Interaction Effects of Infection and Malnutrition on Child Mortality in Scania, Sweden 1766-1894

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1 Background

Child mortality is directly dependent on nutrition and disease, and the combination of these, which in turn depends on family preferences and socio-economic conditions, as well as biological endowments.[1] This paper focuses on nutrition and disease, and the combination of nutrition and disease, for several reasons. Malnutrition and infection, and the interaction of these is important since low nutritional status makes an individual more susceptible to infectious disease, and when illness does strike, it is more severe, prolonged, and carries increased risk of permanent damage or death.[2] Infections on the other hand worsen the nutritional status and body claims are much higher during sickness.[3] But the body is also easier to infect in the first place if malnourished, since immunity is damaged by malnutrition and infection.[4] Thus, it should be of vital interest to include nutrition, disease, and the interaction between nutrition and disease in theoretical mortality models.

In this study, also the empiricial evidence of any effect on mortality of nutrition, disease, and the interaction of nutrition and disease on child mortality will be investigated. The study deals with interaction effects on child mortality, since consequences are more serious for infants and children than adults.[5] The time frame is the 18th and 19th centuries, when food productions was at such low levels that most parts of the European population was chronically malnutritioned, and any short-term food scarcity would have affected the children.[6] The empirical anlaysis uses data from SDD on five parish populations in Scania, Southern Sweden.[7] The database contains data on demographic events and economic conditions for several thousands of individuals from 1766-1894. The child mortality model accounts not only for nutrition and disease and the interaction of nutrition and disease, but also socio-economic conditions, individual characteristics, and family belonging.[8] It is used within a multilevel survival regression framework, and a period subdivision accounts for enclosures, land partitionings, and industrialisation, making it possible to compare child mortality during the transition from one type of society to another.

1.1 Studies of child mortality in Sweden

Infant mortality in Sweden during the mortality decline has been quite extensively studied, for example by Brändström, Sundin, and Tedebrand,[9] but studies of Swedish child mortality are less common. Swedish child mortality in the rural community during the 18th and 19th centuries has been investigated in the dissertations by Winberg and by Eriksson and Rogers.[10] Fridlizius, Bengtsson, and Bengtsson & Lundh have also investigated rural child mortality.[11]

[1] Mosley & Chen (1984).
[2] Scrimshaw, Taylor & Gordon (1968), Pollard (1982), Scrimshaw & SanGiovanni (1997), Mata, Urrita & Garcia (1967), Scrimshaw (2003), Gopolan (2000).
[3] Lunn (1992), Fogel (2000), Scrimshaw, Taylor & Gordon (1968), Osmani (2000).
[4] Gershwin, Beach & Harley (1985), Fogel (1986, 1994a), Pollard (1982), Chandra (1989).
[5] Scrimshaw, Taylor & Gordon (1968).
[6] Fogel (2000).
[7] Scanian Demographic Database.
[8] Based on a theoretical child mortalty model derived in Johansson (2004); Chapter 5.
[9] Bengtsson & Lundh (1999:1), Brändström & Sundin (1981), Sundin & Tedebrand (1981), Brändström (1984), Sundin (1995).
[10] Winberg (1975), Eriksson & Rogers (1978).
[11] Fridlizius (1975, 1979, 1984, 1989), Bengtsson (1999, 2000), Bengtsson & Lundh (1999).

In the dissertation by Edvinsson, mortality in the Sundsvall town is studied, and one of the age groups investigated is children of the ages 1 to 14.[12] Magdalena Bengtsson investigates infant and child mortality in the town of Linköping, but this work mainly focuses on infant mortality rather than child mortality.[13] Lithell also investigates child mortality – especially breast-feeding habits – in her dissertation, but as in the case of Magdalena Bengtsson, her main focus is infant mortality.[14] This is a common feature of many of the investigations: they deal with child mortality only briefly and focus mainly on infant mortality, or have other prime issues. Child mortality in the context of the general mortality decline in Sweden, as well as early life condition effects on child mortality and its impact on the mortality decline is investigated in Johansson (2004).[15] Thus, compared to infant mortality, child mortality in Sweden is not very extensively studied.

2 Theory and theoretical model

Malnutrition and disease, and the combination of these, are the direct causes of child mortality, which in turn depends on family preferences and socio-economic conditions, as well as biological endowments.[16] Here, the effects of malnutrition, and of disease, and the interaction of malnutrition and disease on human health and – thus – ultimately on human mortality will be the main focus.[17] Since malnutrition and infection have been shown to impair immunity, also this will be discussed, as well as interactions between malnutrition, infection, and immunity.

2.1 Malnutrition

Malnutrition causes spontaneous abortions and stillbirths, as well as infant and maternal mortality.[18] Nutritional status is ultimately determined by the availability of all essential nutrients at the cellular level required for growth, maintenance, development, repair, and functioning. Nutritional status is determined by diet and factors that condition the requirement, absorption, assimilation, and utilisation of diet nutrients, including activity level and environmental factors; in particular infections and stress. There is also a time lag between dietary deprivation and clinical undernutrition, and this time lag can vary between different nutrients and clinical sign.[19] It is, however, important to recognise that gross nutrition is in some ways not so important as net nutrition because it is body claims that determine how much nutrition an individual needs to consume.[20]

Nutrition is vital for the survival of humans irrespective of age, but especially for children adequate nutrition intake is important, because children under the age of five are most vulnerable from a nutritional standpoint.[21] For children, the time it takes to reach undernutrition depends on extent and duration of the dietary inadequacy and whether infection is also present, and can vary from four to six months after birth to four or five years.

[12] Edvinsson (1992).
[13] Bengtsson, M (1996).
[14] Lithell (1981).
[15] Johansson (2004).
[16] Mosley & Chen (1994).
[17] In the 18th and 19th centuries, disease was more or less equivalent to infectious disease – especially for children – and the discussion will hence be on infection.
[18] United Nations (1954b:18).
[19] Gopolan (2000:25-26).
[20] Fogel (1986:443-447, 1994a:236-243).
[21] Gopolan (2000:35).

But unlike infectious disease, there is no distinct difference between normalcy and disease.[22] To prevent dietary inadequacy from permanently damaging tissue (catabolism), physical activity is reduced and growth is retarded. For adults, reductions in physical activity limit productivity and thus earning capacity. In children the costs are in fact even higher since they reduce playing and other physical activities. If a child is physical inactive, this reduces stimulation and learning experience opportunities, which may retard both physical growth and mental development.[23]

2.1.1 The effect of malnutrition on disease

Nutrition and disease also interact: low nutritional status makes infections worse. Data from a large number of investigations reviewed by Scrimshaw, Taylor, and Gordon support the statement that moderate to severe undernutrition increases the seriousness of infections in humans.[24] There is also a quite direct connection between mortality from infections and nutrition for children, but not for breastfed infants.[25] Malnutrition makes individuals more susceptible to infectious disease and when illness does strike, it is more severe, prolonged, and carries increased risk of permanent damage or death.[26] Many investigations show a connection between malnutrition and the incidence of infectious disease, but the link is only firmly established for individuals with severe malnutrition – mild malnutrition seems to have less impact on susceptibility to infections.

A more certain relationship is found between nutritional deficiency and disease severity. As an example, diarrhoea can quickly result in life-threatening dehydration due to loss of water and minerals. The duration of illness is also affected by nutrition since the more severe malnutrition is, the longer illness lasts and the longer time it takes to recover. Tissue damage repair is strictly limited by nutrient supply and when demand exceeds supply, the reparative process will be slower. The outcome of infectious disease can be death or recovery, but also recovery with persistent damage or permanent damage. Even in contemporary developed countries, full catch-up in height and weight can take years and in contemporary developing countries, permanent stunting is the norm.[27]

Almost any nutrient deficiency will impair resistance to infection. Essentially all forms of immunity have been shown to be affected by nutrient deficiencies, and these immune responses are not specific to any single nutrient. Studies in immunology have shown that defence mechanisms influenced by nutritional status include interference with the production of antibodies and the bacteriocidal capacity of phagocytes, complement formation, number of T-lymphocytes and T-cell subsets, the complement system, and more.[28] However, even if it is known that undernutrition affects morbidity and mortality by increasing susceptibility to infection and decreasing ability to fight infection, it may not be possible to separate the effects on malnutrition from other effects strongly connected to malnutrition. In environments where malnutrition is common, factors that lead to the spread of infection

[22] Gopolan (2000:18-21).
[23] Gopolan (2000:25-26).
[24] Scrimshaw, Taylor & Gordon (1968:142).
[25] Lithell (1981:40).
[26] Pollard (1982:453).
[27] Lunn (1991:136-141). The possibility that growth retardation can be attributed to genetic differences is dismissed by Gopolan, who states that the genetic potential for growth and development is nearly similar among most populations, and uses the Japanese post-war increase in heights of both children and adults as an example (Gopolan (2000:30-31)).
[28] Scrimshaw & SanGiovanni (1997:464S-475S).

disease are also common. Environments where malnutrition is common are often also crowded and have poor hygiene and sanitary arrangements, which increases the spread of infectious disease. Hence, even though nutrition intake may be known, other factors also common in undernutrition environments complicate any measuring of nutritional effects on morbidity and mortality.[29] It is therefore also important to control for environment and socio-economic conditions in empirical research.

Table 2.1: Nutritional influence on outcomes (morbidity or mortality) of infections.

