LUND UNIVERSITY

The lasting health and income effects of public health formation in Sweden

Lazuka, Volha

2016

[Link to publication](#)

The lasting health and income effects of public health formation in Sweden

Volha Lazuka

Centre for Economic Demography and Department of Economic History,

Lund University

12 October 2016

Abstract

Socio-economic inequalities are remarkable in contemporary developed countries, and continue to grow. The sources of these phenomena are not understood, and there is no agreement about at which part of an individual's life they originate, whether in adulthood or in early childhood. The literature showing that health in infancy may be an important factor of later-life health and income trajectories is expanding, but empirical evidence is still scarce. This paper is the first to link individuals' differences in access to better health care during infancy to income and health outcomes in old ages. Due to the public health care reform that became one of the first elements of the Swedish welfare state, gradually between 1890 and 1917, all rural areas established local health districts which implemented preventive measures with regard to the spread of the infectious diseases. Using administrative longitudinal population data and exploiting variation in the timing of the implementation of the reform across parishes, we examine whether treated individuals have advantages in old ages. Our findings indicate that treatment by the public health care in infancy leads to a significant reduction in mortality, with the largest effects among the cardiovascular diseases, and to an increase in individuals' permanent incomes. The effects are universal across different subpopulations, with somewhat stronger responses among individuals from poor socio-economic backgrounds.

Introduction and literature review

There is a rising concern about the widening socio-economic inequalities in the contemporary developed countries. Socio-economic differences in mortality and disease enlarged throughout the second half of the twentieth century in the US and Western countries (Palloni *et al.* 2009, Mackenbach *et al.* 2008). Almost simultaneously, income and wage inequalities grew and continue to grow within countries (Autor 2014; Deaton 2013). Such similarity between income and health development today has been long placed in parallel with historical improvements in health and economic growth (Haines & Ferrie 2011). Encouraged by Kuznets (1955, 1963), the scholars hypothesized the existence of strong relations between improvements in living conditions due to the industrial revolution and income inequality. In a similar manner, distribution of health in a society had been seen as an outcome of economic growth, where different socio-economic groups receive relatively different gains (McKeown 1976). To date, it has become apparent that empirically such propositions did not find support in case of the historical evolution of either income or mortality inequalities (Piketty & Saez 2003; Bengtsson & van Poppel 2011). The strong commonalities between these phenomena promoted another line of research that is seeking for the link from health to income and income inequalities instead. This course has been to a large extent inspired by Preston (1975) who related the mortality decline between the 1930s and 1960s to the advances in public health and medical science rather than to the gradual increases in incomes. The more promising area of this research is a life course approach, in particular those models which link circumstances in adulthood to the differences in child health induced by the environment or family resources (Kuh & Ben-Schlomo 2004).

The long-term pathway from health to incomes rapidly receives attention in the macroeconomic literature. The bulk of the studies are modeling health and income growth in static relations. In a neoclassical tradition, health is considered as a form of human capital among other inputs which affect labour productivity (Bloom, Canning & Sevilla 2004). In a similar manner, health improvements could additionally act as inputs to population and total factor productivity (Kalemli-Ozcan, Ryder & Weil 2000). Health and human capital are assumed to be crucial for economic growth in endogenous growth models where they are self-perpetuating and thereby enable the labour productivity to grow beyond the provided inputs (Lucas 1988, 2009; Romer 1990; de la Croix, & Licandro 1999). The co-evolution of technological knowledge and institutions, in particular those which promoted public health, could be also central to the long-run economic growth (Mokyr 2005; Galor 2005; Agenor & Neanidis 2011). The majority of the empirical country-level studies examining contemporaneous relations find robust reduced-form effects of

health on income and more moderate effects on income per capita (Sachs 2002; Weil 2007; Becker, Philipson & Soares 2005; Bloom & Canning; Acemoglu & Johnson 2007; Fogel 1994, 1997; see also for review Weil 2013). The emerging strand of growth literature goes beyond period effects of health on income and considers its dynamic nature. The reasoning for this is borrowed from the recent achievements in economic and epidemiological studies which emphasize the relatively larger benefits of the health interventions for the younger segment of population (Bleakley 2010). Under this view, the full benefits of health improvements are realized only in a long run, not least than over the half of a century. The related empirical findings, which are based on simulation methods, demonstrate that the impacts of the health on income per capita account for a sizable portion of their correlation (Ashraf, Lester & Weil 2009; Young 2005).

To date, the microeconomic literature has provided the well-defined reasons for the causation running from health to income. In a purely economic view, the improvements in health could be seen ultimately as direct inputs to workers' efficiency, in both physical and mental terms (Grossman 1972, 2000). There are also several indirect, proximate, channels through which health influences incomes. Regarding those running through human capital, health gains enables to acquire more and better schooling as well as induces the incentives to learn due to longer lives (Ben-Porath 1967; Hazan 2009). Similarly, capital formation and overall productivity are fostered as healthier workers use it more efficiently and partly as increased life prospects encourage individuals' saving rates (Bloom, Canning & Graham 2003). Taking all these arguments, in order to obtain the largest health and income benefits health inputs should be made in younger ages. This notion has been further supported by the testimonies from the epidemiological studies which highlight the lifelong effects of the inputs into the physiological and cognitive development in early childhood (Gluckman *et al.* 2008). There is also an emerging economic literature that demonstrates that beginning from early childhood health and human capital formation occurs in dynamic and self-reinforcing way, pronouncing themselves in health and incomes across the entire lives (Heckman 2007; Cunha & Heckman 2007). An additional mechanism runs through the responses of parental resources to the health improvements of children in a form of changes in fertility or within-family allocation of resources (Soares 2005; Currie 2009; Cunha, Heckman & Schennach 2010). The income effects observed later in life are therefore by-product of both environmental conditions and parental responses to the changes in child health.

In the recent decades, the micro empirical studies on long term health and income effects of changes in child health expanded hastily. The overwhelming part of this literature examines the correlations between the child health and parental socio-economic status and their development which enable to

study broad relations. The evidence from the plentiful studies consistently suggests that differences in socio-economic origin strongly associate with child health status and persist as children age (Currie, Schields, & Price 2007; Currie & Stabile 2003; Flores & Kalwij 2014; see also for review Currie 2009). In a similar manner, parental socio-economic status correlates with education and wealth in working and old ages (Case & Paxson 2010). The long-term causal studies of this kind are rare and concerned with effects for the most disadvantaged groups enhanced due to cash transfers, although they tend to establish to the link of child health to education and incomes in working ages (Aizer *et al.* 2014; Hoynes, Schanzenbach & Almond 2016). Empirically, our knowledge about the causality from early childhood and later health and incomes relies largely on the studies using negative events in environmental conditions as identification strategies. Most consistently, micro level studies based on infectious disease outbreaks or nutrition deprivation demonstrate a strong relationship between exposure in early childhood and health in young and old ages (Banerjee *et al.* 2007; Bengtsson & Lindström 2003; van den Berg, Lindeboom & Portrait 2006; Myrskylä, Mehta & Chang 2013; Helgertz & Persson 2014; Quaranta 2014; see also for review Currie & Almond 2011). Studies connecting variations in disease environment in early life to later-life income or socio-economic status, albeit being scarcer, tend to capture the presence of the effects in the individuals' working ages (Almond 2006; Case & Paxson 2009; Barreca 2010; Bengtsson & Broström 2009; Kelly 2011; see also for review Bleakley 2010). The main concern while concluding from this strand of studies arises from the disease and nutrition insults being local and abrupt, and therefore different from the pervasive long-term changes in environment surrounding birth and childhood.

Our knowledge about the long run income and health effects of public health programs is extremely limited. The scope of the interventions applied in the analysis ranges between those improving nutrition, disease environment and providing general care in early childhood. Feyrer, Politi & Weil (2013) find that cohorts treated by the sharp introduction of iodized salt in the US 1920s have beneficial effects in diseases related to cognitive development. Bleakley (2007) establishes the strong effects of infant health, improved due to the hookworm eradication campaign in the US South, on literacy and earnings. Well-child visit programs initiated in different Scandinavian countries in the 1930s tend to generate positive lasting impacts on health, and less consistently, on incomes of the treated cohorts (Bhalotra, Karlsson & Nilsson 2015; Hoynes *et al.* 2016; Hjort, Solvsten & Wust 2014). Bhalotra & Venkataramani (2013) utilize the sharp arrival of the sulfa therapies in 1937 to examine impacts of pneumonia in infancy on education and income outcomes in working ages and find large impacts on each. Glied & Neidell (2010) find moderate income effects of treatment by water fluoridation in childhood initiated in the 1940s in the US. Some of the

aforementioned studies appear to demonstrate larger benefits of the reforms for the children with poorer backgrounds pointing to the distributional effects of the public health provision at a society level. Defined more broadly than disease and nutrition shocks, the policies used so far in the empirical literature have yet targeted specific diseases and as a result enabled to examine one of the many channels between the influence of child health on later health and income. The investigation of the long-term beneficial effects of the public health reforms, which were initiated along the formation of the welfare state, represents the missing gap in this literature.

Based on the design-based identification strategy for causal inference, our aim is to explore the effects of health in infancy on health and pension incomes in old ages. We exploit the beneficial changes in health endowments among infants induced by the universal public health reform that was implemented in the rural parishes throughout Sweden for the cohorts born between the 1890 and 1917. This reform became one of the first elements of early welfare state and intended to provide access to public health care to total population, and prevent the transmission of infectious diseases at the community level. At that period, around 80 percent of children were born in rural areas, and the reform therefore through targeting all population strata and different parts of the country became nationwide. For the cohorts treated by this reform, we discern no overlapping public health interventions, such as water system improvements or vaccinations. The implementation of the reform occurred exogenously to the local characteristics of the population and socio-economic conditions. The detailed register data allows us to follow all individuals born in rural parishes, with different timing of the implementation of the reform throughout Sweden, between the ages 78 and 95. Both pension income and mortality outcomes are observed, and we therefore capture the benefits of better health in childhood in terms of quantity and quality of life. The rich supplementary dataset on the reform implementation enables to disentangle the effects by initial socio-economic characteristics of the places of birth and by the amounts of public investments into the establishment and maintenance of the health districts.

Our study attempts to make important contributions to the existing literature. To date, the literature studying the link between broad early childhood conditions and later life outcomes is predominantly correlational. The causal studies of this kind employ either specific and abrupt negative conditions or universal reforms implemented in the mid-twentieth century the earliest. Our paper analyzes the impact of the improved access to health care provided to general public along the formation of the welfare state, the policies which could find resemblance in any developed countries in the beginning of the twentieth century or in many developing countries more recently. Furthermore, the existing literature is limited to the adult health as a main outcome of childhood circumstances. The findings

from several studies, where socio-economic outcomes are touched upon, are inconclusive or mixed. Yet, the focus on both disease and income phenomenon as those determined by childhood health is probably the only way to provide the causal link from health to income and income inequalities. This paper is looking both at mortality and income thereby filling this gap. Due to the recent roll-out of the reforms employed, the follow-up period in the contemporary studies is limited to young and middle adulthood. Meanwhile, the focus on the old-age disease and well-being, like this paper is approaching, could shed light on the causes of chronic diseases and conditions that nowadays constitute the main burden of the ageing societies. Policies targeting old-age population through contemporaneous treatments require large costs, and they could be substantially relieved with the accent on favorable early-life conditions induced by the public programs and leading to lasting effects over the life course. In the case of the reform studied in this paper, the provision of the access to health care costed not more than 0.1 percent of national income at the time of the reform implementation. Finally, to date, the current literature has provided two sets of environmental childhood conditions, such as nutrition and disease, which are capable of generating lasting effects. We contribute to this literature by studying the reform that focused on the prevention from the infectious diseases with most discernable treatment effects during infancy.

Context and 1890 Health district reform

In Sweden economic development had accelerated by the last quarter of the nineteenth century. According to the recent estimates, in per capita terms real GDP grew at an annual rate of 1 per cent prior to 1890, and throughout the twentieth century this rate was constant at 2 per cent (Schön & Krantz 2012). Last subsistence crisis occurred in Sweden in the late 1860s (Sandberg & Steckel 1997). Beginning from 1880, the share of employed in industry increased from 15 to 35 per cent, although by 1920s approximately a half of the population still worked in agriculture (Statistiska Centralbyrån 1969). In the following decades, Swedish manufacturing sector became dominant as well as services witnessed their expansion (Schön 2010). The same development is observed in real wages of workers, which began to increase gradually starting from 1880, and accelerated at the middle of the twentieth century (Jörberg 1972). Distinctly for Sweden, the majority of the industrial workers were employed and lived in rural industrial locations. Urbanization followed a similar circuit. Urbanization rate was slow prior to 1890 and afterwards amounted to 2 per cent per year (Statistiska Centralbyrån 1999). Despite the tempo, by the first half of the twentieth century, the majority of the Swedish population was still rural. According to international standards, Swedish cities were small, with the population resided in the biggest cities, such as Stockholm and Gothenburg, not more than 500,000 that accounted for the less than one tenth of total. Taken less

conservatively, the share of population that resided in the countryside (*land*), and therefore outside cities (*stad*) or semi-urban locations (*köping*), declined only from 80 to 70 per cent between 1890 and 1920, and to less than a half of total afterwards (Statistiska Centralbyrån 1999).

The improvements of population health in Sweden exhibited similar pattern. There is no trend change in life expectancy at birth until the middle of the nineteenth century, whereas afterwards it increased almost linearly from 42 to 78 years (Bengtsson 2006). Human stature also demonstrates progressive advances, although according to some estimates it delayed its growth up until 1880 (Sandberg & Steckel 1997). Swedish population enlarged rapidly, from approximately 4.2 to 5.9 mln between 1880 and 1920 and to its double 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 diarrhea (Preston *et al.* 1972). Between 1880 and 1920, infant mortality rate declined dramatically, from 129 to 72 per 1000, and death rates in 1 – 15 ages fell even more rapidly, from 13 to 4 per 1,000 children (Statistiska Centralbyrån 1999). Among the main demographic processes contributed to the slowdown in population growth in this period stand out several ones, such as few waves of Spanish flu, which killed slightly less than one per cent of the population and scarred many more, gradually falling birth rates, and the mass emigration to the United States, which subtracted approximately a million of residents (Hofsten & Lundström 1976). In the later decades, the improvements in life expectancy have been attributed to the decrease in death rates among groups in working and old ages. With regard to the causes of death, in this period the most dramatic reductions were witnessed by mortality rates from pneumonia, degenerative diseases of organs and tissues, vascular and heart diseases (Preston *et al.* 1972; Statistiska Centralbyrån 2010).

