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New and improved ways of measuring conditions during foetal stage and infancy: Early life conditions and mortality later in life in Scania, Sweden 1794-1894

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1 Introduction and background

It is often claimed that human longevity – at least partly – is determined already during intra-uterine life or during infancy.[1] The idea of early life conditions affecting later life health is not new, and early studies based on aggregated historical populations data have shown such a relationship during the general mortality decline.[2] However, data on contemporary populations for testing the early life hypotheses is hard to find, but recent studies of historical populations have established such links with family reconstituted data,[3] where the relationship is formally tested with Cox-regressions based on life-histories of real individuals. This is a huge improvement compared to the tabulations methods used in the early studies. Generally, the use of individual data has several advantages over using aggregated data. The nature of micro data makes it possible to test hypotheses using multivariate statistical tools that allow control for individual characteristics as for example sex, birth-place, family-specific characteristics, and socio-economic group simultaneously in an empirical model. However, even if the life-histories of individuals are known, the conditions in early life *per se* are not known. Nutrition intake and disease histories are usually unknown even in modern data compilations. Hence, in most cases early life conditions has to be measured by some proxy variable or variables, approximating nutrition and/or disease load. This paper discusses such measures, and their advantages/disadvantages, and how to improve them.

A major issue dealt with here is that many studies have been able to establish a link between disease in infancy and later life mortality – what could be called the Fridlizius link from early life conditions to health in later life,[4] but have failed to establish the Barker link from nutrition in the foetal stage to health in later life.[5] In this paper, some new and improved measures for early life conditions are suggested and discussed relative to what has been commonly have been used. The suggested new measures are both supposed to be theoretical and practical improvements compared to previously used measures. Further, one measure approximates foetal stage nutrition and disease effects on later life mortality; thus, the 'true' Barker hypothesis, while another a Fridlizius type of hypothesis regarding early life conditions during infancy.[6]

[1] Barker (1998:5-41, 2001:69-88).
[2] Kermack, McKendrick & McKinlay (1934:702-703), Preston & van de Walle (1978:290-291), Fridlizius (1989:16-17).
[3] Bengtsson (1997:15-19), Bengtsson & Lindström (2000:273-275, 2001:10), Alter & Oris (2000) Bengtsson, Broström & Lindström (2002:1-4, 20-24), Johansson (2004:207-212), Johansson, Beise & Desjardins (2006).
[4] Fridlizius (1989), Barker (1998:Chapter 9).
[5] Barker (1998).
[6] Measuring of early life condition effects was one of the topics that should be more thoroughly investigated in research on early life conditions and later life mortality, as recognized by the commentator Gabriele Doblhammer at the "Early Life Experiences and Mortality" session at PAA 2006.

2 Early life conditions

What do we mean with early life condition effects? It simply means that unfavourable conditions in early life lead to worsened health in later life. There are two prime risk factors during early life: malnutrition and disease.[7] A number of diseases such as rheumatic heart disease, respiratory tuberculosis, bronchitis, hepatitis B and liver cancer have a well-established link to circumstances in early life.[8] However, scholars differ with respect to explanations of the timing and mechanisms conditions through which in early life affect morbidity and mortality in later life.

2.1 Nutrition

A proper development of the foetus is dependent on its mother delivering correct amounts of nutrition and oxygen during the foetal stage. This delivery depends on the size and body composition of the mother, the nutrient stores she has, her food intake during the pregnancy, her ability to transport nutrients to the placenta and transfer across it. If demand of nutrients exceeds supply in the foetal supply line, her foetus will be undernourished. This can be a result of either a low supply from a thin or starving mother or a high demand due to a high foetal growth rate.[9] The foetus' main adaptation to lack of nutrients and oxygen is a lowering of the cell division rate, particularly in tissues undergoing critical periods at that specific time. The cell division due to undernutrition is slowed down either as a direct effect or an indirect effect due to changes in concentrations of growth factors and hormones, of which growth hormone and insulin are especially important. Because different tissues have different critical periods, disproportionate growth may occur.[10]

Animal studies have shown that the formation of many organs and tissues is to a large part completed at or shortly after birth, with regard to cell numbers. Later growth stems from enlargements of these cells rather than the production of new cells.[11] The first 8 weeks after conception consists of an increase in cell number (hyperplasia), while during the third to ninth month cells both increase in number and in size (hypertrophy). Gradually, hypertrophy becomes predominant.[12] Maternal undernutrition at conception in animal experiments leads to fewer cells in the inner cell mass, and this is associated with lower birth weight and lower postnatal growth, as well as changes in the body composition and hypertension development.[13] In late gestation, the foetus responds to undernutrition by redistributing blood flow and changing the production of growth hormones. The immediate adaptation to undernutrition is catabolism, which means that the foetus consumes its own substrates for energy. If undernutrition is prolonged, the growth rate is slowed down to ensure the development of the most important organs, for example the brain, with the result that the liver and kidney may remain underdeveloped. Since these organs develop fast in late gestation, they may be irreparably damaged because cell numbers may be reduced, and after birth there is no possibility for the body to produce cells to catch up.[14] The adaptation to a limited supply of nutrients permanently changes the physiology and metabolism of the human foetus. This programming of the foetus may be the source of several diseases in later life, for example coronary heart disease and the related disorders stroke, diabetes, and hypertension.[15] Thus,

[7] Wadsworth (1999:45).
[8] Elo & Preston (1992:205-206).
[9] Barker (2001:69).
[10] Barker (1995:171).
[11] Hales (1997:115).
[12] Perry (1997:149).
[13] Barker (2001:69).
[14] Barker (2001:70-71).
[15] Barker (1997:96).

there is a relationship between nutrition in the foetal stage and during infancy, and later life morbidity and mortality.[16]

There are also postnatal influences on body growth: both height and body shape are influenced by nutrition in childhood.[17] Several early life conditions – especially nutrition deficiencies – resulting in morbidity and mortality in later life: chronic energy deficiency, vitamin and mineral deficiencies, and the interaction of low nutritional status and infections.[18] But nutrition deficiency also affects the immune response. Nutrition deficiency during the immune system development – late gestation or early post-partum – may compromise the whole mechanism. Small-for-gestational age children with birth weight below 2500 grams have low levels of immunoglobulin with much reduced antigen-induced response. These deficiencies can be persistent and last for years or even become permanent. The increased susceptibility to infection in these children results in much-increased mortality.[19] The immune system development is a well-defined, almost stereotyped, event, and the differentiation steps are mainly genetically regulated. However, environmental influences, such as nutrition deficiency and the occurence of infection, can affect this process.[20] Foetal malnutrition, resulting in small for gestational age children at birth, has a very significant and long lasting effect on cell-mediated immunity.[21] Both upper and lower respiratory infections are three times as likely in small for gestational age infants as in full-term healthy infants up to age three. They also continue to show impaired cell-mediated immune response for several months or years, which correlates with infectious illness.[22] Nutrition deficiency in early life has a prolonged and sometimes permanent effect on the susceptibility to illness.[23] Perry argues that the effect on foetal growth from severe and moderate malnutrition is not seriously disputed, but that the effect of less severe malnutrition is contentious. Controversies rather concern the timing of critical periods during body development and interactions with nutritional status, metabolism, and maternal physiology.[24]

Since it is impossible to measure nutrition intake in the foetal stage, how can we know if an individual have experienced low nutrition in early life? There are several indicators that have been used as signs of unfavourable nutritional conditions during early life, for example adult height. Given too low levels of net nutrition,[25] the body will not develop properly and final height will be less than it would have been with sufficient levels of net nutrition.[26] Fogel reports that mean final height for men in some selected developed countries such as Great Britain, Norway, Denmark and Sweden has risen more than ten centimetres between the late 18th century and late 20th century. More precise data are presented from the Netherlands, where the increase in the same period is between ten and twenty centimetres and Fogel argues that this illustrates a general pattern in Western Europe.[27]

[16] Barker (1998:7-9, 13-20, 46-50, 145).
[17] Perry (1997:159).
[18] Scrimshaw (1985:332-36).
[19] Chandra (1975:450).
[20] Chandra (2000:275).
[21] Chandra (1989:609, 1992:755-756).
[22] Chandra (2002:S73-S74).
[23] Chandra (2000:273).
[24] Perry (1997:158-159).
[25] Fogel (1994a:230-238) states that it is vital to make the distinction between gross nutrition and net nutrition intake. The required nutrition intake depends on the level of activity, so what matters is not gross nutrition intake; it is the net nutrition intake that is of importance.
[26] The use of anthropometric measures such as stature has been common in economic history and development economics, for example in Sandberg & Steckel (1980), Fogel, Engerman, Floud, Friedman, Margo, Sokoloff, Steckel, Trussell, Villaflor & Wachter (1983), Nicholas & Steckel (1991), and Steckel (1995).
[27] Fogel (1994a:249-250).

It has also been argued that birth season indicates conditions in early life: with a nutritional approach to early life condition effects, children born in seasons with nutrition constraints should show higher mortality in later life. Gavrilov and Gavrilova explain a seasonal pattern in longevity by nutrition deficiency in the form of lack of vitamins in late winter and early spring, and that this scarcity coincides with one of two critical child development periods.[28] Another study concludes that month of birth, paternal age, and birth order (firstborn or not) significantly affect adult life span - however, males are not affected.[29]

In recent years, the possibility of high blood pressure having its origin in the uterus or early childhood has received much attention.[30] High blood pressure is a major risk factor for diseases of middle and later life such as stroke and coronary heart disease. A Hong Kong study conclude that both foetal and early postnatal growth affect blood pressure in adults.[31] Animal experiments also show that low birth weights induce hypertension: intrauterine growth-restricted rats had marked elevations in blood pressure.[32]

Another way to check the nutritional hypothesis empirically is by the use of data collected during the World War II Dutch famine and a study of this conclude that many risk factors for chronic heart diseases, such as impaired glucose tolerance, raised blood pressure, obesity, and hypercholesterolaemia, have their origin in the uterus, but that they are programmed at different times. Also, maternal malnutrition may permanently affect adult health for the baby without affecting the baby birth size.[33]

Chronic airflow obstruction is also claimed to have its origin in early life. Animal studies show that undernutrition during critical periods can lead to permanent changes in lung structure, and that the timing of undernutrition in the uterus determines the pattern of growth retardation. The pattern of pulmonary growth retardation in humans is likely to be important for subsequent chronic airflow obstruction, since airway branching is completed in the first trimester and after this only increase in size. This study shows that men with impaired lung function at ages 60-70 were lighter at birth and at age one, and had lower respiratory tract infections during the first two years after birth.[34]

A number of studies of Swedish mortality have formulated hypotheses on low nutrition in early life leading to higher mortality in later life for Scanian adults. However, either the studies show opposite than expected effects,[35] or have expected but insignificant effects.[36] Another study have expected but insignificant effect for foetal stage, and the opposite effect for infancy but also insignificant.[37]

2.2 Disease

Disease in early life will also affect health in later life. Hall and Peckham state that infections during the foetal stage, the prenatal stage, and the postnatal stage can affect the anatomical structure and the development of the immune system. These effects can lead to disease or increase the risk of disease susceptibility in later life. The effect of infection during the foetal stage is dependent on a number of foetal and maternal factors such as nutrition, genetic makeup, foetal development stage, and anatomical factors. Examples of diseases with these effects are influenza and rubella. They conclude that childhood infections are usually

[28] Gavrilov & Gavrilova (1999:365-366).
[29] Gavrilova, Gavrilov, Evdokushkina, & Semyonova (2001:2, 10-13).
[30] Whincup & Cook (1997:121).
[31] Cheung, Low, Osmond, Barker & Karlberg (2000:795-796, 799-800).
[32] Alexander (2003:457).
[33] Roseboom, Meulen, Ravelli, Osmond, Barker & Bleker (2001:94-95, 97).
[34] Shaheen (1997:61-65).
[35] Bengtsson (1997:15-18).
[36] Bengtsson & Lindström (2000:270-276), Bengtsson & Lindström (2001:24-27).
[37] Bengtsson, Broström & Lindström (2002:14-15, 20).

associated with season of birth and birth order, family size and housing characteristics.[38] In the prenatal and postnatal development of the lung and immune system, events in critical periods may influence susceptibility to later infectious, allergenic, or toxic challenges to the airways. However, the critical periods are poorly defined and the relationships between early infectious disease and later lung diseases are complex.[39] Studies have also shown that insults from infectious disease in very early life have a large effect on chronic disease and disability in later life.[40]

Kermack et al argue that the disease load in the first 15 years of life is the determinant of later life mortality.[41] Fridlizius also claims that exposure to diseases during childhood determine later life mortality but that the disease load during the first five years determines health in later life.[42] A study from Gambia shows that events in early life influence adult mortality: disease exposure and malnutrition causing permanent damage during the foetal stage immune system development.[43] Thus, a high disease load in early life is considered to cause damages to the immune system, which triggers increased susceptibility to diseases and thereby higher morbidity and mortality in later life. In a study from contemporary USA, it is shown that adult disease increased for those who had had a major bout of infectious disease in childhood. Any infectious disease in childhood quadruples the incidence of middle age lung conditions like emphysema and bronchitis. Non-infectious diseases have less effect on adult disease.[44]

