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Lazuka, Volha

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Infant Health and Later-Life Labour Market Outcomes: Evidence from the Introduction of Sulpha Antibiotics in Sweden

Volha Lazuka*

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This paper studies the effects of improvements in infant health produced by the introduction of sulphapyridine in the late-1930s as treatment against pneumonia on outcomes in adulthood. Based on longitudinal individual data for the whole population of Sweden 1968–2012 and archival data on the availability of sulphapyridine and applying a difference-in-differences approach, it finds that mitigation of pneumonia infection in infancy increased labour income in late adulthood by 2.8–5.1 percent. The beneficial effects are strong for health, measured by length of stay in hospital, and weaker for years of schooling. These effects are similar between men and women.

JEL: I15, I18, J24, N34

*Volha Lazuka is a post-doctoral researcher at the Department of Economic History and the Centre for Economic Demography, Lund University, Box 7083, 220 07 Lund, Sweden (e-mail: volha.lazuka@ekh.lu.se). All appendixes are online and can be found at http://jhr.uwpress.org/. The author is grateful for comments from Tommy Bengtsson, Petter Lundborg, Anton Nilsson, Luciana Quaranta, and participants at the seminar for the Department of Economic History, Lund University, the workshop at the KORA (Copenhagen), the workshop at the Labour Institute for Economic Research (Helsinki), the European Society of Population Economics conference (Glasgow), and at the XVIIIth World

Economic History Congress (Boston). The individual-level data used in this paper are drawn from Swedish administrative registers and are confidential. However, this access is not unique and others can gain similar access by following a procedure described by Statistics Sweden https://www.scb.se/en/services/guidance-for-researchers-and-universities/. Researchers interested in obtaining this type of data could themselves apply for permission from the Ethical Review Board (ERB) at https://www.epn.se/en/start/. All processing of individual data by the researcher takes place on servers located at Statistics Sweden via secure remote terminal access. The author is willing to assist (volha.lazuka@ekh.lu.se). Models are available in the Online Appendix. For the digitisation of data, excellent assistance of Siqi Zhao is acknowledged. Financial support from Ebbe Kock foundation (Lund University, Sweden, grant EK2016-0004) and Department of Economic History (Lund University, Sweden) is gratefully acknowledged.

I. Introduction

To date, economic and demographic literature has established strong relationships between productivity growth rates and population health (Weil 2013). Recent research has shifted to identifying the causal factors behind labour productivity, among which human capital accumulation throughout the lifetime has gained increasing attention. In a framework proposed by Cunha and Heckman (2007) and Heckman (2007), health and cognitive ability in early life are considered as capacities affecting the production of future capabilities, with consequences on labour productivity later in life. Among the environmental factors strongly influencing early-life health, Finch and Crimmins (2004) suggest that decreased inflammation or exposure to infectious diseases, especially during infancy, leads directly to a decrease in chronic morbidity and mortality in adulthood. The growing empirical literature emphasises early stages of childhood as the time window when public or family investments to health yield the largest rates of return (see Bleakley 2010a; Almond and Currie 2011; Almond et al. 2017 for reviews).

Emerging micro-level literature that connects variations in disease environment in early life to later-life health and socio-economic status finds substantial effects. One strand of this research finds strong influences of epidemics in early life on cognitive ability and labour productivity later in life (Case and Paxson 2009; Barreca 2010; Kelly 2011). Another one combines ecological disease rates with a sharp introduction of intervention programmes to obtain plausible exogenous variation in child health. For instance, according to Bleakley (2007; 2010b), men, who benefited in childhood from the hookworm-eradication campaign (US South 1910) or malaria eradication (US 1920), had better labour market performance as adults. Similar or more moderate results from malaria eradication have been shown for developing countries (Cutler et al. 2010; Lucas 2010; Venkataramani 2012). In the US, salt iodization campaign in 1924 (Adhvaryu et al. 2018) and fluoridation of water beginning from

the 1950s (Glied and Neidell 2010) as early-life resources boosted later-life earnings among women. Bhalotra and Venkataramani (2013) find that reduced exposure to pneumonia in infancy after the introduction of sulphonamides in 1937 US led to gains in educational and labour outcomes among men. For the European context, several studies have found that health policies specifically targeting infants, in particular disease prevention, produce long-term economic effects (Bhalotra, Karlsson, and Nilsson 2016; Hjort, Sølvsten, and Wüst 2017; Lazuka 2018).

To date left unclear, epidemiological literature has suggested several biological mechanisms behind the long-lasting impact of contagion in early life. They include long-run maladaptation to environmental signals, permanent damage to the body, or chronic inflammatory responses (Finch 2007). Such processes are permanent and irreversible especially in a first year of life, making it a critical period, because of rapid development of organs and cells of the body. In the original work, Barker (1994) finds that for the cohorts who did not have access to antibiotics until late adulthood, infant mortality in a year of birth, driven by bronchitis and pneumonia, is strongly associated with subsequent chronic mortality. Recent studies have shown that the dependence of adult respiratory diseases on early-life contagion with pneumonia, largest among infants, is as strong as on adult smoking (Galobardes et al. 2008; Stocks and Sonnappa 2013). Contagion with bacteria *Streptococcus pneumoniae*, the main cause of pneumonia, among infants has been recently linked to the development of arthritis (Colebatch and Edwards 2011), diabetes (Beyerlein et al. 2016), and suggestively to cardiovascular diseases (Willerson and Ridker 2004). Early-life infection, as Landrigan et al. (2005) propose, affects the development of the brain leading to staggered cognitive and behavioural abilities.

In a history of social intervention, the introduction of sulpha antibiotics, in particular *sulphapyridine,* became the first public action that provided the population with treatment of

childhood infectious diseases. Sweden is nearly ideal to study its long-term impacts. In the 1920s–1930s, pneumonia was responsible for the bulk of deaths among infants and children and caused severe and repeated morbidities, and society offered no efficacious treatments equivalent in costs and efficiency to sulpha drugs (Ingvar 1939). In Sweden, these drugs, initially imported and later produced by the national companies, were introduced quickly after medical review and equally across regions due to the high availability of medical personnel, centralisation of drug distribution and low costs of treatment (The Nobel Foundation 1965). For the majority of the countries that adopted sulpha drugs, cohorts born after their introduction are strongly affected by WWII (e.g., Kesternich et al. 2014), while no such disruption occurred in Sweden. Moreover, unlike many other developed countries, from the 1950s, and hence into the adulthood of the affected cohorts, Sweden was characterised by the active inclusion of all population groups into the labour market, in part due to subsidized childcare, flexible working hours, and tax structure among others stimulating labour force participation of married women (Lindert 2004).

By applying a difference-in-differences approach, this paper exploits improvements in infant health, produced by the sharp arrival of sulpha antibiotics in Sweden efficient in treatment against pneumonia, as an early-life experiment and investigates its effects on labour market outcomes in the individuals' later lives. By using high-quality administrative individual data for Sweden, the current study raises the following research questions: (i) What are the long-term effects of exposure to/recovery from pneumonia in infancy (discerned through the introduction of sulpha antibiotics as efficient treatment and reduced peer infection) on labour incomes? (ii) Through what dimension of human capital do these effects operate, through health or acquired schooling, or both?

Having stated this, this study makes several contributions to the existing literature. Primarily, it adds to the emerging literature exploring the long-run economic effects of large-

scale interventions in early life (Almond and Currie 2011; Almond et al. 2017). It does so for the European context that remains underexplored. In relation to sulpha drugs, studies exist only for the US (Yayachandran, Lleras-Muney, and Smith 2010; Bhalotra and Venkataramani 2013). As another contribution, this paper employs both health and labour market outcomes thereby contributing to the discussion on the roots of economic development: whether health is an important determinant of long-run economic growth (Weil 2013). Moreover, it contributes to the multidisciplinary literature (Kuh and Ben-Shlomo 2007) by studying the effects of reduced burden from a particular infectious disease in infancy, namely acute pneumonia, and pointing to specific mechanisms leading to later-life outcomes. The rich data for Sweden, combined from multiple registers, allows me to conduct a more careful analysis than done previously. The longitudinal register-based individual data enables to obtain adult outcomes for the cohorts within the same age ranges and for both sexes. Availability of sibling links negates concerns about unobserved heterogeneity at a family level plausibly affecting the results. Both archival information on sulpha antibiotics, which is unique, and precise measures of pneumonia mortality improve the accuracy of identification. Based on the quasi-experimental design and multiple robustness analyses applied to such data, this paper finds that mitigation of pneumonia infection in infancy due to *sulphapyridine* increased labour income in late adulthood by 2.8–5.1 percent, fairly equally for men and women. The beneficial effects are strong for health, measured by length of stay in hospital, and weaker for years of schooling.

II. Arrival of *Sulphapyridine*

'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 aetiology, 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* (Cronberg 1997). Such disease is characterised by sudden onset, high fever and severe malaise among 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 (Bentley 2009). Figure 1 plots the mortality rates due to pneumonia and other important diseases for the period 1920–1950 in Sweden that clearly demonstrates its significance. In both absolute and relative terms, pneumonia is much more sizable among children, primarily infants, than among adults. In the pre-drug period, pneumonia and bronchitis were responsible for one out of five deaths among infants and amounted to 6.2 per 1000, which is an order of magnitude more than for total population (see also Appendix A). The elderly are also severely affected by pneumonia, although in their case the disease is chronic and associated with other debilitating health conditions (Cronberg 1997). Morbidity due to pneumonia, likely underestimated, accounted for more than 2.7 cases per 1000 (Statistiska Centralbyrån 1920b-1950). Abundant empirical research in medicine that was carried out worldwide prior to drugs documented the pathology of the disease, although it did little in providing an efficient cure.

[Figure 1]

By the late 1930s, scientists revealed a bacteriostatic component of sulphonamides, 'sulpha', that prevented the bacteria from multiplying, by inhibiting the synthesis of folic acid within bacteria, and did not kill it, and experiments with derivatives of sulphonamide preparations were launched elsewhere (The Nobel Foundation 1965). The production and trade of the drugs at a mass and international scale were launched, among which the major success is attributed to *sulphapyridine* (a compound of pyridine and sulphonamides) against pneumonia prepared by the May and Baker Company as M&B 693 in Britain in 1938 (Bentley 2009). Shortly, the invention of sulphonamides was awarded with the Nobel prize,

and WWII shaped their distribution across countries (The Nobel Foundation 1965). For the purposes of this study, I reviewed several archival sources in order to identify when *sulphapyridine* became widely used and how it diffused into medical practice across Sweden. Primarily, the articles published in the Swedish leading medical journal, *Läkartidningen,* indicate that sulphonamides were introduced into therapeutics against pneumonia in Sweden very quickly after their international review, in 1939. This is an example of the testimonials 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, many articles provided evidence on striking reductions in pneumonia mortality among inpatients cured with sulpha antibiotics across Swedish hospitals, at a rate by between 1/2 and 6/7 (e.g., Rahm 1939). Explicit advice to use *sulphapyridine* to treat pneumonia dominates the discussion beginning from 1939, followed by accurate instructions about dosages (Gnosspelius 1939). More specifically, 20 grams of it was needed to cure pneumonia for an adult, and 5 grams was enough for an infant, which could be purchased with recipe from any local doctor. The therapy by sulpha drugs was not only easy to administrate, but also cheap, amounting to 14 SEK for the full episode for an adult (0.4 percent of the annual labour income in industry) compared to 300–400 SEK that had to be spent on antiserum, an alternative treatment (Rahm 1939). By the early 1930s, the medical profession had become connected to the sickness funds, insuring one fourth of morbidity cases, and 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) thereby low-income families were unlikely to be deterred from the use of health care (SOU 1948).

