# On-line Appendixes

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

## Appendix A

CONTEXT OF THE INTRODUCTION OF *SULPHAPYRIDINE*

The necessity of improving infant health, whose advances in Sweden began to stagnate similar to other Western countries (Griffiths and Brock 2003), received wide public attention in the 1920s. The Swedish government in 1929 published a report on public health insurance that in part demonstrated the large inequalities in infant and child mortality across regions, urban and rural areas and socio-economic classes (SOU 1929). Despite the remarkable economic development in the country, measured for instance with real income per capita that had grown at 2.4 percent annually between 1890 and 1930 (Schön and Krantz 2012), these differentials persisted. Among candidate factors responsible for health inequalities several had been listed, such as the standards of living, including housing, family size, infectious disease environment, nutrition, childcare, willingness to use medical services, as well as climatic conditions (Sundin and Willner 2007). Causes associated with infectious diseases clearly dominated both infant and child mortality (see Table). Similar to other countries in Western Europe and North America, respiratory diseases, such as pneumonia, bronchitis and influenza, exhibited stagnation or slow decline for half of the century up until the late 1930s in Sweden (van Hofsten and Lundström 1976). Among exogenous causes, pneumonia and influenza alone accounted for no less than 20 percent of premature deaths below the age of 5 in the 1920s–1930s, emerging as the major cause of death among infants and the major infectious-disease cause of death among children.

The invention and introduction of sulpha antibiotics into medical practice as treatment against pneumonia and other infectious diseases is recognised as one of the major historical breakthrough innovations (WIPO 2015). The efficiency of sulphonamides against many experimental streptococcal and other infections was observed by Gerhard Domagk in Germany in 1932 (Domagk 1957). By the late 1930s, a bacteriostatic component of sulphonamides –

‘sulpha’ – had been revealed, that prevented the bacteria from multiplying, by inhibiting the synthesis of folic acid within bacteria, and did not kill it, and experiments with derivatives of sulphonamide preparations were launched elsewhere (The Nobel Foundation 1965). The production and trade of the drugs at a mass and international scale started by the end of the 1930s, among which the major success is attributed to *sulphapyridine* (a compound of pyridine and sulphonamides) against pneumonia prepared by the May and Baker Company as M&B 693 (Bentley 2009). The clinical trials showed that treatment of pneumonia with sulpha medications on humans reduced the mortality rate by between 1/2 and 6/7, being especially efficacious in treating bacteria-caused pneumonia (Graham et al. 1939). Until the introduction of penicillin in the late 1940s, sulpha antibiotics remained the prime cure against pneumococcal, meningococcal, gonococcal and some other infections.

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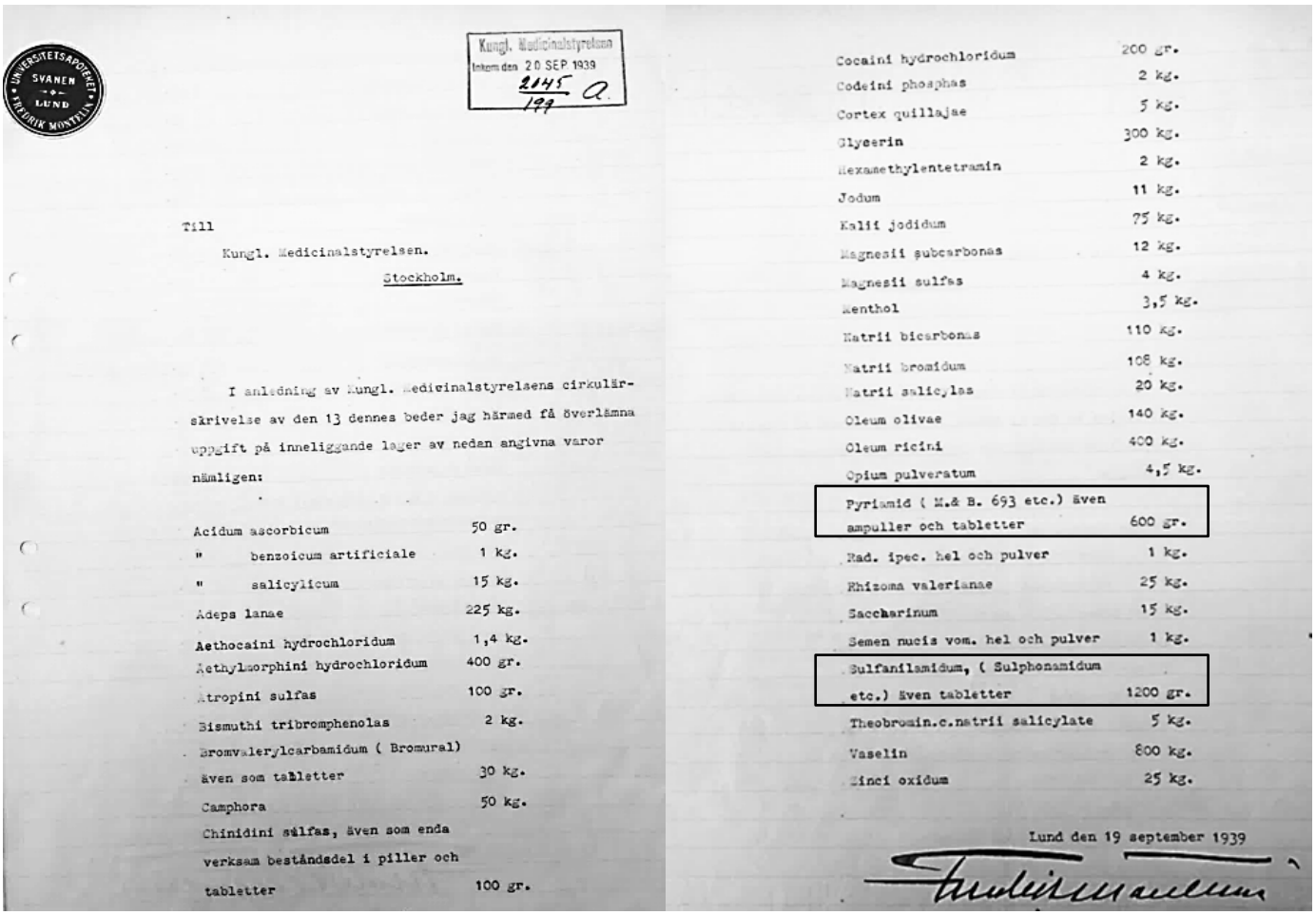
TABLE – COMPOSITION OF THE CAUSES OF DEATH IN SWEDEN, PERCENT, 1920–1950

| | 1920 | | | 1930 | | | 1940 | | | 1950 | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | age<1 | ages 1-5 | all ages | age<1 | ages 1-5 | all ages | age<1 | ages 1-5 | all ages | age<1 | ages 1-5 | all ages |
| malformations | 43 | 2 | 5 | 52 | 4 | 4 | 59 | 3 | 3 | 76 | 8 | 2 |
| infectious | 12 | 45 | 18 | 8 | 30 | 14 | 6 | 24 | 9 | 3 | 7 | 3 |
| pneumonia and influenza | 20 | 25 | 12 | 20 | 26 | 7 | 21 | 24 | 7 | 11 | 22 | 5 |
| diarrheal | 10 | 5 | 2 | 9 | 9 | 1 | 5 | 9 | 0 | 5 | 9 | 0 |
| non-infectious | 5 | 11 | 32 | 6 | 18 | 44 | 5 | 21 | 55 | 4 | 24 | 67 |
| other | 10 | 12 | 32 | 6 | 14 | 30 | 3 | 21 | 27 | 2 | 30 | 23 |
| total | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 |

*Source*: own calculations based on Statistiska Centralbyrån (1920a-1950)

## Appendix B



Kungl. Medicinalstyrelsen
Inkom den 20 SEP 1939
2145 a
199

Till
Kungl. Medicinalstyrelsen.
Stockholm.

I anledning av Kungl. Medicinalstyrelsens cirkulärskrivelse av den 13 dennes beder jag härmed få överlämna uppgift på inneliggande lager av nedan angivna varor nämligen:

| | |
|---|---|
| Acidum ascorbicum | 50 gr. |
| " benzoicum artificiale | 1 kg. |
| " salicylicum | 15 kg. |
| Adeps lanae | 225 kg. |
| Aethocaini hydrochloridum | 1,4 kg. |
| Aethylmorphini hydrochloridum | 400 gr. |
| Atropini sulfas | 100 gr. |
| Bismuthi tribromphenolas | 2 kg. |
| Bromvalerylcarbamidum ( Bromural) även som tabletter | 30 kg. |
| Camphora | 50 kg. |
| Chinidini sulfas, även som enda verksam beståndsdel i piller och tabletter | 100 gr. |
| Cocaini hydrochloridum | 200 gr. |
| Codeini phosphas | 2 kg. |
| Cortex quillajae | 5 kg. |
| Glycerin | 300 kg. |
| Hexamethylentetramin | 2 kg. |
| Jodum | 11 kg. |
| Kalii jodidum | 75 kg. |
| Magnesii subcarbonas | 12 kg. |
| Magnesii sulfas | 4 kg. |
| Menthol | 3,5 kg. |
| Natrii bicarbonas | 110 kg. |
| Natrii bromidum | 108 kg. |
| Natrii salicylas | 20 kg. |
| Oleum olivae | 140 kg. |
| Oleum ricini | 400 kg. |
| Opium pulveratum | 4,5 kg. |
| Pyriamid ( M.& B. 693 etc.) även ampuller och tabletter | 600 gr. |
| Rad. ipec. hel och pulver | 1 kg. |
| Rhizoma valerianae | 25 kg. |
| Saccharinum | 15 kg. |
| Semen nucis vom. hel och pulver | 1 kg. |
| Sulfanilamidum, ( Sulphonamidum etc.) även tabletter | 1200 gr. |
| Theobromin.c.natrii salicylate | 5 kg. |
| Vaselin | 800 kg. |
| Zinci oxidum | 25 kg. |

Lund den 19 september 1939

FIGURE – EXAMPLE OF PHARMACEUTICAL RECORD ON DRUG INVENTORY IN SEPTEMBER 1939, SWEDEN

*Source*: Riksarkivet (1920–1967) (owner)

*Notes:* The amount of sulphapyridine (*Pyriamid*, *M&B 693*, drug against pneumonia) is marked. The amount of sulphonamides (drug against puerperal fever) is recorded separately and marked.

## Appendix C

SURVIVORS OF COHORTS UNDER ANALYSIS

The cohorts born between 1934 and 1943 appear in the SIP dataset from 1968. I therefore do not observe individuals that died or migrated from Sweden prior to age 34. I gathered information on one-year survivors born in rural areas (live births minus infant deaths) of the cohorts born 1920–1950 from Statistics Sweden (Statistiska Centralbyrån, 1920c–1950). In Figure below, I plot them against counts of individuals with places of birth available in SIP by cohort and those who have valid information on the county and parish of birth. A relatively stable fraction of individuals observed in the SIP dataset compared to number of one-year survivors indicates that the individuals born 1934–1943 were dying at a constant rate between the ages 1 and 33. Among the first-year survivors of these cohorts, 96.0 percent were observed in the dataset. The selection to survival to adulthood should not therefore violate the results in the paper.