The Swedish authorities recognized the need of the public health provision already in the nineteenth century. After the establishment of the national vital statistics in 1749, the medical board produced several reports about the mortality in the country and underlined the necessity to prevent deaths from smallpox and other infectious diseases (Johannisson 2006). Under the need to save labour force, in the beginning of the nineteenth century the government set up the foundation for the 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 such 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 a long time, under the fear of epidemics occasionally hitting the neighboring countries, the authorities imposed

quarantine regulations, inspections of the cargoes as well as mobilized medical practitioners (Bourdelais 2002). In the mid-nineteenth century, such initiatives concerned only major Swedish towns which by that time became equipped with hospitals and medical personnel. However, the march of epidemics appeared to fail 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). By disentangling the mode of transmission of the infectious diseases and its causal agent, the international bacteriological discoveries in the 1870 – 1880s made the public efforts targeted and provided exact tools to combat the disease. Consequently, the government authorities had to admit the necessity for the radical programme of the public health care provision throughout the whole country.

Starting from 1890 until 1920s, all communities of Sweden gradually received access to public health care in the form of local health districts. The institute of a provincial doctor district (*provinsialläkardistrikt*), which is organized around an assigned doctor, midwives and a hospital, dates back to 1773. Until the mid-nineteenth century, the number of centrally introduced health districts amounted to 2 per 100,000 inhabitants and disproportionally covered the more urbanized locations (Medicinalstyrelsen 1907). In 1840, industrial elite was granted with the right to organize a local medical district serving their residencies. By establishing a community council representative for all taxpayers, a few decades later the local government reform extended this right to all parishes (Lindblom 1967). The local health administration instructions followed shortly, which prescribed each parish or group of parishes to set up a public committee, including a magistrate and a doctor, to deal with public health matters, in particular in controlling the spread of infectious diseases. The location-initiated creation of health districts accelerated accordingly beginning from the 1880s. However, without any government subsidies, between 1840 and the late 1880s, the process of expansion of provincial doctor districts was sluggish and favorable towards wealthy and industrialized locations (Medicinalstyrelsen 1907). Driven both by international achievements in the municipal government and medicine and by rapid industrial and population growth in the countryside, in 1890 Swedish state authorities announced a reform aiming at creating medical districts in all parts of the country, giving equally this opportunity to the economically disadvantaged areas. According to the reform, each group of parishes, with 8,000 – 12,000 inhabitants, applying for an organization of a public health district could be subsidized with 1,500 SEK from the government and had to accumulate 2,500 SEK from the local sources. Additionally, the state began to stimulate the graduation of the young medical professionals and attract them to the rural parishes, primarily by guaranteeing career promotion and public pension. The reform

therefore was designed centrally to bring access to public health care with more local resources devoted to it to rural population.

Based on the multiple sources, we purposely built the dataset to assess the implementation of the 1890 health district reform (see Appendix A). Rather uniquely, it contains not only information on the administrative division of the parishes into medical districts, abundant parish-level data, but also information on different types of investment, including health care and infrastructure, both prior and after the implementation. Based on several sources, we gathered the dates of the establishment of provincial doctor districts and assured that they coincided with the increases in investment into public health. Due to the primarily reorganization character of the reform in the concluding decades and a few more reasons outlined below, we stop to follow the establishment of medical districts in 1917. In the dataset, we collected information on health districts that were established under modern instructions about disease prevention, since 1881. In order to avoid the overlap with other public health interventions, such as sewerage and water supply improvements, we exclude all urbanized (*stad*) and semi-urbanized (*köping*) parishes from the analysis and therefore consider the adoption of the reform only in rural parishes (*land*). Figure 1 demonstrates the progress of the health care reform across Sweden that discerns the considerable variation of the timing across different regions. According to our data, separate health district doctors were introduced gradually throughout rural parts of Sweden, 124 districts in addition to 163 districts existed prior to the expansion, 17 of them created in 1881 – 1889 and 107 districts, subjected to the subsidies, in 1890 – 1917. Our calculations show that the objectives of the reform were realized (see Appendix B). The average health district costed the public approximately 3,000 SEK in real terms, which were spent on the opening of a hospital or a medical station, employment of one doctor and four-five midwives. Access to health care at a community level was given to 12,000 inhabitants that resided in 6 – 7 parishes on average. At the margin, the 1890 reform doubled the access of the population to public health care. Induced by the reform, public health spending increased by more than 680 SEK per 1000 inhabitants in real terms, whereas in the preceding decade the average public health spending amounted to 300 SEK per 1000 for the whole country and was lower in rural parts. With regard to personnel, parishes additionally employed 6 midwives and 2 doctors per 10,000 inhabitants, and had in their disposition 5 midwives and 1 doctor per 10,000 a decade prior to the reform.

[Figure 1]

Taking advantage of the plentiful parish-level data describing socio-economic, infrastructure, health and demographic domains of the parishes, we can analyze which factors were associated with fostered implementation of the reform. Our concern here is that the forerunning parishes had other

favourable characteristics, such as wealth or health, which could foster the beneficial long-run effects instead of the reform. Our analysis does not support this (Appendix C). Instead, with regard to wealth, the parishes that established the reform earlier were likely to be on average poorer compared to later adopting parishes. More specifically, accelerated implementation of the reform was associated with lower real total investment and investment into health care prior to the reform, larger population and its growth, and a smaller fraction of active population in labour force. Regarding the population health, our analysis shows that public health reform was also introduced independently of the disease conditions in the parishes, measured with the share of infants in total population, share of disabled or the mortality rate under age 15. These results are fully consistent with our earlier work (Lazuka, Quaranta & Bengtsson 2016). Our previous study examined the implementation and the contemporaneous effects of the same set of public initiatives in the rural parishes of southern Sweden in 1870 – 1940. The careful investigation of the public health initiatives showed that improved access to health care was driven by the nationwide fear of the epidemics and the recognition of the lack in local competence in its prevention, and this does not correlate with local prosperity or actual infectious conditions. Similarly, no other health measures, such as water supply installations or food inspections, overlapped with the public health care measures.

The initiatives undertaken in the parishes due to the 1890 reform intended to prevent the spread of infectious diseases. Due to the bacteriological discoveries in the 1870 – 1880s, the intervention was able to target disease in localities, even though the antibiotic therapy and vaccination were adapted only in the late 1930s. In our previous papers (Lazuka *et al* 2016; Lazuka 2016) we discuss the tools available to the public in the pre-drug decades and describe those which came in use due to the health care reform. The newly assigned provincial doctors became responsible for the monitoring and isolation of the infected persons from the rest of the parish population. The cottage hospitals or health stations in the parishes were built for this prime reason, whereas chronic patients for a long time were delivered for the in-patient care to the neighboring cities. Under the control of medical doctors, the organization of health districts encouraged the employment of the midwives, more qualified in disinfection and modern knowledge than previously. Prior to the reform and accompanying introduction of the disinfection instructions in childbirth in 1881, commune midwives participated in the registration of death, did not use antiseptics during birth deliveries and therefore by themselves could be carriers of disease (Pettersson-Lidbom 2014). The public health reform therefore not only increased the overall availability of the midwives, but also shifted the share of the employed midwives towards the competent ones. Additionally, the employed medical practitioners brought in more supportive treatment to the population, such as surveillance and relief

of the disease. Importantly for our identification strategy, beneficial treatment by the reform does not correlate with socio-economic characteristics of the parishes, neither, as our earlier work shows, it did with regard to socio-economic background of a newborn. It is highly expected as health care was provided to the public through redistribution for no or negligible costs for the recipients (Curtis 2011). Our previous studies (Lazuka *et al* 2016; Lazuka 2016) find that these public health initiatives led to more than 50 per cent decreases in under-5 mortality rates, and entirely in pediatric infectious diseases.

Data

Given the roll-out of the health district reform, we require an accurate data on the division of the parishes into health districts and its changes. We collected this information 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). As the passage of the establishment acts may be misleading about the timing of movements in actual policy, we verify these divisions with several sources, such as the provincial doctor reports attained from the National Archive in Sweden (Årberättelse 1893 – 1936), statistical yearbooks on health care (Statistiska Centralbyrån BISOS K, SOS 1880 – 1917, Statistiska Centralbyrån 1880 – 1917a) and public health investment (Statistiska Centralbyrån BISOS U 1874 – 1917). These sources provided information on the number of the medical personnel employed, such as doctors and midwives, and public spending, both in health care and in education, infrastructure and welfare. In the latter case, we gathered investment series for each parish before and after the establishment year, and aggregated them to a health district level, which allowed us to carefully determine the intervention dates. In the analysis, to avoid purely administrative changes, we therefore rely on the implementation dates accommodated with the public health investment series. Our sample includes rural (*land*) parishes only. We also exclude from the analysis the rural parishes that developed through the period into market cities (*köping* or *stad*), parishes that experienced several health district re-allocations or where the adoption dates were uncertain (220 out of 2353 parishes). Although the creation of the medical districts continued from 1890 up until 1940s, we stop to follow the establishment of medical districts in 1917 due to several reasons. Primarily, the organization of districts after 1920 became largely administrative, when several medical units established a few decades prior were merged into a larger unit with no corresponding employment of medical practitioners. The public investment series are also unavailable for the period after 1917. Additionally, the availability of the medical personnel, such as midwives, began to stagnate as the institutional childbirth deliveries grew gradually in rural

locations. The surges of Spanish flu, which came to Sweden in 1918 – 1919, not only affected the subsequent cohorts, but also encouraged the revision of the public control of infectious diseases.

Individual-level data comes from Swedish administrative registers. We utilize the Swedish Interdisciplinary Panel (SIP), which combines the multiple administrative registers for all individuals residing in Sweden from 1968 until 2012 tracked through unique personal identifiers. SIP contains county and parish of birth of the individual which have been easily merged to our data on health districts (see Appendix D). In the period under analysis, parish of birth is accurate and corresponds to the place of mother's residence (Riksskatteverket 1989). Based on the data, to assure the consistency in ages for the cohorts born between 1890 and 1917, we focus on the outcomes at the ages 78 – 95 (see Appendix E). The population and death registers provide records on the survival and the date of death of the individual. In, addition, the cause-of-death register gives the primary cause of death which we further classify into several groups. Guided by the diagnostic groups suggested by the early-life epidemiological literature (e.g. Gluckman *et al.* 2008), we distinguish deaths from infectious diseases, circulatory and heart diseases, diabetes, cancer, degenerative diseases of tissues and organs, and violent and unspecified causes (see Appendix F). During the period under analysis, the registration of the causes of death was mentored by the three revisions in the international classification of death (ICD – 8, ICD – 9, and ICD – 10) among which the long-term follow-ups are reliable and valid (Janssen & Kunst 2004; Ludvigsson *et al.* 2011).

In regard to the socio-economic outcomes, we obtain the data from the income and taxation register. In the observation period, individuals obeyed the pension scheme introduced in 1960 that could be claimed from the age 67. The scheme provided the flat basic rate (*folkpension*) with a supplementary benefit (*allmän tillägspension*) determined as a percentage of the average 15 highest paid years (Kruse 2010). Although the ceilings in the payments existed, the pensions were thus higher with either longer working period or steeper earnings profiles. The pension system also contained a widow pension (*änkepension*), which could be paid out either until the death of widow or remarriage, and similar benefits could be accrued for men. As this pension was rather substantial, amounted to 90 per cent of a base rate, and around 40 per cent of person-years in our sample are under widowhood, we could check the robustness of our results to their exclusion from analysis. To avoid the changes in the registration of different types of income occurred through the period in question, we use the total earned income (*sammanräknad förvärvsinkomst*), which for our age groups includes pension (70 per cent) and property income (23 per cent), and combine it with capital income (*inkomst av capital*, 7 per cent). We rely on the real yearly income as an average between the age 78 and the year before death or the age 95 as our preferred measure of permanent

income. As an alternative measure, we construct a mean residual income, which is a mean residual of the individual's earnings from year of birth and its squared term, and a set of register-year dummies.

To fill the gap in the individual administrative data for the cohorts 1890 – 1917 in terms of the individual's background characteristics, we augment abundant parish-level information from other national records. Swedish decennial censuses 1880 – 1910 emerge as the main source. The counts contain data on the occupation names, their HISCO and status codes, which we further standardized into a historical international social class scheme allowing us to get the measure of socio-economic status consistent between the cohorts (HISCLASS, van Leeuwen & Maas 2011). Among the socio-economic variables at a parish level, we constructed several ones, such as the share of elite and industrial workers and a share of agricultural workers in total male population ages 15 – 55, the share in labour force in total in the ages 15 – 55, the mean family size, and the share married in total population ages 15 – 55. Based on the titles of all local occupations, we were also able to obtain the variable indicating whether the parish had a railway or water supply installations. We supplement these variables with other demographic characteristics of the parishes, such as the size of population, the mean age of the female, and the share of females in total, and with health characteristics, such as the share of infants or children under age 15, the share of population older age 55 in total population and the share of (non)disabled persons in total population. The latter group we complement with information on deaths under the age 15 gathered from the death register, which is possible due to the availability of the year and parish of birth overlapping in the sources. In addition to control for the time-varying factors, all constructed variables for the years 1880 and their changes between 1880 and 1890 can serve as the pre-treatment control variables.

