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Height and the disease environment of children: The association between mortality and height in the Netherlands 1850–1940

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Abstract

Height and infant mortality are both considered health indicators of a population, yet they tend to be much more strongly correlated in high-income, low-mortality populations. This article shows that infant deaths are not representative of the health of survivors as it relates to height because breastfeeding practices shield them from part of the disease environment. Instead, child mortality rates, especially from food and waterborne diseases, capture the disease load that is associated with lower heights better. The period of this study is 1850–1940, with a focus on 1875–1900, as the Netherlands underwent major health and wealth transitions. Individual conscription heights from the Historical Sample of the Netherlands as well as municipal conscription statistics are used. The article takes a diachronic approach to examine how various health indicators have developed over time. The start of the upward trend in heights and the improvement of child mortality rates coincided in four Dutch regions, whereas infant mortality rates followed a different trajectory. Bivariate maps are used to identify municipalities in which infant and child mortality did not correlate. This study adds to both the understanding of heights as a health indicator

and local breastfeeding practices in the nineteenth-century Netherlands.

KEYWORDS
breastfeeding, early life conditions, height, mortality, scarring and selection

Human stature has equipped economic historians with a measure of living standards of populations in the past. Height reflects the bodily condition as a result of nutritional intake, which is affected by economic measures such as real wages. However, stressors on the body, such as childhood diseases, divert energy away from growth and therefore dilute the clear association between income and height.[1] This study will critically examine the most common proxy used to control for the historical disease environment: infant mortality. This examination is needed because empirical evidence for the theoretical relationship between heights and diseases tends to be weaker in historical contexts.[2] With this study, a better understanding is gained regarding how heights are a health measure of historical populations.

Historical heights reflect the health status of those who survived childhood, or, at any rate, those who lived until the age at which they were measured. Historical demographers, however, tend to focus on the most unhealthy members of a population, the dead. In the absence of historical morbidity statistics, mortality statistics provide a good proxy for the severity of the disease environment. The most sensitive indicator is considered to be infant mortality, as infants are the most vulnerable members of a population. While it may be true that infant mortality is the most sensitive indicator of the disease environment in terms of mortality, this article contends that it is not the best indicator of survivors' health in historical, high-mortality contexts.

Infant mortality impacts height in two ways (see figure 1). First, infant mortality has a direct effect on height through selection of the cohort these rates apply to. If shorter height, or factors that are associated with a shorter height, result in a higher chance of infant death, the shortest of a cohort are selectively culled, and therefore survivors are on average taller. Second, chronic or repetitive illnesses may impede growth, resulting in shorter survivors as a result of scarring. Nevertheless, childhood morbidity is seldom recorded, and historians use infant mortality rates of subsequent cohorts as a proxy for the morbidity rates of older children. Hatton, among others, uses these different pathways to disentangle scarring and selection effects. The primary argument for this strategy is that respiratory and gastrointestinal infections, which frequently resulted in infant death, could have caused chronic illness when experienced by older children.[3]

Nevertheless, this argument can be contested if we closely analyse the health transition that occurred in various western European countries from the second half of the nineteenth century on. In the course of this transition, life expectancies increased and people grew taller. However, infant and child mortality rates did not drop concurrently. Within the Netherlands, the declines in post-neonatal and child mortality did not start at the same time. Post-neonatal mortality began to decline rather late, with a modest decline beginning in 1871 and accelerating in 1905. This compares with child mortality, with the rate beginning to decline over a decade earlier, in 1859, and

[1] For example: Harris, 'Anthropometric history'; Steckel, 'Recent developments'.

[2] Bozzoli, Deaton, and Quintana-Domeque, 'Adult height', show this on a global scale.

[3] Hatton, 'Health of survivors'; Sharpe, 'Chronic childhood disease'; Schneider and Ogasawara 'Disease and child growth'.

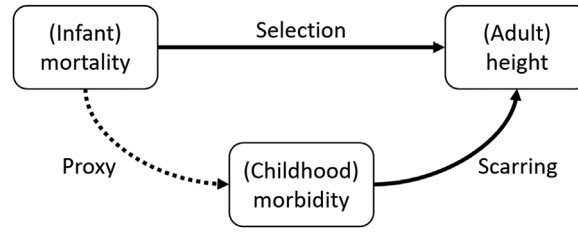


FIGURE 1 Theoretical association between infant mortality and height based on the mechanisms of scarring and selection.

accelerating from 1888.[4] For instance, England exhibits a similar trend, with infant mortality only beginning to decline at the turn of the twentieth century, while child death rates started to decline in the late nineteenth century.[5]

There are two interlinked explanations as to why these trends were different. First, the start of the mortality decline in the Netherlands was significantly influenced by the decline in respiratory tuberculosis mortality.[6] This affected child mortality more than infant mortality, as infants are more susceptible to digestive diseases.[7] This mortality decline was the result of improved living standards rather than medical interventions.[8] Infant mortality rates only significantly decreased as a result of reducing diarrhoeal infections that became a significant factor in the Netherlands' mortality decline at the turn of the century.[9] Therefore, secondly, and related to the first observation, in the absence of medical care, breastfeeding is positively associated with, or even crucial for, infant survival chances.[10] Newborns that are regularly breastfed benefit from stimulation of the immune system, receive antibodies that contribute to anti-inflammatory responses, and obtain receptors that prevent microbes attaching to the mucosa, which all explain why human milk reduces the chances of death from infectious diseases for infants.[11] Furthermore, those infants are shielded from tainted water or food. Infants who are artificially fed lack the protective qualities of mother milk and are more likely to contract infections and die prematurely as a consequence of factors such as infected milk or unsanitary water.[12] The nutritional value of breastmilk is relatively independent of the nutritional status of the mother.[13] Hence, because breastfeeding practices impact an infant's survival, newborns would have been less susceptible to social conditions in the past than other segments of the population. The act of breastfeeding is unrelated to hygienic conditions, food quality, or medical assistance that affect childhood morbidity and height outcomes.[14] Breastfeeding was an important resource for parents to mitigate the disease environment's impact

[4] Wolleswinkel-van den Bosch, Van Poppel, Tabeau, and Mackenbach, 'Mortality decline', post-neonatal with an annual growth rate between 1871 and 1905: −1.2%, and after 1905: −4.3%, child mortality 1859–90: −1.7%, and after 1888: −3.0%.

[5] Floud, Wachter, and Gregory, *Height, health and history*.

[6] Wolleswinkel-van den Bosch, 'Epidemiological transition', p. 105; for example, England see: Floud et al., *Changing body*, pp. 146–7.

[7] Although after six months the susceptibility to airborne infections increases. (Galley and Woods, 'Distribution of deaths'.)

[8] McKeown, *Rise*.

[9] Wolleswinkel-van den Bosch, 'Epidemiological transition', p. 105.

[10] For example, Garðarsdottir, *Saving the child*.

[11] Duijts, Ramadhani, and Moll, 'Breastfeeding protects'.

[12] Walhout, 'Is breast best?'.

[13] For example: Prentice, 'Human milk', only with the exception of extremely deprived mothers.

[14] Van Poppel et al., 'Three Dutch regions'.

on infant health prior to 1900, which they could not apply to their older children.[15] Even after 1900, babies born to mothers who stayed at home had a higher survival rate than babies born to mothers who went to work, probably because they received more frequent and intensive breastfeeding.[16]

Recent research shows that in the nineteenth-century Netherlands, geography, such as province, urban centre, or neighbourhood, explains mortality inequalities better than class, despite the fact that the two are often linked.[17] Therefore, regional variations in infant death rates may be linked to regional variations in breastfeeding practices. Consequently, older children's mortality may differ from local infant mortality rates, particularly if nursing protects infants from deadly gastrointestinal infections. This article will show how infant mortality and childhood mortality rates differed in terms of regional variance in the last quarter of the nineteenth-century Netherlands. This is a strong sign of shielding through breastfeeding, especially if child mortality is high compared with infant mortality. In those regions, child mortality may be more representative of health in early life. Heights may therefore correlate more strongly with child mortality rates, as the environment that kills children can stunt survivors during any stage of their early life.[18] Thus, childhood mortality may be a better proxy for childhood morbidity than infant mortality.

