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Volha Lazuka

DEPARTMENT OF ECONOMIC HISTORY, LUND UNIVERSITY

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Infant health and later-life labour market outcomes: Evidence from the introduction of sulfa antibiotics in Sweden

Lazuka, Volha

Centre for Economic Demography and Department of Economic History, Lund University

Abstract

There is a growing body of literature showing that health in infancy has a strong influence on health and productivity later in life. This paper uses exogenous improvements in infant health generated by the introduction of a medical innovation in the late 1930s as treatment against several infectious diseases, in particular pneumonia reduced by the advent of the sulfa medicaments. Based on rich administrative population data for Sweden 1968–2012 and archival data on the availability of sulfa antibiotics, it explores the effect of reduction in exposure to pneumonia in infancy on labour market outcomes discerned in adulthood of the affected cohorts. Our findings suggest that mitigation of pneumonia disease burden in infancy substantially reduced probability of working disability and increased labour income in late adulthood. Regarding the mechanisms, the beneficial effects are strong for health, measured with reduced number of hospital admissions, and somewhat weaker for years of schooling. These effects are fairly equal among males and females, and larger among individuals from disadvantaged backgrounds. All effects are robust to various specifications including regional and family factors.

Keywords: medical innovation, sulfa antibiotics, early-life effects, infancy, labour productivity, health, human capital, Sweden

JEL: I15, I18, H41, N34

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Introduction and previous research

To date, an economic and demographic literature has established strong relationships between productivity growth rates and population health (Weil 2014). Consequently, recent research has shifted to identifying the causal factors behind labour productivity, among which human and health capital accumulation through the life course gains the increasing an attention. The emerging literature emphasizes early childhood, in particular fetal stage and infancy, as the period when public or family investments yield the largest rates of return (Almond & Currie 2011). When infant or child health improves, for instance due to lowered exposure to infectious diseases, individuals are likely to gain in different aspects of later-life health, such as morbidity and vitality (Finch & Crimmins 2004). The burden of infectious disease in early life affects developing brain and thus cognitive ability (Eppig *et al.* 2010). In a framework proposed by Heckman (2007) and Cunha and Heckman (2007), health and cognitive ability in early life are considered as capacities affecting the production of future capabilities. More specifically, health and abilities in early life are characterized by multiplier effects, which boost both the returns to investments in the health and human capital stock, with consequences for labour productivity later in life. These effects imply especially high productivity of investment in children from disadvantaged families early in life, with such investments therefore leading to a decrease in health and economic inequalities in the long run (Currie 2009). This paper exploits improvements in child health, exogenously affected by the sharp arrival of sulfa drugs in Sweden efficient in treatment against pneumonia, as early-life experiment and investigates its effects on labour market outcomes in individuals' later lives. It additionally enables us to explore both education and health as important channels through which infant health translates into higher earnings in adulthood.

In a history of social intervention, introduction of sulfa drugs became the first public action that provided the population with treatment of several pediatric infectious diseases. In Sweden, the sulfa antibiotics, initially imported and later produced by the national pharmaceutical companies, were introduced into medical practice sharply after the medical review and granted the international recognition to their inventor (The Nobel Foundation 1965). Due to the high availability of medical personnel, centralization of drug distribution and low costs of treatment (Hollingsworth *et al.* 1990), this medical innovation, as we show in this paper, spread quickly across regions of Sweden and led to convergence in death rates from the targeted diseases. The sulfonamides appeared to be extremely efficient against puerperal infections, gonorrhea, meningitis, erysipelas, scarlet fever, dysentery, but especially so against acute pneumococcal pneumonia (Domagk 1957). In the 1920s–1930s, pneumonia took the bulk of deaths and caused severe and repeated morbidities among infants and children (Lindborg 1936), and society offered no efficacious treatments equivalent in costs and efficiency to sulfa drugs (Ingvar 1939). By that time, other childhood infectious diseases treatable by sulfonamides contributed little to the general child mortality (SOS 1930a). The sudden arrival of sulfa antibiotics that initiated convergence in pneumonia mortality among infants therefore suggests a unique natural experiment in early-life environmental conditions. Compared to other countries of North America and Europe, beginning from the 1950s, and hence in the adulthood

of the affected cohorts, Sweden was characterized by the rapid expansion of the welfare state and active inclusion of all population groups into labour market (Lindert 2004). As a result, the institutional setting should not preclude the individuals from full realization of the benefits in health and abilities acquired in early childhood.

The macro-level literature studying the impact of health on labour productivity, recently so using causal tools, has obtained mixed results. In a seminal paper, Preston (1975) indicates a strong impact of public health and medical innovations on gains in life expectancy at birth between the 1930s and the 1960s. There is an abundant empirical literature showing robust associations between initial health conditions and labor productivity increases based on cross-country data (Gallup & Sachs 2001; Bloom *et al.* 2004; Weil 2014 for review). By applying an instrumental variables method and thus obtaining more credible results from a causal perspective, Acemoglu and Johnson (2007) find that abrupt decrease in mortality due to the arrival of efficient cures against major infectious diseases, such as tuberculosis, malaria and pneumonia, in the 1940s increased life expectancy across countries in the long run. These health gains, however, did not translate into economic growth. In contrast, for a similar set of countries, Ashraf *et al.* (2009) show that the long-run responses in economic growth in per capita terms to the application of health and medical technologies are positive but moderate. The scholars simulate the effect of such innovations based on different channels by which health affects income, including early-life mechanisms. Alongside, several studies show that the effects of health improvements in both quantity and quality of life in long term, including decrease in inequalities, appear to be large (Becker *et al.* 2005; Murphy & Topel 2006; Jones & Klenow 2016), and especially so due to the impact of medical innovations, such as drugs or vaccines (Bloom *et al.* 2005; Jayachandran *et al*. 2010; Cutler *et al*. 2012).

In the literature studying the relationship between health and labour productivity, number of the micro-level studies applying a cohort approach is growing. In addition to arguments from economics (Cunha & Heckman 2007; Heckman 2007) mentioned above, micro-level studies are leaning on the assumptions of the epidemiological studies about the life-long importance of early life circumstances (Kuh & Ben-Schlomo 2004; Gluckman *et al.* 2008). There is an emerging empirical micro-level literature that by connecting variations in disease or nutritional environment in early life to later-life health and socio-economic status finds substantial effects. This literature is not only capable of deriving aggregate estimates similar to the ones in macroeconomic studies, but also enables the researcher to apply better identification strategies (Bleakley 2010). One strand of this research finds strong effects of epidemics or episodes of nutrition deprivation in early life on cognitive ability and labour productivity later in life (Almond *et al.* 2006; Bengtsson & Broström 2009; Barreca 2010; Kelly 2011). Another one combines ecological disease rates with sharp introduction of intervention programs to obtain exogenous variation in infant or child health. For instance, Bleakley (2007) shows a substantial impact of exposure to hookworm infection below age 18 on labour market outcomes in adulthood by using hookworm-eradication campaign in the US South in 1910. Based on natural experiment in malaria eradication in the US (1920) and in several developing countries (1955), Bleakley (2010) finds strong effects of childhood exposure on adult literacy and incomes.

Similar or more moderate results from malaria eradication have been shown for other countries (Cutler *et al.* 2010; Lucas 2010; Venkataramani 2012). Bhalotra and Venkataramani (2013) exploit the water reform in 1991 in Mexico to investigate the impact of risk of waterborne disease in early childhood on cognitive outcomes in young adulthood. For the US, Bhalotra and Venkataramani (2015) find that reduced exposure to pneumonia in infancy after the introduction of sulfonamides in 1937 led to gains in educational attainment and socio-economic outcomes of the affected cohorts.

This literature could be seen as a part of the expanding research on the relation between positive, policy-driven interventions in early life and later-life economic and health outcomes. Such literature has grown recently (Almond & Currie 2011; Currie & Rossin-Slater 2015), otherwise our knowledge on what programs generate the long-term positive outcomes remains fragmentary. For instance, in Sweden, in the 1890s-1920s, the nationwide expansion of early public health care accompanied by the establishment of isolation hospitals and the use of antiseptics in midwifery practice in infancy had strong influence on individuals' survival and permanent incomes (Lazuka *et al.* 2016; Lazuka 2017). According to Beach *et al.* (2016), the gradual installation of water purification technologies across the US cities in the beginning of the twentieth century led to higher earnings and education attainment in prime ages of the individuals affected in early life. In the 1930s, as Aizer *et al.* (2016) find, the provision of cash transfers to poor families in the US helped to improve the prospective health and incomes of the affected individuals. The public access to food stamps rolled out in several decades, as Hoynes *et al.* (2016) shows, led to a significant reduction in metabolic syndrome and an increase in labour productivity among individuals treated in childhood. Initiated in the late 1930s, the nationwide implementation of home visiting programs targeting infants positively influenced vitality, survival and earnings of the treated cohorts across the Scandinavian countries (see Bütikofer *et al.* 2015 for Norway; Bhalotra *et al.* 2017 for Sweden; Hjort *et al.* 2017 for Denmark). These programs included various components, such as health examination of infants and provision of information about their proper nutrition and health development. The emerging literature tends therefore to show that specific environmental conditions in childhood, such as nutrition, infection and early health care (Montez & Hayward 2011), are capable of generating lasting effects.

Complementarily, epidemiological literature trying to understand the biological mechanisms behind the long-lasting impact of environmental conditions in early life on health and productivity is expanding. The concept of so-called developmental origins of health and disease has been applied encompassing the connection between early-life events and disease and wellbeing later in life (Gluckman *et al.* 2010). Because of rapid development of organs and cells of the body in the fetal stage and first years of life, adverse or beneficial exposures during early life can leave permanent and irreversible imprints on an individual's health and abilities. The epidemiological literature points at several potential channels, such as long-run maladaptation to environmental signals or permanent damage to the body, and chronic immunity responses launched in early life (Gluckman *et al.* 2008; Hostinar & Gunnar 2013). Exposure to infectious diseases, in particular acute respiratory diseases on health (Finch &

Crimmins 2004), has been emphasized as an important early-life environmental factor. Recent studies have proven that the dependence of abnormalities of pulmonary function in adulthood on childhood exposure to pneumonia is at least as large as those related to adult smoking (Barker 1991; Galobardes *et al.* 2008; Stocks & Sonnappa 2013; Carraro *et al.* 2014). The development of the various types of arthritis has been linked to exposure to streptococcus bacteria in early-life (Colebatch & Edwards 2010). The link between early-life conditions and the cardiovascular diseases and the associated risks is also strong (Barker 1995; Gluckman *et al.* 2008; Sun *et al.* 2013). In particular, contagion with *Streptococcus pneumoniae* in early childhood is expected to cause the major, acute cardiac events, such as myocardial infarction, arrhythmia, and congestive heart failure (Willerson & Ridker 2004; Muscher *et al.* 2007; Hayden *et al.* 2015). Additional studies show how early-life exposure to infection affects the development of brain leading to staggered cognitive and behavioral abilities (Landrigan 2005).

By using high-quality administrative population data for Sweden, the current paper raises the following research questions: (i) What are the long-term effects on exposure to pneumonia in infancy (discerned through the introduction of sulfa antibiotics as its efficient treatment) on labour productivity? (ii) Do these effects operate through health or human capital accumulation, or both? (iii) Are the long-term effects stronger for individuals from disadvantaged families (thus leading to long-term decrease in socio-economic and health disparities)?

Having stated this, this paper makes several contributions to the existing literature. Primarily, it facilitates the emerging literature exploring the long-run effects of shocks or interventions in early life on health and socio-economic outcomes (Almond & Currie 2011; Currie & Rossin-Slater 2015). In doing so, it employs both health and labour market outcomes thereby contributing to the discussion on the causes of economic development; whether health is an important determinant of economic growth and welfare in the long run (Bleakley 2010; Weil 2014). This paper includes the assessment of the mediating role of education and health to income improvements. It also enables us to conclude whether the introduction of sulfa antibiotics as a public good reduced socio-economic disparities in the long run (and therefore exposure to pneumonia increased it) by examining the treatment effects between individuals with different socio-economic backgrounds. Moreover, this paper contributes to the multidisciplinary literature (Gluckman *et al.* 2008) by studying the effects of the reduced burden of a specific infectious disease in infancy, namely acute pneumonia. We exploit the implementation of medical drugs against pneumococcal infection that helped to substantially reduce not only individual mortality and morbidity due to pneumonia (through treatment by drugs) but also peer infection (through improvements in disease environment) (Alm 1942). This paper not only attempts to show the presence of long-lasting effects due to childhood exposure to pneumonia but also – by looking at health outcomes by cause of morbidity – disentangles what biological processes are lying behind this link.

Compared to previous studies, this paper attains several methodological merits. First, the majority of the aforementioned studies in exploring the long-term effects rely on cross-section

data pooled for different census years. This paper constructs the outcomes for the cohorts based on the high-quality longitudinal data for the total population of Sweden. This enables us both to correctly identify the outcomes for individuals across the life course and to measure them within the same age ranges for all cohorts (Watcher 2014). As mentioned above, it also utilizes the larger variety of the outcomes, including not only education and precise measures of labour productivity, but also general health and morbidity by specific conditions. Secondly, as a part of its empirical strategy, this paper will also show that the introduction of sulfa drugs leads to substantial period reductions in infant, child and cause-specific mortality. To our knowledge, there are empirical studies that look at the period effects of antibiotics at the population level for the US (Jayachandran *et al.* 2010), but similar studies are absent for European countries including Sweden. Thirdly, the data provides us parental health and socio-economic characteristics, as well as sibling links, which enable to control for unobserved heterogeneity at a family level, to examine the presence of parental responses to the changes in health endowments of their children and to study the treatment effects by different family or mother sub-groups. Finally, based on the archival sources, we collected information about the distribution of sulfa antibiotics across regions of birth. This allows us to show whether the acquisition of the treatment by drugs differed by socio-economic groups, and thus whether heterogeneous effects between disadvantaged and affluent families stem from differences in take-up or treatment.

The majority of the merits of the current paper apply compared to the study by Bhalotra and Venkataramani (2015) that examines the long-term impact of childhood exposure to pneumonia in the US. The researchers find that the introduction of sulfa antibiotics in treatment against pneumonia led to substantial gains in schooling, incomes and income mobility among the treated cohorts, albeit discerned only among males and being stronger among white males. Compared to other countries of North America and Europe, the case of Sweden – welfare and egalitarian – in adopting sulfa antibiotics to treat pneumonia in infancy and enabling all population groups to benefit from it – is likely to be nearly perfect to study the issue. Both papers rely on the methodology developed by previous studies looking at the long-term effects on interventions targeting eradication of specific diseases (cf. Bleakley 2007), although this paper attains certain important differences. The data availability is strongly favouring our study. Uniquely to the previous literature, we analyse distribution and availability of sulfa antibiotics across all regions (cf. Jayachandran *et al.* 2010). The aggregate data on pneumonia in our case is registered separately from influenza, which is the disease correlating with pneumonia but not treated by sulfa drugs. Such enriched body of information, together with advantages provided by the longitudinal register-based data on individuals and their parents, strengthens an instrumental variables approach adopted in this paper and conventionally used in studies looking at the intervention-led impact of certain diseases in the long run. While the research questions broadly resemble in both papers, this paper enables to pose additional ones. It allows us to explore the long-term impact of pneumonia not only on labour market outcomes, but also on health and human capital stocks. The investigation of the biological mechanisms behind these links emerges as our additional merit.

Context

The necessity to improve infant health, whose advances in Sweden began to stagnate similarly to in other countries such as the US, England or Denmark (Wegman 2001; Griffiths & Brock 2003; Edvinsson *et al.* 2008), received wide public attention in the 1920s. The Swedish government in 1929 presented a report on public health insurance that in part demonstrated the large inequalities in infant and child mortality across regions, urban and rural areas and socio-economic classes (SOU 1929; Wisselgren 2005). Despite the remarkable economic development in the country, measured for instance with real income per capita that had grown at a 2.4 percent annually beginning from 1890 (Schön & Krantz 2014), these differentials persisted. Among candidate factors responsible for health inequalities several ones had been listed, such as the standards of living, including housing, family size, infectious disease environment, nutrition, childcare, willingness to use medical services, as well as climatic conditions (Sundin & Wilner 2007). Causes associated with infectious diseases clearly dominated both infant and child mortality (see Table 1). Similar to other countries of Western Europe and North America, respiratory diseases, such as pneumonia, bronchitis and influenza, exhibited stagnation or slow decline for a half of the century up until the late 1930s in Sweden (Preston *et al.* 1972; Hofsten & Lundström 1976). Among exogenous causes, pneumonia and influenza alone accounted for not less than 20 percent of premature deaths below the age five in the 1920–1930s, protruding as the major cause of death among infants and the major infectious-disease cause of death among children (see Table 1).