An additional source relies on the archival materials of sulpha drug production and distribution in Sweden. Firstly, registers at local pharmaceutical companies and trade show that the imports of medical drugs increased by 50 percent and the Swedish companies began to produce preparates analogous to the M&B 693 on a mass scale in 1938–1939 (Skånes Näringslivsarkiv 1936-1945). No restrictions existed to production because sulpha compounds were available and sulpha drugs could not be patented. Secondly and most importantly, coming from the state inventory of medicaments across pharmacies in Sweden in September 1939, archival records provided information on sulpha drug distribution (Riksarkivet 1920-1967) (see Appendix B for an example). At that time, the distribution of medical drugs among hospitals and local pharmacies was strictly centralised under the auspices of state authorities (SOU 1959). These records testify the existence of *sulphapyridine* in all small geographical units of the country falling within ±2 standard deviations. Complementarily, regional pharmaceutical distribution companies in the largest cities in different parts of the country had a surplus of sulphonamides in stock.

III. Data

A. Individual-Level Data

To explore the long-run impacts of the arrival of *sulphapyridine* and reductions in pneumonia, this study uses individual-level outcome data from a number of administrative registers for individuals born between 1934 and 1943 in Sweden. I do not include the cohorts born beyond these years into the main sample because they are likely to be differentially exposed to public health and schooling reforms, and younger cohorts to the arrival of penicillin, in 1946. This paper uses data from the Swedish Interdisciplinary Panel (SIP)

hosted at the Centre for Economic Demography (Lund University), which is a combination of several administrative individual-level registers with yearly or event date from 1968 to 2012 linked through unique personal identifiers. It contains information for the total population of Sweden born 1930–1980 and their parents and siblings. 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 birth at the maternity hospital gradually became a standard in 1931–1950 (Riksskatteverket 1989). Such recording should not be problematic for this study as hospital delivery implies close proximity to the place of mother's residence, as well as regions are relatively large units. At the aggregate level, I was able to construct 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). I made a distinction between urban and rural areas in full correspondence with classification of those in the statistical yearbooks. The baseline analysis is conditional upon individuals having survived to adulthood and not migrated permanently from Sweden before 1968 or before any respective starting year of the register. Of the cohorts 1934–1943, 96 percent of first-year survivors are recorded in the database (see Appendix C).

As the main outcome, an individual's labour income is available from 1978 onwards on an annual basis through the income and taxation register (*Inkomst- och Taxeringsregistret*). For analysis, I constructed a variable for average real labour income by age interval (between ages 44 until a year prior to death or age 60) and used its logarithmic form to avoid the disproportionate influence of extreme values. Information after age 60 is not included, in order to obtain the measure of lifetime labour earnings, even though the results hold for older ages. The outcomes are constructed for the same age intervals for all cohorts to assure their equal contribution.

Other outcomes include education and health measures. I constructed the variable for education based on the population and housing census 1970 (*Folk- och Bostadsräkningen 1970*) and the education register (*Utbildningsregistret*) available from 1990 which reports information on highest completed schooling and post-schooling degrees respectively. Following Fischer, Karlsson, and Nilsson (2013), I transformed these levels of education into 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, I constructed average length of stay in hospital for ages 53–60. The cause of 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 D).

Individuals are linked to their parents through the multigenerational register (*Flergenerationsregistret*) thereby giving a unique family identifier. Due to the availability of family links (across different outcome samples, 91–94 percent are linked to mothers, and 83–86 percent to both mothers and fathers), I was able to merge socio-economic and demographic information of the family to the individual data (see Appendix C). For individuals without family links, I imputed values based on average values by parental birth cohort and municipality or county of residence. The results by sub-groups did not differ if these individuals were excluded. The parental characteristics included the following information: age of the mother obtained from population register (*Registret över Totalbefolkningen*), education of the mother, socio-economic status and sector of employment of the father obtained from population and housing census 1970 (*Folk- och Bostadsräkningen 1970*). Socio-economic status is further grouped into high (farmers, business owners, higher professionals and managers) and low (workers, military, lower

professionals and managers, and clerical and sales personnel). Descriptive statistics for the estimation samples is presented in Table 1.

[Table 1]

B. Region-of-Birth Data

Demanded by the analysis, an exposure variable for pneumonia infection is obtained for the region and year of birth. For this purpose, I used region-level mortality due to pneumonia, because morbidity data at either individual or region level are not available for the cohorts in question. To construct it, I collected annual death rates from several official statistical sources (see Appendix E). The data are available for the county of birth divided into urban and rural areas. The number of deaths is recorded for all ages jointly. I defined deaths due to pneumonia as a combination of deaths from pneumonia, bronchitis and pleurisy, all treatable with *sulphapyridine*. This was done to avoid plausible regional differences in identification of deaths due to pneumonia among respiratory tract diseases with similar symptoms, as well as because of the change in the cause-of-death nomenclature in 1931 (SOU 1959). I further constructed mortality rates by dividing the number of deaths by the mid-year population in the respective region. Figure 2 displays the constructed mortality rate from pneumonia across counties in Sweden in the pre-drug period of 1932–1936. I chose this period in order to avoid potential misreporting due to nomenclature changes in preceding years and stop before sulphonamides (first type of sulphonamides was efficient against puerperal fever and arrived in such a form to Sweden in 1937–1938) became internationally available. The use of other pre-treatment years yields similar results. The figure discerns considerable geographical variation in pneumonia death rates that ranges from 0.64 to 1.24 per 1000 mid-year population. This baseline pneumonia mortality was higher for disadvantaged regions,

measured for instance with real GDP per capita, and – not surprisingly – was higher for regions with higher infant and general mortality (see Appendix F).

[Figure 2]

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 sulphonamides and some not, allowing me to control for the effects of other factors (demographic, health care and socio-economic) that may have reduced pneumonia mortality beginning from 1939. I use infectious diseases related to childbirth, such as puerperal fever (treatable by sulphonamides, but not by *sulphapyridine*), related to the digestive system, such as typhoid fever and diarrhoea (untreatable by sulphonamides), related to the respiratory system, such as influenza and lung tuberculosis (untreatable by sulphonamides), and non-infectious diseases, such as heart disease, diabetes, and cancer (all untreatable by sulphonamides). Other childhood infectious diseases treatable by sulphonamides contributed little to general child mortality (Statistiska Centralbyrån 1920a-1950).

Additional regional-level variables were also collected from official statistical sources. They included demographic variables varying each year, such as stillbirth rate, crude birth rate, share of women in total population, share of population under age 15, share of population above age 65, 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 GDP per capita, real wage of worker, share of economically active population in agriculture, share of economically active population in industry, medical personnel per 1000, number of pharmacists per 1000, real spending on hospitals per 1000, and number of school-rooms and teachers per 1000 pupils. Mentioned earlier, I collected information on the availability of *sulphapyridine* and other types of sulphonamides in 1939,

price indices of medical drugs in 1939 and their change from the archival sources. Descriptive statistics for the regional-level data is presented in Table 2.

[Table 2]

IV. The Contemporaneous Effects

There are no empirical studies that examine the immediate impact of sulpha antibiotics on pneumonia mortality for Sweden or other countries of Europe. Descriptive studies of this kind suggest that introduction of *sulphapyridine* is associated with decline in pneumonia mortality in the late 1930s–1940s in Sweden, and afterwards is attributed to penicillin (Hemminki and Paakulainen 1976). As shown previously, beginning from the late 1930s, pneumonia mortality exhibited an irretrievable decline in level, trend and variability. There are no similar breaks in other diseases at the same time. In Appendix G, I show that between 1934–1938 (control cohorts) and 1939–1943 (treated cohorts) pneumonia mortality (per 1000) declined most substantially among infants (-1.8 deaths), compared to other age groups (e.g. -0.4 deaths in ages 1–4 or -0.3 deaths in ages 30–59). Figure 3 presents the effects of arrival of sulpha antibiotics on mortality at the aggregate level, where baseline pneumonia mortality in 1932–1936 is plotted against absolute decline in pneumonia mortality for the period until 1943. The results indicate strong convergence in aggregate mortality rates from pneumonia after arrival of sulpha antibiotics. More specifically, a one-unit higher pneumonia mortality rate in 1932–1936 is associated with 0.6 unit reduction in pneumonia mortality afterwards.

[Figure 3]

Results from parametrical analyses also suggest that substantial breaks in pneumonia mortality occurred in year 1939 (see Appendix G). Pneumonia mortality dropped significantly in 1939–1943 by around 30 percent in the level and exhibited a trend decline by

16 percent. Pointing to intervention-led decrease in contagion risk as the only mechanism, I find that decline in pneumonia mortality did not influence socio-economic or demographic indicators. In addition to reduction in pneumonia mortality, results show that the arrival of *sulphapyridine* led to an increase in influenza mortality. Doctors recognised that influenza alone was not responsive to antibiotics at that time (Malmros and Wilander 1941), and this finding could indicate that either influenza cases were prior diagnosed as pneumonia, or that deaths from competing causes were induced. While plausible measurement error is addressed extensively in the further analysis, the latter would imply that the true effect of decline in pneumonia mortality could be underestimated. The analysis shows that sulpha drugs were available in 1939 in all parts of the country in the amounts sufficient to account for the decline in pneumonia (see also Figure 2 above). Such regional distribution of drugs and their prices were not significantly related to different socio-economic characteristics of the regions of birth.

V. Empirical Strategy

This paper follows the strategy previously used to identify the long-run outcomes of exposure to certain infectious diseases targeted by nationwide rapid interventions (e.g., Bleakley 2007; Bleakley 2010b; Cutler et al. 2010; Lucas 2010; Bhalotra and Venkataramani 2013). In order to investigate the impact of infant health on later-life labour market outcomes, this paper exploits the plausibly exogenous variation due to decrease in pneumonia mortality in an individual's birth year and region of birth induced by sharp arrival of *sulphapyridine* in Sweden in 1939. More specifically, it explores the effect of *sulphapyridine* introduction exploiting two sources of variation: the relatively larger benefits for individuals born in regions characterised with higher baseline pneumonia mortality compared to individuals born in regions with lower pneumonia mortality, and varying exposure of different cohorts to the

arrival of *sulphapyridine*. Because I limited the sample to children born in 1934–1943, those born before 1939 were first treated by *sulphapyridine* at ages 1–5 (control group) and those born in 1939 and later in infancy (treated group).

Specifically, the paper estimates the following model:

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

where y_{icb} is the later-life outcome (ln labour income, years of schooling and length of stay in hospital) for individual i born in region c in year b; $post_b$ is a dummy, coded one if an individual was born in 1939–1943, zero if an individual was born in 1934–1938; $P_{pre,c}$ is the baseline pneumonia mortality rate in an individual's region of birth c; X_i is the vector of individual-level characteristics (sex in the baseline specification); δ_c are region-of-birth fixed effects, and λ_b are year-of-birth fixed effects. The parameter β in Eq.1 captures the (reduced-form, intention-to-treat) effect of the arrival of *sulphapyridine,* and if the related decrease in pneumonia in year of birth produced beneficial outcomes in adulthood, I expect to find positive coefficients for ln labour income and years of schooling and negative coefficient for length of stay in hospital. Baseline pneumonia mortality rate is a five-year average of pneumonia mortality rate (pneumonia, bronchitis and pleurisy) for years 1932–1936 obtained separately for each region and, in order to ease an interpretation, normalised by dividing by the range between 95th and 5th percentiles of pneumonia distribution in the country (0.421 deaths per 1000).

