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2026

Document Version:
Other version

Lund Papers in Economic History

No. 267, 2026

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Abstract

At the turn of the twentieth century, infant- and child mortality declined rapidly in many industrializing societies. In Sweden, this decline coincided with industrialization and urbanization, as well as a period of growing social disparities in childhood mortality. The inequality in child survival was connected to a range of factors, including access to water- and sanitation, housing conditions, infant care, and possibly nutrition. We study the importance of socioeconomic neighborhood context for under-five mortality in an industrializing Swedish town (1892–1939). We use individual-level socioeconomic and demographic data from population registers that have been geocoded at the block level and measure neighborhood conditions by the share of white-collar workers in the block. Cox models with time-varying block-level covariates to estimate the association between cumulative social neighborhood variables and the risk of child death. Our findings indicate that the socioeconomic status of the neighborhood was important for the risk of child death even when controlling for social class and family context. The association was present for both boys and girls and got weaker over time in the period we analyze. Social neighborhoods mattered more for infant mortality than for child mortality. In terms of causes of death, the associations were similar for airborne infectious diseases and food/waterborne diseases, while there was no association at all for other causes of death. These findings point to the importance of neighborhoods for child survival during the urban mortality transition and likely reflect both cultural and material causal pathways.

Keywords: Infant mortality, child mortality, neighborhoods, socioeconomic status, health inequality, urban mortality transition, historical demography, Sweden

JEL-codes: I14, N33, N34, J13, R23

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1. Introduction

The turn of the twentieth century was a period of rapid decline in infant- and child mortality and a time of industrialization and urbanization; in Sweden (Hofsten and Lundström 1976; Schön 2010) as well as across the Western World (Davenport 2021; Riely 2001). It was also a period of considerable social differences in childhood mortality, especially in large cities such as Stockholm (Burström and Bernhardt 2001; Molitoris and Dribe 2016; Molitoris 2017) but also in the countryside (Dribe and Karlsson 2022). Similar socioeconomic differences were found in other contexts in Europe and North America (e.g., Hacker et al. 2023; Haines 1995; Jaadla et al. 2020; Mühlichen et al. 2015; Murkens et al. 2023; Muurling and Ekamper 2025; Preston and Haines 1991; Reid et al. 2023), and the socioeconomic gradient in infant mortality persisted as mortality declined in the first half of the twentieth century (Antonovsky and Bernstein 1977). The inequality in child survival was connected to a range of factors, including access to water and sanitation, housing conditions, breastfeeding, infant- and childcare, and possibly nutrition (e.g., Breschi and Pozzi 2004; Corsini and Viazzo 1997; Reid et al. 2023). Furthermore, increased knowledge about disease transmission and protective measures played a role in creating, or enlarging, socioeconomic differences in when mortality declined (see Murkens et al. 2023).

In addition to the parents' socioeconomic status, in most cases mainly the father, neighborhood conditions may have impacted child health through several of these factors (Thornton and Olson 2011; Connor 2017). The extent and quality of the water- and sanitation infrastructure could differ between neighborhoods and, depending on city policies, this may or may not co-vary with the socioeconomic status of the neighborhoods (see Önnerfors 2021). To the extent that there are pronounced differences between neighborhoods in the quality of water- and sewerage systems, we expect differences in childhood mortality, especially in food- and waterborne diseases. Denser neighborhoods with more crowded housing can be expected to promote contagion and increase mortality risks from airborne infectious diseases. We expect such neighborhoods to also have lower socioeconomic status. Besides these factors related to the built environment, cultural differences across neighborhoods are evident in breastfeeding, women's work, and infant care. Therefore, neighborhood status probably has an independent influence on child survival, in addition to the effects of parental socioeconomic status and living standards.

This study examines the importance of the socioeconomic neighborhood context for infant- and child mortality in an industrializing port town in southern Sweden during the period

1882–1939, which was the real breakthrough period of Swedish industrialization and also a time of considerable mortality decline (Schön 2010; Hofsten and Lundström 1976). We use individual-level socioeconomic and demographic data from population registers, which have been geocoded at the block level, and measure neighborhood conditions by the share of white-collar workers within the city block. We use Cox proportional hazards models with monthly time-varying neighborhood measures at the block-level to estimate the association between neighborhood conditions and the risk of childhood death, adjusting for parental social class. We also analyze the neighborhood association by cause of death, distinguishing airborne infectious diseases, food- and waterborne diseases, and other causes.

We contribute to research in several ways. First, analyses of cause-of-death specific infant- and child mortality during mortality decline, using individual-level data, remain rare, despite some previous research (e.g., Jaadla and Puur 2016; contributions in Reid and Janssens 2025). Second, analyses of socioeconomic mortality differences by cause of death are even rarer, and only a few previous studies exist (e.g., Molitoris 2017; Mühlichen and Doblhammer 2025; Murling and Ekamper 2025; Murkens et al. 2023). Third, our study is one of the first to combine the socioeconomic status of the family head with the socioeconomic characteristics of the neighborhood in an analysis of cause-specific infant- and child mortality. To our knowledge, only one study on Montreal has done this previously (Thornton and Olson 2011).

Our findings suggest that the socioeconomic status of the neighborhood was important for the risk of child death, especially for infants, even when controlling for social class and family context. Living in low-status neighborhoods was associated with higher infant- and child mortality. The association was similar for boys and girls, and it got somewhat weaker over time. The socioeconomic status of the neighborhood mattered in a similar way for mortality from airborne infectious diseases and food- and waterborne infectious diseases, whereas it had no association with other causes of death. Different mechanisms could be important for such neighborhood effects. Low-status neighborhoods are likely to have had worse housing quality and more crowded housing, less likely to have had modern sanitation, and it is also possible that there were behavioral differences relating to the care of infants and young children that spread to neighbors through social interaction.

2. Background

2.1. Mortality decline and the urban penalty

There is a vast literature on the decline of infant and child mortality during the demographic transition (e.g., Breschi and Pozzi 2004; Corsini and Viazzo 1997; McKeown 1976; Preston and Haines 1991; Woods 2000; for the Nordic countries, see, e.g., Edvinsson et al. 2008). Several factors have been proposed to explain the decline, focusing on nutrition, water and sanitation, housing standards, public health interventions, parenting and infant care, including breastfeeding.

Much attention has been devoted to urban-rural differences, and especially *the urban penalty* in childhood mortality that was present in most historical contexts before, and early in, the mortality decline (Kearns 1988; Van Poppel 1989; Ramiro Fariñas and Oris 2016), Sweden included (Brändström et al. 2002). Pre-industrial and early-industrial cities were unhealthy places to live in, and the larger the city, usually the worse the conditions (Condran and Crimmins 1980; Haines 2001), although the relationship between city size and mortality is not always linear (Reid 1997; Woods 2003; Pérez-Moreda et al. 2004). Mostly, the urban penalty was connected to higher population density and crowding in urban areas (see, e.g., Beach and Hanlon 2018), which contributed to poor hygiene and spread of infectious diseases (Burström et al. 1999). Moreover, poor water quality and lack of proper sewerage systems promoted the spread of waterborne diseases in urban areas (e.g., Kesztenbaum and Rosenthal 2017), and high levels of air pollution from heating and industries also contributed significantly to high mortality in industrial cities, both among infants and adults (Beach and Hanlon 2018; Hanlon 2024).

Considerable attention has been devoted to issues of data quality and registration of infant deaths and the bias it may introduce into estimates, particularly of infant mortality. In Spain, for example, rural-urban differences in infant mortality were severely distorted by the institution of foundling hospitals; this was most notable in Madrid, where abandoned children were left to be cared for, but where infant mortality was also very high (Revuelta Eugercios 2013). Many of these children came from the surrounding towns and villages, which means that they were registered as born outside the city but then registered as dead in the city. This naturally biases mortality estimates upward for the city and downward for outside the city, thereby exaggerating the urban penalty (Ramiro Fariñas 2007; Revuelta Eugercios and Ramiro Fariñas 2016).