Starting from cohorts 1932 and born later, individuals are linked to their parents through the multigenerational register (*Flergenerationsregistret*) thereby giving a unique family identifier. This information is available for all individuals in our sample conditional on their survival or presence in Sweden to the year 1991. Due to the availability of family links (across different outcome samples, 91–94 percent are linked to mothers, and 83–86 percent to both mothers and fathers), I was able to merge socio-economic and demographic information of the family to the individual data. The parental background characteristics included the following information: age of the mother obtained from population register (*Registret över Totalbefolkningen*), education of the mother, socio-economic status and sector of employment of the father obtained from population and housing census 1970 (*Folk- och Bostadsräkningen 1970*). Socio-economic status has been originally constructed by Statistics Sweden (Statistiska Centralbyrån 1975) based on occupation and occupational status, which I further grouped into high (farmers,

business owners in different sectors of economy, higher professionals, and higher managers) and low (workers in different sectors of economy, military, lower professionals and managers, and clerical and sales personnel). This information was available only for the post-treatment child's ages, in parents' late adulthood (mean age 64 and 99.8 percent are employed), although for paternal cohorts education should have been completed, and socio-economic status and branch of work had to stabilise.

For the cohorts 1925–1929, because the data is conditional on cohorts 1930–1980 and their parents and siblings, no individuals with any younger siblings born starting from 1930 were observed. Among the first-year survivors of these cohorts, I observe 76.6 percent in the dataset. These cohorts do not contain information on sibling links.

## REFERENCES (IN ADDITION TO THE SOURCES NOT CITED IN THE MAIN TEXT)

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FIGURE – FIRST-YEAR SURVIVORS AND ESTIMATION SAMPLE FOR THE COHORTS 1920–1950
*Sources*: own calculations based on SIP and Statistiska Centralbyrån (1920c,d-1950).

## Appendix D

GROUPS OF CAUSES OF MORBIDITY

The cause of admission to the hospital is obtained from the Swedish national inpatient register 1987–2012. It adopted two revisions of the international classifications of the causes of morbidity, such as revision 9 for 1987–1996, and revision 10 for 1997–2012. Following the previous literature (Kuh and Ben-Shlomo 2007), I classified all causes of admissions into six groups, including infectious/respiratory diseases, cardiovascular diseases, diabetes, cancer, degenerative diseases of tissues and organs, and mental diseases and calculated the respective average length of stay in hospital. The same classification is used for cause-specific mortality. The group of degenerative diseases of tissues and organs is dominant with symptoms of respiratory diseases, arthritis and gastro-enteric diseases. In order to measure pathology in health exclusively, I excluded hospital admissions due to violent/accidental causes (2 percent of person-years) and observations with no need for further treatment (0.01 percent of person-years). The exact codes used for these groupings are provided in Table below.

TABLE – DIAGNOSES GROUPS ACROSS TWO REVISIONS OF THE ICD, 1987–2012

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

## Appendix E

DATA SOURCES FOR REGION-OF-BIRTH CHARACTERISTICS, SWEDEN 1934–1943

| Variable | Source | Comments |
|---|---|---|
| Pneumonia mortality rate, per 1000 mid-year population | constructed based on SCB Sveriges officiella statistik. Dödsorsaker [Statistics Sweden. Causes of Deaths]: deaths from respiratory diseases: pneumonia, acute bronchitis, chronic bronchitis, pleurisy and other respiratory diseases (*Pneumonia acuta lobaris. Bronchopnenmonia acuta. Bronchitis capillaris; Bronchitis acuta. Laryngo-tracheitis acuta; Bronchitis chronica; Pleuritis. Empyema pleurae; Alii morbi organoram respirationis*); SCB Sveriges officiella statistik. Befolkningsrörelsen [Statistics Sweden. Population Movement] (mid-year population) | yearly and county urban-rural level, 49 regions, 1920–1950 |
| Cause-specific mortality rates, per 1000 mid-year population | constructed based on Dödsorsaker: typhoid fever (*Febris typhoidea. Febris paratyphoidea*), acute and chronic diarrhoea (*Gastro-enteritia acnta infectiosa; Gastroenteritis chronica*), influenza (*Influenza cum aegrotatione pulmonis. Influenza sine aegrotatione pulmonis*), lung tuberculosis (*Tuberculosis pulmonis, laryngis*), acute and chronic heart disease (*Thrombo-endocarditis acuta; Endocarditis chronica. Hyocarditis chronica*), diabetes (*Diabetes mellitus*), cancer (*Carcinoma oris et linguae, ventriculi, intestine, uteri et ovariorom, mammae, cutis, aliorum organoram; Sarcoma cutis et subcutis, ossiam, viscerum, alioram organoram; Helanosarcoma; Alii tumores*), and puerperal fever (*Septichaemia paerperalis postpartum; Septichaemia puerperalis post abortum*); SCB Sveriges officiella statistik. Befolkningsrörelsen (mid-year population) | yearly and county urban-rural level, 49 regions, 1920–1950 |
| Infant mortality rate, per 1000 live births | constructed based on SCB Sveriges officiella statistik. Befolkningsrörelsen (infant deaths and live births) | yearly and county urban-rural level, 49 regions |
| Crude death rate, per 1000 mid-year population | constructed based on SCB Sveriges officiella statistik. Befolkningsrörelsen (total deaths and mid-year population) | yearly and county urban-rural level, 49 regions |
| Stillbirth rate, per 1000 total births | constructed based on SCB Sveriges officiella statistik. Befolkningsrörelsen (stillbirths and live births) | yearly and county urban-rural level, 49 regions |
| Crude birth rate, per 1000 mid-year population | constructed based on SCB Sveriges officiella statistik. Befolkningsrörelsen (live births and mid-year population) | yearly and county urban-rural level, 49 regions |
| Marital fertility rate, per 1000 | constructed based on SCB Sveriges officiella statistik. Befolkningsrörelsen (yearly legitimate total births and 5-mid-year married women 15-45 ages) | yearly and county urban-rural level, 49 regions |
| Share females in total population | constructed based on SCB Sveriges officiella statistik. Befolkningsrörelsen (female and male mid-year population) | yearly and county urban-rural level, 49 regions |
| Share employed in agriculture | constructed based on SCB Sveriges officiella statistik. Folkräkningen [Statistics Sweden. Population Census] (number employed in agriculture, industry and services) 1930, 1940, 1950 | decadal and county level, 24 counties and Stockholm |
| Share employed in industry | constructed based on SCB Sveriges officiella statistik. Folkräkningen (number employed in agriculture, industry and services) 1930, 1940, 1950 | decadal and county level, 24 counties and Stockholm |
| Real yearly wage of worker | constructed based on Socialstyrelsen Lönestatistisk Årsbok 1930-1950 [Statistics Sweden. Wage Statistics] (average yearly wages for male manufacturing worker and average yearly wages for male servants in agriculture) as a weighted average (share of employed in agriculture and industry as weights from Folkräkningen 1930, 1940, 1950); Edvinsson & Söderberg (2011) (national CPI) | yearly and county level, 24 counties and Stockholm |
| Real regional GDP per capita | Enflo, Henning & Schön (2015) | decadal and county level, 24 counties |

| | | |
|---|---|---|
| Medical personnel, per 1000 mid-year population | constructed based on SCB Sveriges officiella statistik. Allmän om Hålso och sjukvård [Statistics Sweden. Health and Health Care] (number of legitimate doctors, midwives, and medical nurses); SCB Sveriges officiella statistik. Befolkningsrörelsen (mid-year population) | yearly and county urban-rural level, 49 regions |
| Real hospital spending, per 1000 mid-year population | constructed based on SCB Sveriges officiella statistik. Allmän om Hålso och sjukvård (hospitals' receipts); SCB Sveriges officiella statistik. Befolkningsrörelsen (mid-year population); Edvinsson & Söderberg (2011) (national CPI) | yearly and county urban-rural level (24 counties and 3 urban regions: Stockholm, Malmo and Gothenborg), 27 regions |
| Share under age 15 in total population | constructed based on SCB Sveriges officiella statistik. Statistik Årsbok för Sverige [Statistics Sweden. Statistics Yearbook] (population under age 15); SCB Sveriges officiella statistik. Befolkningsrörelsen (mid-year population) | 5-year and county level, 24 counties and Stockholm |
| Share above age 65 in total population | constructed based on SCB Sveriges officiella statistik. Statistik Årsbok för Sverige (population above age 65); SCB Sveriges officiella statistik. Befolkningsrörelsen (mid-year population) | 5-year and county level, 24 counties and Stockholm |
| Number of school-rooms, per 1000 primary-school pupils | constructed based on SCB Sveriges officiella statistik. Statistik Årsbok för Sverige (number of school-rooms and number of pupils in primary schools) | yearly and county level, 24 counties and Stockholm |
| Number of teachers, per 1000 primary-school pupils | constructed based on SCB Sveriges officiella statistik. Statistik Årsbok för Sverige (number of teachers and number of pupils in primary schools) | yearly and county level, 24 counties and Stockholm |
| Sulphapyridine availability, per 1000 mid-year population;<br>Sulphonamide availability, per 1000 mid-year population;<br>Price index of medical drugs | constructed based on Riksarkivet. Medicinalstyrelsens apoteksbyrå [National Archive. National Health Board's Pharmacy Agency], aggregated from pharmacy level, pharmacy-city locations from SCB. Recalculated into adult doses to treat pneumonia episode per 1000 population (1 adult dose = 20 grams of sulphapyridine). Sveriges kommuner åren 1952-1986 | year 1939 and county urban-rural level, 49 regions |
| Absolute change in price of medical drugs | constructed based on Riksarkivet. Medicinalstyrelsens apoteksbyrå [National Archive. National Health Board's Pharmacy Agency], aggregated from pharmacy level, pharmacy-city locations from SCB. Sveriges kommuner åren 1952-1986 | year 1940 relative to 1939 and county urban-rural level, 49 regions |