Definite	**Equivocal or variable**	**Minimal**
Measles	Typhus	Smallpox
Diarrheas	Diphtheria	Malaria
Tuberculosis	Staphylococcus	Plague
Most Respiratory Infections	Streptococcus	Typhoid
Pertussis	Influenza	Tetanus
Most Intestinal Parasites	Systemic Worm Infections	Yellow Fever
Cholera		Encephalitis
Leprosy		Poliomyelitis
Herpes		

Source: Journal of Interdisciplinary History (1983:506); Figure 3.

It is also important to note that not all diseases are affected by nutritional status. Even though malnutrition may increase susceptibility to some diseases, and thus that a well-nourished person may be able to avoid getting infected even if exposed to disease, certain infectious diseases are so virulent that even well-nourished individuals will get infected. This is the case with one of the most influential killers before and during the initial general mortality decline, smallpox. With another of the great killers during especially the 19th century, tuberculosis, the opposite is true.[30] The nutritional status influence on the outcome of infections in humans, either morbidity or mortality, is given in Table 2.1 for a number of important infectious diseases. Infections such as measles, diarrhoea, tuberculosis, and most respiratory infections are definitely influenced by the nutritional status of the host. Other infectious disease such as the aforementioned smallpox, malaria fever, plague, and typhoid are largely unaffected by nutritional status. Nutritional status has a variable or equivocal impact on a third group of infectious disease, e.g. in the case of typhus, diphtheria, and influenza.

It is thus clear that moderate to severe malnutrition has serious consequences for humans both in the short run and the long run, and that it is more serious for infants and children. In well-nourished persons, body reserves and normal dietary intake can avoid malnutrition unless they get prolonged infections.[31] However, during the 18th and 19th centuries, chronic malnutrition was widespread in Europe. National production of food was at such low levels that the lower classes must, under any circumstances, have been malnourished, and high

[29] Pollard (1982:454).
[30] Johansson (2004:Chapter 2).
[31] Scrimshaw, Taylor & Gordon (1968:265).

mortality was caused both by malnutrition and poor diets.[32] Thus, nutrition intake must have been an important determinant of the 18th and 19th centuries mortality; both directly and indirectly.

2.1.2 The effect of disease on malnutrition

Malnutrition and infection interaction also works in the other direction: it is not only malnutrition that affects infections but infections also cause malnutrition; however, it was not until the mid-twentieth century that it was recognised that any infection worsens nutritional status, and that this in turn reduces growth in children. The seriousness of the impact of measles, whooping cough, diarrhoea, and respiratory infections was, however, not recognised. Much of the diarrhoea in contemporary developing country populations is caused by the rotavirus, but this virus was not discovered until 1973. Before this discovery, insufficient diet alone was considered to be responsible for poor growth in children.[33]

There is conclusive evidence that almost all infections produce changes influencing nutritional status. Apart from changing absorption, metabolism, and excretion of specific nutrients, food intake is reduced by loss of appetite during infections.[34] An empirical study of Guatemalan children has shown an increasing frequency of infections and infectious disease in case of malnutrition, as well as an association between infectious disease and failure to gain weight. The study also demonstrates statistical significant synergistic interaction between malnutrition and infection in form of correlation between inadequate diet, poor growth, and high morbidity.[35] Thus, this study showed evidence of an interrelationship between infection and malnutrition in a setting not very different from the conditions during the demographical transition.

In studies of populations in developing countries conducted by Scrimshaw and colleagues during the 1960s, all acute infections resulted in reduced appetite and metabolic losses of nitrogen and other nutrients in the urine. Gastro-intestinal infections decreased nutrient absorption and fever increased metabolism. They also assumed an internal diversion of immune response nutrients.[36] Even with minor infections, the rate of protein breakdown increases at the same time as appetite decreases, and protein and other nutrient deficiencies are accelerated. An infection increases the basal metabolic rate, which may double the energy requirement. Increased demand for glucose may deplete glucose stores in muscle fat. Carbohydrate and fat metabolism are also affected. Since the protein and fat reserves that provide amino acids and energy are low during infection, the outcome of such an infection may be more serious for a malnourished person because his or her reserves would be depleted even under ordinary circumstances; thus, even without the infection.[37]

2.1.3 Immunity

Also specific defence mechanisms are influenced by nutritional status – many forms of immunity are affected by nutrition deficiencies.[38] For the immune response to be able to

[32] Fogel (2000:267, 271).
[33] Scrimshaw (2003:316S-318S).
[34] Scrimshaw, Taylor & Gordon (1968:24, 55, 58-59).
[35] Mata, Urrita & Garcia (1967:124-125).
[36] Scrimshaw (2003:319S).
[37] Pollard (1982:454).
[38] Scrimshaw & SanGiovanni (1997:464S-476S).

work properly, the human body needs nutrition. Apart from skin and mucous membranes, the immune response is supposed to account for the resistance to infectious disease in humans, and since malnutrition decreases the immune response, it also decreases resistance to infection. Hence, a well-nourished person has an immune defence system that can resist attacks from most infectious diseases, while the immune defence system of a malnutritioned person will not be able to resist these diseases.[39] Chandra states that nutrition is a critical determinant of immuno-competence and illness. Young children with protein-energy malnutrition experience increased mortality and morbidity, mainly from infectious disease.[40] One effect of low nutritional status is reduced production of hydrochloric-acid in the stomach, and this makes the pH-level higher than normal, allowing multiplication and passage into the small intestine of pathogens responsible for diarrhoeal disease. It may result in sufficient numbers of *cholera vibrio* organisms to be life threatening.[41] Also, deficiencies of vitamin A, B, and C can impair immunity seriously.[42]

2.1.4 Interaction between malnutrition, disease, and immunity

As is clear from the previous discussion, infections and nutrition deficiency will also affect immunity, but there is also an interaction between these processes. Infection worsens nutritional status and malnutritioned individuals are more easily infected, and both malnutrition and infection impairs immunity. The combination of infectious disease, reduced food intake, and altered metabolism is associated with growth and maturation restriction in young children.[43] The combination of low nutrition intake and disease was probably a larger threat to the 18th and 19th century's populations than either nutrition deficiency or infection alone. This could be caused by several mechanisms, such as decreased nutrient absorption, impaired immunity, etc. Even if food is available and consumed it may not be effectively absorbed due to infectious disease, for example diarrhoeal infections. Such infections are associated with decreased absorption of all three major nutrients: carbohydrate, fat, and protein, but also vitamins and trace elements. Intestinal transit time is reduced, which allows for less absorption time and pathogen-induced damage to the intestinal mucosa.[44] Fogel states that high disease rates during the 18th and 19th centuries must have caused malnutrition because of the high claims the high disease load would have imposed on human bodies, even if diets had been extraordinary.[45]

2.1.5 Vicious circles caused by malnutrition, disease, and impaired immunity interaction

Body claims will be much higher in case of sickness since the body then needs extra nutrition to conquer the disease.[46] But malnutrition would make the body easier to infect in the first place. Undernourished individuals have been shown to have impaired immune responses, and combined with other factors common in societies with food scarcity this leads to more infections, which in turn changes the physiology to worsen nutritional status even more.[47] Hence, several factors affects each other and can lead the individual into a vicious circle where low nutrition leads to impaired immune response, leading to higher susceptibility to

[39] Pollard (1982:454).
[40] Chandra (1989:607-610).
[41] Gershwin, Beach & Harley (1985:95).
[42] Pollard (1982:454).
[43] Scrimshaw, Taylor & Gordon (1968:59).
[44] Lunn (1992:135).
[45] Fogel (2000:277).
[46] Fogel (1986:443-447, 1994a:236-243).
[47] Chandra (1989:607-610).

infectious disease which reduces nutrition intake and so forth, often ending in mortality. This is common in many contemporary developing countries but must also have been the case in 18th and 19th century Europe.

2.2 Conclusion on malnutrition, disease, impaired immunity, and their interaction

It is worth noting that not only one but two or more mechanisms can be in operation at the same time. Chandra claims that epidemiological observations have confirmed that infection and malnutrition aggravate each other,[48] but – as stated previously – nutrition does not affect all infections in the same way.[49] Scrimshaw points to the fact that the combined effect of disease and low nutrition makes it harder for the body to absorb nutrition when in fact extra nutrition is needed because of the sickness.[50] Such combined effects may have a very strong effect and it is not farfetched to have hypohteses that such interaction effects could be very strong, and the conclusion from this section is that such interaction effect should be included in child mortality models. Gopolan draws attention to that it can take children anything from months to years, depending on if infection also is present, to reach undernutrition, which suggests that the theoretical model also should need time lags to account for delayed effects of malnutrition, disease, and their interaction.[51] Hence, this section shows that, additionally of malnutrition and disease load, there is a need to include interaction between malnutrition and disease in the theoretical child mortality model, and also for lagged effects of malnutrition and disease.

2.3 Theoretical model

The starting point for the theoretical child mortality model will be among the medical science approach intermediate determinants: mortality depends directly on nutrition, disease, and combinations of these. These intermediate determinants in turn depend on family socio-economic conditions, parental values and preferences etc, but also biological endowments. These biological endowments are considered to be exogenous.

2.3.1 Determinants of child mortality

The previous section stated that nutrition, infection, and the interaction of nutrition and infection determines mortality, but what determines nutrition and infection? The theoretical child mortality model use here is based on the theoretical model in Johansson (2004) that is in turn building on the Mosley & Chen (2004) framework, and it is showed in the model used here, in Figure 2.1. It states that child mortality is determined by environment, food supply, and disease load at the community level, family wealth, family knowledge, family values and preferences, and genetics at the family level, and maternal factors as well as sex at the individual level.

[48] Chandra (2002:S73).
[49] Table 2.1.
[50] Scrimshaw (1985:332-36).
[51] Gopolan (2000:18-21).