Method

The establishment of the public health districts can be considered as improvements in individual's early-life environmental conditions. Given the gradual implementation of the reform throughout Sweden in 1890-1917, we apply a difference-in-difference approach in the following form:

$$y_{ipb} = \alpha + \beta post_b \times healthdistrict_p + \eta_p + \lambda_b + \varepsilon_{ipb}, \ (1)$$

where y_{ipb} denotes health or income outcomes for individual i, born in parish p, in the birth year b, and $post_b \times healthdistrict_p$ is an indicator for the health district established in parish p in a year of birth b and remained in place in a post-treatment period. The state guaranteed the placement of a provincial doctor for at least 5 years and the majority of the districts were kept longer (Medicinalstyrelsen 1907), we therefore consider pre- and post-treatment periods 5 years each. As a

result, individuals included in a sample are 5 years old or less at the reform implementation. The alternative bounds of 3 or 7 years provide qualitatively similar results (available upon request). Given the numerous changes in demographic and economic conditions in the late-nineteenth and early twentieth century, this empirical strategy has several advantages. The smooth period changes, potentially affecting childhood conditions such as a rise in real wages or decline in fertility rates, that are akin to different locations are ruled out by the introduction of the year of birth fixed effects. The parish-specific differences invariant over time, such as the local wealth, climate or institutions, are also controlled for. As the health district was introduced in a group of parishes, it is possible to include group fixed effects instead, although we settle for the parish fixed effects as more demanding. The parishes that implemented the reform between 1890 and 1917 could be distinct from those which remained unchanged. Our baseline sample therefore includes parishes that established a provincial doctor district at some point in time during the period under analysis. We therefore aim to obtain the estimate *ß* by comparing changes in old-age outcomes across cohorts born in parishes that initiated the establishment of the health district to changes across the same cohorts born in parishes that have not initiated the program yet. In all models, to account for the location-level unobserved correlation, we cluster standard errors by parish of birth. The models additionally introduce sex dummies to control for the sex-specific differences in survival and income trajectories.

As our empirical strategy relies on the random nature of the timing of establishment of health district and parallel development in the outcomes across the parishes, we follow several approaches to address our concern about its retention. As the treated parishes were located in different parts of the country, in order to eliminate the treatment effect from any secular trends at a county of birth level, in our first approach we introduce interactions between county of birth dummies and linear trends in year of birth. Second, based on a multisource parish-level dataset described earlier and following Hoynes *et al* (2016), we are able to control for trends in the observable pre-treatment characteristics by including interactions between parish of birth characteristics, such as levels in 1880 and their changes from 1880 to 1890, and cohort dummies. In using a difference-in-differences method, one should especially be concerned that treated and untreated cohorts before the reform implementation exhibited converging outcomes. We first investigate this with an event study design (Figure 2), and the results indicate no diverging health and income trajectories in the pre-reform years. In the models, in order to explicitly control for the plausibly diverging patterns in the outcomes in the pre-treatment periods across different parishes of birth, we introduce parish-specific linear (or quadratic) time trends. The reform did not affect all rural parishes in Sweden, the majority of them enjoyed the health system existed prior to 1890. As our final approach, following Hjort *et al*

(2014), we can match each eventually treated parish with another one that remained untreated, based on the pre-1890 parish and health district characteristics.

[Figure 2]

The matching procedure, applied to achieve symmetry in the pre-treatment trends, is described in detail in Appendix G. For this approach, we calculate propensity scores and apply a nearest neighbor matching, in which we allow it to find only one control without replacement and impose a common support restriction. Out of 2133 rural parishes, 492 parishes introduced provincial doctor district between 1890 and 1917. As non-implementing parishes are different in plentiful dimensions from the implementing ones, we match on a variety of pre-treatment parish- and health district characteristics. The motivation for the determinants of health district implementation is a standard model of public health care utilization that involves socio-economic, infrastructure, health, demographic and health system domains (Andersen & Aday 1978; Kifmann 2005; Grossman 2000). The list of parish-level characteristics employed in matching procedure is precisely the same as used among pre-treatment characteristics, and includes their levels in 1880 and differences between 1890 and 1880. Among the health-district characteristics, we include those describing pre-treatment wealth and public health system in the parishes, such as logarithm of real investment in public health and logarithm of real investment in education, infrastructure and welfare. As can be seen, the matching procedure allowed us to arrive at 432 treated and 432 matched parishes and therefore substantially enlarge the estimation sample. Using the constructed sample, we assign the implementation dates to the matched parishes based on their treated counterparts, and compare the individual outcomes applying a similar specification as previously:

$$y_{ipb} = \alpha + \beta \, \hat{post_b} \times healthdistrict_p + \delta \, \hat{post_b} + \eta_p + \lambda_b + \varepsilon_{ipb}, \ (2)$$

where additionally included $\hat{post_b}$ denotes post-implementation years for both treated and matched parishes. Taken the strictness of the method, the early-life processes should be therefore extremely strong to reassure the presence of the effects. This is not to say that untreated parishes did not experience enhancement of medical personnel or public health investment between 1890 and 1917. However, these changes were likely to be universal and continuous, and therefore captured by the cohort fixed effects.

To examine the effects of the health district reform on socio-economic inequality, we estimate the heterogeneous effects by several subgroups. The previous research suggested that the investigation of factors of inequalities can be made in a triple difference framework (Bitler, Gelbach & Hoynes 2014). We apply this by introducing additional terms into the eq.1, such as $post_b \times healthdistrict_p \times$

subgroup$_s$, which is an indicator for the health district established in parish *p* in a year of birth *b* and subgroup of parishes *s,* subgroup fixed effects, and the interaction between subgroups (at the parish of birth) and year of birth. The focus is on the estimate for the triple interaction terms, which should provide indication on whether individuals from particular socio-economic groups diverged in their outcomes from the rest of the treated population. As discussed previously, our socio-economic measures are time-varying and defined at a parish-of-birth level. Additionally, health-district data on public investment and the number of employed midwives and doctors enables to relate the early-life effects to the amount of the public resources spent by parishes on the establishment of the district.

The specificities of our estimation sample preclude the use of particular estimators. As we analyze old-age mortality between 78 and 95 ages, we apply the procedures capturing non-linearity of the mortality rates. The simplest way to address the issue is to leave the baseline hazard unspecified by applying Cox proportional hazard model (Cox 1972). Across all specifications, tests based on Schoenfeld residuals indicate no violation in the proportionality of the hazards. The Gompertz regression, where hazard rates can be specified as monotonically increasing over time, has been shown to describe old-age mortality rather well (Thatcher, Kannisto & Vaupel 1998). All duration models are adjusted for left-truncation at age 78. To assure similarity with previous research, we also estimate linear probability models on the indicator whether or not individual is deceased from the beginning of observation at age 78 with different follow-ups, such as the age 78 plus 5 years or 10 years, as eventually these proportions should converge with the completion of lives. For cause-specific mortality, Cox proportional hazards models are also applied. We also utilize logarithms of the survival time as an outcome. Regarding the permanent income, similar concerns are addressed. To provide a comparison between the cohorts with different earnings profiles and impede their disproportionate influence on the results, logarithm of income is used as an outcome in linear least squares models. As the individuals are progressively dying over time, we calculate estimates by using the mean yearly income for different age ranges, such as the full follow-up period the ages 78 and 95, and the shorter ones the age 78 plus 5 years and the age 78 plus 10 years. As an alternative, we use a mean residual income, which reduces variation from measurement errors and therefore artificial income changes compared to other permanent income measures (Lindahl *et al.* 2015).

Table 1 presents descriptive statistics for our estimation samples. Conditional on survival to the age 78, individuals in our samples die in ages 86 – 87 at the mean. This characteristic of our sample does not deviate from the actual life expectancy at the age 78 for the same cohorts in Sweden in total, which is equal to 8.5 years (Human Mortality Database 2016). More than 90 percent of

individuals die within the age range 78 – 95, with the largest fraction due to cardiovascular diseases at 57 percent, followed by cancer at 16 percent, degenerative diseases at 13 percent, and infectious diseases at 9 percent. The share of males in a sample is around 45 percent that is expected due to their lower survival to the old age compared to females. The fraction of individuals treated by the reform is slightly more than a half. By design we follow cohorts born 5 years prior and after the establishment of health districts and those treated might be more likely to survive to the beginning of the follow-up. The logarithm of the permanent income in our estimation samples is 7.7 units that align well with the same cohort measure for the total population (calculated from SIP 2016). With regard to both health and income outcomes and treatment variables, baseline (implemented parishes of birth) and constructed (implemented and matched parishes of birth) samples are similar.

[Table 1]

Results

Table 2 presents results for the long-term effects on mortality from the Cox proportional hazards model in a fashion of specifications discussed previously. The estimates are presented in the exponentiated form and therefore can be interpreted as a percentage change in the mortality risk due to the introduction of the health district in a parish. The results show that the treated individuals, those born within 5 years after the establishment of the health district, are significantly less likely to die in the ages between 78 and 95, more particularly, they exhibit the reduction by around 6 percent in mortality risk. Our estimates keep their size and robustness in different specifications, such as those including the year of birth linear trends across counties of birth, parishes of birth and pre-treatment characteristics varying across cohorts. In the matched sample, the size of the coefficient reduces to 4.2 percent decrease in mortality risk, albeit attains its statistical significance. We additionally estimate the reform effects on mortality by using different estimators (see Appendix H). They all support the presence of the beneficial effects of the reform on mortality. The estimates from the parametric proportional hazard model, the Gompertz model, similarly suggest an effect on the risk of dying in a range of 4 and 6 percent. Additional models indicate that the effects are present in regard to both the incidence of death and the duration until death. Followed between the ages 78 and 82, the share of the deceased among the treated individuals are around 2 percentage points lower compared to the rest, which at the mean dependent variable of around 27 percent implies 7 percent reduction. As expected, this effect on the probability of death significantly reduces with the competition of lives by the age 95. With regard to overall duration, the treated groups of

individuals again experience around 7 per cent longer life until death, which in absolute terms with the mean duration time at 9.4 years is equivalent to 0.7 more years alive.

[Table 2]

The estimates for the income are presented in Table 3. Across all models we use the logarithm of income and the results are thus interpreted as the percentage changes. The estimates suggest the positive effects of the health care reform on the individuals' permanent income. With the full follow-up period at the ages 78 and 95, the treated individuals have a 2 percent higher permanent income compared to their counterparts. The estimates are stable within a range of 1.7 and 2.5 percent across different specifications, including those with parish-specific year of birth trends or exact matching. As individuals are progressively dying at the beginning of observation with age, we check whether shorter follow-up periods affect our results. The subsequent models suggest the effects of similar or slightly larger magnitude (see Appendix I). Similar to the mean income, the use of a mean residual income supports the previous results. The individuals born within 5 years after the launch of the health care reform in a parish exhibit around 2 percent increase in their life-time incomes. Additionally, we exclude person-years under widowhood from the calculation of the mean income, and the results slightly grow in magnitude to 2.5 – 3.0 per cent. We retain from presenting the income effects in absolute terms, as earnings across cohorts under analysis vary considerably.

[Table 3]

The estimates presented above represent the intention-to-treat estimates, and we do not directly observe the probability of the individual to be treated by the reform. In our models, all individuals born in the parishes, where a provincial doctor district was opened in the post-treatment period, are considered to be treated, and the effects are therefore larger if any smaller proportion of them was. In our earlier works (Lazuka et al 2016; Lazuka 2016), we studied the same intervention in a similar rural setting in southern Sweden which provided a detailed records on the participation rates. With more qualified midwives employed at the parishes in 1890 – 1920, the individual-level probability of being treated by a more competent midwife compared to that by a traditional midwife amounted to 20 percent. If to take the total population prior to 1880, including rural and urban areas, the proportion of the licensed midwives amounted to not more than 50 percent (Statistiska Centralbyrån BISOS K 1889-1920). With regard to other preventive initiatives undertaken after the reform, for instance careful monitoring and forced isolation of the infected persons to the hospitals by the provincial doctors, equivalent likelihoods are more difficult to measure. Our previous analysis shows that disease environment, mainly from airborne infectious diseases, improved significantly due to these initiatives. As the effects for the majority of the population occur at a group level due to

the decrease in infectiousness and not due to the individual placement into the isolation hospital, the initial disease panorama, such as the prevalence of airborne or foodborne diseases, weighed the treatment probability. We demonstrated that only 46 percent of infants treated at the location level reacted to the isolation initiatives. Therefore, to transform our intention-to-treat estimates for mortality and income into the treatment effects on the treated, the former should be divided by the proportions in a range of 0.2 to 0.5.

In order to disentangle the biological mechanisms that suspend the development of chronic diseases, we look at the estimates for cause-specific mortality in Table 4. We perform the analysis for the sample of ever implementing parishes and the matched sample. The long-term effects on mortality risk from the health care reform are found predominantly in deaths from cardiovascular diseases, mainly acute myocardial infarction and chronic ischemic heart diseases, which contributed the most overwhelmingly to the general mortality, with 57 percent of all deaths. Dependent on the specification, the individuals treated by the reform have between 5 and 6 percent lower chances of dying from circulatory and heart diseases. In both baseline and matched samples, the decrease in infectious disease mortality is also substantial due to the reform, between 4 and 10 percent, albeit not statistically significant. The results suggest that the reform had a substantial impact on cancer mortality risk, decrease at around 8 – 10 percent, although it is statistically significant only in the matched sample. The estimates in other diagnostic groups, such as diabetes, degenerative diseases of organs and tissues and accidental or ill-defined causes, are not statistically significant.

[Table 4]

Table 5 distinguishes the health and income effects by subgroups based on their local health-care characteristics. As the introduction of the health district in the parishes implied the necessary implementation of the set of initiatives, described earlier, the early-life effects should inevitably emerge at this extensive margin. Improving the intensive domain of the health district, through more midwives employed or larger hospital facilities built, could still produce additional benefits. The estimates for the heterogeneous effects, where the implementing parishes are divided into two equal groups by the public health investment and additional midwives employed per parish population, are supportive of this. The effects in mortality risks are present in groups with low and high investment, and are larger for the latter, at 5 and 7 percent correspondingly. This pattern is less evident for the income effects, with lower responses to more public health spending and higher responses to higher availability of midwives, where probably both direct and indirect channels from early-life treatment operated. The long-term effects are also related to the disease burden in the parishes, measured with the share of infants in parish population and mortality rate under age 15. In

both cases, the health effects are observed for all parishes, but are larger at higher baseline levels of disease. Again, the divergence by these subgroups does not emerge for the permanent income effects.