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## Appendix F

ASSOCIATIONS BETWEEN BASELINE PNEUMONIA MORTALITY AND REGION-OF-BIRTH CHARACTERISTICS (CONTROL VARIABLES), SWEDEN 1932–1936

| **Region-level socio-economic and health care characteristics** | (1) Reduced | (2) Full | **Region-level cause-specific mortality rates** | (1) Reduced | (2) Full |
|---|---|---|---|---|---|
| Stillbirth rate | 0.0429 | -1.423 | Puerperal fever | 1.893 | 0.401 |
| | (0.718) | (0.870) | | (1.721) | (2.087) |
| CBR | 1.667** | -0.449 | Typhoid fever | -0.261 | -1.043 |
| | (0.779) | (1.832) | | (0.725) | (0.682) |
| Share of females | -165.9* | -124.8 | Diarrhoea | 1.282** | 1.623** |
| | (82.90) | (240.5) | | (0.599) | (0.648) |
| Share above age 65 | 105.5 | -6.984 | Influenza | -0.781 | -0.971 |
| | (115.8) | (317.3) | | (0.602) | (0.605) |
| Share below age 15 | 93.36 | 271.7 | Lung tuberculosis | 0.0238 | -0.0453 |
| | (59.66) | (180.0) | | (0.0650) | (0.112) |
| CDR | 7.078*** | 8.914** | Heart disease | -0.0409 | -0.0787 |
| | (2.228) | (3.411) | | (0.0771) | (0.0967) |
| IMR | 0.427* | 0.190 | Diabetes | -0.761 | -0.436 |
| | (0.214) | (0.396) | | (0.531) | (0.671) |
| Ln real regional GDP pc | -20.84* | -38.58 | Cancer | 0.0139 | 0.0648 |
| | (10.42) | (43.65) | | (0.0686) | (0.0923) |
| Ln real wage of worker | 6.412 | -21.00 | | | |
| | (13.34) | (41.23) | | | |
| Share employed in agriculture | 21.67 | -31.45 | | | |
| | (15.06) | (84.03) | | | |
| Share employed in industry | -40.94 | -0.316 | | | |
| | (29.82) | (59.66) | | | |
| Ln medical personnel per 1000 | -6.948 | 5.942 | | | |
| | (4.805) | (12.31) | | | |
| Ln pharmacies per 1000 | -4.424** | 1.101 | | | |
| | (1.930) | (4.082) | | | |
| Ln real hospital spending per 1000 | -0.283 | 9.994 | | | |
| | (5.199) | (8.225) | | | |
| Ln school-rooms per 1000 pupils | -21.56 | -15.19 | | | |
| | (25.19) | (34.20) | | | |
| Ln teachers per 1000 pupils | -14.81 | 6.872 | | | |
| | (26.52) | (57.50) | | | |
| Observations | 49 | 49 | Observations | 49 | 49 |
| R-squared | - | 0.49 | R-squared | - | 0.20 |

*Source:* own estimations based on sources from Appendix E.

*Notes*: OLS regression estimates. Each variable is the arithmetic average for 1932–1936. Mortality rates per 1000 mid-year population. In Model 1 'Reduced', the effect of pneumonia mortality is estimated separately for each variable (plus constant); in Model 2 'Full', all variables are included (plus a constant). Standard errors (in parentheses) are clustered at a region level.

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

## Appendix G

THE CONTEMPORANEOUS EFFECTS OF THE INTRODUCTION OF *SULPHAPYRIDINE*

To start with the graphical analysis, beginning from the late 1930s, total pneumonia mortality exhibited an irretrievable decline in both level and trend (see Figure 1 of the main text). There are no similar breaks in other diseases at the same time. Figure G1 below shows that that in absolute terms pneumonia mortality (per 1000) declined most among infants than among other age groups: -1.8 deaths for infants versus -0.4 deaths in ages 1–4, -0.6 deaths in ages 5–14 or 15–30, -0.3 deaths in ages 30–59, or -1.1 deaths in ages 60+. Figure G2 presents pneumonia mortality across the whole age range in 1933 and 1943, pointing to the same conclusion. Figure 3 of the main text presents the effects of arrival of sulpha antibiotics on mortality at the aggregate level, where baseline pneumonia mortality in 1932–1936 is plotted against absolute decline in pneumonia mortality for the period until 1943. The results indicate strong convergence in aggregate mortality rates from pneumonia after arrival of sulpha antibiotics. More specifically, a one-unit higher pneumonia mortality rate in 1932–1936 is associated with 0.6 unit reduction in pneumonia mortality afterwards.

It is important to investigate whether the breaks in pneumonia mortality occurred in year 1939 using formal tests. As noted above, Figure 1 (in the main text) provides visual evidence that mortality declined substantially, accelerated and never returned to its previous levels after arrival of sulpha medicaments in 1939. The results in both level and logarithmic terms are presented in Table G1. Columns 1 and 2 support that there are both level and trend breaks in pneumonia death rate that year. More specifically, pneumonia mortality dropped significantly in 1939–1943 by around 30 percent in the level and exhibited a trend decline by 16 percent. The beneficial effects of sulpha introduction could also be detected in crude death rate (Columns 3 and 4) and infant mortality rate (Columns 5 and 6), which from 1939 followed an accelerated decrease by 5 percent annually each. I re-estimated the presence of breaks in both

absolute and logarithmic terms using a difference-in-differences approach, where both infectious (Columns 7 and 8) and non-infectious diseases (Columns 9 and 10) are added as control diseases interacted with drug period. The size of the decreases and acceleration in pneumonia mortality from 1939 until 1943 is similar to the one provided in Table G1.

It is plausible that pneumonia reduction due to *sulphapyridine* affected the array of regional characteristics, including demographic, socio-economic and health care characteristics and cause-specific mortality (see Table G2). I additionally studied these effects to shed light on the mechanisms by which the intervention affected child health. Even though results point to the intervention-led decline in crude birth rate, this effects is not robust to the inclusion of the region-specific time trend (point estimate 0.106 and standard error 0.501) or more accurate measures of fertility, such as marital fertility rate. However, in the further analysis based on microdata, I investigate whether fertility was indeed affected across cohorts under analysis (see V. Empirical strategy). Arrival of pneumonia did not affect other socio-economic or demographic indicators. While the results confirmed that the arrival of *sulphapyridine* caused abrupt reduction in pneumonia mortality, it also led to an increase in influenza mortality, by 29– 34 percent of the pre-treatment rate (dependent on specification). At that time, doctors recognised that influenza was not responsive to *sulphapyridine*, only if it was a complication to pneumonia (Malmros and Wilander 1941). This finding could indicate that either influenza cases were prior diagnosed as pneumonia, or that deaths from competing causes were captured. 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.

Sulpha drugs were available in 1939 in all parts of the country in the amounts accounting for this decline (see Figure 2 of the main text). On average across country, pre-treatment pneumonia mortality was 1.043 deaths per 1000 and sulpha drugs were available to save 1.1 deaths per 1000. Importantly, I examined whether regional distribution of *sulphapyridine* was related to

socio-economic characteristics of the regions of birth. As for the variables approximating utilisation of drugs, I employed several, including *sulphapyridine* availability (in adult doses of *sulphapyridine* sufficient to treat full pneumonia episode per 1000 mid-year population) in 1939, average price of a medical drug in 1939 and its change between 1939 and 1940. Table G3 shows that in general there is no systematic association between sulpha drug availability and prices with regional socio-economic variables.

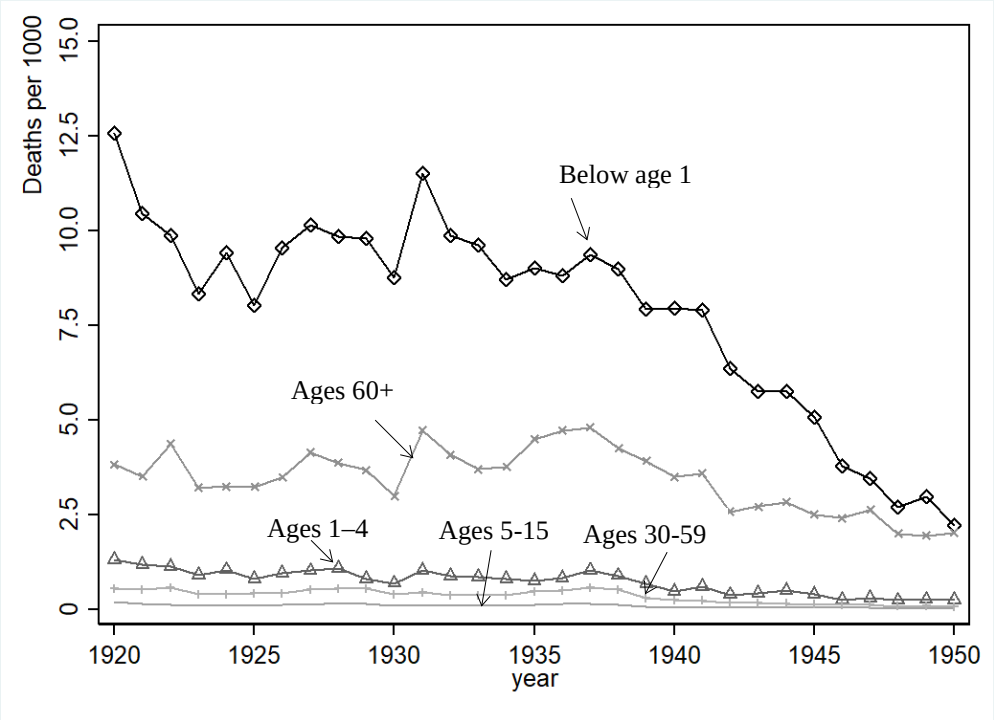



FIGURE G1 – PNEUMONIA MORTALITY BY AGE GROUPS IN 1920–1950

*Sources*: own calculations based on Statistiska Centralbyrån (1920a-1950) and Human Mortality Database (2018) at https://www.mortality.org/.

*Notes*: pneumonia includes pneumonia, acute bronchitis, and chronic bronchitis to avoid differences in nomenclature before and after 1931.

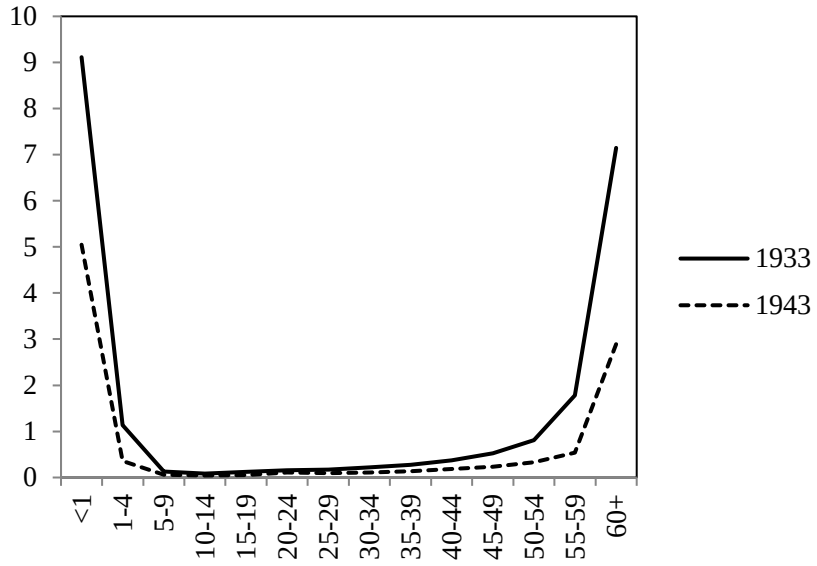



FIGURE G2 – AGE PATTERN OF PNEUMONIA MORTALITY BEFORE AND AFTER INTRODUCTION OF SULPHAPYRIDINE, PER 1000, SWEDEN

*Source:* own calculations based on Statistiska Centralbyrån (1920a-1950)

*Notes:* pneumonia includes pneumonia, acute bronchitis, and chronic bronchitis.

