Maastricht's Missing Girls. A research into patterns and determinants of excess mortality among girls, 1864–1930

1 author:

Wieke Metzlar
Radboud University
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Faculty of Arts

Research Master Historical, Literary and Cultural Studies

Maastricht's Missing Girls
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Wieke Elien Metzlar

Radboud Universiteit

Dankwoord

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Abstract

Despite women having inherently better survival advantages than men, certain groups of females experience significantly higher mortality rates than their male counterparts. Today, our world counts 140 million 'missing' women that would still be alive if not for sex-selective abortions, female infanticide and excess female mortality in childhood and early adulthood.

Excess mortality patterns among girls were also evident in nineteenth- and early twentieth-century Western Europe. Proposed explanations range from (1) biological hypotheses, like girls having less resistance to communicable diseases, to (2) gendered roles, where young females - caring for the sick within households - were more exposed to infectious diseases, and (3) gender discrimination, including reduced access to food and medical care. However, to date, these explanations have not been tested at the individual level, leaving crucial questions regarding the type of girls that were at increased mortality risks and disappearance of excess female mortality unanswered.

This research examines the patterns and determinants of excess female mortality among children aged 0 to 20 between 1864 and 1930. The data of the Maastricht Death and Disease Database is used to identify causes of death that disproportionally affected girls and to study the deaths of girls compared to boys in relation to their specific socioeconomic and demographic characteristics. This research examined the role of the age category, cause of death, individual diseases, socioeconomic status of the parents, occupational exposure to infectious diseases and family composition into mortality differences between boys and girls in Maastricht.

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1. Introduction

Mortality is one of the key indicators of inequalities between people, as disparities in social positions and opportunities in life are associated with differences in the timing and causes of death.[1] One of the groups with strikingly different mortality patterns are young and adolescent women. Women inherently have better survival advantages than men. However, specific groups of girls experienced significantly higher mortality rates compared to their male counterparts, both in nineteenth-century European societies and in today's developing countries.[2] Amartya Sen's publication in the 1990s highlighted this gender inequality in mortality, revealing that over 100 million women were missing from Southern and Western Asian societies.[3] This figure has since increased, with our world today counting 140 million 'missing women', that would still be alive if not for sex-selective abortions, female infanticide and excess female mortality in childhood and early adulthood.[4]

Excess female mortality has been linked to mortality regimes in which infectious diseases are the leading cause of death (such as contemporary developing countries and nineteenth- to early twentieth-century western societies), which disproportionally affect women.[5] Theoretical explanations include (1) *biological factors*, such as girls' increased susceptibility to specific infectious diseases, (2) *gender roles*, like girls taking care for the sick in households, and (3) *gender-specific discrimination* leading to unequal access to food and healthcare.[6] The disappearance of excess female mortality in Western societies is attributed to the epidemiological transition, marked by a decline in infectious diseases with higher female mortality and an increase in degenerative diseases with higher male mortality. Other factors, such as industrialization, the social hygienist movement, and the implementation of social policies like the ban on child labour and compulsory

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[1] Amartya Sen, 'Mortality as an Indicator of Economic Success and Failure', *The Economic Journal* 108:446 (1998) 1-25, there 2-5; F.W.A. van Poppel, De 'statistieke ontleding van de dooden': een spraakzame bron? Inaugural speech (Nijmegen 1999) 1-2; Tommy Bengston, and Frans van Poppel, 'Socioeoconomic Inequalities in Death from Past to Present: An Introduction', *Explorations in Economic History* 48:3 (2011) 343-356.

[2] Isabelle Devos, 'Te jong om te sterven. De levenskansen van meisjes in België omstreeks 1900', *Tijdschrift voor Sociale Geschiedenis* 26:1 (2000) 55-75, there 56; Angélique Janssens, *Sekse, Gender en de Dood.* Inaugural speech (Maastricht 2016) 13-15.

[3] Amartya Sen, 'More than 100 million women are missing', *New York Review Books* 37:20 (1990) 61-66.

[4] Our World in Data. Number of 'missing women' in the world, 2025 to 2050 (2024) https://ourworldindata.org/grapher/global-number-of-missing-women?time=2025..latest

[5] Gregory Hanlon, *Death Control in the West 1500-1800. Sex Ratios at Baptism in Italy, France and England* (Abingdon and New York 2023); Francisco J. Beltrán Tapia and Mikolaj Szoltysek, '"Missing girls" in historical Europe: reopening the debate', *History of the Family* 27:4 (2022) 619-657; Stephan Klasen and Claudia Wink, '"Missing Women." Rivisiting the debate', *Feminist Economics* 9:2 (2003) 263-299, there 280.

[6] Frans van Poppel, Jona Schellekens and Evelien Walhout, 'Oversterfte van jonge meisjes in Nederland in de negentiende en eerste helft twintigste eeuw', *Tijdschrift voor sociale en economische geschiedenis* 6:4 (2009) 37-69, there 44; Devos, 'Te jong om te sterven', 55.

education have also been associated with the relative improvement of women's positions and the disappearance of excess female mortality.[7]

However, these explanations lack individual-level testing, as previous studies relied on aggregate data like death statistics from municipalities or regions that grouped individual data into larger categories organized by age, socio-economic status and/or cause of death. While this data enables the identification of macro-level trends, it does not allow for the disentanglement of characteristics related to the individual. Therefore, critical questions on excess female mortality persist: Did girls from impoverished labouring families face higher premature mortality rates? What role did parental care play in girls' survival chances? Were certain diseases or outbreaks more lethal to girls than to boys? What underlying causes contributed to gender differences in causes-of-death? In essence, who were those girls that died premature?

For the first time, these questions can be examined thanks to the construction of a new crowdsourced historical database, the Maastricht Death and Disease Database, encompassing data on 76,264 individuals who passed away in Maastricht from 1864 to 1955. These burial records not only document the cause of death but also offer detailed information on the deceased, facilitating an examination of their death in relation to their specific socioeconomic and demographic context. This historical research on missing women is pivotal for understanding the causal mechanisms driving excess female mortality, as historians, unlike sociologists and demographers, can employ a long-term perspective. Identifying societal contexts and individual characteristics associated with excess female mortality is crucial, as these insights can contribute to the development of better-targeted policies to protect girls in contemporary societies still experiencing excess female mortality.

This research investigates excess female mortality in the Dutch city of Maastricht from 1864 to 1930. Maastricht presents an intriguing case as the prototypical industrializing city was marked by pronounced patterns of excess female mortality, whereas elsewhere in the Netherlands excess female was predominantly a rural phenomenon.[8] The rich sources available for Maastricht allow for an in-depth examination of mortality patterns while considering changing environmental, socioeconomic, political and cultural contexts over the long term. The focus is on young and adolescent girls, an age group not extensively studied for gender differences in mortality in Maastricht.

The central question to this research is: What were the patterns and determinants of excess mortality among girls aged 0 to 20 in Maastricht between 1864 and 1930? In order to answer this question, three sub-questions will be explored: (1) What patterns of excess female mortality existed in Maastricht among children during the research period? (2) What causes-of-death disproportionally affecting girls can be discerned? (3) What potential underlying factors, including biological factors, gender roles and gender-specific

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[7] Dominque Tabutin and Michel Willems, 'Differential Mortality by Sex from Birth to Adolescence: The Historical Experience of the West (1750-1930), in: United Nations, *Too young to die: Genes or Gender?* (New York 1998) 17-53; Johan P. Mackenbach, 'The Rise and Fall of Diseases: Reflections on the History of Population Health in Europe since ca. 1700', *European Journal of Epidemiology* 36:1 (2021) 1199-1205; L. Wolleswinkel-Van Den Bosch, C. Looman, F. van Poppel and J. Mackenbach, 'Cause-Specific Mortality Trends in the Netherlands, 1875-1992: A Formal Analysis of the Epidemiologic Transition', *International Journal of Epidemilogy* 26:4 (1997) 772-781.

[8] Janssens, *Sekse, Gender en de Dood,* 13; Van Poppel, Schellekens, and Walhout, 'Oversterfte van jonge meisjes', 44; P.J.H. Ubachs, *Tweeduizend jaar Maastricht* (Maastricht 1991) 125-126.

discrimination, can explain the Maastricht patterns of excess female mortality? Using data from the Maastricht Death and Disease Database, this study will primarily utilize descriptive analyses to map out gender differences in children's' mortality patterns. Additionally, it aims to uncover insights into underlying causal mechanisms by observing various factors, such as the cause of death, age, socioeconomic status, profession, education rates and family structure.

This research will begin with an historical overview of Maastricht, detailing its economic and social developments during the late nineteenth and early twentieth century. The literature review will then discuss previous studies on excess female mortality and highlight key debates. Next the data and chosen research method will be elucidated. The fifth research chapter will analyse macro-level of excess female mortality, examining changes in mortality differences between boys and girls over time. Chapter 6 will delve into causes-of-death among girls compared to boys, examining diseases and outbreaks that disproportionally affected girls. This chapter will explore differences in causal groups of death, seasonal patterns, and gender disparities in specific diseases and disease outbreaks. Chapter 7 will investigate potential explanations, including biological factors, gender roles and gender-specific discrimination to discern factors contributing to the observed patterns from Chapters 5 and 6. Finally, the conclusion will summarize main results and suggest future research directions.

2. Living and working conditions during the nineteenth and early twentieth century in Maastricht: A brief discussion of the historical context.

Maastricht, situated in the southern Netherlands near the Meuse river and close to current Belgian and German borders, has a rich history dating back to Roman times. By the sixteenth century, Maastricht evolved into a garrison town accommodating up to six thousand soldiers.[9] At the beginning of the nineteenth century, it was a medium-sized city centring its crafts and production mainly on providing the garrison and trade with the hinterland.[10] During the Belgian Revolution (1830-1839), Maastricht was besieged, causing an economic crisis as the city was cut off from its Belgian hinterland. In response, the Dutch government mandated that materials and unfinished products from Belgium must be finished in Maastricht, laying the initial foundations for the city's industrialization.[11]

Because of high unemployment rates and a readily available cheap labour force, Maastricht became an attractive location for industrialists to establish factories.[12] In 1834, Petrus Regout opened the first factory and subsequent decades saw the establishment of others producing various industrial products like glass, ceramics and nails.[13] The construction of new transport connections, including railways to Aachen in 1853, Liège in 1861 and Venlo in 1863, further fuelled economic growth, leading factories to flourish.[14] However, what began as an agricultural crisis in the 1880s led to an economic downturn that affected the Maastricht industry. Increased prices caused smaller factories to close, while employers of larger factories faced significant wage reductions.[15] In the following decades, the economy did recover to some extent and in 1912, the industrial economy provided around 10,000 jobs in the area.[16] The Maastricht economy was impacted again during World War I – despite the Dutch neutrality – because of the city's proximity to the border. The closure of ways across land, water and rail caused a significant economic crisis in the city. After the war, Maastricht's industrial economy never fully recovered. The decline in production demand and competition from cheaper labourers from Belgium led to high and structural unemployment rates. Throughout the twentieth century, Maastricht gradually transitioned from an industrial economy to one based on service labour.[17]

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[9] Ubachs, *Tweeduizend jaar Maastricht*, 10; A.J.Fr. Maenen, *Petrus Regout 1801-1878. Een bijdrage tot de sociaal-economische geschiedenis van Maastricht* (Nijmegen 1959) 1.

[10] Maenen, *Petrus Regout*, 1.

[11] Mayra Murkens, *Unequal pathways to the grave? Time lags and inequalities in the Dutch health transition, the case of Maastricht, 1864-1955,* Doctoral Thesis (Maastricht 2023) 54.

[12] Ubachs, *Tweeduizend jaar Maastricht,* 202.

[13] Ad Knotter, 'De 'sociale kwestie'. Industrie, arbeid en arbeidsverhoudingen in de negentiende en twintigste eeuw tot circa 1940', in: Paul Tummers, Louis Berkvens, Arnoud-Jan Bijsterveld, Ad Knotter, Leo Wessels, Frans Hermans and Eric van Royen, *Limburg. Een geschiedenis vanaf 1800* (Maastricht 2015) 417-439, there 421.

[14] Maenen, *Petrus Regout,* 31.

[15] Ubachs, *Tweeduizend jaar Maastricht,* 139.

[16] Knotter, 'De 'sociale kwestie'', 421.

[17] Ubachs, *Tweeduizend jaar Maastricht,* 159.

Throughout the nineteenth century working conditions in Maastricht were harsh and hazardous, with high unemployment rates and low wages. More than half of the households were among the lowest income groups.[18] Working days were long, conditions were dangerous, and illiteracy rates were high among the factory workers, as the work did not require high levels of schooling.[19] Even more skilled professions, like pottery and glassmaking were often detrimental to health.[20] However, the industrial economy did provide job opportunities for women, with 46 percent of employees in the Regout pottery factory being women by 1893.[21] Child labour was also prevalent, with in general more boys than girls being employed in the factories, until the 1874 'Kinderwetje van Van Houten (*Childrens' law of Van Houten*) prohibited employment of children under 12 in factories.[22]

The living and working conditions in nineteenth-century Maastricht were deplorable, marked by frequent outbreaks of infectious diseases, leaving clear traces in the mortality pattern.[23] Streets were dirty, rubbish heaps were located in the middle of neighbourhoods and the average single-room house housed 8.8 people in 1851.[24] Disease transmission was facilitated by these poor living conditions and throughout the century, Maastricht suffered from numerous epidemics, including typhus (1848), diphtheria (1854), and cholera (1866). Some efforts were made to improve conditions, yet these mainly benefited the wealthier people. After the garrison's abolishment in 1867, the city could finally expand and battle the overcrowded living conditions in the centre. However, these newly built houses were unaffordable for most, leaving the majority in poor conditions.[25] From 1888 onwards, the city implemented piped water systems to enhance sanitary circumstances, but again, these initiatives prioritized wealthier neighbourhoods and by 1922, only halve of the population had access to piped water.[26]

During the French period (1894-1814), initial initiatives to improve public health were undertaken, leading to more centralized healthcare. Provincial committees documented vaccine distributions of doctors and published annual epidemiological reports to track infectious disease spreads.[27] Some other initiatives aimed to improve air quality, following miasma theory which argued that disease spread was caused by bad airs, while the hygienist (*hygiënisten*) movement advocated for sanitary reforms.[28] The 'Burgerlijk Armbestuur' (*Civil Poor Relief*) was appointed to provide public medical care to whom it was unaffordable, though the quality of care often fell short. Also professional health care often lacked advanced methods and techniques, which caused people to make rarely use of medical care institutions.[29] From 1884, doctor L. Th. van Kleef

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[18] Murkens, *Unequal pathways to the grave?*, 54-55.
[19] Ubachs, *Tweeduizend jaar Maastricht,* 155.
[20] Murkens, *Unequal pathways to the grave?*, 55.
[21] Knotter, 'De 'sociale kwestie'', 422.
[22] Maenen*, Petrus Regout*, 257.
[23] Ubachs*, Tweeduizend jaar Maastricht,* 131.
[24] Maenen, *Petrus Regout*, 50 and 75.
[25] Ubachs*, Tweeduizend jaar Maastricht*, 127-128 and 131.
[26] Murkens, *Unequal pathways to the grave?,* 56.
[27] Ibid, 64 and 67.
[28] Ubachs*, Tweeduizend jaar Maastricht*, 60.
[29] Murkens*, Unequal pathways to the grave?,* 68.

significantly improved healthcare quality at the Calvariënberg hospital, making the hospital one of the frontrunners nationwide and enhancing the public trust.[30]

Nineteenth-century modernisation processes, including advancements in housing, nutrition, and healthcare, led to a gradual improvement in living standards for populations. Life expectancy at birth increased between 1860 and 1948, with women's life expectancy rising from 38 to 69 years and men's from 33 to 66 years.[31] This rise in life expectancy was primarily caused by a shift in the mortality landscape. Maastricht, like the rest of Europe, experienced a health transition during the nineteenth century, marked by a decline in mortality and a shift from infectious to degenerative diseases as the primary cause of death.[32] Figure 1 illustrates this decline in mortality rates for the total male and female population in Maastricht. However, it is important to note that this transition was not uniformly experienced across all segments of the population, as improvements in health and wealth were unevenly distributed among different social classes.[33] Additionally, despite the reduced significance of infectious diseases in mortality patterns, Maastricht continued to face

Figure 1 Mortality pattern of Maastricht, all ages combined,1864-1930.
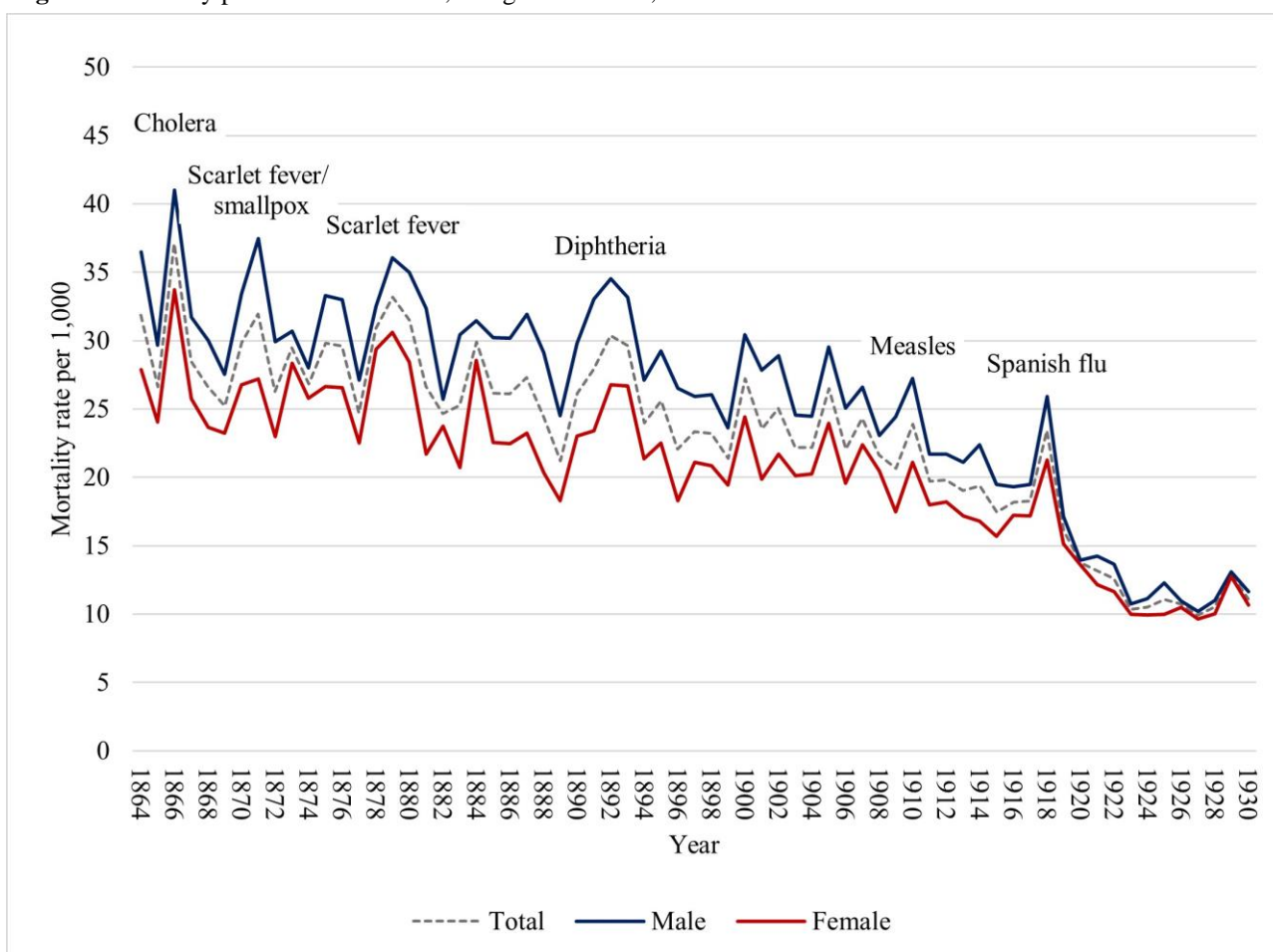
Source: Maastricht Death and Disease Database; Verslag van den toestand der gemeente Maastricht 1864-1930.

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[30] Ubachs, *Tweeduizend jaar Maastricht,* 131.

[31] Murkens, *Unequal pathways to the grave?,* 59.

[32] J. Cleland, 'The Idea of the Health Transition', in: J.C. Caldwell et al. (eds.), *What We Know About the Health Transition: The Cultural, Social and Behavioural Determinants of Health. The Proceedings of an International Workshop* (Canberra 1990) I.

[33] Angus Deaton, *The Great Escape: Health, Wealth and the Origins of Inequality* (Princeton 2013).

significant epidemic outbreak until the early twentieth century, such as measles (1910) and the Spanish flu (1918), indicating that infectious diseases still played a role in the mortality pattern.

The decline in mortality also contributed to a gradual increase of Maastricht's population, from 25,483 in 1840 to 34,220 in 1900.[34] Contrary to other industrializing cities that experienced high levels of rural-to-urban migration, Maastricht's net migration was close to zero (see Annex I).[35] Many migrants who did come for work stayed for only a year, due to the lack of proper and sufficient housing and the lack of improvements in life they had envisioned.[36] The city did experience a large influx of migrants during World War I, as over 14,000 Belgians fled to Maastricht in the first weeks.[37] Alongside the economic crisis caused by the war, this influx of people caused food shortages in Maastricht, leading to malnutrition. This, in turn, increased susceptibility to infectious diseases, which increased the impact of the Spanish Flu in the region.[38] The last large change in the population's structure occurred in 1920 when the total population of Maastricht rapidly surged, but this was mainly caused by the annexation of neighbouring municipalities, which increased the total population.[39]

In short, throughout the long nineteenth century, Maastricht faced poor living and working conditions, and due to industrialization these deteriorated further. The city was overcrowded, work conditions were hazardous and public health was poor. These conditions impacted the quality of life in terms of health and health outcomes, as it gave way to outbreaks of infectious diseases. The question is whether these deplorable conditions were evenly experienced among boys and girls in Maastricht or if gender inequalities occurred. Were younger or older girls more affected by these living circumstances? Did the industrialization of Maastricht improve or deteriorate the position of girls and what changes in the disease environment can be linked to an improvement of female health outcomes?

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[34] Theo Engelen, *Van 2 naar 16 Miljoen Mensen: Demografie van Nederland, 1800-Nu* (Amsterdam 2009) 15.
[35] Ibid, 56; Knotter, 'De 'sociale kwestie'', 422.
[36] Murkens, *Unequal pathways to the grave?,* 63.
[37] Theo Engelen, 'De bevolking van Limburg', in: Paul Tummers, Louis Berkvens, Arnoud-Jan Bijsterveld, Ad Knotter, Leo Wessels, Frans Hermans and Eric van Royen, *Limburg. Een geschiedenis vanaf 1800* (Maastricht 2015 447-460, there 460.
[38] Ubachs, *Tweeduizend jaar Maastricht,* 150.
[39] Murkens, *Unequal pathways to the grave*?, 56.

3. Social disparities and health outcomes: A literature review on causes of mortality differences between boys and girls.

3.1 Introduction

The study of mortality can shed light on one of the most extreme consequences of social disparities. Amartya Sen argued, amongst others, in the 1980s that differences in health outcomes stem not only from biological factors, but also from social positions, shaped by social, economic and cultural factors. These social positions impact experiences and opportunities in life, such as labour opportunities, living conditions, and access to resources, which eventually shape health outcomes. Sen therefore argued that the study of mortality can be used to uncover underlying social disparities in societies.[40]

Since the 1980s, mortality differences have been frequently examined through the lens of two social categories: sex and gender. The study of sex differences mainly focusses on differences between males and female in health outcomes. Gender, meanwhile, encompasses the social-cultural construct of masculinity and femininity, expressed through normative gender roles that shape expectations and behaviour and eventually impact health outcomes.[41] Relative differences between these two genders can be caused by cultural, political and economic contexts and they might show intersections with other social categories like religion, social class, ethnicity and race. Differences in the positions between genders have been present in the past, and continue to cause disparities in today's society, making it one of the most influential social categories in societies.[42] Women have predominantly had a subordinate position to men throughout history, with some periods or places with smaller, but still present differences.[43] This gender inequality affects many aspects of life, from expected behaviour, to clothing, to nutrition, to care and eventually it even causes differences in health outcomes between men and women, which has been discussed in depth in the so called 'missing girls debate'.[44]

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[40] Sen, 'Mortality as an Indicator, 1-25.

[41] Janssens, *Sekse, Gender en de Dood,* 5; Van Poppel, 'De statistieke ontleding van de dooden', 9.

[42] Graziela Dekeyser and Paul Puschmann, 'Women and men in a changing world. Tracing and eliminating the roots of gender disadvantages' in: Jan van Bavel, David de Conick, Paul Puschmann, and Bart van de Putte, *Neurotic Doubt and Sacred Curiosity, Essays in honour of Koen Matthijs* (Leuven 2021) 349-364, there 351.

[43] Ibid, 352.

[44] Sen, 'Mortality as an Indicator, 1-25; Janssens, *Sekse, Gender en de Dood,* 16.

3.2 Missing girls debate

Research on sex and gender differences has been conducted for centuries. As early as 1662, John Graunt noted that women in England had longer life expectancies than men.[45] However, in 1864, William Farr observed that women exhibited higher age-specific mortality rates than men between young childhood and middle ages, indicating that mortality risks were context-specific and can shift throughout the life course.[46] In the latter half of the twentieth century, more historical researchers, such as Sheila Johansson, Michel Poulin and Dominique Tabutin, began focusing on gender differences in historical Europe to examine underlying social position disparities between men and women.[47] From the publication of Sen in the 1990s onwards, the paradigm shifted. Instead of asking why men exhibited higher mortality rates, the focus turned to understanding why, in specific circumstances, certain women experienced higher mortality rates than men.

In a series of publications at the end of the twentieth century, Sen introduced the concept of 'missing women'. This idea revolves around the disparity between sex ratios at birth and later in life. More men are born, causing a male surplus in the sex ratio of populations. Later in life, this ratio turns into a female surplus as women inherently have better survival chances and longer life expectancies than men. However, Sen identified several Asian countries at the end of the twentieth century with pronounced male surpluses in the populations later in life. To quantify this discrepancy, Sen compared the sex ratio at birth to later ages, calculating the number of missing women. These women would have been alive if not for female infanticide and excess female mortality in childhood, adolescence and motherhood.[48]

Sen's estimates revealed that in 1990 over 100 million women were missing from Western and Southern Asian societies, making it one of the largest human catastrophes of the twentieth century. To provide context, Stephan Klasen and Claudia Wink demonstrated that this number surpasses the combined casualties of the First and Second World Wars.[49] Even today, this number of missing women continues to increase, with recent estimates counting over 140 million women that disappeared from contemporary societies.

Since Sen's publication, studies focused on the concept of missing women and girls. Historical studies primarily concentrate on female infanticide and excess female mortality, as sex-selective abortions have only become feasible in more modern times with the advent of prenatal ultrasound technology. Research on European societies has shown that excess female mortality was most prevalent at the end of the nineteenth and beginning of the twentieth century, with spatial variations in intensity and timing. However, due to limited

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[45] John Graunt, *Natural and policital observations mentioned in a following index and made upon the bills of mortality* (London 1662) available on: https://quod.lib.umich.edu/e/eebo/A41827.0001.001?view=toc.

[46] William Farr, *English life talbe. Tables of lifetimes, annuities, and premiums,* (London 1837) available on: https://wellcomecollection.org/works/zxmauyfr.

[47] S. Ryan Johansson, 'Sex and death in Victorian England. An examination of age- and sex-specific death rates, 1840-1910', in: M. Vicinus (ed., *A widening sphere: Changing roles of Victorian women* (Bloomington 1977) 163-181; Michel Poulain and Dominque Tabutin, 'La surmortalité des petites-filles en Belgique, aux XIXe et XXe siècles', *Annales de Démographie Historique* (Paris 1981) 105-118.

[48] Sen, 'More than 100 million women', 61-66.

[49] Klasen and Wink, 'Missing Women', 264.

available sources from earlier periods, it remains unclear whether excess female mortality emerged in nineteenth-century Europe or if its origins date back further into the past.

Research on nineteenth-century European societies observed higher mortality rates among girls aged 5 to 20 and among women of reproductive age.[50] Isabelle Devos identified two distinct traditions in historical research on excess female mortality in Europe. The French school primarily focused on mapping patterns of excess female mortality, emphasizing intensity, developments and spatial differences. The Anglo-Saxo tradition included more economic research and concentrated on determinants of increased mortality risks among women.[51] However, academic interest in excess female mortality gradually waned after numerous studies mapped historical patterns in Europe. This decline was partly due to the limitations of available sources, like aggregated death reports, which allowed for identifying macro-level patterns but did not enable the identification of individual characteristics associated with increased mortality risks.

In the last decade, the debate on missing women in historical Europe has regained attention. With the availability of more individual-level data and more advanced research techniques on chances of survival and death, new dimensions of research have opened-up.[52] Fransisco Beltrán Tapia and Mikolaj Szoltysek emphasized, for instance, in their 2022 article the importance of this development. They suggested that new research should focus on mapping patterns of excess female mortality over time and place, and identifying possible determinants that put girls at greater risks of death, in order to create a deeper understanding of this mortality pattern in historical Europe.[53]

3.3 Determinants of excess female mortality

The body of research on missing girls has proposed various explanations, which will be categorized in this study according to the conceptual distinction by Wiley Henry Mosley and Lincoln Chen between proximate and ultimate determinants. Proximate determinants directly influence the health outcome, while ultimate determinants refer to underlying processes which operate *through* the proximate determinants.[54] Figure 2 illustrates the conceptual framework of explanations for excess female mortality that will be used in this research. This chapter will first discuss proximate determinants and then turn attention to ultimate factors. It is crucial to note the intersectionality among these determinants, as different combinations can lead to varying

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[50] Edward Shorter, *Women's Bodies. A Social History of Women's Encounter with Health, Ill-Health, and Medicine* (New Jersey 1991 228.

[51] Devos, 'Te jong om te sterven', 61.

[52] Paul Puschmann, Hideko Matsuo and Koen Matthijs, 'Historical Life Courses and Family Reconstitutions. The Scientific Impact of the Antwerp COR*-Database', *Historical Life Course Studies* 12:1 (2022) 260-278.