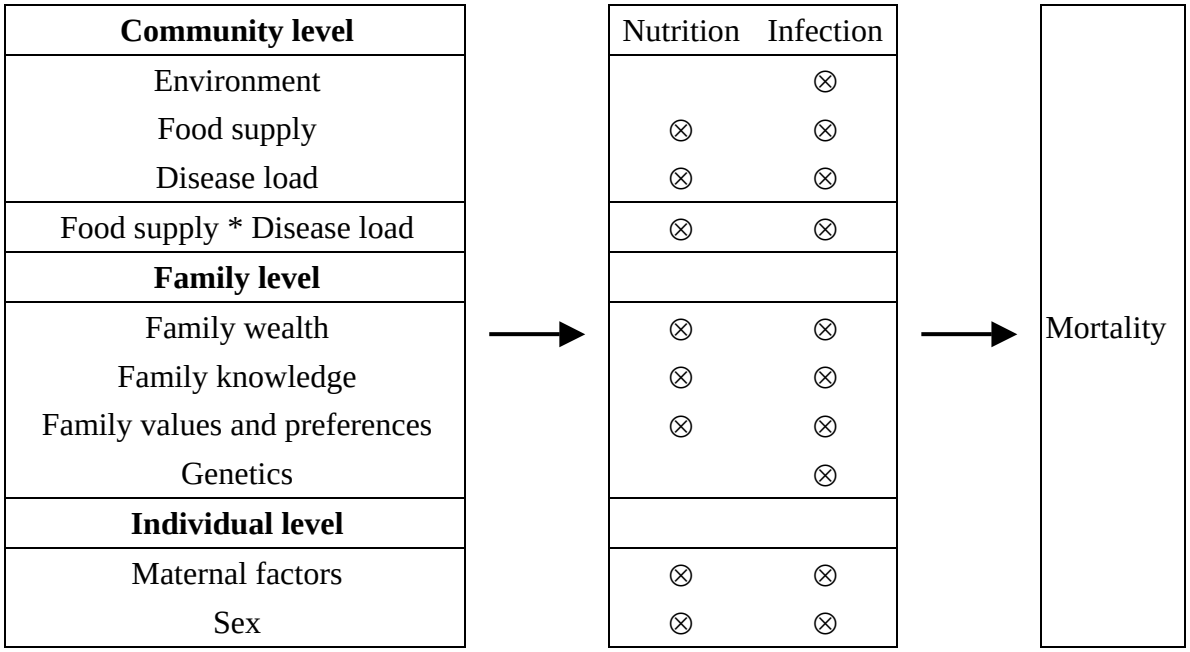


Figure 2.1: A theoretical multi level model for child mortality combining the social science and the medical science approaches.

As seen in the model, these determinants in turn affect the intermediate determinants of mortality disussed earlier; nutrition and infection. The indirect mortality determinants in the left column represent the social science contribution, while the middle column of direct determinants is the medical science contribution to this theoretical model. Most of the background determinants affect both nutrition and infection, but environment and genetics are not assumed to have any direct effect on nutrition.

The theoretical model here uses both the social science and the medical science approaches in a multi level concept, and combines period factors and cohort factors. It explains child mortality differences with background determinants to the intermediate determinants of mortality, nutrition and infection, at three levels: the community level, the family level, and the individual level. At community level, this model includes environment, food supply, and disease load. At the family level, the theoretical model uses family wealth, family knowledge, family values and preferences, and genetics to explain child mortality. The individual level consists of maternal factors unique to each individual and sex. These determinants account for both short-term and long-term effects on child mortality.

2.3.2 Variables in the theoretical model

The variables in the theoretical model here are based on Johansson (2004:Chapter 5) where a thorough discussion is to be found on the selection of variables. Only a short discussion on the food supply variable will be undertaken here.

Section 2 stated that both nutrition and infection determine mortality and that nutrition is crucial for human survival in part because it affects susceptibility to disease and general

immunity. Thus, food supply plays a crucial role for mortality because if food supply is low, nutritional demands cannot be met. Today, food supply is not usually a problem in developed countries, but in developing countries the food supply is usually inadequate and irregular, just as it was in 18th and 19th century Europe. If food supply is highly volatile, food intake and food diversity is also highly variable. Food shortage causes nutrition deficiency and this increases susceptibility to infectious disease, as concluded in section 5.3. As an example, the food supply in 18th and 19th century Sweden was highly volatile since harvests varied greatly from year to year. This was reinforced by the high costs of holding stocks, which has been estimated at 20-30 % of the initial value: wastage was at least 10 %, storage costs 3-5 % and interest rate should be somewhere between 5 and 15 %.[52] The combination of variable harvests and high costs of storing stocks made smoothing out consumption difficult, and both fertility and mortality were affected. Research on modern famines has showed that even small changes in food supply could result in famines.[53] In cases of inadequate food supply in a community, pregnant women and infants are the first to show the effects of food shortage, regardless of whether the shortage is in quality or quantity.[54] An extension of the theoretical model – based on the discussion in the beginning of this paper – is that it should contain a interaction of food supply and disease load.

3 Data

To test the theoretical model in the section above, two things are needed. One is data, and the other is an empirical model; thus, a model that can be used for estimation with the data at hand, and is the operationalisation of the theoretical model. The empirical model is derived in the following section, while this section describes the populations and areas the empirical data set used here is based on. It also derives a periodisation.

3.1 Database, time frame, and age selection

The empirical part of this study uses information on individuals from the five parishes of Halmstad, Hög, Kågeröd, Kävlinge, and Sireköpinge in the Scanian Demographic Database (*Skånes demografiska databas*).[55] Marriages, deaths, and births have been collected and linked together by family reconstitution in this database. Marriages have been recorded and births within these marriages linked to parents, as well as deaths and migration. Additional information from the poll tax registers (*mantalslängderna*) of the size as well as the type of land holding have then been linked to this information. Complementary information from the land registers (*jordeböckerna*) regarding land type and land size as well as property rights have also been added.[56] Bengtsson and Lundh have showed that the database has a very high accuracy.[57]

The time period used here from 1st of January 1766 up to the 31st of December 1894. The time selection starting point stems from the fact that the linking of information on landholding and other information related to economic conditions start in 1766. The end

[52] Persson (1996:692, 699-701).
[53] Persson (1996:692, 701).
[54] United Nations (1954b:18-19).
[55] The database is described (mainly in Swedish) in the Scanian Demographic Database homepage www.ehl.lu.se/database/sdd.htm.
[56] Reuterswärd & Olsson (1993:1-8).
[57] Bengtsson & Lundh (1991:11-13, 1993:21-22).

point selected is equal to when the Scanian Demographic Database is terminated. A sample consisting of children from age 1 to age 14 is selected since the age-group of interest here. The age group 0 is considered to be infants while the population at age 15 and above are regarded as adults.[58]

Only children with known sex and who had mothers with known birth year are selected. Unknown sex of a child or age of a mother at birth of child is taken to be a sign of unreliable data for this individual. Such individuals are therefore removed from the sample to improve data quality. The first case is very unusual: not more than a couple of children have no record of sex. The second case is also quite unusual, and in about half the cases where maternal age is unknown, paternal age is also unknown. No children of higher-class families such as children of teachers, priests, or estate-owners have been included in the sample since they are very few.

3.2 A periodisation of the period of investigation

In the years between 1766 and 1894, Swedish society changed dramatically: in less than 130 years, Sweden experienced three enclosures, the commercialisation of agriculture, and the beginning of the industrialisation. At the end of this period, the country was on its way to being transformed from an agricultural society into an industrialised and urbanised society. As late as 1870, over 70 % worked with agriculture, forestry, or fishing, and only about 15 % in industry. Only 30 years later, these shares had changed to just over 50 % and about 30 % respectively.[59] The demographic changes were also dramatic, with a population increase of over 150 % and a rise in life expectancy by about 14 years. These economic and demographic changes cannot be analysed as independent of each other: the changes in the economy affected the changes in demography and vice versa. Industrialisation rested on the development of wage labour, which would have been impossible without the proletarisation of the rural areas caused by the population increase. On the other hand, development of the economy and a more productive agricultural sector were prerequisites (for the ability) to feed the growing population. This interdependency of the economy and the demography in this period is central to any analysis of Swedish economic history.

