Social Inequality in Cause-Specific Infant Mortality at the Dawn of the Demographic Transition: New Insights from German Church Records

Michael Mühlichen¹ and Gabriele Doblhammer^{2,3}

¹ Federal Institute for Population Research (BiB), Wiesbaden, Germany ² German Centre for Neurodegenerative Diseases ³ University of Rostock, Germany

Abstract

Little is known about social gradients in cause-specific infant mortality in the nineteenth century. To our knowledge, this is the first paper to explore this connection for the time prior to the demographic transition. We used the church records of Rostock, an important port city on the Baltic coast in northern Germany, and prepared and merged the baptismal and burial registers of its largest parish, St. James, for the periods 1815–36 and 1859–82. Based on individual-level data (N=16,880), we classified the fathers' occupations into three social classes and estimated cause-specific infant mortality risks for these groups using event history analysis. We found an almost linear social gradient in neonatal and post-neonatal mortality. This gradient was driven by gastro-intestinal diseases, which suggests severe deficits in nutrition and sanitation among the lower social classes, even before industrialisation (coupled with population growth) led to worsening living environments.

Keywords

Infant mortality; neonatal and post-neonatal mortality; causes of death; historical demography; church records; event history analysis; Germany

Background

Infant mortality is a widely used indicator for mortality and population health (Masuy-Stroobant and Gourbin 1995; Reidpath and Allotey 2003; Gonzalez and Gilleskie 2017). Its decrease in many Western countries from the late nineteenth century onwards is one of the principal drivers of increased life expectancy and, hence, of the first demographic transition (Schofield et al. 1991; Chesnais 1992; Kirk 1996). The determinants of nineteenth-century infant mortality before this decrease have long been among the key issues of historical demographic research, which is due, for instance, to the poor sanitary conditions arising from urbanisation that contemporary populations in a number of less developed countries are currently, similar to those which Western countries faced prior to the first demographic transition (Pozzi and Ramiro Fariñas 2015).

Several studies have explored social differences as well as cause-specific differences in historical infant mortality, whereas little is known about the interplay between social status and cause of death. As to our knowledge, the question if the impact of social status on infant mortality differs by cause of death has not been answered so far for the time before the demographic transition. Given that causes

of death may provide deeper insights into the potential reasons for differentials and trends in infant mortality, this is a surprising gap in the research.

On the one hand, this research gap is due to the fact that official cause-of-death statistics were not introduced until the twentieth century in most Western countries and other sources like church records have the disadvantage that their digitisation, transcription, and preparation for analysis are expensive and time consuming, if they survived at all. On the other hand, these sources frequently do not include useful information on both social status and cause of death. And if they do, the numbers of transcribed cases are usually too small for cause-specific analyses.

Aiming to close this research gap, we used the large, newly available data source of church records from the Hanseatic city of Rostock, Germany, because these data include information on both cause of death and social status for large numbers of residents, and describe an urban setting prior to the demographic and epidemiologic transitions when infectious diseases were still omnipresent, infant mortality was high and industrialisation and urbanisation were just starting to emerge. After classifying causes of death into four groups, and fathers' occupations into three social classes, we used event history methods to estimate the impact of social differences on cause-specific infant mortality in nineteenth-century Germany.

Data and Methods

Our analyses are based on the baptismal and burial registers of St. James, Rostock. We used the data that were available at the RAPHIS database but extended the data by transcribing additional years. After preparing and harmonizing these data and matching the live births from the baptismal registers with the infant deaths from the burial registers, we were able to conduct survival analyses for 16,880 live births born in the periods 1815–1836 and 1859–1882, of whom 2,689 infants died before their first birthday.

We analysed infant death, neonatal death and post-neonatal death as our main outcome variables, each differentiated by the following cause-of-death groups: respiratory diseases, gastro-intestinal diseases, weakness/atrophy and other diseases.

Our main variable of interest is social class, derived from the father's occupation given in the church records. As to our classification, social class A ('high status') includes all high-level officials, merchants, doctors, professors and proprietors; social class B ('medium status') includes craftsmen, medium-level officials, steersmen, skippers, teachers, grocers and wagoners; and social class C ('low status') includes labourers, seamen, low-level officials, day labourers, porters, apprentices, servants, factory workers, field workers, fishermen and artists as well as unknown fathers (particularly in the case of illegitimate births). Other covariates are sex and period of birth (1815–1836 and 1859–1882).

We estimated infant mortality differentials using event history analysis based on the Cox proportional-hazards model. The analysis time was measured by the age in days, from birth to infant death, whereby the age of newborns who survived the first year of life was set at the censored time of 365 days. In the case of neonatal mortality, the censored time was 29 days. The analysis of post-neonatal mortality excluded neonatal deaths by definition and thus involved left-truncated data. For simplicity, we interpreted hazard ratios as relative risks.