The identification strategy in this paper is valid if there are no omitted variables that correlate with both future outcomes and treatment intensity ($post_b \cdot P_{pre,c}$). Because an indicator $post_b$ turns into one for all regions of birth at the same year of birth (1939), differential timing of introduction of *sulphapyridine* into medical practice in the regions will not have an effect on treatment intensity. The identifying assumption does not hold if the introduction of *sulphapyridine* could target particular regions of birth that, nevertheless,

would develop alike (which is not the case as showed before), or if there is pre-treatment convergence across regions of births or overlapping health or schooling programmes that affect cohorts and regions of birth in a manner related to pneumonia treatment intensity. Figures in Appendix H display trends in later-life outcomes by cohort across regions of birth divided into larger areas based on baseline pneumonia mortality (at the quartiles). Before 1939, average later-life outcomes develop similarly across these pneumonic areas of birth. After introduction of *sulphapyridine* in 1939, there is evidence for convergence. The graphs therefore provide a first indication that the empirical strategy should be valid.

The intervention could initiate selective migration or fertility responses among parents of the cohorts under study. If such responses change the composition of cohorts in favour of children with high levels of human capital, this would provide an alternative explanation for the long-term results. I tackle this concern with individual and family data in Appendix I. First, I examine whether the arrival of *sulphapyridine* affected the composition of the parents, distinguishing high- versus low-resource families. I detect no systematic patterns, except for maternal education that, instead, is negatively related to treatment intensity. While the result for maternal schooling should be interpreted with caution, because the share of mothers with unknown education is substantial, the estimates probably pick up the general migration pattern, flowing away from economically disadvantaged regions, rather than the effect of *sulphapyridine*. Second, I analyse whether the drug intervention had heterogeneous effects on the completed fertility of mothers. While there are no statistically significant effects in any subsamples, the results tentatively point to the positive effects on fertility among low-resource families, and negative – for high-resource families. From these analyses, one should discern that a higher proportion of high-risk babies could be present among the treated cohorts, leading to the underestimation of the true long-term effect of the reduced pneumonia.

To provide more reliable estimates for the mitigation of pneumonia, which rule out any intervention-led changes in unobserved heterogeneity at the family level, Eq.1 was further estimated adding mother fixed effects:

$$y_{imcb} = \alpha + \beta post_b \cdot P_{pre,c} + \delta_c + \lambda_b + \eta_m + X_i + \varepsilon_{imcb}, \qquad (2)$$

where η_m are mother (biological mother) fixed effects and all other terms are defined as before. 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-of-birth fixed effects here are identified for families that report different regions of birth for their children. Such analysis is extremely strict as comparisons of the later-life effects of reduction in pneumonia mortality due to arrival of *sulphapyridine* are made only between siblings, born before versus those born during and after the intervention. The separate specifications account for the observable parental characteristics, by controlling for age of the mother, education of mother, socio-economic class and sector of employment of father.

This paper addresses the potential threat to identification in several ways. First, Eq.1 additionally introduces a set of baseline mortality rates from the most substantial infectious and non-infectious diseases (described above) in the period under analysis interacted with $post_b$. These terms should capture the effects of factors other than *sulphapyridine* that affected pneumonia mortality. Adding a larger set of diseases did not affect the results. Second, I include interactions between the array of baseline region-of-birth characteristics and $post_b$ to control for the effects of health, income and other regional factors on development of later-life outcomes across cohorts. It can be also seen as a balancing test for the covariates across regions of birth. Third, I allow for the differential trends across the broad areas of birth ranked by baseline pneumonia mortality (at the quartiles), which should control for the possible influence of unobserved factors that might have affected the development of later-life outcomes. Fourth, I fully control for pre-existing differential trends

across regions of birth by allowing for the region-specific linear time trends (introduction of quadratic trends produces identical results).

An important step in the analysis is the event-study analyses. In the event study specification, I replace the compound indicator for all affected cohorts born 1939–1943 with the year-of-birth indicators of the baseline pneumonia mortality and study their impacts on later-life outcomes. It allows me to explore several issues: the existence of the long-term effects due to the arrival of *sulphapyridine* for individuals treated in other age groups (not older than age 5), and the existence of mean-reverting shocks or pre-treatment differences in later-life outcomes. Importantly, I run such analysis not only for baseline pneumonia mortality as a treatment indicator, but also for baseline mortality from control diseases. The model is estimated as follows:

$$y_{icb} = \alpha + \sum \beta_b post_b \cdot P_{pre,c}\,(D_{pre,c}) + \delta_c + \lambda_b + X_i + \varepsilon_{icb}, \qquad (3)$$

where β_b denotes the cohort-specific impact of baseline pneumonia P_{pre}. The latter is further replaced with baseline mortality rates from control diseases ($D_{pre,c}$: puerperal fever, typhoid fever, diarrhoea, influenza, lung tuberculosis, heart disease, diabetes and cancer; normalised by its 95th–5th percentiles respectively) on later-life outcomes. The arrival of *sulphapyridine* should show up as a shift in the outcomes for the cohorts born in 1939–1943 for baseline pneumonia indicator. The presence of a similar pattern for any other disease could raise concern that the effect of other factors or a pre-treatment convergence process was captured.

VI. Results

A. Main Results

I start by descriptively analysing the difference in the later-life outcomes between the treated and control cohorts (exposed to *sulphapyridine* below age 1 versus those at ages 1–5) for each region of birth. Figure 4 presents the results for the related outcomes under study,

including ln labour income, completed years of schooling and average length of stay in hospital. In the graphs, absolute change in the outcome between cohorts is plotted against region-of-birth baseline pneumonia mortality rates. As shown, consistent with expectations and mirroring the contemporaneous pattern, regions of births with higher baseline pneumonia mortality exposure exhibit larger improvements in all adult outcomes.

[Figure 4]

I proceed to parametrical analyses first with the results from the baseline specification (Eq.1) for individual-level outcomes under study, both sexes jointly and separately. The results presented in Table 3 suggest statistically significant beneficial impacts of reduced exposure to pneumonia in year of birth, due to the arrival of *sulphapyridine*, on ln labour income, completed years of schooling and length of stay in hospital, each observed in late adulthood. The baseline pneumonia mortality has already been normalised by the gap between the 95th and 5th percentiles of its distribution across regions of birth, so the estimates are easily interpretable. The respective sizes of the reduced-form effects due to the introduction of *sulphapyridine* for the outcomes of all sexes jointly are the following: labour income – 4.3 percent increase; years of schooling – 0.148 years increase (1.6 percent of pre-treatment level); and length of stay in hospital – 0.042 nights decrease (5.4 percent of pre-treatment level). The results for men and women separately are fairly equal and not statistically different from each other.

[Table 3]

Table 4 demonstrates the estimates for the specifications which include mother fixed effects. The estimates in Panel A for the baseline specification first show that, in terms of the effects, the sample with known mothers is not different from the full sample. Based on Eq.2, the within-mother comparison (Panel B) confirms the previous findings for all outcomes. Moreover, the magnitude of the estimates for ln labour income and years of schooling

becomes larger. Any heterogeneity of family responses in response to intervention is now controlled for, but it is also possible that parents reinforced the inputs into infant health endowments. In fact the outcomes of siblings from families of stayers are better compared to the allocated ones, implying that reasons other than better prospective health of children in response to arrival of *sulphapyridine* determined the migration of parents. The coefficient for length of stay in hospital becomes marginally insignificant although its size is identical to other specifications. Again, controlling for observable father and mother characteristics improves the estimates, both in terms of the magnitude and their statistical significance (Panel C).

[Table 4]

The estimated effects of pneumonia decline due to *sulphapyridine* are further checked against alternative explanations. I start by accounting for the effects of different region-of-birth observable and unobservable characteristics in the models, and show the results in Table 5. The inclusion of either controls for the breaks in mortality rates in diseases other than pneumonia (Columns 1), or breaks in region-of-birth socio-economic, health care and demographic characteristics (Columns 2) keeps all results statistically significant and changed marginally compared to the baseline specification. In each specification, such added controls are jointly statistically significant (measured with F-test, at 5 percent statistical significance level). It means that they jointly had independent effects on later-life labour income or affected composition of cohorts, although they do not harm the independent effect of pneumonia reduction. When I control for pre-existing differential trends across broad pneumonic areas of birth (Columns 3) or regions-of birth (Columns 4), the effects for ln labour income (varies between 3.0 and 5.1 percent) and length of stay in hospital (varies between -0.054 and -0.041 nights), except for years of education, slightly reduce in magnitude albeit stay statistically significant, at least at the 10 percent level. The effect for

the educational variable reduces from 0.148 to 0.047 years, although it consistently remains statistically significant for women.

[Table 5]

The results for the event study analyses for reduction in pneumonia and control diseases, based on Eq.3, are presented for outcomes under study in Figures 5–7. In line with the a-priori expectations, in case of ln labour income and length of stay in hospital, for the pre-sulpha cohorts, prior to 1939, coefficients fluctuate around zero and never attain statistical significance. Starting from 1939 birth cohort until the last studied cohort, coefficients change rapidly indicating beneficial impact of the introduction of sulpha antibiotics on these outcomes. For the control diseases, I detect no similar patterns; for ln labour income, the results rather indicate that any changes in unobserved health, health care or socio-economic characteristics produced the effects in the direction opposite to the effect of *sulphapyridine*. Regarding the years of schooling, consistent with previous results, I detect the presence of some differences for the pre-treatment cohorts as well as a similar pattern in reduction in diarrhoea mortality, both pointing to pre-treatment convergence. Despite this, the treatment cohorts still enjoy the larger net effects on schooling.

[Figures 5–7]

B. Mechanisms

To explore possible biological mechanisms linking early-life exposure to pneumonia to later-life outcomes, I present the reduced-form estimates of the effects of *sulphapyridine* efficient against pneumonia on length of stay in hospital, by cause of admission, in Table 6. Consistently with empirical literature linking early-life pneumonia to specific chronic morbidities, statistically significant and sizable effects emerge from hospitalisations due to cardiovascular diseases, diabetes and degenerative diseases not specified in other groups. The

magnitudes of the effects are as follows (for the specification with region-of-birth linear time trends): cardiovascular diseases -0.044 nights (7.4 percent of pre-treatment level); diabetes -0.015 nights (16.7 percent of pre-treatment level) and degenerative diseases -0.090 nights (7.6 percent of pre-treatment level). Such strong effects for degenerative diseases are as expected because this group mainly comprises symptoms of respiratory systems and arthritis. The effects are equal between the sexes for almost all outcomes, except for somewhat stronger beneficial effects for men with regard to hospitalisation due to cardiovascular diseases and stronger beneficial effects for women in hospitalisations due to diabetes.