[53] Beltrán Tapia and Szoltysek, 'Missing girls', 619-657.

[54] W. Henry Mosley and Lincoln C. Chen, 'An Analytical Framework for the Study of Child Survival in Developing Countries', *Population and Development Review* 10:1 (1984) 22-45.

health outcomes. As this study focuses on mortality of young and adolescent girls, this literature review will not cover determinants related to later life mortality risks of women, such as pregnancy-related risks.[55]

Figure 2 Framework of proximate and ultimate determinants of excess female mortality.
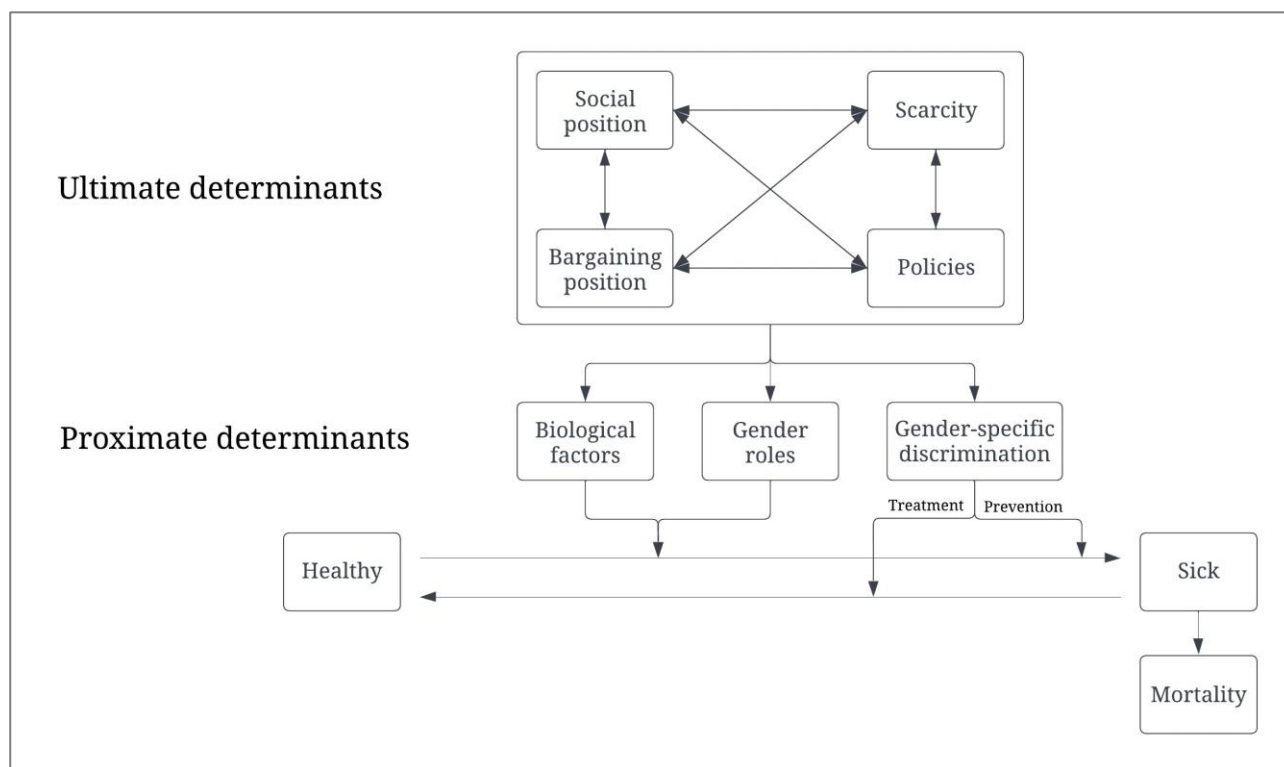
3.4 Proximate determinants

Initially, research predominantly centred on biological differences between men and women, attributing higher susceptibility to certain diseases among women as the reason for mortality disparities. Subsequently, studies emphasized the importance of gender roles, suggesting that behavioural differences influenced mortality risks. Lastly, gender-specific discrimination, resulting in unequal treatment of boys and girls, has been identified as a factor contributing to unequal health outcomes. The following factors of these three groups of proximate factors will be discussed:

- Biological factors: susceptibility; male disadvantage.
- Gender roles: household tasks; working conditions; accident risks.
- Gender-specific discrimination: female infanticide; food distribution; (health)care.

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[55] Although it could be possible that some girls in the research sample were mothers, the average age women received their first child in the Netherlands in the nineteenth century was between 24 and 26 years. The majority of girls up to 20 years were therefore not likely to have conceived a child. Ruben van Gaalen and Frans van Poppel, 'Leeftijd moeder bij eerste geboorte sinds 1850', *Demos* 23:4 (2007).

3.4.1 Biological factors

During the nineteenth and early twentieth centuries, infectious diseases were the primary cause of death in European societies, disproportionally affecting women.[56] Louis Henry demonstrated that girls between the ages of 5 and 19 contracted tuberculosis and other infectious diseases significantly more often than boys, leading to higher mortality rates among girls.[57] Dominique Tabutin and Michel Williams also noted: "Almost everywhere in the Western world, girls and young women aged 5-19 years had at least a 29 per cent higher probability of dying from tuberculosis than their male counterparts."[58] Studies therefore pointed to biological differences between males and female as the key factor contributing to mortality rate disparities.

Susceptibility – Numerous authors argued that women's higher mortality rates from infectious diseases stem from their biologically higher susceptibility.[59] Henry proposed that hormonal changes during puberty increased vulnerability, particularly to tuberculosis. [60] Shorter identified in *Women's Bodies* in 1991 menstruation as a factor, arguing that blood loss during menstruation leads to iron deficiency, which weakened immunity.[61] Henry attributed the increased female mortality during the nineteenth century to industrialization and urbanization, which facilitated disease spread in crowded areas, causing more women to contract diseases due to their higher susceptibility.[62] Tabutin and Willems also emphasized that the pattern of excess female mortality disappeared at the societal level with the decline of infectious diseases as the primary cause of death at the beginning of the twentieth century, yet they emphasized that women continued to have higher mortality rates from such diseases. [63] Others, stressed that while biological factors may contribute, excess female mortality cannot solely be attributed to them. Devos, for example, acknowledged that menarche increases disease susceptibility, but emphasized that this vulnerability is linked to malnutrition. Insufficient protein intake during menarche heightens susceptibility, highlighting the role of nutrition in determining health outcomes.[64]

Male disadvantage – Moreover, numerous studies demonstrated that women inherently have higher survival advantages compared to boys, demonstrating that men, rather than women, appear to be more susceptible to infectious diseases.[65] Beltrán Tapia and Szoltysek argued that more boys than girls die for natural

[56] Shorter, *Women's Bodies,* 231

[57] Louis Henry, 'Men's and Women's Mortality in the Past', *Population* 44:1 (1989) 177-201, there 193.

[58] Tabutin and Willems, 'Differential Mortality', 44; Shorter, *Women's Bodies,* 237.

[59] Tabutin and Willems, 'Differential Mortality', 36-37.

[60] Henry, 'Men's and Women's Mortalty', 193.

[61] Shorter, *Women's Bodies,* 247.

[62] Henry, 'Men's and Women's Mortality', 196-197.

[63] Tabutin and Willems, 'Differential Mortality', 45-46.

[64] Devos, 'Te jong om te sterven', 40.

[65] Hiram Beltrán-Sánchez, Caleb E. Finch and Eileen M. Crimmins, 'Twentieth century surge of excess adult male mortality', *PNAS* 112: 29 (2015) 8993-8998; Greg L. Drevenstedt, Eileen Crimmins, Sarinnapha Vasunilashorn and Caleb E. Finch, 'The rise and fall of excess male infant mortality', *PNAS* 105:13 (2008) 5016-5021.

reasons in utero, during infancy, and during early childhood.[66] Biologist Ingrid Waldron concluded in 1983 that women's genes exhibit greater resistance to infectious diseases.[67] Zarulli et al. observed in 2018 that the female survival advantage persists during extreme mortality periods like epidemics or famines, highlighting women's inherent biological advantage. They attributed variations in this advantage to social factors modulating women's survival advantage over men across different contexts.[68]

3.4.2   Gender roles

The second group of explanatory factors highlights behavioural differences between boys and girls, shaping differences in mortality patterns due to variations in exposure and resistance to diseases. Antonella Pinnelli and Paola Mancini suggested that these gender role difference appear as early as age five, coinciding with the age at which patterns of excess female mortality are typically observed in most Western European historical societies.[69]

Household tasks – The household was one of the places where gender differences between boys and girls were evident. Girls and women were assigned to more tasks and duties within the home, while boys and men engaged more in activities outside the household. Living conditions in the nineteenth century were often unhealthy due to poor ventilation, high humidity, inadequate hygiene facilities, and often high numbers of people living in the same room.[70] Tabutin and Willems argued that transmission of infectious diseases is greatly affected by the number of people living in the room, as well as the ventilation of rooms.[71] Girls, spending more time in these conditions, therefore had higher risks of contracting diseases compared to boys who spent more time outdoors.[72] Additionally, girls and women often cared for the sick in the household, increasing their direct contact with diseases and further raising their risks of contracting diseases themselves.[73]

Working conditions – Female working conditions have been highlighted as another factor. Shorter argued that physically demanding agricultural labour, combined with malnutrition, took a toll on girls and women.[74] Devos identified the textile industry, where many women worked, as having harsh conditions with

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[66] Beltrán Tapia and Szoltysek, 'Missing girls', 621 and 623.

[67] Ingrid Waldron, 'Sex Differences in Human Mortality: The Role of Genetic Factors', *Social Science & Medicine* 17:6 (1983) 321-333, there 325.

[68] Virginia Zarulli, Julia A. Barthold Jones, Anna Oksuzyan, Rune Lindahl-Jacobsen, Kaare Christensen and James W. Vaupel, 'Women live longer than men even during sever famines and epidemics', *PNAS* 115:4 (2018) 832-840.

[69] Antonella Pinnelli and Paola Mancini, 'Mortality Peaks in Italy in the Late 19th and Early 20th Centuries: Trends by Age and Sex', *European Journal of Population / Revue Européenne de Démographie* 14:4 (1998 / 1999 333-365, there 360.

[70] Van Poppel, 'De statistieke ontleding van de dooden', 16.

[71] Tabutin and Willems, 'Differential Mortality', 48.

[72] Devos, 'Te jong om te sterven', 70; Tabutin and Willems, 'Differential Mortality', 48.

[73] Tabutin and Willems, 'Differential Mortality', 48; Pinnelli and Mancini, 'Mortality Peaks in Italy', 355; Paul, Puschmann, 'Discriminatie met dodelijke gevolgen: oversterfte onder Kosovaarse meisjes en vrouwen in de tweede helft van de twintigste eeuw', *Ex Tempore. Verleden Tijdschrift Nijmegen* 2:27 (2008) 141-160, there 142; Van Poppel, 'De statistieke ontleding van de dooden', 16; Murkens, *Unequal pathways to the grave?*, 24.

[74] Shorter, *Women's Bodies,* 237-239.

long hours and intense working rhythms, often in unhygienic workshops with dust and high temperatures, causing health issues.[75] However, some disagree that working conditions explain mortality differences. Henry noted that excess mortality among girls aged 5 to 9 could not be solely attributed to working conditions, as they were unlikely to work full days.[76] Tabutin and Willems did acknowledge the role of hazardous working conditions in increased women's mortality risks at older ages. However, they observed excess female mortality even among very young children (1-4 years old) and in rural pre-industrial societies without the hazardous industrial working conditions, suggesting that working conditions were not the roots of the unequal health outcomes among young children.[77]

Accident risks – Exposure to fatal accidents have also been suggested as a factor influencing different mortality rates between boys and girls. Van Poppel mentioned England as an example, where girls faced more deadly accidents related to burns and boiling water because of household tasks. Doctors attributed these accidents to the highly flammable clothes worn by girls and emphasized the stiffness of these cloths, which restricted their ability to flee from danger.[78] Others, however, argued that boys had higher risks of accidents or other non-natural death. Shorter noted that more men died from alcoholism, violence, accidents, and suicide in the nineteenth century due to more risk-seeking and reckless behaviour.[79] Others emphasized that boys worked more and from younger ages in industries compared to girls, leading boys to be more exposed to hazardous working conditions and potential accidents.[80]

3.4.3   Gender-specific discrimination

Explanations of gender-specific discrimination suggest that the perceived lower value of girls leads to conscious and subconscious gender biases, resulting in unequal parental treatment of daughters and sons.[81] In cultural climates where women are subordinated to men, the birth of a son may be celebrated, while the birth of a daughter could be seen as a burden.[82] Pronounced son preferences leads to preferential treatment of sons and relative discrimination against daughters, which in extreme cases can result in higher morbidity and earlier mortality among them.[83]

Female infanticide – Female infanticide is one of the most pronounced discriminatory practices towards girls, yet its prevalence in historical Europe remains difficult to ascertain due to scarcity of sources.

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[75] Devos, 'Te jong om te sterven', 71.
[76] Henry, 'Men's and Women's Mortality', 195.
[77] Tabutin and Willems, 'Differential Mortality', 49.
[78] Van Poppel, 'De statistieke ontleding van de dooden', 16.
[79] Shorter, *Women's Bodies,* 236.
[80] Tabutin and Willems, 'Differential Mortality', 48.
[81] Mads L. Perner, A.K. Mortensen, H. Castenbrandt, A. Løkke and B.A. Revuelta-Eugercios, 'Gendered Mortality of Children and Adolescents in Nineteenth-Century Denmark. Exploring Patterns of Sex Ratios and Mortality Rates', *The History of the Family* (2022 679-701, there 682; Devos, 'Te jong om te sterven', 70.
[82] Tabutin and Willems, 'Differential Mortality', 47.
[83] Ibid, 47.

Beltrán Tapia and Szoltysek suggested that skewed sex ratios at birth, like in in nineteenth-century Greece, indicated widespread infanticide, whereas in other European societies, such skewed ratios appeared later in life, implying differential treatment of boys and girls during infancy and early childhood.[84] Similarly, Perner et al. concluded that female infanticide was generally uncommon in Europe, and very rare in north-western Europe.[85] However, it is important to note that historical birth records could be imprecise, potentially distorting sex ratios and leading to an over- or underestimation of excess female mortality in the first years of life.[86]

Food distribution – Differential treatment between sons and daughters in later ages could be manifested in the unequal allocation of food within families. After breastfeeding, which protected children equally, children started to compete for scarce household resources, with food being distributed unequally between boys and girls.[87] Devos stated for instance: "Mothers and daughters ate after fathers and sons" and often, the best meat was reserved for the men.[88] These lower quantities and qualities of food have impacted the girls' health, as adequate nutrition is a key factor to health.[89] However, studies by Richard Wall in 1981 and David Courtwright in 1990 showed that boys in respectively England and the United States boys were more often malnourished than girls at the beginning of the twentieth century, suggesting that gender discrimination against girls in food distribution was not prevalent.[90] Additionally, Klasen and Wink argued in 2003 that differences in access to food played a minimal role, if any, in increased mortality risks among girls.[91]

(Health)care – Klasen and Wink argued that inequalities in care and healthcare shaped differences in disease prevention and treatment.[92] Wall's 1981 study showed that at the beginning of the twentieth century, 39 per cent of school girls in Stattforshire had verminous heads compared to 8 per cent of the boys. In Brighton schools during the same period, more boys than girls were found with handkerchiefs, potentially indicating prioritization of sons' personal hygiene by parents.[93] Tabutin and Willems emphasized that this relative neglect of girls' hygiene might have increased their risks of contracting infectious diseases, particularly tuberculosis.[94] Others argued that discrimination in access to health care caused differences in morbidity and mortality rates. Puschmann noted that until the beginning of the twentieth century, girls were less frequently vaccinated against infectious diseases than boys.[95] Tabutin and Willems, as well as Van Poppel mentioned that boys were more often treated in hospitals compared to girls, although details on duration of sickness before hospitalization and

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[84] Beltrán Tapia and Szoltysek, 'Missing girls', 635-636 and 623.
[85] Perner et al., 'Gendered Mortality of Children and Adolescents', 680.
[86] Beltrán Tapia and Szoltysek, 'Missing girls', 629.
[87] Ibid, 635.
[88] Devos, 'Te jong om te sterven', 71.
[89] Sen, 'More than 100 million women', 61-63; Devos, 'Te jong om te sterven', 71; Shorter, *Women's Bodies, 1*9-22.
[90] Richard Wall, 'Inferring Differential Neglect of Females from Mortality Data', *Annales de démographie historique* (1981) 119-140, there 130; David T. Courtwright, 'The Neglect of Female Children and Childhood Sex Ratios in Nineteenth-Century America: A Review of the Evidence', *Journal of the Family* 15:3 (1990) 313-323, there 316.
[91] Klasen and Wink, 'Missing Women', 281.
[92] Ibid, 280.
[93] Wall, 'Inferring Differential Neglect of Females', 130.
[94] Tabutin and Willems, 'Differential Mortality', 47.
[95] Puschmann, 'Discriminatie met dodelijke gevolgen', 142.

treatment were lacking.[96] Henry, however, was unconvinced that differences healthcare access between boys and girls could have led to differences in health outcomes. He argued that excess female mortality occurred when disease treatments were insufficiently developed.[97] Devos also mentioned the general lack of trust in professional health care, which resulted in few children visiting doctors by the end of the nineteenth century, suggesting a minor role for differences in healthcare access.[98]

3.5 Ultimate determinants

Ultimate determinants named in the debate on excess female mortality often included socioeconomic and cultural factors that affected the role of the above discussed biological factors, gender roles and gender-specific discrimination. This section will address four groups of ultimate determinants. However, it is important to note that this conceptual framework may not always capture the complex reality, as certain ultimate determinants may intersect with each other or interact with several proximate determinants simultaneously. The next section will discuss the following four groups of ultimate determinants, and will link them to the proximate determinants they operated through.

- Social position: position of women; position of the child.
- Scarcity: socioeconomic status; economic shocks; family composition.
- Bargaining position: labour opportunities; family system; marriage norms
- Policies

3.5.1   Social position

The social position of girls in the nineteenth century was influenced by two main factors: the position of women and the position of children. Various socioeconomic and cultural developments during this period impacted these factors, leading to both positive and negative changes that ultimately shaped the social position of girls. These changes could have influenced certain gender roles, for instance in the household, and could have also caused changes in the allocation of resources and care between daughters and sons.

Position of women – For centuries, women were confined to subordinate roles to men, with marriage and motherhood often considered their main objectives in life. Nineteenth-century developments, like the emergence of the male breadwinner ideal, pushed women out of the public sphere into the household sphere. This marginalized women further, reinforcing gender divisions in the household and potentially lowering their

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[96] Tabutin and Willems, 'Differential Mortality', 47; Van Poppel, 'De statistieke ontleding van de dooden', 17.
[97] Henry, 'Men's and Women's Mortality', 195.
[98] Devos, 'Te jong om te sterven', 71-72.

perceived value, which could have triggered discriminatory practices in terms of food distribution and (health)care.[99] However, towards the end of the century, societal critiques emerged. The first feminist wave advocated for women's emancipation through initiatives such as improved education and voting rights for women, potentially reducing gender disparities and discriminatory practices.[100]

Position of children – During the nineteenth century, the social position of children underwent changes. Historians argued that childhood began to be seen as a distinct phase of life rather than simply a stage of non-adulthood. Philippe Ariès argued that this 'discovery of the child' led to increased attention to children's upbringing and education, potentially improving parental care and the overall health status of children, particularly for girls if they were previously treated worse.[101] However, this period also saw a rise in child labour in industrial factories, with long working hours, low wages, and hazardous conditions that negatively impacted the children's health. [102] Towards the end of the century, protective policies such as the law of Van Houten in 1874 – prohibiting employment of children under twelve years – and the compulsory education act in 1901 aimed to safeguard children, reducing disparities in gender roles of children under twelve years by mandating education and prohibiting their employment.[103]

3.5.2   Scarcity

Scarcity has been suggested as a crucial ultimate determinant in the debate on excess female mortality. Scarcity of nutrition has been linked to biological factors contributing to girls' increased susceptibility. Additionally, it could affect gender roles within families, with women possibly needing to contribute more in resource-limited households. Finally, scarcity prompts gender-specific discrimination, with parents favouring sons over daughters in times of resource shortages. Additionally, the individual's ability to acquire the necessary resources for survival, known as their bargaining position, also influenced the impact of scarcity. The role of the bargaining position will be discussed in the next section.

Socioeconomic status – Excess female mortality is often associated with poorer families in agricultural settings, as the mortality pattern has primarily been observed in rural regions. [104] Few studies have explored the impact of socioeconomic status on excess female mortality due to data constraints, but those that have examined social class found indeed a relationship between lower classes and excess female mortality. For instance, Kent Johansson's 2010 study in Scania, Sweden, revealed that daughters of landless parents faced

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[99] Stephanie Coontz, *Marriage, a History. How Love Conquered Marriage* (New York 2005) 146.

[100] Ulla Jansz and Tineke van Loosbroek, 'Nieuwe literatuur over de eerste feministische golf: herschrijven van de geschiedenis', in: Jeske Reys, Tineke van Loosbroek, Ulla Jansz, Maria Henneman, Annemarie de Wildt and Mirjam Elias (eds., *De eerste feministische golf* (Nijmegen 1985) 10-29, there 13.

[101] Philippe Ariès, *Centuries of Childhood. A Social History of Family Life* (New York 1962).

[102] Ubachs, *Tweeduizend jaar Maastricht*, 267-270.

[103] Ibid, 257.

[104] C. Ginsberg and A. Swedlund, 'Sex-specific mortality and economic opportunities: Massachusests 1860-1899', *Continuity and Change* 1:1 (1986) 415-445.

higher mortality risks compared to those from smaller or larger farmer families.[105] Similarly, research by Van Poppel, Schellekens and Walhout in 2009 showed that daughters of unskilled labourers had elevated mortality risks, while those from skilled labourer and farmer families had better survival chances in nineteenth-century Netherlands.[106] These increased mortality risks were attributed to poor living and working conditions, including inadequate housing and nutrition, among lower socioeconomic groups, indicating inequalities in gender roles and resource allocation between boys and girls.[107] In addition to gender roles and discrimination, scarcity may bring biological factors to the forefront, as Devos argued that malnutrition could heighten girls' susceptibility during puberty.[108] Klasen and Wink, however, argued: "scarcity of economic resources is a necessary, but not sufficient condition for a gender bias in mortality."[109] They observed that the poorest groups in societies showed fewer inequalities in mortality between the sexes compared to slightly wealthier, but still poor, groups. This was attributed to fewer cultural constraints, such as gendered division of labour, and greater economic independence among women in the poorest groups, leading to fewer inequalities in gender roles and potentially elevating women's social status.[110]

Economic shocks – Economic shocks can impact children's health outcomes by triggering discriminatory practices or reinforcing biases in gender roles. Beltrán Tapia and Szoltysek argued that in areas with strong son preferences, economic shocks may lead to discriminatory practices because of parents favouring sons over daughters when resources were scarce.[111] They also highlighted the role of political events, such as the Spanish Civil War, in causing economic decline, which correlated with a resurgence of excess female mortality in Spain.[112] Additionally, Willibrord Rutten observed a decline in the health status of conscripts in Limburg that grew up during the economic crisis in the 1880s, as these cohorts were on average one centimetre shorter compared to previous cohorts. Rutten attributed this decline to both resource scarcity and increased child contributions during the crisis, whether through work or household chores.[113] Although Rutten focused on men (as conscripts were exclusively men), the impact on resources and increased contributions likely affected the health outcomes of girls ass well, and potentially even more if sons were prioritized over girls.

Family composition – Scarcity has also been linked to the family composition, including the presence of parents and sibling competition, affecting gender roles and resource allocation within families. Björn

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[105] Kent Johansson, 'Sex-specific Child Mortality During the Swedish Mortality Decline: Why is SES Important for Girls Only, and Food Prices for Boys Only?, *paper presented at the European Population Conference* (Vienna 2010) available on: https://epc2010.eaps.nl/abstracts/100247.

[106] Van Poppel, Schellekens and Walhout, 'Oversterfte van jonge meisjes', 65.

[107] Ibid, 68.

[108] Devos, 'Te jong om te sterven', 40.

[109] Klasen and Wink, 'Missing Women', 282.

[110] Ibid, 282.

[111] Beltrán Tapia and Szoltysek, 'Missing girls', 363.

[112] Ibid, 363.

[113] W.J.M.J. Rutten, 'De levensstandaard in Limburg van de Franse tijd tot aan de eerste wereldoorlog. Een analyse van de lichaamslengte van Limburgse lotelingen', in: O.F.M. Remigius Dieteren and J.C.G.M. Jansen (eds.), *Studies over de sociaal-economische geschiedenis van Limburg XL* (Leeuwarden and Maastricht 1995) 123-160, there 151-152.

Quanjer et al. noted, for instance, higher mortality among girls aged 5 to 12 in the Netherlands after parental loss. They suggested that the reduction in resources caused by parental loss triggered unequal resource allocation that prioritized boys over girls.[114] Other studies focused sibling competition, arguing that the number, birth order and ratio of daughter and sons influenced the resource dilution in the family, which could have a gendered dimension if this allocation depended on the child's sex.[115] Klasen and Wink noted increased mortality risks for later-born girls, especially with elder sisters, indicating a gendered resource dilution.[116] While Van Poppel, Schellekens and Walhout found no relationship between siblings and girls' mortality risks at the national level, Riswick found a relation between girls with sisters and elevated mortality rates in certain Dutch regions, which indicates that the impact of sibling competition could vary across contexts.[117]

3.5.3 Bargaining position

Until the age of 10, parental preferences primarily shape children's expected behaviour and resource distribution. As children grow older, individual bargaining positions become influential, increasing the individual's ability to influence life conditions such as the specific household chores they helped in or the resource distribution within the family.[118] As mentioned before, the bargaining position intertwined with the effects of scarcity. For instance, girls without sisters had a better chance to negotiate for their needs compared to those with many siblings, or economic shocks could have caused parents to prioritize sons over daughters, reducing girls' bargaining positions in families.

Labour opportunities – Numerous studies highlighted the significance of labour opportunities for women's bargaining positions.[119] Johansson noted that the emergence of the wage economy in the nineteenth century led to a relative decline in women's position as they became financially dependent on men, with household work being unpaid and undervalued due to its lack of income generation.[120] This Anglo-Saxon tradition argued that female labour opportunities were crucial for women's health outcomes.[121] In nineteenth-century England, McNay, Humphries and Klasen, for instance, found lower excess female mortality rates when more women participated in agricultural labour.[122] Researchers like Devos, as well as Ginsberg and Swedlund

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[114] Quanjer, Björn, Ingrid K. van Dijk and Mattias Rosenbaum-Feldbrügge, 'Short Lives: The impact of Parental Death on Early-Life Mortality and Height in the Netherlands, 1850-1940', *Demography* 60:1 (2023) 255-279.

[115] Klasen and Wink, 'Missing Women', 281; Beltrán Tapia and Szoltysek, 'Missing girls', 633; Riswick, T., '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* 23:4 (2018) 623-655.

[116] Klasen and Wink, 'Missing women', 281.

[117] Van Poppel, Schellekens and Walhout, 'Oversterfte van jonge meisjes', 65; Riswick, 'Testing the conditional resource-dilution hypothesis', 637.

[118] Riswick, 'Testing the conditional resource-dilution hypothesis'.

[119] Puschmann, 'Discriminatie met dodelijke gevolgen', 158-159; Klasen and Wink, 'Missing Women', 282.

[120] Johansson, 'Sex and death in Victorian England', 165.

[121] Klasen and Wink, 'Missing Women', 282.

[122] K. McNay, J. Humphries and S. Klasen, 'Excess Female Mortality in Nineteenth-Century England and Wales, *Social Science History* 29:4 (2005) 649-681, there 669-673.

therefore argued that the disappearance of excess female mortality resulted from industrialization creating labour opportunities, reducing discrimination as women contributed to the family income.[123] However, other researchers, like Tabutin and Henry, argued that industrialization and female labour opportunities were not the solution but the cause of excess female mortality.[124] Tabutin and Willems also suggested that new working conditions, such as those in the textile industries, increased mortality risks for women, which only declined with improved disease control and protective policies.[125]

Family system – The family system also influenced girls' bargaining positions. Beltrán Tapia and Szoltysek linked strong patriarchal values to patterns of excess female mortality, especially in rural areas. They suggested that patrilineal kinship relations could strengthen son preferences and weaken daughter's bargaining positions, as sons could inherit family assets and were expected to support their parents later in life, while girls typically moved in to their husband's household upon marriage.[126] Puschmann highlighted the role of patriarchal family values in twentieth-century Kosovo, where girls consistently held lower positions than boys, resulting in low bargaining positions of girls and unequal resource allocation.[127] Nineteenth-century cultural changes, such as the shift from instrumental to romantic marriages and the rise of age homogamy, indicate a decline in patriarchal family systems in Western societies, potentially leading to more gender equality and reduced discrimination.[128]

Marriage norms – Lastly, cultural values regarding the marriage system could influence girls' bargaining position. The dowry system, for instance, caused girls to be perceived as a financial burden, reducing their ability to negotiate on the resources they need to survive.[129] Additionally, societal expectations of marriage could have impacted girls' bargaining power, as these norms bounded girls more to expected gender roles in the families and caused girls to be perceived as a 'loss' to the family as she was expected to join her husband's family after marriage.[130]

3.5.4   Policies

The political sphere represents the final group of ultimate determinants that can either increase or decrease gender discrimination and influence gendered roles. One of the most illustrating examples of policies invoking

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[123] Devos, 'Te jong om te sterven', 60; Ginsberg and Swedlund, 'Sex-specific mortality', 415-445.

[124] Henry, 'Men's and Women's Mortality', 196; Dominique Tabutin, 'La surmortalité féminine en Europe avaint 1940', *Population* 33:1 (1978 121-148.

[125] Tabutin and Willems, 'Differential Mortality', 17-52.

[126] Beltrán Tapia and Szoltysek, 'Missing girls', 638-639 and 641.

[127] Puschmann, 'Discriminatie met dodelijke gevolgen', 158-159.

[128] Van de Putte, Bart, Frans van Poppel, Sofie Vanassche, Maria Sanchez, Svetlana Jidkova, Mieke Eeckhaut, Michel Oris, Koen Matthijs and Jay Teachman, 'The Rise of Age Homogamy in 19th Century Western Europe', *Journal of Marriage and Family* 5:71 (2009) 1234-1253.