Outside of the safety of breastfeeding, however, extrinsic factors might result in extremely high infant mortality rates. Infant mortality rates of over 300 per 1000 births were recorded in the west of the Netherlands due to endemic malaria, salinization, and contaminated water, as well as local outbreaks of smallpox and cholera in densely populated areas. During the period under study, preventing illnesses rather than curing them would have been a more successful strategy for parents to keep their offspring healthy. Therefore, only households with very protective parenting practices such as a strict hygienic regime, high food quality, less overcrowding, access to smallpox vaccination, and good sanitation, in other words, shielding and protective households, would have been able to protect their offspring against these high rates of over 300 deaths per 1000 births. As a result, a selection effect occurred on the basis of factors that are also associated with a taller average stature rather than a shorter average stature.[19] In a Swedish population, Quaranta discovered indications of selection.[20] Those that were exposed to an epidemic while still in utero were selectively culled and the survivors of such exposure had lower mortality risks well into their 30s. For the Dutch, it was discovered that children who lost their mothers in infancy had such high death rates that there was no significant association with a lower height for survivors of maternal mortality.[21] These findings imply that, at high levels of mortality, selection can at least mask the scarring effect of illnesses, resulting in the absence of a negative correlation with height.[22] This might provide an alternative explanation of why infant mortality rates and height are not strongly correlated in historical populations. The higher mortality rates, the more present this masking can be. This is why, apart from the relative rate of infant and child mortality, the absolute mortality values are also taken into account in this study.

[15] A good example can be found in: Van Poppel, Schellekens, and Liefbroer 'Religious differentials'.

[16] Janssens and Pelzer 'Bad mothers'.

[17] Muurling, Riswick and Buzasi, 'Smallpox epidemic'; Van den Boomen, 'Born close to death'.

[18] Akachi and Canning, 'Conflicting evidence'; Thompson, Quanjer, and Murkens, 'Grow fast'.

[19] Van Dijk, 'Early-life mortality clustering', showed that certain cultural practices unrelated to socio-economic status can protect the household members.

[20] Quaranta, 'Scarred for life'.

[21] Quanjer, Van Dijk, and Rosenbaum-Feldbrügge, 'Short lives'.

[22] Bozzoli et al., 'Adult height'; Spijker, Cámara, and Blanes, 'Health transition'.

Taken together, this article will test the hypothesis that infant mortality rates are not the best proxy for the health of survivors in historical settings. It will argue that childhood mortality rates and conscription height are better measures for health in historical settings, and as a result, also show how conscription height is reflective of childhood health in the nineteenth-century Netherlands. Using the contrast between infant and child mortality rates, it is possible to assess whether external or breastfeeding practices were associated with the secular trend in heights.

I | LINK BETWEEN HEALTH AND HEIGHT

The main point of this paper, that infant mortality is not the best proxy for the health of survivors, is based on a number of studies that found no or at most a very weak association between height and infant mortality rates in settings with an infant mortality rate of over 100 per 1000 births.[23] Such extreme rates are only found in low-income countries and historical contexts. The absence of the association contrasts with results from western and southern European countries in the second half of the twentieth century, where a strong association between height and infant mortality was found.[24] During those decades, these modern countries still experienced an upward trend in heights.[25] However, nutrition was relatively stable, as most diets were likely to have reached an optimal upper limit. Therefore, the upward trend in heights during these decades can be attributed to other factors that divert energy from growth such as diseases. Most plausible, in these modern high income societies, medical interventions had a stronger impact on infant survival than breastfeeding practices. Hence, in these high income societies, infant mortality is a good proxy of childhood morbidity, as breastfeeding was less of a mitigating factor.

The absence of a clear association between infant mortality rates and heights in high-mortality contexts in the literature does not imply that there is no association between childhood morbidity and height. Rather, infant mortality may not be the correct proxy for childhood morbidity, as there is evidence that shows that childhood morbidity, through scarring, is negatively associated with height outcomes, even in high mortality regimes. Particularly, diseases that impact the digestive system likely have a strong, direct impact on height.[26] For example, chronic diarrhoea drains energy from the body while also reducing the body's ability to absorb nutrients.[27] Furthermore, the eradication of hookworm has been found to positively influence heights.[28] Diseases with a less apparent link to nutritional intake, meanwhile, appear to be associated with height outcomes as well. These diseases include pneumonia and tuberculosis.[29] Bailey et al. also found that atmospheric pollution was associated with a shorter stature.[30] These studies indicate that a reduction in airborne infectious diseases may also be associated with a taller stature. It is to be expected that infants can be shielded from food and waterborne diseases through breastfeeding, but may

[23] De Oliveira and Quintana-Domeque, 'Stature in Brazil'; Akachi and Canning, 'Conflicting evidence'; Öberg, 'Direct effect'; Bozzoli et al., 'Adult height'; Stolz et al., 'Living standards'.

[24] Schmidt, Jørgensen, and Michaelsen, 'Height of conscripts'.

[25] Hatton and Bray, 'Long run trends'.

[26] Checkley et al., 'Effects'; Kossmann et al., 'Undernutrition'.

[27] Scrimshaw and SanGiovanni, 'Synergism of nutrition'.

[28] Bleakley, 'Disease and development'.

[29] Bozzoli et al., 'Adult height'; Baten and Murray, 'Heights'.

[30] Bailey, Hatton, and Inwood, 'Atmospheric pollution'.

be very vulnerable to airborne diseases, even in their first year of life.[31] Taking the cause of death into consideration will help to understand the underlying mortality rates' relationship to height.

Although various diseases can hamper growth, it remains unclear if there are particular critical windows in childhood when the scarring effect is the strongest. When growth velocity is highest, the scarring effect of diseases is expected to be strongest, as there is more growth to be hampered, yet various studies found different critical windows. Hatton found that an accumulation of the disease environment, measured between the ages of two and six years, was associated with a shorter height.[32] Depauw and Oxley moved this accumulation effect to span the years of adolescence during which the pubertal growth spurt occurs, and found a negative association between infant mortality rates and heights of Belgian prisoners in the nineteenth century.[33] Already, similar results were found for the nineteenth-century Netherlands.[34] Schneider and Ogasawara found an effect on growth of Japanese children between the ages six and eleven years.[35] Next to the debate on timing, there is no consensus on how energy demanding diseases should be, and for what period they have to affect the body to have a clearly discernible effect on heights. What complicates the debate around critical windows even more is that after a short period of illness, the body might be able to catch up with its previous growth trajectory.[36]

Apart from the critical windows, short periods of illness might not have a clear effect on height, for instance, there is no obvious link between smallpox and short stature, as patients, after a short period of illness, typically develop lifetime immunity.[37] Diseases that are chronic or repetitive in nature are more likely to scar the body in terms of height as there is less time to recover and catch up with the original growth trajectory. It might even be plausible that the variation of critical growth windows found by earlier studies is the result of the same repetitive diseases reoccurring at different stages of childhood.

Considering these earlier studies, height as a cumulative measure of childhood diseases seems to produce the most robust negative association. This is why the timing issue is not extensively studied in this paper, but alternative age windows for the different analyses are provided in figure 2a–c and the appendix.[38]

II | THE DUTCH CONTEXT

The Netherlands witnessed a health transition from high mortality rates and frequent epidemics to low mortality rates between 1850 and 1950. The health of children in particular improved significantly. As previously stated, ecological circumstances in the west of the Netherlands resulted in extremely high death rates. A recent study using nineteenth-century archaeological remains of young children from the Beemster region concluded from the isotopic evidence that breastfeeding

[31] Galley and Woods, 'Distribution of deaths'.

[32] Hatton, 'Health of survivors'.

[33] Depauw and Oxley, 'Toddlers, teenagers'.

[34] De Beer, *Voeding gezondheid en arbeid*.

[35] Schneider and Ogasawara 'Disease and child growth'.

[36] Thompson et al., 'Grow fast'.

[37] Muurling et al., 'Smallpox epidemic'; Oxley 'Pitted'.

