belgium	3,590	1,111
	Germany	1,603	550
	Foreign country, excluding Belgium and Germany	396	153
	Unknown/ill-specified	101	32
Age at death	Stillbirth	5,106	
	<1	15,930	15,930
	1-4	7,674	7,674
	5-19	4,233	
	20-49	11,825	11,825
	50-64	10,446	
	65>	20,821	
	Unknown	229	
Total		**76,264**	**35,429**

* Including age categories of infants (<1), young children (1-4) and adults (29-49), excluding stillbirths, older children (5-19), older adults (50-64), elderly (65>) and age unknown.

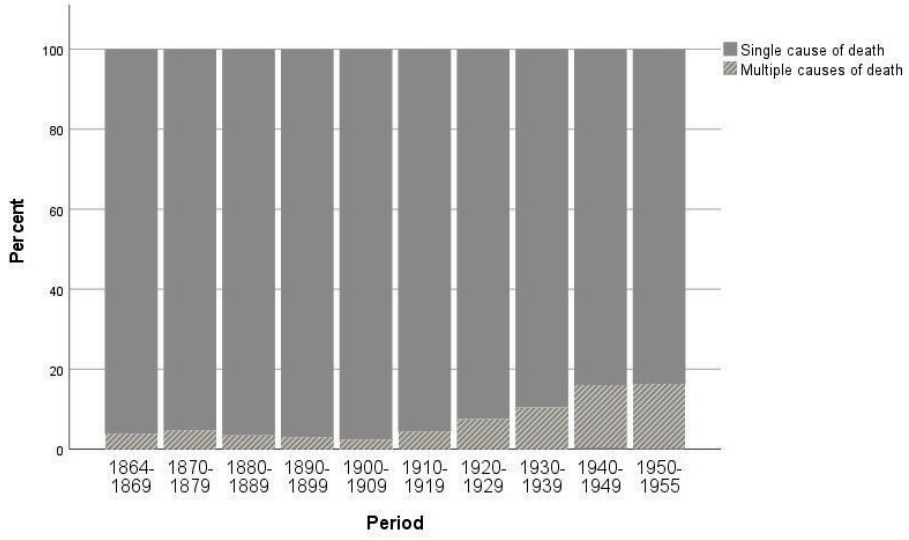


Figure 2.1 Percentage of registered multiple causes of death on total causes of death, Maastricht 1864-1955

Source: Maastricht Death and Disease Database
* Including age categories of infants (<1), young children (1-4) and adults (29-49), excluding stillbirths, older children (5-19), older adults (50-64), elderly (65>) and age unknown.

The vast majority of causes of death were straightforward to interpret as far as assigning it a specific code, except for 0.86% of the causes, which were not interpretable at all (not including unknown or those stating something along the lines of buried elsewhere). In the reporting of multiple causes of death, however, new challenges arose as regards coding. Multiple causes of death were recorded for only 5,268 cases.[122] Until 1919, between 2% to 5% of the cause-of-death entries in each decade contained multiple causes of death (Figure 2.1). Only after 1920 was there an increase of multiple-cause-of-death registration, reaching 16% in the 1940s and 1950s. The likelihood of being ascribed an additional cause of death also increases with age. In 3% to 5% of the causes of death in early childhood, there are multiple causes of death registered, while this is 8% for late childhood and adults, and 9.5% for people in old age (65 years and older). The higher likelihood of having multiple causes of death registered in old age is no surprise because of the higher probability of death caused by comorbidity of several diseases. However, this comorbidity may cause difficulties in deciding in hindsight which cause of death was the primary cause of death.

To decide which cause of death was indicated as the primary cause of death and would thus be used in the analysis, all the multiple causes of death were first divided into

122 This number depends on what would be considered a multiple cause of death. When two or more different causes of death were mentioned, it was indicated as a multiple cause of death. However, deciding when something is a multiple cause of death is not always as straightforward. When, for example, the cause was stated as 'unknown, likely convulsions', the cause was treated as a single cause. Yet when it was stated as 'art. 50 BW, convulsions', it was treated as a multiple cause of death, since there were two indicators of information, although the art.50 BW only provides information on the fact that someone had died outside of the city.

five categories of type of multiple causes of death, in order to establish some hierarchy in the multiple causes of death. The first group contains multiple causes of death which are in fact the same cause. Causes of death which could have multiple locations, such as cancer and tuberculosis, were very common in this category. Causes of death with additional information like bleeding or perforation which clearly belonged to the cause of death were also assigned to this group. Finally, causes of death that appeared to be multiple, but together have a single category in the ICD10H, were put in this group (for example, aortic insufficiency and syphilis is a specific type of syphilis). The second category is similar to the first since it includes multiple causes of death that belong to the same cause of death, the only difference being that it covers complications or subcategories of an ICD10H code instead of, for example, multiple locations. A very common example is measles with pneumonia, pneumonia being a common complication of measles. This category also includes accidents in which the accident and the internal consequence were mentioned, such as a fall with a cranial fracture. The third category comprises multiple causes of death which include only an additional symptom, such as convulsions, exhaustion or old age. For these first three categories, the primary cause was straightforward to decide, since the secondary causes only consisted of a different location of the same disease, a complication or a symptom.

The fourth category consisted of multiple causes of death which were not related to each other in the sense of complications or symptoms. In this category the complexity of deciding which cause of death is the primary cause of death increased, since it is not always clear at first sight. The strategies to determine the primary cause were first to indicate which registered cause of death was most likely to end in death, and which would be a cause of morbidity or physical discomfort, but not as likely to result in death. The differences between acute and chronic diseases, and infectious and non-infectious were often indicative of which cause of death would be the primary cause of death.[123] The cause which results in death would be assigned as the primary cause. The SHiP codebook was consulted to help determine this. When there was no conclusive answer, I looked into the use of indicative words and phrases, such as the use of after or as a consequence of, used by the registrar to establish a hierarchy in the causes of death. If that was not available as well, the last resort was to indicate the first mentioned cause of death as the primary cause of death. Finally, a fifth category comprises the multiple causes of death related to stillbirths or complications at birth. This is a very specific category in which the added cause of death offered additional information, but in which both primary and secondary causes would be classified as complications at birth. In the analyses, the designated primary causes of the entries with multiple causes of death were used to determine the cause of death.

Dealing with multiple causes of death and assigning one as the primary cause of death to use in the analysis challenges the reliability of the data, since the chances of misinterpretation of the actual cause of death increase. However, by adopting a methodological approach to determine the primary cause of death, it became clear that in nearly half of all the cases with multiple causes of death it was straightforward to determine the primary cause of death (Table 2.2, categories 1, 2, 3, 5). It was more

123 For example, someone who had suffered from atherosclerosis and pneumonia is likely to have died of pneumonia, since atherosclerosis would be considered to be less acute.

difficult to establish the exact primary cause of death in the other half of the multiple causes of death. We should not be too pessimistic, since in many instances the two causes of death would both be classified in the same category, for example afflictions of the heart. Moreover, for the analyses performed in this dissertation the multiple causes of death are only a very small proportion of the studied causes of death. Most multiple causes of death occurred in ages over 50 (54%), which is beyond the scope of this research. When studying deaths in old age, we should be extra careful with the multiple causes of death, as the occurrence of multiple causes of death is more common in this age category.

Table 2.2 Determination strategies for multiple causes of death

Category multiple causes of death	n	Percentage of total multiple causes of death	Determination of primary cause
1. Same cause	506	9.6%	Same cause of death
2. Complications/subcategories	786	14.9%	Main cause leading to the complication
3. Symptoms	745	14.1%	Main cause
4. No clear hierarchy in causes of death	2960	56.2%	- Cause leading to death - Indication of disease hierarchy in wording used by registrar - First mentioned cause of death
5. Complications at birth	271	5.1%	Cause leading to death
Total	**5268**	**100%**	

In addition to the coding of causes of death, the occupations also required standardisation, coding and classification as occupations were used to proxy socioeconomic status. There is no information on income or educational levels which could make the socioeconomic status indications more robust. Fortunately, in most cases within our age groups of interest an occupation was available, with the exception of adult women.[124] For each separate chapter, the occupations of interest were coded and classified according to the international HISCO and HISCLASS coding and classification systems for historical occupations.[125] In the case of infant and early childhood mortality, the occupations of the parents were utilised. For adult men, I used the occupation of the deceased. If an occupation was available for adult women, that was used as well. However, since the registration of female occupations was either deemed less important, or because women left the labour market after marriage,[126] an occupation

124 The exact numbers of the known occupations of the deceased or of their parents can be found in the individual chapters.

125 Marco H.D. Van Leeuwen and Ineke Maas, *Hisclass. A Historical International Social Class Scheme* (Leuven: Leuven University Press, 2011); Marco H.D. Van Leeuwen, Ineke Maas, and Andrew Miles, *Hisco. Historical International Standard Classification of Occupations* (Leuven2002).

126 Ad Knotter, *Arbeid Van Vrouwen in Limburg in de Twintigste Eeuw: Een Stille Revolutie* (Zwolle: WBooks/ Sociaal Historisch Centrum Limburg, 2018); Ariadne Schmidt and Elise van Nederveen Meerkerk, 'Reconsidering the 'Firstmale-Breadwinner Economy': Women's Labor Force Participation in the Netherlands, 1600–1900,' *Feminist*

was only available for 19% of the adult women. This is a well-known problem across studies into adult socioeconomic status in Europe.[127] As a consequence, the inclusion of all women with an estimation of socioeconomic status is even more important, because we do not want to leave them aside for the simple reason that it would be easier. Most studies solve this by using the occupation of the husband, since the majority of adult women were married.[128]

For the remaining 80% of adult women, the occupation of the husband was thus used to approximate socioeconomic status of the women. Automatically linking women to their husbands and their husbands' occupations at death was not possible, since only the last names of the spouses are included in the database. Finding an approximation to the socioeconomic status of the women therefore had to be done by hand. Although this is very time-consuming, it has a great advantage as well. Finding the husbands of these women by hand created the opportunity to not only use the occupations mentioned in the death certificates, but also in marriage certificates, the population registers or the death certificates of children. As a result, the validity of the husband's occupation to indicate the woman's socioeconomic status increases, since the certificate closest to the death of the women could be used. In the case a husband died over twenty years after the death of his wife, his occupation at death is less valid compared to when the timing of his death was closer to his wife's death, since he may have moved up or down the social ladder.

In order to find the certificates mentioning the occupation of the husband closest to the woman's death, we used WieWasWie, a digital tool that includes almost all civil certificates in the Netherlands.[129] [130] By entering the name of the deceased woman in *WieWasWie*, we were able to find the digitally available certificates in which she was mentioned. The certificate containing information on the occupation of the husband closest to her death was used, and we kept track of the amount of years between the death of the woman and the timing of the certificate on which we based her socioeconomic status. In cases where there was a long period (over ten years) between the death and the certificate, there was another possibility to check whether another certificate, closer to death, could be found. If women remained unmarried throughout their life, the occupation of the father, the head of the household, or her own occupation in an earlier document were used. In the end, an indication of socioeconomic status was available for 91% of the adult women.

Economics 18, no. 4 (2012).

127 Bengtsson and Van Poppel, 'Socioeconomic Inequalities in Death from Past to Present.'

128 Tommy Bengtsson and Martin Dribe, 'The Late Emergence of Socioeconomic Mortality Differentials: A Micro-Level Study of Adult Mortality in Southern Sweden 1815-1968,' *Explorations in Economic History* 48, no. 3 (2011); Sören Edvinsson and Marie Lindkvist, 'Wealth and Health in 19th Century Sweden: A Study of Social Differences in Adult Mortality in the Sundsvall Region,' *Explorations in Economic History* 48, no. 3 (2011); Robert Woods, *The Demography of Victorian England and Wales* (Cambridge: Cambridge University Press, 2000).

129 *WieWasWie. Iedereen heeft een geschiedenis*, June-December 2021, https://www.wiewaswie.nl/ WieWasWie includes almost all the certificates of the Civil Registry of the Dutch municipalities. However, the certificates from the citizens of Amsterdam are missing.

130 In this case, some extraordinary volunteers and student assistants have assisted me with this painstaking endeavour. I am especially grateful for the help of Nicole Demas, Eva-Britt Rongen, Maria Reinders, Roxali Bijmoer and, last but not least, Dymphie van der Heyden.

2.5 Interpreting historical causes of death

The next step of dealing with historical cause of death data, classifying causes of death, involves a more substantial interpretation of the causes of death. The classification used depends moreover highly on the chosen research design, which in this dissertation focuses primarily on the shifts in the epidemiological regime between different types of infectious diseases or from infectious diseases to non-infectious diseases. These changes depend mainly on the age group, since disease environments were different according to age group. The age groups most affected by infectious diseases prior to the epidemiological transition were infants, young children and adults. The type of infectious diseases most rampant in each age category differed. Whereas infants were highly affected by gastrointestinal infections (water and foodborne infectious diseases), young children were more likely to suffer from typical childhood diseases such as scarlet fever, measles and diphtheria (airborne infectious diseases), and the vast majority of adults suffering from an infectious disease were affected by tuberculosis (airborne infectious diseases).

Distinguishing between different infectious diseases, and preferably even according to their route of transmission, is therefore paramount. Demarcating between infectious and non-infectious diseases in historical causes of death is, however, more complex than it may look at first sight. The definition of an infectious disease may seem very straightforward: The Dutch National Institute for Public Health and the Environment defines infectious diseases as those diseases that are caused by micro-organisms such as bacteria, viruses, fungi or parasites.[131] But how does one identify an infectious disease in an era where ideas about contagion were very different from current medicine, bacteriology was in its infancy and what constituted an infectious disease was still much debated?[132] Historians have discussed the fact that many registered causes of death in the early and mid-nineteenth century were mere symptoms such as fever and convulsions.[133] A thorough discussion of what would be deemed an infectious disease at a time when bacteriology was developing is often lacking, while this is essential for the demarcation between infectious and non-infectious diseases.

The usual suspects of infectious diseases such as smallpox, tuberculosis and scarlet fever are easily identified, and mid to late nineteenth-century physicians were quite capable of identifying these diseases. For symptom-like causes of death it becomes more difficult to establish whether they represented infectious diseases. Beyond the symptoms, the question remains what to do with inflammations? They are often the consequence of an infection, yet not necessarily so and depending much on the location in the body. Unfortunately, there is no conclusive answer whether death was caused by an infectious disease for every single registered cause of death. We should, however, be aware that

131 Rijksinstituut voor Volksgezondheid en Milieu, 'Beschermen tegen infectieziekten', *Rijksvaccinatieprogramma*, (2022), 28-06-2022, https://rijksvaccinatieprogramma.nl/infectieziekten.

132 See for example: Thomas Rütten, 'Introduction,' in *Contagionism and Contagious Diseases. Medicine and Literature 1880-1933*, ed. Thomas Rütten and Martina King (Berlin: De Gruyter, 2014).

133 Alter and Carmichael, 'Studying Causes of Death in the Past.'; Codell Carter, 'Causes of Disease and Causes of Death.'; Günter B. Risse, 'Cause of Death as a Historical Problem.'; Van Poppel and Van Dijk, 'The Development of Cause-of-Death Registration in the Netherlands, 1865-1955.'

not only do symptom-like causes of death create bias, but the foundational difference between infectious diseases and non-infectious diseases is in and of itself complex in historical research.

The demarcation made by Wolleswinkel-van den Bosch was used as a guideline to determine what an infectious disease was and what not, which is based on the modern understanding of what an infectious disease is.[134] In addition, indispensable historical medical literature was used to classify the puzzling causes of death. Some of these causes of death deserve some extra attention. The first, most prominent example is the much-used disease-term convulsions, which is indeed difficult to interpret. Infants suffered most from convulsions (*stuipen* or *eclampsie* in Dutch), although the term eclampsia is also used for women in the early stage of pre-eclampsia, a highly dangerous complication in pregnancy. Convulsions in infancy have been regarded predominantly as representing diarrhoea or gastrointestinal infections.[135] Contemporary medical literature nonetheless discusses a range of other affections that might be related to convulsions in infancy.[136] The usage of convulsions as a cause of death in Maastricht was highest in the period between 1864 and 1869, for which 9% of all causes of death in the dataset consisted of convulsions. The term was already used less often in the 1890s, when only 4% of the causes of death comprised convulsions. The term was mainly used in infancy and early childhood, and was next to non-existent in older age categories. In order to establish how convulsions should be treated, we used seasonality patterns and classified convulsions occurring in the first month of life differently from convulsions occurring in the following months of life. Because this cause of death was very age-specific, a more elaborate discussion of the term follows in chapter 3 on infant mortality.

A second cause of death for which it is difficult to decide whether it was infectious or not, is myocarditis. According to present-day medicine myocarditis is an infection of the heart, often as a consequence of a viral infection. In the Netherlands, until the start of the twentieth century, myocarditis was however used as a catch-all phrase for almost every heart condition not associated with heart murmur.[137] In the early twentieth century, Dutch medical diagnostics turned away from the use of the term myocarditis, unless it was related to rheumatic fever or diphtheria.[138] Myocarditis in the nineteenth century was therefore not necessarily an infectious disease, while its use in the early twentieth century was very likely to indicate an infectious disease. In Maastricht, the expression became only popular during the early twentieth century, mainly among the elderly. Improvements in diagnostics may have caused the phrase to have been used more often in Maastricht, whereas other heart conditions may have been used in earlier decades. The lack of myocarditis as a cause of death in nineteenth-century Maastricht suggests

134 Wolleswinkel-van den Bosch, 'The Epidemiological Transition in the Netherlands,' 287-92.

135 See for example: Hallie J. Kintner, 'Classifying Causes of Death During the Late Nineteenth and Early Twentieth Centuries: The Case of German Infant Mortality,' *Historical Methods: A Journal of Quantitative and Interdisciplinary History* 19, no. 2 (1986); Alice Reid, 'Neonatal Mortality and Stillbirths in Early Twentieth Century Derbyshire, England,' *Population Studies* 55, no. 3 (2001).

136 Lebert, *Handboek Der Praktische Geneeskunde*, 4; Pinkhof, *Vertalend en Verklarend Woordenboek van Uitheemsche Geneeskundige Termen*.

137 A. P. M. Verheugt, 'Myocarditis en Infectieziekten,' *Nederlands Tijdschrift voor Geneeskunde* 92, no. 3 (1948).

138 Ibid.

the opposite mechanism which had been observed in other parts of the country, where there was an overuse of myocarditis. Some nineteenth-century heart conditions which are coded as non-infectious diseases may thus actually have been viral heart infections in Maastricht, since the actual viral myocarditis infections may have been diagnosed as non-infectious heart diseases in this period.

Third, dropsy (*waterzucht*, *hydrops*) is a cause of death which remains a mystery to historians. In Maastricht the expression was used very often, although mainly in combination with other causes of death. When it was used in a combination, it was classified according to the other cause of death, since dropsy is a very indistinct symptom. Dropsy was used the most in the early decades of the research period, in only 3% of all deaths in the late 1860s, without any age-specific limitations. The usage of the term waned after the 1880s, with the improvement of diagnostics.

When diagnostics improved during the research period, more puzzling causes of death became less frequently used. Moreover, whereas Van Poppel and Van Dijk stated that doctors often used a vague *febris* instead of *febris typhoidea*, because the latter had to be reported,[139] this practice seems to have been very limited in Maastricht. Unspecified fever or recurring fever have been used in only 45 cases. Although the majority of the fever cases appear in the 1870s, their numbers altogether are small.

2.6 Changing epidemiological patterns

In order to map the disease environment for each age category, cause-specific mortality rates were calculated in each analysis. The population at risk used in calculating the mortality rates was derived from different sources. For infants, we used the number of liveborn infants per year in Maastricht until 1922.[140] These numbers were not yet digitally available for the years thereafter, hence the published statistics of the Central Bureau for Statistics were used to determine the infant population at risk from 1923 onwards.[141] These numbers were available according to gender until 1930. For the period after 1930, the infant population at risk according to gender was estimated based on the mean sex ratio between 1864 and 1930 and the total number of live births in each year after 1930.

The population at risk for other age categories was derived from the population censuses.[142] Based on the numbers from the census years, the population at risk, calculated

139 Van Poppel and Van Dijk, 'The Development of Cause-of-Death Registration in the Netherlands, 1865-1955.'

140 Registers van geboorte (Registers of birth) [unpublished dataset created by the Regionaal Historisch Centrum Limburg], 1796-1922, collection number 12.059, inventory numbers 14-77, Burgerlijke Stand in Limburg: Maastricht, 1796-1942, Regionaal Historisch Centrum Limburg (Centre for the Regional History of Limburg), Maastricht.

141 Centraal Bureau voor de Statistiek, *Gegevens Per Gemeente Betreffende De Loop Der Bevolking in Het Jaar...* (The Hague: Centraal Bureau voor de Statistiek, 1942-1955); Centraal Bureau voor de Statistiek, *Statistiek Van Den Loop Der Bevolking Van Nederland Over ...* (The Hague: Rijksuitgeverij, 1923-1942).

142 Centraal Bureau voor de Statistiek, *Statistiek Van Den Loop Der Bevolking Van Nederland Over...* (The Hague: Van Weelden & Mingelen, 1900-1947); Centraal Bureau voor de Statistiek, *Volkstelling 31 December 1920* (The Hague: Belinfante, 1922); Centraal Bureau voor de Statistiek, *Volkstelling 31 December 1930* (The Hague: Algemeene Landsdrukkerij, 1932-1934); Centraal Bureau voor de Statistiek, *Statistiek Van De Loop Der Bevolking Van Nederland*

as the mid-year population, was estimated for the years in between. The estimates of the population at risk based on the 1920 census were less reliable, since the census was held on the 31st of December while Maastricht had annexed some neighbouring municipalities. The population increase was therefore much larger. Estimates for the population at the start of 1920 were made based upon the population before the annexation. Thus, for each age category it was possible to calculate the population at risk and the cause-specific mortality rates, while it was not possible to stratify the cause-specific mortality rates according to socioeconomic status. Other methods were therefore used to analyse the changing epidemiological patterns according to socioeconomic status.

In chapter 3 concerning infant mortality and in chapter 5 concerning adult mortality, multinomial logistic regression analyses served as the method to map and analyse the changing epidemiological patterns according to socioeconomic status and gender (when applicable). Multinomial logistic regression analyses are a suitable method when a population at risk is not available. As such, multinomial logistic regression analyses have been used occasionally in historical mortality studies and more frequently in historical marriage studies.[143] Because the changes in epidemiological patters are of prime interest here, the cause-of-death category functions as the dependent variable in this study. The multinomial logistic regression then compares the chances of having died from a specific disease category instead of another to those same chances for a different socioeconomic group. The result is an odds ratio which indicates, for example, whether among all the deceased an upper class individual had lower chances of having died of an infectious disease instead of a non-infectious disease compared to someone from the working classes.

In chapter 4 on early childhood mortality, the multinomial logistic regression analyses were less suitable for two reasons. The first pragmatic reason is that within this age category from 1-4, the lower mortality numbers did not allow for a complex regression analysis. Mortality was still relatively high in this age category, yet much lower

1946-1967 (The Hague: Staatsuitgeverij, 1970); Ministerie van Binnenlandse Zaken, *Uitkomsten der Vierde Tienjarige Volkstelling in het Koninkrijk Der Nederlanden op den Een en Dertigsten December 1859* (The Hague: Van Weelden & Mingelen, 1863); Ministerie van Binnenlandse Zaken, *Uitkomsten der Vijfde Tienjarige Volkstelling in het Koninkrijk Der Nederlanden op den Eersten December 1869* (The Hague: Van Weelden & Mingelen, 1873); Ministerie van Binnenlandse Zaken, *Uitkomsten der Zesde Tienjarige Volkstelling in het Koninkrijk Der Nederlanden op den Een en Dertigsten December 1879* (The Hague: Van Weelden & Mingelen, 1881); Ministerie van Binnenlandse Zaken, *Uitkomsten der Zevende Tienjarige Volkstelling in het Koninkrijk Der Nederlanden op den Een en Dertigsten December 1889 (Met Uitzondering van de Beroepstelling)* (The Hague: Van Weelden & Mingelen, 1898); Ministerie van Binnenlandse Zaken, *Uitkomsten der Achtste Tienjarige Volkstelling in het Koninkrijk Der Nederlanden op den Een en Dertigsten December 1899 (Met Uitzondering van de Beroepstelling en Woningstatistiek)* (The Hague: Belinfante, 1901); Ministerie van Binnenlandse Zaken, *Uitkomsten der Negende Tienjarige Volkstelling in het Koninkrijk Der* Nederlanden op den Een en Dertigsten December 1909 (The Hague: Belinfante, 1910); Afdeling Algemene Tellingen Centraal Bureau voor de Statistiek, *Uitkomsten van de Volks- en Beroepentelling, 31 Mei 1947, Gemeente Maastricht* (The Hague: Centraal Bureau voor de Statistiek, 1948).

143 Hilde Bras, Frans Van Poppel, and Kees Mandemakers, 'Relatives as Spouses. Preferences and Opportunities for Kin Marriage in a Western Society,' *American Journal of Human Biology* 21 (2009); Kristina Thompson, Björn Quanjer, and Mayra Murkens, 'Grow Fast, Die Young? The Causes and Consequences of Adult Height and Prolonged Growth in Nineteenth Century Maastricht,' *Social Science & Medicine* 266 (2020); Bart van de Putte, 'The Influence of Modern City Life on Marriage in Ghent at the Turn of the Twentieth Century: Cultural Struggle and Social Differentiation in Demographic Behavior,' *Journal of Family History* 32, no. 4 (2007); Renzo Derosas and Cristina Munno, 'The Place to Heal and the Place to Die: Patients and Causes of Death in Nineteenth-Century Venice,' *Social History of Medicine* (2020).

compared to infant mortality, and the lifespan under scrutiny is rather short, creating low numbers. Extending the analysis from childhood until puberty, for example, would distort the analysis too much, since mortality regimes were different between ages 1-4 and 5-10. Second, the mortality regime in itself is quite different. Whereas in infancy or adulthood a clear dominant disease existed, causing the majority of deaths as a consequence of a specific infectious disease, this was less true for young children. The majority of infants were culled by gastroenteritis or diarrhoea, which represent the same aetiology, and the majority of adults until 50 suffered from the dragging consequences of a tuberculosis infection. Yet in early childhood, a whole range of airborne infectious diseases could harm the child. These individual diseases differed in severity, and it was more likely the sum of several infections that finally caused death. A detailed analysis of the changes in the specific disease environment of young children is therefore more interesting, while taking into account gender differences and socioeconomic differences when possible. In the end, changes in the epidemiological regimes will help deepen our understanding of ensuing inequalities in gender and socioeconomic status in the course of the health transition.

CHAPTER 3

3

Modernising Maastricht: Historical background of the city of Maastricht in the nineteenth and early twentieth century

3.1 Introduction

> Indeed, Maastricht is now in *distress*. Thousands of people in Maastricht now inhabit miserable *shacks*. In Maastricht, where the relics of our saints have been laid to rest in old medieval churches, now living skeletons of *sufferers of tuberculosis* roam the slums and alleys. In Maastricht, there is now *hunger*. People who had been working in the factories, indeed often from childhood onwards when they were 9 or 10 years old, now find themselves on the streets with no livelihood, not being able to work anymore.[144] – *Noodkistrede* Dr Henri Poels, 12 March 1917

In 1917, Dr Henri Poels (1868-1948), Limburg's best-known priest, theologian and almoner, gave this impassioned speech on the horrendous circumstances the working classes had to endure in Maastricht. The already detrimental living circumstances had deteriorated during the economic hardship of World War I, leading to even greater distress. One of the major concerns of Dr Poels was the gruesome state of housing in the city, with its epitome being the *Groete Bouw* (Maastricht dialect for Big Building), a housing block that had been built in the 1860s on the initiative of Maastricht's leading industrialist Petrus Laurentius Regout (1801-1878), to accommodate his factory workers. By 1917, it had become outdated and even worse, the majority of its two-room units had been split into single-room units, which still accommodated entire families – thus explaining the nickname Dr Poels gave it: The *Mensenpakhuis* (Human Warehouse) of Maastricht.[145] Dr. Poels proclaimed that something had to be actively done to improve these horrible circumstances, instead of priests only preaching against indecency and alcohol abuse.[146]

Maastricht had grown into an industrialised, overcrowded and unhealthy city by the late nineteenth century.[147] A key indicator of poor health, high infant mortality, confirms the unhealthy Maastricht environment. Experiencing the highest infant mortality rates of all the large Dutch cities in the late 1890s, Maastricht was nationwide at the very bottom of the ranks.[148] This historical context is important if we are to understand the transformation in mortality and health that took place from the late nineteenth into the twentieth century. This chapter outlines the historical background of Maastricht, with an emphasis on the socioeconomic, environmental, demographic and medical developments. The chapter starts describing how Maastricht evolved into an industrial city and what this meant for the majority of its population in terms of living circumstances. Next, the focus shifts to how this evolution affected demographic characteristics such as

144 H.A. Poels, *De 'Noodkist'* (Maastricht: De Propaganda 1917): 1-2. Own translation. Dutch original: Ja, te Maastricht heerscht thans *nood*. Te Maastricht wonen duizenden van menschen in armzalige *krotten*. Te Maastricht, waar in de oude middeneeuwsche kerken, de relikwieën onzer heiligen rusten, wandelen thans, in sloppen en stegen, levende geraamten van *tuberculose-lijders* rond. Te Maastricht wordt thans *honger* geleden. Menschen die vanaf hun prille jeugd, ja, vaak, als kinderen van negen of tien jaren reeds, gewerkt hebben in fabrieken, staan thans, nu zij niet langer werken kunnen, hier broodeloos op straat.

145 Ibid.

146 Ibid., 15-16.

147 Philips, *Gezondheidszorg in Limburg,'* 9-11.

148 Wolleswinkel-van den Bosch, 'The Epidemiological Transition in the Netherlands,' 155.

population growth, increasing life expectancy, fertility and migration. Finally, the state of public health and public health initiatives will be discussed.

3.2 Industrialising Maastricht

To understand the vast industrialisation that occurred in Maastricht, as one of the few industrial cities in the Netherlands, its geographical location needs to be taken into account. Maastricht is located in the far south of the country, on the riverbanks of the Meuse, a stone's throw Belgium, and not much farther from Germany (Figure 3.1). The fact that Maastricht bridged the river Meuse has made it a strategic locality for centuries, hence its long-lived status as a garrison town. The river also made it an important transit point for goods. Until the start of industrialisation in Maastricht, the economy predominantly consisted of crafts and services for the garrison, as well as trade.[149] During the Belgian revolution (1830-1839), the Siege of Maastricht resulted in the city being closed off from its Belgian hinterland, with trade only gradually being restored during the revolt. The Dutch government decided that Belgian raw materials and semi-finished products could be imported only if they were finished in Maastricht.[150] This mandated form of trade became a prime incentive for the industrialisation of the city.

In 1834, Maastricht's most famous (and infamous) industrialist Petrus Regout founded his first factory, eventually becoming an industrial magnate in the city. Others followed suit, and new factories mushroomed during the 1840s and 1850s. The industrial entrepreneurs had the luxurious position that high unemployment brought them cheap labour from within the city.[151] In the first industrial period, business was booming and the factories flourished. Yet after the mid-1870s, the economic climate changed and several small factories were forced to close their doors. The larger ones, for example the ones which were part of the Regout empire, survived, and Regout even expanded by opening new factories.[152] The downturn was burdensome on the working classes, since factories tried to counteract the harsh economic climate by docking employees' wages.[153]

The socioeconomic position for the working class population of Maastricht was precarious throughout the nineteenth century. Wages were so low that many struggled to survive. Between 50% and 60% of households found themselves in the lowest income groups, struggling to make ends meet throughout the nineteenth century. Only after the turn of the century did prosperity increase and the number of people living above the

149 A. J. Fr. Maenen, *Maastrichts Sociaal-Economische Structuur Tijdens de Franse Tijd en het Verenigd Koninkrijk (1795-1830)* (Hasselt: Provinciale Bibliotheek, 1962): 1-11.

150 A. J. Fr. Maenen, *Petrus Regout 1801-1878. Een Bijdrage tot de Sociaal-Economische Geschiedenis van Maastricht* (Nijmegen: N. V. Centrale Drukkerij, 1959): 19.

151 Ad Knotter, 'De 'Sociale Kwestie': Industrie, Arbeid en Arbeidsverhoudingen in de Negentiende en Twintigste Eeuw tot Circa 1940,' in *Limburg: Een Geschiedenis,* ed. P. M. J. E. Tummers (Maastricht: Limburgs Geschied- en Oudheidkundig Genootschap, 2015): 418; Pierre J.H. Ubachs and Ingrid M.H. Evers, *Tweeduizend Jaar Maastricht: Een Stadsgeschiedenis*, 3 ed. (Zutphen: Walburg Pers, 2006): 200-02. Maenen, *Petrus Regout 1801-1878*, 29.

152 Constance van Es, 'Migratie Te Maastricht 1850-1920' (Katholieke Universiteit Nijmegen, 1980), 13-18.

153 Ibid., 29.

subsistence minimum grew substantially.[154] Yet a slightly higher wage would not always have been beneficial either, since most skilled occupations were in the pottery and glass industry, which were very hazardous to health.[155] Many male factory workers, who were the only gender allowed to occupy these jobs, suffered from lung conditions such as silicosis and/or tuberculosis,[156] which was popularly known as the '*pottemennekeskrenkde*' (potters' disease).

Improving one's status by social climbing through higher education was very rare in Maastricht. After the Napoleonic years, King William I (reigning from 1815-1840) aimed to improve the education of his subjects. Although primary education improved during his reign, this was to little avail in Limburg, where few people cared about education.[157] The citizens of Maastricht were no different, as children had to supplement the family income, and the predominantly unskilled labour that was in demand required no particular education. After 1901, when compulsory school attendance was introduced at least until 12, the number of children who received primary education increased.[158] Secondary education after 1901 was however still rare, since children were still needed to support the family financially after primary school.[159]

Besides low wages, occupational hazards and low education, the quality of life was severely affected by the poor living circumstances in the city. The quality of housing was fairly low in the overcrowded city, with many families residing in single-room apartments or shacks. Maastricht had been a garrison town at the start of the industrial period. Whereas this had safeguarded its connection to the Netherlands during the Belgian revolution, it became an obstacle once the city needed to expand to accommodate its slowly but steadily increasing working population. The city could not grow beyond its fortifications and slums started to materialise where they could.[160] By 1867, it was finally decided to abolish the garrison. However, this had limited effect on the growth of housing facilities. Instead, the first newly build neighbourhood in 1887 was a luxurious residential neighbourhood designed to meet the wishes of the wealthy, who the municipality feared would flee the city.[161] Building accommodation for the working classes on the other hand was left to the industrials, at least in the opinion of the city council and mayor Pyls (1819-1903) during the 1860s.[162] Apart from some incidental initiatives in the late nineteenth century, housing really began improving in the early twentieth century. New laws in 1901 should have accommodated the building of enough new dwellings, yet it was only after World War I that building actually increased

154 B.P.A. Gales et al., *Het Burgerlijk Armbestuur: Twee Eeuwen Zorg voor Armen, Zieken en Ouderen te Maastricht 1796-1996, 2 vols* (Maastricht: Stichting Historische Reeks Maastricht, 1997), I, 151.

155 Gert-Jan de Groot, *Fabricage van Verschillen: Mannenwerk, Vrouwenwerk in de Nederlandse Industrie (1850-1940)* (Amsterdam: Aksant, 2001): 275; Maenen, *Petrus Regout 1801-1878*, 380.

156 De Groot, *Fabricage Van Verschillen*, 275.

157 P. J. H. Ubachs, *Handboek voor de Geschiedenis van Limburg* (Hilversum: Verloren, 2000): 435.

158 Ibid., 437.

159 Ubachs and Evers, *Tweeduizend Jaar Maastricht. Een Stadsgeschiedenis*, 242.

160 Ibid., 208.

161 Ibid., 208

162 Ibid., 210.

and housing started to improve.[163] The incorporation of nearby localities into the city (Figure 3.1b) may have contributed to the expansion of housing facilities by creating space beyond the city's old borders.

The city council was not much more proactive when it came to providing clean water. When they grudgingly followed the example of other Dutch cities and installed a piped water supply system, it was again principally to satisfy the needs of the wealthy, who they feared would leave a city they considered filthy.[164] Implementation started in 1888, progressing at a snail's pace, so that by 1922, barely half the Maastricht population had access to piped water.[165] The disposal of excrement became more strictly regulated in 1873, when a system of excrement collection via *fosses mobiles*, similar to the Rochdale pail system, was introduced.[166] The filth and overcrowding of the late nineteenth century, which only concerned the city council a sop to the wealthy, thus only really improved substantially in the first decades of the twentieth century.

3.3 Demographic characteristics of Maastricht

During the nineteenth century, population growth in Maastricht was modest, as it increased from a total population of 25,483 in 1840 to 34,220 in 1900 (Figures 3.2). Despite the growing industry in the city from the 1840s onwards, the population did not yet massively increase. Indeed, the provinces of Limburg and Noord-Brabant were lagging in population growth during the nineteenth century, only to catch up in the twentieth century by becoming the fastest-growing provinces.[167] The Maastricht population started to grow more rapidly from the 1910s onwards. The major increase in the Maastricht population between 1910 and 1921 is partially explained by the annexation of neighbouring municipalities (Figure 3.1b), although Figure 3.2b shows that the increasing population was an overall trend in the province.

According to the demographic transition theory, the massive population growth during the transition was caused by a decline in mortality, followed slightly later by a decline in fertility.[168] During the period between the decline in mortality and the decline in fertility, the population is allowed to grow the most. Yet in the Netherlands, the timing of the start of the decline in mortality and fertility appears to have been more or less simultaneous.[169] How did this transition take shape in Maastricht in particular?

163 E.P.M. Ramakers and F.J. Hermans, *Historische Atlas van Maastricht: 2000 Jaar aan Maas en Jeker* (Maastricht: Regionaal Historisch Centrum Limburg, 2005): 46.

164 C. Cillekens, J. J. F. van den Boogaard, and B. P. A. Gales, *Loop naar de Pomp. Geschiedenis van de Watervoorziening en de Waterleiding in Maastricht* (Maastricht: Stichting Historische Reeks Maastricht, 1988): 39.

165 Ibid., 55.

166 I. M. H. Evers and P. J. H. Ubachs, *Voorkomen is Beter dan Genezen: 75 Jaar Georganiseerde Gezondheidszorg te Maastricht* (Maastricht: Stichting Historische Reeks Maastricht, 1996): 15.

167 Theo Engelen, *Van 2 naar 16 Miljoen Mensen: Demografie van Nederland, 1800-Nu* (Amsterdam: Boom, 2009): 15.

168 Chesnais, *The Demographic Transition,* 1-10.

169 D. J. Noordam, 'Demografische Ontwikkelingen,' in *Van Agrarische Samenleving naar Verzorgingsstaat: Demografie, Economie, Maatschappij en Cultuur in West-Europa, 1450-2000*, ed. B. M. A. De Vries, et al. (Groningen: Martinue Nijhoff, 2000): 205.

A major indicator of declining mortality in general can be found in an increasing life expectancy. As such, an overview of increasing life expectancy in Maastricht provides a first impression of declining mortality in the city.

a. The Netherlands and its eleven provinces (ca. 1920)

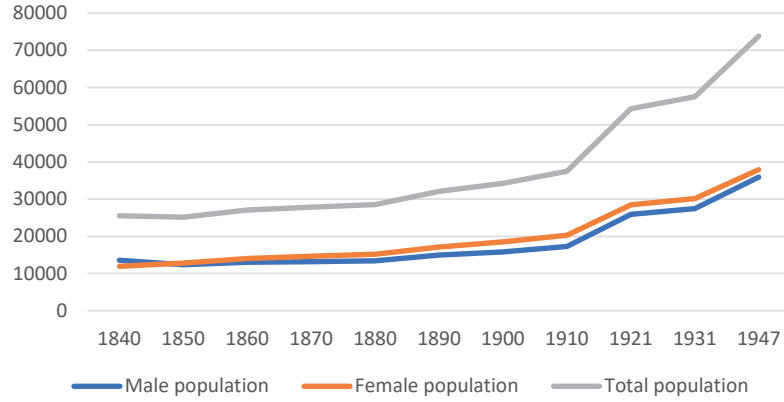
b. Maastricht, before and after the annexation of neighbouring localities in 1920

Figures 3.1a and 3.1b
Source: Thijs van Vugt, Sociaal Historisch Centrum Limburg.

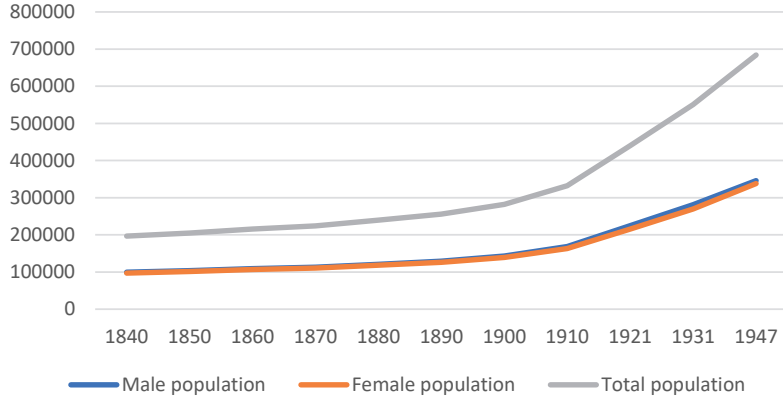
Figure 3.3 shows life expectancy at a certain age for several snapshots in time. Life expectancy was calculated for five-year periods in order to equalise swift changes in life expectancy caused by epidemic outbreaks or an outstanding year with low levels of infectious mortality.[170]

170 The mid-year population used to calculate life expectancy was calculated from the censuses and the estimates of the population for the years between censuses, which in turn were calculated from the surrounding census years. For the age category below 1 the amount of live born was used instead of the census numbers. Populations were slightly adjusted for 1920, which marks the annexation of bordering municipalities. In the case of early childhood, the population estimates in the 1910s were corrected for the rapid infant mortality decline in the period. See chapter xx for more information on the correction for the early childhood population. The unusual circumstances during World War II inhibit the calculation of proper population estimates in the 1940s, hence only the census year 1947 was used to calculate life expectancy in the 1940s.

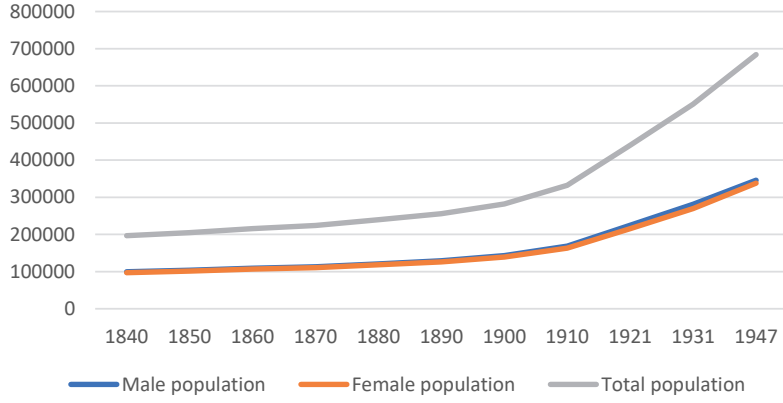
a. Population growth in Maastricht, 1840-1947



b. Population growth in Limburg, 1840-1947



c. Population growth in the Netherlands, 1840-1947



Figures 3.2a, 3.2b and 3.2c
Sources: Gemeente Maastricht, Uitvoerig en Beredeneerd Verslag van den Toestand der Gemeente Maastricht, over het Jaar... (Maastricht: Stedelijk Bestuur van Maastricht, 1852-1874); Gemeente Maastricht, Verslag van den Toestand der Gemeente Maastricht over het Jaar... (Maastricht: Gemeente Maastricht, 1875-1937); Centraal Bureau voor de Statistiek, Gegevens per Gemeente Betreffende de Loop der Bevolking in het Jaar; Centraal Bureau voor de Statistiek, Statistiek Van Den Loop Der Bevolking; Centraal Bureau voor de Statistiek, Bevolking Der Gemeenten Van Nederland Op... (The Hague: Trio, 1920-1955).

Figure 3.3 presents life expectancy at a certain age. As such, it becomes clear that life expectancy at birth was very low until the 1920s. The life expectancy at birth increased for women from 38 in the 1860s to 69 in 1947, and doubled for men from 33 years in the late 1860s to 66 years in 1947. Due to the high infant mortality, many years were gained in life expectancy once infants had survived the first year. The stapled blocks in Figure 3.3 thus represent the years gained in life expectancy once an individual had reached that specific age compared to the previous age category. In general, women had higher life expectancy across the board, at all ages and in all time periods.

The increased life expectancy according to age moreover offers a sneak peek into the age categories contributing most to the longer-lived population of Maastricht. These age categories represent those categories that will be utilised in the analyses in the following chapters. Thus, despite the low life expectancy at birth during the majority of the period, it already slightly increased after the late 1870s, as a consequence of mortality decline among young children (1-4), which will be discussed in chapter 5. In the following decades, life expectancy at birth stagnated until the late 1910s, when infant mortality declined rapidly (chapter 4). Life expectancy at adult ages remained stable throughout the period, until the late 1920s. The 1920s also saw life expectancy at age 20-29 increase, thus adult mortality will be the subject of chapter 6. Moreover, towards the mid-twentieth century, the years added after survival in the previous age category become less, indicating that a larger share of the population survived into old age.

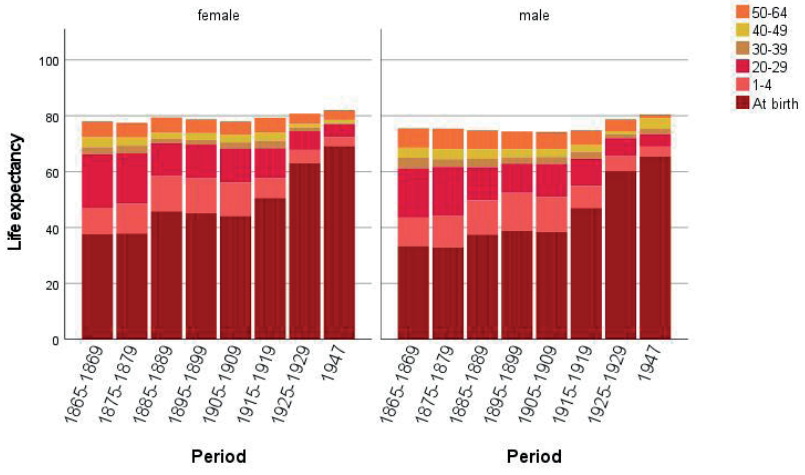


Figure 3.3 Mean life expectancy at age x for women and men, Maastricht 1865-1947
Source: Maastricht Death and Disease Database; Dutch censuses

3.4 Fertility

The other main element of the demographic transition, fertility, is also important in understanding the demographic dynamics of Maastricht and the population growth in the early twentieth century. Due to its almost homogenous Catholic population, the stereotypical Limburg population of the nineteenth century is believed to be highly fertile, with large families consisting of many children. Yet crude birth rates were actually relatively low compared to other Dutch provinces until 1880.[171] Two main fertility measurements can improve this stereotypical understanding of fertility in Maastricht in the nineteenth and early twentieth century. Firstly, the crude birth rates for Maastricht can be calculated. Since crude birth rates do not account for population structures, a second estimation of fertility in Maastricht can be found in the marital fertility rates.

Figure 3.4 Crude birth rate Maastricht, 1860-1955

Source: Statistiek, "Bevolking Der Gemeenten Van Nederland Op..."; Maastricht, "Uitvoerig En Beredeneerd Verslag Van Den Toestand Der Gemeente Maastricht, over Het Jaar..."; "Verslag Van Den Toestand Der Gemeente Maastricht over Het Jaar...". Database RHCL

Crude birth rates in the Netherlands as a whole until 1880 were around or slightly above 35 births per 1,000 persons, while this was lower in Limburg with crude birth rates around 30 births, or at least clearly below 35 births per 1,000 persons.[172] From 1890, the birth rate in Limburg increased substantially, while in the majority of Dutch fertility started to decline.[173] Two main factors can explain the regional differences in crude birth rates, and thus the lower crude birth rates in Limburg. First, Hofstee proposed that the lower birth rates in Limburg were a consequence of an old agrarian-traditional fertility pattern, in which marriage only occurred when the couple had enough resources to

171 Engelen, *Van 2 naar 16 Miljoen Mensen*, 61.

172 Ibid.

173 Ibid., 64-66.

support a family. This resulted in a large proportion of unmarried individuals, which is a natural check on fertility.[174] In the western part of the country, the labour market changed during the nineteenth century and became more volatile, to such an extent that security gained from dependency on the employer dissolved. Without the prospect of any future resource security, a proletarian phase began in which couples tended to marry earlier. As a consequence, crude birth rates increased as a result from first fewer unmarried individuals and second people being married for a longer period during their fertile years.[175] Another explanation for lower birth rates at the end of the nineteenth century might be found in higher levels of breastfeeding, which provokes a biological mechanism of delaying subsequent pregnancies.[176] However, prolonged breastfeeding in Maastricht is not likely the cause for lower fertility, since the assumption is that breastfeeding levels in Maastricht, and in Limburg, were quite low towards the end of the nineteenth century.[177]

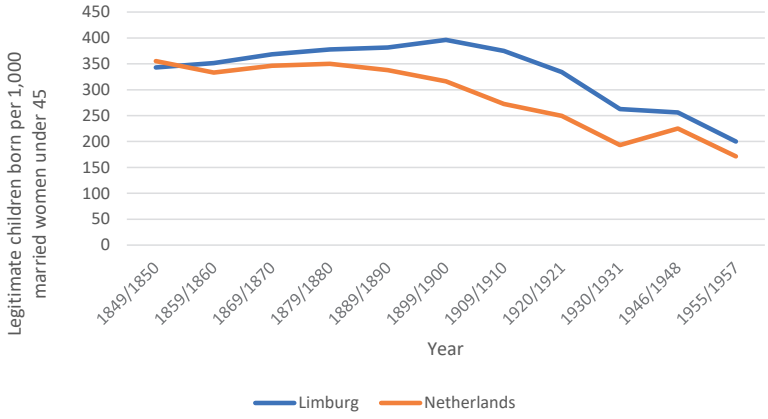


Figure 3.5 Marital fertility in Limburg and the Netherlands, 1849-1957

Source: Centraal Bureau voor de Statistiek, "Regionale Huwelijksvruchtbaarheid. Provinciale Cijfers, 1849/1850-1979," (The Hague1981).

Maastricht was however an outlier in the mainly rural Limburg, with crude birth rates closer to the Dutch average of 35 births until 1880 (Figure 3.4). Perhaps the explanation of Hofstee applies, and as the city industrialised, a proletarian phase began in which couples married earlier. The decline in crude birth rates started later in Maastricht compared to the Netherlands as a whole, from the late 1900s onwards and

174 Ibid., 62; Hofstee, *Korte Demografische Geschiedenis van Nederland*, 44-63.

175 Hofstee, *Korte Demografische Geschiedenis van Nederland*, 40-43.

176 Engelen, *Van 2 naar 16 Miljoen Mensen*, 62-63; Chris Vandenbroeke, Frans van Poppel, and Ad van der Woude, 'De Zuigelingen- en Kindersterfte in België en Nederland in Seculier Perspectief,' *Tijdschrift voor Geschiedenis* 94 (1981): 490-91.

177 Vandenbroeke, Van Poppel, and Van der Woude, 'De Zuigelingen- en Kindersterfte in België en Nederland in Seculier Perspectief,' 490-91; Frans van Poppel, 'Religion and Health: Catholicism and Regional Mortality Differences in Nineteenth-Century Netherlands,' *Social History of Medicine* 5, no. 2 (1992).

only continuing for a relatively short period until the late 1910s. After a brief increase during the 1920s, which may be related to the annexation of bordering municipalities or perhaps a brief baby boom after World War I, crude birth rates declined more slowly until the mid-1930s.

Although the crude birth rates may have been lower in Limburg compared to the rest of the Netherlands, the marital fertility in Limburg rate was consistently higher from 1859/60 until 1955/57 (Figure 3.5). This confirms the stereotype of Limburg families consisting of many children. While in rural Limburg there was a large proportion of unmarried individuals, once they married the couples did have large families. The largest differences in marital fertility developed from 1880 onwards, when indeed marital fertility in the Netherlands as a whole declined, opposed to an actual increase in Limburg. Only after 1900 did marital fertility in the province of Limburg start to decline. The marital fertility rates of Limburg and the Netherlands converged after the 1930s. In Maastricht an initial decline in marital fertility already showed from 1889 to 1910, although the largest decline in marital fertility occurred thereafter, from 1910 to 1930.[178] The slower decline in both crude birth rates and marital fertility may be the consequence of a slower introduction of birth control. The firm grip of the Catholic Church may have withheld people from regulating their family sizes for a longer period compared to the western part of the country.

It becomes clear that in Maastricht the increase of life expectancy (and thus the decline in mortality) also occurred more or less simultaneously with the decrease in fertility, during the early twentieth century. Yet the slower decline in fertility in Limburg compared to the rest of the Netherlands may explain the vaster population growth in the early twentieth century in Limburg, and in Maastricht in particular.

