
Infant Health and Later-Life Labor Market Outcomes

Evidence from the Introduction of Sulfa Antibiotics in Sweden

Volha Lazuka

ABSTRACT

This study examines the effects of improvements in infant health produced by the introduction in the late 1930s of sulfapyridine as treatment against pneumonia on outcomes in adulthood. On the basis of longitudinal individual data for the whole population of Sweden 1968–2012 and archival data on the availability of sulfapyridine, I apply a difference-in-differences approach and find that mitigation of pneumonia infection in infancy increased labor income in late adulthood by 2.8–5.1 percent. The beneficial effects are strong for health, measured by length of stay in hospital, but weaker for years of schooling. These effects are similar for men and women.

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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 labor 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 for labor 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 the 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, Currie, and Duque 2018 for reviews).

Emerging microlevel literature that connects variations in disease environment in early life to later-life health and socioeconomic status finds substantial effects. One strand of this research finds strong influences of epidemics in early life on cognitive ability and labor productivity later in life (Case and Paxson 2009; Barreca 2010; Kelly 2011). Another combines ecological disease rates with a sharp introduction of intervention programs 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 (U.S. South in 1910) or malaria eradication (United States in 1920) had better labor 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 United States, the salt iodization campaign in 1924 (Adhvaryu et al. 2018) and fluoridation of water beginning in 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 in the United States led to gains in educational and labor 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 et al. 2016; Hjort, Sølvsten, and Wüst 2017; Lazuka 2018).

To date the question is unresolved, but epidemiological literature has suggested several biological mechanisms behind the long-lasting impact of infection in early life. These include long-run maladaptation to environmental signals, permanent damage to the body, and chronic inflammatory responses (Finch 2007). Such processes are permanent and irreversible, especially in a first year of life, 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 mortality from chronic diseases among survivors. 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 recently has been 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 behavioral abilities.

In a history of social intervention, the introduction of sulfa antibiotics, in particular sulfapyridine, became the first public action that provided the population with treatment of childhood infectious diseases. Sweden is nearly ideal to study the long-term impacts of the introduction of sulfa antibiotics. In the 1920s and 1930s, pneumonia was responsible for the bulk of deaths among infants and children and caused severe and repeated morbidities, but society offered no efficacious treatments equivalent in costs and efficiency to sulfa drugs (Ingvar 1939). In Sweden, after medical review, these drugs—initially imported and later produced by the national companies—were introduced quickly and equally across regions due to the high availability of medical personnel, centralization of drug distribution, and low costs of treatment (The Nobel Foundation 1965). For the majority of the countries that adopted sulfa drugs, cohorts born after their introduction were strongly affected by World War II (for example, 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 characterized by the active inclusion of all population groups into the labor market, in part due to subsidized childcare, flexible working hours, and tax structure, among others, stimulating labor force participation of married women (Lindert 2004).

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

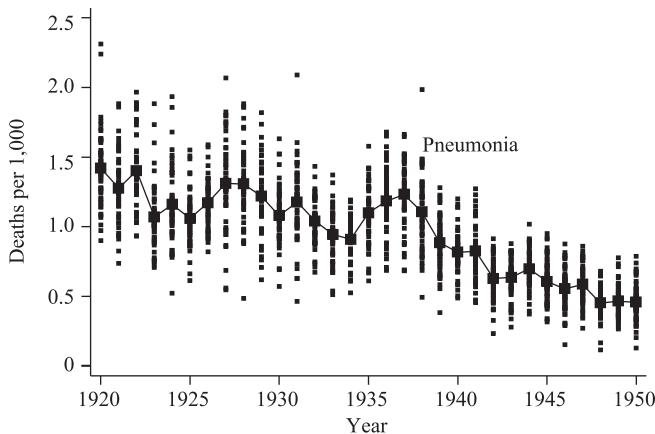
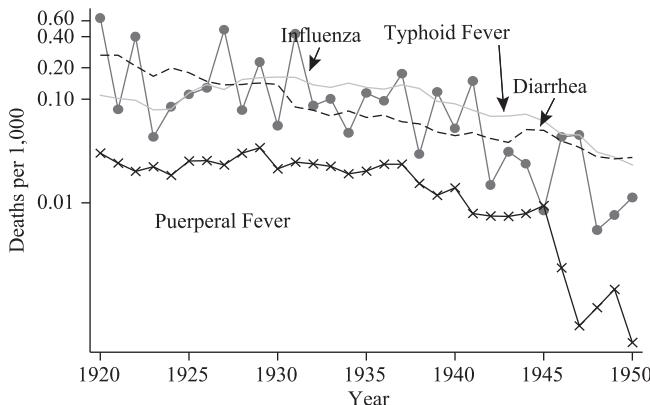
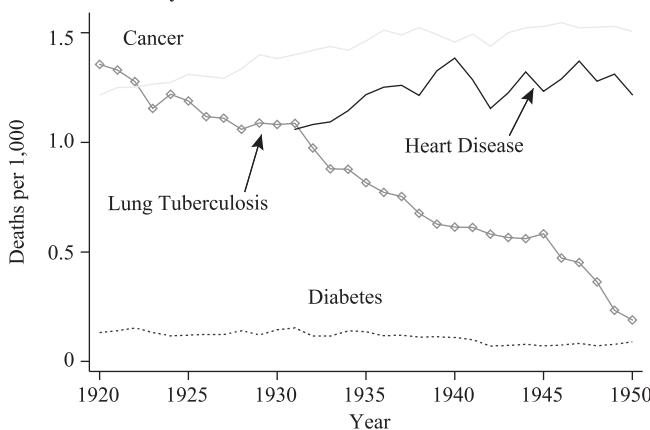
This study makes several contributions to the existing literature. Primarily, it adds to the emerging literature exploring the long-run economic effects of large-scale interventions in early life (Almond and Currie 2011; Almond, Durie, and Duque 2018). It does so for the European context, which remains underexplored. In relation to sulfa drugs, studies exist only for the United States (Yayachandran, Lleras-Muney, and Smith 2010; Bhalotra and Venkataramani 2013). As another contribution, this study employs both health and labor 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, this work 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 has been done previously. The longitudinal register-based individual data enable me 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 sulfa antibiotics, which is unique, and precise measures of pneumonia mortality improve the accuracy of identification. On the basis of the quasi-experimental design and multiple robustness analyses applied to such data, I find that mitigation of pneumonia infection in infancy due to sulfapyridine increased labor 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 Sulfapyridine

“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* (Cronberg 1997). Such disease is characterized by sudden-onset high fever and severe malaise among previously healthy individuals. In 20–30 percent of untreated cases, contagion by pneumonia leads to death, and for survivors, recovery from disease lasts approximately one month (Bentley 2009). Figure 1 plots the mortality rates due to pneumonia and other important diseases for the period 1920–1950 in Sweden, data that clearly demonstrate its significance. In both absolute and relative terms, pneumonia is much more frequent 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 1,000, which is an order of magnitude more than for the total population (see also [Online 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 1,000 (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 to provide an efficient cure.

By the late 1930s, scientists revealed a bacteriostatic component of sulphonamides, “sulfa,” that inhibited the synthesis of folic acid within bacteria, thereby preventing the bacteria from multiplying, but not killing 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, and the major success against pneumonia is attributed to sulfapyridine (a compound of pyridine and sulphonamides), prepared by the May and Baker Company as M&B 693 in Britain in 1938 (Bentley 2009). Shortly, the invention of sulphonamides was recognized with the Nobel prize, and World War II shaped the distribution of sulphonamides across countries (The Nobel Foundation 1965). For the purposes of this study, I reviewed several archival sources in order to identify when sulfapyridine 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

Panel A: Pneumonia Mortality**Panel B: Mortality from Other Communicable Diseases****Panel C: Mortality from Noncommunicable Diseases****Figure 1**

Mortality due to Pneumonia and Control Diseases, Sweden 1920–1950, per 1,000

Source: Own estimations based on sources from [Online 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.

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, page 133)

Similar to clinical trials in other countries, many articles provided evidence on striking reductions in pneumonia mortality among inpatients cured with sulfa antibiotics across Swedish hospitals, at a rate between 1/2 and 6/7 (for example, Rahm 1939). Explicit advice to use sulfapyridine to treat pneumonia dominates the discussion beginning in 1939, followed by accurate instructions about dosages (Gnospelius 1939). More specifically, 20 grams of it was needed to cure pneumonia for an adult, and five grams was enough for an infant, which could be purchased with recipe, from any local doctor. The therapy by sulfa drugs was not only easy to administer, but also affordable, amounting to 14 SEK for the full episode for an adult (0.4 percent of the annual labor 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), so low-income families were unlikely to be deterred from the use of healthcare (SOU 1948).