In several studies, seasons with a high disease load and low nutrition and less diversified food intake are claimed to explain higher mortality, since they are a measure of unfavourable early life conditions. Doblhammer concludes that month of birth has an effect on mortalit.[45] Doblhammer and Vaupel draws similar conclusions.[46] In another study, Doblhammer found that individuals born in autumn live on average 0.44 years longer than individuals born in spring.[47]

Disease of the respiratory system in early life also affects health in later life; a study showed that men who had pneumonia in the first two years of life had a significant, impaired lung function at ages 60 to 70. Another study also shows reduced lung capacity when measured 1986 for individuals who experienced pneumonia in the first 2 years of life.[48] This vulnerability to long-term effects of pneumonia seems to last for the two first years alive. Several reasons may cause this: lungs grow rapidly at this time, lower respiratory tract infection is more severe since the peripheral airways – especially in boys – are small at this point, infants are most likely to be secondary cases of infection and hence infected with a larger dose of virus, and the type of virus common for the respiratory tract at this point may be more damaging than other organisms. Besides, viral infection at an early age may be important since this increases the risk of infections becoming latent or persistent.[49] Most cases of asthma arise in the first four years and asthma inception risk factors may affect individuals in the uterus or first years of life.[50] Bengtsson investigates the hypothesis that a high disease load during early life increases mortality in later life for adults of ages 25 to 55 and the

[38] Hall & Peckham (1997:10-11, 17-19, 21-22).
[39] Strachan (1997:113-114).
[40] Fogel (2004:4).
[41] Kermack, McKendrick & McKinlay (1934:699-702).
[42] Fridlizius (1989:3-4, 8, 11, 16-17).
[43] Moore, Cole, Poskitt, Sonko, Whitehead, McGregor & Prentice (1997:434).
[44] Blackwell, Hayward & Crimmins (2001:1275, 1280).
[45] Doblhammer (1999:4-7).
[46] Doblhammer & Vaupel (2001:2938-2939).
[47] Doblhammer (2002:17-20).
[48] Shaheen (1997:62-63).
[49] Shaheen (1997:66).
[50] von Mutius (2001:156).

disease load hypothesis is supported for infancy.[51] Bengtsson and Lindström study mortality in the same area and time period but for adults aged 55 to 80, and also here disease-load during infancy confirms the early life condition hypothesis,[52] and a second paper confirms this result.[53] Bengtsson *et al* concludes the same.[54]

Hall and Peckham states that infection in early childhood can cause damage to anatomical structures and the development of a number of systems, which may lead to adult disease or increased susceptibility to disease later in life.[55] Many of the immune system components in humans mature in early foetal life and babies of low weight may have sustained immune competence impairment as infants and as children.[56] Early infections can have permanent effects on the immune system.[57] Early life infection can affect the adult central nervous system and that host response to several infections, which may be related to a maturing immune system, has age-differences.[58] Reduced exposure to infections may have effects on the immune system, and that delay of infection exposure affects later life, since age at infection influences both short-term and long-term outcomes of infection. Some specific diseases such as Padget's disease and Parkinson's disease are thought to be delayed consequences of childhood infections of the measles virus and the virus that causes *encephalitis lethargica,* respectively.[59]

2.3 Conclusion

In conclusion, there are multiple studies from various research fields showing a relationship between disease in early life and mortality in later life. The relationship in the reviewed studies seems to be between disease load in infancy and later life mortality rather than during childhood and later life. There are also studies showing a relationship between foetal stage infection and later life mortality, as well as studies where such a relationship cannot be established. In the case of the hypothesised relationship between disease load in the uterus and later life mortality, it is possible that there are effects but that the methods used in these investigations cannot reveal them, as goes for the uterine nutritional explanation. Thus, the way early life conditions are usually measured should be looked further into, since this must be the base for any improvement into measuring conditions in early life, especially in intrauterine life. Therefore we next take a look into the way early life conditions usually are measured.

[51] Bengtsson (1997:16-19).
[52] Bengtsson & Lindström (2000:274-275).
[53] Bengtsson & Lindström (2001:9-13).
[54] Bengtsson, Broström & Lindström (2002:11, 20-23).
[55] Hall & Peckham (1997:10).
[56] Chandra (1974:1393-194).
[57] Aaby, Andersen & Knudsen (1993:156-162).
[58] Martyn (1997:24).
[59] Barker (1998:162).

3 Measures of early life conditions

Theoretically, the 'Barker' or 'early life conditions' hypothesis is rather well established, judging from the bulk of evidence in the previous section. However, looking at for example how few results are found when it comes to the 'true' Barker hypothesis – influence from the foetal stage to later life health – the methods for testing the hypothesis are either wrong, or the hypothesis itself might be questioned. As a start for an improvement of these measures, common ways of measuring conditions in early life are discussed.

3.1 Some commonly used measures for early life conditions

3.1.1 Tabulation of macro data

Early studies are based on aggregated historical populations to measure conditions in early life.[60] Kermack *et al* and Fridlizius argue that a lower disease load during early life improves health in youth and lowers mortality in adult life, while Preston and van de Walle argue that better health in adult life stems from better health through improved sanitation and hygiene during early life.[61] Even though these papers are examples of excellent population research, the empirical foundations rests on simple tabulation methods of macro data, and it is hard to take this as proof of any 'Barker' hypothesis.

3.1.2 Geographical origin and correlation measures

The first investigations in medical research on early life condition effects on morbidity and mortality rest on differences between individuals of different geographical origin.[62] If individuals born in an entirely different environment do not exhibit the same health patterns as individuals living in the same contemporary environment, *ceteris paribus*, it seems straightforward to formulate hypotheses on this health difference being caused by conditions in early life. Several investigations show a significant correlation between conditions during early life and health in later life through place of origin. One of the earliest is a study by Forsdahl from 1977, where he shows different mortality patterns for migrated of Finnish origin compared to natives in a high-mortality part of the very north of Norway, the *Finnmark* county.[63] The study fails to control for both historical and contemporary characteristics, but has been groundbreaking because it has brought attention to the subject of health in early life affecting health in later life. Barker and Osmond produced a number of papers between 1986 and 1990 where early life condition effects are investigated with records of birthplace.[64] Elo and Preston point to problems of research design and that units and dates of analysis change in a haphazard way in the studies by Barker and Osmond.[65] However, a general disadvantage with a place of origin explanation for early life condition effects on morbidity and mortality in later life is that it is a summary measure. Specific problems of the studies by Barker, Osmond, and colleagues is that they used crude statistical techniques such as correlations, and this means that no other factors are controlled for, which is not really acceptable nowadays.

[60] Kermack, McKendrick & McKinlay (1934:702-703), Preston & van de Walle (1978:290-291), Fridlizius (1989:16-17).
[61] Kermack, McKendrick & McKinlay (1934:699-701), Preston & van de Walle (1978:288-291), Fridlizius (1989:3, 16-17).
[62] Barker (1998); Chapter 1.
[63] Forsdahl (2002: 304-307, reprint; originally published in 1977).
[64] Barker & Osmond (1986a, 1986b, 1987), Barker, Osmond, Golding, Kuh & Wadsworth (1989), Osmond, Barker & Slattery (1990).
[65] Elo & Preston (1992:200).

3.1.3 Micro data

Generally, macro data will not be useful if we want to try the 'early life conditions' hypothesis and at the same time also control for other variables. The use of individual data has several advantages over using aggregated data. The nature of micro data makes it possible to test hypotheses using multivariate statistical tools that allow control for individual characteristics as for example sex, birth-place, family-specific characteristics, and socio-economic group simultaneously in an empirical model. But to make a reasonable analysis of early life conditions with individual data, some basic needs have to be met. Besides good data quality and some key variables as sex, we need long enough data sets so they incorporate both conditions in early life as well as in late life. If we are interested in a link to mortality, we need the entire life span of individuals. This means data that spans somewhere about at least 80 years, and in modern societies up to over 110 years, since we nowadays have many centenarians and even super-centenarians. Preferably we would also like the life histories to be spread over time, so the individuals not all are born at the same time, to ensure some variation. We also need exact information about when the individual died and the conditions in early life when it comes to disease exposure and nutrition. With modern data sets, such long life histories are not very common.

3.1.4 Birth season

With the use of individual data, there are a number of different measures to use, as for example final height, place of birth, and birth season. Birth season is supposed to be a combined proxy measure of nutrition and disease load during early life. This method has been used as the sole measure of early life conditions in some studies,[66] but also controlling for birth season while measuring conditions in early life with other proxies.[67] A drawback with the birth season measure is that it is a summary measure – it does not measure any direct link from early life conditions to later life health or mortality, but is rather a summary of everything related to the season/month of birth connected to later life health, just as place of birth and antropomethric measures as adult height.[68] To avoid the summary measure problem, a more direct method of measuring the link between early life conditions and later life health or mortality should be preferred.

3.1.5 Combined micro and macro analysis

Yet another method is based on a model that combines individual data with aggregated data on community food prices; [69] *i e*, what can be called a combined micro and macro analysis model. It uses data on community aggregates as for example fixed food prices during early life as a proxy for nutrition intake in early life.[70] A natural extension of this method is to use the local infant mortality rate as a proxy for disease load. Several investigations have used this approach for measuring the connections between early life disease load conditions and mortality later in life.[71] The method uses individual life histories that could for example be based on church record family reconstitutions. Within a survival regression framework, this

[66] Doblhammer (1999:4-7), Gavrilov & Gavrilova (1999:365-366), Doblhammer & Vaupel (2001:2938-2939), Gavrilova, Gavrilov, Evdokushkina, & Semyonova (2001:2, 10-13), Doblhammer (2002:17-20).

[67] Bengtsson & Lindström (2001:5, 24), Bengtsson, Broström & Lindström (2002:20-23), Johansson (2004:207-212).

[68] Forsdahl (2002: 304-307, reprint; originally published in 1977), Barker & Osmond (1986a, 1986b, 1987), Barker, Osmond, Golding, Kuh & Wadsworth (1989), Osmond, Barker & Slattery (1990, Nyström-Peck (1994), Alter & Oris (2000b), Edvinsson (2001:252-265).

[69] Bengtsson (1993:239-258).

[70] Bengtsson & Lindström (2000:270-276), Bengtsson & Lindström (2001:24-27), Bengtsson, Broström & Lindström (2002:14-15, 20), Johansson (2004:130-134).

[71] Bengtsson (1997:16-19), Bengtsson & Lindström (2000:274-275), Bengtsson & Lindström (2001:9-13), Bengtsson, Broström & Lindström (2002:11, 20-23), Johansson (2004:134-139), Johansson, Beise & Desjardins (2006).

means that this fixed covariate can be used to test if there is a significant relationship between the price of food during infancy and mortality later in life; thus, testing an extra-uterine conditions variant of the Barker hypothesis.[72] In the same way it is possible to test the infancy disease load early life condition hypothesis, as has for example been suggested by Fridlizius but also recognized by Barker.[73] The standard way of approximating infancy disease load is by the use of local infant mortality rates.[74] It is reasonable to assume that local infancy mortality rates gives an indication of how strong the disease load is if we are considering the conditions before the age of sulpha/antibiotics; thus, before WWII. If infants were exposed to various infectious diseases and without any cure for these diseases (thus; antibiotics), many infants would die since no cure was available, making the infant mortality rate a good indicator of the general local disease load. However, an important requisite for this to be reasonable is to use the *local* infant mortality rate, since otherwise this approximation is dubious. If we use infant mortality rates for an entire country in such an analysis, it will surely not approximate any local disease load, even in a small country.

A further extension of both the food price proxy for nutrition intake and the infant mortality rate proxy for disease load is to use these for approximating foetal stage conditions. This can be made by assuming that foetal nutrition intake can be approximated by the price of food during pregnancy, since maternal nutritional intake should be affected by food prices; at least in a historical context.[75] This would then be a test of the Barker foetal origin of later life disease hypothesis.[76] In the same way, it is possible to use local mortality rates for foetal stage exposure to disease. Regarding such exposure, any foetal disease load must be argued to stem from the mother.[77] Thus, it seems quite straightforward to use local female reproductive age mortality rate as a proxy for foetal stage disease load. It can however be hard to use the sex-specific mortality rates since mortality in – say – the age span 15 to 45 is rather low even in historical populations, which can induce problems with too few cases when calculating sex-specific mortality rates. Thus, mortality rates for both sexes together have often been used.[78] Also, it is not unlikely that mortality in adult ages is not related to infectious disease, but rather to accidents, work- and alcohol-related mortality, etc.[79] Hence, these measures are not unproblematic and improvements will therefore be suggested in the following section.