[Table 6]

Regarding economic mechanisms, rather tentatively, I study the contribution of quantity and returns to an individual's schooling to labour income gains. To do this, I rerun the model for ln labour income in Eq.1 while including years of schooling and present the results in Table 7. While keeping in mind that schooling rose in response to the introduction of *sulphapyridine*, the inclusion of schooling accords with our expectation and leads to a decrease in pneumonia exposure coefficient for ln labour income. In relative terms, the results suggest that the increase in years of schooling driven by the reduction of pneumonia accounts for 30–33 percent of the labour productivity results, with no clear differences between sexes. I further investigated whether returns to schooling increased in response to medical intervention (by interacting the treatment intensity with years of schooling), and find no significant effects. The rest of the labour productivity gains produced by the arrival of *sulphapyridine* (67–70 percent of the effect) can be therefore attributed to the direct effect of general and cognitive health. These results are equivalent to those incorporating a squared term for an individual's working experience, in a Mincer equation. So, the early-life effects are mostly driven by health capital accumulation, and I find that they are universal across

individuals with different background characteristics, with somewhat larger effects for those with poor background (see Appendix J).

[Table 7]

C. Plausible Measurement Error

The measure of pneumonia exposure could possibly be subject to systematic measurement error biasing the results towards zero. Table 8 presents the results for several checks that I perform to address this concern (and Appendix K contains more). These robustness checks presented below in general produce estimates which are even larger compared to those reported in the main body of the paper, suggesting that the main estimates could be seen as conservative.

[Table 8]

In the treatment indicator, among the pneumonia deaths I included pneumonia, bronchitis and pleurisy, both acute and chronic, thereby attempting to diminish the plausible regional differences (e.g., between poor and wealthy regions) in registration of particular cause of death due to respiratory tract diseases. On the other hand, in such a form this indicator comprises chronic diseases and other respiratory diseases, and thus might not measure accurately the cases of pneumonia most responsible to the arrival of *sulphapyridine*. I therefore construct the baseline pneumonia mortality while including only deaths due to acute pneumonia and bronchitis and use this indicator instead in the models (Panel A). One more 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-drug period. Instead, I use maternal place of residence from population and housing census 1960 (*Folk- och Bostadsräkningen 1960*) as an indicator of an individual's place of birth (correlation between mother's residence in 1960 and place of birth

obtained from the population register is 0.880, p-value 0.000). I then use this residence indicator for assigning the baseline pneumonia mortality in the models instead (Panel B). Finally, I use the counties of birth (24 counties and Stockholm) instead of the regions of birth (Panel C).

D. Additional Robustness Analyses

In the estimation sample, the control group includes the children aged 1–5 at the introduction of *sulphapyridine*, and looking more closely with event study analyses I detected no beneficial effects for these children. I perform robustness analyses by changing the control group (see Appendix L for more details of this and other checks). First, I expand the cohorts under analysis to those born in 1932–1945, and thus expanding the control group to ages 1–7, thereby stopping before the trials with penicillin were launched across hospitals (in 1946). The results are unaffected by this check. Second, I replace the control group with those born 1925–1929 and thus aged 10–14 at the arrival of *sulphapyridine*. The dataset imposes restrictions in this regard, as control sample is restricted to individuals who had siblings born starting from 1930, and hence likely positively selected because birth intervals have a negative association with income (e.g., Bengtsson and Dribe 2014). Despite this limitation, I find beneficial effects of reduction in pneumonia mortality of sizable magnitude for all outcomes. In support of these variations, assigning placebo treatments based on earlier years than 1939 does not yield significant effects.

Similar to other studies looking at the long-term survival of cohorts treated by different socio-economic conditions in childhood (e.g., Zajacova and Burgard 2013), the bias related to selective mortality is likely to be downward in this 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, I apply a two-stage Heckman selection procedure to analyse whether selective

survival affects the estimates (Heckman 1979), and this procedure does not affect the main findings.

The main results of the paper are unlikely to be explained by other programmes or contemporaneous shocks that overlapped with the introduction of *sulphapyridine*. The children in the estimation samples in the overwhelming majority were exposed to the same compulsory schooling reforms after age 5 (Holmlund 2008; Fischer, Karlsson, and Nilsson 2013); excluding municipalities of birth (<1 percent) treated by the changes in the compulsory schooling leave the results unchanged. Because the expansion of institutions of secondary schooling and vocational training in the 1950s was smooth (Ljungberg and Nilsson 2009), the plausible effects from this educational development are likely to be already controlled by region-of-birth characteristics. The arrival of *sulphapyridine* overlapped with two public health reforms, such as the rollout of government support to maternal and child health in 1937 until its full nationwide coverage in 1960 (Ström 1946) and the gradual expansion of hospital births in 1925–1950 (Vallgårda 1996). In both cases, the correlation between the region-of-birth baseline pneumonia mortality and proportion of infants covered by the policy is too weak to affect the results. During the WWII, Sweden was neutral, but there were regional problems with supply of food and fuels; controlling for the regional price indices for main food products does not affect the results.

I find that the effects of reduced pneumonia infection in infancy exist for other approximations of health, education and labour productivity available in the dataset (whether on disability pension, ever at hospital, total hospital admissions, more than secondary schooling, tertiary degree, ln total income, ln family income, whether employed). In addition to morbidity outcome, I run the models for mortality in ages 34–60 and detect no systematic treatment effects on mortality for the cohorts under study. I also perform the same analysis for mortality by cause of death. Consistent with previous findings for morbidity, the results

point to the beneficial effect of reduced pneumonia in infancy on probability of dying from cardiovascular disease, although it does not attain statistical significance in many specifications. For instance, for the specification with region-of-birth time trends, the reduction in pneumonia infection led to a decrease in cardiovascular mortality by 0.0057 percentage points (26.1 percent of the pre-treatment level). This result is similar if I follow individuals in their mortality outcomes until age 69.

VII. Interpretation

The estimates presented in the paper are reduced-form effects (per pneumonia mortality rates), and I can further estimate the magnitude of the effects produced by the total decline in pneumonia mortality due to *sulphapyridine* for the drug period. Figure 3 presented above gave us the estimate 0.605 (deaths per 1000) for the impact of baseline pneumonia mortality on decrease in pneumonia mortality across regions of birth. I thus scale up the reduced-form coefficients by dividing them by this value. Across different specifications, except those with alternative pneumonia indicators, arrival of *sulphapyridine* efficient in reducing pneumonia exposure in infancy produced the following effects: increase in labour income by 2.8–5.1 percent, increase in years of schooling by 0.047–0.260 years, and decrease in length of stay in hospital by 0.034–0.054 nights. From the interregional viewpoint, it helped to reduce the pre-drug gap in outcomes between the respective region-of-birth groups within the following ranges: ln labour income – 23–43 percent; years of schooling – 5–27 percent; length of stay in hospital – 33–53 percent. These results provide effects similar to those demonstrated in other micro-level studies, which otherwise find results for men only (cf. Bleakley 2010a; Bhalotra and Venkataramani 2013).

Additionally, one 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. If one relates this number to the above effects due to decline in pneumonia, it is clear that – absent the introduction of *sulphapyridine* – the labour income growth continued to develop only at the pre-drug rate. This early-life intervention thus explains 39–67% of the increase in labour income in the productive ages, and thereby suits as one of the factors behind total factor productivity that explains the bulk of income growth in this period (Schön 2004). Similar conclusions could be drawn for human capital stock: had *sulphapyridine* not arrived, growth in education and health would not have accelerated for the cohorts born after 1938.

Investments in provision of *sulphapyridine* against pneumonia among infants, which cost 150 SEK (in SEK 2016) per treatment case, yielded high societal returns. To measure the returns, I can compare the discounted increase in the individual's labour earnings over the lifetime, summed across the cohorts, with costs of treatment. For this calculation, I can rely on wage profiles for the ages 18–38 from the official wage statistics (Socialstyrelsen 1952–2012) and on the cohort- and age-specific labour incomes for the ages 39–60 from the SIP. They can be adjusted with the average of the estimates presented above (4.2 percent) for the exposure to pneumonia and discounted with the real long-term government bond yields 1939–1943 (3.4 percent, based on Waldenström 2014). Among the costs, I consider the costs of treatment of pneumonia (purchase of *sulphapyridine* to treat pneumonia and payment for a doctor's visit, no insurance considered) among the most susceptible age groups. I therefore assume that everyone got infected and underwent treatment, whereas in fact pneumonia incidence rate was much lower in the 1930s–1940s. The calculation also ignores the short-term survival and the lifetime health gains. On the other hand, the costs do not include expenses on invention and distribution of sulpha antibiotics, and only partially cover the costs of the health care system. Yet, based on these conservative numbers, social rate of return

from inception of antibiotics is large, and amounts to a ratio of 34 to 1. This affirms that health technologies with a large public good dimension have substantial economic value not only in short (e.g., Murphy and Topel 2006) but also in long run.

VIII. Conclusions

In recent years, the literature showing that early life circumstances predict health, education and socio-economic status later in life has grown substantially (Almond and Currie 2011; Almond et al. 2017). 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 2013). This study contributes to the emerging line of literature that combines infectious disease exposure in childhood with public efforts to eliminate it (e.g., Bleakley 2007) by studying the effects of exposure to pneumonia in infancy, reduced by a sudden introduction of sulpha antibiotics in Sweden, on labour productivity in late adulthood. The findings suggest the following: (1) decrease in exposure to pneumonia and its treatment in infancy led to gains in labour incomes; (2) it increased an individual's health stock substantially, which productivity also accounts for the bulk of the labour income improvements, whereas responsive increases in schooling are small. This study also links pneumonia exposure in infancy to the development of cardiovascular disease, diabetes, respiratory symptoms and arthritis in adulthood, adding to the epidemiological literature (Kuh and Ben-Shlomo 2007). These results are robust to various robustness checks, including accounting for the influence of pre-existing trends, region-specific arrival of *sulphapyridine*, compositional changes, family factors, overlapping programs and a plausible measurement error.

This study point to several conditions for the efficient implementation of a breakthrough health technology. Certain conditions surround the adoption of sulpha antibiotics. Swedish

health bodies monitored the international medical solutions against pneumonia already in the 1920s, and quickly responded to the invention with beneficial features of the health care system, such as high-quality medical personnel, low costs of treatment, and centralization of drug distribution, allowing all families to access it. Other conditions build up the realisation of the full potential of the acquired early-life benefits. In the 1920s–1930s, pneumonia mortality had a strikingly similar pattern across nations that should have similar long-term consequences. Yet, our findings for total population align well with those reported for white men in the US, characterised with high levels of accumulated human capital (Bhalotra and Venkataramani 2013). Beginning from the 1960s in Sweden, such favourable elements as, for instance, publicly provided childcare or maternity leave, public education, and the progressive taxation of wealth likely supported the realization of the effects among different population groups.

The findings of this paper have relevance for middle- and low-income countries, where the majority of early child deaths occur due to pneumonia, diarrhoea and health problems during the first month of life which could be prevented or treated with access to simple, affordable interventions (WHO 2013). The fact that low-resource families suffer more severely from these conditions, and, according to this paper, their children gain more from reduced exposure in long run, strengthens the importance of these findings even further. Pneumonia infection is also a leading morbidity cause in developed countries, especially in children under age 5, and growing antimicrobial resistance raises new challenges to public action (Rudan et al. 2013). 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 human capital accumulation and income growth in the long run.