An additional source relies on the archival materials of sulfa drug production and distribution in Sweden. First, registers of local pharmaceutical companies and trade show that the imports of medical drugs increased by 50 percent, and the Swedish companies began to produce preparations analogous to the M&B 693 on a mass scale in 1939–1940 (Skånes Näringslivsarkiv 1936–1945). No restrictions existed to production because sulfa compounds were available, and sulfa drugs could not be patented. Second, and most importantly, coming from the state inventory of medicaments across pharmacies in Sweden in September 1939, archival records provide information on sulfa drug distribution (Riksarkivet 1920–1967) (see [Online Appendix B](#) for an example). At that time, the distribution of medical drugs among hospitals and local pharmacies was strictly centralized under the auspices of state authorities (SOU 1959). These records confirm the existence of sulfapyridine in all small geographical units of the country, falling within plus or minus two 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 sulfapyridine 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 in the main sample because they are likely to be differentially exposed to public health and schooling reforms, and, for younger cohorts, to the arrival of penicillin in 1946. This study 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 from 1968–2012 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 data set, 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 standard in 1931–1950 (Riksskatteverket 1989). Such recording should not be problematic for this study because hospital delivery implies close proximity to the place of mother's residence, and regions used 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 \times 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 on 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 [Online Appendix C](#)).

As the main outcome, an individual's labor 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 labor income by age interval (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 the measure of lifetime labor earnings, even though the results hold for older ages. The outcomes are constructed for the same age intervals for all cohorts to ensure their equal contribution.

Other outcomes include education and health measures. I constructed the variable for education based on the population and housing census for 1970 (*Folk- och Bostadsräkningen 1970*) and the education register (*Utbildningsregistret*) available from 1990, which report information on highest level of 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*), which 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 the 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 [Online Appendix D](#)).

Individuals are linked to their parents through the multigenerational register (*Fler-generationsregistret*), 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 are linked to both mothers and fathers), I was able to merge socioeconomic and demographic information of the family to the individual data (see [Online 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 subgroups did not differ if these individuals were excluded. The parental characteristics include the following information: age of the mother obtained from population register (*Registret över Totalbefolningen*), education of the mother, socioeconomic status, and sector of employment of the father obtained from the population and housing census 1970 (*Folk- och Bostadsräkningen 1970*). Socioeconomic 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 are presented in Table 1.

B. Region-of-Birth Data

Demanding 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 the 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 [Online 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 sulfapyridine. 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 to avoid confusion associated with 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 became internationally available (the first type of sulphonamides was efficient against puerperal fever and arrived in such a form to Sweden in 1937–1938). The use of other pre-treatment years yields similar results. The figure discerns considerable geographical variation in pneumonia death rates, ranging from 0.64 to 1.24 per 1,000 mid-year population. This baseline pneumonia mortality was higher for disadvantaged regions, measured with real GDP per capita, for instance, and—not surprisingly—was also higher for regions with higher infant and general mortality (see [Online Appendix F](#)).

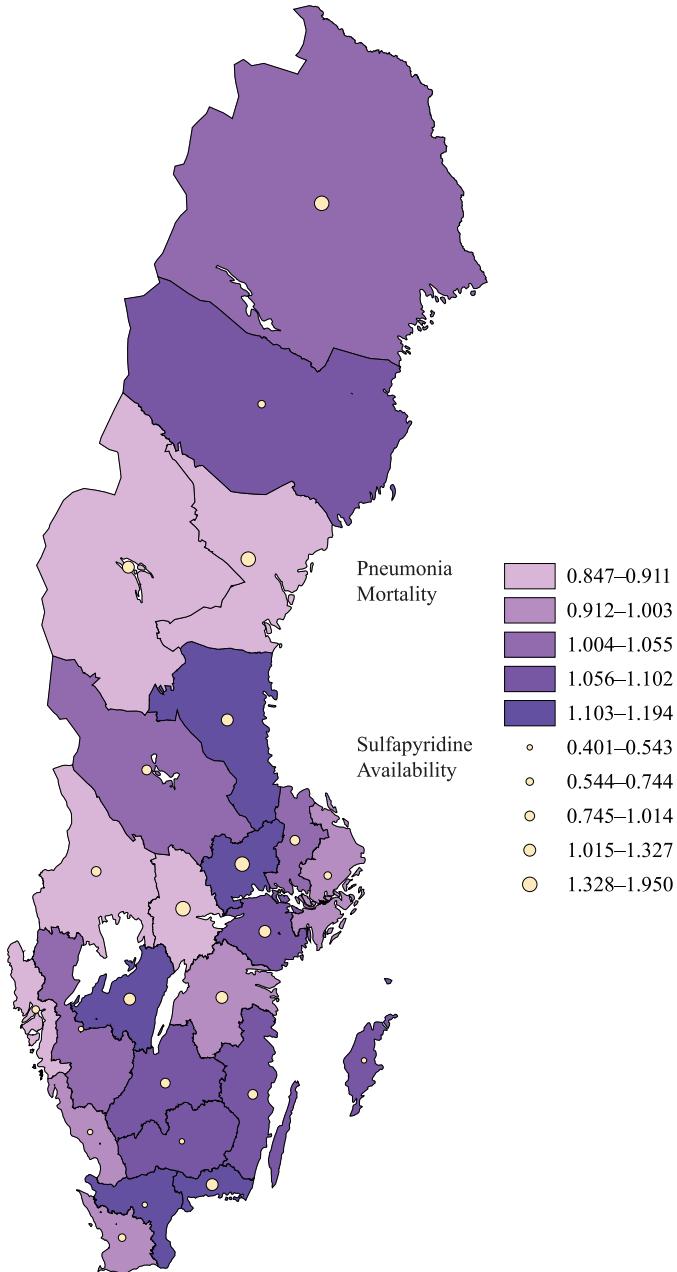
Similar sources provided information across regions on other causes of death deemed important for the analysis. These diseases represent a comparison group, some being previous to sulphonamides and some not, allowing me to control for the effects of other factors (demographic, healthcare, and socioeconomic) that may have reduced pneumonia mortality beginning in 1939. I use communicable diseases related to child-birth, such as puerperal fever (treatable by sulphonamides, but not by sulfapyridine), related to the digestive system, such as typhoid fever and diarrhea (untreatable by

Table 1

*Summary Statistics for Estimation Samples, Individual-Level Data,
Cohorts 1934–1943*

	All	N	Men	N	Women	N
Outcomes						
Ln labor 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

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

**Figure 2**

Geographical Distribution of Pneumonia Mortality 1932–1936 and Sulfapyridine in 1939, Sweden

Sources: Own estimations based on sources from [Online Appendix E](#), with county boundaries from Riksarkivet (1932–1936).

Notes: County pneumonia mortality rates per 1,000 relative to that for the country (1.043); county adult doses of sulfapyridine per 1,000 (20 grams per pneumonia episode) relative to that for the country (1.100).

Table 2
Summary Statistics for Region-of-Birth Data

Variable	All
Pre-treatment mortality rates, per 1,000, 1932–1936 (normalized)	
Pneumonia	2.459 (0.328)
Puerperal fever	0.536 (0.281)
Typhoid fever	1.824 (0.313)
Diarrhea	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 1,000 births	26.643 (5.940)
Crude birth rate, per 1,000	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 1,000	11.170 (1.337)
Infant mortality rate, per 1,000 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 1,000	0.146 (0.419)
Ln pharmacies, per 1,000	-1.496 (1.052)
Ln real hospital spending, per 1,000	8.824 (0.384)
Ln number of school-rooms, per 1,000 pupils	4.364 (0.162)
Ln number of teachers, per 1,000 pupils	3.883 (0.092)
Sulfapyridine availability in 1939, adult doses per 1,000	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 [Online 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 1,000 mid-year population, normalized (dividing by their 95th–5th percentile range, respectively).

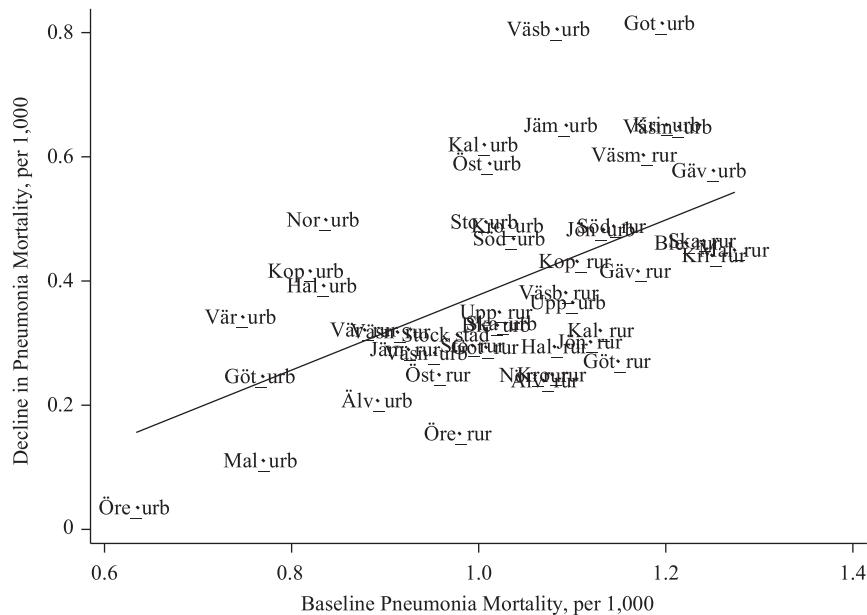
sulphonamides), related to the respiratory system, such as influenza and lung tuberculosis (untreatable by sulphonamides), and noncommunicable diseases, such as heart disease, diabetes, and cancer (all untreatable by sulphonamides). Other childhood infectious diseases treatable by sulphonamides contributed little to general child mortality (Statistiska Centralbyrån 1920a–1950).

