A Natural Female Disadvantage?

Maternal Mortality and the Role of Nutrition Related Causes of Death in the Netherlands, 1875-1899

Angélique Janssens and Elien van Dongen

Abstract

This article addresses the question whether maternal mortality should be excluded from the study of excess female mortality. This phenomenon points to lower survival chances for women in certain age groups as opposed to men in the same age group. The existence of excess female mortality has been established for a number of European countries, primarily for the nineteenth century period, and it has also been observed for the Netherlands between approximately 1850 and 1930. There are strong indications that in this period Dutch women were at a disadvantage compared to men, most notably between the ages of 10 to 19, but also in the adult years after age 20. The survival disadvantage for women between age 20 and 50 may be related to the dangers of pregnancy and childbirth. These maternal mortality risks may seem a natural female disadvantage. However, deficiencies in nutrition may seriously enhance the dangers of pregnancy and childbirth. The results of our analysis indicate that maternal mortality in this period in the Netherlands is partly the effect of the female nutritional disease environment. In particular, the incidence of nutrition-related deaths among women in fertile ages, such as TB, increase maternal mortality. We therefore assume that gender disadvantages in the access to foodstuffs of sufficient nutritional quality increased the level of maternal mortality. Consequently, in research on excess female mortality maternal mortality cannot be simply discounted as a natural disadvantage which should be left out of measures of excess female mortality.

Introduction

In contemporary western populations women have higher survival chances than men, so that it is often assumed that this has been the case throughout most of our past. However, higher female survival has not always been the case, as research on some European countries has identified. This phenomenon, which is called excess female mortality, has also been observed for the Netherlands. There are strong indications that women were at a disadvantage compared to men, most notably between the ages of 10 to 19, but also in the adult years after age 20.[1] In these age groups the mortality hazards for women were higher than for men. Adult female death rates exceeding those of males have been observed for nineteenth century England and Wales, as well as for eighteenth and nineteenth century rural Germany.[2] In quite a few studies a strong relationship has been found with rural areas and the agricultural sector, and authors have hypothesized that the excess female death rates should be attributed to women's reduced access to medical care and adequate nutrition.[3]

Humphries points out that these rural female disadvantages were not related to a traditional rural culture but resulted from the capitalist transformations of the agriculture sector.[4] The scale-up in farming led to the disappearance of small farms and the phenomenon of live-in servants which primarily affected the labour opportunities of women. This economic modernization made women and children more dependent upon men and male breadwinners within a precarious family economy which privileged the male breadwinner in terms of food intakes and other forms of care. Support for this mechanism is also found for the Netherlands.[5] As a result survival chances of young girls and adult wom-

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1 Frans van Poppel, *De 'statistieke ontleding van de dooden': een spraakzame bron?* (Nijmegen 1999).

2 Jane Humphries, ' "Bread and a pennyworth of treacle". Excess female mortality in England in the 1840s', *Cambridge Journal of Economics* 15 (1991) 451-473; Kirsty McNay, Jane Humphries and Stephan Klasen, 'Excess female mortality in nineteenth-century England and Wales. A regional analysis', *Social Science History* 29 (2005) 649-681; Bernard Harris, 'Gender, health, and welfare in England and Wales since industrialisation', *Research in Economic History* 26 (2008) 157-204; Stephan Klasen, 'Marriage, bargaining, and intrahousehold resource allocation: Excess female mortality among adults during early German development, 1740-1860', *Journal of Economic History* 58 (1998) 432-467.

3 Amartya Sen, 'Mortality as an indicator of economic success and failure', *The Economic Journal* 108 (1998) 1-25.

4 Humphries, ' "Bread and a pennyworth of treacle" '.

5 Wiebke Schulz, Ineke Maas and Marco van Leeuwen, 'When women disappear from the labour market: Occupational status of Dutch women at marriage in a modernizing society, 1865-1922', *The History of the Family* 19 (2014) 426-446.

en in England below 60 years of age were seriously depressed. These conclusions were confirmed for nineteenth-century England by McNay, Humphries and Klasen.[6] Klasen reaches a similar conclusion for eighteenth century Germany. Here too the modernization of agriculture was not beneficial for women's survival chances.[7]

However, other studies have indicated that also outside agriculture excess female mortality could and did occur in nineteenth century Europe. In her study of mortality hazards for girls between the ages of 5 and 20 in Belgium around 1900, Isabelle Devos demonstrates that although excess female mortality was highest in rural areas, the industrial textile areas followed closely.[8] According to Devos the negative female survival chances for girls in these latter areas should be attributed to the high proportion of young women in the labour force in the textile industry. Similarly, Eggerickx and Tabutin point towards the important role of unhealthy working conditions in the textile sector in Flanders at that time, which they consider to be an important explanation of excess female mortality in the final decades of the nineteenth century.[9] This shows that excess female mortality is a multi-causal phenomenon, but food intakes, medical care, living and working conditions are factors of prime importance.[10]

For the Netherlands Frans van Poppel was the first to study the occurrence of excess female mortality in different age groups in the period between 1850 and 1996.[11] His results indicate that higher female mortality risks remained in existence throughout the entire period but disappeared in the 1930s. Whether excess female mortality also existed prior to the nineteenth century is unknown. Van Poppel also concludes that especially in the eastern and southern parts of the country girls' and women's survival chances remained behind those for men and boys. Based on similar data Janssens confirms Van Poppel's conclusions regarding female survival disadvantages in the age groups of 14 to 19,

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6   McNay, Humphries and Klasen, 'Excess Female Mortality'.

7   The authors cited above were not the first to have argued that excess female mortality was often found to be related to early modernization in rural areas in European countries. See also: Sheila Ryan Johansson, 'Welfare, mortality and gender. Continuity and change in explanations for male/female mortality differences over three centuries', *Continuity and Change* 6 (1991) 135-177.

8   Isabelle Devos, 'Te jong om te sterven. De levenskansen van meisjes in België omstreeks 1900', *Tijdschrift voor Sociale Geschiedenis* 26 (2000) 55-75.

9   Thierry Eggerickx and Dominique Tabutin, 'La surmortalité des filles en Belgique vers 1890. Une approche régionale', *Population* 49 (1994) 657-683.

10   See: Devos, 'Te jong om te sterven', 70, for an explanatory model for excess female mortality.

11   Van Poppel, *De 'statistieke ontleding van de dooden'*.

and 20 to 50 years.[12] In another study on higher mortality risks for young girls below age 20, Van Poppel, Schellekens and Walhout achieved some mixed results. Excess mortality was only identified for girls below age 10, but not for older girls. Neither could they find a clear connection to agricultural and rural areas, or farming families; it seemed above all that excess mortality for girls was somewhat more prevalent in families headed by unskilled labourers.[13] The dataset that was used in this study may be part of the explanation; for regions for which female disadvantages are assumed to have been more prevalent the dataset is characterized by undersampling whereas oversampling occurs for areas where male disadvantages were more likely to have been the case. On the basis of a comparative analysis of gender differences in physical stature in the Netherlands Hans de Beer argues that it is not likely that the biological living standard for girls was any different from that of boys. Hence, de Beer calls into question the existence of gender differences in the access to food and care, which factor plays an important role in studies regarding excess female mortality.[14] His study does not necessarily have any implications for the issue regarding gender differences in the access to food and care in adulthood: height is determined by nutritional intake in early life. Moreover, de Beer's study is based on height data of prison detainees. Results can therefore not be generalized to the population as a whole.

Excess female mortality for the age group between 5 and 14 also existed in other European countries between roughly 1850 and 1930, after which period excess male mortality came to be the norm. Firm conclusions for earlier periods before the 1850s or earlier are hampered by the lack of systematic and adequate time series data. Nevertheless, it seems that excess female mortality appeared occasionally before 1800 in the younger and adolescent age groups, and that during the nineteenth century excess female mortality became more marked in European countries.[15]

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12 Angélique Janssens, *Sekse, gender en de dood* (Maastricht 2016).

13 Frans van Poppel, Jona Schellekens and Evelien Walhout, 'Oversterfte van jonge meisjes in Nederland in de negentiende en eerste helft van de twintigste eeuw', *Tijdschrift voor Sociale en Economische Geschiedenis* 6 (2009) 37-69.

14 Hans de Beer, 'Physical stature and biological living standards of girls and young women in the Netherlands, born between 1815 and 1865', *History of the Family* 15 (2010) 60-75.