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Tables

Table 1

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

	All	N	Men	N	Women	N
Outcomes						
Ln labour income, ages 44–60	8.133(1.399)	878,606	8.354(1.358)	446,511	7.904(1.404)	432,095
Years of schooling	9.584(2.626)	879,175	9.569(2.799)	446,736	9.600(2.436)	432,439
Length of stay in hospital, ages 53–60	0.687(2.139)	852,460	0.693(2.123)	430,096	0.681(2.157)	422,364
Family-level control variables						
Mother young (age < 29)	0.506(0.500)	878,606	0.506(0.500)	446,511	0.507(0.500)	432,095
Mother old (age ≥ 29)	0.417(0.492)	878,606	0.414(0.493)	446,511	0.419(0.493)	432,095
Mother age unknown	0.077(0.266)	878,606	0.079(0.270)	446,511	0.074(0.261)	432,095
Mother only primary schooling	0.355(0.478)	878,606	0.353(0.478)	446,511	0.356(0.479)	432,095
Mother more than primary schooling	0.220(0.414)	878,606	0.224(0.417)	446,511	0.216(0.412)	432,095
Mother schooling unknown	0.425(0.494)	878,606	0.423(0.494)	446,511	0.428(0.495)	432,095
Father high SES	0.704(0.456)	878,606	0.705(0.456)	446,511	0.703(0.457)	432,095
Father low SES	0.296(0.456)	878,606	0.295(0.456)	446,511	0.297(0.457)	432,095
Father working in agriculture	0.458(0.498)	878,606	0.458(0.498)	446,511	0.458(0.498)	432,095
Father working in industry	0.367(0.481)	878,606	0.366(0.482)	446,511	0.366(0.482)	432,095
Father working in service	0.175(0.380)	878,606	0.177(0.381)	446,511	0.174(0.379)	432,095

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

Table 2

Summary statistics for region-of-birth data

Variable	All
Pre-treatment mortality rates, per 1000, 1932-1936 (normalised)	
Pneumonia	2.459(0.328)
Puerperal fever	0.536(0.281)
Typhoid fever	1.824(0.313)
Diarrhoea	0.695(0.279)
Influenza	0.662(0.308)
Lung tuberculosis	1.066(0.307)
Heart disease	1.452(0.309)
Diabetes	0.918(0.258)
Cancer	2.184(0.299)
Region-of-birth and cohort-level control variables, 1934-1943	
Stillbirth rate, per 1000 births	26.643(5.940)
Crude birth rate, per 1000	15.516(2.931)
Share of women	0.509(0.024)
Share under age 15	0.226(0.031)
Share above age 65	0.096(0.012)
Crude death rate, per 1000	11.170(1.337)
Infant mortality rate, per 1000 live births	39.029(10.698)
Ln real regional GDP, per capita	7.390(0.184)
Ln real wage of worker	7.425(0.116)
Share of employed in agriculture	0.397(0.133)
Share of employed in industry	0.362(0.078)
Ln medical personnel, per 1000	0.146(0.419)
Ln pharmacies, per 1000	-1.496(1.052)
Ln real hospital spending, per 1000	8.824(0.384)
Ln number of school-rooms, per 1000 pupils	4.364(0.162)
Ln number of teachers, per 1000 pupils	3.883(0.092)
Sulphapyridine availability in 1939, adult doses per 1000	1.098(1.177)
Price index of medical drugs in 1939	1.716(0.213)
Change of price index of medical drug, 1940/1939	0.496(0.924)

Sources: see Appendix E.

Notes: Means and standard deviations (in parentheses). Regions-of-birth are counties divided into urban and rural areas and Stockholm (49 in total). Pre-treatment cause-specific mortality rates are per 1000 mid-year population, normalised (dividing by their 95th-5th percentile range respectively).

Table 3

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

	(1) All	(2) Men	(3) Women
Ln labour income			
post1939Xbaseline pneumonia mortality	0.0432***	0.0306	0.0554***
	(0.0151)	(0.0215)	(0.0137)
Pre-mean	8.063	8.321	7.798
Individuals	878,606	446,511	432,095
Rsq	0.031	0.003	0.010
Years of schooling			
post1939Xbaseline pneumonia mortality	0.1475**	0.1447**	0.1515**
	(0.0550)	(0.0561)	(0.0575)
Pre-mean	9.271	9.274	9.268
Individuals	879,175	446,736	432,439
Length of stay in hospital			
post1939Xbaseline pneumonia mortality	-0.0416***	-0.0462**	-0.0369*
	(0.0130)	(0.0203)	(0.0212)
Pre-mean	0.770	0.785	0.775
Individuals	852,460	430,096	422,364

Source: estimations from the *SIP*.

Notes: 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, normalised (dividing by its 95th-5th percentile range). Age interval for ln labour income is ages 44–60, and for the length of stay in hospital is ages 53–60. Models are estimated according to Eq.1. *Pre-mean* denotes mean of the outcome in 1934–1938.

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

Table 4

Reduced-form estimates with mother fixed effects or parental characteristics. Effects of pneumonia exposure in infancy on adult outcomes in Sweden, cohorts 1934–1943

	(1) Ln labour income	(2) Years of schooling	(3) Length of stay in hospital
A – Eq.1 on Mothers' sample			
post1939Xbaseline pneumonia mortality	0.0428***	0.1576***	-0.0543***
	(0.0117)	(0.0578)	(0.0141)
B – With mother fixed effects			
post1939Xbaseline pneumonia mortality	0.0471***	0.1701***	-0.0335
	(0.0149)	(0.0214)	(0.0222)
Pre-mean	8.098	9.330	0.718
Individuals	811,241	804,245	796,818
Mothers	545,318	542,422	538,951
C – With parental characteristics			
post1939Xbaseline pneumonia mortality	0.0472***	0.1998***	-0.0427***
	(0.0142)	(0.0529)	(0.0142)
mother old (ref)			
mother young	-0.0055	-0.0650***	0.0012
	(0.0058)	(0.0197)	(0.0103)
mother's age unknown	-0.3203***	-0.9922***	0.3500***
	(0.0211)	(0.0446)	(0.0255)
mother ≤ primary schooling (ref)			
mother > primary schooling	0.0662***	1.0603***	-0.0235***
	(0.0077)	(0.0655)	(0.0078)
mother schooling unknown	0.0332***	0.6139***	-0.0342***
	(0.0073)	(0.0603)	(0.0106)
father SES low (ref)			
father SES high	0.0366***	0.0694***	-0.0866***
	(0.0046)	(0.0251)	(0.0048)
father sector agriculture (ref)			
father sector industry	0.1284***	0.3643***	-0.0712***
	(0.0057)	(0.0218)	(0.0062)
father sector service	0.2130***	1.0516***	-0.0916***
	(0.0091)	(0.0325)	(0.0069)
Pre-mean	8.063	9.271	0.770
Individuals	878,606	879,175	852,460

Source: estimations from the *SIP*.

Notes: 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, and are estimated for both sexes jointly. Pneumonia mortality rate is per 1000 mid-year population, normalised (dividing by its 95th-5th percentile range). Age interval for ln labour income is ages 44–60, and for length of stay in hospital is ages 53–60. A set of Models in Panel A is estimated according to Eq.1 for the Mothers' sample. A set of Models in Panel B is estimated according to Eq.2. A set of Models in Panel C is estimated according to Eq.1 for the full sample, for both sexes jointly, plus additional controls (shown in table). *Pre-mean* denotes mean of the outcome in 1934–1938.

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

Table 5

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

	(1)	(2)	(3)	(4)	(1)	(2)	(3)	(4)	(1)	(2)	(3)	(4)
	Ln labour income				Years of schooling				Length of stay in hospital			
post1939X baseline pneumonia mortality	0.0507***	0.0279**	0.0468***	0.0296*	0.1323**	0.2599**	0.1452*	0.0469	-0.0406**	-0.0540***	-0.0458*	-0.0469*
	(0.0125)	(0.0122)	(0.0145)	(0.0154)	(0.0497)	(0.1076)	(0.0838)	(0.0351)	(0.0160)	(0.0169)	(0.0254)	(0.0245)
post1939X puerperal fever	-0.0084				0.0012				0.0074			
	(0.0173)				(0.0728)				(0.0176)			
post1939X typhoid fever	-0.0518***				-0.1180				0.0172			
	(0.0139)				(0.0990)				(0.0165)			
post1939X diarrhoea	-0.0102				0.0680				-0.0003			
	(0.0144)				(0.0883)				(0.0124)			
post1939Xinfluenza	0.0365***				0.1060*				0.0081			
	(0.0129)				(0.0529)				(0.0188)			
post1939Xlung tuberculosis	-0.0375*				-0.2276*				-0.0367			
	(0.0195)				(0.1239)				(0.0263)			
post1939Xheart disease	0.0238				0.0652				-0.0177			
	(0.0153)				(0.0773)				(0.0142)			
post1939Xdiabetes	-0.0364				-0.4139*				0.0097			
	(0.0230)				(0.2410)				(0.0262)			
post1939Xcancer	0.0049				0.1015				-0.0480***			
	(0.0140)				(0.1369)				(0.0170)			
post1939Xstillbirth rate		0.0021*				0.0053				-0.0023		
		(0.0011)				(0.0075)				(0.0019)		
post1939XCBR		-0.0043				0.0006				0.0078**		
		(0.0028)				(0.0186)				(0.0033)		
post1939Xshare women		-0.2608				2.8401				-0.0326		
		(0.3725)				(2.7454)				(0.6097)		
post1939Xshare above age 65		1.3015*				9.3500				-0.1446		
		(0.7178)				(6.4933)				(1.0766)		
post1939Xshare under age 15		0.1621				-1.0104				-0.1445		
		(0.3686)				(2.6065)				(0.6022)		
post1939XIMR		0.0006				0.0015				-0.0012		
		(0.0009)				(0.0034)				(0.0010)		
post1939Xln real regional GDP per capita		0.0959				1.6847				-0.0229		
		(0.1007)				(1.2019)				(0.1370)		
post1939Xln real worker wage		0.0372				0.9364				-0.1013		
		(0.0804)				(0.6371)				(0.1246)		
post1939Xshare in agriculture		0.0445				0.8440				0.2979		
		(0.1169)				(1.2432)				(0.2172)		
post1939Xshare in industry		-0.0024				0.2169				0.3407**		

		(0.0955)				(0.8818)				(0.1523)		
post1939Xln medical personnel per 1000		-0.0267				-0.1492				0.0042		
		(0.0191)				(0.1213)				(0.0281)		
post1939Xln pharmacies per 1000		-0.0005				-0.0599				-0.0025		
		(0.0062)				(0.0658)				(0.0130)		
post1939Xln real hospital spending per 1000		-0.0068				-0.0403				0.0227		
		(0.0191)				(0.1236)				(0.0285)		
post1939Xln schoolrooms per 1000 pupils		-0.1854***				-0.1182				0.0133		
		(0.0580)				(0.3831)				(0.0876)		
post1939Xln teachers per 1000 pupils		0.2170				0.6185				-0.1132		
		(0.1361)				(0.9908)				(0.2235)		
Pre-mean	8.063	8.063	8.063	8.063	9.271	9.271	9.271	9.271	0.770	0.770	0.770	0.770
Individuals	878,606	878,606	878,606	878,606	879,175	879,175	879,175	879,175	852,460	852,460	852,460	852,460

Source: estimations from the *SIP*.