[38] The online appendix contains different analyses for different age windows in sec. A.3 (see tables) and A.7.

was less common or even absent in the west, which contrasts to what was previously assumed.[39] Therefore, the shielding mechanism for infants may have been absent as well. Previously, it was widely recognized that Roman Catholic standards discouraged breastfeeding, particularly in the south of the country.[40] Hygiene awareness and breastfeeding practices increased in the second part of the nineteenth century, initially in the north-west and then slowly spreading towards the south-east.[41] Therefore, apart from the external conditions, lack of breastfeeding would have resulted in high infant mortality rates in the west and south. For the current research period, Walhout demonstrated that the number of infant deaths from diarrhoea increased in the southern province of Brabant between 1875 and 1900 due to less frequent breastfeeding.[42] Still, a significantly earlier drop in these death rates is anticipated for the west.

From a medical standpoint, a group of young doctors made strides in boosting sanitation and hygiene awareness, particularly in western towns during the same period.[43] There was minimal access to modern medical treatments in the rural east and south.[44] In addition, awareness was created for child labour, using heights of young factory workers, which resulted in the enactment of strict labour laws. As a result, less energy was diverted to stress and labour for young children which will have enhanced net nutrition. The Netherlands, however, was a late adopter of industrialization, and hence had fewer dark satanic mills than, for example, the United Kingdom.[45] Therefore, the negative effects of industrialization on height are expected to be less pronounced in the Dutch context. Finally, a gradual increase in real wages combined with a significant gender pay gap drove women from the labour market, resulting in a sharp divide between breadwinners and homemakers.[46] The rising real wages might not have compensated fully for the labour market withdrawal of women, but we still see improved living conditions within households, as they could produce non-market products, such as a clean home and a healthy meal.[47] These developments also contributed to the upward trend in heights within the Netherlands.[48]

This study focuses on trends, particularly health trends, associated with heights. As a result, regional contextual circumstances are not included in the scope of this article. Nevertheless, to provide some additional support for the interpretation of the results, a summary of contextual circumstances around 1875 is presented in the appendix.[49] These factors are not included in this study's analyses as covariates, because not all variables can be tracked over time. As a result, including them in a diachronic approach is unfeasible. However, the variables in the appendix go into deeper detail to corroborate the previously indicated context.

[39] Waters-Rist, De Groot, and Hoogland, 'Isotopic reconstruction'.

[40] For example: Walhout, 'An infants'graveyard'.

[41] Hofstee, *Korte demografische geschiedenis*.

[42] Walhout, 'Is breast best?'.

[43] Tassenaar, 'Antropometrie als instrument'.

[44] Van den Boomen, 'Born close to death'.

[45] Van Zanden and Van Riel, *Nederland 1780–1914*.

[46] De Vries, *Industrious revolution*; Boter, 'Dutch divergence'.

[47] Mokyr, 'More work for mother'.

[48] Quanjer and Kok, 'Homemakers and heights'.

[49] See sec. A.8. of the appendix: contextual data for the Netherlands 1875, for the contextual circumstances of this study. In this section, contextual embedding of this study can be found; however, these variables do not confound the association between height and mortality.

III | DATA

This study makes use of a patchwork of several datasets. This is because both height and mortality data are only available for specific time periods at specified levels of aggregation. An overview with exact details of the underlying datasets is provided in the appendix.[50]

Two height samples are used for this article, one at the individual level and one at the municipal level. The first consists of individual heights taken from relatively unbiased conscription records that were linked to the historical sample of the Netherlands (HSN).[51] The HSN allows for a complete life course reconstruction of all research persons (RPs), including a household reconstruction. Furthermore, by the creation of the Male Kin Heights (MKH) database, heights of brothers were added, allowing for within-family comparison of the disease environment.[52] The MKH is biased towards larger families and to the provinces of Noord-Holland and Friesland, in which the digital linkage of conscripted brothers was feasible.[53] Another limitation is that by using conscription records, the analyses are limited to men only.

The individual-level height data provide an excellent source to examine household factors. However, the dataset does not provide enough observations to calculate averages at the municipal level. This is not ideal, as mortality rates are calculated at the municipal level. It is therefore assumed that these households are equally exposed to those external municipal rates. This assumption can easily be refuted, as household factors such as housing quality, sanitary access, clean drinking water, overcrowding, and breastfeeding practices affect mortality rates within the household that almost certainly differ compared with the municipal average. The municipal mortality rates should be considered a crude proxy for the disease environment that is associated with shorter stature. As a result, the association might be underestimated and therefore weaker in the models. To overcome this problem, a second height sample is used independently from the HSN sample.

The second height sample used in this study is based on average height per municipality, which, despite the higher level of aggregation, allows us to obtain a national coverage of heights in the Netherlands and allows for a study of the association with the municipal mortality rates that were aggregated at the same level. Aggregated conscription data per municipality were gathered for the years 1863–75, which consist of the number of conscripts per height group, for example, the number of conscripts between 165 cm and 166 cm.[54] The groups below 150 cm and above 180 cm were open-ended. The collected heights of the HSN were used to calculate group means, allowing an average height per municipality to be calculated. For the period 1921–5, municipal height data are also available.[55] Unfortunately, these heights were rounded to whole centimetres, but there is no reason to expect that this might have biased the results in a particular direction. The two height samples will be used separately in this study.

Three datasets are used for the mortality data. Utilizing the same methodology as Van Poppel et al., the HSN is used to determine neonatal (<28 days), post-neonatal (28 days to 1 year) and

[50] See sec. A.1. of the online appendix: sources on height and mortality used in this study.

[51] Quanjer and Kok, 'Drafting the Dutch'; Mandemakers, 'Historical sample'; HSN release 2010.01 and 2018.02, Dutch conscripts were measured in millimetres which is why all results are reported in millimetres.

[52] HSN release 2019.05.

[53] A logistic regression on the potential biases can be found in the appendix, sec. A.2.

[54] [Anon], 'Ligting', 'Loting' and 'Statistiek'.

[55] Van den Broek, 'Anthropologische samenstelling'.

child (aged 1–5 years) mortality rates for 5-year periods at the provincial level.[56] At the municipal level, only infant (aged <1) and child (aged 1–5 years) mortality rates are available for 5-year periods between 1875 and 1900.[57] Fortunately, these statistics also include the cause of death as well. Therefore, mortality from airborne infectious diseases (e.g. smallpox, measles) as well as food- and water-borne diseases (e.g. diarrhoea, cholera) is used in the analyses. These causes are categorized in accordance with Wolleswinkel-van den Bosch.[58] Given that the causes were determined, registered, and categorized by doctors and clerks from the nineteenth century, the statistics are impeded by the outdated medical understanding of the time.[59] This might mask a clear association, but these statistics are currently the best available. Finally, infant mortality rates at the municipal level for multi-year periods are used outside the 1875–1900 window.[60]

IV | METHOD

The advantage of analysing the association between mortality and height in a historical context is that change over time is considered. Anthropometric studies typically take either a diachronic (across time) or a synchronic (across regions) approach, capturing diverse determinants as a result. For example, a synchronic approach revealed that milk consumption is significantly associated with regional height variance. However, a diachronic approach showed that the beginning of the upward trend in Dutch heights corresponded with a decrease in per capita milk consumption.[61] This study mainly uses a diachronic approach, as it is interested in the development of the various indicators.

The methods used are determined by data availability. They are a compromise between low levels of aggregation and high representativeness. Although this study takes a diachronic approach, a more synchronic approach is initially utilized to determine if indeed a low correlation is found between height and infant mortality when mortality rates are high. Scatterplots, combined with a Pearson's correlation coefficient, are used to establish the association between height and mortality in the relatively high mortality context of 1875 versus the relatively low mortality context of 1925 at the municipal level. Within these graphs, distinct regions are used to assess whether possible contextual factors can be determined.[62]

Since region was a strong determinant of mortality differences, next to these scatterplots, bivariate maps are used to find which regions deviate from the expected negative correlation between height and mortality. The advantage of bivariate maps is that values are compared relative to other values of the same variable (along the *x*-axis) and relative to the value of a second variable (along the *y*-axis). Furthermore, these maps take a perfect correlation as a starting point. Plotting only the residuals when there is no correlation would result in a map of the

[56] Van Poppel et al., 'Three Dutch regions'.