TABLE G1 – LEVEL AND TREND BREAKS IN PNEUMONIA MORTALITY AT THE AGGREGATE LEVEL, 1934–1943

| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) | (9) | (10) |
|---|---|---|---|---|---|---|---|---|---|---|
| | Pneumonia mortality rate | | CDR | | IMR | | Pneumonia mortality rate (treated) and mortality rates from other infectious diseases (control) | | Pneumonia mortality rate (treated) and mortality rates from non-infectious diseases (control) | |
| | levels | logs | levels | logs | levels | logs | levels | logs | levels | logs |
| post1939 | -0.3542*** | -0.2961*** | -0.2309** | -0.0192* | -0.8270 | -0.0225 | | | | |
| | (0.0401) | (0.0402) | (0.1111) | (0.0101) | (1.2298) | (0.0331) | | | | |
| post1939Xyear | -0.1444*** | -0.1593*** | -0.5384*** | -0.0501*** | -1.4741*** | -0.0502*** | | | | |
| | (0.0137) | (0.0138) | (0.0381) | (0.0035) | (0.4218) | (0.0114) | | | | |
| pneumonia mortalityXPost1939 | | | | | | | -0.2878*** | -0.2598*** | -0.3483*** | -0.3530*** |
| | | | | | | | (0.0472) | (0.0659) | (0.0399) | (0.0479) |
| pneumonia mortalityXPost1939Xyear | | | | | | | -0.1153*** | -0.0815*** | -0.0857*** | -0.0920*** |
| | | | | | | | (0.0170) | (0.0257) | (0.0174) | (0.0230) |
| Observations | 490 | 490 | 490 | 490 | 490 | 490 | 2,450 | 2,341 | 1,960 | 1,945 |
| R-sq | 0.64 | 0.66 | 0.82 | 0.82 | 0.65 | 0.63 | 0.90 | 0.87 | 0.90 | 0.94 |

*Sources*: own estimations based on sources from Appendix E.

*Notes*: OLS regression estimates. Models 1–6 additionally include *year* trend and *region* dummies. Models 7–10 additionally include *post1939*, *year*, *pneumonia mortalityXyear* trend, *disease* dummies, *diseaseXyear* trends, and *region* dummies. Infectious diseases added to Models 7 and 8 include typhoid fever, diarrhoea, influenza and lung tuberculosis. Non-infectious diseases added to Models 9 and 10 include puerperal fever, heart disease, diabetes and cancer. Some observations in specifications with logarithmic terms (Models 8 and 10) omitted due to zero mortality from diseases used in the models in some regions. All death rates are calculated per 1000 mid-year population in a corresponding region, and infant mortality rate is calculated per 1000 live births. Year is centred around 1939.

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

TABLE G2 – THE EFFECT OF REDUCED PNEUMONIA MORTALITY ON REGION-OF-BIRTH CHARACTERISTICS (CONTROL VARIABLES), SWEDEN 1934–1943

| | Region-level socio-economic and health care characteristics | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | Stillbirth rate | CBR | Marital fertility rate | Share females | Share <15 ages | Share >65 ages | CDR | IMR | Ln real regional GDP pc |
| post1939Xbaseline pneumonia mortality | 1.299 | -2.181** | -10.916 | 0.004 | -0.000 | -0.001 | -0.264 | -2.835 | -0.008 |
| | (1.725) | (0.954) | (7.091) | (0.004) | (0.002) | (0.001) | (0.265) | (2.505) | (0.027) |
| Pre-mean | 27.795 | 14.126 | 64.960 | 0.511 | 0.235 | 0.095 | 11.614 | 44.186 | 7.346 |
| Observations | 490 | 490 | 490 | 490 | 490 | 490 | 490 | 490 | 490 |
| R-squared | 0.37 | 0.85 | 0.68 | 0.98 | 0.98 | 0.97 | 0.86 | 0.67 | 0.94 |

| | Region-level socio-economic and health care characteristics | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | Ln real wage of worker | Share in agriculture | Share in industry | Ln medical personnel per 1000 | Ln pharmacies per 1000 | Ln real hospital spending per 1000 | Ln school-rooms per 1000 pupils | Ln teachers per 1000 pupils |
| post1939Xbaseline pneumonia mortality | -0.006 | -0.017 | 0.007 | -0.033 | 0.058 | 0.016 | 0.018 | 0.026*** |
| | (0.012) | (0.013) | (0.016) | (0.107) | (0.100) | (0.098) | (0.013) | (0.009) |
| Pre-mean | 7.441 | 0.451 | 0.341 | 0.155 | -1.511 | 8.758 | 4.345 | 3.853 |
| Observations | 490 | 490 | 490 | 490 | 490 | 490 | 490 | 490 |
| R-squared | 0.92 | 0.98 | 0.94 | 0.94 | 0.97 | 0.82 | 0.95 | 0.96 |

| | Region-level cause-specific mortality rates | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | Pneumonia | Puerperal fever | Typhoid fever | Diarrhoea | Influenza | Lung tuberculosis | Heart disease | Diabetes | Cancer |
| post1939Xbaseline pneumonia mortality | -0.223*** | -0.003 | -0.005 | -0.013 | 0.032** | 0.003 | 0.026 | 0.002 | 0.048 |
| | (0.0546) | (0.005) | (0.011) | (0.013) | (0.016) | (0.057) | (0.079) | (0.016) | (0.059) |
| Pre-mean | 1.106 | 0.020 | 0.132 | 0.066 | 0.093 | 0.779 | 1.218 | 0.125 | 1.481 |
| Observations | 490 | 490 | 490 | 490 | 490 | 490 | 490 | 490 | 490 |
| R-squared | 0.70 | 0.26 | 0.39 | 0.29 | 0.55 | 0.79 | 0.69 | 0.41 | 0.60 |

*Source*: own estimations based on sources from Appendix E.

*Notes*: OLS regression estimates. Each model controls for year fixed effects and region fixed effects. Mortality rates per 1000 mid-year population. Pneumonia mortality rate is per 1000 mid-year population, normalized (dividing by its 95th-5th percentile range). Baseline pneumonia mortality is for 1932–1936. Standard errors (in parentheses) are clustered at a region level. *Pre-mean* denotes mean of the outcome in 1934–1938. *** p<0.01, ** p<0.05, * p<0.1. .

TABLE G3 – UTILISATION OF SULPHAPYRIDINE AND REGION-OF-BIRTH SOCIO-ECONOMIC CHARACTERISTICS

| | (1) *Sulphapyridine* drugs per 1000 in 1939 | (2) Price index of medical drugs in 1939 | (3) Change in price index of medical drugs 1939-1940 |
|---|---|---|---|
| Ln real GDP, per capita | -2.193 | 0.516 | -5.048 |
| | (13.036) | (0.430) | (3.464) |
| Ln real wage of worker | -0.548 | -0.865** | 4.681* |
| | (14.375) | (0.420) | (2.770) |
| Share of employed in agriculture | 3.923 | 0.738 | -7.046 |
| | (29.431) | (0.908) | (5.198) |
| Share of employed in industry | 5.050 | 0.628 | 0.849 |
| | (23.513) | (1.001) | (2.571) |
| Ln medical personnel, per 1000 | 3.476 | 0.0537 | -0.221 |
| | (2.629) | (0.0883) | (0.472) |
| Ln pharmacies, per 1000 | 1.573 | -0.0134 | 0.171 |
| | (1.205) | (0.0404) | (0.194) |
| Ln real hospital spending, per 1000 | 0.395 | -0.121 | 0.290 |
| | (1.265) | (0.0867) | (0.334) |
| Regions of birth | 49 | 49 | 49 |
| R-squared | 0.39 | 0.13 | 0.13 |

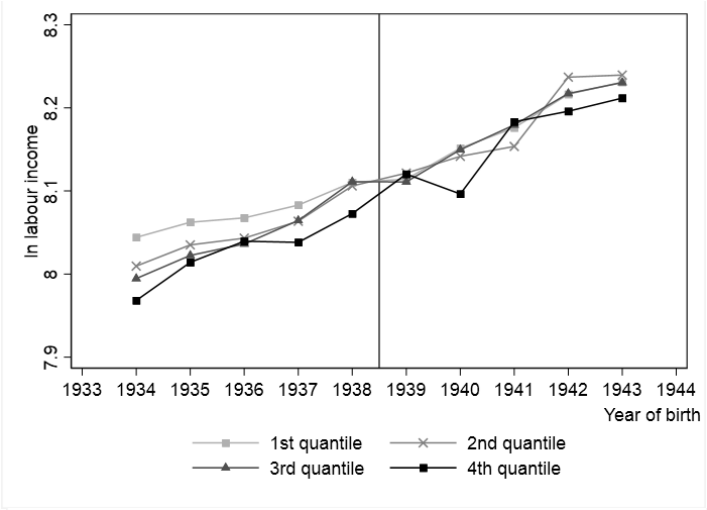
*Sources*: own estimations based on sources from Appendix E.

*Notes*: *Sulphapyridine* drugs is recalculated from original data (in compatible grams) into adult doses sufficient to treat full pneumonia episode per 1000 population (1 adult dose = 20 grams of *sulphapyridine*). Standard errors (in parentheses) are clustered at a region-of-birth level.

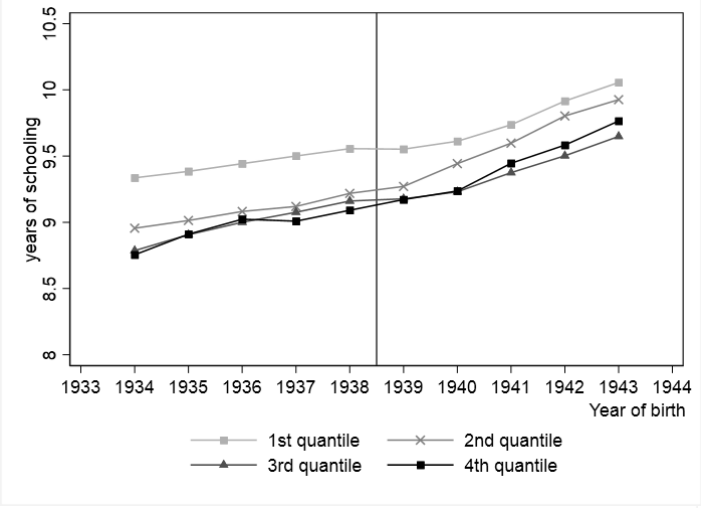
*** p<0.01, ** p<0.05, * p<0.1.