[Table 1]

"Pneumonia" is commonly used to refer to all types of inflammation of the lung. In the majority of cases, it is applied to diseases of infectious origin, such as lobar pneumonia and broncho-pneumonia. Regarding the etiology, pneumonia can be caused by both viruses and bacteria of different kinds, although more than half of the cases are caused by bacteria *Streptococcus pneumoniae* (also called *Pneumococcus*, Cronberg 1976). Such disease is characterized by sudden onset, high fever and severe malaise among the previously healthy individuals. In 20–30 percent of untreated cases, the contagion by pneumonia leads to death, and recovery from disease lasts approximately a month (Klugman & Feldman 2009). It is recognized that the disease is more common among children, primarily among infants, than among adults (Mercer 1990). More specifically, in the 1920–1930s in Sweden infant mortality rates due to pneumonia and bronchitis amounted to around 620 per 100,000 live births that is five times lower than among total population (Figure 1). The elderly are also severely affected by pneumonia, although in their case the disease is chronic and associated with other debilitating health conditions (Cronberg 1976). Morbidity due to pneumonia accounted for more than 200 cases per 100,000 (SOS 1935a), which is the officially recorded number and likely to be underestimated (Ungerleider *et al*. 1943). Abundant empirical research in medicine that was carried out worldwide in the pre-drug era documented the pathology of the disease, although it advanced little in providing the efficient cure (Office of Health Economics 1963). In the beginning of the 1930s, the practice to treat pneumococcal pneumonia with antiserum, that

is immunological therapy, had been approbated by the hospitals of Stockholm, similarly to the practice in large hospitals in other countries (Rahm 1936). However, any intention to implement such procedures throughout the country proved elusive due to the method being labour intensive, hospital-dependent and uncertain in efficacy (Ingvar 1939; Lindau 1939; Watson *et al.* 1993; Podolsky 2005).

[Figure 1]

The invention and introduction of sulfa antibiotics into medical practice of treatment against pneumonia and other infectious diseases is recognized as one of the major historical breakthrough innovations (WIPO 2015). The efficiency of sulfonamides, named Prontosil, against many experimental streptococcal and other infections was observed by G.Domagk in Germany in 1932 (Domagk 1957). At the beginning, sulfonamides were discovered as pro-drugs as they had been found to be efficient only in live animals but not in the test tube (Dahlberg 1939). By the late 1930s, a bacteriostatic component of sulfonamides – "sulfa" – had been revealed, that prevented the bacteria from multiplying and did not kill it, and experiments with derivatives of sulfonamide preparations were elsewhere launched (The Nobel Lectures 1965). The production and trade of the drugs at a mass and international scale started by the end of the 1930s, among which the major success is attributed to sulfapyridine (a compound of pyridine and sulfonamides) prepared by the May and Baker Company as M&B 693 and appeared to be efficient against pneumonia (Bentley 2009). The M&B 693 drug has been officially reported as the first chemical cure against pneumonia and the most noteworthy derivative of sulfonamides (Hager 2006). In 1939, the work of Domagk brought him the Nobel prize in Physiology or Medicine for '*the fact that a red dye-stuff, to which the name 'prontosil rubrum' was given, protected mice and rabbits against lethal doses of staphylococci and haemolytic streptococci*' (The Nobel Foundation 1965). The clinical trials showed that treatment of pneumonia with sulfa medications on humans reduced the mortality rate by between 1/2 and remarkable 6/7 and appeared to be extremely efficacious in treating bacteria-caused pneumonia (Graham *et al.* 1939; Loudon 2008). Until the development and introduction of penicillin in the late 1940s, sulfa antibiotics remained the prime cure against pneumococcal, meningococcal, gonococcal and some other infections. The vaccine against pneumococcal bacteria was developed only in the 1980s (Baker & Katz 2004).

For the purposes of this study, we compiled and reviewed several archival sources in order to identify the exact year when sulfapyridine became widely used and how it diffused into medical practice across regions in Sweden. Primarily, the articles published in the Swedish leading medical journals, such as *Läkartidningen* and *Nordisk Medicinsk Tidskrift* for 1930–1945, indicate that sulfonamides were introduced into Swedish medical practice in therapy against pneumonia in 1939. This is one of the many similar testimonials of the relative efficiency of sulfa antibiotics against pneumonia provided by the medical practitioners of the time:

> "*One should be astonished by the results achieved with this drug. Serum treatment could hardly have a major impact in practice, although it has a theoretically sound basis. Preliminary*

investigations [related to serum] take time and in case of pneumonia there is no time to lose. However, M&B 693 can be truly described as miraculous. After treatment with this drug the patient's temperature falls in a few days and he follows the recovery. Many lives have been already saved by these drugs." (Andersson 1939:133).

Similar to clinical trials in other countries (Loudon 2008), many articles documented evidence on striking reductions in the pneumonia case fatality among inpatients cured with sulfa antibiotics across Swedish hospitals (e.g. Dahlberg 1939; Hesser & Åbom 1939; Rahm 1939; SOS 1939–1941a). An explicit advice to use the sulfa drugs in medical practice thus dominates the discussion of methods to treat pneumonia beginning from 1939. Influenza is clearly recognized being not responsive to sulfapyridine, only in case it comes as a complication to pneumonia (Malmros & Wilander 1941). Accurate instructions about dosage of a sulfapyridine and its analogues to treat pneumonia among children followed (Gnosspelius 1939), and the recipe for it could be prescribed by any local doctor (Lindberg 1939). More specifically, 20 grams of sulfapyridine was needed to cure pneumonia among adults, and 1/4 of this dose was enough for an infant. The therapy by sulfa drugs was not only easy to administrate, but also cheap, amounting to 14 SEK for the full treatment course for an adult (0.4 percent of the annual labour income in industry among males, SOS 1939b) compared to 300–400 SEK that had to be spent on antiserum (Rahm 1939). In 1931 one of the important state acts, connecting the medical profession and the sickness funds, later leading to a universal health insurance system, passed (Ito 1979), and by that time the equal provision of physicians across regions had been assured. No or only a small fee was charged for a doctor's visit (4–5 SEK per visit insured in 1/4 of the cases, SOU 1948) thereby low-income families were unlikely to be deterred from the use of health care (Hort 2014).

An additional source relies on the archival materials of sulfa drug production and distribution in Sweden. Firstly, registers at local pharmaceutical companies and trade show that the imports of medical drugs raised by 50 percent (SOS1920-1950ef) and the Swedish companies began to produce the preparate analogous to the M&B 693 drug (Pyriamid, Sulphan and Septipulmon) at a mass scale in 1938–1939 (Skånes Näringslivsarkiv 2017; Rahm 1939). No restrictions existed to production because sulfa compounds were available and sulfa drugs could not be patented. Secondly and most importantly, the archival pharmacy records provided us with information on drug distribution within the country (Riksarkivet 1920–1967). At that period, the distribution of medical drugs among hospitals and local pharmacies was strictly centralized under the auspices of state authorities (SOS 1920-1940a; Sveriges Farmacevtförbund 1954; SOU 1959). Hospital records indicate the use of sulfonamides in treatment against puerperal fever and gonorrhea beginning from 1938, whereas sulfapyridine was available a year later. In September 1939, the Swedish Health Board made an inventory of all medical preparations in stock across the functioning pharmacies, and we therefore could collect information on the supply of sulfapyridine in compatible units across different regions. The pharmacy records also provided us with information about the turnover and prices of the medical drugs for the later years. As the collected data shows (Riksarkivet 1939), the supply of sulfapyridine varied throughout the country albeit was relatively even among different areas, amounted to 22 grams per 1000 mid-

year population on average (in local pharmacies) with all counties and their urban and rural areas falling within ±2 sd. Complementarily, regional pharmaceutical distribution companies in four largest cities – Stockholm (centre), Göteborg and Malmö (south), and Umeå (north) – had a surplus of sulfonamides in stock.

To the best of our knowledge, there are no empirical studies that examine the period impact of sulfa antibiotics on mortality from pneumonia and other respiratory diseases for Sweden or other countries of Europe. Descriptive studies of this kind suggest that sulfapyridine arrived to Sweden in 1939 (Hemminki & Paakkulainen 1976; Bentley 2009). Similarly, the information presented above documents that the introduction of sulfa antibiotics into the medical practice was sharp and even across the country. Figure 1, demonstrated above, indicates a clear break and convergence in regional death rates from pneumonia, bronchitis and pleurisy in the late 1930s. Despite the light decline in pneumonia mortality before introduction of sulfa antibiotics, recurrent disease outbreaks returned it to the levels of the previous periods. Beginning for the late 1930s, pneumonia mortality exhibited an irretrievable decline in both a level and a trend. The results were similar for aggregate death rates from pneumonia calculated separately for lobar pneumonia and bronchitis and country-level rates of pneumonia death rates for infants and children (SOS 1920-1950a,c). Between 1933 and 1943, mortality due to pneumonia declined primarily among infants, children under age 5, and the elderly (see Figure 2). In absolute terms, the drop is much larger for infants than for children aged 1–5.

[Figure 2]

Data

Individual-level data

This study uses individual-level outcome data from a number of administrative registers for individuals born between 1934 and 1943 in Sweden. Several registers with yearly date from 1968 to 2012 are linked through unique personal identifiers have been combined into the *Swedish Interdisciplinary Panel* (SIP), hosted at the Centre for Economic Demography (Lund University, Sweden). Some individual information is first observed even earlier, in the Swedish *Census 1960*. The SIP contains individual information on date of birth (month and year) and place of birth (county, municipality and parish). In the dataset, place of birth is accurately obtained from the parish records and could indicate either place of mother's (and child's) residence or place of child delivery, as the birth at the maternity hospital gradually became a standard in the 1930–1950 (Riksskatteverket, 1989). Such recording may not be problematic for this study as the delivery at the hospital implies close proximity to the place of mother's residence. We constructed the indicator for the region of birth as an urban or rural area of the county of birth (49 regions: 24 counties x 2 urban/rural and Stockholm), because we use an exposure variable defined at this level. The baseline analysis is conditional upon individuals having survived to the adulthood and not migrated permanently from Sweden before 1960 or before any respective starting year of the register. Of the cohorts 1930–1950, around 80 percent of one-year survivors are recorded in the database (see Appendix A).

The dataset provided abundant information on outcomes, and the paper utilized several indicators measuring labour market outcomes most precisely. For all outcomes, we stopped to follow individuals after age 60 for two reasons, namely to obtain the measure of lifetime labour earnings and to avoid capturing the plausible responses to transition to pension (Kruse 2010). In addition, the outcomes were constructed for the same age intervals for all cohorts to assure their proportionate contribution. An individual's labour income and disability pension is available from 1978 and 1981 onwards on annual basis respectively through the income and taxation register (*Inkomst- och Taxeringsregistret*). Labour income was constructed as a real mean labour income in age interval (between ages 44 until a year prior to death or age 60) and is entered into the models in a logarithmic form to avoid the disproportionate influence of the extreme values. For disability pension, we created an indicator whether an individual was on disability pension in age interval (ages 47–60).

We also utilized information available in the dataset on the mechanisms underlying the links between early-life circumstances and labour productivity later in life. We constructed the variable for education based on the *Census 1970* and the education register (*Utbildningsregistret*) available from 1990 which reports information on highest completed schooling and postschooling degree respectively. Following Fischer *et al.* (2016), we transformed these levels of education into the years of schooling. The health variables were created from the national inpatient register (*Slutenvårdregistret*) that provides information on hospital admissions, their duration and associated diagnoses for the total population from 1987 onwards. From this source and based on population at risk, we constructed the mean hospital nights per year in age interval (ages 53–60). The cause of the hospital admission is given as an ICD code that is adopted from the two revisions of the international classifications of the causes of death throughout 1987–2012 (see Appendix B). Following the previous literature (Kuh & Ben-Schlomo 2004), we classified all causes of admissions into six groups, including infectious diseases, cardiovascular diseases, diabetes, cancer, degenerative diseases of tissues and organs, and mental diseases and calculate the respective mean hospital nights. In order to measure pathology in health exclusively, we excluded hospital admissions due to external causes (2 percent of person-years) and observations with no need for further treatment (0.01 percent of person-years).

Individuals are linked to their parents through the multigenerational register (*Flergenerationsregistret*) thereby giving a unique family identifier. This information is available for all individuals in our sample conditional on their survival to the year 1991. We identified siblings as individuals having the same biological mother. Due to the availability of family links, we were able to merge socio-economic and demographic information of the family to the individual data. For individuals without family links (across different outcome samples, 91–94 percent are not linked to mothers, and 83–86 percent are not linked to both mothers and fathers) we imputed values based on average values by parental birth cohort and municipality of residence. The results by sub-groups did not differ if these individuals are omitted instead. The parental background characteristics included information on mother, such as parity, age of the mother, and family size obtained from population register (*Registret över*

totalbefolkningen) and population movement register (*Förändringsregistret*), and information on father, such as education of father and sector of employment of father (*Census 1970*). In the latter case, information was available only for the post-treatment child's ages, although for paternal cohorts education should have been completed by the child's birth and branch of work had to stabilize (de la Croix *et al.* 2008). Paternal employment in agriculture represents low income group, as the average wages in agriculture were lower compared to those in the industry or the service sector (e.g. SOS 1940b). Descriptive statistics for the estimation samples is presented in Table 2.

[Table 2]

Region-of-birth data

Demanded by the analysis, an exposure variable for pneumonia infection is obtained at the region and year of birth. For this purpose, we use region-specific mortality due to pneumonia, because morbidity data at either individual or disaggregate level are not available for the cohorts in question. To construct it, we collected annual death rates from several official statistical sources (SOS 1920–1950a-d). The data is available for the county of birth divided into urban and rural areas. The number of deaths is recorded for all ages jointly. We defined deaths due to pneumonia as a combination of deaths from pneumonia, bronchitis and pleuritis, all treatable with sulfapyridine. This is because these diseases shared symptoms and could be confused at the registration of death, as well as because of the change in the cause-of-death nomenclature occurred in 1931 (Sveriges Farmacevtförbund 1954). We further constructed death rates by dividing the number of deaths by the mid-year population in the respective region. Figure 3 displays the constructed mortality rate from pneumonia across counties in Sweden in the pre-drug period of 1932–1936. We chose this period in order to avoid potential misreporting due to nomenclature changes in preceding years and to stop before sulfonamides became internationally available. The use of other years (e.g. 1928–1930, 1931–1933 or 1934–1938) or death rates solely due to pneumonia yields results similar to ours (available upon request). The graph discerns considerable geographical variation in pneumonia death rates that ranges from 64 to 124 per 100,000 mid-year population. Similar sources provided information on other causes of death across regions deemed important for the analysis. These diseases represent a comparison group, some being pervious to sulfonamides and some not, allowing us to control for the effects of other factors (public health, medical advances and socio-economic) that may have reduced pneumonia mortality beginning from 1939. We use infectious diseases of the digestive system, such as typhoid fever and diarrhea (non-treatable), of the respiratory system, such as lung tuberculosis and influenza (non-treatable), maternal diseases mainly related to puerperal fever (treatable by sulfonamides), and degenerative diseases, such as heart disease, diabetes, and cancer (non-treatable). Influenza that was not treatable by any derivative of sulfonamides is recorded separately. That is an advantage compared to the data used in previous studies (cf. Bhalotra & Venkataramani 2015). In part because influenza commonly precedes the rise in the pneumonia epidemics it cannot be seen as a white noise component (Office of Health Economics 1963).

[Figure 3]

Additional regional-level variables were also collected from official statistical sources. They included demographic variables varying for each year, such as share of females in total population, share of disabled in total population, crude death rate, and infant mortality rate. Another set of variables included socio-economic variables and those describing public investment of different types such as real regional income per capita, share of working population engaged in agriculture, share of working population engaged in industry, medical personnel per capita, number of hospitals per capita, real spending on hospitals per capita, and schools per capita. Collected from archival pharmacy records, we add number of pharmacists per capita, a price index of medical drugs and its changes as controls. Descriptive statistics for the regional-level data is presented in Table 3.

[Table 3]

Empirical strategy

We follow the strategy previously used to identify the long-run outcomes of exposure to certain infectious diseases targeted by nationwide rapid interventions (Acemoglu & Johnson 2007; Bleakley 2007; Cutler *et al.* 2010; Lucas 2010; Venkataramani 2012; Bhalotra & Venkataramani 2013, 2015). In order to investigate the impact of infant health on the later-life outcomes, this paper exploits the plausibly exogenous variation due to the decrease in mortality from pneumonia in an individual's year of birth induced by sharp arrival of sulfa antibiotics in Sweden in 1939. It obtains the effect of medical innovation using two sources of variation, including the relatively larger benefits of individuals born in regions that experienced higher exposure to pneumonia disease before the introduction of sulfa antibiotics compared to individuals born in regions with lower exposure, and varying exposure of different cohorts to the arrival of antibiotics. Following the recent early-life literature (Almond & Currie 2011), we limited the sample to children born in 1934–1943, and therefore those born before 1939 were at first time treated by sulfa antibiotics in ages 1–5 and those born in 1939 and later – in infancy. Inclusion of more cohorts (e.g. 1931–1946) yielded similar results (available upon request), and cohorts born in 1947 and later were likely to have benefited from the introduction of penicillin that was efficient against all known infectious diseases.