[129] Fransisco J. Beltrán Tapia and Michail Raftakis, 'Sex ratios and gender discrimination in Modern Greece', *Population Studies* (2021) 1-17, there 16; Beltrán Tapia and Szoltysek, 'Missing girls', 640.

[130] Ibid, 639.

excess female mortality was China's one-child policy, which led to extreme son preferences in families.[131] Certain nineteenth-century policies, however, such as the 1874 child employment prohibition and the 1901 compulsory education act in the Netherlands, were associated with improved positions of girls and women and reduced gender biases and discrimination.[132] Nevertheless, Ubachs emphasized that this basic education level was insufficient for girls' emancipation, as the majority of girls ended up working in the household after all.[133] Some policies indirectly targeted other proximate determinants, such as, for example, compulsory education contributing to increased healthcare equality. Since 1823, Dutch children were required to show proof of smallpox vaccination (*'pokkenbriefje'*) to attend school.[134] The act on compulsory education for both boys and girls might have therefore decreased any inequalities in vaccination rates, at least for smallpox.

3.6 Conclusion

In short, this chapter explored theories and determinants related to the debate on excess female mortality in nineteenth- and early twentieth-century Europe. It distinguished between proximate determinants, which directly influenced health outcomes, and ultimate determinants, which operated through these proximate factors.

Initially, research focused on biological factors that might have increased women's susceptibility to infectious diseases. However, later research reached a consensus that while genetic factors play an important role in mortality, a broader set of variables, shaped by human behaviour, contributed to the patterns of excess female mortality. Gender roles, such as differences in household tasks and working conditions, along with behavioural differences between boys and girls were proposed as explanatory factors. However, some argued that these gender roles could not explain excess female mortality among young children, as these gender patterns played minor roles in behaviour of very young children. Others emphasized therefore the differences in treatment between boys and girls that contributed to disparities in mortality rates. They suggested that the perceived lower value of girls led to gender-specific practices affecting their resistance to infectious diseases. However, it should be emphasized that these three groups of proximate determinants do not exclude each other, as the three mechanisms could simultaneously affect health outcomes of children.

The impact of biological factors, gender roles, and gender-specific discrimination on health outcomes was influenced by ultimate determinants such as social position, scarcity, bargaining position and policies, which could either worsen or improve certain roles and perceptions of women. The interplay between these

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[131] Klasen and Wink, 'Missing Women', 284.
[132] Ibid, 282-283; Maenen, *Petrus Regout*, 257.
[133] Ubachs, *Tweeduizend jaar Maastricht*, 155.
[134] Willibrord Rutten, *'De vreselijkste aller harpijen'. Pokkenepidemieën en pokkenbestrijding in Nederland in de achttiende en negentiende eeuw. Een sociaal-historische en historisch-demografische studie.* Doctoral Thesis (Wageningen 1997) 266.

ultimate and proximate determinants is complex, as the ultimate determinants could even intersect with each other, while simultaneously affecting different proximate determinants. The conceptual framework presented in this study does thus not fully capture the complex reality of determinants that affected health outcomes, yet it did provide a more schematic overview of different explanations that have been put forward in the debate on excess female mortality.

After discussing the used data and methodology, this study will first map the Maastricht pattern of excess mortality and identify related causes-of-death in Chapter 1 and 2. Chapter 3 aims to identify any biological factors, gender roles, or gender-specific discrimination associated with the Maastricht pattern, utilizing numerous determinants discussed above as indicators.

4. Data and methodology

This research uses data from the Maastricht Death and Disease Database, compiled by the Centre for Social History of Limburg (*'Sociaal Historisch Centrum Limburg'*). The database comprises burial records of 76,264 individuals that passed away in the municipality of Maastricht between 1864 and 1955. This chapter will first briefly discuss the history of cause-of-death registration in the Netherlands and the challenges that this type of data brings for historical research. Next, it will discuss the Maastricht Death and Disease Database, the construction of used variables and lastly the used methodology in this research.

4.1 Cause-of-death registration in the Netherlands

In 1811, death registration became mandatory in The Netherlands, but it took until 1865 before causes of death were registered. These causes of death were registered to create more insights in mortality patterns, contributing to the development of better-targeted policies aimed at improving the nation's poor health conditions. Initially, the focus of cause-of-death registration was on identifying the spread of infectious and contagious diseases. The classification listed six main diseases: smallpox, scarlet fever, measles, typhus, diphtheria, and cholera, with all other causes of death grouped into a final category.[135] In 1874, the registration process became more precise with the introduction of a new system that categorized 55 standardized causes of death into 35 categories. To ensure the reliability of this registration, the 1865 medical law mandated that doctors issued a medical certificate stating the cause of death. From 1869 onward, the Burial Act required the attendance of a physician after someone's death, leading to an increase in the number of deaths with a medical certificate issued by a professional.[136]

Although the registration was standardized, it was not flawless. For instance, in 1897, concerns were raised about the reporting of stillbirths, as there was no clear guideline on when a foetus should be recorded as a child, leading to potential subjective interpretations. Additionally, some noted that the standardized causes of death had not been updated since 1864, despite advancements in medical knowledge. Furthermore, there were instances where doctors, aiming to protect family reputations, disguised causes related to alcoholism or suicide using another category of the 55 standardized causes of death.[137]

This cause-of-death registration presents two problems for historical research. Firstly, reports often aggregated the data, grouping individual causes of death into larger categories based on age, causes or social

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[135] F. van Poppel and J.P. van Dijk, 'The development of cause-of-death registration in the Netherlands, 1865-1955', *Continuity and Change* 12:2 (1997) 265-287, there 270.
[136] Ibid, 270-272.
[137] Ibid, 273.

classes. This aggregated data cannot be used to study someone's death in relation to their individual socioeconomic and demographic characteristics. Secondly, historical classifications sometimes grouped together causes of death that were not aetiologically linked. For example, diseases like syphilis, rickets, and scrofula were combined into one of the 34 categories, despite their varying infectious and non-infectious natures. As a result, these categories do not allow for the study of underlying causes, such as the route of transmission, that contributed to someone's death.

4.2 Maastricht Death and Disease Database

This study uses the data of the Maastricht Death and Disease Database, compiled by the Centre for the Social History of Limburg ('*Sociaal Historisch Centrum Limburg*'). The data comprises burial notes from the municipal cemetery and death certificates of Maastricht between 1864 and 1955. The cause-of-death administration in the Maastricht burial records provides a unique source, as it differed on two aspects from the cause-of-death registrations of the municipal reports.[138]

Firstly, the dataset from Maastricht contains individual-level data, unlike the aggregated data used in municipality reports. Along with the cause of death, the burial records included detailed socioeconomic and demographic information about the deceased, such as name, gender, age at death, address, profession, parent's names and professions, religion, and place of residence. This comprehensive data enables the study of an individual's death in relation to their specific characteristics, such as socioeconomic class, offering insights into inequalities in mortality patterns.[139]

Secondly, the Maastricht burial records included individual causes of death assigned by a physician. Contrary to the municipal death reports, the Maastricht burial notes did not standardize causes of death into 55 categories, resulting in broader spectrum of recorded causes in the Maastricht data. Additionally, since this data was not grouped into larger categories, the issue of grouping cases that were not aetiologically linked did not arise in the Maastricht data. The Maastricht data can therefore provide insights into underlying causes that could have contributed to someone's cause of death.[140]

4.3 Data sample

This study examines mortality disparities among children aged 0 to 20 years. While Murkens found no excess female mortality among adults (20 years and older), Van Poppel, Schellekens, and Walhout, as well as Janssens

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[138] Murkens, *Unequal pathways to the grave?*, 36.
[139] Ibid, 37.
[140] Ibid, 38.

observed patterns of excess female mortality among children below 20 years in the region of Maastricht.[141] This study will therefore delve into differences in health outcomes among children between 0 and 20 years old in Maastricht to create an in-depth understanding of causes of excess female mortality patterns.

The researched period covers the years from 1864 to 1930. Tabutin and Willems argued in 1996 that excess female mortality patterns disappeared in the Netherlands by 1924, suggesting this study's timeframe may capture this trend and its disappearance in Maastricht.[142] Additionally, population estimates for each year were based on population counts that were held every ten years until 1930. The next following count was held 17 years later, in 1947, which is a much longer time gap and included the Second War Period. Using 1930 as the end of the research period was therefore considered to result in more reliable population estimates.

The sample used in this study includes all children that died between 0 and 20 years in Maastricht between 1864 and 1930, which comprises 33.381 individuals.

4.4 Variables

To study the mortality patterns in Maastricht, numerous variables have been created using data from the Maastricht Death and Disease Database, which will be discussed in this section.[143]

Age categories – This study distinguished five different age groups among children between 0 and 20 years, as mortality patterns and patterns of excess female mortality differed across ages. These categories of ages at death were as followed: infants (<1), young children (1-5), children (6-10), teenagers (11-15), and adolescents (16-20).

Cause of death – The causes of death of children between 1 and 15[144] years were analysed, involving 9477 individuals and encompassing 1,835 unique causes of death. To standardize these causes, the ICD10H coding scheme was utilised. This scheme, a historical adaptation of the *International Statistical Classification of Diseases and Related Health Problems* (ICD-10), was developed by the SHiP research network focussing on mortality dynamics in European port cities during the nineteenth and twentieth centuries.[145] ICD10H-codes accommodate historical variations in disease terminology, such as typhoid fever being referred to as bilious fever, colonial fever, or fog fever in historical Europe. This scheme enables the coding of various terminologies into broader disease categories, while preserving the historical terminology. Therefore, it remains possible to track any historical developments in terminologies of diseases.[146] Some diseases in this dataset were coded

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[141] Janssens, *Sekse, Gender en de Dood,* 13; Van Poppel, Schellekens, and Walhout, 'Oversterfte van jonge meisjes', 44

[142] Dominique Tabutin and Michel Willems, 'La surmortalité des jeunes filles jusqu'en 1940', in: *Santé et mortalité des enfants en Europe* (Louvain-la-Neuve 1996) 96.

[143] See Annex II for an overview.

[144] The results in Chapter 5 showed that excess female mortality occurred in Maastricht among children aged 1-15 years, which became the focus group of the rest of this research.

[145] Angélique Janssens, 'Constructing SHiP and an International Historical Coding System for Causes of Death, *Historical Life Course Studies* 10:3 (2021) 64-70, there 65.

[146] Ibid, 68-69.

differently, due to variations in Dutch terminology compared to the English definitions in the ICD10H-codes. For example, in the Netherlands, angina was used to denote inflammation of the throat in younger ages, while in English, it refers to angina pectoris, a heart condition. Similarly, nervous fever was coded as typhus in the ICD10H-scheme, whereas in the Dutch context, it referred to typhoid fever.[147]

It is important to note that diseases affected age categories differently. Young children were predominantly affected by airborne infectious diseases, such as measles, diphtheria, and scarlet fever, while adults mostly suffered from other infectious disease types such as tuberculosis.[148] Ideally, causes-of-death would be classified differently across age groups. However, for the purpose of enabling comparisons between age groups, this study employed the same classification system for all age categories.

While the burial records also sometimes recorded a second or third cause of death, this study has examined only one cause of death per person to create a better overview. Numerous second or third causes of death were often related to the first, like diarrhoea and gastroenteritis. However, if multiple unrelated causes of death were recorded, the cause of death most likely to result in death was chosen. If no distinction could be made, the order in which the causes were recorded was used, with the first cause of death being chosen as the cause of death of that person.[149]

Murken's classification system has been utilised to categorize causes of death into seven categories: (1) *food- and waterborne infectious diseases* were transmitted through food or water, including amongst others cholera, typhus, typhoid fever, acute diseases of the digestive system and tuberculosis. (2) *Airborne infectious diseases*, that spread via air or close human contact, with diseases like measles, whooping cough, smallpox, scarlet fever, pulmonary tuberculosis, diphtheria or acute diseases of the respiratory system. (3) *Other infectious diseases*, could be transmitted via a mix of the previous two categories, such as endocarditis, nephritis, and syphilis. The (4) *non-infectious diseases* included all other types of diseases or conditions, such as appendicitis, diabetes, cardiac arrest, abscesses, and tumours. Causes related accidents and violence, like fractures, burning wounds, and drownings, were grouped into (5) *non-natural causes of death*. (6) Ill-defined causes of death, included causes like debility, marasmus, or vague descriptions. The last category, (7) *unknown causes of death,* contains all missing causes of death. Since Murkens only coded causes-of-death for children aged 0-4, the causes-of-death for children 5-15 still needed classification. Murkens' categorization served as a guideline, and in remaining cases, the guidelines of Wolleswinkel-Van den Bosch were used to classify causes-of-death into the aforementioned categories.[150]

This categorization was the most adept for studying excess female mortality because it distinguished between infectious diseases and other causes-of-death. This distinction is valuable for determining if infectious diseases were indeed more deadly to girls, as previous studies suggested. Furthermore, Murkens' categorization categorized these infectious diseases into different modes of transmission: (1) food- and

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[147] Murkens, *Unequal pathways to the grave?*, 39.
[148] Murkens 45.
[149] Ibid, 42.
[150] Ibid, 45; Judith Wolleswinkel-van den Bosch, *The epidemiolgocial transition in the Netherlands,* Doctoral Thesis (Rotterdam 1998) 287-292.

waterborne, (2) airborne, and (3) other. Therefore, this classification not only enables the study of causal groups related to excess female mortality, but also allows for an investigation into any different routes of transmission that might have been at play.

Socioeconomic status – The socioeconomic status of children aged 1 to 15 years was determined based on their parent's profession, as children's social status was largely influenced by their parent's socioeconomic status. The parent's professions were coded using the HISCO-codes and classified according to the HISCLASS-12 scheme.[151] In this study, a collapsed version of these 12 socioeconomic classes is used. Table 1 presents an overview of the HISCLASS categories that were merged.

Table 1 Overview of constructed socioeconomic classes.

	HISCLASS-12		**Collapsed scheme**
1	Higher manager	**1+2+3**	Managers and professionals
2	Higher professionals		
3	Lower managers		
4	Lower professionals, and clerical and sales personnel	**4+5+6**	Clericals, sales personnel and foremen
5	Lower clerical and sales personnel		
6	Foremen		
7	Medium skilled workers	**7+9**	Medium and lower skilled workers
9	Lower skilled worker		
8	Farmers and fishermen	**8+10+12**	Lower and unskilled farm workers
10	Lower skilled farm workers		
12	Unskilled farm workers		
11	Unskilled workers	**11+13**	Unskilled unspecified workers
13	Unskilled workers not specified		
-1	Unknown	**-1**	Unknown

Source: Marco H.D. van Leeuwen, Ineke Maas and Andrew Miles, HISCO: Historical International Standard Classification of Occupations (Leuven 2002); Marco H.D. van Leeuwen and Ineke Maas, HISCLASS. A historical international social class scheme (Leuven 2011).

Occupational exposure to infectious diseases – This research uses the index developed by Schalk et al. to investigate the relationship between occupational characteristics and the risk of contracting infectious diseases. This index indicated per HISCO-unit (e.g. 'General managers' or 'Civil Engineers') if the type of work was conducted indoors and if it involved close contact.[152] Based on this classification, four types of occupational characteristics were distinguished: (1) *none*, for occupations not indoors and without close-contact, (2) *indoors,* for indoor professions without close-contact, (3) *contact,* for professions involving close-contact but not indoors, and (4) *both,* for professions that were indoors and involved close-contact. The fifth

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[151] Marco H.D. van Leeuwen, Ineke Maas and Andrew Miles, *HISCO: Historical International Standard Classification of Occupations* (Leuven 2002); Marco H.D. van Leeuwen and Ineke Maas, *HISCLASS. A historical international social class scheme* (Leuven 2011).

[152] Ruben Schalk, Richard Zijdeman, Auke Rijpma, Ingrid van Dijk and Rick Mourits, 'HSNDB index for exposure to infectious disease', IISH Data Collection, V1, accessible on: https://hdl.handle.net/10622/TFG1DQ.

category, *unknown,* included professions with unknown characteristics and parents without a recorded profession.

Family composition – The burial records of Maastricht provided insights into the presence or potential absence of parents, allowing for a robust classification of the family composition of children who passed away between 1 and 15 years old. If both parents were alive and known, the records included their names and professions. If a parent was present but unemployed, 'unknown' was recorded as the profession. If no name and/or profession was recorded or 'deceased' was registered as the profession, the parent was either absent or had passed away. Four types of families were distinguished: (1) a family with both parents alive, (2) with a deceased or absent father but alive mother, (3) a family with the father alive but the mother absent or deceased and lastly, (4) a family with both parents absent or deceased.

4.5 Analytical approach

To study mortality differences between boys and girls, the mortality rates will be compared by computing the male-to-female mortality ratios. These mortality rates are based on the annual male and female age-specific populations in Maastricht and the total number of male and female deaths per year.

In this study, the yearly population at risk of males and females was constructed for numerous years. During the nineteenth and beginning of the twentieth centuries, population censuses were organized in the Netherlands, which registered for larger cities, including Maastricht, the total number of boys and girls per age that were living in that city at the first of December. These censuses were conducted with ten-year intervals in the periods 1859-1930, except for 1909-1920, in which eleven years were in between censuses.[153] To estimate populations for the years between census, proportions per year, age and sex were calculated based on the differences between each census (t=0) and the next (t+1). These proportions were then applied to each intermediate year, assuming a linear population development. While this method provided age-specific populations at risk for each year, it should be stressed that the linear assumption could have caused over- or underestimation of the real population in certain years.

Estimating the population between 1910 and 1920 presented challenges due to Maastricht's annexation of neighbouring municipalities in 1920. Using the difference between 1920 and 1909 to calculate the proportional factor would have resulted in an overestimation of the population. Following Murkens' approach, the proportion each age group represented in the total population in 1909 has been calculated. Then, using this 1909 share, the population for each group has been computed based on the total population count in 1919, as

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[153] Uitkomsten der … tienjarige volkstelling in het Koninkrijk der Nederlanden op den eersten December … (1859-1930) Centraal Bureau voor de Statistiek. www.volkstellingen.nl; Volkstelling 31 december … (1920-1930), Centraal Bureau voor de Statistiek, www.volkstelling.nl

reported in municipal records. The estimates between 1909 and 1919 were constructed similarly to other years, by calculating the proportion per year and adding it to the population counts.

The mortality rates of boys and girls were computed based on the total number of deaths and the population at risk. Often, male-to-female mortality ratios are calculated as followed: (male mortality rate / female mortality rate)*100. Ratios above 100 indicate excess male mortality, while ratios below 100 indicate excess female mortality. However, this method can result in distortions in the intensity of excess male and female mortality. Dividing the male mortality by the female mortality can theoretically yield endlessly positive ratios, but never negative ratios. This means that excess male mortality ratios can go up from 100 to infinity, while excess female mortality can only range between 0 and 100. This could lead to an overestimation of the intensity of excess male mortality and an underestimation of the intensity of excess female mortality.

To address this issue, the mortality ratios in this study were based on an index, using the total mortality rate per year. This total mortality rate was calculated based on the combined number of female and male deaths per year and the population at risk. The male-to-female mortality ratio was then computed as followed: (male mortality rate / total mortality rate)*100.

The male-to-female mortality ratios were calculated for various groups to examine, for example, whether girls or boys died more often in certain age categories, causal groups of death or socioeconomic classes. For each group, the mortality rates of boys and girls were computed, followed by the calculation of the male-to-female mortality ratios. Ratios above 100 indicate excess male mortality, meaning that more boys than girls died in that specific group. Ratios below 100 indicate excess female mortality, meaning that more girls than boys died in that category.

5. Unequal mortality in Maastricht: Mortality differences between boys and girls aged 0-20 years, 1864-1930.

5.1 Introduction

This chapter will examine disparities in mortality between boys and girls in Maastricht at the end of the nineteenth and beginning of the twentieth century. The chapter will examine five different age groups among children between 0 and 20 years old in order to explore possible changes in the male-to-female mortality ratios across age groups. It seeks to answer the following questions: *What patterns of excess female mortality can be discerned in Maastricht across time? Did male-to-female mortality ratios change across age groups? And what factors could have contributed to these patterns?*

The chapter will first explore quinquennial male-to-female mortality ratios, in order to explore in which age groups excess female mortality in Maastricht occurred. Next, the separate age groups will be discussed more in depth to investigate why among certain ages excess female mortality was absent or present and to identify some first potential explanations to the pattern of male-to-female mortality rates in Maastricht.

5.2 Maastricht patterns of excess female mortality

In 1996, Tabutin and Willems calculated differences in mortality rates of boys and girls in nineteenth-century Netherlands and other European countries. They observed excess female mortality among Dutch children aged 5 to 15, peaking between 1888 and 1893, and concluded that this mortality pattern disappeared in the Netherlands by 1925.[154] In 2009, Van Poppel, Schellekens and Walhout conducted a regional analysis of sex differences in mortality, while examining separate age categories. They found excess mortality among girls aged 10-15 during the sample years 1850-1859 and 1901-1902 in the Netherlands. Additionally, they observed to a lesser extent excess mortality among girls aged 5-10 and 15-20, and exclusively in the province of Limburg among girls aged 1-5 during the period of 1901-1902.[155] These regional variations showed that patterns of excess female mortality not only shifted in different spatial contexts, but also across different age categories.

As explained in the data section, this study distinguished five age categories of children between 0-20 years, namely infants (<1), young children (1-5), children (6-10), teenagers (11-15), and adolescents (16-20). In order to create an overview of differences in mortality rates between boys and girls across time and the

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[154]Tabutin and Willems, 'Differential Mortality', 96.
[155] Van Poppel, Schellekens, and Walhout, 'Oversterfte van jonge meisjes', 46-47 and 64-65.

different age groups, the quinquennial male to female mortality ratios have been computed per age category, as shown in Figure 3.

Figure 3 Quinquennial male-to-female mortality ratios per age category, children aged 0-20, Maastricht 1864-1930.
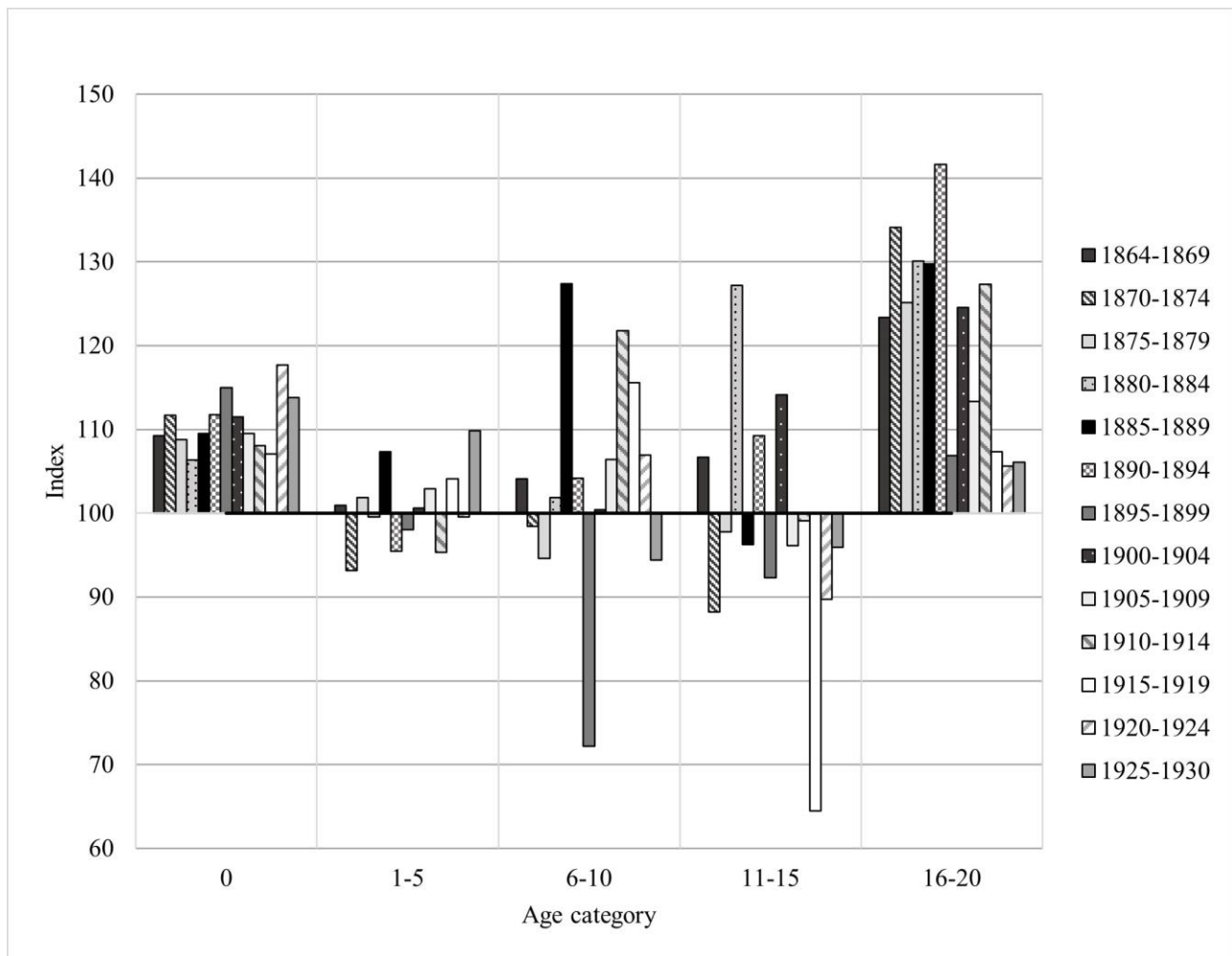
Ratios above 100 indicate excess male mortality, ratios below 100 indicate excess female mortality.
Source: Maastricht Death and Disease Database; Centraal Bureau voor de Statistiek, Volkstelling 1859-1930.

The ratios in Figure 3 indicate that in Maastricht, excess female mortality was observed among children aged 1 to 15 years, while infants and adolescents consistently exhibited higher male mortality rates throughout the researched period. The ratios for children aged 1-5 displayed some fluctuations, with occasional peaks of excess female or male mortality, although these peaks were relatively small. The differences in ratios were more pronounced in the older two age categories, with notable peaks of excess female mortality occurring in 1895-1899 among children aged 6-10 and among children aged 11-15 in the years 1915-1919.

The results somewhat align with earlier observations on the national Dutch excess female mortality pattern. However, there are differences in timing and intensity. While Tabutin and Willems suggested that the pattern disappeared in the Netherlands by 1925, Figure 3 shows that excess female mortality persisted in Maastricht until at least 1930. Additionally, the timing of maximum intensity in Maastricht differs from the national findings of Tabutin and Willems, as peaks spread across various years in different age categories

rather than a clear peak in 1888-1893.[156] These differences could be attributed to differences in the observed regions; while Tabutin and Willems studied the entire Netherlands, this study focuses solely on the municipality of Maastricht. This suggests that although excess female mortality disappeared nationally, the pattern persisted in specific communities in the Netherlands.

The Maastricht patterns of excess female mortality across different age categories also somewhat align with earlier findings. Van Poppel, Schellekens and Walhout observed excess female mortality among children aged 1-5 in Limburg in 1901-1902, while excess male mortality was noted in the preceding and subsequent sample years of 1850-1859 and 1928-1930. However, Figure 3 reveals that children from these ages in Maastricht frequently exhibited higher mortality rates among girls compared to boys. Girls in Limburg aged 5-10 and 10-15 also showed excessive mortality rates in 1850-1859 and 1901-1902, but this shifted to excess male mortality in 1928-1930 at the provincial level. In contrast, both age categories continued to exhibit frequent rates of excess female mortality until the end of the research period in 1930. Finally, while Van Poppel, Schellekens and Walhout observed patterns of excess female mortality among adolescents aged 15-20 in Limburg in 1850-1859 and 1928-1930, in Maastricht boys in this age category had consistently higher mortality rates compared to girls in 1864-1930.[157] The disparities in results between Maastricht and the province of Limburg may stem from the distinct urban context of Maastricht compared to the predominantly rural setting of the rest of the province. However, it is also possible that the use of sample years, with significant gaps spanning numerous decades, might have obscured a more consistent and pronounced pattern of excess female mortality in Limburg.

To gain a more detailed understanding of differences in mortality rates between boys and girls aged 0-20 years, the separate age categories will be examined in depth in the following sections.

5.3 Infants

Infants in Maastricht consistently exhibited excess male mortality ratios. Previous research suggested that male infants are inherently weaker and more susceptible to disease and premature death compared to females. However, Beltrán Tapia and Szoltysek highlighted that in societies with high infant mortality rates, practices of female infanticide could be easily overlooked, as distinguishing between natural deaths and infanticide poses challenges. They proposed examining rates of stillbirths and sex ratios at birth, as parents might disguise female infanticide or mortal neglect as natural (premature) deaths, resulting in distorted mortality rates among infants.[158] If parents reported significantly more stillborn girls compared to boys, it could indicate discriminatory practices.[159] Similarly, a pronounced male surplus in sex ratios at birth might suggest suspicious

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[156] Tabutin and Willems, 'Differential Mortality', 96.
[157] Van Poppel, Schellekens, and Walhout, 'Oversterfte van jonge meisjes', 46-47.
[158] Van Poppel, 'De statistieke ontleding van de dooden', 19; Murkens, *Unequal pathways to the grave*?, 90.
[159] Beltrán Tapia and Szoltysek, 'Missing girls', 621-622.

practices causing fewer girls to be registered as born. Analysing historical sex ratios at birth comes with challenges due to the absence of a benchmark of 'normal' and 'abnormal' ratios, but ratios exceeding 110 are often considered unnatural.[160] The analysis of stillbirth ratios and sex ratios at birth both however suffer from registration issues, as it remains unclear what marked the distinction between a stillbirth and live birth and if miscarriages were included in the registration. Despite these challenges, both methods can provide an initial indication of any suspicious practices not observed from the examination of infant death rates.

In Maastricht, the sex of stillbirths was recorded in burial records between 1864 and 1890. After this period, stillbirths continued to be registered but without information on the sex of the child. Additionally, no names were recorded, making it impossible to derive the sex from the child's name. The annual reports of the municipality (*'Verslagen van den Toestand der Gemeente Maastricht'*) did include the number of births of boys and girls throughout the studied period. However, it remains unclear whether this registration only included live births or also included stillbirths or children that died shortly after birth. Figure 4 displays the male-to-female ratios of stillbirths reported in the burial records of Maastricht. The male and female death rates of stillbirths were calculated based on the total number of male and female births registered in the municipal records of the respective year.