Notes: Standard errors (in parentheses) are clustered at a region-of-birth level. Pneumonia mortality rate is per 1000 mid-year population, normalised (dividing by its 95th-5th percentile range). All models include region-of-birth fixed effects (49 regions) and year-of-birth fixed effects, and are estimated for both sexes jointly. Age interval for ln labour income is ages 44–60, and for length of stay in hospital is ages 53–60. All models are estimated according to Eq.1 plus additional controls. Models 1 additionally include disease controls (normalised, dividing by their 95th-5th percentile range respectively), such as separate interactions between *post1939* and baseline cause-specific mortality (shown in table). Models 2 additionally include interactions between *post1939* and baseline region-of-birth controls (shown in table). Models 3 additionally include interactions between baseline pneumonic regions-of-birth (divided at the quartiles based on baseline pneumonia mortality) and linear time trends. Models 4 additionally include region-of-birth linear time trends. *Pre-mean* denotes mean of the outcome in 1934–1938.

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

Table 6

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)	(5)	(6)	(7)
				Infectious/Respiratory			
post1939Xbaseline pneumonia mortality	0.0057	0.0027	-0.0047	-0.0212	-0.0295	0.0050	-0.0114
	(0.0113)	(0.0125)	(0.0143)	(0.0185)	(0.0217)	(0.0114)	(0.0210)
Pre-mean	0.274	0.274	0.274	0.274	0.274	0.274	0.263
				CVD			
post1939Xbaseline pneumonia mortality	-0.0505**	-0.0491**	-0.0497***	-0.0381	-0.0441	-0.0529**	-0.0535*
	(0.0233)	(0.0215)	(0.0178)	(0.0289)	(0.0330)	(0.0243)	(0.0293)
Pre-mean	0.600	0.600	0.600	0.600	0.600	0.600	0.582
				Diabetes			
post1939Xbaseline pneumonia mortality	-0.0085	-0.0164***	-0.0154***	-0.0155	-0.0151	-0.0089*	-0.0234**
	(0.0053)	(0.0056)	(0.0056)	(0.0142)	(0.0169)	(0.0052)	(0.0115)
Pre-mean	0.090	0.090	0.090	0.090	0.090	0.090	0.085
				Cancer			
post1939Xbaseline pneumonia mortality	-0.0013	0.0084	-0.0131	-0.0419	-0.0514*	-0.0008	0.0158
	(0.0175)	(0.0135)	(0.0166)	(0.0280)	(0.0292)	(0.0182)	(0.0292)
Pre-mean	0.471	0.471	0.471	0.471	0.471	0.471	0.438
				Degenerative			
post1939Xbaseline pneumonia mortality	-0.0971***	-0.0941***	-0.0842***	-0.1100***	-0.0899***	-0.1002***	-0.0776**
	(0.0204)	(0.0193)	(0.0228)	(0.0374)	(0.0294)	(0.0209)	(0.0343)
Pre-mean	1.189	1.189	1.189	1.189	1.189	1.189	1.174
				Mental			
post1939Xbaseline pneumonia mortality	-0.0246	-0.0387*	-0.0233	0.0213	0.0548	-0.0254	0.0090
	(0.0226)	(0.0203)	(0.0205)	(0.0505)	(0.0576)	(0.0221)	(0.0412)
Pre-mean	0.454	0.454	0.454	0.454	0.454	0.454	0.439
Individuals	852,460	852,460	852,460	852,460	852,460	852,460	796,818
Mothers							538,951

Source: estimations from the *SIP*.

Notes: 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, and are estimated for both sexes jointly. Pneumonia mortality rate is per 1000 mid-year population, normalised (dividing by its 95th-5th percentile range). Age interval for ln labour income is ages 44–60, and for length of stay in hospital is ages 53–60. Models 1 correspond to Eq.1. Models 2-7 are estimated according to Eq.1 plus additional controls. Models 2 additionally include disease controls, such as separate interactions between *post1939* and baseline cause-specific mortality. Models 3 additionally include interactions between *post1939* and baseline region-of-birth controls. Models 4 additionally include interactions between baseline pneumonic regions-of-birth (divided at the quartiles of baseline pneumonia mortality) and linear time trends. Models 5 additionally include region-of-birth linear time trends. Models 6 add family-level controls. Models 7 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

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

	(1) All	(2) Men	(3) Women
Ln labour income, years of schooling not included			
post1939Xbaseline pneumonia mortality	0.0497*** (0.0116)	0.0378*** (0.0157)	0.0615*** (0.0134)
Ln labour income, years of schooling included			
post1939Xbaseline pneumonia mortality	0.0346*** (0.0138)	0.0264 (0.0177)	0.0412*** (0.0148)
years of schooling	0.1157*** (0.0023)	0.0996*** (0.0013)	0.1375*** (0.0038)
Individuals	861,772	436,230	425,542

Source: estimations from the *SIP*.

Notes: 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, normalised (dividing by its 95th-5th percentile range). Age interval for ln labour income is ages 44–60. Models correspond to Eq.1 plus a variable shown in Table. Sample is restricted to those for whom information on completed schooling is known.

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

Table 8

Reduced-form estimates. Effects of pneumonia exposure in infancy on adult outcomes while correcting for plausible measurement error, Sweden, cohorts 1934–1943, both sexes

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
A – Lobar and broncho-pneumonia mortality as a baseline pneumonia mortality							
Ln labour income							
post1939Xbaseline pneumonia mortality	0.0477***	0.0570***	0.0331**	0.0334**	0.0350**	0.0525***	0.0517***
	(0.0162)	(0.0134)	(0.0144)	(0.0157)	(0.0158)	(0.0147)	(0.0159)
Years of schooling							
post1939Xbaseline pneumonia mortality	0.1638**	0.1567***	0.3032**	0.1259	0.0701	0.2224***	0.1765***
	(0.0629)	(0.0529)	(0.1274)	(0.0884)	(0.0463)	(0.0598)	(0.0228)
Length of stay in hospital							
post1939Xbaseline pneumonia mortality	-0.0513***	-0.0480***	-0.0711***	-0.0388	-0.0462*	-0.0538***	-0.0509*
	(0.0136)	(0.0168)	(0.0184)	(0.0238)	(0.0245)	(0.0147)	(0.0260)
B – Maternal residence in 1960 as a place of birth							
Ln labour income							
post1939Xbaseline pneumonia mortality	0.0675***	0.0608***	0.0289***	0.0840***	0.0660***	0.0718***	0.0705***
	(0.0139)	(0.0157)	(0.00869)	(0.0181)	(0.0171)	(0.0145)	(0.0172)
Years of schooling							
post1939Xbaseline pneumonia mortality	0.1123***	0.0543	0.1203***	0.0785	0.0169	0.1905***	0.1061***
	(0.0406)	(0.0344)	(0.0408)	(0.0598)	(0.0507)	(0.0476)	(0.0247)
Length of stay in hospital							
post1939Xbaseline pneumonia mortality	-0.0609***	-0.0636***	-0.0778***	-0.0873***	-0.0860**	-0.0630***	-0.0942***
	(0.0150)	(0.0139)	(0.0149)	(0.0297)	(0.0332)	(0.0155)	(0.0281)
C – County of birth as a regional unit							
Ln labour income							
post1939Xbaseline pneumonia mortality	0.0365*	0.0248	0.0152	0.0570***	0.0490***	0.0444**	0.0385***
	(0.0181)	(0.0202)	(0.0123)	(0.0185)	(0.0170)	(0.0201)	(0.0145)
Years of schooling							
post1939Xbaseline pneumonia mortality	0.0644	0.0477	0.1832***	0.0951	0.0333	0.1307*	0.1265***
	(0.0535)	(0.0651)	(0.0531)	(0.0718)	(0.0468)	(0.0712)	(0.0207)
Length of stay in hospital							
post1939Xbaseline pneumonia mortality	-0.0332**	-0.0408**	-0.0570***	-0.0614**	-0.0637*	-0.0371**	-0.0594**
	(0.0138)	(0.0150)	(0.0144)	(0.0271)	(0.0315)	(0.0157)	(0.0237)

Source: estimations from the *SIP*.

Notes: Standard errors (in parentheses) are clustered at a county-of-birth level. All models include region-of-birth fixed effects (49 regions) and year-of-birth fixed effects, and are estimated for both sexes jointly. Pneumonia mortality rate is per 1000 mid-year population, normalised (dividing by its 95th-5th percentile range). Age interval for ln labour income is ages 44–60, and for length of stay in hospital is ages 53–60. Models 1 correspond to Eq.1. Models 2–7 are estimated according to Eq.1 plus additional controls. Models 2 additionally include disease controls, such as separate interactions between *post1939* and baseline cause-specific mortality. Models 3 additionally include interactions between *post1939* and baseline region-of-birth controls. Models 4 additionally include interactions between baseline pneumonic regions-of-birth (divided at the quartiles of baseline pneumonia mortality) and linear time trends. Models 5 additionally include region-of-birth linear time trends. Models 6 add family-level controls. Models 7 are estimated according to Eq.2. Samples and pre-means for Panels A and C are as before. Samples and pre-means for Panel B: 776,362 individuals/520,833 mothers/8.105 for ln labour income; 770,123 individuals/518,307 mothers/9.344 for years of schooling: 762,358 individuals/520,833 mothers/0.712 for length of stay in hospital.

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

Figures

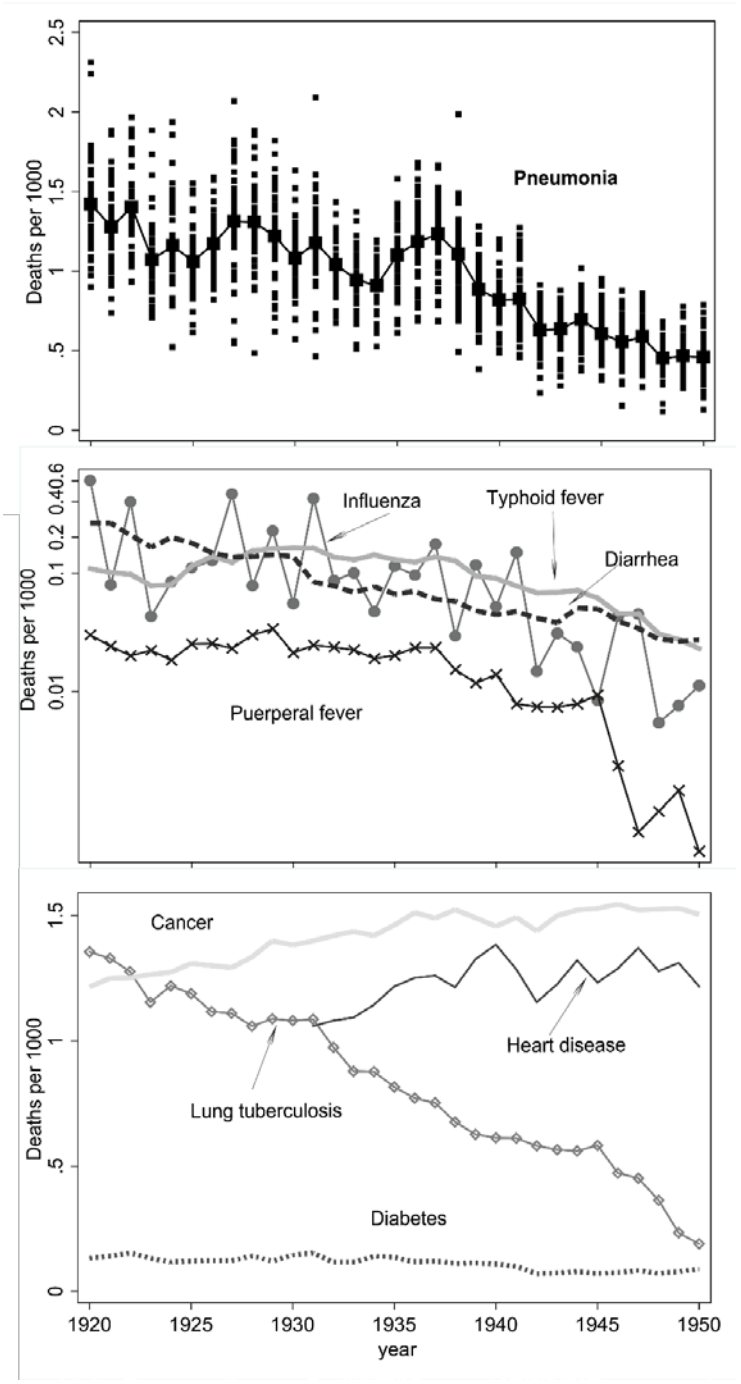

Figure 1

Mortality due to pneumonia and control diseases, Sweden 1920–1950, per 1000

Sources: own presentation based on sources from Appendix E.
Notes: The mortality rates are unadjusted arithmetic averages of mortality rates across Swedish regions (49 in total) by year. For pneumonia, both average (bold) and region-specific mortality rates (dotted) are displayed.