I also collected additional regional-level variables from official statistical sources. These include 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 includes socioeconomic 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 1,000, number of pharmacists per 1,000, real spending on hospitals per 1,000, and number of classrooms and teachers per 1,000 pupils. As mentioned above, I collected information from the archival sources on the availability of sulfapyridine and other types of sulphonamides in 1939 and price indexes of medical drugs in 1939 and their change. Descriptive statistics for the regional-level data are presented in Table 2.

IV. The Contemporaneous Effects

There are no empirical studies that examine the immediate impact of sulfa antibiotics on pneumonia mortality for Sweden or other countries of Europe. Descriptive studies of this kind suggest that introduction of sulfapyridine is associated with decline in pneumonia mortality in the late 1930s–1940s in Sweden, and afterwards the decline is attributed to penicillin (Hemminki and Paakulainen 1976). As mentioned, beginning from the late 1930s, pneumonia mortality exhibited an irreversible decline, and its variability decreased substantially. There are no similar breaks in other diseases at the same time. [Online Appendix G](#) shows that for 1934–1938 (control cohorts) and 1939–1943 (treated cohorts) pneumonia mortality (per 1,000) declined most substantially among infants (−1.8 deaths) compared to other age groups (for example, −0.4 deaths for ages 1–4 or −0.3 deaths for ages 30–59). Figure 3 presents the effects of the introduction of sulfa antibiotics on mortality at the aggregate level, where baseline pneumonia mortality for 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 the arrival of sulfa antibiotics. More specifically, a one-unit higher pneumonia mortality rate in 1932–1936 is associated with 0.6 unit reduction in pneumonia mortality afterwards.

Results from parametrical analyses also suggest that substantial breaks in pneumonia mortality occurred in 1939 (see [Online Appendix G](#)). Pneumonia mortality dropped significantly in 1939–1943, by around 30 percent, and exhibited a trend decline of 16 percent. Pointing to intervention-led decrease in contagion risk as the only mechanism, I find that decline in pneumonia mortality did not influence socioeconomic or demographic indicators. In addition to reduction in pneumonia mortality, results show that the arrival of sulfapyridine led to an increase in influenza mortality. Doctors recognized that influenza was not responsive to antibiotics at that time (Malmros and Wilander 1941), and this finding could indicate that either influenza cases were previously 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.

**Figure 3**

Convergence in Pneumonia Mortality Rates across Regions after Arrival of Sulfapyridine, Sweden

Source: Own estimations based on sources from [Online 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: $\overline{\Delta Rate}_c^{post} = 0.605 \overline{BaseRate}_c - 22.75$

(0.138) (14.417) $N=49, R^2=0.29$

The analysis shows that sulfa drugs were available in 1939 in all parts of the country in 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 socioeconomic 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 (for example, Bleakley 2007, 2010b; Cutler et al. 2010; Lucas 2010; Bhalotra and Venkataramani 2013). In order to investigate the impact of infant health on later-life labor market outcomes, I exploit the plausibly exogenous variation due to decrease in pneumonia mortality in an individual's birth year and region of birth induced by the sharp arrival of sulfapyridine in Sweden in 1939. More specifically, this study

explores the effect of sulfapyridine introduction by exploiting two sources of variation: (i) the relatively larger benefits for individuals born in regions characterized with higher baseline pneumonia mortality compared to individuals born in regions with lower baseline pneumonia mortality and (ii) varying exposure of different cohorts to the arrival of sulfapyridine. Because I limited the sample to children born in 1934–1943, those born before 1939 were first treated by sulfapyridine at ages 1–5 (control group), and those born in 1939 and later were treated in infancy (treated group).

Specifically, the paper estimates the following model:

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

where y_{icb} is the later-life outcome (ln labor 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 and 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 Equation 1 captures the (reduced-form, intention-to-treat) effect of the arrival of sulfapyridine. If the related decrease in pneumonia in year of birth produced beneficial outcomes in adulthood, I expect to find positive coefficients for ln labor 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, normalized by dividing by the range between 95th and 5th percentiles of pneumonia distribution in the country (0.421 deaths per 1,000).

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 sulfapyridine into medical practice in the regions will not have an effect on treatment intensity. The identifying assumption does not hold if the introduction of sulfapyridine could target particular regions of birth that, nevertheless, would develop alike (which is not the case, as shown before) or if there is pre-treatment convergence across regions of birth or overlapping health or schooling programs that affect cohorts and regions of birth in a manner related to pneumonia treatment intensity. Figures in [Online 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 sulfapyridine in 1939, there is evidence for convergence. The graphs therefore provide a first indication that the empirical strategy is 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 favor 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 [Online Appendix I](#). First, I examine whether the arrival of sulfapyridine affected the composition of the parents, distinguishing high- versus low-resource families.

I detect no systematic patterns, except for maternal education, which 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 sulfapyridine. Second, I analyze 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 positive effects on fertility among low-resource families and negative effects 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, Equation 1 is further estimated adding mother fixed effects:

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

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 because 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 sulfapyridine 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 study addresses the potential threat to identification in several ways. First, Equation 1 additionally introduces a set of baseline mortality rates from the most substantial infectious and noninfectious diseases (described above) in the period under analysis interacted with $post_b$. These terms should capture the effects of factors other than sulfapyridine 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 also be 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 preexisting 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 analysis. 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 more issues: the existence of the long-term effects due to the arrival of sulfapyridine for individuals treated in other age

groups (not older than age five) and the existence of mean-reverting shocks or pre-treatment differences in later-life outcomes. Importantly, I run this 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:

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

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, diarrhea, influenza, lung tuberculosis, heart disease, diabetes, and cancer—normalized by its 95th–5th percentiles, respectively) on later-life outcomes. The arrival of sulfapyridine should show up as a shift in the outcomes for the cohorts born in 1939–1943 for the 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 analyzing the difference in the later-life outcomes between the treated and control cohorts (exposed to sulfapyridine below age one versus those at ages 1–5) for each region of birth. Figure 4 presents the results for the related outcomes under study, including ln labor 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 birth with higher baseline pneumonia mortality exposure exhibit larger improvements in all adult outcomes.

I proceed to parametrical analyses first with the results from the baseline specification (Equation 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 sulfapyridine, on ln labor income, completed years of schooling, and length of stay in hospital, each observed in late adulthood. The baseline pneumonia mortality has already been normalized 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 sulfapyridine for the outcomes of all sexes jointly are the following: labor income—4.3 percent increase, years of schooling—0.148 year increase (1.6 percent of pre-treatment level), and length of stay in hospital—0.042 night 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 4 demonstrates the estimates for the specifications that 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 Equation 2, the within-mother comparison (Panel B) confirms the previous findings for all outcomes. Moreover, the magnitude of the estimates for ln labor 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 sulfapyridine determined the

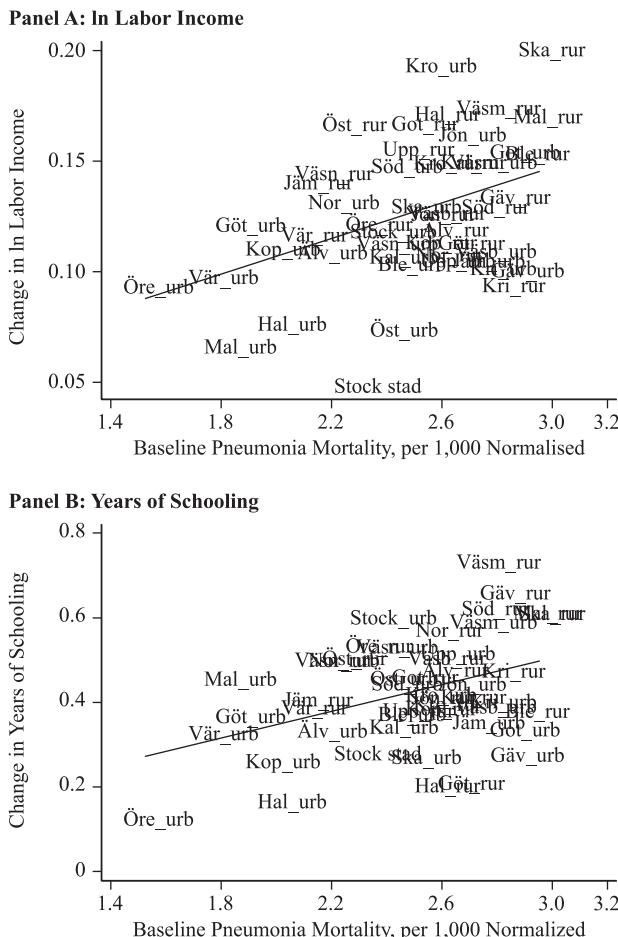


Figure 4

Convergence in Later-Life Outcomes across Regions of Birth after Arrival of Sulfapyridine, 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 normalized dividing by the 95th–5th percentile range, 0.421 deaths per 1,000). One outlier (rural areas of Stockholm county) is excluded from the graph; excluding it from the parametrical analysis does not affect the results.