## Appendix H

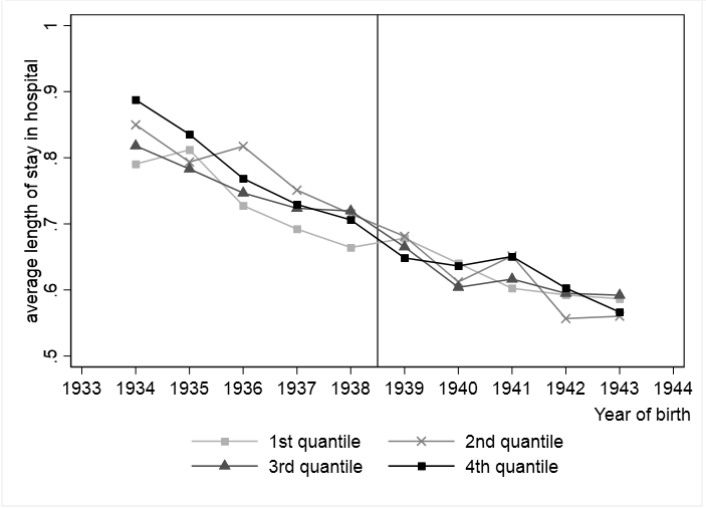
TRENDS IN LATER-LIFE OUTCOMES FOR COHORTS BORN BEFORE AND DURING/AFTER ARRIVAL OF *SULPHAPYRIDINE*, SWEDEN




(1) – Ln labour income




(2) – Years of schooling




(3) – Length of stay in hospital

*Source*: own estimations based on the SIP.
*Notes*: Figure presents the means for the outcome by year of birth and pneumonic regions-of-birth (divided at the quartiles based on baseline pneumonia mortality). Cohort 1939 is the first exposed to *sulphapyridine*.

## Appendix I

ANALYSIS OF CHANGES IN COMPOSITION OF COHORTS DUE TO ARRIVAL OF *SULPHAPYRIDINE*

The intervention could initiate heterogeneous migration or fertility responses among parents of the cohorts under study. If such responses change the composition of cohorts in favour of children with high levels of human capital, this would provide an alternative explanation for the long-term results. I address this concern with individual and family data.

First, in Table I1 I examine whether the arrival of *sulphapyridine* affected the composition of the parents. I estimate the effect of treatment intensity on parental characteristics such as whether mother is older than age 28, whether mother has only primary education, whether father has low socio-economic status and whether he works in agriculture. I detect no systematic pattern on these parental characteristics, except for maternal education. While the result for maternal schooling should be interpreted with caution, because the share of mothers with unknown education is substantial, the estimates probably pick up the general migration pattern, flowing away from economically disadvantaged regions, rather than the effect of *sulphapyridine*. For the long-term effects of pneumonia reduction, if low maternal education is associated with poor infant health, such a finding means that they might be underestimated.

Second, in Table I2 I analyse whether the arrival of *sulphapyridine* affected completed fertility of the mothers. This outcome is more accurate for our estimation sample compared to the regional-level crude birth and marital fertility rates that measure period fertility. The results on family size for the total sample and by subsamples, distinguished on different socio-economic and demographic characteristics are presented based on the specification with region-specific time trends, to diminish concern that they capture pre-treatment convergence. I do not find significant effects of the intervention on completed fertility in any of these samples. While being insignificant, consistently with previous observations, the results tentatively point to the positive effects from intervention among low-resource families, measured with low SES and

low schooling (+1.2-1.6% of the pre-treatment rate) and otherwise for high-resource families (-1.8- -3.8% of the pre-treatment rate). Not only, as before, could a higher proportion of high-risk babies be present among the treated cohorts, but the drug intervention could also pose higher constraints on resources devoted to each child in poorer families (and lower constraints for richer families). In both cases, the true long-term effect of the reduced pneumonia contagion could be underestimated.

TABLE I1 – REDUCED-FORM ESTIMATES. EFFECTS OF PNEUMONIA EXPOSURE IN INFANCY ON PARENTAL CHARACTERISTICS OF COHORTS 1934–1943, SWEDEN

| | (1)<br>*Mother old* | (2)<br>*Mother only primary schooling* | (3)<br>*Father SES low* | (4)<br>*Father in agriculture* |
|---|---|---|---|---|
| Sample 'Ln labour income' | | | | |
| post1939Xbaseline pneumonia mortality | -0.0045 | 0.0272** | 0.0091 | -0.0080 |
| | (0.0066) | (0.0112) | (0.0154) | (0.0079) |
| Individuals | 878,606 | 878,606 | 878,606 | 878,606 |
| R-squared | 0.01 | 0.07 | 0.04 | 0.07 |
| Sample 'Years of schooling' | | | | |
| post1939Xbaseline pneumonia mortality | -0.0042 | 0.0277** | 0.0099 | -0.0083 |
| | (0.0065) | (0.0114) | (0.0153) | (0.0081) |
| Individuals | 879,175 | 879,175 | 879,175 | 879,175 |
| R-squared | 0.01 | 0.07 | 0.04 | 0.04 |
| Sample 'Length of stay in hospital' | | | | |
| post1939Xbaseline pneumonia mortality | -0.0046 | 0.0267** | 0.0105 | -0.0107 |
| | (0.0068) | (0.0113) | (0.0159) | (0.0085) |
| Individuals | 852,460 | 852,460 | 852,460 | 852,460 |
| R-squared | 0.01 | 0.07 | 0.04 | 0.07 |

*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. All Models are estimated according to Eq.1. Pneumonia mortality rate is per 1000 mid-year population, normalised (dividing by its 95th-5th percentile range). Age interval for ln labour income is ages 44–60, and for length of stay in hospital is ages 53–60. *Pre-mean* denotes mean of the outcome in 1934–1938.

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

TABLE I2 – REDUCED-FORM ESTIMATES. EFFECTS OF PNEUMONIA EXPOSURE IN INFANCY ON COMPLETED FERTILITY OF THE MOTHERS BY SUBGROUPS IN SWEDEN, COHORTS 1934–1943

| | (1) | (2) | (3) | (4) | (5) | (6) | (7) |
|---|---|---|---|---|---|---|---|
| | | *Mother's schooling* | | | *Father's SES* | | |
| | Full sample | primary | more than primary | unknown | low | high | unknown |
| post1939Xbaseline pneumonia mortality | -0.0087 | 0.0280 | -0.0382 | -0.0294 | 0.0346 | -0.0898 | -0.0240 |
| | (0.0192) | (0.0282) | (0.0437) | (0.0214) | (0.0229) | (0.0568) | (0.0220) |
| Pre-mean | 2.245 | 2.256 | 2.186 | 2.256 | 2.163 | 2.352 | 2.274 |
| Individuals | 811,241 | 311,387 | 126,205 | 373,649 | 279,541 | 95,382 | 436,318 |
| R-sq | 0.0556 | 0.0746 | 0.0815 | 0.0640 | 0.0620 | 0.0671 | 0.0621 |
| | | (8) | (9) | (10) | (11) | (12) | (13) |
| | | *Mother's age* | | *Father's sector of employment* | | | |
| | | old | young | agriculture | industry | service | unknown |
| post1939Xbaseline pneumonia mortality | | 0.0056 | -0.0193 | -0.0846 | 0.0342 | 0.0126 | -0.0236 |
| | | (0.0202) | (0.0259) | (0.0652) | (0.0253) | (0.0379) | (0.0219) |
| Pre-mean | | 2.234 | 2.254 | 2.586 | 2.175 | 2.083 | 2.275 |
| Individuals | | 366,010 | 445,231 | 64,085 | 183,010 | 127,250 | 436,896 |
| R-sq | | 0.0767 | 0.0834 | 0.0525 | 0.0600 | 0.0528 | 0.0621 |

*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. All Models are estimated according to Eq.1. Pneumonia mortality rate is per 1000 mid-year population, normalised (dividing by its 95th-5th percentile range). Estimates based on ln labour income sample. *Pre-mean* denotes mean of the outcome in 1934–1938.

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

## Appendix J

HETEROGENEOUS EFFECTS

Table presents the results from subsamples distinguished based on father's socio-economic class and mother's age, with the latter approximating differences in infant health. Before, I found that baseline pneumonia was higher for less economically developed and unhealthy regions. The results are generally consistent in finding that individuals from these families gain more from reduction in pneumonia mortality later in life. Children born to mothers aged above the average age in the sample and probably less healthy attain the treatment effects of larger magnitude (Column 1). The treatment effects for the individuals born to families with poor and high socio-economic status are not statistically different, although for ln labour income they are larger for poor families (Column 2). For instance, for labour income, the reduction in pneumonia mortality leads to a 5.7 and 4.3 percent increase for individuals born to older mothers and younger mothers correspondingly; it leads to a 4.8 and 4.2 percent increase for those born to fathers with low and high socio-economic status respectively. Such difference across background characteristics can be plausibly explained by the data limitations, as father's socio-economic status in observed close to retirement ages.

TABLE – REDUCED-FORM ESTIMATES. HETEROGENEOUS EFFECTS OF PNEUMONIA EXPOSURE IN INFANCY BY PARENTAL CHARACTERISTICS ON ADULT OUTCOMES IN SWEDEN, COHORTS 1934–1943

| | (1) Mother's age | | | (2) Father's SES | |
|---|---|---|---|---|---|
| | young | old | unknown | low | high |
| Ln labour income | | | | | |
| post1939Xbaseline | 0.0426*** | 0.0569*** | 0.0427 | 0.0478*** | 0.0417** |
| pneumonia mortality | (0.0115) | (0.0154) | (0.0621) | (0.0176) | (0.0198) |
| Pre-mean | 8.160 | 8.159 | 7.808 | 8.135 | 8.127 |
| Individuals | 445,231 | 366,010 | 67,365 | 618,512 | 260,094 |
| Years of schooling | | | | | |
| post1939Xbaseline | 0.2076*** | 0.2212*** | 0.1914*** | 0.1942*** | 0.1945*** |
| pneumonia mortality | (0.0457) | (0.0624) | (0.0634) | (0.0576) | (0.0563) |
| Pre-mean | 9.525 | 9.778 | 8.994 | 9.634 | 9.465 |
| Individuals | 441,992 | 362,253 | 74,930 | 621,748 | 257,427 |
| Length of stay in hospital | | | | | |
| post1939Xbaseline | -0.0378* | -0.0783*** | 0.0161 | -0.0438*** | -0.0661*** |
| pneumonia mortality | (0.0198) | (0.0166) | (0.0881) | (0.0157) | (0.0221) |
| Pre-mean | 0.671 | 0.645 | 1.086 | 0.707 | 0.640 |
| Individuals | 436,125 | 360,693 | 55,642 | 594,958 | 257,502 |

*Source*: estimations from the *SIP*.

*Notes:* Standard errors (in parentheses) are clustered at a region-of-birth level. All models include region-of-birth fixed effects (49 regions) and year-of-birth fixed effects, and are estimated for both sexes jointly. Pneumonia mortality rate is per 1000 mid-year population, normalised (dividing by its 95th-5th percentile range). Age interval for ln labour income is ages 44–60, and for length of stay in hospital is ages 53–60. All Models are estimated according to Eq.1 plus family-level controls (mother age, mother education, father SES, and father sector of employment) separately for sub-groups defined by parental characteristics. *Pre-mean* denotes mean of the outcome in 1934–1938.