In close connection with the beginning of the period of investigation, the first enclosure movement took place in Sweden, the *Storskifte* act of 1749.[60] The purpose of the *Storskifte* was to decrease the large number of strips. It did not imply any break-up of the village, but the ones who wanted to leave the village were free to do so.[61] The *Storskife* act was followed by the *Enskifte* act of 1803, limited almost entirely to Scania.[62] The purpose of the *Enskifte* was to bring the many small strips into one or a few fields, but this meant that the farms had to be broken up from the original village (*bysprängning*).[63] The *Enskifte* act made it possible to enclose even if only one of the peasants in a village wanted to enclose.[64] As a consequence, the old village was broken up and the new farms were isolated from each other. The *Enskifte* thus had a very large impact on the village but because of the commercialisation

[58] Most figures and tables in this section are (still) based on a sample without Kågeröd; thus on a sample with four parishes (Halmstad, Hög, Kävlinge, and Sireköpinge).
[59] Statistiska Centralbyrån (1955); Figure 3 and Figure 8, Schön (2000:53-63).
[60] Heckscher (1941:199-201).
[61] Fridlizius (1979b:3).
[62] Utterström (1961:193).
[63] Heckscher (1941:205-206). Sometimes it was not possible to enclose so that each peasant got just one piece of land, and they then had to settle for an enclosure solution with two or more pieces of land.
[64] Heckscher (1941:201-204).

of the agricultural sector at the beginning of the 19th century and the increasing grain prices, there was usually at least one peasant who wanted to seize the opportunity and increase his land holding to profit from increased production possibilities. Further, the rent on crown land and the tax on freehold land was fairly stable, which – combined with the grain price increases – stimulated grain production for the market, since increased production did not mean increased tax since tax payment was fixed per unit of land.[65] Since it only required one peasant to pursue enclosure, the *Enskifte* enclosure became very common, despite its dramatic effect on the original village. In 1827, Sweden experienced its third enclosure in the form of the *Laga skifte*.[66]

In Hög and Kävlinge there was *Storskifte* in the 18th century and *Enskifte* in 1804. In the two other parishes, the enclosures came, in general, much later except for parts of Halmstad, where Halmstalund already had the *Enskifte* in 1811, and Halmstad in 1827. Loarp's Village and the Duveke Estate had the *Laga skifte* in the mid-19th century. In Sireköpinge, there was *Laga skifte* in 1849 in Kläsinge, Norraby, Sireköpinge, Spargott, and Tågarp.[67]

The dramatic changes in society due to the enclosures mentioned above, the commercialisation of agriculture, and industrialisation are used for a periodisation of the full 1766-1894 period.[68] The first sub-period concerns the traditional agrarian society in the period after the first enclosure, the *Storskifte* of 1749. It includes the years from 1766 to 1814 and is called the pre-transformation period. The second sub-period begins when the second enclosure, the *Enskifte* of 1803, had affected most of the Scanian villages with freeholders and crown tenants. As stated above, Hög and Kävlinge had the *Enskifte* already 1804. In fact, the peasant regions in the plains were already enclosed within a decade, and in 1822, more than half the peasant land in Scania was enclosed, and almost half of the old holdings moved out.[69] This sub-period also includes the last of the enclosures, the *Laga skifte*. The manorial regions were enclosed later and the process was slower, but as seen above, there were some early enclosures in Halmstad and both Halmstad and Sireköpinge were enclosed before 1850. Most of the other manorial regions in Scania were also enclosed by this point in time.[70] Because of the transformation into a commercialised agriculture, the 1814-1865 sub-period is called the transformation period. In the last sub-period, Sweden started its transformation into an industrialised society and it is thus called the industrialisation period of 1865-1894. The periodisation will make it possible to study how these changes affected child mortality, and is thus vital for the empirical analysis.

3.3 The five parishes

3.3.1 Land structure

In the five Scanian parishes, the land structure can be divided into three groups according to land ownership. One group consisted of tenants on crown land (*kronobönder*), another of tenants on noble land (*frälsebönder*), and a third group of freeholders (*skattebönder*). There were no tenants on church land. Of the three groups, tenants on crown land and freeholders

[65] Herlitz (1974:354-363), Fridlizius (1979b:6).
[66] Heckscher (1941:207).
[67] Bengtsson & Dribe (1997:29); Table 4.2.
[68] This periodisation is based on Bengtsson & Dribe (2002:12).
[69] Heckscher (1941:203), Fridlizius (1979b:3, 7).
[70] Fridlizius (1979b:3).

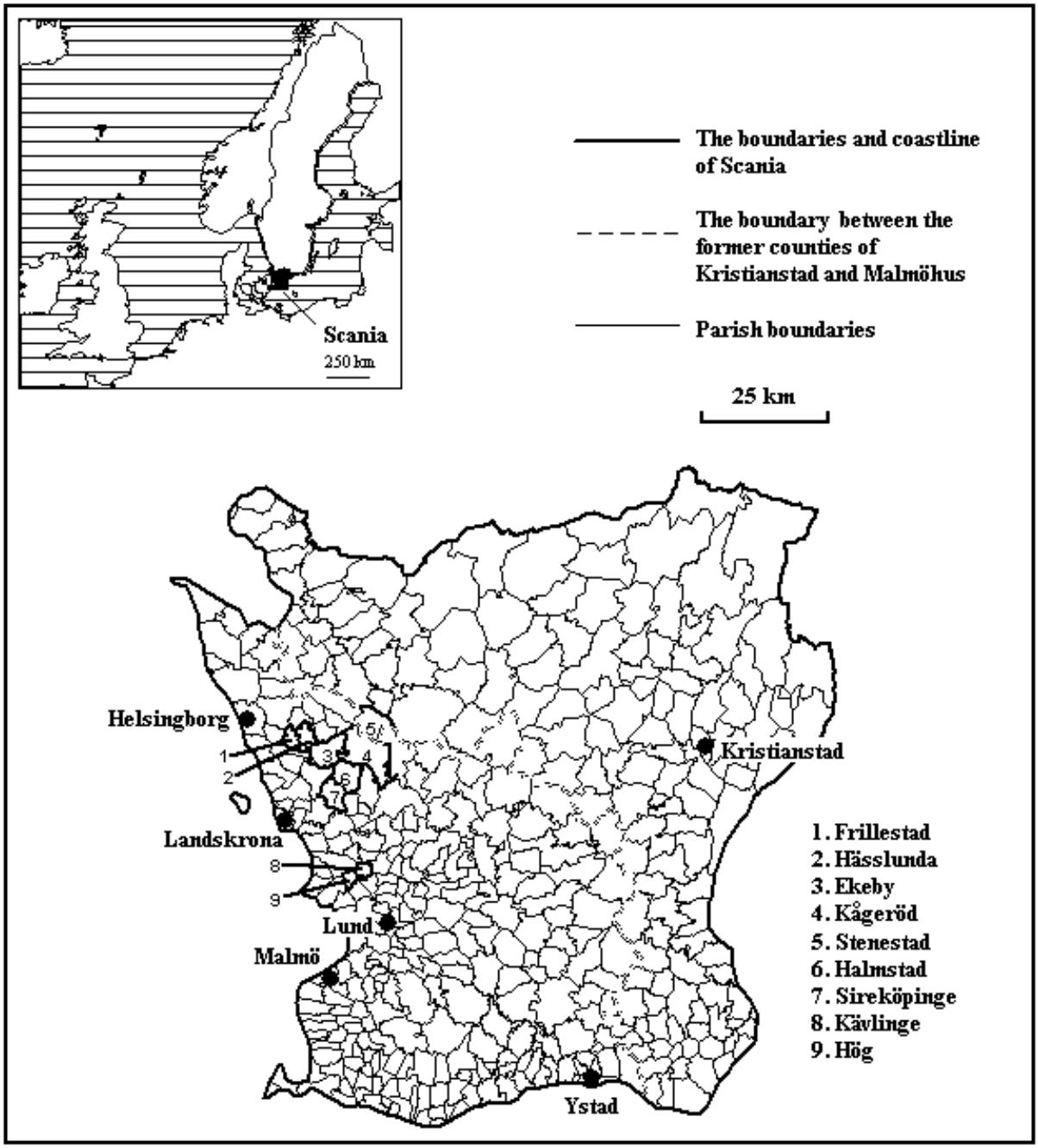


Figure 3.1: Map of Scania, Sweden and the five parishes in the Scanian Demographic Database: Halmstad, Hög, Kågeröd, Kävlinge, and Sireköpinge.

were most alike: both groups held land related to the crown. In the freeholder case, the peasant had bought his land from the crown and had to pay tax to the crown for it. The opportunity to buy the land (*skatteköp*) was introduced in 1701, but it was first after the more favourable new act of 1723 the *skatteköp* became popular. The Scanian freeholder land area more than doubled between 1725 and 1772. In 1773, a new act prohibited the *skatteköp*, but new acts in 1789 and 1793 made it possible to buy land from the crown again. At the turn of the 18th century, the Scanian crown land was almost halved compared to in 1723.[71] In the crown tenant case, the crown was the land-owner, and the peasant paid rent to the crown

[71] Weibull (1923:64-65).

instead of tax. Noble tenants, on the other hand, held land for which they paid rent to the estate owner. This rent was usually in labour (day labour, *dagsverken*) combined with payments in kind, but later also in money.[72] Tenants on noble land dominated in Halmstad and Sireköpinge while tenants on crown land and freeholders dominated in Hög and Kävlinge.[73]

Figure 3.1 shows that the five investigated parishes Hög, Halmstad, Kågeröd, Kävlinge, and Sireköpinge are located between Lund and Helsingborg in western Scania, and Scania is the most southern part of Sweden. The areas in the parishes are open farmland except for the more wooded northern Halmstad.[74]

3.3.2 Food prices

The prices of commodities most important for survival are of vital interest, such as the price of food, as is how much people in general can afford, so wages will also be of importance. Since a large part of the income in the 18th and 19th centuries was spent on food and especially on grain, it is possible to use the price of grain as a proxy for the cost of living.[75] Here, price of rye is used since the price variation of rye was more or less the same as for