Due to the high mortality and comparatively low fertility, many cities relied on large flows of in-migrants to grow and to recruit labor. Such migration flows contributed to contagion, and in addition, rural migrants had less prior exposure to the disease climate of the cities, which might have increased their mortality compared to city dwellers. Moreover, during the urbanization process of the turn of the twentieth century, rural-urban migrants often lived in poorer neighborhoods with low-quality housing and infrastructure (e.g., Olson and Thornton 2011 for Montreal). However, in early twentieth-century Madrid there were only small differentials between in-migrants and city natives in terms of infant mortality, suggesting a fast behavioral adaptation of the migrants (Oris et al. 2023).

During the late nineteenth and early twentieth century many cities started to invest in their water and sewerage infrastructure, which contributed to a faster mortality decline in urban than in rural areas, and hence to a convergence between rural and urban mortality levels (e.g., Alsan and Goldin 2019; Beach et al. 2016; Cutler and Miller 2005; Harris and Helgertz 2019; see Helgertz and Önnerfors 2019 for an analysis Swedish towns and cities between 1875 and 1930). Consequently, a key question is whether and how mortality decline and the urban penalty were stratified by socioeconomic status and neighborhood conditions within cities.

2.2. Socioeconomic differences

Several of the explanations for the decline in infant- and child mortality are also potentially connected to economic wellbeing and socioeconomic status. Burström et al. (2005), for example, show how the decline in diarrheal mortality among children in Stockholm in the 1880s, connected to improved water- and sanitation infrastructure, was faster among the higher-status groups than the lower-status groups. This development widened the class differences, but as investments continued and more households got connected to the modern system, mortality also declined among lower-status groups, which led to a convergence in diarrheal mortality.

However, some previous research on the historical mortality transition has somewhat downplayed socioeconomic status as an important determinant and instead stressed the role of place of residence, race or religion, depending on context (Reid 1997; Reid et al. 2023; Preston and Haines 1991; Garrett et al 2001; Ekamper and Van Poppel 2018). During the pre- and early transition period, there were often only small socioeconomic differences in childhood mortality, while a clearer mortality gradient by socioeconomic status emerged during industrialization and mortality decline (see reviews in Dribe and Karlsson 2022 and Muurling and Ekamper 2025). There are, however, studies showing marked socioeconomic differences also in pre- and early transition contexts (e.g., Mühlichen et al. 2015 for Rostock in Germany; Breschi et al. 2011 for

Algero, Sardinia; Schumacher and Oris 2011 for Geneva; Van Poppel et al. 2005 and Muurling and Ekamper 2025 for the Netherlands).

A previous study of total childhood mortality in a rural area in the same region as this study showed how a socioeconomic gradient in childhood mortality emerged in the second half of the nineteenth century (Dribe and Karlsson 2022). This was clear for both post-neonatal infant mortality and child mortality (1–4 years), whereas there were no systematic class differences in neonatal mortality. It is well known that neonatal mortality is largely related to complications from pregnancy and birth and is usually not closely associated with socioeconomic factors. The emergence of class differences in childhood mortality was partly related to the epidemiological transition and to the declining importance of highly virulent infectious diseases (epidemics), such as smallpox, measles, and whooping cough, and instead a dominance of less virulent infections, both airborne (such as tuberculosis and pneumonia) and food- and waterborne (e.g. typhoid, diarrhea). Morbidity and mortality from several of these infectious diseases were more dependent on nutrition, access to uncontaminated food and water, and crowding. This development may have contributed to a gradual emergence of a mortality gradient by social class.

In the late nineteenth- and early twentieth centuries, knowledge about disease transmission improved, and active treatment against several of the important diseases developed (see Easterlin 1999). This implied increasing possibilities for the socioeconomically more advantaged groups to get ahead in terms of mortality decline, which further increased mortality differentials (Murkens et al. 2023; see also Clouston et al. 2016 for a more theoretical discussion).

Post-neonatal childhood mortality at the turn of the twentieth century was dominated by deaths from infectious diseases, either airborne or food- and waterborne (see Hiltunen Maltesdotter and Edvinsson 2025 for the city of Sundsvall in northern Sweden). Neonatal mortality was more related to the newborn's and mother's health than to the environment (e.g., Reid 2001). Airborne infectious diseases, such as measles, whooping cough, pneumonia, and bronchitis, spread easily in congested living quarters and areas of high population density, while food- and waterborne diseases, for example, diarrhea and gastric diseases, were especially prevalent in contexts lacking clean water and proper sanitation. As previously discussed, cities were especially vulnerable, and the urban penalty has often been explained by the high prevalence of these diseases following poor urban infrastructures (see Harris and Helgertz 2019). However, the link between water supply and mortality is complex and also depends on factors such as breastfeeding, personal hygiene, and sanitation quality, and it is sometimes

difficult to empirically establish an effect on child mortality of improved water supply (see Van Poppel and van der Heijden 1997).

Mühlichen and Doblhammer (2025) found socioeconomic differences in infant mortality (both neonatal and post-neonatal) in nineteenth-century Rostock, which were driven by convulsions (neonatal and post-neonatal) and waterborne diseases (post-neonatal). While there was a clear gradient for neonatal mortality, this was less clear for post-neonatal deaths, where it was mostly the high-status group that had higher mortality than the medium and low-status groups. The existence of a gradient for neonatal mortality is explained by less breastfeeding and unsanitary conditions surrounding childbirth in the lower-status groups. Similar social differences in neonatal mortality have also been shown for some other contexts (e.g. Derosas 2003; Van Poppel et al. 2005). Because socioeconomic status also affected where families lived, it is important to examine how neighborhood conditions mediated or amplified socioeconomic disparities in child survival.

2.3. The role of neighborhoods

Building on these socioeconomic perspectives, and when relevant, also considering religion and race/ethnicity, spatial differences in childhood mortality have been highlighted in previous research. At larger geographical scales, pronounced mortality differences between regions have been linked to both production structure and pollution (see, e.g., Reid 1997; Beach and Hanlon 2018) and infant care including breastfeeding (e.g., Brändström 1984). However, at finer spatial scales, mortality also often varied with access to clean water and sanitation, housing standards and crowding, infant care, and levels of pollution and exposure to contagion (e.g., Connor 2017; Thornton and Olson 2011; Hinman 2017). Breastfeeding practices could differ across neighborhoods depending on, for example, women's work. In areas where women frequently worked outside the home, breastfeeding may have been less prevalent, contributing to higher infant mortality (see, e.g., Lee 1984; Lithell 1988). Sorting into neighborhoods of different quality is not random but structured, for example, by socioeconomic status, creating pronounced patterns of residential segregation. It is not only in historical settings that there is spatial clustering of childhood mortality. Based on a child opportunity index at the level of census tracts, Slopen et al. (2023) found an inverse relationship with childhood mortality in the early twenty-first-century United States.

There are different ways of measuring such neighborhood conditions. In the absence of direct measures of the proximate determinants of mortality, often some kind of segregation index is used. Such indices can be calculated either for pre-defined neighborhoods or city

districts based on administrative boundaries, or using detailed geocoded data at the block, street, or even address level (e.g., Connor 2017; Hinman 2017; Thornton and Olson 2011). Using administrative neighborhoods is plagued with methodological problems (e.g., Kwan 2012), and it is preferable to use more dynamic measures and a finer spatial scale. There has been a growing number of historical studies using such fine-grained spatial measures on various demographic and socioeconomic outcomes (Hedefalk and Dribe 2020, Hedefalk et al. 2023, 2025; Logan and Shin 2012; Logan et al. 2015; Souza Maia et al. 2024, Souza Maia 2025; Xu et al. 2014). We build on this research in our analysis of social neighborhoods and child mortality in turn-of-the-century Landskrona.

3. Context, data and methods

3.1. Landskrona at the turn of the twentieth century

We study Landskrona in southern Sweden between 1892 and 1939. At the time, Landskrona was a mid-sized industrial port town, and although it is not statistically representative of Sweden, it experienced similar social, demographic, and economic developments as other comparable cities in the country during the period under study (Dribe and Svensson 2024). The city underwent an industrial expansion in the early twentieth century, centered on shipbuilding, food processing, and textiles. In 1900, Landskrona had a population of about 14,000, which increased to 20,000 by 1940. It was in many ways a typical working-class town. Average income and the level of education were lower than in most Swedish cities but trends in income inequality broadly coincided with the national trends, at least for the periods when they can be compared (see Brea-Martinez and Dribe 2024). There was clear residential segregation by socioeconomic status in Landskrona in the early twentieth century, even though it was lower than it became at the peak of its industrial development in the mid twentieth century (Brea-Martinez et al. 2024).