3.5 Migration

Maastricht's population growth may also have depended on the factor of migration. Among the main characteristics of the era of industrialisation is the high mobility of people, especially rural-to-urban migration. Not only the pull of the industrial city was responsible for this type of migration, it was also the dissolving of rural industries who could not keep pace with the urban industries that pushed labourers towards the city.[179] Migration was not for all, however, with families with children less likely to migrate and migrants being generally younger since migration required physical strength.[180] Moreover, nowadays migrants are often healthier compared to native-born inhabitants, known as the healthy migrant effect. In historical populations the healthy migrant effect has been observed as well.[181] Alter and Oris distinguished three processes which led to

178 Theo L. M. Engelen, *Fertiliteit, Arbeid, Mentaliteit: De Vruchtbaarheidsdaling in Nederlands-Limburg 1850-1960* (Assen, Maastricht: Van Gorcum, 1987): 234-35.

179 Leslie Page Moch, *Moving Europeans: Migration in Western Europe since 1650*, 2nd ed. (Bloomington: Indiana University Press, 2003): 4.

180 George Alter and Michel Oris, 'Childhood Conditions, Migration and Mortality: Migrants and Natives in 19th Century Cities,' *Social Biology* 52, no. 3-4 (2005).

181 Ibid.; Lionel Kesztenbaum and Jean-Laurent Rosenthal, 'The Health Cost of Living in a City: The Case of France

distinct differences in health between migrants and natives. A healthy migrant effect was first the result of the self-selection of only the healthiest people for migration. Second, in the predominantly rural-to-urban migration, migrants had not been exposed to as many infectious diseases in childhood compared to urban dwellers. This provided the migrants with a health advantage in older age. However, this could also lead to the third process Alter and Oris identified, in which migrants were more disadvantaged in epidemic years due to their lower exposure in younger ages.[182]

Maastricht can be expected to have had a particular attractive power for its rural hinterland, as the growing industries were in need of cheap labour. Yet Maastricht was not characterised by a large settlement surplus, nor by a large migration deficit. Instead, Maastricht is to be considered as a transfer locality, where relatively low numbers of migrants came through and of which the majority stayed for only a year. The pull factors of the city consisted mainly of the hope of employment in its garrison in the early nineteenth century and, later on, in its industry. Yet after roughly a year, the lack of housing facilities and the lack of the materialisation of the imagined better life pushed these migrants outside the city walls again.[183] Moreover, immigration turned out to not be essential for the industry to expand, as Maastricht had enough manpower of its own to fill the factories.[184] Indeed, the proportion of citizens who had been born across the borders – Belgium was closer to Maastricht compared to many rural localities in Dutch Limburg – appears to have declined in the second half of the nineteenth century. Between 1859 and 1889, the amount of Maastricht citizens who had been born in Belgium declined by more than 50%.[185] A similar pattern can be found in the origin of spouses in the nineteenth century, an increasing proportion being of Maastricht origin towards the late nineteenth century, which reinforces the idea that migration was declining throughout the century.[186]

Figure 3.6 shows the immigration, emigration and net migration for each year in Maastricht from 1851 until 1935, based on the reports of the municipality. A small increase in absolute numbers of people migrating can be found, however the net migration stays close to zero. The peak in 1920 should be considered an artificial effect of the annexation of the bordering municipalities, where the municipality apparently regarded the new citizens as migrants. Net migration throughout the period was very small, which affirms the characterisation of Maastricht as a transfer city for relatively few migrants. Although migration both into and out of the city increases slightly over time, there are no major increases or decreases. Clear effects of the agricultural crisis of the 1880s, for example, cannot be found in Maastricht's migration statistics. This affirms

at the End of the 19th Century,' *Explorations in Economic History* 48, no. 2 (2010).

182 Alter and Oris, 'Childhood Conditions, Migration and Mortality.'

183 Van Es, 'Migratie te Maastricht 1850-1920,' 147.

184 Knotter, 'De 'Sociale Kwestie': Industrie, Arbeid en Arbeidsverhoudingen in De Negentiende En Twintigste Eeuw Tot Circa 1940,' 422.

185 Willibrord J.M.J. Rutten, 'Huwelijk en Partnerkeuze in een Grensstad (Circa 1830-1910): Verschuivingen in de Geografische Herkomst van Huwelijkspartners te Maastricht vóór en ná de Scheiding van de Beide Limburgen,' in *Eenheid en Scheiding van de Beide Limburgen,* ed. Maaslandse Monografieën (Leeuwarden, Maastricht: Eisma B.V., 1989): 102.

186 Ibid., 104-05.

the idea that Limburg farmers, even though affected by the crisis, did not massively seek refuge in their industrial provincial capital.

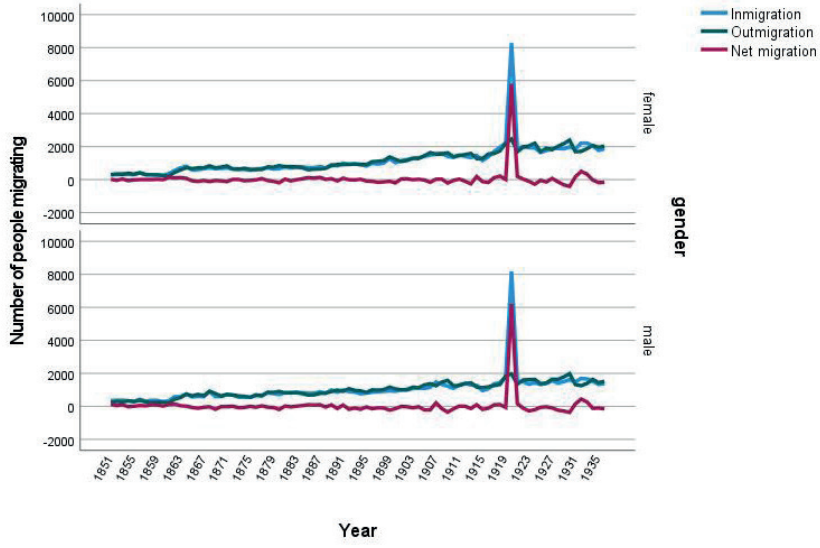


Figure 3.6 Migration by gender, Maastricht 1851-1936

Source: Gemeente Maastricht, Uitvoerig en Beredeneerd *Verslag van den Toestand der Gemeente Maastricht*; Gemeente Maastricht, *Verslag van den Toestand der Gemeente over het Jaar*.

3.6 Medical institutions

Since this dissertation focuses on differences in mortality, a brief discussion of the efforts of healthcare facilities to prevent an early death in its broadest sense is indispensable. Since institutional healthcare and medicine were not as prominent in the nineteenth century as in today's society, nor at the timing of the epidemiological transition, I will concentrate more on the development of public health ideas, and how these transposed to the wider population. In the nineteenth-century Netherlands, medicine and public health belonged predominantly to the realm of private initiatives, with the most noticeable example being the hygienists (*hygiënisten*). From the mid-century onwards, this group of zealous physicians aimed at improving public health, effectuating sanitary reforms and objectifying medicine.[187] Despite their private beginnings, they also aimed at institutionalising public health regulations.

Some first initiatives in regulating public health at an official level had already been instigated by the state from the start of the century onwards. During the Napoleonic period (1794-1814), major changes had occurred and national regulations had become

187 Houwaart, *De Hygiënisten*, 1.

centralised to a much larger extent. As such, the state started to interfere with health in the form of medical police, mainly focusing on the surveillance of the population and of medical practice in the nation.[188] After the defeat of Napoleon and the restoration of the House of Orange, these new medical regulations were not abandoned. As a result, provincial committees tasked with the supervision of medical practice were officially founded in 1818 (Provincial Committees of Medical Research and Supervision/ *Provinciale Commissies van Geneeskundig Onderzoek en Toevoorzicht*), which was a reinforcement of the medical laws that had been issued in the previous years. Local committees were established for cities with at least four practising physicians as well.[189] These committees were primarily preoccupied with the supervision of the licensed and unlicensed medical practitioners in their jurisdiction. They were tasked with overseeing the medical exams, and complaints about the practitioners could be addressed to the committee. The other responsibilities of the committees were compiling overviews of the number of given smallpox vaccines per doctor and providing yearly overviews of the epidemiological state of the province to the state. Finally, in the case of an epidemic outbreak, they were asked to formulate preventive and relief measures.[190]

The effectiveness of the committees was however highly dependent on the good will of local or provincial governments, since the municipalities were responsible for dealing with health issues at the local level. In some parts of the country, the authorities did not follow the advice given by the committee, which left the committees emptyhanded.[191] The endeavours of the private hygienists did not fall on deaf ears, however. New medical laws were issued in 1865, which on one hand meant the end for the committees on medical research and supervision, but on the other hand the start of the Health Inspectorate (*Geneeskundig Staatstoezicht*). The Health Inspectorate was an official body consisting of seven inspectors who had to report directly to the minister. The inspectors were assigned one or two provinces for which they had to report yearly on the state of health, they had an advisory role on public health to local governments and they were obliged with ensuring local compliance with laws and verdicts related to health.[192] The role of the Health Inspectorate was thus mainly advisory, although at a state level they did accomplish the issuing of the Epidemic Diseases Act. With this law, it became mandatory to report infectious diseases and to enact quarantine measures. Nonetheless, municipalities were often still reluctant in taking the inspectors' advice to heart; instead, they mostly viewed the inspectors as an annoyance.[193]

In 1902, the regulations changed again with the issuing of the Health Act (*Gezondheidswet*), which mandated the official organisation of the Health Council and its local health committees.[194] Whereas the Health Inspectorate operated officially only

188 Rutten, 'An Outline of Socio-Medical Care in the Netherlands,' 154.

189 Ibid., 154-55.

190 Ibid., 155. J. K. van der Korst, *Om Lijf en Leven: Gezondheidszorg en Geneeskunst in Nederland Circa 1200-1960* (Utrecht, Antwerpen: Bohn, Scheltema & Holkema, 1988).

191 Rutten, 'An Outline of Socio-Medical Care in the Netherlands,' 155.

192 Houwaart, *De Hygiënisten*, 177; Rigter, *Met Raad en Daad*, 19.

193 Rigter, *Met Raad en Daad*, 20.

194 Ibid.

via its inspectors, the Health Council installed local health committees, who had to report to the health inspectors. The health inspectors now functioned as an intermediary between the Minister of Internal Affairs and the local committees. The committees still had a predominantly advisory role, yet the background of its members was more diverse than the background of the Provincial Committees of Medical Research and Supervision had been in the early nineteenth century. Not only physicians joined the committees, but also engineers, architects, apothecaries, politicians and industrialists attempted to improve living circumstances in their municipalities. The cooperation between the advisory committees and the local governments also improved, by which the endeavour to improve health became a joint project.[195]

Yet the official bodies were not the only, nor the most important, players in the field of improving health. Due to the merely advisory role of the official bodies, and the often hesitant or even reluctant attitude of local governments, citizens took matters into their own hands.[196] An increasing number of private initiatives and organisations with the aim of bettering health and living circumstances was founded during the final decades of the nineteenth century, and they were truly skyrocketing during the early twentieth century.[197] The most well-known of these initiatives were the Cross organisations, the first being the White Cross (*Witte Kruis*), founded in 1875 in the northern province of Noord-Holland, which aimed at fighting contagious diseases.[198]

Maastricht did not diverge greatly from the national tendencies, since the city council remained quite reluctant to act upon health improvements in the late nineteenth century. The city's poor relief organisation *Burgerlijk Armbestuur* was tasked with the medical care for those who were not able to afford medical care, but the poor relief was hesitant in granting their services since that would only cause the poor to rely even more on poor relief.[199] This led to private initiatives emerging during the late nineteenth century. After the Health Inspectorate Act of 1865, the Provincial Committees of Medical Research and Supervision ceased to exist. Their task of reporting to and advising the city council was taken over by the initiative of the newly founded Health Committee in 1873. As a member of the city council, the physician J.H.J. Schreinemacher (1820-1892) believed such a committee could support the Health Inspectorate in overseeing health issues in Limburg.[200] The Committee was among one of the first of its kind in the Netherlands, dedicated to the task of advising the city council and seeing to the compliance of the city council to verdicts concerning public hygiene.[201] Yet the city council consulted the Health Committee only occasionally, leading to frustration within the Committee. The foundation of another public health organisation was fostered by this frustration, in addition to different ideas on the range of topics such an organisation should preoccupy itself with. In 1881, the Maastricht physician Fouquet (1822-1893) founded the

195 Ibid., 30-38.

196 Ibid., 20.

197 Ibid.

198 Ibid.

199 Gales et al., *Het Burgerlijk Armbestuur*.

200 Evers and, *Voorkomen is Beter dan Genezen,* 10.

201 Ibid., 12.

Association for the Advancement of Public Health (*Vereeniging ter Bevordering der Volksgezondheid*). This rival organisation primarily focused on the high child mortality in the city, but also set its sight on other hygienic grievances in the city, such as the quality of foodstuffs and drinking water, the state of the sewage system and the quality of housing.[202]

Based on the extremely high childhood mortality in the city, the Association for the Advancement of Public Health concluded that the health condition was deplorable. The fact that the Health Committee disagreed on this topic, suggests an initial rivalry between the organisations, despite their common goal. The Health Committee explicitly stated that the exceptionally high child mortality was solely the consequence of the outsourcing of the care of their children by working mothers, while the absence of equally high mortality in other age groups was actually an affirmation of a relatively good health condition in the city.[203] Yet in later years, the two organisations appear to have been more collaborative, and the city council sometimes confused the two.[204] Despite their relatively small contribution to actual improvements in health, the two organisations reflect and enhanced the awareness of the importance of hygiene among the higher social classes.[205]

Only in the twentieth century, did organisations with actual vigour emerge. In 1908, *Pro Infantibus* was established, which focused on providing mothers with good-quality milk for infants. Two years later, the Maastricht chapter of the Catholic *Green Cross* was founded, which aimed at improving the health of the Maastricht population with the help of home-visiting nurses. In addition to their preoccupation with infant care, they dedicated themselves to the fight against tuberculosis. These two organisations will receive more attention in the chapter on infant mortality. Other Cross organisations were established in the 1910s as well, but for our purposes here it suffices to say that private organisations preoccupied with public health and with more than just an advisory role flourished in early twentieth-century Maastricht.[206] The year 1920 saw the predominantly private nature of public health change when the city council decided to establish the *Gemeentelijke Geneeskundige Dienst* (Municipal Health Service). They assumed the tasks from many other smaller organisations, such as advising the city council on public hygiene, providing medical care (apart from clinical medical care) and the control of contagious diseases.[207]

Apart from public health, where prevention was the primary concern, institutional healthcare was facilitated by the Civil Poor Relief (*Burgerlijk Armbestuur*). Not only did they facilitate medical care for the poor, they also welcomed paying patients or patients joining a sickness fund into their hospital Calvariënberg, founded in the 1820s. During the nineteenth century, Dutch institutional medical care mainly aimed at retaining the

202 Ibid., 13-14.

203 Report health committee (1881), pages 5-7, collection number 20.150, inventory number 9, Plaatselijke Gezondheidscommissie Maastricht 1873-1902, Regionaal Historisch Centrum Limburg, Maastricht.

204 Evers and Ubachs, *Voorkomen Is Beter Dan Genezen*, 15.

205 Ibid., 16.

206 Ibid., 17-25.

207 Ibid., 31.

physique required to participate in the labour force, specifically for the poor. As a result, the costs of poor relief would be contained.[208] It is therefore no surprise that the first and only hospital in Maastricht was managed by the city's poor relief organisation. The hospital could not count on much appreciation from the population until the 1890s; however, after impressive reorganisation, the hospital became one of the frontrunners in the medical field in the early twentieth century.[209] Moreover, it became a hospital for the entire population during the first decades of the twentieth century as its emphasis on primarily aiding the poor slowly faded.[210]

Thus, whereas Maastricht was an overcrowded, unhealthy city towards the end of the nineteenth century, several improvements materialised during the early twentieth century. Increasing wages brought the majority of the population on the safe side of the subsistence minimum, a piped water supply system slowly expanded and housing conditions improved after the first few decades. Hard-working public health organisations complemented the improvements in living circumstances. These improvements are likely to have been correlated to the demographic changes in the early twentieth century; the population started to grow more rapidly as life expectancy rose and total fertility slowly declined. What was at the root of these developments will be discussed in the following chapters, in which the analysis of causes of death according to different age categories and socioeconomic status will help in gaining a more informed understanding of the health transition in Maastricht.

208 Gales et al., *Het Burgerlijk Armbestuur*, 51-54.

209 Ubachs and Evers, *Tweeduizend Jaar Maastricht*, 212-14.

210 Gales et al., *Het Burgerlijk Armbestuur*, 68.

CHAPTER 4

Transitory inequalities: How individual-level, cause-specific death data can unravel socioeconomic inequalities in infant mortality in Maastricht, the Netherlands, 1864-1955[1]

1 A slightly abbreviated and adapted version of this chapter has been published in the History of the Family and is co-authored by Ben Pelzer and Angelique Janssens.

4.1 Introduction

The decline in infant mortality played a crucial role in the health transition in the Western World. Total mortality rates were highly affected by the deplorably high infant mortality rates (IMR). In a similar fashion, life expectancy at birth was strikingly low due to the culling of many infants in the earliest stages of life. In fact, the decline in infant mortality was responsible for 44% of the increase in life expectancy since 1875 for Dutch men and for 37% of the increase in life expectancy for Dutch women.[211] The health transition was, however, not an evenly dispersed process, with differences across regions, socioeconomic status, cultural factors such as religion, or gender shaping different paths towards a low mortality regime. In the Netherlands, a clear divergence started to appear from the 1860s onwards. While IMR dropped in the north-western provinces, the south-eastern provinces were in fact confronted with an increase in IMR.[212] Local private initiatives, with the aim of improving population health, eventually recognised this time lag in the southern, industrialising city of Maastricht. In their words, the high infant mortality rates casted a shadow over the city, as these caused Maastricht to end up 'in the darkest pages of the reports concerning the health conditions in our country'.[213] In explaining the Dutch regional diverging trends, much of the debate revolves around the role of religion versus region.[214]

The role of socioeconomic status in creating differences should nonetheless not be overlooked, despite the effect of socioeconomic status on mortality differences remaining disputed both in the Netherlands and in a wider Western context.[215] One of the most commonly used theoretical frameworks for determinants affecting infant mortality indeed departs from the idea that socioeconomic status is the fundamental

211 Frans van Poppel, *De 'Statistieke Ontleding Van De Dooden': Een Spraakzame Bron?* (Nijmegen: Uitgeverij KU Nijmegen, 1999): 9.

212 Ekamper and van Poppel, 'Zuigelingensterfte Per Gemeente in Nederland, 1841-1939.'; Hofstee, *Korte Demografische Geschiedenis*.

213 Jaarverslag, (1913), collection number EAN_0930, inventory number 1, (RK Vereniging) Het Limburgse Groene Kruis afdeling Maastricht, Sociaal Historisch Centrum Limburg, Maastricht. Translation of the author. Dutch original: 'Behalve het boven omschreven doel, beoogt de afdeling eveneens de groote zuigelingen sterfte tegen te gaan, waardoor Maastricht met zwarte kool in de verslagen omtrent gezondheidstoestanden in ons land, staat geteekend'.

214 Hofstee, *Korte Demografische Geschiedenis*; Angélique Janssens and Ben Pelzer, 'Lovely Little Angels in Heaven? The Influence of Religiously Determined Cultural Life Scripts on Infant Survival in the Netherlands, 1880-1920,' *Historical Social Research* 39, no. 1 (2014); Nynke van den Boomen and Peter Ekamper, 'Denied Their 'Natural Nourishment': Religion, Causes of Death and Infant Mortality in the Netherlands, 1875-1899,' *The History of the Family* 20, no. 3 (2015); Van Poppel, 'Religion and Health'; Walhout, 'An Infants' Graveyard?'; Wolleswinkel-van den Bosch, 'The Epidemiological Transition in the Netherlands.'

215 Antonovsky, *Social Class, Life Expectancy and Overall Mortality*; Bengtsson and Van Poppel, 'Socioeconomic Inequalities in Death from Past to Present,'; Joseph Molitoris, 'Disparities in Death: Inequality in Cause-Specific Infant and Child Mortality in Stockholm, 1878–1926,' *Demographic Research* 36, no. 15 (2017); Alice Reid, 'Locality or Class? Spatial and Social Differentials in Infant and Child Mortality in England and Wales, 1895-1911,' in *The Decline of Infant and Child Mortality: The European Experience, 1750-1990,* ed. Carlo A. Corsini and Pier Paolo Viazzo (The Hague: Nijhoff, 1997); Frans van Poppel, Marianne Jonker, and Kees Mandemakers, 'Differential Infant and Child Mortality in Three Dutch Regions, 1812-1909,' *The Economic History Review* 58, no. 2 (2005); Woods, Watterson, and Woodward, 'The Causes of Rapid Infant Mortality Decline in England and Wales, 1861–1921 Part I.'

factor influencing direct health determinants.[216] The assumption that socioeconomic status has not been of major importance in differential historical infant mortality may have been driven by two complicating factors for asserting the role of socioeconomic status. First, the focus on very narrow time periods due to data constraints emphasises only the very stationary effect of socioeconomic status on the *level* of infant mortality. A longitudinal approach is essential in order to establish the influence of socioeconomic status on the actual *process* of decline, since the effects of socioeconomic status vary during mortality decline of specific diseases.[217]

Second, many studies are forced to work with all-cause mortality or already grouped mortality data, while Clouston et al. highlight the importance of a cause-specific approach.[218] The impact of socioeconomic status is mainly expressed at a cause-specific level, differing per disease category. Thus, in all-cause mortality analyses, the effects of socioeconomic status can remain hidden. Unfortunately, historical cause-specific mortality data that is longitudinal and at an individual level, specifying socioeconomic status, are very rare.

With this study, we have the rare opportunity to take into account individual causes of death over a long time period for the Dutch city of Maastricht. The unique dataset, containing individual-level data on cause of death, socioeconomic status and age at death of the entire Maastricht population from 1864 through 1955, can contribute to unravelling the intricate process of infant mortality decline. We therefore ask which factors influenced the eventual decline in infant mortality in the industrial city of Maastricht from 1864 until 1955 and how socioeconomic status influenced this decline, by possibly bringing about temporary or permanent inequalities. We aim to map changing epidemiological patterns over time according to socioeconomic status by deploying a multinomial logistic regression analysis. The cause-specific mortality data provide an approximation of the influence of specific direct determinants and the influence of socioeconomic status. Combined with a qualitative analysis of archival material to establish the particular historical context, the analyses provide a well-grounded understanding of the heterogenous process of infant mortality decline in this industrial city.

4.2 Determinants of infant mortality decline in a Western context

In the beginning of the twentieth century, infant mortality and its decline became a priority on national agendas. As state officials and private organisations became more aware of the deplorable state of the health of the national population through emerging statistical research and conscription records, the urgency to improve health and thus foster a strong prospective nation state increased.[219] In some regions, however, the secular

216 W. H. Mosley and L. C. Chen, 'An Analytical Framework for the Study of Child Survival in Developing Countries,' *Population and Development Review* 10 (1984).

217 Clouston et al., 'A Social History of Disease.'

218 Ibid.

219 Eilidh Garrett et al., 'Introduction: Infant Mortality; a Social Problem?,' in *Infant Mortality: A Continuing Social Problem*, ed. Eilidh Garret et al. (Aldershot: Ashgate, 2006); H. Marland and M. Gijswijt-Hofstra ed., *Cultures of*

decline in infant mortality had already started before this realisation. Woods et al. distinguish three European infant mortality decline patterns: A French, Swedish and British pattern. In France, the decline started in 1890, with a temporary reverse in the late 1890s.[220] The Swedish decline started earlier, in 1881, although with another peak in IMR in 1900. In general, the Netherlands adhered to this second pattern. Great Britain experienced a rise in infant mortality rates during the final two decades of the nineteenth century prior to a sudden decline in 1900. Despite regional variation in exact timing and levels, infant mortality started to decline around the turn of the century in most Western European countries.[221]

The secular decline in IMR was certainly not a mono-causal phenomenon, since mortality levels itself were influenced by a variety of possible determinants, as was its decline. Nevertheless, according to the fundamental causes theory, the differing determinants of mortality are all an expression of differences in socioeconomic status. The fundamental health discriminator is socioeconomic status, although its influence can also differ among different diseases.[222] Focusing solely on all-cause mortality or on a limited and static time period could therefore obscure differences underlying overall mortality or differences occurring over longer time periods. As already mentioned in the introduction, Clouston et al. add an explanation of the mechanisms of the fundamental causes theory: Different diseases progress through four different stages of mortality decline (*natural mortality, producing inequality, reducing inequality* and *reduced mortality*), in which the impact of socioeconomic status on mortality differs per stage.[223]

The influence of socioeconomic status on infant mortality in particular has been widely studied, but its importance is still contested. Antonovsky and Bernstein stated '… the undisputed, but rather superficial, fact that infant mortality is closely linked to social class'.[224] This undisputed link proved, however, to be less prominent in other studies. Woods et al. argued that the influence of socioeconomic status indeed affected the *level* of infant mortality, but not the *timing* or the *rate of change* of the mortality decline.[225] Moreover, Garrett et al. claimed that socioeconomic differentials in infant mortality were predominantly a result of segregation in specific neighbourhoods of distinct social classes.[226] Hence, the differences experienced were actually caused by environmental differences. In the Netherlands, the relation between socioeconomic status and the infant mortality rate has been on the chopping block as well. Van Poppel et al. argued

Child Health in Britain and the Netherlands in the Twentieth Century (Amsterdam, New York: Rodopi, 2003).

220 Woods, Watterson, and Woodward, 'The Causes of Rapid Infant Mortality Decline in England and Wales, 1861–1921 Part I.'

221 Ibid.

222 Link and Phelan, 'Social Conditions as Fundamental Causes of Disease.'

223 Clouston et al., 'A Social History of Disease.'

224 Aaron Antonovsky and Judith Bernstein, 'Social Class and Infant Mortality,' *Social Science & Medicine* 11, no. 8 (1977): 453.

225 Woods, Watterson, and Woodward, 'The Causes of Rapid Infant Mortality Decline in England and Wales, 1861–1921 Part I.'; R. I. Woods, P.A. Watterson, and J. H. Woodward, 'The Causes of Rapid Infant Mortality Decline in England and Wales, 1861–1921. Part II,' *Population Studies* 43, no. 1 (1989).

226 Eilidh Garrett, Alice Reid, and Simon Szreter, 'Fertility and Child Mortality in Their Household Setting: A Variety of Perspectives from the Uk Censuses, 1861-1911,' *Popalazione e Storia* 2 (2010).

socioeconomic status was only a partial explanation; while socioeconomic status had some influence on infant mortality, its effect severely diminished when environmental factors were taken into account.[227] In Amsterdam, the effect of class differentials in infant mortality was also much smaller compared to the effect of religion, and again the neighbourhood seemed to be a more decisive factor.[228]

A common denominator of these studies is that they focus on all-cause infant mortality. Clouston et al. have shown that socioeconomic inequalities in mortality may emerge for specific causes of death, depending on the availability of resources to combat the specific disease.[229] Thus, at the level of specific diseases, substantial differences in mortality according to class may have existed, while the sum of all these differences appeared to be minor at an aggregated level. Higher social classes in the Dutch town of Roosendaal were indeed capable of shielding their infants when it came down to specific nutrition-related causes of death.[230] Moreover, Clouston et al. highlight the shifting nature of socioeconomic differences in mortality, instead of envisioning a static influence of socioeconomic status.

Late nineteenth and early twentieth century Stockholm is a good example of where socioeconomic status affected infant mortality differently for specific causes of death.[231] While infant class differentials widened for airborne infectious diseases, they converged for water and foodborne infectious diseases. Whereas discriminating factors such as improved nutrition and environmental circumstances (overcrowding) determined the course of airborne infectious diseases, water and foodborne infectious diseases in Stockholm were mainly influenced by a steadily expanding water supply system, soon available to the majority of the population.[232]

The decline in different causes of death can thus be influenced by different direct determinants, which all depend on socioeconomic status. Information on the specific diseases that declined can improve our understanding of the specific mechanism, i.e. the direct determinants, and the role of socioeconomic status in the decline. A useful model to distinguish different direct determinants which can be connected to certain causes of death and which are affected by socioeconomic status, is the Mosley and Chen analytical framework for studying child mortality in developing countries.[233] The model departs from the premise that 'all social and economic determinants of child mortality necessarily operate through a common set of biological mechanisms, or proximate determinants, to exert an impact on mortality'.[234] The Mosley and Chen framework thus

227 Van Poppel, Jonker, and Mandemakers, 'Differential Infant and Child Mortality in Three Dutch Regions, 1812-1909.'

228 Peter Ekamper and Frans van Poppel, 'Infant Mortality in Mid-19th Century Amsterdam: Religion, Social Class, and Space,' *Population, Space and Place* 25, no. 4 (2019). This study was limited to the year 1851 only, thus the effect could have been different in other periods.

229 Clouston et al., 'A Social History of Disease.'

230 Walhout, 'An Infants' Graveyard?,' 197-99.

231 Molitoris, 'Disparities in Death.'

232 Ibid.

233 Mosley and Chen, 'An Analytical Framework for the Study of Child Survival in Developing Countries.'

234 Ibid., 25.

dovetails with the Clouston et al. model, as they both depart from the same premise that socioeconomic status is a fundamental cause.

The framework comprises five categories through which socioeconomic determinants could work: Maternal factors, environmental contamination, nutrient deficiency, injury, and personal illness control.[235] All five determinants have been extensively researched individually, thus we will discuss them only briefly in the following section. Maternal factors encompass the age of the mother, parity and birth interval. Birth order is also regarded as an important factor, although Knodel and Hermalin found that this effect disappeared after including final sibship size into their analysis.[236] A negative effect of very low and especially high ages of the mother affects mostly neonatal mortality, caused by either a premature or waning reproductive system.[237] The mother's old age could also negatively impact survival chances through maternal depletion and an increasing competition for resources among the increasing number of siblings.[238] In the Dutch province of Zeeland, death clustering was found especially amongst large families with high parity.[239] After the neonatal period,[240] biological (i.e. maternal) factors, start to decrease in importance and give way to social and economic factors after the first semester. Weaning can also bring about this change in influential determinants. Social and economic determinants are not as dominant as biological/maternal factors while the infant is being breastfed.[241]

After weaning, environmental factors therefore play a larger role in the odds of dying, which corresponds to the second category of environmental contamination.[242] This category covers the factors which help infectious diseases spread through contamination. Urban environments with characteristically overcrowded spaces, poor hygienic standards and a higher likelihood of contagion were especially unhealthy environments, causing the 'urban penalty'.[243] Urbanised areas in England and Wales were the most unhealthy places for infants between 1-11 months old, caused by an interaction of climatic

235 Ibid.

236 John Knodel and Albert I. Hermalin, 'Effects of Birth Rank, Maternal Age, Birth Interval, and Sibship Size on Infant and Child Mortality: Evidence from 18th and 19th Century Reproductive Histories,' *American Journal of Public Health* 74, no. 10 (1984).

237 Ibid.; Michel Oris, Renzo Derosas, and Marco Breschi, 'Infant and Child Mortality,' in *Life under Pressure. Mortality and Living Standards in Europe and Asia, 1700-1900,* ed. Tommy Bengtsson, Cameron Campbell, and James Z. Lee (Cambridge, Mass.: The MIT Press, 2004).

238 Knodel and Hermalin, 'Effects of Birth Rank, Maternal Age, Birth Interval, and Sibship Size on Infant and Child Mortality.'; Oris, Derosas, and Breschi, 'Infant and Child Mortality.'

239 Otto W. Hoogerhuis, *Baren op Beverland: Vruchtbaarheid en Zuigelingensterfte in Goes en Omliggende Dorpen Gedurende de 19e Eeuw* (Wageningen University, 2003).

240 Which Oris et al. define as the first nine days of life, although it is also often used to describe the first four weeks of life.

241 Oris, Derosas, and Breschi, 'Infant and Child Mortality.'

242 Ibid.

243 Gerry Kearns, 'The Urban Penalty and the Population History of England,' in *Society, Health and Population During the Demographic Transition*, ed. Anders Brändström and Lars-Göran Tedebrand (Stockholm: Almqvist and Wiksell International, 1988); Simon Szreter and Graham Mooney, 'Urbanization, Mortality, and the Standard of Living Debate: New Estimates of the Expectation of Life at Birth in Nineteenth-Century British Cities,' *The Economic History Review* 51, no. 1 (1998).

conditions and the poor sanitary environment.[244] The 'urban penalty' was only reversed in the 1920s and 1930s, with hygienic improvements starting to outweigh the urban risks, combined with a higher availability of medical care in cities.[245] The availability of a drinking water supply system or a sanitation system may have had a significant positive impact on infant survival, although not all studies could ascertain these effects.[246] In the Netherlands, Van Poppel et al. did not find strong differences between rural and urban areas, with the overall region proving to be more important.[247] However, in the province of Brabant, Walhout did find higher infant mortality rates in towns compared to rural settlements.[248]

Whether the higher infant mortality rates in urbanised Brabant were caused by an urban penalty or by different breastfeeding practices remains unclear, although this does bring us to the third factor of nutritional deficiency.[249] Nutritional deficiency factors, through a lack of sufficient caloric, protein and micronutrient intake, help to determine the physical robustness of infants. When the quality of artificial feeding is not sufficient, breastfeeding practices are increasingly important for this factor. The extensive body of research on breastfeeding patterns demonstrates its impact differed substantially across European countries. Although artificially fed infants were continuously on the losing end, breastfeeding practices influenced infant mortality levels and infant mortality decline differently. Where breastfeeding was already a common practice, such as in England and Wales, breastfeeding was only a supportive factor in infant mortality decline.[250] Conversely, in the Netherlands, France, Iceland and northern Sweden, changes in breastfeeding practices are deemed to have been more decisive in bringing forth a decline in IMR.[251]

The substantial positive effects of breastfeeding on infant health have been widely recognised. Historically, breastfeeding was safer for two main reasons. First, it was nutritionally more suitable compared to cow's milk (often diluted with water) or to

244 Woods, Watterson, and Woodward, 'The Causes of Rapid Infant Mortality Decline in England and Wales, 1861–1921 Part I.'

245 Diego Ramiro Farinas and Michel Oris, 'New Approaches to Death in Cities During the Health Transition: An Introduction,' in *New Approaches to Death in Cities During the Health Transition,* ed. Diego Ramiro Farinas and Michel Oris (Switzerland: Springer, 2016).

246 Ekamper and Van Poppel, 'Infant Mortality in Mid-19th Century Amsterdam.'; Hannaliis Jaadla and Allan Puur, 'The Impact of Water Supply and Sanitation on Infant Mortality: Individual-Level Evidence from Tartu, Estonia, 1897–1900,' *Population Studies* 70, no. 2 (2016); Frans van Poppel and Cor van der Heijden, 'The Effects of Water Supply on Infant and Childhood Mortality: A Review of Historical Evidence,' *Health Transition Review* 7, no. 2 (1997).

247 Van Poppel, Jonker, and Mandemakers, 'Differential Infant and Child Mortality in Three Dutch Regions, 1812-1909.'

248 Evelien Walhout, 'Is Breast Best? Evaluating Breastfeeding Patterns and Causes of Infant Death in a Dutch Province in the Period 1875–1900,' *The History of the Family* 15, no. 1 (2010): 87.

249 Ibid., 89.

250 Woods, Watterson, and Woodward, 'The Causes of Rapid Infant Mortality Decline in England and Wales, 1861–1921. Part II.'

251 Catherine Rollet, 'Childhood Mortality in High-Risk Groups: Some Methodological Reflections Based on French Experience,' in *The Decline of Infant and Child Mortality: The European Experience: 1750-1990*, ed. Carlo A. Corsini and Pier Paolo Viazzo (The Hague: Nijhoff, 1997); Gunnar Thorvaldsen, 'Was There a European Breastfeeding Pattern?,' *The History of the Family* 13 (2008); Van Poppel, 'Religion and Health.'

artificial foodstuffs which were difficult to digest.[252] Second, the use of polluted utensils for artificial feeding, like bottles or teats, put the infant at risk of inflammation of the digestive tract. When the infants were fed with low-nutritious artificial food, other signs of malnutrition like wasting could occur as well. In contemporary research on developing countries the correlation between breastfeeding and lower risk of diarrhoea has been confirmed.[253] Therefore, if communities experience high levels of gastrointestinal disorders or malnutrition amongst infants, it is most likely breastfeeding levels are low.

The negative effects of artificial feeding could be mostly reversed, provided that the milk was of good quality, the water used to dilute the milk was not polluted, and the utensils were used hygienically. Hence not only nutritional quality was important, but also hygiene, leading to the next important category of determinants: Personal illness control.[254] Individual behaviour in taking care of infants, either in the home setting through hygienic behaviour or in taking children to a doctor, influences the chances of survival. For England and Wales, Woods et al. argue improved ante-natal and post-natal care indeed reinforced the continuation of the declining trend in infant mortality.[255] Yet even more decisive than medical care may have been the acceptance of a modern attitude towards health, by incorporating hygienic practices into daily life.[256]

The Mosley and Chen model demonstrates the wide range of potential determinants that can influence childhood mortality. Not all factors will be equally applicable to a historical context, since the model is focused on developing countries. The influence of medical care will be less decisive in a historical context where medical knowledge could not save infants once they had fallen severely ill. On the other hand, an extension of the model as proposed by Kok et al. can be useful when adopting a specification of socioeconomic factors according to determinants operating at an individual/family or a community level.[257] When taking into account the family system level in Maastricht for example, it becomes clear that the fourth factor of Mosley and Chen, injury, is not an important factor in our analysis. Infanticide was not a common practice in nineteenth-century Western Europe, whereas it may have been in Asian countries due to differing family systems.[258] Another factor that Kok et al. added is culture, which appears to be

252 Annemarie de Knecht-van Eekelen, *Naar een Rationele Zuigelingenvoeding: Voedingsleer en Kindergeneeskunde in Nederland (1840-1914),* (Nijmegen: Thieme, 1984); Anna Lökke, 'Infant Mortality in Nineteenth Century Denmark: Regionality, Feeding Habits, Illegitimacy and Causes of Death,' *Hygiea Internationals. An Interdisciplinary Journal for the History of Public Health* 3, no. 1 (2002).

253 Laura M. Lamberti et al., 'Breastfeeding and the Risk for Diarrhea Morbidity and Mortality,' *BMC Public Health* 11, no. 3 (2011).

254 Since non-natural deaths occurred only very rarely among the Maastricht infants, the category of injury is not extensively discussed.

255 Woods, Watterson, and Woodward, 'The Causes of Rapid Infant Mortality Decline in England and Wales, 1861–1921. Part II.'

256 Hofstee, *Korte Demografische Geschiedenis*; Woods, Watterson, and Woodward, 'The Causes of Rapid Infant Mortality Decline in England and Wales, 1861–1921. Part II.'

257 Jan Kok, Mattijs Vandezande, and Kees Mandemakers, 'Household Structure, Resource Allocation and Child Well-Being: A Comparison across Family Systems,' *Tijdschrift voor Sociale en Economische Geschiedenis* 8, no. 4 (2011).

258 Ibid.; Katherine A. Lynch, 'Why Weren't (Many) European Women 'Missing'?,' *The History of the Family* 16, no. 3 (2011).

an important factor in the Netherlands.[259] Indeed, the Dutch debate on differential infant mortality revolves around the importance of cultural factors, more specifically religion versus region. This debate is especially relevant when adopting a comparative research design, in which differences in infant mortality decline within the country are under scrutiny. Our particular case study on Maastricht deals with differences within a religiously homogenous city, making the factor religion less relevant in explaining differences within the city walls. However, the region versus religion debate on infant mortality still provides some essential historical context for studying infant mortality in the Netherlands. We will therefore briefly turn to the Dutch debate before proceeding with the analyses of infant mortality in Maastricht.

4.3 The Dutch region versus religion debate

At a national level, the Dutch decline in infant mortality started relatively early (Figure 4.1).[260] Despite an early onset of the national decline, vast regional differences existed for decades. The differences in infant mortality decline appeared both across provinces and within provinces.[261] At a provincial level, northern and western provinces instigated the national declining trend in infant mortality from the 1870s onwards (Figure 4.1). Only a couple of decades later, the south-eastern provinces followed. Noord-Brabant and Limburg were the two provinces with the most diverging course of infant mortality decline. Whereas infant mortality rates had been below the national average in 1850 in these provinces, they witnessed a marked increase in IMR during the final decades of the nineteenth century, which even lasted until 1900 in Limburg. From the 1920s onwards, levels of infant mortality in Noord-Brabant and Limburg were quite similar to each other, albeit still higher compared to other Dutch provinces.

The infant mortality decline in the province of Limburg was more resemblant of the British pattern, with an increase in the final decades of the nineteenth century, and a decline after 1900. Although the Dutch south was fairly industrialised and in that respect more like Britain than most Dutch provinces, the given explanation for the divergences within the Netherlands are mainly rooted in cultural factors. The founding father of these cultural explanations is the sociologist Hofstee, who proclaimed that the differential infant mortality decline after 1875 was the result of changes in personal behaviour regarding health and hygiene, caused by the gradual dispersion of the acceptance of a modern behavioural pattern.[262] Prior to 1875, infant mortality in the north-western provinces was exceptionally high due to the high salinisation of the coastal provinces. The domestic use of groundwater was therefore inhibited, forcing families to use polluted surface water. The use of polluted water was especially detrimental to the frail gastrointestinal tract of infants, who died predominantly of enteritis and diarrhoea.

259 Kok, Vandezande, and Mandemakers, 'Household Structure, Resource Allocation and Child Well-Being.'

260 Ekamper and Van Poppel, 'Zuigelingensterfte Per Gemeente in Nederland, 1841-1939.'

261 Van den Boomen and Ekamper, 'Denied Their 'Natural Nourishment'.'; Walhout, 'An Infants' Graveyard?'

262 Hofstee, *Korte Demografische Geschiedenis*.

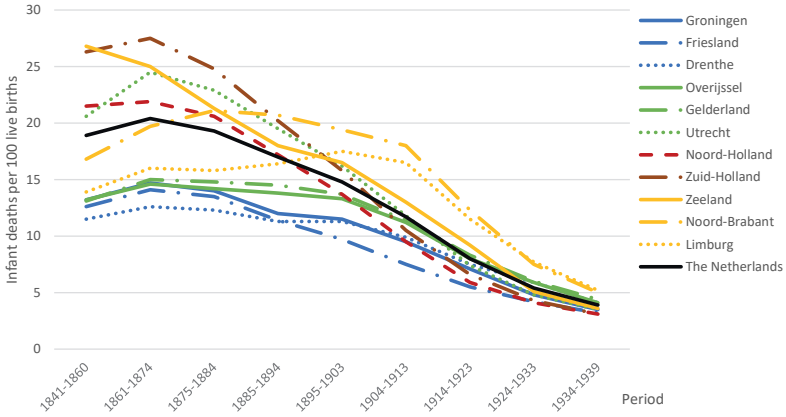


Figure 4.1 Infant mortality decline in the Netherlands by province, 1850-1940

Source: Ekamper and Van Poppel, 'Zuigelingensterfte per Gemeente in Nederland, 1841-1939', 24.

However, the onset of the decline in infant mortality in these coastal provinces was no coincidence. According to Hofstee, the societies in the western Netherlands were more open-minded towards new ideas, as they were accustomed to the influx of new ideas through their already advanced infrastructure and communication networks. This open-mindedness fostered the initial transformation of the cultural setting from traditional to modern.[263] In the southern, and to a lesser extent eastern provinces, inhabitants were less open to new ideas.[264] Communication and infrastructure over longer distances had not yet advanced and expanded as much, keeping families reliant on their traditional cultural habits instead of accepting new hygienic beliefs.[265]

The local communities in Limburg only gradually opened up towards modern ideas, a process which could be more successful if the ideas were transferred via a familiar authority, the Roman-Catholic church.[266] According to Philips, this particular cultural setting was responsible for the shortcomings in nutritional quality and the harmful habits regarding infant feeding practices in the region, causing high infant mortality rates due to gastrointestinal disorders.[267] Yet alongside the cultural explanations, some economic explanations were given as well. De Meere approaches the regional differences from an economic angle, claiming economic prosperity was reflected in nutritional intake, which explained the regional variation.[268] Whereas the western provinces flourished due to shipping, trade and manufacturing, the southern provinces suffered from the agricultural crisis in the 1880s. Consequently, the western parts of the country were

263 Ibid.

264 Ibid.; Philips, *Gezondheidszorg in Limburg.*

265 Hofstee, *Korte Demografische Geschiedenis.*

266 Philips, *Gezondheidszorg in Limburg.*.

267 Ibid.

268 J. M. M. de Meere, *Economische Ontwikkelingen en Levensstandaard in Nederland Gedurende de Eerste Helft van de Negentiende Eeuw: Aspecten en Trends* (The Hague: Martinus Nijhoff BV, 1982).

economically better off and its well-fed inhabitants gained higher resistance to infectious diseases. The opposite was the case for the southern parts, which were subjected to malnutrition and had poor resistance to diseases.[269]

Yet, de Meere's economic explanation does not account for the factor of breastfeeding, which is given at least some attention in many of the other explanations. Vandenbroeke et al. stated that the divide between the north-western and south-eastern regions was an expression of differences in breastfeeding practices, brought about by contrasting female labour participation.[270] Whereas in north-western provinces women increasingly withdrew from the labour market, women in the south-western provinces increasingly joined the emerging industrial labour forces.

Another explanation for different breastfeeding habits between the north and south can be found in the religious divide between the Roman Catholic and Protestant regions. The Catholic faith dominating the south was detrimental to infant health because of its renunciation of breastfeeding after a religious reorientation, issued by the Syllabus Errorum in 1864.[271] After this reorientation, the Catholic clergy propagated a stronger emphasis on morality. Brabantine Catholics developed both a shame complex and a social contamination complex as a consequence of the new moral approach.[272] Catholic women were to cover up as many body parts as possible, especially breasts. Hence, breastfeeding became impossible to practice; if not for covering up the breasts at all times and all costs, than for the even more harmful practice of binding breasts amongst Catholic girls.[273] Van Poppel eventually used the Brabantine case to provide a possible explanation for the rise in infant mortality among Catholics in the Netherlands in general.[274]

Further support was found for the relation between religious denomination and breastfeeding in the Hague, where especially Catholics experienced higher infant mortality rates than the Dutch Reformed and Jews.[275] Wolleswinkel-van den Bosch found the association between Roman Catholicism and higher infant mortality being the strongest among water and foodborne diseases, thus supporting the explanation of a lack of breastfeeding practices amongst Roman Catholic women.[276]

Recently, the conviction on the decisiveness of religion in infant mortality has been upturned. Walhout, focusing on the differences within the Noord-Brabant region, concluded that the association between Roman Catholics and higher infant mortality

269 Ibid.

270 Vandenbroeke, Van Poppel, and Van der Woude, 'De Zuigelingen- En Kindersterfte in België En Nederland in Seculier Perspectief.'

271 P. Meurkens, *Sociale Verandering in het Oude Kempenland (1840-1910): Demografie, Economie en Cultuur van een Preïndustriële Samenleving* (Nijmegen, 1984); Van Poppel, 'Religion and Health.'; Judith Wolleswinkel-van den Bosch et al., 'Determinants of Infant and Early Childhood Mortality Levels and Their Decline in the Netherlands in the Late Nineteenth Century,' *International Journal of Epidemiology* 29, no. 6 (2000).

272 Meurkens, *Sociale Verandering in Het Oude Kempenland.*

273 Ibid.

274 Van Poppel, 'Religion and Health.'

275 Van Poppel, Jonker, and Mandemakers, 'Differential Infant and Child Mortality in Three Dutch Regions, 1812-1909.'

276 Wolleswinkel-van den Bosch, 'The Epidemiological Transition in the Netherlands.'

due to the gastrointestinal infectious diseases was not as straightforward as had been argued.[277] Despite the predominance of Catholicism in the province, diarrhoea was more prevalent in some Protestant (Calvinist) environments than in the predominantly Roman Catholic environments. Furthermore, different Roman Catholic regions in the country followed different trends, and not all Roman Catholic regions were characterised by high infant mortality rates caused by digestive disorders.[278] Moreover, Van den Boomen and Ekamper showed that even when Roman Catholic women started weaning their children earlier, the negative effects of weaning could be reversed by community-level measures such as the implementation of a sewerage system in certain regions.[279] The likelihood of Dutch Catholic women breastfeeding their children did not depend solely on their religion, it depended even more on the region they lived in.[280] Thus, in the region versus religion debate, it appears region *and* religion were influential in the levels and decline of infant mortality.[281]

Clearly, Limburg lagged behind other Dutch regions when it comes to the decline in infant mortality. The Dutch debate on regional differences illuminates the importance of culture and behaviour, expressed through the important factor of breastfeeding. Whilst studying the socioeconomic factors influencing infant mortality, we should keep in mind the importance of these cultural factors in contextualising the infant mortality decline in Maastricht.

4.4 Data

A subset from the *Maastricht Death and Disease Database* (MDDD), including only infants, was used.[282] As discussed in Chapter 2, the dataset contains all the deaths of Maastricht inhabitants and people who died in Maastricht in the period 1864-1955. Some stillbirths have been registered in the MDDD as well, however these have been excluded from the dataset that we used for this chapter. After coding all the causes of death, which is explained in chapter 2 as well, all individual causes of death were classified into nine categories based on aetiology. The first three categories encompass infectious diseases based on their mode of transmission: Water and foodborne infectious diseases (abdominal tuberculosis, cholera, convulsions in children older than 1 month, diarrhoea, gastroenteritis, peritonitis and typhoid fever), airborne infectious diseases (bronchitis, diphtheria, influenza, measles, meningitis, pneumonia, scarlet fever, tuberculosis and whooping cough), and other infectious diseases (nephritis, sepsis, syphilis and tetanus).[283] All the specified non-infectious diseases comprise the fourth

277 Walhout, 'An Infants' Graveyard?'

278 Ibid.

279 Van den Boomen and Ekamper, 'Denied Their 'Natural Nourishment'.'

280 Janssens and Pelzer, 'Lovely Little Angels in Heaven?'

281 Walhout, 'An Infants' Graveyard?'

282 Rutten, *Maastricht Death and Disease Database*.

283 Broadly based on Frans van Poppel, Jona Schellekens, and Aart C. Liefbroer, 'Religious Differentials in Infant and Child Mortality in Holland, 1855-1912,' *Population Studies* 56, no. 3 (2002); Walhout, 'An Infants' Graveyard?'

category, except for causes stemming from endogenous factors such as complications related to birth and congenital malformations (category five), debility (category six) and non-natural causes of death (category seven). Finally, category eight encompasses ill-specified causes of death and category nine unknown causes of death. Categories four to nine were collapsed into one category to ensure enough data power for the regression analysis. The ill-specified and unknown categories may contain some infectious causes of death as well, the differences between infectious diseases and non-infectious diseases may therefore be slightly underestimated in the regression model.

Classifying infant causes of death is however not a straightforward task in all instances. The major conundrum for infants are the often used convulsions (13,1% of infant deaths in our dataset). Although convulsions may refer to gastroenteritis and/or diarrhoea,[284] some studies treated convulsions as an ill-specified disease because of the variety of possible interpretations.[285] Vague causes such as convulsions and atrophy were used for both endogenous deaths (birth traumas and congenital defects, occurring in the first month of life) and exogenous deaths (causes of death caused by environmental causes, occurring after the first month of life).[286] Since endogenous and exogenous mortality stem from different causes, convulsions may refer to different causes of death for the two types of mortality. Jaadla and Puur included convulsions in a digestive infectious disease category on the premise that the death had to have occurred after the first month of life, thus excluding convulsions likely representing endogenous mortality.[287]

Unfortunately, convulsions could represent many other causes of death as well. A German medical handbook, translated and published into Dutch in 1861, explained that '*eklampsie*' could be caused by high temperatures, especially extreme summer heat, unceasing heavy pain, a cold, teething, whooping cough, pneumonia, emerging rash diseases or disorders in the digestive system.[288] In a similar fashion, a 1923 medical dictionary described *eclampsia infantum* as 'convulsions in children with a wide range of causes, organic brain diseases, epilepsy, contagious diseases, nutritional disorders'.[289] Nevertheless, the term convulsions appears to have been frequently used instead of specific gastrointestinal diseases in Limburg.[290]

A solution to classifying convulsions properly may be provided by the analysis of seasonal mortality. When convulsions display a high summer peak, resembling gastroenteritis and diarrhoea, they likely represent digestive infections. Convulsions in our dataset indeed expressed a summer mortality peak in the early stages of the research period. After the 1870s, when the use of convulsions as a cause of death waned, the

284 Kintner, 'Classifying Causes of Death During the Late Nineteenth and Early Twentieth Centuries.'; for neonatal mortality see Reid, 'Neonatal Mortality and Stillbirths in Early Twentieth Century Derbyshire, England.'

285 Molitoris, 'Disparities in Death: Inequality in Cause-Specific Infant and Child Mortality in Stockholm, 1878□1926.'; van Poppel, Schellekens, and Liefbroer, 'Religious Differentials in Infant and Child Mortality in Holland, 1855-1912.'

286 Chris Galley and Robert Woods, 'On the Distribution of Deaths During the First Year of Life,' *Population: An English Selection* 11 (1999).