15 Dominique 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) 17-52.

Reduced female survival chances are especially significant as women are believed to have a modest biological survival advantage over men in all age groups. Hence, under 'normal' conditions excess mortality should be a male phenomenon rather than pertaining to women. Indeed, nowadays men have higher mortality than women, both in terms of overall measures such as life expectancy at birth but also in terms of life expectancy for certain age groups, at least in the western world. The existence of a natural female survival advantage seems undoubted and rests on both genetic and biological mechanisms.[16] Especially genetic factors seem to contribute to a higher female resistances to infectious diseases.[17] However, non-biological factors, which we may broadly define as the individual's living environment, may contribute in substantial ways to actual mortality hazards. Male excess mortality may for instance result from the selection of men into high-risk lifestyles (e.g. alcohol or occupational hazards including fatal work accidents). Furthermore, biological and non-biological factors are also in interaction with each other, as well as with the reigning disease environment. For instance, female hormones may favour women in a disease environment dominated by infectious diseases due to the enhanced immunity effect of oestrogens.[18] This assumption is especially important for historical research into women's position and women's health in the past. In the period before the mid-twentieth century, before the so-called epidemiological transition, infectious diseases were by far the most predominant causes of death. Under these conditions, if all else remains equal, female survival should be higher than male survival. Still, life chances may also be affected, in both direct and indirect ways, by the prevailing economic, social and cultural context in which individuals are living. Hence, researchers such as Samuel Preston have argued that the significant improvements in the social status of women, and hence in their greater survival chances, are amongst the main drivers of the appearance of excess male mortality in the post war western world.[19]

16 Marc Luy, 'The impact of biological factors on sex differences in life expectancy: Insights gained from a natural experiment', in: Martin Dinges and Andreas Weigl (eds.), *Gender-specific life expectancy in Europe 1850-2010* (Stuttgart 2016) 17-46.

17 Ingrid Waldron, 'Sex differences in infant and early childhood mortality: Major causes of death and possible biological causes', in: United Nations, *Too young to die: Genes or gender?* 64-82.

18 Luy, 'The impact of biological factors on sex differences'; Luciana Quaranta, *Scarred for life. How conditions in early life affect socioeconomic status, reproduction and mortality in Southern Sweden, 1813-1968*, Lund Studies in Economic History 59 (Lund 2013).

19 Samuel H. Preston, *Mortality patterns in national populations. With special reference to recorded causes of death* (New York 1976); Carla Medalia and Virginia W. Chang, 'Gender equality, development,

In this study we focus on gender differentials in mortality in the adult age group, that is between age 20 and 50, in the Netherlands in the period 1875-1900. The reasons for the specific time and age boundaries will be explained later on in the article. Higher mortality risks for women in the adult age group may obviously be related to the risks involved in pregnancy and childbearing; especially before the twentieth century and the arrival of penicillin and antisepsis these maternal mortality risks were much higher that they are today.

Indeed, for nineteenth-century England and Wales McNay et al. demonstrate that maternal mortality is an important component of excess female mortality.[20] For the Netherlands Janssens has similarly shown that the exclusion of maternal mortality from gender differentials in adult mortality strongly reduces the observed level of excess female mortality.[21]

It is however questionable whether maternal mortality should indeed be excluded from measures of gender differentials in survival chances. Doing so suggests that maternal mortality should be seen as a 'natural' disadvantage which is entirely unrelated to any gendered pattern of disadvantage or discrimination, for instance regarding access to adequate care and nutritional intake. Another implication of this perspective is that variations in the levels of maternal mortality are seen as exclusively related to the level of medical knowledge and technology, which is applied to all members of society in equal ways.

However, between populations there are large differences in the hazards of motherhood, also in the past, which are not only related to diverging levels of obstetric knowledge and health care, but also to the wider social and economic context, as well as the reigning disease environment. Modern research indicates that pregnancy depresses a woman's immune system, and hence the female biological advantage, so that women have an increased risk of dying from infectious diseases such as influenza, tuberculosis and smallpox in the final stage of the pregnancy.[22] This enhanced risk does not raise the numbers of women dying from *direct* obstetric causes, but it may increase the numbers of associated deaths, deaths due to other causes such as tuberculosis, but af-

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and cross-national sex gaps in life expectancy', *International Journal of Comparative Sociology* 52 (2011) 371-389.

20 McNay, Humphries and Klasen, 'Excess female mortality'.

21 Janssens, *Sekse, gender en de dood*.

22 Irvine Loudon, *Death in childbirth: An international study of maternal care and maternal mortality, 1800-1950* (Oxford 1992) 31.

fecting pregnant or postpartum women. These risks will be enhanced further if women and girls are denied sufficient access to survival-related resources such as adequate food. Poor nutritional levels may directly increase the incidence of certain infectious diseases through enhanced susceptibility. Tuberculosis, which is transmitted through air born particles, is one of those diseases which is considered to have a definite relationship with nutritional levels.[23] Pregnancy and childbirth, combined with poor nutrition therefore poses enhanced mortality risks, certainly in areas where these diseases are endemic. Thus, tuberculosis resulting from reduced nutrition and therefore enhanced susceptibility for the disease may be one of the mechanism in increasing mortality levels for adult women immediately before and after childbirth through the indirect effect of associated deaths.

Contemporary research on Africa in recent years also shows that poverty, malnutrition and adequate care play a role in the sometimes appallingly high rates of maternal mortality in these countries.[24] These studies provide evidence for the fact that tuberculosis and other respiratory diseases (often in association with HIV/AIDS) have become major non-obstetric causes of maternal mortality. As a result a larger proportion of maternal mortality, defined in these studies as women dying during pregnancy, childbirth or in 42 days after delivery, is then due not so much to strictly obstetric causes for maternal mortality but should be considered as resulting from a lack of adequate nutrition.

In this contribution we question the assumption that maternal mortality should be seen as a 'natural' disadvantage. In fact, our aim is to demonstrate that the level of maternal mortality itself may be subject to patterns of gender discrimination involving unequal access to food and health care. We do this by investigating the relationship between maternal mortality and TB, respiratory diseases and other diseases which are known to be related to nutritional intake for adult women in the Netherlands in the age group of 20-50 years during the period 1875-1899. In past societies, TB and respiratory diseases belonged to the major killers

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23 Massimo Livi-Bacci, *Population and nutrition. An essay on European demographic history* (Cambridge 1991); R.I. Rotberg and T.K. Rabb (eds.), *Hunger and history* (Cambridge MA 1985) 305-308: 'The relationship of nutrition, disease, and social conditions: A graphical presentation'; Thomas McKeown, *The modern rise of population* (London 1976).

24 Y. Ahmed et al., 'A study of maternal mortality at the University Teaching Hospital, Lusaka, Zambia: The emergence of tuberculosis as a major non-obstetric cause of maternal mortality', *International Journal of Tuberculosis and Lung Disease*, 3, 8 (1999) 675-680; John Grange et al., 'Tuberculosis in association with HIV/AIDS emerges as a major nonobstetric cause of maternal mortality in Sub-Saharan Africa', *International Journal of Gynecology and Obstetrics* 108 (2010) 181-183.

in the adult age group – although not exclusively so as it was also the primary cause of death amongst adolescents –, and this was no different in the Netherlands as we shall see below. Efficient ways to cure this disease were non-existent, and after infection survival chances were slight.[25] In line with the literature we have cited above we assume that vulnerability to these types of infectious diseases seriously increases as a result of poor nutritional intake, which is especially dangerous during pregnancy and childbirth. Hence, if we find that the impact of the level of TB, respiratory diseases and the group of nutrition related diseases on maternal mortality is strong, we may conclude that maternal mortality is partly the effect of gender disadvantages in the access to sufficient nutritional intake. Or more importantly, the conclusion should then be that maternal mortality should not simply be excluded in assessments of female survival disadvantages in the past.

We investigate the impact of TB, respiratory diseases and other nutrition related diseases on maternal mortality through the use of regression models which are able to indicate the proportion of maternal deaths which may have been due to these so-called associated diseases. Before doing so, we will explore the level of and the regional variation in excess female mortality, as well as in maternal mortality and TB/respiratory diseases. We limit ourselves to the period 1875-1899, which is determined by the time boundaries of the sources. Still, this is a crucial period in the occurrence and gradual disappearance of excess female mortality in the Netherlands.[26] The nature of the source, see below, also determines that we conduct our investigation on the age group between 20 and 50 years which admittedly offers a rough estimation. Not all years between age 20 and 50 pose equal dangers for women of dying in childbirth; it is well known that especially first births are more dangerous for mothers than later births.[27] Finally, the source also determines our definition of maternal mortality; this is discussed in the data section below.