Figure 4 Annual male-to-female stillbirth ratios, Maastricht 1864-1890.
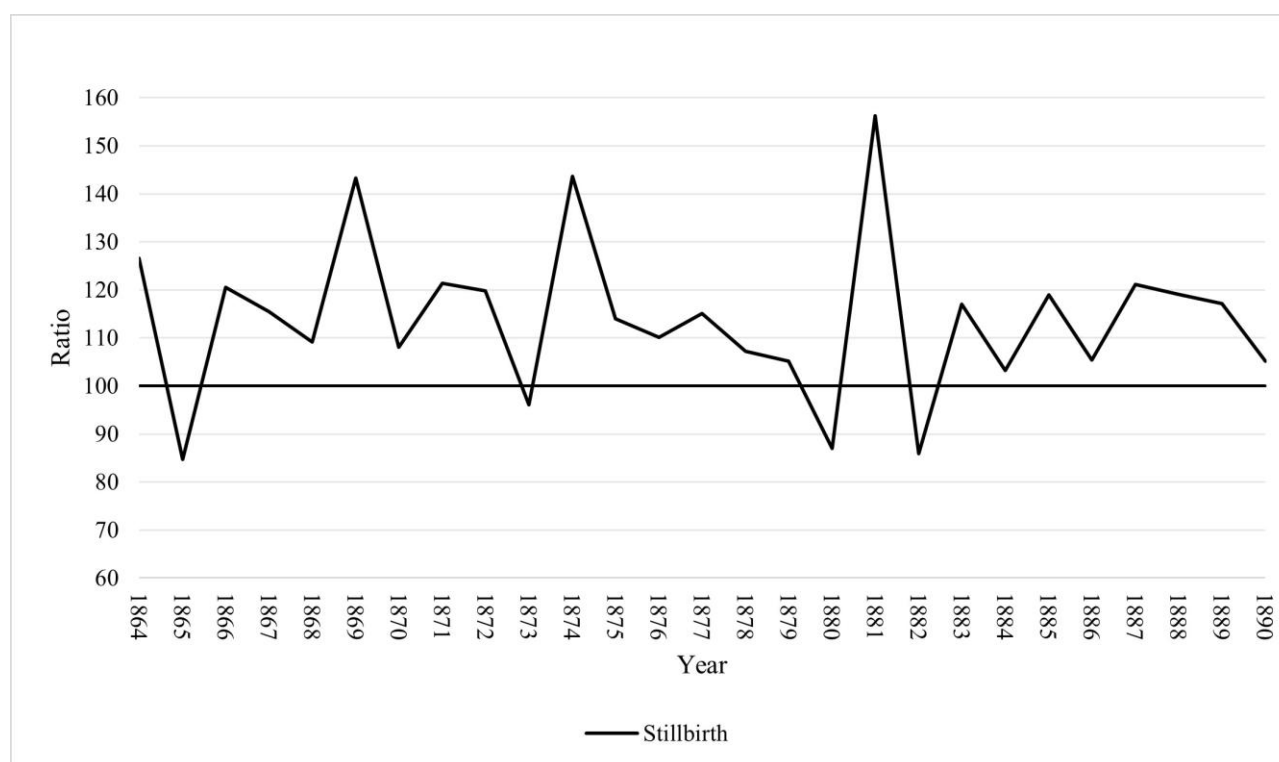
Ratios above 100 indicate excess male mortality, ratios below 100 indicate excess female mortality.
Source: Maastricht Death and Disease Database; Verslag van den toestand der gemeente Maastricht over het jaar 1864-1890.

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[160] Today's societies contain ratios of approximately 105/106 boys to every 100 girls born. In the past, however, infant mortality rates were higher and given the fact that male infants were more prone to die in utero or premature, historical sex ratios at birth are expected to be lower. Often, ratios above 110 are taken as a benchmark, as ratios that exceed this number indicate pronounced differences in numbers of boys and girls reported as born. Beltrán Tapia and Szoltysek, 'Missing girls', 624.

Figure 4 illustrated that in the majority of years, more male than female stillbirths were reported in Maastricht. Given the higher male susceptibility to premature deaths, the reported stillbirths do not suggest consistent suspicious sex rates. While some years did show larger shares of female stillbirths compared to males (e.g., in 1865, 1873, 1880 and 1882), these instances could be due to relatively small numbers reported per year. For example, in 1865, 23 female stillbirths were reported compared to 18 males, and in 1880, these numbers were 29 compared to 23. The rates of stillbirths in Maastricht indicate no consistent practices of female infanticide or mortal neglect of new-born girls.

To provide a more comprehensive view, sex ratios at birth in Maastricht were also calculated, spanning a longer period and involving larger numbers (exceeding at least 450 births annually), which mitigates the issue of fluctuations due to small numbers. As mentioned before, a male surplus at birth is expected. However, pronounced male surpluses in sex ratios at birth could suggest suspicious practices that might have reduced the number of girls being registered as born. These ratios were computed by dividing the total number of male births by the total number of female births each year and are show in Figure 5.

Figure 5 Annual sex ratios at birth, Maastricht 1864-1930.
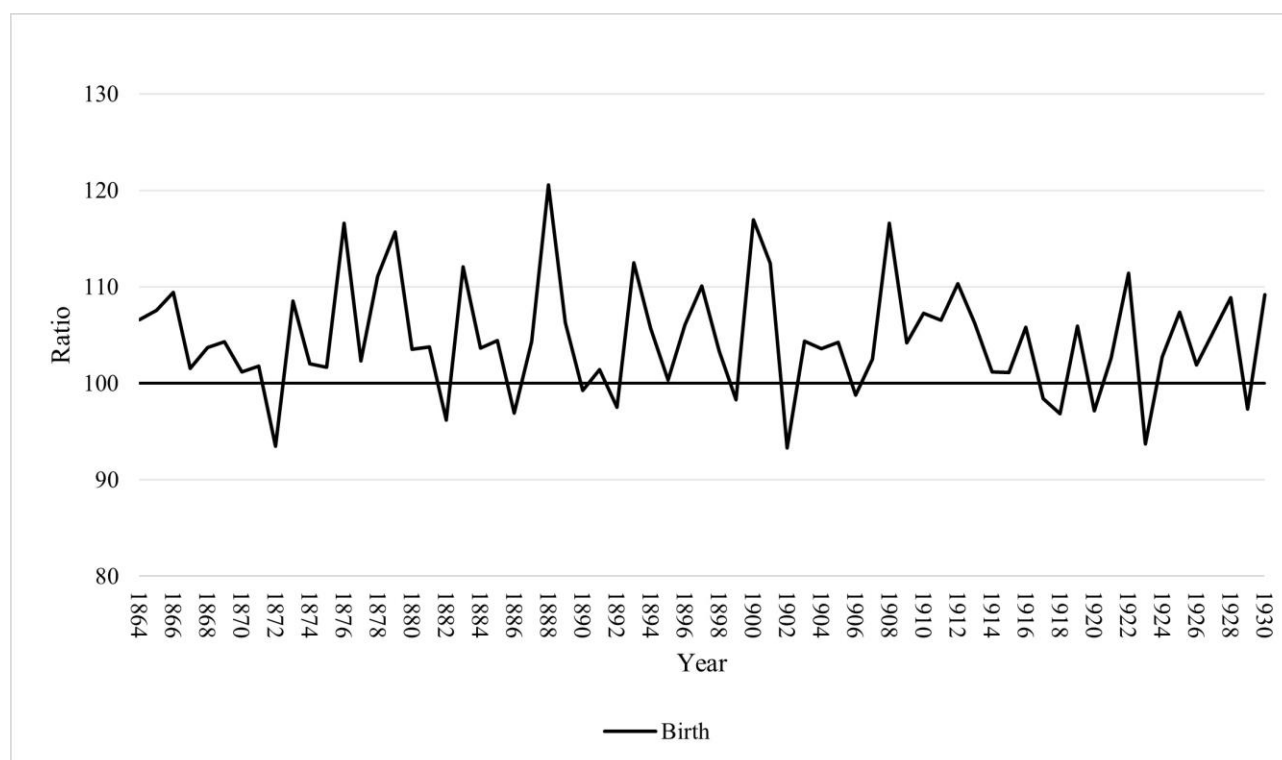
Ratios above 100 indicate a male surplus, ratios below 100 a female surplus.
Source: Verslag van den toestand der gemeente Maastricht, 1864-1930.

In Maastricht, an average sex ratio at birth of 104 with a standard deviation of 5.8 was found. Figure 5 displays some annual fluctuations, notably in 1876, 1879, 1888, 1900, and 1907, where birth ratios exceeded the benchmark of 110. These skewed sex ratios at birth could suggest parental intervention in offspring composition, potentially manifested through female infanticide or neglect of premature girls. However, Figure 5 also shows years with a female surplus, which contradicts the notion of parents favouring sons though infanticide. Beltrán Tapia and Szoltysek concluded that in Western Europe, excess female mortality was not

caused by infanticide, but rather by unequal treatment of boys and girls at later ages.[161] Although inconclusive for Maastricht, the results suggest that if infanticide existed, it was not consistent over the studied period. This study will focus on mortality differences among children aged 1 to 15 because of the pronounced mortality differences in these groups. Future research could, however, delve deeper into the suspicious infant mortality and birth patterns, by comparing it, for instance to a city not affected by excess female mortality, like Amsterdam or by researching the sex ratios at birth and infant death rates over longer periods of time, using parish records or other vital registration.

5.4 Young children (1-5 years)

Tabutin and Willems observed that in historical Europe, excess female mortality emerged around ages 3-4.[162] They noted variations among young children aged 1-5 across different places and periods, with some areas or times exhibiting patterns of excess female mortality, while others showed constant excess male mortality.[163] McNay, Humphries and Klasen concluded that in nineteenth-century England and Wales, excess female mortality did not occur among these ages, attributing it to the inherent survival advantage of women in childhood and infancy.[164] Similarly, Perner et al. found excess male mortality among children of this age group in nineteenth-century Denmark, while Van Poppel, Schellekens and Walhout found in few regions excess female mortality in the Netherlands between 1827 and 1930.[165]

Figure 3 illustrated clear patterns of excess female mortality in Maastricht between 1864 and 1930. To examine differences in mortality rates between boys and girls aged 1 to 5 years, Figure 6 displays annual male-to-female mortality ratios (left axis). The figure also includes the death rates of boys and girls per year (right axis) to assess any relationship between excess female mortality and years with high or low mortality rates. Finally, the disease outbreaks Murkens identified in Maastricht between 1864 and 1955 have been included in the graph.

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[161] Beltrán Tapia and Szoltysek, 'Missing girls', 623.
[162] Tabutin and Willems, 'Differential Mortality', 17.
[163] Ibid, 23.
[164] McNay, Humphries, and Klasen, 'Excess Female Mortaltiy', 654.
[165] Perner et al., 'Gendered Mortality of Children and Adolescents', 69; Van Poppel, Schellekens, and Walhout, 'Oversterfte van jonge meisjes', 46.

Figure 6 Annual male-to-female mortality ratios and sex-specific mortality rates, children 1-5 years old, Maastricht 1864-1930.
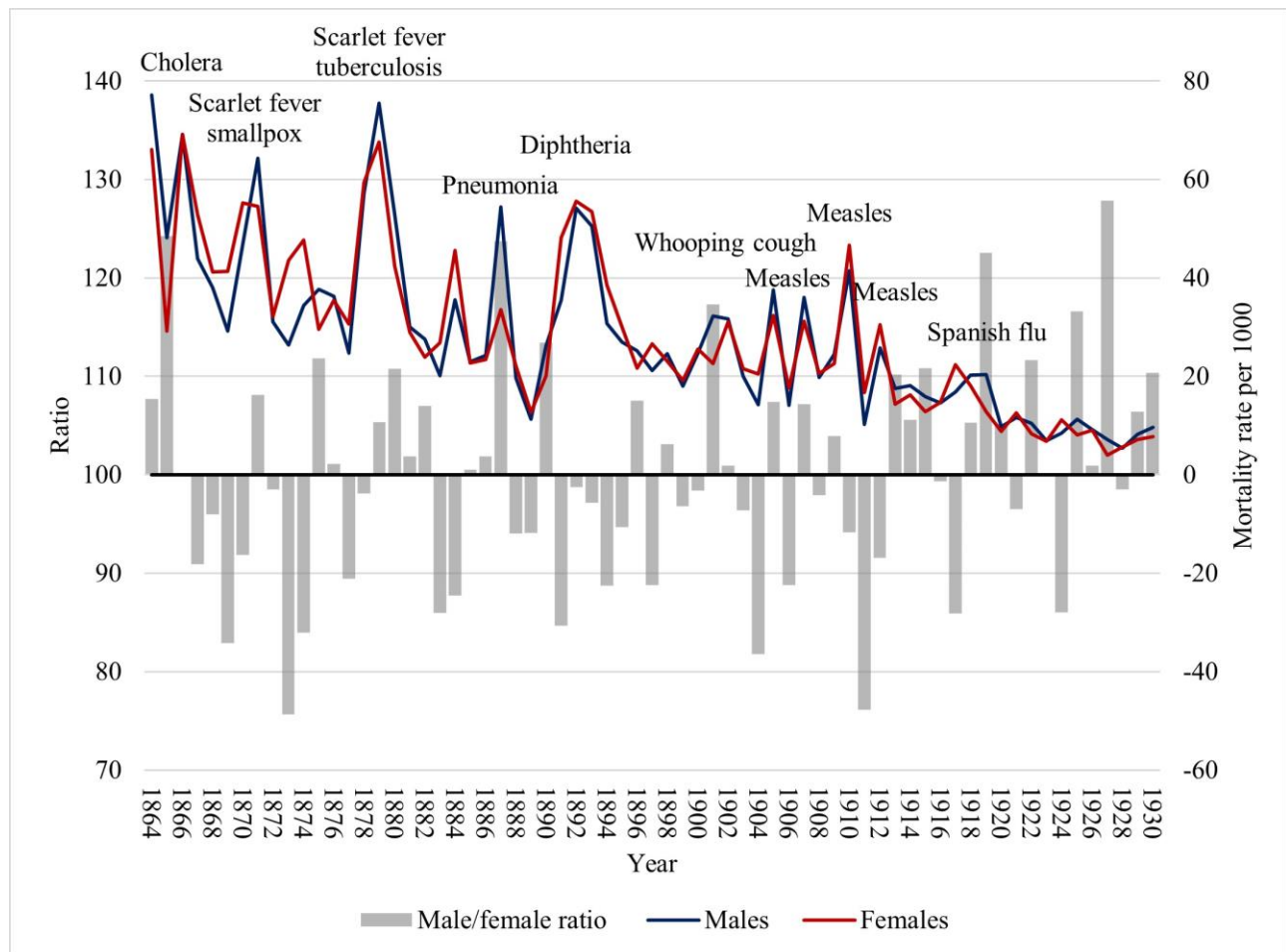
Ratios above 100 indicate excess male mortality, ratios below 100 indicate excess female mortality.
Source: Maastricht Death and Disease Database; Centraal Bureau voor de Statistiek, Volkstelling 1859-1930; Murkens, 'Unequal pathways to the grave', 109 and 223.

The results in Figure 6 reveal frequent patterns of excess female mortality in Maastricht, with notable clusters such as in 1867-1874 and again in 1888-1897. The latter period corresponds to the period Tabutin and Willems identified as the period of maximum intensity of excess female mortality in the Netherlands.[166] From 1912 onwards, the mortality disadvantage of girls appears to gradually diminish, with more consistent ratios of excess male mortality emerging. However, until the end of the researched period, young girls continued to exhibit higher mortality rates in some years compared to boys of the same ages.

The mortality rates of boys and girls suggest that around the turn of the twentieth century, mortality declined as a result of the epidemiological transition. However, even after this transition, excess female mortality persisted. This could indicate that either the disease environment was not a decisive factor in the relative mortality risks of boys and girls, or that certain diseases that disproportionally affected girls continued to prevail even after the epidemiological transition.

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[166] Tabutin and Willems, 'Differential Mortality', 96.

The sex ratios and mortality rates suggest a relationship between high mortality rates and excess male mortality in certain years, such as the peak years of 1871 (scarlet fever and smallpox), 1879 (scarlet fever and pneumonia), 1900 (whooping cough), and 1905 and 1907 (both measles), where the death rates of boys clearly exceeded those of girls. Conversely, some years with high mortality, such as 1874, 1884, and 1918 (Spanish flu), exhibited excess female mortality. The mortality disparities between boy and girls during epidemic outbreak years suggest differing impact of epidemic and endemic diseases on each sex. Epidemic diseases caused sudden mortality peaks in specific periods, while endemic diseases were consistently present, contributing to the mortality pattern almost every year.[167] Murkens also examined gender differences in epidemic years in Maastricht and concluded that boys suffered more from outbreaks of epidemic diseases, while girls suffered more from endemic diseases.[168] This could explain why excess female mortality occurred in low-mortality years, as these endemic diseases were continuously present.

The higher mortality rate of girls from endemic diseases could be due to either increased exposure or increased susceptibility, which could be caused by differences in living conditions. Some researchers suggested, however, that differences in gender roles or discriminatory practices might have played smaller roles in this age category, as children aged 1-5 were not affected by inequalities in work or education.[169] Others pointed therefore to discriminatory practices in resource allocation and (health)care, which could have made girls more susceptible to diseases compared to boys at young ages.[170]

5.5 Older children (6-10 years)

Previous studies have observed more frequent patterns of excess female mortality among children aged 6-10 compared to younger children in Western European historical societies. Tabutin and Willems concluded that excess female mortality among children of these ages was high at the beginning of the nineteenth century, remained high during the century, gradually declined at the end of the century, and disappeared by 1920. However, in France, excess female mortality was noticeable even in the late 1920s among this age group.[171] Similarly, Van Poppel, Schellekens and Walhout observed excess female mortality for this age group in Limburg during the nineteenth century, but in their last sample years (1928-1930), this pattern had shifted to excess male mortality. On the other hand, studies like the one by McNay, Humphries and Klasen, which focused on nineteenth-century England and Wales, concluded that no excess female mortality occurred at these ages.[172]

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[167] Murkens, *Unequal pathways to the grave?*, 223.
[168] Ibid, 136-138.
[169] Ibid, 132; McNay, Humphries, and Klasen, 'Excess Female Mortality', 654.
[170] Tabutin and Willems, 'Differential Mortality', 49.
[171] Ibid, 24; Pinnelli and Mancini, 'Mortality peaks in Italy', 360.
[172] McNay, Humphries, and Klasen, 'Excess Female Mortality', 654.

Figure 3 revealed clear patterns of excess female mortality among children aged 6 to 10 years in Maastricht throughout the studied period. Similarly to the previous age category, Figure 7 shows the annual male-to-female mortality ratios (left axis) and the annual mortality rates of boys and girls (right axis). Additionally, the mortality peaks caused by disease outbreaks identified by Murkens are included in the graph.

Figure 7 Annual male-to-female mortality ratios and sex-specific mortality rates, children 6-10 years old, Maastricht 1864-1930.
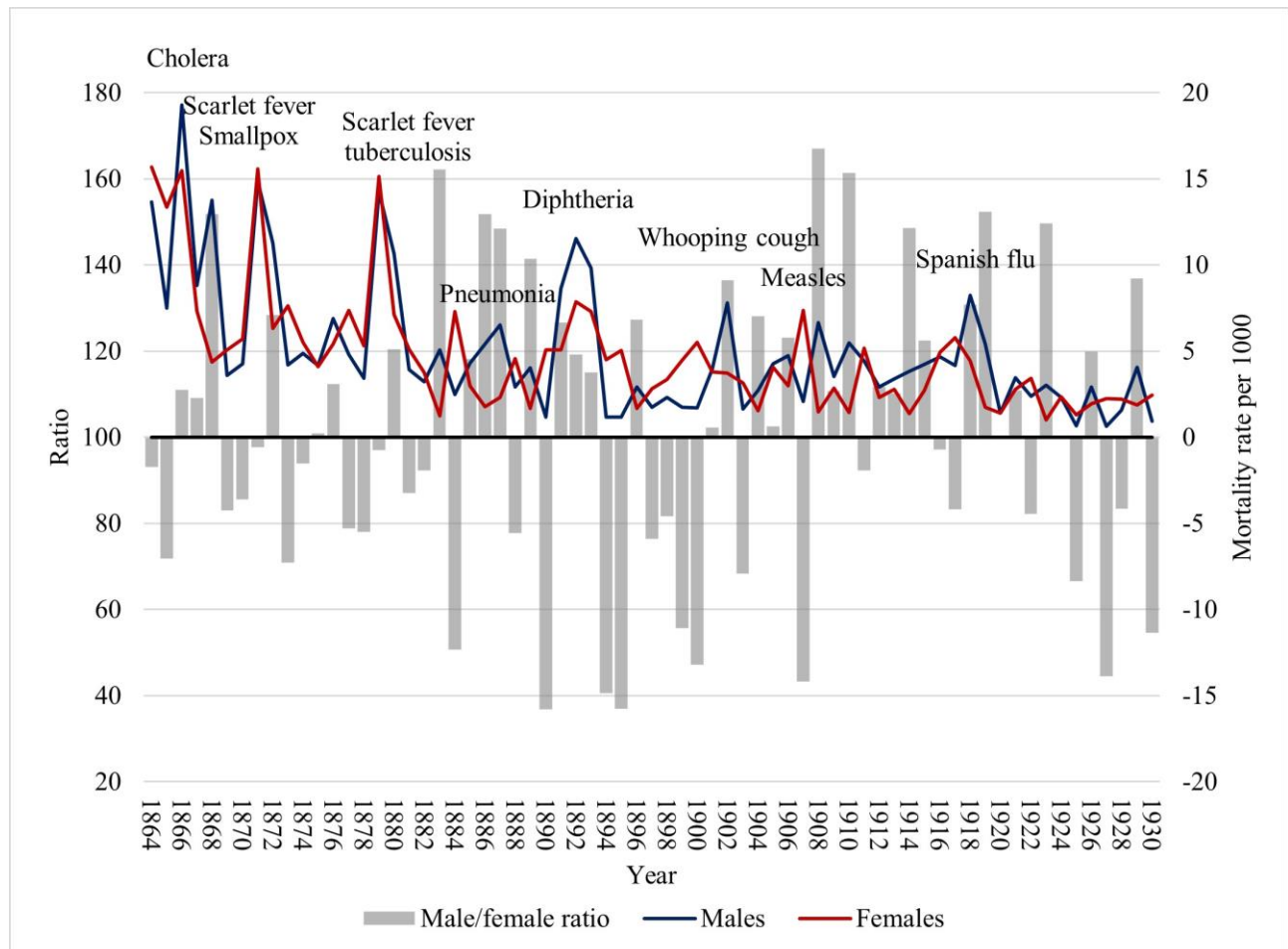
Ratios above 100 indicate excess male mortality, ratios below 100 indicate excess female mortality.
Source: Maastricht Death and Disease Database; Centraal Bureau voor de Statistiek, Volkstelling 1859-1930; Murkens, 'Unequal pathways to the grave', 109 and 223.

Figure 7 illustrates that compared to younger ages, mortality differences among children aged 6-10 years showed larger annual fluctuations, with higher peaks of both male and female excess mortality. Children aged 1-5 years had a maximum excess male mortality ratio of 127.8 compared to a maximum male ratio of 167 among children aged 6-10 years. The maximum excess female mortality ratio was 75.7 for children aged 1-5, whereas for children aged 6-10 years, it was 36.8. However, part of this larger difference in intensity could also be attributed to the lower mortality rates, resulting in larger differences in relative numbers.

Until around the 1880s, mortality ratios fluctuated between years, alternating between excess male and excess female mortality. Between 1882 and 1909, some large peaks of excess female mortality are evident, with a cluster in 1894-1900, slightly later than the cluster observed in younger children between 1888-1897. From 1908 onwards, a relatively stable pattern of excess male mortality is observed, with annual fluctuations recurring from 1920 onwards.

Some epidemic years, such as those with cholera (1866), pneumonia (1887), diphtheria (1892), and Spanish flu (1918) outbreaks, showed a clear male disadvantage. Years with lower overall mortality rates often exhibited excess female mortality, except for peak years like 1878 (tuberculosis) and 1884 (pneumonia). Similarly to the younger children, boys could have suffered more from epidemic diseases and girls from endemic diseases, like Murkens argued.[173] Compared to younger children, the largest decline in mortality rates occurred earlier, with the rates of girls and boys stabilising already in the 1890s, indicating an earlier completion of the epidemiological transition. However, frequent and large peaks due to epidemic outbreaks continued to play a role in the mortality pattern, potentially explaining why patterns of excess female mortality were still observed at the beginning of the twentieth century.

Pinelli and Mancini argued that gender roles begin to shape behaviour after the age of five, potentially impacting health outcomes due to differences in living and working conditions.[174] However, McNay, Humphries and Klasen countered that children under ten were rarely employed, implying minimal influence of gender roles on health outcomes in this age group.[175] Others emphasized the role of discriminatory practices like inequalities in access to the health system and education.[176] Figure 6 illustrated a clear male disadvantage in mortality rates among children aged 1-5 during the smallpox epidemic in 1871. Among children aged 6-10, this male disadvantage disappeared. This disappearance of the relative difference in mortality could indicate that boys from the age of 6 received more medical care, such as vaccinations, compared to girls. The higher mortality rates of girls during the scarlet fever outbreaks in 1871 and 1879 could further indicate differences in care or hygiene between boys and girls in Maastricht. Scarlet fever, a highly contagious disease spread through close contact, had no vaccination available. Proper hygiene is crucial in scarlet fever contraction.[177] Wall argued that in nineteenth-century England, parents prioritized the hygiene of their sons over girls.[178] If this was also the case in Maastricht, differences in care might have caused girls to have higher chances of contracting infectious diseases like scarlet fever compared to boys.

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[173] Murkens, *Unequal pathways to the grave?,* 136-138.

[174] Pinnelli and Mancini, 'Mortality peaks in Italy', 360.

[175] McNay, Humphries, and Klasen, 'Excess Female Mortality', 669.

[176] Murkens, *Unequal pathways to the grave?*, 132-133; Angélique Janssens, M. Messelink and Ariana Need, 'Faulty Genes of Faulty Parents? Gender, family and survival in early and late childhood in the Netherlands, 1860-1900', *History of the Family* 15:1 (2010) 91-108.

[177] Scarlet fever, West Northamptonshire Council, https://www.westnorthants.gov.uk/infectious-diseases/scarlet-fever (accessed on 27-22-2023).

[178] Wall, 'Inferring Differential Neglect of Females', 129.

5.6   Teenagers (11-15 years)

In Maastricht, the last age group exhibiting excess female mortality consisted of children between 11 and 15 years old. This pattern aligns with observations made in the Netherlands during the nineteenth century by Van Poppel, Schellekens, and Walhout. Between 1850-1859 and 1901-1902, all provinces in the Netherlands displayed rates of excess female mortality in this age category, with significant disparities in the latter period, particularly in Gelderland (48), Friesland (50), Drenthe (52), and Limburg (54). In Limburg, the last sample years of 1928-1930 showed excess male mortality rates. [179]

Similar to the previous age groups, the annual male-to-female mortality ratios (left axis) and annual mortality rates of boys and girls (right axes) have been computed, which is displayed in Figure 8. Additionally, the mortality peaks caused by disease outbreaks identified by Murkens are included in the graph.

Figure 8 Annual male-to-female mortality ratios and sex-specific mortality rates, children 11-15 years old, Maastricht 1864-1930.
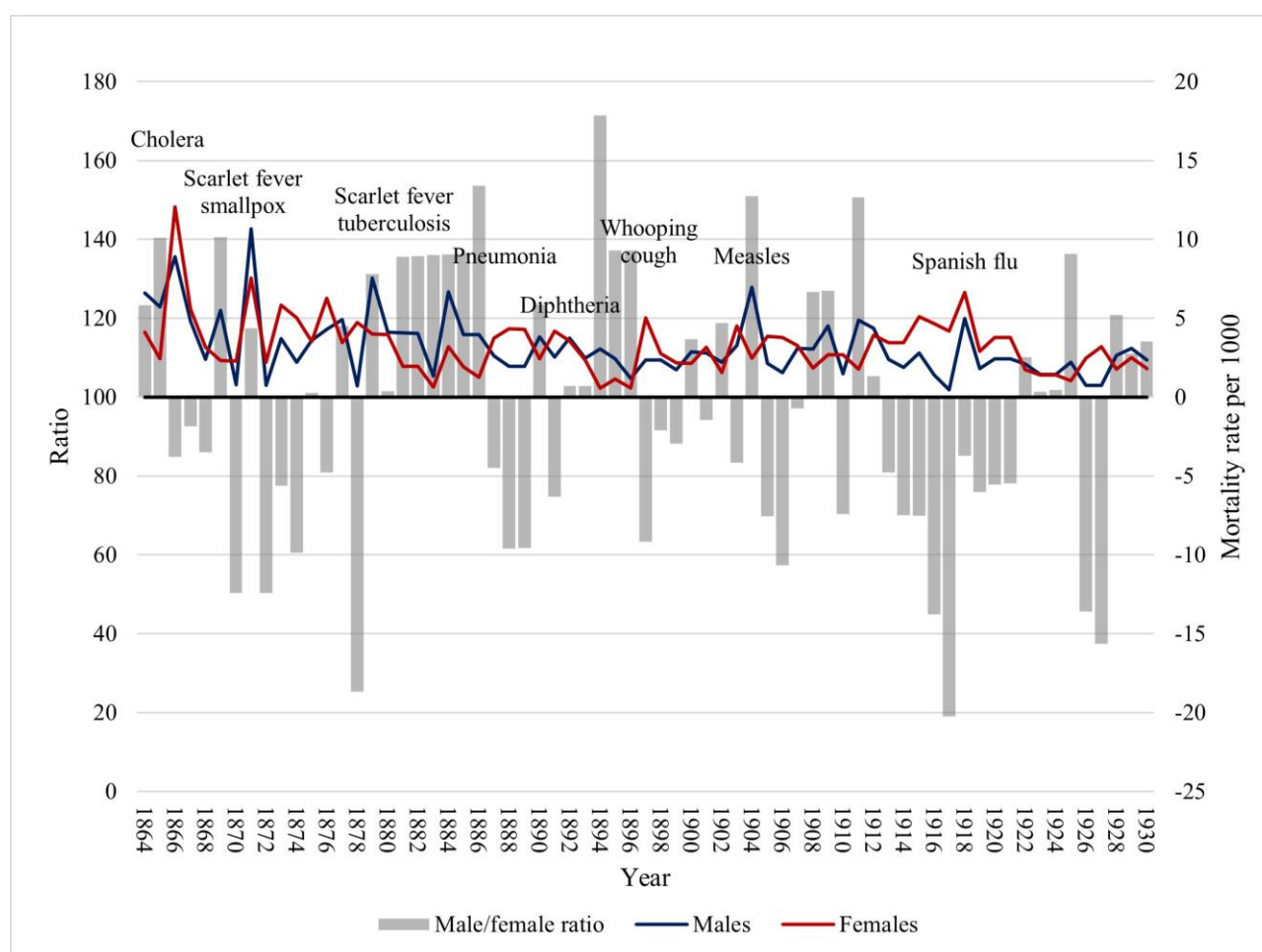
Ratios above 100 indicate excess male mortality ratios below 100 indicate excess female mortality.
Source: Maastricht Death and Disease Database; Centraal Bureau voor de Statistiek, Volkstelling 1859-1930; Murkens, 'Unequal pathways to the grave', 109 and 223.