287 Jaadla and Puur, 'The Impact of Water Supply and Sanitation on Infant Mortality.'

288 Lebert, *Handboek der Praktische Geneeskunde*, 4, 35.

289 Pinkhof, *Vertalend en Verklarend Woordenboek van Uitheemsche Geneeskundige Termen*, 155.

290 Van den Boomen, 'Born Close to Death,' 75.

summer peak became less pronounced. We therefore included convulsions in the digestive infections group if death occurred after the first month of life. This may cause a slight over-estimation of the digestive infections group.[291]

Information on socioeconomic status of the infants was derived from the occupation of the father. In cases where this was not available we used the occupation of the mother if available (Table I). When there was no occupation for either parent, socioeconomic class was classified as unknown. In the MDDD, only 12% of the mothers had a registered occupation. Women in Maastricht often remained working, even when they were married and had given birth to the first couple of children, yet their occupations remained unregistered.[292] The father's occupation is therefore the first choice to work with. All occupations were coded according to HISCO and classified according to the five HISCLASS categories (the elite, lower middle class, skilled workers, farmers, unskilled workers and a sixth unknown category).[293] Typically, most fathers from the elite and middle class were shopkeepers, merchants, clerks or factory overseers. The majority of skilled fathers were *ovenwerkers* (furnace workers), glass blowers, shoemakers, carpenters and *glasslijpers* (glass smoothers). The great majority of parents, both fathers and mothers, among the unskilled workers were day labourers. Among the fathers of the unskilled workers group, one can also commonly find potters, factory workers and excavation workers, while mothers within this group were also employed as factory workers or servants. Other occupational categories than industrial workers were very small, such as the elite and farmers (Table I). In order to ensure enough data power, some categories were again collapsed for the regression analysis. The elite and the lower middle class were merged into the first category, while the farmers and unknown category were combined because of the low numbers of farmers in the urbanised area of Maastricht.

4.5 Methods

Before turning to the analysis of the changing equalities and inequalities according to socioeconomic status, we will identify some major turning points in Maastricht infant mortality decline and establish which cause of death categories were mainly responsible for these turning points. To calculate the infant mortality rates, we used the recorded deaths in the MDDD (excluding stillbirths) and the births based on birth certificates and annual statistics.[294] Furthermore, we will briefly discuss gender differences in

291 A sensitivity check, excluding convulsions in the multinomial logistic regression, showed no substantial differences with the analysis including convulsions.

292 Gales et al., *Het Burgerlijk Armbestuur*; Knotter, *Arbeid van Vrouwen in Limburg in de Twintigste Eeuw*; Schmidt and van Nederveen Meerkerk, 'Reconsidering the 'Firstmale-Breadwinner Economy"; Thijs van Vugt, *Een Arbeidersbuurt Onder de Rook van 'De Sphinx': Een Sociaal-Ruimtelijke Geschiedenis van het Boschstraatkwartier-Oost te Maastricht, 1829-1904* (Hilversum: Verloren, 2015).

293 Van Leeuwen and Maas, *Hisclass*; Van Leeuwen, Maas, and Miles, *Hisco*; Kees Mandemakers et al., 'Hsn Standardized, Hisco Coded and Classified Occupational Titles, Release 2018.01,' (Amsterdam 2018).

294 For the births we used the birth certificates from 1864 until 1922. After 1922, the birth certificates for Maastricht were not yet digitised, which is why the statistical reports of the CBS were used after this period. To ensure consistency in the analysis, we used the deaths from the MDDD, since there were changes in registration practices for the national annual statistics during the research period. Before 1924, the annual statistics reported only live

infant mortality, since boys are well known for having higher infant mortality due to a biological/genetic disadvantage.[295]

We will next turn to the analysis of emerging and receding socioeconomic inequalities in infant mortality, which is based on changing disease patterns along class lines. This provides a good approximation of changing inequalities when cause-specific mortality rates according to socioeconomic status, i.e. a population at risk, are not available. By focusing on the changing mortality patterns we can determine whether changes in mortality patterns were gained in advance by certain socioeconomic groups. We expect the most advantageous group, the upper classes, to display shifts in mortality patterns prior to other groups, and prior to the most pronounced decline in infant mortality.

Since the changing mortality patterns are highly dependable on the actual age of the infant within the first year of life, we stratified the analysis according to age. Within the first month of life, most (but not all) mortality will be due to endogenous causes of death, such as birth traumas or congenital defects. In the following eleven months, deaths are most likely caused by exogenous factors, such as environmental factors and infectious diseases.[296] The level of infant mortality rates according to the month of life can strongly differ between artificially fed children and breastfed children, where artificial feeding practices are often associated with higher mortality from gastrointestinal diseases.[297] The advantages of breastfeeding diminish over the first year of life, as the infant grows stronger.[298] Galley and Woods found that during all the months after the first, digestive diseases were an important cause of deaths for infants, while from the sixth month onwards the infant becomes more susceptible to airborne infectious diseases as well.[299] Different feeding practices causing socioeconomic inequalities may therefore be most visible in the age group of 2-6 months, which is why, in the initial analysis, we stratified according to three age groups (first month, months two up to six, and months seven until eleven). However, the results for differences between water and foodborne mortality and non-infectious diseases after the first month were very similar. To have sufficient observations, we therefore only stratified for neonatal and post-neonatal mortality for water and foodborne mortality. In the case of airborne infectious diseases, clear differences occurred between infants of two to six months and infants of seven to eleven months. The analysis of airborne infectious diseases is therefore presented according to the three age groups.[300]

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births that had survived until their registration. Infants who survived a few hours, but had died before they could be registered were not seen as live births. This changed after 1924 and all live births were used in the national statistics. The birth certificates from 1864 until 1922 contain all live births. See chapter 2 for an overview of the exact sources that were used to calculate the population at risk.

295 Waldron, 'Sex Differences in Human Mortality.'

296 Galley and Woods, 'On the Distribution of Deaths During the First Year of Life'; John Knodel and Hallie Kintner, 'The Impact of Breast Feeding Patterns on the Biometric Analysis of Infant Mortality,' *Demography* 14, no. 4 (1977).

297 Alice Reid, 'Infant Feeding and Post-Neonatal Mortality in Derbyshire, England, in the Early Twentieth Century,' *Population Studies* 56, no. 2 (2002); Rollet, 'Childhood Mortality in High-Risk Groups.'; Walhout, 'Is Breast Best?'

298 Knodel and Kintner, 'The Impact of Breast Feeding Patterns on the Biometric Analysis of Infant Mortality.'; Reid, 'Infant Feeding and Post-Neonatal Mortality in Derbyshire, England, in the Early Twentieth Century'

299 Galley and Woods, 'On the Distribution of Deaths During the First Year of Life.'

300 Age at death was registered in terms of days, weeks, months or years in the MDDD. Since the birth date is

To analyse the socioeconomic differentials in cause of death patterns, we used a multinomial logistic regression. With this regression, we compared the odds for an infant of a specific socioeconomic group of having died of a particular type of infectious disease, compared to having died of a non-infectious cause of death, compared to those odds for an infant of a different socioeconomic group. An important advantage of using multinomial logistic regression, is the fact that we can map socioeconomic inequalities in different periods, check whether these inequalities differed over time, and whether differences were statistically significant.

The dependent variable is the cause of death category, consisting of water and foodborne infectious diseases, airborne infectious diseases, other infectious diseases and non-infectious causes of death. The reference category is the non-infectious disease group, since this is theoretically the final stage of the epidemiological transition. According to the epidemiological transition theory, infectious diseases cease to severely affect mortality rates over the duration of the transition.[301] The share of non-infectious diseases is therefore expected to be larger at the end of the period in comparison to earlier phases of the transition. When infants of a specific socioeconomic group show this proportional change sooner than other groups, this is an indication of an earlier onset of the transition for this group.

We used socioeconomic status and time as independent variables. Gender was not included in the final model, since it remained statistically insignificant in the analysis and excluding it aided in ensuring enough data power. Gender differences in infant mortality may be expected most in the first month of life, however even in that particular group gender had no statistically significant effect. Gender may affect mortality rates, while the disease patterns remain quite similar. Illegitimate children were excluded from the multinomial logistic regression analysis because of their very different circumstances.[302]

Time was treated as a categorical variable, comprising seven periods. The categorisation allows for the separation of periods with diverging trends or which represent special circumstances, for example the World Wars. The seven periods used are: 1864-1883, 1884-1903, 1904-1913, 1914-1919, 1920-1939, 1940-1945 and 1946-1955. At first, we based the periodisation on decennial periods starting in 1864. In order to create enough data power, we collapsed decades with similar trends in infant mortality. While doing so, the differences between the first three periods was safeguarded; the increase in infant mortality rates is captured in the second period, while the first signs of a decline start to appear after the turn of the century in the third period. We also separated the special circumstances during the World Wars, resulting in these seven periods. Finally, we included an interaction term for socioeconomic status and time period, which assesses the effect of socioeconomic status given a certain time period. The interaction thus accounts for time-specific socioeconomic differences in epidemiological patterns.

unknown, we cannot have a more precise indication of age. Children aged 1 year old (which would be the twelfth month) are therefore excluded from the infant mortality analysis, since in this dataset it is impossible to establish whether they had just turned one or were closer to their second birthday.

301 Omran, 'The Epidemiologic Transition.'

302 Jan Kok, Frans van Poppel, and Ellen Kruse, 'Mortality among illegitimate children in mid-nineteenth century The Hague,' in *The Decline of Infant and Child Mortality: The European Experience 1750-1990*, ed. Carlo A. Corsini and Paulo Viazzo (The Hague: Martinus Nijhoff Publishers, 1997).

Table 4.1 Dataset characteristics

Variable		N
Gender	*Male*	8,496
	Female	6,779
Socioeconomic class	*Elite*	320
	Middle class	1,981
	Skilled workers	7,076
	Farmers	248
	Unskilled workers	4,881
	Unknown	770
Socioeconomic status based on	*Father's occupation*	13,919
	Mother's occupation	587
Legitimacy	*Father present*	14,355
	Father not present	921*
Age	*First month*	3,988
	Second to sixth month	7,612
	Seventh to eleventh month	3,676
Period in analysis	*1864-1883*	4,300
	1884-1903	5,280
	1904-1913	2,488
	1914-1919	756
	1920-1939	1,488
	1940-1945	491
	1946-1955	473
Total		**15,276**

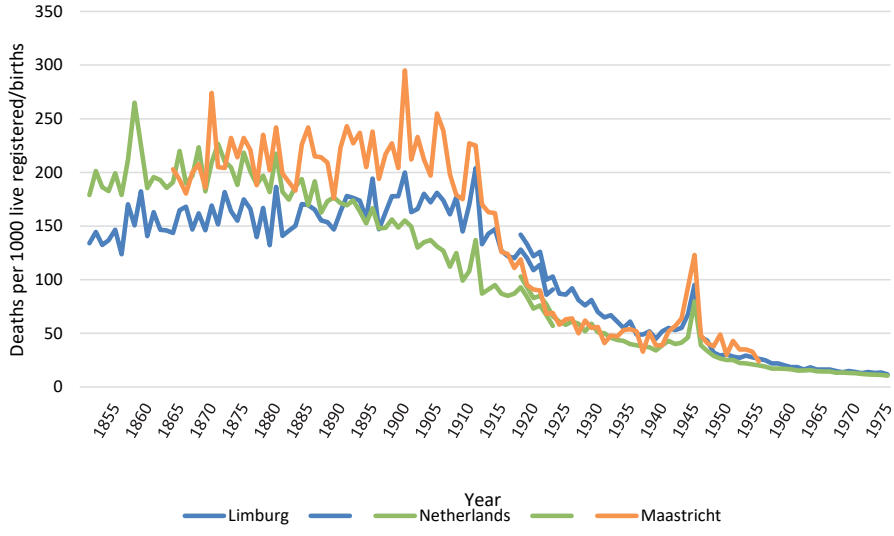


Figure 4.2 Infant mortality in the Netherlands, Limburg and Maastricht, 1850-1970

Sources: Centrale Commissie voor de Statistiek, *Jaarcijfers uitgegeven door de Centrale Commissie voor de Statistiek* (The Hague: Van Weelden & Mingelen, 1893-1898); Centraal Bureau voor de Statistiek, *Jaarcijfers voor het Konink-rijk der Nederlanden: Rijk in Europa* (The Hague: Belinfante, 1899-1924); Centraal Bureau voor de Statistiek, *Statistiek van den loop der bevolking van Nederland*; Centraal Bureau voor de Statistiek, *Statistiek van de loop der bevolking van Nederland*); Centraal Bureau voor de Statistiek, *Statistical Yearbook of the Netherlands* (The Hague, Staatsuitge-verij, 1971-1975); Departement van Binnenlandse Zaken, *Statistisch jaarboek voor het Koninkrijk der Nederlanden* (The Hague, Van Weelden & Mingelen, 1857-1868); Hertogdom Limburg, *Verslag van den toestand van het hertog-dom Limburg over...* (Maastricht: H. Bury,1851-1906); Registers of Birth [unpublished dataset], (1796-1922), RHCL.

4.6 Results

Infant mortality in the southern province of Limburg was characterised by low infant mortality rates during the middle of the nineteenth century compared to the Dutch average. During the final decades of the nineteenth century, the IMR in Limburg and Maastricht increased and only started to decline after the turn of the century (Figure 4.2). The average level of infant mortality between 1864 and 1900 was around 215 deaths per 1000 live births. Yearly variation due to epidemic outbreaks and increased water and foodborne mortality on account of hot summers were, however, substantial. During the decline, three identifiable major turning points occurred. After 1900, an initial small downward trend started, constituting a first turning point. The most decisive turning point came about at the start of the First World War: With an outstanding and swift decline, IMR dropped from 156 to 120 between 1914 and 1915. This impressive decline was not part of a mere fluctuation, but signalled the acceleration of the declining trend during the late 1910s. As a consequence, the Maastricht IMR caught up with the Dutch average in the early 1920s and the high mortality spikes receded. The decline continued until the mid-1930s, indicating a third turning point at the end. Thereafter, IMR peaked temporarily during the Second World War. Instead of being part of the health transition, the dire situation in the Netherlands during the war is the most likely explanation for the peaking mortality during the war years.

The cause of death category responsible for the rapid decline during the second turning point was water and foodborne infectious mortality (Figure 4.3). Debility decreased in the same period, albeit coming from much lower levels already. During the 1920s and 1930s, the decline in airborne infectious diseases drove the remainder of the infant mortality decline. Only during the 1950s was the proportion of non-infectious causes of death for infants larger than the proportion of infectious causes of death. The causes of death related to birth complications or congenital malformations increased slightly, yet this is probably the result of improved registration practices given the decline in debility (Figure 4.3).

Male infant mortality was consistently higher compared to female infant mortality throughout the period (Figure 4.4). Although boys had a continuous disadvantage, the timing of changes in mortality rates did not differ. However, it appears that towards the end of the research period, the differences became smaller, which could be a natural result of lower infant mortality all together.

Thus, while the First World War resulted in dire circumstances for many, it appears that the consequences for infants were the opposite. Since water and foodborne infectious diseases declined slowly from 1900 onwards, with an acceleration during the 1910s, the expectation is that if an advantage had occurred, it would have been in the early twentieth century. The inequalities likely evaporated once the decline had largely set in around 1920. The ensuing decline in airborne infectious diseases may have been characterised by another advantage for the upper classes.

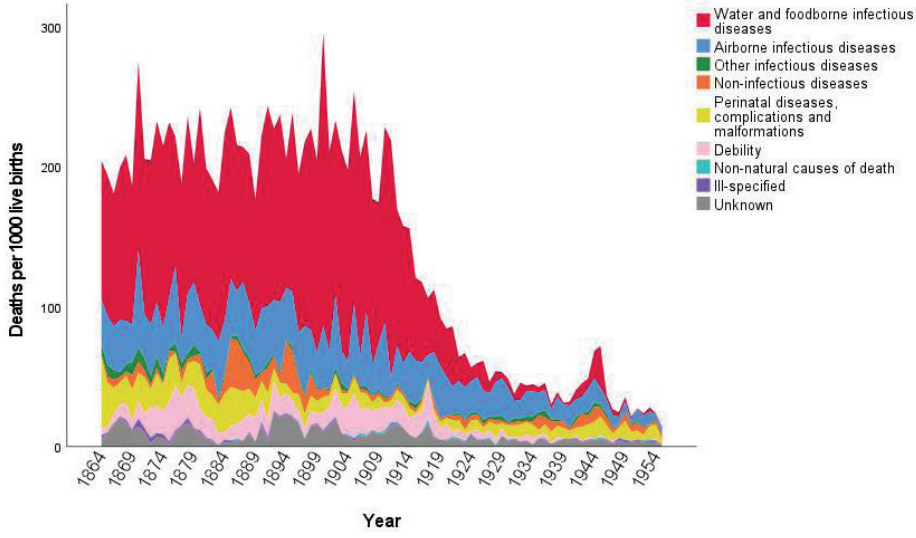


Figure 4.3 Cause-specific infant mortality rates, Maastricht 1864-1955

Sources: Maastricht Death and Disease Database; Dutch censuses

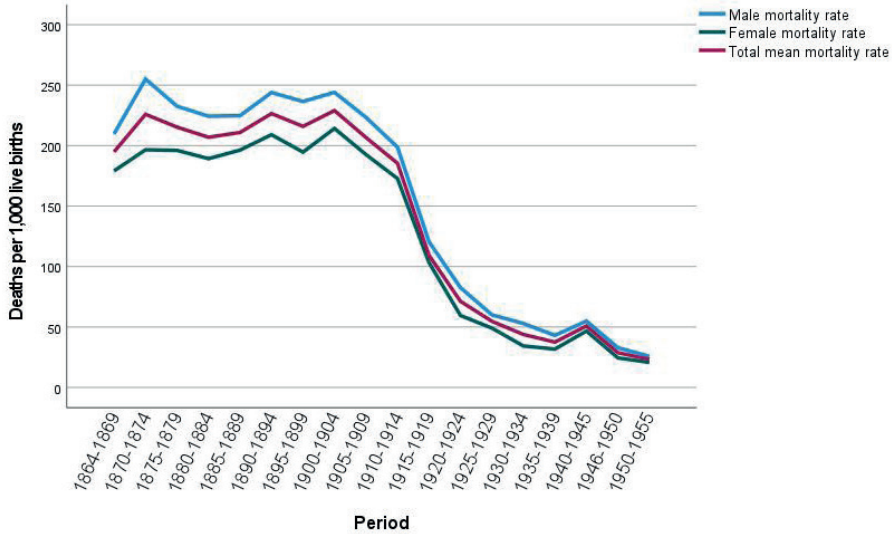


Figure 4.4 Quinquennial gendered infant mortality rates, Maastricht 1864-1955

Source: Maastricht Death and Disease Database; Dutch censuses

To test these hypotheses, we carried out the multinomial logistic regression analyses and presented the results for water and foodborne infectious diseases compared to non-infectious disease, and airborne infectious diseases compared to non-infectious diseases.[303] Figures 4.5 to 4.7 show the proportion of infants, according to a specific age group, that died of a specific infectious disease (y=1) among the total of children having died of that infectious disease group an non-infectious causes of death, for the socioeconomic categories of the upper classes, skilled workers and unskilled workers.

As expected, infant mortality during the first month of life is not as heavily affected by infectious diseases as is the case in the later months within the first year. Regarding the water and foodborne infectious diseases (Figure 4.5a), there were no substantial socioeconomic differences, with the exception being the period between 1904-1913. Contrary to the increasing proportional mortality due to water and foodborne infections among the working classes, neonatal infants from the upper classes had decreasing proportional mortality of water and foodborne infections. These differences were statistically significantly different for infants from skilled workers (p=0.012), and close to statistical significance for infants from unskilled workers (p=0.092) (Annex III).[304]

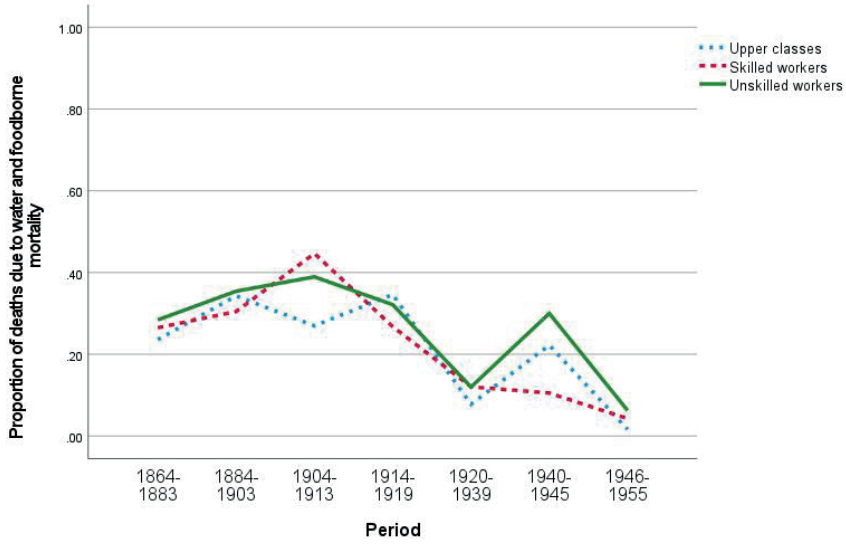
Once infants survived their first month, their susceptibility to infectious diseases increased vastly as the proportions for the infectious diseases were higher compared to their younger counterparts (Figures 4.6 and 4.7). Figure 4.6 depicts the proportional water and foodborne mortality for post-neonatal infants. Infants from the upper classes already experienced lower proportional water and foodborne mortality compared to the working classes in 1864-1883, which might be explained by the cholera epidemic in 1866. This epidemic affected the classes differently according to the municipality report.[305] However, this appears not to be the only discriminator for infants, since the differences remain statistically significant when all cholera cases were excluded from the analysis. The upper class infants subsequently appear to gain an advantage in the early twentieth century. Infants from the unskilled workers had a significantly higher chance of having died of water and foodborne diseases than of non-infectious causes of death compared to the upper-class infants (p=0.044) between 1904-1913. The differences appear to be similar during WWI, although they have lost their statistical significance.

303 The tables of the multinomial logistic regression, including the categories of most interest, can be found in Annex III & IV. The full regression model tables are available from the authors upon request.

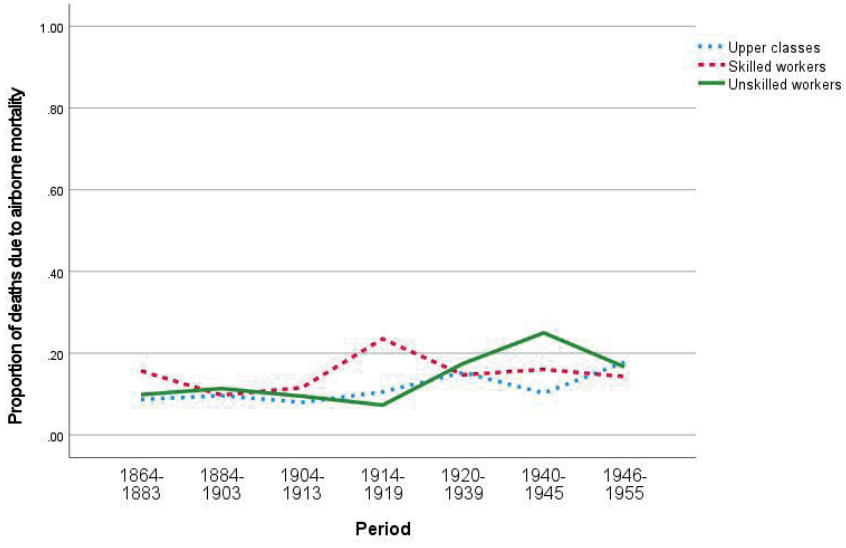
304 The table can be found in Annex III. P-values close to significance are mentioned here as well, since they may also represent an actual difference. Since we are not working with a sample but with a population, these differences were actually present. The p-values mainly indicate whether these differences were more likely based on chance, yet applying the significance levels too strictly may easily lead to an underestimation of inequalities present in the Maastricht infant population.

305 Verslag van Burgemeester en Wethouders nopens de cholera-epidemie in 1866 in Maastricht, (1867), collection number 20.129A, inventory number 5574, Burgerlijke Instellingen van Weldadigheid te Maastricht, Regionaal Historisch Centrum Limburg, Maastricht.

a) Water and foodborne infectious diseases



b) Airborne infectious diseases



Figures 4.5a and 4.5b Proportion of infants up to 1 month old having died of a specific type of infectious disease among the total for infectious disease and non-infectious diseases, 1864-1955

The lower numbers in the period 1914-1919 (Annex III) could explain this loss of statistical significance. When we merge the time periods 1904-1913 and 1914-1919, the proportional water and foodborne mortality for this period between infants from the upper classes and from the unskilled workers gains in statistical significance (p=0.012).

During the first two decades of the twentieth century, infants in this age category with parents belonging to the unskilled working classes had a 83,3% higher chance of having died of a water and foodborne infectious disease instead of a non-infectious cause of death compared to the upper-class infants.

After this short diverging period, mortality patterns regarding water and foodborne infectious diseases for these infants converged again. Differences for airborne infectious diseases appeared to be most pronounced during the First World War, but the results remain statistically insignificant (Figure 4.5b, Annex IV).

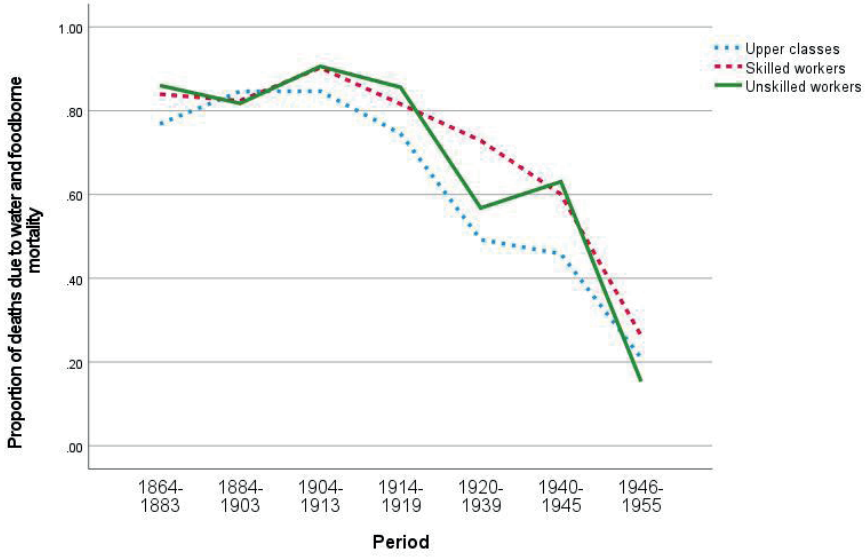


Figure 4.6 Proportion of infants aged two to eleven months of having died of water and food-borne infectious diseases among the total of water and foodborne infections and non-infectious causes of death, 1864-1955

Source: Maastricht Death and Disease Database

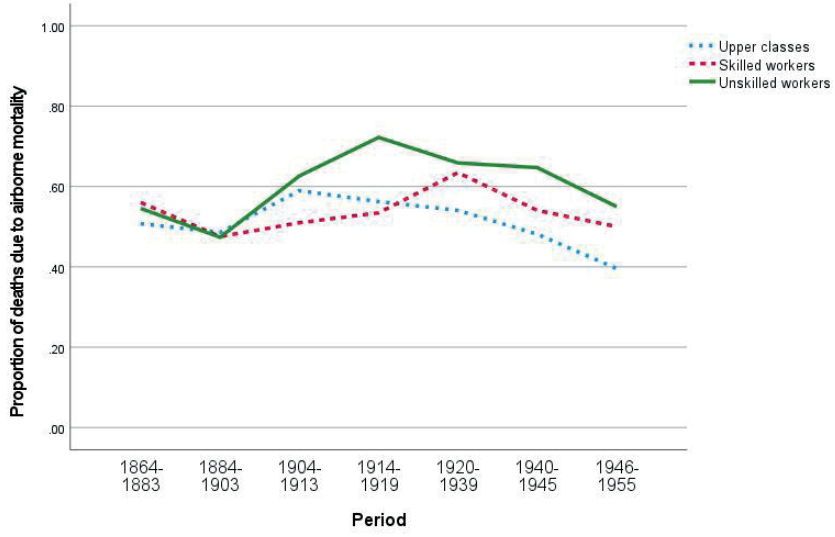
Once the vast decline in infant mortality had started during WWI, proportional water and foodborne mortality declined substantially. Yet for infants over one month of age, the upper-class infants and infants from unskilled-worker families showed a faster decline in proportional mortality than infants from skilled-worker families. These infants lagged behind during the 1920s and 1930s, with an odds ratio of 2.05 compared to infants from the upper classes (p=0.007).

Proportional airborne mortality differed more according to age during the first year of life. Figure 4.7 therefore presents the results of proportional airborne mortality for deceased infants between two and six months (Figure 4.7a), and seven and eleven months (Figure 4.7b). As expected, proportional airborne mortality among the infants aged seven to eleven months is higher compared to their younger counterparts. Differences among the younger infants are small and remain statistically insignificant throughout the period. However, infants between two and six months old from the upper classes started experiencing lower proportional airborne mortality after 1904-1913, while proportional airborne mortality for the infants from skilled workers was increasing. Older infants

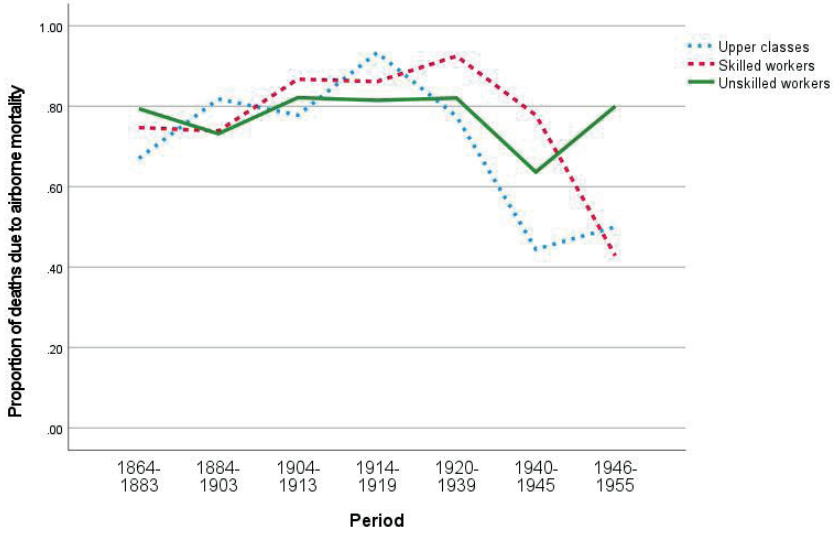
not only had higher proportional airborne mortality, but differences among the classes appear to also have fluctuated to a larger extent (Figure 4.7b). Although the oldest infants from the upper classes had the highest proportional airborne mortality during WWI, they gained an advantage thereafter. Proportional airborne mortality for older upper-class infants declined after WWI, while it increased for infants from the working classes. Especially older infants from skilled-worker families had a disadvantage, with their chances statistically significantly higher ($p=0.020$) of having died of an airborne infectious disease instead of a non-infectious disease compared to infants from the upper classes.

These changes in mortality patterns according to socioeconomic status reflect the mechanism of diverging and converging trends in socioeconomic equalities and inequalities for a specific disease as proposed by Clouston et al. to a certain extent. Prior to the massive decline in infant mortality due to the decrease in water and foodborne infectious diseases during the First World War, it appears that the upper classes already had lower proportional water and foodborne mortality in 1904-1913. The differences in this period between the upper classes and either skilled or unskilled working classes were significant for both neonatal and post-neonatal mortality. Although some differences from the 1940s onwards seem to be large, they all remain statistically insignificant, which is most likely the result of the lower infant mortality rates in that period. Proportional airborne mortality was highest for the oldest infants, and among those infants socioeconomic differences were most pronounced. From the period 1920-1939 onwards, the upper-class infants experienced the lowest proportional infectious mortality, especially after the first month of life. Interestingly, during the 1920s and 1930s, it was not the infants from the unskilled workers who appeared to have been in the least favourable position concerning infectious diseases, but it was actually the infants from the skilled workers. In this period, infants from the skilled workers category have statistically significant higher proportional mortality for both infectious disease groups compared to infants from the upper classes, while infants from the unskilled classes do not differ statistically significantly from the upper classes in this period. This would indicate that the increasing availability of resources to fight infectious diseases made their way from the upper classes to the unskilled workers, and only then to the skilled workers.

a) Two to six months of age



b) Seven to eleven months of age



Figures 4.7a and 4.7b Proportion of infants of having died of airborne infectious diseases among the total of airborne infectious disease and non-infectious diseases according to age category, 1864-1955

Source: Maastricht Death and Disease Database

4.7 Discussion

How can we explain the specific order of socioeconomic inequalities in changing disease patterns that emerged in Maastricht during the early twentieth century? To make sense of the quantitative results, we turn to the historical context of Maastricht to elucidate the different paths the socioeconomic classes took towards a low infant mortality regime. It appears that three events played an important role in the declining infant mortality and the concurrent emerging socioeconomic inequalities in epidemiological patterns. The introduction of a water supply system may have had a first, small effect. More effective, however, was the emerging realisation of the high infant mortality rate and the increased awareness about what could be done to curb it. The dire economic circumstances during World War I may have been the final push for larger proportions of society to seek help, which in turn improved infant survival.

The effects of these three events were highly dependent on both socioeconomic status and sociocultural factors. In order to understand how socioeconomic status and sociocultural factors brought forth inequalities in epidemiological patterns in the course of these events, we will return to the most relevant direct determinants of infant mortality for our case as proposed by the heuristic framework of Mosley and Chen. We focus on the most dominant cause of death category for infants in our study period: The water and foodborne infectious diseases. Causes of death in this category are very closely related to feeding practices, which in turn can be connected to three possible determinants from the Mosley and Chen model: Environmental contamination, nutritional deficiency and personal illness control. In the following section we will therefore first start with a brief recapitulation of what is known about infant feeding practices in nineteenth and early twentieth-century Maastricht. Next, we will move to the three mentioned events that contributed to infant mortality decline. In the discussion of each event, the three determinants from Mosley and Chen serve as a guideline to point out the important elements for socioeconomically differentiated changes in epidemiological patterns. Finally, we will touch upon the declining fertility in the 1910s. While our aim is not to partake in the debate whether a decline in fertility or a decline in infant mortality came first, the declining fertility rate is an important factor to mention at the end of this discussion.

It is assumed that in the southern part of the Netherlands, breastfeeding was uncommon among women due to the sociocultural factor of Catholicism which prohibited the exposure of female body parts.[306] Yet breastfeeding may also have been uncommon because of the necessity for working-class mothers to participate in the labour force.[307] Contemporaries already commented on local infant feeding practices in the early twentieth century. In 1904, the local Health Inspectorate reported that high infant mortality rates were caused amongst others by artificial feeding practices, the quality and preparation of cow's milk to feed the infant, the employment of mothers in

306 Meurkens, *Sociale Verandering in Het Oude Kempenland*; Van Poppel, 'Religion and Health.'

307 Meurkens, *Sociale Verandering in Het Oude Kempenland*; van Poppel, 'Religion and Health'; Vandenbroeke, Van Poppel, and Van der Woude, 'De Zuigelingen- en Kindersterfte in België en Nederland in Seculier Perspectief.'

factories which caused them to outsource the care for their children, and whether or not the child was conceived out of wedlock.[308]

Although exact quantitative data on the number of mothers who did or did not breastfeed are lacking, there are some clear indications that women in Maastricht were often not able to breastfeed their children, and were instead using artificial feeding.[309] Artificial feeding practices opened the door to a range of factors which could harm the infant. Nutritional deficiency may have resulted from the questionable quality and suitability of the milk or porridge that was given to the infant. On top of that, environmental contamination interacting with personal illness control may have heavily influenced the exposure of infants to bacterial microbes. Milk or porridges were often diluted with contaminated water. Yet even with high-quality milk and water, the utensils used to prepare the infants' food could still be contaminated, making hygienic practices equally essential to the survival of the infant. Moreover, the impediments to proper infant feeding demonstrate the strong interaction between the three direct determinants.

Yet some events could operate predominantly through a single determinant. In the case of the first event, the introduction of the water supply system, improvements were made in the area of environmental contamination. The presence of a clean water supply system is generally deemed to be of importance for infant mortality, as it has been shown to have been decisive for infant survival in certain urban areas.[310] For Maastricht, however, the introduction of a piped water system seems to have had little impact on infant mortality rates. The system was introduced to the wealthy in 1888, but its dissemination unfolded only gradually. During the 1910s, when reductions in infant mortality rates were highest and disease patterns changed most quickly, only relatively few new properties were connected to the piped water system.[311] Infants from upper-class families may have gained a small early advantage concerning water and foodborne diseases by reductions in environmental contamination, because these families may already have been connected to the piped water supply system.

The second, more effective event in reducing infant mortality was the raised awareness regarding infant mortality. Organisations preoccupied with improving the public health of the population of Maastricht, as well as individual families, had to become aware of the importance of bringing down infant mortality. Already during the late nineteenth century, local private initiatives were aiming to improve the health of the Maastricht population, yet infant mortality remained out of their scope until the early twentieth century.[312] While the *Société de la Charité/Het Genootschap van Moederlijke Weldadigheid* (founded in 1840), an organisation consisting of upper-class women, did aim to support new mothers in need, they did not directly tackle infant mortality in general.

308 Gemeente Maastricht, 'Verslag van den Toestand der Gemeente Maastricht over het Jaar 1904,' (Maastricht: Gemeente Maastricht, 1905).

309 Jaarverslagen, (1913-1917, 1919-1921), collection number EAN_0930, inventory number 1, (RK Vereniging) Het Limburgse Groene Kruis afdeling Maastricht, Sociaal Historisch Centrum Limburg, Maastricht.

310 Jaadla and Puur, 'The Impact of Water Supply and Sanitation on Infant Mortality'; Molitoris, 'Disparities in Death.'

311 Cillekens, Boogaard, and Gales, *Loop Naar De Pomp*.

312 C.P.F. Andreas, 'Pro Infantibus en Crèche Juliana, Pioniers in de Strijd tegen Kindersterfte,' in *Bonne et Servante: Uit de Geschiedenis van de Maastrichtse Vrouw*, ed. Ingrid M. H. Evers and Th. J. van Rensch (Maastricht: Gemeentelijke Archiefdienst, 1986); Evers and Ubachs, *Voorkomen Is Beter Dan Genezen*.

The local Health Inspectorate in Maastricht also barely mentioned infant mortality in their reports throughout the final decades of the nineteenth century. Attitudes started changing when they included an elaborate report on the issue of infant mortality for the first time in 1904. The Health Inspectorate expressed their concern again in 1911, as did newspapers throughout the country, when Maastricht was struck by an extremely hot summer leading to soaring infant mortality rates.[313] Strikingly, the infant mortality rate in Maastricht had actually been higher in 1900, but at that time it went without any notification. These reports of the local Health Inspectorate reflect the increased awareness of the problematic infant mortality after the first couple of years of the twentieth century among the local upper classes, who filled the ranks of the organisation. Their ideas must have affected the ideas and attitudes of mothers within their social circle as well, which could have induced a small early advantage in proportional water and foodborne mortality among the upper-class families. While working-class families experienced increased proportional water and foodborne mortality in the period between 1904 and 1913, most likely partly due to a series of hot summers, upper-class families appeared to have been able to shield their infants to a certain extent. Whether this was the consequence of improved nutritional quality (switching to breastfeeding, for example) or improved personal illness control remains uncertain, since an advantage was gained both in neonatal and post-neonatal mortality. The Health Inspectorate tried to spread this awareness to other social groups as well. After their first notification of infant mortality in 1904, they decided to hand out leaflets to new parents upon registration of their new-borns, titled *'Onderrichtingen voor moeders'* (Instructions for mothers).[314] With the aim of increasing awareness about childcare among mothers, they operated in the realm of personal illness control.

The changing attitude also manifested itself in the founding of several organisations with the purpose of bringing down infant mortality levels. First and foremost, these organisations concentrated on nutritional deficiency. The organisation *Pro Infantibus*, founded in 1908, provided milk for infants, prepared and sterilised in their own kitchen and diluted according to their best-known ratios. The inclusion of several upper-class women on the organisation's board emphasises the preoccupation of upper-class women with the high infant mortality.[315] The organisation tried to reach lower-class mothers by providing milk to the needy classes without charge, although on the provision that they could hand over a doctor's prescription. Another prerequisite, for all mothers, was that they accepted home visits from female members of the organisation and that they visited the consultation bureau with their new-borns on a weekly basis.[316] The success of the organisation was small at its start, as in the first year only 113 children from the poor classes were provided any milk by *Pro Infantibus*,[317] while over ten times as many infants were born in either 1908 or 1909. Perhaps *Pro Infantibus'* methods were too invasive for

313 Gemeente Maastricht, 'Verslag van den Toestand der Gemeente Maastricht over het Jaar 1911,' (Maastricht: Gemeente Maastricht, 1912).

314 Gemeente Maastricht, 'Verslag van den Toestand der Gemeente Maastricht over het Jaar 1904.'

315 Andreas, 'Pro Infantibus en Crèche Juliana, Pioniers in de Strijd tegen Kindersterfte,' 89.

316 Ibid.

317 Notulen, (07-05-1908 to 01-06-1911), collection number 21.031, inventory number 3, Vereniging sinds 1979 Stichting Pro Infantibus Maastricht 1908-1999, Regionaal Historisch Centrum Limburg, Maastricht.

most mothers. Mothers who had been used to taking care of their own children, with the support from family members or neighbours, now had to hand over some of their authority to the organisation and its doctor. In Noord-Brabant, the Dutch province experiencing similar problematic high infant mortality at the time, the dispersion of knowledge on proper infant feeding and care was highly dependent on an intermediary organisation, bridging between health officials and the higher classes on one side, and the wider population on the other side.[318] Instead of imposing ideas from the top down, new ideas spread through visiting nurses whose success largely depended on the fact that they were religious and female, which gained the trust from the Catholic mothers.[319]

In Maastricht, the *Limburgse Groene Kruis* (Limburg Green Cross), founded in 1910, deployed exactly these Catholic nuns to pay home visits to mothers. The Green Cross' objective was to improve the health of the entire Maastricht population, although with a specific focus on infant mortality evolving around three main principles: Natural feeding, improvement of knowledge in working-class families and hygienic practices.[320] Thus, while nutritional deficiency was one of their primary concerns, they also addressed the realm of personal illness control by concentrating on the improvement of knowledge. The Catholic foundation of the *Green Cross* was an important and appealing asset.[321] Since the visiting nurses already had some common ground with the mothers, their visits may have been less invasive. Over the years, mothers became more proficient in breastfeeding under the guidance of the nurses, for which they previously simply did not have the know-how or experience according to the nurses.[322] Moreover, to improve the knowledge of mothers about infant care, the *Green Cross* started motherhood classes, which became increasingly popular during the 1910s.[323]

The foundation of these organisations demonstrates on the one hand how in the higher social circles the awareness of the problematic infant mortality levels increased, and how they tried to spread that awareness to the working classes to curb the infant mortality on the other. Yet the plummeting infant mortality rate between 1914 and 1915 cannot solely be explained by an increased supply of knowledge on hygiene and feeding practices, and a supply of higher quality milk, since these improvements already existed before 1914. Although the knowledge obtained by an individual during a motherhood class or after a home visit may have spread to her family, friends and neighbours, the very particular timing of the start of the vast infant mortality decline is in need of an additional explanation.

318 Wijnen-Sponselee, *Het Wit-Gele Kruis in Noord Brabant. Intermediair Tussen Medische Verworvenheid En Sociale Acceptatie*.

319 Ibid.

320 Jaarverslagen, (1913-1915), collection number EAN_0930, inventory number 1, (RK Vereniging) Het Limburgse Groene Kruis afdeling Maastricht, Sociaal Historisch Centrum Limburg, Maastricht.

321 Philips, *Gezondheidszorg in Limburg.*

322 Jaarverslagen, (1913-1917, 1919-1921), collection number EAN_0930, inventory number 1, (RK Vereniging) Het Limburgse Groene Kruis afdeling Maastricht, Sociaal Historisch Centrum Limburg, Maastricht.

323 Jaarverslag, (1913-1915), collection number EAN_0930, inventory number 1, (RK Vereniging) Het Limburgse Groene Kruis afdeling Maastricht, Sociaal Historisch Centrum Limburg, Maastricht.

The third event affecting infant mortality decline and its concurrent socioeconomic inequalities, was the start of World War I, despite Dutch neutrality, and the dire socioeconomic circumstances it brought to the city. Counterintuitive as it may seem, since crises like a war can have a negative effect on overall mortality or infant mortality,[324] the First World War was actually not detrimental to Maastricht infants. Contemporaries already discovered that the Dutch infant mortality rates had not increased due to the war, despite the toll of the Spanish flu.[325] The Great War affected countries differently. Whereas in Berlin a crisis in infant mortality emerged, albeit mainly affecting illegitimate children,[326] infants from London did not experience negative consequences.[327]

A contemporaneous protagonist of breastfeeding suggested that the decline in infant mortality during the War resulted from increased milk prices, which forced women to turn to breastfeeding. A precedent for this mechanism could be found in past wars and periods of crises.[328] However, rising milk prices in the Netherlands only became problematic during the later war years,[329] while the steepest infant mortality decline started in the early war years. Other changes in socioeconomic circumstances at the start of the War had larger consequences.

The vast industrial sector of Maastricht, with at its core luxury goods manufactured in the pottery and glass industry, collapsed after the start of the War. Not only the demand for these goods diminished, the British blockade at sea and the U-boat warfare made trade in both raw materials and finished products next to impossible.[330] Factory floors were furthermore partially abandoned due to the mobilisation of men, whereupon many factories decided to close their doors entirely.[331] Employees who were not mobilised lost their jobs and income, hence unemployment numbers rose massively. Since many women remained working after marriage as well, the effects of unemployment on the family income were felt severely. A special committee was founded, the *Steuncomité* (Support Committee), to support families in need due to their lost income. In 1915, 4.520 families, or 18.072 individuals, depended on the *Steuncomité*, nearly half of the total Maastricht population.[332]

The resulting benefits for infants were twofold. First, mothers who had lost their job were now able to take care of their own children at home, instead of relying on others

324 France Meslé, 'La Mortalité Infantile en Europe Pendant les Deux Guerres Mondiales,' in *Bouleversements Démographiques de la Grande Guerre, ed.* Jean-Marc Rohrbasser (Paris: Ined éditions, 2014).

325 Commissie Centrale Gezondheidsraad, *De Invloed Van Den Oorlogstoestand Op Den Gezondheidstoestand Van Het Nederlandsche Volk* (The Hague, Algemeene Landsdrukkerij, 1921).

326 Jay Winter and Joshua Cole, 'Fluctuations in Infant Mortality Rates in Berlin During and after the First World War,' *European Journal of Population / Revue Européenne de Démographie* 9, no. 3 (1993).

327 Jay Winter, Jon Lawrence, and Jackie Ariouat, 'The Impact of the Great War on Infant Mortality in London,' *Annales de Démographie Historique* (1993).

328 'Melknood En Zuigelingensterfte (Letter to the newspaper),' *Rotterdamse Courant*, 27-11-1915 (1915).

329 Paul Moeyes, *Buiten Schot: Nederland Tijdens de Eerste Wereldoorlog 1914-1918*, 4 ed. (Amsterdam: De Arbeiderspers, 2014).

330 Ibid.

331 Ibid.

332 Verslag der werkzaamheden van de Steuncommissie (1914), collection number 20.139, inventory number 9, Steuncommissie 1914 Maastricht, Regionaal Historisch Centrum Limburg, Maastricht.

to care of the young ones. In Belgium, industrial employment of mothers has been shown to have a detrimental effect on infants and young children,[333] which was also mentioned by the Maastricht Health Inspectorate.[334] This detrimental effect was now reversed. Second, the lowered family income may have been a push factor for mothers to seek any kind of support or relief, which drove mothers into the welcoming arms of *Pro Infantibus* or the *Green Cross*. The poorest families, who received support, now started to gain an advantage.

After World War I, the changes in cause of death patterns of infants from unskilled workers and infants from the upper classes were in sync, while infants from skilled workers still showed a more disadvantageous cause of death pattern. A potential explanation for the lagging behind of the skilled workers would be that they were not in the direst of circumstances during WWI, and felt less necessity to seek support. However, the fact that skilled workers were also furnace workers and glass blowers who occupied the factory floors, may argue against that. These men would have been fired as well, while perhaps other skilled workers such as shoemakers and carpenters remained able to make a living throughout the war. Moreover, the target audience of the *Green Cross* were the lower classes, and it was only well into the 1920s that the *Green Cross* provided help for the middle classes too.[335] The question remains what they considered middle class; would a part of that group end up in the skilled workers group according to HISCLASS? It does appear that the group who were not the frontrunners in implementing new hygienic ideas and feeding practices concerning infants, nor in the worst socioeconomic position in society, actually came last in the distribution of resources to prevent water and foodborne infant mortality.

Finally, during the 1910s fertility declined as well. The increased food prices at the start of the First World War provided an economic incentive to lower birth rates, which may have benefitted maternal factors. High food prices have a high impact on fertility, much more than on infant mortality, especially among the poorer classes.[336] The decline in crude birth rates in Maastricht started slowly in 1909 and accelerated from 1914 to 1915. While still declining in 1916, the crude birth rate remained at the same level for the next two years. The marital fertility rates in Limburg illustrate the declining birth rates. After its peak in 1899/1900, marital fertility rates in Limburg had started to decline for the first time in over 50 years and by 1919/1920, the marital fertility rate had dropped even lower than in 1849/1850.[337] Since the crude birth rates indicate that the decline in the 1910s intensified simultaneously with the decline in IMR from 1914 to 1915, it is impossible to assert which of the two came first. Yet marital fertility at least contributed somehow to the continuation of the decline in infant mortality.

333 Tina Van Rossem, *Bruxelles Ma Belle, Bruxelles Mortelle: An Investigation into Excess Mortality in Brussels at the Turn of the Twentieth Century* (Brussels Ghent: Vrije Universiteit Brussel Ghent University, 2018).

334 Gemeente Maastricht, 'Verslag van den Toestand der Gemeente Maastricht over het Jaar 1904.'

335 Jaarverslag, (1913-1917, 1919-1921), collection number EAN_0930, inventory number 1, (RK Vereniging) Het Limburgse Groene Kruis afdeling Maastricht, Sociaal Historisch Centrum Limburg, Maastricht.

336 Oris, Derosas, and Breschi, 'Infant and Child Mortality.'

337 Centraal Bureau voor de Statistiek, 'Regionale Huwelijksvruchtbaarheid: Provinciale Cijfers, 1849/1850-1979,' (The Hague: Staatsuitgeverij, 1981).

After the steepest infant mortality decline in the 1910s, a slower decline in airborne infections continued throughout the 1920s and class differentials emerged in this disease group. Again, the infants from skilled workers experienced the most unfavourable cause of death pattern, with highest proportional airborne mortality in the group aged seven to eleven months. Airborne infectious diseases are closely related to both nutritional deficiency and environmental contamination.[338] The temporary advantage of the upper classes in this case is not likely to be the consequence of improved housing conditions or a quantitative increase in nutrition, as they are expected to already have had access to these resources in an earlier period. The determinant of nutritional deficiency could, however, have been important in regards to a qualitative increase in nutritional intake. Had the upper classes become more knowledgeable about nutritious diets after having started weaning, prior to mothers of infants in the skilled workers group? The route of knowledge transfer from the upper classes to the most needy classes and finally to the intermediate classes may have been decisive here, too. This hypothesis could equally apply to education on dealing with airborne infectious diseases and how to provide care for sick infants, yet this would require further research.

4.8 Conclusion

The decline in infant mortality was one of the key forces in the health transition due to its substantial contribution to overall mortality decline. Once the majority of newborns survived their first year of life, life expectancy rose dramatically. Whereas in many European countries, as in some parts of the Netherlands, the decline in infant mortality already started from the 1880s onwards, Maastricht lagged behind. The decline in the industrialised city started slowly around 1900, only to accelerate during the 1910s on the brink of the First World War. The reason for the 'backwardness' of Maastricht and Limburg in general has been attributed to the Catholic faith; causing society to turn inwards and not being open to new ideas on modern behaviour concerning medicine and hygiene. Mainly cultural factors have therefore been used to explain the diverging pattern in infant mortality decline in the Netherlands. Socioeconomic status is regarded as having played a minimal role in the Dutch decline in infant mortality. Internationally, the effect of socioeconomic status on infant mortality has been highly debated. Region seems to be a more influential determinant than socioeconomic status, although in industrialised areas it does appear to have an effect. Yet one of the most widely used frameworks to study infant mortality, the Mosley and Chen model, departs from the premise that direct determinants are influenced by socioeconomic status. Within the Mosley and Chen model, sociocultural factors should not be forgotten. What was therefore the role of socioeconomic and sociocultural factors on differential mortality patterns within an urban population?

In Maastricht, socioeconomic disparities indeed expressed themselves in the progressing mortality decline, in line with the four stages of disease as proposed by

338 'The Relationship of Nutrition, Disease, and Social Conditions: A Graphical Presentation,' *The Journal of Interdisciplinary History* 14, no. 2 (1983); McKeown, *The Modern Rise of Population*.