3.2 New ways to approximate early life conditions

Infant mortality includes all deaths from 0 to 365 days of age, which means that also the deaths occurring in the first months of life are included in this measure. Mortality during first months is usually considered to be endogenous, which means that the first-month deaths should not have any exogenous causation.[80] These deaths should rather be considered as endogenous mortality, most likely caused by disadvantageous conditions during the foetal stage or congenital defects. Hence, the first months fraction of the infant mortality rate should

[72] Often used as deviation from a 'normal' price estimated by the Hodrick-Prescott short-term-trend – to get relative prices comparable over time; see Johansson (2004) for a discussion.
[73] Fridlizius (1989), Barker (1998:Chapter 9)
[74] Bengtsson (1997:16-19), Bengtsson & Lindström (2000:274-275), Bengtsson & Lindström (2001:9-13), Bengtsson, Broström & Lindström (2002:11, 20-23), Johansson (2004:134-139).
[75] Bengtsson & Lindström (2000:270-276), Bengtsson & Lindström (2001:24-27), Bengtsson, Broström & Lindström (2002:14-15, 20), Johansson (2004:130-134).
[76] Barker (1998)
[77] Johansson (2004:134-136).
[78] Bengtsson (1997:16-19), Bengtsson & Lindström (2000:274-275), Bengtsson & Lindström (2001:9-13), Bengtsson, Broström & Lindström (2002:11, 20-23), Johansson (2004:134-139).
[79] Imhof & Lindskog (1973), Widén (1975), Fridlizius (1983, 1985), Fridlizius & Ohlsson (1983).
[80] It can be debated if one should count the 'extended foetal stage' to one, two or three months; some scholars even suggest that as long as up to 6 months should be regarded as an extension of the foetal stage.

not reflect any exogenous conditions, and should thus not be related to, for example, infancy infectious diseases. Does this mean that the local mortality rate is not a good approximation of local disease load since at least a fraction of it consists of endogenous mortality, not dependent on exogenous conditions? Since empirical evidence shows that first months mortality accounts for quite a large fraction of infant mortality, this may be a more important issue than what one might initially think. Hence, to be a good proxy for disease load, the infant mortality rate should be reduced by these deaths. A useful name for this mortality measure could be the exogenous infant mortality rate. However, an implication is that a reduction in the order of, say, 20 to 40 % of the 0-365 day infant mortality cases can affect the measure in a negative way by more random variation since many cases will be lost. In larger samples, this would not be any major issue, but in small samples, there is reason to be cautious with the endogenous infant mortality measure.

If the standard 0-365 day infant mortality rate is reduced by mortality during the first months and used as proxy for infancy disease load, this would – as discussed above – be the exogenous infant mortality rate. Thus, what could be called the endogenous infant mortality rate would be left out from the infant mortality rate. Since it is seldom a good idea to waste information, it might be a good thing not to just throw this endogenous infant mortality rate away before at least considering what it actually contains. Again, according to the discussion above, mortality during the first months after birth is usually considered to be due to maternal conditions during the foetal stage, for example low nutrition. But the conditions for the foetus during pregnancy is exactly what the 'true' Barker hypothesis is about, and if endogenous mortality stems from foetal conditions, this measure could then be used as an approximate measure of foetal conditions during the foetal stage. Since a quite large fraction of the deaths actually – as stated above – occur in the first months after birth, the number of cases should be enough to make this a good proxy measure for foetal stage conditions.

This new measure of early life conditions should be a much better proxy for foetal stage conditions than the standard measure of foetal stage conditions, the maternal disease load, approximated by the local adult-age mortality, but also of nutritional status, usually approximated by foetal stage food prices. It is not only that it more generally should measure the conditions during the foetal stage, but also that since it is common that mortality in ages 15 to 45 is quite low, so mortality for both sexes has to be used and not female mortality only. Since much of the male mortality in this age group – at least in historical populations – is due to accidents and drinking, and not due to infectious diseases, at least the male part of the mortality rate in this age group does not reflect any general disease load.[81] To a certain extent, it is likely to be the same with women. Most deaths were at least not due to infectious diseases as in the case with infants,[82] which means that this measure most likely is not a good proxy for foetal disease load, or maybe even of general foetal conditions. These doubts about the usefulness of the adult mortality rate during foetal stage have been raised before, and the discussion above could explain the meagre outcome when the foetal stage disease load hypothesis has been tested empirically with the local adult mortality rate.[83] The same is of course true for the nutritional link approximated by foetal stage food prices, where the link is rather far-fetched: foetal stage nutrition is then measured as changes in food prices, supposed to affect net-nutrition for the mother and then in turn her ability to transfer enough nutrition to her foetus.[84]

[81] Imhof & Lindskog (1973), Widén (1975), Fridlizius (1983, 1985), Fridlizius & Ohlsson (1983).
[82] Ibid.
[83] See discussion on this in Johansson (2004).
[84] Ibid.

To summarise, this new foetal stage condition measure would be theoretically correct, and also more accurate than the adult age mortality proxy (for example, local male and female mortality in age 15 to 45). It should also be empirically applicable without any practical problems if the deaths in the data sample are reasonable numerous. The measures could be a solution to some of the problems with approximating foetal stage early life conditions and testing the 'true' Barker hypothesis: the investigations that have tested this hypothesis using the local adult mortality rate as a proxy for foetal stage disease load have not been able to show such a relationship. Hence, the reasons for using these new measures are that

1) the exogenous infant mortality rate is a better measure of early life conditions since it is more pure disease load approximation than with the full 0-365 day infant mortality rate, and

2) the endogenous infant mortality rate provides a measure of nutritional conditions in utero, and can replace the indirect measuring of nutrition and disease load in the foetal stage via food prices and adult mortality, and test the 'true' Barker hypothesis.

Next, we test these new measures, with data from Scania, Sweden during 1794-1894.

4 Data

The empirical part of this study uses information on individuals from the five parishes of Halmstad, Hög, Kävlinge, Kågeröd, and Sireköpinge in the Scanian Demographic Database (*Skånes demografiska databas*).[85] 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.[86] Bengtsson and Lundh have showed that the database has a very high accuracy.[87]

4.1 Time period

The analysis will be from 1794 to when the database is terminated in1894; thus a time span of 100 years. The age group analysed is 55 to 80 years, since these are the ages when the early life experiences usually are said to come into effect. This also affects the starting point for the analysis: since the individuals have to be at least 55 years old when we start them, we need information on conditions in early life (disease load, socio-economic status) from 55 years before the start year of the analysis and with a starting point at 1794, we end up needing early life condition information from about 1750 and onwards. At this point in time, there is enough knowledge in the database for the analysis to work; hence the starting point of analysis.

4.2 Land structure

In the five Scanian parishes, the land structure can be divided into three groups according to land ownership since there were no tenants on church land. One group consisted of tenants on crown land (*kronobönder*), another of tenants on noble land (*frälsebönder*), and a third group of freeholders (*skattebönder*). Of the three groups, tenants on crown land and freeholders 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.[88] In the crown tenant case, the crown was the land-owner, and the peasant paid rent to the crown 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.[89] Tenants on noble land dominated in Halmstad and Sireköpinge while tenants on crown land and freeholders dominated in Hög and Kävlinge.[90]

[85] The database is described (mainly in Swedish) in the Scanian Demographic Database homepage www.ehl.lu.se/database/sdd.htm.
[86] Reuterswärd & Olsson (1993:1-8).
[87] Bengtsson & Lundh (1991:11-13, 1993:21-22).
[88] Weibull (1923:64-65).
[89] Heckscher (1941:213-216).
[90] Bengtsson & Dribe (1997:2).

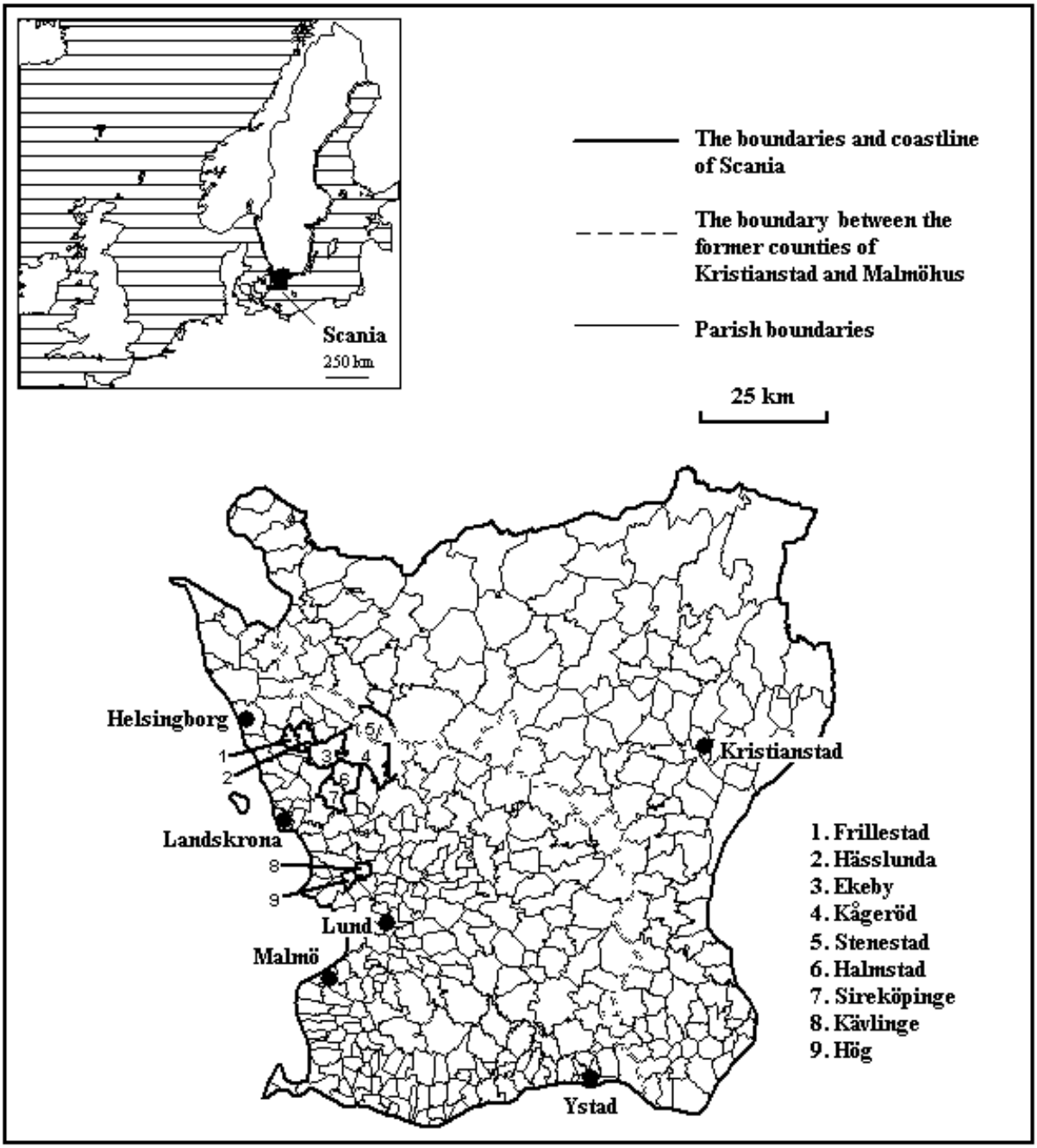


Figure 4.1: Map of Scania, Sweden and the five parishes in the Scanian Demographic Database: Halmstad, Hög, Kågeröd, Kävlinge, and Sireköpinge.

Figure 4.1 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.[91]

[91] Bengtsson & Dribe (1997).

4.3 Food prices

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.[92] Here, price of rye is used since the price variation of rye was more or less the same as for other common grains.[93] Figure 4.2 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.[94] Given the rye prices in Figure 4.2, the cost of living increased up until 1875, when it dropped sharply.[95]