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[179] Van Poppel, Schellekens, and Walhout, 'Oversterfte van jonge meisjes', 47.

Figure 8 shows that excess female mortality persisted in Maastricht among children between 11 and 15 throughout the entire research period, with clusters observed in 1870-1878, 1886-1892, 1896-1907, and 1912-1920. It is important to note that the intensity of these peaks should be interpreted cautiously, as mortality rates among this age group were low, leading to potentially large differences in rates due to small variations in absolute mortality numbers. For example, the peak in 1878 represented the death of 1 boy compared to 7 girls. Nevertheless, the results indicate that during certain periods, the mortality incidence among girls remained consistently higher than that among boys, regardless of the intensity of these differences.

The pattern of excess female mortality among older age groups followed a similar trajectory to that of younger age groups, but diverged towards the end of the researched period. Children aged 1-10 generally exhibited higher male mortality rates between 1910 and 1920. However, among teenagers aged 11-15, a clear cluster of excess female mortality emerged in this period. When examining the mortality rates, the mortality rates of children between 11 and 15 were lower compared to younger ages, and the decline in mortality at the end of the nineteenth century was less steep. Epidemic outbreak peaks were also smaller among this age group. For instance, the mortality peak in 1879 (scarlet fever and tuberculosis) was lower compared to other age groups, and the diphtheria outbreak (1892-1893) barely impacted mortality rates in this age group. Potentially, older children were less susceptible to infectious diseases compared to younger children, possibly due to better immune systems developed from earlier infections during infancy or childhood. High mortality years again showed in this age category in general more excess male ratios, like in 1871 (smallpox and scarlet fever), 1879 (scarlet fever and tuberculosis), 1884 (pneumonia), and 1904 (measles).

The unequal mortality rates between boys and girls in older age groups could be attributed to various factors. Some suggested that gender roles became more pronounced in this age group, as children reached the age to be employed in wage work.[180] Others pointed to girls assisting in household tasks, increasing their exposure to infectious diseases when caring for sick family members.[181] Additionally, the biological factor of increased susceptibility due to menstruation began to play a role in this age category.[182] Finally, unequal allocation of resources and care, as discussed in previous sections, might have further impacted girls' exposure to and resistance against infectious diseases.[183]

5.7 Adolescents (16-20 years)

In the Netherlands, male-to-female mortality ratios among children aged 16-20 varied across regions, with some showing pronounced excess male mortality and others with excessive female mortality rates. Van Poppel,

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[180] Tabutin and Willems, 'Differential Mortality', 46; McNay, Humphries, and Klasen, 'Excess Female Mortality', 669.
[181] Van Poppel, 'De statistieke ontleding van de dooden', 16.
[182] Henry, 'Men's and Women's Mortalty', 193.
[183] Devos, 'Te jong om te sterven', 40.

Schellekens, and Walhout observed in the province of Limburg excess female mortality in this age group in 1850-1859 and 1928-1930.[184] Janssens conducted a more regional analysis and observed in 1875-1890 higher levels of excess female mortality in this age category in southern and eastern regions in Limburg compared to other regions of the province.[185]

The results of this study showed that the mortality pattern of Maastricht differed from the province of Limburg, as no excess female mortality was found between 1864 and 1930. The disparity in male-to-female mortality ratios between Maastricht and Limburg cannot solely be attributed to urban-rural differences. Janssens found, for example, high levels of excess female mortality in urbanized regions and also pronounced excess male mortality patterns in rural areas.[186] Similarly, Van Poppel, Schellekens, and Walhout compared mortality differences among boys and girls in urban and rural municipalities and found no differences in excess female mortality levels between them.[187]

Janssens argued that the patterns of excess male mortality in this age category were likely explained by the professions these adolescents had. She argued that labour-intensive jobs in certain industries, such as heavy industries in cities like Dordrecht and Amsterdam, contributed to higher male mortality rates.[188] In Maastricht, more boys who died between 16 and 20 years old were registered with a profession compared to girls (respectively 76.5 and 30 per cent). Exploration of the types of professions indicated that hazardous working conditions could indeed be related to the higher death rates of boys. For example, pottery work, often registered as a profession among the Maastricht boys, contained harsh and dangerous working conditions, illustrated by the Dutch saying 'aardewerk is paardewerk' (*'pottery work is horse work')*.[189] However, due to the lack of data on surviving adolescents in this study, it is not possible to definitely conclude that working conditions caused the higher male mortality rates.

The excess male mortality rates of adolescents in Maastricht may have indicated either a relative improvement in the position of girls compared to younger age groups or a relative decline in the position of boys. Further comparison with Dutch regions showing excess female mortality could help determine if working conditions were decisive in relative differences in health outcomes between males and females. However, as this research focusses on excess female mortality, it will continue to concentrate on those age groups in which that mortality pattern occurred.

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[184] Van Poppel, Schellekens, and Walhout, 'Oversterfte van jonge meisjes', 47 and 60.
[185] Janssens, *Sekse, Gender en de Dood*, 15-17.
[186] Ibid, 16.
[187] Van Poppel, Schellekens, and Walhout, 'Oversterfte van jonge meisjes', 48.
[188] Janssens, *Sekse, Gender en de Dood,* 18-19.
[189] Jan H. Kompagnie, ''Aardewerk is Paardewerk': een rapport over de aardewerkindustrie uit 1971', *Tidinge van die Goude* 3:1 (2006) 24-28.

5.8 Conclusion

This chapter analysed mortality disparities among children aged between 0 and 20 in Maastricht from 1864 to 1930. The investigation of quinquennial male-to-female mortality rates revealed consistent patterns of excess female mortality among children aged 1-15. While prior research argued that excess female mortality had disappeared in the Netherlands by 1924, this study found that it persisted in Maastricht until at least 1930. This indicates that local-level disparities continued despite their disappearance at the national level. While this study cannot examine factors leading to the disappearance of excess female mortality in Maastricht, this study can explore the determinants of higher female mortality risks in depth, offering insights in the mechanisms of unequal mortality patterns between boys and girls.

The two groups that exhibited higher male than female mortality ratios were infants (<1 year) and adolescents (16-20). Compared to female infants, male infants are inherently more susceptible and weaker, which explains the excess male mortality in this age group. The examination of male-to-female stillbirth ratios and sex ratios at birth examined the potential presence of any discriminatory practices like female infanticide. While some years showed suspicious rates, the results suggested that if discriminatory practices in Maastricht existed, they were not widespread. The second group with constant excess male mortality patterns were adolescents aged 16-20, which contrasted to other Dutch regions where excess female mortality among this age group was observed. The higher male mortality rates in Maastricht may be attributed to the industrial context, with adolescents more likely to be employed in hazardous industrial jobs compared to girls, who might have been more involved in household work.

The analysis of children aged 1-5, 6-10, and 11-15 revealed frequent years where girls had higher mortality rates than boys. Despite some variations in intensity and clustering across age groups, all showed patterns of excess female mortality until the end of the studied period. A comparison to the mortality rates showed that high mortality years seemed to be linked to excess male mortality ratios, while low mortality years tended to exhibit excess female mortality ratios. These results suggested disparities between boys and girls in mortality caused by epidemic outbreaks, which appeared to affect boys more often, compared to endemic diseases that seemed to impact girls more. These differences imply variations in susceptibility and exposure to infectious diseases, influenced by differences in behaviour and caregiving between boys and girls.

As this research focuses on patterns of excess female mortality, the rest of the research will focus on mortality disparities among children aged 1-15 years in Maastricht. The next chapter will explore the causes of death that disproportionally affected girls and the last chapter will explore a set of determinants that could indicate the role of biological factors, gender roles, and discriminatory practices in elevated mortality risks of girls.

6. What killed girls? Identifying causes of death disproportionally affecting girls aged 1-15 in Maastricht, 1864-1930.

6.1 Introduction

This chapter will focus on differences between boys and girls in causes-of-death among children aged 1-15 in Maastricht between 1864 and 1930. Previous studies identified infectious diseases as the primary cause of excess female mortality. However, most studies relied on all-cause mortality data, like municipal or national death statistics, where diseases were often grouped into larger categories which aetiologically were not always linked to each other. Therefore, underlying causes, like the routes of transmission, could often not be examined. Moreover, because of this grouping, the effects of numerous individual diseases could also not be researched. The individual-level cause-of-death data used in this research enables the exploration of different causal groups, differentiating between routes of transmission, and of specific diseases and outbreaks, providing a more in-depth analysis on causes of death that were linked to patterns of excess female mortality.

This chapter seeks to answer three sub-questions. (1) *Were infectious diseases more deadly to girls and were there differences across age groups?* This analysis will focus on differences in mortality rates between infectious and other causes-of-death among boys and girls. Additionally, it will distinguish the routes of transmission of infectious diseases, which might indicate disparities in living or working conditions. (2) *Which specific diseases were more lethal to girls?* To answer this question, the analysis will explore male-to-female mortality ratios of individual diseases. (3) *Did disease outbreaks affect girls and boys differently?* This analysis will examine male-to-female mortality ratios to explore if disease outbreaks changed the observed causal patterns among boys and girls.

6.2 Causal groups of death

Infectious diseases have often been named as a cause of patterns of excess female mortality. During the nineteenth and early twentieth centuries, infectious diseases were the primary causes of death in European societies and they disproportionally affected women.[190] Numerous studies emphasized the unequal mortality rates of infectious diseases between boys and girls.[191] Devos observed, for instance, that in nineteenth-century Belgium, half of all boys aged 5-20 died because of infectious diseases, and even more than half of the girls

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[190] Shorter, *Women's Bodies,* 231

[191] Beltrán Tapia and Szoltysek, 'Missing girls', 619-657; Klasen and Wink, 'Missing women, 280; Janssens, *Sekse, Gender en de Dood,* 15; Pinnelli and Mancini, 'Mortality peaks in Italy', 353.

died from this cause of death.[192] Also Henry emphasized that girls had higher probabilities of dying from infectious diseases, especially from tuberculosis, in the nineteenth century.[193] However, the majority of these researchers relied on aggregated data, making it not possible to examine the role of different routes of transmission of infectious diseases or make adept categories for the other causes of death that could for instance indicate differences in behaviour.

To examine what caused girls to die more often than boys, the causes-of-death among children aged 1-15 will be explored. As explained in the data and methodology section, this study used Murkens' categorization that distinguished seven causal groups: (1) food- and waterborne infectious diseases, (2) airborne infectious diseases, (3) other infectious diseases, (4) non-infectious diseases, (5) non-natural causes of death, (6) ill-defined causes of death, and (7) unknown causes of death. The male-to-female mortality ratios per causal groups were computed to examine differences in mortality rates between boys and girls. First, the annual male and female death rates per causal group were computed. Next, the male-to-female ratios were computed based on the average death rate of all years combined (1864-1930). Table 2 contains the ratios of all ages combined, as well as the separate age categories.

Table 2 Male-to-female mortality ratios per causal group, all years combined, children aged 1-15, Maastricht 1864-1930.

Cause of death	All ages 1-15 years			Young children 1-5 years			Older children 6-10 years			Teenagers 11-15 years		
	Ratio	*N*	*%*	*Ratio*	*N*	*%*	*Ratio*	*N*	*%*	*Ratio*	*N*	*%*
Infectious diseases												
Food- and waterborne	102.8	2295	*24.2*	100.2	2037	*27.0*	**92.1**	154	*13.4*	112.4	104	*13.4*
Airborne	**95.4**	5390	*56.9*	**97.4**	4414	*58.4*	**97.3**	608	*53.1*	**81.9**	368	*47.5*
Other	100.9	297	*3.1*	105.8	162	*2.1*	**96.5**	67	*5.9*	**86.7**	68	*8.8*
Non-infectious diseases	114.2	733	*7.7*	108.4	423	*5.6*	109.4	179	*15.6*	**94.0**	131	*16.9*
Non-natural	310.0	308	*3.2*	133.0	177	*2.3*	168.5	78	*6.8*	189.5	53	*6.8*
Ill-defined	107.5	190	*2.0*	**97.7**	159	*2.1*	128.9	17	*1.5*	126.5	14	*1.8*
Unknown	105.7	264	*2.8*	**98.9**	186	*2.5*	110.2	42	*3.7*	115.2	36	*4.7*
Total N		**9477**	*100*		**7558**	*100*		**1145**	100		**774**	*100*

Ratios above 100 indicate excess male mortality, ratios below 100 indicate excess female mortality.
Source: Maastricht Death and Disease Database; Centraal Bureau voor de Statistiek. Volkstelling 1859-1930.

The ratios of all ages combined in Table 2 show that combined airborne infectious diseases were the only causal group with excess female mortality. When examining separate age groups, results reveal that among young children, older children, and teenagers, these infectious diseases transmitted via air were more deadly to girls compared to boys, with ratios of respectively 97.4, 97.3, and 81.9. These findings demonstrate

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[192] Devos, 'Te jong om te sterven', 65.
[193] Henry, 'Men's and Women's Mortality', 193.

that airborne infectious diseases were a significant contributor to unequal mortality rates between boys and girls in Maastricht, accounting for over half of all deaths (56.9 per cent) and disproportionally affecting girls.

The ratios for all age groups combined reveal that other causal groups affected boys more than girls. However, results from separate age categories show more differences. Among girls aged 6-10, excess female mortality was observed across all types of infectious diseases. Food- and waterborne infectious diseases were even more lethal to girls in this age group compared to airborne infectious diseases, with ratios of 92.1 and 97.3 respectively. Additionally, teenagers (11-15) exhibited excess female mortality not only in airborne infectious diseases but also in the 'other' category. The emergence of excess female mortality ratios at older ages (6-15) in other types of infectious diseases suggests that additional factors may have come into play compared to children aged 1-5. These factors could include gender roles, discriminatory practices, or biological factors during puberty, which, compared to children aged 1-4, created more differences in life conditions between boys and girls, potentially causing patterns of excess female mortality to appear in more causal groups.[194]

The excess female mortality rates in the categories 'ill-defined' and 'unknown' among young children were noteworthy, especially considering that in older age categories, boys were more frequently registered with these types of deaths. The lower precision in registering deaths of young girls compared to boys may suggest that doctors or families paid less attention to assessing the cause of death for young girls, indicating a potential difference in medical care between the genders. Hospital records could give a more detailed picture in differences in treatment between boys and girls at that time. However, it is also possible that doctors were better trained in conditions affecting male bodies, as is the case in today's society where medical knowledge of the female body lags behind that of the male body.[195] This difference in the assessment of cause of death could therefore also stem from a disparity in medical knowledge. An examination of nineteenth-century medical literature could provide further insights into this potential difference.

Another remarkable result from Table 2 is the excessive mortality rates of boys due to non-natural causes-of-death, with an index of 310 for children aged 1-15. This excess male mortality due to violent death causes could indicate differences in behaviour and gender roles. Boys often exhibit more risk-seeking behaviour, increasing the chances of accidents. Additionally, gender roles, such as boys working in factories from a younger age, may have also increased the likelihood of violent deaths. In Chapter 3, the non-natural causes-of-death will be explored in depth, in order to examine underlying differences in behaviour between boys and girls.

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[194] McNay, Humphries, and Klasen, 'Excess Female Mortality', 669; Murkens, *Unequal pathways to the grave?*, 132-133; Janssens, Messelink and Need, 'Faulty Genes of Faulty Parents?', 91-108.
[195] Lea Merone, Komla Tsey, Darren Russel, and Cate Nagle, 'Sex Inequalities in Medical Research: A Systematic Scoping Review of the Literature', *Women's Health Reports* 3:1 (2022) 49-59.

6.3 Specific diseases

Few studies have examined the effects of specific diseases on excess female mortality. Henry argued that pulmonary tuberculosis in nineteenth-century England was linked to patterns of excess female morality, a link also made by McNay, Humphries, and Klasen for nineteenth-century England and Wales.[196] Devos highlighted the role of tuberculosis in excess female mortality patterns in nineteenth-century Belgium, although other groups of diseases, like smallpox, angina, and croup combined or the group of typhus, cholera, and dysentery also showed excess female mortality in her reports. However, Devos did not elaborate on the connection between these diseases and increased female mortality rates. [197] Murkens examined the male-to-female ratios of individual diseases among children aged 1-4 in Maastricht to explore mortality differences between epidemic and endemic diseases. Epidemic diseases caused sudden mortality peaks, while endemic diseases consistently contributed to the mortality pattern.[198] The study revealed excess male mortality ratios from epidemic diseases like measles and scarlet fever, and excess female mortality ratios from endemic diseases like pneumonia, tuberculosis, and bronchitis.[199] However, in the context of studying excess female mortality, an in-depth exploration of individual diseases among children aged 1-15 is lacking, particularly with a focus on differences in transmission routes and age categories.

This section analyses the male-to-female mortality ratios for the most common infectious diseases in Maastricht among children aged 1-15 years. These diseases accounted for approximately 80 per cent of all deaths among children aged 1-5, 60 per cent among those aged 6-10, and roughly half of all deaths among teenagers aged 11-15. Annual male and female death rates for individual diseases were computed, and the male-to-female ratios were calculated based on the average of these annual mortality rates. The ratios for all ages combined and separate age groups are presented in Table 3, with epidemic diseases marked with an asterisk. Annex III provides a list of identified epidemic and endemic diseases in the Maastricht pattern, including outbreak years.

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[196] Henry, 'Men's and women's mortality in the past', 193; McNay Humphries and Klasen, 'Excess Female Mortality', 671-672.
[197] Devos, 'Te jong om te sterven', 66.
[198] Murkens, *Unequal pathways to the grave?*, 223.
[199] Ibid, 138.

Table 3 Male-to-female mortality ratios during disease outbreaks, children 1-15, Maastricht 1864-1930.

	All ages 1-15 years			Young children 1-5 years			Children 6-10 years			Teenagers 11-15 years		
	Ratio	*N*	*%*	*Ratio*	*N*	*%*	*Ratio*	*N*	*%*	*Ratio*	*N*	*%*
Food- and waterborne												
Gastroenteritis	**99.9**	1125	*11.9*	**98.5**	1073	*14.2*	**93.5**	36	*3.1*	109.0	16	*2.1*
Convulsions	107.2	516	*5.4*	104.7	497	*6.6*	108.6	11	*1.0*	151.5	8	*1.0*
Tuberculosis	**98.1**	273	*2.9*	**98.7**	183	*2.4*	**98.2**	49	*4.3*	**86.6**	41	*5.3*
Cholera*	**94.7**	237	*2.5*	**96.3**	182	*2.4*	**74.6**	38	*3.3*	115.8	17	*2.2*
Typhus	**97.3**	92	*1.0*	**94.9**	25	*0.3*	113.7	34	*3.0*	**81.4**	33	*4.3*
Airborne												
Pneumonia	**95.7**	1398	*14.8*	**95.1**	1293	*17.1*	**86.7**	72	*6.3*	**76.1**	33	*4.3*
Pulmonary tuberculosis	**87.2**	803	*8.5*	**95.1**	475	*6.3*	**83.7**	150	*13.1*	**64.9**	178	*23.0*
Diphtheria**	102.4	664	*7.0*	**99.2**	594	*7.9*	121.2	65	*5.7*	**91.0**	5	*0.6*
Meningitis	103.4	543	*5.7*	102.0	402	*5.3*	**98.3**	100	*8.7*	114.0	41	*5.3*
Measles*	112.7	350	*3.7*	110.5	343	*4.5*	103.7	7	*0.6*	-	-	-
Bronchitis	**93.5**	345	*3.6*	**90.3**	333	*4.4*	143.5	7	*0.6*	127.8	5	*0.6*
Scarlet fever*	111.0	310	*3.3*	112.2	242	*3.2*	**99.3**	53	*4.6*	112.5	15	*1.9*
Encephalitis	**97.9**	291	*3.1*	**94.1**	211	*2.8*	103.2	60	*5.2*	110.7	20	*2.6*
Whooping cough*	**50.1**	150	*1.6*	**46.8**	148	*2.0*	202.5	2	*0.2*	-	-	-
Other causes-of-death	110.0	2380	*25.1*	107.4	1557	*20.6*	114.1	461	*40.3*	112.2	362	*46.8*
Total		9477	*100*		7558	*100*		1145	*100*		774	*100*

Ratios above 100 indicate excess male mortality, ratios below 100 indicate excess female mortality.
* Epidemic disease, see Annex I.
** Endemic disease with major epidemic outbreaks, see Annex 1.
Source: Maastricht Death and Disease Database; Centraal Bureau voor de Statistiek. Volkstelling 1859-1930.

Table 3 reveals that almost all food- and waterborne infectious diseases exhibited excess female mortality ratios among children aged 1-15, except for convulsions. Due to the broad spectrum of causes within this category, the reason for the excess male mortality remains unclear. The excess female mortality ratios of some food- and waterborne infectious diseases shifted to excess male mortality across different age groups, however, caution is warranted in interpreting results for children above 5 years due to low death counts. Small absolute differences could lead to significant variations in these ratios. Excess female mortality ratios among food- and waterborne infectious diseases might suggest disparities in exposure between boys and girls. Cholera, for example, transmitted via contaminated water sources, like polluted drinking water or canals used for waste disposal.[200] Girls might have been exposed more or more intense to these polluted water sources due to

[200] Mayra Murkens and Thijs van Vught, 'Een toplocatie aan het water? Een ruimtelijke analyse van de invloed van sociaaleconomische status en woonplaats op de verspreiding van en sterfte door cholera in Maastricht tijdens het epidemiejaar 1866' in: *SHCL Jaarboek* (2022) 59-89, there 65-66.

household chores, which could have exacerbated cholera's impact, resulting in a higher number of female deaths.

Airborne infectious diseases exhibited larger mortality disparities between boys and girls compared to food- and waterborne diseases. Lung-related ailments like pneumonia and bronchitis disproportionally affected young girls aged 1-5 with ratios of 95.1 and 90.3, respectively, constituting combined almost one-fifth of all deaths in this age group. Pulmonary tuberculosis ranked second, displaying excess female mortality across all age groups. Particularly in the 11-15 age group, pulmonary tuberculosis was a significant contributor to mortality disparities between boys and girl, with nearly a quarter of deaths attributed to this disease and a ratio of 64.9 indicating substantial differences in death rates between the sexes. Devos highlighted tuberculosis' role in excess female mortality in nineteenth-century Belgium, emphasizing its multifactorial nature. Infection with tuberculosis does not immediately lead to illness. Instead, a combination of factors such as age, sex, nutrition, and living conditions influences infection progression to illness. Elevated mortality rates among girls due to tuberculosis therefore suggest disparities in life conditions between boys and girls in Maastricht.[201]

Infectious diseases affecting the brain also exhibited relatively higher mortality rates among females. Encephalitis was more lethal to girls aged 1-5, with a ratio of 94.1, while meningitis had an index of 98.3 among children aged 6-10. However, these diseases contributed less to overall mortality, comprising only 3.1 and 5.7 per cent of all deaths, respectively. Studies rarely discuss these diseases in the brain in the past, especially in the context of excess female mortality. Some studies addressed an epidemic outbreak of encephalitis lethargica between 1918 and 1930, affecting an estimated half a million people in Europe. However, encephalitis lethargica was first recognized in 1917, while encephalitis as a cause of death has been recorded in Maastricht since 1864. Moreover, the peak outbreak in Maastricht occurred in 1871, more than forty years before the epidemic outbreak of *encephalitic lethargica*.[202] It remains therefore unknown whether the two causes-of-death were the same or resulted from different diseases. Due to the lack of literature on nineteenth-century encephalitis, questions persist regarding the underlying causes of these infections and why they disproportionally affected girls.

Lastly remain the mortality differences from epidemic and endemic diseases. The results in Table 3 compare to Murkens' observations of airborne infectious diseases. Endemic diseases like pneumonia, pulmonary tuberculosis, and bronchitis exhibited excess female mortality, while epidemic diseases like measles and scarlet fever show excess female mortality rates.[203] Interestingly, among food- and waterborne infectious diseases, cholera and diphtheria demonstrated ratios of excess female mortality in certain age groups, despite being epidemic diseases. Moreover, the average male-to-female ratios fail to explain some of the excess male mortality peaks observed in Figures 6, 7, and 8 from Chapter 1. For instance, the pneumonia outbreak in 1887 exhibited higher male mortality rates across all age groups, contradicting the average male-to-female

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[201] Devos, 'Te jong om te sterven', 66-67.

[202] Leslie A. Hoffman and Joel A. Vilensky, 'Encephalitis lethargica: 100 years after the epidemic', *Brain, A journal of neurology* 40:1 (2017) 2246-2251, there 2246.

[203] Murkens, *Unequal pathways to the grave?*, 136-137.

mortality ratio that indicates higher female mortality rates from pneumonia. This suggests that gender differences may not only occur between epidemic and endemic diseases, but also between endemic diseases and disease outbreaks (both from epidemic and endemic diseases).

6.4 Disease outbreaks

To examine sex differences during disease outbreaks, the male-to-female mortality ratios of all years combined were compared to those during an outbreak year, as displayed in Table 4. Outbreak years were identified if the total death rate for a disease in a year exceeded the mean mortality rate plus three times the standard deviation. Annex III provides a detailed overview of this assessment. The 'average ratio' column presents the average male-to-female mortality ratio of the respective disease across all years (1864-1930), while the 'outbreak year ratio' column displays the male-to-female mortality ratio during the specific year of a disease outbreak.

Table 4 Male-to-female mortality ratios during disease outbreaks, children 1-15, Maastricht 1864-1930.

Disease	Mean mortality rate	Outbreak year	Male mortality rate per 1,000	N	Female Mortality rate per 1000	N	Average ratio	Outbreak year ratio
Endemic								
Bronchitis	0.47	1891	1.50	8	2.19	12	**93.5**	**81.1**
Convulsions	0.76	1864	1.95	8	3.65	15	107.2	**69.6**
		1867	3.06	13	3.07	13		**99.8**
Diphtheria	0.93	1892	10.43	56	10.16	56	102.4	101.3
		1893	9.79	53	9.00	50		104.3
Encephalitis	0.45	1871	2.49	11	3.42	15	**97.9**	**84.4**
Gastroenteritis	1.62	1878	4.30	20	7.74	36	**99.9**	**71.4**
Meningitis	0.74	1879	1.92	9	2.99	14	103.4	**78.2**
Pneumonia	1.84	1887	7.60	39	4.96	26	**95.7**	121.3
Tuberculosis	0.34	1870	2.05	9	1.15	5	**98.1**	128.1
Typhus	0.14	1871	0.45	4	1.82	8	**97.3**	**39.9**
Epidemic								
Cholera	0.40	1866	15.93	67	15.73	66	**94.7**	100.6
Measles	0.43	1907	3.25	20	2.94	19	112.7	105.1
		1910	3.94	25	2.84	19		116.7
Scarlet fever	0.50	1864	10.94	45	9.96	41	111.0	104.7
		1871	8.60	38	3.87	17		137.8
		1879	9.17	43	5.55	26		124.6
Whooping cough	0.19	1897	0.72	4	1.57	9	**50.1**	**62.3**
		1902	0.51	3	1.82	11		**43.6**

Ratios above 100 indicate excess male mortality. ratios below 100 indicate excess female mortality.
Source: Maastricht Death and Disease Database; Centraal Bureau voor de Statistiek. Volkstelling 1859-1930.

The results from Table 4 show that the outbreaks of the epidemic diseases measles and scarlet fever continued to contain excess male mortality rates during the years in which major outbreaks occurred, while cholera also showed a higher male mortality rate during the epidemic outbreak in 1866. Some ratios of endemic diseases remained unchanged during an outbreak year. Bronchitis, encephalitis, gastroenteritis and typhus exhibited on average excess female mortality rates and this persisted during outbreak years. Similarly, diphtheria exhibited both on average and during the outbreak years excess male ratios. Interestingly, some endemic diseases exhibiting on average excess female mortality rates, showed higher male mortality rates during outbreak years, like the 1887 pneumonia outbreak and the tuberculosis outbreak in 1870, although the latter sex ratio should be interpreted with caution due to the low numbers of deaths.

These differences in deaths caused by disease outbreaks versus endemic diseases seem to be an important factor in the overall fluctuation from excess male to excess female mortality. In years with larger outbreaks of diseases, of both endemic or epidemic diseases, boys were struck relatively more compared to girls. However, in 'normal' mortality years, mortality rates of girls were higher compared to boys as girls were affected more by endemic diseases.

Murkens suggested that the difference in mortality between boys and girls during disease outbreaks might stem from girls having a generally poorer health status, leading to higher mortality in 'normal' circumstances. However, during disease outbreaks, boys' biological vulnerability became more pronounced as diseases spread widely, increasing their chances of infection.[204] Alternatively, this difference could also be attributed to variations in exposure between boys and girls, resembling the idea of the 'healthy migrant effect'. Alter and Oris, along with Puschmann et al., observed a healthy migrant effect among rural-to-urban migrants, their lower exposure to infectious diseases previously in life benefitted their overall health, leading to lower mortality rates compared to urban natives. However, during epidemic outbreaks, these migrants were struck more compared to the urban population due to their lack of prior exposure, resulting in lower immunity.[205] Similarly, a 'healthy boy effect' might have occurred in Maastricht. Girls – like the urban population – may have had higher general mortality rates than boys as they were more exposed to infectious diseases because of their gender roles. Boys, meanwhile, could have had lower general mortality rates due to lower exposure – like migrants, but this lower exposure might have caused higher mortality rates during disease outbreaks due to lower immunity build-up.