25 Devos, 'Te jong om te sterven', 66.

26 Van Poppel, *De 'statistieke ontleding van de dooden'.*

27 Brett E. Ory and Frans van Poppel, 'Trends and risk factors in maternal mortality in late-nineteenth-century Netherlands', *The History of the Family* 18 (2013) 481-509.

Data and methods

We make use of community level aggregates for the period 1875-1899 taken from the five-yearly cause of death statistics at the municipal level for the Netherlands produced for the period 1875-1899.[28] For every five-year period the Ministry of Interior Affairs ordered statistics registering the cause of death by age and sex in a nomenclature of 34 different causes of death; see the appendix for the complete list. For adults the following age groups were distinguished: 20-50 years, 50-65 years, 65-80 and those over 80 years of age. In addition to the causes listed here, the source also identified the number of deaths occurring in each age group without medical treatment.

The causes of death reported in this period had to be certified by a medical practitioner. This became mandatory in 1869 when the Burial Act decreed that a body could not be interred before a doctor had provided the civil registry with a cause of death. All civil registries in the Netherlands had to submit monthly overviews of all deaths by age, sex and by cause of death to the Ministry of Interior Affairs. From 1875 until 1900 the Ministry aggregated all these overviews to be published in five-year volumes containing the cause of death overviews for all Dutch municipalities. During this period the nomenclature remained unchanged.

One of the aims of the 1869 regulation was to improve the existing cause of death registration in the Netherlands which was considered to be rather unreliable.[29] The unreliability is for instance evident from the larger numbers of deaths registered as due to unknown causes. Most likely this is a sign that large numbers of death went without medical treatment and that doctors did little to identify the cause of death. As a consequence of the 1869 legislation the quality of the cause of death registration increased as evidenced by the rapidly declining numbers of cases without medical treatment or due to unknown causes. For the purpose of this study it is important to underline that for adults these latter two categories were relatively small, suggesting that medical care for adults was mostly called in, as opposed to the situation for babies,

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28 *Vijfjarig overzicht van de sterfte naar den leeftijd en de oorzaken van den dood in elke gemeente van Nederland*, Ministerie van Binnenlandse Zaken, 's-Gravenhage, Van Weelden en Mingelen, 1882-1901.
29 Nynke van den Boomen and Peter Ekamper, 'Denied their "natural nourishment": Religion, cause of death and infant mortality in the Netherlands, 1875-1899', *The History of the Family* 20 (2015) 391-419; Nynke van den Boomen, 'The impurities in statistics: Interpretations of death and disease in the Netherlands, 1875-1899', paper presented to the ninth WOG workshop *Diseases, causes of death and the epidemiological transition*, Maastricht, 8 December 2016.

young children and the elderly. This becomes understandable when one considers the importance to the family economy of adults of working age: families were more quick to call in medical aid and more prepared to spend money on medical care. Nevertheless, the quality of the diagnosis doctors were giving remains controversial. Since the 1865 Public Health Inspectorate Act and the Medical Practitioners Act the qualifications for medical practitioners were officially defined and only qualified individuals were allowed to practice medicine after successfully passing an official examination. However, medical knowledge changed significantly in this period, as well as did diagnostic and coding practices. We therefore have to remain cautious in terms of the conclusions we can base on these types of data.

In contemporary practice maternal mortality is usually defined as the number of women dying during pregnancy, childbirth or within 42 days after the delivery. This category will then include deaths which are due to obstetric causes as well as non-obstetric ones. We cannot be sure that this is also the case in the source we use here. As the nomenclature states, see the appendix, the category maternal mortality includes deaths due to puerperal diseases or puerperal fever so that it is *possible* that maternal deaths due to associated deaths are hidden in other cause of death categories. The extent to which this may be the case here remains unknown as the source material does not offer information on the way doctors labelled the cause of death of female patients who died during pregnancy and childbirth.

In order to calculate cause specific death rates we have made use of the censuses from this period (1879, 1889, 1899 and 1909) to estimate the population at risk for each 5-year period.[30] As earlier censuses are not available at a sufficiently disaggregated level, we use population sizes at the end of each 5-year period to estimate person-years-lived of the population at risk, rather than the mid-period population sizes commonly used.[31] We do this for single communities but also for larger regions ac-

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[30] We wish to thank DANS (the Netherlands Institute for data archiving) and more in particular Tom Vreugdenhil for their efficient and swift help in obtaining the latest corrected files for the 1879 and 1909 censuses.

[31] The 1899 census does not include age-specific population counts for small municipalities separately, but rather the average age distribution for all municipalities of a certain size, in combination with total population sizes for each municipality. Therefore, the population at risk for the period 1895-1899, which in our specification should ideally be based on the 1899 census, is imputed for all smaller municipalities as:

$$0.5 * \left(\frac{pop_{muni\,1889}^{20-50yo}}{pop_{muni\,1889}^{total}} + \frac{pop_{muni\,1909}^{20-50yo}}{pop_{muni\,1909}^{total}} \right) * pop_{muni\,1899}^{total}$$

cording to the economic-geographic area classification constructed by the Central Bureau of Statistics in the Netherlands for the year 1921.[32] This classification divides the Netherlands into 42 areas which differ from one another on the basis of their dominant economic structures. The classification distinguishes different types of agriculture (grain cultivation, horticulture and livestock farming) and different types of industry. This CBS regional classification enables us to collapse single communities which are largely similar into larger regions. Moreover, the analysis we present below would also not be suitable at the level of single communities because of the large number of zero values for the smaller communities. In addition, at such a level we expect very large random variations to occur. To estimate the effect of TB, respiratory diseases and nutrition related diseases on the level of maternal mortality we make use of a fixed effect regression model which holds constant the average effects of the regions and localities used. The choice for this analytical approach will be explained below in the section on regression results.

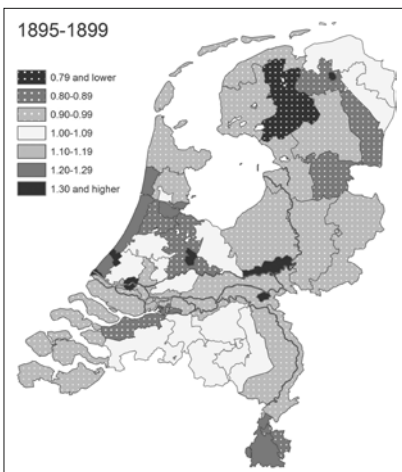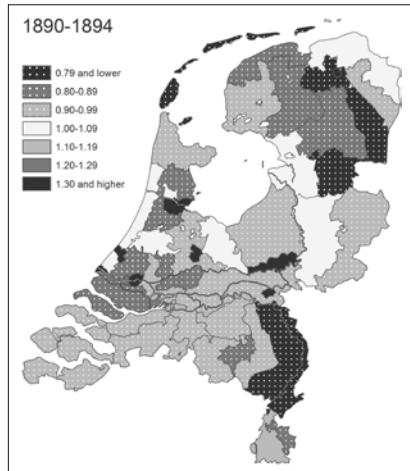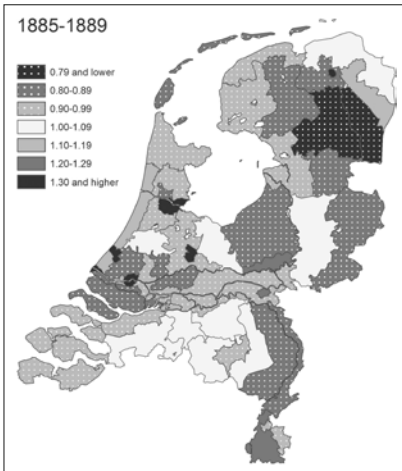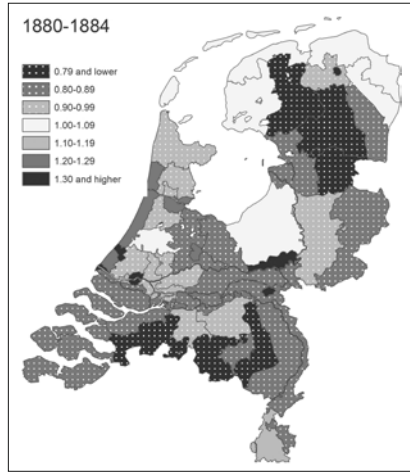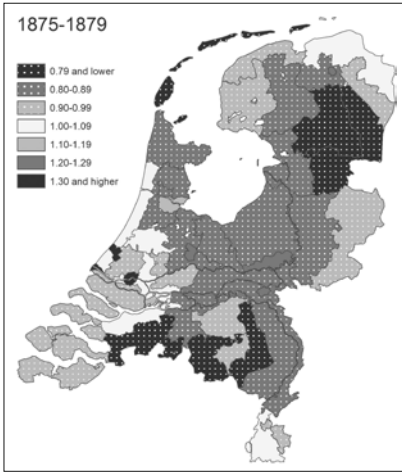## Excess female mortality in the Netherlands, 1875-1899

The first author to note the existence of excess female mortality in the Netherlands in the second half of the nineteenth century was Van Poppel.[33] He demonstrated that excess female mortality was especially prevalent between the ages of 3 and 19, as well as between 25 and 45.[34] On the basis of the cause of death statistics used in this study we are able to confirm his conclusion regarding the adult age group, between age 20 and 50. The first series of maps (maps 1) shows the incidence of excess female mortality by five year period between 1875 and 1899 based on ratios of male and female death rates (male death rate/female death rate). Ratios below 1 indicate an excess of females over males, whereas above 1 are indicative of more men than women dying in this age group. At the start of the period excess female mortality is evident in most regions of the Netherlands, but especially pronounced in some of the eastern and southern areas. Here the ratio decreases to be-

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32 Ronald van der Bie, *De economisch-geografische indelingen van het CBS, 1917-1960* (The Hague, Heerlen 2009).