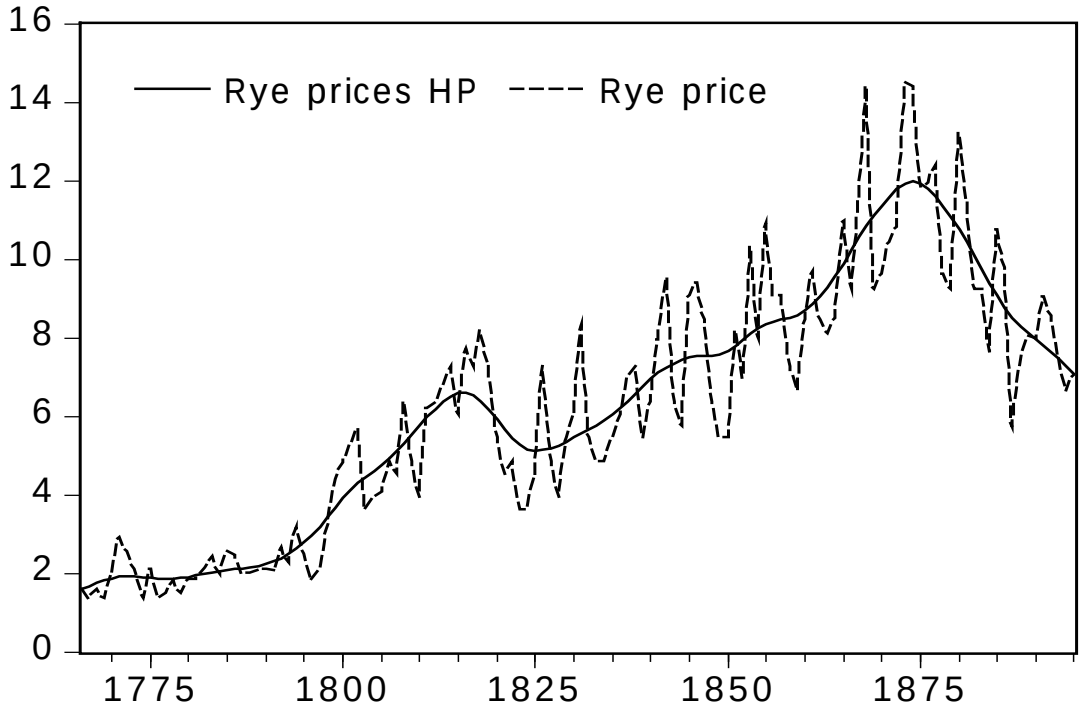


Figure 3.2: Local rye price and HP-trend of local rye price in crowns per hectolitre between 1766 and 1894. *Source*: Bengtsson and Dribe (1997:90-91); Appendix 2, Table 1.[76]

[72] Heckscher (1941:213-216).
[73] Bengtsson & Dribe (1997:2).
[74] Bengtsson & Dribe (1997).
[75] Livi-Bacci (1991:87).
[76] The series is not complete for all years but figures from the lowest level have always been used, which almost entirely means from Harjäger Härad.

other common grains.[77] Figure 3.2 shows the local price of rye together with a smoothed series calculated as the Hodrick-Prescott-trend of rye price. International demand for grain made rye prices rise more or less continuously during the period up to 1875, apart from a sharp fall from 1815 to 1825. After 1875, rye prices dropped dramatically due to increased competition in the world market for grain, especially from Russia and the United States.[78] Given the rye prices in Figure 3.2, the cost of living increased up until 1875, when it dropped sharply. Thus, net consumers would have been better off after this period while net producers had the opposite experience since they would have been better off up until the fourth quarter of the 19th century.[79]

3.3.3 Socio-economic structure

Socio-economic status is derived from the type of land holding the family had, the size of land holding the family held, the type of land the family lived on, and the occupation of the father, or from a combination of two of them.[80] Here, four SES groups are used and they are based on birth family of the child, since children have no socio-economic status of their own. Thus, the lower classes increased their share during this period due to the enclosure movement and the population increase. Many new land holdings had been created by a division of only one or a few land holdings. This process in turn created a group of semilandless peasants who were dependent on wage labour for their income.[81] This period in Swedish history is often referred to as the pauperisation period, when an increasing share of the population had land holdings that were too small to support them, or no land holding at all.[82]

The first landed group includes tenants on crown land (*kronobönder*) and freeholders (*skattebönder*), and the second landed group consists of tenants on noble land (*frälsebönder*). The minimum land holding size for the landed groups is set at 1/16 *mantal* of land since it is supposed to have been enough to support a family on, so the land holder was not forced to take on wage labour also to support his family. However, due to their different relations to the land when it comes to land holding they are divided into two groups. The distinction between this first landed group and the second landed group is that noble tenants instead of rent or tax to the crown paid rent to the estate owner where they had their tenancy. Noble tenants often had to do day labour (*dagsverken*) at the estate. Since they also had to cultivate their own land holding, and day labour at the estate could take up a large share of their time, they often had to hire workers (*drängar*) to do day labour at the estate.[83] Therefore tenants on noble land often had larger households compared to freeholders and crown tenants, because they also had to employ servants to carry out day labour.[84]

The third group has been called semilandless and is quite heterogeneous: it consisted of crofters (*torpare*), cottagers (*husmän, gatehusmän*), peasants with small land holding etc. The people in the smallholder group were forced to work for others since their land holding

[77] Jörberg (1972:323-4).
[78] Jörberg (1972:308).
[79] It is possible to use real wages for the parishes instead of using food prices as a kind of crude measure of standard of living. However, the real wages are not very representative, since wage labour was not commonplace until in the latter part of the 19th century.
[80] Follows Johansson (2004:Chapter 4).
[81] Fridlizius (1975b:128).
[82] Jörberg (1972:336-337).
[83] Dribe (2000:37-8).
[84] Bengtsson & Dribe (1997:21).

was too small to produce what was necessary to support their families.[85] The upper limit for semi-landless is chosen to be 1/16 *mantal* and the lower limit is holding any land at all. Even if they had a very small land holding, the fact that they held at least some land distinguishes them from the group of landless. This group consisted of servants (*tjänstefolk*), lodgers (*inhyses*), soldiers, artisans, and others.[86]

At the same time, the quite large noble tenant group decreased heavily over time as a consequence of the fact that some noble tenants were able to purchase their land holding from the estate owner. Besides, the distinction between members of the semilandless group and the landless group is not always clear, and some of the people in the semilandless group should probably be in the landless group. For example, some crofters only had a small garden to grow potatoes, vegetables, etc while other crofters had quite large land holdings. The semilandless group is therefore somewhat heterogeneous, but the main idea of this grouping is to make the landless group homogenous, so that only people without any land holding at all end up in this group.

3.3.4 Mortality

The most vital mortality patterns are presented in this section: infant mortality, since it will be used as a proxy for local disease load, and child mortality in ages 1-14. Figure 3.3 shows the local infant mortality rate for the five parishes as well as the Hodrick-Prescott trend of

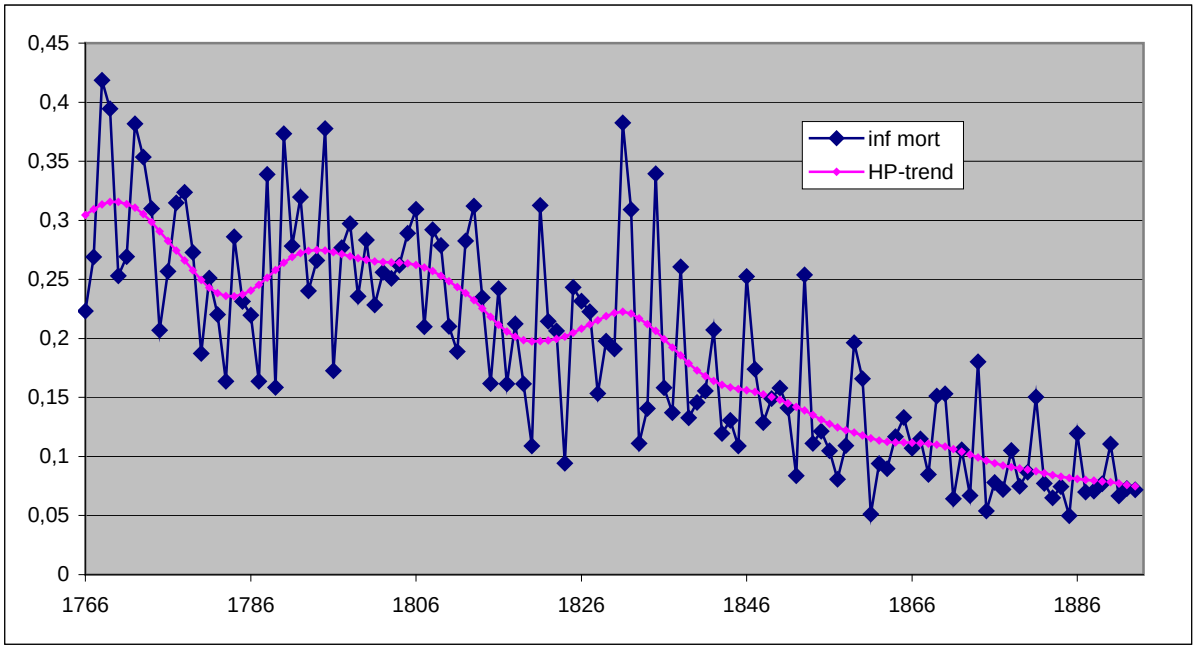


Figure 3.3: Infant mortality in percent for Hög, Kävlinge, Halmstad, Kågeröd and Sireköpinge 1766-1894, computed without stillborn, and Hodrick-Prescott-trend for infant mortality. *Source*: Scanian Demographic Database.

[85] Fridlizius (1975b:128).

