LUND UNIVERSITY

Defeating Disease

Lasting Effects of Public Health and Medical Breakthroughs between 1880 and 1945 on Health and Income in Sweden

Lazuka, Volha

2017

Link to publication

Citation for published version (APA):
Lazuka, V. (2017). *Defeating Disease: Lasting Effects of Public Health and Medical Breakthroughs between 1880 and 1945 on Health and Income in Sweden*. [Doctoral Thesis (compilation), Lund University School of Economics and Management, LUSEM].

Total number of authors:
1

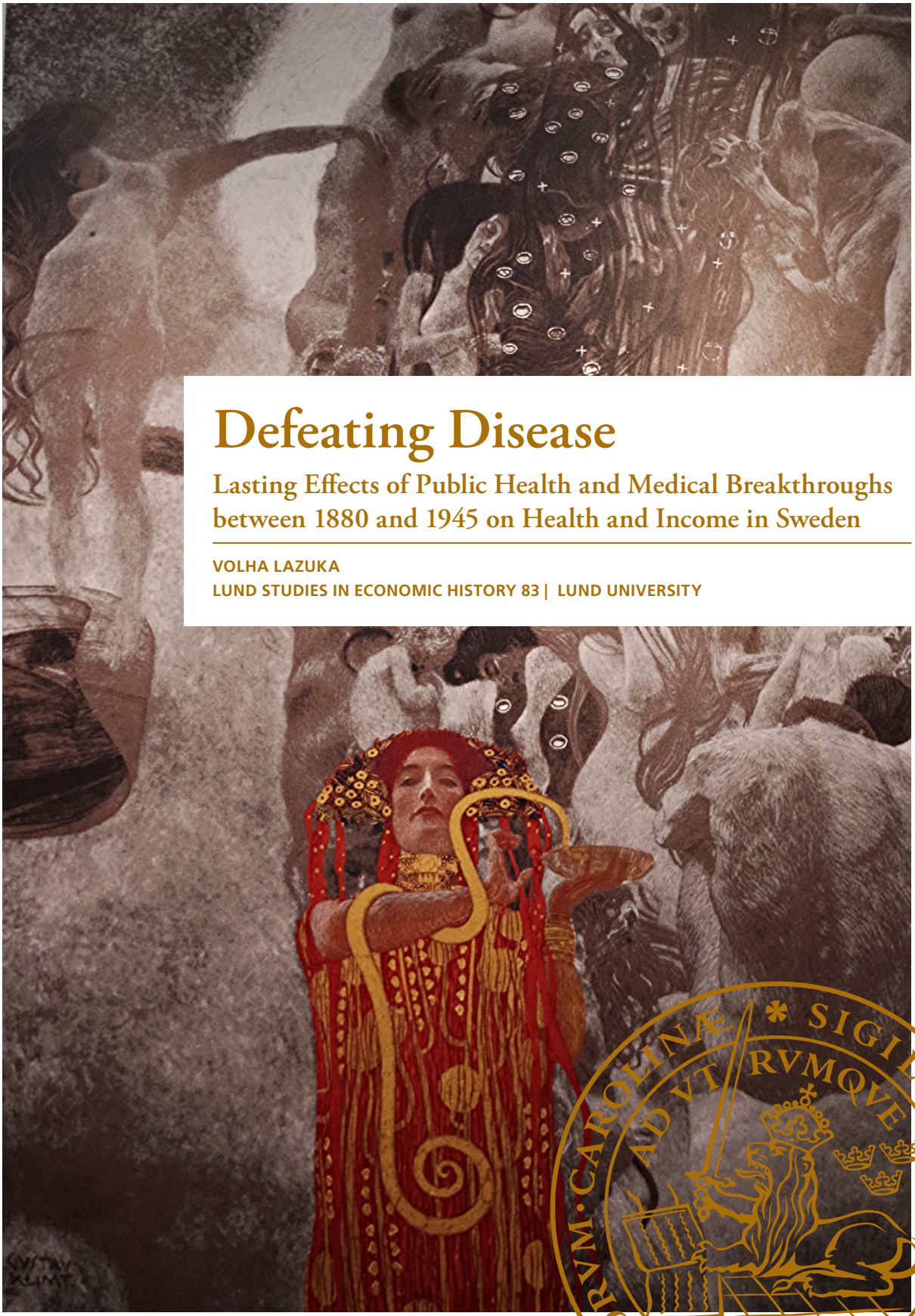
General rights
Unless other specific re-use rights are stated the following general rights apply:
Copyright and moral rights for the publications made accessible in the public portal are retained by the authors and/or other copyright owners and it is a condition of accessing publications that users recognise and abide by the legal requirements associated with these rights.
• Users may download and print one copy of any publication from the public portal for the purpose of private study or research.
• You may not further distribute the material or use it for any profit-making activity or commercial gain
• You may freely distribute the URL identifying the publication in the public portal

Read more about Creative commons licenses: https://creativecommons.org/licenses/

Take down policy
If you believe that this document breaches copyright please contact us providing details, and we will remove access to the work immediately and investigate your claim.

LUND UNIVERSITY

PO Box 117
221 00 Lund
+46 46-222 00 00



Defeating Disease

Lasting Effects of Public Health and Medical Breakthroughs between 1880 and 1945 on Health and Income in Sweden

VOLHA LAZUKA

LUND STUDIES IN ECONOMIC HISTORY 83 | LUND UNIVERSITY

Defeating Disease

Lasting Effects of Public Health and Medical Breakthroughs between 1880 and 1945 on Health and Income in Sweden

Volha Lazuka

LUND
UNIVERSITY

DOCTORAL DISSERTATION
by due permission of the School of Economics and Management,
Lund University, Sweden.
To be defended at EC3:211, November 10 2017 at 10:15.

Faculty opponent
Joseph Ferrie

Organization	Document name
LUND UNIVERSITY	DOCTORAL DISSERTATION
Department of Economic History	Date of issue 06 October 2017
Author Volha Lazuka	Sponsoring organization

Title and subtitle

Defeating Disease: Lasting Effects of Public Health and Medical Breakthroughs between 1880 and 1945 on Health and Income in Sweden

Abstract

This thesis has the general aim of exploring the role of public health and medical interventions between 1880 and 1945, targeting infectious diseases, in health and income improvements in Sweden throughout the twentieth century. It applies methods of causal inference to longitudinal individual-level data from both local and national datasets combined with multisource archival data on the implementation of reforms. The bacteriological discoveries of the latter part of the nineteenth century helped the public to target infectious diseases through isolation, disinfection and treatment. The studies of this thesis show that these societal measures have had a sizable role in the contemporaneous decline in infectious disease rates. In the pre-drug period, public health interventions related to isolation and disinfection substantially reduced infant, child and infectious disease mortality in southern Sweden. In the period after the nationwide introduction of sulpha antibiotics in Sweden, the vast majority of the pneumonia decline was attributable to the antibiotics.

Moreover, these studies find that by reducing the likelihood of infection, societal health measures led to beneficial consequences for the income and long-term health of the affected cohorts, extending through old age. In the long term, the cohorts treated in the early neonatal period by qualified midwifery in southern Sweden obtained strong reductions in all-cause mortality in adulthood and in mortality due to cardiovascular diseases and diabetes in old age. Throughout rural Sweden, similar preventive measures extended the lives and positively affected the lifetime incomes in old ages. The reduction in pneumonia mortality due to the nationwide arrival of sulpha antibiotics boosted the labour income and health of the cohorts that received these antibiotics in infancy. The largest effects on health have been found for hospitalizations due to cardiovascular diseases, diabetes, arthritis and respiratory diseases.

This thesis therefore supports the endogenous growth theory, which posits an interaction between health improvements in early childhood and health technology, based on application of the germ theory of disease, as a determinant of economic growth in the long run. Although the health benefits due to this dynamic process cannot be viewed as a main driver of economic growth, societal health investments nonetheless led to income growth sufficient to yield a high rate of return on the investment. The cause – that is, the reforms under study that are targeting infectious disease – and the consequence, reductions in the rates of specific chronic diseases in adulthood and old age, point to the inflammation mechanisms behind the lasting effects. The studies also suggest that the early neonatal period and infancy are the critical period for intervening such processes.

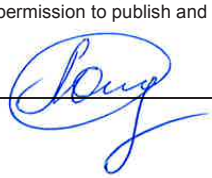
Key words public health, antibiotics, inflammation, early life, health, income, end of the 19th–20th century, Sweden

Classification system and/or index terms I1 J1 J2 N3

Supplementary bibliographical information	Language English
ISSN and key title 1400-4860	ISBN 978-91-87793-38-7

Recipient's notes	Number of pages 363	Price
	Security classification	

I, the undersigned, being the copyright owner of the abstract of the above-mentioned dissertation, hereby grant to all reference sources permission to publish and disseminate the abstract of the above-mentioned dissertation.

Signature  Date 2017-10-06

Defeating Disease

Lasting Effects of Public Health and Medical Breakthroughs between 1880 and 1945 on Health and Income in Sweden

Volha Lazuka

LUND
UNIVERSITY

Lund Studies in Economic History is a series of doctoral dissertations and edited volumes of high scholarly quality in subjects related to the Department of Economic History at the School of Economics and Management, Lund University. All volumes have been reviewed and approved prior to publication.

Coverphoto by Gustav Klimt, 'Medicine' Faculty painting 1901

Copyright Volha Lazuka

School of Economics and Management
Department of Economic History

ISBN 978-91-87793-38-7 (print)
ISBN 978-91-87793-39-4 (pdf)
ISSN 1400-4860

Printed in Sweden by Media-Tryck, Lund University, Lund 2017

To the memory of my father

Content

Aknowledgement....9

List of papers....11

Introduction....13

- Motivation and aim of the thesis....13
- Secular mortality decline: a continuing debate about the determinants....20
- Health as a determinant of economic growth: economic growth theories and empirical findings....29
- Early-life determinants of adult health and labour income....37
  - Epidemiological hypotheses on the lasting importance of early-life environment for adult health....37
  - Economic theoretical models linking early-life environment to adult health and labour income....40
  - Empirical evidence for the early-life hypotheses....44
- Sweden as a laboratory of health and economic development....49
  - Long-run development of health and income....49
  - Provision of public health and antibiotics....53
- Parishes in Scania as a rural representative....56
  - Socio-economic and health development in Scania....56
  - Local government and public health in Scania....59
- Methods....61
  - Quasi-experimental methods used in the literature....61
  - Methods used in the thesis....63
- Data....64
  - Requirements to the data....64
  - Data used in the thesis....65
  - Limitations....69

Results 71
Summary of the papers and the sizes of the effects 71
Social rate of return 77
Discussion and Conclusions 82
On the causality between economic growth and health development 82
Conclusions 85
References 89
Papers I–IV
Appendix

Acknowledgement

This dissertation could not be written without the advice and help of my supervisors. I would like to give my largest and warmest thanks to my main supervisor, Professor Tommy Bengtsson, for his valuable advice, deep intuition, dedication to research, perspective vision and limitless knowledge. Dear Tommy, during these years you became an ideal role model for me as a supervisor, teacher, researcher and a leader of the scientific community. It makes me gifted if I was able to inherit your features even partially, but I am already gifted to become and be your student. I am also cordially grateful to my assistant supervisor, Luciana Quaranta, for the neediest advice, long support and invitation to collaborative research in the future. Many thanks to both of you for personal and professional engagement! Your guidance and cumulative knowledge, to that many preceding years of work at the Department of Economic History and the Centre for Economic Demography in Lund University contributed, helped this thesis realize.

I am also thankful to Martin Lindström for the valuable advice, our discussions of my potential papers and the constant readiness to help. During these years, Anton Nilsson provided me with multiple useful advices for all papers of my dissertation, and thanks to you for this as well as for our friendship and invitation to future research projects. I would also like to thank the persons who were helpful at the initial stages of this dissertation and who forced to always think broadly about contribution of the doctoral research, Martin Dribe and Christer Gunnarsson. Many thanks to Therese Nilsson and Patrick Svensson who were ready to help with any question. I thank a lot the commentators at the final seminar for helpful suggestions as well as for support beyond the seminar, Anders Nilsson, Petter Lundborg, Jonas Helgertz, Anton Nilsson and Astrid Kander. I appreciate and additionally thank Anders Nilsson for generous support. I would like to thankfully acknowledge Martin Dribe, Kirk Scott and Tommy Bengtsson as heads of the Centre for Economic Demography in Lund University, who provided me with a supportive research environment.

The years of doctoral work gave me invaluable friends. I would like to thank my officemate and a friend, Annika Elwert, for your kind heart, generous help and fruitful discussions of my papers. I am thankful for support from Zeyuan Chen, who will always stay in my heart. Thanks to Jens Andersson for our friendly collaboration and that this makes a difference. Special thanks to Sofia Henriques for our joint lunches and friendly talks. I met many other students during these

years at the Department of Economic History, some of them have already become post-doctoral fellows, and the time spent with you and the journey of developing together have been great: Jason Lennard, Sean Kenny, Finn Hedefalk, Kathryn Gary, Hana Nielsen, Joseph Molitoris, Jeff Neilson, Thor Berger, Omar Karlsson, Viktoras Kulionis, Montserrat Lopez Jerez, Siddartha Aradhya, Andrés Palacio, Andy Qi, Anna Tegunimataka, Björn Eriksson, Josef Taalbi and many others. I am grateful to the head of the Department of Economic History, Mats Olsson for creating a productive working atmosphere and for personal support, and to Fay Lundh Nilsson for helping with finding teaching hours. Many thanks to the administrative staff at the Department, Anneli Nilsson Ahlm, Madeleine Jarl, Kristin Fransson, Birgit Olsson, and Tina Wueggertz. I am also thankful to Clas Andersson at the Centre, to Solveig Fagerlund at the Regional Archive in Lund, and to students Federica Braccioli and Siqi Zhao for help with the data.

And finally, I give my warmest thanks to my family. Мама, сёстры Танечка и Юля, подруга Аня, спасибо вам за вашу помощь эти четыре года и понимание. Дорогой Саша, муж мой, спасибо тебе за твою поддержку и любовь!

Lund, 01 October 2017

Olga

List of papers

I Lazuka Volha, Luciana Quaranta and Tommy Bengtsson (2016). Fighting infectious disease: Evidence from Sweden 1870–1940. *Population and Development Review* 42 (1): 27–52.

II Lazuka Volha. The long-term health benefits of receiving treatment from qualified midwives at birth. *Unpublished manuscript.*

III Lazuka Volha. The long-term health and income effects of early public health formation in Sweden. *Unpublished manuscript.*

IV Lazuka Volha. Infant health and later-life labour market outcomes: Evidence from the introduction of sulpha antibiotics in Sweden. *Unpublished manuscript.*

Introduction

Motivation and aim of the thesis

In 1901, Gustav Klimt's painting 'Medicine' appeared at an exhibition in Vienna. A newborn infant, representing life, enters the river of life with the help of the mother. The river of life is flowing and a human passes through the stages of life ending with illness and death. Hygieia, the goddess of prevention of sickness and the continuation of good health, stands at the estuary with the Aesculapian snake and the cup of Lethe. Powerful and oracular, she is turning her back to humankind. Klimt proclaimed the unity of life and death, human fragility, and estrangement of medicine. Despite the high artistic value, the painting came under severe attack from critics of both scientists and the state for misunderstanding of the current contribution of preventive medicine to population health (Fliedl 1998). Like any representative of the upper class, Klimt could not anticipate and value the changes already ongoing in society with regard to the fight for human life and the progress of such a battle. Neither could he foresee that in a few decades, scientists would find a 'miracle' drug against infectious disease, cheap and efficient, which would be able to save countless lives.

In developed countries, life expectancy at birth had already started to improve in the eighteenth century, and beginning from the mid-nineteenth century onward it rose remarkably, with a constant pace of almost 3 months annually (Oeppen and Vaupel 2002). Currently half of all deaths occur after age 80. In these improvements, a rapid decline in both infant and child mortality until around the mid-twentieth-century, predominantly in mortality due to infectious diseases, played the major role. The period afterwards witnessed a more evenly distributed mortality decline across age groups, both due to infectious and chronic diseases, the latter most profoundly since the 1970s. Between 1820 and 2010, real income per capita in Western Europe and the US rose by a factor of more than eighteen (The Maddison-Project 2013). By disentangling the mode of transmission of infectious diseases and the causal agents, international bacteriological discoveries in the 1870s–1880s provided the public with the tools to defeat disease, which were widely implemented. Across developed countries, the infant and child mortality decline that followed could be linked to technology of disease control (Easterlin 1996; Mokyr 2002). The first step in implementing such technology

occurred in the pre-drug period, the 1880s–1930s, and included measures focused on stopping the spread of infectious diseases between communities and individuals by various tools, such as establishment of isolation hospitals, disease monitoring, antiseptic techniques, sanitation measures, and water supply and sewage improvements. The next step, which occurred in the late 1930s–1940s, was driven by the introduction of new drugs for treatment of disease after infection which, as a consequence, led to reduced rates of contagion. It began with the invention and nationwide implementation into medical practice of antibacterial sulpha drugs, which were able to block the development of certain infectious diseases, and later on included penicillin, which killed all known infectious diseases. Other measures included chemical control and immunisations.

Among developed countries, Sweden represents these developments vividly. From 1751 until 2014, life expectancy at birth has risen from 38 to 82 years, among the longest worldwide (Colchero et al. 2016; Human Mortality Database 2017). In the beginning of the nineteenth century, surviving through infancy and childhood was hard compared to the rest of life, even though infant mortality began to fall. Between the 1880s and the 1950s, both infant mortality and mortality for children under age 15 improved at a higher rate than did mortality at other ages. Figure 1 displays changes in life expectancy at different ages: life at birth is increasing much faster than that at age 15 beginning in the 1880s. Between 1850 and 2014, real income per capita increased more than twentyfold (Schön and Krantz 2012; Statistics Sweden 2017). Closely following the achievements of international knowledge and legislation in disease control, Sweden early on adopted measures to fight disease (Niemi 2007). It started with the establishment of local health boards according to the comprehensive Public Health Act 1874, followed by isolation efforts, improved midwifery, water supply and sewage installations and then provision of easy access to antibiotics and later on to vaccines. Public health policies launched in the 1880s in Sweden had a strong emphasis on disease control in the countryside, due to the rural and semi-urban location of the industrialization. Throughout the first half of the twentieth century, regional equality in state-provided health care systems became assured, and Sweden outcompeted other developed countries in terms of public health investment and medical specialization (Hollingsworth, Hage, and Hanneman 1990). Together with monitoring of scientific advances in medical technology, in the late 1930s, such systems inevitably contributed to the cheap provision of antimicrobial drugs to the general public. The most prominent success has been attributed to the propagation of newly invented sulpha antibiotics, as in other Western countries. The sulphonamides appeared to be very efficient against acute pneumococcal pneumonia, the main cause of child deaths at that time, and until the introduction of penicillin in the late 1940s in Sweden remained the primary treatment.

Figure 1 – Life expectancy at birth (e0) and at age 15 (e15) for Sweden, total population
Source: constructed based on data from the Human Mortality Database (2017). Series are smoothed with Hodrick-Prescott filter.

Improvements in health technology enabled the society to escape premature death not only in the short run but also in the long run, and in addition enhance economic growth. Fogel and Costa (1997) and Fogel (2004) famously argue that in the last few centuries countries became healthier and richer because health advances in childhood had translated into higher economic performance in adulthood, leading to better health conditions and higher performance by later generations. To these scholars, health advances were driven by a synergism between technological and physiological improvements; that is the idea which is central to the current thesis. With regard to physiological improvements, it embeds the concept of the so-called developmental origins of disease, according to which environmental conditions in early childhood, through various biological mechanisms, ensure health and productivity throughout life. Formulating a nutritional hypothesis, Fogel and Costa (1997) and Fogel (2004) suggest that among the array of environmental conditions, such as exposure to infectious diseases, malnutrition and other types of biological and socioeconomic stress, advances in nutrition due to advances in food technology and, more broadly, living standards were driving health advances in long run. However, even Fogel calculated that for the first three quarters of the twentieth century roughly half of all changes in mortality are unrelated to improved nutrition, and should be attributed to medical innovations, among other factors (Fogel and Costa 1997).

The proponents of the institutional or knowledge hypothesis, Easterlin (1999, 2004) and Mokyr (2002), suggest that the discovery of the germ theory of disease and its implementation through public health measures was primarily responsible for the escape from premature death, independently of economic growth. The germ theory of disease, whose validity was clear circa 1870–1880, is thus analogous with General Purpose Technology, which is a production technological breakthrough with the potential to alter the societies (Easterlin 2004). Easterlin departs from the findings of Preston (1975), who in a macro-level investigation shows that the expected increase in life expectancy at birth on the assumption of no growth accounts for almost 90% of what actually occurred. Rather speculatively, this fraction can be attributed to the empirical validation of the germ theory of disease. Acceptance of the germ theory of disease has supposedly led to gains in life expectancy which are largely inexpensive, presumably pointing to the importance of knowledge versus income growth in accounting for secular mortality decline (Easterlin 2004; Soares 2007). Finch and Crimmins (2004) and Crimmins and Finch (2006) have recently brought up that the historical decline in exposure to infectious diseases and inflammation, complementarily to nutrition, contributed substantially to the decline in old-age mortality across the developed countries. It thus becomes tempting to establish the extent to which intergenerational improvements in health and income are explained by successes in defeating infectious disease. Unlike the nutritional explanation, the germ theory of disease can account for the reciprocal relationship between health and economic growth.

Despite the clarity of the arguments, to date, empirical literature showing that public health and medical innovations that applied the germ theory of disease contributed to the decline in infectious disease mortality is rare. There is also not a great deal of evidence that cohorts born in and surrounded by better infectious disease conditions as a result of these interventions carry on an advantage in health and income in the long run. The empirical literature is developing in certain directions, with no coherency in investigating the topic under study. Our knowledge about the lasting impact of early-life conditions on health is principally built on studies using negative disease insults as quasi experiments. They elucidate the fact that rate of exposure to disease is a prime mechanism behind the lasting effects. The epidemiological literature, by relying on recent cohorts, brings specificity to this mechanism. However, the clearest evidence regarding the causality and public health importance of early-life factors comes from studies that first show that public health interventions modify early-life conditions and child health and then follow the cohorts in their outcomes throughout the life course, from birth until old age. The focus on both health and income outcomes as determined by childhood health is probably the only way to show a causal link between health and income and income inequalities. In order to be able to draw proper policy implications, several methodological aspects are important here: the

knowledge and identification of an exact developmental period, as well as when the interventions occurred and are most efficient, knowledge of the components of the intervention, and a careful evaluation to identify whether early infectious exposures are independent of other early life exposures. Not only there is no empirical work examining details of early-life treatments and later-life outcomes, but it is also unknown how policy initiatives targeting the disease environment in the period of rapid decline in infant mortality affect health and income later in life; this thesis intends to address these important issues.

The general aim of this thesis is to explore the role of public health and medical breakthroughs guided by the germ theory of disease and enabling to target infectious diseases in health and economic improvements in Sweden since 1880. The interventions under study include those isolating disease – establishment of isolation hospitals and employment of qualified midwifery and disease monitoring – and those treating disease – introduction of the first antibiotics. To carefully identify processes linking early conditions to health and income later in life over such a long period, an investigation has to be made at the level of the individual, and this thesis applies this perspective. By using the methods of causal inference, its ambition is to disentangle a contribution of reduction in exposure to disease that is independent of the improvements in nutrition and living standards. Its hypothesis is that reduction in exposure to infectious disease and its treatment became the mechanism through which public action, guided by knowledge of disease transmission and causal agents, translated into better health, longer survival, and higher incomes. In the short term, this mechanism is supposed to have operated through reduction in infectious disease mortality and better child health. Longer-term, consistent with the infancy inflammation hypothesis, originated from the study by Bengtsson and Lindström (2003), reduced exposure to infectious diseases during individuals' infancy supposedly led to better health and higher incomes in their adulthood. To achieve its objective, this study relies on high-quality register-based longitudinal individual-level data, both local, following individuals from birth until old age, and national, following individuals throughout adulthood and old age with information linked to their date and region of birth. The data on treatment by health interventions is purposely-collected and comes from multiple archival and statistical sources. The estimation of benefits and the availability of precise information about the costs of the reform allows for cost-benefit analysis of these interventions.

The following specific research questions are addressed in this thesis:

(1) To what extent were public health and medical innovations which applied the germ theory of disease responsible for a decrease in mortality due to infectious diseases in Sweden?

(2) Did these interventions, through improvements in disease environmental conditions, generate health advantages among the affected cohorts throughout the life course until death? How large is their contribution to the overall gain in life expectancy and to the decline in chronic diseases in Sweden?

(3) Did these interventions generate advantages in labour incomes? How large is their contribution to economic growth in Sweden?

(4) What is the social rate of return of these interventions, with regard to their short- and long-term outcomes?

Additionally, this thesis examines (5) the relative extent to which isolation of infectious disease versus its treatment produces beneficial health and income consequences in long run. To the extent that both are showed to be important, it will demonstrate that long-term health and economic improvements involve interventions with the same origin – the application of the germ theory of disease. Another integrating ground is that both implementation of measures to isolate infectious disease near the turn of the century and implementation of highly effective treatments during the middle twentieth century had a sizable impact on infectious disease mortality, resulting in rapid decline throughout this period. It points to inflammation in early life as a process leading to the incidence of chronic diseases at older ages.

This thesis could give rise to policy implications for contemporary populations, at least along two dimensions. First, this thesis not only provides an account of the contribution of earlier public initiatives, not specifically targeting infant health but affecting it through reductions in infectious disease rates, to long-term health and income development, and also points to unanticipated benefits of modern targeted programs. Already by the turn of the twentieth century, Sweden, among few countries worldwide, had instituted a comprehensive set of policies including universal maternal and infant health services (SOU 1994; Allender, Cherie, and Warner 2013). Today, these services comprise many components, explored in this thesis, such as preventative health care, monitoring of health development, disease treatment, breastfeeding, and parenting support. Both overall high health care spending and specific programs to strengthen midwifery contribute to infant health. Currently, chronic disease, and cardiovascular disease in particular, is responsible for the majority of deaths in developed countries and globally, and treatment is extremely costly. This thesis emphasizes the causal link between public health programs for infants and long-term reductions in health care costs for treatment of chronic diseases. Second, the evidence provides motivation for increased attention by authorities to infectious disease prevention today. Not only is this thesis highly relevant for middle- and low-income countries, where the majority of child deaths occur due to pneumonia, diarrhoea and health problems during the first month of life which could be prevented or treated with access to affordable interventions, but it is also relevant for high-income countries where pneumonia infection currently represents a leading cause of child morbidity

(Rudan et al. 2013). While antibiotics are indisputably important for child survival, growing antimicrobial resistance raises new challenges to public action, including the intensified use of non-drug initiatives. Internationally, the emergence of new contagious diseases should be dealt with through isolation methods first, before vaccines and drugs are invented. This thesis highlights the fact that improvements in disease conditions in early life with publicly provided medications and interventions are essential not only in reducing the current burden of disease for children, but they are also important in the long run – in promoting life expectancy and income growth and in leading to convergence in health and economic disparities. The condition that should be met is the existence of local administration guided by scientific knowledge and prepared to take timely action in implementing such measures.

The structure of the thesis is as follows. It starts with the introductory chapter (Chapter 1) that presents the common theoretical framework of the papers. It presents the facts about health and income development, discusses the theoretical foundations linking health in early life to later health and income, summarizes the related empirical literature, presents the data and the methods used to collect and analyze it, and positions the results of the papers within the empirical literature. The main body of the thesis is divided into four independent research articles. Paper 1 (Chapter 2) investigates the instantaneous impact of public health interventions in the pre-drug period on infant mortality and mortality under age 15 for Scania. Paper 2 (Chapter 3) explores the long-term effects of access to qualified midwives at birth and in the neonatal period on an individual's morbidity, mortality and skills, for cohorts born 1881–1930 and followed from birth until age 80, in Scania. Turning to rural areas in Sweden, Paper 3 (Chapter 4) studies the impact of the establishment of health districts in 1890–1917 to enhance care during infancy on outcomes from age 78 until death, including both all-cause and cause-specific mortality and pension incomes. For the whole of Sweden, Paper 4 (Chapter 5) investigates the influence of the reduction in pneumonia infection in infancy due to the arrival of sulpha antibiotics on the education, income, and long-term health of individuals in ages 34–60 who were born between 1934 and 1943. The thesis ends with the paper describing and assessing the quality of the data from the midwifery journals used in Paper 2 of the thesis.

Secular mortality decline: a continuing debate about the determinants

This section first presents the features of the mortality decline and then reviews the explanations proposed for the mortality decline and the related empirical literature. The discussion is limited to the explanations for mortality decline that emphasize factors affecting the entire population at the same time regardless of age.

In the Nordic countries, long-term health improvements are marked by universal regularities. In Sweden, Denmark, Norway, and Finland, which have reliable historical data spanning far back in time, mortality had already begun to decline in the eighteenth century (Fridlizius 1984; Perrenoud 1984). Bubonic plague disappeared, incidence of typhus and smallpox declined substantially in severity and frequency, and wars and harvest failures became less disruptive. When long-term mortality decline commenced, it was primarily characterized by a fall in infant and child mortality (Bengtsson and Ohlsson 1994; Bengtsson 2015). Through the mid-nineteenth century, the incidence of various communicable diseases, such as smallpox, scarlet fever, typhoid fever, diphtheria, and whooping cough, was reduced, which translated into lower death rates among children and, to a lesser extent, among young adults (Bengtsson 2015). In the Nordic countries, infant mortality had been uninterruptedly falling throughout the nineteenth century (Perrenoud 1984). Rates of infant mortality and even more so childhood mortality accelerated in decline between 1880 and 1950, whereas mortality in older age groups declined much less, but still significantly. In this period, mortality was reduced the most for tuberculosis, bronchitis, pneumonia, influenza, other respiratory diseases, diarrhoea and enteritis (Kunitz 1986; Schofield and Reher 2002). A final regularity, which began circa 1970 and continues to the present, is the fall in old-age mortality, which gradually began to be responsible for the majority of deaths. It is attributed to reductions in non-communicable diseases, such as cardiovascular diseases and some cancers (Omran 1998). The pattern of mortality development for Western countries (Schofield and Reher 2002), which is based on data from reconstructions of national samples of parish data, and North America (Kunitz 1984; Smith and Bradshaw 2006), which is based on interpolated census data, is generally similar, except for stagnating infant mortality instead of the continuous fall throughout the nineteenth century.