33 Van Poppel, *De 'statistieke ontleding van de dooden'.*

34 Tabutin and Willems show that the intensity of the female disadvantage in the Netherlands was quite moderate compared to some other countries, at least for the age group 5 to 14: Tabutin and Willems, 'Differential mortality by sex'.

Maps 1 Ratio male-female mortality rates, 1875-1899*

1875-1879

0.79 and lower
0.80-0.89
0.90-0.99
1.00-1.09
1.10-1.19
1.20-1.29
1.30 and higher

1880-1884

0.79 and lower
0.80-0.89
0.90-0.99
1.00-1.09
1.10-1.19
1.20-1.29
1.30 and higher

1885-1889

0.79 and lower
0.80-0.89
0.90-0.99
1.00-1.09
1.10-1.19
1.20-1.29
1.30 and higher

1890-1894

0.79 and lower
0.80-0.89
0.90-0.99
1.00-1.09
1.10-1.19
1.20-1.29
1.30 and higher

1895-1899

0.79 and lower
0.80-0.89
0.90-0.99
1.00-1.09
1.10-1.19
1.20-1.29
1.30 and higher

Cartography: Thijs Hermsen, e-Humanities Lab, Radboud University Nijmegen.

low 0.80, which means that for every 100 male deaths more than 125 female deaths occurred, which is a serious survival disadvantage. For ratios between 0.90 and 1 this relationship improves to 110 female deaths or fewer for every 100 male deaths.

Male excess mortality also occurs but this is more or less limited to the urban areas (e.g. cities such as Amsterdam, Rotterdam, Utrecht and The Hague) and a limited number of rural areas in the west, and in the very north and south part of the country. Especially Rotterdam and The Hague, situated in the western part of the country, stand out as places where male excess mortality takes on a serious dimension: here for every 100 males only 75 females deaths occurred. Excess female mortality in the Netherlands is therefore, similar to European countries, a largely rural phenomenon. Over the total period between 1875 and 1899 the level of female survival disadvantage declines to reach a much more moderate level in the final period between 1895 and 1899. In this latter period 'only' 110 females died for every 100 male deaths. Still, it is important to note that the major improvement in the level of excess female mortality occurred in the final period between 1895 and 1899 when there is only one rural region left in the north-eastern part with a survival disadvantage below 0.80.

The regional pattern which restricts excess female mortality to rural areas remains largely the same between 1875 and 1899. Obviously, work on farms and in the countryside was physically demanding for women, especially on the smaller family farms. In addition, the second half of the nineteenth century was also the period in which the participation of women in agriculture declined relative to men, which has been noted by Van Zanden, as well as by Van Nederveen Meerkerk and Paping.[35] Equally, the number of live-in farm servants seems to have declined in this period which may have entailed an increase in the work burden for married women on family farms. In most cases their work activities were not related to the market and therefore went without any monetary value. This negative development for married women is thought to be related to the increasing levels of mechanization due to the agricultural crisis which hit the country in the 1880s.[36] However, the decline of waged work in agriculture must also have been detrimen-

35 Jan Luiten van Zanden, *De economische ontwikkeling van de Nederlandse landbouw in de negentiende eeuw, 1800-1914* (Wageningen 1985); Elise van Nederveen Meerkerk and Richard Paping, 'Beyond the census. Reconstructing Dutch women's labour market participation in agriculture in the Netherlands, ca. 1830-1910', *The History of the Family* 19 (2014) 447-468.

36 Van Zanden, *De economische ontwikkeling*.

tal for single women who were robbed of important employment opportunities. Both developments contributed to a decline in the value of women's work. These negative developments for women may possibly have counteracted and delayed the positive effects of improving hygiene and health care that can also be noted in this period.

It is however not our aim to explain the occurrence of excess female mortality in this period in the Netherlands. The results above serve to demonstrate the clear occurrence of the phenomenon of excess female mortality and to justify our inquiry into the nature of maternal mortality in the Netherlands in this period and its relation with certain infectious diseases. In the next section we will survey the level of and regional variation in maternal mortality in the Netherlands, as well as the occurrence of TB and respiratory diseases.

## Maternal mortality and TB/respiratory diseases, 1875-1899

In the period of investigation maternal mortality in the Netherlands was low compared to many of the surrounding countries. At the start of our period (1875-1879) the Netherlands ranked in the very top of countries compared by Loudon best able to fight maternal mortality, and in the years until 1900-1904 it was able to further reduce maternal mortality to extremely low levels for that time.[37] Whereas England and Wales experienced a maternal mortality rate of 44 (deaths per 10,000 live births), and Sweden even reached the enormously high rate of 89, the maternal mortality rate for the Netherlands was 41. By 1900-1904 this figure for the Netherlands had fallen to 24 (cf. for England and Wales: 44; for Sweden: 23) which indicates that considerable progress had been made in the fight against maternal mortality. It has been suggested that towards the end of the nineteenth century doctors were increasingly reluctant to record cases of maternal mortality as they came to see these as their failure to assist in deliveries, so that the number of 'hidden' maternal mortality deaths in the official figures may have increased.[38] The comparatively favourable position of the Netherlands concerning maternal mortality is generally related to the quality of Dutch midwifery and the relative absence of hospital deliveries.[39]

37 Loudon, *Death in childbirth*.
38 Catharine van Tussenbroek, *De ontwikkeling van de aseptische verloskunde in Nederland* (Haarlem 1911).
39 John R. Shepherd et al., 'Maternal mortality in Taiwan and the Netherlands, 1850-1945', in: Theo

Maps 2   Maternal mortality (per 10,000 person years lived), 1875-1899*
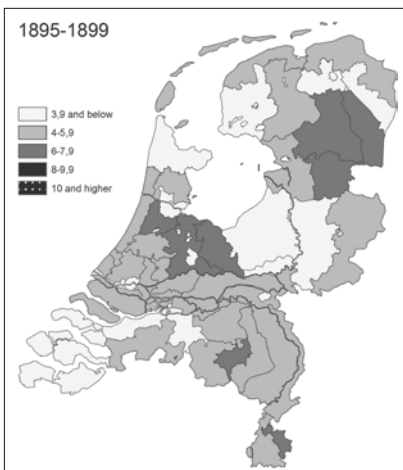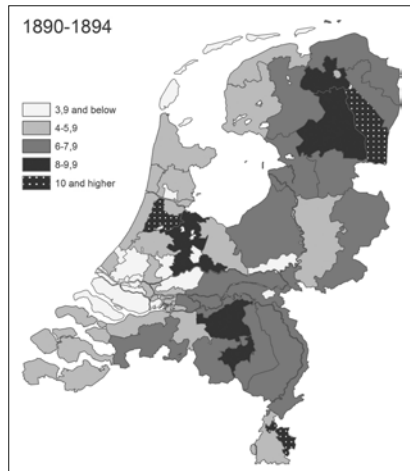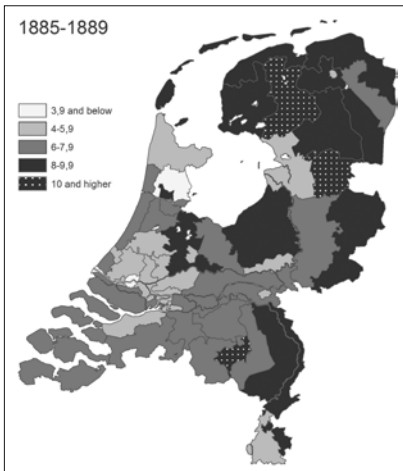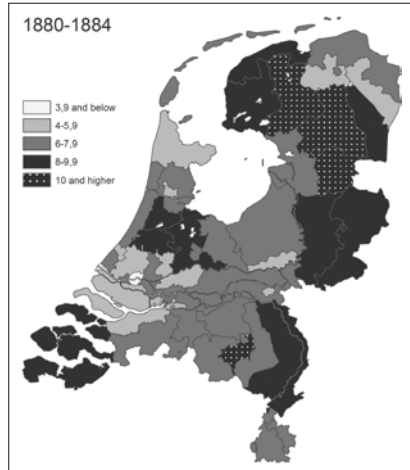
* Cartography: Thijs Hermsen, e-Humanities Lab, Rad-boud University Nijmegen.