Specifically, the paper estimates the following model:

$$y_{icb} = \alpha + \beta post_b \cdot BaseRate_c + \delta_c + \lambda_b + X_i + \varepsilon_{icb} \quad (1)$$

where y_{icb} is the outcome (years of schooling, mean number of hospital admissions, on working disability pension, and natural logarithm of labour income) for individual i born in region c in year b; $post_b$ is a dummy turning one if an individual was born in 1939–1943, zero if an individual was born in 1934–1938; $BaseRate_c$ is the pre-intervention mortality rate from pneumonia in an individual's region of birth c; X_i is the vector of individual-level characteristics; δ_c and λ_b are region- and year of birth fixed effects. By using $post_b$X$BaseRate_c$, the model exploited the cohort-region intervention intensity. An individual-level exposure to

pneumonia infection is not available, and the geographical mortality rate approximates both individual and aggregate exposure to pneumonia. The parameter β in eq.1 captures the effect of intervention, and if intervention-led decrease in pneumonia in year of birth generated beneficial outcomes in adulthood, we expect to find positive relationships. Pre-treatment mortality rate from pneumonia is a five-year average of pneumonia mortality rate (pneumonia, bronchitis and pleuritis) for years 1932–1936 obtained separately for each region. Baseline individual-level characteristics comprise sex, utilized to control for sex-specific health and labour market trajectories, and several other characteristics (parity, age of the mother, family size, education of father, and sector of employment of father) are added in other specifications. For this and other specifications, following the previous literature pointing to the plausibly gender-specific outcomes of exposure to respiratory disease in early life (Kuh & Ben-Schlomo 2004), we present the results for sexes both jointly and separately.

Equation 1 could be seen as the reduced form of the following system (presented schematically):

Baseline pneumonia mortality ⇨ Utilization of sulfapyridine ⇨ Reduction in pneumonia mortality/ ⇨ Later-life outcomes (2)

We therefore replace the reduced form with the 2SLS form, where utilization of sulfapyridine or cohort-region-of-birth death rate (due to pneumonia or among infants) is instrumented with $post_b$X$BaseRate_c$. In doing so, by rescaling the reduced form effects by the first stage effects, we receive evidence for the quantitative impact of exposure to pneumonia in infancy along the particular steps in Eq.2.

The identifying assumption for the model employed in this paper is that the instrument ($post_b$X$BaseRate_c$) does not correlate with the error term in the second-stage equation. To put it differently, there should be no omitted region-of-birthXyear-of-birth variables that correlate with both the long-term outcomes and the instrument (exclusion restriction). Because an indicator $post_b$ turns on for all regions of birth at the same year of birth (1939), the quicker or slower introduction of sulfa antibiotics into medical practice in the regions will not have an effect on the instrument. The main plausible threat to the identifying assumption emerges from any correlation between baseline pneumonia death rates or convergence in pneumonia death rates and potential long-term outcomes, that is they would have developed absent introduction of sulfapyridine. For instance, if introduction of sulfapyridine as treatment against pneumonia – intentionally or coincidentally with other programs or pre-treatment cross-regional convergence – targeted the regions of births that were expected to have beneficial later-life outcomes for the affected cohorts, this assumption is violated.

This paper addresses the potential threat to identification in several ways. First, we investigate empirically the dependence of sulfapyridine availability in 1939 on the socio-economic region-of-birth characteristics. Next, in one of the specifications, the model additionally introduces a set of baseline death rates from the most substantial infectious and degenerative diseases

(described above) in the period under analysis interacted with the post-reform indicator and could be thought as placebo. Such diseases, some affecting children and some adults, could not be treated by sulfa antibiotics, but shared similar risk factors with pneumonia, related to general living conditions, general health care system and targeted public health initiatives. Maternal mortality, mainly due to puerperal fever that was treatable by sulfa antibiotics, was added to control for abrupt region-specific fertility changes. Adding a larger set of diseases did not affect the results. Also, similar omitted variables were controlled for by adding several region-specific socio-economic, health care and demographic variables varying by cohort (described above). Introduction of the pre-treatment averages of these variables interacted with cohort dummies or $post_b$ indicator provided similar results. Also, we directly measured the plausible confounding of the convergence in pneumonia mortality with other health and educational reforms implemented in the period under analysis. Moreover, we control for pre-existing differential trends across regions by allowing for the region-specific linear (quadratic) time trends. Furthermore, additional models account for mean reversion across regions by incorporating the interaction of $post_b$ with the region-of-birth-specific average of the dependent variable in the pre-treatment period.

We further estimate the event-study specification where the compound measure for all affected cohorts 1939–1943 is replaced with the cohort-specific impact of the pre-treatment pneumonia death rates on later-life outcomes. It allows us to explore whether the long-term effects due to the arrival of sulfapyridine emerge for individuals exposed in infancy solely or besides affected other age groups (not older than the age 5). We run the model while substituting the sample of unaffected cohorts, aged 1–5 in 1939 (born in 1934–1938), with those who were aged 10–14 in 1939 (born in 1925–1929), and the results remain qualitatively similar (available upon request). The event study analysis additionally examines the existence of mean-reverting shocks or pre-treatment differences in outcomes. The model is estimated as follows:

$$y_{icb} = \alpha + \sum_b \beta_b BaseRate_c + \delta_c + \lambda_b + X_{icb} + \varepsilon_{icb} \quad (3)$$

where β_b denotes the cohort-specific impact of the pre-treatment pneumonia death rates on later-life outcomes. To pass the check, the arrival of the sulfa antibiotics should show up as a shift in the outcomes for the cohorts born in 1939–1943.

The additional empirical strategy is to account for the parental background characteristics. The parental characteristics from regions with high pretreatment pneumonia death rates might be elevated thereby violating the long-term effects. It is also plausible that non-random migration changes the composition of parents in the region over time. Intervention-led selective migration of high capable families, whose children supposedly have higher capabilities, to the regions with previously high pneumonia exposure can overestimate the true effect of intervention. The maternal characteristics should be also included to control for maternal fertility. Maternal fertility could be affected though several channels, including maternal or family responses to sharp decrease in pneumonia and better infant health (due to sulfapyridine) and improvements in maternal health at childbirth and newborn health at birth (due to sulfonamides). The models

already introduce interaction between intervention indicator and baseline death rates from diseases at childbirth. To address selection issues further, we first control for observable background characteristics, including parity, age of the mother, family size, education of father, and sector of employment of father. Secondly, the eq.1 was estimated adding mother fixed-effects. Mother fixed effects were preferred to mother-and-father fixed effects as the sample of siblings born to the same mother is more comparable to the baseline sample. The region fixed effects are identified for families that report different regions of birth for their children. Therefore, comparisons identifying the long-term effects of exogenous convergence in pneumonia mortality due to arrival of sulfapyridine are made only between siblings. We additionally separated the sample by parental backgrounds and maternal health. This provides a consistency check as the larger effects are expected for individuals with disadvantaged backgrounds due to the higher exposure to pneumonia in infancy.

Results

Impact of the introduction of sulfa antibiotics on mortality

Figure 4 presents the effects of arrival of sulfa antibiotics on mortality at the aggregate level. The results indicate strong convergence in aggregate mortality rates from pneumonia after arrival of sulfa antibiotics. More specifically, one unit higher pneumonia death rate in 1932–1936 is associated with 0.6 unit lower pneumonia death rate in 1943. The results are presented for regions of birth (county x urban/rural) as a geographical unit, and look identical for the county-of-birth level. It should be noted that the effects are likely to be underestimated as the decrease both in morbidity from pneumonia and in mortality/morbidity from other infectious diseases benefited due to such decrease are not taken into account. Data on morbidity is not available for the region-of-birth level, and the approach prevailing in the empirical literature relies on the mortality rates (cf. Bleakley 2010).

[Figure 4]

We can further investigate whether the breaks in pneumonia mortality occurred in year 1939 using formal tests. As noted above, Figure 1 provides visual evidence that mortality declined substantially, accelerated and never returned to its previous levels after arrival of sulfa medicaments in 1939. The results in both level and logarithmic terms are presented in Table 4. Columns 1 and 2 support that there are both level and trend breaks in pneumonia death rate in that year. More specifically, pneumonia mortality dropped beginning from 1939 by around 34 percent and exhibited each year on average an additional decline by 14 percent. The beneficial effects of sulfa introduction could be also detected in crude death rate (Columns 3 and 4) and infant mortality rate (Columns 5 and 6), which from 1939 followed accelerated decrease by 5 percent annually each. We re-estimated the presence of the breaks in both absolute and logarithmic terms using a difference-in-difference approach, where both infectious (Columns 7 and 8) and degenerative diseases (Columns 9 and 10) are added as control diseases interacted with after-intervention period. The size of the decreases and acceleration in

pneumonia mortality from 1939 until 1943 is similar to the one provided in Table 4. The results are also similar for the longer periods (not shown here).

[Table 4]

Main results

We start by descriptively analyzing the difference in the later-life outcomes between the treated and untreated cohorts (that is treated in infancy or ages 1 to 5) for each region of birth. Figure 5 presents the results for the related outcomes under study, including the fraction on working disability, ln labour income, completed years of schooling and mean hospital nights per year. In the graphs, absolute change in the outcome between cohorts is plotted against region-of-birth baseline pneumonia rates. As shown, consistent with our expectations, regions of births with higher baseline pneumonia exposure exhibit larger improvements in all adult outcomes.

[Figure 5]

The following analytical sections present the results from matching exposure to childhood pneumonia, defined at a region and year of birth level, to individual outcomes parametrically, in order of Eq.1-3 to the models with family-level controls and mother fixed effects.

The results from the baseline specification (the reduced-form, ITT effects) for individual-level outcomes under study, both sexes jointly and separately, are presented in Table 5. They suggest statistically significant beneficial impacts of reduced exposure to pneumonia in year of birth, due to the arrival of sulfa drugs, on probability of receiving disability pension, ln labour income, completed years of schooling and mean hospital nights per year, each observed in late adulthood. The magnitude of the effects can be grasped by comparing individuals born in a high and low pneumonic regions, such as region in the 95th percentile of the pre-intervention pneumonia mortality distribution (1.243 deaths per 1000 population) with those born in the 5th (0.642 deaths per 1000 population, and subsequent decrease by 0.455 deaths per 1000 population; overall range is 1.243–0.642=0.601 deaths). The respective sizes for the outcomes of all sexes jointly are the following: probability of receiving disability pension – 1.7 percentage point decrease (4.9 percent of pre-mean value of the outcome); ln labour income – 4.6 percent increase; years of schooling – 0.159 years increase (1.7 percent of pre-mean); and mean hospital nights per year – 0.045 nights decrease (5.8 percent of pre-mean). The results for males and females separately are fairly equal and not statistically different from each other (this difference is estimated in a pooled sample, not shown here).

[Table 5]

Table 6 presents the results for the 2SLS specification of the baseline specifications according to Eq.2. In the table we present the results for the effects of exposure to pneumonia (approximated with pneumonia mortality in Panel A and infant mortality in Panel B) and access to sulfapyridine (Panel C) in infancy, instrumented with the intervention indicator, on later-life outcomes. The 2SLS results give the average effects for individuals for different treatment groups (LATE effects), including those whose mortality from pneumonia or infant mortality

has declined after the arrival of sulfapyridine or who enjoyed greater access to sulfapyridine. The 2SLS results are all statistically significant, at least at 5 percent significance level. The magnitude of the effects is now larger, primarily because the reduced form effects are now rescaled with first stage effects, implying the multiplying the former by a factor of 1.87(1/0.536) for pneumonia mortality, by a factor of 1.060(1/0.943) for infant mortality, and by a factor of 2.78(1/0.360) for sulfapyridine availability. Also importantly for interpretation, the intermediate variables have sizable distribution across different regions of birth implying even a greater range from 95th to 5th percentile (0.948 deaths per 1000 population for pneumonia mortality; 3.536 infant deaths per 100 live births; 6.326 grams per 100 mid-year population for sulfapyridine availability) and a larger fraction of regional convergence explained. Please observe the change in the sign for the coefficients for pneumonia and infant mortality, compared to reduced form effects, as now exposure to infection in year of birth is expected to be negatively associated with later-life outcomes. Similarly to the reduced form effects, the availability of sulfapyridine is positively associated with benefits later in life. These effects are statistically similar to those if to use cohort-varying sulfapyridine availability, defined based on its stock in 1939 and the relative changes in turnover of drugs for successive years (available upon request).

[Table 6]

Robustness analyses

We start the robustness analysis by examining whether regional distribution of sulfapyridine was related to socio-economic characteristics of the regions. As for the variables approximating utilization of drugs, we employ several ones, including sulfapyridine availability (in grams of sulfapyridine per 100 mid-year population) in 1939, average price of a medical drug in 1939 and its change between 1939 and 1940. Table 7 shows the estimates for the effects of different socio-economic variables, such as ln real regional income per 1000, share of employed in agriculture, share of employed in industry, ln medical personnel per 1000, ln number of hospitals per 1000, ln real spending on hospitals per 1000, ln pharmacists per 100,000, share of females, ln number of schools per 1000, crude death rate, and share of disabled, on measures of sulfa drug utilization. As it is evident, in general there is no systematic association between sulfa drug availability and their prices with regional socio-economic variables.

[Table 7]

Next, we proceed to incorporating different region-of-birth level controls directly into the reduced-form models, and show the results in Table 8. The inclusion of either controls for the breaks in mortality rates in other diseases than pneumonia (Column 1), or region-specific cohort-varying socio-economic, health care and demographic characteristics (Column 2) keeps all results statistically significant and unchanged compared to the baseline specification. If region-of-birth-specific linear trends are added (Column 3), the effects for all outcomes, except for years of education, slightly change in magnitude albeit stay statistically significant, at least at 10 percent level. The effect for the educational variable for one standard deviation change in pre-treatment pneumonia (0.138) reduces from 0.048 to 0.015 years, although it consistently

remains statistically significant for females. Table 9 further presents results from the robustness test to mean reversion. The results provide mixed evidence of mean reversion in the sample, although the incorporation of mean-reverting controls does not affect the main results.

[Table 8 and 9]

Table 10 demonstrates the estimates for the specifications which include parental background characteristics and mother fixed effects. Neither of these checks violates the main findings. Controlling for observable father's and mother's characteristics even improves the estimates, both in terms of the magnitude and their statistical significance (Column 1). We use mothers to obtain the family identifying information, and estimates in Column 2 for the baseline specification shows that, in terms of the effects, the sample with known mothers is not different from the full sample. The within-sibling comparison (Column 3) confirms the findings for all outcomes in the cross-section. Moreover, the magnitude of the estimates in the majority of the outcomes, such as probability of receiving disability pension, natural logarithm of labour income, and years of schooling, become larger suggesting that parents reinforced the changes in the child's health endowments. The coefficient for mean hospital nights per year becomes marginally insignificant although its size is identical to other specifications.

[Table 10]

Figure 6 demonstrates the main results of the paper derived from the event study specifications. The figure presents the parameter estimates obtained from replacing the interaction term $post_b X BaseRate_c$ with a separate interaction term for each cohort. To improve precision, we combine two neighboring cohorts into one group. According to the a-priori expectations, for the pre-reform cohorts, prior to 1938/1939, coefficients fluctuate around zero and never attain statistical significance. Starting from 1938/1939 birth cohort until the last studied cohort, coefficients change rapidly indicating beneficial impact of the introduction of sulfa antibiotics on all outcomes under study. These findings are consistent with pneumonia affecting labour market outcomes, health and schooling through its influence on individuals in their infancy. They also show that, conditional on region-of-birth characteristics, there are no pre-treatment differential cohort trends in the outcomes.