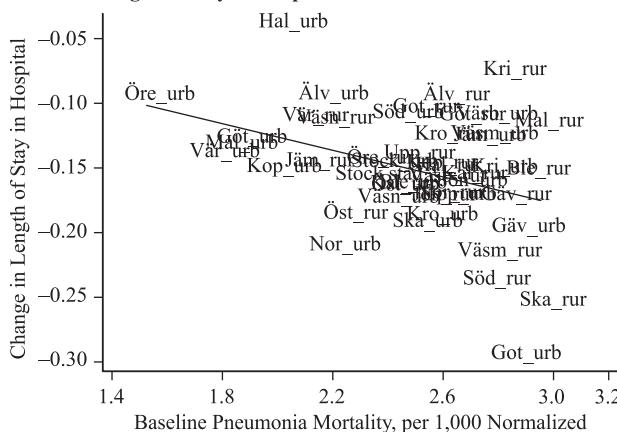


Figure 4 (continued)

Table 3

Reduced-Form Estimates. Effects of Pneumonia Exposure in Infancy on Adult Outcomes in Sweden, Cohorts 1934–1943

	All (1)	Men (2)	Women (3)
Ln labor income			
Post1939 \times baseline pneumonia mortality	0.0432*** (0.0151)	0.0306 (0.0215)	0.0554*** (0.0137)
Pre-mean	8.063	8.321	7.798
Individuals	878,606	446,511	432,095
Years of schooling			
Post1939 \times baseline pneumonia mortality	0.1475** (0.0550)	0.1447** (0.0561)	0.1515** (0.0575)
Pre-mean	9.271	9.274	9.268
Individuals	879,175	446,736	432,439
Length of stay in hospital			
Post1939 \times baseline pneumonia mortality	-0.0416*** (0.0130)	-0.0462** (0.0203)	-0.0369* (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 1,000 mid-year population, normalized (dividing by its 95th–5th percentile range). Age interval for ln labor income is ages 44–60, and for the length of stay in hospital is ages 53–60. Models are estimated according to Equation 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*

	Ln Labor Income (1)	Years of Schooling (2)	Length of Stay in Hospital (3)
Panel A: Equation 1 on Mothers' Sample			
Post1939 × baseline pneumonia mortality	0.0428*** (0.0117)	0.1576*** (0.0578)	-0.0543*** (0.0141)
Panel B: With Mother Fixed Effects			
Post1939 × baseline pneumonia mortality	0.0471*** (0.0149)	0.1701*** (0.0214)	-0.0335 (0.0222)
Pre-mean	8.098	9.330	0.718
Individuals	811,241	804,245	796,818
Mothers	545,318	542,422	538,951
Panel C: With Parental Characteristics			
Post1939 × baseline pneumonia mortality	0.0472*** (0.0142)	0.1998*** (0.0529)	-0.0427*** (0.0142)
Mother old (ref)			
Mother young	-0.0055 (0.0058)	-0.0650*** (0.0197)	0.0012 (0.0103)
Mother's age unknown	-0.3203*** (0.0211)	-0.9922*** (0.0446)	0.3500*** (0.0255)
Mother ≤ primary schooling (ref)			
Mother > primary schooling	0.0662*** (0.0077)	1.0603*** (0.0655)	-0.0235*** (0.0078)
Mother schooling unknown	0.0332*** (0.0073)	0.6139*** (0.0603)	-0.0342*** (0.0106)
Father SES low (ref)			
Father SES high	0.0366*** (0.0046)	0.0694*** (0.0251)	-0.0866*** (0.0048)
Father sector agriculture (ref)			
Father sector industry	0.1284*** (0.0057)	0.3643*** (0.0218)	-0.0712*** (0.0062)

(continued)

Table 4 (continued)

	Ln Labor Income (1)	Years of Schooling (2)	Length of Stay in Hospital (3)
Father sector service	0.2130*** (0.0091)	1.0516*** (0.0325)	-0.0916*** (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 1,000 mid-year population, normalized (dividing by its 95th–5th percentile range). Age interval for ln labor 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 Equation 1 for the mothers' sample. A set of models in Panel B is estimated according to Equation 2. A set of models in Panel C is estimated according to Equation 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. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$.

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).

The estimated effects of pneumonia decline due to sulfapyridine are further checked against alternative explanations. I begin by accounting for the effects of different region-of-birth observable and unobservable characteristics in the models and present 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 socioeconomic, healthcare, and demographic characteristics (Columns 2) keeps all results statistically significant and changing marginally compared to the baseline specification. In each specification, such added controls are jointly statistically significant (measured with F -test, at the 5 percent statistical significance level). This means that they jointly had independent effects on later-life labor income or affected the composition of cohorts, although they do not harm the independent effect of pneumonia reduction. When I control for preexisting differential trends across broad pneumonic areas of birth (Columns 3) or regions of birth (Columns 4), the effects for ln labor 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 decline in magnitude, albeit staying statistically significant, at least at the 10 percent level. The effect for the educational variable decreases from 0.148 to 0.047 years, although it consistently remains statistically significant for women.

The results for the event-study analyses for reduction in pneumonia and control diseases, based on Equation 3, are presented for the outcomes under study in Figures 5–7. In line with the a priori expectations, in the case of ln labor income and length of stay in hospital, for the pre-sulfa cohorts, prior to 1939, coefficients fluctuate around zero

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*

	Ln Labor Income				Years of Schooling				Length of Stay in Hospital			
	(1)	(2)	(3)	(4)	(1)	(2)	(3)	(4)	(1)	(2)	(3)	(4)
Post1939 × baseline pneumonia mortality	0.0507*** (0.0125)	0.0279*** (0.0122)	0.0468*** (0.0145)	0.0296* (0.0154)	0.1323*** (0.0497)	0.2599*** (0.1076)	0.1452* (0.0838)	0.0469 (0.0351)	-0.0406*** (0.0160)	-0.0540*** (0.0169)	-0.0458* (0.0254)	-0.0469* (0.0245)
Post1939 × puerperal fever	-0.0084 (0.0173)				0.0012 (0.0178)				0.0074 (0.0176)			
Post1939 × typhoid fever	-0.0518*** (0.0139)				-0.1180 (0.0990)				0.0172 (0.0165)			
Post1939 × diarrhea	-0.0102 (0.0144)				0.0680 (0.0883)				-0.0003 (0.0124)			
Post1939 × influenza	0.0365*** (0.0129)				0.1060* (0.0529)				0.0081 (0.0188)			
Post1939 × lung tuberculosis	-0.0375* (0.0195)				-0.2276* (0.1239)				-0.0367 (0.0263)			
Post1939 × heart disease	0.0238 (0.0153)				0.0652 (0.0773)				-0.0177 (0.0142)			
Post1939 × diabetes	-0.0364 (0.0230)				-0.4139* (0.2410)				0.0097 (0.0262)			
Post1939 × cancer	0.0049 (0.0140)				0.1015 (0.1369)				-0.0480*** (0.0170)			

(continued)

Post1939 × stillbirth rate	0.0021*	0.0053
	(0.0011)	(0.0075)
Post1939 × CBR	-0.0043	0.0006
	(0.0028)	(0.0186)
Post1939 × share women	-0.2608	2.8401
	(0.3725)	(2.7454)
Post1939 × share above age 65	1.3015*	9.3500
	(0.7178)	(6.4933)
Post1939 × share under age 15	0.1621	-1.0104
	(0.3686)	(2.6065)
Post1939 × IMR	0.0006	0.0015
	(0.0009)	(0.0034)
Post1939 × ln real regional GDP per capita	0.0959	1.6847
	(0.1007)	(1.2019)
Post1939 × ln real worker wage	0.0372	0.9364
	(0.0804)	(0.6371)
Post1939 × share in agriculture	0.0445	0.8440
	(0.1169)	(1.2432)
Post1939 × share in industry	-0.0024	0.2169
	(0.0955)	(0.8818)
Post1939 × ln medical personnel per 1,000	-0.0267	-0.1492
	(0.0191)	(0.1213)

Table 5 (continued)

	Ln Labor Income				Years of Schooling				Length of Stay in Hospital			
	(1)	(2)	(3)	(4)	(1)	(2)	(3)	(4)	(1)	(2)	(3)	(4)
Post1939 × In pharmacies per 1,000	-0.0005 (0.0062)				-0.0599 (0.0658)				-0.0025 (0.0130)			
Post1939 × ln real hospital spending per 1,000	-0.0068 (0.0191)				-0.0403 (0.1236)				0.0227 (0.0285)			
Post1939 × ln schoolrooms per 1,000 pupils	-0.1854*** (0.0580)				-0.1182 (0.3831)				0.0133 (0.0876)			
Post1939 × ln teachers per 1,000 pupils	0.2170 (0.1361)				0.6185 (0.9908)				-0.1132 (0.2235)			
Pre-mean Individuals	8.063 878,606	8.063 878,606	8.063 878,606	8.063 878,606	9.271 879,175	9.271 879,175	9.271 879,175	9.271 879,175	0.770 852,460	0.770 852,460	0.770 852,460	0.770 852,460

Source: Estimations from the SIP.