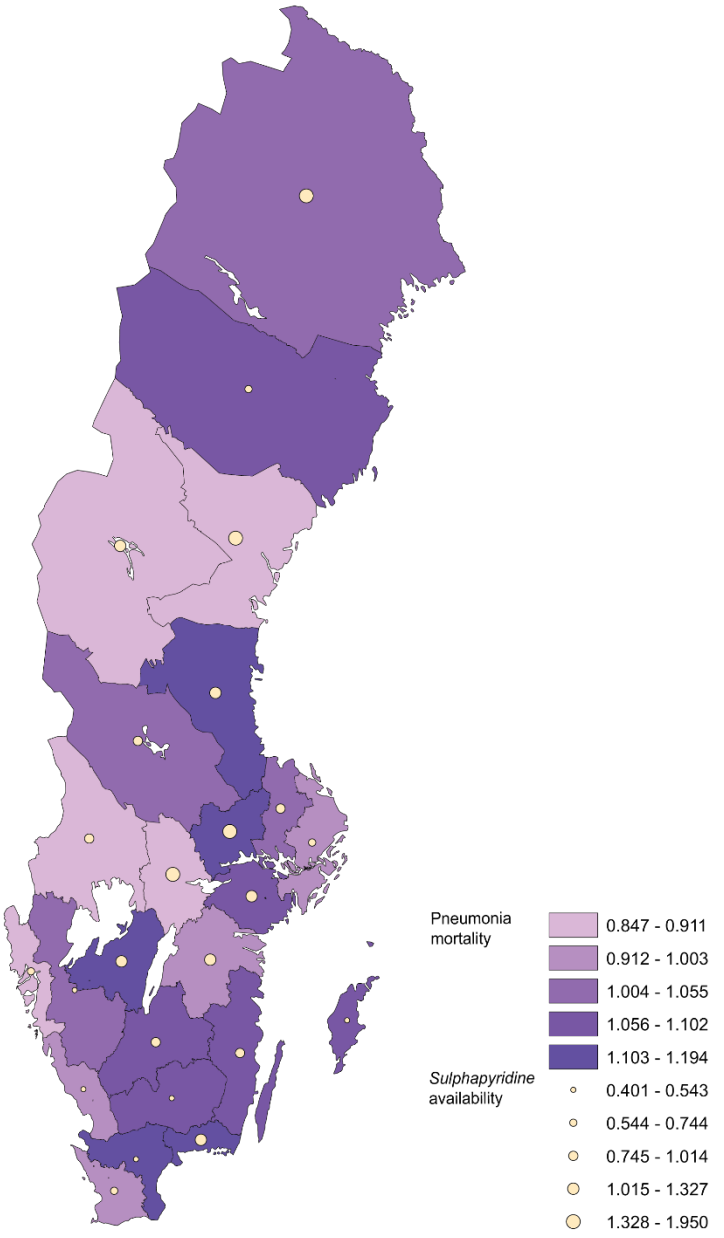

Figure 2

Geographical distribution of pneumonia mortality 1932–1936 and *sulphapyridine* in 1939, Sweden

Sources: own presentation based on sources from Appendix E; county boundaries from Riksarkivet (1932–1936)

Notes: county pneumonia mortality rates per 1000 relative to that for the country (1.043); county adult doses of *sulphapyridine* per 1000 (20 grams per pneumonia episode) relative to that for the country (1.100).

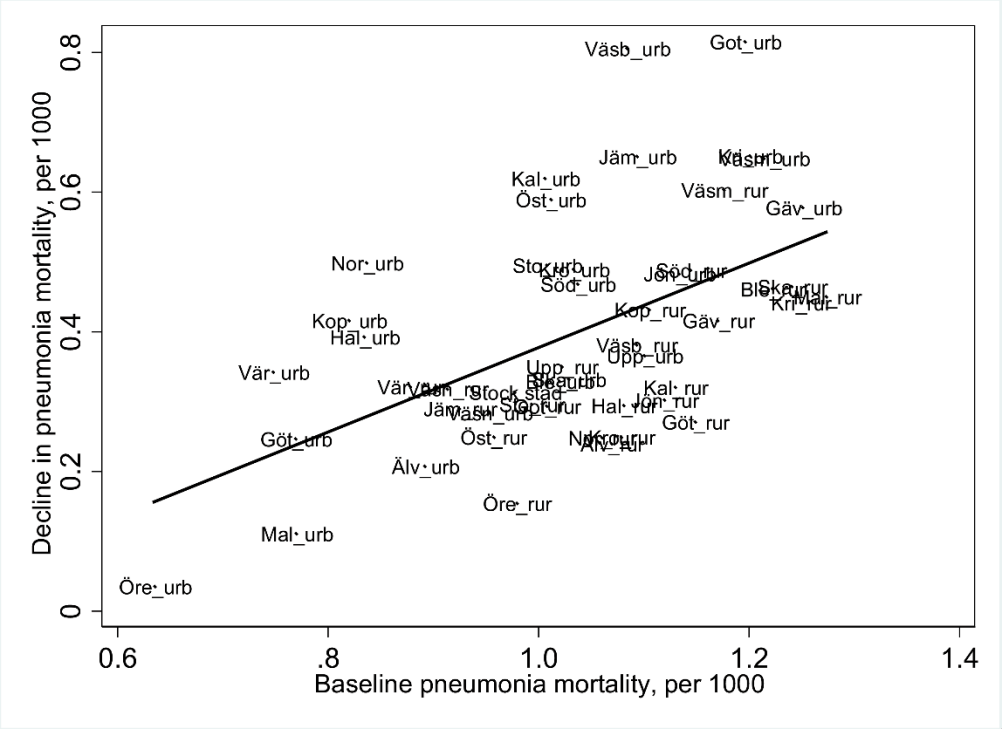

Figure 3

Convergence in pneumonia mortality rates across regions after arrival of *sulphapyridine*, Sweden

Source: own estimations based on sources from Appendix E.

Notes: Figure presents the absolute decline in pneumonia mortality rates (between 1943 and the average of 1932–1936) plotted against the pre-treatment pneumonia mortality rates (average of 1932–1936).

The estimated equation is as follows:

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

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

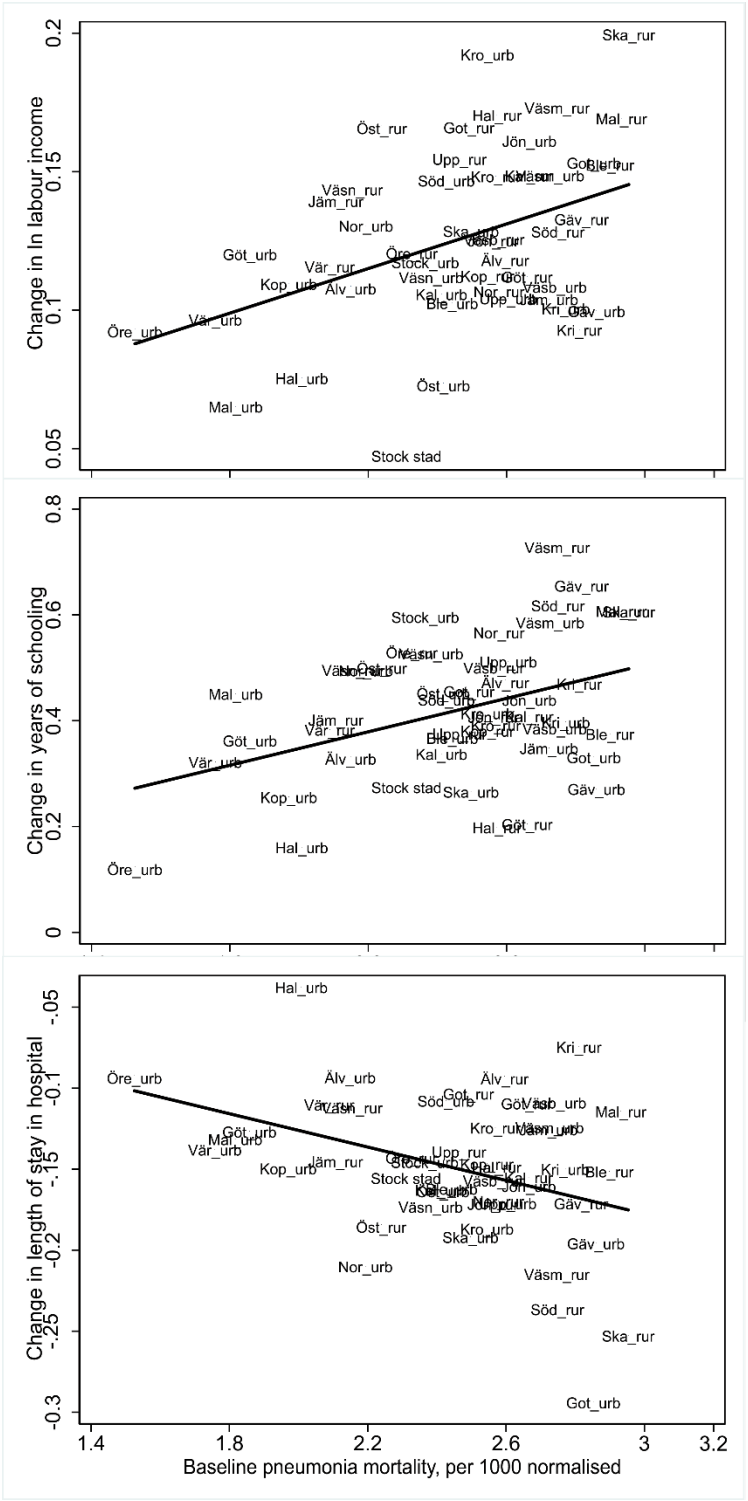

Figure 4

Convergence in later-life outcomes across regions of birth due to arrival of *sulphapyridine*, Sweden

Source: estimations from the *SIP*.

Notes: Figure presents the absolute change in the outcomes under study aggregated at region-of-birth level (between average of 1939–1943 and the average of 1934–1938) plotted against the baseline pneumonia mortality (average of 1932–1936 normalised dividing by the 95th-5th percentile range, 0.421 deaths per 1000). One outlier (rural areas of Stockholm county) is excluded from the graph; excluding it from the parametrical analysis does not affect the results.