As a result of declining mortality, life expectancy increased substantially. Life expectancy is the preferred mortality measure, which is considered throughout the introductory chapter, because it allows for international comparisons and eliminates the influence of the age structure of the population. Figure 2 plots female life expectancy at birth, which is less affected by war losses and out-migration than male life expectancy, for the Nordic countries. In Sweden, up until the beginning of the nineteenth century, female life expectancy at birth improved

from 38 to 40 years. Across the Nordic countries, female life expectancy rose to more than 45 years by 1880, to around 70 years by 1950 and to 80 years by the 2000s. In the best performing countries, throughout the period of health development, life expectancy at birth for females rose, at a fairly steady rate, by 2.92 months per annum and slightly less than that for males (Oeppen and Vaupel 2002). The regularities presented above fit the mortality pattern in Europe and North America, while over the same time frame mortality dropped at an even higher rate in other regions (Omran 1971, 1998). Within the former countries, throughout the nineteenth century, the continuous decline of infectious diseases was a rural phenomenon (Kunitz 1986), whereas urbanized locations experienced relative stagnation in population health (Schofield and Reher 2002). Health indicators, including adult height, BMI, morbidity (Costa and Steckel 1997) and intelligence (Weil 2013), show improvements in parallel to declines in infectious disease mortality.

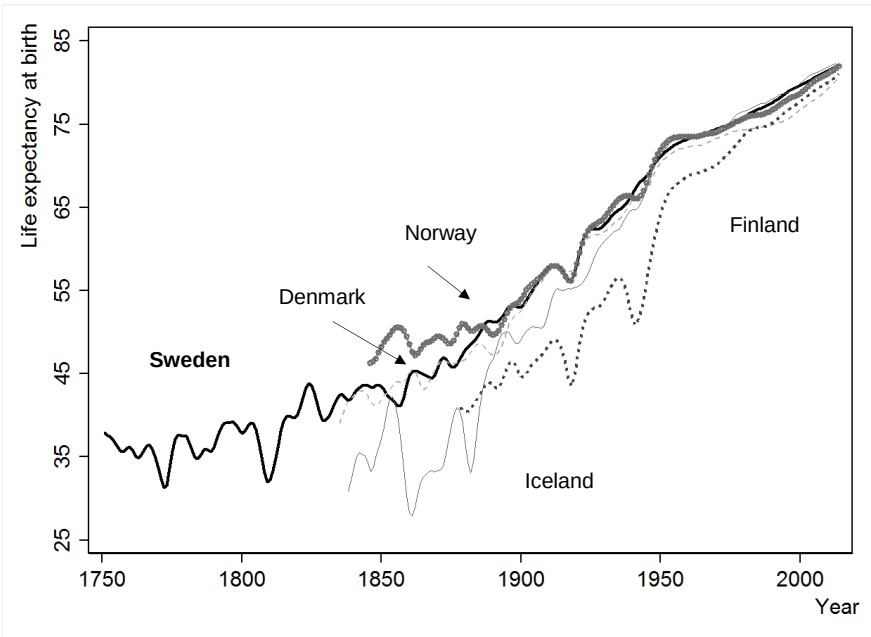


Figure 2 – Life expectancy at birth (female) in the Nordic countries 1751–2014
Source: constructed based on data from Human Mortality Database (2017). Series are smoothed with Hodrick-Prescott filter.

The hypothesis that advances in nutrition were the most prominent factor in reducing secular mortality dominates the works of McKeown (1976, 1980). These works focus on England and Wales in 1850–1971, where secular, that is prolonged as opposed to temporary, mortality decline commenced. Mortality decline in a subset of other European countries was also examined. By decomposing mortality decline by cause of death, McKeown and colleagues calculated that the decline in death rate between 1850 and 1971 is primarily explained by the decline in infectious diseases, among which more than half are airborne diseases (mainly tuberculosis), a third are water- and foodborne disease, and the rest are spread by other means (McKeown, Brown, and Record 1972; McKeown 1976). He further speculated regarding what tools were available in the society to fight specific infectious diseases and their efficiency, based on calculations of fractions in mortality decline attributable to these tools. Isolation from a community or prevention from overcrowding did not play any role in the case of measles and scarlet fever and was secondary in case of tuberculosis, whooping cough, diphtheria and smallpox. The contribution of reduced exposure as a primary influence, in his opinion, was only in control of water- and foodborne diseases, such as the role of improvements in water sanitation and sewage disposal in the decline of enteric and diarrhoeal diseases, a safe milk supply in reduced rates of tuberculosis, and improved personal hygiene in reduced rates of typhus (McKeown 1976, pp.126–127). A spontaneous diminution of the virulence of infectious disease as a prime cause has been downplayed as well: it accounted for the decline of mortality from scarlet fever and may have contributed to that from typhus and cholera, but not significantly to the decline in tuberculosis mortality. Among treatment measures, only treatment of diphtheria with antitoxin circa 1900 could be justified as a credible factor, because its implementation coincided with the drop in death rates due to this disease. Coming as a residual explanation in 'The Modern Rise of Population', a rising standard of living that led to improvements in nutrition, due to greater food supplies, and as a result enhanced resistance to disease emerged as a necessary condition for the prolonged mortality decline. One half of the total reduction in mortality is attributed to nutritional advances, exactly what is left unexplained by other causes.

In the later work, 'The Role of Medicine: Dream, Mirage or Nemesis?', McKeown (1980) completely discounted the role of medical knowledge and medical practitioners in the onset of health advances. His opinion about the contribution of preventive measures in mortality decline is again repeated in this work: while it is relatively easy to control water-borne diseases, and public sewage and water supply initiatives could be important in their reduction, it is usually impossible, and thus of secondary importance, to prevent transmission of airborne infections from one individual to another. In this work, similar to the empirical approach as in the previous works, he further evaluates medical achievements in relation to prevention and treatment of infectious and non-infectious diseases by

matching the timing of the mortality decline and introduction of the measure and assessing its contribution. Undoubtedly, implementation of effective chemotherapy and immunizations correspond to the decline or acceleration in decline in all infectious diseases. For instance, the reduction in death rate due to bacterial pneumonia more than doubled after the introduction of sulphonamides in the late 1930s, 'casting no doubt on its effectiveness'. However, mortality from pneumonia, bronchitis and influenza was falling continuously from circa 1900; by 1971 it had fallen by more than two thirds and only up to one third can be attributed to the use of antibiotics. Similarly, mortality from common infections declined to quite a low level before specific medical tools became available. In case of chronic diseases, medical interventions have been helpful for the relatively uncommon types. McKeown is thus affirming that '… on the one hand medical science has already achieved miracles … and on the other hand an exact evaluation of twentieth-century medicine would do much to restore nineteenth-century faith in prayer' (p.176). As before, the work concludes with the only remaining possibility, that mortality declined primarily because the response to infections was modified by improved nutrition.

Providing counterevidence to the nutritional explanation of the secular mortality decline, several scholars brought in changes in the pathogens favourable to humans as a leading hypothesis. Wrigley and Schofield (1981) reconstruct English population data from parish records for 1541–1871 and focus on the upward trend of life expectancy at birth beginning from the 1770s. Visual inspection of trends indicates no pattern linking life expectancy at birth and mortality with real wages. Such an association exists only in the short term: a regression analysis suggests that short-term variation in economic variables, such as wheat prices, accounts for not more than one sixth of the variance in mortality and is positively associated (Lee 1981). Wrigley and Schofield (1981) argue that the mortality changes were thus independent of the economic system and, in particular, of living standards. To these scholars, the dominant factor in mortality reductions is the balance between the population and microorganisms, developed from sporadic and lethal to endemic and less lethal, which is 'outside the consciousness of men and … quite outside their power of influence' (p.416). Similar conclusions based on the same empirical approach were drawn in the investigation of mortality patterns for Sweden from 1751–1914 based on a stratified parish sample, grouped according to geographical and socio-economic structure (Fridlizius 1984). Fridlizius shows that the onsets of increases in real wages, introduction of public health and medical measures as well as climate changes did not coincide with the timing of mortality changes. Based on the residual argument, he argues that a dominant role in mortality decline should be given to a changed relation between the various infectious diseases and the population in its favour, either due to a change in virulence or mutation of the disease. Perrenoud (1984) arrives at a similar conclusion in a comparison of Swedish experience with those of Norway and

France, and so does Schofield (1984) in a comparison between Sweden, France and England.

In exploring the issue for different European countries in 1350–1900, Livi-Bacci (1991) in 'Population and Nutrition: An Essay on European Demographic History' strongly downplays the long-term importance of nutrition. The novelty of his analysis is disaggregation of life expectancy at birth by social class; social classes differ with regard to malnutrition just as they do for other material conditions. At least for the case of England from the sixteenth to early eighteenth centuries, his analysis demonstrates no advantage for the privileged classes compared to the total population as a result of dietary differences, raising doubt as to the primacy of nutrition. Based on theoretical assertions, Livi-Bacci puts emphasis on the disease-population balance, which is, in his opinion, not strictly exogenous. It is partially influenced by demographic and economic factors such as population density, urban expansion and mobility. This is an argument most convincingly elaborated in the discussion of global population history as a coherent phenomenon from the Palaeolithic to modern times by McNeill (1989) in 'Plagues and Peoples'. In a narrative with references to the rich contextual information and empirical data from secondary sources, McNeill connects the development of common infectious diseases, such as bubonic plague, smallpox, measles, tuberculosis, diphtheria, cholera and influenza, to population distributions and movements. As he argues, while it took 120–150 years for the population to stabilize in response to the new infection, other important endogenous factors of mortality decline operated, including improvements in agriculture, organization of states and their public health efforts. Even though McNeill mentions nutrition as a determinant, he gives much more credit to protection from disease. For instance, he highlights the importance of quarantining and disinfecting measures to prevent the spread of cholera beginning from the 1880s in developed countries.

Based on inspection of mortality trends in Europe and North America during the eighteenth and nineteenth centuries and in Latin America in the twentieth century, Kunitz (1984, 1986) asserts that both exposure and resistance to disease contributed to the mortality decline. Examining the major infectious diseases chronologically, Kunitz believes that infectious diseases are all 'product of social systems and that civilized societies had much reduced the spectre of death', and that the means of control differed by time and place (p.299). In the seventeenth and eighteenth centuries, establishment of stable governments, control of state borders, discipline in the armies and enforced sanitary regulations brought epidemics of plague, typhus and smallpox under control. With the growth of population and mobility in the nineteenth century, pathogens began to manifest themselves in endemic diseases, such as tuberculosis, pneumonia, and diarrhoea. These infectious diseases are beyond control by administrative means, and here long-term improvements in the standard of living helped to eliminate them. Kunitz avoids mentioning nutrition only, invoking such terms as 'economic development'

or ‘wealth’ and adding other aspects of standards of living, such as sterilization of milk, public health and fluid therapy. Efficient preventive or therapeutic treatment at a medical practitioner level became available only in the 1940s and cannot have had a measurable impact on the mortality decline prior (Kunitz 2002). In these studies, Kunitz is concerned that the relative contribution to mortality decline of reduced exposure and increased recovery from disease, both endogenous to population, is difficult to ascertain in practice.

According to Szreter (1988), social intervention, not nutrition, was a critical determinant of mortality decline in 1848–1914. Based on his examination of the case of England and Wales, Szreter criticizes McKeown for his residual approach to identification of the main determinants of mortality decline and ignorance of discontinuities in development of infectious disease mortality. He proposes to analyze the mortality decline from common infectious diseases separately between urban and rural areas, and indicates that the fall in mortality was continuous in rural areas, whereas residents of urban areas experienced an increase in mortality due to food- and waterborne as well as airborne diseases. Szreter proposes that various public health measures provide effective protection against area-specific mortality from disease, and include water supply and sewage installations and sanitation measures against cholera and typhoid, the effective national system of notification against scarlet fever, cholera and smallpox, and expansion of local health and maternity services, such as qualified midwives, quarantining and isolation, and personal hygiene against different airborne diseases. Such area- and cause-specific inspection of trends rather suggests that ‘the invisible hand of rising living standards, conceived as an impersonal and ultimately inevitable by-product of general economic growth, no longer takes the leading role as historical guarantor of the nation’s mortality decline’ (p.35). More recently, Szreter (2003, 2004) highlighted the importance of vital registration and effective social security systems in the sixteenth and seventeenth centuries and of the system of local governments in the nineteenth century for mortality decline. Unfortunately, despite the detailed argumentation, Szreter (1988, 2004) does not provide a quantitative account of the role of public health measures in mortality decline. Mercer (1990) more explicitly matches the onset of mortality decreases in specific diseases to particular public initiatives, but does not address their quantitative contribution.

Preston (1975, 1996) not only stresses the leading role of public health in reducing mortality, but also explicitly links it to a key scientific breakthrough – the germ theory of disease. Preston’s seminal contribution is a thorough empirical analysis of the associations between life expectancy at birth and national income per capita across different countries from the 1900s–1960s. He finds that current level of income accounts for only 10–25% of the rise in life expectancy at birth between the 1930s and 1960s. Increased living standards, nutrition and literacy do not explain the residual. Instead, 75–90% of the relationship is explained by the factors exogenous to a country’s level of income. This fraction, as Preston (1975)

argues, is attributed primarily to the public health efforts stimulated by the germ theory of disease. More specifically, while antimicrobial drugs were not available until after 1935, the germ theory of disease stimulated other important public health efforts, such as improved antiseptic practices, quarantining, isolation of infectious patients, food and water supply improvements, personal hygiene and improved infant feeding. According to Preston, preventive and supportive medicine in its traditional form was not helpful. In empirical work about child mortality using the 1900 US census, Preston and Haines (1991) conclude that broader geographical variables, such as size and region of residence and state income are its most important correlates, all linked to exposure to disease, but not family socio-economic characteristics. In one of the chapters they compare the experience of the US and England and Wales and demonstrate that in 1895–1905 the children of physicians and teachers had mortality similar to the average, while by 1925 it dropped to a third below the average (Ch.5). It emphasizes a close relation between dissemination of information about scientific advances, such as public officials' awareness of the germ theory of disease, and child mortality. Tomes (1998) for the US and Mooney (2015) for England provide detailed narratives of public health interventions focused on the mitigation of exposure to disease and implemented under the guidance of the new scientific knowledge. Both works highlight the potential importance of preventive measures taken by the authorities against infectious diseases in the late nineteenth century, such as surveillance of infectious disease, including notifications, analysis and the design of the targeted public health measures, disinfection of the birthing environment by the birth attendants, of the people, their belongings and apartments, and isolation of pathologically dangerous patients in isolation hospitals.

Easterlin (1999, 2004) in 'The Reluctant Economist: Perspectives on Economics, Economic History, and Demography' pushes the notion of the primacy of targeted social intervention much further, arguing that innovations in health technologies are dissociated from those in production methods. To Easterlin, major medical breakthroughs of the nineteenth and twentieth centuries, which were based on the germ theory of disease and led to mortality decline, fall into three broad categories: new methods of preventing the transmission of disease through sanitation and education (from the 1850s), new vaccines (from the 1890s), and antibiotics (from the late 1930s). The implementation of sanitary measures (inspection, quarantining, establishment of water supplies and sewage, regulation of food and milk, improvements in housing standards and general cleanliness) in addition required institutional innovation, namely the establishment of a network of local health boards under the supervision of the state, which could put the reforms in place (Easterlin 1999). Public subsidies to health services in the form of hospitals and medical personnel should have had a sizable impact on mortality beginning from the 1870s, as they supplied information about new medical discoveries to the general public. As Easterlin (2004) speculates, dramatic declines

in infant and child mortality in this period – the 'mortality revolution' – occurred independently of modern economic growth, and required 'much more modest resource commitment'. Moreover, market developments and institutions rather counteracted the health policy, through increasing exposure to disease in the growing cities or direct opposition from a side of the individuals and firms. Continuing to trumpet the significance of medical science, Easterlin (2000) relates the decline in cardiovascular disease in the second half of the twentieth century to the medical and research advances translated into improved lifestyles and new drug treatments. Despite the rich argumentation, Easterlin (1999, 2004) does not provide reliable empirical analysis. In line with these arguments, Hardy (1993, 2001) matches the timing of the advances in preventive and curative medicine, including practice of disease surveillance and supportive treatment from the 1870s, introduction of antitoxin against diphtheria in the 1890s, expansion of hospital births from the 1930s, and sulpha antibiotics against pneumonia in the late 1930s amongst earlier measures, with trends in infectious and non-infectious diseases.

There are explanations for the mortality decline which attempt to avoid the competing hypotheses of exposure versus resistance to disease and instead outline the whole range of possible factors. Such studies are less equipped with empirical data and analytical tools and rely instead on secondary sources. In the studies of the United Nations, several determinants of the global mortality decline are named, such as public-health reforms, advances in medical knowledge, improved personal hygiene, and rising income and standards of living (United Nations 1953), with decline in the virulence of pathogens added later (United Nations 1973). More recently, preventive, curative and surgical therapies have been suggested to determine the commencement of declines in chronic diseases (Bongaarts and Bulatao 2000). When presented in more detail, this multifactorial explanation is consistent with the postulates of the classical demographic transition theory, according to which high levels of mortality in traditional societies existed due to poor social and economic conditions, and were replaced with low mortality as soon as these conditions improved. In the probably most vivid expression of this theory, Omran (1971, 1998) argues that the long-term mortality decline should be understood as an epidemiological transition, from the prevalence of the deadliest epidemic diseases, to stabilization in infectious death rates, and to the prevalence of degenerative diseases and decline in certain types, where transition between the stages occurs due to the changes in a 'modernization complex'. Riley (2001) in 'Rising Life Expectancy: A Global History' makes a detailed survey of these determinants, concluding that progress in life expectancy represents 'the success of efforts to modify human behaviours, to reshape the environment, and to invest the means of avoiding, preventing, and ameliorating diseases and injuries' (p.221). This 'transition' explanation differs from those discussed before, e.g. Kunitz (1984, 1986) or McNeill (1989), primarily in the line

of causation, by assigning economic growth as a necessary condition for the initiation of mortality transition.

Despite the lively theoretical debates, to date supportive empirical evidence remains predominantly descriptive. Both inspection of trends in total and cause-specific country mortality and their matching to the introduction of various reforms or trends in living standards are the main analytical tools of the studies discussed above. Nation-level data hide potentially important discontinuities; as highlighted by Szreter (1988), rather arbitrary conclusions can be (and have been) drawn regarding the relative contribution of different factors. Incorporation of region-specific detailed data provides one solution. For instance, Woods, Watterson, and Woodward (1989) conduct an associational analysis between infant mortality and environmental and socio-economic district characteristics for England and Wales from 1880–1910. Both sanitation measures and provision of safe milk and food have been found to be of special importance. Kunitz and Engerman (1992) disaggregate mortality by social class for England and the Netherlands in 1877–1915, the period examined by McKeown (1976), and find no systematic patterns. Any social differences in mortality that were depicted could be explained by the spatial composition of social groups, mainly between urban and rural areas. One further step taken in the recent literature is investigation of detailed individual-level data covering total populations. Recently, several studies for different European countries, the US and Canada with such data analyse associations between mortality, social class and residence for long periods – covering the nineteenth and twentieth centuries (Bengtsson and van Poppel 2011). In Sweden, a social gradient in adult mortality is most likely to be a very recent phenomenon, beginning from the 1950s (Bengtsson and Dribe 2011). The consistent finding of these empirical studies is that geographical location rather than social class is associated with mortality. A reasonable explanation is that there are differences in exposure to infectious disease associated with location, and the availability of tools to combat disease, rather than in resistance to disease and nutrition, captured by social class. Rich in micro details and contextual information, these studies do not answer the main question – about causation.

Another step has been recently made by studies looking at the impact of public health reforms with region-specific data and using methods credible from a causal perspective. These studies consistently establish the important role of public initiatives and thus exposure to disease in reducing mortality. At the county level, Petterson-Lidbom (2014) finds that the employment of qualified midwives led to 15–30% reduction in maternal mortality in Sweden between 1830 and 1894. No effect of expansion of midwifery on infant mortality was found, Petterson-Lidbom suggested, because hygienic instructions regarding the birthing environment had not yet been put in place. Several quasi-experimental studies investigated the impact of water purification technologies on mortality for the largest cities, such as Stockholm (Macassa, de Leon, and Burström 2006), Chicago (Ferrie and Troesken

2008), and Paris (Kesztenbaum and Rosenthal 2017), and found sizable effects. For instance, Cutler and Miller (2005) show that filtration and chlorination technologies reduced typhoid mortality by one fourth and child mortality by one half for a set of US cities between 1900–1936. Hansen, Jensen and Madsen (2017) studied the impact of openings of tuberculosis dispensaries in cities in Denmark from 1890–1936, and found that it led to a 16% decline in tuberculosis mortality. For the later period, based on state-level US data, Yayachandran, Lleras-Muney and Smith (2010) show that introduction of sulpha antibiotics led to decline in maternal mortality by at least one fourth, in pneumonia mortality by one sixth, and in scarlet fever mortality by a half. To date, there are no studies of a similar kind relying on individual-level population data, the sort of data which provides correct information about the populations at risk and accounts for individual-level socio-economic characteristics. Moreover, empirical evidence for the efficacy of specific public health initiatives, such as isolation or disinfection, which were applied on a much broader scale, is absent.

Health as a determinant of economic growth: economic growth theories and empirical findings

After showing the relationship between the historical progression of health and income in developed countries, this section discusses economic growth theories. It attempts to focus on those models where health improvements, public health initiatives and scientific advances are recognized as determinants of economic growth. While the papers of the thesis primarily rely on the microeconomic analytical literature (e.g., Grossman 1972; Cunha et al. 2006; Heckman 2007; see below), macroeconomic growth models – neoclassical and endogenous growth models – provide an essential theoretical framework. They position the positive productivity effects of population health, induced by public health measures and other intervening factors, at the macro level. In addition, they uncover the interaction between population health and accumulation of (medical) knowledge as a determinant of economic growth.

The experience of developed countries in the nineteenth-twentieth centuries is not limited to the remarkable health advances; real income increased even more rapidly. Internationally, a marked sustained increase in growth rate in income per capita occurred in the nineteenth century, and it has been hastening since then (0.7% for the UK and 1.0% for the US in 1800–1870, and 1.5 and 1.8% in 1870–2010 respectively; Lucas 2000; The Maddison-Project 2013). In this respect, economic growth points to the analogy with long-term health development outlined above, although its timing is delayed compared to that of the mortality

decline. I have constructed the Preston curves, which are the plots of life expectancy versus real income, between the countries for 1880, 1930, 1960, and 2010 (see Figure 3). It becomes clear that the period 1880–1960 marks a remarkable shift in the curve, where life expectancy at birth grew rapidly whereas there were comparably much smaller increases in real incomes. Such an observation is similar to that of Preston (1975), who conducted analysis for 1900s–1960s and argued that the rise in living standards may not be responsible for the mortality decline in the nineteenth and the beginning of the twentieth centuries. Instead, the strong commonalities between these phenomena are in line with the growth theories which place health and health interventions as determinants of economic growth.

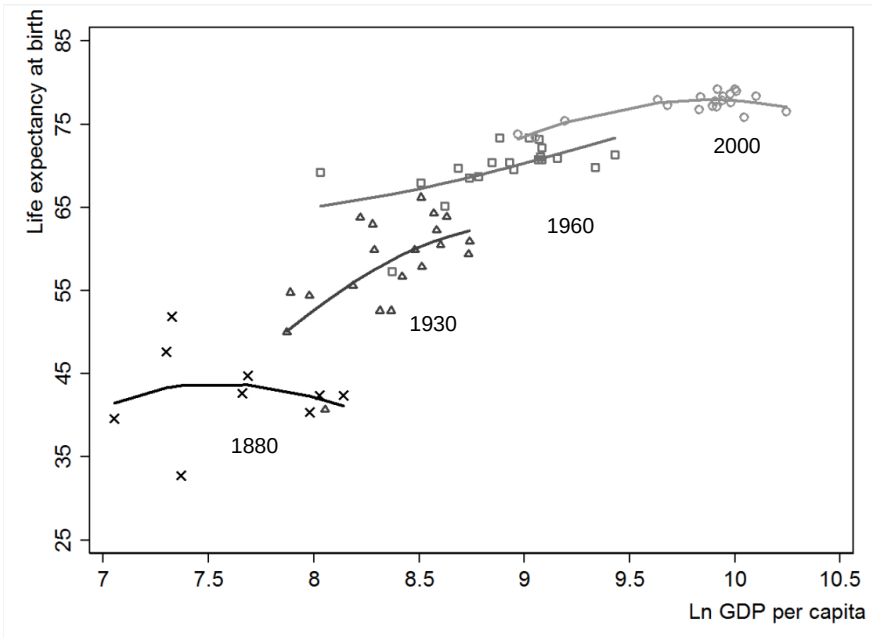


Figure 3 – Preston curves 1880–2000: life expectancy at birth and ln GDP per capita (1990 international Geary–Khamis dollar)
Sources: constructed based on data from Acemoglu and Johnson (2007); The Maddison-Project 2(013); Human Mortality Database (2017).
Note: The sample is limited to the countries similar in income per capita to Sweden in 1890 [1635, 1990 international Geary–Khamis dollar].

Health has been incorporated among the reproducible factors in the neoclassical growth models. According to Solow (1956) and Swan (1956), the inputs of physical capital and labour exhibit diminishing returns each and constant returns overall; technological change appears to be the only source of sustained economic growth, which is treated as an exogenous phenomenon, or 'manna from heaven'. In the traditional neoclassical models, knowledge, or 'effectiveness of labour'

operates multiplicatively with labour and not capital, thereby contributing to economic growth by augmenting labour productivity (Solow 1956). Later development of the concept of capital in the neoclassical model has been usefully broadened from physical goods to include human capital in the form of education, experience and health. In these models, all forms of capital – physical, human and health capital – are characterised by decreasing returns to scale, and the same is true for labour, together assuring constant returns for all factors. For instance, testing the model empirically for growth rate in GDP per working-age person across a set of countries in 1960–1985, Mankiw, Romer and Weil (1992) include human capital, measured as the share of the working population with secondary education, as an input alongside physical capital and labour and find that the model accurately describes existing international differences. Barro and Sala-i-Martin (2004) (Ch.12) in 'Economic Growth' and Barro (2013) additionally incorporate a concept of health capital. Health is assumed to affect productivity through various channels: for a given amount of labour hours, physical capital, and schooling, improvements in health raise labour productivity directly both physically and mentally; because poor health raises levels of mortality and morbidity, it affects labour productivity indirectly, thereby decreasing the effective rate of depreciation of human capital and raising the demand for human capital; better health and lower mortality also affect population and fertility, with various influences on productivity. Empirically, Barro and Sala-i-Martin (2004) (Ch.12) find that health, measured by life expectancy at age 1, emerges as a statistically and economically significant correlate of growth rate of GDP per capita, similar to labour and capital, in a set of countries from 1965–1995. In a growth accounting framework, which allocates growth rates in national output to its determinants based on the assumptions of the neoclassical models, Nordhaus (2003) proposes to measure the economic value of improved health status, which is gains in life expectancy at birth times the discounted labour income per year – value of an additional life-year. Such a value, called 'health income' or 'returns to health capital', accounts for more of growth in income per capita for the US for 1900–1950 than non-health goods and services, and marginally less since 1950. By looking more closely at causes of death and life gains at different ages, Murphy and Topel (2006) attribute the bulk of this value to medical interventions against infectious diseases.

Health as a determinant of economic growth has been explicitly asserted in the endogenous growth models. In endogenous growth models, long-term economic growth will depend on the rate of technological change, similarly to the assumptions of neoclassical theory, but here technological progress is an outcome of the economy. By integrating microeconomic foundations (the behaviour of firms and households), all endogenous growth models envisaged the idea of their very first version developed by Romer (1986): capital (physical and intellectual) accumulation exhibits constant returns to scale, technology is growing in line with

such accumulation, and together they lead to increasing returns at the aggregate level. The further developed growth models recognize innovation, which encourages the growth of intellectual capital, as a primary source of technological progress. To become a long-term economic determinant, health among other inputs is required to affect the equilibrium rate of innovation. Aghion and Howitt (1992) and Howitt (2005) develop a theoretical model that contains various channels through which population health influences economic performance, such as improvements due to productive efficiency, improvements in skill level due to changes in life expectancy, improvements in learning capacity, creativity and coping skills, and reductions in inequality. Not all of these factors enhance long-run economic growth, but only creativity and coping skills through the influence of innovation capacity, which – as Howitt and Aghion highlight, leaning towards the epidemiological literature – can be especially fostered in early childhood. The empirical work by Aghion, Howitt and Murtin (2011) establishes that both levels and improvements in life expectancy at birth across developed countries affect per capita income growth in 1960–2000. van Zon and Muysken (2005) construct a theoretical model where provision of health services affects economic growth and provide empirical support for it. The model recognizes that health affects labour efficiency, that the health services require the allocation of labour, that improvements in health influence the utility directly, and all of them through various channels affect human capital accumulation.

In the endogenous growth models, knowledge accumulation drives economic growth. According to Romer (1990), goods are produced with a single kind of capital – 'knowledge capital' – and the output of producers will depend on their own knowledge, and the knowledge possessed by the society, and the stock of knowledge is virtually free like a public good. While internal knowledge yields diminishing returns, society's knowledge possesses increasing returns to scale at the aggregate level. Lucas (1988) develops the model of the accumulation of knowledge or human capital, where this capital can be accumulated intentionally. For Lucas, growth in stock of 'useful' knowledge does not generate sustained increase in living standards unless it raises the return on investment in human capital among individuals, who can increase own human capital by devoting time to education or learning. With human capital accumulation as a main driver of growth, Lucas (2002) in the 'Lectures on Economic Growth' provides his explanation of long-term economic growth in Western countries by additionally incorporating fertility decisions: 'the industrial revolution required a change in the way people viewed the possibilities for the lives of their children that was widespread enough to reduce fertility across economic classes' (p.160). This model of long-term economic development is close to the theoretical view and empirical findings of Galor (2011) in 'Unified Growth Theory'. To Galor, sustained economic growth is explained by the interaction between several groups of factors, such as the Malthusian elements, the engines of technological progress,

the origins of human capital formation, and the determinants of families' choices regarding the quantity and quality of their children. In the model, the size of the population stimulated technological progress in both industry and medicine until 1870, whereas afterwards the resulting increase in prolongation of life and industrial demand for human capital encouraged human capital accumulation, with positive consequences for economic growth.