Clouston et al..[339] By using multinomial logistic regression analysis to approximate socioeconomic differences through diverging disease patterns, we concluded that the upper classes gained an advantage, followed by infants from the unskilled workers and finally the infants from the skilled-worker families. The uneven changes in disease pattern indicate first of all that it was not an all-encompassing, non-discriminating measure that influenced the mortality decline, such as a quick roll-out of piped water supply would offer. Conversely, the upper classes gained a first advantage regarding the predominant cause of death for infants – gastrointestinal infections – from the turn of the century onwards. The advantage among the upper classes was caused primarily by an increased awareness about the issue of high infant mortality, which in turn influenced most likely first feeding practices and some hygienic practices. Not only the Maastricht Health Inspectorate demonstrated this increased awareness, but also the foundation of two private organisations with the objective of increasing infant health by providing high-quality milk or support symbolises the recognition of a high infant mortality problem. Upper-class mothers may have been in closer contact with these organisations, as they moved within the same social circles. Another secondary advantage for the upper classes may have been gained by the introduction of the water supply system, although its effect is most likely small due to its a-synchronic timing of implementation.

In the end, it was religion that eventually bridged the two sides of the *health system*, the demand and supply, and the provision of medical/hygienic care and practices, and the receptiveness to these, thus bringing down the high infant mortality rates in the lower social classes. The Catholic underpinnings of the *Limburg Green Cross* organisation were the key to the homes of mothers. However, it was not solely the sociocultural factor of the Catholic faith that incentivised mothers to seek for help. A third event, affecting the economic circumstances of working-class families, helps to explain the causes of the very swift infant mortality decline from 1914 to 1915. Economic hardship caused by the outbreak of the First World War, causing massive unemployment in a city that highly relied on the industries of luxury goods, pushed the poorest households into the arms of support organisations. Infants from the skilled workers nonetheless lagged behind infants from the unskilled workers and upper classes in the 1920s and 1930s, which was expressed in both proportional water and foodborne mortality and proportional airborne mortality. We should keep in mind that HISCLASS coding practices may severely influence this finding. However, it does appear that the group which was not the frontrunner in declining infant mortality, nor in the worst position socioeconomically, was the group lagging behind most and the last to gain access to essential resources.

Although there were several instances when there were no substantial socioeconomic inequalities in mortality among infants, the analysis of cause-specific mortality over an extended period illuminates the mechanisms of socioeconomic inequalities in the course of the health transition. Advantages were gained by privileged groups in the process of decline, as reflected by an earlier transition in epidemiological patterns. Instead of being non-existent, these socioeconomic mechanisms may only be hidden in aggregated or all-cause mortality analyses. In understanding the effects of socioeconomic status on infant mortality, we are in need of more detailed, cause-specific analyses that take

339 Clouston et al., 'A Social History of Disease.'

into account the changing nature of the influence of class differentials. Moreover, a simple socioeconomic gradient does not always apply, since infants from skilled workers were lagging behind most. The very specific order of improving cause of death patterns indicates that we should look further into the specific characteristics of socioeconomic groups, in order to understand the mechanisms causing socioeconomic inequalities.

CHAPTER 5

5

Changing diseases, changing inequalities? Transitioning early childhood mortality in Maastricht, 1864-1955

5.1 Introduction

Once new-borns had made it through the hazardous first year, either by surviving or evading wracking gastrointestinal infections, new challenges where ahead as they entered their second year of life. Although many children aged one could still suffer from gastrointestinal infections, it were the characteristic outbreaks of childhood diseases which caused great havoc in the years between one and five. Recurring outbreaks of scarlet fever, diphtheria, measles and whooping cough added to the mortality rates of young children, on top of more endemically raging respiratory infections like pneumonia, bronchitis and tuberculosis. The disease environment in early childhood was predominantly characterised by these different airborne infections, in contrast to the dominant position of gastrointestinal infections determining the odds of survival for infants.[340]

In studying the health transition, much attention has understandably been given to infant mortality decline and the large contribution it made to the overall mortality decline and increasing life expectancy. Whereas prior to the health transition all age groups were affected by infectious diseases, at the start of the transitional phase the hazards had shifted to the most vulnerable groups within the population at both ends of life: Infants, young children and the elderly. For this reason, the mechanisms of early childhood mortality decline are also crucial in gaining a better understanding of how the health transition took place and which role the decline of epidemic outbreaks of infectious diseases, haunting young children, played during the transition. Moreover, since mortality patterns differed between infants and young children, it is essential to study young children separately from infants.

Since young children were susceptible to a variety of typical childhood diseases, the decline of one particular cause of death may have had little effect on the overall early childhood mortality decline. It is therefore crucial to take into account the wider spectrum of diseases in explaining the mortality decline, which I will refer to as the disease environment. This disease environment entails more than the occurrence of certain diseases at a specific location, it takes into account the interplay between those diseases, where a decline in one may cause an increase in another. Moreover, although very difficult to study reliably, morbidity should not be lost from sight. Even when mortality declines, morbidity from a particular disease can still contribute to harming the immune system of children. In that way, morbidity adds to the total burden of disease, which could end in death, even from something as 'simple' as pneumonia.

Adhering to the wider objective of this dissertation, the inequalities created and lost due to the decline in early childhood mortality will be a focus point here. For the Netherlands, is has been found that socioeconomic inequalities were more pronounced in early childhood mortality compared to infant mortality, although the socioeconomic gradient was very small.[341] For Britain, it has been argued that socioeconomic differences were not on par with the impact of regional disease environments. In heavily industrialised

340 See for example Robert Woods and Nicola Shelton, *An Atlas of Victorian Mortality* (Liverpool: Liverpool University Press, 1997), 34-37, for an overview on the highest cause specific mortality rates for each age group.

341 Van Poppel, Jonker, and Mandemakers, 'Differential Infant and Child Mortality in Three Dutch Regions, 1812-1909,' 303.

regions, socioeconomic inequalities, however, did have an effect on the health of young children.[342] Apart from these inequalities, many studies have demonstrated the existence of gender inequalities in early childhood, in particular regarding typical infectious childhood diseases.[343] With the decline of precisely these infectious diseases, gender inequalities may have changed during the nineteenth and early twentieth century.

Studying the changing disease environment over a long period of time for young children will shed light on how the mortality transition influenced inequalities between the sexes. Central to this chapter will therefore be two questions. First, how did the decline in early childhood mortality progress and how did the disease environment change with this decline, between the mid-nineteenth century and the first half of the twentieth century? Second, which inequalities resulted from or disappeared due to the decline in early childhood mortality? In order to answer my two main questions, this chapter will be divided into two main sections. In the first section, I will examine the theoretical constitution of the disease environment, and analyse early childhood mortality decline and the role of specific diseases within the disease environment of Maastricht. In the second section, the relation of this disease environment with health inequalities will be central, to understand how the changes in disease environment affected health inequalities among young children. Due to data constraints, the data do not allow for a complex modelling approach of socioeconomic inequalities, but the chapter will consist of an exploration of both socioeconomic and gender inequalities in cause-specific childhood mortality.

5. 2 The secular decline in early childhood mortality

Early childhood mortality rates (ECMR) started to decline in some European regions from the 1840s and 1850s onwards, and by the 1870s most European countries began to follow this trend.[344] However, determining the start of the decline in ECMR also depends on how one deals with a previous upswing in childhood mortality followed by a decline a few decades later. The rise in early childhood mortality in the middle of the nineteenth century has been found in several European countries. [345] Because of this rise in mortality, Woods argues in the case of England that the true secular decline in ECMR

342 Hannaliis Jaadla and Alice Reid, 'The Geography of Early Childhood Mortality in England and Wales, 1881–1911,' *Demographic Research* 37/58 (2017).

343 Graham Mooney, 'Shifting Sex Differentials in Mortality During Urban Epidemiological Transition: The Case of Victorian London,' *International Journal of Population Geography* 8, no. 1 (2002), Peter Aaby, 'Are Men Weaker or Do Their Sisters Talk Too Much? Sex Differences in Childhood Mortality and the Construction of 'Biological' Differences,' in *The Methods and Uses of Atnhropological Demography*, ed. Alaka Malwade Basu and Peter Aaby (Oxford: Clarendon Press, 1998). United Nations Department of Economic and Social Affairs Population Division, *Too Young to Die: Genes or Gender?* (New York: United Nations, 1998).

344 Romola J. Davenport, 'Urbanization and Mortality in Britain, C. 1800–50,' *The Economic History Review* 73, no. 2 (2020), Woods, *The Demography of Victorian England and Wales*, Andrew Hinde and Bernard Harris, 'Mortality Decline by Cause in Urban and Rural England and Wales, 1851–1910,' *The History of the Family* 24, no. 2 (2019), Woods and Shelton, *An Atlas of Victorian Mortality*.

345 Davenport, 'Urbanization and Mortality in Britain,' 19-22.

only started around 1900, simultaneously with IMR, since the apparent decline of the 1860s in England was only a result of the earlier upswing in ECMR.[346]

Whereas in some areas infant mortality declined from the 1880s onwards, in some other countries this trend only started after 1900.[347] Simultaneous declines in IMR and ECMR may not come as a surprise, since their decline can be influenced by similar determinants to a certain extent. For England, there was a correlation between infant mortality rates and early childhood mortality rates, although with geographically differing ratios. For the Netherlands, Wolleswinkel-van den Bosch has found correlations between both the levels of infant mortality and early childhood mortality, as well as for the decline in both age-specific mortality categories.[348]

Despite the correlation between ECMR and IMR, regional variety in early childhood mortality could be caused by factors influencing only ECMR, and not IMR. Jaadla and Reid studied the strong regional variety, independent of infant mortality, in early childhood mortality in Britain. They found that the widening gap between ECMR and IMR during the final decades of the nineteenth century was caused by the stronger influence of the local disease environment on ECMR.[349] Thus, when factors influencing mortality changed for young children, yet not for infants, the 1870s may actually be regarded as the start of mortality decline among young children.

The related factor of the disease environment has not only influenced the decline in ECMR after the middle of the nineteenth century, but it has also been held accountable for the initial upswing in early childhood mortality at the start of the century. Szreter and Mooney argued that the increase in mortality was caused by a lesser interest in the smallpox vaccine in the early nineteenth century in large urban centres, causing once more a rise in smallpox mortality rates.[350] This is part of their wider argument, that in the 1830s and 1840s living conditions deteriorated in the British urban environment due to industrialisation.[351] In contrast, Davenport has recently argued that both the increase in the early nineteenth century and the decline from 1870 onwards in ECMR were caused by rather independent changes in the virulence of scarlet fever.[352] These changes in the virulence of scarlet fever have been established before.[353] Davenport argues that similar trends were recognisable in many regions of the European continent.[354] For the Netherlands, these patterns can be found in the provinces of Zeeland and Utrecht; there was both an increase in ECMR during the early decades of the nineteenth century and a

346 Davenport, 'Urbanization and Mortality in Britain'; Szreter and Mooney, 'Urbanization, Mortality, and the Standard of Living Debate'; Woods, *The Demography of Victorian England and Wales*; E.A. Wrigley et al., *English Population History from Family Reconstitution 1580-1837* (Cambridge: Cambridge University Press, 1997), 261.

347 Woods, Watterson, and Woodward, 'The Causes of Rapid Infant Mortality Decline in England and Wales, 1861–1921 Part I.'

348 Wolleswinkel-van den Bosch, 'The Epidemiological Transition in the Netherlands,' 157.

349 Jaadla and Reid, 'The Geography of Early Childhood Mortality in England and Wales, 1881–1911.'

350 Szreter and Mooney, 'Urbanization, Mortality, and the Standard of Living Debate,' 102.

351 Ibid., 110.

352 Romola J. Davenport, 'Urbanization and Mortality in Britain.'

353 Anne Hardy, *The Epidemic Streets. Infectious Disease and the Rise of Preventive Medicine, 1856-1900* (Oxford: Clarendon Press, 1993).

354 Davenport, 'Urbanization and Mortality in Britain.'

decline from the 1850s and 1860 onwards, respectively. In Friesland, the overall ECMR was lower and this pattern was less pronounced, yet still present.[355]

Whether the diminished virulence of scarlet fever on its own could bring about a large decline in early childhood mortality can be questioned. It may be that scarlet fever receded after the 1870s, only to make room for other infectious diseases to take over. The diseases taking over may in turn have reversed a part of the decline instigated by the decreased virulence of scarlet fever. As such, scarlet fever contributed to the decline, but was not the single cause of the decline in ECMR. Understanding the decline in ECMR will therefore require an understanding of the co-dependent changes in the disease environment, since the decline in mortality due to a specific cause may have influenced the disease environment in more than one way.

5.3 The disease environment

The large variety of childhood diseases which could put the child in jeopardy is one of the most interesting features of historical early childhood mortality. Some categories of disease were more hazardous than others, partly depending on the age of the child. In the younger age group of infants, the main hazard were gastrointestinal diseases, which could still be a threat in the second year of life at age one. Infants could, however, be relatively shielded from airborne infectious diseases through their mother's immune system during their first months of life.[356] Once this protection waned, the child was most vulnerable to airborne infections, until it had gained immunity by surviving the disease itself. The highest childhood mortality rates, excluding infant mortality, therefore occurred in the years between one and five, predominantly from these childhood airborne infections. This category alone still constituted a wide variety of possible diseases.

In order to fully grasp the importance of this variety, a further exploration and clear definition of the concept of the disease environment is indispensable. Although within the field of historical demography the term reappears in many studies, which also attribute quite some importance to it, a clear and comprehensive definition often remains unmentioned. Disease environment could simply mean the set of diseases present in a given population, but it appears to entail more than just that. The concept of *pathocenosis*, coined in 1966 by the historian Mirko Grmek,[357] provides a solution to the understanding of the idea of a disease environment. Grmek introduced the term *pathocenosis* to promote the synthetic – instead of analytical – study of diseases. A synthetic approach takes into account the entire spectrum of diseases in the study of the history of medicine, whereas an analytical approach would only focus on an individual disease. The definition of *pathocenosis* according to Grmek was: '…the ensemble of pathological states present in a specific population at a given moment in space and

355 Van Poppel, Jonker, and Mandemakers, 'Differential Infant and Child Mortality in Three Dutch Regions, 1812-1909,' 285.

356 A. Katharina Simon, Georg A. Hollander, and Andrew McMichael, 'Evolution of the Immune System in Humans from Infancy to Old Age,' *Proceedings of the Royal Society B: Biological Sciences* 282, no. 1821 (2015): 20143085.

357 Pierre-Olivier Méthot, 'Introduction: Mirko Grmek's Investigative Pathway,' in Mirko D. Grmek, *Pathological Realities Essays on Disease, Experiments, and History, ed.* Pierre-Olivier Méthot (New York: Fordham University Press, 2019): 19.

time'.[358] Within this ensemble, the frequency and distribution of specific diseases were dependent on each other, as well as on endogenous and ecological factors, whilst not being stationary but subject to change over time. Certain diseases could either behave in symbiosis to each other, antagonistically, or indifferently;[359] thus creating very particular disease environments in different settings. More recent epidemiological research indeed suggests that epidemiological patterns of individual diseases change when they interfere with other diseases in a given population, especially when those infections have fatal consequences.[360] Later in his career, Grmek articulated the importance of ecological or environmental features in the pathocenosis when he described the concept of emerging infectious and other diseases. The emergence of an apparently new disease depends partly on new ecological developments, where an already pre-existing germ may be given the opportunity to become hazardous enough to be noticed, due to human-driven developments.[361]

In that respect, the more tangible phrasing of disease environment means more than just the diseases that were present in a particular population or how this was related to environmental issues. The disease environment also entails the interplay between the different diseases and how changes in either biology or environment influenced the hazardous nature of a particular disease, which in turn influenced the range of diseases an individual was exposed to. Diseases should not be understood as individual agents only within the disease environment itself, but we should also take the interconnectedness of diseases into account in resulting mortality. Co-morbidity is one such phenomenon that stresses the importance of disease relationships. Measles for example, are not very lethal on their own.[362] Yet the devil is in the details, since death is often caused by the complications due to the immune suppression caused by the measles virus.[363] The accompanying pneumonia, whooping cough or meningitis, to name a few, cause the measles to be a hazardous disease for children after all. Although smallpox was already receding during the research period, a similar mechanism is observed for smallpox.[364] During the stage of recovery from a smallpox infection, the respiratory organs are highly susceptible to a second bacterial infection. Bronchopneumonia was therefore a common complication of smallpox. The nineteenth-century Health Inspectors were not oblivious to the relation between high smallpox mortality rates, and other

358 Grmek, *Pathological Realities*, 33.

359 Ibid.

360 P. Rohani et al., 'Ecological Interference between Fatal Diseases,' *Nature* 422, no. 6934 (2003).

361 Grmek, *Pathological Realities*, 47-48. An illustrative example is the emergence of Legionnaires disease, in which the microbe could become very lethal due to ideal conditions to multiply in newly installed air conditioning systems.

362 Hardy, *The Epidemic Streets.*

363 Laksono, Brigitta M., Rory D. de Vries, R. Joyce Verburgh, Eline G. Visser, Alwin de Jong, Pieter L. A. Fraaij, Wilhemina L. M. Ruijs, *et al.* 'Studies into the Mechanism of Measles-Associated Immune Suppression During a Measles Outbreak in the Netherlands,' *Nature Communications* 9, no. 1 (2018/11/23 2018): 4944.
Noori Navideh, and Pejman Rohani, 'Quantifying the Consequences of Measles-Induced Immune Modulation for Whooping Cough Epidemiology,' *Philosophical Transactions of the Royal Society B: Biological Sciences* 374, no. 1775 (2019): 20180270.

364 Willibrord J. M. J. Rutten, '*De Vreselijkste Aller Harpijen : Pokkenepidemieën en Pokkenbestrijding in Nederland in de Achttiende en Negentiende Eeuw: Een Sociaal-Historische en Historisch-Demografische Studie* (Wageningen University, 1997): 399.

diseases. They observed a relation between smallpox and cholera. While a part of the simultaneous higher occurrence of infectious diseases may be explained from a common set of determinants affecting the susceptibility to infectious diseases as a whole, Rutten finds such strong statistically significant correlations between smallpox mortality and high overall mortality rates, that part of this correlation is likely to be explained from an overall deplorable state of health, and thus from co-morbidity.[365] Child mortality was, therefore, often the consequence of a sort of symbiosis of diseases, instead of being caused by a single agent.

Whereas co-morbidity is one example of the interconnectedness of diseases, infections could also strain the young body subsequently instead of simultaneously. Even when a child recovered from a potentially lethal disease, suffering from it could already have strained the body severely. This mechanism is often used for explanations of higher mortality in adulthood, known as the scarring mechanism.[366] However, if a child had endured multiple diseases successively, the consequences of scarring may already occur in childhood, causing death even from a relatively mild infection such as, for example, pneumonia. Thus, even when a particular disease became less lethal, it could still have contributed to the overall burden of disease and subsequently high death rates. In childhood mortality, the final cause of death was most likely only the last in a longer chain of childhood diseases, and simply the straw that broke the camel's back. This is illustrated by Aaby's findings for modern-day underdeveloped countries; with the introduction of the measles vaccine the amount of deaths that had been prevented exceeded the number of deaths ascribed to measles prior to the vaccinations.[367] Both co-morbidity or scarring could be an explanation for this mechanism, as in both cases measles had contributed to more deaths than strictly registered. The question arises whether a specific disease's decline in mortality alone, but not in morbidity, can even be effective enough to influence the total burden of disease and cause all-cause mortality to decline.

Because of the complicated mechanisms operating in the disease environment, quantifying the explicit gains in lives when a particular disease receded proves to be complicated. Reduction in fatality does not necessarily mean a smaller burden of disease if morbidity remained high. Even if morbidity had declined as well, the disease could easily be replaced by one of the other rivalling diseases eager to infect some of the children that managed to escape the devastating consequences of the first disease. Still, over the course of the eighteenth and nineteenth century, the replacement of diseases followed a continuous pattern in which the most prevalent diseases were replaced by the prevalence of less lethal diseases,[368] which did contribute to the decline in overall mortality.

365 Ibid., 399-400.

366 Luciana Quaranta, *Scarred for Life: How Conditions in Early Life Affect Socioeconomic Status, Reproduction and Mortality in Southern Sweden, 1813-1968* (Lund: Media-Tryck, 2013), 5.

367 Peter Aaby, 'Lessons for the Past: Third World Evidence and the Reinterpretation of Developed World Mortality Declines,' *Health Transition Review* 2 (1992): 155-83.

368 Romola J. Davenport, 'The First Stages of the Mortality Transition in England. A Perspective from Evolutionary Biology,' Working paper (2017).

Davenport explained how this trend in declining lethality was related to the introduction of interventions and how these interventions needed to be increasingly severe in order to be effective for the less lethal diseases. Fundamental to this idea is the assumption that, within pathogens, there is a relation between their mode of transmission and their virulence, with its eventual lethality. Pathogens which can survive outside of a human host, for example in other animal hosts such as fleas, or which can 'sit and wait' on other objects, do not depend on the human host for transmission. They can therefore afford to be very lethal and kill their hosts in many cases, often doing so very quickly. Examples of these diseases are the bubonic plague, smallpox and cholera. On the other side of the spectrum are pathogens which cannot survive outside of a human host and for which the transmission route is via humans only. These pathogens are dependent for their own survival on a sufficient survival of their hosts for transmission. Examples are measles, whooping cough, diphtheria and scarlet fever. These typical childhood diseases are thus highly contagious and affect many, but are less lethal and have more survivors than the diseases which can exist outside of a human host. Consequently, they are also harder to eradicate because they spread so easily. A weak quarantine policy may have been effective against the very lethal diseases, while stronger measures proved to be necessary to prevent the spread of the typical childhood diseases. This is why, over the course of the eighteenth and nineteenth century, the incidence of very lethal diseases was replaced by a higher incidence of the milder, but easier-spreading typical childhood diseases.[369]

The mechanism of diseases replacing each other further complicates making estimations of the effect of the decline of one particular disease on the mortality decline as a whole. This is once more a reminder that the decline of one particular disease should not be analysed individually, but only by taking the broader disease environment into account.[370] The decline of a particular disease has both direct and indirect effects on total mortality decline. The direct effect of the eradication of a disease is a decline in mortality, whereas the indirect effect is an increased availability of hosts in the population for other diseases which can reverse part of the direct effect of mortality decline.[371] Thus, the rise in the incidence of other diseases can be an indirect effect of the eradication of one. In the long run, the direct effects of the eradication of a disease should outbalance the indirect effects of a greater availability of new hosts for other diseases, especially when incorporating Davenport's argument that highly lethal diseases are gradually replaced by less lethal diseases. After all, if the diseases causing the indirect effect of partially or fully reversing mortality decline are less lethal compared to the disease that has been eradicated and morbidity does not cause too much scarring, total mortality should decline.

In this fashion, Hinde and Harris have computed the contribution of individual diseases to the increase in life expectancy at birth in late nineteenth century Britain. They concluded that in the period from the 1850s to 1901-1910 in Britain, life expectancy

369 Ibid.

370 Hiram Beltrán-Sánchez, Samuel Preston, and Vladimir Canudas-Romo, 'An Integrated Approach to Cause-of-Death Analysis: Cause-Deleted Life Tables and Decompositions of Life Expectancy,' *Demographic Research* 19, no. 35 (2008): 1323-50.

371 See also Hinde and Harris, 'Mortality Decline by Cause in Urban and Rural England and Wales, 1851–1910.'

rose with nine years. The increase was predominantly caused by the decline in water, food and airborne diseases. Pulmonary tuberculosis, waterborne diseases, scarlet fever and diseases of the lung (which included bronchitis, pneumonia and influenza) were the main responsible diseases within these groups.[372] These results are more or less in line with Woods' conclusions for this period,[373] although Hinde and Harris found the childhood diseases (measles, scarlet fever, whooping cough) playing a larger role in the period from the 1860s to the 1890s, and smaller effects in this period from adult causes of death like cancer and violence.[374] The impact of the decline in scarlet fever was strongest in the shorter period from 1861-1870 to 1891-1900, and after 1900 the effect of the decline in scarlet fever was much smaller.[375] This is in line with other findings from Woods and Davenport, that the major childhood disease scarlet fever declined rapidly after the 1870s.

Wolleswinkel-van den Bosch has calculated the contributions of disease groups and individual diseases to the total mortality decline for the Netherlands in the period 1875-1970. The greatest all-cause, all-age mortality decline took place between 1901 and 1931. Yet an already substantial mortality decline occurred from 1875 to 1901, which was only slightly smaller compared to the decline in the following period.[376] Infectious childhood mortality (ages 1-4 and 5-14) declined most rapidly in the final decades of the nineteenth century.[377] In this period, the other infectious diseases were the largest contributor of all the infectious diseases to the total mortality decline, with 18.42 percent. At the level of individual diseases, it was mainly diphtheria, whooping cough and scarlet fever that declined in 1875-1901, while this period still faced increases in mortality from acute digestive disorders and measles.[378] In line with the findings of Hinde and Harris, mainly the childhood diseases declined in this period.

Although Hinde and Harris, and Wolleswinkel-van den Bosch did not stratify according to age in their analyses of the contributions of individual diseases to mortality decline, most of the diseases they identified as major contributors to the mortality decline in the final decades of the nineteenth century were predominant in early childhood. Yet how the disease environment exactly changed for young children in this period remains unclear. For Maastricht, however, it is possible to analyse how the disease environment for young children changed, in order to analyse which groups of children gained a first advantage thereafter.

372 Ibid., 389.

373 Woods, *The Demography of Victorian England and Wales*, 350-1.

374 Hinde and Harris, 'Mortality Decline by Cause in Urban and Rural England and Wales, 1851–1910,' 389.

375 Ibid., 392.

376 Wolleswinkel-van den Bosch, 'The Epidemiological Transition in the Netherlands,' 106-111.

377 Ibid., 107-11.

378 Ibid.

5. 4 Early childhood mortality decline in Maastricht

In the Netherlands, early childhood mortality was on average declining from the early 1870s onwards (Figure 5.1).[379] The levels of ECMR were highest in urban areas, although the urban-rural differences were smaller in the period 1895/99 than in 1875/79.[380] Compared to Amsterdam, Maastricht had a high ECMR in 1875/79, but had caught up to the capital and to the average ECMR for sixteen large Dutch cities in 1895/99.[381] In the following section, I will analyse the decline in ECM in Maastricht, and determine which diseases and/or disease categories were driving the early childhood mortality decline. Figure 5.2 shows the ECMR for both Maastricht and all the other localities of the province of Limburg from 1812 through 1910, based on the mortality rate by birth cohort.[382] Throughout the period, Maastricht was in a worse position compared to the average early childhood mortality rates of the other Limburg localities. In fact, early childhood mortality appears to have increased during the first half of the nineteenth century.

A subset of all young children having died in Maastricht was created again from the *Maastricht Death and Disease Database*. In the research period, a total of 7,357 young children found an early death, of which 3698 were boys and 3659 were girls (Table 5.1). For a first overview of the causes of death, I used seven categories of causes of death: water and foodborne infectious diseases, airborne infectious diseases, other infectious diseases, non-infectious diseases, non-natural causes of death, ill-specified causes of death, and unknown causes of death. Some categories were collapsed to ensure enough data power for further analysis. In this latter categorisation, the non-infectious diseases, non-natural causes of death, ill-specified causes of death and unknown causes are taken together in the category labelled 'all non-infectious diseases'. In order to analyse the relative contribution of specific causes of death and their interconnectedness within the disease environment, the most important childhood diseases are taken separately as well. In line with most literature these diseases are: (gastro)enteritis, cholera, convulsions, scarlet fever, measles, diphtheria, whooping cough, tuberculosis, pneumonia and bronchitis.[383]

379 J.H. de Haas, *Kindersterfte in Nederland Atlas* (Assen: Van Gorcum, 1956).

380 Wolleswinkel-van den Bosch, 'The Epidemiological Transition in the Netherlands,' 155.

381 Ibid.

382 The data for this figure is derived from the Links dataset: Kees Mandemakers and Fons Laan, 'Linked Civil Registry, Limburg, Release 2018_01,' (IISH Data Collection, 2022). Because of the different nature of the dataset, ECMR is calculated by birth cohort. To calculate this ECMR the number of children born in for example 1812 who made it past their first year of life, but passed away before the age of 5, were divided by the number of live-born, minus the deceased infants. This figure can thus provide an overview of the differences between Maastricht and the rest of the province, and of the general trends. However, the levels of the mortality rates cannot be compared one on one to the figures derived from the Maastricht Death and Disease Database.

383 For example: Hinde and Harris, 'Mortality Decline by Cause in Urban and Rural England and Wales, 1851–1910'; Wolleswinkel-van den Bosch, 'The Epidemiological Transition in the Netherlands'; Woods and Shelton, *An Atlas of Victorian Mortality*.

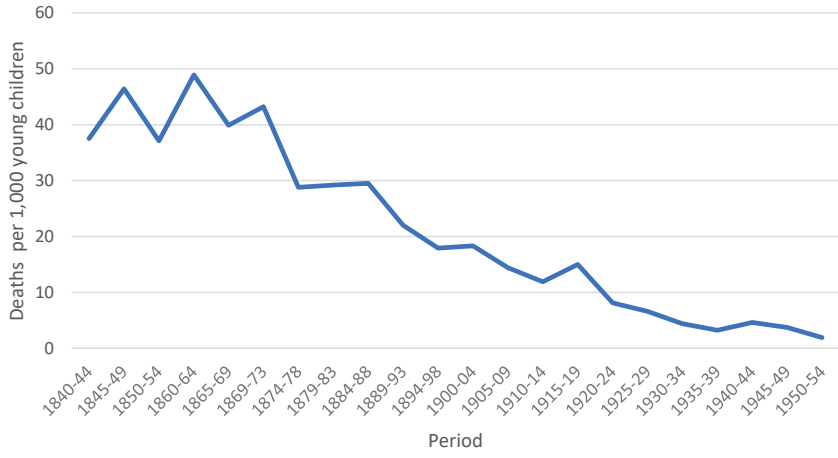


Figure 5.1 Early childhood mortality in the Netherlands, 1840-1954

Source: J.H. De Haas, Kindersterfte in Nederland Atlas, (1956).

Table 5.1 Dataset characteristics, young children aged 1-4

Variable		**N**
Gender	*Male*	3,698
	Female	3,659
Socioeconomic class	*Elite*	134
	Middle class	820
	Skilled workers	3254
	Farmers	113
	Unskilled workers	2458
	Unknown	578
Age	*One year*	3,916
	Two year	1,697
	Three year	1,017
	Four year	727
Period in analysis	*1864-1869*	894
	1870-1879	1,420
	1880-1889	999
	1890-1899	1264
	1900-1909	1059
	1910-1919	825
	1920-1929	413
	1930-1939	230
	1940-1945	147
	1946-1955	106
Total		**7357**

The population at risk used for all the analyses is based on the decennial censuses.[384] These contain age categories for either each early childhood year, or the group from one through four. The population for the years between the decennial censuses is based on estimations derived from the adjacent census years. The period between 1910 and 1920 were more complicated to estimate, since the new census was held in 1921, right after the annexation of some of the small municipalities at the borders of Maastricht. For these years, the population estimates were checked with a second computation,[385] to make the results more robust. After calculating the mortality rates based on the two different estimates, the differences only occurred at the decimal level, which ensures the reliability of the rates at least so far as at the integer level. Finally, the mid-year population based on these calculations was used for the computation of the total early childhood mortality rate (ECMR) and the cause and sex-specific early childhood mortality rates.

Figure 5.3 presents the decline in overall ECMR for the period between 1864 and 1955 for each year separately, while Figure 5.4 is more illustrative of the general trends as it shows the quinquennial ECMR. As could be expected, Figure 5.3 is characterised by regular spikes, often caused by epidemic outbreaks of diseases.[386] The occurrence of regular disease outbreaks lasted until the middle of the 1910s. However, as the quinquennial data show, overall ECMR was already declining from the 1880s onwards. The decline in ECMR in Maastricht followed European-wide patterns. In many European regions this decline started in the 1870s; in Maastricht a subtle decline appears to have started from the mid-1860s, and accelerated during the 1880s until 1890. After 1890, ECMR increased for a short period, to return again to the level of 1885/89 after one five-year period in 1895/99. During the following two decades, ECMR remained quite stable, to only start declining again from 1915/19 onwards. The slope of the decline diminished after 1920/24, although the declining trend continued until the Second World War,

384 See Chapter 2.

385 The first computation being equal to the estimation of other years, taking the population at the beginning and the end of the period and increasing equally each year. For the population of young children at the start of 1920, when the inhabitants of the annexed municipalities were not yet included, I used the (known) whole population and took 9% (in 1910 the young children made up 9% of the total population, in 1921 the percentage is 10%) as representing the young children. Then I estimated the population in each year in between with a fixed number of increase. However, this computation may hide decline in specific years in this period. The second computation is based on only the population of young children in 1910. From this number I subtracted the deaths between one and five, after which I multiplied it with a factor 0,78, representing the number of children turning five the next year and not belonging to the category of young children anymore. The factor 0,78 gives the best estimation of the final population in 1920 (instead of for example 0,75). Finally, to this number the amount of surviving infants in that year are added, resulting in the population estimation for the next year. I repeated this step for each year. In the end, method two gave a more precise estimation, so that is the one I have used in all the models. However, the differences with method one only showed at the decimal level. Equation of method 2: Population year x= (Population year x-1- deaths 1-4 year x-1)*0,78 +(Births year x-1-infant deaths year x-1).

386 See Annex V for an overview of the epidemic outbreaks of diseases. The definition of an epidemic outbreak is quite straightforward, as it constitutes the occurrence of more cases of a disease than expected in a given area of among a specific group of people over a particular time period. Yet quantifying what the threshold is for an increase in lethal incidence of a disease to turn it into an epidemic is not as straightforward and depends on the disease, time and place where it occurs. Cause-specific mortality spikes were considered to be an epidemic outbreak when the cause specific mortality rate for that disease was larger than three standard deviations from the mean mortality rate over the period. Since some diseases already receded before the end of the research period, some overestimation of epidemics can occur.

which itself caused a minor rise in ECMR. After the war, the levels prior to the war were reached again and slowly declined.

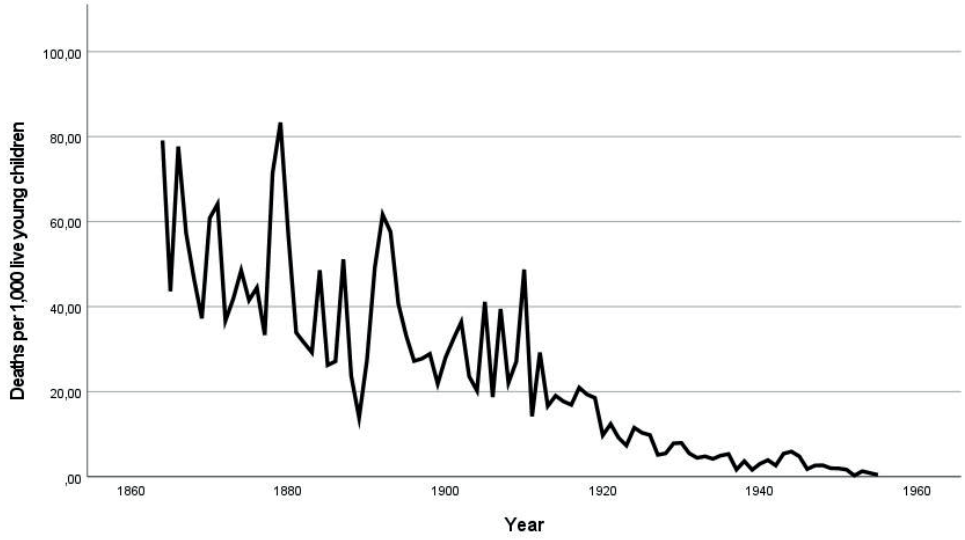


Figure 5.3 Early childhood mortality rate, Maastricht 1864-1955

Source: Maastricht Death and Disease Database, Dutch censuses

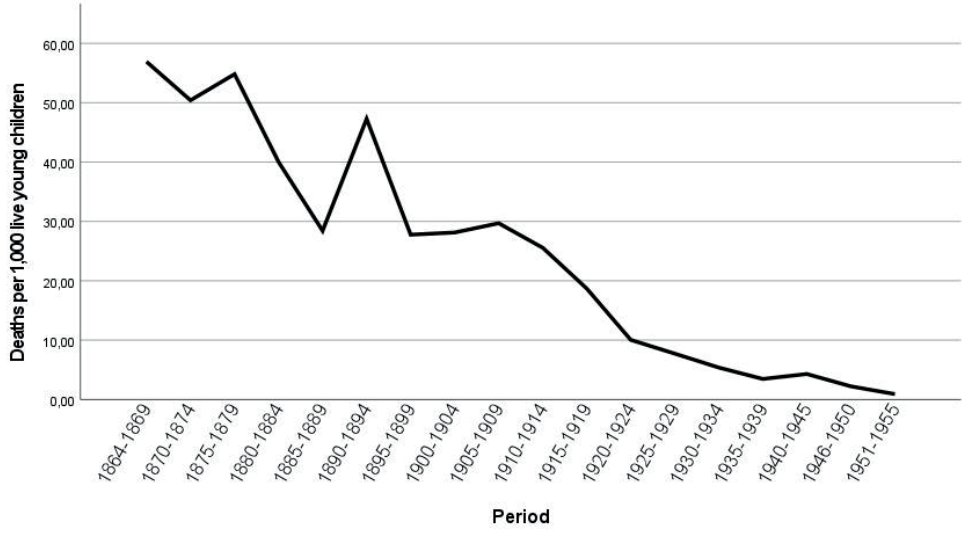


Figure 5.4 Quinquennial early childhood mortality rate, Maastricht 1864-1955

Source: Maastricht Death and Disease Database, Dutch censuses

Figure 5.5a shows which of the seven categories of causes of death were responsible for the ECMR, and which disease categories caused the epidemic outbreaks. In addition to that, Figure 5.5b specifies the actual mortality decline in the four larger disease categories for each decade. Airborne infectious diseases make up for a large share of the ECMR throughout the entire period, as they were responsible for many spikes in mortality. Airborne infectious diseases were, however, predominant, also in lower-mortality years. Both characteristically endemic (constantly prevalent) and epidemic (occurring once every couple of years) diseases contributed to the burden of disease caused by airborne infectious diseases (Annex V). Water and foodborne infectious diseases contributed substantially to mortality at the beginning of the period as well. Among young children a decline in water and foodborne diseases already occurred from the 1860s until the 1880s. Water and foodborne infections remained stable thereafter until 1900, which is remarkable since infant mortality due to water and foodborne disease actually rose in the same period. After 1900, the water and foodborne infectious diseases declined further to a point where they lose their importance completely after 1920.

The recurring spikes in early childhood mortality were caused by a variety of epidemic outbreaks. Since these epidemic outbreaks had a substantial impact on the health of young children, taking a closer look at them may help to understand the changes that occurred in the disease environment of young children during the research period. An epidemic outbreak ensues when there are more cases of a disease than would be expected based on the normal occurrence of the disease, and it is geographically and time-bound. Conversely, we speak of an endemic disease when a disease is constantly prevalent within a population in a certain geographical area. Although the definitions appear to be quite straightforward, quantifying a threshold of what constituted an epidemic outbreak in retrospect is, however, very complex. Nowadays, epidemiologists are mainly interested in establishing which number of cases will cause an outbreak becoming hazardous in terms of spreading, often referred to as the reproduction rate.[387] Yet what the crucial threshold is for calling a disease an epidemic one is highly dependent on the disease itself. For this study, mortality rates which were three standard deviations higher than the mean mortality rate over the entire period were regarded as epidemic outbreaks, since this would indicate a mortality rate exceeding what is statistically expected to be normal (Annex V). In the water and foodborne category, only cholera caused an epidemic outbreak in 1866, while gastroenteritis was rather endemic and was constantly prevalent until 1920. The majority of the mortality spikes were the result of epidemic outbreaks of airborne infectious diseases.

Scarlet fever struck the city in 1864, 1871 and 1879 with high mortality rates, after which its lethality receded as it had done in other European countries.[388] Morbidity had not yet waned completely, since the annual reports of the municipality still reported epidemics of scarlet fever after these three massive outbreaks.[389] It appears the epidemic outbreaks of scarlet fever generally became less lethal, although they could have

387 Erik Volz and Lauren Ancel Meyers, 'Epidemic thresholds in dynamic contact networks,' *Journal of the Royal Society Interface* 6 (2008).

388 Davenport, 'Urbanization and Mortality in Britain'; Anne Hardy, *The Epidemic Streets.*

389 Gemeente Maastricht, *Uitvoerig en Beredeneerd Verslag van den Toestand der Gemeente Maastricht*; Gemeente Maastricht, *Verslag van den Toestand der Gemeente over het Jaar*.

continued to partially contribute to the total disease burden. Diphtheria was slightly more endemic than scarlet fever, with some regular lethal cases every year. In 1892/1893, a massive diphtheria outbreak occurred, which caused the high spike in mortality. The municipality felt the need to take severe measures to fight this outbreak, and even closed the schools for a period of time.[390] Finally, measles caused some mortality peaks in the early 1900s, in 1907, 1910 and 1912. Measles mortality patterns resembled scarlet fever mortality patterns more than those of diphtheria. Large measles outbreaks occurred only occasionally, in the other years almost all young children escaped the lethal consequences of measles. Measles behaved as an epidemic disease when taking the mortality rates into account, which could have been the result of Maastricht's population being too small to sustain the disease constantly. It is estimated that urban populations of 250,000 people or more are needed to ensure the survival of the infective chain of measles. With only a total population of nearly 55,000 in 1921, after Maastricht's annexation of adjacent municipalities, measles needed to be 'imported' once in a while, since it could not sustain itself within this population.[391]

However, in some of the in-between years, there were still outbreaks of measles, only not highly lethal. The municipality reports even mention measles in some years with very low or even no measles mortality among young children. Measles in these years must have been either lethal in other age groups, or had no lethal consequences at all. It is remarkable that measles appears to have become more lethal after the turn of the century. While measles clearly appeared to be present in the nineteenth century according to the reports of the health committee, the cause-specific mortality rates of measles only increased after the turn of the century, causing measles epidemics.

390 Gemeente Maastricht, *Verslag van den Toestand der Gemeente over het Jaar 1893* (Maastricht: Gemeente Maastricht, 1894).

391 Andrew David Cliff, Peter Haggett, and Matthew Smallman-Raynor, *Measles: A Historical Geography of a Major Human Viral Disease from Global Expansion to Local Retreat, 1840-1990* (Oxford: Blackwell, 1993). Ministerie van Binnenlandse Zaken, *Statistiek Van Den Loop Der Bevolking Van Nederland Over.*

Figure 5.5a Yearly mortality rates

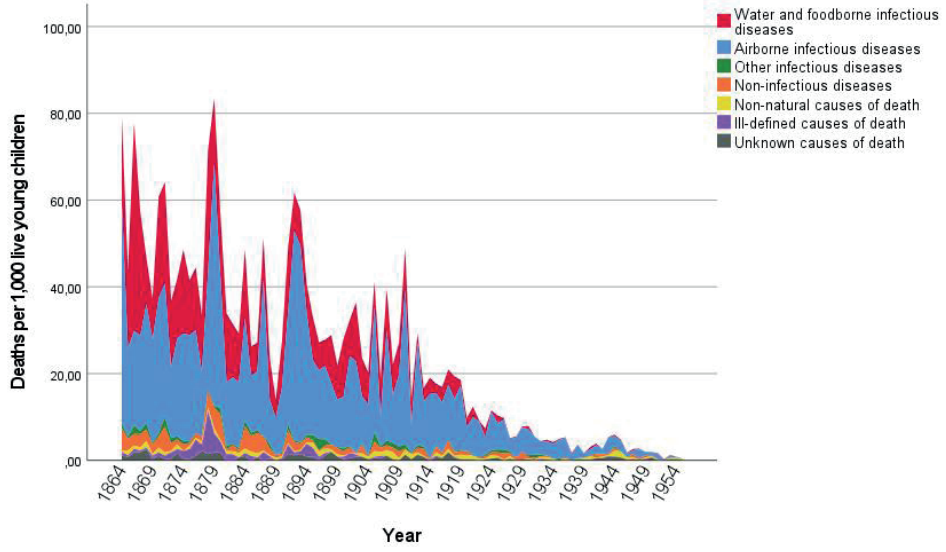
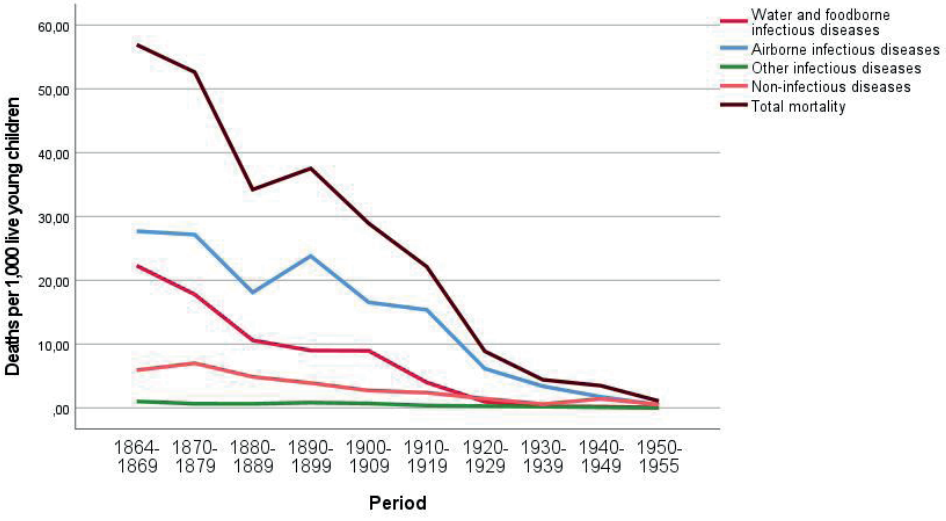


Figure 5.5 b Decennial mortality rates



Figures 5.5a and 5.5b Category-specific early childhood mortality rates, Maastricht 1864-1955

Source: Maastricht Death and Disease Database, Dutch censuses

Figure 5.6a Per cent contribution of cause-of-death categories

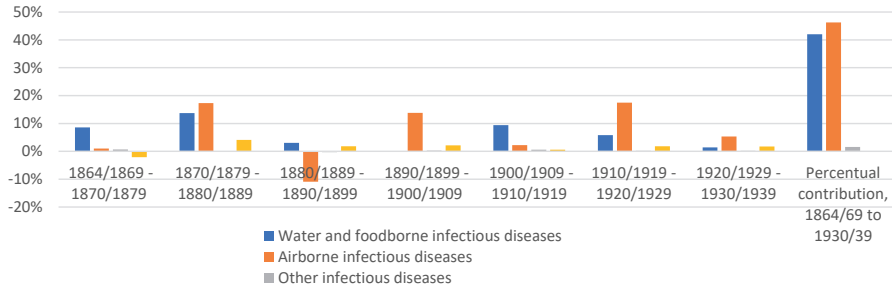


Figure 5.6b Per cent contribution of individual water and foodborne infectious diseases

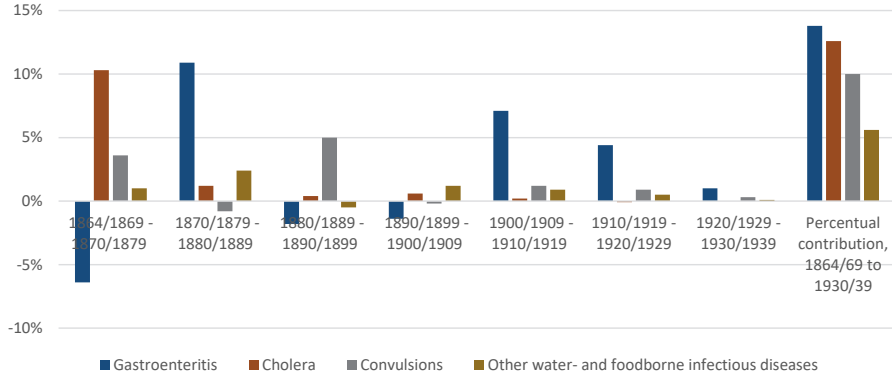
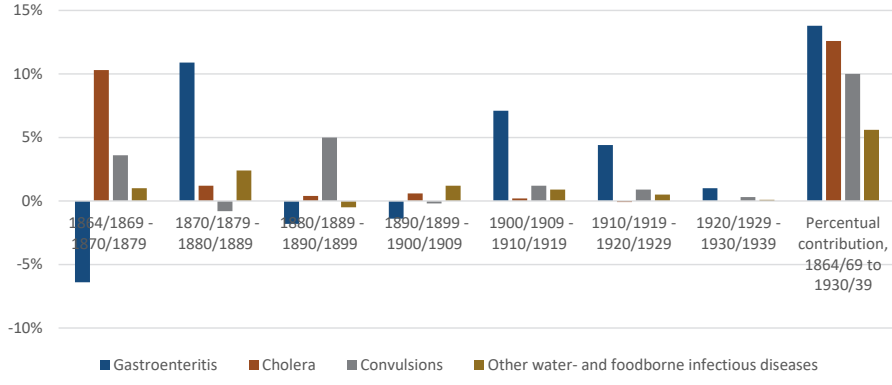


Figure 5.6c Per cent contribution of individual airborne infectious diseases



Figures 5.6a, 5.6b and 5.6c Per cent contribution of causes of death to total early childhood mortality, 1864-1939

Source: Maastricht Death and Disease Database

The question arises which mechanisms were at the root of the overall decline in early childhood mortality, and how the decline of one disease interacted with the wider disease environment. Were diseases replaced by other, less lethal diseases, which in the end caused overall mortality to decline? Or was the final breakthrough a flipping point,

where children became robust enough to endure and survive the entire chain of diseases? Following from the specific epidemic outbreaks in Maastricht is at least the idea of the airborne diseases replacing each other to a certain extent. At the beginning of the research period, it was mainly scarlet fever which caused high mortality peaks among the children. Once this disease had receded, diphtheria was an important contributor to mortality and finally measles caused some more lethal outbreaks in the early twentieth century. However, the fact that mortality from one cause of death declined, does not immediately imply that its contribution to the overall burden of disease had disappeared. A child may not have died of scarlet fever anymore, but the child could still have been scarred by it, contributing to increasing mortality rates for other causes of death such as, for example, measles. In order to analyse the contribution of the decline of a certain disease to the overall mortality decline, we should preferably take both the direct and indirect effects into account, as clearly explained by Hinde and Harris,[392] and keep in mind that morbidity may have influenced mortality rates as well.

In the following analysis, I calculated the per cent contribution of the four separate disease categories, as well as of some individual diseases, by decade to the overall mortality decline from 1864 until 1939. After 1939, there was a small increase in ECMR during the Second World War, to return to its previous level afterwards. Since this is not part of the secular decline in ECMR, this analysis only runs until 1939, when the majority of the mortality decline as part of the health transition took place. Figures 5.6a-c represent the per cent contributions to the overall mortality decline of the four disease categories (Figure 5.6a), of the specific water and foodborne infectious diseases (Figure 5.6b) and the airborne infectious diseases (Figure 5.6c).[393]

In general, there was one distinct period in which both the per cent contribution of water and foodborne and airborne infectious diseases to overall mortality decline was the highest: From 1870/79 until 1880/89. Water and foodborne infectious diseases moreover contributed substantially to the decline from 1864/69 until 1870/79 and from 1900/09 until 1910/19. The second period of substantial airborne infectious disease decline came after that, from 1910/19 to 1920/29. The apparent decline of airborne infectious diseases between 1890/99 to 1900/09 is only a side effect of the diphtheria outbreak in 1892/1893 (see Figure 5.6c) and should therefore not be regarded as an important period of decline. The decline in the specific disease categories complimented each other, rather than that they counteracted each other. Either one category declined on its own without being counteracted by another, or they reinforced each other and declined simultaneously. At the level of individual diseases, a similar mechanism was in place, with only some exceptions, in the period from 1864/69 until 1870/79.

The responsible individual diseases for the decline of the water and foodborne category were mainly cholera and gastroenteritis. The decline in cholera after the cholera epidemic in 1866 was counteracted by a small increase in gastroenteritis (Figure 5.6b). However, in the following period, the decline in gastroenteritis ensured a steady decline in water and foodborne infectious diseases. After the turn of the century, another decline in gastroenteritis set in, albeit a bit smaller. The declines in gastroenteritis were not

392 Hinde and Harris, 'Mortality Decline by Cause in Urban and Rural England and Wales, 1851–1910.'

393 Annex V contains the underlying tables.

counteracted by any other disease from this disease category, thus truly contributing to the mortality decline.