[86] This differs from some of the other studies made with the Scanian data, where the landless group has been the heterogeneous one (thus; not the semilandless group, as here) and, due to this, the landless group is much larger than the grouping made here. Others studies have used the same definition as here.

local infant mortality rate, representing the short-term trend in local infant mortality rate. There were quite large short-term fluctuations (solid line), but the general picture is of a steady decline in infant mortality rate all through the period (dotted line), except for the end of the 18th century and also a temporary increase around 1825. Figure 3.4 shows mortality in ages 1-14 in the five parishes:

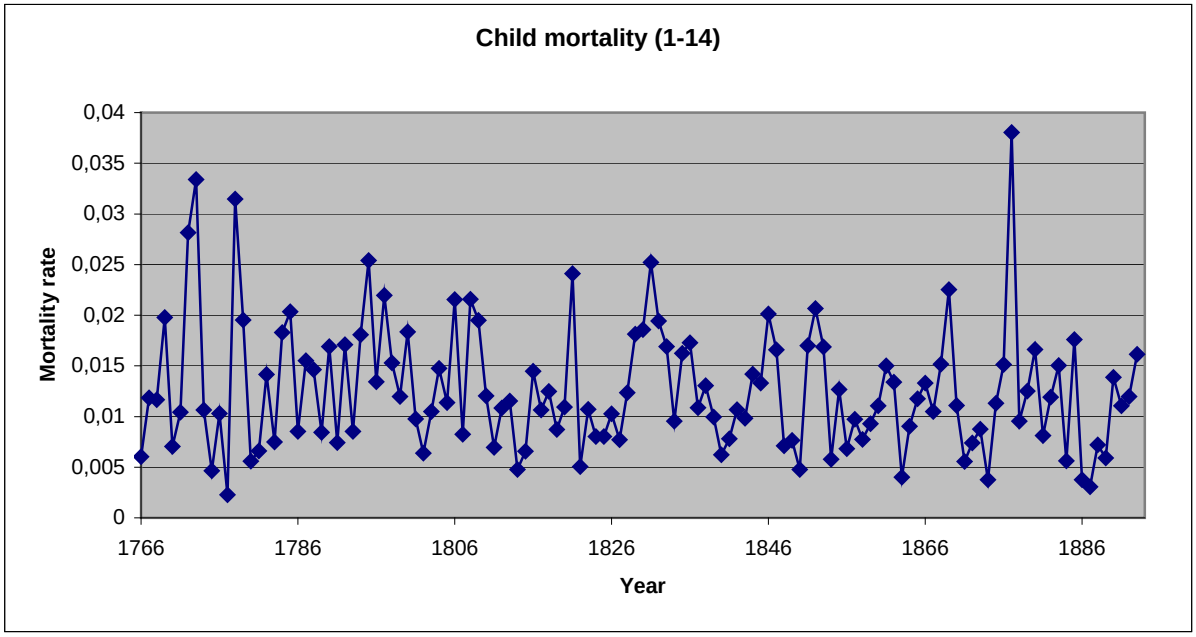


Figure 3.4: Mortality rate in ages 1-14 year old children in Hög, Sireköpinge, Halmstad, Kågeröd, and Kävlinge in Scania, Sweden 1766-1894 and Hodrick-Prescott- trend for child mortality; own calculations. *Source*: Scanian Demographic Database.

4 Empirical model

The empirical model is based on the theoretical model given earlier here in this paper, as well as the empirical child mortality model in Johansson (2004:Chapter 6), where all variables used are derived, and the discussion regarding the empirical model will concentrate on the most important variables here; the food price and the disease load variables.

4.1 Local food prices

The theoretical model uses food supply as the underlying determinant of nutrition but since food supply is unknown, local food prices will be used as a proxy for food supply. The operationalisation of local food prices is the price of rye. It would, of course, have been possible to construct a composite index for food prices but since a large part of the family income was spent on some kind of grain and that the price of rye varied in the same way as other grain like barley,[87] the local price of rye is used as a proxy for food supply. However, changes in food supply due to harvest fluctuations will have different effects on prices when

[87] Jörberg (1972:323-4), Livi-Bacci (1991:87).

endogenous prices rather than exogenous prices are assumed.[88] Here, prices are assumed to be exogenous, because of the market integration during this period.[89] It is possible that if the price of grain rose, people would start consuming foodstuff that could work as substitutes.
Table 4.1: The operationalisation of the theoretical child mortality model from the theorietical child mortality model in section 3.

Theoretical model	**Operationalisation**	**Variable in empirical model**
Community level		
Environment	Parish	Hög (reference group) Kävlinge Halmstad Kågeröd Sireköpinge
Nutrition	Food price	Local rye price in time *t*
	(same with 1 year lag)	Local rye price in time *t*-1
Disease load	Mortality rate	Local infant mortalty rate in time *t*
	(same with 1 year lag)	Local infant mortalty rate in time *t* -1
Nutrition & disase load interaction	Food price * mortality rate	Local rye price in time *t* * local infant mortality rate in time *t*
	(same with 1 year lag)	Local rye price in time *t*-1 * local infant mortality rate in time *t*-1
Family level		
Family wealth	Socio-economic status	Landless (reference group) Smallholder with LT 1/16 *mantal* Freeholder/crown tenant GE 1/16 *mantal* Noble tenant with GE 1/16 *mantal*
Family knowledge	Shared components	Family-based frailty
Family values/preferences	Shared components	Family-based frailty
Genetics	Shared components	Family-based frailty
Individual level		
	Birth cohort	Year of birth
Maternal factors	Low maternal age	Maternal age at childbirth under 22
	Normal maternal age	Maternal age at childbirth 22 to 34 (ref grp)
	High maternal age	Maternal age at childbirth 35 and over
Sex		Male (reference group) Female

Note: Abbreviations in table: GE = Greater or Equal to. LT = Less Than.

This could, for example, be to consume potatoes. But potatoes were not widely consumed in the 18th century and potato production in the first decades of the 19th century mostly went

[88] Bengtsson & Dribe (1997:62-65), Dribe (2000:166-175). With endogenous prices, local changes in demand and supply (thus, inside the local economic system) will affect prices. With exogenous prices, prices are assumed to be given outside the local economic system and will not be affected by changes in demand and supply inside the system.
[89] Jörberg (1972:193-205).

into alcohol production.[90] Thus, there is the possibility that people substituted grain for potatoes in the later part of the transformation period and the industrialisation period, but the price of rye is argued to be useful as a general indicator of food prices during this period.

Several investigations of Scanian mortality study the effect of current nutrition on mortality, but only one investigation has been able to establish a link between current rye prices and mortality. In an investigation of adult mortality in the period 1766-1895 for the age groups 25 to 55, high current rye prices increase mortality.[91] In a similar study, only one interaction term with occupation and rye price was significant.[92] In another study, no significant effect was found.[93] There have also been studies relating food prices in early life to mortality in adult life but none of these has been able to establish a link between early life food price and later life mortality.[94] Further, all these studies have been on adult mortality. Children are more sensitive to malnutrition and have been found to be affected by price changes.[95]

The operationalisation of food supply to food prices concerns the effect of food prices[96] on childhood mortality. This is operationalised into a time-varying variable measuring rye prices during age 1 up to age 14 to represent effects on mortality induced by current food prices. Hence, the purpose of including current rye prices in the model is to test if short-term fluctuations in food prices affect child mortality. Higher food prices than normal means the family can afford to buy less food for net consumers, and less food means reduced food supply and thereby less gross nutrition. Assuming unchanged levels of body nutrition claims, less gross nutrition will cause growth retardation and reduced immune response, which induces a higher risk of morbidity. Besides, in case of sickness, the body needs more nutrition since it still needs the usual level of nutrition plus additional nutrition for the extra energy needed to conquer the disease.[97] Thus, the effect of a reduced gross intake with unchanged claims is twofold: first a higher risk of reduced growth and increased morbidity and then – in case the higher risk of morbidity results in sickness – even higher nutrition claims. Hence, higher food prices are expected to increase mortality during childhood for net-consumers, and the current rye price coefficient is therefore expected to be positive. The price of food might be argued to only be considered for the various socio-economic status groups, with the expectations for the different socio-economic groups that net-producers should not be affected or have had decreased mortality, while net-consumers should have experienced increased mortality. However, socio-ecomic status will also be controlled for in the model and it is also often hard to show any interaction effects in a rather small sample with several groups interacting a continous variable.

Nutrition-deficiency-induced weaker health may, however, not be sufficient to cause the death of a child directly, since the result may only be morbidity and not mortality. Low nutrition intake could cause death indirectly via a reduced immune response as a result of morbidity. Of course, there is the possibility that this process will be slow, and that the

[90] Fridlizius (1988).
[91] Bengtsson (1997:9-10, 16-20).
[92] Bengtsson & Lindström (2000:270-276).
[93] Bengtsson & Lindström (2001:24-27).
[94] Bengtsson (1997:15-21), Bengtsson & Lindström (2000:270-276), Bengtsson & Lindström (2001:24-27), Bengtsson, Broström & Lindström (2002:20).
[95] Johansson (2004:176-182).
[96] This could be called ‘current’ food prices, since it refers to a time-varying price of food – thus, it changes yearly over the life of an individual.
[97] Pollard (1982:454).

effects of a longer period of starvation and sickness will not result in immediate death but rather death one or several years later. Thus, there is reason to believe that there can be a lag