[57] Van den Boomen, 'Born close to death'.

[58] Wolleswinkel-van den Bosch, 'Epidemiological transition'.

[59] Van den Boomen, 'Born close to death', chapter: 'Imperfections in statistics'.

[60] Ekamper and Van Poppel, 'Zuigelingensterfte'.

[61] Knibbe, 'Levensstandaard in Nederland'; Groote and Tassenaar,'Living standards in a dairy region'.

[62] The provinces of Groningen and Friesland are grouped as north; Drenthe, Overijssel, and Gelderland are east; Noord-Holland, Zuid-Holland, Utrecht, and Zeeland are west; and Noord-Brabant and Limburg are south. In the appendix, sec. A.3. the scatterplots per region can be found.

y variable. Since there is a risk of a very low correlation in 1875, bivariate maps are the better option.

The association with child mortality is also depicted in a bivariate map because the key hypothesis is that infant mortality is not a useful health proxy in a high mortality scenario. A map showing the link between height and child death rates is not feasible because child mortality data at the municipal level are only available for the years 1875–1900. This map can, however, demonstrate whether the areas that differ from the height and infant mortality association before 1875 also differ from the anticipated positive association between infant and child mortality between 1875 and 1880.

After that, a diachronic approach is used to investigate whether height changed concurrently with neonatal, post-neonatal, and child mortality in four Dutch regions. In particular, the turning point of the various trends is studied.[63] Did the various trends start to improve at the same time? Any discrepancy in the starting points gives us a clue as to how these indicators reflect health in history. The individual heights can be used to calculate height trends for the four Dutch regions to find the starting point for the upward trend in heights.

To test these changes in a linear regression model, only the period 1875–1900 is available. The provincial or regional mortality rates from the HSN cannot be used, as the differences within these regions are too great to use the HSN's provincial mortality rates as a proxy for the disease environment.[64] Therefore, municipal infant and child mortality rates that are only available for 1875–1900 are used. For this same period, height is only available at an individual level. Hence, as mentioned in the data section, municipal mortality rates will be linked to individual heights as a proxy for the disease environment. Since cause of death is available for the period as well, it is possible to establish the best proxy for the disease environment that is negatively associated with heights.

Finally, if child mortality is more closely associated with height in the past, this might indicate that the period between the age of one and five years is a critical stage in which health conditions affect growth, and ultimately terminal height. However, it might also be about child mortality being the most sensitive proxy for the disease environment that affects height regardless at which age in early life this exposure proxy is used. Therefore, the final model compares the heights of brothers who experienced a sibling death at a different age in their childhood in a family fixed effects model. This helps to identify the crucial stages in which the disease environment has an impact.

V | RESULTS

First, the correlation between height and infant mortality is examined in two periods. Second, bivariate maps are presented to identify regions that deviated from the expected association. Third, figures show the development of mortality rates and height over time. Finally, the regression models test the association between mortality and height.

Figure 2a shows the association between infant mortality rate and conscription height for the cohorts who are considered the last to be born before the epidemiological transition. These cohorts experienced high levels of infant mortality during their early life. Infant mortality rates were particularly high in the western part of the Netherlands, but considerably lower in the northern

[63] Similar to Wolleswinkel-Van den Bosch et al., 'Mortality decline'.

[64] The regional variation in rates is shown in the appendix, sec. A.4. and A.8.

regions. Overall, there is a −0.12 Pearson correlation between the two variables. For the north, the correlation was −0.41.[65] The difference in the correlation coefficient when infant mortality rates from the years 1861–74 are used was rather small (−0.06). Only for the western regions did the correlation based on infant mortality rates experienced during infancy of −0.21 decrease to −0.01 for the rates they experienced during their teenage years.

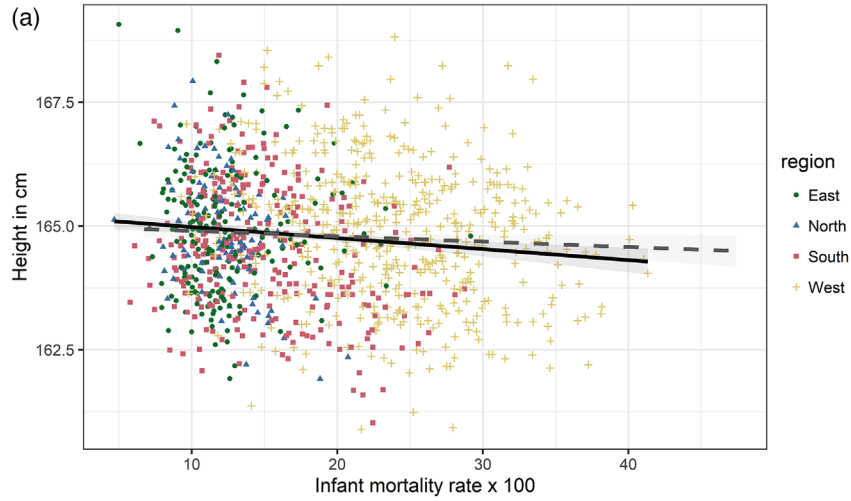
Although the epidemiological transition did not end in 1925, cohorts who reached conscription heights in 1925 experienced significantly lower infant mortality rates during their early lives. This is represented in figure 2b, which shows a Pearson correlation of −0.58 [−0.60 if the infant mortality rate (IMR) of 1914–23 was used]. The provinces in the south were lagging behind in their transition, as seen by higher infant mortality rates. For this region alone the correlation of −0.06 was lower than for the nation as a whole, and still similar to the pre-transitional stage of 50 years earlier.

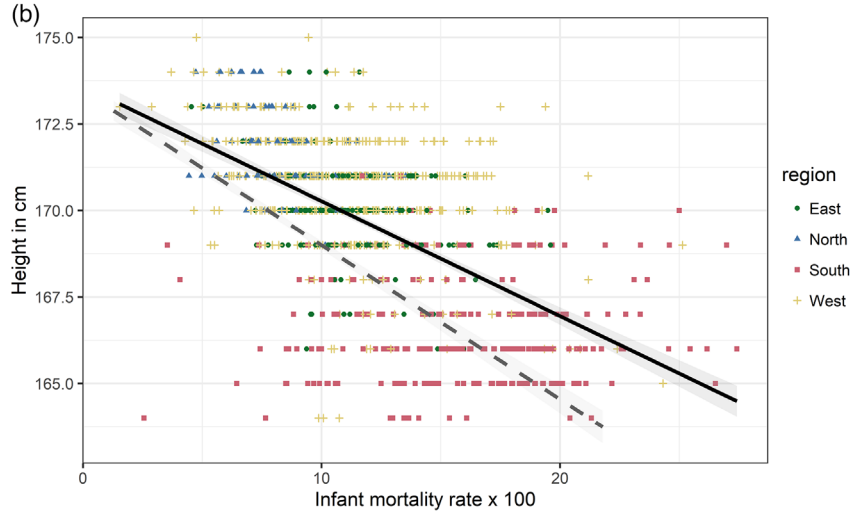
Since this article is interested in how these two variables developed together over time, figure 2c shows how these figures changed between 1875 and 1925 per municipality. This figure shows the most clear distinction between the regions of the three figures. The west is notable for its rapid decrease in death rates. The south stands out for its more modest trend in height. The correlation is highest in the north (−0.68) and lowest in the west (−0.04).

To explore the correlation further, figure 3 shows bivariate maps that combine both infant mortality rates and conscription height per municipality. Because the regression line for 1875 is virtually horizontal (see figure 2a), depicting the residuals would result in a map that only shows height differences. These maps illustrate whether areas exhibit the hypothesized association between height and infant mortality rates. The municipalities that do meet this expectation are coloured using the scheme along the diagonal line from the left top corner (yellow, tall stature, low IMR) to the right bottom (blue, short stature, high IMR) of the legend. Because the correlation was determined to be high in 1925, most municipalities in the right panel match the colour scheme along this diagonal line. However, in 1875, the white and black areas deviated from the expected correlation. The white band in the heart of the country's eastern half, along with a small section in the south, has low infant mortality rates and low conscription heights. The black areas in the country's mid-west, on the other hand, combine high infant mortality rates with taller stature. This is consistent with the hypothesis that in high-mortality contexts, infant mortality rates are not reflected in heights.