However, there is a very distinct regional variation in the incidence of maternal mortality. Loudon suggested that progress was largest in Amsterdam but that is not entirely true.[40] The municipal cause of death data for the Netherlands allow us to show the regional variation of maternal mortality between 1875 and 1900. Maps 2 show the regional variation of maternal mortality rate per 10,000 person-years lived, between the ages of 20 and 50. Two things stand out from these maps. Between 1875 and 1900 the fall in maternal mortality was considerable: it dropped by 50 per cent or even more. Secondly, especially at the start of the period there is a strong regional variation in the level of maternal mortality. Comparatively high levels can be found in the northern and southern parts of the country as well as along the eastern borders with Germany. These are primarily rural and agricultural areas, with quite some variation however in terms of soil type, farm sizes, the level of commercialization of farming, and also in terms of wage levels.[41] Nevertheless, also in the mid-western part of the country to the south of Amsterdam, not a particularly rural area, the level of maternal mortality was quite high.

Towards the end of the century the entire western part of the country has clearly taken the lead. Levels have dropped to a maternal mortality rate of 4 or even less per 10,000 person-years lived. By 1900 a few spots have remained where this rate was still between 6 and 8 per 10,000 person-years, located in the north-eastern part (the relatively poor region of Drenthe), the south and also in the west, to the south of Amsterdam. It is worth noting that the maternal mortality rates of the Catholic south (the provinces of North-Brabant and Limburg in the south and the south-east) do not appear in the top positions by the end of the century, despite the high fertility levels found in these provinces in this period. We also produced alternative measures of maternal mortality based on the number of live births rather than person-years lived. These results (not shown here) show that the Catholic south fares even better compared to the rest of the country.

Engelen, John R. Shepherd and Yang Wen-shan (eds.), *Death at the opposite ends of the Eurasian continent. Mortality trends in Taiwan and the Netherlands 1850-1945* (Amsterdam 2011) 229-273; Ory and Van Poppel, 'Trends and risk factors in maternal mortality'; V. Lazuka, L. Quaranta and T. Bengtsson, 'Fighting infectious disease: Evidence from Sweden 1870-1940', *Population and Development Review* 42 (2016) 27-52.

40 Loudon, *Death in childbirth*.

41 Hans Knippenberg and Ben de Pater, *De eenwording van Nederland* (Nijmegen 1988) 92-134.

Maps 3 Mortality due to TB and other respiratory diseases for females and males (per 10,000 years lived), 1875-1899 *
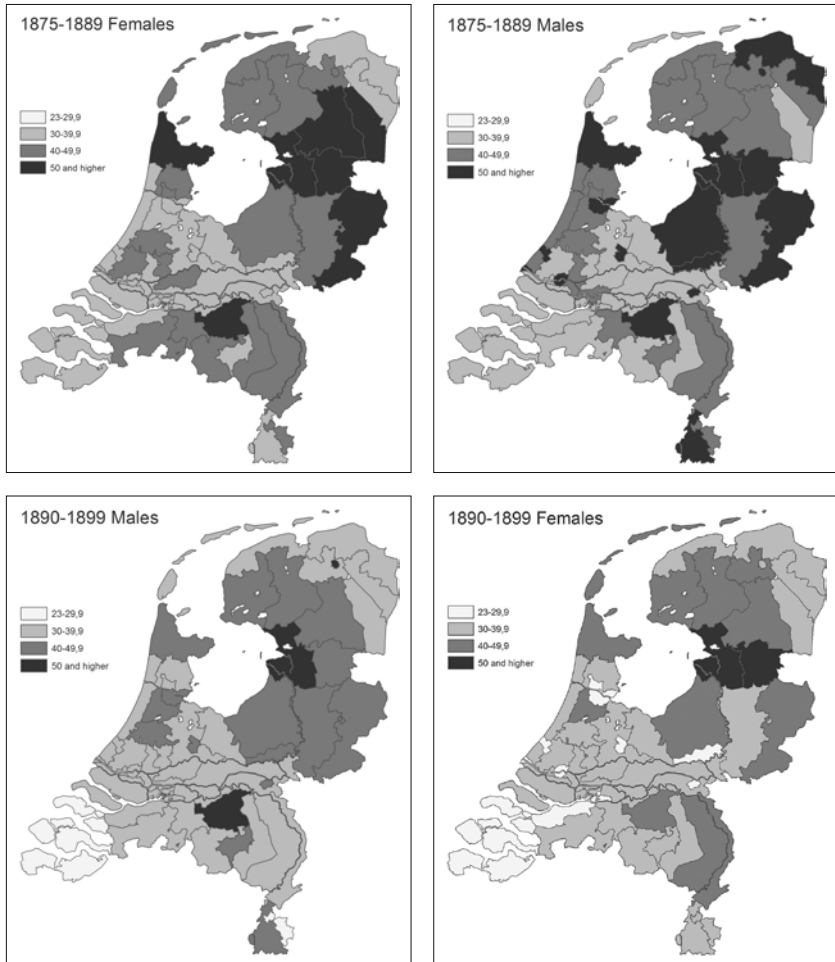
* *Cartography: Thijs Hermsen, e-Humanities Lab, Radboud University Nijmegen.*

The risk of dying in childbirth was not the major killer of women aged 20-50 years. This role was played by the category of tuberculosis and other respiratory diseases, which conclusion also pertains to men in this age group. Maps 3 show the mortality rates from TB and other respiratory diseases for both men and women; to limit the number of maps we collapsed the five-year periods into two periods, from 1875 to 1889 and from 1890 to 1899.[42] For both men and women cause specif-

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42 Differences within these two periods were only minimal.

ic mortality rates for TB/respiratory diseases varied regionally between 35 and 75 per 10,000 person-years lived for the period 1875-1879, and between 22 to 51 for the 1895-1899 period. It is clear that progress in these contagious diseases was a lot more difficult to achieve before the age of penicillin. Regional variation is important here as well, and especially the pattern for women suggests some degree of correlation between TB/respiratory diseases and maternal mortality. This is highly relevant to the purpose of this article as we are primarily interested in the contribution of TB/respiratory diseases to the level of maternal mortality.

The importance of regional variation in both TB/respiratory diseases and maternal mortality remained relatively constant over the twenty-five year period: while the region with highest levels of maternal mortality generally had four times as high mortality as the region with lowest maternal mortality, the highest rate of TB/respiratory was about 2.5 times that of the lowest rate in both the first and the last five-year period. However, which regions had lowest and highest rates varied. This implies that cause-specific mortality decline occurred at different paces in different regions.[43] We will take this regional variation as well as the time trend into account in our regression models.

In order to investigate the relationship between the two causes of death categories we present a scatter plot in figure 1 showing the correlation for all regions and all five-year periods between 1875 and 1900. The numbers on the X and Y axis of this graph present the cause specific mortality rate for each disease, that is the numbers of women dying from this specific disease between the age of 20 and 50 per 1,000 person-years lived. The graph shows that we may assume a strong correlation between the two groups of causes of death; the separate trend lines demonstrate that this relation exists for each of the five periods. For all five periods the maternal mortality rate is high where the rate for TB is high and vice versa.

To emphasize the role time plays in this association, different periods are represented by different symbols. Both mortality due to TB/respiratory diseases and maternal mortality decrease substantially during the last quarter of the nineteenth century. Thus, any analysis of the association between these causes should control carefully for general time

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43 For example: Maternal mortality per 1,000 live births ranges from 2.15-7.24 in 1875-1879 to 1.25-3.98 in 1895-1899. The minimum and maximum regions in these two periods are respectively: region 32 (min 1875), region 8 (max 1875), region 37 (min 1895), region 6 (max 1895).