Notes: Standard errors (in parentheses) are clustered at a region-of-birth level. Pneumonia mortality rate is per 1,000 mid-year population, normalized (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 labor income is ages 44–60, and for length of stay in hospital is ages 53–60. All models are estimated according to Equation 1 plus additional controls. Model 1 additionally includes disease controls (normalized, dividing by their 95th–5th percentile range respectively), such as separate interactions between post1939 and baseline cause-specific mortality (shown in table). Model 2 additionally includes interactions between post1939 and baseline region-of-birth controls (shown in table). Model 3 additionally includes interactions between baseline pneumonia regions-of-birth (divided at the quartiles based on baseline pneumonia mortality) and linear time trends. Model 4 additionally includes region-of-birth linear time trends. Pre-mean denotes mean of the outcome in 1934–1938. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$.

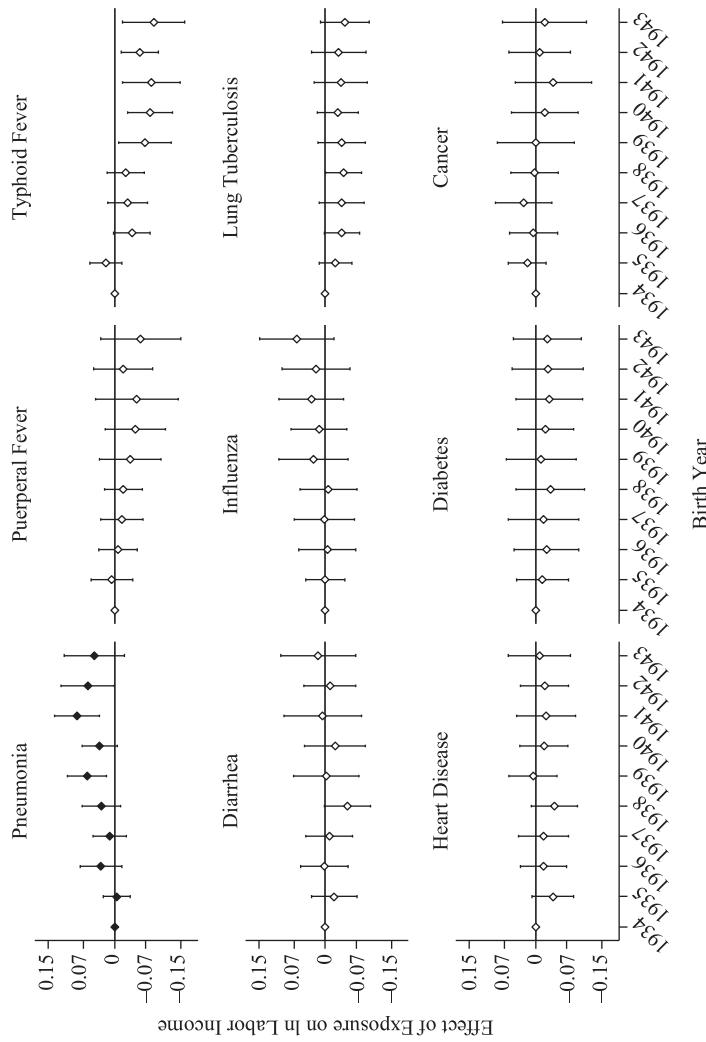


Figure 5
Ln Labor Income. Event-Study Analyses of the Effect of Pneumonia Exposure in Infancy, Sweden Cohorts 1934–1943

Source: Estimations from the SIP.

Notes: Models are estimated according to Equation 3. Cohort 1934 is a reference category. Cohort 1939 is the first exposed to sulfaipyridine. Point estimates and 95 percent confidence interval.

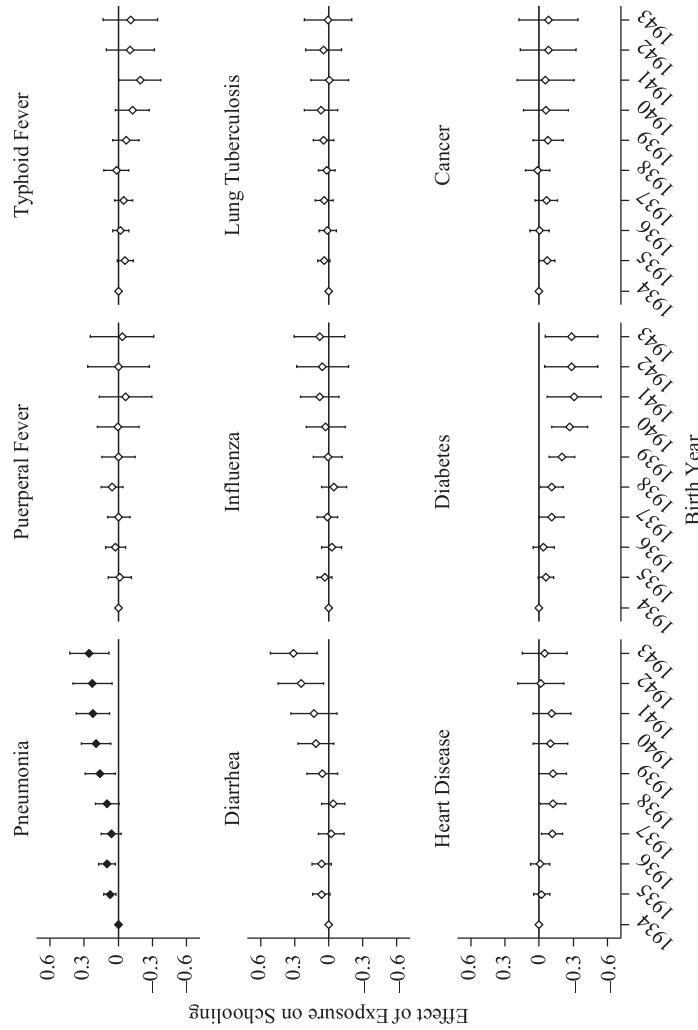


Figure 6
Years of Schooling. Event-Study Analyses of the Effect of Pneumonia Exposure in Infancy, Sweden Cohorts 1934–1943

Source: Estimations from the SIP.

Notes: Models are estimated according to Equation 3. Cohort 1939 is a reference category. Cohort 1939 is the first exposed to sulfapyridine. Point estimates and 95 percent confidence interval.

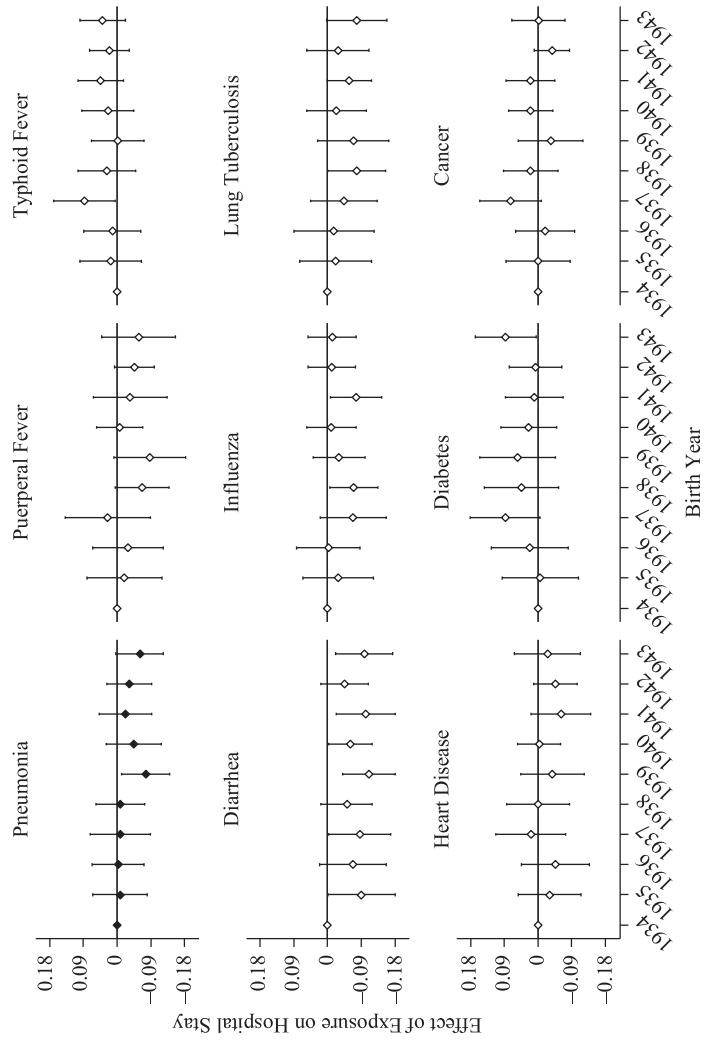


Figure 7
Length of Stay in Hospital. Event-Study Analyses of the Effect of Pneumonia Exposure in Infancy, Sweden Cohorts 1934-1943

Source: Estimations from the SIP.
 Notes: Models are estimated according to Equation 3. Cohort 1934 is a reference category. Cohort 1939 is the first exposed to sulfapyridine. Point estimates and 95 percent confidence interval.

and never attain statistical significance. Starting with the 1939 birth cohort until the last studied cohort, coefficients change rapidly, indicating beneficial impact of the introduction of sulfa antibiotics on these outcomes. For the control diseases, I detect no similar patterns. For ln labor income, the results indicate that any changes in unobserved health, healthcare, or socioeconomic characteristics produced the effects in the direction opposite to the effect of sulfapyridine. 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 diarrhea mortality, both pointing to pre-treatment convergence. Despite this, the treatment cohorts still enjoy the larger net effects on schooling.