[Figure 6]

Mechanisms

To explore biological mechanisms linking early-life exposure to pneumonia to later-life outcomes, we present the reduced-form estimates of the effects of of sulfa antibiotics efficient against pneumonia on mean hospital nights per year, by cause of admission, in Table 11. The estimates attain expected signs for all cause-specific outcomes. However, statistically significant and sizable effects emerge from hospitalizations due to infectious, cardiovascular diseases, and degenerative diseases not specified in other groups. The magnitudes of the respective effects, by comparing individuals born in a region in the 95th percentile of the pre-intervention pneumonia mortality with those born in the 5th (0.455 deaths per 1000 population), can be presented as follows: infectious diseases and pneumonia – 0.012 mean

hospital nights per year decrease (9.7 percent of pre-mean); cardiovascular diseases – 0.055 mean hospital nights per year decrease (9.1 percent of pre-mean); and degenerative diseases – 0.105 mean hospital nights per year decrease (8.8 percent of pre-mean). The effects in a degenerative group are explained by the effects in hospitalizations due to symptoms of respiratory systems, and arthritis. The effects are equal between the sexes for almost all outcomes, except for somewhat stronger beneficial effects for males with regard to hospitalization due to cardiovascular diseases and stronger beneficial effects for females in hospitalizations due to diabetes (not shown here).

[Table 11]

Regarding economic mechanisms, we study the contribution of quantity and returns to an individual's schooling to labour income gains. To do this, we rerun the model while including years of schooling. The models are equivalent to Eq.1 while adding region-level varying controls and presented in Table 12 for sexes both jointly and separately. The results reported in Table 12 are equivalent to those incorporating a squared term for an individual's working experience (a Mincer-type equation). While keeping in mind that schooling rose in response to the intervention, the inclusion of schooling accords with our expectation and leads to a decrease in the pneumonia exposure coefficient for both the natural logarithm of labour income and the probability of working disability. In relative terms, the results suggest that the increase in years of schooling accounts for 21–34 percent of the labour productivity results, with no clear differences between sexes. We can further investigate whether returns to schooling increased in response to medical intervention. This can be done by interacting the pneumonia exposure term with education variable, while adding respective interactions of education with other terms in the models. The coefficient for triple interaction ($post_b X BaseRate_c X schooling$) is small and never statistically significant (not shown here). The rest of the labour productivity increases (66–79 percent) can be therefore attributed to the direct effect of general and cognitive health.

[Table 12]

Heterogeneous effects

Table 13 presents the reduced-from results from subsamples distinguished based on parental socio-economic and health characteristics. Pneumonia was more prevalent among disadvantaged families and the intervention effects should be larger for them. The results are consistent in finding that individuals from these families gain more from reduction in pneumonia mortality later in life. Children born to mothers aged above the average age in the sample and probably less healthy attain the treatment effects of larger magnitude (Column 1). In addition, as expected, the treatment effects for the individuals born to families with poor socio-economic background, such as less educated fathers (Column 2) or fathers working in agriculture (Column 3), indicate systematically stronger magnitudes. For instance, in a 95/5 percentile fashion of the presentation of the results, a decrease in in pre-treatment pneumonia mortality rate by 0.455 deaths per 1000 population leads to a 0.023 percentage point decrease in the probability of disability (6.7 percent of pre-mean) for individuals born to fathers with less

than primary schooling. In contrast, it leads to a 0.015 percentage point decrease in probability of disability (4.3 percent of pre-mean) for individuals born to fathers with more schooling. The statistical difference between the coefficients is estimated in the pooled samples, in which dummy for a certain sub-group is interacted with all terms in the specification (not shown here).

[Table 13]

Additional robustness analyses

Our analyses shows that the effects of exposure to pneumonia and its sharp reduction due to the nationwide arrival of sulfa antibiotics are marginally sensitive to the inclusion of various region-of-birth and parental controls. The estimates including family fixed effects are even larger, in part suggesting that selective migration across regions or fertility that changed in response to the introduction of drugs against pneumonia does not affect the main findings. In fact, families reporting different region of birth for their children after the intervention have worse outcomes compared to the children of stayers. It implies that the reasons other than better prospective health in places that advanced more after arrival of sulfapyridine determined the migration of parents between childbirth, for instance the structure of the local labour force (Enflo *et al*. 2014). Similar to other studies looking at the long-term survival of cohorts treated by different socio-economic conditions in childhood (cf. Zajacova & Burgard 2013), the bias related to selective mortality is likely to be downward in our case, as the weakest members of cohorts are more likely to survive in the after-drug period and observed in the registers. To assess it formally, we apply a two-stage Heckman selection procedure to analyze whether a selective survival affects our estimates (Heckman 1979). In the first stage, the probability of being observed in the estimation sample is modelled as a function of cohort fixed effects, region of birth fixed effects and sex for all individuals whom we observe as early as in the year 1960 (1960 Census) in a probit model. An inverse Mills' ratio originating for each individual from the estimates of the probit model is further included as a covariate into the baseline specification, and this procedure does not affect our main findings (see Appendix C).

The main results of the paper are unlikely to be explained by other programs that overlapped with introduction of sulfapyridine. The confounding by the compulsory schooling reforms that were also coordinated with child labour laws do not appear to be problematic for our estimates, as the children in the estimation samples in the overwhelming majority were exposed to the same reforms after age 5. The introduction of seventh compulsory grade has been completed for the studied cohorts, with the exception of several municipalities (out of 949) that introduced the reform in 1934–1936 (Fischer *et al.* 2013). Similarly, an introduction of a nine-year comprehensive school did hardly affect the cohorts under analysis, although some forerunning municipalities, which had such system in place early, did implement it fully in 1943 (Homlund 2008). Nevertheless, we rerun the models while excluding these municipalities of birth (less than 1 percent of population) treated by the changes in the compulsory schooling and the results remain unchanged (not shown here). Besides the

compulsory schooling reforms, up to 1950, institutions of secondary schooling and vocational training gradually began to educate pupils of both sexes (Ljungberg & Nilsson 2009). Such process was, however, very smooth and demand-driven. The plausible effects from this educational development is thus likely to be controlled by several socio-economic and demographic region-of-birth characteristics that we added to the models.

The arrival of sulfa antibiotics overlapped with two other public health reforms. One reform was related to the rollout of the government support to maternal and child health in 1937 until its full nationwide coverage in 1960 (Ström 1946). Based on the official statistical sources (SOS 1937–1945a,c), we collected information on the coverage of infant population in the regions of birth by this program. The correlation between the region-of-birth pneumonia pre-treatment mortality and proportion of infants enrolled in the institutional care activities in 1938–1943 is –0.055 (p-value is 0.708), indicating unlikely influence of their effects on our results. Another institutional change has been occurring with regard to the gradual expansion of the hospital births in 1920–1950 (Wisselgren 2005; Vallgårda 1996). It is not problematic in case this expansion occurred gradually across the cohorts in a manner unrelated to pre-treatment mortality, only its acceleration for the cohorts treated by sulfa antibiotics could potentially violate our results. Based on the same statistical sources, we collected information on the regional proportions of hospital deliveries in total. The correlation between the rate of change in these fractions in 1938–1943 and the region-of-birth pneumonia pre-treatment mortality is 0.0215 (p-value is 0.883), that is too weak to affect our results. One potential concern is whether the use of place of birth, which in the period under analysis might record either place of mother's residence or hospital location, reflects true pneumonia exposure in the pre-intervention period. As a robustness check, we use the counties of birth instead and assign the respective values for the regional disease burden, which give the estimates lightly larger, especially for the specification with region-specific linear/quadratic time trends, but not statistically different from our main results.

[Table 14]

Additionally, we test the robustness of our results to the region-specific influence of the WWII. During the war, Sweden was neutral otherwise there were problems with supply of food and fuels (Wangel 1982). Studies looking at the impact of food shortage on either childhood anthropometric measures, such as birth weight, height and BMI (Abolins 1962; Angell-Andersen *et al.* 2004), or in female labour force participation (Gustafsson & Jacobsson 1985), by comparing the surrounding cohorts, do not find any differences. In our case, any potential country-level changes in child health due to food shortage are ruled out by the birth cohort fixed effects. Differences in price changes of basic products in 1930s-1940s were also indicated across regions of Sweden; food prices increased more considerably in central parts compared to the rest (SOS 1931-1959f). Based on the official statistical sources, we collected the regional prices indices for main food products for years 1934–1943 (SOS 1931-1959f) and added them into the specification. Our results stay unaffected by this check (see Appendix D).

We have shown that results are present for different approximations of health, education and

labour productivity available in the dataset (ever at hospital, total hospital admissions, more than secondary schooling, tertiary degree, ln total income, ln family income, employed). In addition to the morbidity outcome, we run the models for mortality in ages 34–60 and detect no systematic treatment effects on mortality for the cohorts under study (see Appendix E). We also perform the same analysis for mortality by cause of death (see Appendix F). The results point to the beneficial effect of reduced pneumonia exposure in infancy on probability of dying from cardiovascular disease, although it does not attain statistical significance in many specifications. The effects on cardiovascular mortality become more evident if to follow individuals until age 69 (not shown here).

Discussion and conclusions

In recent years, the literature showing that early life circumstances predict health, education and socio-economic status later in life has grown substantially. In particular, it has been shown that disease environment in younger ages, especially in infancy, shapes income growth in the long run across countries and individuals (Weil 2014; Bleakley 2010). The majority of the related causal studies use abrupt infectious shocks, such as epidemics or disease outbreaks, as a source of exogenous variation in infant health (e.g. Barecca 2010). Another emerging line of research combines infectious disease exposure in childhood with public efforts to eliminate it (e.g. Bleakley 2007). Our study contributes to this line of literature by studying the effects of exposure to pneumonia in infancy, reduced by a sudden introduction of sulfa antibiotics in Sweden, on labour productivity in late adulthood. Our findings suggest the following: (1) decrease in exposure to pneumonia in infancy led to gains in labour productivity both in terms of entry to the labour market and labour intensity; (2) it increased an individual's health stock substantially, which productivity also accounts for the bulk of the income improvements, whereas responsive increases in human capital stock are small; (3) the effects of reduced exposure to pneumonia infection on productivity are higher for individuals raised in disadvantaged families.

Our results indicate that arrival of sulfa antibiotics as treatment against pneumonia among children had beneficial effects, although it is important to provide their interpretation comparable to other studies. We have already presented the magnitudes of the coefficients in terms of the differences between the most and least (95th and 5th percentiles) pneumonic regions of birth in Sweden obtained from the reduced-from model (ITT effects). To put the results differently, dependent on specification, arrival of sulfa antibiotics efficient in reducing pneumonia exposure in infancy helped to reduce the pre-drug gap in outcomes between the respective regional groups within the following ranges: probability of working disability – 14–29 percent (95/5 difference in outcome in 1934–1938 is 0.098); ln labour income – 15–26 percent (95/5 difference in outcome in 1934–1938 is -0.223); years of schooling – 3–14 percent (95/5 difference in outcome in 1934–1938 is -1.619); mean hospital nights per year – 21–29 percent (95/5 difference in outcome in 1934–1938 is 0.163). Another way to interpret the results is to relate them to the changes in exposure to disease, approximated with either

pneumonia or infant mortality rates, due to introduction of sulfapyridine into medical practice or directly to drug availability. We have reported the related results from the instrumented variables model, where intervention serves as an instrument to exposure to pneumonia and access to sulfapyridine in their effects on labour productivity (LATE effects). Both approaches applied in this paper provide effects similar to those demonstrated in other micro-level studies (cf. Bleakley 2010) and together with those are larger compared to the macroeconomic studies (cf. Weil 2014).

The above results are plausibly underestimated as arrival of sulfa antibiotics reduced not only mortality, but also morbidity due to pneumonia, as well as reduction in pneumonia among children has been more rapid compared to that among total population. Consequently, one can relate outcome effects to the probability of childhood infection thereby obtaining the treatment effects for those who got infected by pneumonia and were treated by sulfa antibiotics (LATE effects). If to use pneumonia incidence rate 15 per 100 children (Lindborg 1936), we arrive at the following ranges of results from different reduced-from specifications coming from one standard deviation (0.138) decrease in pneumonia mortality: 0.028–0.057 unit decrease in probability of working disability (8.2–16.5 percent of pre-mean), 6.7–11.7 percent increase in ln labour income, 0.101–0.450 years increase in schooling (1.1–4.9 percent of pre-mean), and 0.068–0.096 units decrease in mean hospital nights per year (8.8–12.4 percent of pre-mean). In this fashion, our results are substantial and identical to those found in the study of the long-run impact of sulfa antibiotics on the males in the US by Bhalotra and Venkataramani (2015).

Additionally, we can interpret the results adding an intertemporal viewpoint. Taken roughly from the data, the growth in labour income was rather stable for the cohorts born in 1934–1938 amounting to 6.2 percent increase in total, and it increased up to 11.5 percent for the cohorts 1939–1943 (see Appendix G). If one relates this number to our reduced form estimates (one sd higher baseline pneumonia (0.138) is responsible for 1.0–1.7 percent increase in labour income, and the mean and the range of baseline pneumonia is 1.038 and 0.601 respectively), it is clear that – absent the introduction of sulfa antibiotics – the income growth continued to develop only at the pre-drug rate. Similar conclusions could be drawn for health and human capital stocks. Schooling increased for the treated cohorts by 0.188 years compared to the untreated ones (one sd higher baseline pneumonia is responsible for 0.015–0.067 years increase in schooling), and hospitalizations decreased by 0.035 mean hospital nights between the treatment cohorts (one sd higher baseline pneumonia is responsible for 0.010–0.014 years increase in mean hospital nights per year). For both mechanisms, had sulfapyridine not arrived, the cohorts born after 1938 would have enjoyed gains in education and health in size equivalent only to those experienced by their older counterparts and not higher.

The findings of the current paper stress the importance of the context for realization of the full potential of the acquired early-life benefits. In the 1920s–1930s, like worldwide today, pneumonia was mainly caused by the *Streptococcus pneumoniae* bacteria (Blasi *et al.* 2007), and the pattern of pneumonia mortality was strikingly similar across nations (Mercer 1990). It

is thus anticipated that the results of our paper align well with those reported for the US cohorts (Bhalotra & Venkataramani 2015). An important difference is that in their case, due to the presence of institutions impeding the effects to realize, more disadvantaged population groups benefit less. In contrast, beginning from the 1960s, the labour market in Sweden did not only favour females due to publicly provided childcare (Hort 2014) and expansion in the married women's labour force participation (Stanfors 2007), but also matched individuals according to their abilities regardless of the socio-economic origin (Breen & Jonsson 2005). In line with it, our paper demonstrates the benefits of the medical intervention for each child born after arrival of sulfa antibiotics, which are especially large for children from disadvantaged families. There are no socio-economic differences in utilization of sulfa drugs, and therefore these heterogeneous effects stem from the differences in treatment. To put our findings differently, while exposure to pneumonia infection in infancy primarily hit the poor families, their children translated this disadvantage to own adulthood. Such a process has been consistent with the development of increasing socio-economic and health disparities beginning from the mid-twentieth century Sweden (Mackenbach 2008; Keeley 2015).

Our results clearly point to certain biological mechanisms behind the influence of early-life conditions on later-life wellbeing. The existing explanations emphasize exposure to infectious diseases, malnutrition or socio-economic conditions in general as important early-life environmental factors. Our study disentangles this mechanism by finding that exposure to pneumonia in the year of birth leads to depletion of adult general health. Additionally, consistent with the recent epidemiological literature (e.g. Muscher *et al.* 2007; Colebatch & Edwards 2010; Carraro *et al.* 2014), we find the most discernible effects of exposure to pneumonia in infancy on development of infectious disease/pneumonia, cardiovascular disease, and arthritis. Our results additionally show that acquired schooling might be affected by early-life pneumonia conditions, which is in line with literature showing how early-life exposure to infection affects the development of brain in the long run (Landrigan 2005). While we detect sizable effects on morbidity, there are no clear effects on mortality. For the cohorts under analysis, mortality was rather low and declining slowly in ages 34–60 (7.6 percent of population at risk died). Otherwise, in accordance with the development of specific chronic diseases due to early-life conditions (Gluckman et al. 2008; Sun *et al.* 2013), there is a light indication that reduction in pneumonia exposure in infancy led to decrease in probability of dying from cardiovascular diseases. One should presumably follow our cohorts to old age to detect such effects on mortality consistently, while until age 60 the treated cohorts demonstrate benefits only in sickness and disability.

We find that the economic pathway from early childhood conditions to labour productivity is largely determined by the productivity of general and cognitive health rather than that of education. In responses to reduction in pneumonia infection as infants, individuals enjoyed higher incomes and better health in adulthood, and the treatment effect on years of schooling is relatively small and not robust across different specifications. Bleakley (2010) points to the economic theory behind it, reminding that increase in quantity of schooling is not an important channel through which infant health affected incomes rather being a direct effect of health.

Consistent with it and with evidence from previous empirical studies (Bleakley 2007; Cutler *et al.* 2010), we show that increases in health accounts for at least two thirds of the gains in labour income, and the rest is attributed to schooling. One should admit that quality of schooling should be measured in addition to its quantity. Unfortunately, we are limited in the data of this kind and thus open up the opportunity for the future research. In doing this, it is important to keep in mind the note from the previous studies pointing to the first-order importance of health improvements compared to schooling in driving income benefits regardless of what indicators of the latter are used.