The hypothesis about the interaction between different types of knowledge as a determinant of economic growth in Western countries is extensively elaborated by Mokyr (2002) in 'The Gifts to Athena: Historical Origins of the Knowledge Economy'. 'Useful' knowledge, that with technological implications, is divided into 'propositional' knowledge, which involves regularities that can be discerned by chemistry, biology and physics, and 'prescriptive' knowledge, which encompasses practical techniques. Encouraged by the development of institutions, 'propositional' knowledge, which interacted with 'prescriptive' knowledge, grew stronger in the nineteenth century and 'created a positive feedback that never existed before', determining the rate of technological progress and sustained increase in living standards (Ch.3). According to Mokyr (2005), such institutions include universities, financial institutions and local government involved in public sanitation. Mokyr and Stein (1996) and Mokyr (1998, 2002) also suggest viewing the changes in medical technology as a special case of induced technological change. With the peoples' 'desire to survive, be disease-free, pain-free, and have one's children and relatives enjoy the same', three major scientific revolutions – the sanitarian and hygienic movement from 1815, the applications of the germ theory of disease after 1870, and curative medicine and composition of diet – were the most important increases in 'useful' knowledge that helped to avoid infection both in the domain of public health and in that of household technology. They all are based on the scientific understanding and gradual eradication of the infectious diseases. As a result of these changes in 1871–1911 across European countries, Mokyr and Stein (1996) present an estimation of the economic value of improvements in life expectancy at birth, as a fraction of growth rate in utility (defined as life expectancy adjusted with discount rate and elasticity of annual utility with respect to consumption) in growth in real wages. This contribution is substantial: in England, Germany, and France 33–40% of growth was due to increased life expectancy; in Sweden, where increase in real wages was extremely fast compared to other countries, it equals to 8–13%. Mokyr and Stein contend that these results 'place Pasteur's [germ theory] revolution at center stage in the history of economic progress' for half a century after 1870 (p.196). The notion of primacy of institutions and the germ theory of disease in determining mortality decline and the following increase in the pace of economic growth strongly resembles the viewpoint of Easterlin (1999, 2004) in 'Reluctant Economist: Perspectives on Economics, Economic History, and Demography', outlined above. Easterlin (1996) in 'Growth Triumphant: The Twenty-First Century in Historical

Perspective' suggests that the demonstrable success in improving health of preventive methods may promote favourable attitudes towards innovation more generally, as well as increase motivation for long-term investment and innovation.

Net-nutrition, that is the difference between gross nutrition and its claims because of infectious disease, can be explicitly encountered as a health input in the endogenous growth models. Fogel and Costa (1997) (and later Fogel 2004 and Floud et al. 2011) suggest that over the eighteenth and twentieth centuries 'a synergism between technological and physiological improvements has produced a form of human evolution that is biological but not genetic, rapid, culturally transmitted, and not necessarily stable', and call this process 'techno-physio evolution'. In their opinion, the technological breakthroughs affecting long-term mortality and driving economic growth are mainly in food production, but also include those in manufacturing, transportation, trade, communications, energy production, leisure-time services and medical services. Such interactions between technological and health improvements do not act as period factors, but as cohort factors, that is, they affect only certain age groups with long-lasting effects. Fogel and Costa (1997) and Fogel (1994, 2004) suggest that malnutrition (and less stressed but mentioned, exposure to infectious diseases and other types of environmental and socioeconomic stress) prenatally and during early childhood determines survivorship and performance in middle and late ages. They develop a concept of 'physiological capital' to denote an individual's capacity to learn and work, implying the interplay of environment and a body starting from early life until old age. They consider the view that the epidemiology of chronic diseases is more separate from that of infectious diseases to be 'a significant misinterpretation'. In 'The Changing Body: Health, Nutrition, and Human Development in the Western World since 1700', Floud et al. (2011) extend this notion beyond one generation, arguing that the nutritional status of a generation determines not only its own longevity and productivity, but also the nutritional status, living standards and performance of the next generations, ad infinitum.

As net-nutrition consists of two components, in practice it is hard to disentangle the mortality developments due to increases in gross nutrition from those due to decreases in infectious disease. Finding a strong relationship between the development of heights, BMI and caloric intakes in Western countries, Fogel (1994, 1997) argues that improvements in gross nutrition are strongly linked to secular declines in mortality and morbidity and improvements in economic performance, and that declines in infectious disease were less important. In France and England, improved gross nutrition can account for 90% of the mortality decline between 1785 and 1870 and about 50% between 1870 and 1975. In England, the increase in the number of calories available for work made two contributions to economic growth in 1780–1980: it first increased the labour force participation rate by including the labour force at the bottom 20% of the caloric consumption, and second, by raising the average consumption of calories, it

increased the productivity of those in the labour force by about 60%. This yields economically large effects: in combination, Fogel contends, these two processes contributed 0.34% per year to the annual growth rate of per capita income in Britain and accounted for 20–30% of growth in 1780–1979. By relying on the most recent data for the period 1800–2000, Floud et al. (2011) have updated this number to 54%. Fogel and Costa (1997) and Fogel (2004) in 'The Escape from Hunger and Premature Death, 1700–2100: Europe, America and the Third World' speculate that their findings are not linked exclusively to food consumption, but also to clothing, shelter, incidence of infectious diseases and public health, although inevitably they turn toward the primacy of nutrition, both conceptually and empirically.

Epidemiological development defined broadly has been explicitly incorporated in the endogenous growth models. Arora (2015) in 'The Transitions of Aging' presents a theoretical model where the improvements in health due to the decline in infectious diseases circa 1870 through intergenerational effects are linked to a permanent increase in economic growth. Based on the data for England and Wales from 1848–1992, Arora presents evidence on the close relation between mortality decreases due to infectious diseases in the late nineteenth century and mortality decreases due to non-infectious diseases in the twentieth century. In the Malthusian epidemiologic regime, the persistence of infections and instability of diets resulted in high death rates from all diseases. As soon as new knowledge about disease prevention arrives, human durability can improve both contemporaneously and as a cohort phenomenon, leading to decline in later-life chronic mortality. He indicates that for England and Wales, the decline in mortality from non-infectious diseases commenced around 30 years after that in infectious diseases, already beginning in 1900, pointing to the existence of the cohort mechanism linking both processes. Due to the lasting effects from improved public health conditions and the status of health as human capital, health leads to sustained economic growth. By applying a cointegration approach, Arora (2001) tests his model for a sample of developed countries in 1870–1994 and concludes that health improvements are associated with the increase in long-term growth by 30–40%. He relates health improvements to public health infrastructure, the germ theory of disease and advances in food production, arguing that they have not been a by-product but rather a driver of economic growth.

Empirical studies outlined above provide evidence for the existence of strong associations between health improvements and economic growth. Recently, several macroeconomic studies applying methods more credible from a causal perspective have addressed whether such associations might be causal, at least for the interventions after the late 1930s–1940s. Yet, they show rather different results with regard to the existence and size of the effects. A population's health, size and age structure are strongly affected by deadly diseases, and several scholars estimated the effect of specific diseases on economic growth. For instance, Gallup

and Sachs (2001) show sizable and robust estimates for the fractions of population at malaria risk in cross-country regressions of income growth for the 1965–1990. Based on these associations, the elimination of malaria in a country where it was endemic would increase income per capita by 1.3% per year. Several studies with similar samples, such as Sachs (2003) and Lorentzen, McMillan, and Wacziarg (2008), propose to account for endogeneity of mortality by instrumenting its impact on economic growth with biological and climate characteristics of malaria transmission. The results are again sizable and suggest that elimination of disease would raise GDP per capita by at least 2.7 times. There are several studies looking at the economic effects of a broader set of public health interventions more applicable to developed countries. For instance, Ashraf, Lester, and Weil (2009) applied a simulation approach to cross-country data, in which different channels are specified with microeconomic estimates to the effects of health interventions implemented based on the germ theory of disease after the 1940s: of those various interventions leading to rise in life expectancy at birth from 40 to 60 years, and of eradication of malaria and tuberculosis. They find that health benefits raise GDP per capita by roughly 15% in the long run, but reduce it in the short run due to population growth. In contrast, consistent with the neoclassical growth model, as Acemoglu and Johnson (2007) show, health gains do not cause long-term economic growth. For a set of developed and developing countries, they find that an abrupt decrease in mortality due to the arrival of pesticides for eradicating malaria-bearing mosquitos, drugs and health campaigns efficient against major infectious diseases in the 1940s, such as malaria, tuberculosis and pneumonia, indeed increased life expectancy across countries between 1940–1980, but left growth of income per capita unaffected.

The theoretical macroeconomic model most suitable for the current thesis is therefore provided by the endogenous growth theory by Fogel and Costa (1997) and Fogel (2004), which posits synergism between technological and health improvements as a determinant of health and income in long run. This model is dynamic, because the related benefits emerge as a cohort phenomenon and accrue in adulthood of the individuals. An important difference is that the scholars put emphasis on adequate nutritional intake, and thus on living standards, as a prime determinant. Instead, the current thesis emphasizes the economic significance of public health and medical technologies targeting infectious diseases. From this perspective, it is closely related to the theoretical arguments provided by Easterlin (1999) and Mokyr (2002), who suggest that the germ theory of disease and the related public initiatives appear as an important determinant of economic growth, although their model is static. The current thesis places this theory in a dynamic, namely cohort, perspective. In doing so, it also follows the emerging trend in the macroeconomic empirical literature which studies the relationship between exogenous health improvements and labour productivity in a cohort perspective, at times when the benefits can be fully realized for all age groups.

Early-life determinants of adult health and labour income

The reasons why better environmental conditions might positively affect population health and income in the long run are broader than those demographic and economic ones outlined in the macroeconomic literature. They concern the specificities of human physiology, which relate to the development of the body and its functioning. To understand such processes, scholars need to theorize and run investigations at the level of an individual. To date, theoretical postulates about the relationship between early-life conditions and adulthood outcomes have been provided by both epidemiology and microeconomics.

Epidemiological hypotheses on the lasting importance of early-life environment for adult health

This thesis focuses on interventions affecting early-life health which relate to biological processes mediating the relationship between early-life and adult health. The epidemiological literature pursued this issue beginning in the 1920s–1930s. The theoretical models, refined since then, fall into a so-called life-course approach, which emphasizes the long-term importance of environmental exposures that occur early in life (Kuh and Ben-Shlomo 2007). Two conceptual models of adult health have been developed (Ben-Shlomo and Kun 2002; Lynch and Davey Smith 2005) which are generally in line with economic models to be reviewed below. The 'critical period model' – specifically tested in the research conducted for this thesis – suggests that exposures or treatments acting during specific periods, such as the fetal stage or infancy, have lasting or delayed effects on health which cannot be modified in any substantial way by later-life experience. Within this framework, the literature introduces the concept of 'sensitive period', that is, the period when environmental events have a stronger influence on health in terms of later outcomes compared to other periods, and thus later interventions could potentially modify the effects. The models of 'accumulation of risks' consider that exposures affecting health accumulate over time, and both the accumulation and the related effects on health can occur at any period of life. Such literature is exceedingly focused on biological exposures as determinants of health and mortality, and approximates the intervening tools with various childhood socio-economic conditions, recognizing the existence of direct and indirect (through human capital accumulation) pathways to adult outcomes (Preston, Hill, and Drevenstedt 1998; Kuh and Ben-Shlomo 2007).

The current understanding of biological influences of early-life conditions on health in adulthood and old age remains hypothetical. Several such hypotheses are

suggested – examples include the developmental origins hypothesis (incorporated within the 'critical/ sensitive period' model) and the allostatic load hypothesis (complementary to the 'accumulation of risks' model), both pointing to early life conditions as an origin of later-life health (review in e.g., Hostinar and Gunnar 2013). The developmental origins hypothesis has grown from the studies of Barker and the colleagues, summarized in the seminal work of Barker (1994) in 'Mothers, Babies, and Disease in Later Life', that showed that various indicators of child health, more specifically, fetal growth (pointing to maternal nutrition), infant growth (pointing to nutrition in infancy) and exposure to infections in infancy are associated in a bivariate fashion with mortality from chronic diseases in adulthood, such as coronary heart disease, type II diabetes and osteoporosis. Only earlier work by Barker was explicit about the importance of early infections for later-life mortality, whereas his later work focused entirely on the role of maternal nutrition (Barker 2012). The analysis by Barker has spawned a large nonexperimental empirical literature in epidemiology trying to connect specific conditions at specific ages in childhood to the development of later disease and by these means elucidate the underlying processes (Gluckman and Hanson 2006). The current understanding of the 'developmental origins' of adult disease focuses on the distinction between body characteristics, such as genotype (inheritable) and phenotype (heritable), and the 'memory' of the body of the childhood environment expressed in the latter (e.g., Waterland and Michels 2007). It suggests that disease may be latent until adulthood and old age and then emerge in higher chronic disease probabilities: positive and negative environmental shocks during early-life critical stages lead the body to compromise its functions, metabolism and chronic disease susceptibility that are important in later stages of lifetime.
A complementary hypothesis – the allostatic load hypothesis – also suggests that having faced environmental shocks, the growing body undergoes physiological changes, for instance produces stress hormones or launches immune responses (e.g., Danese and McEwen 2012). These adjustments are optimal in the very short run, but if frequent and chronic, such 'allostatic load' leads to higher morbidity and mortality, remaining as a permanent disadvantage from childhood to adulthood.

The early neonatal period and infancy – the focus of this thesis – are suggested to be critical time windows for health development. Even though the related epidemiological studies overwhelmingly rely on recent cohorts, they provide important insights on the origins of the long-term repercussions of policy-generated improvements in environmental conditions under study in this thesis. All of these interventions reduced exposure to various infectious diseases, especially respiratory (isolation hospitals, disease monitoring and improved midwifery) and in particular pneumonia (sulpha antibiotics). A strong note in the literature is that exposure to respiratory or diarrhoeal infection among infants, who are more susceptible to disease than other age groups, is linked to later-life chronic

diseases (see review in Bengtsson and Lindström 2003; Davey Smith, Leary, and Ness 2006). Adhering to an inflammation hypothesis, Finch and Crimmins (2004), Crimmins and Finch (2006), and Finch (2007) provided a set of related arguments, in which infectious conditions in early life, especially among infants, cause direct damage to organs and cells, which launches a chronic inflammatory response or diverts resources away from normal body development, thereby leading to chronic diseases later in life. Exposure to respiratory infections in early life supposedly leads to underdevelopment of lungs and to lung damage, which persist throughout life and is revealed in chronic respiratory diseases (Bengtsson and Lindström 2003). A characteristic of respiratory and stomach infections is their ability to persist in tissues for many years since the insult of contagion in early life, sometimes throughout the whole life (Liuba and Pesonen 2005). Such invasion is followed by the dissemination of the microbes into the blood and arterial walls, causing chronic inflammation and eventually cardiovascular and metabolic diseases.

The empirical medical studies supporting the hypotheses overwhelmingly rely on associations between childhood disease history or childhood anthropometry and later-life disease development. Barker et al. (1991) studied cohorts born between 1911 and 1930 in England and found that pneumonia, bronchitis and whooping cough infections in infancy, correlated with retarded infant growth and with chronic obstructive airway disease in late adulthood (ages 59–70), and there are no such effects for children in ages 1 to 5. This result was independent of birth weight, smoking habits, and social class. The investigators concluded that infection of the lower respiratory tract during a critical period in infancy has persisting adverse effects. In several contemporary studies, reduced exposure to microbial infectious diseases in the early neonatal period has been associated with markers of chronic inflammation, which are linked to the process of early atherosclerosis and are predictive of risks of cardiovascular and metabolic diseases (McDade et al. 2010). Contagion with pneumonia infection in infancy through the above mechanisms is linked to various chronic diseases in adulthood, such as abnormalities of pulmonary function (Carraro et al. 2014), arthritis (Colebatch and Edwards 2011), cardiovascular disease (Willerson and Ridker 2004), and diabetes (Beyerlein et al. 2016). There are several studies showing that treatment of individuals infected with pneumonia with antibiotics is associated with improvement of markers of atherosclerosis and the reduction of the risk of atherosclerosis-related ischemic events (Liuba and Pesonen 2005). An additional line of epidemiological literature shows that early-life exposure to infection affects the development of the brain, leading to staggered development of cognitive and behavioural abilities (Landrigan et al. 2005).

The papers comprising this thesis examine health interventions which not only reduced exposure to infection, but also provided beneficial treatments to neonates (qualified midwifery). The connected empirical evidence is sparser, albeit it sheds

light on intervening factors linking early- to later-life health. The level of maternal and midwifery care, such as time spent nursing, in the early neonatal period could have a critical influence on the development of stress responses and may alter the long-term inflammatory process with links to chronic diseases and cognitive dysfunction (Miller, Chen, and Parker 2011). Disease surveillance by medical personnel, doctors and midwives provides supportive treatment and could help to divert additional maternal resources from a healthier mother to the child, especially those fragile at birth, potentially discouraging the development of future disease (Calkins and Devaskar 2011). All environmental factors acting in early life can affect almost any aspect of the immune system, leading to misregulated innate immune responses, inflammatory dysfunction, and an increased risk of chronic inflammatory diseases (Leifer and Dietert 2011). Through this mechanism, prolonged breastfeeding has been shown to decrease later-life risk of cardiovascular disease and diabetes (Andersson et al. 2009).

The basis for understanding biological mechanisms behind early- and later-life health – in such a long term perspective as in this thesis – is not complete without hypotheses explaining secular mortality decline. These hypotheses related to the instantaneous improvements in health conditions historically have been discussed in detail above, and empirical evidence connecting cohort improvements with long-term health and mortality is presented below. Each paper in this thesis reviews the related medical literature in more detail.

Economic theoretical models linking early-life environment to adult health and labour income

Early-life epidemiological literature has spawned a recent theoretical literature on health economics, which emphasizes the role of early-life conditions for adult outcomes. Their review is important in this thesis for several reasons. The microeconomic theoretical models that were developed not only related general and cognitive health at different stages of life to health in childhood, parental investments and environmental shocks, but also extended it to human capital accumulation and income. Theoretically, health economics made a broad distinction between health in early and late childhood and adulthood, but relied on empirical literature for a more definite answer regarding what stages of childhood are the most productive. Microeconomic models focus exclusively on intervening factors in later-life outcomes, which are under study in this thesis.

Theoretical perspectives on the microeconomic foundations of health as a determinant of productivity and labour earnings have roots in traditional neoclassical theory. Schultz (1961), Becker (1964), Ben-Porath (1967), and Mincer (1974) developed human capital theory and the theory of allocation of time, which place the individual as a producer of wealth, the one who allocates

their own resources so as to attain the highest possible outcomes over the life cycle. Human capital is a broad term that encompasses 'the knowledge, information, ideas, skills, and health of individuals' (Becker 1964). It is 'the stock that produces labour services … and is thus the analogue of machines in the case of tangible capital' (Ben-Porath 1967), as well as produces commodities in non-market (household) sectors (Becker 1964). All parts of human capital exhibit diminishing returns as a function of scale. According to human capital theory, increases in individuals' stock of human capital raise their productivity (through raising wage rate) in market and non-market sectors, and thus an individual has an incentive to invest in their own human capital in the form of schooling, on-the-job training, improvements in emotional and physical health, migration or searching for information about prices and incomes. More specifically for adult health, as Becker (1964) writes, 'a decline in the death rate at working ages may improve labour earning prospects by extending the period during which earnings are received; a better diet adds strength and stamina, and thus earning capacity, or an improvement in working conditions may affect morale and productivity'. Becker (1964) and Ben-Porath (1967) develop models that determine the optimal quantity of investment in human capital at any age, in general contending that investment in human capital should fall with age as the period over which returns can be accrued decreases.

Investments in children's health have been suggested as an important determinant of their future earnings. In a seminal book on family economics, 'A Treatise on the Family', Becker (1981) (Ch.6) introduces a theoretical model of an individual's adult income in relation to parents' investment behaviour:

$$y = I + A \tag{1}$$

where A denotes the market value of the child's endowments ('different defects and abilities, different accidents, luck, and other experiences as they interact with their environment') and of the capital gained due to 'the luck in the market sector'; I denotes the market value of the family's investments in human capital ('education, nutrition, shelter, child care and other human capital') on behalf of their children. Through investments in children, which are a function of family income, a market rate of return on parental investments in human capital and the market value of endowments and luck of children, parents can influence the income of their children. The model may additionally recognize the role of public programs in equalizing the opportunities for children from poor and rich families.

As an important extension to Becker/Ben-Porath's human capital theory, Grossman (1972, 2000) constructed the health capital model. Grossman argues that an individual's stock of health capital differs from other forms of human capital as it does not simply increase wage rates, but determines the total amount of 'healthy' time (morbidity and more broadly longevity), which is used for producing labour income and commodities. The development of health capital in

the current period h_t is assumed to depend on its value in the previous period of health h_{t-1} in the following relation:

$$h_t = A[(1 - \delta)h_{t-1} + I_t] \quad (2)$$

where I_{t-1} – health investments; δ – depreciation rate; A captures all the external factors that affect health. Health stock is understood as resistance to disease; it varies over time, typically depreciating at an increasing rate, but is influenced by investment in the form of time and market goods such as 'medical care, diet, exercise, recreation and housing'. With age, depreciation rate increases, making health investment too costly, and the resulting decrease in health stock leads to death. To Grossman (1972), all environmental factors (demographic characteristics and education, which could be extended to include all background characteristics, disease and public health environment), in analogy to forces such as 'technology and entrepreneurial capacity in firms', are exogenous. Shifts in such factors influence the individual's productivity in labour market and non-market activities. Following the model, Bleakley (2010a) explains that when education is allowed to depend on health capital, the direct effect of health capital on income (more 'healthy' days and higher mental and physical productivity) remains of first order importance compared to indirect effects – changes in quantity of education.

A contrasting view has been recently presented by early-investment theories. Unlike in Grossman's (1972) models, where the positive effect of health in the previous period vanishes over time, the theoretical models developed by Cunha et al. (2006), Cuhna and Heckman (2007), and Heckman (2007) suggest that health and investment in childhood have sustained or even increasing effects on the stock of human capital, its various components and labour income. As analogues to human capital, Cunha, Heckman and their colleagues invoke the term 'human capabilities' to describe the following components: pure cognitive abilities (skills), non-cognitive abilities (skills), and health (propensities for mortality and morbidity). Similar to Becker (1981), Cunha et al. (2006) relocate the attention from adult health investment decisions, where child health endowments are treated as exogenous, to investments in early life, where parents make decisions about investments. Unlike Ben-Porath (1967) or Becker (1981), they argue that 'human capability' formation is governed by a multistage technology: different inputs and technologies can be used at different stages, and inputs at each stage produce qualitatively different outputs at the next stage. Heckman (2007) divide individuals' lives into three stages: early childhood, late childhood, and adulthood. The adult stock of capability h is described by the neoclassical production function with constant elasticity of substitution (CES):

$$h = A[\gamma I^{\varphi}_1 + (1 - \gamma)\, I^{\varphi}_2]^{1/\varphi} \quad (3)$$

where I_1 = health investments during the first childhood period, I_2 = health investments during second childhood period, A captures all the external factors that affect child health, such as genetic, background and environmental characteristics; $\phi \leq 1$ and $0 \leq \gamma \leq 1$. γ = the CES share parameter (showing how health investment are distributed throughout the childhood), which is a skill multiplier, and captures the net outcome of two effects. One is the effect of self-productivity: higher levels of 'human capabilities' in early childhood boost levels in late childhood; with regard to health, health in early childhood beget health in late childhood. Another is the effect of direct complementarity: a higher level of 'human capabilities' in early childhood makes investment in late childhood more productive; with regard to health, health in early childhood raises productivity of schooling; health is multidimensional, and one component of health may affect the formation and productivity of other components. The term $1/(1 - \phi)$ is the elasticity of substitution between I_1 and I_2.

In a framework of Heckman (2007) and Cunha and Heckman (2007), as Almond and Currie (2011) describe, one can consider the effect of exogenous shocks to health investment in early childhood, while treating parental investment as exogenous:

$$h = A[\gamma(I^{\varphi}_1 + \mu_g) + (1 - \gamma)\, I^{\varphi}_2]^{1/\varphi} \quad (4)$$

where μ_g denotes the exogenous shock (a subscript g refers to the group/ geographic area consistent with the shock usually occurring at a certain group/ geographical level), and all other terms are as above. In light of the papers in this thesis, one can assume that the shock on early-life health from a public health or medical treatment is positive, that is, $I^{\varphi}_1 + \mu_g > 0$. The impact of an early-life positive shock on adult 'human capabilities' – to be estimated in this thesis – is then described as follows:

$$\delta h/\delta \mu_g = \gamma A[\gamma(I_1 + \mu_g)^{\varphi} + (1 - \gamma)\, I^{\varphi}_2]^{(1-\varphi)/\varphi} \cdot (I_1 + \mu_g)^{\varphi - 1} \quad (5)$$

If $\phi = 1$ (early and late investments are perfect substitutes), the effect reduces to γA, and the benefit from the positive shock will depend only on γ (the extent to which early childhood is more productive than late childhood). For other values of ϕ (early and late investments are complements), there is decreasing marginal productivity of the investment: individuals born to families with higher levels of investment (rich families) will exhibit milder effects from a positive shock compared to those with lower levels (poor families).

Empirical evidence for the early-life hypotheses

In recent decades, the empirical literature that explores the early-life hypotheses, covering economics and economic history and relying on epidemiological studies for an interpretation, has grown substantially and continues to expand. While the scope of these studies is substantial and reviewed elsewhere (e.g., Galobardes, Lynch, and Davey Smith 2004; Davey Smith and Lynch 2005; Almond and Currie 2011; Montez and Hayward 2011; Currie and Rossin-Slater 2015; Currie and Vogl 2013), this section provides the outline of the results of the studies relevant for this thesis, those that linked (i) episodes of disease insults and (ii) beneficial policy-driven treatments in early life, targeting exposure to infectious disease and other aspects, to adult health and labour income. Properly discerned, exogenous differences among individuals or changes over time and place in specific health policies linked to individuals provide a good identifying variation, allowing the investigators to obtain estimates more credible from a causal perspective. By focusing on negative disease events, the first group of studies provides support for exposure to infectious disease being a mechanism behind long-term health and income improvements. So does the second group of studies, but also they explicitly elucidate the intervening factors, which is of particular interest in this thesis.

The earlier historical studies established consistent evidence for an association between infant and childhood mortality and mortality later in life using macro-level data. As early as the 1920s–1930s, birth cohort studies, based on age-specific death rates for England and Wales, Scotland, and Sweden, uncovered the presence of firm regularities in the relationship between relative childhood mortality and the mortality that each generation experienced in adulthood (Derrick 1927; Kermack, McKinlay, and McKendrick 1934). From the inspection of relative mortality for cohorts circa 1805–1925, Kermack, McKinlay, and McKendrick (1934) suggest that these cohort effects were attributed to the improved epidemiological environmental conditions as ‘each generation after the age of five years seems to carry along with it the same relative mortality throughout adult life, and even into extreme old age’ (p.679). Having replicated a similar analysis for Sweden and extended cohorts up to the born in 1960 in his conclusions, Fridlizius (1989) claimed that ‘the mortality decline during nineteenth century contained a marked cohort element and that interpretations of the causes and consequences of this decline might be oversimplified unless this aspect is not taken into account’ (pp.16–17). His findings thus gave priority both to a changed balance between pathogens and population and to sanitary factors, and exclude improvements in nutrition as the cohort factor of mortality decline. In interpreting the strong cohort effect in later-life mortality, Fridlizius (1989) proposed that decreased exposure to certain infectious diseases, such as smallpox or scarlet fever, in childhood through

improved immunity decreases susceptibility to infection in adulthood and as a result decreases mortality.

A more recent research in this area employed region-specific infant mortality rates as a composite measure of living standards in childhood, such as malnutrition, infectious diseases and poverty. Such rates may indicate broad social conditions for those born at that time, and if partitioned into, for instance, neonatal and postneonatal mortality and correlated with specific disease risks among adults, can point to more specific mechanisms. The findings for cohorts born 1896–1925 in Norway (Forsdahl 1977) and for cohorts born 1917–1921 in the US (Buck and Simpson 1982) suggested a strong association between high infant mortality rates and death rates from cardiovascular diseases during adult ages, after the 40s in the respective cohorts, pointing to the undernutrition in childhood as a mechanism. For cohorts 1911–1925 in England and Wales, Barker and his colleagues empirically connected neonatal mortality rates to cardiovascular disease in adulthood (suggesting importance of maternal nutrition), and postneonatal mortality rates to subsequent chronic respiratory disease in adulthood (suggesting importance of infection in infancy), both for those aged 35 and older (Barker, Osmond, and Law 1989; Barker 1991). In later studies, Barker (2012) ignored infection as a mechanism and strongly argued that in-utero growth and nutrition lead to chronic disease. Outlined earlier, the works of Fogel (1994, 1997) could be seen as suggestive the same, nutritional, mechanism.

Other scholars have claimed the relative importance of exposure to infectious diseases early in life as a cohort factor of mortality. Based on the macro-level data for England, France, Sweden and Switzerland, Finch and Crimmins (2004) and Crimmins and Finch (2006) were able to anew relate the mortality in ages 70–74 and height for the cohorts born up to 1899 to infant and child mortality in a year of birth, and find that these relations are strong. They argue that infant and child mortality for the cohorts in question are primarily explained by infectious-disease mortality. This enables the scholars and Finch (2007) in 'The Biology of Human Longevity', contend that inflammation ('cohort morbidity phenotype') resulting from exposure to infectious diseases in early life, fetal stage and infancy, shapes later-life morbidity and mortality and that the historical decline in infectious diseases stands behind the modern mortality decline in old age. Finch (2007) pushes this idea even further arguing that the relations observed by Fogel and Costa (1997) are all linked to improved life expectancy because all of the chronic diseases of aging have inflammatory components (p.15). Albeit being of high relevance, their empirical findings indirectly point to this, as the scholars cannot explicitly distinguish infectious versus other environmental causes. Such estimates should be also interpreted as associations, because when correlating data in levels there is always a possibility that the estimates are biased by period or time effects. As a methodologically important addition, Catalano and Bruckner (2006) apply methods enabling to control for trends, seasonal cycles, and other forms of

autocorrelation for the available country data in Sweden, Denmark, and England and Wales up until the cohort born in 1912. The exogenous component of child mortality (under age 5) was strongly correlated with cohort life expectancy at age 5, supporting the importance of early-life, supposedly infectious disease, conditions for survival of birth cohorts.