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

## Appendix K

ADDITIONAL TESTS FOR PLAUSIBLE MEASUREMENT ERROR

In Table K1 I estimate the impact of pneumonia mortality in a region and a year of birth on later-life outcomes, while instrumenting it with treatment intensity. Any measurement error should be absorbed from the 2SLS estimates with this check. The 2SLS results give the average effects for individuals for different treatment groups (LATE effects), including those whose mortality from pneumonia mortality has declined after the arrival of *sulphapyridine*. The 2SLS results are all statistically significant, at least at 5 percent significance level, and their magnitudes are now larger. In Table K2 I split baseline pneumonia mortality at the quartiles (4 groups), rank and normalise them and use this indicator in the models (Panel A). The rationale for this test is that regions with certain levels of pneumonia mortality, even measured with some error, are unlikely to fall into wrong quartiles. I further adjusted the pneumonia indicator by dividing it by region-of-birth crude death rate in order to exclude the influence of possible regional differences in registration of deaths (Panel B). While it is unlikely that deaths were not registered in regions in full, because they were aggregated from parish records, these models estimate the impact of pneumonia with the share of pneumonia deaths in total deaths. Similarly to the tests presented in the main body of the text, these robustness checks in general produce estimates which are even larger compared to the main estimates, suggesting that they could be seen as conservative.

TABLE K1 – 2SLS ESTIMATES. EFFECTS OF PNEUMONIA EXPOSURE IN INFANCY ON ADULT OUTCOMES, SWEDEN, COHORTS 1934–1943, BOTH SEXES

| | (1) | (2) | (3) | (4) | (5) | (6) | (7) |
|---|---|---|---|---|---|---|---|
| *Ln labour income* | | | | | | | |
| pneumonia mortality | -0.1228*** | -0.1556*** | -0.0687*** | - | - | -0.1342*** | -0.1458*** |
| | (0.0447) | (0.0360) | (0.0299) | | | (0.0442) | (0.0502) |
| First stage | | | | | | | |
| post1939Xbaseline | -0.3513*** | -0.3258*** | -0.4050*** | -0.1694* | -0.1236 | -0.3514*** | -0.3195*** |
| pneumonia mortality | (0.0611) | (0.0646) | (0.0563) | (0.0963) | (0.1067) | (0.0611) | (0.0022) |
| F-stats | 32.98 | 25.36 | 51.61 | 3.07 | 1.34 | 33.01 | 20221.28 |
| Pre-mean | 8.063 | 8.063 | 8.063 | 8.063 | 8.063 | 8.063 | 8.098 |
| Individuals | 878,606 | 878,606 | 878,606 | 878,606 | 878,606 | 878,606 | 811,241 |
| Mothers | | | | | | | 545,318 |
| *Years of schooling* | | | | | | | |
| pneumonia mortality | -0.4198** | -0.4058** | -0.6436** | - | - | -0.1217** | -0.0187 |
| | (0.2112) | (0.1990) | (0.2872) | | | (0.0504) | (0.0834) |
| First stage | | | | | | | |
| post1939Xbaseline | -0.3512*** | -0.3260*** | -0.4038*** | -0.1688* | -0.1226 | -0.3513*** | -0.3197*** |
| pneumonia mortality | (0.0611) | (0.0646) | (0.0564) | (0.0961) | (0.1069) | (0.0612) | (0.0023) |
| F-stats | 32.95 | 25.45 | 51.30 | 3.08 | 1.30 | 32.76 | 18763.79 |
| Pre-mean | 9.271 | 9.271 | 9.271 | 9.271 | 9.271 | 9.271 | 9.330 |
| Individuals | 879,175 | 879,175 | 879,175 | 879,175 | 879,175 | 879,175 | 804,245 |
| Mothers | | | | | | | 542,422 |
| *Length of stay in hospital* | | | | | | | |
| pneumonia mortality | 0.1186** | 0.1248** | 0.1337*** | - | - | 0.5865** | 0.6256*** |
| | (0.0460) | (0.0624) | (0.0502) | | | (0.2261) | (0.0657) |
| First stage | | | | | | | |
| post1939Xbaseline | -0.3506*** | -0.3251*** | -0.4042*** | -0.1685* | -0.1221 | -0.3507*** | -0.3174*** |
| pneumonia mortality | (0.0611) | (0.0648) | (0.0564) | (0.0963) | (0.1068) | (0.0612) | (0.0022) |
| F-stats | 32.73 | 25.16 | 51.91 | 3.06 | 1.32 | 32.98 | 20548.50 |
| Pre-mean | 0.770 | 0.770 | 0.770 | 0.770 | 0.770 | 0.770 | 0.718 |
| 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. Pneumonia mortality rate is per 1000 mid-year population, normalised (dividing by its 95th-5th percentile range). All models include region-of-birth fixed effects (49 regions) and year-of-birth fixed effects, and are estimated for both sexes jointly. Age interval for ln labour income is ages 44–60, and for the length of stay in hospital is ages 53–60. Model 1 is estimated according to Eq.1 Models 2-6 are estimated according to Eq.1 plus additional controls. Models 2 additionally include disease controls, such as separate interactions between *post1939* and baseline cause-specific mortality (puerperal fever, typhoid fever, diarrhoea, influenza, lung tuberculosis, heart disease, diabetes, and cancer). Models 3 additionally include interactions between *post1939* and baseline region-of-birth controls (stillbirth rate, crude birth rate, share of females, share under age 15, share above age 65, infant mortality rate, ln real regional GDP per capita, ln real wage of worker, share of employed in agriculture, share of employed in industry, ln medical personnel per 1000, ln pharmacies per 1000, ln real hospital spending per 1000, ln number of school-rooms per 1000 pupils, and ln number of teachers per 1000 pupils). Models 4 additionally include interactions between baseline pneumonic regions-of-birth (at the quartiles of baseline pneumonia mortality) and linear time trends. Models 5 additionally include region-of-birth linear time trends. Models 6 add family-level controls (mother age, mother education, father SES, and father sector of employment). Models 7 are estimated according to Eq.2. *Pre-mean* denotes mean of the outcome in 1934–1938.

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

TABLE K2 – REDUCED-FORM ESTIMATES. EFFECTS OF PNEUMONIA EXPOSURE IN INFANCY ON ADULT OUTCOMES WHILE CORRECTING FOR PLAUSIBLE MEASUREMENT ERROR, SWEDEN, COHORTS 1934–1943, BOTH SEXES

| | (1) | (2) | (3) | (4) | (5) | (6) | (7) |
|---|---|---|---|---|---|---|---|
| A – Pneumonic categories as a baseline pneumonia mortality | | | | | | | |
| *Ln labour income* | | | | | | | |
| post1939Xbaseline pneumonia mortality | 0.0486** | 0.0559*** | 0.0269* | 0.0445** | 0.0438** | 0.0542*** | 0.0479*** |
| | (0.0193) | (0.0154) | (0.0141) | (0.0179) | (0.0179) | (0.0198) | (0.0174) |
| Pre-mean | 8.063 | 8.063 | 8.063 | 8.063 | 8.063 | 8.063 | 8.098 |
| Individuals | 878,606 | 878,606 | 878,606 | 878,606 | 878,606 | 878,606 | 811,241 |
| Mothers | | | | | | | 545,318 |
| *Years of schooling* | | | | | | | |
| post1939Xbaseline pneumonia mortality | 0.1357** | 0.1284** | 0.2476** | 0.0527 | 0.0427 | 0.1987*** | 0.1797*** |
| | (0.0657) | (0.0546) | (0.1023) | (0.0466) | (0.0477) | (0.0702) | (0.0251) |
| Pre-mean | 9.271 | 9.271 | 9.271 | 9.271 | 9.271 | 9.271 | 9.330 |
| Individuals | 879,175 | 879,175 | 879,175 | 879,175 | 879,175 | 879,175 | 804,245 |
| Mothers | | | | | | | 542,422 |
| *Length of stay in hospital* | | | | | | | |
| post1939Xbaseline pneumonia mortality | -0.0446*** | -0.0436** | -0.0548*** | -0.0428 | -0.0421 | -0.0460** | -0.0382 |
| | (0.0165) | (0.0180) | (0.0185) | (0.0337) | (0.0339) | (0.0175) | (0.0288) |
| Pre-mean | 0.770 | 0.770 | 0.770 | 0.770 | 0.770 | 0.770 | 0.718 |
| Individuals | 852,460 | 852,460 | 852,460 | 852,460 | 852,460 | 852,460 | 796,818 |
| Mothers | | | | | | | 538,951 |
| B – Adjusting for crude death rate | | | | | | | |
| *Ln labour income* | | | | | | | |
| post1939Xbaseline pneumonia mortality | 0.0443 | 0.0638* | 0.0478** | 0.0295 | 0.0296 | 0.0466* | 0.0686** |
| | (0.0282) | (0.0321) | (0.0229) | (0.0278) | (0.0278) | (0.0277) | (0.0295) |
| Pre-mean | 8.063 | 8.063 | 8.063 | 8.063 | 8.063 | 8.063 | 8.098 |
| Individuals | 878,606 | 878,606 | 878,606 | 878,606 | 878,606 | 878,606 | 811,241 |
| Mothers | | | | | | | 545,318 |
| *Years of schooling* | | | | | | | |
| post1939Xbaseline pneumonia mortality | 0.2065** | 0.2034* | 0.4897** | 0.1894* | 0.0504 | 0.2586** | 0.2780*** |
| | (0.1007) | (0.1128) | (0.2199) | (0.0963) | (0.0843) | (0.1083) | (0.0434) |
| Pre-mean | 9.271 | 9.271 | 9.271 | 9.271 | 9.271 | 9.271 | 9.330 |
| Individuals | 879,175 | 879,175 | 879,175 | 879,175 | 879,175 | 879,175 | 804,245 |
| Mothers | | | | | | | 542,422 |
| *Length of stay in hospital* | | | | | | | |
| post1939Xbaseline pneumonia mortality | -0.0561** | -0.0736** | -0.0801** | -0.0466* | -0.0504 | -0.0537** | -0.0495 |
| | (0.0246) | (0.0281) | (0.0324) | (0.0241) | (0.0591) | (0.0264) | (0.0487) |
| Pre-mean | 0.770 | 0.770 | 0.770 | 0.770 | 0.770 | 0.770 | 0.718 |
| 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 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. In Panel A pneumonia mortality rate is per 1000 mid-year population divided by crude death rate per 1000, normalised (dividing by its 95th-5th percentile range). In Panel B pneumonia mortality rate is ranked within pneumonia mortality per 1000 mid-year population divided at the quartiles, normalised (dividing by its 95th-5th percentile range). Age interval for ln labour income is ages 44–60, and for length of stay in hospital is ages 53–60. Models 1 correspond to Eq.1. Models 2–6 are estimated according to Eq.1 plus additional controls. Models 2 additionally include disease controls, such as separate interactions between *post1939* and baseline cause-specific mortality. Models 3 additionally include interactions between *post1939* and baseline region-of-birth controls. Models 4 additionally include interactions between baseline pneumonic regions-of-birth (divided at the quartiles of baseline pneumonia mortality) and linear time trends. Models 5 additionally include region-of-birth linear time trends. Models 6 add family-level controls. Models 7 are estimated according to Eq.2. *Pre-mean* denotes mean of the outcome in 1934–1938.