Figure 4 Scatter plot showing the correlation between maternal mortality and TB/respiratory diseases (per 1,000 PYL 20-50 years old), for all 42 regions and all 5-year periods 1875-1900
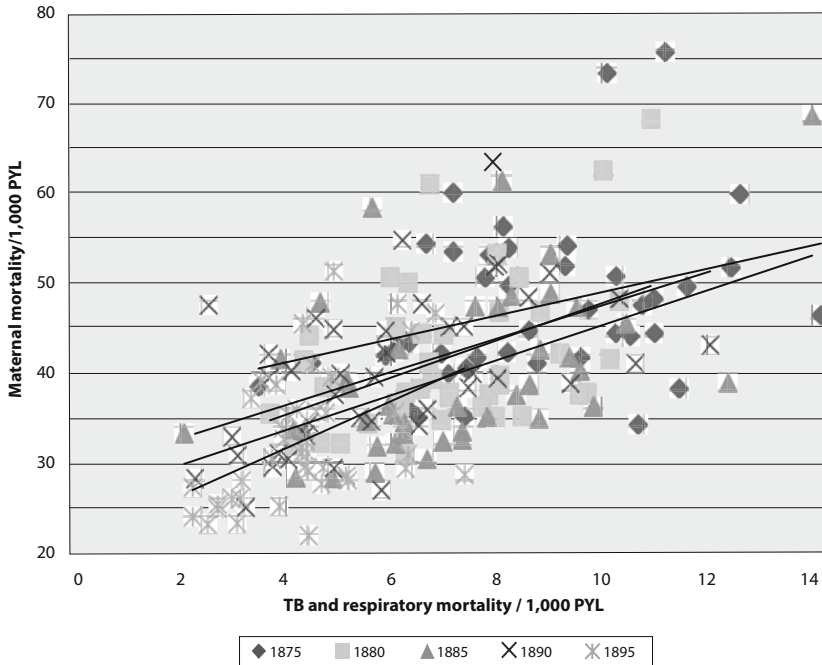
trends in the mortality decline. However, as we can see an association within each time period as well, a further examination of the role played by TB in the incidence of maternal mortality appears justified.

In the subsequent section we will conduct a regression analysis to investigate the impact of TB/respiratory diseases, and nutrition related diseases, on maternal mortality. For this purpose, it is vital to control for regional and temporal differences in disease environment, medical conditions, female labour market participation, demographic conditions such as mortality decline and fertility levels et cetera. As there is no perfect data on the local level for all these characteristics, we will do this in an indirect way using a fixed effects model instead of a simple OLS regression. Using this approach we control for time-invariant regional differences and differences over time without them being included as separate covariates. We should also add here that it is not our intention to offer explanations for regional or time differences in the level of maternal mortality, nor for the variation in the impact of TB and other diseases on the level of maternal mortality. Our aim is to show that the level

of maternal mortality is influenced by the incidence of female TB (and other diseases) while *taking into account* various types of regional and time differences. Further details on this approach are discussed in the regression section.

Regression analysis

We explore the role played by TB and its effect on the incidence of maternal mortality in the Netherlands in a number of fixed effects regression models. As opposed to OLS regression models, the fixed effects models used here control for the variation between regions and periods. The approach is similar to adding dummies for each region and period in an OLS model. Only the association between independent and dependent variable within each region and period is estimated. Thus, any time-invariant characteristics of regions, or nation-wide characteristics of periods that affect both the dependent and independent variable (so-called confounding variables), are controlled for without adding them as covariates to the model. In this way we can estimate the relationship between TB and maternal mortality without actually having all the data on regional differences, e.g. the economic structure of a region, and differences in time, e.g. the speed with which general mortality declines. Thus, fixed effects models control *indirectly* for these so-called omitted variables.

There remains, however, some concern that we do not control for certain confounding variables: those that differ between regions and over time simultaneously. Examples of possible confounders of this form would be regional differences in the speed of the mortality decline, or regional differences over time in the improvement of health care facilities. We partly resolve this by adding a linear time trend for each region. But as will become clear in the results section below, such factors either have little effect or are not captured well by a region-specific linear time trend.

First, we conduct an OLS regression without any of these period and regional controls. The results of this are shown in column (1) of table 1. The OLS model does *not* control for the variation between the 42 regions nor for differences between periods (1875-1879, 1880-1884, 1885-1889, 1890-1894, 1895-1899). The only control added here is the general disease environment for TB/respiratory and nutritional diseases, operationalised through male mortality of these diseases. In the

OLS model we find a strong correlation between deaths due to female TB/respiratory diseases and maternal mortality; this is indicated by the 0.128 coefficient on this variable. This can be interpreted in the following way. If per 10,000 of the population at risk there is one additional female death due to TB/respiratory disease then there will be 0.128 additional cases of maternal mortality per 10,000 of the population at risk (the coefficient in table 1, column (1)). As TB/respiratory mortality rates are about six times as high as maternal mortality, this means that maternal mortality increases proportionally to TB/respiratory mortality (0.13*6=0.78). Hence, if the numbers of TB/respiratory deaths double, the number of cases of maternal mortality increases with 80 per cent. As we explained above, OLS regression does not take into account differences over time and space. So we expect that the results of model (1) will be biased. As we discussed in earlier sections, we assume the relationship to be strongly confounded by the general mortality decline. Hence, the OLS result will overestimate the actual effect of TB/respiratory diseases on maternal mortality.

In further models – columns (2)-(6) in table 1 – the fixed effects are added step by step in order to remove the bias due to time and space. In column (2) we control only for secular time trends. In column (3) we control, instead, for time-invariant differences between regions. Column (4) combines these period and region controls. This approach, in column (4), follows the model most commonly applied in social sciences when these types of panel data are used.[44] We will first discuss the motivation behind these models, and subsequently their results.

The region controls, which are added in model (3), capture the differences between the 42 regions, but only in as far as these differences do not vary over time. The period controls, first introduced in model (2), capture the downward trend in mortality rates for the Netherlands as a whole, as well as other changes over time, e.g. in the level of medical care. The period controls however do not capture possible differences between the 42 regions in the speed of this process.

To control for these time differences in the pace of mortality decline between regions linear time trends are added in model (6). Under the assumption that the speed of mortality decline for different locations *within* each region is fairly similar, the regional linear time trend *together* with the period controls should thus capture the major omitted variable

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44 For an easy to read introduction to these models, see: J.D. Angrist and J.S. Pischke, *Mostly harmless econometrics* (Princeton 2008) 221-226, 243-244.

here: the overall mortality decline. We believe this assumption is justified, as the regions are defined to capture municipalities which are similar with regard to their economic structure, labour market and urbanization rate. In this way we control for nation-wide changes over time between 1875 and 1900, such as advances in medical care, improvements in nutritional status, or in important variables such as changes in female labour market participation and general living conditions for women (housing, hygiene et cetera). The regional linear time trends add variation between regions in this process of change over time.

Beside tuberculosis – our main independent variable of interest –, we added other respiratory mortality rates (due to acute and chronic respiratory diseases) as well as an aggregate for the mortality rate due to other nutrition-related diseases. This latter group of diseases consists of measles, whooping cough, diarrhoea, dysentery, cholera asiatica and cholera nostra, and various acute diseases of the digestive system.[45] Deficiencies in nutritional intake increase the susceptibility to these diseases.[46] Cause-specific *female* mortality rates for nutrition-related diseases capture the cause-specific *female* disease environment in each region. The general disease environment for nutrition-related diseases is controlled for by adding similar cause-specific *male* mortality rates. For all causes of death these rates reflect deaths within the population aged 20-50 years old as a share of the number of person-years-lived (population at risk * exposure time) in the same age group. We specify different models in which tuberculosis, acute and chronic respiratory diseases are either aggregated or included separately. The disaggregated models are shown in the appendix, see table 2.

As classification practices are likely to have varied between doctors, as well as the ability of doctors to distinguish between some of these individual respiratory causes of death, we believe that the aggregated category (TB, acute and chronic respiratory diseases) may be better able to capture regional differences in the incidence of tuberculosis.[47] To sum-

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45 See the appendix for the various disease categories and the different groups of diseases we are using here. TB falls under disease number 18. Acute and chronic respiratory diseases are listed under numbers 21 and 22 respectively. The nutrition related diseases listed here can be found in the appendix under numbers 12, 20, 26, 26* 27, 27* and 28. See the following literature on this: Rotberg and Rabb (eds.), *Hunger and history*, 305-308; Livi-Bacci, *Population and nutrition*, 38.

46 Nutritional deficiencies are capable of reducing resistance to infection and increasing the severity of many infections through a variety of mechanisms such as: a reduced production of humoral antibodies, an impaired cell-mediated immunity or less effective phagocytosis (a mechanism which is able to destroy invading micro-organisms).