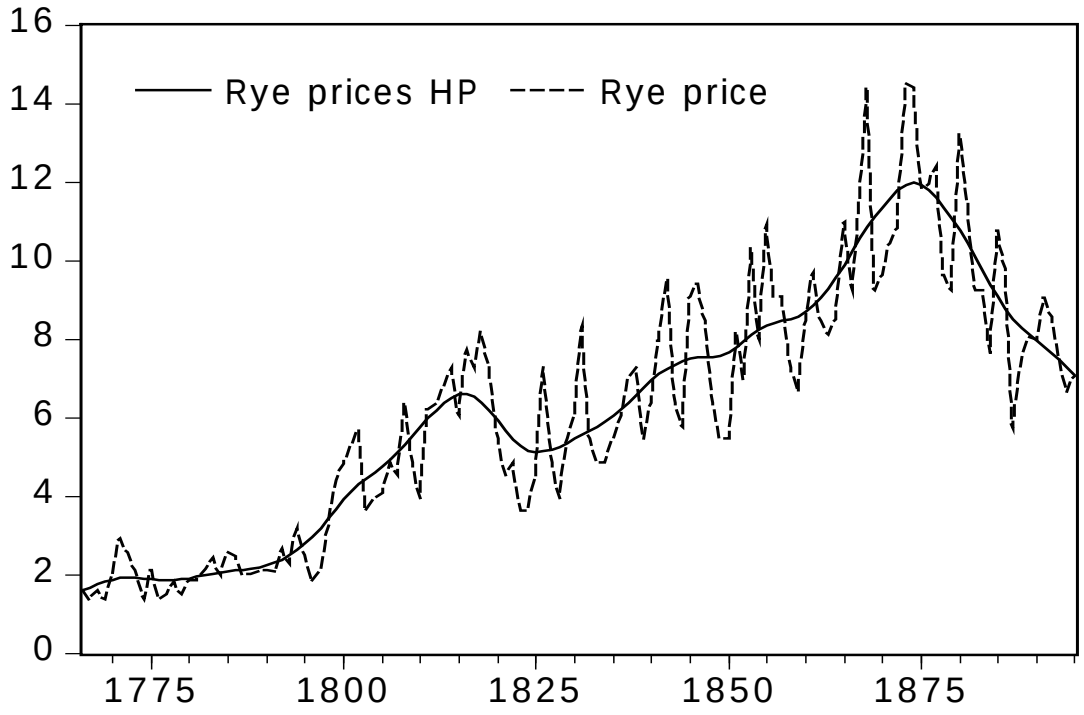


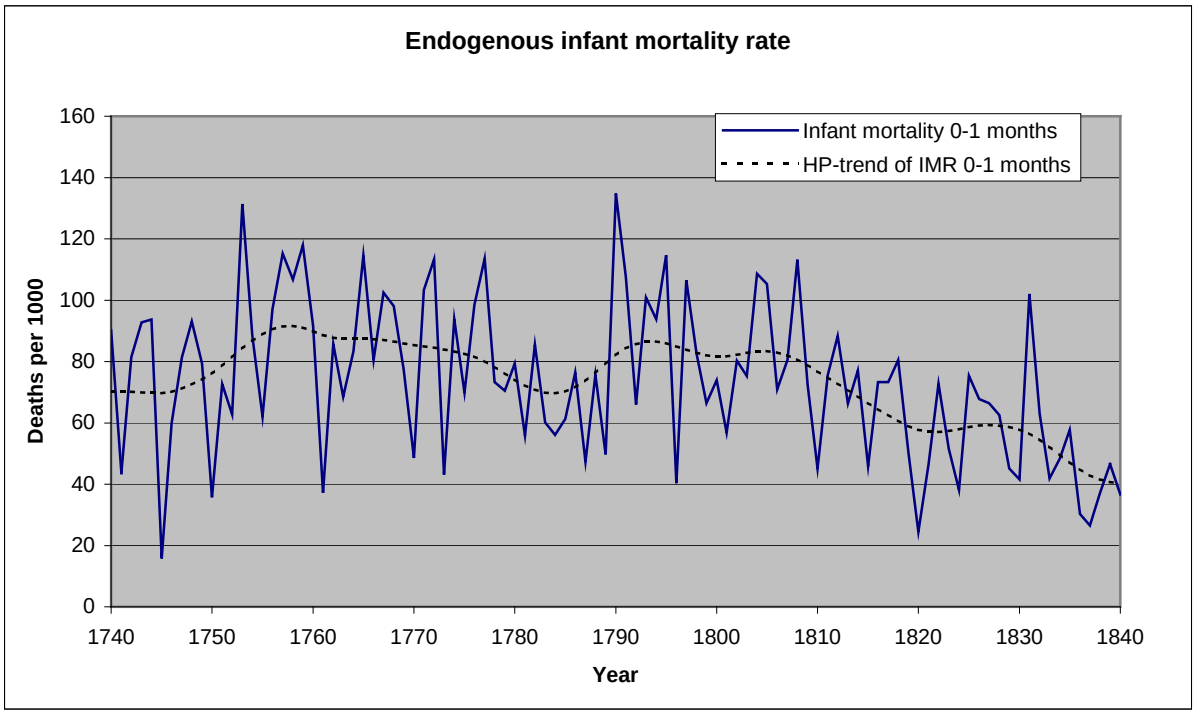
Figure 4.2: Local rye price and HP-trend of local rye price in crowns per hectolitre between 1766 and 1894. *Source*: Bengtsson and Dribe (1997:90-91); Appendix 2, Table 1.[96]

4.4 Mortality

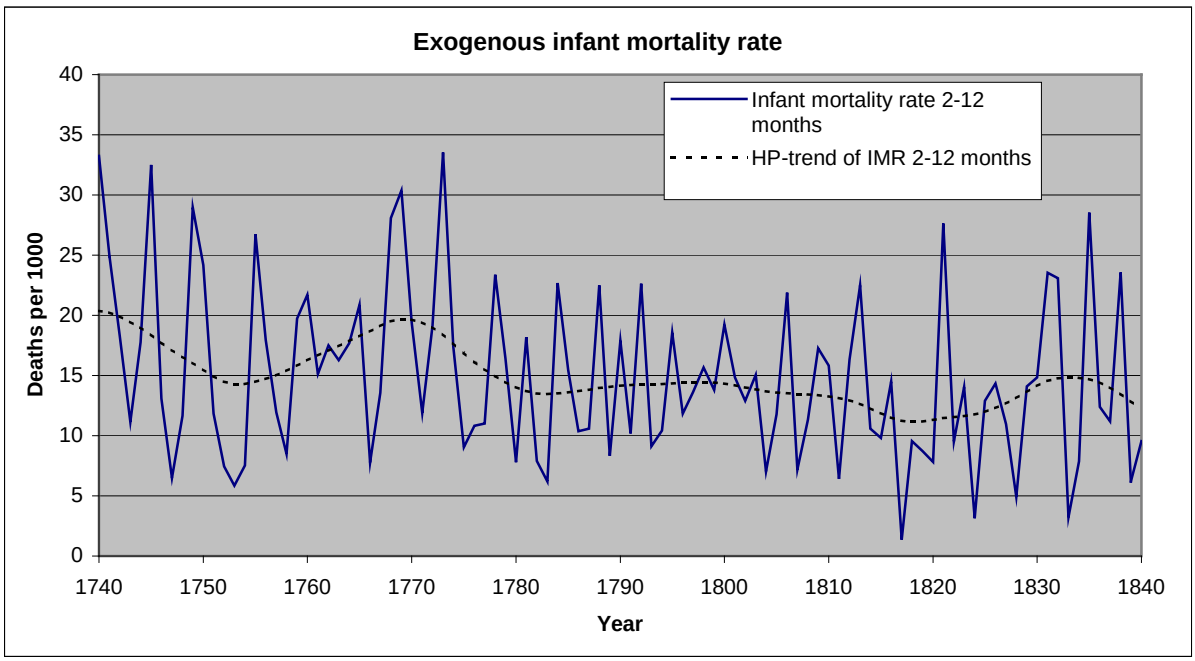
Below, the new measures suggested earlier in this paper, the endogenous and the exogenous infant mortality rate, are shown. Also a short-term trend is given, using a Hodrick-Prescott-trend to reveal a more general picture, without the most erratic movements. The graphs starts at about the time where the youngest individuals could be born; that is, at about 1794 minus 55 years. In this period, it is known that infant mortality was rather low in Sweden, and we also see this here for the five parishes in Scania, and that we have an increase during the

[92] Livi-Bacci (1991:87).
[93] Jörberg (1972:323-4).
[94] Jörberg (1972:308).
[95] It is possible to use real wages for the parishes instead of using food, however, the real wages are not very representative, since wage labour was not commonplace until in the latter part of the 19th century.
[96] 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.

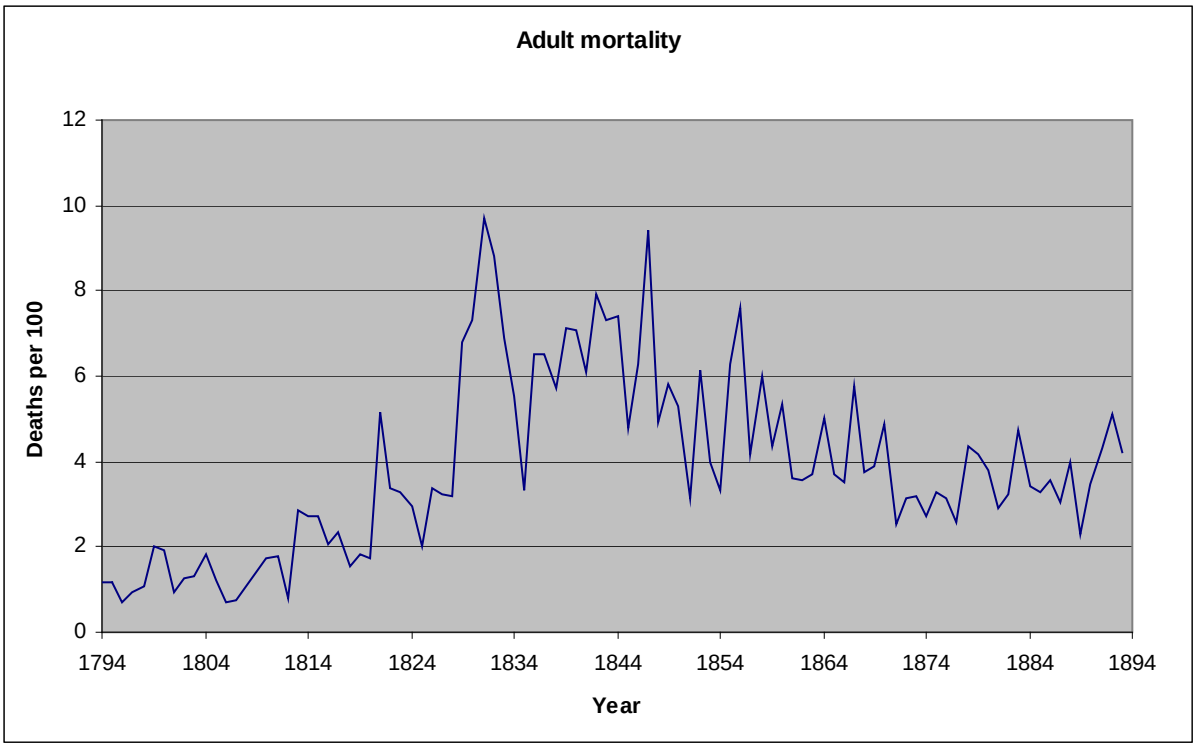
1750's and 60's. After this, we have a period up to the end of the 80's with decreasing mortality, and after a sharp rise in the 90's, mortality is more or less declining continuously in the 19th century. However, over the entire period, there are pronounced yearly fluctuations.



For the exogenous infant mortality, the same pattern is visible up to about 1790, but it does not rise in the 90's – it is more or less steady for the rest of the period. Also the exogenous mortality shows heavy yearly variation.



Below, mortality in ages 55-80 in the five parishes is given. There is a quite strong upward movement in mortality after about 1810 up to about 1840, and then a decline that last until about 1875, when mortality more or less levels off. This late decline in mortality, compared to for infant mortality, is quite similar to how it looks for the entire Sweden.



5 Theoretical and empirical model

5.1 Theoretical model

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 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. These determinants account for both short-term and long-term effects on mortality.

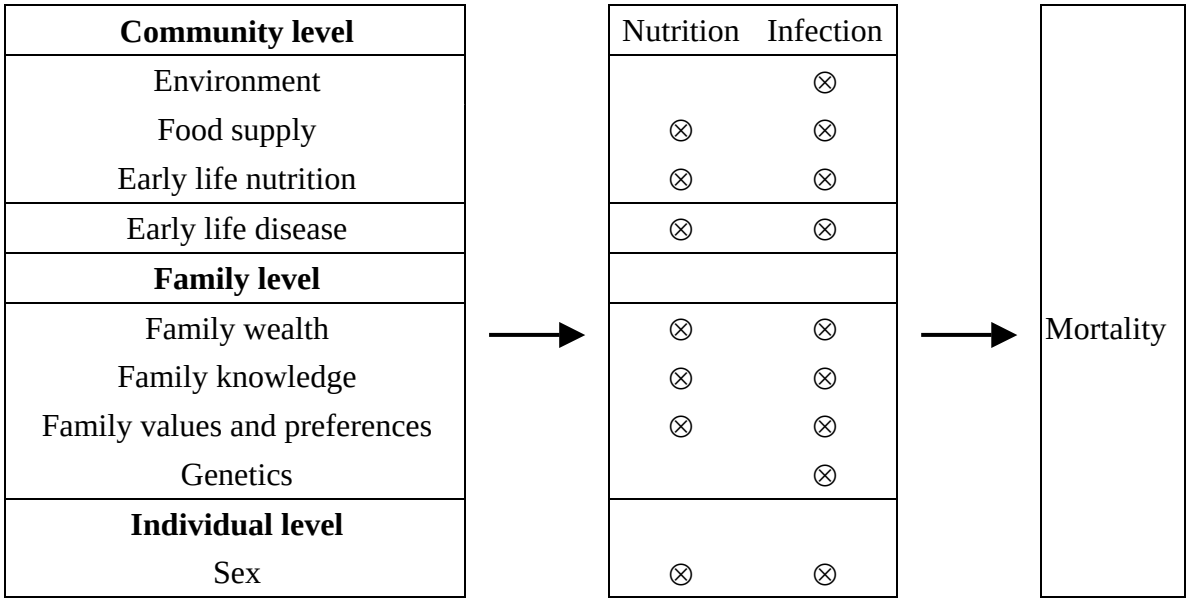


Figure 5.1: A theoretical multi level model for adult mortality combining the social science and the medical science approaches.