Unfortunately, no child mortality rates are available at the municipal level prior to 1875. Nonetheless, the available rates between 1875 and 1880 may represent the municipal pattern in child mortality rates before 1875 to a great extent, even though certain places did experience a decline in death rates over time. Figure 4 shows that the locations in 1875 that were regarded as outliers to the expected association between height and infant mortality can be explained in some part by the disparity in infant and child mortality rates. In contrast to the previous maps, figure 4 is intended to show a positive correlation, as evidenced by the diagonal line in the legend changing from white to black. The outliers in the east can be found with low infant mortality rates followed by high child mortality rates (in yellow). In comparison with their western counterparts, infants in these municipalities tend to survive their first year of life. They do, however, die more frequently as a result of the adverse conditions they face as children. This might indicate that certain measures, for example, breastfeeding practices, protect infants from an unhealthy disease environment. The blue areas in the mid-west, in contrast, have high infant mortality and low child mortality. This

[65] Sec. A.3. of the appendix shows the figures with a regression line per region separately. In addition, the correlation coefficients are provided in a table.





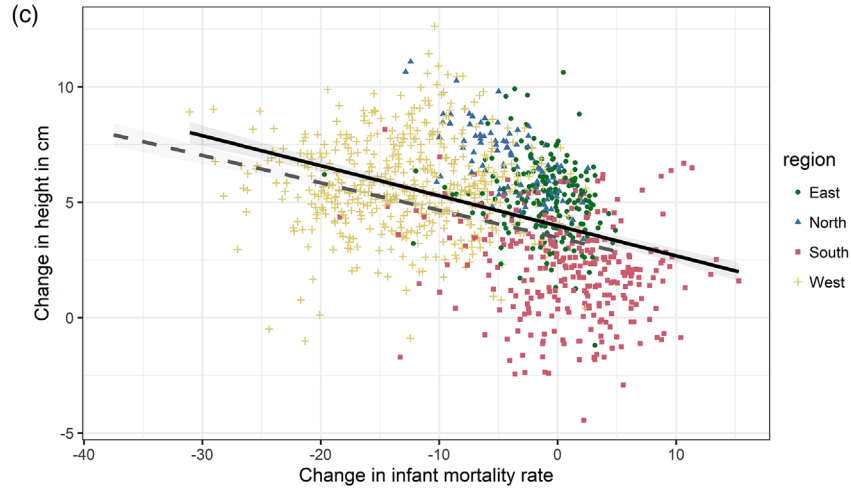


FIGURE 2 (a) Scatterplot on the association between infant mortality rate per 100 births 1840–60 and average conscription height per municipality (measurement years: 1863–75). *Sources*: Ekamper, P. & van Poppel, F. (2008); N.d. (1869;1873;1877) ligting voor de nationale militie 1863–75.
Note: Next to the regression line a dashed line shows what the association would have looked like if infant mortality rates of 1861–74 would have been used. To place these figures in a modern perspective, the United Nations reported that in 2012 Sierra Leone was the last country to reach an infant mortality rate below 10 per 100. (b) Scatterplot on the association between infant mortality rate per 100 births 1904–13 and average conscription height per municipality (measurement years: 1921–5). *Sources*: Ekamper, P. & van Poppel, F. (2008); Van den Broek (1930). *Note*: Next to the regression line a dashed line shows what the association would have looked like if infant mortality rates of 1914–23 would have been used. (c) Scatterplot on the association between the change in infant mortality rate per 100 births between the periods 1840–61 and 1904–13 and the change in conscription height (measurement years: 1863–75 to 1921–5). *Sources*: Ekamper, P. & van Poppel, F. (2008); N.d. (1869;1873;1877) ligting voor de nationale militie 1863–75; Van den Broek (1930). *Note*: The change is calculated between the periods given in the title, not within. Furthermore, next to the regression line a dashed line shows what the association would have looked like if infant mortality rates of 1861–74 and 1914–23 would have been used. [Colour figure can be viewed at wileyonlinelibrary.com]

pattern may be the result of more favourable conditions for children compared with infants, or perhaps this pattern is related to diseases that only affect the mortality of infants. Alternatively, the infant mortality rates could also selectively cull unhealthy infants (or infants in an unhealthy or unprotective household). The surviving children who were exposed to the same diseases (or grew up in a protective household) were selected on the basis of their health (or their parents' ability to provide a healthy home) and as a result had a higher chance of survival. Regardless of the mechanism, these maps suggest that child mortality rates in particular areas might predict

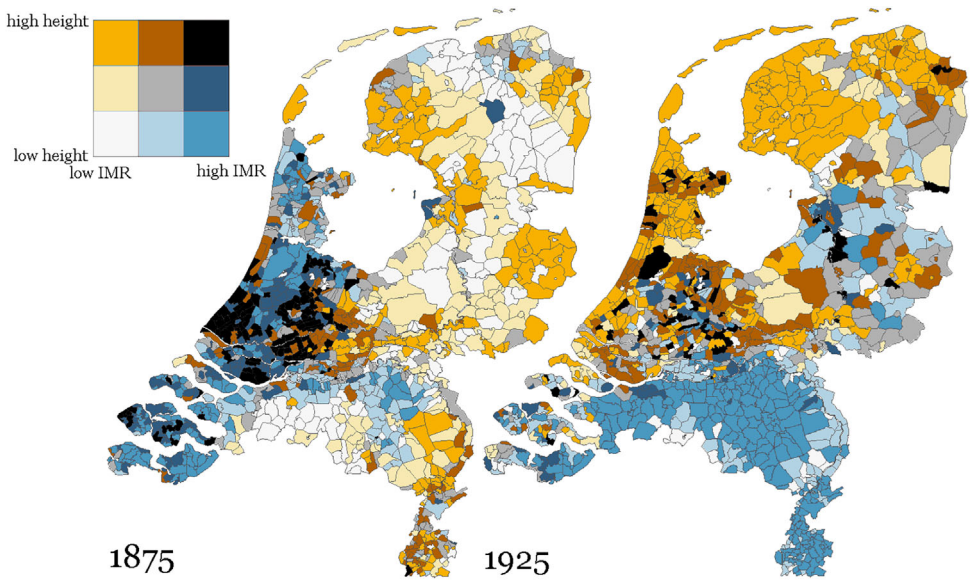


FIGURE 3 Bivariate maps on the association between conscription stature (measurement years: 1863–75) and infant mortality rate (1840–61) in the left panel, and conscription stature (measurement years: 1921–5) and infant mortality rate (1904–13) in the right panel in the Netherlands. [Colour figure can be viewed at wileyonlinelibrary.com]