The higher susceptibility mentioned by Murkens and/or greater exposure among girls could explain their generally higher mortality rates, particularly from endemic diseases. Increased risk of infection raised the likelihood of co-morbidity, either simultaneously or subsequently. For instance, measles, while not inherently lethal, can lead to death due to secondary infectious like pneumonia, that easier struck the child due to a

[204] Murkens, *Unequal pathways to the grave?*, 139.
[205] George Alter and Michel Oris, 'Childhood Conditions. Migration and Mortality. Migrants and Natives in 19th Century Cities', *Social Biology* 3:4 (2005) 178-191. Paul Puschmann, Robyn Donrovich, Per-Olof Grönberg, Graziela Dekeyser and Koen Matthijs, 'Disfavored in Life, Favored in Death? Later-Life Mortality Differences (Ages 30+) between Migrants and Natives in Antwerp, Rotterdam and Stockholm, 1850-1930', *Historical Social Research / Historische Sozialforschung* 41:4 (2016) 257-290.

suppressed immune system caused by the measles infection.[206] Subsequent co-morbidity, also known as the scarring mechanism, occurs when a child survives multiple infections, leaving a child scarred and more vulnerable which potentially turns even mild infections into lethal ones.[207]

The higher susceptibility Murkens named and/or a higher exposure among girls could explain why girls in general had higher mortality rates, especially from endemic diseases. A higher risk of infection increased the chances of co-morbidity, either simultaneously or subsequently. An example of simultaneous co-morbidity is measles, which often on its own not a particularly lethal disease, yet it can result in death because of other infections like pneumonia that easier struck the child due to the measles infection suppressing its immune disease. Subsequent co-morbidity, on the other hand, is also known as scarring-mechanism. If a child survives multiple infections subsequently, it could have been scarred and become more susceptible and weaker, which could lead to even milder infections becoming lethal

Future research could further explore the impact of co-morbidity on the male-to-female ratios in Maastricht. While this study primarily focused on the primary cause of death, investigating secondary and tertiary causes could offer insights into simultaneous co-morbidity in girls' mortality rates. However, examining subsequent co-morbidity comes with challenges, as the Maastricht Death and Disease Database only covers the end stage of life and not earlier health issues. Hospital reports might provide valuable information on health conditions preceding death, although they may not include data on less severe infections that could still scar children's' health.

6.5 Conclusion

This chapter examined the causes of death of children aged 1-15 years in Maastricht to understand what causes of death caused girls to die more often compared to boys. By analysing individual-level cause-of-death data, it aimed to uncover factors contributing to excess female mortality, focusing on different routes of transmission of infectious diseases, male-to-female mortality ratios of individual diseases, and differences in mortality rates during disease outbreaks.

The results showed a significant role of airborne infectious diseases in excess female mortality patterns. Particularly lung-related diseases, like pneumonia and bronchitis among young children and pulmonary tuberculosis among older children, played important roles in the observed mortality disparities. These diseases accounted for relatively large shares of deaths and exhibited substantial mortality disparities between boys and girls. Food- and waterborne infectious diseases also contributed to the excess female mortality pattern, with diseases like gastroenteritis, typhus and tuberculosis exhibiting higher female mortality rates. The higher

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[206] Rutten, *De vreselijkste aller harpijen*, 399.
[207] Luciana Quaranta, *Scarred for Life. How conditions in early life affect socioeconomic status, reproduction and mortality in Southern Sweden, 1813-1968*, Doctoral Thesis, Lund University (2013) 5; Murkens, *Unequal pathways to the grave?*, 113-114.

female mortality rates of infectious diseases indicate that girls were either exposed to infectious diseases more often compared to boys or that they had a higher susceptibility to these types of diseases.

The examination of differences in mortality rates between epidemic and endemic diseases underscored the differences in exposure and susceptibility between boys and girls. Boys exhibited higher mortality rates from epidemic diseases, while girls died more often from endemic diseases. Additionally, during disease outbreaks, boys also died more often from some endemic diseases.

The higher mortality rates of girls form endemic diseases could explain the patterns of excess female mortality. Endemic diseases were always prevalent, causing girls to generally die more often than boys. These higher female mortality rates indicate that girls were more exposed to infectious diseases and/or had a higher susceptibility, causing an overall lower health status of girls. A higher infection rate could have increased the risks of co-morbidity, as infections could have suppressed the immunity system, potentially causing even milder infections to be deadly, or could scar the body, leading to increased susceptibility to new infections.

The lower mortality rates of boys from endemic diseases and higher rates among disease outbreaks indicate that boys were less exposed to infections, suggesting a 'healthy boy effect'. Boys may have had a better health status due to lower overall exposure to infectious diseases, resulting in lower mortality rates than girls. However, in years of disease outbreaks, boys may have been more vulnerable due to a lack of immunity from previous infections, causing their mortality rates to exceed the girls'.

The next chapter will aim to identify the determinants causing these differences in health status and health outcomes between boys and girls. Could biological factors have caused these differences? Were the differences in exposure caused by differences in gender roles and expectations? Or did gender-specific discrimination lead to a lower health status of girls, increasing their susceptibility?

7. Biological factors, gender roles or discrimination? Examining the causes of the Maastricht excess female mortality pattern, children aged 1-15, 1864-1930.

7.1 Introduction

This chapter, explores three potential groups of explanatory factors: biological factors, gendered roles and discriminatory practices. The results from previous chapters indicate that excess female mortality occurred among children aged 1 to 15 between 1864 and 1930. The examination of causes-of-death in Chapter 2 revealed that infectious diseases, particularly endemic airborne infectious diseases like pneumonia and pulmonary tuberculosis, significantly contributed to patterns of excess female mortality in Maastricht. This raises the question if any underlying factors causing the increased mortality risks can be discerned: Were girls biologically more susceptible to these diseases and how affected changes in the disease environment general mortality patterns? Were gendered roles, like differences in behaviour or working conditions, important factors creating different health outcomes among boys and girls? Or lastly, could gender-specific discrimination, such as unequal allocation of resources, healthcare or parental care, be the driving mechanism behind the Maastricht pattern of excess female mortality?

This chapter will explore a broad set of potential indicators that could indicate a role of these biological, gender or discriminatory factors. These factors could concern both proximate determinants or ultimate determinants that have been discussed in the literature review.

7.2 Biological factors

The first group of explanations for excess female mortality focused on biological factors that could increase girls' susceptibility to infectious diseases. Henry suggested that hormonal changes during menstruation make the female body more susceptible to infections, while Devos mentioned menarche as a potential factor.[208] However, the average age of first menstruation in Europe was around 15 years at the end of the nineteenth century, and even if some girls experienced menarche at younger age, this would not explain the mortality patterns observed among girls between 1 and 10 years old.[209] Therefore, this explanation does not seem suitable for explaining the mortality difference between boys and girls in Maastricht.

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[208] Henry, 'Men's and Women's Mortality', 193; Devos, 'Te jong om te sterven', 40.
[209] Andreas Lehmann, Christiane Scheffler, Michael Hermanussen, 'The variation in age at menarche: an indicator of historic developmental tempo', *Anthropologischer Anzeiger* 68:1 (2016) 85-99.

Tabutin and Willems also highlighted biological factors, suggesting that women were more susceptible to infectious diseases, a trend that persisted until the 1950s, even after patterns of excess female mortality at the societal level had disappeared in Western European societies. They argued that this disappearance was primarily caused by the epidemiological transition, where infectious diseases ceased to be the dominant cause of death, leading to a decrease in overall female mortality rates.[210] Despite the epidemiological transition taking place, the patterns of excess female mortality did not disappear in Maastricht and they persisted at least until 1930. While the exact relationship between the changing environment and the disappearance of excess female mortality in Maastricht cannot be examined, it is possible to identify diseases that have *not* played a decisive role in the pattern. If a disease rarely or never occurred but excess female mortality persisted, it is likely that the disease was not a dominant factor in the mortality differences between boys and girls at a societal level.

To investigate the relationship between the course of specific infectious diseases and excess female mortality in Maastricht, the annual death rate per disease among children aged 1-15 were computed over time. Figure 9 displays the mortality rate trends of diseases that exhibited excess female mortality ratios during the studied period (as shown in Table 3). For clarity, the crude death rate of cholera is omitted from the graph. While cholera had a large outbreak in 1866, sporadic mortality occurred in other years, distorting the overall picture. While the disease did exhibit higher female mortality rates compared to males, the epidemic outbreak at the beginning of the researched period could not have been the primary cause of the general mortality rate discrepancy between the sexes until 1930.

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[210] Tabutin and Willems, 'Differential Mortality', 45-46.

Figure 9 Annual mortality rates of diseases with excess female mortality, children aged 1-15, Maastricht 1864-1930.
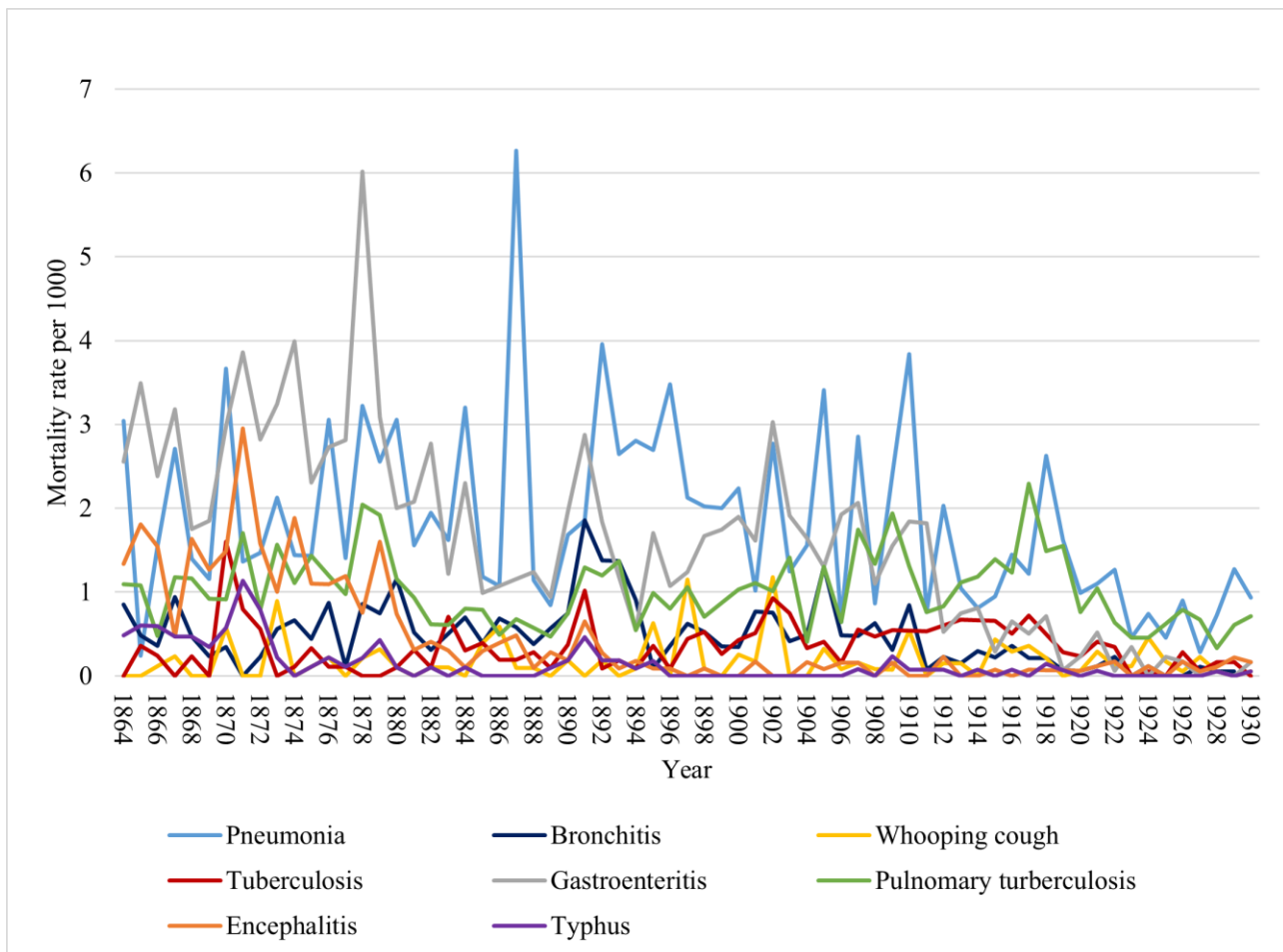
Source: Maastricht Death and Disease Database, Centraal Bureau voor de Statistiek. Volkstelling 1859-1930.

The results depicted in Figure 9 illustrate a decline in death rates for several diseases over time. For example, encephalitis was prevalent at the beginning of the research period but nearly disappeared after 1880. Similarly, gastroenteritis, which frequently caused mortality among children in Maastricht during the nineteenth and early twentieth centuries, saw a significant decline after 1910, and in certain years after 1918, it was absent from the mortality pattern entirely. Two diseases with pronounced female mortality ratios – pneumonia and pulmonary tuberculosis – persisted in the Maastricht mortality pattern until the end of the researched period, when the majority of deceases declined or disappeared. These diseases accounted for a significant share of overall mortality, with pneumonia causing 17.1 per cent of all deaths among children aged 1-5 and pulmonary tuberculosis contributing to 23 per cent of deaths among children aged 11-15. Therefore, these diseases seemed to have played a crucial role in the excess female mortality patterns in Maastricht. While this research examined mortality patterns in Maastricht until 1930, future studies could explore developments beyond this period and further investigate the relationship between patterns of excess female mortality and pneumonia and pulmonary tuberculosis.

The decline or disappearance of diseases with excess female mortality does not necessarily lead to a reduction in overall mortality rates for girls. Murkens highlighted that as very lethal infectious diseases such

as scarlet fever and diphtheria disappeared during the nineteenth century, milder diseases became more prominent in the mortality pattern.[211] The disappearance of numerous infectious diseases with higher female mortality rates did not guarantee the elimination of excess female mortality. If girls continued to be disproportionally exposed and/or affected by these milder diseases, it could perpetuate higher mortality rates among girls compared to boys due to co-morbidity, weakening the body and making it more susceptible to other potentially fatal infections. Future research could therefore investigate the role of co-morbidity, particularly during the early twentieth century, to determine whether persisting patterns of excess female mortality can be attributed to the prevalence of pneumonia and pulmonary tuberculosis, or if co-morbidity mechanisms contributed to higher mortality rates among girls despite the decline of diseases that previously disproportionally affected girls in the mortality pattern.

7.3 Gender roles

The second group of explanations suggests that disparities in behaviour between boys and girls caused varying exposure to and resilience against infectious diseases. Gender roles within the household, such as girls caring for the sick, along with female working conditions and disparities in accident risks, have been cited as factors influencing divergent health outcomes between boys and girls. [212] Shorter highlighted the physically demanding labour conditions in agriculture, while Devos argued that the textile industry, with significant female employment rates, posed particular hazards due to unhygienic and poorly ventilated workshops, as well as long and intense working days.[213] Tabutin and Willems, however, concluded that these hazardous working conditions primarily affected older age groups.[214]

Nineteenth-century Maastricht was a prototypical industrializing city that generated new job opportunities and conditions for people of various ages, including both man and women. However, examining the impact of working conditions on health outcomes of children comes with challenges. Burial records in Maastricht rarely noted the professions of deceased children. Only 1.7 per cent of boys aged 1-15 and 0.7 per cent of girls within the same age group had their profession recorded at death between 1864-1930. Additional sources, like a 1841 survey in Regout's factories, counted 12 employed children under 10 years (11 boys and 1 girl), and 165 boys and 46 girls aged 10-15.[215] The survey counts show that low numbers of children were employed and more boys compared to girls worked in Regout's factories, indicating that working conditions in these factories could not have caused the higher girls' mortality rates. However, it is important to note that employment numbers for other Maastricht factories and workplaces, especially smaller ones, remain unknown.

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[211] Murkens, *Unequal pathways to the grave?,* 115.
[212] Tabutin and Willems, 'Differential Mortality', 48; Pinnelli and Mancini, 'Mortality Peaks in Italy', 355; Puschmann, 'Discriminatie met dodelijke gevolgen', 142; Van Poppel, 'De statistieke ontleding van de dooden', 16.
[213] Shorter, *Women's Bodies,* 237-239; Devos, 'Te jong om te sterven', 71.
[214] Tabutin and Willems, 'Differential Mortality', 48.
[215] Maenen, *Petrus Regout,* 293.

Smaller enterprises might have hired more children and women, given their lower wages compared to adult men.[216]

In 1874, the prohibition of employing children under 12 years old in factories led to a decline in child labour in Regout's enterprises in Maastricht (see Table 5). The compulsory education law in 1901 likely further reduced the number of employed children, such as those in workshops or households, as they were required to attend school between the ages of 6 and 12. As patterns of excess female mortality persisted until 1930, working conditions might not have been the primary cause of increased mortality risks for girls, at least for children below 12 years. Henry also emphasized that potential differences in working conditions did not explain why more girls between five and nine years old died, as children from these ages were less likely to be employed.[217] Furthermore, the personnel counts in Table 5 indicate that more boys than girls aged 12-15 were employed. While the total employment figures for girls and boys remain unknown, it appears that boys in Maastricht were more exposed to hazardous working conditions than boys in larger industrial factories, like Tabutin and Willems also argued.[218] To fully understand employment rates among children in Maastricht during the nineteenth and twentieth centuries, personnel overviews of smaller factories or workshops should be examined. Only then can working conditions be decisively ruled out as an explanatory factor for elevated mortality rates among girls.

Table 5 Personnel counts in Regout's factories, Maastricht 1874-1877.

Age	May 1874 Male	May 1874 Female	December 1874 Male	December 1874 Female	December 1875 Male	December 1875 Female	December 1876 Male	December 1876 Female	December 1877 Male	December 1877 Female
<10	11	-	-	-	-	-	-	-	-	-
10-12	63	13	52	12	15	2	-	-	-	-
12-15	176	96	196	117	234	124	219	115	229	133

Source: A.J.Fr. Maenen, Petrus Regout 1801-1878, Een bijdrage tot de sociaal-economische geschiedenis van Maastricht (Maastricht 1959) 328-329.

Another type of work not likely to be registered in burial records or personnel counts is household work, to which girls were more often assigned to. These tasks caused girls to easier contract infectious diseases, as they spent more time in poorly ventilated houses and they took care of the sick.[219] Due to the lack of sources, researching differences in household roles between boys and girls is challenging. Van Poppel suggested that certain accidents might be associated with household work. For example, in England, accidents related to burns and boiling water were more common among girls, indicating that they were more exposed to fire and hot substances during activities like cooking. Van Poppel also highlighted differences in clothing, with girls wearing stiffer and longer clothes that could hinder their ability to escape dangers and increase the risk of

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[216] Ibid, 261.
[217] Henry, 'Men's and Women's Mortality', 195.
[218] Tabutin and Willems, 'Differential Mortality', 48.
[219] Tabutin and Willems, 'Differential Mortality', 48; Puschmann, 'Discriminatie met dodelijke gevolgen', 142.

severe accidents.[220] Therefore, a higher incidence of burning wounds would be expected among girls compared to boys. While chapter 2 showed that boys died more often a violent death, the exact causes-of-death have not been thoroughly explored. Table 6 presents the registered causes-of-death for children (aged 1-15) who died from non-natural causes in Maastricht during the researched period.

Table 6 Categories of non-natural causes of death, all years combined, children aged 1-15 years, Maastricht 1864-1930.

Cause of death	Male		Female		Total	
	N	*%*	*N*	*%*	*N*	*%*
Burning wounds	74	*31.5*	47	*64.4*	121	*39.3*
Drowning	89	*37.9*	8	*11.0*	97	*31.5*
Brain trauma	30	*12.8*	10	*13.7*	40	*13.0*
Traffic accidents	15	*6.4*	3	*4.1*	18	*5.8*
Accidents	9	*3.8*	2	*2.7*	11	*3.6*
Inflicted violence	7	*3.0*	3	*4.1*	10	*3.2*
Fractures	7	*3.0*	-	-	7	*2.3*
Other	4	*1.7*	-	-	4	*1.3*
Total	**235**	*100*	**73**	*100*	**308**	*100*

Source: Maastricht Death and Disease Database.

The results from table 6 suggest underlying differences in behaviour. Boys died more frequently and from a wider range of non-natural causes, possibly indicating more risk-taking or reckless behaviour compared to girls. For example, 89 boys drowned compared to 8 girls, with most of these incidents occurring during summer months (see Annex IV), suggesting that boys may have been more likely to swim or take greater risks while swimming, leading to more fatalities. Among all violent deaths, burning wounds accounted for a significant portion for both boys and girls. While girls generally died less frequently from violent causes, it is noteworthy that 65 per cent of all violent deaths among them was due to burning wounds. This high percentage among girls may indeed suggest their involvement in household tasks, exposing them to specific accident risks. Meanwhile, one-third of violent deaths among boys was caused by burning wounds. The previous section suggested that boys might have been more employed in industrial factories compared to girls, which could have caused these burning wounds among boys. However, these assumptions cannot be verified, as only 1 out of 121 children who died from burning wounds had a recorded profession (1 girl identified as a maid). Therefore, it remains unclear whether boys sustained burning wounds in different contexts compared to girls.

Others argued that the socioeconomic status of the family was an important ultimate determinant in shaping gender roles of boys and girls, as differences in this status led to varying types and levels of household tasks and other assigned work. For example, Klasen and Wink suggested that among the poorest groups, there were fewer cultural constraints like gendered division of labour, and women had greater economic

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[220] Van Poppel, 'Statistieke ontleding van de dooden', 16.

independence compared to slightly wealthier but still poor groups, as the poorest groups required all available help or labour power to survive.[221] Similarly, Van Poppel, Schellekens and Walhout observed that in nineteenth-century Netherlands, daughters of unskilled labourers had higher mortality risks compared to daughters of skilled labourers and farmers. They attributed these higher mortality risks across different social classes to differences in living and working conditions, including inadequate housing, which caused girls in specific socioeconomic classes to be at higher risks of death.[222]

To investigate how socioeconomic status influenced mortality differences between boys and girls, the professions of the parents were examined. As explained in the data and methodology section, this study coded the parents' professions into a simplified HISCLASS-scheme, consisting of six socioeconomic classes. Table 7 presents the average male-to-female mortality ratios that were derived from the annual male and female death rates of each socioeconomic class.

Table 7 Male-to-female mortality ratios per socioeconomic class, all years combined, children aged 1-15, Maastricht 1864-1930.

	All ages 1-15 years			Young children 1-5 years			Children 6-10 years			Teenagers 11-15 years		
	Ratio	*N*	*%*	*Ratio*	*N*	*%*	*Ratio*	*N*	*%*	*Ratio*	*N*	*%*
Father												
Managers and professionals	103.7	440	4.6	**98.5**	308	4.1	111.4	89	7.8	119.9	43	5.6
Clericals, sales personnel and foremen	**96.5**	820	8.7	**95.2**	645	8.5	**99.3**	102	8.9	**93.6**	73	9.4
Medium and lower skilled workers	101.2	4072	43.0	**99.7**	3372	44.6	100.3	440	38.4	103.0	260	33.6
Lower and unskilled farm workers	114.2	204	2.2	113.4	147	1.9	110.1	28	2.4	118.5	29	3.7
Unskilled unspecified workers	103.0	3012	31.8	101.0	2501	33.1	109.4	324	28.3	**98.2**	187	24.2
Unknown	**96.3**	929	9.8	**97.2**	585	7.7	**98.2**	162	14.1	**87.6**	182	23.5
Total		**9477**	*100*		**7558**	*100*		**1145**	*100*		**774**	*100*
Mother												
Managers and professionals	128.8	28	0.3	116.3	15	0.2	113.8	7	0.6	167.6	6	0.8
Clericals, sales personnel and foremen	**95.6**	96	1.0	**96.8**	69	0.9	**75.9**	14	1.2	**99.9**	13	1.7
Medium and lower skilled workers	103.5	182	1.9	105.4	152	2.0	**69.1**	20	1.7	122.5	10	1.3
Lower and unskilled farm workers	135.2	52	0.5	143.9	30	0.4	102.4	8	0.7	131.9	14	1.8
Unskilled unspecified workers	102.6	1051	11.1	101.3	864	11.4	106.3	123	10.7	**93.3**	64	8.3
Unknown	100.8	8068	85.1	**99.2**	6428	85.0	104.3	973	85.0	**97.4**	667	86.2
Total		**9477**	*100*		**7558**	*100*		**1145**	*100*		**774**	*100*

Ratios above 100 indicate excess male mortality, ratios below 100 indicate excess female mortality.
Source: Maastricht Death and Disease Database; Centraal Bureau voor de Statistiek. Volkstelling 1859-1930.

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[221] Klasen and Wink, 'Missing women', 282.
[222] Van Poppel, Schellekens and Walhout, 'Oversterfte van jonge meisjes', 65.

The results in Table 7 reveal that in the 'Clericals, sales personnel, and foremen' as well as the 'Unknown' socioeconomic classes, higher female mortality ratios occurred across all age groups. These results diverge from earlier studies, which often associated excess female mortality with farming, agricultural and/or poorer families, mostly found in rural areas. The absence of excess female mortality rates in Maastricht among farmers suggest that excess female mortality may not be universally linked to farmer families, or that urban hazards differ from those in rural regions, potentially shifting hazards for girls to other socioeconomic classes in both contexts. Furthermore, among unskilled labourers, excess female mortality rates emerged in the oldest age category (11-15 years), suggesting that girls from this socioeconomic class might have been more frequently expected to contribute to the household compared to other social classes, potentially leading to higher mortality rates. Additionally, as Van Poppel, Schellekens and Walhout argued, poorer life conditions among this social class could have also contributed to the higher female mortality rates.[223]

Table 7 shows that the profession of the mother was not recorded in the majority of burial records (85.1 per cent). Female labour participation has been identified as an ultimate determinant contributing to improved bargaining and social positions for women, which could potentially reduce gender biases.[224] The high proportion of mothers without a recorded profession could indicate low female participation rates among mothers, possibly increasing gender biases and contributing to higher mortality rates among girls. However, it is also plausible that mothers worked in unpaid roles such as household work, or their occupations were simply not documented. Future research should further investigate mothers' professions to ascertain whether they were indeed unemployed or if additional vital registration sources, like marriage records, could provide more comprehensive employment information. Lastly, the category 'Unknown' includes also parents that were deceased or absent. The higher rates of excess female mortality in this category may therefore also indicate that the absence of a parent increased mortality risks for girls compared to boys. The role of parental presence or absence will be explored further in the following section on gender-specific discrimination.

The rates of excess female mortality among the upper working classes could imply that scarcity placed a smaller role in increased mortality risks for girls in Maastricht, as these classes possessed more assets compared to lower social classes. Possibly, gender biases might have been more pronounced in these higher classes as there were more assets to be divided, which could exclusively be inherited by boys, potentially leading to son preferences. However, it is also plausible that instead of social position and specific gender roles across different socioeconomic classes, another mechanism was at play. Rijpma et al. suggested that the spread of the Spanish flu in 1918 could have been influenced by occupational characteristics rather than socioeconomic status, similar to the increased infection risks faced by healthcare professionals and other close-contact professions during the Covid-19 outbreaks. To investigate the influence of occupational characteristics, they designed an occupational index based on exposure to infectious diseases, considering factors like indoor

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[223] Van Poppel, Schellekens and Walhout, 'Oversterfte van jonge meisjes', 65.
[224] Puschmann, 'Discriminatie met dodelijke gevolgen', 158-159; Klasen and Wink, 'Missing Women', 282.

work and close-contact during work.[225] Given the significance of infectious diseases in patterns of excess female mortality, parental work characteristics might have been more influential on health outcomes compared to family socioeconomic status.

As explained in the data and methodology section, the professions of the parents were coded into the index of occupational exposure to infectious diseases and four types of occupations were distinguished: (1) *none*, for occupations not indoors and without close-contact, (2) *indoors,* for indoor professions without close-contact, (3) *contact,* for professions involving close-contact but not indoors, and (4) *both,* for professions that were indoors and involved close-contact. The fifth category, *unknown,* included professions with unknown characteristics and parents without a recorded profession. After coding, annual male and female death rates were computed per category, distinguishing between infectious and non-infectious diseases as the cause of death of the child. Table 8 presents the average male-to-female mortality ratios based on the annual death rate.

Table 8 Male-to-female mortality ratios per type of occupational exposure and type of death, all years combined, children aged 1-15, Maastricht 1864-1930.

	Infectious diseases			Non-infectious diseases			Unknown		
Profession	*Ratio*	*N*	*%*	*Ratio*	*N*	*%*	*Ratio*	*N*	*%*
Father									
None	104.5	940	*11.8*	125.3	134	*12.9*	122.5	43	*9.5*
Indoors	**97.6**	2603	*32.6*	119.9	310	*29.8*	**92.8**	133	*29.3*
Contact	100.6	2316	*29.0*	117.3	288	*27.7*	110.4	125	*27.5*
Both	**98.8**	1386	*17.4*	115.6	188	*18.1*	**96.3**	72	*15.9*
Unknown	**91.3**	737	*9.2*	123.3	121	*11.6*	105.1	81	*17.8*
Total		**7982**	*100*		**1041**	*100*		**454**	*100*
Mother									
None	138.5	44	*0.6*	128.8	12	*1.2*	-	-	-
Indoors	**95.9**	145	*1.8*	173.2	7	*0.7*	146.6	8	*1.8*
Contact	**99.0**	850	*10.6*	128.7	114	*11.0*	109.7	53	*11.7*
Both	**97.9**	131	*1.6*	139.1	26	*2.5*	119.9	8	*1.8*
Unknown	**98.7**	6812	*85.3*	117.0	882	*84.7*	101.0	385	*84.8*
Total		**7982**	*100*		**1041**	*100*		**454**	*100*

Ratios above 100 indicate excess male mortality, ratios below 100 indicate excess female mortality.
Source: Maastricht Death and Disease Database, Centraal Bureau voor de Statistiek. Volkstelling 1859-1930, Ruben Schalk, Richard Zijdeman, Auke Rijpma, Ingrid van Dijk and Rick Mourits, 'HSNDB index for exposure to infectious disease', https://hdl.handle.net/10622/TFG1DQ, IISH Data Collection, V1.