47 A comparison of differences in mortality rates for each of the three respiratory categories at the mu-

Illustration 1 Granulin, the anti-tuberculosis drug prepared according to the prescription of the inventor, Dr J.H. van Grafhorst. Granulin Company (Apeldoorn) (source: ReclameArsenaal, Koninklijke Bibliotheek Nederland, BG D30/971, affiche).

marise, models (2), (4) and (5) include period controls, while model (6) additionally includes region-specific linear time trends. Furthermore, in models (3), (4) and (5) we control for regional differences. Let us now discuss the results of these models.

In model (2) we see that period is indeed essential to the association between TB/respiratory and other nutrition-related disease environments and maternal mortality, indicated by the 'R-squared between' of 0.93. This number shows that most of the variation in maternal mortality can be attributed to change over time. This fits in with the decline in maternal mortality we have seen in maps 2. However, controlled for this change over time (by the period control variable) a strong association between mortality due to TB/respiratory diseases and maternal mortality remains. Models (3), (4) and (6) include the control for region, in model (4) and (6) in combination with period controls. In

nicipal level suggests that, to a certain extent, the categories were probably used interchangeably. This can however not be formally assessed without information on the doctor reporting each cause of death.

these three models the 'R-squared between' indicates the proportion of the variation in maternal mortality attributable to regional variation which is stable over time. We see that regional variation is less substantial than temporal variation, as the 'R-squared between' in the models including period fixed effects is initially around 0.19 and virtually disappears when regional time trends are added (model (6)).[48] Note also that all models have a significant F-statistic.[49]

Our main result is model (6), which indicates that whilst controlling for the variance in regions, time and the linear time trends there remains a substantial positive and significant association between maternal mortality and our female death causes of interest. As expected, the effect size is somewhat smaller here than in the OLS model; 0.104 instead of 0.128. This implies that if the number of TB/respiratory deaths doubles, the number of cases of maternal mortality increases with 60 per cent.[50]

Additionally, model (6) shows a small positive effect of the general disease environment as indicated by the male TB/respiratory mortality variable. This effect is inconsistent over the different model specifications; it changes sign moving from OLS and model (2) to the full fixed effects models (4)-(6). To further investigate the relationship between maternal mortality and the general disease environment, the same group of other nutritional diseases that is included for women, is also included for men. We see that the coefficient for male other nutritional mortality is close to zero in all models. On the other hand, other nutritional mortality rates among women may increase maternal mortality. This result, although insignificant, is consistent over model specifications. The coefficient for other female nutritional mortality is around 0.3 in specifications (4)-(6) and thus much larger than the coefficient for TB/respiratory mortality. This should not be taken to mean that the impact of this group of diseases is larger than that of TB/respiratory mortality. The incidence of nutrition-related mortality among 20-50 year old

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48 The 'R-squared within' in model (2) and (3) refers to the proportion of the variance in maternal mortality attributable to TB/respiratory and nutrition-related mortality, whilst in model (4) this measure refers to the same mortality rates plus period controls and in model (6) it includes the variance due to TB, period plus the linear time trend. It has a lower value for model (2) – the only model which does not include period effects. This confirms the importance of the general mortality decline during this period.
49 This implies that the models are a significant improvement on the null-hypothesis where none of the included covariates affect maternal mortality. Calculating the F-statistic in model (6) is not possible/unreliable because of a high covariate to observation ratio.
50 As before, this calculation takes into account that the incidence of TB/nutritional mortality is six times higher than for maternal mortality.

Table 1 Fixed effects models for Dutch maternal mortality 1875-1900 per region (42) and period (5), including imputed populations

Maternal mortality /10,000 person-years lived (20-50 year old women)	(1) OLS	(2) FE	(3) FE	(4) FE	(5) first diff.	(6) FE
Female TB and respiratory	0.128***	0.113***	0.125***	0.0812*	0.0797*	0.104**
mortality rate	(0.0159)	(0.0133)	(0.0340)	(0.0431)	(0.0408)	(0.0504)
Fem. nutritional	0.379***	0.193	0.393***	0.280*	0.347**	0.296
mortality rate	(0.127)	(0.128)	(0.135)	(0.149)	(0.143)	(0.180)
Male TB and respiratory	-0.0119	-0.0386*	0.0782*	0.0536	0.0863**	0.0889*
mortality rate	(0.0219)	(0.0160)	(0.0448)	(0.0462)	(0.0402)	(0.0479)
Male nutritional	-0.00248	0.0122	0.0255	0.0455	-0.102	-0.0861
mortality rate	(0.148)	(0.177)	(0.134)	(0.137)	(0.111)	(0.141)
Period controls	No	Yes	No	Yes	Yes	Yes
Regional controls	No	No	Yes	Yes	Yes	Yes
Regional linear time trend	No	No	No	No	No	Yes
Observations	210	210	210	210	168	210
R-squared within	–	0.231	0.557	0.595	–	0.771
between	–	0.939	0.189	0.182	–	0.008
overall	0.401	0.381	0.364	0.423	0.241	0.284
R-squared adj.	–	–	–	–	0.207	–
F statistic	37.29	[68.28][1]	44.37	22.18	4.90	–
p-value F test	0.000	[0.001]	0.000	0.000	0.001	–

*Robust standard errors in parentheses *** p<0.01, ** p<0.05, * p<0.1.*

1   *Model excluding male nutritional mortality taye-current model includes too many covariates.*

women is somewhat lower than the incidence of maternal mortality, and more than six times as low as the incidence of TB/respiratory mortality.[51] Thus, the much larger coefficient for female other nutritional mortality reflects a proportional effect of the female other nutritional disease environment on maternal mortality which is less than half the proportional effect of female TB/respiratory disease environment on maternal mortality.

51 More specifically, the rate of nutritional-related mortality (excluding TB) shows a higher regional variation than the rate of maternal mortality: the maximum observed value of female nutrition-related mortality across all regions is similar to the maximum observed value of maternal mortality, while the minimum observed value of female nutrition-related mortality is more than five times as small as the minimum observed value of maternal mortality.

However, model (6) may be overspecified. As we only have five distinct periods the linear time trends and regional controls may largely capture the same phenomena (resulting in collinearity). This is also suggested by the very low 'R-squared between' in model (6): controlled for regional linear time trends close to none of the variation in maternal mortality can be attributed to regional controls. This may indicate that the most important difference between regions is in their speed of mortality decline. Given that model (6) may be overspecified, it is important to include model (4) and (5), which exclude linear time trends, in our main conclusions. The results here are very similar to those in model (6). Moreover, models (4) and (5) do not entirely miss out on the regional variation in the speed of the general mortality decline. This variation is partly captured by the covariate for male cause-specific mortality rates.

Thus, we regard models (4) and (5) as reliable specifications. Both models control for region and period but are estimated using different techniques: fixed effects versus first-differences.[52] If the models are specified correctly (i.e. are consistent) the results of these estimation techniques should converge when the number of observations is large enough. Our sample size is rather small, nevertheless we obtain similar results in model (4) and (5). This justifies our assumption that the results in models (4) and (5) can be regarded as plausible.

Finally, we conducted various other regression models and analytical techniques to further test our results. We have relegated the information on these test to the footnotes to keep the main body of the text more readable.[53] Generally, on the basis of our analysis we may conclude

[52] While in region-fixed effects models the regional average is distracted from each observation for all included variables (dependent and independent), in the first-difference model the differences between observations at time t and time t-1 are used instead of the observations themselves, again for all included variables (dependent and independent). In fact, there is reason to believe the first-difference approach is superior in our case (as the error terms may be serially correlated). The fixed effects approach is discussed as our main approach for sake of simplicity, because results are similar, and because of the possibility to add regional linear time trends.

[53] The regressions shown here make use of a limited number of observations (42 regions in 5 time periods). This approach is preferred to an analysis at the more disaggregated level of individual municipalities. Regressions such as we use here would not be suitable at such a level because of the large number of zero values. In addition, at such a level we expect very large random variations to occur. Nevertheless, we did run models at the municipal level (not shown here) which gave similar results to the regional models: with, as expected, smaller coefficients and higher significance. Furthermore, the models used here define maternal mortality in terms of the population at risk (person-years lived of 20-50 year old women). As a robustness check, we created a similar model (not shown in this article but available upon request) using the number of maternal deaths per 1,000 live births, which gave almost identical results

that the effect of female cause-specific mortality on maternal mortality seems considerably stronger – and less ambiguous – than the effect of male-cause specific mortality. This makes an interpretation relating these findings to a female disadvantage in access to food plausible.