A modern water- and sewerage system, with piped water and underground sewerage, was under development since the end of the nineteenth century, and, at the beginning of the twentieth century, more than 90 percent of houses were connected (1907 for sewerage and 1909 for water).[1] The main water source was underground wells, which meant that it was of good quality, and this had also been the case in Landskrona before the building of the modern system

[1] Information about the water and sewerage system in Landskrona comes from official reports and statistics and was collected by Jonas Helgertz and Martin Önnerfors (see Helgertz and Önnerfors 2019). We are grateful to Jonas Helgertz for sharing the data for Landskrona.

(see Dribe and Svensson 2024). Filtration was the main method of purifying the water. According to the statistical reports, the quantity of good-quality water was also sufficient.

Infant mortality in Malmöhus county, which included Landskrona, was in the mid- to high range in Sweden compared to other counties in the late nineteenth century, as well as in the mid-twentieth century (Brändström et al. 2002). Compared to ten other cities in Sweden, Landskrona shared a declining trend and had medium or lower infant mortality in the late nineteenth century and the first decade of the twentieth century, but from about 1910 until 1935, it was among the cities with the highest infant mortality. For child mortality, Landskrona was among the cities with lower mortality levels, except during some years when mortality spiked, and the city had the highest infant mortality among the cities compared (Dribe and Svensson 2024).

3.2. Data and variables

We use data from the Scanian Economic-Demographic Database (SEDD) (Bengtsson et al. 2025; Dribe and Quaranta 2020). The sources are continuous population registers linked to income and taxation registers. SEDD contains information on births, marriages, deaths, occupations, and in- and out-migrations. We use a spell-based extraction from the database (see Quaranta, 2015, 2016) and add neighborhood data. Neighborhoods are created from geocoded street blocks from the population registers 1892–1939, reconstructed using historical maps and aerial photos from Landskrona City Archives and the Land Survey (Hedefalk and Souza Maia 2023). We continuously follow the residential histories of all individuals in the town.

We examine how block-level neighborhood socioeconomic status is associated with cause-specific mortality of children under five, including separate analyses for the periods 1892–1915 and 1916–1939. Approximately 90 percent of children's time-at-risk could be linked to the block they lived in during the period 1892–1915, and 94 percent during the period 1916–1939. We excluded children residing in very small blocks with few people, as these were likely single properties (about 0.5 percent of the original sample). Additionally, we excluded children who could not be linked to their mothers (about 2 percent of the original sample). Our final sample consists of 10,678 boys and 10,244 girls.

We measure the children's socioeconomic status using *social class* (HISCLASS, Van Leeuwen and Maas 2011), which is derived from HISCO-coded occupations (Van Leeuwen et

al. 2002).[2] The time-varying social class is based on the father's occupation and grouped into four classes:

- White-collar workers (higher professionals and managers; lower professionals and lower managers, clerical and sales personnel; HISCLASS 1–5),
- Skilled workers, foremen and farmers (HISCLASS 6–8),
- Lower-skilled workers (HISCLASS 9–10),
- Unskilled workers (HISCLASS 11–12).

As the data mainly cover the town, the sample includes only a few farmers. They lived and worked in the rural area at the outskirts of town, which was part of the city parish. They were merged with the class of skilled workers and foremen. Table 1 shows the distribution of the social classes. In the first period, 14 percent belonged to white-collar workers, who constituted the city's economic and social elite. The proportion of the population in this class increased to almost 20 percent in the second period. The blue-collar workers were quite evenly distributed over the three classes: skilled, lower-skilled, and unskilled workers, with minor changes in the distribution over time.

[2] The coding of occupations in SEDD has been harmonized within SwedPop (see: http://www.swedpop.se).

Table 1. Distribution of time at risk (person-years) of the explanatory variables (%).

	1892–1915	1916–1939
WC-share in block		
Q1	22.8	23.9
Q2-Q4	68.1	70.1
Unlinked	9.1	5.0
Social class		
White-collar workers	14.0	19.6
Skilled workers	26.4	27.4
Lower-skilled workers	20.9	23.7
Unskilled workers	30.0	23.8
Missing occupation	8.7	5.5
Mother present in household	96.0	96.0
Father present in household	88.1	86.7
Birth order (mean (min, max))	2.9 (1, 19)	2.5 (1, 15)
No. of siblings		
0	18.2	31.4
1	22.2	26.0
2-4	45.4	32.7
5+	14.2	9.9
Birth year (mean (min, max))	1901.2 (1887, 1915)	1923.7 (1911, 1939)
Mothers age at birth*		
<20	1.8	2.9
20–24	17.5	21.7
25–29	28.9	30.0
30–34	24.8	23.7
35–39	17.8	14.7
40–45	9.1	6.9
Population density (all individuals)		
Q1	22.5	23.4
IQR	45.8	47.9
Q4	22.5	23.9
Not linked to block	9.1	5.0
Season		
Dec–Feb	24.8	24.7
March–May	25.2	25.2
June–Aug	25.2	25.2
Sep–Nov	24.8	24.9
Adult surv. years per year & block (mean, (min, max))	224 (10, 719)	265 (10, 744)
Number of individuals	12,401	10,071

Source: The Scanian Economic-Demographic Database, Version 8.2 (Bengtsson et al. 2025; Hedefalk and Souza Maia 2023).

Our measure of the neighborhood socioeconomic status, the *WC-share*, is the average monthly share of adult white-collar workers (18 years and older) within the block of residence. This is calculated by dividing the total time-at-risk for all white-collar residents by the total time-at-risk for all adult residents in the block. These monthly WC-shares are categorized into

quartiles based on the distribution for each given month, from Q1 with the lowest share and Q4 with the highest share. Blocks with less than 10 years of time-at-risk for a given year (approximately 0.5 percent of the risk time) were excluded, as these primarily represented single-family homes. Children who could not be linked to a block were categorized as "unlinked" (9 percent of the total time-at-risk, 1892–1915, and 5 percent, 1916–1939, see Table 1).

Figure 1 illustrates the various blocks in Landskrona, along with the WC-share, for the two periods. The low-status areas are concentrated in the northern parts of the city, towards the northwest, while the highest-status areas are in the city center and in the outlying area of Borstahusen in the west. Over time, the eastern part of the city, close to nearby industries, becomes increasingly low-status (for a more detailed analysis of the socioeconomic segregation over time in Landskrona, see Brea-Martinez et al. 2024).

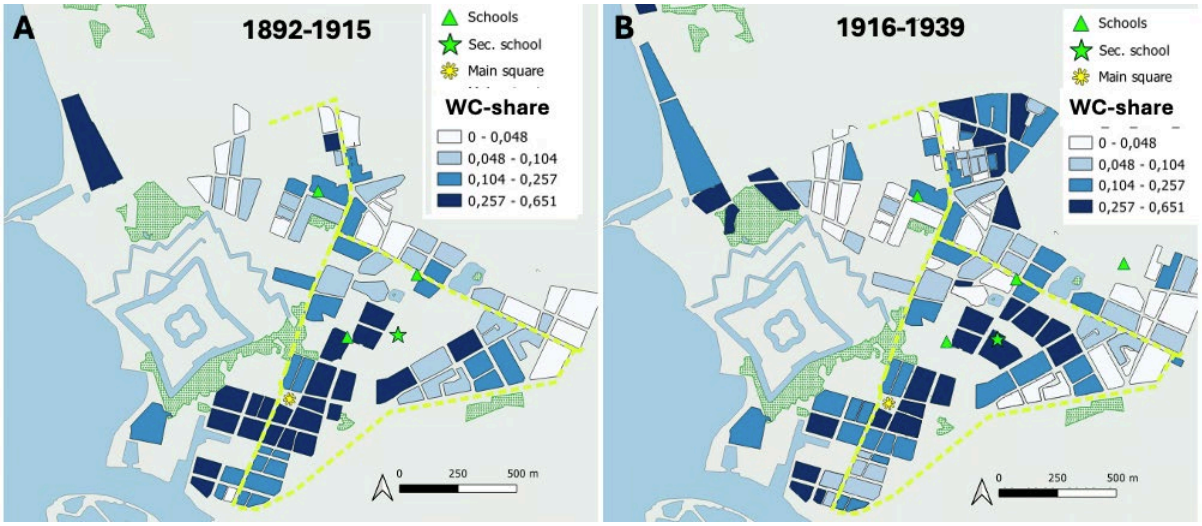


Figure 1: Adult average White collar (WC) share by blocks in Landskrona. A) 1892–1915; B) 1916–1939.
Source: See Table 1.