The results from Table 8 show that among all types of professions, both among fathers and mothers, boys had higher mortality rates than girls from non-infectious diseases. Among the infectious diseases, girls also had better survival chances than boys if either parent worked in a profession that was not indoors and

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[225] Auke Rijpma, Ingrid van Dijk, Ruben Schalk, Richard L. Zijdeman, Rick J. Mourits, ' Unequal excess mortality during the Spanish Flu pandemic in the Netherlands', *Economics and Human Biology* 47:1 (2022) 1-17, there 2; Ruben Schalk, Richard Zijdeman, Auke Rijpma, Ingrid van Dijk and Rick Mourits, 'HSNDB index for exposure to infectious disease', https://hdl.handle.net/10622/TFG1DQ, IISH Data Collection, V1

contained no close-contact, with ratios of 104.5 for fathers and 138.5 for mothers. However, excess female mortality ratios were observed among children who died from infectious diseases and whose parents worked in professions with exposure to infectious diseases. Specifically, if the father worked in an indoors profession or both indoors and close-contact profession, their daughters had a higher risk of dying from infectious diseases compared to their sons. Similarly, among mothers, all types of occupations involving exposure to infectious diseases led to higher mortality rates from infectious diseases among girls compared to boys.

Differences in mortality rates related to parents' profession exposure to infectious diseases may suggest variations in the exposure to infections among daughters and sons or differences between their overall health status. Parents exposed to infectious disease at work could potentially bring home these infections, affecting other family members. However, the results indicate an unequal distribution of infections (or at least mortality from these infections) among sons and daughters. According to Peter Aaby, a crucial factor for some infectious diseases could be the distinction between index cases and secondary cases of infection. Index cases are the first infected individuals in a family, contracting the disease outside the home, while secondary cases are family members infected inside the home through the index case. Secondary cases have been exposed longer and more intensely to the disease, which causes more severe infections and higher mortality rates compared to the index cases.[226] The higher mortality ratios of girls among parents with higher exposure to infectious diseases could suggest that girls were more likely to stay at home and/or care for sick parents, which increased their risk of contracting the disease and increased the risk of a more severe infection compared to boys.

7.4 Gender discrimination

The last group of potential explanations focuses on gender-specific discrimination, caused by perceived lower value of girls, leading to unequal treatment of sons and daughters in families.[227] Devos argued that women received a lower quantity and quality of food compared to men, contributing to a poorer health status among women and increasing their susceptibility to diseases.[228] Others, like Klasen and Wink, emphasized inequalities in care and healthcare as significant factors causing differences in the prevention and treatment of illnesses between boys and girls.[229] Puschmann suggested, for instance, that girls were less frequently vaccinated than boys until the early twentieth century, while Wall proposed that parents in England may have prioritized personal hygiene of their sons.[230] Van Poppel, meanwhile, suggested that girls were less frequently

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[226] Peter Aaby, 'Are Men Weaker or Do Their Sisters Talk Too Much? Sex Differences in Childhood Mortality and the Construction of 'Biological' Differences', in: Alaka Malwade Basu and ibid (eds.), *The Methods and Uses of Anthropological Demography* (Oxford 1998) 223-245, there 223; Murkens, *Unequal pathways to the grave?*, 132.
Perner et al., 'Gendered Mortality of Children and Adolescents', 682; Devos, 'Te jong om te sterven', 70.
[228] Ibid, 71.
[229] Klasen and Wink, 'Missing Women', 281.
[230] Puschmann, 'Discriminatie met dodelijke gevolgen', 142; Wall, 'Inferring Differential Neglect of Females', 130.

treated in hospitals compared to boys, contributing to higher mortality rates among girls.[231] Additionally, this gender bias could have been influenced by various ultimate determinants, such as improvements in the social position of girls reducing discriminatory practices, or changes in family composition like parental loss, which might have accentuated son preferences to the detriment of girls.[232]

Gender-specific discrimination is often associated to the social position of women, which can be linked to female education rates. During the nineteenth and early twentieth centuries, two policies were implemented in the Netherlands to increase education levels among children: the 1874 policy prohibiting employment of children under 12 in factories, and the 1901 law mandating compulsory education for children aged 6-12.[233] Education rates provide insights not only into inequalities between boys and girls in access to school, but could also function as a proxy for smallpox vaccination rates. Since 1823, Dutch children were required to have proof of smallpox vaccination, known as a '*pokkenbriefje*', to attend school.[234] Differences in school attendance rates between boys and girls can therefore indicate disparities in smallpox vaccination rates, shedding light on potential gender-specific discrimination. However, it should be noted that school attendance rates do not provide insights into vaccination rates among children who did not attend school, nor do they reflect vaccination rates for infectious diseases other than smallpox.

The municipal registers ('*Verslagen van den toestand der gemeente Maastricht*') provided data on primary school attendance for boys and girls between 1864 and 1920. The school attendance rates in Figure 10 were calculated based on the number of recorded children attending school and the number of boys and girls between 6 and 12 years in the Maastricht population in the concerned year. During the twentieth century, the number of children attending school exceeded the population estimates, suggesting a potential underestimation of the population in certain years between censuses. Another possibility is that children from neighbouring municipalities attended primary schools in Maastricht, but were not included in the population counts of the Maastricht municipality.

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[231] Van Poppel, 'De statistieke ontleding van de dooden', 17.

[232] Jansz and van Loosbroek, 'Nieuwe literatuur over de eerste feministische golf', 13; Quanjer et al., 'Short Lives', 255-279.

[233] Ubachs, *Tweeduizend jaar Maastricht,* 257.

[234] Rutten, *De vreselijkste aller harpijen,* 266.

Figure 10 Primary school attendance rates boys and girls 6-12 years, Maastricht 1864-1920.
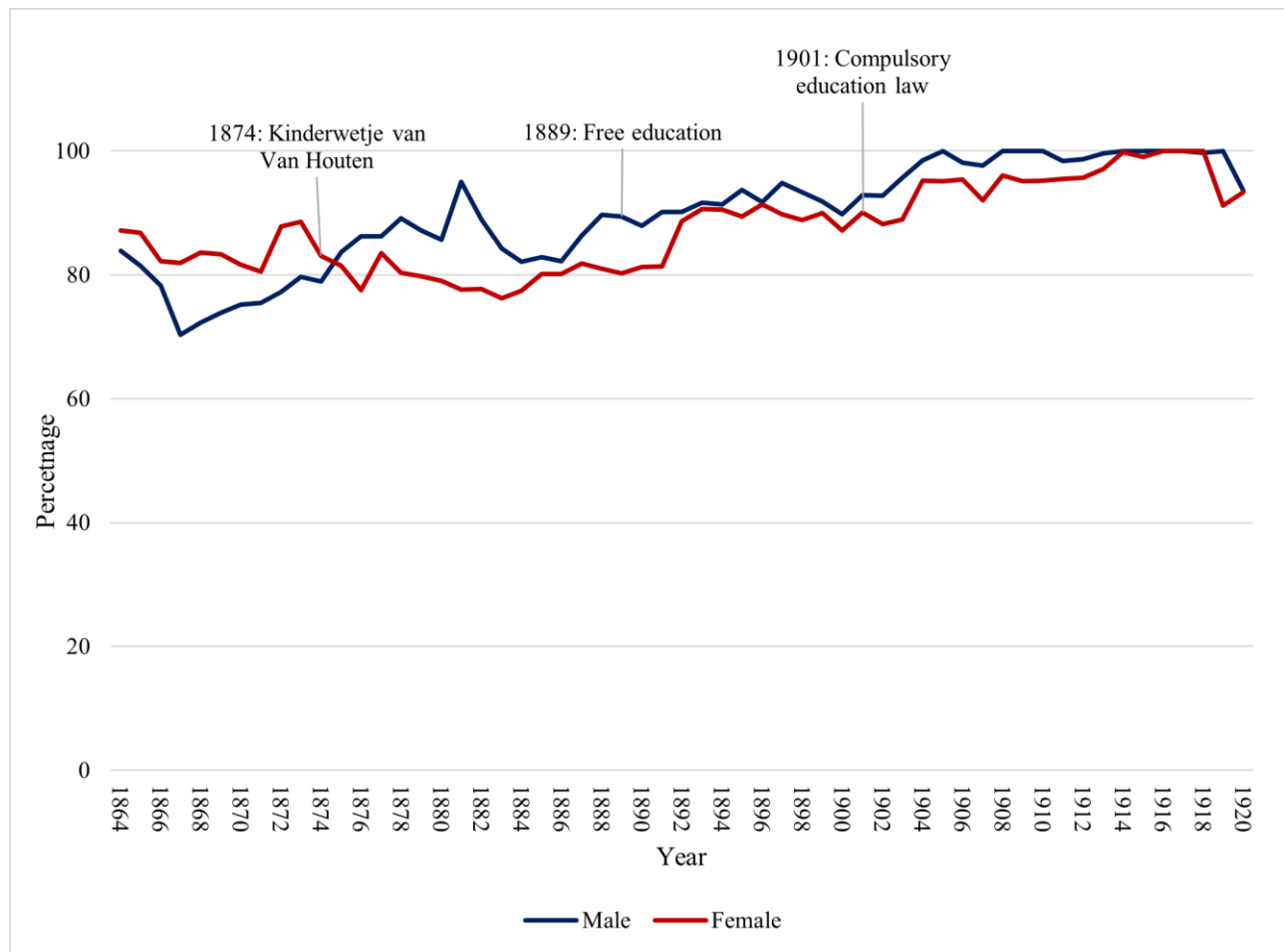
Source: Verslag van den toestand der gemeente Maastricht, 1864-1920; Centraal Bureau voor de Statistiek. Volkstelling 1859-1920.

The results in Figure 10 demonstrate high attendance rates in Maastricht. Initially, more girls than boys aged 6 to 12 attended primary school, but this changed in 1874, when factory employment for children was prohibited. Subsequently, there was a surge in boys attending school, indicating that in the years before, boys were more often employed in Maastricht factories compared to girls. From 1892 onwards, attendance rates for boys and girls began to converge, with almost all children attending school by the early twentieth century. However, despite these high rates of female education, excess female mortality persisted throughout the studied period. Ubachs argued that this basic level of education was insufficient for girls to achieve emancipation, as the majority of them ended up in the household.[235] The high primary school attendance rates of girls were therefore presumably insufficient to achieve equality in health outcomes between boys and girls in Maastricht.

The school attendance rates in Figure 10 suggest that smallpox vaccination rates were relatively equal for boys and girls in Maastricht. Initially, higher female school attendance rates could even indicate higher vaccination rates among girls, although it remains unknown if boys who did not attend school were vaccinated.

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[235] Ubachs, *Tweeduizend jaar Maastricht*, 155.

By the end of the researched period, both girls' and boys' school attendance rates reached (almost) 100 per cent, indicating minimal or no differences in smallpox vaccination rates. This equality in vaccination rates, however, did not prevent girls from dying more frequently than boys in Maastricht, also because the disease was by the end of the nineteenth century no longer predominant in the city's mortality pattern.[236] Future research could explore other sources, such as reports of provincial committees documenting vaccination rates of doctors, to investigate potential inequalities in vaccination rates of diseases that were more dominant during the researched period in Maastricht, in order to examine if any inequalities in access to healthcare might have contributed to the excess female mortality pattern in the city.

Scarcity is another ultimate determinant often associated with gender-specific discrimination, as son preferences may become more pronounced during resource shortages. Excess female mortality has often been linked to poorer families where resources are scarce. However, the results from the previous section showed that among the poorest families in Maastricht, excess female mortality only occurred among children aged 11-15 years, suggesting that general resource scarcity may not have influenced treatment differences between sons and daughters in the city. Nevertheless, other forms of scarcity, caused by sudden events like economic crises or wars, could have intensified son preferences.[237] Beltrán Tapia and Szoltysek argued that economic shocks could lead to discriminatory practices by parents, affecting children's' health outcomes.[238] Rutten also observed a decline in the health status of conscript cohorts during the economic crisis in Limburg in the 1880s, indicated by average body lengths that were 1 centimetre shorter compared to previous cohorts. Rutten attributed this to general resource scarcity during the crisis, or by an increase of work assigned to the children to contribute more to the family's income during these crisis years.[239]

To investigate the potential link between economic developments and child mortality patterns, the relationship between the death rate of children and wheat prices in Maastricht will be analysed. If economic shocks influenced discriminatory practices such as unequal food distributions among sons and daughters, it is expected that girls' mortality rates will correlate more closely with wheat prices than those of boys. Annual wheat prices were based on the average of June and December prices until 1914. After this year, the prices from the province of Limburg were used due to the unavailability of Maastricht's prices. Between 1864 and 1914, Limburg and Maastricht prices showed similar trends (see Annex V), indicating the reliability of using Limburg prices from 1914 onward.[240] In addition to male and female death rate of children aged 1-15 (left axis) and annual wheat prices (right axis), Figure 11 also includes prices in t+2 (right axis) as reductions in food availability during an economic crisis may not immediately result in children's deaths but could affect their general health status, contributing to mortality in subsequent years.

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[236] Murkens *Unequal pathways to the grave?,* 113.
[237] Beltrán Tapia and Szoltysek, 'Missing girls', 363.
[238] Ibid, 363.
[239] Rutten, 'De levensstandaard in Limburg', there 151-152.
[240] *W. Tijms, 'Prijzen van Granen en Peulvruchten te Arnhem, Breda, Deventer, 's-Hertogenbosch, Kampen, Koevorden, Maastricht, Nijmegen', Historia Agriculturae (Groningen 1983) 71-73, 79-80, 89-90.*

Figure 11 Annual wheat prices and male and female death rates, children aged 1-15, Maastricht 1864-1930.
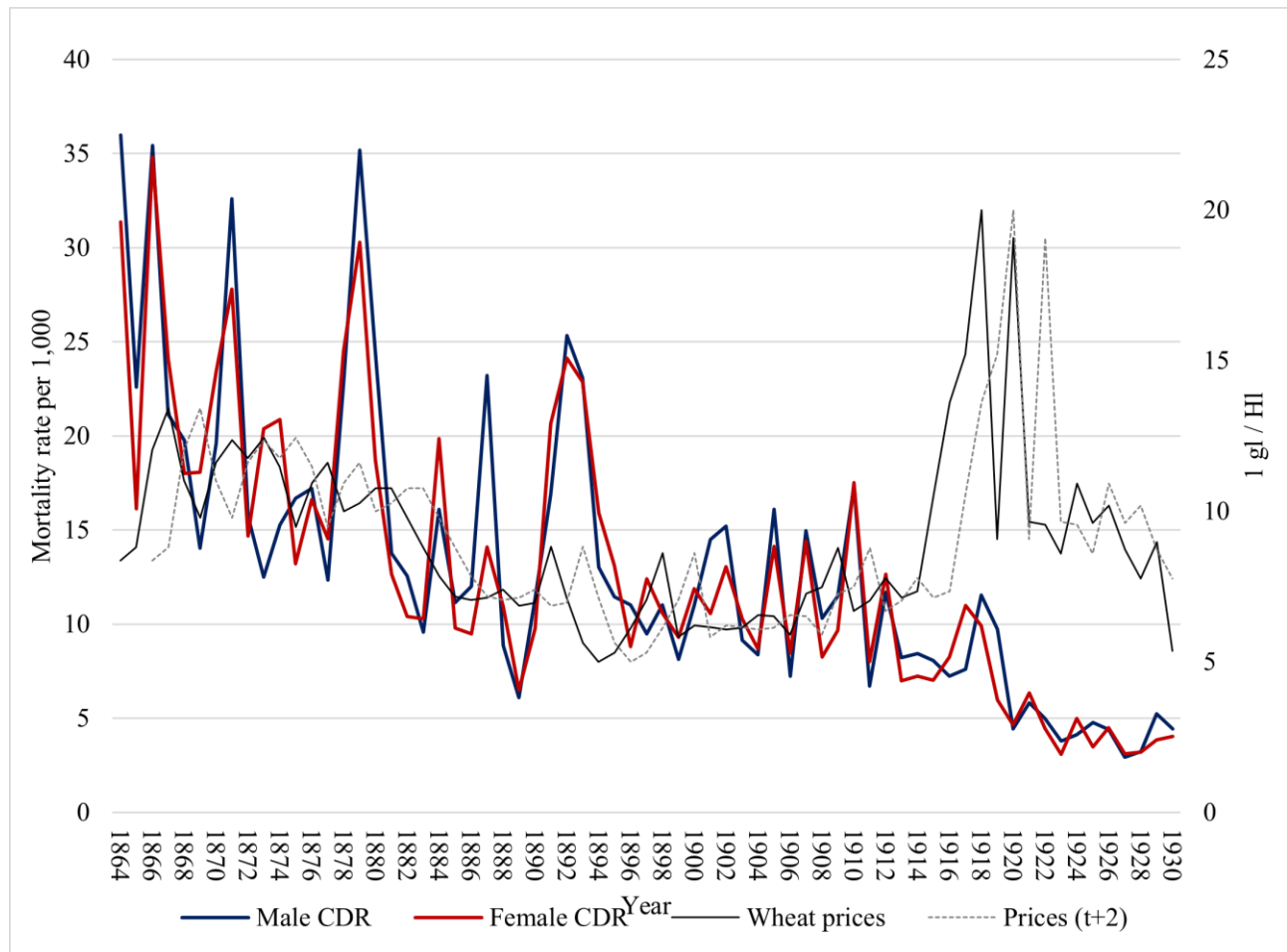
Source: Maastricht Death and Disease Database; Centraal Bureau voor de Statistiek. Volkstelling 1859-1930; W. Tijms, 'Prijzen van Granen en Peulvruchten te Arnhem, Breda, Deventer, 's-Hertogenbosch, Kampen, Koevorden, Maastricht, Nijmegen', Historia Agriculturae (Groningen 1983) 71-73, 79-80, 89-90.

Figure 11 indicates a positive relationship between mortality and economic developments at the beginning of the researched period: mortality rates increased when wheat prices increased. However, by the early twentieth century, the general standard of living appeared to have improved to such extent that mortality rates and food prices were no longer as closely linked as before, which indicates that other mechanisms, like social welfare and a general improvement of people's health, prevented economic developments to directly affect health outcomes. Mortality and wheat prices continued to have some association, as they both increased during the First World War, but this relationship was not as straightforward as it was at the beginning of the researched period.

Due to the frequent mortality peaks, it is challenging to determine whether girls' mortality rates correlated more with wheat prices than boys. To explore this relationship further, linear regressions were conducted with the death rate as the dependent and wheat prices as the independent variable. Separate models were created for boys and girls across different age categories and for wheat prices in t=0, t+1 and t+2. The correlation coefficient R represents the correlation between the dependent and independent variable, while R^2 indicates the proportion of mortality variation (dependent variable) explained by wheat prices (independent

variable). A 95 per cent confidence interval was used, with p-values <0.05 considered statistically significant. Effect sizes (indicated by *B*) were interpreted only if the model was statistically significant. Table 9 displays the models examining the relationship between the death rate of boys and girls across different age groups and wheat prices across all years combined.

Table 9 Linear regression mortality rates and wheat prices per period, children aged 1-15, Maastricht 1864-1930.

Age category	Sex	Year prices	*R*	*R²*	*P model*	B	*p*
1-5	*Male*	t=0	0.48	.002	.705		
		t+1	.031	.001	.807		
		t+2	.025	.001	.844		
	Female	t=0	.081	.007	.514		
		t+1	.044	.002	.726		
		t+2	.008	.000	.947		
6-10	*Male*	t=0	.274	.075	.025	.361	.025
		t+1	.241	.058	.052		
		t+2	.097	.009	.440		
	Female	t=0	.212	.045	.086		
		t+1	.114	.013	.360		
		t+2	.092	.009	.465		
11-15	*Male*	t=0	.134	.018	.281		
		t+1	.003	.000	.981		
		t+2	.024	.001	.847		
	Female	t=0	.464	.215	<.001	.290	<.001
		t+1	.232	.054	.061		
		t+2	.074	.006	.557		

Source: Maastricht Death and Disease Database; Centraal Bureau voor de Statistiek. Volkstelling 1859-1930; W. Tijms, 'Prijzen van Granen en Peulvruchten te Arnhem, Breda, Deventer, 's-Hertogenbosch, Kampen, Koevorden, Maastricht, Nijmegen', Historia Agriculturae (Groningen 1983) 71-73, 79-80, 89-90.

Table 9 reveals that most models were not significant, suggesting that mortality rates during the studied period were not influenced by wheat prices. However, it is also possible that the opposite trends observed in Figure 11, with a positive relationship at the beginning of the research period compared to a convergent trend at the end, may have led to few significant models for all studied years combined. The results of girls aged 11-15 show a positive relationship between prices and mortality: if prices increased, mortality among girls aged 11-15 increased. This suggests that among this age group, economic shocks may have caused differences in the food allocation between boys and girls, as the boys' mortality rates did not show a relationship. The significant relationship for boys' mortality rates between 6 and 10 years and wheat prices is unexpected, as literature would predict a relationship for girls instead. This could indicate the inherently weaker position of boys, becoming more pronounced in times of scarcity when prices increased. In this scenario, food may have been distributed equally among boys and girls in scarce times, but in 'normal' times boys might have received more food compared to girls, concealing their biologically disadvantageous position.

Based on Rutten's, as well as Klasen and Wink's findings, a stronger association between mortality rates and wheat prices will be expected during the 1880s when Maastricht, like the rest of Limburg, faced an economic crisis. To explore this relationship further, separate linear regressions were conducted for each decade. However, the majority of periods yielded no significant results. Table 10 presents only the significant findings from these models that examined the relationship between mortality rates of boys and girls and wheat prices (t=0, t+1, and t+2) in separate decades. The complete models can be found in Annex VI.

Table 10 Linear regression mortality rates and wheat prices per period, children aged 1-15, Maastricht 1864-1930.

Year	Age category	Sex	Price	R	R²	*p model*	*B*	*p*
1864-1879	6-10	Female	t+1	.527	.278	.044	-1.573	.044
1864-1879	6-10	Female	t+2	.538	.289	.047	-1.545	.047
1864-1879	11-15	Female	t+2	.628	.394	.016	-1.124	.016
1891-1900	6-10	Female	t+2	.726	.528	.017	1.069	.017
1891-1900	11-15	Female	t=0	.695	.482	.026	.783	.026
1911-1920	11-15	Female	t=0	.666	.443	.036	.176	.036

Source: Maastricht Death and Disease Database; Centraal Bureau voor de Statistiek. Volkstelling 1859-1930; W. Tijms, 'Prijzen van Granen en Peulvruchten te Arnhem, Breda, Deventer, 's-Hertogenbosch, Kampen, Koevorden, Maastricht, Nijmegen', Historia Agriculturae (Groningen 1983) 71-73, 79-80, 89-90.

The results in Table 10 indicate that the relationship between wheat prices and boys' mortality between 6 and 10 years, observed over the entire research period, disappears when examined across separata decades, which could indicate that several underlying mechanisms not included in the model were at play. Contrary to expectations based on previous research, there was no significant relationship between the economic crisis of the 1880s and children's mortality. Interestingly, during these crisis years, none of the models yielded significant results, suggesting no link between economic developments and mortality during that period. Possibly, the decline in health status Rutten observed did not translate to the mortality pattern, although increased mortality, especially among girls, would be expected among children if their health status deteriorated.

Moreover, Table 10 reveals an unexpected negative relationship between girls' mortality rates and wheat prices during the period 1864-1879. In this period, mortality decreased among girls as wheat prices increased, contrary to expectations and the results of Table 8. This negative relationship for girls aged 6 to 10 years may explain the lack of significant effects in this age category when considering all years combined, as opposite trends occurred in this age group across different decades. Girls aged 11 to 15 years exhibited a strong relationship between mortality rates and wheat prices, particularly in the period 1891-1900 and also in 1911-1920, suggesting that around the turn of the twentieth century, teenage girls' mortality was affected more by economic developments compared to other age groups and compared to boys, which could indicate differences in terms of food allocation between them.

The regressions in this study offered an initial investigation into the relationship between food prices and children's mortality rates in Maastricht. However, these models lacked additional variables that could help explain the observed relationships. Future research could include variables like adult mortality, or more

specifically parental loss, particularly after high-mortality years among adults, as the positive relations between mortality and prices in t+2 might also be attributed to reduced care following epidemic outbreaks that led to parental loss in some families. Additionally, incorporating causes-of-death would be valuable in further understanding the connections between economic crisis, epidemic outbreaks, increased susceptibility due to malnutrition, and mortality rates.

Based on the findings in this preliminary exploration, it appears that major economic shocks did not result in elevated female mortality, indicating that the food distribution in families may not have been impacted by food prices. However, it remains plausible that food was consistently distributed unequally between boys and girls, leading to increased susceptibility among girls throughout the entire studied period rather than during specific decades with economic shocks.

Lastly, scarcity has been linked to the family composition. Quanjer et al. noted, for instance, higher mortality among girls aged 5 to 12 in the Netherlands after parental loss. They suggested that the reduction in resources caused by parental loss triggered unequal resource allocation that prioritized boys over girls.[241] As explained in the data and methodology section, four types of family compositions were constructed based on the information of the burial records: (1) a family with both parents alive, (2) with a deceased or absent father but alive mother, (3) a family with the father alive but the mother absent or deceased and lastly, (4) a family with both parents absent or deceased. Table 11 shows the male-to-female mortality ratios of the four different types of family compositions that have been distinguished and across different age categories. These ratios are the averages of the annual male and female death rate per family type.

Table 11 Male-to-female mortality ratios per family composition, all years combined, children aged 1-15, Maastricht 1864-1930.

Family composition	All ages 1-15 years			Young children 1-5 years			Children 6-10 years			Teenagers 11-15 years		
	Ratio	*N*	*%*	*Ratio*	*N*	*%*	*Ratio*	*N*	*%*	*Ratio*	*N*	*%*
Both alive	101.9	8433	89.0	100.0	6948	91.9	104.6	949	82.9	104.5	536	69.3
Father deceased or absent	**94.3**	215	2.3	**93.3**	197	2.6	100.6	10	0.9	**76.4**	8	1.0
Mother deceased or absent	**97.4**	291	3.1	**99.6**	148	2.0	104.8	61	5.3	**85.5**	82	10.6
Both deceased or absent	**95.6**	538	5.7	**98.7**	265	3.5	**96.3**	125	10.9	**87.5**	148	19.1
Total		**9477**			**7558**			**1145**			**774**	

Ratios above 100 indicate excess male mortality, ratios below 100 indicate excess female mortality.
Source: Maastricht Death and Disease Database; Centraal Bureau voor de Statistiek. Volkstelling 1859-1930.

The results in Table 11 indicate that excess female mortality was absent in families where both parents were alive. However, when one or both parents were deceased or absent, girls exhibited higher mortality rates compared to boys, like Quanjer et al. observed. This suggests that girls with both parents alive received relatively better care than girls that experienced parental loss. Among children between 1 and 5 years, the

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[241] Quanjer et al., 'Short Lives', 255-279.

reduction in resources could have caused a more unequal distribution of food and (health)care between daughters and sons, which could have decreased the girls' survival chances. Meanwhile, among older girls, next to potential inequalities in the allocation of resources and/or care, the loss of one or both parents may have led to increased household responsibilities, replacing the role of a parent, which potentially elevated their exposure and subsequently mortality rates.

Quanjer et al. argued that the death of a mother is more harmful for children than the death of the father, as the loss of a mother primarily results in a decline in household care, while the loss of a father mainly affects economic resources.[242] However, Table 11 reveals greater disparities in mortality rates between boys and girls in families without the father compared to those without the mother. Both scenarios of parental loss or absence led to higher mortality risks for girls compared to boys and girls without a father had even higher mortality risks compared to girls without a mother. Single mothers may have faced greater scarcity if they needed to work for family income. Female wages were lower, resulting in fewer economic resources of the family, and her employment hindered her work in the household, reducing also household care. The higher female mortality rates among single mothers could stem from maternal preferences for sons, as daughters typically left the household upon marriage, whereas sons were expected to inherit family assets and provide support to the mother later in life.[243]

However, it is important to note that the information of these different family compositions was referred from burial notes, meaning that this analysis was an initial investigation of potential differences in family systems and health outcomes of boys compared to girls. It remains unknown to what extent this construction reflects reality. Parents could have remarried with a new partner, which means that there were two caregivers in a family instead of one. Quanjer et al. observed for instance lower mortality rates among children that experienced the arrival of a stepparent in the family after parental loss compared to children exhibiting higher mortality rates whose parent remained alone.[244] Moreover, in case of illegitimacy, a parent could have been absent from official vital registration, like the burial notes, but could still have been involved in the upbringing of a child.

Future research could further explore the role of family dynamics on gender roles of girls and parental discriminatory practices within families. Linking the Maastricht Death and Disease Database with LINKS-Limburg, a family reconstruction database, would facilitate this approach. It would allow to examine the role of the family at large, as child mortality was often clustered in families, and the impact of sibling competition, influenced by the number and ratio of brothers and sisters, on individual survival chances of girls in nineteenth- and early twentieth-century Maastricht.

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[242] Quanjer et al., 'Short Lives', 257.
[243] Beltrán Tapia and Szoltysek, 'Missing girls', 638-639 and 641.
[244] Quanjer et al., 'Short Lives', 271.

7.5 Conclusion

This chapter explored various indictors that could elucidate the influence of biological factors, gender roles, or gender-specific discrimination on the excess female mortality patterns observed in Maastricht. Since the pattern of excess female mortality persisted throughout the researched period, only factors contributing its existence were investigated, rather than those associated with its disappearance.

The first group of explanations focused on biological factors contributing to girls' higher susceptibility to infectious diseases compared to boys. It linked the disappearance of excess female mortality patterns to the epidemiological transition, suggesting that while the higher female susceptibility persisted, the mortality pattern disappeared at the societal level because infectious diseases were no longer the primary cause of death. In Maastricht, most diseases showing excess female mortality ratios declined by the early twentieth century, except for pneumonia and pulmonary tuberculosis. These diseases, with relatively high shares in the mortality pattern and notable excess female mortality ratios, presumably contributed to the persistence of the Maastricht excess female mortality pattern.

Some researchers proposed that hormonal changes during puberty increased female susceptibility, but this alone could not explain excess female mortality among girls aged 1-10 years, as these children had not reached the age of puberty yet. Others, moreover, argued that these hormonal changes only come to play when girls were malnourished, implying that other factors contributed to increased vulnerabilities. Therefore, besides biological factors, behavioural differences and varying exposure and resistance to infectious diseases likely contributed to higher infection rates among girls, leading to increased female mortality.