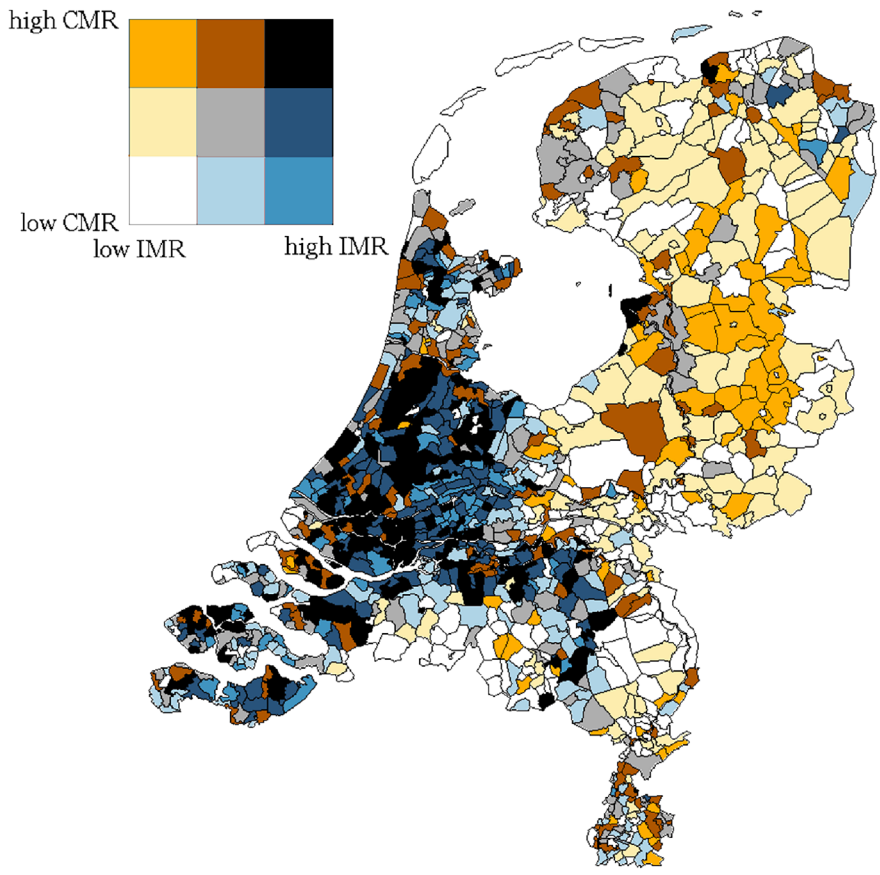


FIGURE 4 Bivariate map on the association between infant mortality rate (ages 0–1) and child mortality rate (ages 1–5) per 1000 inhabitants in the Netherlands 1875–80. [Colour figure can be viewed at wileyonlinelibrary.com]

height in 1875 better than infant mortality rates.[66] Especially in regions where infant mortality rates are not closely associated with heights, child mortality rates would help to explain why that is the case.

So far, only the regional variation in infant and child mortality has been examined. However, these inequalities did not remain constant over time, and researching their trend will assist in understanding how the health transition in the Netherlands is linked to the secular rise in heights. Figure 5a provides the trends for neonatal, post-neonatal, and child mortality in four Dutch regions between 1815 and 1925. The figures show how different regions differ not only in terms of absolute mortality rates, but also in terms of mortality transition trends.

Except for the west, child mortality rates began to decline roughly 20 years before post-neonatal rates. Furthermore, child mortality was higher than post-neonatal mortality in the north, south, and east in the early part of the nineteenth century. The disparity is particularly noticeable in the east, as shown in figure 4. In the west, both rates were the highest in the country until at least 1890,

[66] Despite the mismatch in period, a bivariate map between height and child mortality is provided in sec. A.5. of the appendix. It should be taken into consideration that child mortality rates reflect diseases after conscription heights used for the map were reached.

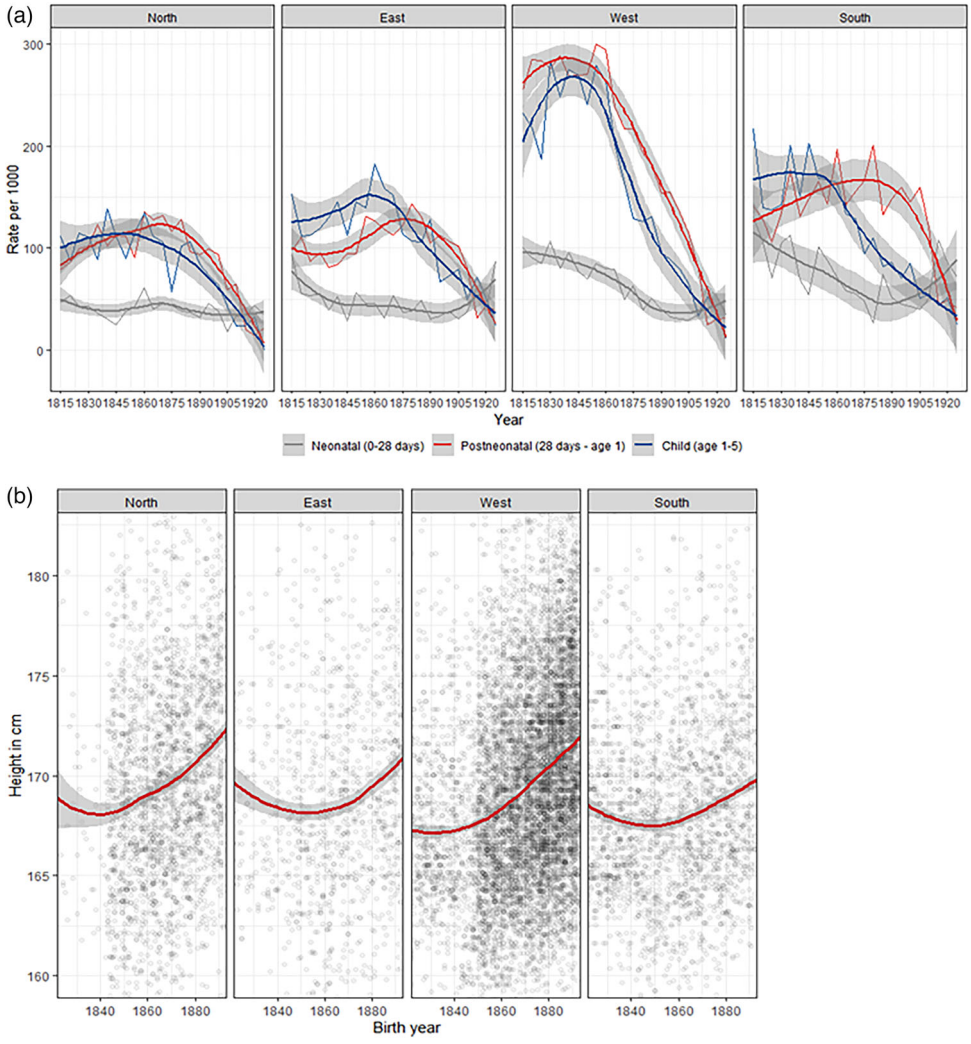


FIGURE 5 (a) Neonatal, post-neonatal, and child mortality rates in four Dutch regions 1815–1925. *Source*: Historical sample of the Netherlands. *Note*: Figures with the mortality rates per province and figures with the full height trend per province can be found in the Appendix, section A.4 (online only). (b) Height trend in four Dutch regions. *Source*: Historical sample of the Netherlands. *Note*: Figures with the mortality rates per province and figures with the full height trend per province can be found in the Appendix, section A.4. (online only). [Colour figure can be viewed at wileyonlinelibrary.com]

but they started to decline the earliest. In this region, post-neonatal mortality has been higher than child mortality throughout the nineteenth century. Finally, the west surpasses the south first and the east later as the most unhealthy regions for young children, while the north remains the most healthy region throughout the period.

To explore how mortality and height trends coincided, figure 5b provides four panels in which the start of the secular trend in heights for each region is zoomed in on. The height trend starts together with the decline in child mortality in all regions. In the west, the upward trend in heights already begins in the 1830s albeit from a relatively low level. The north follows around 1840. This specific starting point should be reviewed with caution because of the low number of observations

TABLE 1 Linear regression models on the association between post-neonatal mortality and child mortality and height 1850–1940 per region.

	North	East	West	South
Post-neonatal mortality rate	−0.24*	−0.07	−0.22***	−0.15***
Child mortality rate	−0.45***	−0.37***	−0.05	−0.27***
Intercept	1750.93	1731.10	1728.10	1719.27
R^2	0.656	0.406	0.623	0.743

Estimates in mm. Signif. codes: 0 *** 0.001 ** 0.01 * 0.05 ‘ 0.1.

TABLE 2 Linear regression models on the association between age and cause specific mortality rates and height 1875–1900.