5.2 Empirical model

This model is a model where the theoretical variables are operationalised so they can be used to test the hypotheses concerning the new measurement for early life conditions with a regression model. The dependent variable is exact age at death. The independent variables are presented and discussed below.

5.2.1 Parish

Mortality differs especially between rural and urban environments, so environment has been operationalised into a variable indicating the birth parish of the individual. This could mean a higher risk for individuals born in Kävlinge compared to the other parishes, since it grew into a town in second half of 19th century. On the other hand, since this variable stems from birth, almost all were born before Kävlinge became a town, this might not be true. It could very well be that Kågeröd, which is very large (about 70 % of the others together), instead has a higher

Table 5.2: The operationalisation of the theoretical mortality model.

Theoretical model	Operationalisation	Variable in empirical model
Community level		
Environment	Parish	Hög (reference group) Kävlinge Halmstad Kågeröd Sireköpinge
Current nutrition	Food price	Local rye price in time *t*
	(same with 1 year lag)	Local rye price in time *t*-1
Foetal stage conditions	Mortality rate	Local infant mortality rate 0-1 months
Infancy conditions	Mortality rate	Local infant mortality rate 2-12 months
Family level		
Family wealth at birth	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
Sex		Male (reference group) Female

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

mortality due to its more dense population. Thus, there is not too much of a priori knowledge to have certain expectations on this variable: the parish variable is foremost included to control for any differences when it comes to birth place.

5.2.2 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,[97] 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 endogenous prices rather than exogenous prices are assumed.[98] Here, prices are assumed to be exogenous, because of the market integration during this period.[99] It is possible that if the

[97] Jörberg (1972:323-4), Livi-Bacci (1991:87).
[98] 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.
[99] Jörberg (1972:193-205).

price of grain rose, people would start consuming foodstuff that could work as substitutes. This could, for example, be to consume potatoes, but potatoes mostly went into alcohol production in the first decades of the 19th century.[100] Several investigations of Scanian adult mortality study the effect of current nutrition on mortality, but only one investigation has shown that high current rye prices increase mortality.[101] In a similar study, only one interaction term with occupation and rye price was significant.[102] In another study, no significant effect was found.[103]

The operationalisation of food supply to food prices concerns the effect of food prices on mortality. This is operationalised into a time-varying variable measuring rye prices during age 55 to 80 to represent effects on mortality induced by current food prices. Hence, the purpose of including current rye prices[104] in the model is to test if short-term fluctuations in food prices affect old-age mortality. Higher food prices than normal means the individuals can afford to buy less food if they are 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 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.[105] Hence, higher food prices are expected to increase mortality for net-consumers, and the current rye price coefficient is therefore expected to be positive. At the age of 55 and above, individuals are assumed to be net-consumers, since the retirement age in this area was about 59 years of age.

Nutrition-deficiency-induced weaker health may, however, not be sufficient to cause the death of an adult 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 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 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 delayed effects, and for adults, there is even more reason to believe in a slow process since the body of an adult is stronger to resist undernutrition.[106]

[100] Fridlizius (1988).
[101] Bengtsson (1997:9-10, 16-20).
[102] Bengtsson & Lindström (2000:270-276).
[103] Bengtsson & Lindström (2001:24-27).
[104] 'Current' food prices means that it refers to a time-varying price of food – thus, it changes yearly over the life of an individual. Here, it is the price that change every year at ages 55 to 80; thus, a period or 'current' effect.
[105] Pollard (1982:454).
[106] Johansson (2004:182).

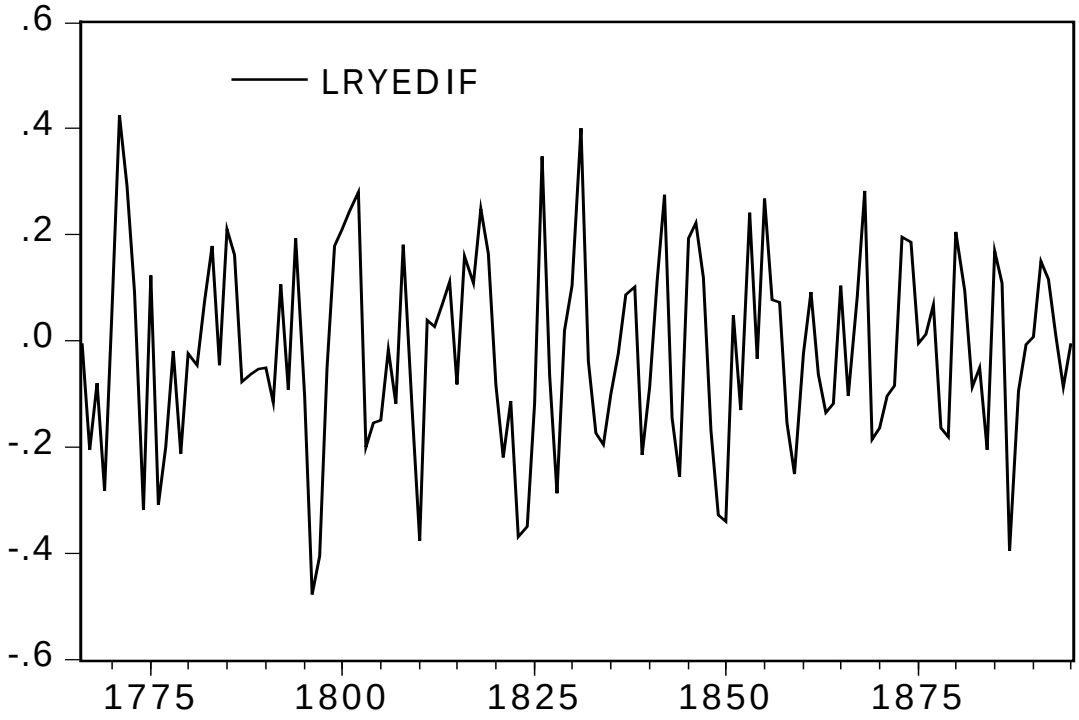
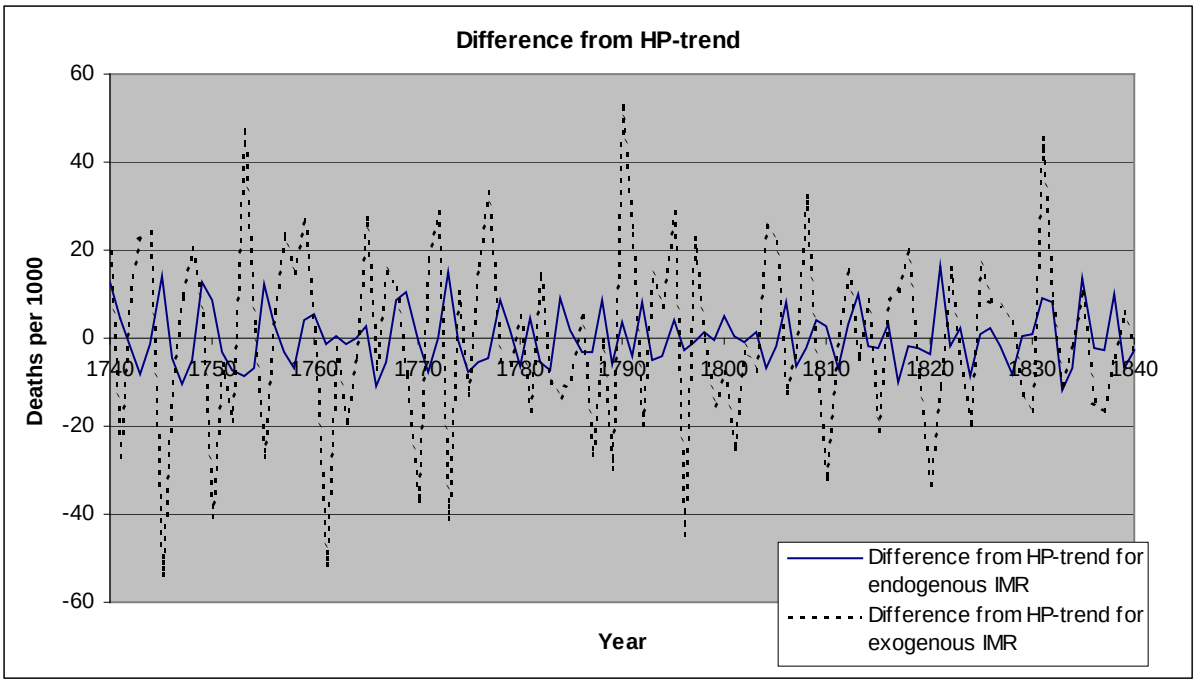


Figure 5.2: Log of local rye prices minus log of HP-trend of local rye prices for the five parishes 1766 -1894. *Source*: Bengtsson and Dribe (1997), Appendix 2.

The original local rye price was shown in a previous section. To capture short-term price variations in food prices, rye prices will be measured as the 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 5.2 below shows this logarithm deviance series and, as seen in the figure, the variance here is decently homoskedastic over time. Thus, what is measured here is the difference from what could be called a normal value of food prices, so that long-term changes will not dominate over short-term changes in food prices and we can compare conditions over a long time period. The expectation for these variable (whether lagged or not) is that it is positive, since that means that higher than normal prices increase old-age mortality.

5.2.3 Local mortality rates

In the same fashion as with local food prices, the local mortality rates and their HP-trends shown in Section 4, will be used to calculate what could be called differences from normal values. The difference between actual value and the HP-trend value for this value is given for both the endogenous and exogenous infant mortality rate in the graph below. It looks like there is more variation in the endogenous rate than the exogenous, and this is partly because of larger values. However, the important thing here is that with the difference from a normal value, all years become comparable and there is no trend left in these variables, so any correlation between these series and adult mortality cannot be due to any trend.



5.2.4 Socio-economic status

The socio-economic status variable in this paper is a fixed variable that is used as an early life condition variable for socio-economic conditions, so we can control for such conditions at birth. Thus, this variable stems from the family the individual was born into, not his or her own socio-economic status later in life. The variable 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.[107] Here, four SES groups are used and they are based on birth family of the individual, since new-borns have no socio-economic status of their own.

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.[108] 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.[109]

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 was too small to produce what was necessary to support their families.[110] The

[107] Follows Johansson (2004:Chapter 4).
[108] Dribe (2000:37-8).
[109] Bengtsson & Dribe (1997:21).
[110] Fridlizius (1975b:128).

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.

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.

5.2.5 Shared components

Since family belonging is an important carrier of information, it is vital to account for heterogeneity between different families to avoid biased estimates, regardless of whether this may stem from family values and preferences, genetics, or knowledge.[111] In the demography literature, this is usually referred to as frailty. In its original context, frailty means that some individuals are weaker than other individuals and tend to have higher mortality without any apparent or observable reason.[112] These unobserved characteristics result in a different mortality pattern when compared to other individuals with the same observable prerequisites. Here, frailty is considered to be biological endowments shared with siblings, while frailty originally refers to individual frailty. Thus, these unobserved characteristics are supposed to be shared by siblings and are measured as frailty derived from their family.

5.2.6 Birth cohort

Factors hard to quantify, such as improvements in hygiene and sanitation or even unobservable variables such as better immunity in the human host are needed to explain at least parts of the general mortality decline. Since these variables are hard to quantify or even unobservable, a relationship between mortality and time can be assumed to account for these unobservable changes during the general mortality decline. To include such a relationship between mortality and time, a cohort factor will be put into the empirical mortality model as a proxy for these unobservables. This proxy can be said to represent a general birth cohort effect; hence, an effect of being born at a specific time, shared by everyone born at this point in time and it is expected to be small but the sign is uncertain: the graph of old-age mortality in Section 4 revealed both increasing and thereafter decreasing mortality.

5.2.7 Sex

The last of the variables in the empirical model is a simple control for sex. Males are here defined as the reference group so mortality will be relative to males. In the case of adult men, Fridlizius finds excessive male morality in the beginning of the 19th century due to the enormous alcohol consumption at that point in time,[113] but over the entire time period, only a smaller disadvantage for males is expected.

[111] Schultz (1984:217-218).
[112] Vaupel, Manton & Stallard (1979:389-397), Vaupel (1988:277-287), Andersen, Borgan, Gill & Keiding (1997:660-674).
[113] Fridlizius (1989:5, 8-11).

6 Results

The empirical model was estimated with a Cox survival regression with shared frailty at the family level and there are 2317 events. The results shows a significant cohort factor but it is positive, meaning that on average, the mortality risk for 55-80 year olds increases over the period 1794-1894. Further, it shows that being born in any parishes compared to Hög results in a significant higher mortality. Especially in the large parish of Kågeröd, this is a bad thing since mortality risk doubles. When it comes to socio-economic status, the only significant difference compared to landless is among the smallholders, who have about 58 % higher risk of morality in adult ages. This is a bit surprising, since it is compared to the landless group. Also the sex variable is a bit unexpected, since there is no significant difference between the reference group of women compared to men.

When it comes to the variables of specific interest in this paper, results are more in line of what was expected. The current rye price variable, measuring deviation from a normal food price, both the coefficient for time t and for the lag, time t-1, are positive. It is otherwise common that we get opposite signs when we use the same variable in time t and t-1. Only the lagged rye price is significant, so the assumption that there is a delay from undernutition to death for old-age adults is confirmed. The effect is also quite strong. If we exclude lag 1, the lag 0 coefficient is still insignificant at conventional significance levels.