[Table 5]

To address the role of treatment in inequality development, we present the effects of the introduction of health district by subgroups with different socio-economic characteristics at birth in Table 6. As discussed, we consider several measures of socio-economic status at a parish of birth obtained from the decennial censuses. The estimates for both mortality and log income suggest that the treated individuals with all socio-economic backgrounds benefited from the reform, whereas the benefits are larger for the more disadvantaged groups. In case of mortality, the affected individuals with wealthier and poorer socio-economic origins obtain 3 and 10 percent decreases in all-cause mortality risks accordingly. The availability of a railway can serve as a measure of parish of birth infrastructure and communications, and here the effects in mortality are to a larger extent concentrated among the poorer parishes. Initial socio-economic conditions were stressed as important mediator of the early-life effects especially for income outcomes (Flores & Kalwij 2014; Currie 2009), and here the pattern towards individuals with more disadvantaged childhood conditions is also obvious. Depending on the measure, the treated individuals with lower socio-economic status in childhood attain 3 – 4 percent and the increases among the initially richer treated individuals are twice lower. Similar difference in income outcomes is observed between the treated individuals born in parishes of birth with or no access to the railway. In using the parish-level characteristics to build the measures of socio-economic status in infancy, we cannot entirely distinguish whether the individuals from poor families benefited more from the introduction of health district or whether those born in parishes with more poor families in total population benefited more. However, the different patterns for socio-economic inequality and infrastructure indicators may point to the former premise.

[Table 6]

Robustness Analyses

So far, the treatment effects for both old age mortality and income appear to have been robust to the inclusion into the models different controls for the pre-treatment secular trends specific for locations of birth. They hold with regard to the additional interactions between year of birth trends and county of birth dummies, parish of birth dummies, and level and trend changes in a set of pre-intervention observable parish of birth characteristics. In the baseline and matched sample, where the pre-

treatment trends are equalized based on these characteristics by the virtue of the matching procedure, the significant effects remain. One concern still could be raised on coinciding social spending occurring alongside the introduction of health districts that could explain the results instead. Additional concern is whether any discontinuous changes in specific locations coincided with the timing of the health care reform thereby changing the composition of the individuals' parents towards wealthier or healthier ones. Finally, it is important to test whether the reform by itself affected the composition of the parents. In what further follows, we analyze the robustness of our results to the potential influence of these factors.

Given the rise in overall social spending starting from the mid-1880s, one might question whether the effects captured emerge entirely due to the health care reform and not due to other overlapping public initiatives. We have already discussed that the local governments put in place no other public health measures, such as improvements in water facilities, vaccinations or food hygiene control, and did it not until the 1930s. From our dataset, we observe that increase in public health investment associated with the introduction of health districts also implied increase in overall social spending. A more careful look suggests that the overwhelming part of the residual local investment was spent on primary schools that do not directly affect infant or health under age 5. Additional resources were also accumulated for poor relief, although they covered only 1.2 percent of children under age 15 with direct support (Statistiska Centralbyrån BISOS U 1890 – 1917). The rise in social expenditures other than public health care is therefore not responsible for our results.

In the period under analysis, it seems possible that some abrupt changes affected parishes differently, thus potentially harming our identification strategy. One potential threat to our estimates arises from the mass emigration of the Swedish residents to the US and other countries, which was rather discontinuous in the period in question. If the emigrating population was selective towards poorer socio-economic classes, thus affecting families of our cohorts, and occurred in a sharp manner across different regions, one might expect the disturbance in our estimates. The emigration to other countries from Sweden was massive between the 1880 and 1910, and around 80 percent of all migrants left to the US (Statistiska Centralbyrån 1967 p.47). Regarding the age composition of the migrants, the persons in ages between 15 and 29 weighted the most. The prime sending counties, where the countryside experienced the mass migration to the US, were Värmland and Halland (Bohlin & Eurenius 2010). Panel A of Table 7 presents the results for the sample where we excluded the individuals with the counties of birth mostly affected by emigration to the US. As can be seen, our results stay unaffected by this check. Furthermore, the location-specific influence of the World War I might be important for our estimates. Even though Sweden was neutral during the war,

the treated parishes could be those mostly affected by its threat, forcing the parents to postpone fertility until the peaceful times, or those agricultural regions that witnessed an increase in the exports of the raw materials to Europe thereby boosting the local wages (Siney 1975; Qvarnström 2014). We therefore run the analysis while omitting from the sample individuals born in Norrbotten and Västerbotten counties, the mostly affected by the war, and present the estimates in panel B. Again, both long-term income and mortality effects attain their statistical significance and sizes analogous to the baseline coefficients. To supplement these analyses, we added to the models the parish of birth characteristics that more carefully describe the age structure of the population and vary across cohorts, in addition to those included previously. As panel C shows, our results were also not sensitive to this check.

[Table 7]

We perform additional robustness analyses with regard to whether the parents responded to the reform as such. It might be possible that families, willing to improve the life chances of their newborns, moved to the areas with access to health care and had some advantageous characteristics which could instead explain our results. The internal migration flows in rural areas in the period under analysis were explained by the structure of the local labour force (Enflo, Lundh, & Prado 2014). Previously we find that the implementation of the health care reform did not correlate with shares of industrial or agricultural workers in total or share of the married in total. To account for residential selection, we repeat our analysis by additionally controlling for several time-varying indicators of the local labour markets, such as the share of industrial and agricultural workers in total male population ages 15 – 55, share of skilled workers in total males population ages 15 – 55, share of the married in total population ages 15 – 55, mean age of the female, and share of population with non-Swedish origin in total. Panel D reports the results from the models including these controls, which appear to be unaffected compared to the baseline estimates. We additionally analyze the reform-driven migration responses by using the parish-level information on migrant structure obtained from censuses 1880 – 1910 (see Appendix K, Model 1). The results indicate that there were no effects of the reform on migration flows between the parishes. Finally, it is plausible that the reform generated the fertility responses favouring the delivery of healthy newborns. Again, we can test it with parish-level data from censuses (see Appendix K, Model 2), and our results suggest no presence of these responses.

Conclusions and discussion

To date, our knowledge about the long-run influence of the public health care reforms that were initiated across the developed countries along the formation of the welfare states is extremely scarce. The focus on individuals in their old ages, and therefore on the prolongation of lives, ultimate incomes, and their development across socio-economic groups, disentangles the quantity and quality benefits of any such programs (Weil 2014; Deaton 2010). Taking a causal approach, this paper examined the long term effects of the universal expansion of health districts in rural areas of Sweden between 1890 and 1917 on income and health in the ages 78 – 95. The differences-in-differences approach assured that we compare the outcomes of the individuals born within 5 years after the establishment of health district in the parishes with the same cohorts in other parishes that did not establish a district yet. We find effects for both old-age mortality and incomes. Due to the reform, individuals attain decreases in all-cause mortality risk at around 5 – 7 percent that is equivalent to 0.7 additional years spent alive. The positive effects on the permanent income of the treated individuals amount to 2 – 3 percent. These results passed all robustness analyses, including those controlling for the secular cohort trends specific to locations and those examining the plausible compositional differences between the treated and untreated cohorts. In order to obtain the treatment effects on the treated based on our estimates, one should divide them by the fraction in a range between one fifth and a half.

To our knowledge, our study is first that find the effects of the health care reform on both individual health and income in old ages. We can briefly align the findings of the policy-based studies focusing on age and cohort groups similar or overlapping with ours. Based on the cash transfer program initiated in the US in 1911-1935, Aizer *et al* (2014) estimates the treatment effects on the individuals accepted to the program that at the ages 80 and older are at around 1.4 additional years. Bhalotra *et al* (2015) studies the long-term health effects of the well-child visit program implemented in 1931 – 1933 Sweden, and demonstrates 7 percent reduction in mortality between the ages 40 - 70 among the cohorts treated in a parish during infancy. Similar individual-level studies for long-term income effects are difficult to find. Albeit derived for the cohorts older than ours, using the well-child visit program in the 1936-1955 Norway, Bütikofer *et al* (2014) finds that the program had the intention-to-treat effects on the earnings in young and late adulthood in a range of 1 – 2 percent. Applying a simulation-based method to the health improvements in the first half of the twentieth century and its dynamic demographic consequences, Ashraf *et al* (2009) find that long-run effects on income per capita stabilize at additional 15 percent after 80 and more years. Broadly speaking, our effects on health and income are therefore within a scope of those demonstrated in the previous studies.

Our findings suggest that particular biological mechanisms can underlie the long-term effects. In the previous discussion, we highlighted that the establishment of a health district led to improvement in prevention from infectious diseases of the affected cohorts. In this paper, years of the establishment of the districts are used as the approximations of the starting dates of their functioning. As a result, individuals could be treated by the reform in pre-, postnatal periods or both. The epidemiological literature emphasized these ages as most critical in lasting responses to the influence of infections (Gluckman *et al* 2008). More specifically, the body produces stress hormones or launches chronic immune responses that mobilize and withdraw nutrients away from body and brain development thereby gradually affecting mental and physical health later in life (Danese & McEwen 2012). Through different channels, either improvements in productivity or human capital accumulation, health responses translate into acquired incomes (Heckman 2007). Complementarily, the human body programmed for a particular environment, once faced with a new environment, can be maladaptive in long run and thus generate various health, behaviour and socio-economic outcomes (Boyce & Ellis 2005). Consistent with our results, indicators of infection and inflammation are related to chronic pulmonary disease, vascular and heart disease and other diseases of aging (Barker 1991; Finch & Crimmins 2004).

In addition to the methodological issues discussed earlier, we conclude with those plausibly leading to the underestimation of our results. The first one relates to selective mortality and its potential effects in our results. It is obvious that we examined the group of individuals in their old ages, between 78 and 95, where selective mortality could cause some cohort differences to change. This methodological issue was specifically addressed by the recent studies for similar cohorts as ours (Zajacova & Burgard 2013; Cutler & Lleras-Muney 2010) and most studies of long-term outcomes (Almond 2006; Bhalotra & Venkataramani 2013). The overall findings suggest that our results might be underestimated. More specifically, the individuals untreated by the health care reform and survived to age 78 may be a more selected group, with better health and earnings, than the treated individuals who survived to that age. In support of this, we clearly observe that the marginal effect of the health care reform on income and health is weakening with age. The medical treatments against certain diseases, such as antibiotics, drugs against cardiovascular, heart diseases and diabetes, emerged as the contemporaneous events, should not affect our results. The second methodological issue relates to the income variable that we utilized. It can be questioned whether a mean income in old ages can approximate for permanent income, that is income in early and middle adulthood. Previously, we discussed that the pension schemes, covering the cohorts under analysis, relied substantially on the economic performance in adulthood. The income in old age can be also viewed a measure of individual's economic well-being (Netuveli *et al.* 2006).

Our results indicate that implementation of the health care reform, through improved child health, led to larger effects for individuals with poorer socio-economic backgrounds. In line with our expectations, given the design of the health care reform, we find that the local preventive initiatives generated the long-run benefits across all subpopulations. If socio-economic differences in health and incomes persist and even accumulate with age (Hayward & Gorman 2004; Currie & Schwandt 2016; Case, Lubotsky & Paxson 2002), one should discern the stronger effects for the less affluent individuals. Our results provide several indications in support of this notion. Primarily, in relative terms, individuals with poor family socio-economic classes enjoy larger effects in both longevity and permanent incomes. Similarly, in absolute terms, more generous public health investments per capita, devoted to the creation and functioning of the health district, generated stronger individuals' responses. Previously, we also discussed that the utilization of the health services occurred in an equal manner across families of different socio-economic characteristics. This body of evidence offers some evidence on the intergenerational transmission of health, where child health is linked to adult and old-age health and well-being.

Our findings provide policy recommendations which could substantially relieve the burden of costs related to the support of the ageing population. Nowadays, chronic disease is responsible for the majority of the deaths in developed countries and globally, among which cardiovascular disease takes the bulk of all deaths (WHO 2014). The treatment of the chronic diseases and recovery and the costs targeting adulthood behavior are extremely costly to the societies. The accent on those early life conditions that have lasting health and income effects over the life course could reduce these contemporary costs. In most conservative terms, the public costs of the reform, studied in this paper, including continuous expenditures on health care maintenance in parishes not affected by the reforms, constitute not more than 0.1 percent of national income annually between 1890 and 1917. The health district reform was designed to provide all parts of Sweden with access to public health care, giving equally this opportunity to the disadvantaged regions, and became one of the first elements of the modern welfare state. In our previous work, we showed that the immediate decreases in infant and child mortality and as a result social returns to a corresponding set of public initiatives, focusing on prevention of the infectious disease from transmission, were large (Lazuka *et al* 2016). This study provides evidence on additional gains in old age in terms of longer lives, lower probability of death from cardiovascular disease and higher permanent incomes for the cohorts affected by the reform in infancy.

References

Acemoglu, D. & Johnson, S., 2007, 'Disease and Development: The Effect of Life Expectancy on Economic Growth' *Journal of Political Economy*, 2007.

Agenor, P.-R. & Neanidis, K.C., 2011, 'The allocation of public expenditure and economic growth', *Manchester School (14636786)* 79(4), 899–931.

Aizer, A., Eli, S., Ferrie, J.P. & Lleras-Muney, A., 2014, 'The long-term impact of cash transfers to poor families', *NBER Working Papers* 20103.

Almond, D., 2006, 'Is the 1918 influenza pandemic over? Long-term effects of in utero influenza exposure in the post-1940 US population', *Journal of Political Economy* 114(4), 672–712.

Andersen, R. & Aday, L.A., 1978, 'Access to Medical Care in the U.S.: Realized and Potential', *Medical Care,* 1978.

Ashraf, Q.H., Lester, A. & Weil, D.N., 2009, 'When does improving health raise GDP?', *NBER Working Papers*(1), 157–204.

Autor, D.H., 2014, 'Skills, education, and the rise of earnings inequality among the "other 99 percent"', *Science* 344(6186).

Banerjee, A., Duflo, E., Postel-Vinay, G. & Watts, T.M., 2007, 'Long Run Health Impacts of Income Shocks: Wine and Phylloxera in 19th Century France', *NBER Working Papers* 2007.

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

Barreca, A., 2010, 'The long-term economic impact of in utero and postnatal exposure to malaria', *The Journal of Human Resources* 45(4), 865–892.

Becker, G.S., Philipson, T.J. & Soares, R.R., 2005, 'The Quantity and Quality of Life and the Evolution of World Inequality', *The American Economic Review*, 2005, p. 277.

Bengtsson, T., 2006, *Perspectives on mortality forecasting. 3, The linear rise of life expectancy: history and prospects / editor Tommy Bengtsson*, Stockholm : Swedish National Social Insurance Board, 2006.