The findings of this paper give rise to direct policy implications that extend far beyond the populations under study. Our findings on the gains in labour productivity are substantial. We conclude that the investments in provision of antibiotics against pneumonia among infants, which costed 150 SEK (in SEK 2016) per treatment case, likely yielded high societal returns. The findings have relevance for middle- and low-income countries, where the majority of early child deaths occur due to pneumonia (0.9 mln deaths annually equivalent to 16 percent of all deaths under age 5), diarrhea and health problems during the first month of life which could be prevented or treated with access to simple, affordable interventions (WHO 2013, 2016). The fact that poor households suffer more severely from these conditions strengthens the importance of our findings even further. Pneumonia infection is a leading morbidity cause also in developed countries, especially in children under age five (2.6 million cases annually equivalent to 29 percent of all morbidity episodes under age 5, Rudan *et al.* 2013), including in Sweden (Socialstyrelsen 2015), and growing antimicrobial resistance raises new challenges to public action (WHO 2014). This study highlights that improvements in disease conditions in early life with publicly provided medications and interventions are essential not only in reducing the current burden of disease, but also in promoting life expectancy and income growth and in leading to convergence in health and economic disparities in the long run.

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TABLES

Table 1 – *Composition of the causes of death in Sweden, percent, 1920–1950*

	1920		1930		1940		1950	
	age<1	ages 1-5	age<1	ages 1-5	age<1	ages 1-5	age<1	ages 1-5
malformations	43	2	52	4	59	3	76	8
infectious	12	45	8	30	6	24	3	7
pneumonia and influenza	20	25	20	26	21	24	11	22
diarrheal	10	5	9	9	5	9	5	9
degenerative	5	11	6	18	5	21	4	24
other	10	12	6	14	3	21	2	30
total	100	100	100	100	100	100	100	100

Source: SOS 1920–1950c

Table 2 – *Summary statistics for estimation samples, individual-level data, cohorts 1934–1943*

	All	N	Males	N	Females	N
Outcomes						
Years of schooling	9.584(2.626)	879,175	9.569 (2.799)	446,736	9.600(2.436)	432,439
Mean hospital nights per year, ages 53–60	0.687(2.139)	852,460	0.693(2.123)	430,096	0.681(2.157)	422,364
On disability pension, ages 47–60	0.337(0.473)	872,917	0.299(0.458)	442,926	0.378(0.485)	429,991
Ln labour income, ages 44–60	8.133(1.399)	878,606	8.354(1.358)	446,511	7.904(1.404)	432,095
Family-level control variables						
First parity child	0.561(0.496)					
Second and higher parity child	0.438(0.496)					
One child family	0.290(0.453)					
More than two children family	0.623(0.484)					
Father only primary schooling	0.627(0.483)					
Father more than primary schooling	0.372(0.483)					
Father working in agriculture	0.642(0.479)					
Father working in industry/service	0.357(0.479)					
Mother young (age < 29)	0.497(0.499)					
Mother old (age >=29)	0.503(0.499)					

Note: SIP. Means and standard deviations (in parentheses). Family-level control variables are provided for mortality sample.

Table 3 – *Summary statistics for region-of-birth data*

Variable	Means(sd)
Pre-treatment mortality rates, per 1000, 1932-1936	
Pneumonia	1.035(0.138)
Typhoid fever	0.132(0.018)
Diarrhea	0.057(0.022)
Lung tuberculosis	0.863(0.236)
Influenza	0.089(0.031)
Heart disease	2.731(0.351)
Diabetes	0.124(0.030)
Cancer	1.450(0.174)
Maternal (childbirth)	0.046(0.018)
Region-of-birth and cohort-level control variables, 1934-1943	
Ln real regional income, per 1000	13.083(0.480)
Share employed in agriculture	0.235(0.212)
Share employed in industry	0.461(0.115)
Ln medical personnel, per 1000	0.146 (0.419)
Ln number of hospitals, per 100,000	1.159(0.864)
Ln real expenditures on public health, per 1000	12.703(10.099)
Ln number of pharmacists, per 100,000	3.109(0.948)
Sulfapyridine availability in 1939, per 1000	2.181(2.317)
Price index of medical drugs	1.716(0.211)
Change of price index of medical drug	0.496(0.917)
Ln number of schools, per 1000	1.700(1.043)
Share of females	0.509(0.024)
Crude death rate, per 1000	11.169(1.337)
Infant mortality rate, per 1000 live births	39.029(10.698)
Share of disabled	0.057(0.054)

Note: Means and standard deviations (in parentheses). Regions-of-birth are counties divided into urban and rural areas (49 in total).

Table 4 – *Level and trend breaks in pneumonia mortality at the aggregate level, 1934–1943*

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
	Pneumonia death rate		CDR		IMR		Pneumonia death rate (treated) and death rates from other infectious diseases (control)		Pneumonia death rate (treated) and death rates from degenerative diseases (control)	
	levels	logs	levels	logs	levels	logs	levels	logs	levels	logs
Post1939	-36.86***	-0.340***	-23.08**	-0.019*	-82.70	-0.022				
	(3.393)	(0.0374)	(11.108)	(0.0101)	(122.97)	(0.0331)				
Post1939Xyear	-12.10***	-0.142***	-53.84***	-0.0501***	-147.4***	-0.0502***				
	(1.164)	(0.0128)	(3.810)	(0.00347)	(42.18)	(0.0114)				
PneumoniaXPost1939							-28.81***	-0.275***	-37.47***	-0.353***
							(4.698)	(0.070)	(4.636)	(0.057)
PneumoniaXPost1939Xyear							-11.46***	-0.0787***	-8.56***	-0.114***
							(1.711)	(0.0243)	(2.112)	(0.0242)
Observations	490	490	490	490	490	490	2,450	2,318	2,450	2,389
R-squared	0.644	0.663	0.822	0.828	0.659	0.634	0.910	0.881	0.963	0.957

Note: OLS regression estimates. Models 1–6 additionally include *year* trend and *region* dummies. Models 7–10 additionally include *Post1939*, *year*, *PneumoniaXyear* trend, *disease* dummies, *diseaseXyear* trends, and *region* dummies. Infectious diseases added to Models 7 and 8 include lung tuberculosis, influenza, diarrhea, and typhoid fever. Degenerative diseases added to Models 9 and 10 include cancer, diabetes, heart disease and maternal causes. Some observations in specifications with logarithmic terms (Models 8 and 10) omitted due to zero mortality from diseases used in the models in some regions. All death rates are calculated per 100,000 mid-year population in a respective region, and infant mortality rate is calculated per 100,000 live births. Year is centered around 1939.

Table 5 – *Reduced-form estimates. Effects of pneumonia exposure in infancy on adult outcomes in Sweden, cohorts 1934–1943*

	(1) All	(2) Males	(3) Females
On disability pension			
post1939Xbaseline pneumonia mortality	-0.0367***	-0.0349***	-0.0394*
	(0.0129)	(0.0112)	(0.0198)
Pre-mean	0.343	0.316	0.342
Individuals	872,917	442,926	429,991
Ln labour income			
post1939Xbaseline pneumonia mortality	0.102***	0.0726	0.131***
	(0.0358)	(0.0512)	(0.0326)
Pre-mean	8.063	8.321	7.798
Individuals	878,606	446,511	432,095
Years of schooling			
post1939Xbaseline pneumonia mortality	0.350**	0.343**	0.359***
	(0.1306)	(0.1333)	(0.1366)
Pre-mean	9.271	9.274	9.268
Individuals	879,175	446,736	432,439
Mean hospital nights per year			
post1939Xbaseline pneumonia mortality	-0.0988***	-0.110**	-0.0876*
	(0.0309)	(0.0484)	(0.0505)
Pre-mean	0.770	0.785	0.775
Individuals	852,460	430,096	422,364

Note: estimations from *SIP*.
Standard errors (in parentheses) are clustered at a region-of-birth level. All models include region-of-birth fixed effects (49 regions) and year-of-birth fixed effects. Pneumonia mortality rate is per 1000 mid-year population. Age interval for disability outcome is age 47–60, for ln labour income is ages 44–60, and for the mean hospital nights per year is ages 53–60. Models are estimated according to Eq.1, for both sexes, males and females. *Pre-mean* denotes mean of the outcome in 1934–1938.
*** p<0.01, ** p<0.05, * p<0.1

Table 6 – *2SLS estimates. Effects of pneumonia exposure and access to sulfapyridine in infancy on adult outcomes in Sweden, cohorts 1934–1943*

	Panel A - Effect of pneumonia mortality instrumented with *post1939Xbaseline pneumonia mortality*				Panel B - Effect of infant mortality instrumented with *post1939Xbaseline pneumonia mortality*				Panel C - Effect of sulfapyridine availability instrumented with *post1939Xbaseline pneumonia mortality*			
	(1)	(2)	(3)		(1)	(2)	(3)		(1)	(2)	(3)	
	All	Males	Females		All	Males	Females		All	Males	Females	
On disability pension												
pneumonia mortality	0.0685***	0.0652***	0.0734*	infant mortality	0.0389***	0.0374***	0.0414*	sulfapyridine availability	-0.103***	-0.101***	-0.107*	
	(0.0240)	(0.0209)	(0.0370)		(0.0137)	(0.0120)	(0.0209)		(0.0361)	(0.0323)	(0.0540)	
Pre-mean	0.343	0.316	0.342	Pre-mean	0.343	0.316	0.342	Pre-mean	0.343	0.316	0.342	
Individuals	872,917	442,926	429,991	Individuals	872,917	442,926	429,991	Individuals	872,917	442,926	429,991	
Ln labour income												
pneumonia mortality	-0.191***	-0.135	-0.245***	infant mortality	-0.109***	-0.0776	-0.138***	sulfapyridine availability	0.285***	0.206	0.357***	
	(0.0668)	(0.0954)	(0.0608)		(0.0380)	(0.0547)	(0.0343)		(0.0996)	(0.1454)	(0.0886)	
Pre-mean	8.063	8.321	7.798	Pre-mean	8.063	8.321	7.798	Pre-mean	8.063	8.321	7.798	
Individuals	878,606	446,511	432,095	Individuals	878,606	446,511	432,095	Individuals	878,606	446,511	432,095	
Years of schooling												
pneumonia mortality	-0.653**	-0.641**	-0.670**	infant mortality	-0.371**	-0.367**	-0.378**	sulfapyridine availability	0.949**	0.944**	0.961**	
	(0.243)	(0.249)	(0.254)		(0.138)	(0.142)	(0.144)		(0.354)	(0.366)	(0.365)	
Pre-mean	9.271	9.274	9.268	Pre-mean	9.271	9.274	9.268	Pre-mean	9.271	9.274	9.268	
Individuals	879,175	446,736	432,439	Individuals	879,175	446,736	432,439	Individuals	879,175	446,736	432,439	
Mean hospital nights per year												
pneumonia mortality	0.184***	0.205**	0.164*	infant mortality	0.105***	0.118**	0.0924*	sulfapyridine availability	-0.285***	-0.324**	-0.246*	
	(0.0578)	(0.0905)	(0.0943)		(0.0329)	(0.0520)	(0.0533)		(0.0892)	(0.143)	(0.142)	
Pre-mean	0.770	0.785	0.775	Pre-mean	0.770	0.785	0.775	Pre-mean	0.770	0.785	0.775	
Individuals	852,460	430,096	422,364	Individuals	852,460	430,096	422,364	Individuals	852,460	430,096	422,364	

Note: estimations from *SIP*.

Standard errors (in parentheses) are clustered at a region-of-birth level. All models include region-of-birth fixed effects (49 regions) and year-of-birth fixed effects. Pneumonia mortality rate (1934–1943) is per 1000 mid-year population (mean 1.035 and sd 0.138). Infant mortality rate (1934–1943) is per 100 live births (mean 3.902 and sd 1.069). Sulfapyridine availability (region-of-birth-level in 1939) is in grams per 1 mortality rate (1939–1943) is per 100 mid-year population (mean 2.181 and sd 2.317). Sulfapyridine availability enters the model as *post1939Xsulfapyridine availability*. In the first stage (for all sexes ln labour income, similar to other estimation samples), coefficient for *post1939Xbaseline pneumonia mortality* in regression for (Panel A) pneumonia mortality is -0.536(se 0.0934); in regression for infant mortality (Panel B) is -0.943(se 0.392); in regression for sulfapyridine availability (Panel C) is 0.369(se 1.941). Age interval for disability outcome is age 47–60, for ln labour income is ages 44–60, and for the mean hospital nights per year is ages 53–60. The 2SLS models are estimated according to Eq.2. *Pre-mean* denotes mean of the outcome in 1934–1938.

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

Table 7 – *Utilisation of sulfapyridine and region-of-birth characteristics*

	(1) Sulfapyridine in grams per 100	(2) Price index of medical drugs in 1939	(3) Change in price index of medical drugs 1939-1940
Ln real regional income, per 1000	-0.697	-0.194	-0.456
	(2.728)	(0.342)	(1.650)
Share employed in agriculture	-6.727	-0.0441	2.289
	(5.515)	(0.690)	(3.336)
Share employed in industry	-6.409	0.759	2.701
	(4.836)	(0.605)	(2.926)
Ln medical personnel, per 1000	-0.786	0.155	-0.318
	(1.327)	(0.166)	(0.803)
Ln number of hospitals, per 100,000	-0.259	0.0737	-0.0680
	(0.634)	(0.0794)	(0.384)
Ln real expenditures on public health, per 1000	0.685	0.000119	0.184
	(0.834)	(0.104)	(0.504)
Share of females	-12.12	3.273	5.908
	(35.11)	(4.396)	(21.24)
Crude death rate, per 1000	405.5	-59.83	-8.238
	(350.9)	(43.92)	(212.2)
Share of disabled	3.335	0.774	-0.757
	(12.06)	(1.509)	(7.294)
Ln number of schools, per 1000	0.299	-0.0535	-0.148
	(0.479)	(0.0600)	(0.290)
Ln pharmacists, per 100,000	1.571	-0.221*	0.459
	(0.951)	(0.119)	(0.575)
R-squared	0.273	0.012	0.013
Regions of birth	49	49	49

Note: Standard errors (in parentheses)

Sources: for drug utilization – Riksarkivet 1939–1940; for region-of-birth characteristics – SOSa-e 1939.

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

Table 8 – *Robustness analyses (region-of-birth characteristics). Reduced-form estimates. Effects of pneumonia exposure in infancy on adult outcomes in Sweden, cohorts 1934–1943*

	(1) Control diseases			(2) Region-of-birth characteristics			(3) Region-of-birth linear time trends		
	All	Males	Females	All	Males	Females	All	Males	Females
On disability pension									
post1939Xbaseline pneumonia mortality	-0.0411***	-0.0357***	-0.0475***	-0.0358***	-0.0367***	-0.0355*	-0.0306***	-0.0175	-0.0460***
	(0.0100)	(0.0117)	(0.0147)	(0.0121)	(0.0119)	(0.0199)	(0.00926)	(0.0161)	(0.0157)
Pre-mean	0.343	0.316	0.342	0.343	0.316	0.342	0.343	0.316	0.342
Individuals	872,917	442,926	429,991	872,917	442,926	429,991	872,917	442,926	429,991
Ln labour income									
post1939Xbaseline pneumonia mortality	0.127***	0.0898*	0.165***	0.089***	0.0504	0.129***	0.0703*	0.107**	0.0342
	(0.0318)	(0.0461)	(0.0350)	(0.0323)	(0.0388)	(0.0350)	(0.0365)	(0.0500)	(0.0625)
Pre-mean	8.063	8.321	7.798	8.063	8.321	7.798	8.063	8.321	7.798
Individuals	878,606	446,511	432,095	878,606	446,511	432,095	878,606	446,511	432,095
Years of schooling									
post1939Xbaseline pneumonia mortality	0.489***	0.456**	0.525***	0.272**	0.244*	0.306**	0.111	0.057	0.164*
	(0.1778)	(0.2029)	(0.1611)	(0.1241)	(0.1301)	(0.1285)	(0.0834)	(0.1153)	(0.0952)
Pre-mean	9.271	9.274	9.268	9.271	9.274	9.268	9.271	9.274	9.268
Individuals	879,175	446,736	432,439	879,175	446,736	432,439	879,175	446,736	432,439
Mean hospital nights per year									
post1939Xbaseline pneumonia mortality	-0.0859**	-0.0999	-0.0876*	-0.0930***	-0.110**	-0.0748	-0.111*	-0.111	-0.127*
	(0.0409)	(0.0635)	(0.0505)	(0.0328)	(0.0506)	(0.0572)	(0.0581)	(0.0887)	(0.0641)
Pre-mean	0.770	0.785	0.775	0.770	0.785	0.775	0.770	0.785	0.775
Individuals	852,460	430,096	422,364	852,460	430,096	422,364	852,460	430,096	422,364

Note: estimations from *SIP*. Standard errors (in parentheses) are clustered at a region-of-birth level. Pneumonia mortality rate is per 1000 mid-year population. Age interval for disability outcome is age 47–60, for ln labour income is ages 44–60, and for the mean hospital nights per year is ages 53–60. All models include region-of-birth fixed effects (49 regions) and year-of-birth fixed effects. All models are estimated according to Eq.1 plus additional controls. A set of Models 1 additionally includes disease controls, such as separate interactions between *post1939* and baseline mortality from typhoid fever, diarrhea, lung tuberculosis, influenza, heart disease, diabetes, cancer, and maternal causes. A set of Models 2 additionally includes region-specific controls, such as ln real region income per 1000, share of employed in agriculture, share of employed in industry, ln medical personnel per 1000, ln number of hospitals per 1000, ln real spending on hospitals per 1000, ln pharmacists per 100,000, price index of medical drugs in 1939, change in price index of medical drugs in 1939–1940, share of females, ln number of schools per 1000, crude death rate, share of disabled, and infant mortality rate per 1000 live births. A set of Models 3 additionally includes region-specific linear time trends. *Pre-mean* denotes mean of the outcome in 1934–1938. *** p<0.01, ** p<0.05, * p<0.1.