Conclusions

In this study we have first of all confirmed the existence of excess female mortality in the adult years in the Netherlands for the period between 1875 and 1900. These enhanced female mortality risks between age 20 and 50 are particularly significant given the assumed biological advantage and the higher resilience of women to infectious diseases as compared to men. In conformity to findings elsewhere, the higher female mortality risks were mainly found for rural areas which is suggestive of a link with agriculture. The fact that these female disadvantages were not limited to rural areas with lower levels of market integration and commercialization suggest the existence of an overall rural penalty for women in this period. There are strong differences in the levels of this female mortality disadvantage between regions, but these do not follow in a neat way divisions between the more advanced rural areas and the more backward ones.

These findings clearly diverge from those found for other countries, for instance for nineteenth-century England, where higher female mortality risks were primarily found in the more developed rural areas. An explanation for this specific regional pattern of excess female mortality in the Netherlands is outside the scope of this article. However, based on the existing literature on rural work opportunities for women and girls in the later parts of the nineteenth century we suggest that this overall rural female penalty may be connected to the disappearance of

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to the model presented above. The model specification using more disaggregated cause-specific mortality rates, shown in appendix table 2, should be seen as another robustness check. The most important findings here are that chronic respiratory diseases seem entirely unrelated to maternal mortality, contrary to acute respiratory diseases which show a very strong association. Furthermore, the coefficient for TB narrowly defined (death cause no. 18 in the appendix) becomes insignificant in models (4)-(6) due to high variation, although it is consistently positive. Furthermore, the effect size of TB should be considered large, as about three-quarter of mortality due to TB and other respiratory diseases falls under this narrow definition of TB. A coefficient of 0.07 then means that as many as three in every ten cases of maternal mortality may be due to TB (0.07/0.22). We assume that the insignificance of TB in this specification is largely due to variation among doctors in classification practices, where tuberculosis and other respiratory classifications might have been used by different doctors for similar pathologies.

female employment opportunities in the countryside. Further research is needed on this issue. Towards the end of the period under investigation the adult female mortality disadvantage diminished significantly, but had still not disappeared entirely.

Obviously, the enhanced adult female mortality risks we have found seriously implicate the negative effects of pregnancy and childbirth. This makes the question urgent and legitimate whether we should regard maternal mortality as a natural female disadvantage which may simply be ignored when considering excess female mortality in the past. Based on the literature on contemporary societies we have followed the assumption that part of the maternal mortality of women between age 20 and 50 in this period in the Netherlands might be due to so-called associated deaths; deaths due to diseases such as TB and a number of other diseases which are in a similar way as TB related to a lack of adequate nutritional intakes. If nutrition is insufficient, resistance to these diseases is seriously reduced and the severity of the disease, after infection, is increased. This is especially dangerous in combination with pregnancy and childbirth which depresses a woman's immune system. Hence, these infectious diseases contribute indirectly to higher levels of maternal mortality through the phenomenon of associated deaths. The associated deaths would then occur during pregnancy and childbirth as a result of diseases such as influenza, TB, cholera or other nutrition related diseases.

Indeed, in this study we have first of all established a correlation between TB and respiratory diseases on the one hand and maternal mortality on the other hand. For both types of diseases the mortality rates decline within our study period but within each time period a close association exists between maternal mortality and TB/respiratory diseases. When maternal mortality is high, the death rate due to TB and respiratory diseases is also high. This indicates that the two groups of diseases are related to each other in some way or another.

Secondly, we have been able to ascertain that indeed a considerable part of maternal mortality in the period 1875-1900 can be attributed to TB, respiratory diseases and other diseases for which adequate nutritional intakes are highly relevant. It also appeared that the *general* disease environment, as represented via male cause specific mortality rates, was irrelevant to the regression outcomes. Hence, health conditions which were specific for women in this age group partly determined outcomes for maternal mortality risks.

We may therefore conclude that an important part of maternal mortality is related to the quality of life, that is access to food of sufficient

nutritional quality, of adult women in the Netherlands in the second half of the nineteenth century. In studies on the phenomenon of excess female mortality in this period in the Netherlands maternal mortality should be implicated as part of the problem. Maternal mortality cannot simply be seen as a natural female disadvantage which may be ignored. Further research at the individual level in which individually registered causes of death can be connected to life course and vital events information is required.[54] Such an approach will yield more insight into the precise connections between causes of death, nutritional disadvantages, childbirth and the female life course.

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54 Projects are underway to research individual level causes of death for the cities of Amsterdam and Maastricht at the Radboud Group for Historical Demography, Radboud University, and the Centre for Social History of Limburg, at Maastricht University.

Appendices

Table 1 List of causes of death and their categorization

	Violent, ill-defined and unknown
32	Violent death
33	Drowning
34	Unknown
32*	Suicide
33*	Suicide by drowning
34*	Sudden death
6	Dropsy
2	Debility, phthisis
1	Premature births, congenital malformations
	Tuberculosis and other respiratory diseases
18	Tuberculosis of the lungs & larynx
18*	Coughing up blood/haemoptysis , diabetes
21	Acute respiratory diseases (influenza/acute bronchitis, pneumonia, diseases of the pleural cavity)
22	Chronic respiratory diseases (diseases of larynx, pharynx, nasal cavity, and oral cavity; chronic bronchitis, asthma, other diseases of the lung)
	Other airborne infectious diseases
10	Smallpox
11	Scarlet fever
12	Measles
19	Croup
20	Whooping cough
25	Diphtheria
	Food and waterborn infectious diseases
7	Typhoid fever
26	Diarrhoea
26*	Dysentry
27	Cholera asiatica
27*	Cholera nostra
28	Acute diseases of the digestive system (appendicitis, peritonitis)
	Other infectious disease
3*	Syphilis

9	Intermittent fever
9*	Pernicious fever (febris interna pernicios)
16	Dementia, delirium tremens, acute & chronic brain diseases
17	Diseases of the spinal cord/paralysis
14	Convulsions, trismus, epilepsy
	Neoplasms & circulatory
5	Cancer
23	Circulatory diseases, rheumatism, arthritis
24	Diseases of the heart, aneurysm
	Other non-infectious diseases
3	Scrofula/rhachitis
4	Abscess, ulcer, gangrene, pyaemia, haemorrhage
6*	Scurvy
8	Continuous fever
13	Skin diseases
15	Apoplexy
29	Chronic diseases of the digestive system
30	Acute & chronic diseases of the genital-urinary system
	Maternal mortality
31	Puerperal diseases
31*	Febris puerperal

Table 2 Full general model: fixed effects models for Dutch maternal mortality 1875-1900 per region (42) and period (5), including imputed populations*

Maternal mortality /10,000 person-years lived (20-50 year old women)	(1)	(2)	(3)	(4)	(5) (first diff.)	(6)
Female TB	0.118***	0.0816***	0.155***	0.0649	0.0697	0.0823
mortality rate	(0.0191)	(0.0160)	(0.0376)	(0.0445)	(0.0431)	(0.0519)
Female chronic respiratory	0.325***	0.114	0.140	-0.0106	-0.0755	-0.0236
mortality rate	(0.0937)	(0.0620)	(0.111)	(0.116)	(0.130)	(0.137)
Fem. acute respiratory	0.132**	0.306**	0.170***	0.304***	0.269***	0.325***
mortality rate	(0.0654)	(0.0681)	(0.0582)	(0.0720)	(0.0824)	(0.0821)
Fem. Nutritional	0.400***	0.0891	0.466***	0.171	0.242**	0.191
mortality rate	(0.0883)	(0.0635)	(0.104)	(0.114)	(0.120)	(0.140)

Maternal mortality /10,000 person-years lived (20-50 year old women)	(1)	(2)	(3)	(4)	(5) (first diff.)	(6)
Male TB	-0.0598**	-0.0845**	0.0492	-0.0187	0.0288	0.0383
mortality rate	(0.0262)	(0.0214)	(0.0543)	(0.0464)	(0.0483)	(0.0541)
Period controls	No	Yes	No	Yes	Yes	Yes
Regional controls	No	No	Yes	Yes	Yes	Yes
Regional time trend	No	No	No	No	No	Yes
Observations	210	210	210	210	168	210
R-squared within	–	0.305	0.544	0.616	–	0.774
Between	–	0.486	0.229	0.326	–	0.006
Overall	0.430	0.325	0.372	0.505	0.242	0.307
R-squared adj.	–	–	–	–	0.204	–
F statistic	34.88	[69.20[1]]	39.80	22.14	3.84	–
p-value F test	0.000	[(0.001)]	0.000	0.000	0.003	–

* Robust standard errors in parentheses *** p<0.01, ** p<0.05, * p<0.1.
1 Model excluding chronic respiratory mortality rate-current model includes too many covariates.