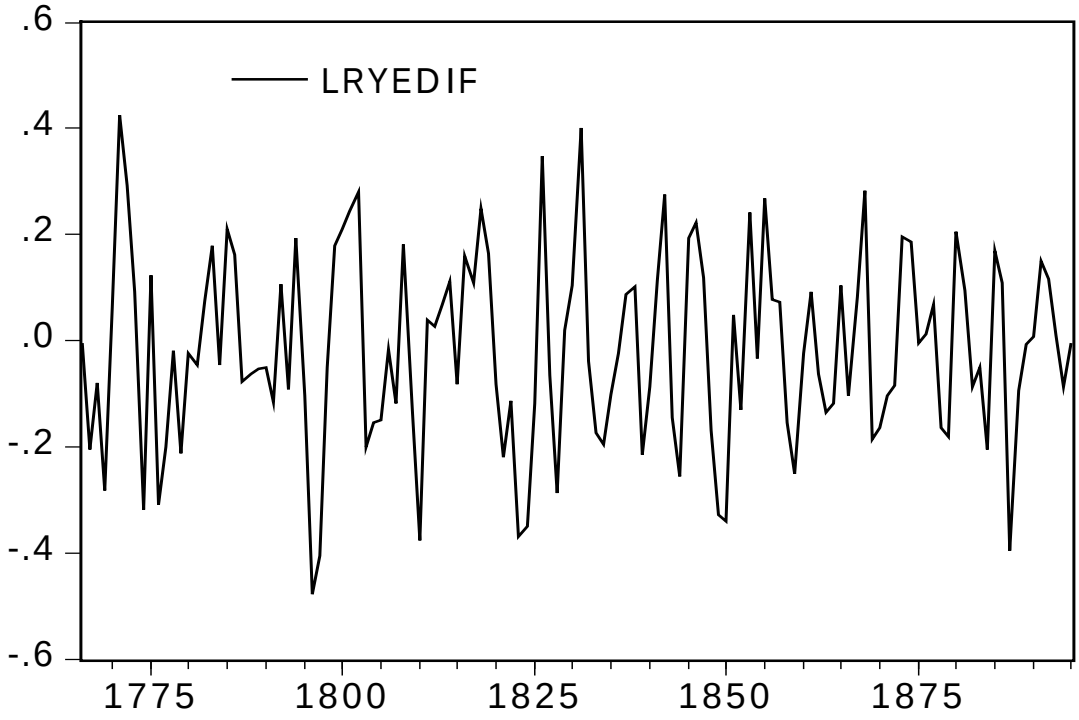


Figure 4.1: Log of local rye prices minus log of HP-trend of local rye prices for the five parishes 1766 -1894. *Source*: Bengtsson and Dribe (1997), Appendix 2.

in the effect from malnutrition to mortality, so the food price variable is also lagged by one year in the empirical model. For example, during the second part of the 19th century, a common killer was tuberculosis, which was a slow-working, nutrition-dependent disease that might need more time to affect mortality. Previous research on child mortality in Scania has shown such effects.[98]

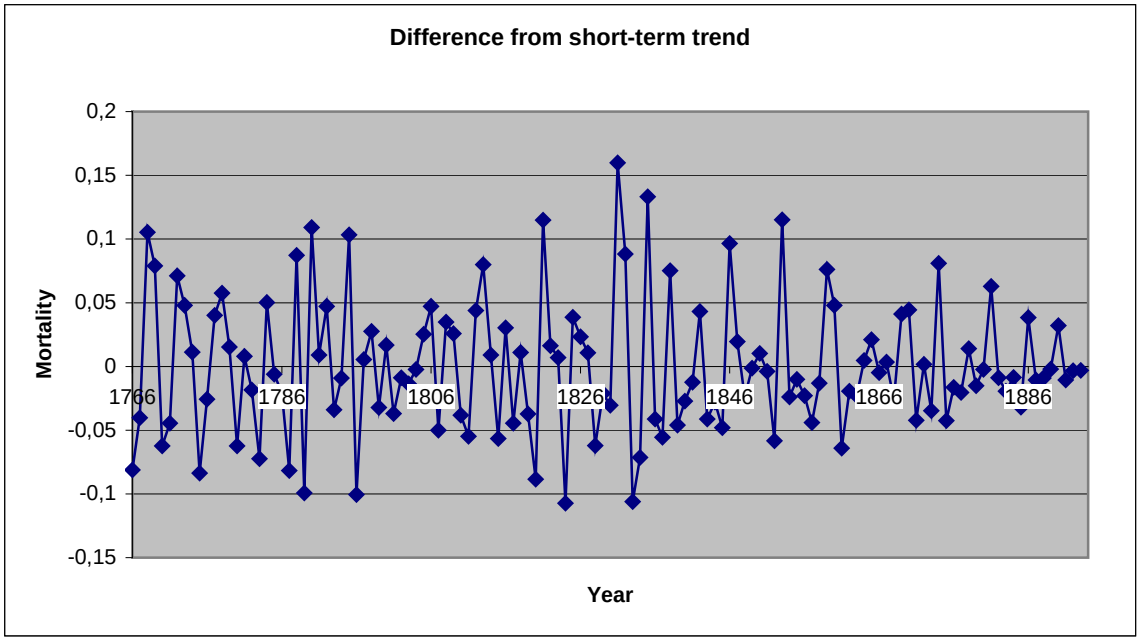
The local rye price was shown in Figure 3.2. To capture short-term price variations in food prices, rye prices will be measured as a deviation from a short-term trend. The rye price series used is therefore calculated as yearly fluctuations from the Hodrick-Prescott-trend. The variance is, however, increasing quite heavily over time in this series. To smooth out this uneven variance, the natural logarithm of the rye prices series, minus the natural logarithm of the Hodrick-Prescott-trend of the rye price series, is used instead of the original series. Figure 4.1 below shows this logarithm deviance series and, as seen in the figure, the variance here is decently homoskedastic over time.

4.2 Local mortality rates

Local area mortality rates have been used as a proxy for disease in research on historical adult mortality as well as child mortality.[99] However, in these investigations, local mortality

[98] Johansson (2004:182).
[99] Bengtsson (1997), Bengtsson & Lindström (2000, 2001), Bengtsson, Broström & Lindström (2002), Johansson (2004).

is used as a fixed measure for disease load in early life. In this study, disease load is operationalised into a period measure. This variable is the local mortality rate over time, and is thus a time-varying variable, used to catch changes in local disease load experienced by the children. To avoid any problems with trends since of the general decline in mortality and to make the different time periods comparable, also local infant mortality is measured as the deviation form the short-term trend. The yearly value is thus the actual value of local infant mortality minus the Hodrick-Prescott-trend; shown below. There is a variant without any lag and one that has been lagged one year to capture any delayed effects of disease load.



4.3 Local food price and mortality interaction

The variables for local food prices and for local infant mortality rates are also used to test the prime hypothesis of this paper: when interacted with each other they show the combined effects of malnutrition and disease load. The expectation is the same as for the stand-alone variable expectations: the interaction effect of the deviation from the short-term trend in local rye price and the deviation from the short-term trend in local infant mortality is expected to have a positive impact on the risk of mortality for children. This means that higher positive values than what were normal increases the risk of mortality for the children with this experience. Also, as in the case with the stand-alone variables, this interaction is hypohthesised to possibly have a delayed effect, and the lag of these variables is therefore also interacted.

5 Results

Here, the results from the Cox regressions are reported, and even though several variants of the models were run, but not all results are shown. Significance level is set to 10 % due to the sample and the interactions.

In the pre-transformation period, the results show that there is a significant general cohort effect in form of a decrease in the general risk of mortality over time, shown by the ½ % decrease per birthyear. There are some non-significant differences in mortality risk between the parishes. This variable is mostly used as control for environmental conditions we don't know that much about, except in the late period, when Kävlinge develops into a town and mortality is expected to increase. Food prices has a significant and positive influence on the mortality risk, and this was in line with expectations – a higher than normal price of food should increase the risk of mortality for the children. The local infant mortality rate also has a significant increase on child mortality and the effect is positive (= increases the risk of mortality) and confirms the hypothesis of a high disease load increasing mortality for the children.

The estimates also show some SES differences but they are non-significant. The conclusion is as shown in several investigation earlier: socio-economic status did not matter very much for mortality in 18th century Sweden since wealth could not buy you health – knowledge and technology could not be bought, and smallpox was so virulent and not nutrition-dependent that it killed without a socio-economic differentiation.[100] Boys have a significant 7 % higher mortality risk than the girls, which is consistent with previous research.[101] There is also a significant family effect shown by the shared frailty term. In the interaction version of this model, the effect of the interaction between food price and infant mortality rate is postivite, significant and rather large. Thus, the estimate of the hypothesised interaction effect is very much in line of what was expected. The variants with lagged food price and lagged mortality rates are not shown since they shown small non-significant effects.

Cox regression for child mortality age 1-14 in the pre-transformation period 1766-1815:

Covariate	Mean	Coef	Rel.Risk	S.E.	Wald p
bthdate	1783.399	-0.005	0.995	0.003	0.059
parish					
Hög	0.091	0	1 (reference)		
Kävlinge	0.088	0.176	1.193	0.189	0.351
Halmstad	0.173	-0.073	0.929	0.221	0.740
Sireköpinge	0.189	-0.020	0.980	0.263	0.940
Kågeröd	0.459	-0.414	0.661	0.371	0.264
ryeprice	0.447	0.643	1.902	0.201	0.001
inf.mortality	10.381	0.048	1.049	0.008	0.000
socio-ec.status					
Landless	0.258	0	1 (reference)		
Smallhold	0.295	0.052	1.054	0.113	0.644
Free+Crown	0.353	-0.150	0.860	0.116	0.195
Noble	0.094	0.198	1.219	0.173	0.252
male	0.486	0.162	1.176	0.081	0.046
frailty(fam)	844.810	0.000	1.000	0.000	0.063

Events	612
Total time at risk	44418
Max. log. likelihood	-4939.6
LR test statistic	71.4
Degrees of freedom	12
Overall p-value	1.73751e-10

[100] Johansson (2004:Chapter 8).
[101] Johansson (2004:Chapter 8).