Table 9 – *Robustness analysis (mean reversion). Reduced-form estimates. Effects of pneumonia exposure in infancy on adult outcomes in Sweden, cohorts 1934–1943*

	(1) On disability pension	(2) Ln labour income	(3) Years of schooling	(4) Mean hospital nights per year
post1939Xbaseline pneumonia mortality	-0.0615* (0.0344)	0.126** (0.0587)	0.258* (0.1441)	-0.0739** (0.0324)
post1939Xbaseline fraction on disability	0.995*** (0.0005)			
post1939X baseline ln labour income		0.987*** (0.0007)		
post1939Xbaseline years of schooling			-0.0868** (0.0331)	
post1939Xbaseline mean hospital nights per year				0.395** (0.1540)
Individuals	872,917	878,606	879,175	852,460
R-squared	0.549	0.198	0.066	0.002

Note: estimations from *SIP*.
Standard errors (in parentheses) are clustered at a region-of-birth level. All models include region-of-birth fixed effects (49 regions) and year-of-birth fixed effects. Pneumonia mortality rate is per 1000 mid-year population. Age interval for disability outcome is age 47–60, for ln labour income is ages 44–60, and for the mean hospital nights per year is ages 53–60. Baseline outcomes are calculated as mean outcomes for the cohorts 1934–1938. *Pre-mean* denotes mean of the outcome in 1934–1938.
*** p<0.01, ** p<0.05, * p<0.1

Table 10 – *Robustness analyses (parental characteristics and family fixed effects). Reduced-form estimates. Effects of pneumonia exposure in infancy on adult outcomes in Sweden, cohorts 1934–1943*

	(1) Parental characteristics			(2) Eq.1 on Mothers' sample			(3) Mother FE		
	All	Males	Females	All	Males	Females	All	Males	Females
On disability pension									
post1939Xbaseline pneumonia mortality	-0.0419***	-0.0410***	-0.0437**	-0.0381***	-0.0396***	-0.0375**	-0.0563***	-0.0507**	-0.0847***
	(0.0124)	(0.0114)	(0.0190)	(0.0117)	(0.0127)	(0.0165)	(0.0126)	(0.0222)	(0.0242)
Pre-mean	0.343	0.316	0.342	0.341	0.314	0.369	0.341	0.314	0.369
Individuals	872,917	442,926	429,991	808,643	409,455	399,188	808,643	409,455	399,188
Mothers							544,177	330,854	324,047
Ln labour income									
post1939Xbaseline pneumonia mortality	0.119***	0.0943*	0.143***	0.109***	0.0892**	0.128***	0.112***	0.0623	0.189***
	(0.0329)	(0.0481)	(0.0307)	(0.0266)	(0.0354)	(0.0266)	(0.0353)	(0.0614)	(0.0677)
Pre-mean	8.063	8.321	7.798	8.098	8.362	7.830	8.098	8.362	7.830
Individuals	878,606	446,511	432,095	811,241	411,022	400,219	811,241	411,022	400,219
Mothers							545,318	331,911	324,746
Years of schooling									
post1939Xbaseline pneumonia mortality	0.445***	0.454***	0.440***	0.375***	0.394***	0.360**	0.404***	0.544***	0.309***
	(0.132)	(0.136)	(0.136)	(0.138)	(0.138)	(0.147)	(0.0508)	(0.0983)	(0.0855)
Pre-mean	9.271	9.274	9.268	9.330	9.347	9.314	9.330	9.347	9.314
Individuals	879,175	446,736	432,439	804,245	406,459	397,786	804,245	406,459	397,786
Mothers							542,422	329,028	323,263
Mean hospital nights per year									
post1939Xbaseline pneumonia mortality	-0.104***	-0.115**	-0.0922*	-0.129***	-0.141**	-0.117**	-0.0816	-0.0461	-0.120
	(0.0315)	(0.0502)	(0.0489)	(0.0337)	(0.0530)	(0.0463)	(0.0579)	(0.106)	(0.107)
Pre-mean	0.770	0.785	0.775	0.718	0.729	0.707	0.718	0.729	0.707
Individuals	852,460	430,096	422,364	796,818	402,146	394,672	796,818	402,146	394,672
Mothers							538,951	326,039	321,068

Note: estimations from *SIP*. Standard errors (in parentheses) are clustered at a region-of-birth level. All models include region-of-birth fixed effects (49 regions) and year-of-birth fixed effects. Pneumonia mortality rate is per 1000 mid-year population. Age interval for disability outcome is age 47–60, for ln labour income is ages 44–60, and for the mean hospital nights per year is ages 53–60. A set of Models 1 is estimated according to Eq.1, for both sexes, males and females, plus additional controls (parity, family size, education of father, sector of father, age of mother). A set of Models 2 is estimated according to Eq.1 for the Mothers' sample. A set of Models 3 is estimated according to Eq.5. A family identifier is the same biological mother. *Pre-mean* denotes mean of the outcome in 1934–1938. *** p<0.01, ** p<0.05, * p<0.1

Table 11 –*Reduced-form estimates. Effects of pneumonia exposure in infancy on adult health by cause of morbidity in Sweden, cohorts 1934–1943*

	(1)	(2)	(3)	(4)
	infectious	infectious	infectious	infectious
post1939Xbaseline pneumonia mortality	-0.0255*	-0.00747	-0.0261*	-0.0183
	(0.0143)	(0.0148)	(0.0141)	(0.0191)
Pre-mean	0.120	0.120	0.120	0.120
	CVD	CVD	CVD	CVD
post1939Xbaseline pneumonia mortality	-0.120**	-0.103**	-0.119***	-0.105
	(0.0555)	(0.0441)	(0.0424)	(0.0786)
Pre-mean	0.600	0.600	0.600	0.600
	diabetes	diabetes	diabetes	diabetes
post1939Xbaseline pneumonia mortality	-0.0202	-0.0497***	-0.0186	-0.0360
	(0.0126)	(0.0177)	(0.0149)	(0.0402)
Pre-mean	0.090	0.090	0.090	0.090
	cancer	cancer	cancer	cancer
post1939Xbaseline pneumonia mortality	-0.00312	0.0162	-0.0138	-0.122*
	(0.0417)	(0.0369)	(0.0463)	(0.0694)
Pre-mean	0.471	0.471	0.471	0.471
	degenerative	degenerative	degenerative	degenerative
post1939Xbaseline pneumonia mortality	-0.231***	-0.205***	-0.177***	-0.213***
	(0.0486)	(0.0551)	(0.0469)	(0.0699)
Pre-mean	1.189	1.189	1.189	1.189
	mental	mental	mental	mental
post1939Xbaseline pneumonia mortality	-0.0585	-0.0745	-0.0545	0.130
	(0.0537)	(0.0513)	(0.0499)	(0.137)
Pre-mean	0.454	0.454	0.454	0.454
Individuals	852,460	852,460	852,460	852,460

Note: estimations from *SIP*.
Standard errors (in parentheses) are clustered at a region-of-birth level. Pneumonia mortality rate is per 1000 mid-year population. Age interval for the mean hospital nights per year is ages 53–60. All models include region-of-birth fixed effects (49 regions) and year-of-birth fixed effects. A set of Models 1 correspond to Eq.1, for both sexes. All other models are estimated according to Eq.1 plus additional controls. A set of Models 2 additionally includes disease controls, such as separate interactions between *post1939* and baseline mortality from typhoid fever, diarrhea, lung tuberculosis, influenza, heart disease, diabetes, cancer, and maternal causes. A set of Models 3 additionally includes region-specific controls, such as ln real region income per 1000, share of employed in agriculture, share of employed in industry, ln medical personnel per 1000, ln number of hospitals per 1000, ln real spending on hospitals per 1000, ln pharmacists per 100,000, price index of medical drugs in 1939, change in price index of medical drugs in 1939–1940 share of females, ln number of schools per 1000, crude death rate, share of disabled, and infant mortality rate per 1000 live births. A set of Models 4 additionally includes region-specific linear trends. *Pre-mean* denotes mean of the outcome in 1934–1938.
*** p<0.01, ** p<0.05, * p<0.1

Table 12 – *Reduced-form estimates. Effects of pneumonia exposure in infancy on labour productivity holding education constant, Sweden cohorts 1934–1943*

	(1) All	(2) Male	(3) Female
Ln labour income, years of schooling not included			
post1939Xbaseline pneumonia mortality	0.0908*** (0.0271)	0.0623* (0.0328)	0.119*** (0.0305)
Ln labour income, years of schooling included			
post1939Xbaseline pneumonia mortality	0.0599** (0.0284)	0.0388 (0.0362)	0.0777*** (0.0274)
Individuals	861,772	436,230	425,542
On disability pension, years of schooling not included			
post1939Xbaseline pneumonia mortality	-0.0357*** (0.0121)	-0.0379*** (0.0128)	-0.0340* (0.0193)
On disability pension, years of schooling included			
post1939Xbaseline pneumonia mortality	-0.0283** (0.0116)	-0.0314*** (0.0114)	-0.0253 (0.0187)
Individuals	856,574	432,958	423,616

Note: estimations from *SIP*.
Standard errors (in parentheses) are clustered at a region-of-birth level. Pneumonia mortality rate is per 1000 mid-year population. All models include region-of-birth fixed effects (49 regions) and year-of-birth fixed effects. A set of Models 1 correspond to Eq.1, for both sexes. All other models are estimated according to Eq.1 plus region-specific controls, such as ln real region income per 1000, share of employed in agriculture, share of employed in industry, ln medical personnel per 1000, ln number of hospitals per 1000, ln real spending on hospitals per 1000, share of females, ln number of schools per 1000, ln pharmacists per 100,000, price index of medical drugs in 1939, change in price index of medical drugs in 1939–1940, crude death rate, share of disabled, and infant mortality rate per 1000 live births. Sample is retricted to those for whom information of education is provided.
*** p<0.01, ** p<0.05, * p<0.1

Table 13 – *Reduced-form estimates. Heterogeneous effects of pneumonia exposure in infancy by parental characteristics on adult outcomes in Sweden, cohorts 1934–1943*

	(1) Mother's age		(2) Father's education		(3) Father's sector of employment	
	young	old	only primary	> primary	agriculture	industry/ service
On disability pension						
post1939Xbaseline pneumonia mortality	-0.0443***	-0.0380**	-0.0495***	-0.0330	-0.0508***	-0.0336*
	(0.0118)	(0.0145)	(0.0110)	(0.0221)	(0.0136)	(0.0178)
Pre-mean	0.345	0.342	0.337	0.353	0.353	0.317
Individuals	443,476	429,441	557,974	314,943	556,125	316,792
Ln labour income						
post1939Xbaseline pneumonia mortality	0.102***	0.132***	0.135***	0.119***	0.141***	0.119***
	(0.0252)	(0.0449)	(0.0369)	(0.0284)	(0.0390)	(0.0314)
Pre-mean	8.104	8.024	8.111	7.998	8.015	8.205
Individuals	445,231	433,375	561,016	317,590	561,426	317,893
Years of schooling						
post1939Xbaseline pneumonia mortality	0.389***	0.493***	0.534***	0.390*	0.489***	0.401***
	(0.116)	(0.154)	(0.116)	(0.195)	(0.151)	(0.136)
Pre-mean	9.234	9.306	9.208	9.356	9.167	9.579
Individuals	441,992	437,183	554,331	324,844	563,519	315,656
Mean hospital nights per year						
post1939Xbaseline pneumonia mortality	-0.0877*	-0.120**	-0.124***	-0.131*	-0.128**	-0.0863
	(0.0454)	(0.0481)	(0.0420)	(0.0752)	(0.0513)	(0.0567)
Pre-mean	0.735	0.805	0.685	0.893	0.802	0.681
Individuals	436,125	416,335	551,586	300,874	540,214	312,246

Note: estimations from *SIP*.
Standard errors (in parentheses) are clustered at a region-of-birth level. All models include region-of-birth fixed effects (49 regions) and year-of-birth fixed effects. Pneumonia mortality rate is per 1000 mid-year population. Age interval for disability outcome is age 47–60, for ln labour income is ages 44–60, and for the mean hospital nights per year is ages 53–60. All Models are estimated according to Eq.1 plus additional controls (parity, family size, education of father, sector of father, age of mother) separately for sub-groups defined by parental characteristics. *Pre-mean* denotes mean of the outcome in 1934–1938.
*** p<0.01, ** p<0.05, * p<0.1

Table 14 – *Reduced-form estimates. Effects of pneumonia exposure in infancy on adult outcomes with county of birth as a regional unit, Sweden, cohorts 1934–1943*

	(1) Baseline	(2) Control diseases	(3) County-of-birth characteristics	(4) County-of-birth linear time trends	(5) Parental characteristics	(6) Mother FE
On disability pension						
post1939Xbaseline pneumonia mortality, both sexes	-0.0400*	-0.0416**	-0.0558***	-0.0683***	-0.0462**	-0.0417*
	(0.0226)	(0.0160)	(0.0164)	(0.0148)	(0.0223)	(0.0223)
Pre-mean	0.343	0.343	0.343	0.343	0.343	0.341
Individuals	872,917	872,917	872,917	872,917	872,917	808,643
Mothers						544,177
Ln labour income						
post1939Xbaseline pneumonia mortality, both sexes	0.127*	0.0951**	0.0943*	0.170***	0.151**	0.133**
	(0.0628)	(0.0456)	(0.0526)	(0.0593)	(0.0682)	(0.0590)
Pre-mean	8.063	8.063	8.063	8.063	8.063	8.098
Individuals	878,606	878,606	878,606	878,606	878,606	811,241
Mothers						545,318
Years of schooling						
post1939Xbaseline pneumonia mortality, both sexes	0.224	0.191	0.208	0.116	0.328	0.280
	(0.186)	(0.242)	(0.1644)	(0.163)	(0.208)	(0.208)
Pre-mean	9.271	9.271	9.271	9.271	9.271	9.330
Individuals	879,175	879,175	879,175	879,175	879,175	804,245
Mothers						542,422
Mean hospital nights per year						
post1939Xbaseline pneumonia mortality, both sexes	-0.115**	-0.145**	-0.0885**	-0.222*	-0.127**	-0.145**
	(0.0481)	(0.0576)	(0.0362)	(0.109)	(0.0488)	(0.0557)
Pre-mean	0.770	0.770	0.770	0.770	0.770	0.718
Individuals	852,460	852,460	852,460	852,460	852,460	796,818
Mothers						538,951

Note: estimations from *SIP*. Standard errors (in parentheses) are clustered at a county-of-birth level. Samples include both sexes jointly. Pneumonia mortality rate is per 1000 mid-year population. Mean of baseline pneumonia mortality rate is 1.043 (se 0.097) per 1000. All models include county-of-birth fixed effects (25counties) and year-of-birth fixed effects. A set of Models 1 correspond to Eq.1. All other models are estimated according to Eq.1 plus additional controls. A set of Models 2 additionally includes disease controls, such as separate interactions between *post1939* and baseline mortality from typhoid fever, diarrhea, lung tuberculosis, influenza, heart disease, diabetes, cancer, and maternal causes. A set of Models 3 additionally includes county-specific controls, such as ln real region income per 1000, share of employed in agriculture, share of employed in industry, ln medical personnel per 1000, ln number of hospitals per 1000, ln real spending on hospitals per 1000, ln pharmacists per 100,000, price index of medical drugs in 1939, change in price index of medical drugs in 1939–1940, share of females, ln number of schools per 1000, crude death rate, share of disabled, and infant mortality rate per 1000 live births. A set of Models 4 additionally includes county-of-birth linear time trends. A set of Models 5 additionally control for parental characteristics (parity, family size, education of father, sector of father, age of mother). A set of Models 6 additionally includes mother fixed effects. *Pre-mean* denotes mean of the outcome in 1934–1938. *** p<0.01, ** p<0.05, * p<0.1

FIGURES

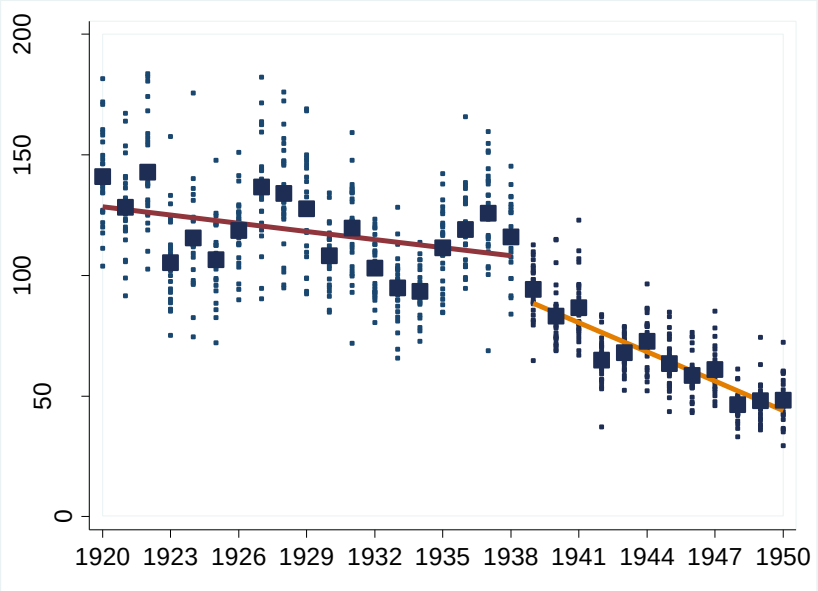


Figure 1 – *Pneumonia mortality in Sweden in 1920–1950, per 100,000*

Note: tiny dots represent the county-level death rates, the bold dots represent the country-level averages of death rates; trend lines are separate for 1920-1938 and 1939-1950.
Source: SOS 1920-1950c, d.