Bengtsson, T. & Broström, G., 2009, 'Do conditions in early life affect old-age mortality directly and indirectly? Evidence from 19th-century rural Sweden', *Social Science & Medicine* 68(9), 1583–1590.

Bengtsson, T. & Lindström, M., 2003, 'Airborne infectious diseases during infancy and mortality in later life in southern Sweden, 1766–1894', *International Journal of Epidemiology* 32(2), 286–294.

Bengtsson, T. & van Poppel, F., 2011, 'Socioeconomic inequalities in death from past to present: An introduction', *Explorations in Economic History* 48(3), 343–356.

Ben-Porath, Y., 1967, 'The Production of Human Capital and the Life Cycle of Earnings', *Journal of Political Economy*, 1967.

Bhalotra, S., Karlsson, M. & Nilsson, T., 2015, 'Infant health and longevity: evidence from a historical trial in Sweden', *IZA Working Papers* 8969.

Bhalotra, S. & Venkataramani, A., 2013, 'Shadows of the captain of the men of death: early life health interventions, human capital investments, and institutions', *IZA Working Papers* 7833.

Bitler, M.P., Gelbach, J.B. & Hoynes, H.W., 2014, 'Can variation in subgroups' average treatment effects explain treatment effects heterogeneity? Evidence from a social experiment', *NBER Working Papers* 2014, 1.

Bleakley, H., 2007, 'Disease and development: evidence from hookworm eradication in the south', *Quarterly Journal of Economics* 122(1), 73–117.

Bleakley, H., 2010, 'Health, Human Capital, and Development', Annual Review of Economics, 2010.

Bloom, D.E. & Canning, D., 'Health and economic growth: Reconciling the micro and macro evidence', *CDDRL Working Papers*, 1–25.

Bloom, D.E., Canning, D. & Graham, B., 2003, 'Longevity and life-cycle savings', The Scandinavian *Journal of Economics*, 2003, pp. 319–338.

Bloom, D.E., Canning, D. & Sevilla, J., 2004, 'The Effect of Health on Economic Growth: A Production Function Approach', *World Development* 32, 1–13.

Bohlin, J. & Eurenius, A.-M., 2010, 'Why they moved — Emigration from the Swedish countryside to the United States, 1881–1910', *Explorations in Economic History* 47, 533–551.

Bourdelais, P., 2002, 'Cholera: a victory for medicine?', in R.S. Schofield, D. Reher, & A. Bideau (eds.), *The decline of mortality in Europe*, pp. 118–130, Clarendon Press, Oxford.

Boyce, W. & Ellis, B., 2005, 'Biological sensitivity to context: I. An evolutionary–developmental theory of the origins and functions of stress reactivity', *Development and Psychopathology* 17, 271–301.

Case, A., Lubotsky, D. & Paxson, C., 2002, 'Economic Status and Health in Childhood: The Origins of the Gradient', The American Economic Review, 2002.

Case, A. & Paxson, C., 2009, 'Early life health and cognitive function in old age', *American Economic Review* 99(2), 104–109.

Case, A. & Paxson, C., 2010, 'Causes and consequences of early-life health', Demography, 2010.

Cox, D. R., 1972, 'Regression Models and Life-Tables', *Journal of the Royal Statistical Society*. Series B (Methodological), 1972.

Cunha, F. & Heckman, J., 2007, 'The technology of skill formation', *The American Economic Review*, 2007, pp. 31–47.

Cunha, F., Heckman, J.J. & Schennach, S.M., 2010, 'Estimating the technology of cognitive and noncognitive skill formation', *Econometrica*, 2010, pp. 883–931.

Currie, J., 2009, 'Healthy, wealthy, and wise: socioeconomic status, poor health in childhood, and human capital development', *Journal of Economic Literature* 47(1), 87-122.

Currie, J. & Almond, D., 'Human capital development before age five', vol. 4, pp. 1315–1486.

Currie, A., Shields, M., & Price, S. 2007, 'The child health/family income gradient: Evidence from England', *Journal of Health Economics,* 26 (2007), 213–232.

Currie, J. & Schwandt, H., 2016, 'Inequality in mortality decreased among the young while increasing for older adults, 1990-2010', *Science* 352(6286), 708-712.

Currie, J. & Stabile, M., 2003, 'Socioeconomic Status and Child Health: Why Is the Relationship Stronger for Older Children?', *The American Economic Review*, 2003.

Curtis, S., 2011, 'The arrival and diffusion of academic medicine in rural Sweden: the case of the Sundsvall region in the late nineteenth century', *An Interdisciplinary Journal for the History of Public Health* 10(1), 7–32.

Cutler, D. & Lleras-Muney, A., 2010, 'The education gradient in old age disability', in D. Wise (ed.), *Research findings in the economics of ageing*, pp. 101–120, The University of Chicago Press, Chicago.

Danese, A. & McEwen, B.S., 2012, 'Adverse childhood experiences, allostasis, allostatic load, and age-related disease', *Physiology & Behavior* 106(1), 29–39.

de la Croix, David & Licandro, O., 1999, 'Life expectancy and endogenous growth', *Economics Letters* 65(2), 255–263.

de la Croix, David, Lindh, T. & Malmberg, B., 2008, 'Swedish Economic Growth and Education since 1800', *The Canadian Journal of Economics* / Revue canadienne d'Economique, 2008.

Deaton, A., 2013, *The great escape: Health, wealth, and the origins of inequality*, Princeton, NJ : Princeton University Press, 2013.

Deaton, A., 2010, 'Income, Aging, Health and Well-being around the World: Evidence from the Gallup World Poll' in, in D. Wise (ed.), *Research findings in the economics of ageing*, pp. 235–263, The University of Chicago Press, Chicago.

Enflo, K., Lundh, C., & Prado, S., 2014. 'The role of migration in regional wage convergence: Evidence from Sweden 1860–1940', *Explorations in Economic History* 52, 93-110.

Feyrer, J., Politi, D. & Weil, D.N., 2013, 'The cognitive effects of micronutrient deficiency: Evidence from salt iodization in the United States', *NBER Working Papers* 2013.

Finch, C.E. & Crimmins, E.M., 2004, 'Inflammatory exposure and historical changes in human life-spans', *Science* 305(5691), 1736–1739.

Fine, J.P. & Gray, R.J., 1999, 'A proportional hazards model for the subdistribution of a competing risk', *Journal of the American Statistical Association*, 1999, pp. 496–509.

Flores, M. & Kalwij, A., 2014, 'The associations between early life circumstances and later life health and employment in Europe', *Empirical Economics* 47(4).

Fogel, R.W., 1994, 'Economic growth, population theory, and physiology: the bearing of long-term processes on the making of economic policy', *The American Economic Review*(3), 369–395.

Fogel, R.W., 1997, 'Secular trends in nutrition and mortality', *Handbook of population and family economics*, pp. 433–481, Amsterdam : Elsevier, 1997.

Galor, O., 2005, 'From stagnation to growth: Unified growth theory', in P. Aghion and S.N. Duralf (eds.), *Handbook of Economic Growth*, 1A, pp. 171–293, Elsevier.

Glied, S. & Neidell, M., 2010, 'The Economic Value of Teeth', The Journal of Human Resources, 2010, p. 468.

Gluckman, P.D., Hanson, M.A., Cooper, C. & Thornburg, K.L., 2008, 'Effect of in utero and early-life conditions on adult health and disease', *New England Journal of Medicine* 359(1), 61–73.

Grossman, M., 1972, 'On the concept of health capital and the demand for health', *Journal of Political Economy* 80(2), 223–255.

Grossman, M., 2000, 'The Human Capital Model', *Handbook of health economics*, pp. 347–408, Amsterdam : Elsevier, cop. 2000.

Haines, M.R. & Ferrie, J.P., 2011, 'Socioeconomic inequalities in death from past to present: A postscript', *Explorations in Economic History* 48(3).

Hayward, M., & Gorman, B. 2004, 'The long arm of childhood: the influence of early-life social conditions on men's mortality', *Demography*, 41(1), 87-107.

Hazan, M., 2009, 'Longevity and Lifetime Labor Supply: Evidence and Implications', *Econometrica*, 2009, p. 1829.

Heckman, J.J., 2007, 'The economics, technology, and neuroscience of human capability formation: The economics, technology, and neuroscience of human capability formation', *Proceedings of the National Academy of Sciences of the United States of America* 104(33), 13250-13255.

Helgertz, J. & Persson, M.R., 2014, 'Early life conditions and long-term sickness absence during adulthood - A longitudinal study of 9000 siblings in Sweden: Early life conditions and long-

term sickness absence during adulthood - A longitudinal study of 9000 siblings in Sweden', *Social Science and Medicine* 119, 224-231.

Hjort, J., Solvsten, M. & Wust, M., 2014, 'Universal investment in infants and long-run health: evidence from Denmark's 1937 home visiting program', *SFI Working Paper* 08.

Hofsten, E. & Lundström, H., 1976, *Swedish population history. Main trends from 1750 to 1970.*

Hoynes, H., Schanzenbach, D.W. & Almond, D., 2016, 'Long-Run Impacts of Childhood Access to the Safety Net', *American Economic Review* 106(4), 903–934.

Human Mortality Database, 2016, *http://www.mortality.org/.*

Janssen F & Kunst AE, 2004, 'ICD coding changes and discontinuities in trends in cause-specific mortality in six European countries, 1950-99', *Bulletin of the World Health Organization* 82(12), 904–B.

Johannisson, K., 2006, 'The people's health: public health policies in Sweden', in D. Porter (ed.), *The history of public health and the modern state*, pp. 165–182, GA, Amsterdam-Atlanta.

Jörberg, L., 1972, *A history of prices in Sweden, 1732-1914*, Lund.

Kalemli-Ozcan, S., Ryder, H.E. & Weil, D.N., 2000, 'Mortality decline, human capital investment, and economic growth', *Journal of Development Economics* 62, 1–23.

Karlsson, M., Nilsson, T. & Pichler, S., 2014, 'The impact of the 1918 Spanish flu epidemic on economic performance in Sweden. An investigation into the consequences of an extraordinary mortality shock', *Journal of Health Economics* 36, 1–19.

Kelly, E., 2011, 'The Scourge of Asian Flu: In utero Exposure to Pandemic Influenza and the Development of a Cohort of British Children', *Journal of Human Resources*(4), 669.

Kifmann, M., 2005, 'Health insurance in a democracy: Why is it public and why are premiums income related?', *Public Choice* 124(3/4), 283–308.

Kruse, A., 'A stable pension system: The eighth wonder', *Population Ageing - a Threat to the Welfare State?: The Case of Sweden (Demographic research monographs)*, pp. 47–64, Dordrecht, Netherlands : Springer Berlin Heidelberg.

Kuznets, S., 1955, 'Economic growth and income inequality', *American Economic Review* 45(1), 1.

Kuznets, S., 1963, 'Quantitative Aspects of the Economic Growth of Nations: VIII. Distribution of Income by Size', Economic Development and Cultural Change, 1963.

Kuh, D., & Ben-Shlomo, Y. 2004, *A Life Course Approach to Chronic Disease Epidemiology*, Oxford Scholarship Online (Public Health and Epidemiology) 2004.

Lazuka, V., 2016, 'The long-term health benefits of receiving treatment from qualified midwives at birth', *Lund University Working Papers in Economic History. Population Economics* 2016, 1–36.

Lazuka, V., Quaranta, L. & Bengtsson, T., 2016, 'Fighting infectious disease: Evidence from Sweden 1870-1940', *Population & Development Review* 42(1), 27–52.

Lindahl, M., Palme, M., Sandgren Massih, S. & Sjogren, A., 2015, 'Long-Term Intergenerational Persistence of Human Capital: An Empirical Analysis of Four Generations', *Journal of Human Resources* 50(1), 1–33.

Lindblom, P., 1967, *Malmöhus läns landsting 1863-1962*, Malmö, 1967.

Lucas, R.E., 2009, 'Ideas and Growth', *Economica*, 2009.

Lucas Jr. , R. E., 1988, 'On the mechanics of economic development: On the mechanics of economic development', *Journal of Monetary Economics* 22(1), 3-42.

Ludvigsson, J. (2010.) External review and validation of the Swedish national inpatient register. *BMC: Public Health* 11(450), 1-16.

Mackenbach, J.P., Stirbu, I., Roskam, A.-J.R., Schaap, M.M., Menvielle, G., Leinsalu, M. & Kunst, A.E., 2008, 'Socioeconomic inequalities in health in 22 European countries', *The New England Journal of Medicine* 358(23), 2468–2481.

McKeown, T., 1976, *The modern rise of population / Thomas McKeown*, London: Arnold, 1976.

Medicinalstyrelsen, 1939, *Rikets indelning i provinsialläkardistrikt före 1/7 1939 och Medicinalstyrelsens yttrande och förslag till stadsläkarsakuniga den maj 1932*, Kungl. Boktryckeriet, P.A.Norstedt & Söner.

Mokyr, J., 2005, 'Long-term economic growth and the history of technology', in P. Aghion and S.N. Duralf (eds.), *Handbook of Economic Growth*, 1B, pp. 1–74, Elsevier.

Myrskylä, M., Mehta, N.K. & Chang, V.W., 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-e90.

Netuveli, G., Wiggins, R.D., Hildon, Z., Montgomery, S.M. & Blane, D., 2006, 'Quality of life at older ages: evidence from the English longitudinal study of aging (wave 1)', *Journal of Epidemiology and Community Health* (1979-), 2006, p. 357.

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

Palloni, A., Milesi, C., White, R.G. & Turner, A., 2009, 'Early childhood health, reproduction of economic inequalities and the persistence of health and mortality differentials', *Social Science and Medicine* 68(9), 1574-1582.

Pettersson-Lidbom, P., 2014, 'Midwifes and maternal mortality: evidence from a midwifery policy experiment in Sweden in the 19th century', *Unpublished manuscript* 2014.

Piketty, T. & Saez, E., 2003, 'Income Inequality in the United States, 1913-1998', *Quarterly Journal of Economics* 118(1), 1–39.

Preston, S.H., 1975, 'The changing relation between mortality and level of economic development', *Population Studies* 29(2), 484–490.