We divide causes of death into three groups based on the age-specific historical ICD-10h framework developed by Reid et al. (2024)[3]: 1) Airborne infections, which cover respiratory diseases transmitted through droplets or aerosols and include illnesses such as tuberculosis, measles, whooping cough, influenza, pneumonia, and scarlet fever; 2) Food- and waterborne infections, which cover enteric diseases spread via the fecal-oral route, including cholera, typhoid, and various bacterial gastroenteritis; and 3) Other causes, including all remaining ICD-

[3] The coding of causes of death were made within SwedPop, see Hiltunen, Maltesdotter and Edvinsson (2025) and swedpop.se. See also Janssens (2021) for a discussion on the historical coding of causes of death.

10h codes, such as injuries, congenital anomalies, ill-defined diagnoses, and unidentified causes. This classification highlights two main environmental factors contributing to child mortality: crowding and air quality, and sanitation and water supply. This approach enables us to examine how neighborhood contexts influence child mortality through different mechanisms over the study period.

3.3. Statistical models

We estimate Cox proportional hazards models to analyze the impact of neighborhood adult WC-share on all-cause under-five mortality, and Fine-Gray competing-risk models to analyze cause-specific mortality (Fine and Gray 1999). Separate models are also run for males and females, infants and children (1–4), neonatal and post-neonatal infant mortality, and the two time periods (1892–1915 and 1916–1939). Right censoring occurs at out-migration or when residing in a block with less than 10 years of time-at-risk for a given year.

We include the following control variables: birth year, birth order (continuous and time-invariant), presence of mother and father (binary and time-varying), number of siblings (categorical and time-varying: 0, 1, 2–4, 5+), mother's age at childbirth (categorical and time-invariant: <20, 20–24, 25–29, 30–34, 35–39, 40–45), season (categorical and time-varying: winter (December–February), spring (March–May), summer (June–August), autumn (September–November)), and block population density (monthly total time-at-risk for all individuals in the block, categorized into quartiles for each month and displayed using the interquartile range). The individual and family variables are commonly included in analyses of infant and child mortality (see, e.g., Lynch and Greenhouse 1994 for an analysis of Sweden), and the population density is included to adjust for higher population density, which could be an important mechanism in the social neighborhood effect. The season variable is included to account for seasonality in exposure to infectious diseases and in mortality. The distributions of all variables in the two sub-periods used in the analysis are shown in Table 1.

The basic model (Model 1) adjusts only for neighborhood WC-share, sex (for combined models), and birth year. Model 2 adds social class and Model 3 adds neighborhood WC-share. Finally, Model 4 adds family-level controls, including birth order, presence of parents, number of siblings, mother's age at birth, season, and block population density. This stepwise modeling approach allows us to assess both the gross neighborhood associations and to the extent to which they are accounted for by the variables included in the full model.

4. Results

4.1. Descriptive patterns

First, we look at the distribution of the grouped causes of death. We observe a total of 1,855 deaths (see Table 2). Overall, other causes constitute the most frequent cause-of-death group, followed by airborne infections and food- and waterborne infections. Among food- and waterborne deaths, the highest concentration occurs among infants 1892–1915; the highest concentration of airborne causes appears among child deaths 1892–1915, and the highest concentration in other causes occurs for child deaths 1916–1939.

Table 2. Distribution of cause-specific deaths by period, age and sex.

	1892–1915 (%)				1916–1939 (%)			
	Infants		Ages 1–4		Infants		Ages 1–4	
	Boys n=449	Girls n=335	Boys n=227	Girls n=240	Boys n=284	Girls n=177	Boys n=77	Girls n=66
Cause of death								
Airborne (incl TBC)	25.8	31.0	37.4	55.0	27.1	32.8	28.6	31.8
Food-/waterborne	21.6	20.9	7.9	5.0	10.6	8.5	6.5	3.0
Other	52.6	48.1	54.6	40.0	62.3	58.8	64.9	65.2
Total deaths in period	1,251				604			
Number of individuals in period	12,401				10,071			

Source: See Table 1.

Table 3 shows a more detailed subdivision of causes of death, where neonatal mortality (first 28 days) is also separated from post-neonatal infant mortality (29-365 days). Neonatal mortality is dominated by congenital and birth disorders (68 percent), while post-neonatal infant mortality is dominated by airborne diseases (41 percent) and food- and waterborne diseases (25 percent). There has been some concern in previous research on historical cause-specific infant mortality about convulsions, which sometimes constituted a large proportion of all infant deaths (see, e.g., Mühlichen and Cilek 2024 for Rostock; see also discussion in Reid and Janssens 2025), but in other cases were very rare as a stated cause (e.g., in Sundsvall, Sweden, see Hiltunen Maltesdotter and Edvinsson 2025). As is clear from Table 3, only about 2 percent of infant deaths, both neonatal and post-neonatal, were classified as convulsions in our sample. For child mortality, airborne, respiratory, tuberculosis, and other causes are the most frequent, while food- and waterborne diseases are much less prevalent, as was also shown in the aggregate grouping in Table 2.

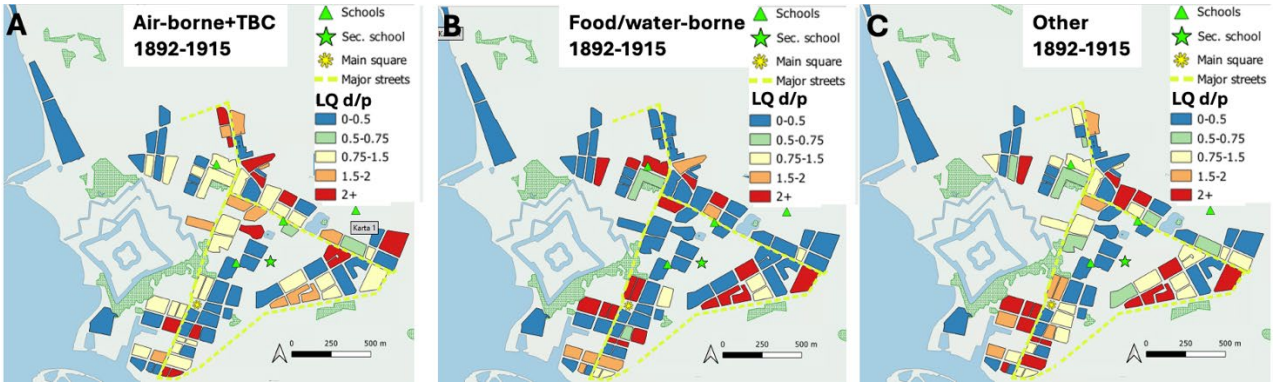
Table 3. Distribution of detailed cause-specific deaths, 1892–1939 (%).

Cause of death	Neonatal 0–28 days (n = 457)	Postneonatal 29d–1y (n = 788)	Ages 1–4 years (n = 610)
Airborne			
Airborne[1]	6.3	41.4	25.7
Tuberculosis	–	–	16.9
Food-/waterborne			
Food-/waterborne	3.3	25.0	6.1
Other			
Respiratory	–	–	22.8
Congenital and birth disorders	67.6	10.2	–
Other infectious	8.1	2.4	1.1
Other	9.4	15.1	23.4
Convulsions	2.4	2.2	0.2
Weakness	0.4	0.6	0.2
External causes	0.7	2.0	3.3
Ill-defined and unknown	0.7	0.4	0.2
No cause given / blank	0.4	0.1	–
Stated to be “unknown”	0.4	0.4	0.2
Unknown	0.2	0.3	–
Total	100	100	100

Note: [1] This group includes the causes “Airborne,” “Other airborne,” and “airborne infectious” for ages 1-4. For infants, only the cause “Airborne” is included.