The contemporary economic and historical literature testing the early-life hypotheses for health outcomes by employing negative shocks to the individual-level data is substantial. Seasonal variations in infectious diseases during infancy, measured with season of birth, are associated with improvements in remaining life expectancy among the cohorts born at the turn of the twentieth century in Austria, Denmark, the US or Australia (Doblhammer and Vaupel 2001; Doblhammer 2007). The scholars find that the individuals born in the northern hemisphere live 0.36 years longer after age 50 if they were born in the fourth quarter instead of the second quarter. For the US, Costa and Lahey (2005) find that the effect of season of birth on mortality in ages 60–79 declines between the cohorts born in 1864–1865 and in 1900. The scholars attribute the effects to the seasonal differences in maternal diets and infectious disease incidence in early infancy and in pregnancy, which both improved between the cohorts; such design gives no further room to disentangle a unite biological mechanism. Suggesting the long-lasting influence of inflammation in infancy, Schellekens and van Poppel (2016) show that in the Netherlands a declining trend in infant mortality at birth and increase in height contributed to 3–5 years to rise in life expectancy at age 30 for cohorts born in 1812–1921, which is more than a fourth of overall improvements. Again, the unobserved factors driving the trends, which are not controlled, could be affecting these results. Neighboring cohorts, exposed to different disease conditions, can be also compared in their outcomes, albeit the investigator has to assume that they are similar on all aspects other than infection. For instance, Myrskylä, Mehta, and Chang (2013) follow the cohorts exposed to the Spanish influenza pandemic in 1918–1920 in the US from ages 63 to 95 and find that those exposed at late gestation and at birth experience 8% higher mortality risks compared to the later born cohorts.

A credible approach is to decompose infant disease mortality into permanent and transitory components and use the latter as a source of exogenous variation in early life conditions. Studies adopting this approach implicitly make an assumption that the determinants of short-term fluctuations in mortality are the same as those of secular trends in mortality. In the eighteenth-nineteenth centuries, the bulk of variation in both components is attributed to infectious disease mortality, and this assumption is highly valid. Pioneering the approach in the study of individuals' mortality in rural parishes of Scania, Bengtsson and Lindström (2000, 2003) examine whether the individual's mortality risks in ages 55–80 for cohorts 1760–1895 are influenced by a transitory component in regional infant mortality rate in year of birth, food prices in a year prior to birth and a year of

birth, conditional on socio-economic background characteristics. While no systematic effects are found for the food prices, high infant mortality rates in infancy, predominantly the outbreaks of smallpox and whooping cough, strongly elevated all-cause and respiratory-disease mortality risks in old age. The scholars interpret their findings as support of the hypothesis that inflammation due to infectious disease during particular time window in childhood – infancy, not nutrition during fetal stage, has long-lasting effects on old-age health. For the same area, Quaranta (2013, 2014) studies at what ages the effects of early-life exposure to infectious disease for individuals born 1813–1898 emerges, by tracing them from age 1 until 70. The scholar finds that individuals exposed to epidemics of infectious diseases in late prenatal stage and early infancy, in particular whooping cough, have greater risks of dying starting already from ages 20–49 and in ages 50–70, from all causes and from respiratory diseases, and females have worse reproductive health. These effects are summing up to 1.1– 2.1 years lost between ages 1 and 70. Similar results from exposure to high infant mortality in the year of birth were obtained for old-age mortality in nineteenth-century Belgium (Bengtsson and Alter 2007), Canada (Gagnon and Mazan 2009), and in case of Ireland resulted in around 20% of higher probability of being disabled (Delaney, McGovern, and Smith 2011).

The literature linking negative events due to infection in early life to cognitive and labour outcomes is much sparser. For the rural parishes in Scania, by applying the same approach to identifying infectious-disease conditions as in Bengtsson and Lindström (2000, 2003), Bengtsson and Broström (2009) find that for cohorts 1813–1894 high infant mortality rate in a year of birth resulted in 30% lower odds of being wealthy at age 50. For the US cohorts born 1900–1936, Case and Paxson (2009) find significant correlation between infant mortality at ages 1–2, driven by typhoid, malaria, measles, influenza, and diarrhoea, and cognitive function in ages 50–90. However, these results were not robust to adding region–specific time trends. For the US, Barecca (2010) shows that in-utero and postnatal exposure to malaria can explain up to 25% of the difference in education attainment and poverty rates between those born in high versus low malaria regions in 1900–1936.

Another more recent and sparse line of empirical research employs variations in early life environmental conditions due to societal health interventions, such as eradication campaigns or medical innovations adopted in fight against specific infectious diseases. Such studies have to carefully control for the pre-existing trends across cohorts, which instead could explain the observed differences in later-life outcomes. By using hookworm-eradication campaign in the US South in 1910, Bleakley (2007) shows that the exposure to hookworm infection below age 18 explains around 20% of gap in labour market outcomes in adulthood between high and low hookworm regions. Bleakley (2010b) finds that cohorts born after the anti-malaria campaign earned as adults around 10% (the US circa 1920) and

25% (several developing countries circa 1955) more than the previous generation. Similar or more moderate results from malaria eradication have been shown for other countries (Cutler et al. 2010; Lucas 2010; Venkataramani 2012). Beach et al. (2016) find that the elimination of early-life exposure to typhoid fever in the US cities in 1895–1920 increased labour earnings by 1% and educational attainments by 1 month. Fluoridation of water, efficient against caries and implemented in 1950s–1978 US, as Glied and Neidell (2010) show, affected female earnings by around 4%. For the US, Bhalotra and Venkataramani (2013) find that reduced exposure to pneumonia in infancy after the introduction of sulphonamides in 1937 led to 0.7 more years of schooling and 10% higher incomes of the affected cohorts. By using quasi-experimental variation in salt iodization in the 1920s US, Feyrer, Politi, and Weil (2013) find that reduction in iodine deficiency in year of birth leads to higher IQ among military recruits and equals to the decadal rise in IQ.

The beneficial impact of broader public programs in early life on adult health and economic outcomes has been also investigated. Our knowledge about the effects of such programs is scattered. Aizer et al. (2016) study the impact of the provision of cash transfers to poor families, affecting children under age 18 in the US in the 1930s and find that it improved survival in ages 60–90s by 0.7–1.4 additional years and enhanced incomes in young adulthood by 14%. Such families did not have access to public health services, so this study points to the long-lasting importance of income enhancement. Child visiting programmes, initiated in different Scandinavian countries in the 1930s, enhanced nutrition and improved disease environment and health monitoring of infants. For Norway, Bütikofer, Løken, and Slavanes (2015) find that exposure to child-care centres in infancy implied around 0.1–0.2 more years of schooling and 1–2% higher earnings by age 40, with no effects on health. Hjort, Sølvsten, Wüst (2017) find that Danish cohorts treated in infancy by a similar program are 5–8% less likely to die between ages 50–70, and are less likely to be diagnosed with cardiovascular disease, with no effects on earnings. In Sweden, as Bhalotra et al. (2016) and Bhalotra, Karlsson and Nilsson (2017) establish, treatment by a home-visiting program in infancy led to 7% lower mortality risk by age 75 and decreased mortality risk due to cardiovascular disease and cancer, and positively influenced female schooling and income at ages around 30–40 and 70. While the programmes had multiple components, the scholars suggest that preventive care, better hygiene, and nutrition were among the most influential. To date, the long-term health effects of development of qualified midwifery, connected to the introduction of antiseptic routines and disease monitoring, has not been previously investigated. Based on a contemporary developing setting, recent studies show that expansion of access to midwives, who provided healthcare services across rural villages during both the prenatal and early childhood periods, led to short-term improvements in height in

early childhood (Frankenberg, Suriastini, and Thomas 2005) and in cognitive performance in late childhood (Cas 2012).

To summarize, the research on the early-life effects of economic history and economics has been recently widened by the investigation of how policy-driven treatments in early life affect health and labour market outcomes later in life. The prior evidence is primarily based on studies exploiting negative conditions or events, where emerging studies began to show that intervening in early-life development launches long-term mechanisms. Earlier, macro-level studies established strong links between cohort factors, such as infant mortality, and adult and old-age mortality. Later on, based on microdata, empirical studies connected regional-level cohort factors to various indicators of health and socio-economic position throughout individuals' lifetimes. The effects found by these empirical studies are substantial. Due to declines in infant mortality, the cohorts born at the turn of the twentieth century have been found to gain up to one fourth of total improvements in life expectancy and real incomes in the adult ages. The effects reported in the scarce studies exploiting public interventions, more systematically conducted for the cohorts born close to the mid-twentieth century, are of a similar magnitude. There is also some evidence that due to these programs, mortality declined, especially for cardiovascular diseases. Yet, our knowledge of the lasting impact of policy initiatives targeting disease environment around the time of acceleration of decline in infant mortality and throughout the path of its rapid decline on health and income is absent.

Sweden as a laboratory of health and economic development

Long-run development of health and income

In the above discussion, the late nineteenth–twentieth century experience of Sweden in health and economic development has been mentioned with reference to its profound increase in life expectancy and economic growth, representative of the Nordic countries, and to active provision of public health and medical innovations, representative of the countries of West Europe and North America. Two studies in this thesis (Paper 1 and Paper 2) were conducted for a rural area in Scania (southern Sweden), and two other studies (Paper 3 and Paper 4) are based on the whole of Sweden (see Figure 4). I first present the survey of the contextual information related to the issue studied in more detail for Sweden in general, and later on – for Scania.

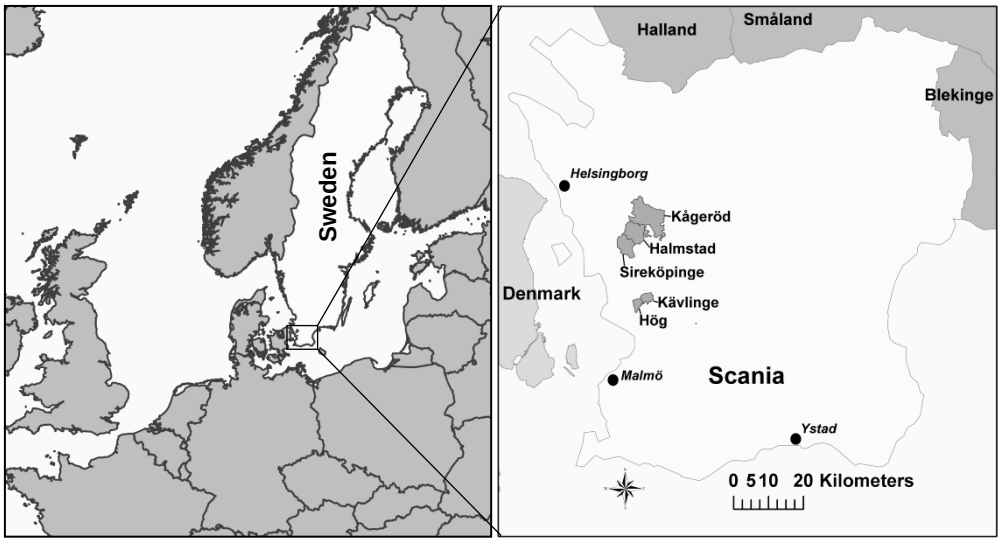


Figure 4 – Maps of Sweden (to the left) and parishes in Scania (to the right)
Source: Hedefalk, Harrie, and Svensson (2017)

In Sweden, economic development had accelerated by the last quarter of the nineteenth century. According to recent estimates, in Sweden real GDP per capita grew constantly at an annual rate of 0.5% between 1800 and 1869, and between 1870 and 2010 overall growth amounted to around 2%, and its rate of growth was increasing over time (Schön and Krantz 2012). The last subsistence crisis occurred in Sweden in the late 1860s (Sandberg and Steckel 1997). Among the developed countries, Sweden experienced a rather late but rapid industrialization (Jörberg 1994). Beginning in 1880, employment in industry has increased from 15 to 35 per cent, although by the 1920s approximately half of the population still worked in agriculture (Statistiska Centralbyrån 1969). In the following decades, the Swedish manufacturing sector became dominant and services witnessed an expansion. The same development is observed in the real wages of workers, which began to gradually increase in 1880, and accelerated in the middle of the twentieth century (Jörberg 1972; Prado 2010). Plotting real GDP per capita versus life expectancy at birth in 1800–2010 for Sweden demonstrates a positive relationship between the phenomena. It is most profound for the period 1880–1950 (see Figure 5). Urbanization followed a similar pattern. The urbanization rate was slow prior to 1890 and afterwards amounted to 2 per cent per year (Statistiska Centralbyrån 1999). Despite this increase, by the first half of the twentieth century, the majority of the Swedish population was still rural. Industrialization in Sweden had a nonurban location; the majority of industrial workers were employed and lived in rural or semi-urban industrial locations. According to international standards, Swedish cities were small, with the population of the biggest cities, such as Stockholm and Gothenburg, not more than 500,000, which accounted for less than

one-tenth of the total population. The share of population that resided in the countryside, and therefore outside cities or semi-urban locations, declined only from 80 to 70 per cent between 1890 and 1920 and to less than one-half of the total by 1960 and to one sixth of the total by 1995 (Statistiska Centralbyrån 1999).

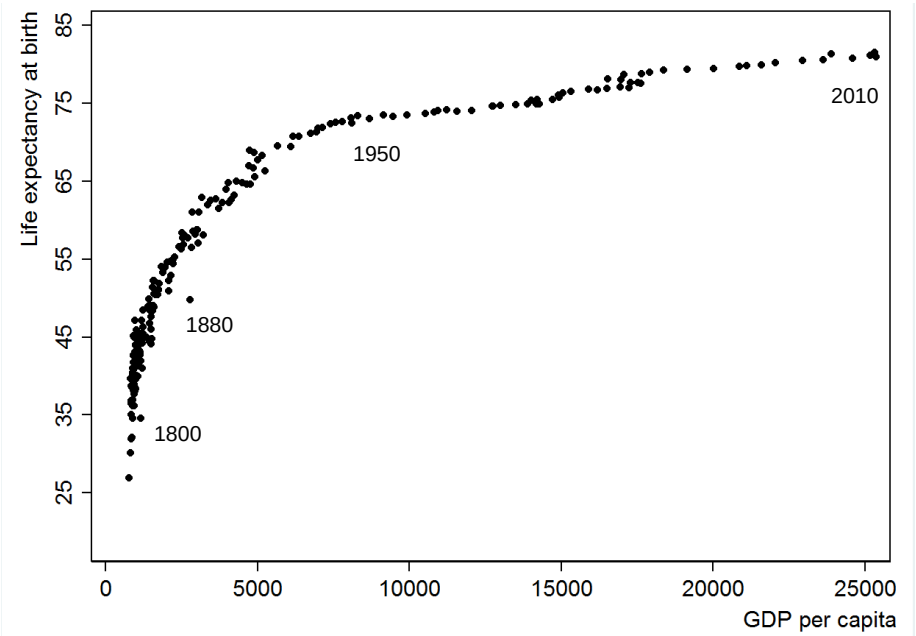


Figure 5 – Preston curve for Sweden 1751–2010: life expectancy at birth and real GDP per capita (1990 international Geary–Khamis dollar)
Sources: The Maddison-Project (2013); Human Mortality Database (2017)

The improvements in population health in Sweden exhibited a similar pattern, although they began earlier. Life expectancy at birth increased slightly until the mid–nineteenth century, and up until 2015 it increased almost linearly from age 42 to 82 (Pliktverket 2000; Human Mortality Database 2017). The Swedish population increased rapidly, from approximately 4.2 to 5.9 million between 1880 and 1920, and doubled by the turn of the century (Statistiska Centralbyrån 1999). Prior to the first quarter of the twentieth century, such rapid population growth was largely a result of declining death rates, mainly among children and infants, from airborne and foodborne infectious diseases such as whooping cough, measles, diphtheria and diarrhoea (Preston, Keyfitz, and Schoen 1972; see Table 1). Afterwards, respiratory diseases, such as pneumonia and influenza, accounted for not less than one fifth of premature deaths below the age five in the 1920–1930s, and the major cause of death among infants and the major infectious-disease cause of death among children. Between 1880 and 1950, the infant mortality rate declined dramatically, from 129 to 72 per 1000, and death rates in

the 1–15 year-old population fell even more rapidly, from 13 to 4 per 1000 children (Statistiska Centralbyrån 1999). The Swedish government in 1929 presented a report on public health insurance that in part demonstrated the large inequalities in infant and child mortality across regions, urban and rural areas and socio-economic classes (SOU 1929; Wisselgren 2005). Between the mid–1880s–1930s, rural infant mortality declined by 70%, urban infant mortality converged to its level, and both stagnated for a decade and dropped again from the 1940s (Statistiska Centralbyrån 1999). Following infectious death rates, non-infectious death rates also began to decline from the 1970s (van Hofsten and Lundström 1976). In the second half of the twentieth century, the improvements in life expectancy have been attributed largely to the decrease in death rates among the working population and the elderly. With regard to the causes of death, in this period the most dramatic reductions were witnessed in mortality rates from pneumonia, degenerative diseases of organs and tissues, and cardiovascular diseases.

Table 1 – Age- and cause-specific death rates per 100,000 for selected years, 1820–2000

	1820	1850	1880	1911	1951	1980	2000
<1							
Infectious diseases	n/a	n/a	n/a	6008	1646	574	271
CVD/cancer/degenerative	n/a	n/a	n/a	1446	470	109	69
Accidents	n/a	n/a	n/a	68	36	12	4
All causes	19017	16808	12734	7521	2152	695	345
1–14							
Infectious diseases	n/a	n/a	n/a	324	13	5	3
CVD/cancer/degenerative	n/a	n/a	n/a	135	37	10	6
Accidents	n/a	n/a	n/a	34	25	10	3
All causes	1549	1130	1299	493	76	25	12
15–44							
Infectious diseases	n/a	n/a	n/a	342	30	6	3
CVD/cancer/degenerative	n/a	n/a	n/a	167	79	49	33
Accidents	n/a	n/a	n/a	61	41	52	31
All causes	985	818	668	570	149	107	67
45–74							
Infectious diseases	n/a	n/a	n/a	446	102	62	52
CVD/cancer/degenerative	n/a	n/a	n/a	1461	1306	1215	723
Accidents	n/a	n/a	n/a	84	69	79	48
All causes	3733	3072	2382	1991	1476	1356	824
75+							
Infectious diseases	n/a	n/a	n/a	1295	914	662	784
CVD/cancer/degenerative	n/a	n/a	n/a	10458	11128	8549	7387
Accidents	n/a	n/a	n/a	154	320	312	218
All causes	17637	15984	14530	11907	12362	9523	8388
All ages combined							
Infectious diseases*	210	198	135	114	8	4	1
Influenza/pneumonia/diarrheal	2160	1998	1632	405	96	74	50
CVD/cancer/degenerative				797	828	958	955
Accidents	76	52	60	61	56	69	48
All causes	2446	2248	1827	1377	987	1105	1054

Sources: calculations based on data from Preston, Keyfitz, and Schoen (1972); van Hofsten and Lundström (1976); Statistiska Centralbyrån (1982-1983, 2013); Human Mortality Database (2017); data for both sexes combined.
Note: *In 1820–1880, infectious diseases recorded include smallpox, measles, scarlet fever, whooping cough, malaria and dysentery, and the rest infectious diseases are recorded in the category of non-infectious diseases. In 1911–2000, influenza, pneumonia, bronchitis, respiratory tuberculosis, diarrhoeal diseases, maternal diseases and certain diseases of infancy are allotted into a separate category.

Provision of public health and antibiotics

The Swedish authorities recognized the need for the provision of public health in the nineteenth century. After the establishment of the national vital statistics in 1749, the medical board produced several reports about mortality in the country and underlined the necessity to prevent deaths from smallpox and other infectious diseases (Johannisson 2006). Driven by the need to save the labour force, in the beginning of the nineteenth century, the government set up the foundation for public health care, which introduced compulsory vaccination against smallpox and obliged the parishes to open poorhouses. Local hospitals and physicians received much less focus in these measures, although their free public offer became assured. However, with regard to the overall panorama of infectious diseases, any public measures before the 1880s remained responsive. For many years, under the fear of epidemics spreading to neighbouring countries, the authorities imposed quarantine regulations and the inspection of cargo, in addition to mobilizing medical practitioners (Bourdelais 2002). In the mid-nineteenth century, such initiatives covered only the major Swedish towns, which by that time were equipped with hospitals and medical personnel. However, the epidemics appeared to resist any broad and costly quarantine and surveillance efforts in the urban localities and killed many more in rural areas, where no measures were in place (Niemi 2007). In 1862, a local government reform established county councils, with one extra provincial doctor per each, as a complement to the central administration that took over responsibility for hospitals (Lindblom 1967). In 1874, the public health movement expanded to each Swedish town and village: a new act on public health required every commune throughout Sweden to establish a local health committee that was obliged to combat poor sanitary conditions (Kullberg 1877). This meant a crucial change in the Swedish public health system that was dominated by acute medical care, and inefficient due to the absence of drugs. In the cities, the duties of the public health committee primarily focused on improvements in water supply and sewage in 1875–1935, whereas in the countryside any important efforts of the board covered public health care and local infrastructure. By disentangling the mode of transmission of infectious diseases and the causal agents, international bacteriological discoveries in the 1870–1880s helped to target public efforts and provided tools to combat disease (see Table 2). Compared to the broad and radical public health measures, the targeted initiatives were less costly and more realizable.

Table 2 – Investment into health care and medical personnel, Sweden 1855–1944

Year	Midwives per 1000 born, urban	Doctors per 10,000 population, urban	Midwives per 1000 born, rural	Doctors per 10,000 population, rural	Public health care investment per capita, total (SEK 1900=100)	Pharmacies per 10,000 population, total
1855*	37,0	1,0	11,9	0,5	0,7	0,4
1865	27,5	0,9	10,7	0,3	0,8	0,5
1875	19,7	1,9	14,6	0,6	0,9	0,5
1885	18,3	1,6	16,5	0,5	1,5	0,5
1895	24,1	1,4	17,8	0,7	2,5	0,6
1905	22,8	1,3	20,3	0,8	3,8	0,7
1915	22,7	1,2	24,6	0,7	6,1	0,7
1925	23,4	4,1	28,3	1,1	9,8	0,7
1935	19,1	5,0	29,4	1,4	13,7	0,7
1944	9,1	6,0	15,1	1,6	20,1	0,7

Sources: calculations based on data from Statistiska Centralbyrån (1855-1944; 1855-1910; 1856-1905; 1880-1917; 1911-1944; 1918-1944)
Note: *Stockholm instead of urban in 1855

Between the 1880s and mid–1930s, in the absence of efficient cures for infectious diseases, except for drugs introduced to treat syphilis and diphtheria, public health efforts focused on the prevention of the spread of disease. Inspired by fear of cholera, a state law in 1893 required each commune to prepare hospitals that could serve for isolation of the sick (Svensk författningssamling 1893). In parallel, the number and quality of medical personnel, including provincial doctors, medical nurses and qualified midwives improved markedly after the 1890s in the rural parishes. By the 1880s, the skills of licensed midwives were extensive, covering childbirth, the use of obstetrical instruments, supportive treatment of newborns, child and maternal health and related advice (Romlid 1998). In the late 1870s, thanks to bacteriological discoveries, careful instructions about antiseptic and aseptic techniques, including eye treatment, were introduced in hospitals, and in 1881 they became obligatory at home deliveries in rural areas (Svensk författningssamling 1881). Around the turn of the century, public policies not only increased the overall availability of midwives but also shifted the share of employed midwives towards competent ones. From 1890 until the 1920s, all communities in Sweden gradually received access to public health care in the form of local health districts, which are organized around an assigned doctor, midwives and a hospital. Until the mid-nineteenth century, the number of centrally introduced health districts amounted to 2 per 100,000 inhabitants and disproportionately covered the more urbanized locations (Medicinalstyrelsen 1907). The location-initiated creation of health districts accelerated due to institutional changes beginning in the 1880s. However, without any government subsidies, between 1840 and the late 1880s, the process of expansion of provincial doctor districts was sluggish and favoured wealthy and industrialized locations (Medicinalstyrelsen 1907). In 1890, Swedish state authorities began to subsidize the establishment and maintenance of medical districts in all parts of the country, giving this opportunity equally to economically disadvantaged areas, and to stimulate the graduation of young medical professionals and attract them to rural

parishes. In addition to improved midwifery, the newly assigned provincial doctors became responsible for the monitoring and isolation of infected persons from the rest of the parish population (Engberg 2009). The cottage hospitals or health stations in the parishes were built for this reason, whereas chronic patients for many years were transported for in-patient care to neighbouring cities. At a minimum, the 1890 reform doubled the access of the population to public health care. This system stayed unchanged until the late 1940s, when together with the introduction of the maternity hospital system, the duties of public midwives evolved into prenatal and infant care (Sundin and Willner 2007).

In the mid–1930s–1940s, as in other Western countries, the first antibiotics – sulphonamides – were introduced as treatments of infectious disease across Sweden. As mentioned before, pneumonia dominated the causes of child deaths in the 1920–1930s, but abundant empirical research in medicine that was carried out worldwide advanced little in providing an effective cure. In the beginning of the 1930s, the practice to treat pneumococcal pneumonia with antiserum, that is, immunological therapy, had been approbated by the hospitals of Stockholm, similar to the practice in large hospitals in other countries (Rahm 1939). However, any intention to implement such procedures throughout the country proved elusive due to the method being labour intensive, hospital-dependent and uncertain in efficacy (Ingvar 1939). The efficiency of sulphonamides in treating many experimental streptococcal and other infections was observed by Domagk in Germany in 1932 (Domagk 1957). The production and trade of the drugs on a mass and international scale started by the end of the 1930s; the greatest success is attributed to sulphapyridine (a compound of pyridine and sulphonamides), prepared in the UK and a very efficient treatment for pneumonia (Bentley 2009). In Sweden, the sulpha antibiotics, initially imported and later produced by the national pharmaceutical companies, were introduced into medical practice sharply after the medical review in 1939 and granting of international recognition to their inventor. Therapy with sulpha drugs was not only easy to administer, but also cheap, amounting to 150 SEK (in SEK 2016) for the full treatment course for an infant compared to 300–400 SEK that had to be spent on antiserum, an alternative, immunological treatment (Rahm 1939). Due to the high availability of medical personnel, centralization of drug distribution and low costs of treatment (Hollingsworth, Hage, and Hanneman 1990), this medical innovation spread quickly across regions of Sweden and led to regional convergence in death rates from the targeted diseases, namely pneumonia. Until the development and introduction of penicillin in the late 1940s, sulpha antibiotics remained the prime cure against infections. The vaccine against pneumococcal bacteria was developed only in the 1980s.

Parishes in Scania as a rural representative

Two studies in this thesis (Paper 1 and Paper 2) are based on microdata from five parishes in Scania, and this section provides background information about the areas and discusses whether these parishes are representative of rural localities in Sweden.

Socio-economic and health development in Scania

Between 1880 and 1940, the parishes under study could be considered rural and rural-located industrial areas. The five parishes – Hög, Kävlinge, Halmstad, Sireköpinge and Kågeröd – are located in close proximity to each other and to the west of Scania, which is the southernmost province of Sweden. In terms of geography and climate, Scania differs from the rest of Sweden: its landscape is generally not mountainous and open and the climate is far less cold during the winter. The fertile land made the Scanian region the granary of Sweden; by 1900, mechanization was introduced into agriculture across the whole of Sweden and most extensively in this region (Moberg 1989). The sampled parishes possess these natural features. The nearby towns, which surrounded the parishes at a distance of between 10 and 30 km, are small. The five parishes comprised around 2,500 inhabitants in 1766 and by 1880 had increased to around 5,100 inhabitants, resulting in a 0.6% annual growth rate of population that is somewhat lower than that for other rural parts of Sweden (Bengtsson and Dribe 2010). In 1895–1915, one of the southern parishes – Kävlinge – became partially industrialized, something which affected the neighbouring Hög as well. During this period, due to the building of several factories and construction of a railway, the population in Kävlinge increased rapidly from 600 to approximately 2,900 inhabitants, primarily due to the inflow of industrial workers. During the same period, Kågeröd also experienced an increase in the number of immigrated workers, albeit in more moderate terms. By 1940, the parishes had grown to around 6,400 inhabitants. Similar to other rural industrializing parts of Sweden, the manufacturing and service sectors grew rapidly and the employment structure changed in the Scanian parishes, with a higher proportion of supervisors and skilled industrial workers and lower proportion of farmers (Quaranta 2013). Unlike other parts of Sweden, the rural area where the five parishes are located did not experience the large-scale migration, primarily to the US, that peaked in 1870–1900 and continued until the 1920s (Bengtsson and Dribe 2014). Real wages began to increase rapidly in the mid–1860s, and since then have witnessed a substantial increase at an average rate of 1.8%, following the pattern of real wages for the whole of Sweden (Bengtsson and Dribe 2005; calculations based on Bengtsson et al. 2014).

The five parishes in Scania have similar health development to the whole country, especially the rural areas. In 1880–2010, life expectancy at birth followed the same development as in Sweden as a whole, but was about one year higher (Bengtsson and Dribe 1997; Quaranta 2013; calculations based on Bengtsson et al. 2014). The linear trends in heights in 1880–1968 for the Scanian parishes and for Sweden are almost identical (Öberg 2014). Historically, the infant mortality rate had been lower in southern Sweden for reasons unknown (Brändström, Edvinsson, and Rogers 2002). The development of infant mortality differs slightly from the national pattern. More specifically, a steep decline in infant mortality, with short term upswings, prior to 1870 is followed by a levelling-off at a rate around 100 per 1,000 infants up until the mid–1910s, plausibly related to the compositional changes due to the inflow of population, and then by decrease by two times. After around 1880, the child mortality rate, mainly due to the decline in mortality from airborne and foodborne infectious disease, such as whooping cough, measles, diphtheria and diarrhoea, plummeted with almost no interruptions and reached a level of 2 per 1,000 children up to 1920, followed by a less rapid decline thereafter. For cohorts born from 1881–1930, cohort life expectancy at birth in general develops similar to the national pattern but is higher by one year on average (see Figure 6). Partially explained by the influence of migration on such a small area, it likely represents rural cohort life expectancy that outcompeted that in urban areas due to the lower infant mortality rates (Statistiska Centralbyrån 1969). The Scanian and overall Swedish mortality had an identical cause-of-death composition (see Figure 7). Both levels and development of mortality in working and older ages resembled those of Sweden in total (calculations based on Bengtsson et al. 2014). From ages 15–39, in 1895–1950 mortality was dominated by infectious diseases, including diarrhoea and pneumonia, whereas afterwards chronic diseases such as cardiovascular disease, diabetes and cancers took the largest toll of deaths. Mortality from ages 40–80, which was declining throughout the twentieth century, was dominated by deaths due to chronic diseases, among which CVD and diabetes were the leading causes (calculations based on Bengtsson et al. 2014).