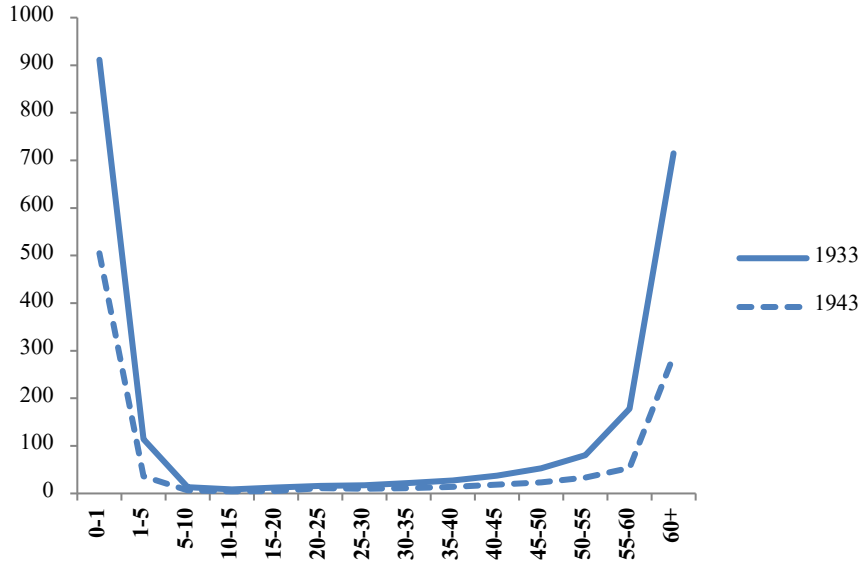


Figure 2 – *Age pattern of pneumonia mortality before and after introduction of sulfa antibiotics, per 100,000, Sweden*

Note: pneumonia includes pneumonia, bronchitis and pleurisy.
Source: SOS 1933cd, 1943cd.

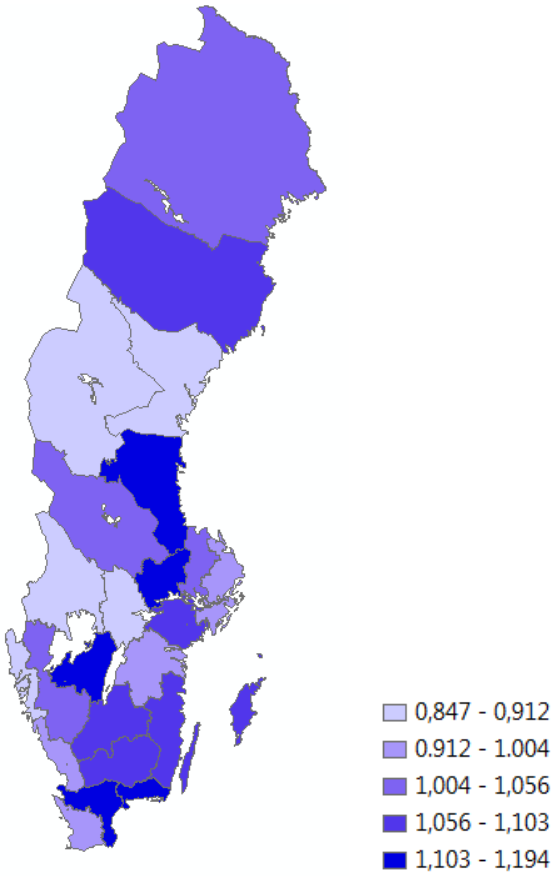


Figure 3 – *Geographical distribution of pneumonia death rate, Sweden 1932–1936*
Note: county pneumonia death rates relative to country pneumonia death rate
Sources: SOS 1932–1936cd; county boundaries from Riksarkivet (1932–1936)

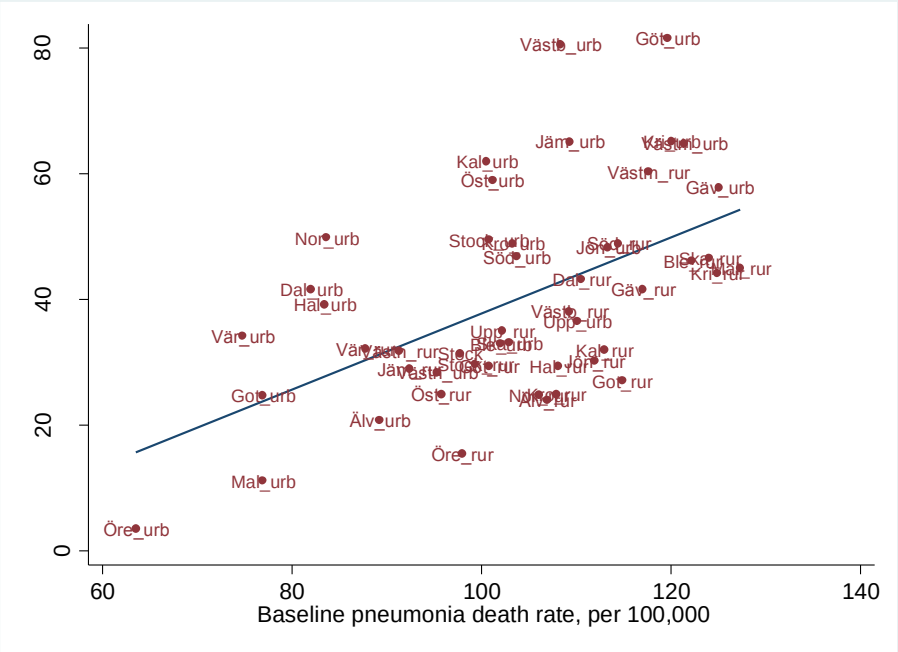


Figure 4 – *Convergence in pneumonia mortality rates across regions after arrival of sulfapyridine, Sweden*

Note: Figure presents the absolute decline in death rate due to pneumonia (between 1943 and the average of 1932–1936) plotted against the pre-treatment death rate from pneumonia (average of 1932–1936). This is analogous specification to Eq.2, re-written in the first-difference form:

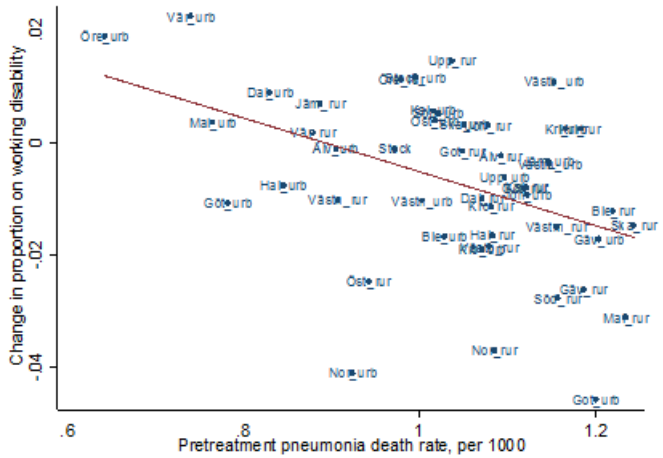
$$\Delta Rate_c^{post} = \beta_{11} BaseRate_c + c + \nu_{11cb}$$

The estimated equation is as follows:

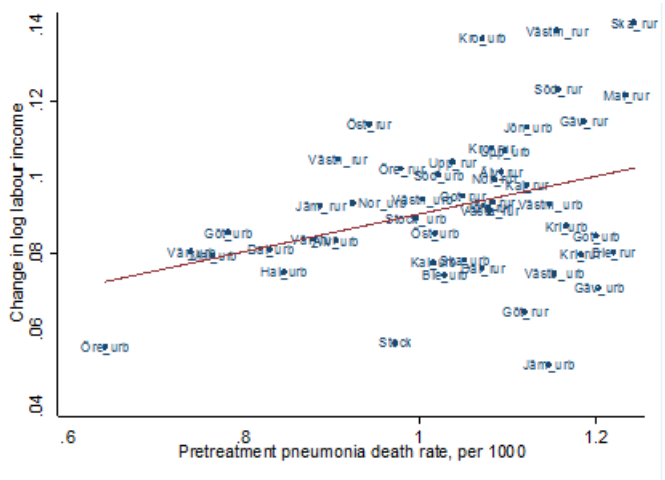
$$\Delta \overline{Rate_c^{post}} = 0.605 \overline{BaseRate_c} - 22.75$$

$$(0.138) \qquad (14.417) \qquad N = 49, Rsq = 0.29$$

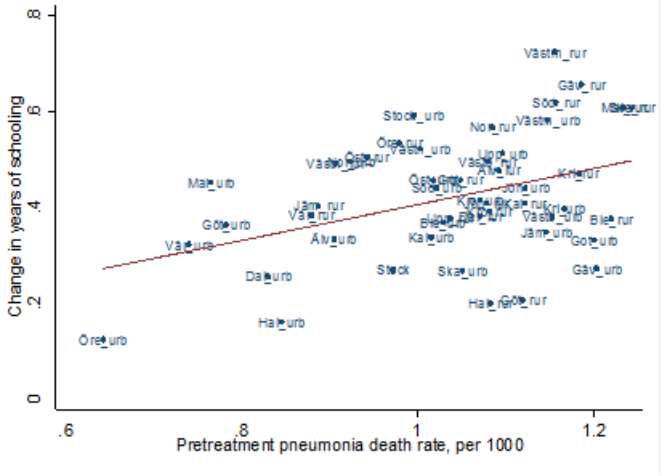
Source: own estimations based on the data from Statistiska Centralbyrån 1930–1950



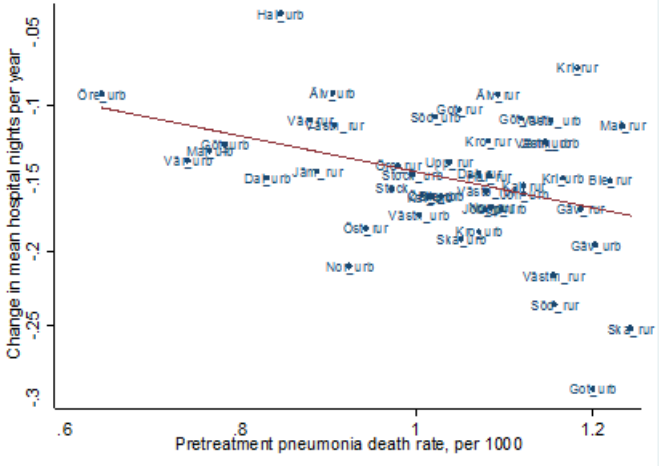
(a) – Probability of working disability



(b) – Ln labour income



(c) – Years of schooling



(d) – Mean hospital nights per year

Figure 5 – *Convergence in later-life outcomes across regions of birth after arrival of sulfapyridine, Sweden*

Note: Figure presents the absolute change in the outcomes under study aggregated at region-of-birth level rate due to pneumonia (between average of 1939–1943 and the average of 1934–1938) plotted against the pre-treatment death rate from pneumonia (average of 1932–1936). One outlier (Stockholm rural) is excluded from the graph; excluding this region from the parametrical analysis does not affect the results. Source: SIP

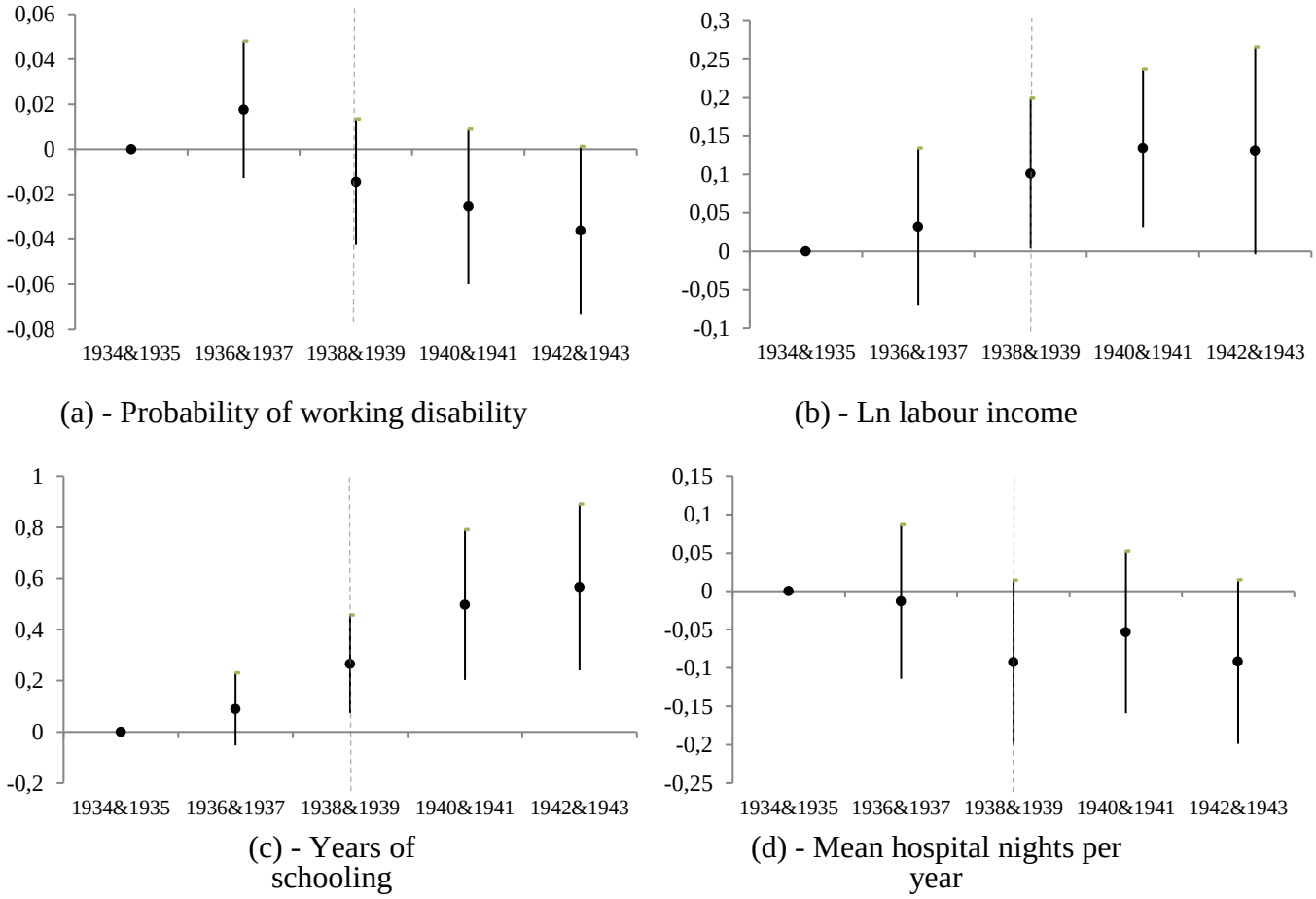


Figure 6 – *Event study analyses of the effect of pneumonia exposure in infancy on adult outcomes in Sweden, cohorts 1934–1943*

Note: The models are estimated according to Eq.3. Cohorts 1934–1935 are a reference category. For the cohorts, point estimates and 95% confidence intervals are presented. Cohorts 1938/1939 are the first exposed to sulfa antibiotics.