	All-cause mortality			Airborne infectious mortality			Food and waterborne mortality		
	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 8	Model 9
Infant mortality rate per 1000 inhabitants	−0.02	–	0.02	−0.15	–	−0.16	0.02	–	0.10
Child mortality rate per 1000 inhabitants	–	−0.29	−0.34	–	−0.05	-0.01	–	−0.34‘	−0.45*
R^2	0.043	0.047	0.046	0.044	0.046	0.046	0.043	0.044	0.045

Estimates in mm. Signif. codes: 0 *** 0.001 ** 0.01 * 0.05 ‘ 0.1. All models are controlled for SES, birth region, and birth decade. The model uses 1900 height observations. The correlation between IMR and CMR is 0.51.

before 1840. The east and south are the last to start their height trend. If both the height and mortality figures are closely examined, it becomes apparent that the height trend begins slightly earlier than the child mortality trend. This is due to the fact that a child death rate cannot be assessed until they reach the age of five years. Finally, the association between infant mortality and height cannot be dismissed altogether. The southern panel shows high infant mortality rates until the end of the nineteenth century, which may explain the region’s less steep secular height trend.

Table 1 uses the same variables as in figure 4a,b. For each individual region, a regression model examines the association between post-neonatal, child mortality, and height.[67] The estimate for child mortality is larger in the north, east, and south of the country. Furthermore, the regions that were identified earlier as deviating from the expected correlation show interesting outcomes. In the east, which had low levels of infant mortality followed by relatively high levels of child mortality, only child mortality is significantly associated with height. In the west, which had high levels of infant mortality followed by low levels of child mortality, only post-neonatal mortality is negatively associated with height. This will be explored at lower levels of aggregation below.

Table 2 provides the results of the regression of infant and child mortality at the municipal level on conscription height at the individual level between 1875 and 1900. In line with the scatterplot

[67] At these high levels of aggregation, using an average for the region and five year period, the R^2 will be higher. Furthermore, the averages reduce variation in all variables and therefore the association tends to be significant when compared with the results from tab. 2.

TABLE 3 Family fixed effects model of the timing of a sibling death on height.

	Ages 0–1			Ages 1–5			Ages 5–12			Ages 14–19		
Model	**OLS**	**FFE**	**FFE[a]**	**OLS**	**FFE**	**FFE[a]**	**OLS**	**FFE**	**FFE[a]**	**OLS**	**FFE**	**FFE[a]**
No death	Ref.	Ref.	Ref.	Ref.	Ref.	Ref.	Ref.	Ref.	Ref.	Ref.	Ref.	Ref.
Sibling death	−6.89	4.74	−1.57	−7.62 **	−2.06	−6.55 *	−8.01 **	−0.43	0.35	−5.95	−3.02	1.43
Families	3938	144	144	3938	502	502	3938	532	532	3938	380	380
Observations	3938	455	455	3938	1564	1564	3938	1700	1700	3938	1239	1239

A sibling death occurred within the given interval. Estimates in mm.

Note: In the app. overall models for all age groups can be found (online only). Signif. codes: 0*** 0.001** 0.01 * 0.05 ‘ 0.1.

[a]The last model of each age group is controlled for birth year.

of 1875, no association was found between infant mortality rates and height. In contrast, child mortality is negatively associated with height. Nevertheless, despite the clear negative association, the estimate was not statistically significant.

Cause-specific infant and child death rates provide a clearer understanding of the mechanism behind the time trend in both mortality and height for the period 1875–1900. The infant and child mortality rates for airborne infectious deaths, as well as food and waterborne deaths, are presented in table 2. The strongest negative estimated effect for airborne infectious mortality on height outcomes is found in infant mortality, although the association is not significant. Child mortality is negatively associated with shorter stature when it comes to food and waterborne death, and these findings are significant. The negative estimate of diseases that are related to food and water quality indicates that the scarcity of sanitary and hygienic measures, as well as poor food and water quality, may have stunted young children. Breastfeeding could have prevented death from these causes, especially gastrointestinal infection deaths for infants, which is why there is no significant association with infant mortality rates and shorter stature.[68] As older children are not breastfed and display a clear association with height and their mortality rates, breastfeeding is a reason why this cause specific infant mortality is less representative of the overall childhood disease environment.

The results in table 2 provide some evidence that child mortality is a stronger predictor of adult heights in the past than infant health. Even so, there could be spillover effects to children of different ages. For instance, the spread of a disease among children aged one to five years has the potential to stunt children in other age groups, without resulting in the same mortality rates for these older children.[69] To study this effect, the final model is entirely centred on the household unit, as this is the ideal site to study these issues. Within a household, a sibling death indicates a high disease load within this household.[70] Holding all other factors constant, brothers experience the death of a sibling at a different stage of their early life, and as a consequence, the association with height can vary.

Table 3 presents whether the death of a sibling, regardless which age the deceased sibling had, stunts surviving siblings within the household for the age intervals 0–1 years, 1–5 years, 5–12 years,

[68] Galley and Woods, ‘Distribution of deaths’.

[69] For example: Hatton, ‘Health of survivors’.

[70] Ho, ‘Estimating sibling spillovers’.

and 14–19 years of the surviving sibling.[71] The ordinary least squares model shows that boys who survived a sibling death between the ages of 1 year and 12 years are likely to be shorter. However, the family fixed effects model comparing brothers within a household shows that the negative estimate of a sibling death on the height of survivors is found between the ages of one year and five years.

VI | DISCUSSION

This study has demonstrated that infant deaths are not representative for the health of survivors in a high mortality context. Indeed, infants are the most sensitive and vulnerable, but they can also be shielded through breastfeeding from contextual factors that affect health, causing a deviation in their disease and mortality pattern compared with older children. Contrastingly, heights are taken from those who survived the high mortality context and are therefore reflective of the health of surviving children. This is why, both in earlier studies and in this article, a very low correlation was found between infant mortality rates and height.

This does not imply that height does not reflect health in early childhood. Both the start of the upward height trend and the decrease in child mortality were found to have occurred simultaneously. Therefore, we can conclude that child mortality rates are a good proxy for the health of survivors. Childhood diseases divert energy from growth, resulting in a shorter stature. In particular, diseases that impact the digestive system hamper growth, as child deaths from water and foodborne diseases were negatively associated with the health of survivors.

The family fixed effect models showed that the critical stage of the impact of disease on height is in early childhood as well. Only those who experienced sibling mortality within the household when they were themselves between the ages of one year and five years were negatively associated with lower height outcomes. Most likely, infants would not have survived an extreme disease environment, whereas older children were affected less.

The bivariate maps indicated that infant mortality can be both higher and lower compared with child mortality rates. Especially in the regions where relatively low infant mortality rates were followed by high child mortality rates, infants were most likely shielded via breastfeeding practices. Although possible, it is highly unlikely that diseases that kill children would spare even more vulnerable infants. Furthermore, the presence of local doctors was low in these parts of the Netherlands.[72] It seems likely that in the east of the Netherlands breastfeeding was a common practice, and therefore the lower infant mortality rates should be viewed with caution as they do not reflect the health conditions survivors experienced. Furthermore, from the development over time, we learn that child mortality was even higher than infant mortality in the east. The east also saw a relatively late improvement in child mortality that coincided with the late onset of the secular trend in heights. It is especially in these regions that historians should use child mortality rather than infant mortality, as was also shown in the regression models.

In regions that experience high infant mortality followed by low child mortality, we could argue this is linked to a lack of breastfeeding. As a result, infant deaths were high, leaving only the strongest children to survive. In the Dutch south, it was found that fewer infants were breastfed

[71] The HSN only provides information of members of the household of the RP; therefore, the diseased sibling is likely to be younger than 20 and is still living within the household.

[72] See sec. A.8., fifth map on the top row in the appendix.

frequently, and as a consequence, the number of digestive disorder deaths increased.[73] Also in the west, a recent study linked infant deaths to the lack of breastfeeding.[74] As infants were not shielded trough weening in the west and south, the infant mortality rates were higher, and infants were exposed earlier to the same external factors that killed children in other regions; the external factors which infants in the east and north were shielded from. This implies that the argument that infant mortality rates are not reflective of children's health is only applicable when infant mortality rates are low because of breastfeeding, but does not work in the opposite direction.