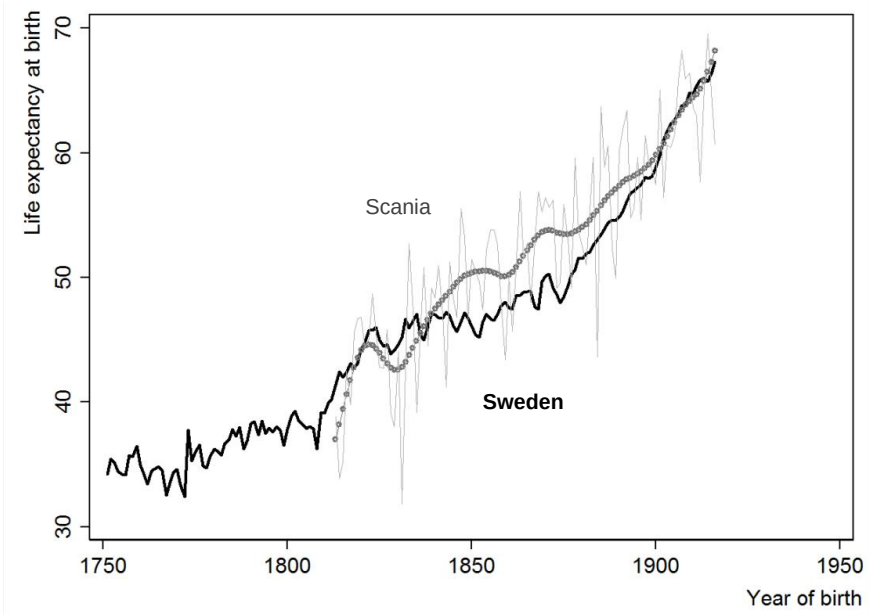


Figure 6 – Cohort life expectancy at birth in Sweden and Scanian parishes, cohorts 1751–1917
*Source*s: Sweden: Statistiska Centralbyrån (2010); Human Mortality Database (2017); Scanian parishes: calculations based on data from SEDD version 4.0.

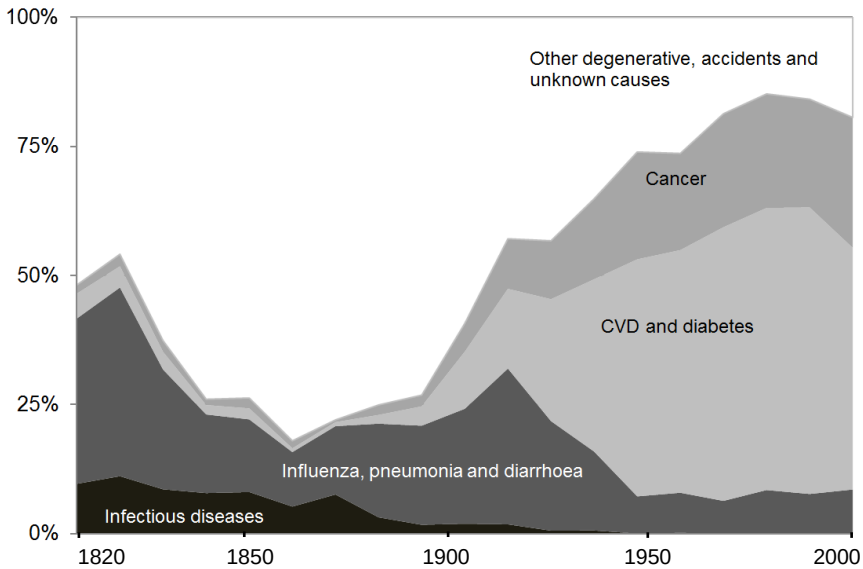


Figure 7 – Deaths by cause as proportions of all deaths, Scanian parishes, cohorts 1813–2004
Source: calculations based on data from SEDD version 4.0.
Note: 'Infectious diseases' recorded include smallpox, measles, scarlet fever, whooping cough, malaria and dysentery. Influenza, pneumonia, bronchitis, respiratory tuberculosis, diarrhoeal diseases, and maternal diseases are allotted into the category 'Influenza, pneumonia and diarrhoea'.

Local government and public health in Scania

The parishes in southern Sweden underwent the same institutional and public health changes as other rural parts of Sweden in the 1860s–1940s. The county council administering the area was established in 1863 and resided in the city that was located south of the studied parishes, Malmö. As in other areas of Sweden, local authorities in the southern region had to establish the local health boards responsible for sanitary conditions according to the 1874 Health Act. Until 1889, only one provincial doctor took care of the rural parishes in the county. According to a decision taken, separate district doctors were introduced gradually thereafter, 6 in total, each for 8–12,000 inhabitants (Lindblom 1967). In 1889, a local doctoral district serving three out of five studied parishes – Halmstad, Sireköpinge and Kågeröd – was introduced in the neighboring parish, Teckomatorp; in 1900, a separate doctor's district was opened in other parishes – Kävlinge – that began to administer Hög as well. Major improvements with regard to public health management came into place here around that time. A local health board was obliged to make an annual report to the provincial doctors concerning the general health conditions in the parishes, including employment of additional medical personnel, measures with regard to isolation of the infected, and food and water supplies. In the 1890s, however, the local health committee of the Malmöhus county began to subsidize the health care service in the rural parishes, in the form of provincial doctoral districts, isolation hospitals, and quantity and quality of the assisting medical personnel. Modern medical knowledge – through better trained medical nurses and midwives with knowledge about the use of antiseptics and isolation from the infected – did reach the rural population of the parishes under study in the 1890s–1900s. In the 1940s, the area witnessed other public health interventions such as improvements of water supply, control of the sanitary conditions in factories, grocery stores, and animal husbandry, the use of antibiotics as well as spread of in-patient care including hospital birth deliveries.

In 1880–1930, similar to other rural areas, the number of trained midwives in the Scanian parishes increased, although the composition of birth attendants stayed diverse. From 1819, the law obliged cities to employ trained midwives but allowed traditional birth attendants when a licensed midwife was not available in rural parishes until 1908 (Högberg 2004). In the beginning of the 20th century, in large cities almost all deliveries were carried out in lying-in and maternity hospitals, whereas in small cities and villages they took place at home up until the 1940s (Curtis 2005). Local cottage hospitals, which were established in the area, served for the isolation of the sick and their supportive cure, but never admitted women in labour. Only the women with the most severe complications during childbirth (a few cases per year) were transported to the maternity hospitals in the neighbouring cities. For a long period, throughout rural Sweden, the majority of women delivered their children with the assistance of traditional birth attendants whose

experience was otherwise greatly recognized by the population, district medical officers and the clergy. For instance, after the establishment of provincial doctoral districts in the 1890s, newly assigned doctors from different parts of the country documented the presence of numerous traditional practitioners working throughout the areas but regarded them as a common feature of rural life (Romlid 1997). Their knowledge in disinfection and after-delivery care and skills in the use of obstetrical instruments, however, were not sufficient. For instance, the newly assigned provincial doctor in Kävlinge in 1900 indicated that the midwives working in the district were 'incompetent in the use of antiseptics and modern knowledge' and they rarely used instruments (Regionarkivet Lund 1900-1946). The 'qualified' midwives also worked in the parishes under the supervision of the local doctors, and had formal training either in manual childbirth or in addition in the use of obstetrical instruments, and had the related license to conduct these duties. These qualifications could be obtained in the midwifery schools in Stockholm beginning in 1792 and in Gothenburg beginning in 1856; a midwifery school in Lund, established in 1783 to meet the need to cover southern Sweden, was accorded only to the right to teach basic courses (Högberg 2004). Despite this, the majority of graduated midwives found employment in the cities, and met a demand in the countryside. Due to the proximity of the parishes to Gothenburg and Lund, where the midwifery schools provided only training in manual childbirth, the proportion of midwives with a license to use instruments and thus equipped with pain-killing drugs and disinfectants, was at least two times lower than in other parts of Sweden (calculated based on Statistiska Centralbyrån 1855-1944). Up until the extension of the maternity hospital system in the late 1940s, when the deliveries could be taken in hospitals located in the neighbouring cities, such as Helsingborg, Landskrona, Lund and Ängelholm, rural midwifery remained diverse in core qualifications.

To summarize, despite the differences in geography, climate and soil productivity, the parishes in Scania under study witnessed similar economic and health development throughout the twentieth century. The new public health institutions of the 1880s–1940s were established strictly following state regulations and were representative of those in the countryside throughout Sweden.

Methods

As the above outline of the empirical studies demonstrates, the empirical economic and historical literature of recent decades had an emphasis on designing studies that could potentially disentangle causal pathways, either by applying quasi-experimental designs or applying non-experimental designs that could avoid the influence of important confounding variables. Compared to the macro data, the use of individual-level data already provides better identification, as health and labour outcomes can be precisely matched to the differences among individuals (Weil 2013). In light of the reviewed literature, testing the critical period and inflammation hypotheses require careful design of the studies regarding the environmental and intervening factors. It also requires careful evaluation to identify whether these associations are independent of other early life exposures. This section presents the most prevalent empirical strategies used in the recent literature further enabling randomization of these differences. It further presents the methods applied in the studies of this thesis.

Quasi-experimental methods used in the literature

The majority of studies have applied a quasi-experimental design by exploiting plausibly exogenous variation in the economic or health environment surrounding individuals. Obviously, background socio-economic information about the individual (e.g., distinctions between poor and wealthy families that differ in exposure to pathogens and nutrition) cannot be instrumental as early-life experimental conditions, as it by itself to some extent is determined by unobserved factors that affect health and labour market outcomes of individuals at adult and old ages. Instead, one could rely on the group (geographical)-level variation in the timing of negative disease outbreaks, episodes of malnourishment or policy. One way of implementing this approach is to compare individuals who lived in the same area but were born in different years, and who therefore were exposed at different ages to sharply occurring epidemics, in the form of mild or severe insults. Such environmental conditions 'are exogenous from an individual point of view' (Bengtsson and Mineau 2009; van den Berg, Doblhammer, and Christensen 2009). Another approach, more relevant from the viewpoint that motivates this thesis, is to compare the outcomes of individuals both across cohorts and across geographic units, who are similar but were differentially exposed to certain health policies at some point in time. One of the most appropriate methods is difference-in-differences (DD) estimates; fixed effects, which generalize DD estimates when there are more than two time periods (year of birth) or more than two treatment groups, may be equally applied (later I refer to it as to the DD method). This

empirical strategy is attractive, as it cancels out the potentially confounding differences across groups (e.g., broad institutions, geography or climate) and across cohorts (e.g., improvements in nutrition and living standards). In applying the DD method, it is important to assure that the implementation of the health policy or innovation in certain regions is not driven by the observed and unobserved characteristics of the regions, which could potentially lead to the same outcomes (Rosenzweig and Wolpin 2000). Strauss and Thomas (2008) highlight that the choice of the correct comparison groups is especially important for the analysis of the long-term effects of public health policies which are usually targeted to problematic regions or, otherwise, the wealthy regions where it is easy to implement the policy due to the available infrastructure. To assure the validity of the method, such differences across the parishes should be therefore identified and properly controlled.

The contemporary early-life empirical studies set additional requirements for the non-experimental methods. Based on controlling for observable characteristics, the studies could still make use of the abundant background information, especially if it covers many socio-economic and demographic dimensions. One can put them into the model and obtain the average effect weighted across the various possible values of controls, or implement matching on probabilities of being assigned to the treatment groups based on these controls ('propensity scores'). As Duflo, Glennerster, and Kremer (2008) point out, the validity of the assumption that the control variables contain 'all relevant differences between the study groups must be evaluated from study to study, but in many situations the variables that are controlled for are just those that happen to be available in the dataset, and "omitted variable" bias remains an issue'. Another method that can eliminate the influence of unobserved characteristics at a family (mother) level, such as those from shared family background, is sibling fixed effects (mother fixed effects) estimation. However, with regard to the studies in question, it should be used with caution, as family conditions (e.g. maternal health) are likely to change across siblings, or parents may invest differentially in the health of each sibling dependent on his/her health endowments in early life (Almond and Currie 2011). The family-fixed-effects estimates should be interpreted with these potential influences in mind. Finally, one can improve the methodology by employing the variables that correlate with early-life environmental differences under study and do not correlate with unobserved characteristics ('instruments'). For instance, regional-level variables were successfully used as instruments in previous research (Brookhart and Schneeweiss 2007). Under certain assumptions, the instrumental variables (IV) methods that could be further implemented provide the effect for individuals whose environmental conditions were affected by the instrument. One such basic assumption is testable (IV is strongly related to exposure), whereas the other ones, such as conditional independence (IV is independent of confounders) and exclusion restriction (IV has no direct effect on outcomes) are not verifiable.

When environmental differences are identified at a geographic level, the study only allows for obtaining reduced-form ('Intention-to-Treat') estimates. This occurs because in fact the researcher does not observe which individuals are at risk of an environmental shock or which are affected by it. However, the gains could be rescaled by the participation rate, giving us the Wald estimate (which is the IV estimate), where there is only one single solution to get it from the first stage and reduced form coefficients. From the viewpoint of the thesis, this empirical strategy might be more important because, by defining the 'complier' group, the researcher is eager to disentangle the size of the impact of treatment delivered a particular channel (cf. Duflo, Glennerster, and Kremer 2008).

Methods used in the thesis

The studies in this thesis have applied a quasi-experimental design. In three studies, it is based on a comparison of the groups exposed differentially to public health and medical interventions by date and region of birth, which predisposes the use of DD as an empirical method. One paper is based on an adjusted comparison of the individuals for whom individual-level treatment status is known, and also exploits variation in the number of qualified midwives employed at the commune level across cohorts and regions of birth as an instrument of this individual-level treatment; thus, it uses an IV empirical method.

Aiming to explore the permanent effects of public health interventions on mortality, Paper 1 examines changes in mortality risk due to establishment of isolation hospitals and employment of qualified midwifery over a decade by comparing changes in the risk experienced by children living in treated and non-treated parishes. While applying non-experimental design as the main empirical strategy to analyse the long-term effects on health, such as controlling for a variety of background childhood characteristics, Paper 2 in addition applies an IV approach. The plausibly exogenous variation in individual treatment by midwifery is derived due to the sharp increase in employment of qualified midwives across parishes and over time. The mother-fixed-effects estimation is also utilized, although in this case the within-mother variations in treatment status at birth are likely to reflect that some unobservable maternal characteristics changed over time, such as health during pregnancy or delivery conditions. In a DD specification, Paper 3 examines the long-term effect of the reform by comparing health and income outcomes of individuals who received access to better health care in infancy compared to those who were aged 1 to 5 at the time of the reform implementation. As a robustness check, it compares the individuals actually treated in infancy with those born in the same year and in a 'twin' parish that were not treated, identified by means of propensity score matching. In order to investigate the impact of infant health on a variety of later-life outcomes, Paper 4

exploits the plausibly exogenous variation due to the decrease in mortality from pneumonia in an individual's year and region of birth induced by sudden arrival of sulpha antibiotics in Sweden in 1939, comparing individuals exposed in infancy to those exposed at ages 1 to 5. The sharp changes in mortality from diseases other than pneumonia, some being pervious to sulphonamides and some not, are controlled for in order to differentiate the effects of the drugs from the effects of other factors (demographic, health care and socio-economic) that may have reduced pneumonia mortality beginning from 1939. This is a methodological advantage of studying the effects of introduction of sulpha antibiotics, because the later antibiotics such as penicillin, introduced in the late 1940s, affected mortality from all infectious diseases. In all papers, additional specifications introduce controls for pre-treatment region-specific trends based on observable and unobservable characteristics, as well as parental information. Finally, all reduced-form effects are rescaled by the participation rates, defined for instance based on the fraction of infants treated by qualified midwifery or decline in pneumonia mortality due to the arrival of antibiotics.

Data

This section first introduces the data used in the most recent empirical literature, and then presents the datasets and samples used in the studies of this thesis.

Requirements to the data

Regarding the investigation of instantaneous and lasting outcomes of early-life health interventions, the availability of the individual-level data is necessary but not sufficient. Obviously, in addition to general demographic and cause-of-death information, such a dataset should contain information on the socio-economic background of a child, primarily in order to investigate and control for the self-selection of the family into the treatment. To suffice for the implementation of the method, it additionally requires the collection of detailed information about health policy or other environmental shocks (e.g. infant mortality rate or timing of the reform), possibly from several historical and administrative sources, accompanied by their thorough examination. Further challenges pursue the investigation of long-term effects of the environmental shocks. An attempt to follow individuals in their outcomes requires data on long time spans, which enables one to observe the effects in the ages when these effects, while being latent at younger ages, can potentially reveal themselves (Bengtsson and Mineau 2009). One way to approach this challenge is to rely on historical datasets. Albeit limited in area coverage and

sample size, they not only possess important individual-level information, such as household- or community-level circumstances surrounding birth and childhood and, uniquely, information about individual-level policy treatment, but also refer to particular historical settings that provide precision to the underlying early-life mechanisms (Lawlor 2008). Another way is to use prospective information from the register data, typically available from the late 1960s in the Nordic countries. Based on place and time of birth, information on the reform implementation can be easily merged with these data. The fact that the data are left-truncated imposes the need for discussion of selection into survivorship due to pre-term death or out-migration. Mortality selection should be discussed with regard to the severity of the exposure (morbidity versus mortality), as no data on stillbirths or deaths in the population under study before the start of observation are available. In case of right-censoring, for instance due to out-migration, it is possible to tentatively test it. As Almond and Currie (2011) highlight, when in addition the existing data merges administrative records from multiple sources at the individual level through personal identifiers, this data provides additional merits in studying both health and socio-economic outcomes of early-life environmental conditions and underlying biological and economic mechanisms.

Data used in the thesis

Following the guidelines of the methodological literature, the studies of the thesis rely on two individual-level register-type longitudinal datasets – with local and national coverage – which are complementary in approaching the issue. They both are hosted at the Centre for Economic Demography (Lund University, Sweden). The local database is unique in its characteristics, in both the age depth and details available, and allows short- and long-term follow-ups in various outcomes. The use of the local dataset, with unique information on individual characteristics and medical treatments and a lifetime follow-up, diminish concerns about the confounding variables acting in early life or in adulthood. However, a follow-up into adulthood in economic outcomes was not sufficient to conduct analysis that strongly motivated the use of the national individual-level data. In addition, the use of the national dataset widens the external validity of the results and, combined with archival data, makes the investigation of policy treatments identified at the regional level possible.

The local dataset (from which samples are used in Paper 1 and Paper 2), the Scanian Economic Demographic Dataset (SEDD), provides full individual demographic and socio-economic information for the total population residing in 1646–2012 in five rural parishes in southern Sweden (Kävlinge, Hög, Halmstad, Sireköpinge and Kågeröd) and followed until old age. The database contains records on all individuals who were born in or immigrated into the five parishes

until their death or out-migration. The main source of the data is catechetical examination registers with information on demographic events, which have been checked against underreporting with data from church books, and linked to tax registers that provided annual information on occupation of the family head and landholding. The quality of the data is very high. The source material is described in Reuterswärd and Olsson (1993), and the quality of the data is analyzed in Bengtsson and Lundh (1994). The data in the SEDD followed individuals within five parishes until 1968 and for 1968–2012 – regardless of an individual's geographical location in Sweden, based on information merged through personal identifiers from various Swedish registers by Statistics Sweden.

Another dataset (used in Paper 3 and Paper 4), the Swedish Interdisciplinary Panel (SIP), provides information compiled from multiple Swedish administrative registers, such as population movement, income and taxation, occupation, inpatient and outpatient visits, and education, for all individuals who are alive and have not migrated permanently from Sweden between 1968 and 2012, with further restrictions regarding belonging to the cohorts born before and after 1930. For cohorts born before 1930, the dataset is restricted to those having a family member (sibling or child) who is born from 1930 onwards; for cohorts born between 1930–1980, the database contains all born individuals. Figure 8 compares the cohort life expectancy at age 78 for cohorts born from 1890–1917 based on data from SIP and for the country as a whole. The life expectancies follow a similar development, with absolute difference between the two amounting to 0.074 years, which is diminutive; the pattern is the same for other ages. This comparison indicates that for cohorts born from 1890–1917 SIP should be representative of the country. SIP provides parental information and family links, accessed from the mutigenerational register, for the cohorts born from 1932 onwards. In all studies, to investigate the progression, quality and outcomes of the reforms under study, additional archival and statistical sources have been gathered at a geographical level and merged to individual-level data through the parish and date of birth.

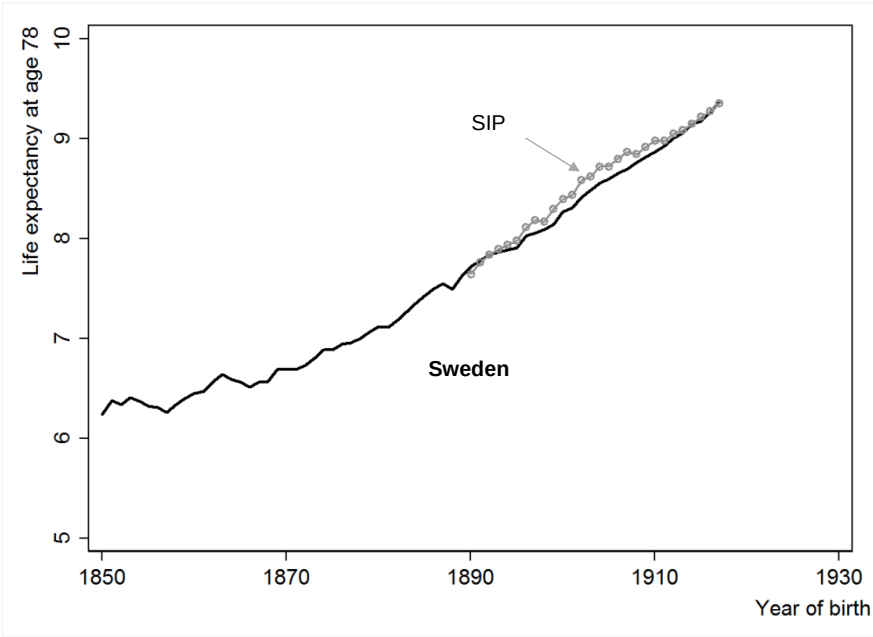


Figure 8 – Cohort life expectancy at age 78 for data from SIP and Sweden, cohorts 1850(1890)-1917
Sources: calculations based on data from SIP and Human Mortality Database (2017).

In Paper 1, individual-level data for individuals below age 15 come from the sample of all individuals under age 15 residing in the five parishes between 1870 and 1940 drawn from the SEDD version 3.0 (Bengtsson, Dribe, and Svensson 2012). In addition to all-cause mortality, the database includes high-quality data on the causes of death, which in 1870–1940 was certified by a doctor or a midwife and registered by the clergyman (Bengtsson and Lindström 2000). Additional data on treatment by public health interventions have been added at a geographic level from three sources. The parish-level information on total public and public health expenditures and on the number of medical personnel comes from official statistical sources (Statistiska Centralbyrån 1874-1917; 1880-1910; 1911-1920). At the health-district level (a group of parishes), contextual data on the health interventions undertaken by local authorities come from the local annual health reports (Regionarkivet Lund 1881-1888; 1889a-1941; 1889b-1941; 1901; 1913-1941). Importantly, the data from the local ledger books, allowing me to observe the costs of interventions, were available for the two central parishes and local hospitals, Kävlinge and Kågeröd (Kommunarkivet Kågeröd 1876-1930; 1901-1931; Kommunarkivet Kävlinge 1881-1930).

Paper 2 relies on longitudinal individual-level data from the SEDD that have been expanded for the period 1968–2012, SEDD version 4.0 (Bengtsson et al. 2014). As a result, the cohorts in question, individuals born in the parishes in

1881–1930, could be followed from birth, or even prenatally, until age 80. In terms of treatment, this study utilizes a unique piece of information, such as the individual data from local midwife reports that has been merged to both newborns and mothers in the SEDD, originally covering the cohorts from 1881–1946 (see Chapter 6). From this source, cohorts from 1881–1930 are considered. It contains information on the type of treatment at birth and during the three weeks after birth (e.g., use of antiseptics and residential distance of midwife from a patient's house) and child's health status at birth (e.g., birth weight). In addition to detailed information on socio-economic background, the study makes use of abundant information on mothers (e.g., maternal age and midwifery treatment at previous childbirth). In terms of health outcomes, as previously, there is information on all-cause and cause-specific mortality; in addition, the database contains information on height, morbidity and skills for males at conscription examination (19–21 years of age), which has been merged to the SEDD from the military records (Öberg 2014). As an instrument to the individual-level treatment by midwifery, this study relies on archival information about the number of qualified midwives employed at the level of a parish or a group of parishes across the cohorts gathered from archival sources, such as annual provincial doctor's reports and ledger books, which is further used to instrument the individual treatment by midwifery at birth (Regionarkivet Lund 1881-1888; 1881-1899; 1889-1946; 1900-1946; Kommunarkivet Kävlinge 1881-1930; Kommunarkivet Kågeröd 1876-1930). Moreover, from the dataset, I constructed various parish-level socio-economic and demographic characteristics varying by year of birth and complemented it with parish-level real investment per capita for 1881–1930 from official statistical sources (Statistiska Centralbyrån 1880-1917; 1918-1930).

The longitudinal individual-level data for the Paper 3 come from the SIP. To assure consistency in ages for the cohorts under study – those born between 1890 and 1917, – the study focuses on outcomes at ages 78–95. For the old-age outcomes, the study endeavors to study mortality, cause-specific mortality and average income. Regarding income, to avoid changes in the registration of different types of income occurring through the period under study and because pension could be capitalized, I used total income, which for the age groups under study includes pension (90.7 percent) and capital income (9.3 percent). The pension scheme covering the cohorts under study provided a flat basic rate with a supplementary benefit determined as a percentage of the average 15 highest paid years (Kruse 2010). For those individuals for whom both a base pension and a contribution dependent on labour productivity are known (from 1991), the latter constitutes 43.7 percent of the final income measure. Information on the rollout of the health district reform, merged to individual-level data based on parish and date of birth, has been collected from several sources. Primarily, the reports of the health board on provincial doctor districts contain detailed data on the allocation of the parishes as well as the creation dates and funding of the new districts

(Medicinalstyrelsen, 1907, 1939). Additionally, the official statistical sources provided parish- and health-district information on the number of medical personnel employed, such as doctors and midwives, and public total investments and investment in health care (Statistiska Centralbyrån 1880-1917; 1880-1910; 1911-1920). To fill the gap in the individual's background characteristics for the cohorts from 1890–1917, the paper gathered abundant parish-level socio-economic (e.g., the share of elite and industrial workers and whether the parish had a railway) and demographic (e.g., the size of population and the share of infants) information from the Swedish decennial censuses for 1880, 1890, 1900 and 1910 (Riksarkivet 2014).

Relying on the SIP as a source of longitudinal individual-level data, Paper 4 utilizes its data on multiple outcomes. As for the main outcomes, for the cohorts born from 1934–1943, the SIP contains data on labour income (ages 44–60). The study also utilized information on the mechanisms underlying the links between early-life circumstances and labour income later in life, such as years of education, average length of stay in hospital in total and by cause of admission (ages 53–60), and all-cause and cause-specific mortality (ages 34–60). Almost all individuals are linked to their parents, thereby giving a unique family identifier. Due to the availability of family links, it was possible to merge socio-economic and demographic information of the family to the individual data (e.g., maternal age and paternal education). Treatment information has been gathered from several sources at a geographical (region, that is, an urban or rural part of county) and cohort level. For this purpose, the study utilizes region-specific mortality due to pneumonia, collected from several official statistical sources (Statistiska Centralbyrån 1911-1944; 1918-1944; 1920a-1950; 1920b-1950; 1920c-1950; 1920d-1950; 1925-1950; 1931-1959). Similar sources provided regional and cohort-level demographic information (e.g., mortality by cause of death and share of females in total population) and socio-economic information (e.g., income per capita and health care expenditures). The study gathers pharmacy-level data on drugs by type, number of pharmacists and price indices of medical drugs, a rather unique source of information collected from archival pharmacy records (Riksarkivet 1920-1967).

Limitations

The combinatory use of individual and archival data in the thesis helps to systematically study the phenomena of embedded medical knowledge as the origin of decline in infectious disease in short- and long-run health and economic development in Sweden. However, despite the richness and unique features discussed above, these data had certain inevitable limitations plausibly affecting the validity of the results. One is the absence of total and labour income in

adulthood as an outcome in an investigation of the effects of a rural health district reform in Paper 3. Even though the pension income for the cohorts under study is related to productivity in adulthood, it is not ideal, and results on its basis – in the strictest sense – should be thought of as evidence for the presence of the treatment effects on income rather than a precise indication of the magnitudes of such effects. The datasets for the cohorts under study are also lacking good measures of cognitive ability and quality of schooling, limiting the opportunities to investigate the mechanisms of the reforms beyond health. While in Paper 2 the timing of treatment is precise (at birth and three weeks after birth), in other studies individuals are linked through the year of birth, not taking month of birth into account. The latter studies compare individuals exposed in a year of birth to other groups in childhood, so the effects should be interpreted as for those treated in infancy, albeit with no further precision. One more limitation is the losses of individuals in the follow-up, in both local and national datasets. Specific characteristics, if associated with the outcomes of interest, of the individuals not observed at later ages raise the possibility of selection bias. In the Scanian dataset, permanently migrating individuals are not traced, and in the national dataset, some individuals permanently migrated or died before entering the sample. The reader can consult the studies of this thesis for discussion of these issues and the related robustness checks.

Results

Summary of the papers and the sizes of the effects

Paper 1 – Lazuka Volha, Luciana Quaranta and Tommy Bengtsson. 2016. 'Fighting infectious disease: Evidence from Sweden 1870–1940.' *Population and Development Review* 42 (1): 27–52. This paper is co-authored with two supervisors, Tommy Bengtsson (main) and Luciana Quaranta (assistant). Volha Lazuka was a main author, both in producing (at all stages and of all components) and publishing the paper.

New insights into the modes of transmission and causal agents in the mid-nineteenth century, together with fear of new epidemic outbreaks, motivated public investments aimed at reducing mortality from infectious disease. Following state regulations, rural and semi-urban parishes in Scania established local health boards in the 1860s and 1870s which implemented a set of public health initiatives. They opened isolation hospitals for isolation of sick residents and travelers under the auspices of newly employed medical doctors in the most populous parishes (two out of five), and employed midwives qualified in bacteriological knowledge (under contracts with two separate health districts) with different timing between 1870 and 1940. We take a causal (DD) approach to estimating the effects of these public health interventions on mortality under age 15 (17,000 children) using longitudinal individual-level data combined with purposely collected parish-level data on public health investment from local ledger registers for southern Sweden during this period. The data used in this study allow us to take into account children's socioeconomic status, which is of importance for the period of interest owing to the nutritional sensitivity of certain infectious diseases, such as measles, diarrhoea, and respiratory infections. It also enables us to study distributional effects of the interventions. Public health measures under study were not accompanied by sanitary or vaccination reforms, and these measures appeared to be independent of local wealth and infectious disease conditions.