Figure 2 displays maps of Location Quotients (LQs) for the three cause-of-death groups in Landskrona during the periods 1892–1915 and 1915–1939. These are relative cause-specific death rates in the neighborhood compared to the city, and values above 1 indicate a higher-than-average risk of death for that cause. For example, a value of 1.5 means that the mortality rate in the block from the specific cause is 50 percent higher than the city average. For all causes, deaths are geographically evenly distributed throughout the city. However, mortality from airborne and food- and waterborne diseases is lower than the city average in the newly developed areas in the outskirts of Landskrona during the last period, and consistently lower in both periods in the more affluent part of the city center near the secondary school (panels A and B).

a. 1892–1915



b. 1916–1939

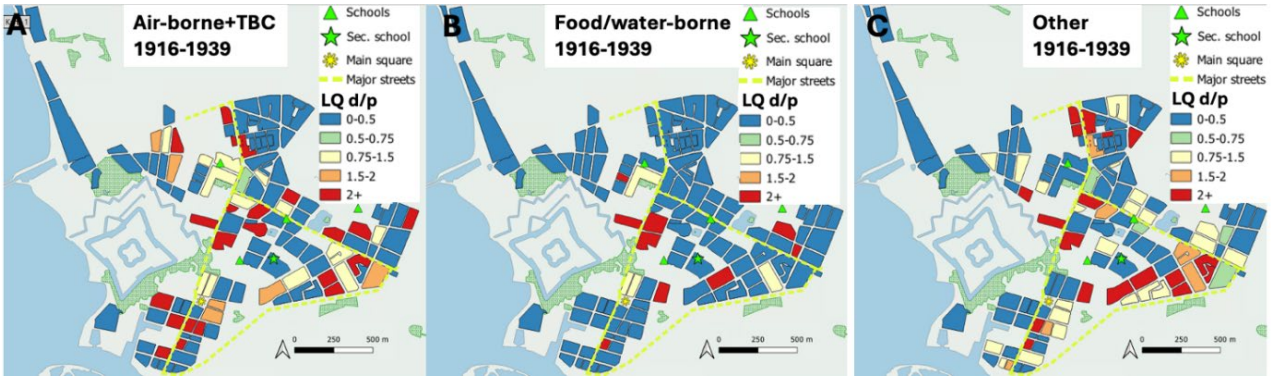


Figure 2: Cause-specific deaths of infants and children shown in Location Quotients (LQ). A) Airborne, incl. TB; B) Food/waterborne; C) Other causes.
Note: Location Quotient (LQ) for a given period = (cause-specific child deaths in the block ÷ children who ever lived in the block during the period) ÷ (cause-specific child deaths city-wide ÷ all children in the city); LQ > 1 signals higher-than-average risk, LQ < 1 signals lower. LQ = 1.5 means that the block's mortality rate is 50 % higher than the city average; LQ = 0.50 means that the block's mortality rate is 50 % lower than the city average.
Source: See Table 1.

Figure 3 shows the seasonal pattern by age, period and cause of death. Looking first at the entire period (panels A and B), there is a winter and early spring peak in airborne mortality for both infants and children, but more pronounced for infants, and a late summer peak in mortality from food- and waterborne diseases, especially for infants. These patterns do not change much over time when mortality declines and they are similar to those reported for Sundsvall in a somewhat earlier period (Hiltunen Maltesdotter and Edvinsson 2025).

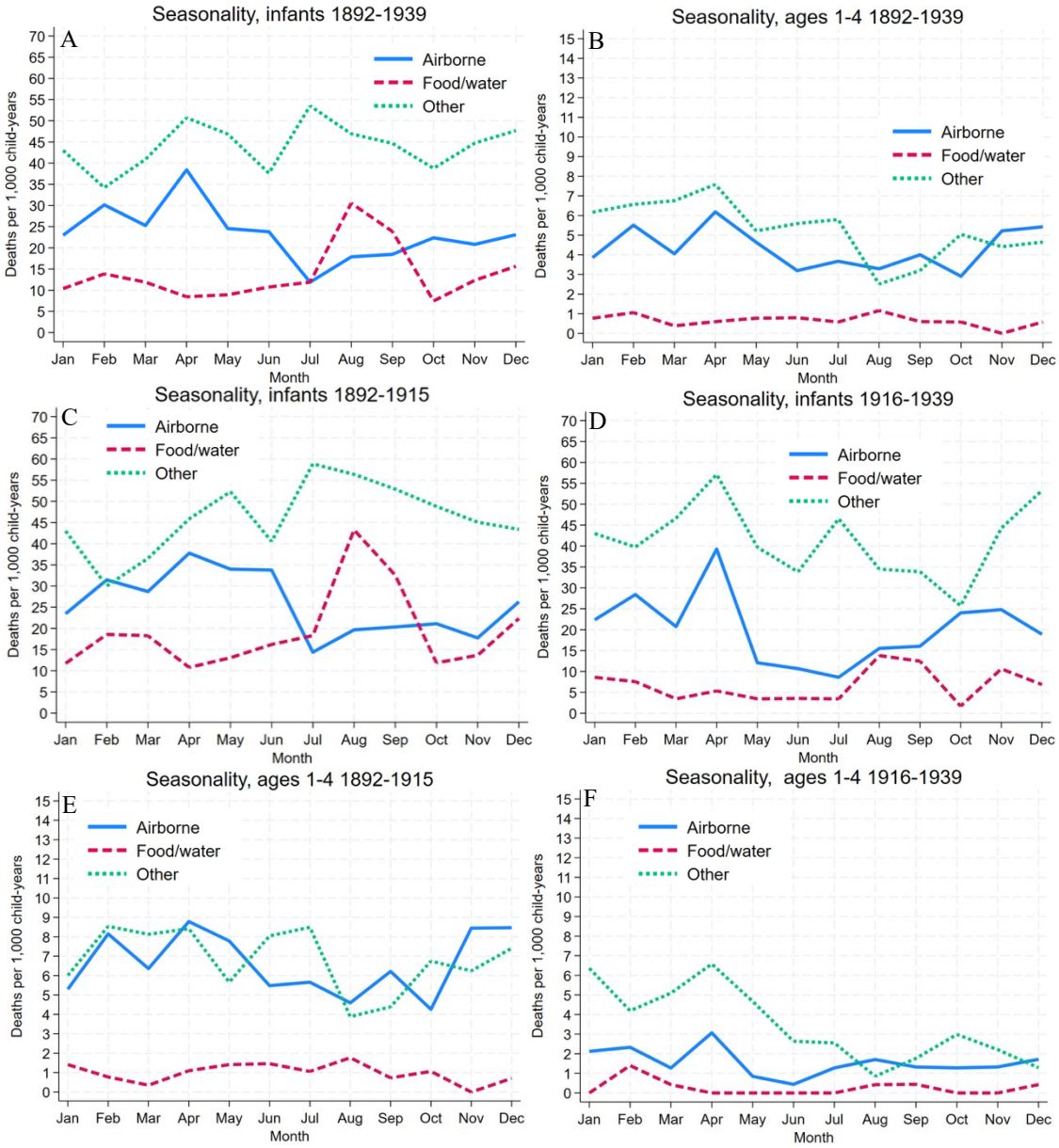


Figure 3: Deaths per 1,000 child-years and month, infants and children, 1892–1939. A) infants 1892–1939; B) ages 1–4 1892–1939; C) infants 1892–1915; D) infants 1916–1939; E) ages 1–4 1892–1915; F) ages 1–4 1916–1939.
Source: See Table 1.

We now examine mortality patterns in various neighborhoods. Figure 4 displays cumulative hazards of death under age five by the share of white-collar workers in the block. It clearly demonstrates higher childhood mortality for boys compared to girls, especially for infant mortality. Boys have between 10 and 15 percent cumulative mortality at age five, and girls between 8 and 12 percent, depending on the neighborhood. These numbers are reasonable in comparison with the national development in this period. Looking at aggregate life-table data for Sweden as a whole, the probability of dying before age five ($_5q_0$) in 1901–1910 was 13.5

percent for boys and 11.7 percent for girls. The corresponding figures for 1931–1940 was 6.4 percent for boys and 5.0 percent for girls (Statistics Sweden 1999: Table 5.6).