Preston, S.H., Keyfitz, N., Schoen, R. & Nelson, V.E., 1972, 'Causes of death: Life tables for national populations', *Studies in Population* 1972.

Quaranta, L., 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, 266–273.

Qvarnström, S., 2014, 'Sweden', *1914-1918-online: International Encyclopedia of the First World War*, Freie Universität Berlin.

Riksskatteverket, 1989, *Sveriges församlingar genom tiderna*, Stockholm: Riksskatteverket, Skatteförvaltningen : Allmänna förl.

Romer, P.M., 1990, 'Endogenous Technological Change', *Journal of Political Economy*, 1990, pp. S71.

Sachs Jeffrey D., 2002, 'Macroeconomics and health: investing in health for economic development', *Revista Panamericana de Salud Pública*(2), 143.

Sandberg, L. & Steckel, R.H., 1997, 'Was Industralization hazardous to your health? Not in Sweden', in R.H. Steckel and R. Floud (eds.), *Health and welfare during industrialization*, pp. 127–159, NBER Project Report series; Chicago and London; University of Chicago Press.

Schön, L., 2010, *Sweden's road to modernity: An economic history*, SNS förlag, Stockholm.

Schön, L. & Krantz, O., 2012, 'Swedish Historical National Accounts 1560-2010', *Lund Papers in Economic History* 123.

Siney, M., 1975, 'Swedish neutrality and economic warfare in World War I', *Conspects of History* 1(2), 13–28.

Soares, R.R., 2005, 'Mortality Reductions, Educational Attainment, and Fertility Choice', *The American Economic Review,* 2005, p. 580.

Statistiska Centralbyrån, 1874-1917., *BISOS U: Bidrag till Sveriges officiella statistik U. Kommunernas fattigvård och finanser, Statistiska centralbyrån, P.A. Norstedt & Söner, Stockholm*.

Statistiska Centralbyrån, 1889-1920, *BISOS K: Bidrag till Sveriges officiella statistik K. Helso och sjukvården. Statistiska centralbyrån, P.A. Norstedt & Söner, Stockholm*.

Statistiska Centralbyrån, 1911-1920, *SOS: Sveriges officiella statistik. Allmän om Hålso och sjukvård. Statistiska centralbyrån, P.A. Norstedt & Söner, Stockholm*.

Statistiska Centralbyrån, 1969, *Historisk statistik för Sverige. Del 1. Befolkning 1720-1967*.

Statistiska Centralbyrån, 1999, 'Befolkningsutvecklingen under 250 år : historisk statistik för Sverige = Population development in Sweden in a 250-year perspective.' 1999.

Thatcher, R., Kannisto, V. & Vaupel, J.W., 1998, *The force of mortality at ages 80 to 120*, Odense University Press.

van Den Berg, G. J., Lindeboom, M. & Portrait, F., 2006, 'Economic conditions early in life and individual mortality', *American Economic Review* 96(1), 290-302.

van Leeuwen, M. & Maas, I., 2011, *HISCLASS. A historical international socil class scheme.*, Leuven University Press.

Weil, D.N., 2007, 'Accounting for the effect of health on economic growth', *The Quarterly Journal of Economics*, 2007, p. 1265.

Weil, D.N., 2013, 'Health and economic growth', *Handbook of Economic Growth. [Elektronisk resurs]*, 2B, pp. 623–682, 2013.

WHO 2014, *Global status report on noncommunicable diseases 2014*. World Health Organization, Switzerland.

Young, A., 2005, 'The gift of the dying: the tragedy of aids and the welfare of future African generations', *The Quarterly Journal of Economics*, 2005, p. 423.

Zajacova, A. & Burgard, S.A., 2013, 'Healthier, wealthier, and wiser: a demonstration of compositional changes in aging cohorts due to selective mortality', *Population Research and Policy Review* 2013, 1-14.
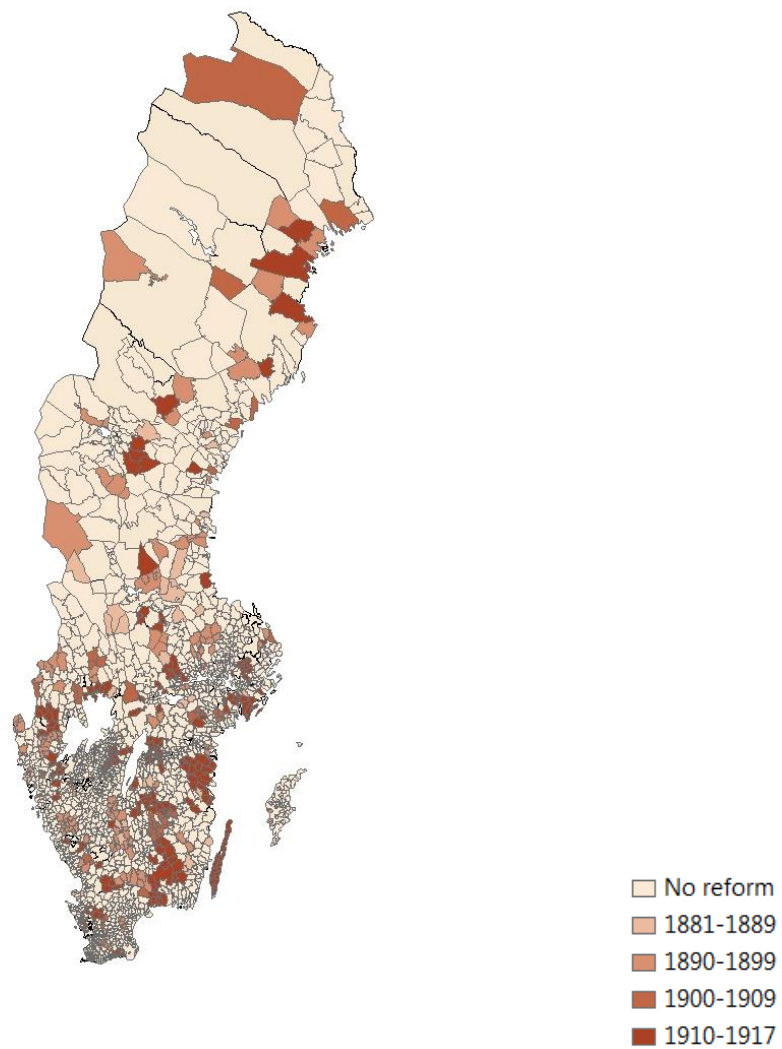
Figure 1 – Implementation of the 1890 Health district reform in Sweden

Source: Historical GIS maps from the Swedish National Archives merged with data on reform dates (see Appendix A)
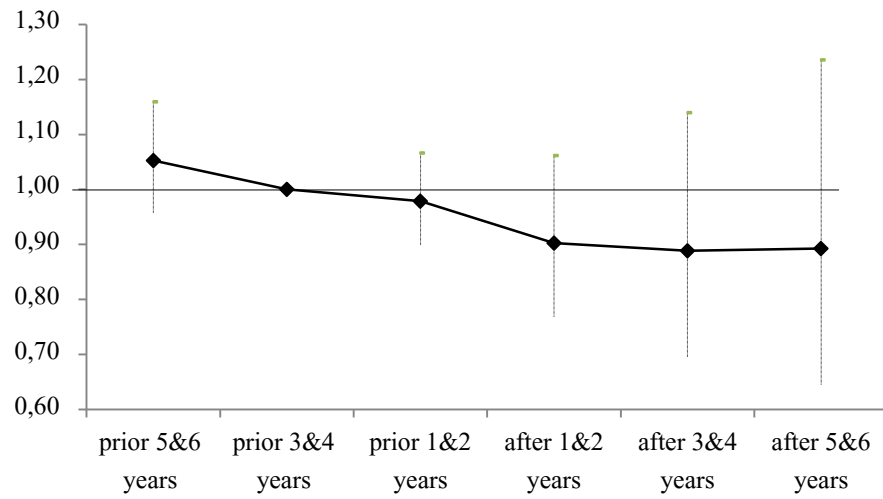
(a) Mortality risk
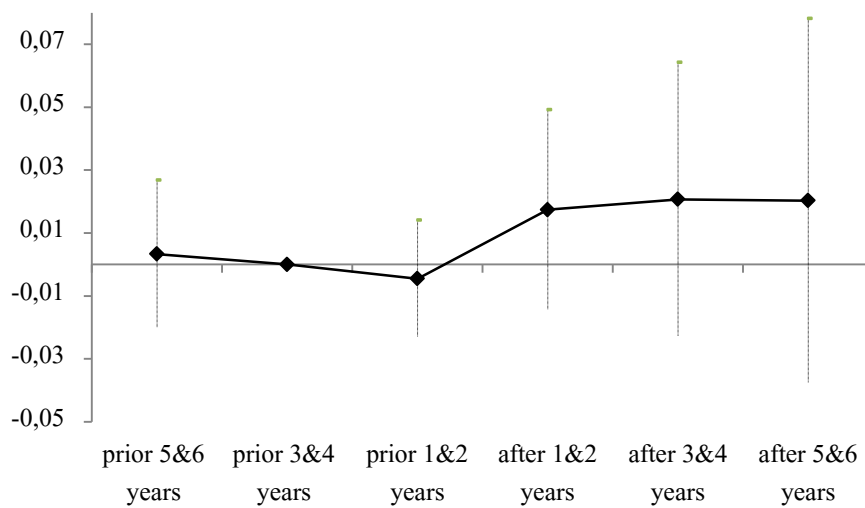
(b) Log income

Figure 2 – Coefficient estimates before and after the reform

Table 1 – Descriptive statistics

	Sample of implemented (*I*)	Sample of implemented and matched (*M*)
Mortality sample, ages 78 - 95		
post X health district	0.540	0.254
	(0.498)	(0.435)
mean death age (non-censored)	85.85	85.81
	(4.799)	(4.805)
mean censoring age	86.55	86.50
	(5.220)	(5.224)
fraction of observed deaths	0.920	0.922
	(0.272)	(0.269)
due to infectious diseases	0.091	0.091
	(0.287)	(0.288)
due to cardiovascular diseases	0.569	0.568
	(0.495)	(0.495)
due to diabetes	0.018	0.018
	(0.132)	(0.134)
due to cancer	0.156	0.155
	(0.363)	(0.362)
due to degenerative diseases	0.133	0.135
	(0.340)	(0.342)
due to other causes	0.034	0.033
	(0.180)	(0.179)
male	0.451	0.448
	(0.498)	(0.497)
Number of individuals	39,604	69,939
Permanent income sample, ages 78 - 95		
post X health district	0.538	0.253
	(0.499)	(0.434)
log income, mean yearly (including 0s)	7.707	7.704
	(0.551)	(0.536)
male	0.448	0.445
	(0.497)	(0.497)
Number of individuals	38,618	68,224

Note: means of the variables and standard deviations (in parentheses). Data from Swedish Interdisciplinary Panel

Table 2 – Hazard ratios. Effect of the reform on mortality in ages 78 – 95, cohorts 1890 – 1917
Sweden

	I	*I*	*I*	*I*	*M*
post X health district	0.940***	0.941***	0.940**	0.948**	0.958**
p-value	(0.006)	(0.007)	(0.035)	(0.022)	(0.012)
Cohort FE	yes	yes	yes	yes	yes
Parish of birth FE	yes	yes	yes	yes	yes
County of birth x cohort linear trends		yes			
Parish of birth Xs x cohort FE			yes		
Parish of birth x cohort linear trends				yes	
Individuals	39,604	39,604	39,604	39,604	69,939
Deaths	36,429	36,429	36,429	36,429	64,451

Note: exponentiated coefficients from Cox proportional hazards models. Models are adjusted for the left-truncation at age 78. *I* denotes a sample of implemented parishes of birth, *M* - sample of implemented and matched. Standard errors clustered at the parish of birth level (414 parishes). See main text for further description.

P-values in parentheses

*** p<0.01, ** p<0.05, * p<0.1

Table 3 – OLS estimates. Effect of the reform on log income in ages 78 – 95, cohorts 1890 – 1917 Sweden

	I	*I*	*I*	*I*	*M*
post X health district	0.0203**	0.0212**	0.0228**	0.0246**	0.0170**
p-value	(0.041)	(0.042)	(0.028)	(0.033)	(0.025)
Cohort FE	yes	yes	yes	yes	yes
Parish of birth FE	yes	yes	yes	yes	yes
County of birth x cohort linear trends		yes			
Parish of birth Xs x cohort FE			yes		
Parish of birth x cohort linear trends				yes	
Individuals	38,618	38,618	38,618	38,618	68,224

Note: OLS regression estimates. *I* denotes the sample of implemented parishes of birth, *M* sample of implemented and matched. Standard errors clustered at the parish of birth level (414 parishes). See main text for further description.

P-values in parentheses

*** p<0.01, ** p<0.05, * p<0.1

Table 4 – Hazard ratios. Effect of the reform on cause-specific mortality in ages 78 – 95, cohorts 1890 – 1917 Sweden

	Infectious	Cardiovasc.	Diabetes	Cancer	Degener.	Other
I						
post X health district	0.899	0.939**	0.946	0.922	1.003	0.930
p-value	(0.151)	(0.035)	(0.693)	(0.105)	(0.963)	(0.589)
Cohort FE	yes	yes	yes	yes	yes	yes
Parish of birth FE	yes	yes	yes	yes	yes	yes
Individuals	39,604	39,604	39,604	39,604	39,604	39,604
Deaths	3,304	20,733	649	5,675	4,847	1,221
M						
post X health district	0.961	0.953**	0.969	0.898**	1.030	1.058
p-value	(0.454)	(0.034)	(0.772)	(0.010)	(0.512)	(0.571)
Cohort FE	yes	yes	yes	yes	yes	yes
Parish of birth FE	yes	yes	yes	yes	yes	yes
Individuals	69,939	69,939	69,939	69,939	69,939	69,939
Deaths	5,862	36,579	1,185	9,961	8,723	2,141

Note: exponentiated coefficients from Cox proportional hazard models. Models are adjusted for the left-truncation at age 78. *I* denotes a sample of implemented parishes of birth, *M* - sample of implemented and matched. Standard errors clustered at the parish of birth level (414 parishes). See main text for further description.