The second set of explanations focused on behavioural differences, shaped by different gender roles between boys and girls. In Maastricht, the impact of hazardous working conditions, such as in the textile industry, on health outcomes likely were not related to excess female mortality. Results suggested that boys were more often employed in wage work compared to girls and the mandated laws prohibiting child labour in factories diminished the effects of industrial working conditions on the health status of children. The role of nonmarket labour, like household chores, could not be examined due to absence of sources registering this type of work. However, some results indicated that girls were indeed more assigned to household tasks, which increased their exposure to infectious diseases. Analysis of mortality differences across socioeconomic classes revealed that excess female mortality occurred in higher classes, suggesting that parental occupational characteristics, rather than resource scarcity in poorer classes, contributed to increased infection risks of girls. Girls with parents in professions with high infectious disease exposure had higher mortality. This suggests that that in these types of households, parents had higher chances of contracting a disease and bringing it home, potentially infecting other household members. Longer and more intense exposure to an infection causes secondary cases to contract more severe infections, leading to higher mortality rates. The higher mortality levels among daughters compared to sons suggest that these girls could have spent more indoor time with this

sick person, potentially even in close contact as girls often had to take care of the sick, which could have caused more severe infections among them compared to boys.

The final set of explanations focused on gender-specific discrimination within families, highlighting unequal treatment of sons and daughters influenced by inequalities in gender positions. Despite high female education rates in Maastricht, patterns of excess female mortality persisted, indicating that basic education alone did not mitigate health disparities. Additionally, these high female education rates served as a proxy of vaccination rates, suggesting no inequalities in access to healthcare in Maastricht, at least regarding smallpox vaccinations. Analysis of economic shocks and mortality rates yielded few significant results. Combined with the absence of excess female mortality among the poorest classes, the results in this research suggested that economic scarcity might not have triggered unequal food allocation in families. A more important factor causing differences in health outcomes was the family composition. Girls exhibited higher mortality rates in families with one or no parent alive, possibly due to the remaining parent's focus on securing economic resources, affecting the quality and quantity of child care in the family. Potentially, girls in these families were expected to contribute more to the family compared to their brothers, which could have caused the differences in mortality outcomes.

8. Conclusion

In nineteenth-century Europe, young and adolescent girls and women of reproductive age exhibited significantly higher mortality rates compared to their male counterparts. Research has linked these patterns of excess female mortality to infectious diseases as they disproportionally affect girls. Proposed explanations argued that biological factors, gender roles or gender-specific discrimination caused differences in susceptibility and vulnerability between boys and girls, leading to higher female mortality rates. However, these explanations have yet to be tested at the individual level, as the majority of research has relied on aggregated data, leaving important questions about the characteristics of girls and their families unanswered. The newly-created Maastricht Death and Disease Database offered a unique opportunity to incorporate individual-level data into the study of mortality differences between males and females. This study aimed to identify patterns of excess female mortality among children aged 0 to 20 years between 1864 and 1930.

The first sub-question examined mortality differences between boys and girls across different age groups. Findings revealed that while infants and adolescents (aged 16-20) showed higher male mortality rates, children aged 1-15 years exhibited excess female mortality, making this age group the focal point of this study. While previous studies indicated the disappearance of excess female mortality in the Netherlands by 1924, the results of this study demonstrated that this pattern persisted in Maastricht at least until 1930. This suggested that disparities between the genders persisted at the local level, while they had disappeared at the national level. Additionally, comparison of male-to-female mortality ratios with male and female death rates suggested a correlation between high mortality years and excess male mortality, while low mortality years were associated with excess female mortality, suggesting differences in the underlying causes of death between boys and girls.

The second sub-question investigated the specific causes of death contributing to excess female mortality. Previous studies observed higher female mortality rates from infectious diseases, particularly tuberculosis. Utilising individual-level cause-of-death data, this study delved deeper into these causes of death, not only by distinguishing between routes of transmission, but also by examining the role of individual diseases. Results revealed that airborne infectious diseases, particularly lung-related illnesses such as pneumonia and bronchitis among young children, and pulmonary tuberculosis among older children were significant contributors to excess female mortality rates, due to their large shares in the mortality pattern and their higher death rates among girls compared to boys. Additionally, food- and waterborne diseases like typhus and tuberculosis played also a substantial role in the excess female mortality pattern, as these diseases also disproportionally affected girls and were accountable for considerate shares of deaths in the overall mortality pattern.

Additionally, the results revealed a disparity in mortality between endemic diseases in low mortality years, resulting in more deaths among girls, and disease outbreaks of both endemic and epidemic diseases in high mortality years, affecting more boys. The results suggested that in general, girls contracted infectious

diseases more often, leading to generally higher mortality rates. Boys, meanwhile, could have had a 'healthy boy effect', where lower exposure to infectious diseases generally led to lower mortality rates among boys compared to girls. However, during disease outbreaks, boys were disproportionally affected due to their lack of immunity from previous infections.

The final sub-question aimed to identify the underlying biological factors, gender roles, and discriminatory practices contributing to the observed excess female mortality in Maastricht. The epidemiological transition around the turn of the twentieth century has been linked to the disappearance of excess female mortality patterns, as infectious diseases were no longer the predominant cause of death. Two infectious diseases with excess female mortality ratios, pneumonia and pulmonary tuberculosis however continued to prevail in the Maastricht, which presumably caused the excess female mortality pattern in the city to prevail.

The higher mortality rates among girls from endemic diseases suggested either a higher susceptibility or a greater exposure compared to boys. The results supported previous research indicated that differences in gender roles and treatment, rather than biological factors, were important contributors to excess female mortality patterns. In Maastricht, factory working conditions were not associated with increased female mortality, nor were poor families. However, the parents' occupational exposure to infectious diseases did impact health outcomes of girls, with children of such parents exhibiting excess female mortality ratios. This suggests that daughter spent more time indoor with the sick parent, potentially in close contact as they took care of the sick, which could have caused more severe infections among daughters compared to sons.

Lastly, the study examined the role of unequal treatment. Results indicated no disparities in smallpox vaccination rates. Additionally, scarcity in resources did not appear to lead to discriminatory practices, as there was no clear correlation between food prices and female mortality rates. Instead, the absence of one or both parents appeared as an important contributor to increased female mortality risks, potentially leading to inequalities in terms of care and household tasks, as girls in single-parent families may have been required to contribute more to the household compared to boys.

Using individual-level cause-of-death data from the Maastricht Death and Disease Database enabled a thorough analysis of the specific causes of death that disproportionally affected girls. Moreover, as the data also contained socioeconomic and demographic information on the deceased persons, their deaths could be examined in relation to their specific socioeconomic and demographic characteristics, which provided a comprehensive understanding of the girls at increased risks of death in Maastricht during the late nineteenth and early twentieth centuries.

However, this study did not examine family dynamics. Future research should incorporate a family approach in studying patterns of excess female mortality, not only because child mortality was highly clustered in families, but also because family dynamics determined for a great deal the gender roles and allocation of resources and care of the family members. The Maastricht Death and Disease Database could be linked to the death certificates in the LINKS-Limburg family reconstruction database, which would facilitate the use of this family approach. This linkage would not only allow to identify mortality differences across families, but also

examine the role of the number, sex ratio and birth interval of siblings, as well as age differences between the parents and time differences between parental loss and child mortality.

Finally, as this study focused on mortality, it only captured disparities in the final stage of life. To develop a comprehensive understanding of health outcome differences between boys and girls, morbidity should also be considered. Sources on morbidity, such as hospital or doctor reports, could offer insights into infection rates and the severity of infections and other illnesses during life. This additional data would provide a deeper understanding of any inequalities in health statuses during life, which eventually contribute to inequalities in the overall health outcomes.

Creating a more comprehensive understanding of the determinants of excess female mortality is crucial as disparities between men and women continue to cause unequal health outcomes. Today, our world counts more than 140 million missing women that would still be alive if not for sex-selective abortions, female infanticide, and excess female mortality. Identifying the determinants that put individual girls at larger risks of death and the societal factors related to the disappearance of excess female mortality would contribute to create better-targeted policies that would better protect girls in societies that still experience higher mortality levels compared to boys.

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Appendices

Annex I

The population has been computed on the annual changes that happened in birth, death, in-migration and out-migration. To compare the natural population course to the total population course, the graph below contains both population developments. The natural population has been computed as followed: population = population (t-1) + (number of births – number of deaths). The population with migration has been computed as followed: population = population (t-1) + (number of births – number of deaths) + (number of in-migration – number of out-migration). As suggested in literature, Maastricht indeed had a net-migration close to zero, as in the majority of years, the population without and with migration were almost the same.
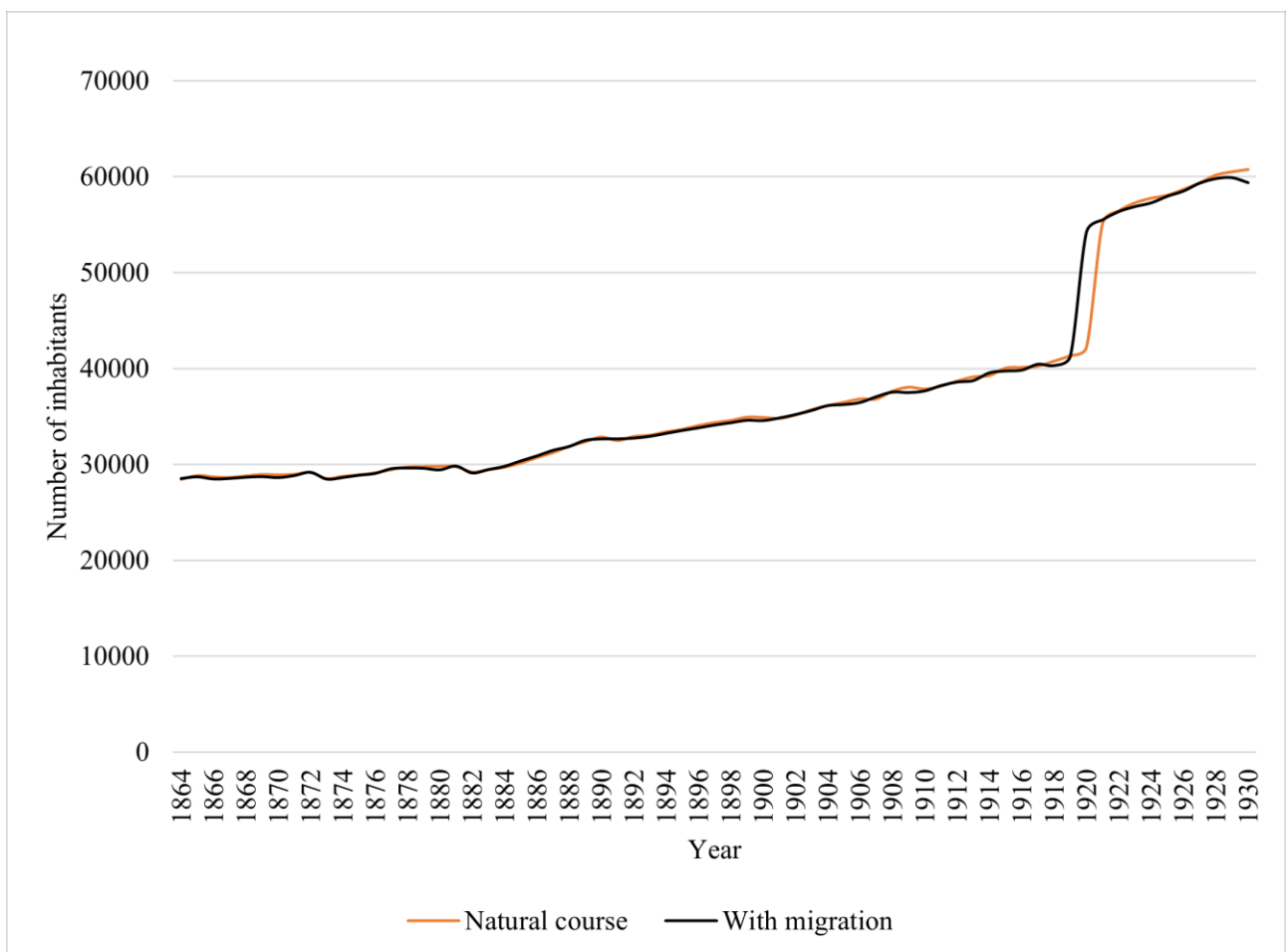
Source: Verslag over den toestand der gemeente Maastricht 1864-1930.

Variable	Infants <1 year N	Young children 1-5 years N	Children 6-10 years N	Teenagers 11-15 years N	Adolescents 16-20 years N
Gender					
Male	8869	4187	763	497	919
Female	7060	4071	670	507	731
Causal groups of death					
Food- and waterborne infectious diseases	793	361	114	55	93
Airborne infectious diseases	1669	759	150	113	164
Other infectious diseases	7152	3657	554	345	454
Non-infectious diseases	352	191	47	44	93
Non-natural causes of death	4555	2647	372	226	230
Ill-defined causes of death	1409	643	196	221	616
Unknown	793	361	114	55	93
Socioeconomic status father					
Managers and professionals	640	308	89	43	55
Clericals, sales personnel and foremen	1336	645	102	73	107
Medium and lower skilled workers	6275	3372	440	260	332
Lower and unskilled farm workers	244	147	28	29	69
Unskilled unspecified workers	4179	2501	324	187	178
Unknown	1294	585	162	182	492
Socioeconomic status mother					
Managers and professionals	22	15	7	6	13
Clericals, sales personnel and foremen	101	69	14	13	31
Medium and lower skilled workers	306	152	20	10	9
Lower and unskilled farm workers	49	30	8	14	34
Unskilled unspecified workers	1395	864	123	64	49
Unknown	12095	6428	973	667	1097
Occupational exposure father					
Both	2498	1282	216	148	193
Contact	3663	2240	302	187	192
Indoors	4723	2547	327	172	230
Unknown	1711	901	130	86	122
Occupational exposure mother					
Both	309	116	30	19	43
Contact	1242	836	120	61	45
Indoors	259	140	11	9	6
Unknown	51	33	8	15	34
Family composition					
Both alive	12654	6948	949	536	651
Father deceased or absent	865	197	10	8	10
Mother deceased or absent	219	148	61	82	152
Both parents deceased or absent	230	265	125	148	420
Total	**13968**	**7558**	**1145**	**774**	**1233**

In order to distinguish between epidemic and endemic diseases. the death rates of diseases per year were compared to the average crude death of the concerned disease throughout the research period. Following Murkens. a score of the mean mortality per 1.000 +3 standard deviations was used as a threshold for an epidemic outbreak.[245] As the crude death rates of children between 1 and 15 were used. some outbreaks among specific age categories could have been under- or overestimated as certain diseases occurred mostly among younger ages whereas others mostly affected older ages. Lastly. by using a cut-off-score based on the mean mortality. outbreaks of diseases that barely appeared at the end of the research period could have been overestimated.

The majority of lethal diseases for children were endemic diseases. which were always prevalent and caused mortality in most years. Epidemic diseases. on the other hand. caused large mortality spikes but did not or barely occur in the years in between outbreaks. Lastly. a disease could be both endemic and epidemic if it was always prevalent. caused a number of deaths in most years but also caused mortality spikes in certain years of large outbreaks.

Infectious disease	N in total	Mean mortality rate per year	Standard deviation	Threshold epidemic outbreak	Epidemic outbreak	Epidemic or endemic
Food- and waterborne						
Cholera	237	0.40	1.96	6.29	1866	Epidemic
Convulsions	516	0.76	0.67	2.78	1864 1867	Endemic
Gastroenteritis	1125	1.62	1.18	5.17	1878	Endemic
Tuberculosis	273	0.34	0.29	1.23	1870	Endemic
Typhus	92	0.14	0.22	0.81	1871	Endemic
Airborne						
Bronchitis	345	0.47	0.38	1.61	1891	Endemic
Diphtheria	664	0.93	1.74	6.15	1892 1893	Endemic with major outbreaks
Encephalitis	291	0.45	0.62	2.32	1871	Endemic
Pulmonary tuberculosis	803	1.02	0.43	2.30	-	Endemic
Measles	350	0.43	0.81	2.86	1907 1910	Epidemic
Meningitis	543	0.74	0.48	2.18	1879	Endemic
Pneumonia	1398	1.84	1.09	5.11	1887	Endemic
Scarlet fever	310	0.50	1.72	5.65	1864 1871 1879	Epidemic
Whooping cough	150	0.19	0.25	0.95	1897 1902	Epidemic

Source: Maastricht Death and Disease Database.

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[245] Murkens, *Unequal pathways to the grave?¸* 223.

Annex III

The seasonality of causal groups has been computed. First, the monthly average per causal group were computed, based on this average, the monthly numbers of deaths per causal group have been indexed.
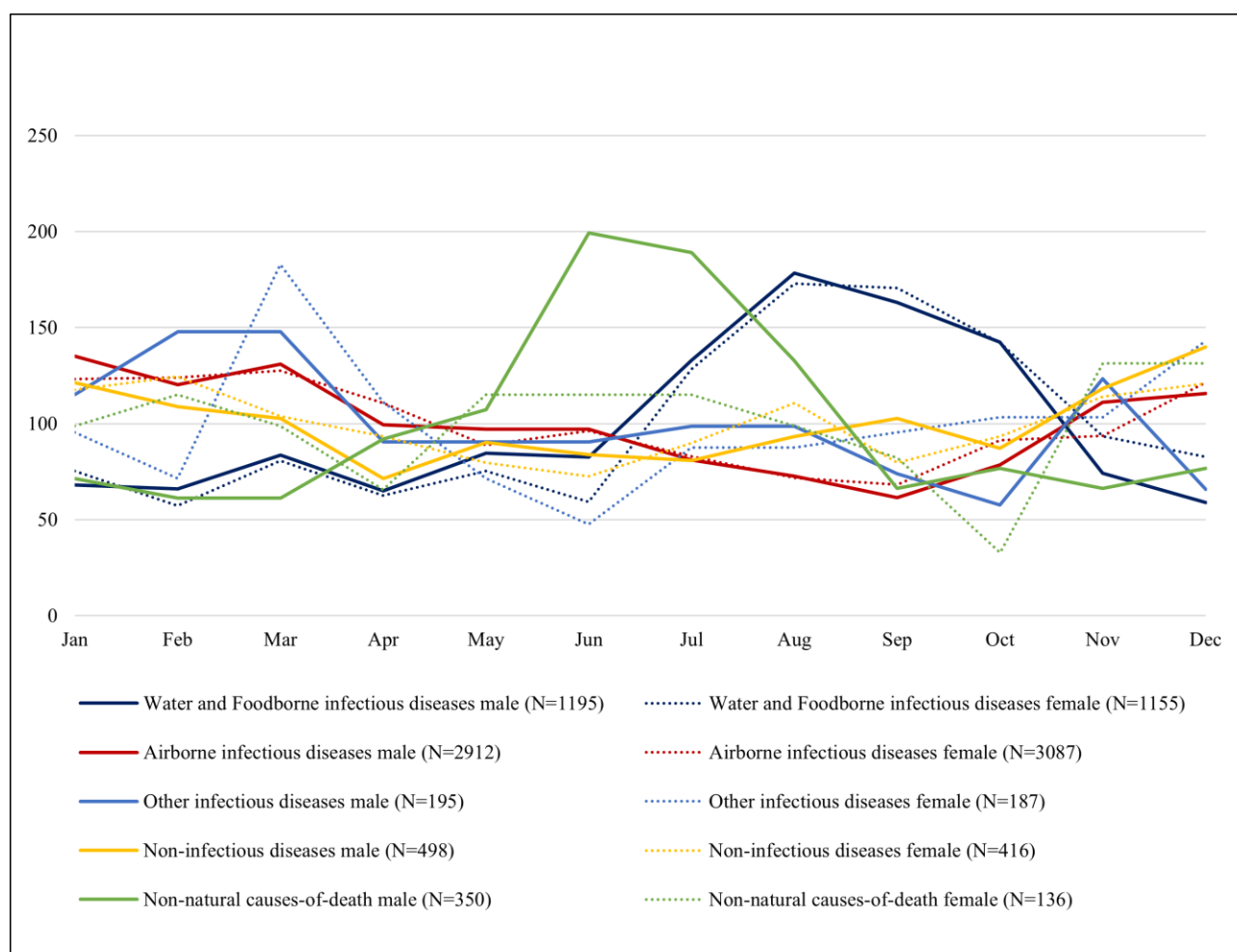
Source: Maastricht Death and Disease Database

Annex IV
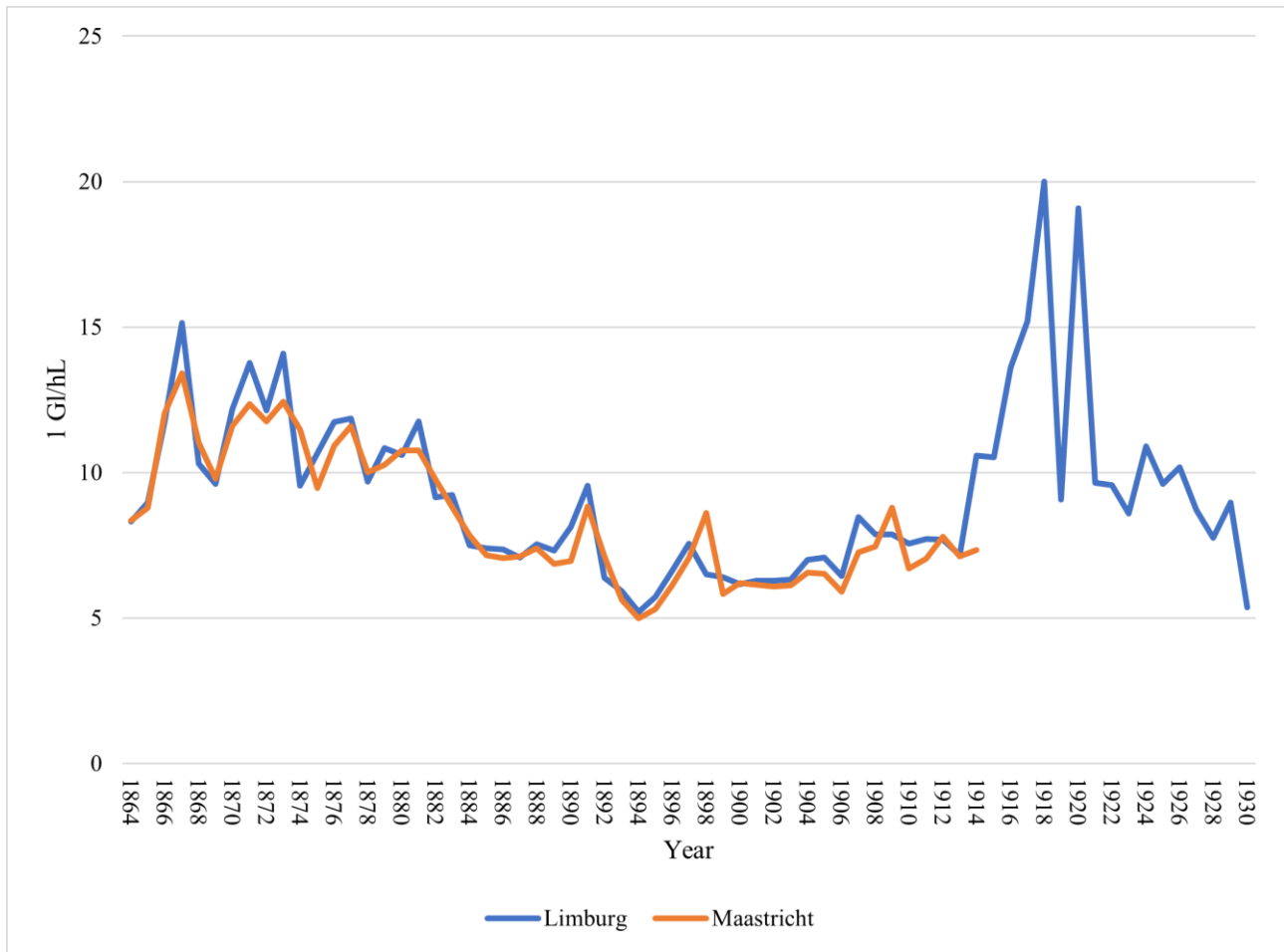
Source: W. Tijms, 'Prijzen van Granen en Peulvruchten te Arnhem, Breda, Deventer, 's-Hertogenbosch, Kampen, Koevorden, Maastricht, Nijmegen', Historia Agriculturae (Groningen 1983) 71-73, 79-80, 89-90.

Period	Age category	Sex	Year prices	R	R²	p model	B	p
All years	*1-5*	*Male*	t=0	0.48	.002	.705		
			t+1	.031	.001	.807		
			t+2	.025	.001	.844		
		Female	t=0	.081	.007	.514		
			t+1	.044	.002	.726		
			t+2	.008	.000	.947		
	6-10	*Male*	t=0	.274	.075	.025	.361	.025
			t+1	.241	.058	.052		
			t+2	.097	.009	.440		
		Female	t=0	.212	.045	.086		
			t+1	.114	.013	.360		
			t+2	.092	.009	.465		
	11-15	*Male*	t=0	.134	.018	.281		
			t+1	.003	.000	.981		
			t+2	.024	.001	.847		
		Female	t=0	.464	.215	<.001	.290	<.001
			t+1	.232	.054	.061		
			t+2	.074	.006	.557		
1864-1879	*1-5*	*Male*	t=0	.272	.074	.309		
			t+1	.408	.167	.131		
			t+2	.438	.191	.118		
		Female	t=0	.075	.006	.783		
			t+1	.147	.022	.600		
			t+2	.430	.185	.125		
	6-10	*Male*	t=0	.116	.013	.669		
			t+1	.118	.014	.675		
			t+2	.477	.227	.085		
		Female	t=0	.135	.018	.619		
			t+1	.527	.278	.044	-1.573	.044
			t+2	.538	.289	.047	-1.545	.047
	11-15	*Male*	t=0	.002	.000	.995		
			t+1	.399	.159	.141		
			t+2	.421	.177	.134		
		Female	t=0	.429	.184	.097		
			t+1	.240	.058	.388		
			t+2	.628	.394	.016	-1.124	.016
1880-1890	*1-5*	*Male*	t=0	.352	.124	.288		
			t+1	.196	.039	.563		
			t+2	.139	.019	.684		

		Female	t=0	.441	.194	.175		
			t+1	.399	.159	.224		
			t+2	.450	.203	.165		
	6-10	*Male*	t=0	.427	.183	.190		
			t+1	.275	.076	.413		
			t+2	.188	.035	.579		
		Female	t=0	.441	.194	.175		
			t+1	.329	.108	.323		
			t+2	.272	.074	.419		
	11-15	*Male*	t=0	.188	.035	.580		
			t+1	.235	.055	.487		
			t+2	.321	.103	.336		
		Female	t=0	.148	.022	.665		
			t+1	.301	.091	.368		
			t+2	.510	.260	.109		
1891-1900	*1-5*	*Male*	t=0	.049	.002	.894		
			t+1	.402	.162	.249		
			t+2	.444	.197	.199		
		Female	t=0	.107	.012	.768		
			t+1	.347	.120	.326		
			t+2	.448	.200	.195		
	6-10	*Male*	t=0	.329	.109	.353		
			t+1	.577	.333	.081		
			t+2	.373	.139	.288		
		Female	t=0	.108	.012	.767		
			t+1	.502	.252	.139		
			t+2	.726	.528	.017	1.069	.017
	11-15	*Male*	t=0	.078	.006	.831		
			t+1	.274	.075	.444		
			t+2	.438	.192	.206		
		Female	t=0	.695	.482	.026	.783	.026
			t+1	.428	.205	.189		
			t+2	.020	.000	.956		
1901-1910	*1-5*	*Male*	t=0	.032	.001	.929		
			t+1	.303	.092	.395		
			t+2	.297	.088	.405		
		Female	t=0	.017	.000	.962		
			t+1	.548	.300	.101		
			t+2	.525	.276	.119		
	6-10	*Male*	t=0	.105	.011	.774		
			t+1	.363	.132	.302		
			t+2	.008	.000	.983		
		Female	t=0	.003	.000	.993		
			t+1	.561	.315	.092		
			t+2	.114	.013	.755		

	11-15	*Male*	t=0	.339	.115	.339		
			t+1	.275	.076	.442		
			t+2	.170	.029	.639		
		Female	t=0	.293	.086	.412		
			t+1	.262	.069	.464		
			t+2	.026	.001	.944		
1911-1920	*1-5*	*Male*	t=0	.236	.056	.512		
			t+1	.260	.068	.468		
			t+2	.307	.094	.388		
		Female	t=0	.209	.043	.563		
			t+1	.120	.014	.742		
			t+2	.465	.216	.176		
	6-10	*Male*	t=0	.246	.060	.493		
			t+1	.582	.338	.078		
			t+2	.022	.000	.952		
		Female	t=0	.240	.058	.504		
			t+1	.106	.011	.770		
			t+2	.260	.067	.469		
	11-15	*Male*	t=0	.043	.002	.905		
			t+1	.247	.061	.491		
			t+2	.048	.002	.895		
		Female	t=0	.666	.443	.036	.176	.036
			t+1	.214	.046	.552		
			t+2	.047	.002	.898		
1921-1930	*1-5*	*Male*	t=0	.239	.057	.506		
			t+1	.575	.330	.082		
			t+2	.152	.023	.675		
		Female	t=0	.464	.215	.177		
			t+1	.569	.323	.086		
			t+2	.009	.000	.980		
	6-10	*Male*	t=0	.392	.153	.263		
			t+1	.195	.038	.589		
			t+2	.115	.013	.751		
		Female	t=0	.003	.000	.993		
			t+1	.254	.065	.478		
			t+2	.601	.361	.066		
	11-15	*Male*	t=0	.331	.110	.350		
			t+1	.089	.008	.807		
			t+2	.071	.005	.845		
		Female	t=0	.071	.005	.846		
			t+1	.630	.397	.051		
			t+2	.114	.013	.755		

Source: *Source: Maastricht Death and Disease Database; Centraal Bureau voor de Statistiek. Volkstelling 1859-1930; W. Tijms, 'Prijzen van Granen en Peulvruchten te Arnhem, Breda, Deventer, 's-Hertogenbosch, Kampen, Koevorden, Maastricht, Nijmegen', Historia Agriculturae (Groningen 1983) 71-73, 79-80, 89-90.*