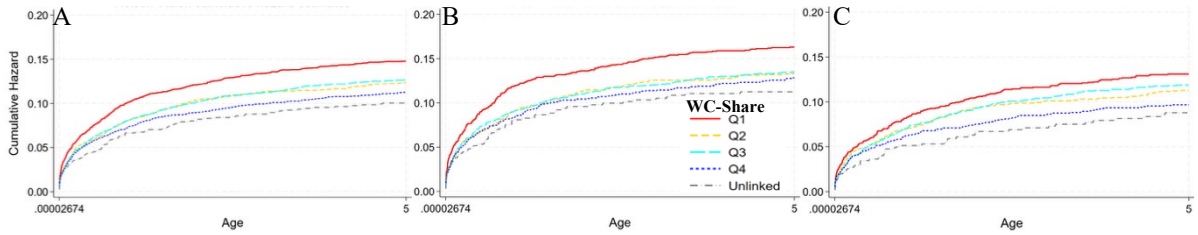


Figure 4: Nelson-Aalen cumulative hazard curves by block WC-share quartiles, infants and children, 1892–1939. A) girls and boys; B) boys; C) girls.
Source: See Table 1.

The neighborhoods with the lowest white-collar share stand out with much higher childhood mortality, and this is true for both boys and girls. Neighborhoods with the highest socioeconomic status (Q4) have the lowest mortality. Hence, especially between the highest- and lowest-status neighborhoods, mortality differences are substantial. Cumulative mortality at age five for boys in the lowest-status neighborhoods exceeds 15 percent, while it is around 11 percent in the highest-status neighborhoods. For girls, the corresponding figures are about 12 percent and 8 percent, respectively. It is also clear from the figures that differences across neighborhoods are established early in infancy and remain throughout childhood.

Figure 5 displays the cumulative hazards by neighborhood status in the two periods (panels A and B) and for infant and child mortality separately (panels C and D). Mortality declined in all neighborhoods, somewhat more in the highest-status neighborhood, leaving the absolute mortality differentials similar (between 8 and 12 percent for Q4 and Q1 in the first period and 6 to 11 percent in the final period). The unliked category had much lower mortality in the final period, but this category consists of a small group of people living outside the city itself.

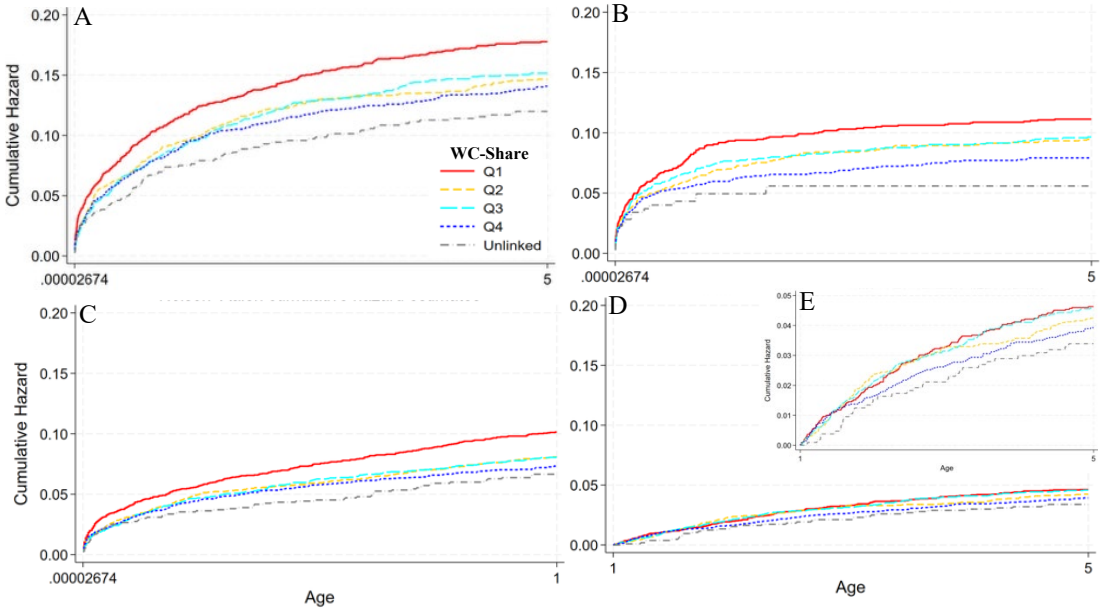


Figure 5: Nelson-Aalen cumulative hazard curves by block WC-share quartiles. A) infants and children, 1892–1915; B) infants and children, 1916–1939; C) infants, 1892–1939; D) children aged 1–4, 1892–1939; E) children aged 1–4, 1892–1939 with y-scale ranging from 0 to 0.05 for the cumulative hazard.
Source: See Table 1.

The mortality differentials by neighborhoods are more consistent and pronounced for infant mortality than for child mortality. However, also for child mortality, there is a clear difference between the highest- and lowest-status neighborhoods.

In Figure 6, we turn to the mortality patterns by cause of death. For airborne diseases and food- and waterborne diseases, there is a clear difference, especially between the highest-status and lowest-status neighborhoods, and the same is true for other causes, but the differences are smaller in this group. The patterns are less clear for neighborhoods in-between the top and the bottom (Q2 and Q3).

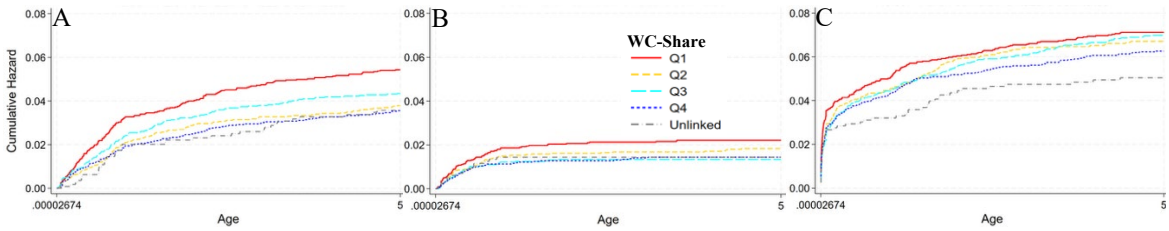


Figure 6: Cause-specific Nelson-Aalen cumulative hazard curves by block WC-share quartiles, infants and children, 1892–1939. A) Airborne; B) Food/waterborne; C) Other causes.
Source: See Table 1.

4.2. Regression results

Table 4 displays the regression models for both sexes combined, for the entire study period. Model 1 includes the neighborhood variable, sex, and birth year, and shows a similar pattern as in Figure 4. Children in the lowest-status neighborhoods (Q1) have higher mortality 0–4 years than children in the other neighborhoods (HR=1.22), and children in unlinked blocks have even lower mortality. As already mentioned, this could be because these places are located outside the city's urban area, where real city blocks do not exist. Hence, children in these places may experience more rural-like living conditions. It is also clear, as expected, that boys have much higher childhood mortality than girls (HR=1.23), and the cohort variable indicates a declining trend in childhood mortality over time.

In Model 2, we instead look at family social class, based on the occupation of the family head (usually the father). Children in white-collar families have lower mortality than the other groups (HR=0.72 compared to the unskilled), and skilled workers also have lower mortality than unskilled workers (HR=0.85). This pattern aligns with the findings reported by Dribe and Karlsson (2022) for a nearby largely rural area.

Model 3 includes both social class and the neighborhood variable, and in Model 4, other family and contextual variables are added in the full model. Importantly, the neighborhood effect remains largely unchanged when adding the control variables (HR=1.19 in the full model), and the main pattern for social class also remains. Therefore, the white-collar share in the neighborhood operates quite independently of social class and family context.

Table 4: Hazard ratios for under-5 mortality, Landskrona, 1892–1939.