The picture for airborne infectious diseases is more complex. While a decline emerged in the first decade in diphtheria and scarlet fever, this was almost completely counteracted by increases in tuberculosis, pneumonia and whooping cough. The two most remarkable declines in airborne infectious diseases appeared in the periods from 1870/79 until 1880/89 and 1910/19 until 1920/29. In both periods, a characteristically epidemic disease declined, combined with the decline of some more endemically occurring airborne diseases. Scarlet fever led the first decline, while measles led the second. Both diseases were accompanied by a decline in tuberculosis, and for scarlet fever other airborne infectious diseases declined simultaneously, while measles was accompanied by a decline in pneumonia (which could be a complication of measles as well). Scarlet fever and measles do not seem to have left a gap for other diseases to fill, it actually appears that their decline indeed lessened the total burden of disease.[394]

Preliminary conclusions hint at direct contributions to mortality decline from cholera, gastroenteritis, scarlet fever and measles, where the latter two declining diseases were accompanied by a decline of other, more endemic, airborne diseases. The decline in scarlet fever around the 1860s and 1870s is reflected in Europe as well, and has been attributed to a decline in virulence of the disease.[395] The fact that other airborne diseases (and not to forget, water and foodborne diseases as well) declined simultaneously, can be explained by the fact that scarlet fever was not adding much to the total burden of disease anymore, causing children to be more robust in fighting other diseases. Whether the decline of measles, with the consequential decline in tuberculosis and pneumonia, can be explained by the same mechanism is less clear. Since mortality due to airborne infectious diseases declined for infants as well in this period, more generic explanations such as improved nutrition and housing conditions could explain the second decline in airborne infectious diseases as well.

At the beginning of the period, the cholera epidemic caused a spike in mortality. It is however debatable whether cholera itself truly contributed to the overall mortality decline. The decline in water and foodborne mortality after the 1860s may have been an artificial decline if levels of water and foodborne mortality were as high prior to the severe epidemic outbreak of cholera in 1866, as after this event. However, the fact that cholera epidemics had been more common during the middle of the nineteenth century whilst not returning to Maastricht afterwards, except for 1894 (and with only few fatalities among the young children), indicates that improvements had been made. Especially cholera serves as a good indicator of the water quality in cities.[396] By 1894, the piped water supply system was still underdeveloped in Maastricht, but changes had been made in waste disposal.[397] These may have been sufficient in improving the water quality and thus shielded children from cholera in the final cholera outbreak of 1894.

394 Which has been noticed by Aaby as well, after the measles vaccine was introduced in the global south. Aaby, 'Lessons for the Past'.

395 Davenport, 'Urbanization and Mortality in Britain'; Woods, *The Demography of Victorian England and Wales*.

396 Romola J. Davenport, Max Satchell, and Leigh Matthew William Shaw-Taylor, 'Cholera as a 'Sanitary Test' of British Cities, 1831–1866', *The History of the Family* 24, no. 2 (2019): 404-438.

397 Evers and Ubachs, *Voorkomen Is Beter Dan Genezen*, 15.

The remaining important individual contributor to the decline in ECMR is (gastro) enteritis. Although water and foodborne mortality for infants declined somewhat simultaneously with early childhood mortality in the 1910s, the young children already benefitted from a decline in this category in an earlier period. While infant mortality, mainly due to water and foodborne infections, was actually on the rise in the late nineteenth century, there was a substantial decline in this category for young children from 1864/69 to 1880/89, in particular from 1870/79 to 1880/89. What caused this impressive decline in these causes of death for children, while infants did not benefit from these factors? A rise in breastfeeding practices is not very likely to explain improved conditions for young children, when the conditions for infants in fact deteriorated. However, feeding practices can still be part of the explanation. Although De Beer argued that nutrition did not significantly impact mortality from the second year of life onwards in the Netherlands, his analysis focuses only on the quantity of the caloric and protein intake.[398] What if it was not the quantity, but the quality that made a difference for young children? Perhaps there was a shift in the quality of feeding practices for young children in the final decades of the nineteenth century.

Whereas little is known about breastfeeding practices versus artificial feeding practices for infants, there is even less known about feeding practices for young children. The advice given by physicians was to make a gradual transition from weaning to easily digestible porridges, to slowly joining the family table and consuming food that was heavier to digest.[399] Whether people indeed adhered to this advice remains unknown. Yet, during the 1870s and 1880s, many new artificial foodstuffs to feed infants and young children were introduced. In the Netherlands, especially condensed milk and Nestlé's '*farine lactee*' were popular as artificial substitutes for breastmilk.[400] In 1867, Nestlé's product was put on the market, becoming popular especially among higher classes in the northern regions of the country.[401] Although they mainly aimed at infant feeding, Nestlé also had a special product for one-year-olds.[402] Could it be the case that these new products were harmful to very young infants, but were actually improving the nutritional status of the one-year-olds? Perhaps the decline in water and foodborne mortality in the late nineteenth century was a higher-class phenomenon, since the higher classes could afford new products or adjusted their feeding practices for young children in a different way, improving the quality of the food given to their children. Once children joined the family table, the quantity and quality of the family dinner was more important than the quality of milk and milk substitutes for the survival of young children.

Nutrition could be an explanation for the simultaneous decline of the group of airborne diseases, which also includes diphtheria, measles, tuberculosis and pneumonia. The decline in this group took a leap forward from 1920 onwards, which indicates that

398 Hans de Beer, *Voeding, Gezondheid en Arbeid in Nederland Tijdens de Negentiende Eeuw: Een Bijdrage tot de Antropometrische Geschiedschrijving* (Amsterdam: Aksant, 2001).

399 Douwe Lubach and Samuel Senior Coronel, *De Opvoeding van den Mensch van zijne Kindsheid tot den Volwassen Leeftijd: Eene Handleiding voor Ouders en Onderwijzers* (Haarlem: De Erven F. Bohn, 1870): 15-16.

400 De Knecht- van Eekelen, *Naar een Rationele Zuigelingenvoeding.*

401 Ibid., 350.

402 Ibid., 281.

a common denominator was responsible for that decline. The origin of the common denominator is most likely to be found in factors involving the host or non-specific exposure, as the concomitant decline is less likely to be explained by a simultaneous decline in virulence of all individual infections. One of the explanations for a decline in several airborne infectious diseases at the same time could have been an improvement in living conditions such as housing and nutrition. Particularly measles mortality indicates overcrowding,[403] although overcrowding is also an important determinant for mortality from airborne infections as a whole. Combatting overcrowding, which the municipality had slowly taken up, could thus have been one of the explanations for the mortality decline from the 1920s onwards.[404]

The other living condition that could have improved was the already mentioned nutritional intake, in line with McKeown's explanation of the decline in airborne infectious disease mortality during the epidemiological transition.[405] Improved nutritional intake, either in quantity or in quality, can improve the host's resistance to infections; better-nourished children would therefore have been more robust when affected by the total burden of diseases. Even if net calories per household did not change, fertility declined during the 1910s in Maastricht, which could have caused less strain on households and increased the size of the share of an individual child within the household. However, for the Netherlands, the relation between nutritional intake and mortality is not very straightforward. De Beer has found no relationship between nutrition, proxied by real wages, and mortality from the second year of life onwards for the late nineteenth century,[406] although such a relation could have existed in the twentieth century. Finally, an important factor to keep in mind is that the robustness of young children is affected by their wellbeing as infants. During the 1910s, infant mortality rates declined rapidly, which probably indicates that infants suffered less severely from gastrointestinal infections, which thus strained the body less than before. The children who turned one were then more apt to endure and survive childhood diseases. Whether the different changes in the disease environment affected inequalities and equalities in early mortality as well, either according to gender or socioeconomic status, will be elaborated upon in the following section.

5.5 Inequalities in early childhood mortality

Whereas the role of socioeconomic status in mortality differentials for infant mortality is disputed, a stronger social gradient can be found in early childhood mortality.[407] However, there is often no substantial role ascribed to socioeconomic status in differences in early childhood mortality. Whereas Oris et al. did find an effect of economic factors on mortality

403 Woods, *The Demography of Victorian England and Wales*, 311.

404 Gemeente Maastricht, *Verslag van den Toestand der Gemeente over het Jaar*.

405 McKeown, *The Modern Rise of Population*.

406 De Beer, *Voeding, Gezondheid en Arbeid*, 78.

407 Oris, Derosas, and Breschi, 'Infant and Child Mortality'; Van Poppel, Jonker, and Mandemakers, 'Differential Infant and Child Mortality in Three Dutch Regions, 1812-1909.'

risks from the second semester of life onwards, with the largest influence after the first birthday, they also argued that ecological factors were most decisive in early childhood mortality differentials, at least in pre-transitional populations.[408] Differing spatial patterns in childhood mortality in England were not so much the result of the influence of social and economic factors, but instead of the particular disease environment. Manufacturing towns were the exception; there class did matter for children's survival chances.[409] In these manufacturing towns, the environment was not conducive to child health. The social gradient discovered in these towns suggests that the higher social classes were indeed more able to shield their children from the environment because of, for example, their financial situation and knowledge.[410] In the United States, the influence of social capital, in the form of the available knowledge, proved more important than economic factors. Economic circumstances in the United States were fairly adequate, as higher wages and the abundancy of food compared to other Western countries demonstrated. However, the United States were not able to reach lower levels of childhood mortality compared to Britain, indicating that economic factors were not conclusive in early childhood mortality decline. In fact, the lack of knowledge of infectious diseases and hygienic practices among public officials, physicians and parents impeded the United States from gaining an advantageous position.[411]

Socioeconomic status in the Netherlands has been argued to have been of only minor importance as a determinant for differences in early childhood mortality throughout the nineteenth century.[412] However, in the Netherlands, a small social gradient in early childhood mortality did start to express itself from the 1850s onwards, which was larger compared to the one for infancy.[413] The three studied provinces in the Netherlands (out of eleven) – Zeeland, Utrecht and Friesland – were moreover not characterised by much industry and differed greatly from Maastricht, which was well known for its industrial expansion. As Reid has argued, class could have influenced early childhood mortality in such manufacturing cities.[414] While socioeconomic differentials have not been found to be of much importance for other regions of the Netherlands, it may have been more influential in industrialised Maastricht. Due to data limitations, it is not possible to conduct complex modelling of the role of socioeconomic status in ECM for Maastricht. However, descriptive techniques may give an indication whether socioeconomic factors were a noteworthy factor in childhood mortality differentials.

Changes in the disease environment could have influenced other inequalities as well, most prominently between the sexes. Sex differentials have been found in many age

408 Oris, Derosas, and Breschi, 'Infant and Child Mortality, ' 388.

409 Reid, 'Locality or Class? Spatial and Social Differentials in Infant and Child Mortality in England and Wales, 1895-1911.'

410 Ibid., 141.

411 Samuel H. Preston and Michael R. Haines, *Fatal Years: Child Mortality in Late Nineteenth-Century America* (Princeton: Princeton University Press, 1991): 208-10.

412 Van Poppel, Jonker, and Mandemakers, 'Differential Infant and Child Mortality in Three Dutch Regions, 1812-1909.'

413 Ibid., Oris, Derosas, and Breschi, 'Infant and Child Mortality,' 394.

414 Reid, 'Locality or Class? Spatial and Social Differentials in Infant and Child Mortality in England and Wales, 1895-1911.'

categories throughout time and may differ from one disease to another. The general assumption is that boys are biologically weaker than their female counterparts, which is mainly expressed through a lower immune response in boys.[415] They are thus more vulnerable to infectious diseases. In a disease environment characterised by a wide range of infectious diseases, boys may have been on the losing end. In assuming there is a real biological disadvantage for boys, presumed equality as expressed by similar gender-specific mortality rates may actually have masked female disadvantages.[416] However, sex differentials can also have a behavioural, socioeconomic or cultural origin, which is why a debate on the origin of gendered disadvantages in mortality is still ongoing; is this a biological phenomenon or is it more dependent on contextual factors such as socioeconomic or cultural differences?

The fact that gender disadvantages differed across populations adds to the complexity of this question. In the nineteenth and early twentieth century, Dutch boys from infancy to five years old had higher mortality chances than girls, while after age three a clear disadvantage for girls started to express.[417] This female disadvantage only disappeared after 1930. In the Twente region in the Netherlands, Janssens et al. found mainly excess male mortality for children between one and five in the rural areas in the period from 1875-1900.[418] In industrial areas, sex differences in childhood mortality were limited, except for the excess male mortality in the period 1880-1885, which flipped to female excess mortality in the period thereafter. The opposite was the case in nineteenth-century London. Here girls had a disadvantage, in particular regarding infectious diseases in childhood. However, women did already have higher life expectancies overall. The decline in infectious diseases in early childhood mortality only caused a widening gap in life expectancy between London men and women towards the turn of the century, as girls benefitted disproportionally from the decline.[419] The United Nations underline this ambiguous phenomenon where boys are clearly more susceptible to infectious diseases in infancy, but where the direction of sex differentials for young children remains inconclusive. However, for the north-western European regions as a whole, there was a marked increase in male excess mortality in early childhood between 1860 and 1930, with a particular strong increase after 1900.[420]

Departing from the idea that biological differences indeed exist to a certain extent, contextual factors such as cultural and behavioural influences start to manifest themselves progressively from early childhood onwards. Aaby, for example, has argued that the

415 Annechien Bouman, Maas Jan Heineman, and Marijke M. Faas, 'Sex Hormones and the Immune Response in Humans,' *Human Reproduction Update* 11, no. 4 (2005); Waldron, 'Sex Differences in Human Mortality: The Role of Genetic Factors,' 411–423.

416 The United Nations Department of Economic and Social Affairs Population Division, 'The Extent and Causes of Female Disadvantage in Mortality: An Overview.,' in *Too Young to Die: Genes of Gender?, ed.* The United Nations Department of Economic and Social Affairs Population Division (New York: United Nations, 1998).

417 Van Poppel, *De 'Statistieke Ontleding Van De Dooden': Een Spraakzame Bron?,* 15.

418 Angélique Janssens, Maaike Messelink, and Ariana Need, 'Faulty Genes of Faulty Parents? Gender, Family and Survival in Early and Late Childhood in the Netherlands, 1860-1900,' *The History of the Family* 15, no. 1 (2010).

419 Mooney, 'Shifting Sex Differentials in Mortality During Urban Epidemiological Transition.'

420 Dominique Tabutin and Michel Willems, 'Differential Mortality by Sex from Birth to Adolescence: The Historical Experience of the West (1750-1930),' in *Too Young to Die: Genes or Gender?,* ed. The United Nations Department of economic and social affairs Population division (New York: United Nations, 1998): 26-27.

seemingly genetic disadvantage for boys in measles mortality, was actually a culturally-induced behavioural phenomenon.[421] Female disadvantages can be the consequence of socioeconomic factors as well, when limited resources are unevenly allocated to different household members.[422] Some even go as far as claiming that the genetic vulnerability of boys is the result of more discursive phenomena than of biological differences.[423] Undeniably, gender inequalities in mortality exist, albeit having different origins. While taking into account the related social and cultural aspects, it would be too radical to reject the idea of a biological origin of disadvantages for boys entirely, given the extensive biological research that has been done.[424]

After the first year of life, however, discriminating environmental influences started to dominate biological influences on gender-specific mortality.[425] Thus, despite the biological disadvantage of boys, female excess mortality for all ages has been linked to higher female mortality from infectious diseases.[426] Five main factors likely discriminated against girls in childhood and adolescence: Nutritional intake, housing conditions and hygiene, access to education, access to the health system, and conditions at the workplace.[427] These five factors can in turn influence the three more direct factors which determine the severity and outcome of disease: The extent to which one is exposed to the disease, the host resistance, and the response to contracting the disease.[428]

Sufficient nutrition shields children from infections, both severe and less severe, which improves the host's resistance to the disease. In the case of large poor families, the sufficient nutritional intake of children could be put in jeopardy. According to the resource dilution hypothesis, with every child born into the family, a limited amount of resources (both material and immaterial) had to be divided over an increasing number of family members.[429] Apart from the distribution of food, less tangible resources like care also had to be divided over multiple siblings. In many countries, there was a preference for boys, which led to the lower nutritional intake of girls.[430] Contextual factors are, however, very important in using the resource dilution hypothesis as a framework.[431] Depending on the regional sociocultural setting, gender preferences could differ. According to Riswick, a mixed sibship-set preference dominated in the Netherlands,[432] which may have been less detrimental to girls. In general, Dutch boys had higher chances of dying when there was

421 Aaby, 'Are Men Weaker or Do Their Sisters Talk Too Much?'

422 Tim Riswick and Theo Engelen, 'Siblings and Life Transitions: Investigating the Resource Dilution Hypothesis across Historical Contexts and Outcomes,' *The History of the Family* 23, no. 4 (2018).

423 Heather T. Battles, 'The Biologically Vulnerable Boy,' *Boyhood Studies* 9, no. 2 (2016).

424 For a good overview, see Cullen et al., 'The Weaker Sex? Vulnerable Men and Women's Resilience to Socio-Economic Disadvantage.'

425 Tabutin and Willems, 'Differential Mortality by Sex from Birth to Adolescence,' 47.

426 Ibid., 36.

427 Ibid., 48.

428 See also van Poppel, *De 'Statistieke Ontleding van de Dooden': Een Spraakzame Bron?*

429 Riswick and Engelen, 'Siblings and Life Transitions.'

430 Edward Shorter, *A History of Women's Bodies* (New York: Basic Books, 1982).

431 Riswick and Engelen, 'Siblings and Life Transitions.'

432 Tim Riswick, 'Testing the Conditional Resource-Dilution Hypothesis: The Impact of Sibship Size and Composition on Infant and Child Mortality in the Netherlands, 1863–1910,' *The History of the Family* 23, no. 4 (2018).

an older brother in the household in the final two decades of the nineteenth century, while Dutch girls in this period had lower chances of dying when they had an older brother. For the Limburg region, where mainly large stem families resided, this effect was however not significant. In this region, the risks of dying for boys of girls did not differ significantly according to sibship composition.[433]

The second discriminating factor of housing conditions and hygiene is strongly related to the cultural expectations of boys and girls, and can be decisive for the extent to which girls and boys were exposed to pathogens. Girls were often expected to help inside the house, while the opposite was the case for boys, who had to help outside of the house. Poorly ventilated and small houses could then affect girls negatively.[434] Excess female measles mortality in contemporary underdeveloped countries has been explained by this type of reasoning. Aaby, however, finds that in many historical populations it was actually males who suffered from excess measles mortality. Whooping cough on the other hand was particularly high in female children.[435] The explanation for the historical higher male measles mortality should not be looked for in biological differences, since worldwide mortality patterns through time vary too much to be caused by a general biological factor.[436] According to Aaby, mainly culturally-induced behavioural patterns influence the transmission patterns, which in most cases caused boys to be more susceptible. An important biological influence in measles mortality concerns the distinction between index cases and secondary cases. An index case is the first case within a family who contracts the disease outside of the home environment. Secondary cases often contract the disease in the home environment. Because secondary cases constitute a more intense infection (as the result of longer exposure to the virus because of the extensive contact within the home), they suffer from higher case fatality rates. Moreover, secondary cases tended to be less severe later on in childhood, where contracting measles from a family member in early childhood was the most hazardous. Aaby argues that the behavioural component is related to the mode of transmission, since girls were more involved in close social interaction outside of their homes compared to boys. Girls were therefore more likely to be the index case in the family in early childhood than boys. This pattern is reversed when girls are more restricted to the home and have no close contact with other families outside of their own.[437] Thus, in historical populations where excess male measles mortality is dominant, girls were likely not as confined to their homes as in some current-day underdeveloped countries, according to Aaby.

The other three factors through which discrimination can affect mortality inequalities – access to the health system, education, and conditions at work – may have less effect on the age category of early childhood. This may explain why excess female mortality is sometimes only expressed very clearly in the later age categories, between five and

433 Ibid.

434 Tabutin and Willems, 'Differential Mortality by Sex from Birth to Adolescence,' 48/49; Van Poppel, *De 'Statistieke Ontleding van de Dooden': Een Spraakzame Bron?*

435 Aaby, 'Are Men Weaker or Do Their Sisters Talk Too Much? ,' 223.

436 Ibid., 224.

437 Ibid.

nineteen years old.[438] In these later ages, access to school or working conditions are more relevant discriminating factors. Access to health care may have already been an important factor in the response to contracting a disease in the early ages. In Belgium, it appeared that boys were taken to medical services more often than girls.[439] In the Dutch city of Tilburg, there was however no reason to believe that there was gender discrimination in accessibility of health services for the early ages at the start of the twentieth century.[440] Moreover, Rutten has shown that medical care for young children, regardless of their sex, was less likely to be sought for than for other age categories. For both the very young and the old, the family was not very prone to call for professional medical care.[441] In Maastricht, this is exemplified by the few beds available for children in the hospital until well into the twentieth century, with only one ward with twelve beds intended for children, in contrast to four wards for adult men. Until the turn of the century, infants were not welcome at all at the hospital, although an occasional infant patient appeared in the lists of treated patients in the nineteenth century.[442] However, even if there had been no discrimination in accessibility to health services, this may have had little effect, since there were only few effective medical remedies for the infectious childhood diseases during the majority of the researched period. It does, however, mirror the potentially uneven distribution of care that was given to sick family members.

5.6 Inequalities in early childhood mortality in Maastricht

In order to look into socioeconomic inequalities in epidemiological patterns, the occupations of the fathers of the children were used. The same categories as for infants were used: Upper class, skilled workers and unskilled workers. Out of the total of 7,357 young children that had died in the research period, 6,666 children could be classified as one of these three classes (Table 5.1). In the city of Maastricht, at least some socioeconomic differences in early childhood mortality occurred when taking into account different disease patterns. As Figure 5.7 shows, the distribution of deaths due to specific disease categories began to change for upper-class children from 1870/79 onwards. This change was less apparent for children in skilled and unskilled working-class families. In 1870/79, children from the upper classes already showed a lower percentage of deaths due to water and foodborne infectious diseases compared to the skilled and unskilled workers. The disease distributions in 1880/89 were more similar to each other, although they diverged yet again in 1890/99. The differences remained quite large for the two decades thereafter. Whereas upper-class children's deaths were less often caused by water and foodborne infectious diseases, this category still made up for one-third of children's deaths of the unskilled workers throughout the period, and for

438 Janssens, Messelink, and Need, 'Faulty Genes of Faulty Parents?'; Tabutin and Willems, 'Differential Mortality by Sex from Birth to Adolescence.'

439 Tabutin and Willems, 'Differential Mortality by Sex from Birth to Adolescence.'

440 C. G. W. P. Van der Heijden, *Het Heeft Niet Willen Groeien: Zuigelingen- en Kindersterfte in Tilburg, 1820-1930. Omvang, Oorzaken en Maatschappelijke Context* (Tilburg: Stichting Zuidelijk Historisch Contact, 1995).

441 Rutten, 'Ongelijke Behandeling Binnen het Gezin,' 254-255.

442 Gemeente Maastricht, *Verslag van den Toestand der Gemeente over het Jaar.*

the skilled workers in 1900/09. In 1910/19, the distribution of causes of death equalised again, when in all segments of society approximately three-quarters of the deceased children had died of airborne infectious diseases.

The next shift in mortality from airborne infectious diseases to non-infectious diseases occurred rather simultaneously for all classes. From 1940/49 onwards, only about fifty per cent of the deceased children had died of an airborne infectious disease. While the upper classes gained an advantage regarding water and foodborne infectious mortality by the 1870s, the shift from airborne infectious diseases to non-infectious diseases was thus a more uniform event.

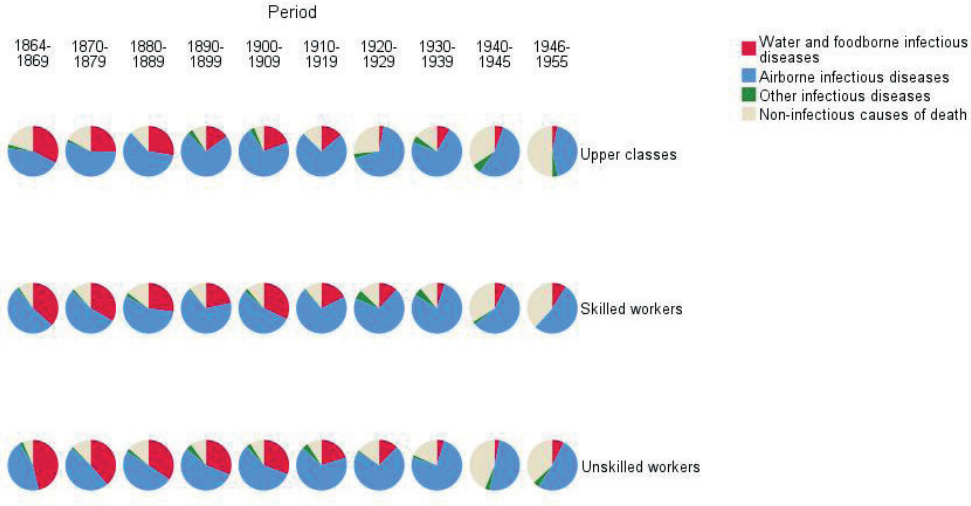


Figure 5.7 Distribution of deaths according to disease category, by socioeconomic status, Maastricht 1864-1955

N upper classes =954, n skilled workers = 3,254, n unskilled workers = 2,458
Source: Maastricht Death and Disease Database

The smaller contribution of water and foodborne infectious diseases to the total deaths for only upper-class children from 1870/79 onwards, would support the idea that improved feeding practices aided this change. As De Knecht-van Eekelen showed, artificial foodstuff production expanded in the late nineteenth century, and the elite were the most important purchasers.[443] This may have been beneficial to upper-class children, while it was not beneficial to upper-class infants. Other changes in nutritional intake do not provide us with a sufficient explanation for the diverging trend in disease patterns. The agricultural crisis, taking place in the same period and improving the food standard for people living in urbanised areas because of the cheaper imported grain, would have provided a more egalitarian effect. Improvements in hygiene would also not offer a suitable explanation for this trend, as such improvements should have affected infant

443 De Knecht-van Eekelen, *Naar Een Rationele Zuigelingenvoeding*.

mortality as well, which did not show a change in disease patterns for the upper classes in this period, and overall infant mortality rates were rising. The main trend Figure 5.7 shows is that a diverging disease pattern for different social groups began to be visible from the 1870s onwards. The decline in water and foodborne mortality from the 1870s to the 1880s may therefore have been associated with gained advantages among the upper classes. The privileges of the upper-class families may have helped them less in the next change in the disease environment, when airborne infectious diseases became a less dominant category of deaths. The decline in mortality due to airborne infectious diseases appears to have occurred simultaneously for all classes.

The other prominent potential inequalities and equalities which could be influenced by the changing disease environment were gender differences. In order to analyse these, we will first look at the gender-specific mortality rates (Figure 5.8). As Figure 5.8 illustrates, during some specific high-mortality years, male mortality was higher compared to female mortality. This was most evident in the nineteenth century in the years 1864, 1866, 1879 and 1887, although also twice in the early twentieth century, in 1905 and 1907. During lower mortality years, the opposite was the case: The male mortality rate was often actually lower compared to the female mortality rate. Yet in two semi-peak years, 1874 and 1884, female mortality was substantially higher than male mortality, and in the peak years 1892 and 1910, female mortality was slightly higher than male mortality. After the mid-1910s, it appears the large differences between the sexes had waned. Could boys have been more affected by epidemic outbreaks, while girls were more disadvantaged under 'normal' mortality circumstances? Would the latter disadvantage stem from a form of discrimination against girls under normal circumstances, since males are biologically more vulnerable?

If that is the case, mortality ratios between the sexes should show a male disadvantage for the characteristically epidemic diseases, and a female disadvantage for the characteristically endemic diseases. Would disease categories themselves, moreover, differ in the extent to which they discriminated between the sexes? Figure 5.9 shows the sex ratio (M/F) based on mortality rates, according to all-cause mortality and the disease categories water and foodborne infections and airborne infections. Ratios around one indicate no differences between the sexes, while ratios above one indicate higher male mortality and ratios below one indicate higher female mortality.

The all-cause mortality sex ratios remain around one, sometimes tending slightly more to male excess mortality, only to flip over to slight female excess mortality in other periods. It is only after 1920/25 that there is a clear trend in male excess mortality, until after the Second World War. The number of deaths had, however, declined to such a low number by that period, that the sex ratios in this period are more subject to chance. This is also the case for water and foodborne mortality after 1920, hence the massive peak in excess male mortality in 1925/29. The trend prior to 1920 shows both male and female excess mortality, and at times equal mortality rates. A clear male disadvantage was apparent in the 1880s, the same period when the plateau in water and foodborne mortality had been reached, after a period of decline. However, after the 1880s, the disadvantage turns to girls during the 1890s. During the early twentieth century, the sex ratios remain quite equal, until the deaths due to this disease group drop massively after 1920.

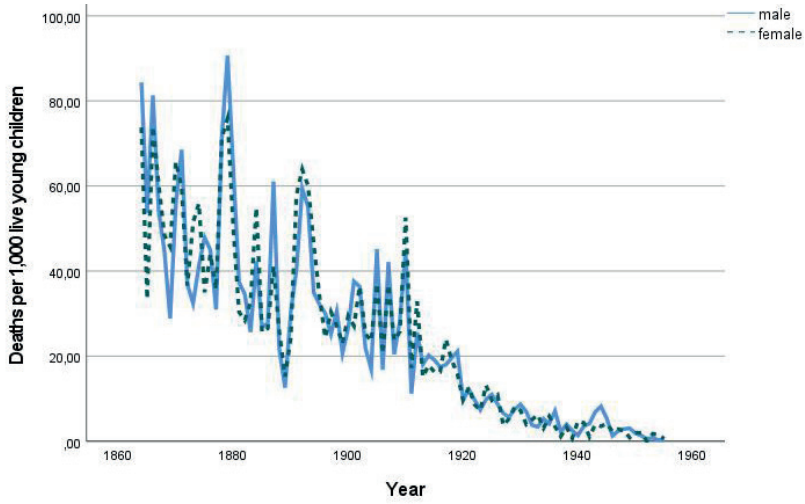


Figure 5.8 Gender-specific mortality rates, Maastricht 1864-1955

Source: Maastricht Death and Disease Database

There is less male excess mortality for airborne infectious disease mortality. The sex ratios either remain mostly equal, or show a slight disadvantage for girls. Only after 1935 does a male disadvantage appear, although this is again likely to be distorted by the low mortality rates achieved by that time. It is noteworthy that there is no male excess mortality for airborne infectious diseases, despite the peaks in mortality caused by epidemic outbreaks, for which airborne infections were most often responsible (Figure 5.8). While there are no clear trends in the sex ratios at the level of disease categories, this may be the case for individual diseases. Did the very specific disease environment affect boys and girls differently?

In order to investigate the male/female ratio for individual diseases, I calculated cause-specific male/female ratios for the most recurring individual causes of death or the causes of death known for some severe epidemic outbreaks, being gastroenteritis, pneumonia, tuberculosis, diphtheria, measles, scarlet fever, cholera, convulsions and whooping cough.[444] As discussed earlier, measles, scarlet fever and cholera behaved as epidemic diseases in Maastricht, only occurring occasionally with high mortality spikes, while causing no deaths in other years. On the other hand, tuberculosis, pneumonia, bronchitis, meningitis, convulsions and gastroenteritis behaved as endemic diseases (Annex V). I calculated the male/female ratios for these causes of death based on the mean cause-specific mortality rate per year by gender for the entire research period. I performed a robustness check which excluded the years without any incidence of the particular disease, which turned out to affect the results minimally. Table 2 presents the sex ratios for the individual diseases in Maastricht.

For the individual water and foodborne infectious diseases gastroenteritis and cholera, the male/female ratio was almost one, despite the at-times high male/female ratio

444 Whooping cough had only a small share in the total mortality rates, however, in line with most literature on gendered cause specific mortality in early childhood, it is included in the analysis.

presented in Figure 5.9. It appears that on average, there was no clear male disadvantage in this disease category, except for convulsions, which did show a male disadvantage. The convulsions category may therefore explain the instances when there was a higher male disadvantage as represented in Figure 5.9, especially during the nineteenth century when convulsions was still a regularly used cause of death.

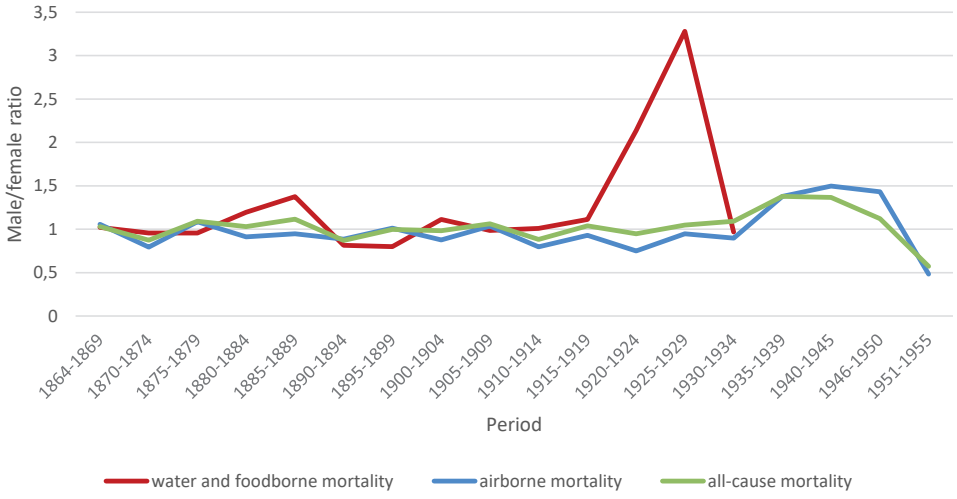


Figure 5.9 Male/female mortality ratios by cause-of-death category, Maastricht 1864-1955

Source: Maastricht Death and Disease Database

The airborne infectious diseases present more pronounced differences. The more endemically raging afflictions such as pneumonia, tuberculosis and bronchitis all show a disadvantage for girls, as does whooping cough. The diseases characterised by more sporadic epidemic outbreaks such as measles, and even more clearly scarlet fever, demonstrate a male disadvantage. Diphtheria was a disease which behaved endemically, yet with some substantial epidemic outbreaks as well. As such, diphtheria does not show a clear disadvantage for either sex with a sex ratio very close to one.

Table 5.1 Male/female ratio by individual disease for total research period

Cause of death	M/F ratio
Pneumonia	0.90
Gastroenteritis	0.98
Tuberculosis	0.90
Diphtheria	0.98
Convulsions	1.08
Meningitis	1.08
Measles	1.20
Bronchitis	0.87
Scarlet fever	1.34
Cholera	0.99
Whooping cough	0.35

These ratios confirm the idea that boys were more likely to suffer from epidemic outbreaks, while girls were not as much affected by those as boys. Girls, on the other hand, did have a disadvantage regarding endemic diseases. The fluctuations in the male/female ratio can be partly explained by this phenomenon. In the periods including the largest scarlet fever outbreaks, disadvantaging boys, the category of airborne infections indeed showed a male disadvantage according to the male/female ratio. In the periods in between, with less large outbreaks of scarlet fever or measles, the airborne male/female ratio was disadvantaging girls. In the period 1905-1909, there was again a male disadvantage, which is likely to have been caused by a series of measles outbreaks. In the period 1910-1914, however, there was again a female disadvantage, despite some recurring measles outbreaks. Contrary to the average measles male/female ratio and the measles male/female ratios for other outbreaks, measles was disadvantaging girls in the outbreaks of 1912 and 1914. Thus, despite a general trend where epidemically raging diseases affected boys disproportionally and endemic airborne afflictions were disproportionally causing havoc to girls, at times the coin flipped to the other side. In general, however, it is clear that once the more endemic airborne diseases also declined during the twentieth century, the male/female ratio gradually changed to favour girls.

A biological explanation of boys having a lower immune response may explain why they suffer more from sudden epidemic outbreaks than girls. Yet what explains the disadvantage for girls regarding the endemic airborne diseases pneumonia, tuberculosis and bronchitis? A more structural explanation is likely to explain this disadvantage. Riswick argued that, within the Netherlands, there was a preference for a mixed sibship, thus no clear preferences for boys existed in late nineteenth-century Netherlands.[445] However, in the southern part of the Netherlands, Riswick's results were less conclusive and thus the male/female ratios presented here may hint to a different story for the southern part of the Netherlands. A lower nutritional status for girls may explain their very particular disadvantages. Lower nutritional intake may have caused them to be less robust in general, which resulted in higher mortality under 'normal' circumstances, with the endemic diseases being constantly present in the population. Mainly when an

445 Riswick, 'Testing the Conditional Resource-Dilution Hypothesis.'

epidemic outbreak occurred, boys' biological disadvantages started to express, resulting in a disadvantage for boys.

5.7 Conclusion

In many European regions, early childhood mortality already declined from the 1870s onwards. The Netherlands were no exception to that trend, and neither was Maastricht. Whereas Maastricht lagged behind in infant mortality compared to the national trend, this was not the case for early childhood mortality. Thus, although a correlation between infant and early childhood mortality emerges from the literature, there were clear differences between mortality in these age categories in Maastricht. Highly important to the fate of young children was the very particular disease environment they were exposed to and how the disease environment changed over time.

The mechanisms of the disease environment are manifold. In analysing the changing disease environment in the course of the decline in early childhood mortality, a central question is what happens when mortality caused by a particular disease is reduced. Mortality declines as a direct effect, but an indirect effect is that other diseases can fill the gap and increase in mortality. Moreover, what is the role of morbidity? Through co-morbidity or scarring, certain diseases can still add to the burden of disease, resulting in death nonetheless. On the other hand, however, if a disease is eliminated, the total burden of disease may be lessened, which may reinforce a decline in other causes of death as well. As Grmek described, diseases in the disease environment can act in symbiosis, antagonistically or indifferently.[446]

The first conclusion of this chapter is that at least two of these mechanisms occurred in the changing disease environment of Maastricht. Driving the decline in early childhood mortality were two disease groups, namely gastrointestinal infectious diseases and airborne infectious diseases. During the first period of substantial decline from the 1870s to the 1880s, mortality of both disease categories declined. The categories acted either indifferently or in symbiosis. Certainly mortality declined, and no other disease appears to have filled the gap, thus the mechanism at play was likely an overall decline of the burden of diseases. Thereafter, the two disease categories declined individually, although the decline in airborne infectious diseases in the period from the 1910s to the 1920s may have been a side effect of the decline in gastrointestinal infections for both young children and infants in the same period, causing more children to be increasingly robust and able to fight airborne diseases.

At the level of individual diseases, a different mechanism of the disease environment appears. Here it seems some diseases did increase in lethality after the receding of another disease. When cholera declined in the 1860s to 1870s, gastroenteritis increased, partially counteracting the decline in cholera. However, after the increase in gastroenteritis, it declined in the following period. Among the airborne infectious diseases something similar happened: From the late 1860s to the 1870s, diphtheria and scarlet fever declined, while antagonistically tuberculosis and pneumonia increased. However, in the

446 Grmek, *Pathological Realities*.

next period the decline in scarlet fever truly gained ground, and it appears to have acted in symbiosis with the declining tuberculosis and other airborne infectious diseases. Even so, after the decline of scarlet fever, another epidemic disease came to the fore and increased in its lethal consequences. Measles mortality increased in the early twentieth century, while the disease had already been present in the late nineteenth century. When measles declined from the 1910s to the 1920s, it acted in symbiosis with the declining tuberculosis and pneumonia.

With the changing disease environment, inequalities changed as well. Whereas in much of the literature a substantial effect of socioeconomic status on early childhood mortality is absent, an exception has been found in manufacturing towns. The second conclusion of this chapter concerns the emergence of socioeconomic differences in early childhood mortality decline around the 1870s. During that period, the upper-class disease pattern showed a smaller contribution from water and foodborne infectious diseases compared to the disease patterns of the skilled and unskilled workers. Since there was an actual decline in water and foodborne mortality in this period as well, this decline could have expressed itself more strongly among the higher social strata. The decline in airborne infectious diseases was, on the other hand, fairly uniform among the different social strata in terms of disease patterns.

Other important potential inequalities could arise between genders. A clear-cut pattern in gender inequalities in historical mortality for young children has not yet been found. While boys appear to have a biological disadvantage, in some societies their disadvantage is counteracted by a more sociocultural disadvantage for girls, with girls having limited access to resources. While Riswick did not find any sociocultural preferences for boys in the Netherlands, this may have been slightly different in Maastricht. The third conclusion of this chapter hints at fascinating mortality differences according to gender, based on a biological disadvantage for boys when characteristically epidemic diseases struck, while girls appeared to have a more continuous disadvantage caused by higher mortality from endemic airborne diseases. Once the majority of airborne infectious diseases had receded, a stronger male disadvantage started to express, indicating that the biological disadvantage remained.

When studying mortality decline and the inherent inequalities, it proves to be important to take into account the entire disease environment. In order to do so, we do not only need individual cause-specific mortality data, but we also need to consider the relations among different diseases. Questions remain about what indeed caused the mortality decline in early childhood from water and foodborne infectious diseases in the late nineteenth century, while infant mortality still increased. Moreover, the gender differences in mortality from more characteristically epidemic airborne and other diseases and more commonly endemic diseases present a new dimension to the puzzle, and it would be interesting to research whether this phenomenon occurred in other European regions as well.

CHAPTER 6

6

Wealthy or poor; male or female; married or unmarried? Mortality inequalities in cause-specific adult mortality in Maastricht 1864-1955

6.1 Introduction

Whereas the decline of infant, and to a slightly lesser extent, early childhood mortality has been given considerable attention in historical-demographical research over the past decades, the decline in adult mortality has received less attention. Given the large share of infant and childhood mortality in total mortality, this may come as no surprise. Living conditions for many adults in the nineteenth century and early twentieth century were nonetheless far from ideal either. Likewise, improvements in those circumstances must have affected the adult population as well. According to Omran, mainly children and women in their reproductive ages experienced the early benefits of the epidemiological transition.[447] Apparently, adult men had lower probabilities of dying compared to women when life expectancies were low. Yet other studies have found that gender differences in adult mortality were highly dependent on a rural or urban environment; adult men had higher mortality risks in urban areas, while women often experienced a relative mortality disadvantage in rural areas.[448]

Recently, historical adult mortality has gained more attention in light of the debate on socioeconomic inequalities. Nowadays, a clear socioeconomic gradient in adult mortality can be observed.[449] The existence of such a socioeconomic gradient is however less straightforward for historical populations, and highly debated. Recent studies on adult mortality in Sweden have shown that the observed socioeconomic mortality differentials among adults are only a phenomenon of the last couple of decades and bear no resemblance to the past.[450] These Swedish findings contest the research that argues that either a temporary socioeconomic gradient in mortality existed among adults in the past, or that socioeconomic status is a fundamental cause of differences in mortality.[451] Antonovsky argued that socioeconomic inequalities in mortality increased during the industrialisation period (1650-1850), especially among the middle years of life,[452] while others argued that such a social gradient emerged during a later period (between 1750-1850).[453] This idea that the social gradient was most apparent in the middle years of life

447 Omran, 'The Epidemiologic Transition,' 41-42.

448 Isabelle Devos, 'Te Jong om te Sterven: De Levenskansen van Meisjes in België omstreeks 1900,' *Tijdschrift voor Sociale Geschiedenis* 26 (2000); Humphries, "Bread and a Pennyworth of Treacle"; Janssens and van Dongen, 'A Natural Female Disadvantage?'

449 Mackenbach et al., 'Widening Socioeconomic Inequalities in Mortality in Six Western European Countries'; Marmot, *The Status Syndrome*; Johan P. Mackenbach, 'Nordic Paradox, Southern Miracle, Eastern Disaster: Persistence of Inequalities in Mortality in Europe,' *European Journal of Public Health* 27, no. Supplement 4 (2017).

450 Bengtsson and Dribe, 'The Late Emergence of Socioeconomic Mortality Differentials'; Tommy Bengtsson, Martin Dribe, and Jonas Helgertz, 'When Did the Health Gradient Emerge? Social Class and Adult Mortality in Southern Sweden, 1813–2015,' *Demography* 57, no. 3 (2020); Enrico Debiasi and Martin Dribe, 'Ses Inequalities in Cause-Specific Adult Mortality: A Study of the Long-Term Trends Using Longitudinal Individual Data for Sweden (1813–2014),' *European Journal of Epidemiology* 35, no. 11 (2020).

451 Antonovsky, *Social Class, Life Expectancy and Overall Mortality*; Clouston et al., 'A Social History of Disease'; Link and Phelan, 'Social Conditions as Fundamental Causes of Disease.'

452 Antonovsky, *Social Class, Life Expectancy and Overall Mortality*, 67; Bengtsson and Van Poppel, 'Socioeconomic Inequalities in Death from Past to Present,' 346.

453 Peter Razzell and Christine Spence, 'The Hazards of Wealth: Adult Mortality in Pre-Twentieth-Century England,' *Social History of Medicine* 19, no. 3 (2006); Riley, *Rising Life Expectancy*; Woods, *The Demography of Victorian England and*

has also been contested; infants and children would actually be more prone to experience socioeconomic advantages and disadvantages.[454]

Complicating the conundrum of historical health inequalities among adults is the interaction between gender and socioeconomic status. Some studies have shown distinct socioeconomic differences in mortality for men and women.[455] Yet these studies are unfortunately scarce, caused by a lack of reliable data especially on female socioeconomic status.[456] Moreover, the causes of differential mortality along socioeconomic and gender lines combined remains obscure. Were the discriminating causes of death the usual suspects, such as maternal mortality disadvantaging women in their reproductive ages and non-natural causes of death having a disproportional share among mostly young men? Or were other causes of death overrepresented in a distinct fragment of the population, indicating other factors influenced differential mortality? By studying cause-specific mortality according to both gender and socioeconomic status, we can enhance our understanding of the roots of adult health inequalities in the past. This chapter therefore seeks to answer the question which gendered socioeconomic inequalities appeared in cause-specific adult mortality in the course of the health transition in Maastricht and what could possibly explain these potential inequalities? Changes in mortality patters according to class and gender can be observed by adopting a longitudinal and cause-specific approach. This will result in an approximation of what kind of inequalities existed based on changing epidemiological patterns. When these changes occurred a-synchronically, it is most likely that a certain part of the population gained an advantage.

Before turning to the analysis itself, I will briefly discuss the decline in adult mortality in the course of the health transition. Subsequently, this chapter will turn to a more elaborate discussion of the literature on socioeconomic differential adult mortality and gender differences in mortality. Arriving at the analysis itself, the initial focus will be on adult mortality decline in Maastricht according to gender and specific causes of death. The second part of the analysis focuses on the inequalities in cause-of-death patterns by once more deploying a multinomial logistic regression model. As we will see, the paths towards a low-infectious mortality regime were not equal and in addition to socioeconomic status, other factors interacted with gender in creating temporary health inequalities.

6.2 The decline in adult mortality

One of the elements central to the decline in adult mortality in the course of the nineteenth century and early twentieth century, is the improvement in the standard of living and

Wales; Robert Woods and Naomi Williams, 'Must the Gap Widen before It Can Be Narrowed? Long-Term Trends in Social Class Mortality Differentials,' *Continuity and Change* 10, no. 1 (1995).

454 Woods, *The Demography of Victorian England and Wales*, 245; Woods and Williams, 'Must the Gap Widen before It Can Be Narrowed?'

455 Edvinsson and Lindkvist, 'Wealth and Health in 19th Century Sweden'; Razzell and Spence, 'The Hazards of Wealth.'

456 Bengtsson and Van Poppel, 'Socioeconomic Inequalities in Death from Past to Present,' 348.

the subsequent increase in life expectancy.[457] The improvements in the standard of living were closely related to economic growth, although the effects of economic growth can be explained in different ways. Either economic growth supported improving living circumstances by causing a rise in real wages, or economic growth was detrimental to living standards as a consequence of the industrialisation and urbanisation process. Migration into urban areas to find employment in the rapidly expanding industries resulted in cramped cities. Not surprisingly, an urban penalty has been observed, where cities were known for their substantially higher mortality rates compared to rural areas, due to the unhealthy urban environments.[458] Not only infants and children fell victim to the hazards of cities, adults were at risk as well as they had to persevere within an environment full of dangerous working conditions and living circumstances. When did these circumstances improve and when did the increasing income start to outweigh the deteriorated living circumstances for adults specifically?

According to Wrigley et al. the secular decline in mortality in England took off in the second half of the eighteenth century, continuing into the early decades of the nineteenth century. After a period of stagnation in the decades between 1820 and 1860, mortality declined again from the late 1860s onwards.[459] Yet adult mortality already declined early in the eighteenth century. Razzell designated this period as the start of a declining mortality trend, while Wrigley et al. argue that this decline only represented a change in demographic regime.[460] The decline in adult mortality was countervailed by an increase in other age-specific mortality, for example among young children, resulting in no improvements in mortality by and large.[461]

Based on more widely available mortality statistics for the nineteenth century, Woods argues in disagreement with Wrigley et al. that the demographic transition only started in the second half of the nineteenth century.[462] After the period of stagnation, and even a decline in life expectancy at birth in the second quarter of the nineteenth century,[463] circumstances seemed to improve from the late 1860s and 1870s onwards. Not only higher social classes benefitted from the improved circumstances, the working population was affected by these improvements as well, according to Szreter and Mooney.[464] Yet these observations mainly rely on total mortality, which is highly dependent on the very high infant and early childhood mortality rates. Infant mortality in England remained high until the start of the twentieth century, but early childhood mortality already declined

457 Floud et al., *The Changing Body*, 6-14.

458 Kearns, 'The Urban Penalty and the Population History of England'; Lionel Kesztenbaum and Jean-Laurent Rosenthal, 'The Democratization of Longevity: How the Poor Became Old in Paris, 1880-1913,' in *New Approaches to Death in Cities During the Health Transition*, ed. Michel Oris and Diego Ramiro Farinas (Switzerland: Springer, 2016); Szreter and Mooney, 'Urbanization, Mortality, and the Standard of Living Debate.'

459 Edward Anthony Wrigley et al., *The Population History of England, 1541-1871: A Reconstruction* (London: Cambridge Group for the History of Population and Social Structure, 1981), 231.

460 Peter Razzell, 'The Conundrum of Eighteenth-Century English Population Growth,' *Social History of Medicine* 11, no. 3 (1998); Wrigley et al., *English Population History from Family Reconstitution 1580-1837*, 284.

461 Wrigley et al., *English Population History from Family Reconstitution 1580-1837*, 282-84.

462 Woods, *The Demography of Victorian England and Wales*, 391.

463 Szreter and Mooney, 'Urbanization, Mortality, and the Standard of Living Debate.'

464 Ibid.

from the 1870s onwards. Where in the decline of total mortality should we therefore precisely situate the decline of adult mortality? And, additionally, how representative is the British experience of other European regions?

In general, mortality among young adults in their twenties started to improve in England and Wales from what Woods designated the start of the demographic transition, around the 1870s. Adults up to the age of 44 followed suit in the years shortly after that.[465] Woods finds a similar trend in his analysis of occupational mortality: Almost all occupations show a marked decline in mortality between the 1860s and 1900s.[466] Changes in adult mortality in Belgium appear to have started slightly earlier based on rising life expectancy at adult ages. Life expectancy at age twenty rose between 1846 and 1856, but briefly declined in 1866.[467] The level from 1856 was picked up again and rose from 1880 onwards. Life expectancy at age forty briefly increased between 1846 and 1856, to decrease again and remain at that level for the final decades of the nineteenth century.[468] It appears that, just as in England and Wales, mortality among younger adults improved at an earlier stage in Belgium than it did for older adults. In France the adult mortality decline already started in the second half of the eighteenth century, and the mortality decline among younger adults once again preceded the mortality decline among older adults. Whereas mortality between ages 15-45 started to decline between 1740-49 and 1780-89, mortality between ages 35-65 declined between 1780-89 and 1820-1829. After a period of stagnation until 1877/81, all mortality below age 45 declined again.[469] Life expectancy at five started to rise again from the 1880s onwards as well.[470] After the war, mainly young adults gained from the mortality decline in France.[471]

Adult mortality decline in Italy started much later than in England, the Scandinavian countries and France, yet Vallin finds that adult mortality in Italy was already much lower than in, for example, France, while child mortality was of a similar level. Apparently, adults in Italy had an advantage over their counterparts in northern European countries, which Vallin proposes may be due to a different lifestyle.[472] In the Scandinavian countries, overall mortality appeared to already decline from the early nineteenth century onwards or after the first couple of decades of the nineteenth century.[473] In Sweden, adult mortality specifically started to decline in the period between 1865 and 1894, according to Bengtsson and Dribe.[474] The timing was not very different for the Netherlands, where female adult mortality started to decline from 1858 onwards; male mortality followed

465 Floud et al., *The Changing Body*, 147.

466 Woods, *The Demography of Victorian England and Wales*, 219.

467 Van Rossem, *Bruxelles Ma Belle, Bruxelles Mortelle*, 220.

468 Ibid.

469 Jacques Vallin, 'Mortality Decline in Europe from 1720 to 1914. Long-Term Trends and Changes in Patterns by Age and Sex,' in *The Decline of Mortality in Europe,* ed. Roger Schofield, David Sven Reher, and Alain Bideau (Oxford: Oxford University Press, 1991): 54.

470 Kesztenbaum and Rosenthal, 'The Democratization of Longevity,' 140.

471 Vallin, 'Mortality Decline in Europe from 1720 to 1914.'

472 Ibid., 56-67.

473 Floud et al., *The Changing Body*, 244; Vallin, 'Mortality Decline in Europe from 1720 to 1914,' 40-41.

474 Bengtsson and Dribe, 'The Late Emergence of Socioeconomic Mortality Differentials,' 390.

suit after 1881. However, the most important period for Dutch adult mortality decline was between 1920 and 1955, having the highest rate of decline.[475] The decline in adult mortality therefore started around the mid-nineteenth century or slightly later in most countries, although in some countries adult mortality already improved in the eighteenth century. For the countries where a distinction between young adults and older adults has been made, it appears that young adults gained the advantage first.