Covariate	Mean	Coef	Rel.Risk	S.E.	Wald p
Cohort factor	1771.252	0.011	1.011	0.001	0.000
Parish:					
Hög	0.113	0	1 (reference)		
Kävlinge	0.118	0.173	1.189	0.092	0.060
Halmstad	0.177	0.230	1.259	0.103	0.025
Sireköpinge	0.189	0.347	1.415	0.138	0.012
Kågeröd	0.402	0.741	2.097	0.203	0.000
Socio-economic Status:					
Landless	0.713	0	1 (reference)		
Smallholders	0.183	0.457	1.580	0.051	0.000
Free+Crown	0.061	0.030	1.031	0.104	0.772
Noble	0.043	-0.019	0.981	0.116	0.868
Male	0.494	0.058	1.060	0.042	0.163
Rye price (t)	-0.021	0.087	1.091	0.118	0.460
Rye price (t-1)	-0.022	0.395	1.484	0.118	0.001
Endogen. inf.mort	-0.940	0.002	1.002	0.001	0.071
Exogen. Inf mort	-0.059	0.005	1.005	0.003	0.093
Frailty(family)	1710.247	-0.000	1.000	0.000	0.007

Events	2317
Total time at risk	74875
Max. log. likelihood	-18183
LR test statistic	425
Degrees of freedom	14
Overall p-value	0

For the most interesting two variables, results are also as good. When divided in 0-1 and 1-12 months of mortality, what here is called endogenous infant morality and exogenous infant mortality, both these coefficients are according to expectations; both are positive, so a high value in early life means higher mortality later in life, and both of them are significant at conventional significance levels. Hence, this should mean that the suggested new and improved measures are working, and that they are also confirming both an increased mortality in later life due to unfavourable conditions during the foetal stage and due to unfavourable

conditions during infancy. Thus, the results in this paper confirm both the ‘Barker’ hypothesis as well as the ‘Fridlizius’ hypothesis.

Looking at the relative risks, the effects seem small but since the endogenous and the exogenous infant mortality variables have quite large deviations, the effects are not so small at all. Minimum endogenous infant mortality rate is about –45, which means that the ones with the most favourable foetal stage conditions have about 9 % lower risk of mortality in old age than for the average. For the least favourable, it is +54 which means about 11 % higher risk than for the average, so the difference between the least favourable and the most favourable is about 20 % higher mortality risk in old age. For the exogenous mortality rate it is –10 for the most favourable; thus 5 %, and +28 for the least favourable; thus 14 %, so again we end up at about a 20 % difference between most and least favourable conditions.

The last estimate reported in the table is the family frailty effect, and it is highly significant. This means that siblings share mortality risk, but if this is due to genetics, family preferences, or other shared components is impossible to know. However, there is an effect still after controlling for many variables, and that is still in effect after 55 years of life and more.

7 References

Aaby, P, Andersen, M & Knudsen, K (1993), 'Excess mortality after early exposure to measles'. *International Journal of Epidemiology*, 22(1), pp 156-162.

Adair, Linda S & Cole, Tim J (2003), 'Rapid Child Growth Raises Blood Pressure in Adolescent Boys Who Were Thin at Birth'. *Hypertension*, 41, Part 1, 451-456.

Ahlström, Göran (1974), *Svensk ekonomisk politik och prisutveckling 1776 – 1802*. Doctoral thesis. Skrifter utgivna av Ekonomisk-Historiska föreningen i Lund, Vol XIV, Lund.

Alexander, Barbara T (2003), 'Placental Insufficiency Leads to Development of Hypertension in Growth-Restricted Offspring'. *Hypertension*, 41, 457.

Alter, George and Oris, Michel (2000a), 'Mortality and Economic Stress: Individual and Household Responses in a Nineteenth-century Belgian Village' in Bengtsson, Tommy & Saito, Osamu (eds), *Population and Economy. From Hunger to Modern Economic Growth*. Oxford University Press, Oxford 2000, pp 335-370.

Alter, George and Oris, Michel (2000b), 'Early life conditions and the decline of adult mortality in the Belgian Ardennes, 1812 – 1890'. *PIRT/ELC Working Paper Series 2002*, Indiana University, No 02.

Ashorn, P, Maleta, K, Espo, M & Kulmala, T (2002), 'Male biased mortality among 1-2 year old children in rural Malawi'. *Archives of Disease in Children*, 87, pp 386-387.

Banggaard, Grethe (2004), *Befolkningsfremmende foranstaltninger og faldende børnedødelighed. Danmark ca 1750-1850*. Doctoral Thesis. University of Southern Denmark, Syddansk Universitetsforlag, Odense.

Barker, David J P (1995), 'Fetal origins of coronary heart disease'. *British Medical Journal*, 311, pp 171-184.

Barker, David J P (1997), 'Fetal nutrition and cardiovascular disease in later life' in Marmot, Michael G & Wadsworth, Michael E J (eds), *Fetal and early childhood environment: long-term health implications.* British Medical Bulletin, Volume 53, Number 1, pp 96-108.

Barker, David J P (1998), *Mothers, Babies, and Health in Later Life*. Churchill Livingstone, London.

Barker, David J P (2001), 'The malnourished baby and infant'. *British Medical Bulletin*, 60, pp 69-80.

Barker, David J P & Osmond, Clive (1986a), 'Infant mortality, childhood nutrition, and ischaemic heart disease in England and Wales'. *Lancet*, 10:1, pp 77-81.

Barker, David J P & Osmond, Clive (1986b), 'Childhood respiratory infection and adult chronic bronchitis in England and Wales'. *British Medical Journal*, 293, pp 1271-1275.

Barker, David J P & Osmond, Clive (1987), 'Death rates from stroke in England and Wales predicted from past maternal mortality'. *British Medical Journal*, 295(6590), pp 83-86.

Barker, D J P, Osmond, C & Law, C M (1989), 'The intrauterine and early postnatal origins of cardiovascular disease and chronic bronchitis'. *Journal of Epidemiology and Community Health*, 43, pp 237-240.

Barker, D J P, Osmond, C, Golding, J, Kuh, D & Wadsworth, M E J (1989), 'Growth in utero, blood pressure in childhood and adult life, and mortality from cardiovascular disease'. *British Medical Journal*, 298(6673), pp 564-567.

Barker, D J P, Forsén, T, Uutela, A, Osmond, C & Eriksson, J G (2001), 'Size at birth and resilience to effects of poor living conditions in adult life: a longitudinal study'. *British Medical Journal*, Volume 323, pp 1-5.

Bengtsson, Tommy (1993), 'Combined time-series and life event analysis. The impact of economic fluctuations and air temperature on adult mortality by sex and occupation in a Swedish mining parish, 1757-1850.' in Reher, David and Schofield, S (eds), *Old and new methods in historical demography*. Oxford University Press, Oxford, pp 239-258.

Bengtsson, Tommy (1997), 'Adult Mortality in Rural Sweden 1760-1895'. *EAP Working Series*, No 8, International Research Centre for Japanese Studies, Kyoto.

Bengtsson, Tommy (1999), 'The vulnerable child. Economic insecurity and child mortality in pre-industrial Sweden. A case study of Västanfors, 1757-1850'. *European Journal of Population*, 15, pp 117-151.

Bengtsson, Tommy (2000), 'Inequality in Death: Effects of the Agrarian Revolution in Southern Sweden 1765-1865', in Bengtsson, Tommy & Saito, Osamu (eds), *Population and Economy. From Hunger to Modern Economic Growth.* Oxford University Press, Oxford, pp 301-334.

Bengtsson, Tommy (ed) (2004), *Life Under Pressure. Mortality and Living Standards in Europe and Asia, 1700-1900*. Massachusetts Institute of Technology Press, Cambridge.

Bengtsson, Tommy & Lundh, Christer (1991), 'Evaluation of a Swedish Computer Program for Automatic Family Reconstitution'. *Lund Papers in Economic History*, No 8, Department of Economic History, Lund University, Lund.

Bengtsson, Tommy and Dribe, Martin (1997), 'Economy and Demography in Western Scania, Sweden,1650-1900.' *EAP Working Paper Series*, No. 10. International Research Center for Japanese Studies, Kyoto.

Bengtsson, Tommy and Lindström, Martin (2000), 'Childhood Misery and Disease in Later Life: The Effects on Mortality in Old Age of Hazards Experienced in Early Life, Southern Sweden, 1760-1894.'. *Population Studies*, Volume 54, pp 263-277.

Bengtsson, Tommy and Saito, Osamu (2000), 'Introduction' in Bengtsson, Tommy and Saito, Osamu (eds), *Population and Economy. From Hunger to Modern Economic Growth.* Oxford University Press, Oxford, pp 1-20.

Bengtsson, Tommy and Lindström, Martin (2001), 'Early-life Conditions and Mortality in Later Life: Southern Sweden, 1765-1894.'. Paper presented at the Social Science and History conference, Chicago, 15-18 November.

Bengtsson, Tommy, Broström, Göran and Lindström, Martin (2002), 'Effects of Conditions in Early-Life on Old Age Mortality in Southern Sweden 1766-1894: Functional Form and Frailty'. Paper for The European Social Science and History Association Conference, Den Haag, 27 February - 2 March 2002.

Bengtsson, Tommy and Dribe, Martin (2002), 'New Evidence on the Standard of Living in Sweden during the 18th and 19th Centuries: Long-term Development of the Demographic Response to Short-term Economic Stress among the Landless in Western Scania.' *Lund Papers in Economic History*, Department of Economic History, Lund University.

Blackwell, Debra L, Hayward, Mark D & Crimmins, Eileen M (2001), 'Does childhood health affect chronic morbidity in later life?'. *Social Science and Medicine*, 52, pp 1269-1284.

Blane, D, Hart, C L, Davey Smith, G, Gillis, C R, Hole, D J & Hawthorne, V M (1996), 'Association of cardiovascular disease risk factors with socio-economic position during childhood and during adulthood'. *British Medical Journal*, Volume 313, pp 1434-1438.

Brunner, Eric, Shipley, Martin J, Blane, David, Davey Smith, George & Marmot, Michael G (1999), 'When does cardiovascular risk start? Past and present socioeconomic circumstances and risk factors in adulthood'. *Journal of Epidemiology and Community Health*, Volume 53, pp 757-764.

Caldwell, John C (1979), 'Education as a Factor in Mortality Decline An Examination of Nigerian Data'. *Population Studies*, Volume 33, No 3, pp 395-413.

Campbell, Cameron D & Lee, James Z (2000), 'Price Fluctuations, Family Structure, and Mortality in Two Rural Chinese Populations: Household Responses to Economic Stress in Eighteenth- and Nineteenth-century Liaoning', in Bengtsson, Tommy & Saito, Osamu (eds), *Population and Economy. From Hunger to Modern Economic Growth.* Oxford University Press, Oxford, pp 371-419.

Chandra, Ranjit Kumar (1974), 'Immunocompetence in low-birth-weight infants after intrauterine malnutrition'. *Lancet*, ii, pp 1393-1394.

Chandra, Ranjit Kumar (1975), 'Fetal malnutrition and postnatal immunocompetence'. *American Journal of Disease in Childhood*, 129(4), pp 450-454.

Chandra, Ranjit Kumar (1984), 'Parasitic infection, nutrition, and immune response'. *Fed Proc*, 43(2), pp 251-255.

Chandra, Ranjit Kumar (1989), 'Nutritional regulation of immunity and risk of illness', *Indian Journal of Pediatrics*, 56(5), pp 607-11.

Chandra, Ranjit Kumar (1992), 'Nutrition and immunoregulation. Significance for host resistance to tumours and infectious disease in humans and rodents'. *Journal of Nutrition*, 122(3), Supplement, pp 754-757.

Chandra, Ranjit Kumar (2000), 'Food allergy and nutrition in early life: implications for later health'. *Proceedings of the Nutrition Society*, 59, pp 273-277.

Chandra, Ranjit Kumar (2002), 'Nutrition and the immune system from birth to old age'. *European Journal of Clinical Nutrition*, 56, Supplement 3, pp S73-S76.

Cheung, Yin Bun, Low, Louise, Osmond, Clive, Barker, David & Karlberg, Johan (2000), 'Fetal Growth and Early Postnatal Growth Are Related to Blood Pressure in Adults'. *Hypertension*, 36, pp 795-800.

Dezateux, Carol & Stocks, Janet (1997), 'Lung development and early origins of childhood respiratory diseases' in Marmot, Michael G & Wadsworth, Michael E J (eds), *Fetal and early childhood environment: long-term health implications*, British Medical Bulletin, Volume 53, Number 1, pp 40-57.

Doblhammer, Gabriele (1999), 'Longevity and Month of Birth: Evidence from Austria and Denmark'. *Demographic Research*, Volume 1:3.

Doblhammer, Gabriele (2002), 'Differences in Lifespan by Month of Birth for the United States: The impact of early life events and conditions on late life mortality'. MPIDR Working Paper WP 2002-019, Max-Planck Institute for Demographic Research, Rostock, May.