	Model 1		Model 2		Model 3		Model 4	
	HR	95% CI	HR	95% CI	HR	95% CI	HR	95% CI
WC-share in block								
Q1	1.22***	1.10-1.35			1.18**	1.06-1.31	1.19**	1.07-1.33
Q2–Q4	1	RC			1	RC	1	RC
Unlinked	0.74**	0.61-0.90			0.74**	0.61-0.90	0.73**	0.59-0.90
Sex								
Boys	1.23***	1.12-1.34	1.23***	1.12-1.34	1.23***	1.12-1.34	1.23***	1.12-1.34
Girls	1	RC	1	RC	1	RC	1	RC
BirthYear	0.97***	0.97-0.98	0.98***	0.97-0.98	0.98***	0.97-0.98	0.98***	0.97-0.98
Social class								
White-collar			0.72***	0.61-0.84	0.76***	0.64-0.89	0.80**	0.68-0.94
Skilled workers			0.85*	0.75-0.97	0.86*	0.76-0.97	0.89+	0.78-1.01
Lower skilled workers			0.94	0.82-1.07	0.95	0.83-1.08	0.87*	0.76-0.99
Unskilled workers			1	RC	1	RC	1	RC
Missing occupation			0.95	0.82-1.10	0.96	0.82-1.11	0.81**	0.69-0.95
Mothers age at birth								
<20							1.24	0.95-1.63
20–24							1	RC
25–29							0.88+	0.77-1.01
30–34							0.83*	0.71-0.97
35–39							0.83*	0.69-0.99
40–45							0.78*	0.63-0.98
No. of siblings								
0							0.79	0.51-1.21
1							0.88	0.61-1.28
2–4							0.94	0.72-1.22
5+							1	RC
Birth order							1.02	0.95-1.10
Father present							0.64***	0.55-0.73
Mother present							0.69***	0.58-0.83
Population density								
Q1							1	RC
IQR							0.93	0.83-1.04
Q4							1.02	0.90-1.17
Season								
Dec–Feb							1.07	0.94-1.22
March–May							1.10	0.97-1.25
June–Aug							1	RC
Sep–Nov							0.94	0.82-1.07
Individuals	20,917		20,917		20,917		20,917	
Deaths	1,855		1,855		1,855		1,855	
Surival time	71,567		71,567		71,567		71,567	

Note: Hazard ratios from estimates of Cox proportional hazards models.
Source: See Table 1.

In Table 5, we analyze the patterns separately for boys and girls and by period based on estimates from the full model. The overall pattern is similar for both sexes. There is a clear association between the social neighborhood and childhood mortality, which is independent of social class and the other control variables. The magnitude of the differential is also similar (HR=1.29 for boys and HR=1.18 for girls). The hazard ratios for social class are also quite similar for boys and girls.

When looking at differences between the two periods, the neighborhood difference is larger in the first period (HR=1.25) than in the second period (HR=1.14, not statistically significant), whereas it is the opposite for the unlinked, where the difference is larger in the second period than in the first. The survival advantage for children from white-collar origins also grows larger between the periods, whereas the estimates for the other classes remain similar.

Table 5: Hazard ratios for under-5 mortality by sex and period, Landskrona, 1892–1939.

	Boys		Girls		1892–1915		1916–1939	
	HR	95% CI	HR	95% CI	HR	95% CI	HR	95% CI
WC-share in block								
Q1	1.20*	1.04-1.39	1.18*	1.00-1.39	1.25**	1.09-1.43	1.14	0.95-1.37
Q2-Q4	1	RC	1	RC	1	RC	1	RC
Unlinked	0.76+	0.57-1.01	0.68*	0.50-0.95	0.75*	0.59-0.96	0.63+	0.39-1.03
Sex								
Boys					1.14*	1.02-1.27	1.44***	1.22-1.69
Girls					1	RC	1	RC
Social class								
White-collar	0.79*	0.63-0.99	0.80+	0.62-1.02	0.86	0.70-1.05	0.68**	0.51-0.90
Skilled workers	0.90	0.76-1.07	0.86	0.71-1.04	0.88	0.76-1.03	0.88	0.70-1.11
Lower skilled workers	0.86+	0.72-1.02	0.89	0.73-1.08	0.88	0.75-1.03	0.84	0.66-1.06
Unskilled workers	1	RC	1	RC	1	RC	1	RC
Missing occupation	0.79*	0.65-0.98	0.82	0.65-1.04	0.82*	0.68-0.99	0.90	0.68-1.20
Individuals	10,673		10,244		12,401		10,071	
Deaths	1,037		818		1,251		604	
Survival time	36,159		35,407		39,476		32,090	

Note. See Table 4. The models control for the same variables as in model 4 of Table 4.

Table 6 presents hazard ratios for infant and child mortality separately. The neighborhood difference is only present for infant mortality (HR=1.26), while for child mortality, the hazard ratio is close to 1 (HR=1.07) and not statistically significant. There is only a gender difference in infant mortality, with higher mortality for boys. Social class differences are similar across

both age groups, with statistically significant lower mortality among white-collar workers than among unskilled workers. The hazard ratios also indicate a gradient between the two ends of the hierarchy, but they are not statistically significant in most cases.

Table 6: Hazard ratios for under-5 mortality by age, Landskrona, 1892–1939.

	Infants		Ages 1–4	
	HR	95% CI	HR	95% CI
WC-share in block				
Q1	1.26***	1.11-1.44	1.07	0.88-1.30
Q2–Q4	1	RC	1	RC
Unlinked	0.76*	0.59-0.98	0.67*	0.46-0.97
Sex				
Boys	1.37***	1.23-1.54	0.98	0.83-1.15
Girls	1	RC	1	RC
Social class				
White-collar	0.80*	0.65-0.99	0.77*	0.59-1.00
Skilled workers	0.91	0.77-1.06	0.84+	0.68-1.03
Lower skilled workers	0.88	0.74-1.03	0.85	0.68-1.06
Unskilled workers	1	RC	1	RC
Missing occupation	0.83*	0.69-0.99	0.67*	0.46-0.98
Individuals	17,035		18,516	
Deaths	1,245		610	
Survival time	14,819		56,747	

Note. See Table 5.

In Table 7, we look at neonatal mortality and post-neonatal infant mortality separately. The hazard ratio for the high-status neighborhood is larger for neonatal (HR=1.35) than for post-neonatal mortality (HR=1.22), whereas the class differences are most pronounced for post-neonatal infant mortality.

Table 7: Hazard ratios for infant mortality by age, Landskrona, 1892–1939.

	Nenonatal		Post-neonatal	
	HR	95% CI	HR	95% CI
WC-share in block				
Q1	1.35**	1.09-1.66	1.22*	1.04-1.44
Q2–Q4	1	RC	1	RC
Unlinked	0.82	0.55-1.22	0.73+	0.52-1.01
Sex	1	RC	1	RC
Boys	1.46***	1.21-1.76	1.33***	1.15-1.53
Girls	1	RC	1	RC
Social class				
White-collar	0.96	0.69-1.33	0.71*	0.55-0.94
Skilled workers	0.90	0.69-1.19	0.91	0.74-1.11
Lower skilled workers	0.78+	0.58-1.03	0.93	0.76-1.14
Unskilled workers	1	RC	1	RC
Missing occupation	0.96	0.74-1.29	0.74*	0.59-0.93
Individuals	15,780		16,502	
Deaths	457		788	
Survival time	1,221		13,599	

Note. See Table 5. Neonatal mortality refers to first 28 days of life, and post neonatal to 29–265 days.

Finally, we turn to the cause-specific analysis in Table 8. The neighborhood differences are similar for airborne and food- and waterborne diseases (HR=1.37 vs 1.39), whereas there is no difference for other diseases when controlling for all the variables in the model. In this group, the hazard ratio is close to 1 and not statistically significant. While there is no gender difference for airborne diseases, male mortality is higher for both food- and waterborne diseases and other diseases (HR=1.44 and 1.40, respectively). The class pattern is also similar for airborne and food/waterborne diseases, with lower mortality for white-collar workers, even if the estimate is not statistically significant for the latter disease group.

Table 8: Hazard ratios for cause-specific under-5 mortality, Landskrona, 1892–1939.