B. Mechanisms

To explore possible biological mechanisms linking early-life exposure to pneumonia to later-life outcomes, in Table 6 I present the reduced-form estimates of the effects of sulfapyridine efficient against pneumonia on length of stay in hospital, by cause of admission. Consistently with empirical literature linking early-life pneumonia to specific chronic morbidities, statistically significant and sizable effects emerge from hospitalizations 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 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 hospitalization due to cardiovascular diseases and stronger beneficial effects for women in hospitalizations due to diabetes.

Regarding economic mechanisms, I tentatively study the contribution of quantity and returns to an individual's schooling to labor income gains. To do this, I rerun the model for ln labor income in Equation 1 while including years of schooling and present the results in Table 7. Keeping in mind that schooling rose in response to the introduction of sulfapyridine, the inclusion of schooling, as expected, leads to a decrease in the pneumonia exposure coefficient for ln labor 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 labor 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 labor productivity gains produced by the arrival of sulfapyridine (67–70 percent of the effect) can therefore be 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 these effects are universal across individuals with different background characteristics, with somewhat larger effects for those with poor background (see Online Appendix J).

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							
Post1939 × baseline	0.0057 (0.0113)	0.0027 (0.0125)	-0.0047 (0.0143)	-0.0212 (0.0185)	-0.0295 (0.0217)	0.0050 (0.0114)	-0.0114 (0.0210)
Pre-mean	0.274	0.274	0.274	0.274	0.274	0.274	0.263
Cardiovascular diseases							
Post1939 × baseline	-0.0505** (0.0233)	-0.0491** (0.0215)	-0.0497*** (0.0178)	-0.0381 (0.0289)	-0.0441 (0.0330)	-0.0529** (0.0243)	-0.0535* (0.0293)
Pre-mean	0.600	0.600	0.600	0.600	0.600	0.600	0.582
Diabetes							
Post1939 × baseline	-0.0085 (0.0053)	-0.0164*** (0.0056)	-0.0154*** (0.0056)	-0.0155 (0.0142)	-0.0151 (0.0169)	-0.0089* (0.0052)	-0.0234*** (0.0115)
Pre-mean	0.090	0.090	0.090	0.090	0.090	0.090	0.085
Cancer							
Post1939 × baseline	-0.0013 (0.0175)	0.0084 (0.0135)	-0.0131 (0.0166)	-0.0419 (0.0280)	-0.0514* (0.0292)	-0.0008 (0.0182)	0.0158 (0.0292)
Pre-mean	0.471	0.471	0.471	0.471	0.471	0.471	0.438

(continued)

Table 6 (continued)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Degenerative							
Post1939 × baseline	-0.0971*** (0.0204)	-0.0941*** (0.0193)	-0.0842*** (0.0228)	-0.1100*** (0.0374)	-0.0899*** (0.0294)	-0.1002*** (0.0209)	-0.0776*** (0.0343)
pneumonia mortality							
Pre-mean	1.189	1.189	1.189	1.189	1.189	1.189	1.174
Mental							
Post1939 × baseline	-0.0246 (0.0226)	-0.0387* (0.0203)	-0.0233 (0.0205)	0.0213 (0.0505)	0.0548 (0.0576)	-0.0254 (0.0221)	0.0090 (0.0412)
pneumonia mortality							
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 1,000 mid-year population, normalized (dividing by its 95th–5th percentile range). Age interval for in labor income is ages 44–60, and for length of stay in hospital is ages 53–60. Model 1 corresponds to Equation 1. Models 2–7 are estimated according to Equation 1 plus additional controls. Model 2 additionally includes disease controls, such as separate interactions between post1939 and baseline cause-specific mortality. Model 3 additionally includes interactions between post1939 and baseline region-of-birth controls. Model 4 additionally includes interactions between baseline pneumonia regions-of-birth (divided at the quartiles of baseline pneumonia mortality) and linear time trends. Model 5 additionally includes region-of-birth linear time trends. Model 6 adds family-level controls. Model 7 is estimated according to Equation 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 Labor Productivity Holding Education Constant, Sweden Cohorts 1934–1943

	All (1)	Men (2)	Women (3)
Ln labor income, years of schooling not included			
Post1939 × baseline pneumonia mortality	0.0497*** (0.0116)	0.0378*** (0.0157)	0.0615*** (0.0134)
Ln labor income, years of schooling included			
Post1939 × baseline 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 1,000 mid-year population, normalized (dividing by its 95th–5th percentile range). Age interval for ln labor income is ages 44–60. Models correspond to Equation 1 plus a variable shown in the table. Sample is restricted to those for whom information on completed schooling is known. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$.

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 [Online Appendix K](#) contains more). These robustness checks presented below in general produce estimates that are even larger than those reported in the main body of the paper, suggesting that the main estimates could be viewed as conservative.

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 (for example, between poor and wealthy regions) in the registration of the particular cause of death due to respiratory tract diseases. On the other hand, in this form the indicator comprises chronic diseases and other respiratory diseases and thus might not measure accurately the cases of pneumonia most responsive to the arrival of sulfapyridine. 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

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)
Panel A: Lobar and Broncho-Pneumonia Mortality as a Baseline Pneumonia Mortality							
Ln labor income							
Post1939 × baseline pneumonia mortality	0.0477*** (0.0162)	0.0570*** (0.0134)	0.0331** (0.0144)	0.0334** (0.0157)	0.0350** (0.0158)	0.0525*** (0.0147)	0.0517*** (0.0159)
Years of schooling							
Post1939 × baseline pneumonia mortality	0.1638** (0.0629)	0.1567*** (0.0529)	0.3032** (0.1274)	0.1259 (0.0884)	0.0701 (0.0463)	0.2224*** (0.0598)	0.1765*** (0.0228)
Length of stay in hospital							
Post1939 × baseline pneumonia mortality	-0.0513*** (0.0136)	-0.0480*** (0.0168)	-0.0711*** (0.0184)	-0.0388 (0.0238)	-0.0462* (0.0245)	-0.0538*** (0.0147)	-0.0509* (0.0260)
Panel B: Maternal Residence in 1960 as a Place of Birth							
Ln labor income							
Post1939 × baseline pneumonia mortality	0.0675*** (0.0139)	0.0608*** (0.0157)	0.0289*** (0.00869)	0.0840*** (0.0181)	0.0660*** (0.0171)	0.0718*** (0.0145)	0.0705*** (0.0172)
Years of schooling							
Post1939 × baseline pneumonia mortality	0.1123*** (0.0406)	0.0543 (0.0344)	0.1203*** (0.0408)	0.0785 (0.0598)	0.0169 (0.0507)	0.1905*** (0.0476)	0.1061*** (0.0247)

Length of stay in hospital						
Post1939 × baseline	-0.0609*** (0.0150)	-0.0636*** (0.0139)	-0.0778*** (0.0149)	-0.0873*** (0.0297)	-0.0860** (0.0332)	-0.0630*** (0.0155)
pneumonia mortality						
Ln labor income						
Post1939 × baseline	0.0365* (0.0181)	0.0248 (0.0202)	0.0152 (0.0123)	0.0570*** (0.0185)	0.0490*** (0.0170)	0.0444** (0.0201)
pneumonia mortality						
Years of schooling						
Post1939 × baseline	0.0644 (0.0535)	0.0477 (0.0651)	0.1832*** (0.0531)	0.0951 (0.0718)	0.0333 (0.0468)	0.1307* (0.0712)
pneumonia mortality						
Length of stay in hospital						
Post1939 × baseline	-0.0332** (0.0138)	-0.0408** (0.0150)	-0.0570*** (0.0144)	-0.0614** (0.0271)	-0.0637* (0.0315)	-0.0371** (0.0157)
pneumonia mortality						

Panel C: County of Birth as a Regional Unit

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 1,000 mid-year population, normalized (dividing by its 95th–5th percentile range). Age interval for in labor income is ages 44–60, and for length of stay in hospital is ages 53–60. Model 1 corresponds to Equation 1. Models 2–7 are estimated according to Equation 1 plus additional controls. Model 2 additionally includes disease controls, such as separate interactions between post1939 and baseline region-of-birth controls. Model 3 additionally includes interactions between post1939 and baseline cause-specific mortality. Model 4 additionally includes interactions between baseline pneumonia regions-of-birth (divided at the quartiles of baseline pneumonia mortality) and linear time trends. Model 5 additionally includes interactions between baseline pneumonia regions-of-birth and linear time trends. Model 6 adds family-level controls. Model 7 is estimated according to Equation 2. Samples and pre-means for Panels A and C are as before. Samples and pre-means for Panel B: 762,358 individuals/520,833 mothers/8,105 for ln labor income; 770,123 individuals/518,307 mothers/9,344 for years of schooling; 776,362 individuals/520,833 mothers/0.712 for length of stay in hospital.