Appendices

Appendix A – *Survivors of cohorts under analysis*

The cohorts born between 1934 and 1943 appear in the SIP dataset from 1968. We therefore do not observe individuals that died or migrated from Sweden prior to age 34. We gathered information on one-year survivors born in rural areas (live births minus infant deaths) of the cohorts born 1930-1950 from Statistics Sweden (SOS 1930-1950). In Figure we plot them against counts of individuals with places of birth available in SIP by cohort and those which have valid information on the parish of birth. A relatively stable fraction of individuals observed in the SIP dataset compared to a number of one-year survivors indicate that the individuals born 1934-1943 were dying at a constant rate between the ages 1 and 33. The selection to survival to adulthood should not therefore violate the results in our paper (in the paper we additionally assess it with a two-stage Heckman selection procedure).

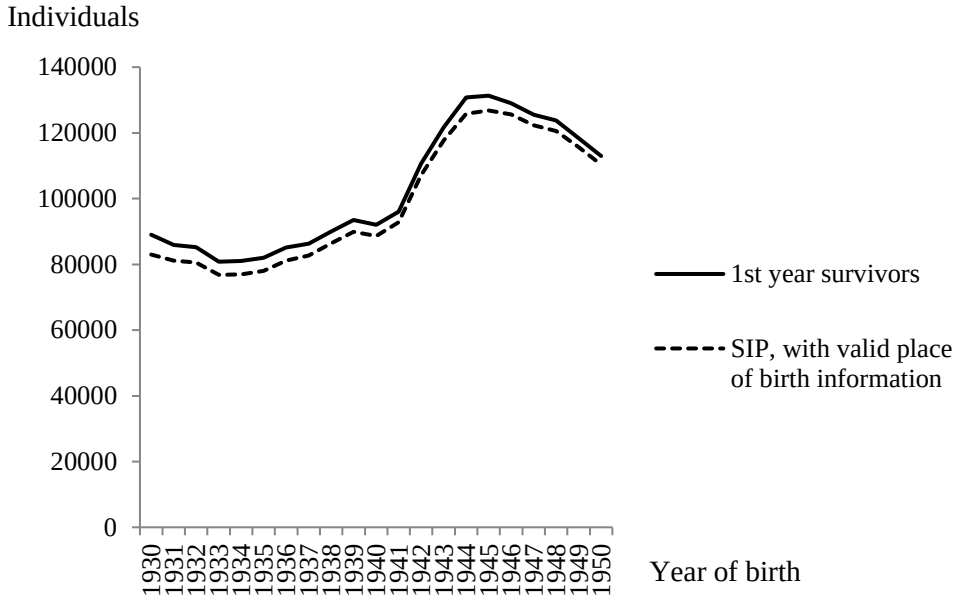


Figure – *One-year survivors and estimation sample for the cohorts 1930–1950*
Source: SIP and SCB.

Appendix B – *Groups of causes of morbidity*

The cause of admission is obtained from the Swedish national inpatient register 1987–2012. It adopted two revisions of the international classifications of the causes of morbidity, such as the revision 9 for 1987–1996, and the revision 10 for 1997–2012. We classify all causes of morbidity into five groups, such as infectious diseases, cardiovascular diseases, diabetes, cancer, degenerative diseases of tissues and organs, and mental diseases. The group of degenerative diseases of tissues and organs is dominant with symptoms of respiratory diseases, arthritis and gastroenteric diseases. The exact codes used for these groupings are provided in the following table:

Table – *Diagnoses groups across two revisions of the ICD, 1987–2012*

	ICD-9	ICD-10
Infectious diseases	001-139; 320-324; 460-519	A00-B99; G00-G09; J00-J99
Cardiovascular diseases	390-459	I00-I99
Diabetes	250	E10-E14
Cancer	140-239	C00-D48
Degenerative diseases	240-246; 251-289; 325-330; 332-389; 520-796	D50-E07; E15-E90; F10-F99; G10-G26; G31-H95; K00-R94
Mental diseases	290-319; 331	F00-F09; G30

Appendix C

Table – *Reduced-form estimates. Effects of pneumonia exposure in infancy on outcomes in later life adjusting for selective survival, Sweden, cohorts 1934–1943*

	(1)
On disability pension	
post1939Xbaseline pneumonia mortality, both sexes	-0.0328***
	(0.00929)
Individuals	872,917
Ln labour income	
post1939Xbaseline pneumonia mortality, both sexes	0.0759**
	(0.0367)
Individuals	878,606
Years of schooling	
post1939Xbaseline pneumonia mortality, both sexes	0.112
	(0.0828)
Individuals	879,175
Mean hospital nights per year	
post1939Xbaseline pneumonia mortality, both sexes	-0.112*
	(0.0581)
Individuals	852,460

Note: estimations from *SIP*.
Models adjust for selective survival by using a two-stage Heckman selection procedure. Both sexes jointly. Standard errors (in parentheses) are clustered at a region-of-birth level. Pneumonia mortality rate is per 1000 mid-year population. All models include region-of-birth fixed effects (49 regions) and year-of-birth fixed effects. A set of Models correspond to Eq.1 plus includes region-of-birth linear time trends.
*** p<0.01, ** p<0.05, * p<0.1

Appendix D

Table – *Reduced-form estimates. Effects of pneumonia exposure in infancy on adult outcomes controlling for regional food prices, Sweden, cohorts 1934–1943*

	(1) Both sexes	(2) Males	(3) Females
On disability pension			
post1939Xbaseline pneumonia mortality, both sexes	-0.0359***	-0.0350***	-0.0374**
	(0.0122)	(0.0113)	(0.0192)
Pre-mean	0.343	0.343	0.343
Individuals	872,917	872,917	872,917
Ln labour income			
post1939Xbaseline pneumonia mortality, both sexes	0.0907***	0.0520	0.130***
	(0.0317)	(0.0386)	(0.0348)
Pre-mean	8.063	8.063	8.063
Individuals	878,606	878,606	878,606
Years of schooling			
post1939Xbaseline pneumonia mortality, both sexes	0.279**	0.250*	0.313**
	(0.125)	(0.132)	(0.128)
Pre-mean	9.271	9.271	9.271
Individuals	879,175	879,175	879,175
Mean hospital nights per year			
post1939Xbaseline pneumonia mortality, both sexes	-0.0920***	-0.107**	-0.0767
	(0.0328)	(0.0493)	(0.0564)
Pre-mean	0.770	0.770	0.770
Individuals	852,460	852,460	852,460

Note: estimations from *SIP*. Standard errors (in parentheses) are clustered at a region-of-birth level (49 regions). Pneumonia mortality rate is per 1000 mid-year population. A set of Models includes region-of-birth fixed effects, year of birth fixed effects, and region-of-birth controls, such as ln real region income per 1000, share of employed in agriculture, share of employed in industry, ln medical personnel per 1000, ln number of hospitals per 1000, ln real spending on hospitals per 1000, ln pharmacists per 100,000, price index of medical drugs in 1939, change in price index of medical drugs in 1939–1940, share of females, ln number of schools per 1000, crude death rate, share of disabled, infant mortality rate per 1000 live births, and price index of main food products. *Pre-mean* denotes mean of the outcome in 1934–1938. *** p<0.01, ** p<0.05, * p<0.1

Appendix E

Table – *Reduced-form estimates. Effects of pneumonia exposure in infancy on mortality in ages 34–60, Sweden, cohorts 1934–1943*

	(1) Baseline	(2) Control diseases	(3) Region-of-birth characteristics	(4) Region-of-birth linear trends	(5) Parental characteristics	(6) Mother FE
post1939Xbaseline pneumonia mortality, both sexes	0.000648	0.00336	-0.00182	-0.0120	-0.000780	-0.00315
	(0.00450)	(0.00513)	(0.00572)	(0.00983)	(0.00515)	(0.00610)
Pre-mean	0.0804	0.0804	0.0804	0.0804	0.0804	0.0464
Individuals	895,701	895,701	895,701	895,701	895,701	818,237
Mothers						548,422
post1939Xbaseline pneumonia mortality, males	-0.00418	-0.00579	-0.00662	-0.0292*	-0.0061	0.0112
	(0.00713)	(0.00933)	(0.00959)	(0.0152)	(0.0073)	(0.0122)
Pre-mean	0.0994	0.0994	0.0994	0.0994	0.0994	0.0576
Individuals	456,960	456,960	456,960	456,960	456,960	414,926
Mothers						334,536
post1939Xbaseline pneumonia mortality, females	0.00551	0.0128***	0.00305	0.00587	0.0046	-0.00551
	(0.00403)	(0.00460)	(0.00472)	(0.00770)	(0.0043)	(0.0102)
Pre-mean	0.0607	0.0607	0.0607	0.0607	0.0607	0.0351
Individuals	438,741	438,741	438,741	438,741	438,741	403,311
Mothers						326,869

Note: estimations from *SIP*.

Standard errors (in parentheses) are clustered at a region-of-birth level. Pneumonia mortality rate is per 1000 mid-year population. All models include region-of-birth fixed effects (49 regions) and year-of-birth fixed effects. A set of Models 1 correspond to Eq.1. All other models are estimated according to Eq.1 plus additional controls. A set of Models 2 additionally includes disease controls, such as separate interactions between *post1939* and baseline mortality from typhoid fever, diarrhea, lung tuberculosis, influenza, heart disease, diabetes, cancer, and maternal causes. A set of Models 3 additionally includes region-specific controls, such as ln real region income per 1000, share of employed in agriculture, share of employed in industry, ln medical personnel per 1000, ln number of hospitals per 1000, ln real spending on hospitals per 1000, ln pharmacists per 100,000, price index of medical drugs in 1939, change in price index of medical drugs in 1939–1940, share of females, ln number of schools per 1000, crude death rate, share of disabled, and infant mortality rate per 1000 live births. A set of Models 4 additionally includes region-of-birth linear time trends. A set of Models 5 additionally control for parental characteristics (parity, family size, education of father, sector of father, age of mother). A set of Models 6 additionally includes mother fixed effects. *Pre-mean* denotes mean of the outcome in 1934–1938.

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

Appendix F

Table – *Reduced-form estimates. Effects of pneumonia exposure in infancy on cause-specific mortality in ages 34–60, both sexes, Sweden, cohorts 1934–1943*

	(1) Baseline	(2) Control diseases	(3) Region-of-birth characteristics	(4) Region-of-birth linear trends	(5) Parental characteristics	(6) Mother FE
Infectious diseases						
post1939Xbaseline pneumonia mortality	0.000963	0.000886	0.000862	0.00203	0.000880	0.000799
	(0.000805)	(0.000854)	(0.000863)	(0.00163)	(0.000767)	(0.00130)
Pre-mean	0.0033	0.0033	0.0033	0.0033	0.0033	0.0019
CVD						
post1939Xbaseline pneumonia mortality	-0.00329	-0.00333	-0.00485	-0.0136***	-0.00356	0.000374
	(0.00288)	(0.00313)	(0.00292)	(0.00400)	(0.00308)	(0.00323)
Pre-mean	0.0218	0.0218	0.0218	0.0218	0.0218	0.0133
Diabetes						
post1939Xbaseline pneumonia mortality	0.000139	-0.000671	0.000491	0.000344	0.000114	-0.000337
	(0.000457)	(0.000545)	(0.000604)	(0.00136)	(0.000477)	(0.000761)
Pre-mean	0.0015	0.0015	0.0015	0.0015	0.0015	0.0008
Cancer						
post1939Xbaseline pneumonia mortality	0.00354	0.00871***	0.00334	0.00413	0.00315	-0.000188
	(0.00250)	(0.00256)	(0.00282)	(0.00605)	(0.00250)	(0.00408)
Pre-mean	0.0293	0.0293	0.0293	0.0293	0.0293	0.0191
Degenerative						
post1939Xbaseline pneumonia mortality	-0.000898	-0.000494	-0.00147	-0.00389	-0.00106	-0.00399**
	(0.00129)	(0.00137)	(0.00165)	(0.00258)	(0.00125)	(0.00181)
Pre-mean	0.0077	0.0077	0.0077	0.0077	0.0077	0.0040
Mental						
post1939Xbaseline pneumonia mortality	-0.000592	-0.000993	-0.000765	-0.00123	-0.000642	-0.00198**
	(0.000817)	(0.000727)	(0.000835)	(0.00118)	(0.000778)	(0.000921)
Pre-mean	0.0019	0.0019	0.0019	0.0019	0.0019	0.0009
Individuals	895,701	895,701	895,701	895,701	895,701	818,237
Mothers						548,422

Note: estimations from *SIP*.

Standard errors (in parentheses) are clustered at a region-of-birth level. Pneumonia mortality rate is per 1000 mid-year population. All models include region-of-birth fixed effects (49 regions) and year-of-birth fixed effects. A set of Models 1 correspond to Eq.1. All other models are estimated according to Eq.1 plus additional controls. A set of Models 2 additionally includes disease controls, such as separate interactions between *post1939* and baseline mortality from typhoid fever, diarrhea, lung tuberculosis, influenza, heart disease, diabetes, cancer, and maternal causes. A set of Models 3 additionally includes region-specific controls, such as ln real region income per 1000, share of employed in agriculture, share of employed in industry, ln medical personnel per 1000, ln number of hospitals per 1000, ln real spending on hospitals per 1000, ln pharmacists per 100,000, price index of medical drugs in 1939, change in price index of medical drugs in 1939–1940, share of females, ln number of schools per 1000, crude death rate, share of disabled, and infant mortality rate per 1000 live births. A set of Models 4 additionally includes region-of-birth linear time trends. A set of Models 5 additionally control for parental characteristics (parity, family size, education of father, sector of father, age of mother). A set of Models 6 additionally includes mother fixed effects. *Pre-mean* denotes mean of the outcome in 1934–1938. *** p<0.01, ** p<0.05, * p<0.1.

Appendix G

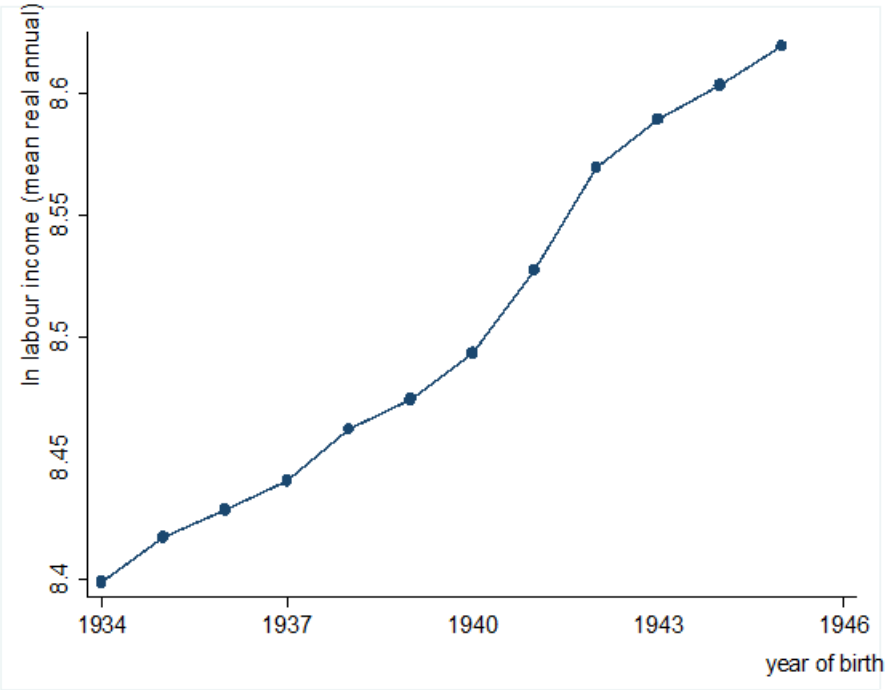


Figure – *Development of ln labour income in ages 44–60 for the cohorts 1934–1943*
Souce: SIP