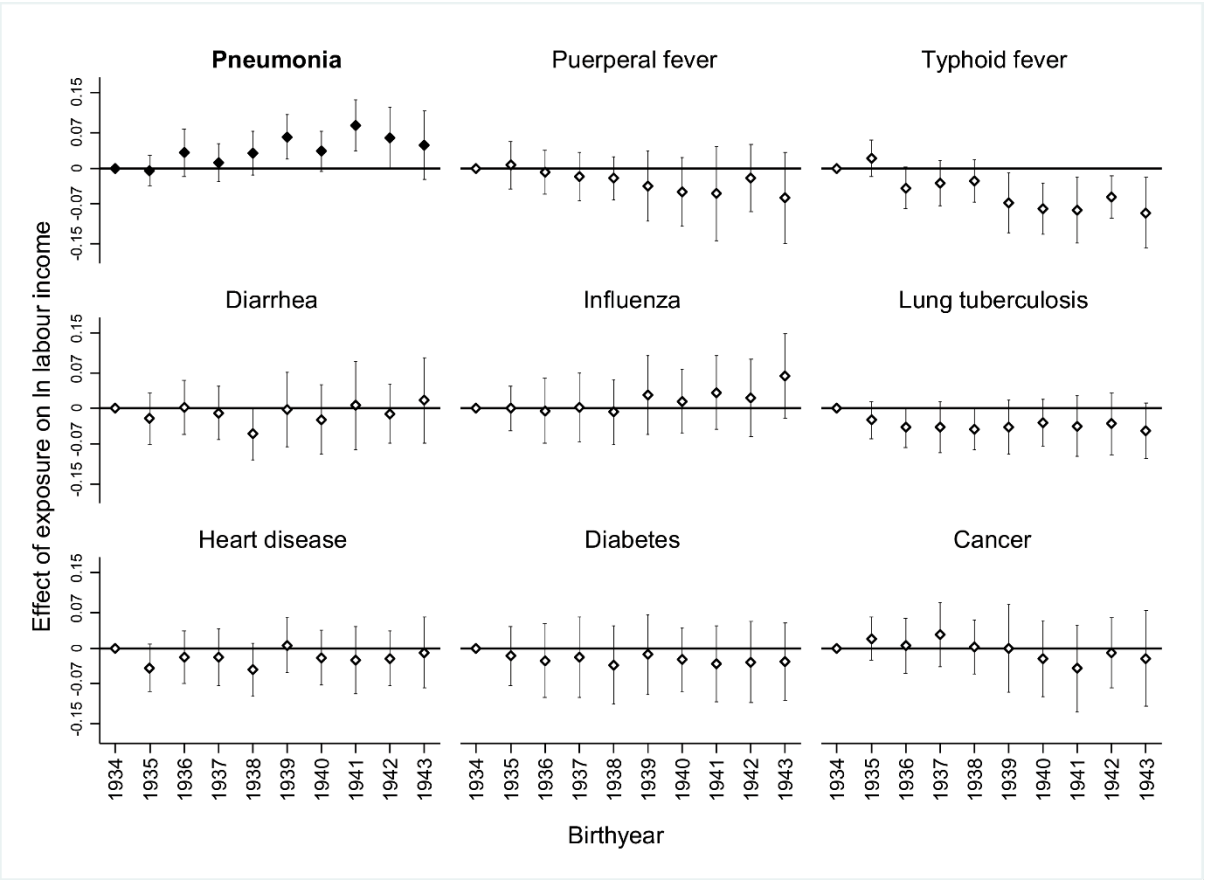

Figure 5
Ln labour income. Event study analyses of the effect of pneumonia exposure in infancy, Sweden cohorts 1934–1943

Source: estimations from the *SIP*.
Notes: Models are estimated according to Eq.3. Cohort 1934 is a reference category. Cohort 1939 is the first exposed to *sulphapyridine*. Point estimates and 95 percent CI.

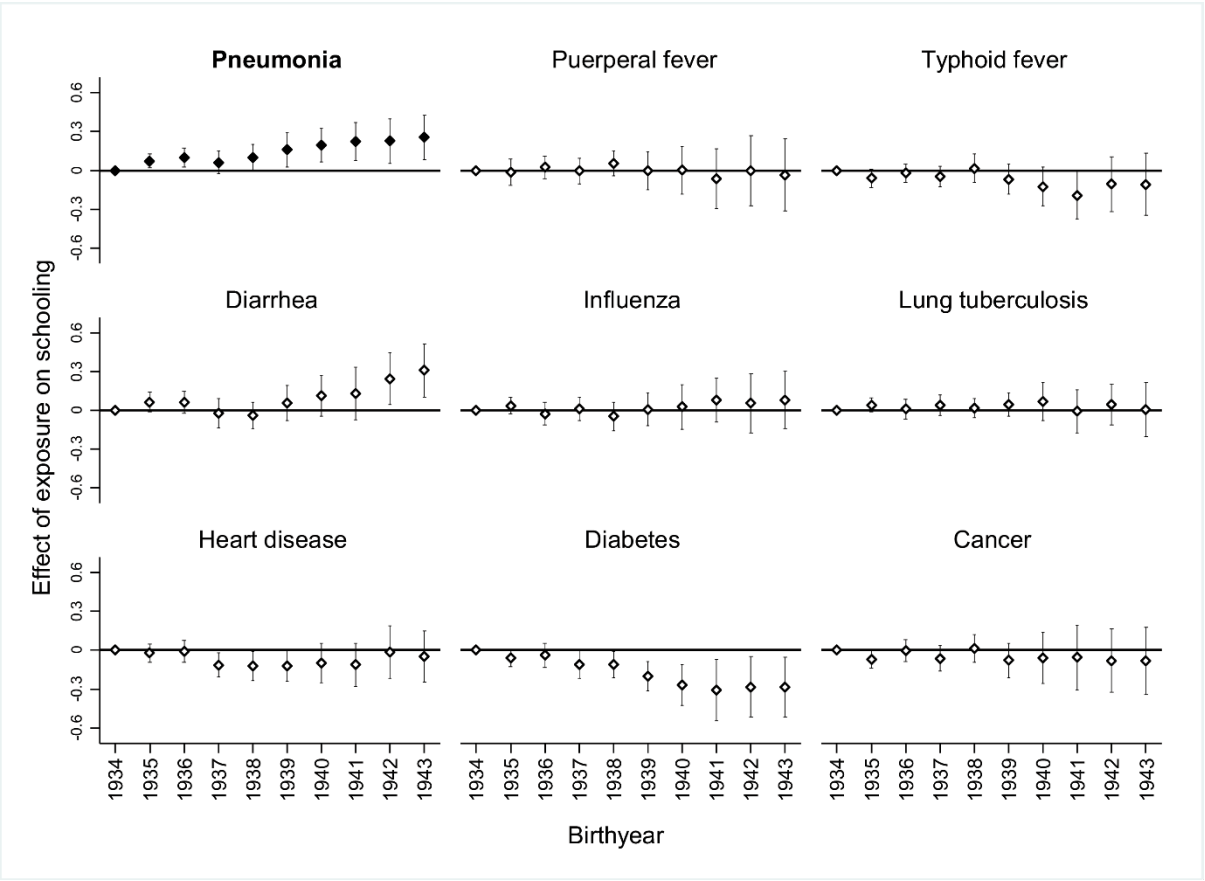

Figure 6
Years of schooling. Event study analyses of the effect of pneumonia exposure in infancy, Sweden cohorts 1934–1943

Source: estimations from the *SIP*.
Notes: Models are estimated according to Eq.3. Cohort 1934 is a reference category. Cohort 1939 is the first exposed to *sulphapyridine*. Point estimates and 95 percent CI.

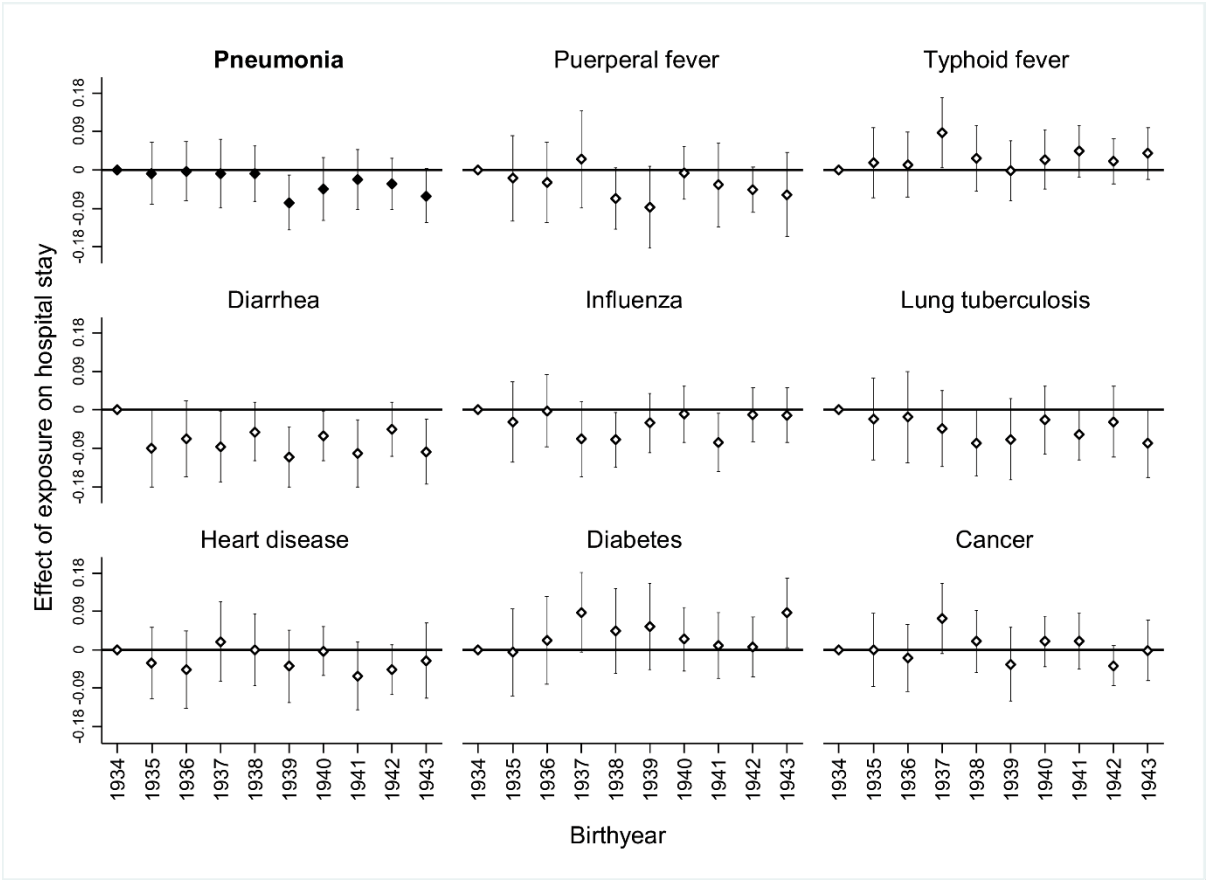

Figure 7
Length of stay in hospital. Event study analyses of the effect of pneumonia exposure in infancy, Sweden cohorts 1934–1943

Source: estimations from the *SIP*.
Notes: Models are estimated according to Eq.3. Cohort 1934 is a reference category. Cohort 1939 is the first exposed to *sulphapyridine*. Point estimates and 95 percent CI.