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

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 sulfapyridine, 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 [Online 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 sulfapyridine. The data set imposes restrictions in this regard, as the 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 (for example, 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 years earlier than 1939 does not yield significant effects.

Similar to other studies looking at the long-term survival of cohorts treated by different socioeconomic conditions in childhood (for example, Zajacova and Burgard 2013), in this case the bias related to selective mortality is likely to be downward, as the weakest members of cohorts are more likely to survive in the after-drug period and be observed in the registers. To assess this formally, I apply a two-stage Heckman selection procedure to analyze whether selective survival affects the estimates (Heckman 1979), and this procedure does not affect the main findings.

The main results of the study are unlikely to be explained by other programs or contemporaneous shocks that overlapped with the introduction of sulfapyridine. The children in the estimation samples in the overwhelming majority were exposed to the same compulsory schooling reforms after age five (Holmlund 2008; Fischer, Karlsson, and Nilsson 2013); excluding municipalities of birth (<1 percent) treated by the changes in the compulsory schooling leaves 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 sulfapyridine overlapped with two public health reforms: 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 World War II, Sweden was neutral, but there were regional problems with supply of food and fuels. Controlling for the regional price indexes 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 labor productivity available in the data set

(whether on disability pension, ever at a hospital, total hospital admissions, more than secondary schooling, tertiary degree, ln total income, ln family income, and whether employed). In addition to morbidity outcome, I run the models for mortality at 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 the 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 sulfapyridine for the drug period. Across different specifications, except those with alternative pneumonia indicators, the arrival of sulfapyridine efficient in reducing pneumonia exposure in infancy produced the following reduced-form effects: an increase in labor income by 2.8–5.1 percent, an increase in years of schooling by 0.047–0.260 years, and a decrease in length of stay in hospital by 0.034–0.054 nights. Figure 3 presented above gave us the estimate 0.605 (deaths per 1,000) for the impact of baseline pneumonia mortality on decrease in pneumonia mortality across regions of birth. Using indirect least squares, I thus scale up the reduced-form coefficients by dividing them by this value that gives the following effects: an increase in labor income by 4.6–8.4 percent, an increase in years of schooling by 0.078–0.430 years, and a decrease in length of stay in hospital by 0.056–0.089 nights. From an interregional viewpoint, the arrival of sulfapyridine helped to reduce the pre-drug gap in outcomes between the respective region-of-birth groups within the following ranges: ln labor income 23–43 percent, years of schooling 5–27 percent, and length of stay in hospital 33–53 percent. These results provide effects similar to those demonstrated in other microlevel studies, which otherwise find results for men only (see Bleakley 2010a; Bhalotra and Venkataramani 2013).

Additionally, one can interpret the results from an intertemporal viewpoint. Estimated roughly from the data, the growth in labor 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 sulfapyridine—the labor income growth continued to develop only at the pre-drug rate. This early-life intervention thus explains 39–67% of the increase in labor 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 sulfapyridine not arrived, growth in education and health would not have accelerated for the cohorts born after 1938.

Investments in the provision of sulfapyridine against pneumonia among infants, which cost 150 SEK (in SEK 2016) per treatment case, yielded high societal returns. To measure the returns, I compare the discounted increase in the individual's labor earnings over the lifetime, summed across the cohorts, with costs of treatment. For this calculation, I rely on wage profiles for ages 18–38 from the official wage statistics (Socialstyrelsen 1952–2012) and on the cohort- and age-specific labor 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 sulfapyridine 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 sulfa antibiotics and only partially cover the costs of the healthcare system. Yet, based on these conservative numbers, the social rate of return from inception of antibiotics is large and amounts to a ratio of 34/1. This affirms that health technologies with a large public-good dimension have substantial economic value not only in the short term (for example, Murphy and Topel 2006) but also in the long run.

VIII. Conclusions

In recent years, the literature showing that early-life circumstances predict health, education, and socioeconomic status later in life has grown substantially (Almond and Currie 2011; Almond, Currie, and Duque 2017). In particular, it has been shown that disease environment at 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 (for example, Bleakley 2007) by studying the effects of exposure to pneumonia in infancy, reduced by a sudden introduction of sulfa antibiotics in Sweden, on labor productivity in late adulthood. The findings suggest the following: (i) decrease in exposure to pneumonia and its treatment in infancy led to gains in labor incomes; (ii) it increased an individual's health stock substantially, with associated productivity that accounts for the bulk of the labor 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 preexisting trends, region-specific arrival of sulfapyridine, compositional changes, family factors, overlapping programs, and a plausible measurement error.

This study points to several conditions for the efficient implementation of a breakthrough health technology. Certain conditions surround the adoption of sulfa 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 healthcare system, such as high-quality medical personnel, low costs of treatment, and centralization of drug distribution, allowing all families to access it. Furthermore, all population groups should have equal opportunities to fully realize the acquired early-life benefits. In the 1920s–1930s, pneumonia mortality had a strikingly similar pattern across nations that should yield similar long-term consequences. Yet, in the United States, due to available institutions, only white men benefited from decreased pneumonia in terms of schooling, income, and social mobility in adulthood, unlike women and black men (Bhalotra and Venkataramani 2013). For Sweden, the present paper finds long-term benefits for everyone. Beginning from the 1960s in Sweden, such favorable elements as, for instance, publicly provided childcare or maternity leave, public education, and the progressive taxation of wealth likely supported the realization of the early-life effects among men and women.

The findings of this paper have relevance for middle- and low-income countries, where the majority of early childhood deaths occur due to pneumonia, diarrhea, 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 five, 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 as a result of 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.

References

- Adhvaryu, Achyuta, Steven Bednar, Anant Nyshadham, Teresa Molina, and Quynh Nguyen. 2018. "When It Rains It Pours: The Long-Run Economic Impacts of Salt Iodization in the United States." NBER Working Paper 24847. Cambridge, MA: NBER.
- Almond, Douglas, and Janet Currie. 2011. "Human Capital Development before Age Five." In *Handbook of Labor Economics, Volume 4B, Developments in Research Methods and Their Application*, ed. David Card and Orley Ashenfelter, 1315–468. New York: Elsevier.
- Almond, Douglas, Janet Currie, and Valentina Duque. 2018. "Childhood Circumstances and Adult Outcomes: Act II." *Journal of Economic Literature* 56(4):1360–446.
- Andersson, Oscar. 1939. ("Practice and Modern Pneumonia Treatment.") [In Swedish.] *Läkartidningen* 23:1190–5.
- Barker, David. 1994. *Mothers, Babies, and Disease in Later Life*. London: British Medical Journal Publishing Group.
- Barreca, Alan I. 2010. "The Long-Term Economic Impact of In Utero and Postnatal Exposure to Malaria." *The Journal of Human Resources* 45(4):865–92.
- Bengtsson, Tommy, and Martin Dribe. 2014. "The Historical Fertility Transition at the Micro Level: Southern Sweden 1815–1939." *Demographic Research* S14(17):493–534.
- Bentley, Ronald. 2009. "Different Roads to Discovery; Prontosil (Hence Sulfa Drugs) and Penicillin (Hence Beta-Lactams)." *Journal of Industrial Microbiology & Biotechnology* 36(6):775–86.