Other similarities in adult mortality across European countries can be found in the most prominent causes of death. Death due to tuberculosis, with maternal mortality and violent deaths in second place, were the most prominent causes of death among adults in many European countries.[476] Phthisis was the first and foremost cause of death among adults aged 20-44 in England and Wales between 1861 and 1870. Although phthisis mortality had declined substantially, especially among younger adults in 1891-1900, it remained the most important cause of death. Phthisis continued to be a risk for older adult ages, but it shared its position with diseases of the respiratory system.[477] While remaining an important cause of death, tuberculosis mortality rates among adults declined during the final quarter of the nineteenth century in England and Wales. A similar trend can be found in Finland and Sweden, where tuberculosis mortality started to fall from the 1860s and early 1870s onwards. This must have been beneficial to adults, since high tuberculosis incidence was found for women in their early youth or during their reproductive period and for men in the upper age groups.[478]

Next to tuberculosis, maternal mortality and violent deaths among men were two of the most common causes of death in adulthood. For women, giving birth was one of the most hazardous endeavours, especially when they were undernourished themselves, or when having very large families. The more well-off women were also at risk if they were taken to the hospital, where puerperal fever could easily spread. When antisepsis and asepsis practices became more common around the early 1880s, maternal mortality started to drop across Europe.[479] In England and Wales, mortality from childbirth declined during the final quarter of the nineteenth century.[480] While maternal mortality rates declined in the same period in the Netherlands as well, the Dutch levels of maternal mortality were already among the lowest in Europe. The lower levels were most likely a result of the common practice of giving birth at home opposed to more hazardous hospital births, and the exceptional high quality of Dutch midwifes.[481] While maternal

475 Wolleswinkel-van den Bosch, 'The Epidemiological Transition in the Netherlands,' 68.

476 McKeown, *The Modern Rise of Population*; Philips, *Gezondheidszorg in Limburg*; Woods and Shelton, *An Atlas of Victorian Mortality*; Brett E. Ory and Frans W. A. van Poppel, 'Trends and Risk Factors of Maternal Mortality in Late-Nineteenth-Century Netherlands,' *The History of the Family* 18, no. 4 (2013).

477 Woods and Shelton, *An Atlas of Victorian Mortality*, 34-38.

478 Bi Puranen, 'Tuberculosis and the Decline of Mortality in Sweden,' in *The Decline of Mortality in Europe*, ed. Roger Schofield, David Sven Reher, and Alain Bideau (Oxford: Oxford University Press, 1991).

479 Shorter, *A History of Women's Bodies*, 132.

480 Woods and Shelton, *An Atlas of Victorian Mortality*, 34-38.

481 Ory and Van Poppel, 'Trends and Risk Factors of Maternal Mortality in Late-Nineteenth-Century Netherlands,' 482-83; John R. Shepherd et al., 'Maternal Mortality in Taiwan and the Netherlands, 1850-1945,' in *Death at the Opposite of the Eurasian Continent. Mortality Trends in Taiwan and the Netherlands 1850-1945*, ed. Theo Engelen, John R. Shepherd, and Yang When-Shan (Amsterdam: Amsterdam University Press, 2011); Irvine Loudon, *Death in*

mortality was one of the greatest risks for women, violent deaths were more common among men.[482] In England and Wales, violent deaths declined in the same period as mortality from childbirth, yet to a lesser extent. Thus, while there was a clear set of usual suspects causing death in adulthood, these could differ between the sexes and classes. We will therefore continue with a discussion of differential adult mortality according to socioeconomic status and gender.

6.3 The socioeconomic gradient in adult mortality

There is a twofold, still-ongoing discussion on the socioeconomic mortality gradient in historical populations, more specifically on when it appeared in the course of the health transition. On one side, there is the question whether a social gradient in adult mortality as we know it has actually ever existed before the past few decades.[483] On the other side, if there was indeed such a social gradient, how did it evolve? Two main theories emerged for the latter side of the discussion. As discussed earlier, Antonovsky proposed the divergence-convergence hypothesis of social inequality in mortality. Between 1650 and 1850, social inequalities in mortality became increasingly larger as the higher classes profited from the growing economy, while the lower classes mainly encountered the downside of emerging industries and expanding economies. As the nineteenth century progressed, this trend was reversed and the mortality gap between the higher and the lower classes started to diminish. [484] Eventually, class differences in mortality had become very small in the mid-twentieth century, although we can now see them increasing again.[485] Contrasting Antonovsky were Link and Phelan, and later Clouston et al., advocating the constancy hypothesis.[486] The constancy hypothesis presupposes omnipresent existing social differences in mortality. While socioeconomic inequalities will remain, the specific diseases at the root of the differences change throughout time with expanding medical knowledge and remedies becoming available to first small and later larger segments of society. Yet, in the end, the two theories are only two sides of the same coin. Both are based on the premise that socioeconomic inequalities in mortality did exist in the past.

These theories have recently been challenged. Predominantly based on research on Swedish population statistics, the idea has arisen that a socioeconomic gradient in adult mortality is only a phenomenon of recent decades, without a clear precedent

Childbirth. An International Study of Maternal Care and Maternal Mortality 1800-1950 (Oxford: Clarendon Press, 1992), 421-22.

482 Woods and Shelton, *An Atlas of Victorian Mortality*.

483 Mackenbach, 'Nordic Paradox, Southern Miracle, Eastern Disaster'; Mackenbach et al., 'Widening Socioeconomic Inequalities in Mortality in Six Western European Countries'; Marmot, *The Status Syndrome*.

484 Antonovsky, *Social Class, Life Expectancy and Overall Mortality*.

485 Mackenbach, 'Nordic Paradox, Southern Miracle, Eastern Disaster'; Mackenbach et al., 'Widening Socioeconomic Inequalities in Mortality in Six Western European Countries'; Marmot, *The Status Syndrome*.

486 Clouston et al., 'A Social History of Disease'; Link and Phelan, 'Social Conditions as Fundamental Causes of Disease.'

further back during the twentieth and nineteenth centuries.[487] A social gradient in adult mortality could only be found in Sweden from the 1950s or the 1970s onwards.[488] However, just as the British experience does not exactly represent the experience of other European countries, the Swedish cannot either, despite the very rich available datasets. A social gradient did emerge in other historical European adult populations, albeit small at times.[489] In England, Woods and Williams already observe lower mortality among upper-class adult men from the mid-eighteenth century onwards,[490] while Razzell and Spence only observe a social gradient emerging at the end of the nineteenth century. The later emergence of social disparities between the rich and poor at the turn of the century are not unique to Britain, as also in Italy (Sardinia) and Paris the rich seem to have fared better at that moment in time.[491] A similar observation has been made for the Netherlands, where Van Reek and Van Zutphen discovered the most pronounced socioeconomic gradient in male adult mortality at the end of the nineteenth century, in the period 1896-1903.[492] Conversely, Van Poppel et al. determined that the advantage of the wealthy Dutch was already present earlier in the nineteenth century, and only decreased towards the twentieth century.[493]

The absence of a socioeconomic gradient in adult mortality might stem from a selection effect. The culling of young children in their early stages of life, caused by dire circumstances in the lower social strata, could result in the survival of the strongest, thus creating healthier and stronger groups of adults despite a low socioeconomic status. However, both in a population with and without observed socioeconomic differences in mortality in the nineteenth century, evidence for such a selection effect has not been found.[494]

487 Bengtsson and Dribe, 'The Late Emergence of Socioeconomic Mortality Differentials'; Bengtsson, Dribe, and Helgertz, 'When Did the Health Gradient Emerge?'; Debiasi and Dribe, 'Ses Inequalities in Cause-Specific Adult Mortality'; Enrico Debiasi, Martin Dribe, and Gabriel Brea-Martinez, 'Has It Always Paid to Be Rich? Income and Cause-Specific Mortality in Southern Sweden 1905-2014,' *Lund papers in economic demography* 2 (2021); Edvinsson and Lindkvist, 'Wealth and Health in 19th Century Sweden.'

488 Bengtsson and Dribe, 'The Late Emergence of Socioeconomic Mortality Differentials'; Bengtsson, Dribe, and Helgertz, 'When Did the Health Gradient Emerge?'; Debiasi and Dribe, 'Ses Inequalities in Cause-Specific Adult Mortality.'

489 M. Breschi et al., 'Socioeconomic Conditions, Health and Mortality from Birth to Adulthood, Alghero 1866–1925,' *Explorations in Economic History* 48, no. 3 (2011); Floud et al., *The Changing Body*, 148-49; Kesztenbaum and Rosenthal, 'The Democratization of Longevity'; Razzell and Spence, 'The Hazards of Wealth'; Frans van Poppel, Roel Jennissen, and Kees Mandemakers, 'Time Trends in Social Class Mortality Differentials in the Netherlands, 1820-1920: An Assessment Based on Indirect Estimation Techniques,' *Social Science History* 33, no. 2 (2009); Woods, *The Demography of Victorian England and Wales*.

490 Woods and Williams, 'Must the Gap Widen before It Can Be Narrowed?,' 129.

491 Breschi et al., 'Socioeconomic Conditions, Health and Mortality from Birth to Adulthood, Alghero 1866–1925.'; Kesztenbaum and Rosenthal, 'The Democratization of Longevity.'

492 J. van Reek and W.M. van Zutphen, 'Sterfte naar Sociale Klasse bij Volwassenen in Nederland sinds de Negentiende Eeuw,' *Bevolking en Gezin*, no. 2 (1985): 84.

493 Van Poppel, Jennissen, and Mandemakers, 'Time Trends in Social Class Mortality Differentials in the Netherlands, 1820-1920' 144-45.

494 Breschi et al., 'Socioeconomic Conditions, Health and Mortality from Birth to Adulthood, Alghero 1866–1925.'; Martin Dribe and Björn Eriksson, 'Socioeconomic Status and Adult Life Expectancy in Early 20th-Century Sweden. Evidence from Full-Count Micro Census Data,' *Lund Papers in Economic Demography*, no. 1 (2018).

The absence of a socioeconomic gradient could also be due to the exclusive focus on all-cause mortality. Existing socioeconomic differences at the cause-specific level may be levelled out at the aggregated level of total mortality. This appears to be the case with the Swedish population studies. Debiasi, Dribe and Martinez did not find a social gradient in all-cause adult mortality prior to the 1950s, but they did discover statistically significant differences at a cause-specific level in the period from 1922 until 1947. Lower mortality due to infectious diseases among men with the highest 20% of incomes, combined with higher mortality due to circulatory diseases within this group and period, resulted in equal total mortality rates.[495] Debiasi and Dribe also found higher mortality due to circulatory causes of death among the higher social classes in the nineteenth century.[496] Yet during the nineteenth century, the most expected cause-of-death category to be affected by socioeconomic status, the infectious diseases, did not show a social gradient in mortality in Sweden, nor in a part of the United States.[497] In the United States, however, the most common cause of death, tuberculosis, did affect particularly men differently in the 1850s-1860s, while all-cause mortality did not show a mortality gradient.[498] The direction of these differences was nevertheless different from the general expectation: Male white-collar workers actually had higher mortality from tuberculosis compared to labourers.[499] The work environment was likely a decisive factor in the risks of dying of tuberculosis among the white-collar workers, who did not experience the advantages of working outside. Van Reek and Van Zutphen adopted a cause-specific approach for their analysis of the Netherlands as well, and found a social gradient for both acute and chronic diseases in 1891-1895. The most prominent differences occurred along the lines of age categories; the age groups 18-24 and 36-50 were most affected by this gradient. The unemployed were an exception, having disproportionally high mortality rates for all causes in all age categories between 18 and 50.[500]

Another issue with the search for a social gradient lies in its definition. For a gradient to exist, socioeconomic status must be positively associated with survival chances. This theoretically means that mortality must be smaller with every step up the social ladder, while the historical reality may have been less linear. Woods and Williams highlighted that, prior to 1750, socioeconomic differences in mortality already existed, albeit without there being a clear gradient: '…in which life-chances were consistently and positively related to social status'.[501] A similar problem can be found in the data for nineteenth-century Brussels, where higher average ages at death for adults were found among the

495 Debiasi, Dribe, and Brea-Martinez, 'Has It Always Paid to Be Rich?,' 17-18.

496 Debiasi and Dribe, 'Ses Inequalities in Cause-Specific Adult Mortality,' 1051.

497 Ibid.; Susan Hautaniemi Leonard et al., 'The Effects of Wealth, Occupation, and Immigration on Epidemic Mortality from Selected Infectious Diseases and Epidemics in Holyoke Township, Massachusetts, 1850-1912,' *Demographic Research* 33 (2015): 1035-46.

498 Joseph P. Ferrie, 'The Rich and the Dead: Socioeconomic Status and Mortality in the United States, 1850-1860,' in *Health and Labor Force Participation over the Life Cycle. Evidence from the Past*, ed. Dora L. Costa (Chicago: University of Chicago Press, 2003): 26-27.

499 Ibid.

500 Van Reek and van Zutphen, 'Sterfte naar Sociale Klasse bij Volwassenen in Nederland sinds de Negentiende Eeuw,' 183.

501 Woods and Williams, 'Must the Gap Widen before It Can Be Narrowed?,' 132.

elite, the retired and the higher professionals. Even so, a gradient could not be found because the group of unskilled workers had lived longer on average than the group of skilled and lower-skilled workers.[502] While a clear-cut social gradient is missing, it would be a bridge too far to conclude there were no important socioeconomic differences at all.

On top of that, the gendered influence of socioeconomic status complicates the study of socioeconomic disparities. The Swedish studies find that, when the social gradient emerged, this occurred two decades earlier for women than for men.[503] In the historical populations too, there were different effects of socioeconomic status along gender lines. While wealthy adult and elderly (over 60 years of age) women in Sweden experienced a health advantage, this was absent for their wealthy male counterparts.[504] The unhealthy lifestyle among wealthy men in particular could explain their lack of a health advantage.[505] High caloric meals, smoking and excessive use of alcohol, with not much physical exercise might be as abusive to the human body as working long hours on crowded factory floors in dangerous circumstances. Research on the combined effects of socioeconomic status and gender is unfortunately still very rare. The scarcity can be partially explained by the lack of sufficient data,[506] but it would be worthwhile to strive for a better understanding of these gendered socioeconomic mortality differentials, since it seems that the experiences of men and women interacted with their social standing.

6.4 Gender differences in adult mortality

In modern-day societies across the globe, women tend to outlive men. With higher life expectancies in all countries, women have a very clear health advantage over men.[507] Biology and genetics in general favour women over men when it comes to infectious diseases, since men find themselves having higher susceptibility to those diseases.[508] Pregnancy is an exception to this, because pregnancy causes women's immune system to become repressed and hence creates a biological disadvantage for women.[509] These biological and genetic advantages do not, however, operate in a vacuum; they interact with environmental, economic and cultural factors.[510] Historically, women had higher life expectancies as well, although these did conceal concrete female health disadvantages in certain age groups.[511] In particular young girls, adolescent women and women in their

502 Van Rossem, *Bruxelles Ma Belle, Bruxelles Mortelle*, 447.

503 Bengtsson, Dribe, and Helgertz, 'When Did the Health Gradient Emerge?'

504 Dribe and Eriksson, 'Socioeconomic Status and Adult Life Expectancy in Early 20th-Century Sweden'; Edvinsson and Lindkvist, 'Wealth and Health in 19th Century Sweden,' 13.

505 Razzell and Spence, 'The Hazards of Wealth.'

506 Breschi et al., 'Socioeconomic Conditions, Health and Mortality from Birth to Adulthood, Alghero 1866–1925.'

507 Anna Barford et al., 'Life Expectancy: Women Now on Top Everywhere,' *BMJ* 332, no. 7545 (2006).

508 Waldron, 'Sex Differences in Human Mortality: The Role of Genetic Factors,' 24-25.

509 Loudon, *Death in Childbirth*, 31.

510 Alter, Manfredini, and Nystedt, 'Gender Differences in Mortality,' 328; Waldron, 'Sex Differences in Human Mortality.'

511 Devos, 'Te Jong om te Sterven,' 56.

reproductive ages were on the losing end.[512] Only towards the age of 50 did gendered mortality rates converge again.[513] In the nineteenth-century Netherlands, it appears that women had a disadvantage as well in the age categories between 10 and 19, and 20 years and older.[514] A clear male disadvantage started to occur with the decline in infectious diseases, and the increase in cardiovascular diseases, starting with birth cohorts from the late nineteenth century.[515]

Before the emergence of a clear male disadvantage, childbearing caused women to fare less well. Maternal mortality in itself contributed to the higher female mortality, but maternal depletion caused by the many pregnancies and low nutritional status was another contributing factor to excess female mortality. Whereas in the Netherlands maternal mortality was relatively low,[516] maternal depletion was very likely to be an important factor nonetheless. Especially in the Catholic south, where large family sizes prevailed, exhausted female bodies were likely common. For mothers to be, the risk of dying was even higher due to possible associated deaths. The immune system was repressed during pregnancy, resulting in increased risks of dying of an infection during these nine months. In the Netherlands, Janssens and Van Dongen indeed discovered a correlation between maternal deaths and deaths caused by the most prominent infectious diseases, namely tuberculosis and respiratory infections.[517] On top of the biological disadvantage, other cultural, social and economic factors could contribute to higher risks for women.

The very specific historical circumstances of the nineteenth and early twentieth century could lead to less robust women in general. Historical excess mortality for women has mainly been found in rural areas in England.[518] During the nineteenth century, capitalist reforms in the agricultural sector disfavoured women. By scaling up and hiring farm labourers, women had less opportunity to contribute financially to the farm income. As a consequence, women and children became more dependent on the labour of the male breadwinner, their husband and father. This economic reorganisation, combined with culturally dependent ideas on the worth of female life, provided the male breadwinner with a privileged position within the household regarding resource allocation.[519] Lower nutritional intake can result in lower robustness when combatting infectious diseases, which puts the ones receiving the smallest plates at the highest risk. In the Netherlands, excess female mortality appeared to be mainly a rural phenomenon as well.[520]

512 Alter, Manfredini, and Nystedt, 'Gender Differences in Mortality'; Devos, 'Te Jong om te Sterven'; Dominique Tabutin, 'La Surmortalité Féminine en Europe Avant 1940,' *Population (French Edition)* 33, no. 1 (1978); Van Poppel, *De 'Statistieke Ontleding Van De Dooden': Een Spraakzame Bron?*

513 Alter, Manfredini, and Nystedt, 'Gender Differences in Mortality,' 328.

514 Van Poppel, *De 'Statistieke Ontleding Van De Dooden': Een Spraakzame Bron?*

515 Hiram Beltrán-Sánchez, Caleb E. Finch, and Eileen M. Crimmins, 'Twentieth Century Surge of Excess Adult Male Mortality,' *Proceedings of the National Academy of Sciences* 112, no. 29 (2015).

516 Ory and Van Poppel, 'Trends and Risk Factors of Maternal Mortality in Late-Nineteenth-Century Netherlands.'

517 Janssens and Van Dongen, 'A Natural Female Disadvantage?,' 84-115.

518 Devos, 'Te Jong om te Sterven'; Janssens and Van Dongen, 'A Natural Female Disadvantage?'; Shorter, *A History of Women's Bodies*.

519 Humphries, "Bread and a Pennyworth of Treacle',' 62-66, 71.

520 Janssens and Van Dongen, 'A Natural Female Disadvantage?,' 96.

In France and Belgium, however, excess female mortality was not restricted to the rural part of the country. French demographers have rather connected the excess female mortality to modernisation, specifically industrialisation, creating an unfavourable position for women in society in general.[521] Excess female mortality for young girls was highest in rural areas in Belgium, although closely followed by clear excess female mortality in the urban and textile regions.[522] For England, Humphries instead argues that increased female participation in industrial areas could improve the position of women within the household, thus causing lower excess female mortality. This was, however, not always necessarily the case. In England too, women could suffer disproportionally from factory employment in particular.[523] Even so, Alter et al. stress that, in general, excess female mortality was lower in urban areas, since women had more opportunities to contribute to the family income in those environments.[524] Hinde argues that in the case of the most common cause of death in the adult ages, tuberculosis, the bargaining-nutrition hypothesis does not hold.[525] In urban areas, it was actually young men who experienced a disadvantage from tuberculosis deaths. The disadvantage for women in rural areas was therefore more likely the effect of return migration of sickly women out of service, instead of an effect of their smaller claim on the household's resources. Hinde furthermore argues that the link between nutrition and tuberculosis is in fact quite weak.

The division of tasks within the household and leisure activities did not aid women either. Men not only worked outside the home, they also often spent their leisure time outside.[526] Whereas this increased the risks of men suffering from injuries while working outside, women were affected disproportionally by dire living circumstances. Women spent more hours within the home in damp rooms, and because women cared for the sick they were also more often exposed to infectious agents within the household.[527] Other disadvantages for men existed as well, however. Risky behaviour such as smoking and violence were more prevalent among men. As a consequence, there was a 'trauma bump' among men in young adolescence due to violent deaths and accidents.[528] The emergence of smoking was also mainly deleterious for men, fuelling an increasing male health disadvantage into the twentieth century. In the nineteenth century, the death of a spouse appeared to still affect widowers more severely than widows. Widowers seem to have been more dependent on the care of their wives, whereas widows were more effective in finding support.[529]

521 Devos, 'Te Jong om te Sterven,' 136-37.

522 Ibid., 64.

523 Humphries, "Bread and a Pennyworth of Treacle'," 468.

524 Alter, Manfredini, and Nystedt, 'Gender Differences in Mortality,' 332.

525 Hinde, 'Sex Differentials in Phthisis Mortality in England and Wales, 1861-1870.'

526 Humphries, "Bread and a Pennyworth of Treacle'," 465.

527 Alter, Manfredini, and Nystedt, 'Gender Differences in Mortality,' 331.

528 James C. Riley, 'Excess Mortality in Youth,' in *Old and New Methods in Historical Demography*, ed. David Reher and Roger Schofield (Oxford: Clarendon Press, 1993).

529 Alter, Manfredini, and Nystedt, 'Gender Differences in Mortality,' 349; Edvinsson and Lindkvist, 'Wealth and Health in 19th Century Sweden,' 383.

Different mechanisms could have caused different mortality rates among women and men. Whereas women were more likely to be in an unfavourable position in rural areas, the pattern is less clear for urban areas. Cities could be dangerous to both men and women, but could also offer economic opportunities. Although Janssens and Van Dongen found excess female mortality in the southern part of the Netherlands, it remains unclear whether Maastricht was also a more dangerous place for women than for men. The city's thriving industry may have had a very distinct effect on its inhabitants' health.

6.5 Adult mortality decline in Maastricht

The focus of this chapter will be on adults and adult mortality between twenty and fifty years of age. This age categorisation allows for the separation of adulthood from adolescence on the one hand, and the separation of the reproductive ages from old age on the other hand. Moreover, the indication of socioeconomic status is based on occupation, and within this age categorisation the amount of people that had retired and therefore had 'no occupation' or 'retired' written behind their names is minimal. Since deaths due to degenerative diseases are less likely to occur in this age category compared to older ages, it should be easier to identify key turning points in the epidemiological transition for adults. Total adult mortality was substantially smaller compared to mortality in the age groups of infants and young children, but it did decline as well in the nineteenth and twentieth century.

For the scope of this study, it appears there were two key turning points in adult mortality in Maastricht: First in the late 1890s and second in the early 1910s, if we regard the WWI increase as an exceptional incident (Figure 6.1). [530] Overall adult mortality started to decline gradually from the start of the research period onwards.[531] Given the highly lethal cholera epidemic in 1866, the mortality rate of the first quinquennial period presented in Figure 6.1 may have been higher than mortality rates in previous periods.

The driving factor behind the early mortality decline was the decline in female mortality. Male mortality plateaued until there was a rapid decline from the late 1890s onwards. Thereafter, both male and female mortality remained quite stable until the 1910s. Mortality for both genders started to decline rapidly again from the early 1910s onwards, with the exception of a peak in mortality during the First World War. The adult mortality decline in Maastricht thus more or less resembles the Dutch pattern, in

530 Mortality rates were calculated on the basis of the Maastricht Death and Disease database, the censuses and the reports of the municipality of Maastricht. The final reliable census within the research period was held in 1930. There are no censuses from the Second World War, and the next census was held in May 1947. Whereas for infants and children estimations could be made quite easily for the period after WW II, this was a too shaky endeavour for adults. I calculated the population at risk for 1930-1944 on the basis of the value of the entire population of Maastricht for each year, based on the adult ratio of the population of the 1930 ratio. After 1944 this ratio would be less reliable, because of the death toll and the baby boom of the late 1940s. Moreover, in 1920 the municipality of Maastricht annexed parts of its surrounding municipalities. This annexation was accounted for in calculating and estimating the population at risk

531 Whether the decline had already started earlier in Maastricht is difficult to assess, due to the lack of sufficiently specific data.

which Wolleswinkel-van den Bosch found that female adult mortality started to decline from 1858 onwards, and for men from 1881 onwards.[532] Men slightly lagged behind the national average.

Female mortality did not only decline earlier than male mortality, it appears that the industrial city of Maastricht was also not disfavouring women when it comes to mortality hazards; rather the opposite was the case. Male mortality rates remained higher than female mortality rates until the First World War. Female mortality increased during WWI almost to the level of male mortality, and thereafter the gendered mortality rates behaved synchronically. The disproportional rise in mortality among women in the First World War brings forth new questions. Did the effects of the war, while the Netherlands remained neutral, affect women more than men, despite the mobilisation and closing of many factories in this period which would weigh particularly heavy on men? The Second World War also shows a marked effect on mortality, although this time men were disproportionally affected. The fact that the Netherlands played a much more active part in WWII may be a plausible explanation for the increased mortality at that time.

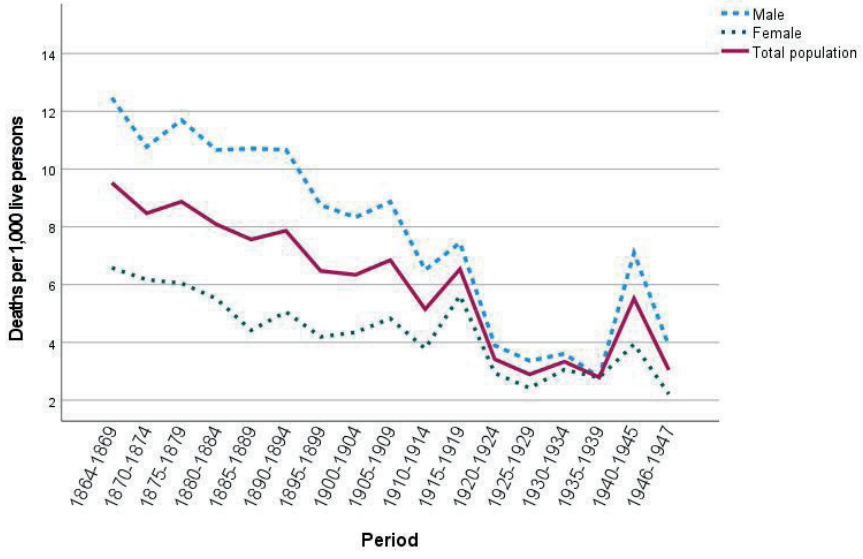


Figure 6.1 Quinquennial average all-cause adult mortality rate (20-49) by gender, Maastricht 1864-1944

Source: Maastricht Death and Disease Database, Dutch censuses

These all-cause mortality rates may obscure differences in cause-specific patterns. The analysis of specific causes of death will help to gain more insight into these specific gendered mortality rates. For the analysis of the cause-of-death patterns, I classified the individual causes of death into an initial eleven categories. Infectious diseases were again divided into water and foodborne infectious diseases (1), airborne infectious diseases (2)

532 Wolleswinkel-van den Bosch, 'The Epidemiological Transition in the Netherlands,' 68.

and other infectious diseases (3). All other defined causes of death belong to the category of non-infectious diseases (4), except for cancers (5), cardiovascular causes of death (6), causes of death indicating a mental disorder (7), deaths related to childbirth (8) and external causes of death (9). Finally, ill-defined causes of death (10) and unknown causes of death (11) were given a separate category as well.

Figure 6.2 demonstrates that, until the early twentieth century, infectious diseases indeed reigned. It was predominantly airborne infectious diseases that created great havoc among both genders, although in larger numbers among men compared to women. Only during their sharper rise in mortality during WWI, female mortality due to airborne diseases approximated male mortality due to the same cause. This was solely the result of the Spanish flu pandemic of 1918, when 27.7 % of total female mortality was caused by either the Spanish flu or influenza.

More specifically gendered causes of death are clearly visible as well. External mortality seems almost absent among women, while being clearly present among men. The increasing mortality during WWII is also mostly the result of increasing mortality from external causes such as bomb attacks or grenades, besides a larger amount of unknown causes. Whereas many Dutch citizens suffered from famine during the final winter of the war in 1944-45 (*hongerwinter*), this was less the case for Maastricht citizens, since Maastricht had already been liberated in September 1944. Yet while the collective memory mainly recollects the famine and cold of the final war years, apart from the atrocities of the Holocaust, Ekamper et al. have recently shown that the forgotten citizen casualties from the war in the non-western parts of the county contributed equally to the experienced excess mortality as the famine and cold victims in the west.[533] Next to the increased external mortality, the increased unknown causes mainly consist of the mentioning of 'Art. 50 BW.' This indicated that a citizen of Maastricht had died in another locality, this being a different municipality or country. The number of causes of death described as 'missing' had also increased substantially during the war years. The Jewish population in Maastricht was disproportionally affected by the war, although some of the missing cases also occurred among the rest of the population. Nonetheless, in the total dataset only 0.96% of the deceased were of the Jewish faith, while during WWII people of Jewish faith made up 12.26% of the adult female mortality and 7.12% of the adult male mortality.

533 Peter Ekamper et al., 'War-Related Excess Mortality in the Netherlands, 1944-45: New Estimates of Famine- and non-Famine-Related Deaths from National Death Records', *Historical Methods: A Journal of Quantitative and Interdisciplinary History* 50, no. 2 (2017).

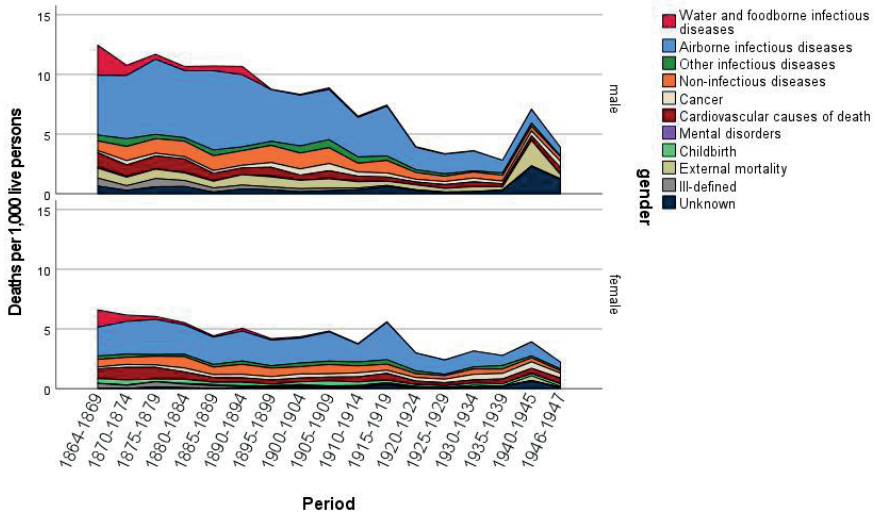


Figure 6.2 Cause-specific mortality rates by gender, aged 20-49, Maastricht 1864-1947

Source: Maastricht Death and Disease Database, Dutch censuses

In the majority of the research period, female-specific mortality due to causes related to complications in childbirth were indeed present, yet only in small numbers and declining even further after the 1920s. However, a critical note should be raised in assessing the mortality rates related to childbirth. During the process of estimating female socioeconomic status, it appeared that on multiple occasions a woman had died shortly after giving birth (stillbirths and live births) without any sign of that in the designated cause of death. For example, in 1900, when the 44-year-old Anna Maria died two days after giving birth, the registered cause of death did not mention anything about a pregnancy, childbirth or a related complication; instead, the registered cause of death was chronic hepatitis. According to the WHO, '*deaths from any cause related or aggravated by pregnancy or its management during pregnancy and childbirth or within 42 days of termination of pregnancy*' are to be counted as maternal mortality.[534] It is therefore very likely that maternal mortality is underrepresented in the dataset. Infectious diseases, on the other hand, may be overrepresented, as in many occasions the registered causes of death of women who had died shortly after giving birth were infectious diseases.

Nevertheless, deaths in childbirth did not disadvantage women to such an extent that they had higher risks of dying compared to men in Maastricht. This disadvantage did not only stem from higher mortality among men as a consequence of external factors such as accidents, but also from mortality rates of airborne infectious diseases being much higher among men. Was the hazardous work environment of men to blame? Would socioeconomic inequalities also be more pronounced among adult men because of their overall higher mortality as a consequence of airborne infectious diseases?

534 Definition of maternal mortality as found on the World Health Organization webpage: https://www.who.int/data/gho/indicator-metadata-registry/imr-details/4622, consulted on 17-02-2022

Unfortunately, the data do not allow for a calculation of cause-specific mortality rates by socioeconomic class. However, with a multinomial logistic regression analysis we are able to probe the emergence of changes in epidemiological patterns. This will allow for a deeper understanding of the potentially emerging inequalities in the course of the health transition, in times of changes in the disease environment or when remedies become increasingly available.

6.6 Data & methods

In order to find which groups of people gained an advantage in the process of the health transition at first, based on changing epidemiological patterns, it is crucial to first define what would be perceived as an advantage in the analysis. Most mortality in a high mortality regime is the result of infectious diseases, while in a low mortality regime infectious diseases have largely receded and non-infectious diseases remain. An earlier onset of changing epidemiological patterns towards a dominance of non-infectious diseases within a certain group may therefore indicate an advantage for that subpopulation at large. More concretely, for both female and male mortality the age at death was moreover higher on average for non-infectious mortality compared to infectious mortality.[535]

A multinomial logistic regression analysis will help assess whether such advantages indeed emerged. The multinomial logistic regression analysis allows for an analysis of the different cause-specific disease patterns among specifically characterised groups of all deceased adults within the research period. It provides the odds ratio for a particular deceased person of having died of a specific cause-of-death category compared to another cause-of-death category. It also allows for comparisons between specific socioeconomic groups; more concretely, it would provide the odds ratio for a deceased person from, for example, the elite of having died of an airborne infectious disease instead of a non-infectious cause of death, compared to a deceased individual from another socioeconomic group, such as the unskilled workers. The model covers the entire population instead of only a sample and is therefore not just a model, but a direct representation of the Maastricht situation. Running such a model has a clear advantage over presenting only the percentages of deaths from each cause for every subgroup in the population. Namely that the model can give an estimation on whether these differences are statistically significant, or more likely the result of mere chance.

The dependent variable of the model, the cause-of-death variable, comprises three categories: Airborne infectious diseases, other infectious diseases and non-infectious causes of death. The categories of the eleven-category model had to be collapsed in order to ensure enough data power. The omnipresent airborne infectious diseases are the most important category, with a shared aetiology to distinguish it from other causes of death. Other infectious diseases accounted for a much smaller part of the total mortality rates, but are important to keep separate in the analysis due to their different aetiology from airborne infectious diseases and non-infectious diseases. The non-infectious disease

535 A t-test showed that the differences in average mortality were indeed statistically significant.

group is also important to distinguish, as a relative increase in these causes of death may be expected in the course of the health transition. Since the cause-of-death patterns differed between the genders, the analyses were stratified according to gender.

Time, socioeconomic status, an interaction term for socioeconomic status and time, and birthplace were included as independent variables. Marital status was included initially in both analyses. However, for men this appeared to be statistically insignificant in a basic model, while for women this was statistically significant. The variable marital status was therefore only included in the final analyses for women, distinguishing between never married, married, or divorced/widowed. Due to the healthy migrant effect, the expectation is that the proportional mortality also differed between the native-born Maastricht population and the part of the population migrating into the city.[536] Birthplace was therefore included in the initial model to test whether there were statistically significant differences, which were confirmed for men, yet for women they disappeared in a more sophisticated model. Migration was therefore only included in the final analysis for men, in which it was used to stratify according to migrants and native-born. Time was included as a periodical variable in order to provide the most straightforward, yet specific enough interpretation. The final model contains a five-period categorisation: 1864-1879, 1880-1899, 1900-1919, 1920-1939 and 1940-1955. However, to check for the robustness of the model, I ran several models with a different periodisation, such as one based on ten-year periods and separating the wartime periods.

The classification and interpretation of socioeconomic status is based on occupations, using HISCO and HISCLASS.[537] The occupations were the only available indicator of socioeconomic status within the dataset, but for historical populations this is the preferred indicator of socioeconomic status.[538] Even if available, education as an indicator of socioeconomic status may not yet be a very discriminatory factor, since the majority of the population only received primary education. The HISCO and HISCLASS system were used because they are a widely used and straightforward system. The classification of socioeconomic status according to HISCLASS for adults was, however, not as unchallenging as it may seem. Firstly, the interpretation of historical occupations can be a complicated matter; people may have wanted to create a better image for themselves, calling themselves a merchant (*koopman* in Dutch) instead of a 'simple' peddler (*venter*). In many cases the trade was however specified, which may increase the accuracy of the classification.

Secondly, the fact that not everyone remained in the same trade for the entire duration of their working life may be a pitfall. It was not uncommon to work on the same factory floor for your entire working life in Maastricht, but some people did have an upward career. Especially for men, for which we used occupation at death, the listed occupation was likely the one at the height of their career. As a result, we miss out on possible changes throughout their career, which may have influenced one's health. The converse is also possible: Some factory workers may have ended their career as a day labourer instead of

536 See for the healthy migrant effect Alter and Oris, 'Childhood Conditions, Migration and Mortality'; Kesztenbaum and Rosenthal, 'The Health Cost of Living in a City.'

537 Van Leeuwen and Maas, *Hisclass. A Historical International Social Class Scheme*; Van Leeuwen, Maas, and Miles, *Hisco. Historical International Standard Classification of Occupations*.

538 Bengtsson and Van Poppel, 'Socioeconomic Inequalities in Death from Past to Present,' 348.

as the skilled worker they had been for the majority of their working life.[539] This therefore makes the boundary between skilled and unskilled workers somewhat artificial, which should be taken into account in the interpretation of the results. Moreover, by creating categories that are large enough to allow for meaningful distinctions between the groups and enough data power for the analyses, I aim to minimise any bias as much as possible. The elite and middle class were therefore collapsed into one group, which will again be referred to as the upper classes. The second and third categories are skilled and unskilled workers, and a final residual category contains the unknown occupations and farmers.[540]

In the case of men, it was fairly straightforward to classify their socioeconomic status on the basis of their occupation at death. Finding a good indication of socioeconomic status for women was more demanding. Even though women did work the factory floors of Maastricht, not all of them continued doing so until their death, nor were their occupations always registered if they did remain working.[541] As a consequence, of the total adult female Maastricht population present in the dataset within the research period, only a rough 20% had an occupation at the time of death. It was possible to find an indication of occupation based on the occupation of the husband for an additional 64%.[542] Instead of simply using the occupation of the husband at his death, I used the occupation of the husband I could find in the documentation of the civil registry closest to the actual death of the woman. In this way, the listed occupation represents the socioeconomic status of the woman as accurately as possible. In the 16% of unmarried woman without an occupation, the occupation of the father, or in rare occasions the head of the household, was used as an indication. In the case of young women who were still living with their parents this may be quite reliable, but for older women this would be less the case. However, it does provide some information on whether someone belonged to the working classes, or was a wealthy heiress instead. Since marital status is included in the final model for women, this particular group of unmarried women can be separated from the groups for which there is a more reliable indication of socioeconomic status. Table I presents the number of individuals used in the analysis according to their socioeconomic status.

539 Van Rossem, *Bruxelles Ma Belle, Bruxelles Mortelle. An Investigation into Excess Mortality in Brussels at the Turn of the Twentieth Century*.

540 Farmers were such a small group that it would only cause a loss of data power to make a separate category for them.

541 Gales et al., *Het Burgerlijk Armbestuur; Knotter, Arbeid Van Vrouwen in Limburg in De Twintigste Eeuw. Een Stille Revolutie*; Schmidt and van Nederveen Meerkerk, 'Reconsidering the 'Firstmale-Breadwinner Economy': Women's Labor Force Participation in the Netherlands, 1600–1900.'

542 The occupation of the husband was found in the available, digitized civil registry document closest to the death of the woman. It was relatively easy to browse information from marriage certificates and death certificates either within the dataset (information from the death certificate of the husband or of a child when the death of a child was the closest event to the death of the woman of interest), or on WieWasWie.nl (information from marriage certificates from the couple itself, or from their children).

Table 6.1 Dataset socioeconomic status, marital status and migration characteristics

		Men	Women
Socioeconomic status	*Upper classes (elite + middle class)*	1391	1241
	Skilled workers	2414	1913
	Unskilled workers	1280	1019
	Unknown and farmers	789	426
Occupation based on	*Occupation at death*	5085	887
	Occupation of husband		2796
	Occupation of father		608
	Occupation other (head of the household or mother)		13
	Occupation unknown	789	295
Marital status*	*Married*	3310	2840
	Unmarried	2364	1505
	Divorced	19	15
	Widowed	181	239
Migrants	*Maastricht native*	3506	2711
	Born in Limburg	1096	895
	Born outside of Limburg	1272	993
Total n		**5874**	**4599**

*Meaning marital status at death. In some cases individuals had been divorced or widowed before. However, if they were married at death, their status was regarded as married.

6.7 Changing cause of death patterns

For the largest part of the nineteenth and early twentieth century, the single most lethal disease affecting the adult population of Maastricht was tuberculosis. Combined with other airborne infectious diseases, around 50% of all deaths for both men and women were caused by airborne infectious diseases until the start of the 1920s. However, some men and women were able to escape this faith earlier than others. Thus, when the mortality patterns of a particular subgroup within the population started to change from predominantly airborne towards more non-infectious diseases first, this subgroup had a head start in the process of the health transition. In line with the model Clouston et al. presented,[543] this advantage was a temporary one, in which a privileged group gained the resources to conquer this type of diseases first. In the case of tuberculosis and airborne infectious diseases, the expectation is that the elite gained an advantage first, due to their easier access to resources such as improved nutrition, housing quality and medical knowledge; factors which greatly influence the exposure and resistance to airborne infectious diseases.

To start with the group that suffered the most from these airborne infectious diseases – men – the main results of the multinomial logistic regression are displayed in Table 6.2. For the sake of clarity, the table contains only the odds ratio for differences between

543 Clouston et al., 'A Social History of Disease.'

socioeconomic status, which is the main variable of interest.[544] The results for migrants and Maastricht natives are displayed in different columns. To further illustrate the results, Figures 6.3 provide a visualisation of these results. The lines represent the proportional deaths of each socioeconomic group of airborne infectious diseases within the total of airborne infectious diseases and non-infectious diseases for the Maastricht native-born men (Figure 6.3a) and migrants (Figure 6.3b). Figures 6.3 clearly show the differences between migrants and native-born men.

Whereas differences between the classes can be observed for the native-born men, there are far less distinct cause-of-death patterns between the classes for the migrants. The migrants also appear to have lower proportional mortality due to airborne infectious diseases compared to native-born men. The healthy migrant effect could explain the differences, since the migrants would be less prone to die of airborne infections due their better health, and thus if they died they probably died of less preventable non-infectious diseases. However, the observed lower proportional airborne mortality may also be a result of return migration of the migrant men. Migrants suffering from tuberculosis in Scotland, for example, returned home when they needed care after contracting the disease.[545] Consequently, the migrants suffering from tuberculosis are less included in the mortality statistics of the city where they had been working. The migrants who did die in that city may therefore misrepresent the health of the total group of migrants.

The larger proportional share in airborne infectious diseases during the 1920s and 1930s among the migrant men without an occupation diverts from the general trend. Although it is not possible to determine what caused this with certainty, it may have been related to the economic crisis of 1929 and the subsequent years. Instead of only the healthiest people migrating to find work, men with lesser constitutions may also have searched for new occupations further away. Apart from that group, there were no substantial nor statistically significant differences between the socioeconomic classes in cause-of-death patterns among migrant men.

Among native-born Maastricht men, this was quite different. In the period 1864 until 1879 only small differences existed in epidemiological patterns between the upper classes on the one hand and the working classes on the other hand. The diversion between the lines in Figures 6.3 is only small, and none of the odds ratios of the workers indicate statistically significant differences from the upper classes. The residual socioeconomic status group is a clear exception to that, with significantly lower proportions of airborne infectious diseases. During the next period, inequalities start to emerge, due to a proportional increase in airborne infectious causes of death among the deceased from the working classes, while the deceased from the upper classes show a small decline. The differences between the upper classes and the skilled workers become nearly statistically significant in this second period, and are statistically significant at the level of $p > 0.05$ after the turn of the century. During the first decades of the twentieth century, a deceased skilled worker had a 57% higher chance of having died of an airborne infectious disease instead of a non-infectious cause of death, compared to someone who had died from

544 Results for the entire model are available upon request.

545 Alice Reid and Eilidh Garret, 'Mortality, Work and Migration. A Consideration of Age-Specific Mortality from Tuberculosis in Scotland, 1861-1901,' *Historical Life Course Studies* 6, no. Special issue 1 (2018).

the upper classes. While unskilled workers have higher proportional airborne mortality in these two periods as well, the differences between them and the upper classes are not statistically significant. During the 1920s and 1930s, the differences between the socioeconomic classes converged as regards proportional airborne mortality compared to non-infectious diseases.

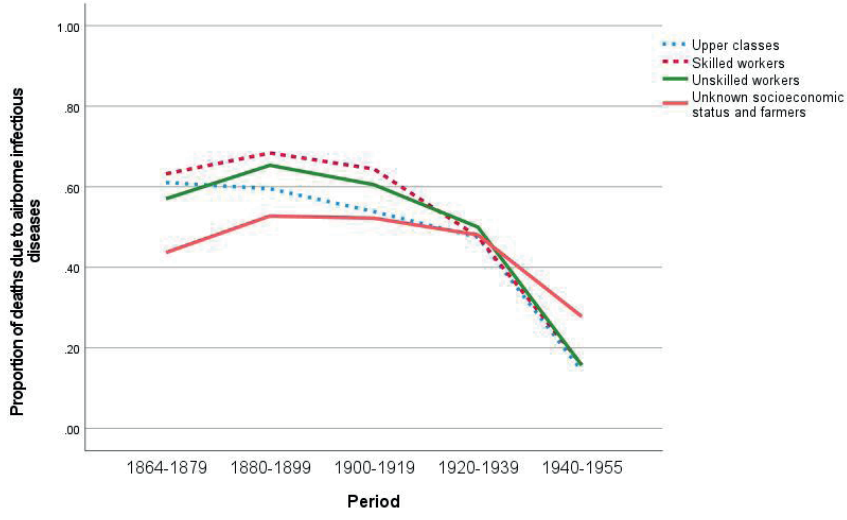
A remarkable outlier in this analysis is the 'residual' socioeconomic status group. During the period from 1864 until 1879, they experience statistically significant lower proportions of airborne infectious diseases within the total of airborne infectious and non-infectious diseases compared to the upper classes. While these differences between the residual group and the upper classes lose their significance in the following periods, the proportional airborne mortality among the residual group remains significantly lower compared to the working classes during the final two decades of the nineteenth century, and also in the first two decades of the twentieth compared to the skilled workers. From the 1920s onwards, they converge with all the other groups as well. If this group were to consist predominantly of farmers, the explanation would be quite logical. Farmers are known for their lower mortality from airborne infectious diseases. However, the group consists mainly of men without an occupation. What causes this group to have such a lower proportional airborne mortality? Would this stem from a registration practice? Having no occupation could mean that someone was in need of a job, retired, or living off inherited fortunes. Perhaps in the earlier decades of the research period, men who had no occupation registered on their death certificates were the ones living of inherited wealth and were therefore less prone to die of airborne infectious diseases when they died. Perhaps they were actually the 'real' elite?

For women, the picture was slightly different. First, it immediately became clear that whereas marital status had no effect on disease patterns for men, it did have a significant effect on women. Unmarried women had statistically significant higher chances of having died of an airborne infectious disease instead of a non-infectious cause of death when compared to married or divorced/widowed women. Second, although there were at times large differences in epidemiological patterns between the social classes, just as was the case among men, the timing of these differences was different for both genders. Third, whereas migration was an important factor for men, this was less the case for women. In a very simplistic model it appeared to be statistically significant; however, in the final model, after including all the variables and the interaction term of socioeconomic status and time, it lost its statistical significance.

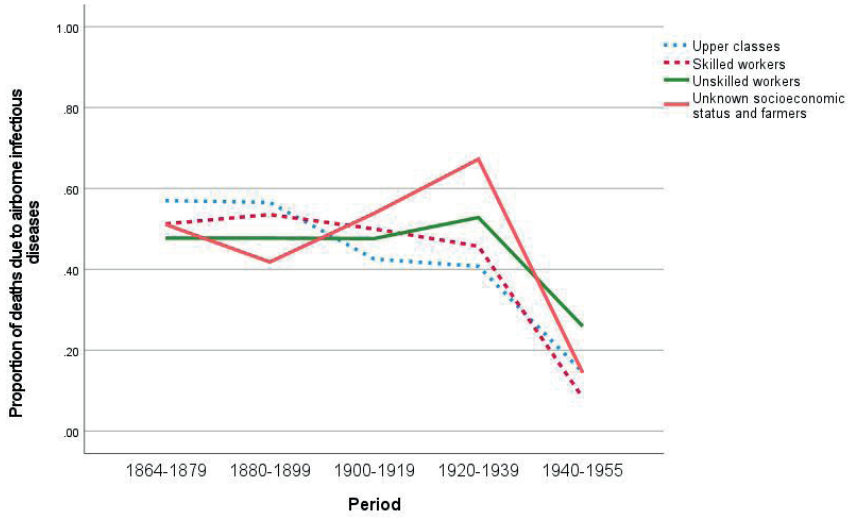
Table 6.2. Results of the multinomial logistic regression analysis for airborne infectious diseases compared to non-infectious diseases for Maastricht-born men, Maastricht 1864-1955

Period	Socioeconomic status	Odds ratio Maastricht-born	p-value	Odds ratio migrant	p value
1864-1879	*Upper classes*	ref.		ref.	
	Skilled workers	1.096	0.709	0.793	0.355
	Unskilled workers	0.847	0.547	0.689	0.197
	Unknown socioeconomic status and farmers	0.495	0.020	0.788	0.505
1880-1889	*Upper classes*	ref.		ref.	
	Skilled workers	1.471	0.070	0.886	0.561
	Unskilled workers	1.282	0.279	0.702	0.180
	Unknown socioeconomic status and farmers	0.759	0.321	0.552	0.059
1900-1919	*Upper classes*	ref.		ref.	
	Skilled workers	1.577	0.029	1.350	0.209
	Unskilled workers	1.351	0.175	1.227	0.501
	Unknown socioeconomic status and farmers	0.952	0.849	1.575	0.164
1920-1939	*Upper classes*	ref.		ref.	
	Skilled workers	1.006	0.981	1.223	0.447
	Unskilled workers	1.106	0.706	1.627	0.138
	Unknown socioeconomic status and farmers	1.023	0.939	2.982	0.001
1940-1955	*Upper classes*	ref.		ref.	
	Skilled workers	1.125	0.706	0.530	0.079
	Unskilled workers	1.116	0.768	2.052	0.063
	Unknown socioeconomic status and farmers	2.308	0.022	0.989	0.976

a. Native-born Maastricht men



b. Migrant men



Figures 6.3a and 6.3b Proportional mortality of airborne infectious diseases, as part of the combined group of deaths due to airborne infectious diseases and non-infectious causes of death, by socioeconomic status of the adult male Maastricht population, 1864-1955

Source: Maastricht Death and Disease Database

The analysis for women is therefore a slightly adjusted version of the one for men. In the initial general model, it turned out that there were mainly statistically significant differences between married and unmarried women, while the cause-of-death patterns of divorced and widowed women did not differ statistically significant from those of married women. Moreover, the group of divorced and/or widowed women was very small (Table 6.1). In order to account for the substantial differences between married and unmarried women, the multinomial logistic regression analyses were stratified according to married and unmarried. Such a separate analysis for the divorced/widowed group was impossible because of the lack of sufficient data, but they did not behave statistically significantly different from married women in the more general model. Migration was not included in the final model, nor presented here. However, all the tables and figures have been checked with the inclusion of migration, but no substantial differences were found between the results presented here and the results including migration.[546]

The results of the analysis of married women can be found in Table 6.3, and of unmarried women in Table 6.4. Figures 6.4a (married women) and 6.4b (unmarried women) again illustrate these results by presenting the proportional deaths of each socioeconomic group of airborne infectious diseases within the total of airborne infectious diseases and non-infectious diseases. Clearly, deceased married women had

546 There is one exception to be mentioned here. In the period 1880-1899, the married, migrated women in the unskilled workers group behaved more like the elite, while in the model excluding migration married women from the group of unskilled workers found themselves in a position between the skilled workers and the elite. In the early twentieth century, the married migrated women behaved more in synch with the married women from the skilled workers group again.

lower chances of having died of airborne infectious diseases instead of non-infectious causes of death regardless of their socioeconomic class, for almost the entire research period. An earlier, general model (not presented here) showed that these differences were indeed statistically significant from the 1880s onwards until the 1940s. Since higher proportions of deaths due to airborne infections are an indication of lesser resources to resist infection or battle the infection among the specified group, it appears that unmarried women had a clear disadvantageous position in Maastricht. This effect was especially strong during the early twentieth century, when the proportional mortality due to airborne infections declined for married upper-class women and married women from the skilled-worker group, while the unmarried women in these two groups showed an actual increase in proportional airborne infectious mortality. A logical explanation for a higher proportional airborne mortality among unmarried women would be that married women would naturally have higher proportions of non-infectious mortality because they would suffer more from mortality related to childbearing. Yet even when this particular cause-of-death category was removed from the analysis, deceased unmarried women still had a 45% higher chance of having died of airborne infectious diseases instead of non-infectious causes of death, compared to married women. A selection effect for marital status may offer another explanation for the higher chances of unmarried women to have died of an airborne infectious disease. Other studies on historical populations have shown that men and women were selected for marriage based on height – being taller increased the chances of marrying – and on weight – men with slightly higher weight had higher chances of marrying.[547] A similar selection effect may have existed for health, especially since height and weight are often indicators of health. In the case of the most common cause of death among adults – tuberculosis – an extensive period of sickness, wearing the patient down, often preceded death. As a result, a sickly woman may have had lower chances of getting married.