Doblhammer, Gabriele & Vaupel, James W (2001), 'Lifespan Depends on Month of Birth'. *Proceedings of the National Academy of Sciences of the United States of America*, 98(5), pp 2934-2939.

Donker, G A, Labarthe, D R, Harrist, R B, Selwyn, B J, Wattigney, W & Berenson, G S (1997), 'Low birth weight and blood

pressure at age 7-11 in a biracial sample'. *American Journal of Epidemiology*, Volume 145, Issue 5, pp 387-397.

Doull, Iolo J M & Holgate, Stephen T (1997), 'Asthma: early predisposing factors' in Marmot, Michael G & Wadsworth, Michael E J (eds), *Fetal and early childhood environment: long-term health implications.* British Medical Bulletin, Volume 53, Number 1, pp 71-80.

Dribe, Martin (2000), *Leaving Home in a Peasant Society. Economic Fluctuations, Household Dynamics and Youth Migration in Southern Sweden, 1829-1866*. Doctoral Thesis. Lund Studies in Economic History 13, Almqvist & Wiksell International, Södertälje.

Edvinsson, Sören (1992), *Den osunda staden. Sociala skillnader i dödlighet i 1800-talets Sundsvall*. Doctoral thesis. Umeå University, Umeå.

Edvinsson, Sören (2001), 'Adult Mortality and Childhood Conditions' in Tedebrand, Lars-Göran & Sköld, Peter (eds), *Nordic Demography in History and Present-Day Society*. Scandinavian Population Studies, Volume 12 & Report no. 18 from the Demographic Data Base, Umeå University, Umeå, pp 247-268.

Elo, Irma T & Preston, Samuel H (1992), 'Effects of Early-life Conditions on Adult Mortality: A Review'. *Population Index*¸ 58(2), pp 186 - 212.

Eskenazi, B & Bergmann, J J (1995), 'Passive and active maternal smoking during pregnancy, as measured by serum cotinine, and postnatal smoke esposure. I. Effects on physical growth at 5 years'. *American Journal of Epidemiology*, Volume 142, Issue 9, pp S10-S18.

Ezzati, Majid & Kammen, Daniel M (2001), 'Indoor air pollution from biomass combustion and acute respiratory infections in Kenya: an exposure-response stud'. *Lancet*, Volume 358, pp 619-624.

Fogel, Robert W (1986), 'Nutrition and the Decline in Mortality since 1700: Some Preliminary Findings' in Engerman, Stanley L & Gallman, Robert E, *Long-Term Factors in American Economic Growth*. National Bureau of Economic Research, Chicago, pp 439-527.

Fogel, Robert W (1988), 'Nutrition and the Decline in Mortality since 1700: Some Additional Preliminary Findings' in Brändström, Anders and Tedebrand, Lars-Göran (eds), *Society, Health and Population during the Demographic Transition*. Almquist & Wiksell International, Stockholm, pp 369-384.

Fogel, Robert W (1994a), 'The Relevance of Malthus for the Study of Mortality Today: Long-Run Influences on Health, Mortality, Labour Force Participation, and Population Growth' in Lindahl-Kiessling, Kerstin and Landberg, Hans (eds), *Population, Economic Development, and the Environment. The making of our common future.* Oxford University Press, Oxford, pp 231-284.

Fogel, Robert W (1994b), 'Economic Growth, Population Theory, and Physiology: The Bearing of Long-Term Processes on the Making of Economic Policy'. *The American Economic Review*, Volume 84, Issue 3, pp 369-395.

Fogel, Robert W (2000), 'Second thoughts on the European Escape from Hunger: Famines, Chronic Malnutrition, and Mortality Rates' in Osmani, Siddiqur Rahman (ed). *Nutrition and Poverty.* Oxford University Press, New Dehli, pp 243-286.

Fogel, Robert W (2004), 'Changes in the disparities in chronic disease during the course of twentieth century'. NBER Working paper Nr 10311, National Bureau of Economic Research.

Fogel, Robert W, Engerman, Stanley L, Floud, Roderick, Friedman, Gerald, Margo, Robert A, Sokoloff, Kenneth, Steckel, Richard H, Trussell, T James, Villaflor, Georgia & Wachter, Kenneth W (1983), 'Secular Changes in American and British Stature and Nutrition'. *Journal of Interdisciplinary History*, XIV:2, pp 445-481.

Fogel, Robert W & Costa, Dora L (1997), 'A theory of technophysio evolution, with some implications for forecasting population, health care costs, and pension costs'. *Demography*, Volume 34, Nr 1, pp 49-66.

Forsdahl, Anders (2002; first printed in 1973), 'Observations throwing light on the high mortality in the county of Finnmark. Is the high mortality today a late effect of very poor living donations in childhood and adolescence?'. *International Journal of Epidemiology*, Nr 2002:31, pp 302-308.

Fridlizius, Gunnar (1975a), 'Some New Aspects on Swedish Population Growth. I. A Study at Country Level.'. *Economy and History*, Volume XVIII:1, pp 3 - 33.

Fridlizius, Gunnar (1975b), 'Some New Aspects on Swedish Population Growth. II. A Study at the Parish Level.'. *Economy and History*, Volume XVIII:2, pp 126 - 154.

Fridlizius, Gunnar (1979a), 'Sweden.' in Lee, W Robert (ed), *European Demography and Economic Growth.* Croom Helm, London, pp 340-405.

Fridlizius, Gunnar (1979b), 'Population, Enclosure and Property Rigths'. *Economy and History*, Volume XXII:1, pp 3 - 37.

Fridlizius, Gunnar (1984), 'The Mortality Decline in the First Phase of the Demographic Transition: Swedish Experiences.' in Bengtsson, Tommy, Fridlizius, Gunnar and Ohlsson, Rolf (eds), *Pre-industrial population change: The mortality decline and short-term population movements.* Amqvist & Wiksell International, Lund, pp 71-114.

Fridlizius, Gunnar (1988): 'Sex-Differential Mortality and Socio-Economic Change. Sweden 1750-1910' in Brändström, Anders

and Tedebrand, Lars-Göran (eds), *Society, Health and Population during the Demographic Transition.* Almquist & Wiksell International, Stockholm, pp 237-272.

Fridlizius, Gunnar (1989), 'The deformation of cohorts. Nineteenth century mortality in a generational perspective.'. *Scandinavian Economic History Review*, 37(3), pp 3 - 17.

Fridlizius, Gunnar & Ohlsson, Rolf (1984), 'Mortality patterns in Sweden 1751-1802 – a regional analysis' in Bengtsson, Tommy, Fridlizius, Gunnar and Ohlsson, Rolf (eds), *Pre-industrial population change: The mortality decline and short-term population movements.* Amqvist & Wiksell International, Lund, pp 299-328.

Gavrilov, Leonid A & Gavrilova, Natalia S (1999), 'Season of Birth and Human Longevity.'. *Journal of Anti-aging Medicine*, Volume 2, Number 4, pp 365-366.

Gavrilova, Natalia S, Gavrilov, Leonind A, Evdokushkina, Galina N & Semyonova, Victoria G (2001), 'Early-life predictors of human longevity'. Paper presented at the 2001 annual meeting of the Social Science History Association, Chicago Nov 15 - 18 2001.

Gershwin, M E, Beach, R S & Harley, L S (1985), *Nutrition and Immunity*. Academic Press, Orlando.

Gogos, C A & Kalfarentzos, F (1995), 'Total parenteral nutrition and immune system activity: a review'. *Nutrition*, 11(4), pp 339-344.

Gopolan, C (2000), 'Undernutrition: Measurement and Implications' in Osmani, Siddiqur Rahman (ed), *Nutrition and Poverty.* Oxford University Press, New Dehli, pp 17-47.

Graham, Neil M H (1994), 'Respiratory Infections' in Pless, Ivan Barry (ed), *The Epidemilogy of Childhood Disorders*. Oxford University Press, New York.

Hack, Maureen, Schluchter, Mark, Cartar, Lydia, Rahman, Mahboob, Cuttler, Leona & Borawski, Elaine (2003), 'Growth of Very Low Birth Weight Infants to Age 20 Years'. *Pediatrics*, Volume 112, No 1, pp E30-E38.

Hales, C N (1997), 'Non-insulin-dependent diabetes mellitus' in Marmot, Michael G & Wadsworth, Michael E J (eds), *Fetal and early childhood environment: long-term health implications.* British Medical Bulletin, Volume 53, Number 1, pp 109-122.

Hall, A J & Peckham, C S (1997), 'Infections in childhood and pregnancy as a cause of adult disease – methods and examples' in Marmot, Michael G & Wadsworth, Michael E J (eds), *Fetal and early childhood environment: long-term health implications.* British Medical Bulletin, Volume 53, Number 1, pp 10-23.

Honickey, R E, Osborne, J S & Akpom, C A, (1985), 'Symptoms of respiratory illness in young children and the use of wood-burning stoves for indoor heating.'. *Pediatrics*, 75, pp 587-593.

Hull, H F, Williams, P J & Oldfield, F (1983), 'Measles mortality and vaccine efficacy in rural West Africa. *Lancet*, 1, 972-975.

Imhof, Arthur E & Lindskog, Bengt I (1973), 'Dödsorsakerna i Sydsverige 1749-1773'. *Sydsvenska medicinhistoriska sällskapets årsskrift 1973*, pp 120-143.

Johansson, Kent (2004), *Childhod mortality during the demographic transition.* Almquist & Wiksell, Lund.

Johansson, Kent & Beise, Jan (2006) , 'Early life conditions in the Quebec area', conference paper, PAA, Los Angeles.

Jones, Peter (1997), 'The early origins of schizophrenia' in Marmot, Michael G & Wadsworth, Michael E J (eds), *Fetal and early childhood environment: long-term health implications.* British Medical Bulletin, Volume 53, Number 1, pp 135-155.

Jörberg, Lennart (1972), *A History of Prices in Sweden 1732-1914.* Volume 2. C W K Gleerup, Lund.

Journal of Interdisciplinary History (1983), 'The Relationship of Nutrition, Disease, and Social Conditions: A Graphical Presentation' in 'Hunger and History: The Impact of Changing Food Production and Consumption Patterns on Society'. *Journal of Interdisciplinary History*, Volume 14, Issue 2, pp 503-506.

Kermack, W O, McKendrick, A G & McKinlay, P L (1934), 'Death Rates in Great Britain and Sweden: Some Regularities and Their Significance.'. *Lancet*, pp 698–703.

Knodel, John (1988): 'Two Centuries of Infant, Child and Maternal Mortality in German Village Populations' in Brändström, Anders and Tedebrand, Lars-Göran (eds), *Society, Health and Population during the Demographic Transition*. Almquist & Wiksell International, Stockholm, pp 23-48.

Lee, James Z & Campbell, Cameron D (1997), *Fate and fortune in rural China. Social organization and population behaviour in Liaoning 1774-1873*. Cambridge University Press, Cambridge.

Lee, James Z & Feng, Wang (1999), *One Quarter of Humanity. Malthusian Mythology and Chinese Realities, 1700-2000.* Harvard University Press, Cambridge.

Lithell, Ulla-Britt (1981), *Breast-feeding and Reproduction. Studies in marital fertility and infant mortality in 19th century Finland and Sweden*. Doctoral Thesis. Studia Historica Upsalensia 120, University of Uppsala, Uppsala.

Livi-Bacci, Massimo (1983), 'The Nutrition-Mortality Link in Past Times: A Comment.' *Journal of Interdisciplinary History*, XIV:2, pp 293-298.

Livi-Bacci, Massimo (1991), *Population and nutrition. An Essay on European Demographic History*. Cambridge University Press, Cambridge.

Lunn, Peter (1991), 'Nutrition, Immunity and Infection' in Schofield, R, Reher, D & Bideau, A (eds), *The Decline of Mortality in Europe*. Clarendon Press, Oxford, pp 131-145.

Lurbe, Empar, Torro, Isabel, Rodríguez, Concepción, Alvarez, Vicente & Redón, Josep (2001), 'Birth Weight Influences Blood Pressure Values and Variability in Children and Adolescents', *Hypertension*, 38, 389.

Lurbe, Empar, Torro, Isabel, Carvajal, Eva, Alvarez, Vicente & Redón, Josep (2003), 'Birth Weight Impacts on Wave Reflections in Children and Adolescents', *Hypertension*, 41, 646-650.

Lynch, John W, Kaplan, George A, Cohen, Richard D, Kauhanen, Jussi, Wilson, Thomas W, Smith, Nicholas L & Salonen, Jukka T (1994), 'Childhood and adult socio-economic status as predictors of mortality in Finland'. *Lancet*, Volume 343, pp 524-527.

Lynch, Katherine A & Greenhouse, Joel B (1994), 'Risk Factors for Infant Mortality in Nineteenth-Century Sweden'. *Population Studies*, Volume 48, No 1, pp 117-133.

Marmot, Michael G (1997); 'Early life and adult disorder: research themes'. *British Medical Bulletin*, Volume 53, Number 1, January, pp 3-9.