- Beyerlein, Andreas, Ewan Donnachie, Sibille Jergens, and Anette-Gabriele Ziegler. 2016. "Infections in Early Life and Development of Type 1 Diabetes." *JAMA* 315(17):1899–901.
- Bhalotra, Sonia, Martin Karlsson, Therese Nilsson, and Nina Schwarz. 2016. "Infant Health, Cognitive Performance and Earnings: Evidence from Inception of the Welfare State in Sweden." IZA Working Paper 10339. Bonn, Germany: IZA.
- Bhalotra, Sonia, and Athendar S. Venkataramani. 2013. "Shadows of the Captain of the Men of Death: Early Life Health Interventions, Human Capital Investments, and Institutions." IZA Working Paper 7833. Bonn, Germany: IZA.
- Bleakley, Hoyt. 2007. "Disease and Development: Evidence from Hookworm Eradication in the American South." *Quarterly Journal of Economics* 122(1):73–117.
- Bleakley, Hoyt. 2010a. "Health, Human Capital, and Development." *Annual Review of Economics* 2:283–310.
- Bleakley, Hoyt. 2010b. "Malaria Eradication in the Americas: A Retrospective Analysis of Childhood Exposure." *American Economic Journal: Applied Economics* 2(2):1–45.
- Case, Anne, and Christina Paxson. 2009. "Early Life Health and Cognitive Function in Old Age." *American Economic Review* 99(2):104–9.
- Colebatch, Alexandra N., and Chris J. Edwards. 2011. "The Influence of Early Life Factors on the Risk of Developing Rheumatoid Arthritis." *Clinical and Experimental Immunology* 163(1):11–16.
- Cronberg, Stig. 1997. ("Infections: Illness, Environment, Treatment.") [In Swedish.] Stockholm: Liber.
- Cunha, Flavio, and James Heckman. 2007. "The Technology of Skill Formation." *The American Economic Review* 97(2):31–47.
- Cutler, David, Winnie Fung, Michael Kremer, Monica Singhal, and Tom Vogl. 2010. "Early-Life Malaria Exposure and Adult Outcomes: Evidence from Malaria Eradication in India." *American Economic Journal: Applied Economics* 2(2):72–94.
- Finch, Caleb E. 2007. *The Biology of Human Longevity*. Oxford: Elsevier.
- Finch, Caleb E., and Eileen Crimmins. 2004. "Inflammatory Exposure and Historical Changes in Human Life-Spans." *Science* 305(5691):1736–9.
- Fischer, Martin, Martin Karlsson, and Therese Nilsson. 2013. "Effects of Compulsory Schooling on Mortality: Evidence from Sweden." *International Journal of Environmental Research and Public Health* 10(8):3596–618.
- Galobardes, Bruna, Peter McCarron, Mona Jeffreys, and George Davey-Smith. 2008. "Association between Early Life History of Respiratory Disease and Morbidity and Mortality in Adulthood." *Thorax* 63(5):423–29.
- Glied, Sherry, and Matthew Neidell. 2010. "The Economic Value of Teeth." *The Journal of Human Resources* 45(2):468–96.
- Gnosspelius, A. 1939. ("Pneumonia Treatment in Children with '693.'") [In Swedish.] *Läkartidningen* 21:1028–34.
- Heckman, James J. 1979. "Sample Selection Bias as a Specification Error." *Econometrica* 47(1):153–61.
- Heckman, James J. 2007. "The Economics, Technology, and Neuroscience of Human Capability Formation." *Proceedings of the National Academy of Sciences of the United States of America* 104(33):13250–55.
- Hemminki, Elina, and Anneli Paakulainen. 1976. "The Effect of Antibiotics on Mortality from Infectious Diseases in Sweden and Finland." *American Journal of Public Health* 66(12):1180–84.
- Hjort, Jonas, Mikkel Sølvsten, and Miriam Wüst. 2017. "Universal Investment in Infants and Long-Run Health: Evidence from Denmark's 1937 Home Visiting Program." *American Economic Journal: Applied Economics* 9(4):78–104.

- Holmlund, Helena. 2008. "A Researcher's Guide to the Swedish Compulsory School Reform." CEE DP Working Paper 87. London, UK: Centre for the Economics of Education, London School of Economics and Political Science.
- Ingvar, Sven. 1939. ("On the Treatment of Croupous Pneumonia.") [In Swedish.] *Läkartidningen* 1:11–57.
- Kelly, Elaine. 2011. "The Scourge of Asian Flu: In Utero Exposure to Pandemic Influenza and the Development of a Cohort of British Children." *Journal of Human Resources* 46(4):669–94.
- Kesternich, Iris, Bettina Siflinger, Joachim K. Winter, and James P. Smith. 2014. "The Effects of World War II on Economic and Health Outcomes across Europe." *Review of Economics and Statistics* 96(1):103–18.
- Kuh, Diana, and Yoav Ben-Shlomo. 2007. *A Life Course Approach to Chronic Disease Epidemiology*. 2nd edition. Oxford, UK: Oxford University Press.
- Landrigan, Philip J., Babasaheb Sonawane, Robert N. Butler, Leonardo Trasande, Richard Callan, and Daniel Droller. 2005. "Early Environmental Origins of Neurodegenerative Disease in Later Life." *Environmental Health Perspectives* 113(9):1230–33.
- Lazuka, Volha. 2018. "The Long-Term Health Benefits of Receiving Treatment from Qualified Midwives at Birth." *Journal of Development Economics* 133(July):415–33.
- Lindert, Peter H. 2004. *Growing Public: Social Spending and Economic Growth since the Eighteenth Century*. Cambridge, UK: Cambridge University Press.
- Ljungberg, Jonas, and Anders Nilsson. 2009. "Human Capital and Economic Growth: Sweden 1870–2000." *Cliometrica* 3(1):71–95.
- Lucas, Adrienne M. 2010. "Malaria Eradication and Educational Attainment: Evidence from Paraguay and Sri Lanka." *American Economic Journal: Applied Economics* 2(2):46–71.
- Malmros, Haqvin, and Olof Wilander. 1941. ("Should Sulfaipyridine Be Used against Influenza?") [In Swedish.] *Läkartidningen* 4:185–89.
- Murphy, Kevin M., and Robert H. Topel. 2006. "The Value of Health and Longevity." *Journal of Political Economy* 114(5):871–904.
- Rahm, Nils. 1939. ("Pneumonia Treatment.") [In Swedish.] *Läkartidningen* 28:2044–60.
- Riksarkivet. 1920–1967. (*Records of the Pharmaceutical Agency*.) [In Swedish.]
- Riksarkivet. 1932–1936. (*Historical GIS Maps in Sweden, 1569–1998*.) [In Swedish.]
- Riksskatteverket. 1989. (*Swedish Parishes throughout the Centuries*.) [In Swedish.]
- Rudan, Igor, Katherine L. O'Brien, Harish Nair, Li Liu, Evropi Theodoratou, Shamim Qazi, and Ivana Lukšić. 2013. "Epidemiology and Etiology of Childhood Pneumonia in 2010: Estimates of Incidence, Severe Morbidity, Mortality, Underlying Risk Factors and Causative Pathogens for 192 Countries." *Journal of Global Health* 3(1):46–59.
- Schön, Lennart. 2004. "Total Factor Productivity in Swedish Manufacturing in the Period 1870–2000." In *Exploring Economic Growth: Essays in Measurement and Analysis*, ed. Sakari Heikkilä and Jan Luiten van Zanden, 273–97. Amsterdam: Aksant.
- Skånes Näringslivsarkiv. 1936–1945. (*Leo AB and Ferrosan AB. Pharmaceutical Production*.) [In Swedish.]
- Socialstyrelsen. 1952–2012. (*Wage Statistics Yearbook for Sweden*.) [In Swedish.] Stockholm: Isaac Marcus Bocktryckeri-Aktiebolag.
- SOU. 1948. (*About Open Medical Care in the Country*.) 14. [In Swedish.] Stockholm: P.A. Norstedt & Söner.
- SOU. 1959. (*Organization of Pharmaceutical Supply*.) 5. [In Swedish.] Stockholm: P.A. Norstedt & Söner.
- Statistiska Centralbyrån. 1920a–1950. (*Causes of Death*.) [In Swedish.] Stockholm: P.A. Norstedt & Söner.
- Statistiska Centralbyrån. 1920b–1950. (*Yearbook on Health and Health Care*.) [In Swedish.] Stockholm: P.A. Norstedt & Söner.

- Stocks, Janet, and Samatha Sonnappa. 2013. "Early Life Influences on the Development of Chronic Obstructive Pulmonary Disease." *Therapeutic Advances in Respiratory Disease* 7(3):161–73.
- Ström, Justus. 1946. ("The Social Support of Future Mothers and Infants in Sweden.") [In Swedish.] *Social-Medicinsk Tidskrift* 19:22–43.
- The Nobel Foundation. 1965. *Nobel Lectures, Physiology or Medicine 1922–1941*. Amsterdam: Elsevier.
- Vallgårda, Signild. 1996. "Hospitalization of Deliveries: The Change of Place of Birth in Denmark and Sweden from the Late Nineteenth Century to 1970." *Medicinal History* 40(2): 173–96.
- Venkataramani, Athendar S. 2012. "Early Life Exposure to Malaria and Cognition in Adulthood: Evidence from Mexico." *Journal of Health Economics* 31(5):767–80.
- Waldenström, Daniel. 2014. "Swedish Stock and Bond Returns, 1856–2012." In *Historical Monetary and Financial Statistics for Sweden*, Volume II, ed. Rodney Edvinsson and Daniel Waldenström, 223–92. Stockholm: Sveriges Riksbank and Ekerlids.
- Weil, David N. 2013. "Health and Economic Growth." In *Handbook of Economic Growth*, ed. Philippe Aghion and Steven N. Durlauf, 623–82. San Diego, CA: North Holland, Elsevier.
- WHO. 2013. *Ending Preventable Child Deaths from Pneumonia and Diarrhoea by 2025. The Integrated Global Action Plan for Pneumonia and Diarrhoea (GAPPD)*. Geneva: WHO.
- Willerson, James T., and Paul M. Ridker. 2004. "Inflammation as a Cardiovascular Risk Factor." *Circulation* 109(21):II-2–II-10.
- Yayachandran, Seema, Adriana Lleras-Muney, and Kimberly V. Smith. 2010. "Modern Medicine and the Twentieth Century Decline in Mortality: Evidence on the Impact of Sulfa Drugs." *American Economic Journal: Applied Economics* 2(2):118–46.
- Zajacova, Anna, and Sarah A. Burgard. 2013. "Healthier, Wealthier, and Wiser: A Demonstration of Compositional Changes in Aging Cohorts due to Selective Mortality." *Population Research and Policy Review* 32(3):1–14.

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