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

## Appendix L

ADDITIONAL ROBUSTNESS ANALYSES

The analyses show that the effects of exposure to pneumonia and its sharp reduction due to the nationwide arrival of sulpha antibiotics are marginally sensitive to the inclusion of various region-of-birth and parental controls. The estimates including family fixed effects are even larger, in part suggesting that selective migration across regions or fertility that changed in response to the introduction of drugs against pneumonia does not affect the main findings. In fact, families reporting different regions of birth for their children after the intervention have worse outcomes compared to the children of stayers. It implies that reasons other than better prospective health in places that advanced further after arrival of *sulphapyridine* determined the migration of parents between childbirth, for instance the structure of the local labour force (Enflo, Lundh, and Prado 2014). An additionally conducted test for mean reversion leaves the effects for ln labour income and length of stay in hospital unchanged, albeit reduces the effects for years of schooling (not shown here).

In the estimation sample, the control group includes the children aged 1–5 at the introduction of *sulphapyridine*, and looking more closely with event study analyses I detected no beneficial effects for these children. I perform robustness analyses by changing the control group (see Table L1). 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 (1946), and the results are unaffected by this check (Panel A). Second, I narrow cohorts to those born in 1935–1942, thereby comparing infants to children in ages 1–4 that gives similar results (Panel B). Second, I replace the control group with those born 1925–1929 and thus aged 10–14 at the arrival of *sulphapyridine* (Panel C). The dataset imposes restrictions in this regard, as for these older cohorts I do not observe individuals with any younger siblings born starting from 1930. For these cohorts, because birth intervals have a

negative association with income (e.g., Bengtsson and Dribe 2014), the control group could be positively selected. Despite this limitation, I find beneficial effects of reduction in pneumonia mortality of sizable magnitude for all outcomes.

Similar to other studies looking at the long-term survival of cohorts treated by different socio-economic conditions in childhood (cf. Zajacova and Burgard 2013), the bias related to selective mortality is likely to be downward in this case, as the weakest members of cohorts are more likely to survive in the after-drug period and observed in the registers. To assess it formally, I apply a two-stage Heckman selection procedure to analyse whether selective survival affects the estimates (Heckman 1979). In the first stage, the probability of being observed in the estimation sample is modelled as a function of cohort fixed effects, region of birth fixed effects and sex for all individuals observed as early as the year 1960 (*Folk- och Bostadsräkningen 1960*) in a probit model. An inverse Mills' ratio originating for each individual from the estimates of the probit model is further included as a covariate into the baseline specification, and this procedure does not affect the main findings (see Table L2).

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

compulsory schooling and the results remain unchanged (not shown here). Besides the compulsory schooling reforms, up to 1950, institutions of secondary schooling and vocational training gradually began to educate pupils of both sexes (Ljungberg and Nilsson 2009). Because this process was smooth, the plausible effects from this educational development are likely to be controlled by several socio-economic and demographic region-of-birth characteristics that have already been included in the models.

The arrival of *sulphapyridine* overlapped with two other public health reforms. One reform was related to the rollout of government support to maternal and child health in 1937 until its full nationwide coverage in 1960 (Ström 1946). Based on the official statistical sources (Statistiska Centralbyrån 1920b-1950), I collected information on the coverage of infant population in the regions of birth by this programme. The correlation between the region-of-birth baseline pneumonia mortality and proportion of infants enrolled in the institutional care activities in 1938–1943 is –0.055 (p-value is 0.708), indicating its unlikely influence on the results. Another institutional change occurred with regard to the gradual expansion of hospital births in 1925–1950 (Vallgårda 1996; Wisselgren 2005). It is not problematic if this expansion occurred gradually across the cohorts in a manner unrelated to baseline pneumonia mortality, only its acceleration for the cohorts treated by sulpha antibiotics could potentially violate the results. Based on the same statistical sources, I collected information on the regional proportions of hospital deliveries in total. The correlation between the rate of change in these fractions in 1938–1943 and the region-of-birth baseline pneumonia mortality is 0.022 (p-value is 0.883), which is too weak to affect the results.

Additionally, I test the robustness of the results to the region-specific influence of WWII. During the war, Sweden was neutral, but there were problems with supply of food and fuels (Wangel 1982). Studies looking at the impact of food shortage on either childhood anthropometric measures, such as birth weight, height and BMI (Abolins 1962; Angell-

Andersen et al. 2004), or in female labour force participation (Gustafsson and Jacobsson 1985), by comparing the surrounding cohorts, do not reveal any differences. In this case, any potential country-level changes in child health due to food shortage are ruled out by the birth cohort fixed effects. Differences in price changes of basic products in 1930s–1940s were also indicated across regions of Sweden; food prices increased more considerably in central parts compared to others (Statistiska Centralbyrån 1931-1959). Based on the official statistical sources, I collected the regional price indices for main food products for years 1934–1943 (Statistiska Centralbyrån 1931-1959) and added them into the specification. As evident (see Table L3), the results stay unaffected by this check. Given the importance of month of birth for the later-life outcomes (Bound, Jaeger, and Baker 1995; Buckles and Hungerman 2013), I introduce month of birth and it interactions with control variables that leaves the estimates unaffected (not shown here).

In Table L4, I perform the placebo analysis when the post-treatment period is assigned to earlier cohorts (*post* is defined as 1 for those born during and after 1935, 1936, 1937, and 1938 instead of during and after 1939). With these checks, I detect no significant effects and they all are smaller in magnitudes than those in the main analyses, consistent with arrival of *sulphapyridine* in 1939.

I find that the effects of reduced pneumonia infection in infancy exist for other approximations of health, education and labour productivity available in the dataset (whether on disability pension, ever at hospital, total hospital admissions, more than secondary schooling, tertiary degree, ln total income, ln family income, whether employed). In addition to morbidity outcome, I run the models for mortality in ages 34–60 and detect no systematic treatment effects on mortality for the cohorts under study (see Table L5). I also perform the same analysis for mortality by cause of death (see Table L6). Consistent with previous findings for cause-specific morbidity, the results point to the beneficial effect of reduced pneumonia exposure in infancy

on probability of dying from cardiovascular disease, although it does not attain statistical significance in many specifications. For instance, for the specification with region-of-birth linear 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). The results are similar if I follow these individuals in their mortality outcomes until age 69.

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TABLE L1 – REDUCED-FORM ESTIMATES. EFFECTS OF PNEUMONIA EXPOSURE IN INFANCY ON ADULT OUTCOMES (VARIATIONS WITH TREATMENT AND CONTROL GROUPS), SWEDEN, COHORTS 1925–1945, BOTH SEXES

| | (1) | (2) | (3) | (4) | (5) | (6) | (7) |
|---|---|---|---|---|---|---|---|
| *A - Expanding cohorts: cohorts 1932–1938 and 1939–1945* | | | | | | | |
| *Ln labour income* | | | | | | | |
| post1939Xbaseline pneumonia mortality | 0.0477*** | 0.0569*** | 0.0371*** | 0.0474*** | 0.0412*** | 0.0547*** | 0.0484*** |
| | (0.0140) | (0.0128) | (0.0121) | (0.0155) | (0.0131) | (0.0140) | (0.0136) |
| Pre-mean | 8.021 | 8.021 | 8.021 | 8.021 | 8.021 | 8.021 | 8.069 |
| Individuals | 1,271,553 | 1,271,553 | 1,271,553 | 1,271,553 | 1,271,553 | 1,271,553 | 1,129,255 |
| Mothers | | | | | | | 695,674 |
| *Years of schooling* | | | | | | | |
| post1939Xbaseline pneumonia mortality | 0.1865*** | 0.1673*** | 0.2980*** | 0.1691* | 0.0553 | 0.2653*** | 0.1513*** |
| | (0.0622) | (0.0594) | (0.1177) | (0.0980) | (0.0400) | (0.0626) | (0.0181) |
| Pre-mean | 9.180 | 9.180 | 9.180 | 9.180 | 9.180 | 9.180 | 9.282 |
| Individuals | 1,278,988 | 1,278,988 | 1,278,988 | 1,278,988 | 1,278,988 | 1,278,988 | 1,123,680 |
| Mothers | | | | | | | 694,017 |
| *Length of stay in hospital* | | | | | | | |
| post1939Xbaseline pneumonia mortality | -0.0384*** | -0.0343*** | -0.0525*** | -0.0770*** | -0.0810*** | -0.0395*** | -0.0380* |
| | (0.0115) | (0.0126) | (0.0116) | (0.0243) | (0.0263) | (0.0124) | (0.0215) |
| Pre-mean | 0.776 | 0.776 | 0.776 | 0.776 | 0.776 | 0.776 | 0.717 |
| Individuals | 1,227,889 | 1,227,889 | 1,227,889 | 1,227,889 | 1,227,889 | 1,227,889 | 1,102,011 |
| Mothers | | | | | | | 685,451 |
| *B - Narrowing cohorts: cohorts 1935–1938 and 1939–1942* | | | | | | | |
| *Ln labour income* | | | | | | | |
| post1939Xbaseline pneumonia mortality | 0.0432*** | 0.0496*** | 0.0274** | 0.0381** | 0.0194 | 0.0458*** | 0.0411** |
| | (0.0125) | (0.0106) | (0.0124) | (0.0150) | (0.0153) | (0.0122) | (0.0174) |
| Pre-mean | 8.076 | 8.076 | 8.076 | 8.076 | 8.076 | 8.076 | 8.108 |
| Individuals | 689,170 | 689,170 | 689,170 | 689,170 | 689,170 | 689,170 | 638,458 |
| Mothers | | | | | | | 458,367 |
| *Years of schooling* | | | | | | | |
| post1939Xbaseline pneumonia mortality | 0.1190** | 0.1020** | 0.2256** | 0.1157* | 0.0658 | 0.1567*** | 0.1843*** |
| | (0.0491) | (0.0467) | (0.1000) | (0.0664) | (0.0394) | (0.0470) | (0.0247) |
| Pre-mean | 9.324 | 9.324 | 9.324 | 9.324 | 9.324 | 9.324 | 9.376 |
| Individuals | 689,414 | 689,414 | 689,414 | 689,414 | 689,414 | 689,414 | 632,683 |
| Mothers | | | | | | | 455,612 |
| *Length of stay in hospital* | | | | | | | |
| post1939Xbaseline pneumonia mortality | -0.0369*** | -0.0414** | -0.0520** | -0.0608** | -0.0667** | -0.0377** | -0.0423 |
| | (0.0134) | (0.0159) | (0.0198) | (0.0275) | (0.0304) | (0.0142) | (0.0280) |
| Pre-mean | 0.754 | 0.754 | 0.754 | 0.754 | 0.754 | 0.754 | 0.714 |
| Individuals | 668,728 | 668,728 | 668,728 | 668,728 | 668,728 | 668,728 | 627,461 |
| Mothers | | | | | | | 452,813 |
| *C - Changing a control group: cohorts 1925–1929 and 1939–1943* | | | | | | | |
| *Ln labour income* | | | | | | | |
| post1939Xbaseline pneumonia mortality | 0.0904*** | 0.1136*** | 0.0467** | 0.1163 | 0.0784 | 0.1196*** | *na* |
| | (0.0318) | (0.0278) | (0.0213) | (0.0740) | (0.0753) | (0.0313) | |
| Pre-mean | 7.430 | 7.430 | 7.430 | 7.430 | 7.430 | 7.430 | |
| Individuals | 815,285 | 815,285 | 815,285 | 815,285 | 815,285 | 815,285 | |
| *Years of schooling* | | | | | | | |
| post1939Xbaseline pneumonia mortality | 0.0944 | 0.0902 | 0.2021 | 0.2993 | 0.0462 | 0.2780*** | *na* |
| | (0.0664) | (0.0642) | (0.1367) | (0.2121) | (0.1025) | (0.0761) | |
| Pre-mean | 8.527 | 8.527 | 8.527 | 8.527 | 8.527 | 8.527 | |
| Individuals | 834,447 | 834,447 | 834,447 | 834,447 | 834,447 | 834,447 | |
| *Length of stay in hospital* | | | | | | | |
| post1939Xbaseline pneumonia mortality | -0.0661* | -0.0503* | -0.0273 | -0.0600 | -0.0163 | -0.0650 | *na* |
| | (0.0075) | (0.0287) | (0.0234) | (0.0731) | (0.0976) | (0.0400) | |
| Pre-mean | 1.348 | 1.348 | 1.348 | 1.348 | 1.348 | 1.348 | |
| Individuals | 770,907 | 770,907 | 770,907 | 770,907 | 770,907 | 770,907 | |