Martinez, F D, Wright, A L, Taussig, L M, Holberg, C J, Halonen, M & Morgan, W J (1995), 'Asthma and wheezing in the first six years of life'. *New England Journal of Medicine*, 332(3), pp 133-138.

Martyn, C N (1997), 'Infection in childhood and neurological diseases in adult life' in Marmot, Michael G & Wadsworth, Michael E J (eds), *Fetal and early childhood environment: long-term health implications*. British Medical Bulletin, Volume 53, Number 1, pp 24-39.

Mata, Leonardo J, Urrutia, Juan J & Garcia, Berhta (1967), 'Effect of infection and diet on child growth: experience in Guatemalan village' in Wolstenholme, G E W & O'Connor, Maeve, *Nutrition and Infection*, Ciba Foundation Study Group No 31, J & A Churchill Ltd, London, pp 112-126.

Maughan, Barbara & McCarthy, Gerard (1997), 'Childhood adversities and psychosocial disorders' in Marmot, Michael G & Wadsworth, Michael E J (eds), *Fetal and early childhood environment: long-term health implications*. British Medical Bulletin, Volume 53, Number 1, pp 156-169.

Moore, Sophie E, Cole, Timothy J, Poskitt, Elizabeth M E, Sonko, Bakary J, Whitehead, Roger G, McGregor, Ian A & Prentice, Andrew M (1997), 'Season of birth predicts mortality in rural Gambia'. *Nature*, Volume 388, p 434.

Morris, K, Morgenlander, M, Coulehan J L, Gahagen, S, Arena, V C, Morgenlander, M (1990), 'Wood-burning stoves and lower respiratory tract infections in American Indian Children'. *American Journal of Disease of Children*, 144(4), pp 105-108.

Mosely, Henry & Chen, Lincoln (1984), 'An analytical framework for the study of child survival in developing countries'. *Population and Development Review*, 10, Supplement, pp 25-45.

Nicholas, Stephen & Steckel, Richard H (1991), 'Heights and Living Standards of English Workers During the Early Years of Industrialization, 1770-1815'. *The Journal of Economic History*, Volume 51, No 4, pp 937-957.

Olsson, Franceska & Reuterswärd, Elisabeth (1993), 'Skånes demografiska databas 1646-1894. En källbeskrivning'. *Lund Papers in Economic History/Population Economics*, No 33, Department of Economic History, Lund University, Lund.

Osmond, C, Barker, D J P & Slattery, J M (1990), 'Risk of death from cardiovascular disease and chronic bronchitis determined by place of birth in England and Wales.'. *Journal of Epidemiology and Community Health*, 44(2), pp 139-141.

Perry, Ivan J (1997), 'Fetal growth and development: the role of nutrition and other factors' in Kuh, Diana & Ben-Shlomon, Yoav (eds), *A Life Course Approach to Chronic Disease Epidemiology. Tracing the origins of ill-health from early to adult life*. Oxford Medical Publications, Oxford, pp 145-168.

Persson, Karl Gunnar (1996), 'The Seven Lean Years, Elasticity Traps, and Intervention in Grain Markets in Pre-Industrial Europe'. *The Economic History Review*, New Series, Volume 49, No 4, pp 692-714.

Pharoah, P O D, Stevenson, C J & West, C R (1998), 'Association of blood pressure in adolescence with birthweight'. *Archives of Disease in Childhood. Fetal and Neonatal Edition*, 79, pp F114-F118.

Pollard, John H (1982), 'Morbidity and longevity' in Ross, John A (ed), *International Encyclopedia of Population.* Volume II. The Free Press, Macmillan, New York, pp 452-458.

Preston, Samuel H & van de Walle, Etienne (1978), 'Urban French Mortality in the Nineteenth Century.'. *Population Studies*, Volume 32, Issue 2, pp 275-297.

Preston, Samuel H & Haines, Michael R (1991), *Fatal Years: Child Mortality in Late Nineteenth-century America.* Princeton University Press, Princeton.

Reading, Richard (1997), 'Poverty and the health of children and adolescents'. *Archives of Disease in Childhood*, Volume 76, pp 463-467.

Rona, R J, Gulliford, M C & Chinn, S (1993), 'Effects of prematurity and intrauterine growth on respiratory health and lung

function in childhood'. *British Medical Journal*, 306, pp 817-820.

Roseboom, Tessa J, van der Meulen, Jan H P, Ravelli, Anita C J, Osmond, Clive, Barker, David J P & Bleker, Otto P (2001), 'Effects of prenatal exposure to the Dutch famine on adult disease in later life: an overview'. *Molecular and Cellular Endocrinology*, 185, pp 93-98.

Rosenberg, Margit (1988): 'Birth Weight, Breast-Feeding, Postpartum 'Amenorrhea and Infant Mortality in Three Norwegian Cities during Late Nineteenth and Early Twentieth Century' in Brändström, Anders and Tedebrand, Lars-Göran (eds), *Society, Health and Population during the Demographic Transition.* Almquist & Wiksell International, Stockholm, pp 49-60.

Sandberg, Lars G & Steckel, Richard H (1980), 'Soldier, Soldier, what Made You Grow so Tall? A Study of Height, Health, and Nutrition in Sweden, 1720-1881'. Economy and History, Volume XXIII:2, pp 91-105.

Schultz, T Paul (1984), 'Studying the Impact of Household Economic and Community Variables on Child Mortality' in Mosley, W Henry and Chen, Lincoln C (eds), *Child Survival. Strategies for Research*, Population and Development Review, Supplement to Volume 10, pp 215-235.

Scrimshaw, Nevin S (1985), 'The Value of Contemporary Food and Nutrition Studies for Historians' in Rotberg, Robert I and Rabb, Thoeodore K (eds), *Hunger and History. The Impact of Changing Food Production and Consumption Patterns of Society*, Cambridge University Press, New York.

Scrimshaw, Nevin S (2003), 'Historical Concepts of Interactions, Synergism and Antagonism between Nutrition and Infection'. *Journal of Nutrition*, 133, pp 316S-321S.

Scrimshaw, Nevin S, Taylor, Carl E & Gordon, John E (1968), *Interactions of nutrition and infection.* World Health Organization, Geneva.

Scrimshaw, Nevin S & SanGiovanni, J P (1977), 'Synergism of nutrition, infection, and immunity: an overview'. *American Journal of Clinical Nutrition*, 66(2), pp 464S-477S.

Shaheen, Seif (1997), 'The beginnings of chronic airflow obstuction' in Marmot, Michael G & Wadsworth, Michael E J (eds), *Fetal and early childhood environment: long-term health implications.* British Medical Bulletin, Volume 53, Number 1, pp 58-70

Stevenson, C J, West, C R & Pharoah, P O D (2001), 'Dermatoglyphic patterns, very low birth weight, and blood pressure in adolescence'. *Archives of Disease in Childhood: Fetal Neonatal Edition*, 84, pp F18-F22.

Stevenson, D K, Verter, J, Fanaroff, A A, Oh, W, Ehrenkranz, R A, Shankaran, S, Donovan, E F, Wright L L, Lemons, J A, Tyson, J E, Korones, S B, Baurer, C R, Stoll B & Papile, L-A (2000), 'Sex differences in outcomes of very low birhtweight infants: the newborn male disadvantage'. *Achives of Disease in Children. Neonatal Edition*, 83, pp F182-F185.

Strachan, David P (1997), 'Respiratory and allergic diseases' in Kuh, Diana & Ben-Shlomon, Yoav (eds), *A Life Course Approach to Chronic Disease Epidemiology. Tracing the origins of ill-health from early to adult life*. Oxford Medical Publications, Oxford, pp 101-120.

Strauss, Richard S (1997), 'Effects of the intrauterine environment on childhood growht' in Marmot, Michael G & Wadsworth, Michael E J (eds), *Fetal and early childhood environment: long-term health implications*, British Medical Bulletin. Volume 53, Number 1, pp 81-95.

Sundin, Jan (1977), 'The demographic data base at the university of Umeå' in Sundin, Jan & Söderlund, Erik, *Time Space and Man. Essays in Microdemography.* Amquist & Wiksell International, Stockholm, pp 251-259.

Sundin, Jan (1995), 'Culture, Class, and Infant Mortality during the Swedish Mortality Transition, 1750-1850.' *Social Science History*, 19:1, pp 117-145.

Sundin, Jan & Tedebrand, Lars-Göran (1981), 'Mortality and Morbidity in Swedish Iron Foundries 1750-1875' in Brändström, Anders & Sundin, Jan (eds), *Tradition and Transition: Studies in Microdemography and Social Change*, Umeå, pp 105-160.

Suomi, Stephen J (1997), 'Early determinants of behaviour: evidence from primate studies' in Marmot, Michael G & Wadsworth, Michael E J (eds), *Fetal and early childhood environment: long-term health implications*. British Medical Bulletin, Volume 53, Number 1, pp 170-184.

Sylva, Kathy (1997), 'Critical periods in childhood learning' in Marmot, Michael G & Wadsworth, Michael E J (eds), *Fetal and early childhood environment: long-term health implications*. British Medical Bulletin, Volume 53, Number 1, pp 185-197.

United Nations (1953), *The Determinants and Consequences of Population Trends. A Summary of the Findings of Studies on the Relationships between Population Changes and Economic and Social Conditions.* Population Studies No 17, Department of Social Affairs, Population Division, United Nations, New York.

United Nations (1954a), *Foetal, Infant and Early Childhood Mortality. Volume I. The Statistics*. Population Studies No 13:1, Population Division, United Nations, New York.

United Nations (1954b), *Foetal, Infant and Early Childhood Mortality. Volume II. Biological, Social and Economic Factors*. Population Studies No 13:2, Population Division, United Nations, New York.

United Nations (1973), *The Determinants and Consequences of Population Trends. New Summary of the Findings on Interaction of Demographic, Economic and Social Factors.* Volume I. Population Studies No 50, Department of Social Affairs, Population

Division, United Nations, New York.

United Nations (1988), 'Sex differentials in life expectancy and mortality in developed countries: an analysis by age groups and causes of death from recent and historical data'. *Population Bulletin of the United Nations*, 25, pp 65-106.

van der Sande, M A B, Kidd, I M, Gopetghebuer, T, Martynoga, R A. Magnusen, A, Allen, S, Weber, M W, Fielding, K L, Marchant, A & Whittle, H C (2002), 'Severe respiratory syncytial virus infection in early life is associated with increased typ 2 cytokine production in Gambian children'. *Clinical and Experimental Allergy*, 32, pp 1430-1435.

Vaupel, James W (1988), 'Inherited frailty and longevity'. *Demography*, 25, pp 227-287.

Vaupel, James W, Manton, Kenneth G & Stallard, Eric (1979), 'The impact of heterogeneity in individual frailty on the dynamics of mortality'. *Demography*, 16, 439-454.

Venables, W N & Ripley, B D (2001), *Modern Applied Statistics with S-PLUS*. 3rd edition. Springer Verlag, New York.

von Mutius, E (2001), 'Paediatric origins of adult lung disease'. *Thorax*, 56, pp 153-156.

Wadsworth, Michael E J (1997), 'Changing social factors and their long-term implications for health' in Marmot, Michael G & Wadsworth, Michael E J (eds), *Fetal and early childhood environment: long-term health implications*. British Medical Bulletin, Volume 53, Number 1, pp 198-210.

Wadsworth, Michael E J (1999), 'Early Life' in Marmot, Michael and Wilkinson, Richard (eds), *Social Determinants of Health*. Oxford University Press, Oxford, pp 44-63.

Walker, S P, Gaskin, P, Powell, C A, Bennett, F I, Forrester, T E & Grantham-McGregor, S (2001), 'The effects of birth weight and postnatal linear growth retardation on blood pressure at age 11-12 years'. *Journal of Epidemiology and Community Health*, 55, pp 394-398.

Wannamethee, Goya, Whincup, Peter H, Sharper, Gerald & Walker, Mary (1996), 'Influence of father's social class on cardiovascular disease in middle-aged men'. *Lancet*, Volume 348, pp 1259-1263.

Whincup, Peter & Cook, Derek (1997), 'Blood pressure and hypertension', in Kuh, Diane and Ben-Schlomo, Yoav (1997) (eds), *A life Course Approach to Chronic Disease Epidemiology*. Oxford University Press, Oxford, pp 121-141.

Whincup, P H, Bredow, M, Payne, F, Sadler, S & Golding, J (1999), 'Size at birth and blood pressure at 3 years of age. The Avon Longitudianl Study of Pregnancy and Childhood (ALSPAC)'. *American Journal of Epidemiology*, Volume 149, Issue 8, pp 730-739.

Widén, Lars (1975), 'Mortality and Causes of Death in Sweden During the 18th Century'. *Statistisk tidskrift* (Statistical Review), 13, Nr 2, pp 93-104.

Widén, Lars (1976), 'A retrojection back to 1700', in Hofsten, Erland & Lundström, Hans, *Swedish Population History. Main trends from 1750 to 1970.* National Central Bureau of Statistics, Stockholm, pp 169-176.

Young, S, O'Keefee, P T, Arnott, J & Landau, L I (1995), 'Lung function, airway responsiveness and respiratory symptoms before and after bronchoiolitis'. *Archives of Disease in Childhood*, 72, pp 16-24.