Cox regression with interactions for child mortality age 1-14 in the pre-transformation period 1766-1815:

Covariate	Mean	Coef	Rel.Risk	S.E.	Wald p
Bthdate	1783.399	-0.006	0.994	0.003	0.051
parish					
Hög	0.091	0	1 (reference)		
Kävlinge	0.088	0.180	1.197	0.189	0.341
Halmstad	0.173	-0.076	0.927	0.221	0.731
Sireköpinge	0.189	-0.021	0.979	0.262	0.935
Kågeröd	0.459	-0.416	0.660	0.371	0.262
ryeprices	0.447	-0.780	0.458	0.478	0.103
inf.mortality	10.381	-0.005	0.995	0.018	0.787
as.factor(ses)					
Landl	0.258	0	1 (reference)		
Small	0.295	0.057	1.059	0.113	0.613
Fr+Cr	0.353	-0.147	0.864	0.116	0.206
Noble	0.094	0.197	1.218	0.173	0.254
male	0.486	0.161	1.175	0.081	0.047
frailty(fam)	844.810	0.000	1.000	0.000	0.065
rye*inf.mort		0.119	1.126	0.036	0.001

Events	612
Total time at risk	44418
Max. log. likelihood	-4934.5
LR test statistic	81.8
Degrees of freedom	13
Overall p-value	5.05629e-12

For the transformation period, the estimated regression coefficients show that the birth year (cohort) effect is about the same as in the first sub-period, and that children living in Kävlinge already in this period has a 31 % significant risk premium over Hög already in this period. The effect of current rye price is again positive and significant, and thus in line with expectations. The effect of mortality rate is as in the pre-transformation period positive and significant. The effect of socio-economic status is that the noble tentants have a significant 25 % lower mortality than the landless, which is what could be expected. In this period, the

Cox regression for child mortality age 1-14 in the transformation period 1815-1865:

Covariate	Mean	Coef	Rel.Risk	S.E.	Wald p
birthdate	1833.798	-0.004	0.996	0.003	0.118
parish					
Hög	0.110	0	1 (reference)		
Kävlinge	0.137	0.271	1.312	0.151	0.071
Halmstad	0.180	0.068	1.071	0.201	0.734
Sireköpinge	0.203	-0.017	0.983	0.296	0.954
Kågeröd	0.371	-0.168	0.845	0.441	0.702
ryeprice	0.349	0.464	1.591	0.182	0.011
inf.mortality	10.519	0.023	1.023	0.006	0.000
SES					
Landl	0.388	0	1 (reference)		
Small	0.333	-0.101	0.904	0.084	0.227
Fr+Cr	0.173	0.056	1.058	0.105	0.591
Noble	0.106	-0.280	0.756	0.133	0.036
male	0.495	-0.147	0.863	0.070	0.036
frailty(fam)	1384.749	-0.000	1.000	0.000	0.864

Events	819
Total time at risk	68637
Max. log. likelihood	-6994.7
LR test statistic	55.2
Degrees of freedom	12
Overall p-value	1.68554e-07

girls have about the same level of risk premium over the boys as the boys had over the girls in the pre-transformation period; it is over 13 % and highly significant, and in line with the results in Johansson (2004). The family effect is however non-significant in this period. For the interaction version, the effect of food price and mortality is also in this sub-period in line with expectations: it is positive and higly significant. Thus, also the estimates from this sub-period model show a strong interaction effect of nutrition and disease that increases the risk of mortality for children. The variants with lagged food price and lagged mortality rates are not shown since they shown small non-significant effects.

Covariate	Mean	Coef	Rel.Risk	S.E.	Wald p
birthdate	1833.798	-0.003	0.997	0.003	0.247
parish					
Hög	0.110	0	1 (reference)		
Kävlinge	0.137	0.270	1.309	0.151	0.073
Halmstad	0.180	0.065	1.067	0.201	0.748
Sireköpinge	0.203	-0.011	0.989	0.296	0.969
Kågeröd	0.371	-0.172	0.842	0.441	0.696
ryeprice	0.349	-0.871	0.418	0.368	0.018
inf.mortality	10.519	-0.020	0.980	0.012	0.093
SES					
Landl	0.388	0	1 (reference)		
Small	0.333	-0.103	0.902	0.084	0.219
Fr+Cr	0.173	0.055	1.057	0.105	0.598
Noble	0.106	-0.282	0.755	0.133	0.035
male	0.495	-0.148	0.862	0.070	0.035
frailty(fam)	1384.749	-0.000	1.000	0.000	0.871
rye*inf.mort		0.109	1.116	0.026	0.000

Events	819
Total time at risk	68637
Max. log. likelihood	-6986.5
LR test statistic	71.5
Degrees of freedom	13
Overall p-value	4.16531e-10

For the industrialisation period, the estimates show that the general cohort factor captured by the year of birth is as expected negative and significant, with a yearly decrease of relative risk of mortality with 1.2 %. It also shows the negative effect on health of a town in the relative risk estimates for parish – Kävlinge has a significant mortality premium of 80 % over Hög in the industrialisation period. This was in line with the expectations, but also Sireköpinge and Kågeröd have significantly higher mortality than Hög. As in the other sub-periods and as expected, the effect of food prices is positive and here it also very strong; note however that this is for the delayed effect, thus for lag t-1. The non-lagged model estimates didn't show any effects at all. Also, no interaction model is given since the interaction effect is far from significant.

The effect of current local mortality rate is here significant but with unexpected sign, meaning that a high local disease load in lag 1 would lower the risk of mortality. In this period, the effect of socio-economic conditions is expected to have the largest effect, and the noble tenants have almost 50 % lower mortality risk compared to the landless group. As expected and in line with previous reseach on child mortality in Scania (Johansson (2004)), there are not sex-differentials in mortality in this period. Last, there is a significant effect of family belonging in this period. This could confirm hyptheses regarding increasing information in society on diseases, hygiene, germs, and the increase in differences in the

ability to use this knowledge due to different abilities in reading and writing, as well as access to such knowledge.

Cox regression for child mortality age 1-14 in the pre-transformation period 1865-1894:

Covariate	Mean	Coef	Rel.Risk	S.E.	Wald p
Bthdate	1871.200	-0.012	0.988	0.005	0.019
parish					
Hög	0.088	0	1 (reference)		
Kävlinge	0.114	0.595	1.813	0.198	0.003
Halmstad	0.202	0.170	1.185	0.267	0.525
Sireköpinge	0.283	0.687	1.988	0.394	0.081
Kågeröd	0.313	0.957	2.604	0.600	0.111
Ryeprice(t-1)	0.380	1.201	3.323	0.284	0.000
inf.mortality(t-1)	4.622	-0.032	0.968	0.013	0.016
SES					
Landl	0.603	0	1 (reference)		
Small	0.199	-0.036	0.965	0.110	0.745
Fr+Cr	0.083	-0.052	0.949	0.170	0.758
Noble	0.116	-0.649	0.523	0.169	0.000
male	0.485	0.016	1.016	0.084	0.850
frailty(fam)	1318.806	-0.000	1.000	0.000	0.074

Events	564
Total time at risk	47497
Max. log. likelihood	-4582
LR test statistic	61
Degrees of freedom	12
Overall p-value	1.47117e-08

since of the commonness of slow-working diseases as tuberculosis in the period, the process from malnutrition and disease to mortality might take longer time, and that this delayed effect is captured in the lag model, but only for food prices, and the interaction is non-significant.

6 Conclusion

The estimates for the pre-transformation period, most results were in line with what was expected, and the main interest variables here – the nutrition and disease variables – both had the expected sign and were significant. Also the interaction between nutrition and disease was positive and was significant. Hence, nutrition intake during childhood had a significant impact on the mortality risk of children in 1766-1815, and the same goes for the disease load, and the interaction between nutrition intake and disease load. The variants with lagged food price and lagged mortality rates didn't show any significant effects.

The conclusion for the transformation period is about the same as for the pre-transformation period: most results were in line with what was expected, and the nutrition and disease variables had the expected signs and were signficant. The interaction between nutrition and disease was also significant and positive. Thus, nutrition intake and disease load during childhood had a significant impact on the mortality risk of children also in 1815-1865, as well as the interaction between nutrition intake and disease load. The variants with lagged food price and lagged mortality rates are were all non-significant. In the industrialisation period no effects were significant in lag 0, but in lag 1, food prices are significant and positive; however, the mortality rate has the unexpected sign and the interaction is insignificant. Thus, this period is different from the other two that showed very similar results to each other.

What conclusions can we draw from the theoretical considerations and the empirical results in this paper?

The theoretical part of this paper shows that there is theoretical evidence for that malnutrition and disease are important variables to explain child mortality and should thus be included in theoretical child mortality models. The same goes for the interaction of malnutrition and disease. It is also reasonable to assume that there could be a delayed effect, suggesting that assuming lags in the theoretical model would be appropriate.

The empirical part of this paper confirms most of these theoretical suggestions, at least for children and for the Scanian population sample in the 18th and 19th centuries:

- current malnutrition significantly increases the risk of mortality, which might not be a surprise since it has been shown before,

- current disease load significantly increases the risk of mortality, which might not have been show before with the use of local infant mortality rates, and

- the interaction between current malnutrition and current disease load significantly increases mortality risk, which is probably not shown before either with the use of local food prices and local infant mortality rates.

The results are very consistent for the pre-transition period and the transformation period, while the industrialisation period only shows some results in lag 1. In the other two periods, there seem to be no delayed effect. Thus, there is empirical evidence supporting the theoretical arguments in the first part of this paper, with some reservation for the delayed effects. Implementation of the use of proxies for malnutrition and disease load in other studies of child mortality where nutrition intake and disease exposure is unknown should be fairly straightforward. Local disease load measured as local infant mortality rate is fairly easy to calculate. Estimates of local food prices or of real wages might be harder but not impossible to find or to approximate.

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