Still the question remains as to why infant mortality rates were negatively associated with height but with an absent child mortality effect in the west. If infants were killed by the same external factors as children, one would expect both mortality rates to be a similar proxy for health. This might hint at mortality selection in which only the healthiest, or children from the most protective households, survived, resulting in a relatively low child mortality rate. Nevertheless, the association between infant mortality and height was negative, and one would expect these selected survivors to be taller. This might have to do with a defect in the scarring and selection mechanism as spelled out in the introduction. High infant mortality rates do not separate infants into a clearly separated healthy and unhealthy group. Still, scarring in infancy might occur among survivors, and is even very likely when mortality rates are high. Possibly short-term benefits in mortality rates might be absent at conscription when the scarring effect comes to the forefront. Furthermore, it is also a matter of perspective: the child mortality rates in the west were low compared with those in the north and east that were driven by children who had been shielded during their first year of life.

The significantly negative association between child death from food and waterborne diseases and height can also be linked to the various height trends in the Netherlands. Mortality from food and waterborne diseases remained at a relatively high level in the south throughout the nineteenth century.[75] This would explain, after the initial onset of the secular trend in heights around 1850, why average height increased much slower in this area. Furthermore, since breastfeeding shields infants particularly well against this type of disease, it also explains the relatively low correlation between infant mortality rates and heights in 1925 in the south. In the west, meanwhile, the largest sanitary improvements were made, together with the encouragement of breastfeeding. This region also saw the most rapid increase in heights. In sum, this study has shown that infants can be shielded by breastfeeding from a high mortality regime, which is when a historian should use child mortality rates as a proxy for the disease environment of older children. Nevertheless, in the absence of breastfeeding, it is highly likely that infant mortality rates are a sufficient proxy. Still, researchers should be aware that these breastfeeding practices could change over time and cloud the association between infant mortality and height.

VII | LIMITATIONS

One of the major pitfalls of this paper is, theoretically, that wealth confounds the association between diseases and heights.[76] Wealthier families may be able to reduce exposure to infection

[73] Walhout, 'Is breast best'.

[74] Waters-Rist et al., 'Isotopic reconstruction'.

[75] Van den Boomen, 'Born close to death'.

[76] A previous version of this study tested for this confounder, the results are included in the appendix, sec. A.7.

as they can afford better environments. Moreover, their ability to afford more and better food increases their ability to fight off diseases. Yet there is mixed evidence as to whether the latter also results in less energy being diverted from growth.[77] Furthermore, studies in historical demography provide little evidence for such a confounding factor overall. In high mortality contexts, no or at best a weak socio-economic gradient was found in infant or child mortality rates.[78] Rather, local environmental conditions determined these rates. Van Poppel et al. showed that even the lowest social groups in the northern province of Friesland were better off than the highest social group in the western provinces of Utrecht and Zeeland in terms of post-neonatal and child mortality.[79] Alternatively, a clear socio-economic gradient in heights was found.[80] As this gradient is less clear or even absent for mortality, this indicates that the height gradient is most likely the result of better nutrition and not so much of lesser diseases. Therefore, the association between health and height is not fully confounded by income.

Another potential problem of why the association between infant death and height is weak and not significant is because the infant mortality rates might not be reflective of the underlying population as a whole, as mentioned in section IV. Confounding factors could result in mortality rates that differ compared with the municipal average. To illustrate this, it has been found that to a certain extent, mortality is clustered in certain high-risk families which cannot be explained by other factors such as socio-economic status.[81] This does not directly imply that morbidity, and therefore the effect of the disease environment on height, is also clustered. It might be an indication of bad parenting or genetics that influence both mortality and heights in particular high-risk clusters. Therefore, to add to the family fixed effect models, the number of deceased siblings was tested.[82] It was found that there is a negative linear gradient on the height of survivors for the number of siblings that died in a household. This explains the association better than the labelling of high-risk families alone.

The finding in the family fixed effect models that those who experienced a sibling death in early childhood were shorter shows that these children did not benefit from less diluted resources with the reduction of sibship size. However, they were deprived of sibling support in extra care and resources for the household. A study into adult support on the same data found strikingly similar results as a result of paternal death.[83] Furthermore, the stress of a sibling death might reduce appetite or be associated with a later onset of puberty.[84] The results show that the loss of a sibling is negatively associated with height and that surviving children do not benefit from one plate less on the table.

Furthermore, data availability might have hampered or biased the outcomes. In the appendix, logistic regression models are added that show the likelihood that data are selected for a particular analysis. The starting point of this paper is the HSN conscription data which are a representative measure for heights within the Netherlands.[85] A logistic regression was used to test which HSN

[77] Schneider, 'Effect of nutritional status'.

[78] Bengtsson and Van Poppel, 'Socioeconomic inequalities'; Dribe and Karlsson, 'Inequality in early life'.

[79] Van Poppel et al., 'Three Dutch regions'.

[80] Jaadla, Shaw-Taylor and Davenport, 'Height and health'; Quanjer and Kok, 'Homemakers and heights'.

[81] See Van Dijk 'Early-life mortality', for and extensive overview.

[82] These models can be found in the appendix, sec. A.6.

[83] Quanjer et al., 'Short lives'.

[84] For example: Sheppard, Garcia and Sear, 'Childhood family disruption'.

[85] Quanjer and Kok, 'Drafting the Dutch'.

RPs were linked to their brothers in the first place. It was reassuring that height did not influence the likelihood of being linked at all. Still, the linkage was biased towards the north-west of the Netherlands for birth cohorts 1860–80, because of the digital linking process that increased the chances of a successful link.[86] This regional and periodical bias had no effect on the socio-economic composition of the data. However, the data linkage was biased towards larger families, as the likelihood of having a brother in the dataset increases with sibship size. Furthermore, higher within-family mortality was associated with lower chances of a successful brother link, as brothers were less likely to survive until the age at which they were subject to conscription. These factors should be kept in mind when interpreting the results. The most reassuring point is that height was not biased in any way via the data selection process; that is, in none of the data selections was height biased in any way.

Finally, why are infant mortality rates associated with much of the secular trend in heights in the second half of the twentieth century in high-income countries? Most likely, medical interventions and increased understanding of nutrients and hygiene from 1900 onwards gained importance for the population as a whole and therefore reduced the mediating role parents could play in infant survival.[87] Large-scale vaccination campaigns, modern medical interventions, and close monitoring of child health are as important for infants' health as for children's health.[88] This also increased the role for income as a confounder as these medical interventions can be bought. The way we therefore proxy health with the net nutrition equation should be very much context dependent.

VIII | CONCLUSION

Separating infant mortality from child mortality provides better insight as to how height is associated with the disease environment. Infants are vulnerable and do not necessarily respond to a disease environment in a way that is representative for the other children of a population. Hygienic measurements might not have improved infant health in the short run, but increasing heights are an indication of a healthier population during the second half of the nineteenth-century Netherlands. The major improvement in child mortality can be associated with a strong improvement in heights. Although the coefficients found were rather small, the size of the health transition translates into a 6 cm increase in heights in the west between 1830 and 1920, and a 1.5 cm increase in the north between 1840 and 1920.[89] These are substantial increases in height that can be linked to improved health. In other words, the coastal region in the west, the richest part of the country, was held back in its biological living standards due to unhealthy conditions.[90] Current ongoing in-depth projects into the details of the health transitions, for example, in Amsterdam, will provide answers to what the drivers are behind the health improvements.[91] For now, it has become apparent that these studies should not only focus on infants, but should include the health of children

[86] Ibidem, see for a more in depth explanation of the linking process.

[87] For example: Harris, *Health of the schoolchild*.

[88] De Pree-Geerlings, De Pree and Bulk-Bunschoten, '100 jaar artsen'.

[89] This is based on the estimate of −0.3 of child mortality rates on heights in millimetres. In the west, child mortality rates fell from around 250 per 1000 to 50 per 1000; in the north, rates fell from 100 per 1000 to around 50 per 1000.

[90] The west was on average 1.2 cm shorter than the north in the nineteenth century; when controlled for child mortality, the west was 0.7 cm taller.

[91] For example: https://doodinamsterdam.nl/

as well, as it will be these children who become the conscripts that determine our understanding of living standards in the past.

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