The study finds that the effects of both establishment of isolation hospitals and employment of qualified midwives on infant and child mortality from 1870–1940 are substantial. It shows that after the employment of more qualified midwives, the infant mortality rate dropped by more than 50 per 1,000, that is by almost 60 per cent, net of period and parish level changes. The effects were greatest for neonates, and may be directly related to the employment of better-educated midwives, but were also strong for infants in the post-neonatal period, indicating that basic changes in infant care were helpful, such as the isolation of infants from sick family members, disinfection of the umbilicus and prolonged breastfeeding.

The impact of the establishment of isolation hospitals on child mortality has a similar relative magnitude but is somewhat smaller in absolute terms. The isolation of people in separate buildings prevented deaths from scarlet fever, measles, and lung inflammation. The mortality rate of children aged 1–4 and 5–14 decreased by approximately 50%; in absolute terms, this represents a decrease of 12 and 4 deaths per 1,000, respectively. Unlike for rural parishes, the effects of isolation were not robust for semi-urban areas, indicating that the adverse effects on mortality as a result of partial industrialization outweighed the benefits of the public health intervention. Hence, the public health interventions focusing on reduction of exposure to disease explain all declines in infant mortality and at least two thirds of contemporaneous decline in mortality under age 15. Together with the absence of socio-economic differences in access to these public services, it finds that mortality from airborne infectious diseases declined the most.

Paper 2 – Lazuka Volha. 'The long-term health benefits of receiving treatment from qualified midwives at birth'. *Under review.*

For the same area as in the previous study and inspired by findings for the benefits for infant health due to the service of newly employed midwives, Paper 2 explores effects of being born with the assistance of a qualified midwife on health and skills throughout the life course. In addition to parish-level information about the employment of midwives and the related investments as well as family-level information, SEDD possesses data about individual-level treatment by qualified midwives, which is unique, about maternal birth history, and follows individuals from birth, or even prenatally, until age 80. I use such information for all individuals born between 1881, the year when a midwifery birth registry was introduced, until 1930, before the introduction of antibiotics, expansion of maternity hospitals and water supply reforms were launched in the parishes (7,211 individuals). All individuals found in the diaries whose mothers resided in the five parishes under analysis were successfully identified in the subsequent population of newborns. Individuals who were not observed in these diaries were assumed to have been assisted at birth by traditional midwives or no midwives. Both controlling-for-observables and instrumental-variables empirical strategies are applied, with the latter exploiting variation in the number of qualified midwives locally employed under contract with the commune as an instrument. In the setting of home deliveries, midwives used antiseptics, provided information about the importance of isolation of infants, early breastfeeding, boiling of milk, warmth and hygiene, and monitored women's and newborn's health for several weeks after birth. Qualified midwives did not supervise women during pregnancy, so the setup allows me to clearly identify a time window for intervention – from birth until three weeks after birth.

The study first supports the previous finding that the employment of qualified midwives reduced both neonatal and postneonatal mortality, with the strongest impacts on the former. In 1881–1930 in southern Sweden, neonatal mortality dropped from 241 to 70 per 1,000, or by two thirds. The estimates from the study suggest that between 70 to 100% of this decrease can be attributed to the service of qualified midwifery. As the core analysis, the study then finds that treatment by qualified midwifery at birth yielded significant decreases in mortality throughout the lifetime of the individuals. Introduction of this high-quality treatment led to at least a 40% lower all-cause mortality rate from ages 15–40 (mainly mortality from respiratory diseases, such as pneumonia and pulmonary tuberculosis), and 45% lower mortality from cardiovascular diseases and diabetes (mainly ischaemic heart disease and cerebral haemorrhage) from ages 40–80. For the cohorts born in the parishes under study in 1881–1930, mortality from ages 15–39 declined from 510 to 111 per 100,000, and mortality from cardiovascular diseases and diabetes from ages 40–80 declined from 736 to 488 per 100,000. Hence, the employment of midwives qualified in 'modern knowledge' explains half of the overall reduction in mortality for those aged 15–39 and total reduction in chronic mortality from ages 40–80. Additionally, males treated by qualified midwifery at birth had lower morbidity (at least 17% lower probability), including lower rates of tuberculosis, heart disease, and 'general weakness', and better technical or language skills (at least 10% higher probability), measuring likelihood of being able to drive and handle a machine or horse, knowledge of foreign languages, or being suited for desk duty, among other differences, from ages 19–21. In total, treatment by qualified midwifery extends the lives by 1.2–1.9 more years between ages 15 and 80, which is around a fourth of overall gain in cohort life expectancy in adulthood. The study found that hygiene techniques and disease monitoring appear to be responsible for the long-lasting effects. There is no indication that the effects of midwifery were strongly modified by family or socio-economic conditions in childhood and adulthood.

Paper 3 – Lazuka Volha. 'The long-term health and income effects of early public health formation in Sweden'. *Under review.*

Similar public health initiatives exploited in the previous papers were launched throughout all rural parts of Sweden, usually connected to the establishment of a provincial health district. In 1890, the Swedish state began to subsidize the establishment of health districts in rural parishes, which implied the employment of a doctor and several midwives, opening of a hospital or a health station, and accumulation of local spending on health care; the health districts were established gradually throughout Sweden. In this reform, the state subsidized almost half of the expanses related to the organization of health districts for each group of parishes with 8,000–12,000 inhabitants. The study follows the reform

implementation through 1917; after this year it encompassed reorganizations rather than additional public health investments. Rural health districts were created under conditions of new bacteriological regulations imposing local control of the spread of infectious disease. Using register-based individual-level longitudinal data with national coverage (1968–2012) and exploiting plausibly exogenous variation in the timing of the implementation of the reform across parishes, I examine whether individuals treated in their infancy have an advantage in old age, namely ages 78–95 (69,939 individuals). The study relies on a DD method as a main empirical strategy combined with propensity score matching. In terms of the outcomes, there is information on all-cause and cause-specific mortality as well as on pension and capital income. The rich supplementary dataset on the reform implementation, purposely built for this study, enables disentanglement of the effects of background socio-economic characteristics, the quality of the health initiatives undertaken at the place of birth and mechanisms linking them to long-term outcomes.

This study shows that preventive measures undertaken in infancy of the individuals accrue beneficial effects for all-cause and cardiovascular disease mortality (mainly acute myocardial infarction and chronic ischaemic heart diseases) from ages 78–95 for those who survived to these ages. While Paper 2 finds reductions in mortality risk due to cardiovascular disease and diabetes from ages 40–80 for individuals treated in the early neonatal period by qualified midwifery, this paper supports that such benefits extend to older ages. Due to the reform, individuals treated in infancy attain decreases in all-cause mortality risk of approximately 4.2–6.0%, equivalent to 0.54–0.71 additional years spent alive. In the rural setting in Sweden, cohort remaining life expectancy at age 78 increased from 8.4 to 10.1 years. The reform thus explains at least 30–40% of lifetime gain in those ages. Among all causes of death, the effects emerge due to reduction in cardiovascular mortality by 4.6–7.1%. Mortality due to cardiovascular disease constitutes more than half of general mortality, and declined across cohorts from 3,746 to 2,895 deaths per 100,000; 20–30% of this decline occurred due to the reform in question. In addition to mortality, the study finds that the positive effects on lifetime income (average real income between the ages 78 and 95 or a year before death) amount to 1.7–2.5 percent, albeit accrued based on pension and capital incomes. Across the rural-born cohorts, increase in real income was extremely rapid, 77%, and such increase was representative of the growth in national income per capita during the cohorts' adulthood (1925–1955). So the reduced-from effects of the reform explain only 2–3% of the total increase. To transform the intention-to-treat estimates for mortality and income into the treatment effects on the treated, the former should be divided by the proportion in a range of 0.2 to 0.5. There are no beneficial effects for individuals exposed to preventive measures in older ages in childhood, beyond infancy. The rise in social expenditures other than public health care is not responsible for the results. The

mortality effects are stronger in places which employed more medical personnel following the reform and where disease burden was higher. They are also somewhat stronger among individuals from poor socio-economic backgrounds.

Paper 4 – Lazuka Volha. 'Infant health and later-life labour market outcomes: Evidence from the introduction of sulpha antibiotics in Sweden'. *Under review.*

The late 1930s in Sweden witnessed the invention and introduction into medical practice of the first antibiotics – sulpha antibiotics – which appeared to be very efficacious again pneumonia, the main cause of infant death in that period. In Sweden, the sulpha drugs, initially imported and later produced by the national pharmaceutical companies, were introduced quickly after medical review. Due to the high availability of medical personnel, centralisation of drug distribution and low costs of treatment and administration, this medical innovation spread quickly across regions of Sweden and led to regional convergence in death rates from the targeted diseases. Based on rich administrative population data for Sweden from 1968–2012 and archival data on the availability of sulpha antibiotics in the late 1930s and applying a DD empirical method, it explores the effect of reduction in exposure to pneumonia in infancy on health, schooling and income discerned in adulthood of the affected cohorts (878,606 individuals). It studies the effects of sulphapyridine introduction exploiting two sources of variation: the relatively larger benefits for individuals born in regions characterised with higher baseline pneumonia mortality compared to individuals born in regions with lower pneumonia mortality, and varying exposure of different cohorts to the arrival of sulphapyridine. The cohorts include those born 1934–1943. The cohorts born beyond these years are likely to be differentially exposed to public health and schooling reforms, and younger cohorts to the arrival of penicillin. Health is measured with average length of stay in hospital and with mortality, both all-cause and cause-specific. The data provide family information, as well as sibling links, which enable controlling for unobserved heterogeneity at the family level, and to study treatment effects by different family or mother sub-groups. As demanded by the analysis, an exposure variable for pneumonia infection is obtained for the region and year of birth, collected from official statistical sources. Similar sources provided information on other mortality by causes of death, treatable and untreatable by sulpha antibiotics, across regions and socio-economic and demographic regional-level variables.

As a part of the empirical strategy, this study makes an assessment of the immediate effects of introduction of sulpha antibiotics on pneumonia mortality at the regional level. It shows that pneumonia mortality dropped by around 31 deaths per 100,000, which is around 90 percent of the pneumonia decline between pre-sulpha and sulpha periods (1934–1938 versus 1939–1943 respectively). As a core analysis, the paper finds that the arrival of sulpha antibiotics, efficient in treatment

against pneumonia, in the individual's year of birth led to larger income gains (average real labour income from ages 44–60), and fewer hospitalizations in adulthood (ages 53–60); effects for schooling were weak and not robust. Taken roughly from the data, real income increased by 7.6% for post-drug cohorts (1939–1943) compared to those born in pre-drug period (1934–1938). If one relates this number to the reduced form estimates of the impact of arrival of sulpha antibiotics, which are in a range of 3.0–5.1%, this intervention explains 39–67% of the total increase from the older to the younger cohorts. In the late adulthood of the affected cohorts, 1983–2010, real GDP per capita grew by 44%, and 7–12% of this increase can be attributed to the cohort effects due to introduction of sulpha antibiotics. Between the cohorts, average length of stay in a hospital decreased by 0.150 nights, and the intervention explains 23–31% of it. The most sizable effects on health have been found for hospitalizations due to cardiovascular diseases, diabetes and degenerative diseases of tissues and organs (the latter mainly symptoms of respiratory systems and arthritis). While there are no effects on mortality (only 7.7% are dying between ages 34–60), the results, albeit not statistically significant in some specifications, suggest that the reduction in pneumonia infection led to a decrease in cardiovascular mortality by 0.007 percentage points (26% of the pre-treatment level). The paper also finds that the positive effects on health, not schooling, are driving the bulk of the income gains emerged due to the intervention. These effects are fairly equal among males and females, and larger among individuals with poorer socio-economic backgrounds.

To summarize, the studies in this thesis find that public health and medical interventions yielded sizable reductions in infectious disease mortality in the short run, and in the long run the cohorts who experienced better disease conditions in infancy accrued additional benefits in terms of lower mortality, lower morbidity and higher incomes. They therefore support endogenous growth theory, which posits an interaction between health improvements and health technology, based on application of the germ theory of disease, as a determinant of economic growth in the long run. The magnitudes of the influence of interventions in question on mortality under age 15 are consistent with non-causal estimates calculated by Preston (1975, 1996), which he linked to various public health initiatives and medical treatments that implemented the germ theory of disease across the world. They also leave no or negligible room for the influence of nutrition on infectious disease mortality, which was claimed by McKeown (1976, 1980) and Fogel (1994, 1997) to be a major influence. The magnitudes of the long-term effects are similar to those shown by the empirical micro-level studies testing infancy inflammation hypotheses for the cohorts close to those under study. However, fair comparison to the previous micro studies, especially those studying pre-drug interventions, is limited by the rarity of studies explicitly testing the influence of interventions based on the germ theory of disease. The sizes of the lasting effects of infant health on income in adulthood and old age, which are established in this thesis, are

to a large extent consistent with the estimations presented in macro studies, such as Aghion, Howitt, and Murtin (2011) and Arora (2001, 2015). The cause – that is, the reforms under study that are targeting infectious disease – and the consequence, the rates of specific chronic diseases such as respiratory and cardiovascular diseases in adulthood and old age, point to the inflammation mechanisms behind the lasting effects suggested by Finch and Crimmins (2004) and Crimmins and Finch (2006). Consistent with the proposition of Bengtsson and Lindström (2003), the studies also suggest that the early neonatal period and infancy are the critical period for effecting the mitigation of such processes.

Social rate of return

Approach to measurement

To evaluate the monetary value and costs of health reforms, the concept of economic 'value of life' was developed. The value of statistical life is defined as the value of reducing mortality risk such that one statistical (hypothetical) life is saved in a population where the health reform is expected and across which this risk is spread (Weil 2013). Two approaches have been adopted to calculating economic 'values of life' – a human capital approach, where value of a statistical life is measured as discounted present value of lifetime labour earnings lost due to premature mortality, and a willingness-to-pay approach, which is deemed to represent individual preferences and estimates the value by looking at the wage premium associated with jobs that evolve extra mortality risks or from market prices for products that reduce the probability of fatal injury (Kenkel 2003). Unlike with discounted earnings, the value of a statistical life varies drastically across the studies, dependent on, for instance, the characteristics of the population, type of mortality risk and health policy in question. Both human capital and willingness-to-pay approaches are designed for and applied to measuring instantaneous effects of health policies. In assessing the net benefits of the public health interventions relevant for this thesis, they have been applied, for instance, in the studies looking at the short-term mortality effects of water purification technologies (Cutler and Miller 2005; Ferrie and Troesken 2008).

In the studies conducted in this thesis, where social rates of return of both instantaneous and long-term effects of public health and medical interventions are of interest, I adopt the hybrid approach, combining the two. In this chapter, the instantaneous economic benefits of a health policy are measured while employing values of a statistical life calculated within a willingness-to-pay approach. The ones adopted here focus on quantity of life (mortality reduction) and ignore quality of life (e.g., health improvements), and if not ignored they should be larger by approximately two thirds (Ryen and Svensson 2015). This chapter employs the value of a statistical life suggested by the Swedish authorities (22 mln SEK in

2012 price level, Hultkrantz and Svensson 2012), which is a moderate number, and further adjusts it for income level. It is calculated for contemporary populations, and was lower historically; as suggested, for rapidly industrializing economies such as Sweden in 1890–1930, the elasticity of the real value of life is suggested to be 2.3 relative to the GDP per capita (Haacker 2012). The economic benefits due to the emergence of latent effects are measured as the discounted increase in earnings or improvements in mortality probabilities across the life course. This long-term value should be seen as a rough measure of both quantity and quality benefits, due to better health, lower mortality risks and better cognitive ability, resulting from the improved early-life conditions. The approach has been recently applied in research studying the long-term effects of improved childhood conditions (e.g. Beach et al. 2016; Bütikofer, Mølland, and Salvanes 2016). The calculations in this chapter are made in a most conservative manner; where both reduced-form and IV estimates are available, the reduced-form estimates are used. The costs are available for each health reform under study, purposely gathered from archival and statistical sources. The values and costs are transferred to those in Swedish kroner (SEK) 1900 constant price level.

Short-term effects

The monetary value of period improvements in infant and child mortality is large and outweighs the costs of the related public health interventions. Paper 1 calculates the social rates of return of public health investment in the establishment of isolation hospitals and employment of qualified midwifery. It does so by comparing the real value of lives saved due to interventions with real costs, thus taking into account reductions in mortality. It first calculates the variable and fixed costs over the 10-year post-treatment period per treated child in 1900 constant price level SEK, relying on information from the parish ledgers, such as wages of midwives and a provincial doctor, costs of training of midwives, and payment for a child delivery; costs of isolation hospitals include initial costs for building a hospital and annual maintenance costs. To obtain the total benefits, it then calculates the number of deaths averted as a result of the public health interventions and multiplies them by the real value of a life in 1900 constant price level SEK to obtain the total benefits. The real value of life adjusted for elasticity is 1,480 SEK at the 1900 price level. It is noteworthy that this value of life does not take into account age, and for infants (with remaining life expectancy around 59 years for the 1900 cohort) should be 90 percent larger (Viscusi 2010), but this is ignored for the current calculations. Having made all necessary adjustments, a pairwise comparison of the benefits and costs showed that the social rate of return for health care interventions is large (see Table 3). Each public kroner spent on health care gave a return of more than 11 SEK from investment in establishment of isolation hospitals and 12 SEK from investment in employment of qualified midwives. The rough approximation of the investments in infrastructure shows

that the most expensive hospital established in the parishes costs the public less than one-fifth of what was invested in other local infrastructure. The size of the return is not surprising if one considers that expenses for public health care were not substantial compared to other costs, particularly at the initial stages.

Table 3 – Social gains from the public health and medical innovations: Short-term effects, Sweden 1883–1943

	Isolation hospitals, 1883–1905	Midwifery, 1893–1925	Health districts, 1890–1917	Sulpha antibiotics, 1934–1943
	rural Scania	rural Scania	rural Sweden	Sweden
	all-cause mortality ages 1–14	all-cause mortality <1	all-cause mortality <1	pneumonia mortality all ages
Total benefits from intervention, SEK 1900 price level	134 065	147 405	12 285 888	29 037 464
Total costs of intervention, SEK 1900 price level	12 701	12 866	994 716	1 018 069
Social rate of return	11:1	12:1	12:1	29:1

Sources: own calculations based on the estimates from the papers of this thesis and various archival sources.

Paper 3, which studies the lasting effects of establishment of health districts across the countryside of Sweden from 1890–1917, does not contain the calculation of the short-term effects of such policy due to the unavailability of the relevant data. However, the components of the health district are the same as those studied in Papers 1 and 2, such as employment of qualified personnel, midwives and doctors, and disinfection and isolation measures undertaken. Given that previously rural parishes in Scania were assessed to be similar to other rural parts of Sweden in terms of health and health care characteristics, I can rely on the policy estimates obtained in these papers. The focus here is on infants, and in 1890–1917 in rural parts of Sweden infant mortality dropped from 95 to 67 per 1,000 live births (Statistiska Centralbyrån 1969). Hence, the reform – rather tentatively – can explain all reductions over this period. With around 2.8 million live births and 23 percent of the rural parishes establishing health districts between 1890–1917, the reform therefore prevented around 8,300 deaths. Using the adjusted real value of life (1,480 SEK in 1900 price level), the total benefits due to the reform will amount to 12.3 million SEK at the 1900 price level. The database purposely collected for the study contains information about the costs of all newly established provincial health districts, and if one further assigns a quarter of the same amount (roughly a salary to a doctor and two midwives) as the costs for maintenance of a district for 10 years (5 or 7 years used in the paper would inflate the value), the total costs are around 1.0 million SEK at the 1900 price level. The calculations give the social rate of return of 12 to 1, which is very similar to that for the rural parishes in Scania.

In studying the economic effects of the introduction of sulpha antibiotics on rates of pneumonia, as a part of the empirical strategy, Paper 4 makes an assessment of its immediate effects on pneumonia mortality at the regional level. Based on the estimates mentioned previously, with the population 6.2–6.5 million in 1934–1943, the decline in pneumonia mortality due to arrival of sulpha antibiotics represents the saving of 19,620 lives of all ages. The total benefits from mortality reductions due to sulpha antibiotics, with the real value of life as that used previously, amount to 29.0 million SEK at the 1900 price level. Among the costs, I consider the costs of treatment of pneumonia (purchase of sulpha antibiotics to treat pneumonia and payment for a doctor's visit, no insurance considered). Among the pneumonia cases, not only those prevented from death are included, but also all morbidity cases due to pneumonia, 112,648 cases in 1939–1943 (Statistiska Centralbyrån 1920d-1950). The costs therefore do not include expenses of invention and distribution of sulpha antibiotics, which is difficult to quantify given its international origin, and only partially cover the costs of the health care system. The total costs amount to 1.0 million SEK at the 1900 price level. The comparison of total benefits due to instantaneous reduction in pneumonia mortality versus costs for the introduction of sulpha antibiotics provides a ratio of 29 to 1, which is large, albeit expected.

Long-term effects

The public health interventions in the pre- and after-drug periods not only accrued beneficial effects in the short run but also positively affected survival, health, skills and incomes of individuals throughout their adulthood and old age. This section monetizes such long-term benefits by using a human capital approach as follows:

$$NPV = \sum_{t=t_0}^{t_n} \frac{(S_t^{/} I_t^{/} - S_t I_t)N}{(1+r)^t} \tag{6}$$

where $S_t^{/}$ and S_t are survival probabilities with and without a reform, $I_t^{/}$ and I_t are labour income with and without a reform, N = cohort size; r = real interest rate. I use yearly long-term government bond yields adjusted for inflation as real interest rates from Waldenström (2014). The differences between the adjusted income profiles (with adjusted survival probabilities or/and adjusted incomes) and baseline income profiles are therefore discounted by the interest rate, based on a compound measure for the ages $t_0 - t_n$. This difference, summed across ages and cohorts, could then be compared with the costs of public health/medical initiatives. Similar to the previous calculation, the social benefits and costs, including the interest rate, can be calculated in real terms with 1900 as a base year. To preclude any overestimation, among the measures of costs and estimates, I rely on the most conservative values.

The discounted net benefits of the pre-drug interventions in the long run are considerable. To make a calculation, I obtained the data for cohorts from 1881–1930 directly from the SEDD 4.0: cohort- and age-specific survival probabilities and incomes (income earned from employment and business), for the latter covering the period 1902–2010. Based on Waldenström (2014), the real interest rate amounts to 3.2% for the cohorts 1880–1930. It is reasonable to take into account two effects of the introduction of qualified midwifery: on survival probabilities during working ages (15–39), estimates of which can be directly employed, and on skills for males (ages 19–21). Differences in skills in question, which plausibly approximate those in vocational education, should yield differences in labour earnings. I further take the lowest of estimates for the rate of return to vocational education calculated by Palme and Wright (1998) for Swedish males (6.6%), and apply it to the earnings of males aged 19–21. Regarding the public costs of improved midwifery for this period, I summate salaries of midwives, doctors, costs of midwifery training, payments for deliveries and costs of all cottage hospitals that were built. Table 4 presents the social benefits, costs and social rate of return for this intervention. Based on the reduced-form estimates, benefits from qualified midwifery outweigh costs by a ratio of 33 to 1.

Table 4 – Social gains from the public health and medical innovations: Long-term effects, Sweden cohorts1881–1943

	Midwifery, cohorts 1881–1930 rural Scania ages 15–40	Health districts, cohorts 1890–1917 rural Sweden ages 78–95	Sulpha antibiotics, cohorts 1934–1943 Sweden ages 44–60
Total benefits from intervention, SEK 1900 price level	3 298 455	6 107 451	401 966 590
Total costs of intervention, SEK 1900 price level	101 104	994 716	11 790 478
Social rate of return	33:1	6:1	34:1

Sources: own calculations based on the estimates from the papers of this thesis and various archival sources.

The net benefits of the establishment of a rural health care district, discounted from a period when the affected cohorts are in their old age, are positive. To make a calculation, I obtained 1890–1917 cohort survival probabilities for ages 78–95 for Sweden from the Human Mortality Database (2017). Cohort- and age-specific incomes (real pension and capital income in ages 78–95) are gathered from SIP. The real interest rate amounts to 2.6% for the cohorts from 1890–1917. Noteworthy is that the discount rate is based on the interest rate compounded by years 78–95 respectively. For the reform, as provided by the study, both improvements in survival probabilities and in incomes can be taken into account. The discounted incomes equal 6.1 million SEK at the 1900 price level. The costs are the same as those for the short run: investment in newly established provincial health districts summated with a quarter of the same amount (roughly a salary to a

doctor and two midwives) as the costs for maintenance of a district for 10 years, in total around 1.0 million SEK at the 1900 price level. The costs are not discounted. As shown, based on the reduced-form estimates, each public kroner spent on the establishment of health districts yielded a return of 6 kroners.

To calculate the net monetary value of the long-term effects of introduction of sulpha antibiotics, I again rely on the comparison between discounted long-term earnings and costs of treatment. I do not take into account the effects on health, and adjust the labour incomes across working ages by the estimates (cohorts 1939–1943). Cohort- and age-specific labour incomes, covering the period 1979–2012 and ages 44–60, are gathered from the SIP. For an adjustment, I rely on the lowest among the reduced-form estimates of the reduced exposure to pneumonia on labour income. As previously, I used real interest rates, that is, the average of long-term government bond yields adjusted for inflation in 1939–1943 from Waldenström (2014). The real interest rate amounts to 3.4% for the cohorts 1939–1943. The discounted benefits amount to 401.9 million SEK at the 1900 price level. Among the costs, as previously, I considered the costs of treatment of pneumonia cases in 1939–1943 (1.0 million SEK at the 1900 price level), but also added costs of treatment for all infants and children under age 15 (10.8 million SEK at the 1900 price level), assuming that they all at some point in childhood underwent treatment. Yet, based on these conservative numbers, the social rate of return for introduction of sulpha antibiotics is large, and amounts to a ratio of 34 to 1.

To summarize, in accordance with interpretations by Easterlin (1999) and Mokyr (1998, 2002) and macro-level calculations by Nordhaus (2003) and Murphy and Topel (2006), as the cost-benefit analysis shows, health and economic gains of public health and medical interventions, based on the germ theory of disease, not only were inexpensive but also largely outweighed the related public costs, both in the short- and long run. The net benefits are still positive even if discounted from those received more than seven decades after the intervention.

Discussion and Conclusions

On the causality between economic growth and health development

The above calculations show that isolation and disinfection public initiatives and provision of sulpha antibiotics yielded sizable net social gains since 1880 in Sweden. The methods applied in the studies allowed elimination of the influence of income growth. Yet, the question that remains is to what extent the health interventions in question are independent of long-term increases in income.

Consistent with the debate on the primacy of improvements in living standards compared to preventive knowledge or institutional advances, one may argue that long-term income growth *preceded* invention and application of the germ theory of disease, became its source and thus a necessary condition. From this viewpoint, income growth inevitably becomes a long-term driver of health advances. The previous macro literature discussed this possibility. For instance, Easterlin (2004) matches the onset of modern economic growth and improvements in life expectancy in a set of developed and developing countries. As he shows, although it occurs in all countries, the contrast between the UK and Sweden appears to be the most striking: the onsets of rapid increases in life expectancy are identical, but those in economic growth differ by three-quarters of a century, being later for Sweden. In fact, among the countries in Europe and North America that adopted the new public health measures in the 1880s, in terms of real income per capita Sweden was among the poorest and became a median-earner only in the mid-twentieth century (The Maddison-Project 2013). The interventions were also inexpensive: the public costs of either rural health district reform or the costs of sulpha antibiotics calculated previously constituted a diminutive share of contemporaneous GDP in Sweden. Therefore, based on these comparisons and calculations in this chapter, I could agree that 'the measures necessary to implement advances in health technology do not seem to have required … the capital expenditures necessary for modern economic growth' (Easterlin 2004, p.91). Unlike in income levels and income growth, these countries resembled each other in the timing of implementation of public health measures focusing on stopping the spread of infectious disease (e.g., Niemi 2007).

One more argument in favour of the income explanation is that societal health measures required entrepreneurial skill and initiative, like those in the endogenous growth model of Aghion and Howitt (1992). In this perspective, as Weil (2013) argues, incentives in microbiology become a function of market and income growth, because they produce better scientific tools and enhance the pursuit of profit. For instance, the sulpha antibiotics were produced from the materials of the chemical industry, developed as a result of industrialization, and at the laboratories of the profit-seeking pharmaceutical companies. Again, Easterlin (1999) refutes such a view. He claims that for the application of the sanitary and isolation public health measures, private incentives were an obstacle rather than the impetus, and local government had to impose the measures not due to but despite market interests. The public authorities also had to regulate the distribution of antimicrobial drugs, due to the side effects, misuse of the drugs and growing drug resistance. To further disapprove the income explanation, one may look at the knowledge accumulation that preceded these inventions. In line with claims by Mokyr (1998, 2002), medical knowledge has a long history of human effort and trial, and develops cumulatively by interacting with environmental changes, and so did the germ theory of disease. The long tradition of Sweden controlling disease in

society, dating back to the eighteenth century, became fruitful for acceptance of internationally growing medical knowledge. The public and scientific measures, based on the germ theory of disease, together with institutions realizing them can thus be viewed as an independent force behind economic growth.

At the micro level, a probably credible way to support the contention that income does not underlie the benefits from public health and medical interventions of applying the germ theory of disease is to investigate the presence of socio-economic differences in their access and adoption. On may argue that when a new health technology is provided in the society, richer households, due to power, material resources or education, may more willingly apply it (e.g., Link and Phelan 1996). The application of public knowledge thus depends on household income and knowledge availability and cannot be regarded as truly 'revolutionary' or 'exogenous'. The qualitative evidence rather suggests the opposite. The narratives by Mokyr (2002) and Easterlin (1999, 2004) show that households across all socio-economic classes in Western countries at the time of adoption of public interventions were instead following or even resisting a variety of campaigns against infectious disease in the nineteenth-beginning of the twentieth centuries. The studies of this thesis explore the issue quantitatively.