P-values in parentheses

*** p<0.01, ** p<0.05, * p<0.1

Table 5 – Effects of the reform on mortality and log income by public health investments in ages 78 – 95, cohorts 1890 – 1917 Sweden

	Mortality risk	Log income
Investments into health care per capita		
post X health district X low	0.949**	0.0275**
	(0.048)	(0.034)
post X health district X high	0.935***	0.0158
	(0.004)	(0.109)
Midwives employed per capita		
post X health district X low	0.951**	0.0167
	(0.045)	(0.163)
post X health district X high	0.931***	0.0234**
	(0.003)	(0.024)
Share of infants		
post X health district X large	0.896***	0.0195
	(0.001)	(0.225)
post X health district X small	0.973	0.0247*
	(0.353)	(0.063)
Mortality rate under age 15		
post X health district X high	0.923**	0.0097
	(0.015)	(0.431)
post X health district X low	0.963	0.0107
	(0.216)	(0.411)
Cohort FE	yes	yes
Parish of birth FE	yes	yes
Parish of birth X x cohort FE	yes	yes
Parish of birth X x parish of birth FE	yes	yes
Individuals	39,604	38,618
Deaths	36,429	

Note: exponentiated coefficients from Cox proportional hazards models for mortality, adjusted for left-truncation at age 78, and OLS regression estimates for log income. The background characteristics are at the level of parish of birth. All groups divided at the median. Standard errors clustered at the parish of birth level (414 parishes). See main text for further description.

P-values in parentheses

*** p<0.01, ** p<0.05, * p<0.1

Table 6 – Effects of the reform on mortality and log income by socio-economic parish-of-birth characteristics, ages 78 – 95, cohorts 1890 – 1917 Sweden

	Mortality risk	Log income
SES, share of skilled		
post X health district X low	0.900***	0.0297
	(0.005)	(0.135)
post X health district X high	0.972	0.016
	(0.331)	(0.103)
SES, share in labour force		
post X health district X low	0.896***	0.0431**
	(0.002)	(0.029)
post X health district X high	0.976	0.0049
	(0.400)	(0.637)
Mean family size		
post X health district X large	0.932**	0.0187
	(0.027)	(0.200)
post X health district X small	0.941**	0.0180
	(0.049)	(0.179)
Railway		
post X health district X no	0.886***	0.0371**
	(0.004)	(0.049)
post X health district X yes	0.970	0.0112
	(0.250)	(0.297)
Cohort FE	yes	yes
Parish of birth FE	yes	yes
Parish of birth X x cohort FE	yes	yes
Parish of birth X x parish of birth FE	yes	yes
Individuals	39,604	38,618
Deaths	36,429	

Note: exponentiated coefficients from Cox proportional hazards models for mortality, adjusted for left-truncation at age 78, and OLS regression estimates for log income. The background characteristics are at the level of parish of birth. All groups divided at the median. Standard errors clustered at the parish of birth level (414 parishes). See main text for further description.

P-values in parentheses

*** p<0.01, ** p<0.05, * p<0.1

Table 7 – Robustness analyses for mortality and income in ages 78 – 95, cohorts 1890 – 1917 Sweden

	Mortality risk	Log income
A – Excluding counties of birth affected by emigration		
post X health district	0.938***	0.0186*
	(0.006)	(0.081)
Cohort FE	yes	yes
Parish of birth FE	yes	yes
Individuals	36,023	35,134
Deaths	33,127	
Parishes of birth	385	385
B - Excluding counties of birth affected by WWI		
post X health district	0.933***	0.0248**
	(0.004)	(0.018)
Cohort FE	yes	yes
Parish of birth FE	yes	yes
Individuals	35,320	34,453
Deaths	32,391	
Parishes of birth	409	409
C – Adding parish-specific controls for age structure		
post X health district	0.943***	0.0208**
	(0.009)	(0.024)
Cohort FE	yes	yes
Parish of birth FE	yes	yes
Individuals	39,604	38,618
Deaths	36,429	
Parishes of birth	414	414
D – Adding parish-specific controls for local labour market and migrant structure		
post X health district	0.930***	0.0251**
	(0.004)	(0.016)
Cohort FE	yes	yes
Parish of birth FE	yes	yes
Individuals	39,604	38,618
Deaths	36,429	
Parishes of birth	414	414

Note: exponentiated coefficients from Cox proportional hazards models for mortality, adjusted for left-truncation at age 78, and OLS regression estimates for log income. Standard errors clustered at the parish of birth level. See main text for further description.

P-values in parentheses. *** p<0.01, ** p<0.05, * p<0.1

APPENDICES

Appendix A - Description of the data sources

We gathered data on the division of the parishes into health districts and its changes from several sources. Primarily, governmental reports 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 collected from the health board acts (Medicinalstyrelsen 1907, 1939). We additionally verify these divisions with several sources, such as the provincial doctor reports attained from the National Archive in Sweden (Årberättelse 1893 – 1936), statistical yearbooks on health care (Statistiska Centralbyrån BISOS K, SOS 1880 – 1917, Statistiska Centralbyrån 1880 – 1917a) and on public health investment (Statistiska Centralbyrån BISOS U 1874 – 1917). These sources provided information on the number of the medical personnel employed, such as doctors and midwives, and public spending, both in health care and in education, infrastructure and welfare. In the latter case, we obtained the investment series for each parish before and after the establishment year, and aggregated them to a health district level, which allowed us to carefully determine the intervention dates. All urbanized (*stad*) and semi-urbanized (*köping*) parishes are excluded and therefore the sample comprises only rural parishes (*land*). We also exclude from the analysis the rural parishes that were developed throughout the period into small towns (*köping*), parishes that experienced several health district re-allocations or those where the adoption dates were uncertain (220 out of 2353 parishes). Although the creation of the medical districts continued from 1890 up until 1940s, we stop to follow the establishment of medical districts in 1917.

Data for health districts are gathered from:

1. Statistiska Centralbyrån, (1880 – 1917). BISOS U: Bidrag till Sveriges officiella statistik U. Kommunernas fattigvård och finanser, Statistiska centralbyrån, P.A. Norstedt & Söner, Stockholm (annual volumes).
2. ———, (1880 – 1910). BISOS K: Bidrag till Sveriges officiella statistik K. Hälso- och sjukvården. Statistiska centralbyrån, P.A. Norstedt & Söner, Stockholm (annual volumes).
3. ———, (1911 – 1917). SOS: Sveriges officiella statistik. Allmän om Hälso- och sjukvård. Statistiska centralbyrån, P.A. Norstedt&Söner, Stockholm (annual volumes).
4. Archival sources from the National Archive of Sweden, Medicinalstyrelsen 'Årberättelse från Förste provinsialläkare' i Malmohus län och Kristianstads län 1881-1946.

Administrative divisions of health districts into parishes are gathered from:

1. Betänkande angående rikets indelning in läkardistrikt samt tjänsteläkarens anställning och åligganden afgigtet af den af Kungl. Maj:t för sådant ändamål tillsatta kommitté, Stockholm, Kungl. Boktryckeriet, P.A.Norstedt & Söner, 1907.
2. Rikets indelning i provinsialläkardistrikt före 1/7 1939 och Medicinalstyrelsens yttrande och förslag till stadsläkarsakuniga den maj 1932, National Arkiv, Sverige, 1939.

Appendix B

Table - Characteristics of the old and newly established provincial health districts

Characteristic	Existed prior to 1880	Established 1881-1917
Number of health districts	163	124
Number of parishes per health district	14,1	6,9
Total population per health district	22,617	11,779
Number of midwives employed	-	4,4
Midwives employed per 10,000 population	-	5,6
Number of (extra or provincial) doctors employed per 10,000 population	-	1,9
Real investments into health care prior, 1900SEK	18,266	16,148
Real investments into health care prior per 1000 population, 1900SEK	715	1,611
Real investments into health care after, 1900SEK	-	19,267
Δ in real investments into health care, 1900SEK	-	4,928
Δ in real investments into health care per 1,000 population, 1900SEK	-	684
Real total public spending prior, 1900SEK	196,182	142,668
Real total public spending prior per 1,000 population, 1900SEK	6,792	11,638
Real total public spending after, 1900SEK	-	130,857
Δ in total public spending, 1900SEK	-	8,317
Δ in total public spending per 1,000 population, 1900SEK		1,272

Note: Parish- and health-district indicators gathered from Statistiska Centralbyrån, BISOS U and K 1880 – 1917.

Appendix C

Table – Factors of staggered implementation of the reform, 1890 – 1917

	after1907=1	
	levels 1880	differences 1890 to 1880
log real investment into health care per parish	0.198***	-
	(0.000)	
log real education, infrastructure and welfare spending per parish	0.136***	-
	(0.000)	
log population	-0.0688**	-0.904***
	(0.013)	(0.000)
share of elite and industrial workers in male population 15 − 55 ages	0.298	-0.346
	(0.123)	(0.197)
share of agricultural workers in male population 15 − 55 ages	0.0936	0.558**
	(0.647)	(0.019)
mean age of female	0.0118	0.0216
	(0.291)	(0.146)
share females in total	1.755	2.121
	(0.147)	(0.134)
share in labour force in total 15 − 55 ages	0.567**	0.0105
	(0.010)	(0.970)
share married in total 15 − 55 ages	-0.169	0.0840
	(0.657)	(0.884)
mean family size	-0.212***	-0.0344
	(0.000)	(0.618)
share under age 1 in total	-4.232	-3.656
	(0.291)	(0.269)
share above age 55 in total	0.882	1.939*
	(0.306)	(0.0549)
mortality rate under age 15	0.00239	0.000332
	(0.765)	(0.775)
share (non)disabled	0.0720	3.890
	(0.985)	(0.297)
Railway	0.0533	0.00900
	(0.284)	(0.849)
water supplies improvements	0.216*	0.112
	(0.053)	(0.372)
Observations	492	492

P-values in parentheses.

*** p<0.01, ** p<0.05, * p<0.1

Note: OLS regression estimates. All characteristics are parish-level. The mean implementation year is 1907. Each coefficient is estimated separately. See Appendix for data sources and description. Parish-level indicators gathered from Censuses 1880, 1890, 1900 and 1910, and from Statistiska Centralbyrån, BISOS U and K 1880 – 1917.

Appendix D

The cohorts born between 1890 and 1917 appear in the SIP dataset from 1968, consistently between the ages 78 and 95. We therefore do not observe individuals that died or migrated from Sweden prior to these ages. In SIP, the data on parish of birth (together with county of birth) are given in text format, and we used an automatic procedure to match these names to parish names in our treatment health district dataset that we further check manually. In the estimation sample, out of 492 parishes treated by the reform, we observe representatives of the 414 parishes.

We gathered information on one-year survivors born in rural areas (live births minus infant deaths) of the cohorts born 1890-1917 from Statistiska Centralbyrån, BISOS A. In Figure we plot them against counts of individuals with rural places of birth available in SIP by cohort and those which have valid information on the parish of birth. Increasing fraction of individuals observed in the SIP dataset compared to a rather stable fraction of one-year survivors from rural places indicate that the individuals born 1890 – 1917 were dying at an increasing rate between the years 1 and 77. As the proportion of the individuals treated by the reform is increasing over time, the effects of the reform can be thus underestimated.
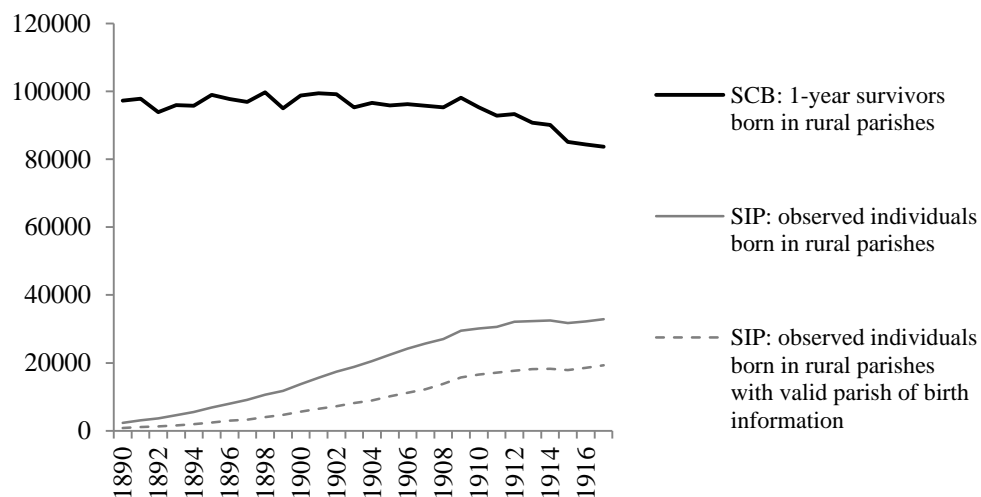

Figure – One-year survivors and estimation sample for the cohorts 1890 – 1917
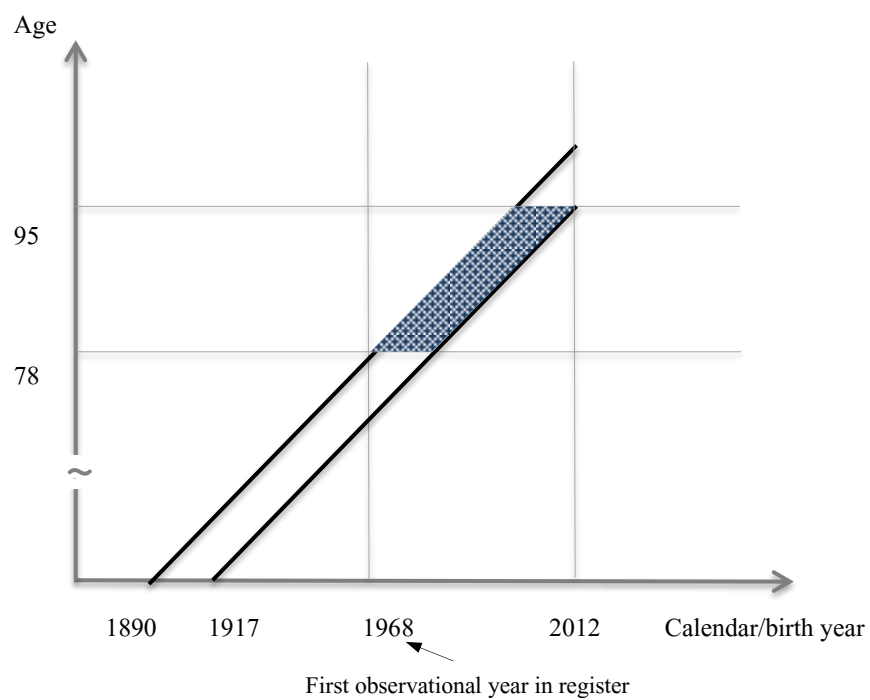Source: SIP and SCB.