Within the group of unmarried women, differences in cause-of-death patterns between the different social strata were however small. Only for the residual socioeconomic status group is the pattern quite different. The lack of substantial differences between the classes among unmarried women can partially be caused by coding issues. For half of the unmarried group, the socioeconomic status is based on the occupation of the father. However, the other half of unmarried women had an occupation of their own, and the occupation of the father gives at least a rough indication of the social class, which should be sufficient in working with large enough socioeconomic groups. What causes the residual group to fare better and have lower proportional airborne mortality remains unclear, since it is not possible to distinguish exactly who had an occupation of their own, and who did not.

The results for married women present us with a clearer pattern. Among this group, distinct differences in cause-of-death patterns occurred between the different socioeconomic groups (Figure 6.4a). These differences started to express themselves earlier than they did for men, namely in the final two decades of the nineteenth century. Whereas in the late nineteenth century married women from the class of skilled workers

547 Jörg Baten and John E. Murray, 'Women's Stature and Marriage Markets in Preindustrial Bavaria,' *Journal of Family History* 23, no. 2 (1998); John E. Murray, 'Marital Protection and Marital Selection. Evidence from a Historical-Prospective Sample of American Men,' *Demography* 37, no. 4 (2000).

fared worst regarding airborne proportional mortality, in the early twentieth century women from the group of unskilled workers had the largest airborne proportional mortality. The differences between the upper classes on the one hand, and the unskilled and skilled workers group on the other hand, remained statistically significant from the 1880s onwards through the 1910s. The differences were even almost statistically significant in the period thereafter for the skilled-worker group and the upper classes. The higher proportional mortality among women from the unskilled workers in the final period (1940-1955) turns out to be mainly the result from circumstances related to the Second World War.[548]

Table 6.3. Results of the multinomial logistic regression analysis for airborne infectious diseases compared to non-infectious diseases for married women, Maastricht 1864-1955

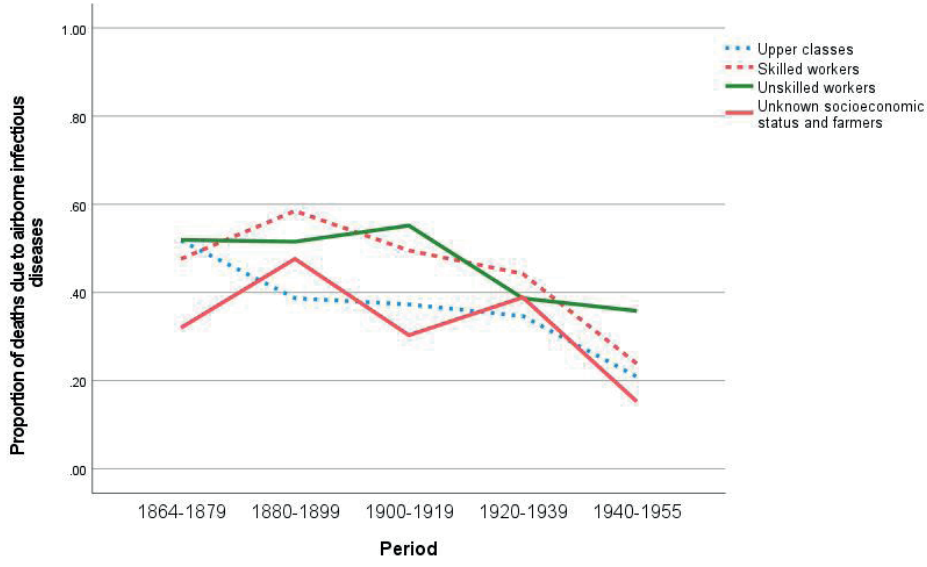
Period	Socioeconomic status	Odds ratio	p value
1864-1879	*Upper classes*	ref.	
	Skilled workers	0.862	0.533
	Unskilled workers	1.024	0.930
	Unknown socioeconomic status and farmers	0.446	0.085
1880-1889	*Upper classes*	ref.	
	Skilled workers	2.232	0.000
	Unskilled workers	1.684	0.041
	Unknown socioeconomic status and farmers	1.439	0.311
1900-1919	*Upper classes*	ref.	
	Skilled workers	1.650	0.020
	Unskilled workers	2.069	0.003
	Unknown socioeconomic status and farmers	0.731	0.457
1920-1939	*Upper classes*	ref.	
	Skilled workers	1.496	0.074
	Unskilled workers	1.190	0.524
	Unknown socioeconomic status and farmers	1.200	0.639
1940-1955	*Upper classes*	ref.	
	Skilled workers	1.185	0.555
	Unskilled workers	2.111	0.031
	Unknown socioeconomic status and farmers	0.681	0.373

548 According to a model with the wartime periods separated, not presented here.

Table 6.4. Results of the multinomial logistic regression analysis for airborne infectious diseases compared to non-infectious diseases for unmarried women, Maastricht 1864-1955

Period	Socioeconomic status	Odds ratio	p value
1864-1879	*Upper classes*	ref.	
	Skilled workers	1.044	0.895
	Unskilled workers	0.765	0.472
	Unknown socioeconomic status and farmers	0.653	0.260
1880-1889	*Upper classes*	ref.	
	Skilled workers	1.146	0.646
	Unskilled workers	1.560	0.185
	Unknown socioeconomic status and farmers	0.691	0.343
1900-1919	*Upper classes*	ref.	
	Skilled workers	1.309	0.330
	Unskilled workers	0.790	0.429
	Unknown socioeconomic status and farmers	0.324	0.009
1920-1939	*Upper classes*	ref.	
	Skilled workers	1.169	0.566
	Unskilled workers	1.154	0.665
	Unknown socioeconomic status and farmers	0.679	0.383
1940-1955	*Upper classes*	ref.	
	Skilled workers	1.283	0.560
	Unskilled workers	1.076	0.876
	Unknown socioeconomic status and farmers	0.412	0.193

a. married women



b. unmarried women

Figures 6.4a and 6.4b Proportional mortality of airborne infectious diseases, as part of the com-bined deaths due to airborne infectious diseases and non-infectious causes of death, by socio-economic status of the female population of Maastricht, 1864-1955

Source: Maastricht Death and Disease Database

The inequalities in cause-of-death pattern for married women start to appear simultaneously with the initial decline in female mortality from the 1880s onwards. For men, too, the patterns for the different socioeconomic strata start to diverge when the decline in male mortality starts. For men, this decline starts later than for women, around 1895 (Figure 6.1). The effect of this decline can be found in the multinomial logistic regression model for men in the period of 1900-1919, due to the periodisation. The results thereby demonstrate that the most substantial inequalities according to socioeconomic status in cause-of-death patterns appeared when the decline in mortality started, indicating a disadvantageous position for the working classes, since they continued to suffer from the infectious diseases which occur in a high mortality regime.

6.8 Important factors in the adult mortality decline in Maastricht

The previous section has shown that mortality decline for men and women did not occur simultaneously, nor did the inequalities among socioeconomic groups in cause-of-death patterns occur in tandem for the two genders. Could that mean that different factors played a role in the decline in mortality for men and women? The historical context of Maastricht, combined with the information we have on cause-specific mortality, may deepen our understanding of the adult mortality decline in Maastricht.

Since the adult mortality decline was predominantly a consequence of the decline in airborne infectious diseases and their lethality, we must look to improved nutritional

status, improved housing conditions (overcrowding) and probably also better working conditions on the factory floors as important factors.[549] The multinomial logistic regression analyses moreover demonstrate the graduality of the changes in cause-of-death patterns, being unequally dispersed. The factors influencing mortality decline must therefore have had a number of discriminating characteristics. If they would have affected the entire population at the same time, which Molitoris has shown for water and foodborne infant mortality in Stockholm,[550] the divergence of cause-of-death patterns between the classes would not have been as strong.

The first and foremost potentially discriminatory factor influencing especially airborne infectious diseases would be nutrition. McKeown has received his fair share of critique for his theory on the role of nutrition in mortality decline, but we should not throw away the baby with the bathwater. Scholars have argued more recently that while nutrition may not have been the only important factor in the decline of mortality, it was definitely an important factor in synergy with other factors.[551] The main airborne cause of death was tuberculosis, for which effective treatment only became available during the Second World War. In the Netherlands, as in other countries, tuberculosis mortality had already declined before the emergence of effective treatment. The main factors behind the drop in tuberculosis mortality would have been improved nutrition, improved housing conditions and improvements in the field of hygiene.[552] Yet for the Netherlands as a whole, De Beer argues that improved nutrition had only a minor or even no effect at all on the mortality decline in the late nineteenth century.[553] After all, modern standards regarding the available amount of calories and protein per head of the population had already been reached early in the second half of the nineteenth century.[554] The specific circumstances in Maastricht may have been different, however, especially for adults. National mortality decline most substantially as a result of the decline in infant mortality, which indeed was mostly related to the improvement of hygienic feeding *practices*, and not the amount of calories. The decline in adult mortality has been much less researched in the Netherlands, with adult mortality possibly being more affected by nutrition.

Provincial availability of beef improved in the province of Limburg after the turn of the century and even exceeded the national average availability of beef per capita by the 1890s.[555] Consumption of meat in particular may have been restricted to the higher classes, only slowly expanding to other classes when wages increased. Gales'

549 'The Relationship of Nutrition, Disease, and Social Conditions: A Graphical Presentation.'; Robert William Fogel, *The Escape from Hunger and Premature Death, 1700-2100. Europe, America and the Third World* (Cambridge: Cambridge University Press, 2004); Harris, 'Public Health, Nutrition, and the Decline of Mortality'; McKeown, *The Modern Rise of Population*.

550 Molitoris, 'Disparities in Death.'

551 Fogel, *The Escape from Hunger and Premature Death, 1700-2100*; Harris, 'Public Health, Nutrition, and the Decline of Mortality.'

552 Ernest Hueting and Agnes Dessing, *Tuberculose. Negentig Jaar Tuberculosebestrijding in Nederland* (Zutphen: Walburg Press, 1993).

553 De Beer, *Voeding, Gezondheid En Arbeid in Nederland Tijdens De Negentiende Eeuw*, 85.

554 Ibid.

555 Ibid., 64.

characterisation of the Maastricht population reinforces the idea that increased meat consumption in the nineteenth century may have been restricted to the wealthy few only. Until the turn of the century, between 50% and 60% of the Maastricht population found itself on the wrong side of the poverty line.[556] The majority of the population in Maastricht was probably already content if they could afford basic needs such as bread and potatoes. Only from the twentieth century onwards did real wages increase and a larger share of the Maastricht population became able to properly fend for themselves.[557] Improved meat nutrition could explain an advantage gained by upper-class married women, since the timing of improved nutrition dovetails with the timing of changing cause-of-death patterns for this group.

Men of all classes and women from the working classes, on the other hand, had to wait for improvements to start later. Upper-class men started to diverge from the cause-of-death pattern of the men from the working classes in the early twentieth century. Since the divergence is based on differences in epidemiological patterns, most importantly the proportional mortality of airborne infectious diseases and non-infectious diseases, a proportional lower mortality due to airborne infectious diseases among the upper-class men might have actually been the result of an increase in mortality due to non-infectious diseases. In England, it appeared upper-class men were not better off than the working classes, due to their health-depriving lifestyles which consisted of heavy, fatty diets, smoking and alcohol.[558] However, the change in epidemiological patterns among adult men coincided with the initial decline in mortality, thus making an increase in mortality due to non-infectious diseases among upper-class men not a very likely explanation for the changing epidemiological patterns.

The working classes, both men and women, had to wait until the 1920s before their cause-of-death patterns truly started to change. According to Gales, more people slowly but steadily ended up on the right side of the poverty line, towards what was called the socioeconomic position of '*gewone mensjes*' (ordinary people, who previously had been in poverty), from the twentieth century onwards.[559] Lower-middle-class families probably experienced this expanded availability of resources earlier, causing them to gain a head start in the changing epidemiological patterns. The working classes profited from these improvements shortly thereafter. The worst position for men who belonged to the skilled workers does not adhere to the idea of a social gradient, but can be explained from the particular historical context of Maastricht. The majority of the working classes had a position in one of the factories, the majority being active in the pottery and glass industries.[560] In these industries, the occupations which required skilled work were the most harmful. In the pottery industry, these were the '*vormers*', the moulders. Not only did the work require physical strength, but the constantly lingering dust from the pottery often led to affections of the lungs. Due to gender segregation in the workforce, only men could start a career as moulder. Once they had acquired the status of a fully trained

556 Gales et al., *Het Burgerlijk Armbestuur,* 152.

557 Ibid., 153.

558 Razzell and Spence, 'The Hazards of Wealth: Adult Mortality in Pre-Twentieth-Century England.'

559 Gales et al., *Het Burgerlijk Armbestuur*, 151.

560 De Groot, *Fabricage Van Verschillen*, 214-16.

moulder, the wages were fairly decent, although at the expense of their health. The men had to enter the profession as young boys, from the age of 12 onwards, and follow a long training process. By the time they had reached the age of forty, they had gained a fair wage in combination with an affection of the lung, often noted down as '*longtering*', tuberculosis of the lung.[561] The gender segregation on the work floor may explain why among women the worst position in connection to epidemiological patterns was not to be found among the skilled workers; they were not allowed to perform these most health-hazardous jobs.

Social medicine and medical care increased in the early twentieth century as well, as described in chapter 3. Important to mention here is that tuberculosis care was the spearhead of the *Gemeentelijke Geneeskundige Dienst* (*GGD*, Municipal Health Service). The Green Cross already provided care for sufferers of tuberculosis from 1913 onwards, and in 1920 the GGD adopted this task. They opened a consultation bureau for tuberculosis and sent affected people to sanatoria in the countryside.[562] Although this may not have cured the illness, it may have stunted the constant circulation of the disease in the population.

6.9 Conclusion

The aim of this chapter on adult mortality was to deepen our understanding of the combined effects of socioeconomic status and gender on inequality in mortality. By tracing changes in epidemiological patterns for distinct groups in the population of Maastricht, I was able to show how different inequalities in health, as measured by mortality, emerged in the course of the health transition for adults. The existence of these historical inequalities in health have been widely debated in the literature. Recently the historical socioeconomic inequalities among adults have been on the chopping block. Older theories either advocate a constancy hypothesis, with enduring socioeconomic inequalities in health over time, or the divergence-convergence hypothesis, which states that inequalities first increased before diminishing again. Recent research has nonetheless pointed out that these mortality inequalities along class lines among adults cannot always be found for the nineteenth century. Health inequalities are therefore a recent phenomenon.

The picture for gender inequalities in historical adult mortality is equally complex. In England and the Netherlands, excess female adult mortality is mainly a rural phenomenon, due to a worsened bargaining position of women within the household as a consequence of modernisation in agriculture. Women in urban areas would have had a relatively better position compared to men, since they had more opportunities to contribute to the household income and hence had a better bargaining position. Yet in France and Belgium, excess female mortality was found in urbanised regions as well. Deteriorating circumstances as a consequence of the industrial revolution would have affected women disproportionally, resulting in their excess mortality.

561 Ibid., 230-76.

562 Evers and Ubachs, *Voorkomen Is Beter Dan Genezen*, 46.

A number of studies have also combined gender and socioeconomic status, in order to study the combined effect of these factors on mortality. Among men, socioeconomic status may effect overall mortality less than among women, on account of excessive life styles being more present among wealthy men: Their eating, smoking and drinking habits counteracted the advantages their privileged position provided them. The fact that overall mortality may level cause-specific differences in mortality highlights the importance of using cause-specific data in studying historical inequalities in mortality. Only few studies have adopted such an approach, especially in the case of adult mortality. The lack of sufficient data is the first and foremost reason for the scarcity of these studies. The dearth in adult mortality studies specifically may also be a consequence of the fact that adult mortality decline was not the protagonist in mortality decline in the process of the health transition; that position was reserved for infant and early childhood mortality.

The Maastricht mortality decline experience was definitely characterised by certain health inequalities, advantages gained and disadvantages overcome. The mortality decline was predominantly a result of a decline in airborne infectious diseases, among both men and women. The first important conclusion of this chapter concerns the interaction between gender and socioeconomic status. While socioeconomic inequalities emerged in the initial phase of the adult mortality decline, the timing of when this initial phase began was different for the two genders. Once the decline took off for one of the two genders, the upper classes started to experience lower proportional mortality due to airborne infectious diseases, as opposed to non-infectious diseases, compared to the working classes. Since infectious diseases correlate with a high mortality regime according to the epidemiological transition theory, and airborne infectious diseases are associated with poor living circumstances and poor nutrition, it is reasonable to conceive higher proportional airborne mortality among the deceased as a disadvantageous position, as opposed to non-infectious causes of death.

Upper-class married women started to gain an advantage over their working-class peers during the final two decades of the nineteenth century. Men had to wait a little longer, but upper-class men gained a similar advantage during the first two decades of the twentieth century. The working classes caught up to those with an advantageous position roughly during the 1920s and 1930s. The inequality patterns in mortality for Maastricht are therefore different from what Van Poppel and Van Gaalen have found for the Netherlands as a whole, namely that socioeconomic inequalities in adult mortality were present but small in the nineteenth century, and only diminished towards the end of the century. The results that Van Reek and Van Zutphen found for the Netherlands are more in sync, as they did find inequalities for male mortality in the 1890s. In Maastricht, the turn of the century saw the culmination of socioeconomic inequalities in adult mortality.

Secondly, other differences in gender existed as well. Until the First World War, there was clear excess male mortality. After the First World War, mortality rates for men and women became more or less equal, due to a decline in male mortality from 1895 onwards, and a short-lived increase in female mortality during the wartime years, mainly caused by a large number of Spanish flu cases among women.

A third distinction between male and female mortality was the marked difference in the impact of marital status. Whereas for men their marital status had little effect on their cause-of-death patterns, for women it mattered a great deal. Unmarried deceased

women had an especially larger proportional mortality due to airborne causes of death instead of non-infectious causes of death when compared to married women. Even when deaths related to childbearing were omitted, as they may cause higher proportional non-infectious mortality among married women, the unfavourable position of unmarried women held firm. A likely explanation for this phenomenon would be a positive selection in marriage for healthier women, who suffered less from draining airborne infections.

Fourthly and finally, the impact of migration also differed between the genders. For men, clear differences between men born in Maastricht and those born elsewhere appeared. These differences were in line with the healthy migrant effect; migrants had a substantial lower proportional mortality due to airborne infectious diseases in general until the decline in airborne mortality during the 1920s and 1930s. This phenomenon was not restricted to the upper classes: Men from the working classes also experienced lower proportional airborne mortality. Within the group of migrants, no substantial differences in cause-of-death patterns by socioeconomic status existed. Yet the healthy migrant effect observed here may have been a consequence of return migration as well, since in England migrants suffering from tuberculosis returned to their place of birth to be cared for.

The factors causing the decline in adult mortality in Maastricht cannot be established in a fully conclusive manner. However, the fact that it was mainly airborne infectious diseases that declined, and the existence of socioeconomic inequalities in the course of the health transition, can help to narrow down the potentially most influential factors. In the case of airborne infectious diseases, the improvement in nutrition in the late nineteenth century in Limburg and the increasing economic position of the Maastricht population at the start of the twentieth century have most likely contributed to adult mortality decline in Maastricht, since these occurred simultaneously with the decline. Yet the increasing preoccupation with the treatment of tuberculosis in the early twentieth century may have also aided further by restricting the circulation of tuberculosis. Yet the exact causes of the decline in tuberculosis mortality are difficult to establish.

Finally, the results indicate that the Swedish experience is not fully applicable to other European regions. Socioeconomic inequalities in adult mortality are not only a phenomenon of the past couple of decades. Whether socioeconomic status was a fundamental cause of inequality, or socioeconomic inequalities behaved more in a diverging-converging patterns, cannot conclusively be answered. The Maastricht data on adults allowed for the study of differences between airborne infectious diseases and non-infectious diseases, simply because 'enough' people died of them. The pattern for these causes of death shows a clear divergence-convergence pattern at the level of disease categories. However, it does not tell us whether new divergence-convergence patterns for other specific causes of death occurred prior to or after this wave. The cause-specific approach allowed for studying inequalities at a more specific level, whereas at an aggregated level these results might have been obscured. It has indeed turned out, though, that in some periods in the nineteenth and early twentieth century, certain groups in Maastricht society gained a temporary health advantage related to their socioeconomic status and/or gender.

CHAPTER 7

7

Conclusion

7.1 Main conclusions

The central aims of this dissertation were to examine the association between the mortality decline as part of the health transition on the one hand, and inequalities in health by age, socioeconomic status and gender on the other hand, and to understand the ways these inequalities evolved during the mortality decline. Who gained a health advantage early on in the health transition, and who lagged behind and continued to suffer from high mortality until the final stages of the health transition? These questions were answered by studying individual-level, cause-specific mortality, based on the unique sources available for Maastricht. With these sources, it was possible to approximate the effects of individual factors such as age, socioeconomic status and gender on changing epidemiological patterns. The health transition functioned as the fundamental conceptual framework for this study, due to its focus on the shift from predominantly infectious to non-infectious diseases in both mortality *and* morbidity, while accounting for the influence of social, economic *and* cultural factors on this transition. Thus, within the concept of the health transition, the importance of culturally-induced behaviour and religion are also fully acknowledged. The epidemiological transition theory can be viewed as a highly important element of the health transition, and has a strong methodological function in this study. The epidemiological transition is premised on the idea that a high mortality regime in historical populations is characterised by omnipresent infectious diseases, while a low mortality regime is reached when the infectious diseases have declined and given way to mortality due to non-infectious diseases. As such, the epidemiological transition functions as a baseline premise to estimate when changes from a high mortality to a low mortality regime have occurred, based upon changing epidemiological patterns. When specified mortality rates, for example by socioeconomic status, are not available, this method offers a solution to look into specific inequalities in the course of this transition. Since the epidemiological transition first and foremost affected infants, young children and adults, these age categories were studied in this dissertation.

The main focus of this study is therefore first the timing of changes in the health transition and the way these changes may have created inequalities across groups, and second the differences in mortality levels. In order to study these potential inequalities, the first question this dissertation seeks to answer is how the health transition took shape in Maastricht in the period from 1864 through 1955. When did it occur exactly, and more specifically, which causes of death were responsible for the mortality decline? Additionally, since the health transition did not take place for each age group at the same time, what were the age-specific pathways towards a low mortality regime at the end of the health transition? Next are questions on other individual characteristics: Did inequalities arise in the course of the health transition according to gender or socioeconomic status? And, finally, what can we learn about the determinants of these inequalities based on the cause-specific pattern of declining mortality?

This conclusion consists of two main elements. First the overarching conclusions will be discussed, in combination with how these contribute to the debate on historical inequalities in health. I will then broaden the view and discuss the scientific relevance of this study, and how that feeds into suggestions for the direction of future research in historical demography and medical history.

7.1.1 Inequalities in cause-specific mortality

The likelihood of meeting an early death, in times prior to the health transition, was fairly large for many. However, some were able to escape raging infectious diseases and prolong their lives. Socioeconomic status, gender and age are the three main factors studied in this dissertation which could potentially determine whether one might meet an early death. By finding that all three factors contributed to unequal pathways to the grave in Maastricht, this study contributes substantially to our understanding of health inequalities. However, the three factors did not always exert their influence on health inequalities in a similar fashion.

Age dictated a great deal of the disease hazards an individual was exposed to. Some causes of death occurred in all age categories discussed here (infancy, early childhood and adulthood), although the predominance of certain infectious diseases differed across ages. The factors influencing the decline in mortality could also differ among age groups, in particular when these factors were targeted at specific age groups. Age therefore determined when the pathways to death started to change, and thus when the mortality decline started. Adult mortality and early childhood mortality declined relatively early compared to infant mortality. The role of socioeconomic status on the shift towards a low mortality regime was fairly consistent. Socioeconomic inequalities emerged in the initial phase of the health transition. As such, socioeconomic status created fast tracks and congestions early on *en route* to a low mortality regime. Whereas socioeconomic status caused the emergence of disparities in the initial phase of the health transition and age category mainly dictated the start of the health transition, gender was a more long-lasting discriminatory factor; gender continued to be associated with higher levels of mortality among males after a low mortality regime had been reached. However, the influence of gender on the health transition was quite complex. Depending on the age group, gender affected overall mortality levels, yet without causing different epidemiological routes towards a low mortality regime. In other age groups, gender could be as important as age in determining the start of the health transition or the predominance of certain diseases. In the sections below these overarching conclusions will be articulated further.

7.1.2 Dynamics of socioeconomic inequalities

Over the past few decades, there has been a great deal of debate around the existence of socioeconomic inequalities in health during the health transition. Although it was assumed that socioeconomic status was either a constant discriminatory factor in health or that there was a converging-diverging pattern of socioeconomic health inequalities, recent studies on Swedish mortality have found no socioeconomic health inequalities before 1950. Yet Clouston et al. argued that although socioeconomic status is a constant discriminatory factor, its effects alternate from one individual disease to the next. This mechanism may cause some inequalities at the individual disease level to be obscured because other disease inequalities at a more aggregated level can outweigh them. In contrast to the recent Swedish studies, this study did find socioeconomic inequalities in mortality prior to 1950. However, the observed socioeconomic inequalities existed only temporarily, emerging during the initial phase of the transition, to recede again once the transition progressed.

The occurrence of socioeconomic inequalities was not limited to one specific age group; in fact all age groups experienced diverging epidemiological patterns according to socioeconomic status. This also holds true men and women. Among all age groups, the epidemiological pattern amongst the upper classes changed prior to change in the epidemiological pattern of the other classes. The upper classes therefore gained an early health advantage, since an earlier lower proportional mortality due to one of the predominant causes of death would imply that the pathway towards a lower mortality regime had been taken earlier. This created time lags in the onset of changes in epidemiological patterns, since other classes experienced these changes later. However, the emergence of socioeconomic inequalities depended on the cause of death. For infants and young children, the upper classes showed lower proportional mortality due to water and foodborne infectious diseases at the start of the decline of this cause-of-death category. Socioeconomic inequalities in airborne infectious diseases were not observed for young children, and only somewhat for infants, but were strongly observed for adults. Therefore, socioeconomic status as a fundamental cause of health inequalities as put forth by Link and Phelan, does not apply to Maastricht. However, Maastricht does seem to fit the mechanism proposed by Clouston et al., which emphasises that socioeconomic disparities start to appear at the moment when certain resources begin to emerge that are capable of curbing mortality from a particular disease.

The absence of socioeconomic inequalities at a cause-specific level in some instances, while being present for other causes of death, highlights a different important feature of the analysis of socioeconomic status. While social inequalities themselves are seen as one of the great injustices of the world, and rightly so, and are therefore a legitimate subject of study, the purposes of their analysis can be extended. The analysis of socioeconomic inequalities can also be used as an analytical tool to gain a better understanding of the determinants driving the mortality decline. In his study of Stockholm, Molitoris already explained that the absence of socioeconomic inequalities among infant mortality due to water and foodborne diseases was likely the result of an indiscriminate measure to fight these diseases. If socioeconomic inequalities did emerge when a specific cause of death declined, the factors effectuating the decline are more likely to have been restricted or targeted at specific groups. The occurrence of socioeconomic inequalities in the early phases of decline of a specific disease can therefore direct attention to a set of potential determinants. When this information is combined with the information on the specific cause of death, we gain a better understanding of the determinants of the mortality transition.

In this study the cause-specific analysis has produced the following insights concerning the determinants driving the mortality decline. First, from the occurrence of socioeconomic disparities in some disease and age categories, and the absence of these disparities in other categories, we can deduct that the determinants responsible for the decline in mortality were multifaceted and differed in their strength. Second, while in a given disease category, socioeconomic inequalities emerged in different age groups, the different timings of the decline for these age groups may indicate that the responsible determinants were dissimilar. This applies to the decline in water and foodborne infectious diseases for both infants and young children; in both age groups socioeconomic inequalities emerged, although at different times. It is therefore likely that the determinants influencing the decline in water and foodborne mortality were

not exactly the same for infants and young children. Moreover, from the emergence of socioeconomic inequalities for both infants and young children, we learn that these determinants must indeed have been restricted in their initial availability. Increased awareness of high infant mortality and new hygienic practices in taking care of infants reached the upper classes first. Only later did these ideas become more widespread. In that sense, cultural behaviour could be seen as an initially restricted determinant.

Socioeconomic inequalities did not always emerge in the decline of airborne infectious diseases. This was the case among young children in the 1920s, which could indicate that a socioeconomically non-discriminatory measure would have been responsible for the decline, or that multiple factors influenced the changing epidemiological pattern simultaneously. Otherwise, a privileged group of young children would have likely displayed a change in proportional mortality due to airborne infectious diseases prior to other socioeconomic groups of young children. However, the factors responsible for the decline in airborne mortality did have a discriminatory effect among infants. During the same period, the 1920s, infants from skilled workers had a higher chance of having died of airborne infectious diseases as opposed to non-infectious diseases, compared to infants in upper-class families. Adult mortality due to airborne infectious diseases had started to decline earlier, and continued to decline in the 1920s. Since the socioeconomic disparities became apparent in the initial phase of the decline, there were no longer significant differences in epidemiological patterns regarding airborne infectious diseases among adults in the 1920s. Thus, differences in the timing of decline in airborne infectious diseases among different age groups occurred as well, mainly between adults on the one hand and infants and young children on the other hand. Therefore, the determinants responsible for the decline in airborne mortality may have differed among age groups, or may have affected adults first.

The particular historical context may have generated a set of factors influencing the decline in mortality. These factors may have varied by disease and age. Within a specific age category these factors could (or could not) vary by socioeconomic status. Riley argued how potential determinants for mortality decline can alternate in importance across different societies.[563] This study also shows how these determinants can alternate within a certain society, depending on the subpopulation. At the city level, the historical context may exert a distinct effect on the mortality hazards of different age groups, even though the mortality is caused by the same type of disease.

Another important conclusion is that the observed socioeconomic inequalities did not always follow a pattern of a linear social gradient. The assumption that socioeconomic inequalities appear as a linear social gradient seems to be ubiquitous in the studies in this field. According to the seminal words of Marmot, the observed social inequalities in health *are* the social gradient, with an inherent social hierarchy.[564] The social hierarchy is linear, implying that with each step up the socioeconomic ladder, health increases and with each step down, health decreases, and this is observed across the globe. Yet if we would observe a process which contains emerging and receding socioeconomic health inequalities as Clouston et al. theorise, would it make sense to assume by default

563 Riley, *Rising Life Expectancy. A Global History*.

564 Marmot, *The Status Syndrome*, 1-12.

that the inequalities are rigidly tied in a hierarchical manner to socioeconomic status? In other words, do or did the poorest in society always have the last and least access to the necessary resources to improve their health when inequalities were observed? This study demonstrates that such a linear hierarchy of a social gradient may not always have materialised.

While in Maastricht the upper classes always seemed to gain the first advantage, the health advantages in the next stage of declining mortality did not always extend first to the skilled workers, and only thereafter to the unskilled workers. Infants in skilled working-class families were lagged behind most, compared to the other socioeconomic groups. One might expect this position to have been reserved for the poorer socioeconomic group of unskilled workers. From qualitative sources the idea arises that the unskilled workers were the first group targeted by public and private aid institutions, which perhaps would give them an earlier advantage. Adult men from the group of skilled workers were also the one group that showed a statistically significant different disease pattern compared to the upper classes, while the differences between the unskilled workers and upper classes were not statistically significant. The proportional mortality due to airborne infectious diseases of unskilled workers was situated between that of skilled workers and the upper classes during the initial phase of the health transition, and therefore probably not diverging enough to be statistically significantly different from the proportional airborne mortality of the upper classes. The nature of the skilled workers' occupations, such as the manufacturing of pottery or glass products, may explain this phenomenon. These social classes may have consisted of the most dangerous or health-risking occupations, due to dust inhalation, harming the respiratory system and thus causing the lungs to be more vulnerable to tuberculosis. The occupations therefore not only reflect one's socioeconomic status, but also the health hazards that were connected to the specific occupations.

Yet occupational hazards cannot fully explain the lack of a social *gradient* when socioeconomic inequalities in health emerge, since this lack of a gradient was also observed among infants. The disadvantages of skilled workers thus transcended specific working conditions related to the individual. A slightly higher position on the social ladder may have caused these families to actually be the last to gain access to resources, since they may not have been the target group of institutional care or the first public health initiatives, such as the Cross organisations. Once again, the particular historical context proves to be highly important for how the observed inequalities took shape. In other localities, in which public health initiatives were similarly trying to bring mortality levels down, such non-linear socioeconomic disparities may have emerged as well. In the analysis of socioeconomic inequalities, we should not focus too narrowly on looking for a linear social gradient. Other socioeconomic inequalities may have existed, and are legitimate objects of study, even if they may not fit our theoretical expectations of a linear social gradient.

7.1.3 Gender inequalities

Socioeconomic differences were not the only factos paving unequal pathways to the grave; gender differences also caused inequalities in health. In general, the literature has found a biological disadvantage for men due to a weaker immune response to infectious diseases. As a result, men are thought to de disadvantaged in a disease environment in

which infectious diseases are predominant. Yet in historical populations, many social and cultural factors had a substantial influence on gendered health as well. From the historiography we learn that women could be in a disadvantageous position due to different gender roles, in which men often earned an income while women worked in and around the home. Since the family was dependent on the male breadwinner's income, he came first when available resources were allocated. However, in urban industrial areas, men were often exposed to a disproportional amount of health risks due to their working conditions, which could reverse the sex ratios in mortality.

In the case of gender inequality, the data for Maastricht allowed for an analysis of gender disparities in mortality levels. In the exceptionally industrialised city of Maastricht, clear excess mortality existed for adult men. In this way, Maastricht reflected the British situation and other Dutch studies, in which female excess mortality is mainly a rural phenomenon instead an of urban one. This is not surprising; Maastricht's factories had a highly gendered occupational structure, leaving men with the most health hazardous occupations. Maastricht was also no exception when it came to gender differences in infant mortality. Male infants experienced excess mortality as well, which is found in all studies, whether in urban or rural contexts, and is considered to be related to biological disadvantage. The picture was more complicated among young children, amongst whom only a small male disadvantage existed after mortality had declined substantially. However, before the health transition no clear differences in mortality levels between girls and boys could be found. This again corresponds to other studies' findings on early childhood: From a general male disadvantage in infancy, gender starts to influence early childhood differently. No uniform pattern for gender inequalities in early childhood has been found up until now, although many find if a female disadvantage exists, it starts to be expressed at this age.

Thus, findings from Maastricht mirrored other studies' findings regarding gender differences in mortality levels. Yet several other important new insights regarding gender inequalities in health can be gained from the study of Maastricht. First, while socioeconomic inequalities in mortality increased during the initial phase of the health transition, the opposite happened with gender inequalities. For infants and adults, gender inequalities already existed prior to the health transition and decreased during the middle phases of the health transition. Once the greatest decline in mortality had taken place, only a negligible male disadvantage remained in all three age categories. Whether the persistent male disadvantage, even after the health transition, is a purely biological phenomenon is an open question. Although men in general have been found to have a weaker immune response to infectious diseases, the effects of this may have been diminished after the health transition, when infectious diseases deaths were receding. What caused men to have a health disadvantage *after* the health transition has taken place, is beyond the scope of this study, especially since disease patterns have changed substantially by then.

Second, although gender interacted with several other factors, such as migration and marital status, there was no support found in this study that gender interacted with socioeconomic status *directly*. Among adults, the emergence of socioeconomic inequalities in mortality differed between genders. However, that is more likely to be the result of a different timing in the start of the health transition. Especially the change in epidemiological patterns started earlier for upper-class women. Moreover, married

women clearly had lower proportional mortality from airborne infectious diseases compared to unmarried women, while a health benefit of being married was absent for men. This could have been caused by a selection effect, where healthy women were positively selected for marriage. A selection effect for marital status based on health has been found in historical populations for both men and women, yet in Maastricht marital status did not affect changes in epidemiological patterns for men. A confounding factor affecting only men was migration status. Men who had migrated into the city displayed lower proportional mortality due to airborne infectious diseases compared to native-born men.

Whereas gender clearly interacted with migration status and marital status in effectuating health inequalities among the adult Maastricht population, the relationship between socioeconomic status and gender is more intricate. Socioeconomic inequalities emerged for both genders once the mortality decline started. Since the mortality decline started in a different period for both women and men, socioeconomic disparities also occurred in different periods for women and men. This does not necessarily mean that gender and socioeconomic status interacted; after all, the emergence of socioeconomic inequalities depended on when the initial phase of the health transition occurred. However, socioeconomic status did appear to interact with migration status and marital status, since socioeconomic inequalities were not found for migrant men or unmarried women. Both migration and marital status also interacted with gender. There were thus several intricate processes at play that determined the different impact gender could have on health inequalities, in which the combined effects of gender and socioeconomic status were mediated by marital status or migration. A clear interaction between gender and socioeconomic status impacting cause-specific mortality, as has been proposed for Britain, where upper-class men had a higher chance of dying of non-infectious diseases, is not directly found in this research. However, the increase in proportional mortality due to non-infectious diseases among adult men might have been the consequence of an increase in degenerative diseases, instead of a decline in infectious diseases. We would need cause and class-specific mortality rates to assess whether this was the case. In neither of the other two age categories, of infants and young children, did gender and socioeconomic status interact clearly with each other.

A third important conclusion can be drawn from the analysis of gender inequalities in health amongst young children. Although gender did not appear to have had a substantial influence on the overall chances of survival among young children in the nineteenth and early twentieth centuries, gender differences existed regarding the particular diseases young children were suffering from. Early childhood mortality was characterised by typical childhood diseases such as scarlet fever, measles and diphtheria, but tuberculosis and pneumonia also culled young ones regularly. Some of these diseases were endemic; they were lethal on a regular basis, continuously adding to total mortality. Other diseases did not sustain themselves in the population, and were instead epidemic diseases that only occasionally had lethal consequences. However, these diseases caused large mortality spikes when they occurred. This study revealed that epidemic and endemic diseases differently impacted young children. Diseases that were typically epidemic disadvantaged boys, while endemic diseases appear to have disfavoured girls, based on mortality ratios.

The causes for these differences may be found in a slightly disadvantageous position in the distribution of resources for girls. The disadvantage in endemic diseases, such as tuberculosis, pneumonia and bronchitis were related to nutrition. This points towards a structural disadvantage for girls. The disadvantage for boys in epidemic outbreaks may indicate that in these instances the male biological disadvantage kicked in, a disadvantage they may have been able to avoid in the more endemic diseases due to a greater availability of resources. Whereas Riswick has found no particular favour for boys or girls in the Netherlands, these disease patterns may point to a different historical reality, where girls were indeed slightly disadvantaged in Maastricht. It would be interesting to see whether this epidemic/endemic dichotomy is unique to Maastricht, or was present in other populations as well.

Gender is a multifaceted factor. How it influences mortality differences depends heavily on age, historical context and the interaction with other factors, of which socioeconomic status might be one example. As such, gender has a potentially strong explanatory power in understanding different mortality levels, as well as different epidemiological pathways to lower mortality levels. It is therefore crucial to not only focus on mortality among men, but also on women. Fortunately, many studies take women into account as well, even though some still focus solely on male mortality, due to the larger availability of historical data for men. In accordance with Bengtsson and Van Poppel,[565] I argue that we should go the extra mile to be able to include women in our studies, and include their specific characteristics. Some historians may feel that finding an indication of socioeconomic status for women is too time-consuming, if it is even possible. However, this study demonstrates that finding ways to include female characteristics such as socioeconomic status is not only possible, but leads to many new insights which would be overlooked if we would only use male data.

7.1.4 Maastricht in the Dutch context

Maastricht, and the province of Limburg in which it is situated, have been known in historiography for their unhealthy environment compared to other Dutch regions in the late nineteenth century. Not only the heavily industrialised urban environment was a hazard to health, but also the lack of breastfeeding practices and the late adoption of hygienic practices due to Roman Catholicism have been put forth in the literature to explain the backward position of Limburg. Maastricht indeed lagged behind the national mortality decline, especially regarding infant mortality in the late nineteenth and the very early twentieth century; the decline in infant mortality in Maastricht started much later compared to other Dutch regions. However, does Maastricht deserve to be viewed as an extremely unhealthy place *for all* in comparison to the north-western part of the nation?

According to Wolleswinkel-van den Bosch, the Dutch mortality decline started for infants from 1871 onwards, for young children from 1888 onwards and for adults from 1880 onwards. Maastricht substantially lagged behind the rest of the Netherlands in terms of infant mortality decline, which only started in Maastricht from 1900 onwards. Yet the start of the mortality decline among young children was not so different from

565 Bengtsson and Van Poppel, 'Socioeconomic Inequalities in Death from Past to Present. An Introduction,' 348.

the Dutch average. Already from 1880 onwards, the retreat of scarlet fever and water and foodborne diseases resulted in a substantial decline in early childhood mortality in Maastricht. The beginning of the mortality decline among adult women even preceded the start of the Dutch average adult mortality decline, with female adult mortality declining in Maastricht from the start of the research period (1864). This decline started slightly later for men, in the late 1890s. All-age female mortality in the Netherlands started to decline in 1858, which is in line with the earlier start of female adult mortality decline in Maastricht. The all-age male mortality decline started earlier in the Netherlands in 1855, but intensified after 1881. Whereas infants and adult men lagged behind the national average mortality decline, this was not the case for young children and adult women.

Next to the start of the health transition, it is also relevant to consider the *level* of mortality when discussing which regions in the Netherlands were healthier than others. Regarding infant mortality, Maastricht experienced similar levels as the Dutch capital of Amsterdam in the 1870s, which was also close to the urban Dutch average. Since the decline started earlier in Amsterdam, it may come as no surprise that, by the late 1890s, the levels of infant mortality in Maastricht were substantially higher compared to Amsterdam and the urban Dutch average at that time.[566] The opposite was the case for early childhood mortality. While Maastricht had higher levels of early childhood mortality than Amsterdam in the late 1870s, the levels were equal to the levels found for Amsterdam and for the urban Dutch average in the late 1890s. Maastricht thus actually experienced a stronger decline in early childhood mortality in the late nineteenth century compared to the majority of other Dutch cities.

Does the higher level of infant mortality and the later onset of mortality decline for infants and adult men legitimise the characterisation of late-nineteenth and early-twentieth-century Maastricht as extremely unhealthy? To a certain extent it appears it does. It is undeniable that for these age and gender groups Maastricht suffered some of the highest mortality rates of the nation at the end of the nineteenth century. However, I argue that to establish such an overall characterisation, we need to look beyond infant mortality. Early childhood mortality in Maastricht at the end of the century does not reflect the characterisation of the city as unhealthy, since mortality levels did not deviate from the Dutch urban average. Although a selection effect among infants may have resulted in a more robust population of young children, patterns found for adult women also seem to have followed the national average turning point for the decline in mortality. Living in Maastricht may have been more hazardous for some subpopulations compared to other Dutch cities, yet for others this may not have been the case. It is time, therefore, to move beyond the dominant focus on infant mortality and take other population groups into account in establishing which regional differences existed in the Dutch health transition.

566 Angélique Janssens and Tim Riswick, 'What Was Killing Babies in Amsterdam? A Study on Infant Mortality Patterns Using Individual Level Cause-of-Death Data, 1856-1904,' *Historical Life Course Studies* (Forthcoming); Wolleswinkel-van den Bosch, 'The Epidemiological Transition in the Netherlands.'

7.2 Scientific relevance

According to Amartya Sen, a key characteristic of social justice is what one is able to do, the freedom one enjoys to pursue the life one wants to live.[567] One of these essential freedoms is to be free of premature mortality.[568] Inequalities in mortality, whether they are based on gender or socioeconomic status, can therefore be considered as great social injustices. Indeed, socioeconomic and gender inequalities in mortality are one of the main preoccupations of scholars.[569] An in-depth understanding of these inequalities in health is an essential first step to minimise them to the greatest extent possible. In order to gain that understanding, we need to grasp the historical roots of inequalities in health and death. This case study on Maastricht has contributed to that understanding by revealing how socioeconomic inequalities emerged once resources became available to curb ravaging infectious diseases. Socioeconomic inequality in health is not a static phenomenon, it is dynamic and develops when remedies for diseases start to become available. Socioeconomic inequalities are not only connected to the availability of resources, but also to the individual's receptiveness – as at least partially determined by socioeconomic status – towards new ideas on hygiene or treatment, which is highlighted in this study's chapter on infant mortality.

The recent COVID-19 pandemic made it painfully clear how socioeconomic inequality in health is still a problem today. Not only were there differences in the availability of resources, for example when first world countries reserved all the first vaccines for themselves, and third-world countries had to wait, but also inequalities from the lack of acceptance of these resources emerged. In the Netherlands, people with a migration background in three large Dutch cities had higher chances of dying of COVID-19.[570] This may partially have been a socioeconomic problem, since people with a migration background tend to have lower incomes. However, the willingness to be vaccinated also was less present in this population. While these issues are unfortunately not new, historical research can help in dealing with these issues today, especially by theorising which groups are most at risk of infection and death.

Moreover, COVID-19 has once again reinforced a statement already made in the late 1990s: contrary to popular belief in the mid-twentieth century, we are not able to eradicate preventable infectious diseases. Rather, we face a potentially whole range of new infectious diseases, and we could consider ourselves to be in the fifth stage of the epidemiological transition: The phase of re-emerging infectious diseases.[571] The past years have shown the different strategies deployed by countries to manage the disease until a vaccine was available, from extremely restrictive to extremely lenient. Modern-day

567 Amartya Sen and James Eric Foster, *On Economic Inequality. Enlarged Edition with a Substantial Annex 'on Economic Inequality after a Quarter Century'* (Oxford: Clarendon Press, 1997).

568 Amartya Sen, 'Mortality as an Indicator of Economic Success and Failure,' *The Economic Journal* 108, no. January (1998); Amartya Sen, *The Idea of Justice* (London, New York: Allen Lane/Penguin Books, 2009), 226.

569 To only name a few: Deaton, *The Great Escape*; Mackenbach, 'Nordic Paradox, Southern Miracle, Eastern Disaster'; Mackenbach et al., 'Widening Socioeconomic Inequalities in Mortality in Six Western European Countries.'

570 Karien Stronks, Maria Prins, and Charles Agyemang, 'Bevolkingsgroepen Met Migratieachtergrond Zwaarder Getroffen Door Covid-19,' (2021).

571 Olshansky et al., 'Emerging Infectious Diseases. The Fifth Stage of the Epidemiologic Transition.'

society had no idea what strategies to use when dealing with an emergent infectious disease, perhaps because people believed those infectious diseases to be a distant memory. The field of historical demography, and also this study, have been preoccupied with the question of which determinants were effective in lowering mortality, especially in a period when antibiotics and vaccines were mostly non-existent. These determinants go beyond only medical determinants, and can thus provide us with strategies when medicine may find itself empty-handed at the beginning of a spreading infectious disease.

We also have dealt continuously with gender inequalities in the recent past. We may feel that gender inequalities in health are also only reminiscent of the past, but this is far from the case. Medical research and technological innovations have been carried out from an androcentric point of view,[572] which still poses greater risks for women due to a lack of understanding gender differences. By prioritising the inclusion of women in historical demographic studies, we can aid the understanding of female health experiences, whether these are rooted in biological, social, or cultural influences.

The insights gained from this dissertation stem from the unique and exceptionally rich dataset that was available for Maastricht. The use of individual-level, cause-specific mortality data allows for more analytical opportunities, compared to the usage of aggregated mortality data,[573] that have been used by many other studies into the Dutch health transition and its features. Whereas aggregated data can offer a broad understanding of demographic trends, these individual-level data have the great potential to check and to amend overarching theories and illuminate which mechanisms are obscured by higher-level aggregations. Several distinct characteristics of the *Maastricht Death and Disease Database* (MDDD) added substantially to our understanding of the health transition.

The first and foremost advantage of the MDDD is the fact that it contains information on the specific causes of death, as registered by a trained physician. Most studies on cause-specific mortality in the Netherlands have used aggregated statistics, which are available for each Dutch municipality from 1875 onwards. The main problem with the published cause-of-death statistics is that they deploy grouped causes of death. These groupings are based on a contemporary understanding of disease mechanisms and aetiology, which is different from our modern-day understanding. As a result, for the purpose of our analyses, some of these groups should ideally be disentangled. With the individual-level data, convulsions could be analysed separately, while in the published cause-of-death statistics convulsions are grouped together with trismus and epilepsy, two different causes of death. Similarly, diabetes and tuberculosis of the lung could be disentangled, as well as debility and extrapulmonary tuberculosis. If only mortality data at the aggregated level had existed for Maastricht, we would have had a less accurate understanding of which causes of death were predominant in the research period, and how certain diagnostic practices and/or mortality patterns evolved. Among infants, the use of convulsions already declined after the 1870s, however we would not have been certain that a decline in its disease category would be due to a lesser use of the term

572 See for example the popular-scientific Caroline Criado Perez, *Invisible Women: Data Bias in a World Designed for Men* (London: Chatto & Windus, 2019).

573 Janssens and Devos, 'Introduction to the Special Issue the Limits and Possibilities of Cause of Death Categorization for Understanding Late Nineteenth Century Mortality.'

convulsions, or of trismus or epilepsy instead, if only the aggregated causes of death had been available.

Another major advantage of using individual-level data is the variety of subpopulations one can analyse. Other studies have been capable of highlighting several individual features of cause-specific mortality, such as cause-specific mortality by age, or by socioeconomic status, or by religion. The individual-level data make it possible to combine these characteristics and study their interactions. Especially in the case of adult mortality, the combination of different individual characteristics was key in creating health advantages and disadvantages. The analysis of changing epidemiological patterns by socioeconomic groups as a whole would not have been possible, were it not for the individual-level data. Studies using individual-level data therefore contribute to our understanding of the health transition in major ways.

7.3 Future research

The data underpinning this research have not been used exhaustively. The dataset contains the address where death occurred, which was often also the address of residence. Spatial analyses would be a first step in expanding the research on the health transition for Maastricht. There are even greater opportunities ahead if the dataset would be enlarged by data from the Civil Registry, such as birth and marriage certificates. By linking the data, life course analyses would then become possible. Moreover, in this research the emergence and retreat of socioeconomic inequalities based on changing epidemiological patterns have been demonstrated. These socioeconomic inequalities are inequalities based on the *timing* of changes in mortality during the health transition. To what extent these inequalities correspond to disparities in the *levels* of mortality remains an open question. By extending the dataset with birth certificates, the population at risk could be determined by socioeconomic class, which would allow for the computation of cause-specific *and* class-specific mortality rates. We could greatly enhance our understanding of socioeconomic inequalities in health by studying both inequalities in the timing of the health transition, and in the levels of specific mortality rates.

Naturally, these endeavours should not be contained to Maastricht. I have argued throughout this dissertation that the British and Swedish experience of the health transition do not necessarily represent the experience of other western countries. Based on the characteristics of Maastricht –an industrialised city – one might expect that the British experience may have been closer to Maastricht than the experience of the western Netherlands. However, we would need many other case studies to determine whether the experiences of different localities resemble each other. Moreover, could we find certain characteristics that are related to similar experiences of the health transition? Because these characteristics may not be shared with localities within national borders, but perhaps instead with localities abroad, large comparisons between localities on at least a European level would be necessary. Several of these individual-level datasets have been constructed for localities throughout Europe. With the SHiP+ initiative, researchers are endeavouring to make mortality data comparable, despite differences in historical

context and nosologies of the localities.[574] By building and expanding mortality datasets with the help of data scientists and citizen science, the understanding of the health transition can be greatly improved. The Amsterdam cause of death project with its many volunteers is a great example.[575] However, for the Netherlands we currently have complete cause-specific individual-level data over a long time horizon for only a few cities. We would need to mine the archives further to see if we could build these kinds of datasets for other localities as well.