The papers of this thesis show the following: in none of the public health and medical interventions under study – isolation and disease surveillance efforts, qualified midwifery or provision of sulpha antibiotics – was it found that socio-economic class and its different dimensions determine the access and take-up of the treatment. In Paper 1, the mortality risks for the different socioeconomic groups treated by isolation efforts and increase in qualified midwifery indicate no apparent differences in the mortality responses among infants and older children. In Paper 2, there is no full gradient in treatment by qualified midwifery at birth, and the neonatal mortality decreases are similar among the socio-economic groups. Paper 4 showed that the distribution of sulpha antibiotics across regions of Sweden did not depend on the regional income and infrastructure. Moreover, in the long run the public health and medical measures under study produced larger health and income effects among the individuals with poorer backgrounds. For instance, as Paper 3 shows, less affluent parishes became forerunners in the organisation of health districts, implementing isolation, disinfection and surveillance measures, and they gained more from it. Due to the low costs of the drug and spillovers from reduced peer infection, both poor and more affluent families received access to treatment by sulpha antibiotics, and the former benefited more in terms of productivity gains. The findings of this thesis are therefore consistent with theoretical considerations where health and health technology, driven by new public knowledge about reducing exposure to infectious disease in a manner independent of income growth, become a noticeable determinant of long-term health and economic growth.

Conclusions

This thesis has a general aim to explore the role of public health and medical interventions between 1880 and 1945, enabling reduced exposure to infectious disease, in health and income improvements throughout the twentieth century. The bacteriological discoveries of the latter part of the nineteenth century, specifically the validation of the germ theory of disease, provided scientific grounds for control of infectious diseases, such as isolation, disinfection and treatment, and helped the public to target and, as this thesis finds, by the 1950s to defeat most infectious diseases. The role of public interventions in controlling infectious disease, undergirded by the germ theory, in contemporaneous decline in infectious disease is substantial. Not only were these interventions successful in the short run, but they interacted with individuals' health and thereby led to beneficial consequences for health and income for the cohorts born to better environmental conditions in their adulthood and old age. In the long run, societal health investments through health advances in a cohort perspective undoubtedly led to income growth and yielded high rate of return, albeit they cannot be viewed as a main driver of economic growth. This thesis provides support for the endogenous growth theory that posits an interaction between health improvements and societal health technology, based on the germ theory of disease, as a determinant of economic growth in long run.

In the pre-drug period, public health interventions related to the isolation of the contagious persons in hospitals and employment of midwives qualified in bacteriological knowledge reduced infant, child and infectious disease mortality in Sweden, by 50% each. In the long term, the cohorts treated in the early neonatal period by qualified midwifery obtained sizable advantages in health: at least a 40% decrease in mortality risks in all-cause mortality in adulthood and a 45% decrease in cardiovascular diseases and diabetes in old age. Treatment by qualified midwifery extends the lives by 1.2–1.9 more years between ages 15 and 80, which is a fourth of overall gain in cohort life expectancy in adulthood. As early as young adulthood, males treated by these interventions also had lower morbidity and better skills. Throughout rural Sweden, the cohorts affected in infancy by preventive measures and improved midwifery care in the community, after the establishment of health districts, gained between 0.5 and 0.7 additional years of life at age 78. The reform explains at least 30–40% of the total increase of lifetime gain. The lifetime incomes of these cohorts have been also positively affected, by 1.7–2.5 percent, although it accounts for a small fraction (2–3%) of the overall increase.

In the drug period, a nationwide introduction of sulpha antibiotics in Sweden, a first medical innovation in the treatment of child infectious diseases such as pneumonia, led to an observable decline in pneumonia mortality, by around 30%, thereby explaining almost the totality of pneumonia mortality reductions in that

period. Moreover, reductions in pneumonia mortality due to the arrival of sulpha antibiotics enhanced the income and health of cohorts affected in infancy. In the long run, this medical innovation increased real labour income by 3.0–5.1%, and explained at least 40% of the total increase between the cohorts and 7–12% of the increase in real GDP per capita. Between the pre- and post-drug cohorts, average length of stay in a hospital decreased by 20% in adulthood, and the intervention explains around a fourth of it. The most sizable effects on health have been found for hospitalizations due to cardiovascular diseases, diabetes, arthritis and respiratory diseases. These effects are responsible for acceleration in growth in labour income for the affected cohorts, where improvements in adult health, rather than schooling, account for its bulk.

In monetary terms, the benefits resulting from these public health and medical breakthroughs outweighed the associated costs. This holds for both short- and long-run effects of reduced mortality and increased income. Taken together, improved midwifery and isolation hospitals in Scania yielded a social rate of return of 34 to 1, establishment of rural health districts accompanied by employment of qualified medical personnel and the implementation of preventive measures throughout the rural parishes in Sweden – 19 to 1, and introduction of sulpha antibiotics throughout Sweden – 37 to 1. The reforms were specific, low-cost and effective, and the associated gains appeared inexpensive. A portion of the growth in real national income since the second quarter of the twentieth century can be thus attributed to the health investments made a half a century earlier. The revenues due to these public investments add to the value of goods and services produced.

The results of this thesis point to the decreased exposure to infectious diseases and inflammation in the individuals' infancy through both public health (prevention) and medical (treatment) initiatives in 1880–1945 resulting in positive consequences for health and income in the long run. The application of the advances in health technology studied in this thesis, one close to the turn of the century and another to the middle twentieth century, both affected infectious disease mortality, which declined rapidly throughout this period. The effects emerge for individuals treated in their infancy, when infectious disease mortality is the highest and thus consequences of the reforms are the largest, and not in other stages of childhood. Such effects are apparent for all-cause mortality and morbidity, with the most pronounced influence on cardiovascular causes. Cardiovascular disease is the leading cause of death nowadays, and this thesis shows that its current decline embeds a cohort inflammatory component. The initiation of such dynamics became realized due to the cumulative scientific effort of humans in finding the tools to defeat the deadliest diseases of era and their institutionalized adoption.

Infectious disease mortality, including from pneumonia, has been declining throughout the twentieth century in Sweden, only recently dropping to a few cases

per 100,000 among infants. It is appealing to attribute the decline since 1945 to the application of medical advances, such as introduction of penicillin and streptomycin, mass vaccinations and neonatal care, which is something to be proven in future research. In developing countries, mortality due to infectious disease has declined since 1950 but remains high today and could be prevented by affordable and efficient interventions, such as antibiotics, qualified midwifery and isolation of the sick. Morbidity due to respiratory infections, pneumonia the leading one, remains the major cause of death among children under age 5 in Sweden and other developed countries. The implications of this thesis therefore extend beyond the cohorts born at a time of rapid improvements in life expectancy. For those born throughout the whole twentieth century, public action based on up-to-date medical knowledge is likely to lead not only to an increase in health and productivity when people reach working ages, but also to improvements in health and well-being in their old age. This thesis draws a policy attention to public health and medical policies for infants and emphasizes the importance of a long-term, namely cohort, perspective for the countries that lag behind in health and economic improvements.

References

Acemoglu, Daron, and Simon Johnson. 2007. "Disease and development: The effect of life expectancy on economic growth." *Journal of Political Economy* 115 (6): 925–85.

Aghion, Philippe, and Peter Howitt. 1992. "A model of growth through creative destruction." *Econometrica* 60 (2): 323–51.

Aghion, Philippe, Peter Howitt, and Fabrice Murtin. 2011. "The relationship between health and growth: When Lucas meets Nelson-Phelps." *Review of Economics and Institutions* 2 (1): 1–24.

Aizer, Anna, Shari Eli, Joseph Ferrie, and Adriana Lleras-Muney. 2016. "The long-run impact of cash transfers to poor families." *American Economic Review* 106 (4): 935–71.

Allender, Judith A., Rector Cherie, and Kristine D. Warner, eds. 2013. *Community Health Nursing: Promoting and Protecting the Public's Health.* 8th. Philadelphia: Lippincott Williams & Wilkins.

Almond, Douglas, and Janet Currie. 2011. "Human capital development before age five." In *Handbook of Labor Economics. Vol. 4B, Developments in Research Methods and Their Application*, edited by Orley Ashenfelter and David E. Card. 1315–468. New York: Elsevier.

Andersson, Yvonne, Marie-Louise Hammarström, Bo Lönnerdal, Gitte Graverholt, Helen Fält, and Olle Hernell. 2009. "Formula feeding skews immune cell composition toward adaptive immunity compared to breastfeeding." *Journal of Immunology* 183 (7): 4322–8.

Arora, Suchit. 2001. "Health, human productivity, and long-term economic growth." *The Journal of Economic History* 61 (3): 699–749.

Arora, Suchit. 2015. *The Transitions of Aging.* Cham: Springer.

Ashraf, Quamrul H., Ashley Lester, and David N. Weil. 2009. "When does improving health raise GDP?" *NBER Working Papers* 14449.

Barker, David J. 1991. "The foetal and infant origins of inequalities in health in Britain." *Journal of Public Health Medicine* 13 (2): 64–68.

Barker, David J. 1994. *Mothers, Babies, and Disease in Later Life.* London: British Medical Journal Publishing Group.

Barker, David J. 2012. "Sir Richard Doll Lecture: Developmental origins of chronic disease." *Public Health* 126 (3): 185–89.

Barker, David J., Clive Osmond, and C. Law. 1989. "The intrauterine and early postnatal origins of cardiovascular disease and chronic bronchitis." *Journal of Epidemiology and Community Health* 43 (3): 237–40.

Barker, David J., Godfrey K.M., Fall C., Osmond Clive, Winter P.D., Shaheen S.O. 1991. "Relation of birth weight and childhood respiratory infection to adult lung function and death from chronic obstructive airways disease." *British Medical Journal* 303 (6804): 671–5.

Barreca, Alan I. 2010. “The long-term economic impact of in utero and postnatal exposure to malaria.” *The Journal of Human Resources* 45 (4): 865–92.

Barro, Robert J. 2013. “Health and economic growth.” *Annals of Economics and Finance* 14 (2): 305–42.

Barro, Robert J., and Xavier Sala-i-Martin. 2004. *Economic Growth.* 2nd. Cambridge: MIT Press.

Beach, Brian, Joseph Ferrie, Martin Saavedra, and Werner Troesken. 2016. “Typhoid fever, water quality, and human capital formation.” *Journal of Economic History* 76 (1): 41–75.

Becker, Gary S. 1964. *Human Capital.* 2nd. New York: Columbia University Press.

Becker, Gary S. 1981(1991). *A Treatise on the Family.* Cambridge: Harvard University Press.

Bengtsson, Tommy. 2015. “Mortality: The great historical decline.” In *International Encyclopedia of the Social & Behavioral Sciences,* edited by Linda L. Wright, 868–73. 2nd. Amsterdam: Elsevier.

Bengtsson, Tommy, and Christer Lundh. 1994. “La mortalite infantile et post-infantile dans les pays Nordiques avant 1900.” *Annales de Demographie Historique* 1: 23–43.

Bengtsson, Tommy, and Frans van Poppel. 2011. “Socioeconomic inequalities in death from past to present: An introduction.” *Explorations in Economic History* 48 (3): 343–56.

Bengtsson, Tommy, and George Alter. 2007. “Early-life conditions and old-age mortality in a comparative perspective: 19th century Sweden and Belgium.” In *Perspectives on Mortality Forecasting. 5. Cohort Factors: How Conditions in Early Life Influence Mortality Later in Life,* edited by Tommy Bengtsson, 61–80. Stockholm: Swedish Social Insurance Agency.

Bengtsson, Tommy, and Geraldine P. Mineau. 2009. “Early-life effects on socio-economic performance and mortality in later life: A full life-course approach using contemporary and historical sources. Introduction.” *Social Science & Medicine* 68 (9): 1561–64.

Bengtsson, Tommy, and Göran Broström. 2009. “Do conditions in early life affect old-age mortality directly and indirectly? Evidence from 19th-century rural Sweden.” *Social Science & Medicine* 68 (9): 1583–90.

Bengtsson, Tommy, and Martin Dribe. 1997. “Economy and demography in Western Scania, Sweden, 1650–1900.” *EAP Working Paper Series* 10.

Bengtsson, Tommy, and Martin Dribe. 2005. “New evidence on the standard of living in Sweden during the eighteenth and nineteenth centuries: Long-term development of the demographic response to short-term economic stress.” In *Living Standards in the Past: New Perspectives on Well-Being in Asia and Europe,* edited by Robert C. Allen, Tommy Bengtsson, and Martin Dribe, 341–72. Oxford: Oxford University Press.

Bengtsson, Tommy, and Martin Dribe. 2010. “Agency, social class, fertility in southern Sweden, 1766–1865.” In *Prudence and Pressure: Reproduction and Human Agency in Europe and Asia, 1700–1900,* edited by Noriko O. Tsuya, Wang Feng, and James Z. Lee, 159–94. Cambridge: MIT Press.

Bengtsson, Tommy, and Martin Dribe. 2011. "The late emergence of socioeconomic mortality differentials: A micro-level study of adult mortality in southern Sweden 1815–1968." *Explorations in Economic History* 48 (3): 389–400.

Bengtsson, Tommy, and Martin Dribe. 2014. "The historical fertility transition at the micro level: Southern Sweden 1815–1939." *Demographic Research* 30 (1): 493–534.

Bengtsson, Tommy, and Martin Lindström. 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* 54 (3): 263–77.

Bengtsson, Tommy, and Martin Lindström. 2003. "Airborne infectious diseases during infancy and mortality in later life in southern Sweden, 1766–1894." *International Journal of Epidemiology* 32 (2): 286–94.

Bengtsson, Tommy, and Rolf Ohlsson. 1994. "The demographic transition revisited." In *Population, Economy, and Welfare in Sweden*, edited by Tommy Bengtsson, 11–36. Berlin: Springer-Verlag.

Bengtsson, Tommy, Martin Dribe, and Patrick Svensson. 2012. *The Scanian Economic Demographic Database: Version 3.0 (Machine-Readable Database).* Lund: Lund University, Centre for Economic Demography *[Data]*.

Bengtsson, Tommy, Martin Dribe, Patrick Svensson, and Luciana Quaranta. 2014. *The Scanian Economic Demographic Database: Version 4.0 (Machine-Readable Database).* Lund: Lund University, Centre for Economic Demography *[Data]*.

Ben-Porath, Yoram. 1967. "The production of human capital and the life cycle of earnings." *Journal of Political Economy* 75 (4): 352–65.

Ben-Shlomo, Yoav, and Diana Kuh. 2002. "A life course approach to chronic disease epidemiology: Conceptual models, empirical challenges and interdisciplinary perspectives." *International Journal of Epidemiology* 21 (2): 285–93.

Bentley, Ronald. 2009. "Different roads to discovery; Prontosil (hence sulfa drugs) and Penicillin (hence beta-lactams)." *Journal of Industrial Microbiology & Biotechnology* 36 (6): p775–86.

Beyerlein, Andreas, Ewan Donnachie, Sibille Jergens, and Anette-Gabriele Ziegler. 2016. "Infections in early life and development of type 1 diabetes." *JAMA* 315 (17): 1899–1901.

Bhalotra, Sonia, and Atheendar S. Venkataramani. 2013. "Shadows of the captain of the men of death: Early life health interventions, human capital investments, and institutions." *IZA Working Papers* 7833.

Bhalotra, Sonia, Martin Karlsson, and Therese Nilsson. 2017. "Infant health and longevity: Evidence from a historical trial in Sweden." *Journal of the European Economic Association* In press.

Bhalotra, Sonia, Martin Karlsson, Therese Nilsson, and Nina Schwarz. 2016. "Infant health, cognitive performance and earnings: Evidence from inception of the welfare state in Sweden." *IZA Working Papers* 10339.

Bleakley, Hoyt. 2007. "Disease and development: Evidence from hookworm eradication in the American South." *Quarterly Journal of Economics* 122 (1): 73–117.

Bleakley, Hoyt. 2010a. “Health, human capital, and development.” *Annual Review of Economics* September (2): 283–310.

Bleakley, Hoyt. 2010b. “Malaria eradication in the Americas: A retrospective analysis of childhood exposure.” *American Economic Journal: Applied Economics* 2 (2): 1–45.

Bongaarts, John, and Rodolfo A. Bulatao. 2000. *Beyond Six Billion: Forecasting the World's Population.* Washington: National Academy Press.

Bourdelais, Patrice. 2002. “Cholera: A victory for medicine?” In *The decline of mortality in Europe*, edited by Roger Schofield, David Reher, and Alain Bideau, 118–30. Oxford: Clarendon Press.

Brändström, Anders, Sören Edvinsson, and John Rogers. 2002. “Illegitimacy, infant feeding practices and infant survival in Sweden 1750–1950: A regional analysis.” *An Interdisciplinary Journal for the History of Public Health* 3 (1): 13–52.

Brookhart, Alan M., and Sebastian Schneeweiss. 2007. “Preference-based instrumental variable methods for the estimation of treatment effects: Assessing validity and interpreting results.” *International Journal of Biostatistics* 3 (1): 1–14.

Buck, Carol, and Helen Simpson. 1982. “Infant diarrhoea and subsequent mortality from heart disease and cancer.” *Journal of Epidemiology and Community Health* 36 (1): 27–30.

Bütikofer, Aline, Eirin Mølland, and Kjell G. Salvanes. 2016. “Introducing a free nutritious school breakfast: Long-term impacts on education and adult earnings.” *Working Paper, Norwegian School of Economics.*

Bütikofer, Aline, Katrine V. Løken, and Kjell G. Salvanes. 2015. “Long-term consequences of access to well-child visits.” *IZA Working Papers* 9546.

Calkins, Kara, and Sherin U. Devaskar. 2011. “Fetal origins of adult disease.” *Current Problems in Pediatric and Adolecent Health Care* 41 (6): 158–76.

Carraro, Silvia, Eugenio Baraldi, Nienke Scheltema, and Louis Bont. 2014. “Early-life origins of chronic respiratory diseases: Understanding and promoting healthy ageing.” *European Respiratory Journal* 44 (6): 1682–96.

Cas, Ava G. 2012. “Early life public health intervention and adolescent cognition: Evidence from the Safe Motherhood program in Indonesia.” *Working Paper, Duke University.*

Case, Anne, and Christina Paxson. 2009. “Early life health and cognitive function in old age.” *American Economic Review* 99 (2): 104–9.

Catalano, Ralph, and Tim Bruckner. 2006. “Child mortality and cohort lifespan: A test of diminished entelechy.” *International Journal of Epidemiology* 35 (5): 1264–9.

Colchero, Fernando, Roland Rau, Owen R. Jones, Julia A. Barthold, Dalia A. Conde, Adam Lenart … James W. Vaupel. 2016. “The emergence of longevous populations.” *Proceedings of the National Academy of Sciences* 113 (48): E7681–90.

Colebatch, A. N., and C. J. Edwards. 2011. “The influence of early life factors on the risk of developing rheumatoid arthritis.” *Clinical and Experimental Immunology* 163 (1): 11–16.

Costa, Dora L., and Joanna N. Lahey. 2005. “Predicting older age mortality trends.” *Journal of the European Economic Association* 3 (2/3): 487–93.

Costa, Dora L., and Richard H. Steckel. 1997. “Long-term trends in health, welfare, and economic growth in the United States.” In *Health and Welfare during Industrialization*, edited by Richard H. Steckel and Roderick Flouds, 47–90. Chicago: University of Chicago Press.

Crimmins, Eileen M., and Caleb E. Finch. 2006. “Infection, inflammation, height, and longevity.” *Proceedings of the National Academy of Sciences of the United States of America* 103 (2): 498–503.

Cunha, Flavio, and James Heckman. 2007. “The technology of skill formation.” *The American Economic Review* 97 (2): 31–47.

Cunha, Flavio, James J. Heckman, Lance Lochner, and Dimitriy V. Masterov. 2006. “Interpreting the evidence on life cycle skill formation.” In *Handbook of the Economics of Education*, edited by Erik A. Hanushek and F. Welch, 697–812. St. Louis: Elsevier.

Currie, Janet, and Maya Rossin-Slater. 2015. “Early-life origins of life-cycle well-being: Research and policy implications.” *Journal of Policy Analysis and Management* 34 (1): 208–42.

Currie, Janet, and Tom Vogl. 2013. “Early-life health and adult circumstance in developing countries.” *Annual Review of Economics* 5: 1–36.

Curtis, Stephan. 2005. “Midwives and their role in the reduction of direct obstetric deaths during the late nineteenth century: The Sundsvall region of Sweden (1860–1890).” *Medical History* 49 (3): 321–50.

Cutler, David M., and Grant Miller. 2005. “The role of public health improvements in health advances: The twentieth-century United States.” *Demography* 42 (1): 1–22.

Cutler, David, Winnie Fung, Michael Kremer, Monica Singhal, and Tom Vogl. 2010. “Early-life malaria exposure and adult outcomes: Evidence from malaria eradication in India.” *American Economic Journal: Applied Economics* 2 (2): 72–94.

Danese, Andrea, and Bruce S. McEwen. 2012. “Adverse childhood experiences, allostasis, allostatic load, and age-related disease.” *Physiology & Behavior* 106 (1): 29–39.

Davey Smith, George, and John Lynch. 2005. “Life course influences on coronary heart disease.” In *Coronary Heart Disease Epidemiology: From Aetiology to Public Health*, edited by Michael Marmot and Paul Elliott, 1–46. Oxford: Oxford University Press.

Davey Smith, George, Sam Leary, and Andy Ness. 2006. “Could dehydration in infancy lead to high blood pressure?” *Journal of Epidemiology and Community Health* 60 (2): 142–3.

Delaney, Liam, Mark McGovern, and James P. Smith. 2011. “From Angela’s Ashes to the Celtic Tiger: Early life conditions and adult health in Ireland.” *Journal of Health Economics* 30 (1): 1–10.

Derrick, V. P. 1927. “Observations on (1) errors of age in the population statistics of England and Wales, and (2) the changes in mortality indicated by the national records.” *Journal of the Institute of Actuaries* 58 (2): 117–59.

Doblhammer, Gabriele. 2007. “The month of birth: Evidence for declining but persistent cohort effects in lifespan.” In *Perspectives on Mortality Forecasting. 5. Cohort Factors: How Conditions in Early Life Influence Mortality Later in Life*, edited by Tommy Bengtsson, 41–60. Stockholm: Swedish Social Insurance Agency.

Doblhammer, Gabrielle, and James W. Vaupel. 2001. “Lifespan depends on month of birth.” *Proceedings of the National Academy of Sciences of the United States of America* 98 (5): 29–34.

Domagk, Gerhard. 1957. “Twenty-five years of sulfonamide therapy.” *Annals of the New York Academy of Sciences* 69 (3): 380–84.

Duflo, Esther, Rachel Clennerster, and Michael Kremer. 2008. “Using randomization in development economics research: A toolkit.” In Paul T. Schultz and John Strauss, *Handbook of Development Economics*, 3895–962. North Holland: Elsevier.

Easterlin, Richard A. 1996. *Growth Triumphant: The Twenty-First Century in Historical Perspective.* Michigan: Ann Arbor, The University of Michigan Press.

Easterlin, Richard A. 1999. “How beneficent is the market? A look at the modern history of mortality.” *European Review of Economic History* 3 (3): 257–94.

Easterlin, Richard A. 2000. “Twentieth-century American population growth.” In *The Cambridge Economic History of the United States. Vol. 3. The Twentieth Century*, edited by Stanley L. Engerman, Robert E. Gallman, 505–48. Cambridge: Cambridge University Press.

Easterlin, Richard A. 2004. *The Reluctant Economist: Perspectives on Economics, Economic History, and Demography.* Cambridge: Cambridge University Press.

Engberg, Elisabeth. 2009. “The invisible pandemic: Community response to pandemic influenza in rural northern Sweden 1918–20.” *Varia Historia* 25 (42): 429–56.

Ferrie, Joseph P., and Werner Troesken. 2008. “Water and Chicago’s mortality transition, 1850-1925.” *Explorations in Economic History* 45 (1): 1–16.

Feyrer, James, Dimitra Politi, and David N. Weil. 2017. “The cognitive effects of micronutrient deficiency: Evidence from salt iodization in the United States.” *Journal of the European Economic Association* 15 (2): 355–87.

Finch, Caleb E. 2007. *The Biology of Human Longevity.* Oxford: Elsevier Inc.

Finch, Caleb E., and Eileen M. Crimmins. 2004. “Inflammatory exposure and historical changes in human life-spans.” *Science* 305 (5691): 1736–39.

Fliedl, Gottfried. 1998. *Gustav Klimt, 1862–1918: The World in Female Form.* Köln: Taschen.

Floud, Roderick, Robert W. Fogel, Bernard Harris, and Sok C. Hong. 2011. *The Changing Body: Health, Nutrition, and Human Development in the Western World since 1700.* Cambridge: Cambridge University Press.

Fogel, Robert W. 1994. “Economic growth, population theory, and physiology: The bearing of long-term processes on the making of economic policy.” *The American Economic Review* 84 (3): 369–95.

Fogel, Robert W. 1997. “Secular trends in nutrition and mortality.” In *Handbook of Population and Family Economics*, edited by Mark Rosenzweig and Oded Stark, 433–81. Amsterdam: Elsevier.

Fogel, Robert W. 2004. *The Escape from Hunger and Premature Death, 1700-2100: Europe, America and the Third World.* Cambridge: Cambridge University Press.

Fogel, Robert W., and Dora Costa. 1997. "A theory of technophysio evolution, with some implications for forecasting population, health care costs, and pension costs." *Demography* 34 (1): 49–66.

Forsdahl, A. 1977. "Are poor living conditions in childhood and adolescence an important risk factor for arteriosclerotic heart disease?" *British Journal of Preventive and Social Medicine* 31 (2): 91–5.

Frankenberg, Elizabeth, Wayan Suriastini, and Duncan Thomas. 2005. "Can expanding access to basic healthcare improve children's health status? Lessons from Indonesia's "Midwife in the Village" programme." *Population Studies* 59 (1): 5–19.

Fridlizius, Gunnar. 1984. "The mortality decline in the first phase of the demographic transition: Swedish experiences." In *Pre-Industrial Population Change: The Mortality Decline and Short-Term Population Movements*, edited by Tommy Bengtsson, 71–114. Stockholm: Almquist and Wiksell.

Fridlizius, Gunnar. 1989. "The deformation of cohorts: Nineteenth-century decline in a generational perspective." *Scandinavian Economic History Review* 37 (3): 3–17.

Gagnon, Alain, and Ryan Mazan. 2009. "Does exposure to infectious diseases in infancy affect old-age mortality? Evidence from a pre-industrial population." *Social Science & Medicine* 68 (9): 1609–16.

Gallup, John L., and Jeffrey D. Sachs. 2001. "The economic burden of malaria." *American Journal of Tropical Medicine and Hygeine* 64 (1-2): 85–96.

Galobardes, Bruna, John W. Lynch, and George Davey Smith. 2004. "Childhood socioeconomic circumstances and cause-specific mortality in adulthood: Systematic Review and interpretation." *Epidemiologic Reviews* 26 (1): p7–21.

Galor, Oded. 2011. *Unified Growth Theory.* Princeton: Princeton University Press.

Glied, Sherry, and Matthew Neidell. 2010. "The economic value of teeth." *The Journal of Human Resources* 45 (2): 468–96.

Gluckman, Peter D., and Mark Hanson. 2006. *Developmental Origins of Health and Disease.* Cambridge: Cambridge University Press.

Grossman, Michael. 1972. "On the concept of health capital and the demand for health." *Journal of Political Economy* 80 (2): 223–55.

Grossman, Michael. 2000. "The human capital model." In *Handbook of Health Economics*, edited by Anthony Culyer and Joseph Newhouse, 347–408. Amsterdam: Elsevier.

Haacker, Markus. 2012. "Contribution of increased life expectancy to living standards." *Working Paper, London School of Hygiene and Tropical Medicine.*

Hansen, Casper W., Peter S. Jensen, and Peter E. Madsen. 2017. "Preventing the white death: Tuberculosis dispensaries." *Working Paper, University of Copenhagen.*

Hardy, Anne. 1993. *The Epidemic Streets: Infectious Diseases and the Rise of Preventive Medicine 1856–1900.* Oxford: Oxford University Press.

Hardy, Anne. 2001. *Health and Medicine in Britain since 1860.* New York: Palgrave Macmillan.

Heckman, James J. 2007. “The economics, technology, and neuroscience of human capability formation.” *Proceedings of the National Academy of Sciences of the United States of America* 104 (33): 13250–5.

Hedefalk, Finn, Lars Harrie, and Patrick Svensson. 2017. “Spatiotemporal historical datasets at micro-level for geocoded individuals in five Swedish parishes, 1813–1914.” *Scientific Data* 4 (170046): 1–13.

Hjort, Jonas, Mikkel Sølvsten, and Miriam Wüst. 2017. “Universal investment in infants and long-run health: Evidence from Denmark’s 1937 home visiting program.” *American Economic Journal: Applied Economics* 9 (4): 78–104.

Högberg, Ulf. 2004. “The decline in maternal mortality in Sweden: The role of community midwifery.” *American Journal of Public Health* 94 (8): 1312–20.

Hollingsworth, Rogers J., Jerald Hage, and Robert A. Hanneman. 1990. *State Intervention in Medical Care: Consequences for Britain, France, Sweden, and the United States, 1890–1970.* London: Cornell University Press.

Hostinar, Camelia E., and Megan R. Gunnar. 2013. “The developmental effects of early life stress: An overview of current theoretical frameworks.” *Current Directions in Psychological Science* 22 (5): 400–406.

Howitt, Peter. 2005. “Health, human capital, and economic growth: A Schumpeterian perspective.” In *Health and Economic Growth: Findings and Policy Implications*, edited by Guillem López-Casasnovas, Berta Rivera, and Luis Currais, 19–40. Cambridge: MIT Press.

Hultkrantz, Lars, and Mikael Svensson. 2012. “The value of a statistical life in Sweden: A review of the empirical literature.” *Health Policy* 108 (2–3): 302–10.

Human Mortality Database. 2017. *http://www.mortality.org/ [Data]*.

Ingvar, Sven. 1939. “Om den croupösa pneumoniens behandling: Terapeutiska betraktelser med anledning av letalitetskurvan för 1,635 fall av croupös lunginflammation vid medicinska kliniken i Lund åren 1900–1939.” [On the treatment of croupous pneumonia: Therapeutic considerations from the lethality curve of 1,635 cases of croupous pneumonia at Lund medical clinic 1900–1939] *Läkartidningen* 1: 11–57.

Johannisson, Karin. 2006. “The people’s health: Public health policies in Sweden.” In *The History of Public Health and the Modern State*, edited by Dorothy Porter, 165–82. Amsterdam: GA Rodopi.

Jörberg, Lennart. 1972. *A History of Prices in Sweden, 1732–1914.* Lund: Lund University.