	Airborne (incl TBC)		Food/water-borne		Other	
	HR	95% CI	HR	95% CI	HR	95% CI
WC-share in block						
Q1	1.37***	1.14-1.65	1.39*	1.05-1.85	1.04	0.89-1.20
Q2-Q4	1.00	1.00-1.00	1.00	1.00-1.00	1.00	1.00-1.00
Unlinked	0.81	0.57-1.16	0.79	0.45-1.40	0.68*	0.51-0.91
Sex						
Boys	0.90	0.77-1.06	1.44**	1.12-1.85	1.40***	1.24-1.59
Girls	1	RC	1	RC	1	RC
Social class						
White-collar	0.76+	0.57-1.02	0.77	0.48-1.22	0.84	0.67-1.04
Skilled workers	0.87	0.70-1.08	0.89	0.62-1.26	0.91	0.76-1.08
Lower skilled workers	0.97	0.77-1.20	0.85	0.59-1.24	0.82*	0.68-0.99
Unskilled workers	1.00	1.00-1.00	1.00	1.00-1.00	1.00	1.00-1.00
Missing occupation	0.74+	0.55-1.00	0.73	0.48-1.11	0.86	0.70-1.05
Individuals	20,917		20,917		20,917	
Deaths	615		249		991	
Competing	1,240		1,606		864	
Survival time	71,567		71,567		71,567	

Note: Hazard ratios from estimates from competing-risk proportional hazards models (Fine and Gray 1999). Models control for the same variables as model 4 of Table 4.
Source: See Table 1.

5. Discussion

We analyzed how the socioeconomic characteristics of urban neighborhoods related to childhood mortality during a period of mortality decline and industrialization at the turn of the twentieth century in urban southern Sweden. Previous research has mainly focused on spatial differences at broader levels, such as rural-urban differences or differences between regions with different characteristics. There has been less research on mortality differentials at smaller spatial scales during the mortality transition, such as neighborhoods in cities. Existing research for Dublin and Montreal shows that neighborhood amenities, ethnic and religious composition, as well as socioeconomic status of neighborhoods, were associated with childhood mortality (e.g., Connor 2017; Thornton and Olson 2011).

In Landskrona around the turn of the twentieth century, children living in low-status neighborhoods had higher mortality than other children. This neighborhood association was independent of parental social class and persisted after controlling for several family and contextual factors. The association was present for both boys and girls and got somewhat weaker over the period we analyzed. Social neighborhoods mattered more for infant mortality than for child mortality. In terms of causes of death, the associations were similar for airborne

infectious diseases and food- and waterborne diseases, while no clear link was found for other causes of death. Beyond neighborhood socioeconomic status, our findings also support previous research in highlighting the importance of the child's sex and family context, including socioeconomic status, presence of parents, and mother's age, while birth order and number of siblings seem less consequential.

Landskrona was a mid-sized industrial port town with a homogenous religious and ethnic makeup (Swedish-born Protestants). We cannot empirically establish exactly through which mechanisms these neighborhood effects operated, but they were probably linked to housing standards and water and sanitation systems. However, it is also likely that infant- and childcare practices, such as breastfeeding, which was possibly related to women's work, were an important mechanism.

Looking at infant mortality, one important determinant in historical contexts is breastfeeding, or rather the lack of it (e.g., Brändström 1984; Brändström et al. 2002). Early weaning implies reliance on substitutes for breast milk, as infants are dependent on liquid nutrition. Such substitutes, in turn, are easily contaminated by impure water. Cow milk, which was a common substitute, is also vulnerable to high temperatures, which, in a time before access to refrigeration, was a serious problem in the summer months. Moreover, artificial feeding removes the immunity protection the infant gets through breast milk, which further contributes to the negative effects of the termination of breastfeeding on infant survival. In addition, artificially fed infants are also more often undernourished, as it is difficult to replace all the nutrition available in breast milk, and this was more problematic in the past than it is today, when nutrients can easily be added to various milk substitutes.

We have no direct evidence on breastfeeding at the individual level, but we know that Landskrona was located in an area where breastfeeding was generally common (Brändström et al. 2002). However, the actual practice of breastfeeding within an area likely varied by socioeconomic status, especially related to women's participation in the labor force (cf. Lee 1984; Lithell 1988). While high-status women often did not breastfeed their children themselves, they hired wetnurses to do so, which helped avoid some of the most serious negative effects of early weaning. Working-class women, however, who needed to work to supplement family income, could not afford to hire wetnurses, and had to rely on artificial feeding. This is one important reason behind socioeconomic differences in infant mortality. From a neighborhood perspective, this could also be an important mechanism. In neighborhoods where many women worked, a more accepting culture toward early weaning

may have developed, which could have affected infant survival even after accounting for actual socioeconomic status and women's labor force participation.

Another important determinant of childhood mortality is housing standards and crowding. Large families living in very small apartments promoted the spread of airborne infections and also made it more difficult to keep the living quarters clean, further contributing to contagion. Low-standard housing was also more difficult to heat in the wintertime, which made them cold and increased the risk of contracting disease. Even if we lack direct evidence on housing quality, it is reasonable to assume that neighborhood socioeconomic status was strongly associated with housing standards, with poorer neighborhoods having smaller living spaces and lower-quality housing. This could partly explain the observed neighborhood effect. The fact that mortality from airborne infectious diseases was clearly associated with social neighborhoods is consistent with housing standards and crowding being important mechanisms. Additionally, the strong neighborhood effect for neonatal mortality may be related to housing quality and especially unsanitary conditions at childbirth.

In addition to housing quality, access to clean drinking water and sewerage systems has often been highlighted as important determinants of mortality from food- and waterborne diseases. Our results showed clear neighborhood effects for childhood mortality from these diseases, which suggests that differences across neighborhoods in access to safe water and proper sanitation might have been important in accounting for the neighborhood mortality differentials in Landskrona at this time. Due to its reliance on groundwater, Landskrona appears to have had good-quality drinking water even before installing a modern piped water system (Dribe and Svensson 2024). This suggests that water quality as such may not have been as important as sanitation, but we lack detailed neighborhood-level data on this. The fact that the neighborhood association weakened over time aligns with this mechanism being important, as most of the population becamse connected to the modern system after about 1910.

We have no information available to discriminate between these potential explanations behind the neighborhood effects on childhood mortality, and there is no reason to believe that they could not all have been important. Culture and norms surrounding infant care, especially breastfeeding but also hygienic practices and cleanliness, have often been shown to be important in explaining infant mortality in high-mortality contexts, and such norms and behaviors might have varied by neighborhood socioeconomic status, beyond the immediate impact of family socioeconomic status. Similarly, it is likely that both housing standards and access to modern sanitation differed between neighborhoods, and possibly also access to water, even if this seems less likely in the case of Landskrona. Most probably, these factors interacted

to create better or worse neighborhoods for child survival. Early weaning combined with cramped, cold, and dirty housing and poor sanitation, created unhealthy environments for infants by both increasing the prevalence of infectious diseases of various kinds and the risk of getting infected and contracting disease. The nutrition and general health status, together with larger families, added to these adverse neighborhood conditions in increasing mortality risks for working-class children in industrial cities.

6. Conclusions

Our study is one of the first to analyze the interplay between social class, neighborhood socioeconomic status, and cause-specific infant- and child mortality during the mortality transition. We use detailed individual-level, geocoded demographic and socioeconomic data for a mid-sized industrial town in Sweden around the turn of the twentieth century. Our findings show an important role for the neighborhood's socioeconomic composition beyond the influence of social class and other family-specific factors. Living in a low-status neighborhood, measured by a low share of white-collar workers in the block, was associated with considerably higher infant mortality from both airborne diseases and food- and waterborne diseases. These findings underscore the importance of neighborhoods and socio-spatial inequalities in understanding infant mortality during the age of mortality decline. Although we cannot directly observe the underlying mechanisms, the cause-specific pattern is consistent with material conditions such as housing, water, and sanitation, as well as norms and behaviors related to breastfeeding, hygiene, and infant care.

Acknowledgements

This study forms part of projects funded by the Knut and Alice Wallenberg Foundation (2023.0206), the Swedish Research Council (2021-01578 and 2023-01250), the Swedish Research Council for Environment, Agricultural Sciences and Spatial Planning (2021-01409), and the Jan Wallander and Tom Hedelius Foundation and Tore Browaldh Foundation (P21-0191). A previous version of this paper was presented at the International Population Conference (IUSSP), Brisbane, Australia, July 2025, and the European Society for Historical Demography, Bologna, Italy, September 2025. We are grateful to session participants for comments and suggestions.

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