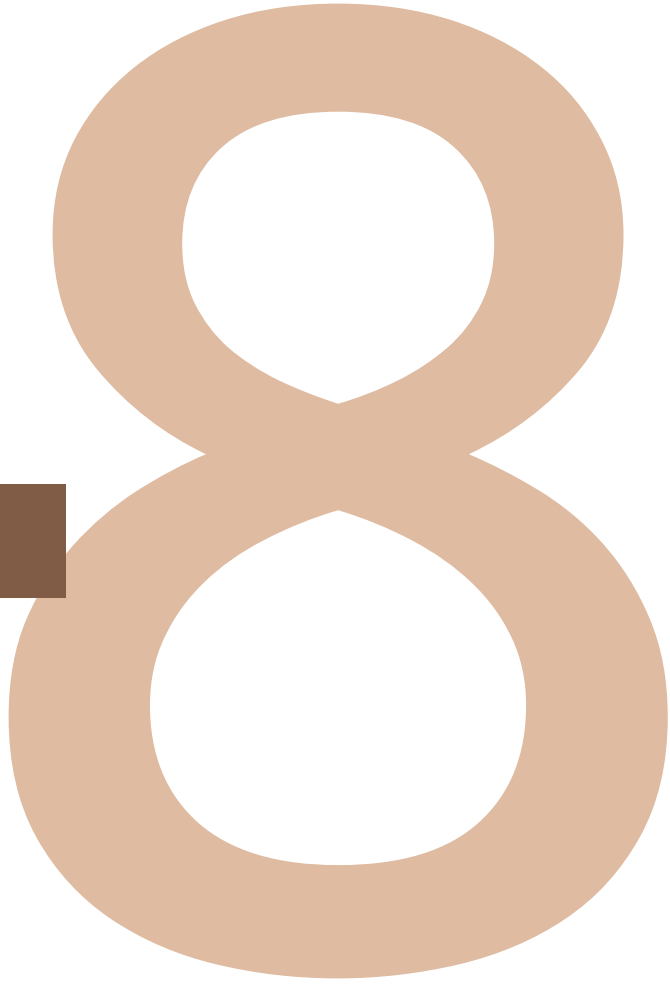
Next to further linking existing datasets and eventually extending the number of existing datasets, quantitative historical demographical analyses would benefit greatly from including more qualitative data. While quantitative analyses may reveal trends in historical developments and help to assess whether identified trends were indeed significant changes in society, qualitative source material puts the quantitative analyses into broader perspective. Ideally, both analyses would therefore be combined.

Yet the combination of quantitative and qualitative analyses goes beyond methodological considerations. Historical demography as a field appears to be the only historical branch that studies causes of death. Medical historians often keep themselves far away from undertaking the tricky study of disease itself, because from their perspective causes of death should be seen as social constructs. However, perceiving historical diseases and causes of death as social constructs may actually enhance demographic analyses; doing so may lead to more thoroughly scrutinised mortality data. On the other hand, medical history could learn from historical demography by not losing sight of these large population trends in mortality as part of the health transition. A combination of a realist and constructivist approach would improve research, and may lead to new questions and insights in both fields. Ultimately, this may lead to an improved understanding of the health transition, of its determinants, of the inequalities in the process, of what moved people, of how they dealt with both morbidity and mortality, and of how that in turn affected the health transition again.

574 Ibid.

575 https://doodinamsterdam.nl/ accessed July 2022.

CHAPTER 8



Impact paragraph

Over the past couple of years, the world has faced the hazards of the COVID-19 pandemic and its disruptive consequences, which makes it feel as if the relevance of studying historical epidemiological patterns has almost become self-evident. The effects of epidemic outbreaks on society are and have been immense. In combatting these epidemic diseases, one of our main interests is also minimising inequalities in death: The less privileged groups in society who are highly vulnerable to these diseases should be actively protected and supported. Studying historical inequalities in mortality can therefore aid our modern-day efforts in fighting inequalities in health and death. The historical studies provide deeper insights into potential inequalities and potential solutions to lessen the inequalities in death. The specific scientific relevance of this study in the fields of historical demography and medical history have been stressed extensively in the conclusion of this dissertation, thus it will suffice to only recapitulate some of the main elements.

By deploying unique individual-level cause-of-death data for the period from 1864 until 1955 for the city of Maastricht, this study contributes substantially to studies in the field of historical demography. Many of these studies have been forced to use aggregated mortality data. With individual-level datasets slowly becoming available for many European countries, our understanding of the health transition can improve considerably. Moreover, until now, studies on inequalities have not been able to find conclusive answers on whether inequalities existed and, if so, how they emerged or how they behaved. This study offers new pieces of the puzzle of how socioeconomic, age, and gender inequalities occur, emerge and recede in the course of the health transition. These findings have not only been presented here on paper, but they have been presented at different international conferences, both live and virtually.

Further, this research is a case study, and therefore deals with the history of a single city, the city of Maastricht. Whereas the wider public may be less interested in the details of the analyses, it turned out that the more general outcomes of the research did not fall on deaf ears with this group. The research's findings have been communicated to the general public via different news outlets, such as the local radio, local television, the local newspaper, and on social media. Public interest increased during the COVID-19 pandemic, when we were able to provide historical contextualisation on dealing with epidemic diseases. This resulted in an interview in the newspaper *De Limburger* with both my supervisor Willibrord Rutten and myself.

During the course of my PhD I have worked together with the *Sociaal Historisch Centrum Limburg* (Centre for the Social History of Limburg, SHCL), which resulted in a fruitful public event on trend reversals in health in the past and present at the SHCL in April 2022. During this event we fostered a dialogue about past and present health issues. The main question of the event was how the historical trend reversal in exceedingly high mortality took shape, and how such a trend reversal could be accomplished again in the future, given the health lags in southern Limburg today. During the public event, presenters from the local *Gemeentelijke Geneeskundige Dienst* (Municipal Health Service), a public historian and myself covered different aspects of these trend reversals in mortality. At the end of the day, we could look back on a successful event, with thought-provoking presentations, an engaged audience and a typical Limburg coffee break with a lot of networking over a piece of *vlaai*.

Finally, there is one other remark I would like to make. While working on the gender inequalities in mortality, I came across a different type of gender inequality at a more meta level. In historical demography, we distinguish between multiple gendered demographic characteristics, due to the highly different experiences and contexts of men and women. However, in our research it appears that many studies still, most likely unconsciously, work from an androcentric point of view.[576] The male is taken as standard, and the female only as diverting from the male standard experience. The fact that data are more widely available for men than for women only reinforces this kind of androcentric understanding. Few studies actually acknowledge their own androcentric point of view, which does not aid the still-ongoing emancipation of women. Studies, for example, sometimes only research men, without fully acknowledging that this is not representative of society as a whole. Or when both genders are taken into account, researchers by default start discussing the results for men first. Although I have tried to be aware of these mechanisms, I have occasionally fallen victim to this latter practice as well.

As researchers, we have much to gain in acknowledging our own androcentric views, and we should aim to shake off those views in order to improve our understanding of different historical realities. In this dissertation, I have taken a first step in this regard by trying to be aware of my own unconscious androcentric ideas. A tangible example is that I did not allow myself to start with men by default when discussing my results. Starting with women instead, however, felt like an empty statement. I therefore forced myself to come up with a different rationale for the way I ordered the discussion of my results. A fairly simple, yet logical rationale was to start with the result, of the group which experienced the highest outcome of the variable I was interested in. In the end I therefore often start the discussion with men, since they often experienced the highest mortality rates. However, in some other instances I started with women, for example when discussing the proportion of Jewish people who died during the Second World War, which was higher for women compared to men.

Although such a rationale may not be consistent in mentioning both genders in the same order in every analysis, it is consistent in mentioning the gender that was most affected first. In doing so, I hope to become more aware of our own ingrained androcentric points of view, and to find different rationales than just applying the 'easy' men-first rationale. In this way, we hopefully become more aware of other potential androcentric practices in our research, which we are not yet aware of.

576 Angélique Janssens, "Op De Snijtafel Van De Genderhistoricus. Androcentrisme in De Historische Demografie," *TSEG- The Low Countries Journal of Social and Economic History* 17, no. 1 (2020).

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Appendices

Annex I: Maastricht Death and Disease Database variables.

The variables listed here are the variables that were included in the Maastricht Death and Disease Database. The burial registers, on which – next to the civil registration- the database is based, contain more information which could provide additional information, yet not in all cases for the entire duration of the research period. The other available information in the burial registers include information on the location of the grave on the graveyard, the burial class (adhering to the social class scheme, except that people tried to buy a grave from a higher class than they could actually afford), the duration of the grave rights, fees due, name of the gravedigger and the name of the responsible physician for the death certificate.

Variable	Explanation
Type of document	In the majority of the cases, this referred to the death certificate. However, sometimes there was a correction.
Document number	The burial registers and death certificates were linked based on the document number of the death certificate, since these were reported in the burial registers.
Document date	In the majority of the cases, there was a one to two day difference between the actual date of death and the date of the document.
Date of death	
Religion*	
Place of death (municipality level)	
Place of residence	This variable only includes an entry when the place of residence was other than Maastricht.
Address of death	Often includes the Abtstraat (numbers 2 or 6 in the following variable) as well, where the hospital was located.
Street number	
Addition to address	
First name deceased	
Insertion of names deceased	The often used *van*, *von*, *van der*, or *de* for example.
Family name deceased	
Gender deceased	
Occupation of the deceased	
First name of the father of the deceased	
Insertion of names the father of the deceased	
Family name of the father of the deceased	
Occupation of the father of the deceased	
First name of the mother of the deceased	
Insertion of names of the mother of the deceased	
Family name of the mother of the deceased	
Occupation of the mother of the deceased	

Variable	Explanation
First name of the partner of the deceased	
Insertion of names of the partner of the deceased	
Family name of the partner of the deceased	
Relationship	Indicating whether someone was married, divorced, or only the son or daughter of others.
Remarks	This variable could include a variety of different additional remarks, for example the names of the spouses when someone had been married multiple times, or the number of days, weeks or months old an infant was at the time of death or whether the death was actually a stillbirth, or whether someone had been found in the river.
Age	Age was included only in years, and was not included for infants. A numerical expression for age had to be calculated based on the information on the infants age given under the previous variable, remarks.
Cause of death*	
Place of birth (municipality level)	
Feedback	Includes a host of information, mainly when there were two addresses mentioned. Often one of the two addresses was the Abtstraat 2 or 6, or St. Annadal 1, which was the address of the hospital (St. Annadal after 1940). Also when someone had died outside of Maastricht, this was mentioned under this heading.

*The information for these variables was only listed in the burial registers, not in the death certificates.

Annex II: Table of the hundred most frequent tidied causes of death.

The Dutch causes of death between brackets are the tidied, but not yet standardised, phrasings as they occurred in the registers.

Cause of death	Frequency
Tuberculosis of the lung (*Longtuberculose*)	2942
Gastroenteritis (*Gastro-enteritis*)	2274
Pneumonia (*Pneumonie*)	2202
Bronchopneumonia (*Bronchopneumonie*)	1861
Marasmus senilis (*Marasmus senilis*)	1831
Enteritis (*Enteritis*)	1781
Cancer ventriculi (*Kanker ventriculi*)	1747
Degeneration of the myocard (*Myocarddegeneratie*)	1633
Convulsions (*Stuipen*)	1579
Article 50. of the Civil Code (*Art. 50 BW*)	1474
Apoplexy cerebri (*Apoplexia cerebri*)	1312
Cause unknown (*Onbekende oorzaak*)	1250
Apoplexy (*Apoplexie*)	1207
Consumption of the lung (*Longtering*)	1094
Inflammation of the bowel (*Darmontsteking*)	1069
Congenital debility (*Aangeboren lichaamszwakte*)	950
Marasmus (*Marasmus*)	812
Phthisis pulmonum (*Phthisis pulmonum*)	783
Viable (*Levensvatbaar*)	774
Meningitis (*Meningitis*)	689
Convulsions (*Convulsies*)	642
Vitium cordis (*Vitium cordis*)	640
Croup pneumonia (*Croupeuse pneumonie*)	637
Heart disease (*Hartziekte*)	624
Encephalitis (*Encephalitis*)	622
Pedatrophy (*Paedatrofie*)	616
Atherosclerosis (*Atheriosclerose*)	544
Diphtheria (*Difterie*)	506
Bronchitis (*Bronchitis*)	492
Catarrh pneumonia (*Catharr pneumonie*)	488
Drowning (*Verdrinking*)	474
Old age (*Ouderdom)*	449
Cause unknown (*Causa ignota*)	445
Lifeless (*Levenloos*)	442
Chronic nephritis (*Chronische nefritis*)	409
Capillary bronchitis (*Capillaire bronchitis*)	408
Chronic myocarditis (*Chronische myocarditis*)	384
Myocarditis (*Myocarditis*)	357
Stroke (*Beroerte*)	346
Cholera (*Cholera*)	341
Tuberculous meningitis (*Tuberculeuze meningitis*)	322

Cause of death	Frequency
Premature birth (*Vroeggeboorte*)	321
Uraemia (*Uremie*)	314
Stomach cancer (*Maagkanker*)	311
Acute gastroenteritis (*Acute gastro-enteritis*)	307
Liver cancer (*Leverkanker*)	307
Diabetes (*Diabetes*)	305
Measles (*Mazelen*)	302
Meningitis tuberculosis (*Meningitis tuberculose*)	300
Chronic bronchitis (*Chronische bronchitis*)	296
Peritonitis (*Peritonitis*)	293
Cerebral haemorrhage (*Hersenbloeding*)	291
Cholerine (*Cholerine*)	290
Stillbirth (*Doodgeboren*)	290
Angina pectoris (*Angina pectoris*)	285
Convulsions of the infant (*Ecclampsia infantum*)	278
Cancer (*Kanker*)	275
Sudden death (*Plotselinge dood*)	268
Organic cardiac deficiency (*Organisch hartgebrek*)	260
Brain attack (*Hersenberoerte*)	258
Croup (*Croup*)	248
Not viable (*Niet levensvatbaar*)	245
Paralysis cordis (*Paralysis cordis*)	243
Bright's disease (*Morbus Bright*)	234
Dropsy (*Waterzucht*)	230
Breast cancer (*Borstkanker*)	229
Premature (*Prematuur*)	229
Cancer of the rectum (*Kanker rectum*)	227
Decompensatio cordis (*Decompensatio cordis*)	216
Cirrhosis of the liver (*Levercirrose*)	207
Chronic enteritis (*Chronische enteritis*)	205
Uterine cancer (*Baarmoederkanker*)	195
Scarlet fever (*Roodvonk*)	192
Acute bronchitis (*Acute bronchitis*)	189
Diarrhoea (*Diarrhée*)	182
Without medical treatment (*Zonder geneeskundige hulp*)	181
Myocardial infarction (*Hartinfarct*)	180
Cancer of the oesophagus (*Kanker oesofagus*)	180
Insufficientia cordis (*Insufficiëntia cordis*)	164
Heart failure (*Hartverlamming*)	160
Whooping cough (*Kinkhoest*)	160
Chronic pneumonia (*Chronische pneumonie*)	159
Typhoid fever (*Febris typhoidea*)	156
Oedema of the lung (*Longoedeem*)	154
Nephritis (*Nefritis*)	154
Phthisis (*Phthisis*)	147
Scrofula (*Klierziekte*)	146
Sepsis (*Sepsis*)	146
Influenza (*Influenza*)	143

Cause of death	Frequency
Atrophy (*Uittering*)	141
Tuberculosis (*Tuberculose*)	140
Cardiac aneurysm (*Aneurysma cordis*)	139
Softening of the brain (*Hersenverweking*)	135
Cancer of the colon (*Kanker van de dikke darm*)	133
Not listed (*Niet vermeld*)	129
Illness of the bowel (*Darmziekte*)	126
Placenta praevia (*Placenta praevia*)	123
Act of war (*Oorlogshandeling*)	122
Intoxication of the infant (*Intoxicatie [zuigeling]*)	121
Lung cancer (*Longkanker*)	118

Annex III: Results of the multinomial logistic regression analysis for water- and foodborne infectious compared to non-infectious causes of death, legitimate infants.

Period	Socioeconomic class	Odds-ratio first month	p-value	N* first month	Odds-ratio 2-11 month	p-value	N* 2-11 month
1864-1883	Elite	1,000		178	1,000		411
	Skilled workers	1,168	0,466	366	1,503	0,005	1014
	Unskilled workers	1,286	0,247	306	1,771	0,000	634
	Unknown	2,331	0,017	43	1,339	0,267	120
1884-1903	Elite	1,000		143	1,000		406
	Skilled workers	0,841	0,392	479	0,906	0,509	1514
	Unskilled workers	1,053	0,810	302	0,870	0,363	1091
	Unknown	1,279	0,551	30	0,726	0,237	106
1904-1913	Elite	1,000		63	1,000		144
	Skilled workers	2,183	0,012	262	1,406	0,187	774
	Unskilled workers	1,727	0,092	172	1,744	0,044	523
	Unknown	1,353	0,654	12	1,939	0,247	38
1914-1919	Elite	1,000		26	1,000		31
	Skilled workers	0,690	0,451	71	1,507	0,362	192
	Unskilled workers	0,895	0,825	56	2,157	0,121	108
	Unknown	1,889	0,557	4	1,739	0,528	12
1920-1939	Elite	1,000		90	1,000		84
	Skilled workers	1,623	0,284	191	2,005	0,007	230
	Unskilled workers	1,617	0,365	75	1,325	0,317	129
	Unknown	1,912	0,297	36	1,048	0,909	32
1940-1945	Elite	1,000		45	1,000		34
	Skilled workers	0,412	0,087	76	1,567	0,272	85
	Unskilled workers	1,500	0,449	30	2,138	0,104	43
	Unknown	---	---	2	1,267	0,790	6
1946-1955	Elite	1,000		61	1,000		45
	Skilled workers	2,667	0,386	94	1,936	0,169	61
	Unskilled workers	4,000	0,265	32	1,859	0,385	14
	Unknown	---	---	7	0,925	0,947	6

Annex IV: Results of the multinomial logistic regression analysis for airborne infectious compared to non-infectious causes of death, legitimate infants.

Period	Socioeconomic class	Odds-ratio first month	p-value	N* first month	Odds-ratio 2-6 months	p-value	N* 2-6 months	Odds-ratio 7-11 months	p-value	N* 7-11 months
1864-1883	Elite	1,000		149	1,000		136	1,000		85
	Skilled workers	1,945	0,044	319	1,237	0,309	282	1,452	0,196	178
	Unskilled workers	1,146	0,705	243	1,165	0,524	143	1,892	0,044	131
	Unknown	2,092	0,195	30	0,822	0,658	24	0,982	0,970	27
1884-1903	Elite	1,000		104	1,000		105	1,000		77
	Skilled workers	1,016	0,966	369	0,959	0,850	406	0,627	0,158	237
	Unskilled workers	1,205	0,636	220	0,953	0,833	285	0,606	0,135	205
	Unknown	1,567	0,525	21	1,006	0,988	39	1,556	0,518	24
1904-1913	Elite	1,000		50	1,000		39	1,000		27
	Skilled workers	1,507	0,476	164	0,723	0,373	153	1,868	0,256	98
	Unskilled workers	1,205	0,760	116	1,166	0,694	91	1,314	0,615	84
	Unknown	1,437	0,759	9	0,928	0,928	7	1,714	0,646	7
1914-1919	Elite	1,000		19	1,000		16	1,000		15
	Skilled workers	2,615	0,230	68	0,893	0,842	58	0,444	0,459	65
	Unskilled workers	0,671	0,677	41	2,022	0,261	36	0,314	0,313	27
	Unknown	0,001	0,927	2	0,778	0,867	2	0,500	0,641	8
1920-1939	Elite	1,000		98	1,000		74	1,000		40
	Skilled workers	0,955	0,894	197	1,476	0,159	197	3,567	0,020	93
	Unskilled workers	1,174	0,694	80	1,641	0,098	126	1,327	0,555	78
	Unknown	1,428	0,463	39	1,322	0,566	23	0,747	0,617	25
1940-1945	Elite	1,000		39	1,000		27	1,000		9
	Skilled workers	1,673	0,398	81	1,267	0,599	74	4,375	0,093	18
	Unskilled workers	2,917	0,118	28	1,974	0,196	34	2,188	0,394	11
	Unknown	13,125	0,015	5	1,077	0,935	6	---	---	3
1946-1955	Elite	1,000		73	1,000		48	1,000		16
	Skilled workers	0,769	0,526	105	1,526	0,278	62	0,750	0,666	21
	Unskilled workers	0,923	0,883	36	1,865	0,246	20	4,000	0,258	5
	Unknown	0,769	0,815	7	0,916	0,911	8	---	---	0

Annex V: Epidemic and endemic mortality among young children.

Cause-specific mortality spikes were considered to be an epidemic outbreak when the cause-specific mortality rate for that disease was larger than three standard deviations from the mean mortality rate over the entire period. Since some diseases already receded before the end of the research period, some overestimation of epidemics can occur. Moreover, the main diseases among children were characterised as being epidemic, endemic of a combination of both. The epidemic diseases not only caused spikes in early childhood mortality, but were completely absent in the mortality statistics in the years in between. Endemic diseases were, on the other hand, always prevalent, and thus caused deaths in almost every year. A mix between epidemic and endemic diseases is a disease which occurs regularly and caused some deaths in most years, yet with major spikes in some instances.

	N in total period	Mean mortality rate per year (deaths per 1,000 children)	Standard deviation	Threshold mortality rate for epidemic (mean +3 standard deviation)	Number of epidemic outbreaks	Epidemic or endemic behaviour
Pneumonia	1,256	4.02	3.71	15.15	1: - 1887	Endemic
Gastroenteritis	1,134	3.92	4,16	16.4	1: - 1878	Endemic
Tuberculosis	632	2.05	1.90	7.75	1: - 1879	Endemic
Diphtheria	565	1.88	3.80	13.28	3: - 1868 - 1892 - 1893	Endemic, with major epidemic outbreaks
Convulsions	433	1.56	2.18	8.10	2: - 1864 - 1867	Endemic
Meningitis	370	1.22	1.38	5.36	1: - 1879	Endemic
Measles	349	1.05	2.40	8.25	3: - 1907 - 1910 - 1912	Epidemic
Bronchitis	316	1.06	1.31	4.99	1: - 1891	Endemic (until 1920)
Scarlet fever	194	0.75	3.25	10.50	3: - 1864 - 1871 - 1879	Epidemic
Cholera	170	0.67	3.33	10.66	1: - 1866	Epidemic
Whooping cough	151	0.46	0,93	3.25	2: - 1897 - 1902	Epidemic*

* Difficult to establish. Whooping cough occurred only once every couple of years, yet with very few lethal cases. Its morbidity could therefore be endemic, yet only resulting in lethal cases once every few years.

Annex VI: Table with per cent contribution of disease groups and individual diseases by decade to early childhood mortality decline of as a whole (1864/69 until 1930/39).*

Cause of death	1864/1869 - 1870/1879	1870/1879 - 1880/1889	1880/1889 - 1890/1899	1890/1899 - 1900/1909	1900/1909 - 1910/1919	1910/1919 - 1920/1929	1920/1929 - 1930/1939	Total per cent contribution, 1864/69 to 1930/39
Water and foodborne infectious diseases	8.6%	13.7%	3.0%	0.1%	9.4%	5.8%	1.4%	42.0%
Airborne infectious diseases	1.0%	17.3%	-10.9%	13.8%	2.2%	17,5%	5.3%	46.3%
Other infectious diseases	0.7%	0.0%	-0.3%	0.2%	0.6%	0.2%	0.2%	1.6%
Non-infectious diseases	-2.1%	4.1%	1.8%	2.2%	0.6%	1.8%	1.7%	10.1%
(Gastro)enteritis	-6.4%	10.9%	-1.8%	-1.4%	7.1%	4.4%	1.0%	13.8%
Cholera	10.3%	1.2%	0.4%	0.6%	0.2%	0.0%	0.0%	12.6%
Convulsions	3.6%	-0.8%	5.0%	-0.2%	1.2%	0.9%	0.3%	10.0%
Other water and foodborne infectious diseases	1.0%	2.4%	-0,5%	1.2%	0.9%	0.5%	0.1%	5.6%
Scarlet fever	2.4%	5.1%	0.6%	-0.2%	0,0%	0.2%	0.2%	8.3%
Diphtheria	7.7%	1.8%	-8.0%	9.4%	0.0%	1.0%	-0.2%	11.7%
Measles	0.0%	-0.4%	-0.7%	-3.0%	-1.1%	5.7%	0.3%	0.7%
Whooping cough	-1.1%	0.7%	-0.7%	0.1%	0.0%	0.5%	0.8%	0.2%
Tuberculosis	-4.2%	4.9%	0.1%	-0.8%	-1.5%	4.5%	1.1%	3.9%
Pneumonia	-3.0%	0.1%	-1.4%	2.9%	1.6%	4.7%	2.6%	7.4%
Bronchitis	0.0%	-0.7%	-1.3%	1.6%	1.5%	1.4%	0.0%	2.6%
Other airborne infectious diseases	-0.6%	5.9%	0.6%	4.0%	1.7%	-0.5%	0.5%	11.5%

Positive percentages indicate a positive contribution to the decline. i.e. the cause of death or category-specific rate declined. Negative percentages indicate a negative contribution to the decline. i.e. the cause of death or category-specific rate increased.

Summary

This dissertation is concerned with the changes in health and mortality that have occurred over the past two centuries in modern societies known as the health transition. Whereas in the pre-transitional eras infectious diseases were the dominant cause of death for the majority of the population, causing high premature mortality rates, in post-transitional societies most people live into old age and eventually die of degenerative and man-made diseases. This change was not only a result of medical innovations and increasing wealth, but also a consequence of cultural changes. However, the transition was not evenly dispersed throughout society and inequalities in health and mortality could exist, arise or recede due to differences in age, gender and socioeconomic status. Although the health transition has been broadly theorised based on mainly aggregated mortality data, the underlying changes in particular diseases or among distinct subpopulations are still not fully understood.

The aim of this study is to further expand our understanding of the health transition by looking beyond the aggregated mortality data. The individual-level cause-of-death data that are available for the industrial Dutch city of Maastricht allows for a more detailed analysis of the changes in mortality that occurred between 1864 and 1955. The data not only include the exact cause of death, as written down by a physician, but it also contains information on the age, religion, gender and occupation of the deceased. The richness of the dataset therefore enables the analysis of the combined effects of these characteristics on cause-specific mortality. The specific causes of death are furthermore very informative of the determinants leading to the health transition. When certain diseases recede, the aetiology of the disease can offer a clearer sense of the related factors transforming health.

The central research question for this study is threefold: First, how did the health transition in the industrialising city of Maastricht take shape; second, did the transition lead to new inequalities in cause-specific mortality in terms of socioeconomic status, age and gender; and, third, which determinants were at the root of these changes in health? In order to answer these questions, different methods are used. A common denominator of these methods, however, is the focus on changing epidemiological patterns, i.e. the study of informative changes in the dominant pattern of circulating diseases. The unique data available for Maastricht allow for a focus on cause-specific mortality, despite a population at risk not being available according to all the specific individual characteristics, such as social group. Nevertheless, by analysing changing epidemiological patterns for these particular groups instead of the changes in cause-specific mortality *rates* for these groups, it becomes possible to approximate emerging inequalities and health advantages and disadvantages. The epidemiological patterns thus help to locate changes in mortality regimes for distinct groups in society.

The reasoning behind the methodological approach that focuses on changing epidemiological patterns is derived from Omran's epidemiological transition theory, which can be viewed as one of the crucial elements of the wider health transition theory. To understand the analysis of changing epidemiological patterns in order to identify possible inequalities, two main characteristics of the epidemiological transition theory are needed. The epidemiological transition theory postulates that during the transition

the dominant causes of death changed from being infectious diseases to man-made and degenerative causes of death. Moreover, when infectious diseases are rampant, there is a high mortality regime in which many young children and adults find an early death. A low mortality regime is reached when not only the dominant causes of death have changed to non-infectious diseases, but death also predominantly occurs in old age. When cause-specific mortality rates according to socioeconomic group are lacking, it may seem impossible to establish whether the specific group was experiencing a high or low mortality regime. However, the epidemiological patterns for specific groups can provide an indication of the reigning mortality regimes. When the majority of causes of death is due to infectious diseases, a high mortality regime is most likely to have been in place. A comparison of the changes in epidemiological patterns for specific groups within society over time could therefore indicate whether one or more groups gained an advantage or were disadvantaged compared to the other groups. An advantageous position would be one in which the proportion of infectious diseases in that group has decreased earlier compared to the rest of society. The expectation would be that mortality has declined at that moment as well. On the other hand, a disadvantageous position would mean the lingering of infectious diseases compared to other groups in society, and the continuation of a high mortality regime.

Central to the first analytical chapter, chapter 4, are the changes in health in the late nineteenth and early twentieth century among infants. Infant mortality remained extremely high in Maastricht for a longer period of time compared to the northern and western provinces of the country. Although boys had a slightly higher mortality rate compared to girls, most likely stemming from a biological disadvantage, these differences did not affect the start of the mortality decline, nor did they interact with socioeconomic inequalities. The main focus of the chapter is the emerging socioeconomic inequalities once the decline in mortality had started. The dominant causes of death in this age category were water and foodborne diseases, more precisely gastroenteritis and diarrhoea. By deploying a multinomial logistic regression analysis, it became clear that in the initial phase of the decline of these causes of death specifically, the upper-class infants gained an advantage. The proportion of water and foodborne causes of death of the total of water and foodborne and non-infectious causes of death was lower among upper-class infants in the initial phase of the decline compared to the proportional mortality of infants from working-class families. However, once the decline sped up and infant mortality declined immensely during the early years of World War I, the socioeconomic inequalities receded once more. The inequalities emerging during the early phase can be explained by an earlier awareness of the high infant mortality rates as problematic, yet resolvable. Most likely by adopting hygienic practices and/or breastfeeding, upper-class mothers were able to shield their infants earlier than mothers from the working classes. Working-class mothers adopted hygienic practices and/or breastfeeding *en masse* once aid at a community level offered by the *Green Cross* became available, for which they may have felt an increased need due to the hardship caused by high unemployment during WWI.

In chapter 5, the attention shifts towards a slightly older age category, the young children from age one through four. Once children had survived their first year, new hazards emerged. Mortality among young children was still high, due to the many typical childhood diseases they were exposed to. The composition of the very particular

disease environment for young children was highly important in determining the risks of death for young ones. The analysis of per cent changes in disease categories and individual diseases demonstrated that different disease mechanisms occurred. Driving the decline in early childhood mortality were two disease groups, namely gastrointestinal infectious diseases and airborne infectious diseases. During the first period of substantial decline from the 1870s to the 1880s, mortality of both disease categories declined. The categories acted either indifferently or in symbiosis. Thereafter, the two disease categories declined individually, although the decline in airborne infectious diseases in the period from the 1910s to the 1920s may have been a side effect of the decline in gastrointestinal infections for both young children and infants in the same period, causing more children to be increasingly robust and able to fight airborne diseases. At the level of individual diseases, a different mechanism of the disease environment appears. Here it seems some diseases did increase in lethality after the receding of another disease, for example when mortality from scarlet fever declined, mortality from tuberculosis and pneumonia actually increased, and later on measles became increasingly lethal.

Socioeconomic inequalities among young children seem to have appeared in the first stages of mortality decline as well, mainly when mortality from gastrointestinal infectious diseases declined. Gender differences seemed to be absent at first, there being no clear excess mortality prior or during the health transition for either of the sexes. However, at the level of individual diseases, it was found that there were a number of differences. Girls may have suffered a more structural disadvantage after all, since they experienced excess mortality regarding endemic diseases. Only for the occasional epidemic diseases did boys have higher mortality levels. Apparently, when an epidemic occurred, the biological disadvantage of boys kicked in, while in other, more normal years, girls were affected disproportionally.

Among adults, men were clearly in the worst position. Chapter 6 focuses on adult mortality and its decline. Until the health transition started, the main disease category causing a premature death among adults was that of airborne infectious diseases. In particular tuberculosis was the disease wearying the already tired bodies of industry workers, with an early death as its result. Although women had to endure the dangers of many pregnancies and births, men experienced the highest mortality rates, mainly due to these airborne infectious diseases. Mortality also declined earlier for women than it did for men. Women's mortality already declined from the late mid-century onwards, while men had to wait until the end of the nineteenth century. Once mortality declined, inequalities in socioeconomic status again emerged for both genders. However, other factors were important in determining the epidemiological patterns as well. Among men, migrants showed an advantageous disease pattern compared to native-born Maastricht men, even across the entire socioeconomic spectrum. An effect of migration status was absent for women – for women, however, it mattered whether they were married or not. Unmarried women were in the worst position, which is most likely explained by a selection effect positively selecting the most healthy women for marriage.

Several overarching conclusions emerge when combining the three analytical chapters. All three included factors – age, gender and socioeconomic status – that contributed to unequal pathways to the grave in Maastricht. However, these three factors did not always exert their influence on health inequalities in a similar fashion. Age dictated in large part the degree to which individuals were exposed to disease hazards. Some

causes of death occurred in all age categories discussed here (infancy, early childhood and adulthood), although the predominance of certain infectious diseases differed between ages. The factors influencing the decline in mortality could also differ between age groups, in particular when these factors were targeted at specific age groups. Age therefore determined when the pathways to death started to change, and thus when the mortality decline started. The influence of socioeconomic status on the shift towards a low mortality regime was fairly consistent. Socioeconomic inequalities emerged in the initial phase of the health transition. As such, socioeconomic status created fast tracks and congestions early on *en route* to a low mortality regime. Whereas socioeconomic status caused the emergence of disparities in the initial phase of the health transition and age category mainly dictated the start of the health transition, gender was a more long-lasting discriminatory factor, still creating higher levels of mortality among males after a low mortality regime had been reached. The influence of gender on the health transition was however quite complex. Depending on the age group, it could affect only different levels of mortality, yet without causing different routes towards a low mortality regime. In other age groups, gender could be as important as age in determining the start of the health transition or the predominance of certain diseases.

This case study on Maastricht has contributed substantially to our understanding of the more detailed working of the health transition and its ensuing inequalities. Socioeconomic inequalities are not a static phenomenon, they are dynamic and develop when remedies start to become available. Socioeconomic inequalities are not only connected to the availability of resources, but also the individual's receptiveness – as at least partially determined by socioeconomic status – towards new ideas on hygiene or treatment mattered. Gender inequalities have been shown to have been fairly present as well, not only stemming from biological factors, on which we have the least influence. We may feel that gender inequalities in health are nowadays only reminiscent of the past, but this is far from the case. By prioritising the inclusion of women in historical demographical studies, we can aid the understanding of the singular female health experience as well, whether this may be rooted in biological, social, or cultural influences. Using individual-level data, which allows for a highly detailed analysis of all these factors and their potential interactions, contributes to our understanding of the health transition in major ways.

Samenvatting

Dit proefschrift bestudeert de veranderingen in gezondheid en sterfte die zich de afgelopen twee eeuwen hebben voorgedaan in moderne samenlevingen, die beter bekend staan onder de overkoepelende term van de *gezondheidstransitie* (*health transition*). In de periode voordat deze transitie plaatsvond, overleed het merendeel van de populatie op relatief vroege leeftijd aan infectieziekten. In samenlevingen waarin deze transitie is voltooid, bereikt het merendeel van de populatie een hoge leeftijd en overlijden mensen met name aan degeneratieve en zogeheten *man-made diseases*, zoals kanker en hart- en vaatziekten. De veranderingen die worden verstaan onder deze gezondheidstransitie waren niet zonder meer het resultaat van ontwikkelingen in de geneeskunde of de verbetering van de welvaart, maar waren ook het gevolg van culturele veranderingen. Desalniettemin waren deze veranderingen geen gelijkmatig optredend fenomeen; ongelijkheden in gezondheid en sterfte ten gevolge van verschillen in leeftijd, geslacht en sociaaleconomische status konden bestaan, optreden of verminderen tijdens het proces. Deze gezondheidstransitie is reeds getheoretiseerd op basis van de beschikbare historische sterfte data, veelal op een geaggregeerd niveau. De veranderingen die ten grondslag lagen aan deze geaggregeerde veranderingen, op het gebied van individuele ziekten of subpopulaties, worden echter nog niet volledig begrepen.

Het doel van dit onderzoek is om het begrip van de gezondheidstransitie verder uit te breiden, door middel van het gebruik van data over doodsoorzaken op een individueel niveau. Deze individuele doodsoorzaken data zijn beschikbaar voor de Nederlandse industriestad Maastricht tussen 1864 en 1955. Hierdoor is een meer gedetailleerde analyse van de gezondheidstransitie in Maastricht mogelijk. De beschikbare data bevatten niet alleen informatie over de exacte doodsoorzaak, zoals hoe deze was vermeld door een arts, maar bevatten ook informatie over de leeftijd van overlijden, religie, sekse en het beroep van de overledene. Deze rijkdom aan data maakt het daardoor mogelijk om naar de gecombineerde effecten van deze variabelen op doodsoorzaak-specifieke sterfte te kijken. Bovendien kunnen deze doodsoorzaak-specifieke data meer inzicht geven in de determinanten die ten grondslag lagen aan het verloop van de gezondheidstransitie. Immers, wanneer de incidentie van specifieke doodsoorzaken afneemt, kan dit helpen om te begrijpen welke factoren daarvoor van belang waren, op basis van de etiologie van deze specifieke ziekten.

De centrale onderzoeksvraag van dit onderzoek is drievoudig. Ten eerste vraagt deze hoe de gezondheidstransitie plaats vond in het snel industrialiserende Maastricht. Ten tweede is de vraag of deze gezondheidstransitie tot nieuwe ongelijkheden leidde in doodsoorzaak-specifieke sterfte als gevolg van verschillen in sociaaleconomische status, leeftijd en geslacht. Ten derde is de vraag welke determinanten ten grondslag lagen aan deze gezondheidstransitie. Om deze vragen te beantwoorden, worden er verschillende onderzoeksmethoden gebruikt. De gemene deler van deze methoden is de nadruk op veranderende epidemiologische patronen, wat inhoudt dat informatieve veranderingen in de dominantie van bepaalde ziektes wordt bestudeerd. De unieke data die beschikbaar zijn voor Maastricht maken het mogelijk om op een individueel niveau doodsoorzaken te bestuderen, ondanks het gebrek van informatie over een gespecificeerde *population at risk* naar al de beschikbare karakteristieken, zoals sociale klasse. Door het bestuderen

van de veranderingen in epidemiologisch patroon voor deze specifieke groepen, in plaats van het bestuderen van de sterfte cijfers (*mortality rates*) voor deze specifieke groepen, is het mogelijk om opkomende ongelijkheden en gezondheidsvoordelen en -nadelen te benaderen. De epidemiologische patronen helpen dus om veranderingen in sterfte regimes voor specifieke groepen te lokaliseren.

De redenering die ten grondslag ligt aan deze methodologische benadering is gebaseerd op Omran's epidemiologische transitie theorie, die als een van de cruciale elementen van de bredere gezondheidstransitie theorie gezien kan worden. Om de analyse van veranderende epidemiologische patronen, als middel om potentiële ongelijkheden in gezondheid te identificeren, te begrijpen, zijn er twee belangrijke elementen van de epidemiologische transitie theorie nodig. Allereerst stelt de epidemiologische transitietheorie dat de dominantie van infectieziekten als doodsoorzaak werd vervangen door een dominantie van degeneratieve ziekten en *man-made diseases* gedurende deze transitie. Ten tweede stelt de theorie dat wanneer infectieziekten dominant zijn, er in het algeheel een hoog mortaliteitsregime is, waarbij veel jonge kinderen en volwassenen een vroegtijdige dood sterven. Een laag mortaliteitsregime is bereikt wanneer de dominantie in doodsoorzaken is veranderd, en sterfte voornamelijk nog op late leeftijd plaatsvindt. Wanneer doodsoorzaken-specifieke sterftecijfers naar sociaaleconomische status ontbreken, lijkt het onmogelijk om vast te stellen wanneer specifieke sociaaleconomische groepen gekenmerkt worden door een laag- of hoog mortaliteitsregime. Echter, de epidemiologische patronen kunnen een indicatie bieden voor de heersende mortaliteitsregimes binnen deze gespecificeerde groepen. Wanneer de meerderheid van de doodsoorzaken het gevolg is van een infectieziekte, is het waarschijnlijk dat er een hoog mortaliteitsregime was. Een vergelijking van de veranderingen in deze epidemiologische patronen tussen verschillende groepen over een langere periode, kan dus een indicatie geven of bepaalde groepen een voorsprong behaalden, of juist achterliepen. Groepen waarin de proportie van sterfte aan infectieziekten binnen het geheel van doodsoorzaken eerder daalde vergeleken met de gehele samenleving, hadden waarschijnlijk een voorsprong behaald. De verwachting is dat de sterftecijfers als geheel op dat moment namelijk ook zijn gedaald. Daartegenover staat dat een achterlopende groep waarschijnlijk langer een hogere proportie infectieziekten binnen het geheel van doodsoorzaken zal hebben ervaren, en daarmee hogere sterftecijfers, vergeleken met de samenleving als geheel.

In het eerste analytische hoofdstuk van dit proefschrift, hoofdstuk vier, staan de veranderingen in gezondheid in de late negentiende- en vroege twintigste eeuw onder zuigelingen centraal. Maastricht werd gekenmerkt door de voortduring van een bijzonder hoge zuigelingensterfte, terwijl in het westen en noorden van het land de daling van de zuigelingensterfte al goed onderweg was. Jongetjes hadden een wat hogere sterfte dan meisjes, waarschijnlijk als gevolg van een biologisch nadeel. Deze verschillen uitten zich echter niet in een verschillend startpunt van de daling in sterfte, noch hadden de sekse verschillen een duidelijk aantoonbaar verband met sociaaleconomische verschillen in sterfte. De voornaamste focus in dit hoofdstuk ligt dan ook bij de wel opkomende ongelijkheden in sterfte ten gevolge van sociaaleconomische verschillen, bij de aanvang van de sterftedaling. De dominante doodsoorzaken binnen deze leeftijdsgroep waren infectieziekten die via water en voedsel werden verspreid, in het bijzonder gastro-enteritis en diarree. Door middel van een multinomiale logistische regressie analyse

werd het duidelijk dat vooral bij de aanvang van de sterftedaling, de zuigelingen uit de hogere klassen een voordeel behaalden. In de eerste fase van de gezondheidstransitie was de proportie van deze infectieziekten die via water en voedsel werden verspreid, binnen het totaal aantal doodsoorzaken, lager voor zuigelingen uit de hogere klassen, dan voor zuigelingen uit de werkende klassen. Wanneer de daling echter verder vorderde en sterk daalde tijdens de Eerste Wereldoorlog, verdwenen de sociaaleconomische ongelijkheden in gezondheid weer. De ongelijkheden aan het begin van de sterftedaling kunnen uitgelegd worden als het gevolg van een vergroot bewustzijn van het probleem zuigelingensterfte, gepaard gaande met de gewaarwording dat dit probleem ook opgelost kon en moest worden. Door het aanleren van hygiënische praktijken en/of de jonge kindjes borstvoeding te geven, konden moeders uit de hogere klassen hun kinderen waarschijnlijk als eerste beschermen tegen de maagdarminfecties. Moeders uit de arbeidende klassen adopteerden deze hygiënische praktijken en borstvoeding op grotere schaal vanaf het moment dat hulp vanuit de eigen gemeenschap werd geboden via het *Groene Kruis*. Dit gebeurde bovendien op een moment wanneer door de hoge werkeloosheid in Maastricht ten gevolge van de Eerste Wereldoorlog, deze moeders wellicht extra genoodzaakt waren om verlichting van hun zware omstandigheden te zoeken.

In hoofdstuk vijf verschuift de aandacht naar een iets oudere leeftijdscategorie, die van jonge kinderen tussen de één en vijf jaar oud. Als kinderen het eerste jaar eenmaal hadden overleefd, dienden zich er nieuwe gezondheidsrisico's aan. Sterfte onder deze jonge kinderen was nog altijd hoog in de negentiende eeuw, met name door de typische kinderziektes waar deze kinderen aan blootgesteld waren. De samenstelling van de specifieke ziekte omgeving (*disease environment*) was van groot belang in de kansen op overlijden voor jonge kinderen. De analyse van percentuele veranderingen in ziektecategorieën en van individuele ziekten, toonde aan dat verschillende mechanismen aan het werk waren. De daling in kindersterfte werd gedreven door met name twee ziektecategorieën; aan de ene kant die van de infectieziekten die via water en voedsel werden verspreid, en aan de andere kant die van infectieziekten die via de lucht werden verspreid. Beide ziektecategorieën toonden een daling tijdens de eerste periode van substantiële sterftedaling onder jonge kinderen, tussen de jaren 1870 en de jaren 1880. De ziektecategorieën daalden ofwel zonder invloed op elkaar te hebben, ofwel in een soort symbiose. Daarna daalden de twee categorieën zonder duidelijk invloed op elkaar te hebben, al kan de daling in infectieziekten die via de lucht werden verspreid van de jaren 1910 tot de jaren 1920 deels een effect zijn geweest van de daling aan maagdarminfecties onder zuigelingen en jonge kinderen in dezelfde periode. Door deze laatste daling kunnen zuigelingen minder geschaad zijn doordat zij geen maagdarminfecties meer doormaakten, en daardoor sterker waren om met andere infectieziekten de strijd aan te gaan wanneer zij iets ouder waren. Op het niveau van individuele ziekten is een ander mechanisme in de ziekte omgeving te zien. Het lijkt dat sommige ziekten dodelijker werden op het moment dat andere ziekten waren gedaald, bijvoorbeeld wanneer de sterfte aan roodvonk daalde. Hierna steeg de sterfte aan tuberculose en longontsteking, en later leek mazelen ook dodelijker te worden.

Sociaaleconomische verschillen in sterfte onder jonge kinderen lijken wederom in de eerste fase van de gezondheidstransitie te zijn opgetreden, met name wanneer de sterfte aan maagdarminfecties daalde. Op het eerste gezicht leken ongelijkheden op

basis van geslacht in sterfte afwezig, aangezien er geen duidelijk sterfteoverschot voor een van de beide seksen was voor of tijdens de sterftedaling. Echter, kijkende naar individuele doodsoorzaken, bleek dat er toch een aantal gender verschillen was. Meisjes lijken een meer structureel nadeel gehad te hebben, aangezien zij een sterfteoverschot aan endemische infectieziekten kenden. Alleen wanneer een epidemie uitbrak, hadden jongens een sterfteoverschot. Het lijkt dat in epidemische jaren het biologische nadeel van jongetjes zich liet gelden, terwijl in niet-uitbraak jaren meisjes een disproportionele sterfte hadden.

Onder volwassenen was een duidelijker verschil tussen mannen en vrouwen te zien; de gezondheid en sterfte was duidelijk het slechtst onder mannen. Hoofdstuk zes richt zich op volwassenensterfte en de daling daarvan. Totdat de gezondheidstransitie van start ging, leidden voornamelijk infectieziekten die via de lucht werden verspreid tot een vroegtijdige dood onder volwassenen. Tuberculose was met name de ziekte die de al verzwakte lichamen van Maastrichts arbeiders aanviel, leidend tot een vroege dood. Hoewel vrouwen de gevaren van vele zwangerschappen en bevallingen moesten proberen te overleven, hadden mannen een sterfteoverschot ten gevolge van de via de lucht verspreide infectieziekten. De sterftedaling zette bovendien eerder in voor vrouwen dan voor mannen. Vanaf het midden van de negentiende eeuw daalde de vrouwensterfte al, terwijl mannen tot het einde van de negentiende eeuw moesten wachten op de sterftedaling. Voor beide geslachten ontstonden er sociaaleconomische verschillen in sterfte bij de eerste fase van de gezondheidstransitie. Onder volwassenen bestonden echter ook andere duidelijke factoren die de epidemiologische patronen bepaalden. Bij mannen was migratie een bepalende factor, waarbij mannen die de stad in gemigreerd waren een gezondheidsvoordeel lijken te hebben gehad in vergelijking met mannen die in de stad geboren waren, over het gehele sociaaleconomische spectrum. Dit effect lijkt afwezig voor vrouwen, maar voor vrouwen was de huwelijkse staat een invloedrijke factor. Ongetrouwde vrouwen bevonden zich in de slechtste positie, wat waarschijnlijk voortkomt uit een selectie effect dat de meest gezonde vrouwen positief selecteerde voor het huwelijk.

Een aantal overkoepelende conclusies kan getrokken worden wanneer de resultaten van de drie empirische hoofdstukken worden gecombineerd. Alle drie de factoren leeftijd, geslacht en sociaaleconomische status droegen bij aan ongelijke trajecten richting het graf. Deze drie factoren oefenden echter lang niet altijd dezelfde invloed uit op gezondheid en sterfte. Leeftijd bepaalde voornamelijk de mate waarin individuelen vatbaar waren voor bepaalde ziekten. Sommige doodsoorzaken kwamen voor in alle leeftijdscategorieën, al verschilde de dominantie van bepaalde ziekten per leeftijdscategorie. De factoren die van invloed waren op de sterftedaling kunnen ook per leeftijdscategorie verschillen, zeker wanneer bepaalde factoren gericht waren op specifieke leeftijdsgroepen. Leeftijd bepaalde dus wanneer de trajecten richting de dood begonnen te veranderen, en dus de sterftedaling inzette. De invloed van sociaaleconomische status op de verandering richting een laag mortaliteitsregime was redelijk consistent. Onder alle leeftijdsgroepen traden sociaaleconomische verschillen in sterfte op ten tijde van de initiële fase van de gezondheidstransitie. Op die manier creëerde sociaaleconomische status snelle wegen en opstoppingen, op de weg naar een laag mortaliteitsregime.

Geslacht was in tegenstelling tot de tijdelijke invloed van leeftijd en sociaaleconomische status op ongelijkheden in gezondheid, een meer blijvende discriminerende factor. Ook

wanneer een laag mortaliteitsregime was bereikt, bleven hogere mortaliteitslevels bestaan voor mannen. De invloed van geslacht op de sterftetransitie is echter vrij complex. Geslacht kon ofwel enkel de mortaliteitslevels beïnvloeden zonder verschillende wegen richting een lager mortaliteitsregime te creëren, ofwel de start van de sterftedaling mede bepalen, ofwel de dominantie van bepaalde ziekten beïnvloeden, afhankelijk van de leeftijdsgroep.

Deze *case-study* van Maastricht heeft substantieel bijgedragen tot ons begrip van de meer gedetailleerde processen die ten grondslag lagen aan de gezondheidstransitie, en de bijkomende ongelijkheden. Sociaaleconomische ongelijkheden zijn, zoals blijkt, geen statisch fenomeen. Ze zijn daarentegen dynamisch en ontstaan op het moment dat het mogelijk wordt om met bepaalde middelen een ziekte te lijf te gaan. Echter, sociaaleconomische verschillen in sterfte zijn niet enkel verbonden met de beschikbare remedies of middelen tegen een ziekte, maar ook met de mate waarin mensen open staan tegenover deze nieuwe remedies of hygiënische praktijken. Ongelijkheden in gezondheid als gevolg van verschillen in gender waren ook duidelijk aanwezig. Deze ongelijkheden kwamen niet alleen voort uit een biologisch mannelijk nadeel, waarop we het minste invloed hebben als mens, maar lijken ook deels voort te zijn gekomen uit ongelijke behandeling. We mogen nu wellicht het gevoel hebben dat gezondheidsongelijkheden als gevolg van verschillen in geslacht enkel een herinnering van het verleden zijn, maar dit is zeker niet het geval. Door juist de bestudering van vrouwen in de historische demografie te prioriteren, wordt het mogelijk om ook ons begrip van de uitzonderlijke historische vrouwelijke gezondheidservaring te vergroten. Met behulp van data op een individueel niveau, die een gedetailleerde analyse van biologische, sociale en culturele invloeden en hun interactie verder mogelijk maakt, kunnen we onze kennis over de gezondheidstransitie nog sterk vergroten.

Curriculum Vitae

Mayra Murkens was born in Amsterdam in 1992, where she finished her secondary education at the Fons Vitae Lyceum in 2010. She started her bachelor's degree in history in the same year at the University of Amsterdam, and continued with her Research Master's degree from 2013 to 2015 at the same university. During her bachelor, she obtained a teaching certificate of the second degree for secondary education at the University of Amsterdam by completing the educational minor. Towards the end of her bachelor's degree, her research interests had started to focus on medical history of the nineteenth and early twentieth century. This specialisation was intensified during her Research Master, when she followed and successfully completed the Medical History Master Trajectory at the VU University in Amsterdam (Vrije Universiteit). As part of her master's programme, she also did an internship at the University of Amsterdam's research group Medicine and Society, where she co-organised a symposium with this research group and the Meertens Institute on the dread of epidemics (*Angst voor de epidemie*). Her master thesis studied the practice of Dutch consultation bureaus which offered medical testing before marriage, from a eugenic perspective, in the Interwar period. In 2018 she obtained a teaching certificate of the first degree for secondary education at the University of Amsterdam, shortly before she started her PhD research at Maastricht University and the Centre for the Social History of Limburg (SHCL).

While working on the PhD, Murkens followed post-graduate education at the Posthumus research school for economic and social historians. At Posthumus, she completed a course in statistical analysis in early 2018, and she completed the Basic Training Programme in 2020. After the PhD research, Murkens started working as a postdoctoral researcher in September 2022 at Radboud University in the project 'Lifting the Burden of Disease', headed by prof. dr. Angélique Janssens. In this project, she continues to study trends in historical mortality, at an individual level, for the city of Amsterdam.

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