Jörberg, Lennart. 1994. “Structural change and economic growth in Sweden in the nineteenth century.” In *The Industrial Revolutions: The Industrial Revolution in Europe*, edited by Roy A. Church, Edward A. Wrigley, Lennart Jörberg, and Patrick K. O’Brien, 411–54. Oxford: Blackwell.

Kenkel, Don. 2003. “Using estimates of the value of a statistical life in evaluating consumer policy regulations.” *Journal of Consumer Policy* 26 (1): 1–21.

Kermack, W. O., P. L. McKinlay, and A. G. McKendrick. 1934. “Death rates in Great Britain and Sweden. Some general regularities and their significance.” *Lancet* 30 (31): 698–703.

Kesztenbaum, Lionel, and Jean-Laurent Rosenthal. 2017. “Sewers’ diffusion and the decline of mortality: The case of Paris, 1880–1914.” *Journal of Urban Economics* 98: 174–86.

Kommunarkivet Kågeröd. 1876–1930. *Kågeröd Kommunalnämnd Huvudbok 1876–1930 [Kågeröd’s Commune Ledger Book 1876–1930]*.

Kommunarkivet Kågeröd. 1901–1931. *Norra Krerflinge Kommunalnämnd Huvudbok 1901–1931* [*Norra Krerflinge’s Commune Ledger Book* 1901–1931].

Kommunarkivet Kävlinge. 1881–1930. *Kommunal Räkenskapsbok i Kjeflinge 1881–1930 [Kjeflinge’s Commune Ledger Book 1881–1930]*.

Kruse, Agneta. 2010. “A stable pension system: The eighth wonder.” In *Population Ageing - a Threat to the Welfare State? The Case of Sweden*, edited by Tommy Bengtsson and James W. Vaupel, 47–64. Dordrecht: Springer Berlin Heidelberg.

Kuh, Diana, and Yoav Ben-Shlomo. 2007. *A Life Course Approach to Chronic Disease Epidemiology*. 2nd. Oxford: Oxford University Press.

Kullberg, Anders F. 1877. *Författningar m.m. angående Medicinalväsendet i Sverige. 1860/1876 [Laws Related to Health Care in Sweden. 1860/1876]*. Stockholm: P.A. Norstedt & Söner.

Kunitz, Stephen J. 1984. “Mortality change in America, 1620–1920.” *Human Biology* 56 (3): 559–82.

Kunitz, Stephen J. 1986. “Mortality since Malthus.” In *The State of Population Theory: Forward from Malthus*, edited by D. Codelam and Roger S. Schofield, 279–302. Oxford: Basil Blackwell.

Kunitz, Stephen J. 2002. “The personal physician and the decline of mortality.” In *The Decline of Mortality in Europe*, edited by Roger S. Schofield, David Reher, and Alain Bideau, 248–62. Oxford: Clarendon Press.

Kunitz, Stephen J., and Stanley Engerman. 1992. “The ranks of death: Secular trends in income and mortality.” *Health Transition Review* 2: 29–46.

Landrigan, Philip J., Babasaheb Sonawane, Robert N. Butler, Leonardo Trasande, Richard Callan, and Daniel Droller. 2005. “Early environmental origins of neurodegenerative disease in later life.” *Environmental Health Perspectives* 113 (9): 1230–33.

Lawlor, Debbie A. 2008. “Commentary: The developmental origins of health and disease: Where do we go from here?” *Epidemiology* 19 (2): 206–8.

Lee, Ronald. 1981. “Short-term variation: Vital rates, prices, and weather.” In *The Population History of England 1541–1871*, edited by Edward A. Wrigley and Roger S. Schofield, 356–401. Cambridge: Cambridge University Press.

Leifer, Cynthia A., and Rodney R. Dietert. 2011. “Early life environment and developmental immunotoxicity in inflammatory dysfunction and disease.” *Toxilogical and Environmental Chemistry* 93 (7): p1463–85.

Lindblom, Paul. 1967. *Malmöhus Läns Landsting 1863–1962 [Malmöhus County Councils]*. Stockholm: Godvil.

Link, Bruce G., and Jo C. Phelan. 1996. “Understanding sociodemographic differences in health - the role of fundamental social causes.” *American Journal of Public Health* 86 (4): 471–73.

Liuba, Petru, and Erkki Pesonen. 2005. “Infection and early atherosclerosis: Does the evidence support causation?” *Acta Paediatrica* 94 (6): 643–51.

Livi-Bacci, Massimo. 1991. *Population and Nutrition: An Essay on European Demographic History*. Cambridge: Cambridge University Press.

Lorentzen, Peter, John McMillan, and Romain Wacziarg. 2008. “Death and development.” *Journal of Economic Growth* 13 (2): 81–124.

Lucas, Adrienne M. 2010. “Malaria eradication and educational attainment: Evidence from Paraguay and Sri Lanka.” *American Economic Journal: Applied Economics* 2 (2): 46–71.

Lucas, Robert E. 1988. “On the mechanics of economic development.” *Journal of Monetary Economics* 22 (1): 3–42.

Lucas, Robert E. 2000. “Some macroeconomics for the 21st century.” *The Journal of Economic Perspectives* 14 (1): 159–68.

Lucas, Robert E. 2002. *Lectures on Economic Growth:* Cambridge, Mass: Harvard University Press.

Lynch, John, and George Davey Smith. 2005. “A life course approach to chronic disease epidemiology.” *Annual Review of Public Health* 26: 1–35.

Macassa, Gloria, Antonio P. de Leon, and Bo Burström. 2006. “The impact of water supply and sanitation on area differentials in the decline of diarrhoeal disease mortality among infants in Stockholm 1878–1925.” *Scandinavian Journal of Public Health* 34 (5): 526–33.

Mankiw, N. Gregory, David Romer, and David N. Weil. 1992. “A contribution to the empirics of economic growth.” *The Quarterly Journal of Economics* 2: 407–37.

McDade, Thomas W., Julienne Rutherford, Linda Adair, and Christopher W. Kuzawa. 2010. “Early origins of inflammation: Microbial exposures in infancy predict lower levels of c-reactive protein in adulthood.” *Proceedings: Biological Sciences* 277 (1684): 1129–37.

McKeown, Thomas. 1976. *The Modern Rise of Population*. London: Arnold.

McKeown, Thomas. 1980. *The Role of Medicine: Dream, Mirage or Nemesis?* Princeton: Princeton University Press.

McKeown, Thomas, R. G. Brown, and R. G. Record. 1972. “An interpretation of the modern rise of population in Europe.” *Population Studies* 26 (3): 345–82.

McNeill, William H. 1989. *Plagues and Peoples.* New York: Anchor Books.

Medicinalstyrelsen. 1907. *Betänkande Angående Rikets Indelning i Läkardistrikt Samt Tjänstläkarnes Anställning och Åligganden [Report on the Division of the Country on Doctor Districts and the Employment Services of the Doctors]*. Stockholm: P.A. Norstedt & Söner.

Medicinalstyrelsen. 1939. *Rikets Indelning i Provinsialläkardistrikt före 1/7 1939 och Medicinalstyrelsens Yttrande och Förslag till Stadsläkarsakuniga den Maj 1932 [Division of the Country on Provincial Doctor Districts before 1/7 1939 and the Health Board’s Opinion and Proposals to Provincial Doctors on May 1932]*. Stockholm: P.A. Norstedt & Söner.

Mercer, Alex. 1990. *Disease, Mortality and Population in Transition: Epidemiological-Demographic Change in England since the Eighteenth Century as part of a Global Phenomenon*. Leicester: Leicester University Press.

Miller, Gregory E., Edith Chen, and Karen J. Parker. 2011. "Psychological stress in childhood and susceptibility to the chronic diseases of aging: Moving toward a model of behavioral and biological mechanisms." *Psychological Bulletin* 137 (6): 959–97.

Mincer, Jacob. 1974. *Schooling, Experience, and Earnings*. New York: Columbia University Press.

Moberg, Harald A. 1989. *Jordbrukets Mekanisering i Sverige under Tre Sekel [Agricultural Mechanization in Sweden during Three Centuries]*. Stockholm: LTs förlag.

Mokyr, Joel. 1998. "Induced technical innovation and medical history: An evolutionary approach." *Journal of Evolutionary Economics* 8 (2): 119–37.

Mokyr, Joel. 2002. *The Gifts of Athena: Historical Origins of the Knowledge Economy*. Princeton: Princeton University Press.

Mokyr, Joel. 2005. "Long-term economic growth and the history of technology." In *Handbook of Economic Growth*, edited by Philippe Aghion and Steven N. Duralf, 1–74. North Holland: Elsevier.

Mokyr, Joel, and Rebecca Stein. 1996. "Science, health, and household technology: The effect of Pasteur revolution on consumer demand." In *The Economics of New Goods*, edited by Timothy F. Bresnahan and Robert J. Gordon, 143–206. Chicago: University of Chicago Press.

Montez, Jennifer K., and Mark D. Hayward. 2011. "Early life conditions and later life mortality." In *International Handbook of Adult Mortality*, edited by Richard G. Rogers and Eileen M. Crimmins, 187–206. New York: Springer Science and Business Media.

Mooney, Graham. 2015. *Intrusive Interventions: Public Health, Domestic Space, and Infectious Disease Surveillance in England, 1840–1914*. Rochester: University of Rochester Press.

Murphy, Kevin M., and Robert H. Topel. 2006. "The value of health and longevity." *Journal of Political Economy* 114 (5): 871–904.

Myrskylä, Mykko, Neil K. Mehta, and Virginia W. Chang. 2013. "Early life exposure to the 1918 influenza pandemic and old-age mortality by cause of death." *American Journal of Public Health* 103 (7): e83–90.

Niemi, Miriam. 2007. *Public Health and Municipal Policy Making. Britain and Sweden, 1900-1940*. Aldershot: Ashgate.

Nordhaus, William D. 2003. "The health of nations: The contribution of improved health to living standards." In *Measuring the Gains from Medical Research: An Economic Approach*, edited by Kevin M. Murphy and Robert Topel, 9–40. Chicago: The University of Chicago Press.

Öberg, Stefan. 2014. *Social Bodies: Family and Community Level Influences on Height and Weight, Southern Sweden 1818–1968*. Gotheborg: University of Gotheborg.

Oeppen, Jim, and James W. Vaupel. 2002. “Broken limits to life expectancy.” *Science* 296: 1029–31.

Omran, Abdel R. 1971. “The epidemiologic transition: A theory of the epidemiology of population change.” *Milbank Memorial Fund* 29: 509–38.

Omran, Abdel R. 1998. “The epidemiologic transition theory revisited thirty years later.” *World Health Statistics Quarterly* 51 (2–4): 99–119.

Palme, Marten O., and Robert E. Wright. 1998. “Changes in the rate of return to education in Sweden: 1968–1991.” *Applied Economics* 30 (12): 1653–63.

Perrenoud, Alfred. 1984. “The mortality decline in a long term perspective.” In *Pre-Industrial Population Change: The Mortality Decline and Short-Term Population Movements*, edited by Tommy Bengtsson, Gunnar Fridlizius, and Rolf Ohlsson, 41–70. Stockholm: Almqvist & Wiksell International.

Pettersson-Lidbom, Per. 2014. “Midwifes and maternal mortality: Evidence from a midwifery policy experiment in Sweden in the 19th century.” *Unpublished manuscript*.

Pliktverket. 2000. *Resultat från Mönstringen. Statistik från 2000 [Reports from the Recruitment Commitee. Statistics from 2000]*. Pliktverket.

Prado, Svante. 2010. “Nominal and real wages of manufacturing workers, 1860–2007.” In *Historical Monetary and Financial Statistics for Sweden*, edited by R. Edvinsson, T. Jacobson, and Daniel Waldenström, 479–527. Stockholm: Sveriges Riksbank and Ekerlids.

Preston, Samuel H. 1975. “The changing relation between mortality and level of economic development.” *Population Studies* 29 (2): 484–90.

Preston, Samuel H. 1996. “American longevity: Past, present and future.” *Syracuse University Policy Brief* 7: 3–18.

Preston, Samuel H., and Michael R. Haines. 1991. *Fatal Years: Child Mortality in Late Nineteenth-Century America*. Princeton: Princeton University Press.

Preston, Samuel H., M. E. Hill, and G. L. Drevenstedt. 1998. “Childhood conditions that predict survival to advanced ages among African-Americans.” *Social Science and Medicine* 47 (9): 1231–46.

Preston, Samuel H., Nathan Keyfitz, and Robert Schoen. 1972. *Causes of Death: Life Tables for National Populations*. New York: Seminar Press.

Quaranta, Luciana. 2013. *Scarred for Life: How Conditions in Early Life Affect Socioeconomic Status, Reproduction and Mortality in Southern Sweden, 1813–1968*. Lund: Lund University.

Quaranta, Luciana. 2014. “Early life effects across the life course: The impact of individually defined exogenous measures of disease exposure on mortality by sex in 19th- and 20th-century southern Sweden.” *Social Science & Medicine* 119: 266–73.

Rahm, Nils. 1939. “Pneumonibehandling.” [Treatment of Pneumonia] *Läkartidningen* 28: 2044–60.

Regionarkivet Lund. 1881–1888. *Sammandrag av Provinsialläkarens Årsberättelser i Landskrona/Helsingborg [Summary Reports of the Provincial Doctors in Landskrona/Helsingborg]*.

Regionarkivet Lund. 1881–1899. *Sammandrag av Provinsialläkarens Årsberättelser i Malmö [Summary Reports of the Provincial Doctors in Malmö]*.

Regionarkivet Lund. 1889a–1941. *Årsberättelser från Hälsovårdsnämnderna i Halmstad [Report of the Local Health Board in Halmstad]*.

Regionarkivet Lund. 1889b–1941. *Årsberättelser från Hälsovårdsnämnderna i Kågeröd. [Report of the Local Health Board in Kågeröd]*.

Regionarkivet Lund. 1889–1946. *Sammandrag av Provinsialläkarens Årsberättelser i Teckomatorp [Summary Reports of the Provincial Doctors in Teckomatorp]*.

Regionarkivet Lund. 1900–1946. *Sammandrag av Extra Provinsialläkarens Årsberättelser i Kävlinge [Summary Reports of the Extra Provincial Doctors in Kävlinge]*.

Regionarkivet Lund. 1901. *Sammandrag av Extra Provinsialläkarens Årsberättelser i Kävlinge [Summary Reports of the Extra Provincial Doctors in Kävlinge District]*.

Regionarkivet Lund. 1913–1941. *Årsberättelser från Hälsovårdsnämnderna i Sireköpinge. [Report of the Local Health Board in Sireköpinge]*.

Reuterswärd, Elisabeth, and Franceska Olsson. 1993. "Skånes Demografiska Databas 1646–1894: En Källbeskrivning." [Scanian Demographic Database 1646–1894: A Source Description] *Lund Papers in Economic History* 33.

Riksarkivet. 1920–1967. *Medicinalstyrelsens apoteksbyrå. Arkiv Kungliga Medicinalstyrelsen [National Archive of the National Health Board. Records of the Pharmaceutical Agency]*.

Riksarkivet. 2014. *National Sample of the 1880–1910 Census of Sweden [Decennial]*.

Riley, James C. 2001. *Rising Life Expectancy: A Global History*. New York: Cambridge University Press.

Romer, Paul M. 1986. "Increasing returns and long-run growth." *Journal of Political Economy* 94 (5): 1002–37.

Romer, Paul M. 1990. "Endogenous technological change." *Journal of Political Economy* 98(5): S71–102.

Romlid, Christina. 1997. "Swedish midwives and their instruments in the eighteenth and nineteenth centuries." In *Midwives, Society and Childbirth: Debates and Controversies in the Modern Period*, edited by Hilary Marland and Anne M. Rafferty, 38–61. London: Routledge.

Romlid, Christina. 1998. *Makt, Motstånd och Förändring: Vårdens Historia Speglad Genom Det Svenska Barnmorskeväsendet 1663–1908. [Power, Resistance and Change: The History of Service Reflected by the Swedish Midwifery 1663–1908]* Stockholm: Vårdförbundet.

Rosenzweig, Mark R., and Kenneth I. Wolpin. 2000. "Natural "natural experiments" in economics." *Journal of Economic Literature* 38 (4): 827–74.

Rudan, Igor, Katherine L. O'Brien, Harish Nair, Li Liu, Evropi Theodoratou, Shamim Qazi, and Ivana Lukšić. 2013. "Epidemiology and etiology of childhood pneumonia in 2010: Estimates of incidence, severe morbidity, mortality, underlying risk factors and causative pathogens for 192 countries." *Journal of Global Health* 3 (1): 46–59.

Ryen, Linda, and Mikael Svensson. 2015. "The willingness to pay for a quality adjusted life year: A review of the empirical literature." *Health Economics* 24 (10): 1289–1301.

Sachs, Jeffrey D. 2003. "Institutions don't rule: Direct effects of geography on per capita income." *NBER Working Papers* 9490.

Sandberg, Lars, and Richard H. Steckel. 1997. "Was industralization hazardous to your health? Not in Sweden." In *Health and Welfare during Industrialization*, edited by Richard H. Steckel and Roderick Floud, 127–59. Chicago: University of Chicago Press.

Schellekens, Jona, and Frans van Poppel. 2016. "Early-life conditions and adult mortality decline in Dutch cohorts born 1812–1921." *Population Studies* 70 (3): 327–43.

Schofield, Roger S. 1984. "Population growth in the century after 1750: The role of mortality decline." In *Pre-industrial Population Change*, edited by Tommy Bengtsson, Gunnar Fridlizius, and Rolf Ohlsson, 17–39. Stockholm: Almquist and Wiskell International.

Schofield, Roger S., and David Reher. 2002. "The decline of mortality in Europe." In *The Decline of Mortality in Europe*, edited by Roger S. Schofield, David Reher, and Alain Bideau, 1–17. Oxford: Clarendon Press.

Schön, Lennart, and Olle Krantz. 2012. "Swedish historical national accounts 1560-2010." *Lund Papers in Economic History* 123.

Schultz, Theodore W. 1961. "Investment in human capital." *The American Economic Review* 51 (1): 1–17.

Smith, David W., and Benjamin S. Bradshaw. 2006. "Variation in life expectancy during the twentieth century in the United States." *Demography* 43 (4): 647–57.

Soares, Rodrigo R. 2007. "On the determinants of mortality reductions in the developing world." *Population and Development Review* 33 (2): 247–87.

Solow, Robert M. 1956. "A contribution to the theory of economic growth." *The Quarterly Journal of Economics* 1: 65–94.

SOU. 1929. *Statens Offentliga Utredningar: Betäkande Angående Moderkapsskydd avgivet den 26 September 1929. [Ministry of Health and Social Affairs: Statement of Parental Protection Issued on September 26, 1929*] 28. Stockholm: Socialdepartementet.

SOU. 1994. *Statens Offentliga Utredningar: Kvinnor, Barn och Arbete i Sverige 1850–1993. [Ministry of Foreign Affairs: Women, Children and Work in Sweden 1850–1993*] 38. Stockholm: Utrikesdepartementet.

Statistics Sweden. 2017. *National Accounts: Quarterly and Annual Estimates 1980–2016.* Stockholm: Statistics Sweden.

Statistiska Centralbyrån. 1855–1910. *BISOS A: Bidrag till Sveriges Officiella Statistik A. Befolkningsstatistik. [Contribution to Sweden's Official Statistics A. Population Statistics]*. Stockholm: P.A. Norstedt & Söner [Annual Volumes].

Statistiska Centralbyrån. 1855–1944. *SOS: Sveriges Officiella Statistik. Allmän om Hålso och Sjukvård [Sweden's Official Statistics. Yearbook on Health and Health Care]*. Stockholm: P.A. Norstedt & Söner [Annual Volumes].

Statistiska Centralbyrån. 1856–1905. *BISOS H. Bidrag till Sveriges Officiella Statistik. Befallningshavandes Femårsberättelser [Contribution to Sweden's Official Statistics H. Economy]*. Stockholm: P.A. Norstedt & Söner [Annual Volumes].

Statistiska Centralbyrån. 1874–1917. *BISOS U: Bidrag till Sveriges Officiella Statistik U. Kommunernas Fattigvård och Finanser [Contribution to Sweden's Official Statistics U. Commune Poor relief and Finances]*. Stockholm: P.A. Norstedt & Söner [Annual Volumes].

Statistiska Centralbyrån. 1880–1910. *BISOS K: Bidrag till Sveriges Officiella Statistik K. Helso och Sjukvården [Contribution to Sweden's Official Statistics K. Health and Health Care]*. Stockholm: P.A. Norstedt & Söner [Annual Volumes].

Statistiska Centralbyrån. 1880–1917. *BISOS U: Bidrag till Sveriges Officiella Statistik U. Kommunernas Fattigvård och Finanser [Contribution to Sweden's Official Statistics U. Commune Poor relief and Finances]*. Stockholm: Norstedt & Söner [Annual Volumes].

Statistiska Centralbyrån. 1911–1920. *Sveriges Officiella Statistik. Allmän om Hålso och Sjukvård [Sweden's Official Statistics. Yearbook on Health and Health Care]*. Stockholm: Norstedt & Söner [Annual Volumes].

Statistiska Centralbyrån. 1911–1944. *Sveriges Officiella Statistik. Befolkningsrörelsen [Sweden's Official Statistics. Population Movement]*. Stockholm: P.A. Norstedt & Söner [Annual Volumes].

Statistiska Centralbyrån. 1918–1930. *Sveriges Officiella Statistik. Kommunernas Finanser [Sweden's Official Statistics. Commune Finances]*. Stockholm: P.A. Norstedt & Söner [Annual Volumes].

Statistiska Centralbyrån. 1918–1944. *Sveriges Officiella Statistik. Kommunernas Finanser [Sweden's Official Statistics. Commune Finances]*. Stockholm: P.A. Norstedt & Söner [Annual Volumes].

Statistiska Centralbyrån. 1920a–1950. *Sveriges Officiella Statistik. Befolkningsrörelsen [Sweden's Official Statistics. Population Movement]*. Stockholm: P.A. Norstedt & Söner [Annual Volumes].

Statistiska Centralbyrån. 1920b–1950. *Sveriges Officiella Statistik. Dödsorsaker [Sweden's Official Statistics. Causes of Death]*. Stockholm: P.A. Norstedt & Söner [Annual Volumes].

Statistiska Centralbyrån. 1920c–1950. *Sveriges Officiella Statistik. Statistik Årsbok för Sverige [Sweden's Official Statistics. Statistics Yearbook for Sweden]*. Stockholm: P.A. Norstedt & Söner [Annual Volumes].

Statistiska Centralbyrån. 1920d–1950. *Sveriges Officiella Statistik. Allmän om Hålso och Sjukvård [Sweden's Official Statistics. Yearbook on Health and Health Care]*. Stockholm: P.A. Norstedt & Söner [Annual Volumes].

Statistiska Centralbyrån. 1925–1950. *Sveriges Officiella Statistik. Industri och Bergshantering [Sweden's Official Statistics. Industry and Mining]*. Stockholm: Isaac Marcus Boktryckeri-Aktiebolag [Annual Volumes].

Statistiska Centralbyrån. 1931–1959. *Sveriges Officiella Statistik. Konsumentpriser och Indexberäkningar [Sweden's Official Statistics. Consumer Prices and Indices]*. Stockholm: P.A. Norstedt & Söner.

Statistiska Centralbyrån. 1969. *Historisk Statistik för Sverige. Del 1. Befolkning 1720–1967 [Historical Statistics for Sweden. Part 1. Population 1720–1967]*. Stockholm: KL Beckmans Tryckerier AB.

Statistiska Centralbyrån. 1982–1983, 2013. *Sveriges Officiella Statistik. Statistik Årsbok för Sverige [Sweden's Official Statistics. Statistics Yearbook for Sweden]*. Stockholm: P.A. Norstedt & Söner [Annual Volumes].

Statistiska Centralbyrån. 1999. *Befolkningsutvecklingen under 250 År: Historisk Statistik för Sverige [Population Development for 250 Years: Historical Statistics for Sweden]*. Halmstad: Bulls Tryckeri.

Statistiska Centralbyrån. 2010. *Cohort Mortality in Sweden: Mortality Statistics since 1861*. Stockholm: Statistiska Centralbyrån.

Strauss, John, and Duncan Thomas. 2008. "Health over the life course." In *Handbook of Development Economics*, edited by Thomas P. Schultz and John Strauss, 3375–474. North Holland: Elsevier.

Sundin, Jan, and Sam Willner. 2007. *Social Change and Health in Sweden – 250 years of Politics and Practice.* Stockholm: Swedish National Institute of Public Health.

Svensk författningssamling 1881: 30. *Dagboksplikt. Kongl. Medicialstyrelsens Cirkulär till Barnmorskorna i Riket, Angående Försigtighetsmått, som Böra af Dem Iakttagas till Förekommande Utfärdadt den 13 Juni 1881 [Swedish Code of Statutes. Diaries. Circular of the National Health Board to Midwives in the Country from 13 June 1881]*. Stockholm: Svensk författningssamling.

Svensk författningssamling 1893: 61. *Kongl. Maj:ts Nådiga Kungörelse, Angående Vissa Åtgärder till Förekommande af Kolerans Utbredning bland Rikets Invånare [Swedish Code of Statutes. Certain Measures to Prevent the Spread of Cholera among the Residents]*. Stockholm: Svensk författningssamling.

Swan, Trevor W. 1956. "Economic growth and capital accumulation." *Economic Record* 32 (2): 334–61.

Szreter, Simon. 1988. "The importance of social intervention in Britain's mortality decline c.1850–1914: A Re-interpretation of the role of public health." *Social History of Medicine* 1 (1): 1–38.

Szreter, Simon. 2003. "The population health approach in historical perspective." *American Journal of Public Health* 93 (3): 421–31.

Szreter, Simon. 2004. "Health, economy, state and society in modern Britain: The long-run perspective." *Hygiea Internationalis* 4 (1): 205–27.

The Maddison-Project. 2013. *http://www.ggdc.net/maddison/maddison-project/home.htm [Data]*.

Tomes, Nancy. 1998. *The Gospel of Germs: Men, Women, and the Microbe in American Life.* Cambridge: Harvard University Press.

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*. New York: United Nations.

United Nations. 1973. *The Determinants and Consequences of Population Trends: New Summary of Findings on Interaction of Demographic, Economic and Social Factors*. New York: United Nations.

van den Berg, Gerard J., Gabriele Doblhammer, and Kaare Christensen. 2009. "Exogenous determinants of early-life conditions, and mortality later in life." *Social Science and Medicine* 68 (9): 1591–98.

van Hofsten, Erland A., and Hans Lundström. 1976. *Swedish Population History. Main Trends from 1750 to 1970.* Stockholm: Statistiska Centralbyrån.

van Zon, Adriaan, and Joah Muysken. 2005. "Health as a principal determinant of economic growth." In *Health and Economic Growth: Findings and Policy Implications*, edited by Guillem López-Casasnovas, Berta Rivera and Luis Currais, 41–66. Cambridge: MIT Press.

Venkataramani, Atheendar S. 2012. "Early life exposure to malaria and cognition in adulthood: Evidence from Mexico." *Journal of Health Economics* 31 (5): 767–80.

Viscusi, Joseph E. 2010. "Policy challenges of the heterogeneity of the value of statistical life." *Foundations and Trends in Microeconomics* 6 (2): 99–172.

Waldenström, Daniel. 2014. "Swedish stock and bond returns, 1856–2012." In *Historical Monetary and Financial Statistics for Sweden, Volume II: House Prices, Stock Returns, National Accounts, and the Riksbank Balance Sheet, 1620–2012*, edited by Rodney Edvinsson and Daniel Waldenström. Stockholm: Sveriges Riksbank and Ekerlids.

Waterland, Robert A., and Karin B. Michels. 2007. "Epigenetic epidemiology of the developmental origins hypothesis." *Annual Review of Nutrition* 27: 363–88.

Weil, David N. 2013. "Health and economic growth." In *Handbook of Economic Growth*, edited by Philippe Aghion and Steven N. Durlauf, 623–82. North Holland: Elsevier.

Willerson, James T., and Paul M. Ridker. 2004. "Inflammation as a cardiovascular risk factor." *Circulation* 109 (21): II-2–10.

Wisselgren, Maria J. 2005. *Att Föda Barn – Från Privat till Offentlig Angelägenhet [To Bear Children – From Private to Public Concern]*. Umeå: Umeå University.

Woods, Robert I., Peter A. Watterson, and Joan H. Woodward. 1989. "The causes of rapid infant mortality decline in England and Wales, 1861–1921 Part II." *Population Studies* 43: 113–32.

Wrigley, Edward A., and Roger S. Schofield. 1981. *The Population History of England 1541–1871: A Reconstruction*, edited by Edward A. Wrigley and Roger S. Schofield, with contrib. by Ronald Lee and Jim Oeppen. London: Arnold.

Yayachandran, Seema, Adriana Lleras-Muney, and Kimberly V. Smith. 2010. "Modern medicine and the twentieth century decline in mortality: Evidence on the impact of sulfa drugs." *American Economic Journal: Applied Economics* 2 (2): 118–146.

Defeating Disease

This thesis has the general aim of exploring the role of public health and medical innovations between 1880 and 1945, targeting infectious diseases, in health and income improvements in Sweden throughout the twentieth century. It applies methods of causal inference to longitudinal individual-level data from both local and national datasets combined with multisource archival data on the implementation of reforms. The bacteriological discoveries of the latter part of the nineteenth century helped the public to target infectious diseases through isolation, disinfection and treatment with antibiotics. The studies of this thesis show that health interventions have had a sizable role in the contemporaneous decline in infectious disease rates. Moreover, these studies find that by reducing the likelihood of infection, societal health measures led to beneficial consequences for the income and long-term health of the affected cohorts, extending through old age.

This thesis therefore supports the endogenous growth theory, which posits an interaction between health improvements in early childhood and health technology, based on application of the germ theory of disease, as a determinant of economic growth in the long run. Although the health benefits due to this dynamic process cannot be viewed as a main driver of economic growth, societal health investments nonetheless led to income growth sufficient to yield a high rate of return on the investment. The cause – that is, the reforms under study that are targeting infectious disease – and the consequence, reductions in the rates of specific chronic diseases in adulthood and old age, point to the inflammation mechanisms behind the lasting effects. The studies also suggest that the early neonatal period and infancy are the critical period for intervening such processes.

Printed by Media-Tryck, Lund 2017 NORDIC ECOLABEL, 3041 0903

LUND UNIVERSITY

Department of Ecomomic History
School of Economics and Management

Lund Studies in Economic History 83
ISBN 978-91-87793-38-7
ISSN 1400-4860