Results

The risk of infant death is connected with social class, period of birth and sex but the differences are not evident in every cause-of-death group (Table 1). With regard to all causes, the risk of dying in the first year of life is 30 % higher in the medium social class B (hazard ratio HR = 1.30) and 49 % higher in the low social class C (HR = 1.49) compared to the reference group of the high social class A. In comparison with the cohorts born between 1815 and 1836, the hazard ratio for the cohorts born in the

period 1859–1882 is 47 % higher. Furthermore, the risk of infant death is 15 % lower for female infants than for male ones. From a cause-of-death perspective, we see that these differences are mainly attributable to gastro-intestinal diseases, where the differences are even stronger. The hazard ratio grew by 64 % from the first to the second period. Among the social classes, it is 82 % higher in social class B and 113 % higher in social class C compared to social class A. This social gradient is not evident in other cause-of-death groups. The sex gradient is strongest in 'other causes'. The period effect is evident in all cause-specific groups, albeit statistically insignificant in respiratory diseases.

Variable	Category	All causes	Gastro- intestinal	Respiratory diseases	Weakness/ atrophy	Other causes
Sex	Men	1	1	1	1	1
	Women	0.85 ***	0.85 ***	0.95	0.87	0.71 ***
Period	1815–1836	1	1	1	1	1
of birth	1859–1882	1.47 ***	1.64 ***	1.16	1.46 ***	1.39 ***
Social class	A: High status B: Middle status C: Low status	1 1.30 *** 1.49 ***	1 1.82 *** 2.13 ***	1 0.94 0.99	1 0.96 1.17	1 1.17 1.26

Table 1Infant mortality by cause of death (hazard ratios); Cox proportional-hazards model;
Rostock, St. James parish, 1815–1836 and 1859–1882

* $p \le 0.1$; ** $p \le 0.05$; *** $p \le 0.01$.

Regarding neonatal mortality (Table 2), the social gradient in gastro-intestinal diseases is even stronger than in total infant mortality: The hazard ratio is 109 % higher in social class B and even 222 % higher in social class B compared to social class A. The sex gradient to the disadvantage of male infants is also more pronounced in neonatal mortality than in overall infant mortality, whereas the period effect is considerably weaker and only significant in connection with weakness/atrophy.

Table 2	Neonatal mortality by cause of death (hazard ratios); Cox proportional-hazards model;
	Rostock, St. James parish, 1815–1836 and 1859–1882

Variable	Category	All causes	Gastro- intestinal	Respiratory diseases	Weakness/ atrophy	Other causes
Sex	Men	1	1	1	1	1
	Women	0.77 ***	0.70 ***	1.07	0.81 **	0.86
Period	1815–1836	1	1	1	1	1
of birth	1859–1882	1.20 **	1.08	2.35	1.40 **	0.86
Social class	A: High status B: Middle status C: Low status	1 1.21 1.60 ***	1 2.09 ** 3.22 ***	1 0.83 1.11	1 0.99 1.10	1 0.71 1.15

* $p \le 0.1$; ** $p \le 0.05$; *** $p \le 0.01$.

In post-neonatal mortality (Table 3), the social gradient is evident in gastro-intestinal diseases as well, where the hazard ratio is 75 % higher in social class B and 86 % higher in social class C compared to social class A. The sex differences are less pronounced in post-neonatal mortality than in neonatal mortality, whereas the increase in the hazard ratios from the first to the second period is stronger in post-neonatal mortality and significant in all cause-of-death groups, except for respiratory diseases.

Variable	Category	All causes	Gastro- intestinal	Respiratory diseases	Weakness/ atrophy	Other causes
Sex	Men	1	1	1	1	1
	Women	0.89 ***	0.91	0.95	1.11 **	0.68 ***
Period	1815–1836	1	1	1	1	1
of birth	1859–1882	1.62 ***	1.94 ***	1.13	1.68 **	1.57 ***
Social class	A: High status B: Middle status C: Low status	1 1.34 *** 1.44 ***	1 1.75 *** 1.86 ***	1 0.94 0.98	1 0.86 1.40	1 1.31 1.29

Table 3Post-neonatal mortality by cause of death (hazard ratios); Cox proportional-hazards model;
Rostock, St. James parish, 1815–1836 and 1859–1882

* $p \le 0.1$; ** $p \le 0.05$; *** $p \le 0.01$.

Conclusion

Our preliminary results show a remarkable, almost linear social gradient in infant mortality for nineteenth-century Rostock, which is true for both neonatal and post-neonatal mortality. However, the consideration of causes of death reveals that this gradient is driven entirely by gastro-intestinal diseases. To our knowledge, this is the first study to explore this connection for the time prior to the first demographic transition. The social gradient is particularly pronounced in neonatal mortality, although gastro-intestinal and other exogenous diseases were relatively rare in the first four weeks of life. Following Bengtsson and van Poppel (2011), we do not assume that this is a direct effect of social class. Especially the close connection with gastro-intestinal diseases indicates that the social class is, rather, a proxy for nutrition and sanitation. Therefore, we assume that infants of lower social status were more likely to be exposed to substitute nutrition (instead of breastfeeding), malnourishment, poor access to clean water, and neglect. Moreover, following Derosas (2009), the strong social gradient in neonatal mortality suggests severe deficits in maternal care in the low social class.

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