Figure – Lexis chart illustrating the estimation sample

Appendix F - The cause-of-death groupings

The death and cause-of-death data is obtained from the Swedish death register and the Swedish cause-of-death register. These registers adopted the different revisions of the international classifications of the causes of death throughout 1968 – 2012, such as the revision 8 for 1968-1986, the revision 9 for 1987 – 1996, and the revision 10 for 1997 – 2012. We classify all causes of death into five groups, such as infectious diseases, cardiovascular diseases, diabetes, cancer, degenerative diseases of tissues and organs, and other causes including violent and ill-defined causes. The exact codes used for these groupings are provided in the following table:

Table – Diagnoses groups across different revisions of the ICD, 1968 – 2012

	ICD-8	ICD-9	ICD-10
Infectious diseases	000-136; 320-324; 460-519	001-139; 320-324; 460-519	A00-B99; G00-G09; J00-J99
Cardiovascular diseases	390-458	390-459	I00-I99
Diabetes	250	250	E10-E14
Cancer	140-239	140-239	C00-D48
Degenerative diseases	240-246; 251-315; 325-389; 520-789	240-246; 251-319; 325-389; 520-796	D50-E07; E15-F99; G10-H95; K00-R94
Other causes	790-796; E800-Y87	797-999; E800-V82	R95-Z99

Appendix G - Matching procedure

Figure 1 plots a cumulative number of parishes by their dates of the reform implementation. 492 out of 2133 rural parishes established new health districts. From a sample of parishes untreated by the reform we select one-to-one matches to each treated parish, based on the variety of the pre-treatment parish and health-district characteristics at both levels and trends. The motivation for the determinants of health district implementation is a standard model of public health care utilization that involves socio-economic, infrastructure, health, demographic and health system domains. For this approach, we calculate propensity scores and apply a nearest neighbor matching, in which we allow it to find only one control without replacement and impose a common support restriction, with a caliper 0.10. Narrowing or widening of the caliper gives qualitatively analogous results. After the matching, the control and treated groups exhibit a more similar distribution by propensity scores (see Figure 2). As can be seen in Table, the matching procedure allowed us to arrive at 432 treated and 432 matched parishes and therefore substantially enlarge the estimation sample. After the procedure, there are no significant differences between the treated and control groups of parishes by all parish- and health-district level characteristics.
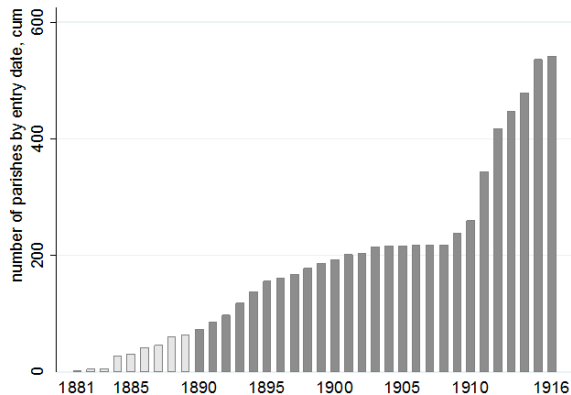

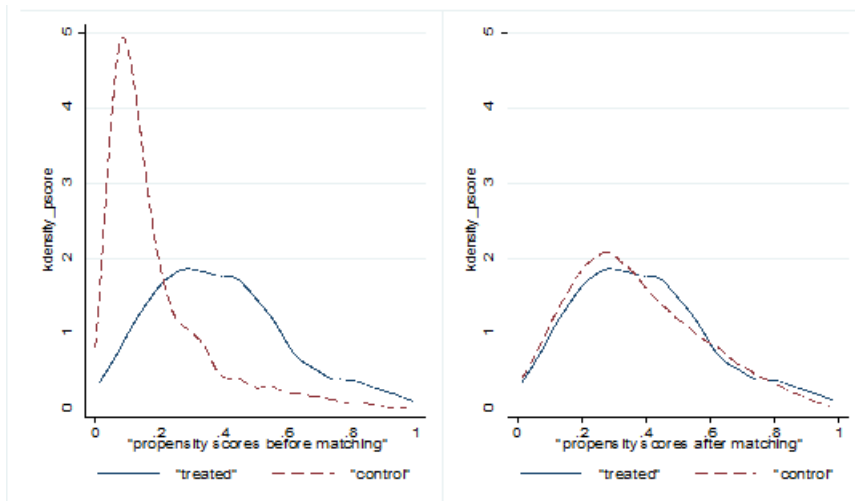Figure 1 - Number of parishes by their date of entry into treatment, 1881 – 1917

Figure 2 – Density of the parishes of birth over the propensity scores

Table – Parish and health-district pre-treatment characteristics used for the full sample (control and implemented) and matched sample

	Before matching			After matching		
	Control	Treated		Control	Treated	
Variable	Mean	Mean	p-value	Mean	Mean	p-value
	1	2	3	4	5	6
Levels			1 versus 2			5 versus 6
log real health care investment per parish	6,422	7,260	0,000	7,208	7,126	0,153
log real education, infrastructure and welfare spending per parish	8,988	9,402	0,000	9,363	9,330	0,408
log total population 1880	7,130	7,232	0,011	7,277	7,226	0,335
share elite and industrial workers 1880	0,256	0,266	0,045	0,262	0,264	0,731
share agricultural workers 1880	0,421	0,406	0,005	0,410	0,408	0,867
mean age of female 1880	29,295	29,250	0,638	29,241	29,250	0,943
share females 1880	0,510	0,507	0,010	0,507	0,507	0,500
share in labour force 1880	0,674	0,666	0,074	0,666	0,666	0,996
share married 1880	0,480	0,480	0,800	0,483	0,482	0,859
share infants 1880	0,025	0,025	0,706	0,025	0,025	0,573
share older 55 ages 1880	0,150	0,150	0,670	0,150	0,150	0,883
under 15 mortality rate 1880	0,695	0,753	0,617	0,868	0,818	0,788
share (non)disabled 1880	0.992	0.993	0,024	0,992	0,992	0,560
mean family size 1880	4,037	4,015	0,316	4,030	4,025	0,890
railway1880	0,235	0,234	0,968	0,229	0,226	0,935
water supplies 1880	0,045	0,037	0,415	0,035	0,037	0,855
Differences						
diff log total population 1880-1890	-0,027	-0,027	0,792	-0,016	-0,020	0,657
diff share elite and industrial workers 1880-1890	-0,015	-0,009	0,093	-0,004	-0,008	0,408
diff share agricultural workers 1880-1890	0,021	0,013	0,106	0,010	0,014	0,547
diff mean age of female 1880-1890	1,140	1,319	0,017	1,262	1,202	0,561
diff share females 1880-1890	0,001	0,001	0,590	-0,001	0,001	0,312
diff share in labour force 1880-1890	0,023	0,028	0,136	0,028	0,028	0,985
diff share married 1880-1890	0,004	0,002	0,264	0,003	0,003	0,940
diff share infants 1880-1890	-0,001	-0,002	0,223	-0,002	-0,002	0,348
diff share older 55 ages 1880-1890	0,026	0,028	0,070	0,026	0,026	0,994
diff under 15 mortality rate 1880-1890	6,604	6,226	0,629	6,637	6,359	0,811
diff share (non)disabled 1880-1890	-0.001	-0.001	0,369	-0,001	-0,001	0,991
diff mean family size 1880-1890	-0,185	-0,189	0,806	-0,192	-0,183	0,656
diff railway 1880-1890	-0,090	-0,077	0,560	-0,069	-0,085	0,596
diff water supplies 1880-1890	0,010	0,016	0,555	0,019	0,016	0,847
Number of parishes	**1641**	**492**		**432**	**432**	
Number of health districts	**163**	**107**		**131**	**96**	

Appendix H - Additional results for mortality

Table – Robustness analyses. Gompertz proportional hazards model. Effect of the reform on mortality 78 – 95 ages, cohorts 1890 – 1917

	I	*I*	*I*	*I*	*M*
post X health district	0.944**	0.945**	0.943*	0.959*	0.957**
p-value	(0.016)	(0.019)	(0.060)	(0.088)	(0.019)
Cohort FE	yes	yes	yes	yes	yes
Parish of birth FE	yes	yes	yes	yes	yes
County of birth x cohort linear trends		yes			
Parish of birth Xs x cohort FE			yes		
Parish of birth x cohort linear trends				yes	
Individuals	39,604	39,604	39,604	39,604	69,939
Deaths	36,429	36,429	36,429	36,429	64,451

Note: Models are adjusted for the left-truncation at age 78. *I* denotes a sample of implemented parishes of birth, *M* - sample of implemented and matched. Standard errors clustered at the parish of birth level (414 parishes). For parish-specific trends age interval is 78 – 96. See main text for further description.

Table – Robustness analyses. Effect of the reform on alternative measures of survival 78 – 95 ages, cohorts 1890 – 1917

	I	*I*	*I*	*I*	*M*
dead=1, OLS, **78 – 82 ages**					
post X health district	-0.0187**	-0.0205**	-0.0165	-0.0208**	-0.0117**
p-value	(0.048)	(0.033)	(0.193)	(0.041)	(0.090)
dead=1, OLS, **78 – 87 ages**					
post X health district	-0.0239**	-0.0230**	-0.0255**	-0.0208*	-0.0201***
p-value	(0.026)	(0.030)	(0.043)	(0.056)	(0.006)
dead=1, OLS, **78 – 95 ages**					
post X health district	-0.0019	-0.0017	-0.0074	-0.0015	-0.0034
p-value	(0.717)	(0.751)	(0.313)	(0.804)	(0.431)
log time alive, Tobit, **78 – 95 ages**					
post X health district	0.0769**	0.0761**	0.0572	0.0704***	0.0466**
p-value	(0.014)	(0.017)	(0.128)	(0.000)	(0.037)
Cohort FE	yes	yes	yes	yes	yes
Parish of birth FE	yes	yes	yes	yes	yes
County of birth x cohort linear trends		yes			
Parish of birth Xs x cohort FE			yes		
Parish of birth x cohort linear trends				yes	
Individuals	39,604	39,604	39,604	39,604	69,939

Note: *I* denotes a sample of implemented parishes of birth, *M* - sample of implemented and matched. Standard errors clustered at the parish of birth level (414 parishes). See main text for further description. For the tobit models, the observations are right-censored at 2.890 (log of 18 years). Mean in not-treated is 9.4 years.

Appendix I - Additional results for log income

Table – Robustness analyses. OLS models. Effect of the reform on permanent income 78 – 95 ages, cohorts 1890 – 1917

	I	I	I	I	M
life-time earnings 78 − 95 ages					
post X health district	0.0199**	0.0217**	0.0196**	0.0238**	0.0215***
p-value	(0.037)	(0.032)	(0.045)	(0.027)	(0.007)
Cohort FE	yes	yes	yes	yes	yes
Parish of birth FE	yes	yes	yes	yes	yes
County of birth x cohort linear trends		yes			
Parish of birth Xs x cohort FE			yes		
Parish of birth x cohort linear trends				yes	
Individuals	38,618	38,618	38,618	38,618	68,224
log income 78 − 82 ages					
post X health district	0.0222**	0.0246**	0.0261*	0.0236**	0.0193**
p-value	(0.028)	(0.024)	(0.019)	(0.041)	(0.017)
Cohort FE	yes	yes	yes	yes	yes
Parish of birth FE	yes	yes	yes	yes	yes
County of birth x cohort linear trends		yes			
Parish of birth Xs x cohort FE			yes		
Parish of birth x cohort linear trends				yes	
Individuals	38,609	38,609	38,609	38,609	68,208
log income 78 − 87 ages					
post X health district	0.0208**	0.0214**	0.0227**	0.0245**	0.0178**
p-value	(0.035)	(0.040)	(0.029)	(0.034)	(0.021)
Cohort FE	yes	yes	yes	yes	yes
Parish of birth FE	yes	yes	yes	yes	yes
County of birth x cohort linear trends		yes	yes	yes	
Parish of birth Xs x cohort FE			yes		
Parish of birth x cohort linear trends				yes	
Individuals	38,615	38,615	38,615	38,615	68,218
log income 78 − 95 ages, closest value for income instead of the null					
post X health district	0.0156*	0.0161*	0.0218**	0.0210**	0.0150**
p-value	(0.066)	(0.074)	(0.017)	(0.040)	(0.029)
Cohort FE	yes	yes	yes	yes	yes
Parish of birth FE	yes	yes	yes	yes	yes
County of birth x cohort linear trends		yes			
Parish of birth Xs x cohort FE			yes		
Parish of birth x cohort linear trends				yes	
Individuals	38,618	38,618	38,619	38,619	68,224
log income 78 − 95 ages, Excluding years under widowhood					
post X health district	0.0225*	0.0241*	0.0261*	0.0307**	0.0235**
p-value	(0.086)	(0.080)	(0.065)	(0.045)	(0.013)
Cohort FE	yes	yes	yes	yes	yes
Parish of birth FE	yes	yes	yes	yes	yes
County of birth x cohort linear trends		yes			
Parish of birth Xs x cohort FE			yes		
Parish of birth x cohort linear trends				yes	
Individuals	27,824	27,824	27,824	27,824	48,941

Appendix K – Additional robustness analyses for selection

Table – Robustness analyses. Effect of the reform on migrant structure and household size of the parishes of birth, 1890 – 1917

	(1) Share of migrants	(2) Household size
post X health district	0.00100	0.00141
	(0.372)	(0.851)
Cohort FE	yes	yes
Parish of birth FE	yes	yes
Observations	3,108	3,108
R-squared	0.082	0.006

Note: The analyses restricted to parish-cohorts used in the sample. Data is obtained from censuses 1880 – 1910 and recalculated to parish-of-birthXcohort level.