*Source*: estimations from the *SIP*.

*Notes*: Standard errors (in parentheses) are clustered at a region-of-birth level. Pneumonia mortality rate is per 1000 mid-year population, normalised (dividing by its 95th-5th percentile range). All models include region-of-birth fixed effects (49 regions) and year-of-birth fixed effects. In the 'expanded' sample, age interval for ln labour

income is ages 46–60, and for length of stay in hospital is ages 55–60. In the 'narrow' sample, age interval for ln labour income is ages 44–60, and for length of stay in hospital is ages 53–60. In the 'change control' sample, age interval for ln labour income is ages 53–60, and for length of stay in hospital is ages 62–69. All models are estimated for both sexes jointly. Models 1 are estimated according to Eq.1 Models 2-6 are estimated according to Eq.1 plus additional controls. Models 2 additionally include disease controls, such as separate interactions between *post1939* and baseline cause-specific mortality (puerperal fever, typhoid fever, diarrhoea, influenza, lung tuberculosis, heart disease, diabetes, and cancer). Models 3 additionally include interactions between *post1939* and baseline region-of-birth controls (stillbirth rate, crude birth rate, share of females, share under age 15, share above age 65, infant mortality rate, ln real regional GDP per capita, ln real wage of worker, share of employed in agriculture, share of employed in industry, ln medical personnel per 1000, ln pharmacies per 1000, ln real hospital spending per 1000, ln number of school-rooms per 1000 pupils, and ln number of teachers per 1000 pupils). Models 4 additionally include interactions between baseline pneumonic regions-of-birth (at the quartiles of baseline pneumonia mortality) and linear time trends. Models 5 additionally include region-of-birth linear time trends. Models 6 add family-level controls (mother age, mother education, father SES, and father sector of employment). Models 7 are estimated according to Eq.2. *Pre-mean* denotes mean of the outcome in 1932–1938 for 'expanded' sample, that in 1934–1938 for 'narrow' sample, and that in 1925–1929 for 'change control' sample.

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

TABLE L2 – REDUCED-FORM ESTIMATES. EFFECTS OF PNEUMONIA EXPOSURE IN INFANCY ON LATER-LIFE OUTCOMES WHILE ADJUSTING FOR SELECTIVE SURVIVAL, SWEDEN, COHORTS 1934–1943

| | (1) |
|---|---|
| Ln labour income | |
| post1939Xbaseline pneumonia mortality, both sexes | 0.0319** |
| | (0.0154) |
| Individuals | 878,606 |
| Years of schooling | |
| post1939Xbaseline pneumonia mortality, both sexes | 0.0472 |
| | (0.0348) |
| Individuals | 879,175 |
| Length of stay in hospital | |
| post1939Xbaseline pneumonia mortality, both sexes | -0.0472* |
| | (0.0244) |
| Individuals | 852,460 |

*Source*: estimations from the *SIP*.

*Notes*: Models adjust for selective survival by using a two-stage Heckman selection procedure. Standard errors (in parentheses) are clustered at a region-of-birth level. All models include region-of-birth fixed effects (49 regions) and year-of-birth fixed effects, and are estimated for both sexes jointly. Pneumonia mortality rate is per 1000 mid-year population, normalized (dividing by its 95th-5th percentile range). Models correspond to Eq.1 plus region-of-birth linear time trends.

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

TABLE L3 – REDUCED-FORM ESTIMATES. PLACEBO TREND BREAKS. EFFECTS OF PNEUMONIA EXPOSURE IN INFANCY ON ADULT OUTCOMES IN SWEDEN, COHORTS 1934–1943 BOTH SEXES

| | (1)<br>Ln labour income | (2)<br>Years of schooling | (3)<br>Length of stay in hospital |
|---|---|---|---|
| post1935Xbaseline pneumonia mortality | 0.0023 | 0.0353 | -0.0053 |
| | (0.0147) | (0.0319) | (0.0344) |
| Pre-mean | 8.013 | 9.049 | 0.839 |
| Rsq | 0.031 | 0.068 | 0.002 |
| Individuals | 852,460 | 879,175 | 852,460 |
| post1936Xbaseline pneumonia mortality | 0.0184 | -0.0045 | -0.0028 |
| | (0.0139) | (0.0303) | (0.0219) |
| Pre-mean | 8.031 | 9.108 | 0.827 |
| Rsq | 0.031 | 0.068 | 0.002 |
| Individuals | 852,460 | 879,175 | 852,460 |
| post1937Xbaseline pneumonia mortality | 0.0027 | -0.0406 | -0.0128 |
| | (0.0133) | (0.0483) | (0.0272) |
| Pre-mean | 8.041 | 9.168 | 0.807 |
| Rsq | 0.031 | 0.068 | 0.002 |
| Individuals | 852,460 | 879,175 | 852,460 |
| post1938Xbaseline pneumonia mortality | 0.0232 | 0.0051 | -0.0250 |
| | (0.0139) | (0.0467) | (0.0279) |
| Pre-mean | 8.050 | 9.217 | 0.787 |
| Rsq | 0.031 | 0.068 | 0.002 |
| Individuals | 852,460 | 879,175 | 852,460 |

*Source*: estimations from the *SIP*.

*Notes*: Standard errors (in parentheses) are clustered at a region-of-birth level. All models include region-of-birth fixed effects (49 regions) and year-of-birth fixed effects, and are estimated for both sexes jointly. Pneumonia mortality rate is per 1000 mid-year population, normalized (dividing by its 95th-5th percentile range). Models correspond to Eq.1 plus region-of-birth linear time trends. Age interval for ln labour income is ages 44–60, and for length of stay in hospital is ages 53–60. *Pre-mean* denotes mean of the outcome in 1934–1938.

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

TABLE L4– REDUCED-FORM ESTIMATES. EFFECTS OF PNEUMONIA EXPOSURE IN INFANCY ON ADULT OUTCOMES WHILE CONTROLLING FOR REGIONAL FOOD PRICES, SWEDEN, COHORTS 1934–1943

| | (1) All | (2) Men | (3) Women |
|---|---|---|---|
| Ln labour income | | | |
| post1939Xbaseline pneumonia mortality | 0.0429*** | 0.0302 | 0.0553*** |
| | (0.0143) | (0.0202) | (0.0136) |
| Pre-mean | 8.063 | 8.321 | 7.798 |
| Individuals | 878,606 | 446,511 | 432,095 |
| Years of schooling | | | |
| post1939Xbaseline pneumonia mortality | 0.1472** | 0.1446** | 0.1510** |
| | (0.0562) | (0.0567) | (0.0591) |
| Pre-mean | 9.271 | 9.274 | 9.268 |
| Individuals | 879,175 | 446,736 | 432,439 |
| Length of stay in hospital | | | |
| post1939Xbaseline pneumonia mortality | -0.0417*** | -0.0465** | -0.0369* |
| | (0.0129) | (0.0198) | (0.0213) |
| Pre-mean | 0.770 | 0.785 | 0.775 |
| Individuals | 852,460 | 430,096 | 422,364 |

*Source*: estimations from the *SIP*.

*Notes*: Standard errors (in parentheses) are clustered at a region-of-birth level. All models include region-of-birth fixed effects (49 regions) and year-of-birth fixed effects. Pneumonia mortality rate is per 1000 mid-year population, normalized (dividing by its 95th-5th percentile range). Age interval for ln labour income is ages 44–60, and for the length of stay in hospital is ages 53–60. Models correspond to Eq.1 plus price the index of main food products. *Pre-mean* denotes mean of the outcome in 1934–1938.

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

TABLE L5 – REDUCED-FORM ESTIMATES. EFFECTS OF PNEUMONIA EXPOSURE IN INFANCY ON MORTALITY IN AGES 34–60, SWEDEN, COHORTS 1934–1943

| | (1) | (2) | (3) | (4) | (5) | (6) | (7) |
|---|---|---|---|---|---|---|---|
| post1939Xbaseline pneumonia mortality, *both sexes* | 0.0003 | 0.0005 | -0.0009 | -0.0029 | -0.0051 | 0.0003 | -0.0013 |
| | (0.0019) | (0.0016) | (0.0018) | (0.0033) | (0.0041) | (0.0026) | (0.0026) |
| Pre-mean | 0.0804 | 0.0804 | 0.0804 | 0.0804 | 0.0804 | 0.0804 | 0.0464 |
| Individuals | 895,701 | 895,701 | 895,701 | 895,701 | 895,701 | 895,701 | 818,237 |
| Mothers | | | | | | | 548,422 |
| post1939Xbaseline pneumonia mortality, *men* | -0.0018 | -0.0027 | -0.0035 | -0.0079 | -0.0123* | -0.0017 | 0.0047 |
| | (0.0030) | (0.0028) | (0.0033) | (0.0053) | (0.0064) | (0.0034) | (0.0051) |
| Pre-mean | 0.0994 | 0.0994 | 0.0994 | 0.0994 | 0.0994 | 0.0994 | 0.0576 |
| Individuals | 456,960 | 456,960 | 456,960 | 456,960 | 456,960 | 456,960 | 414,926 |
| Mothers | | | | | | | 334,536 |
| post1939Xbaseline pneumonia mortality, *women* | 0.0023 | 0.0038** | 0.0018 | 0.0023 | 0.0025 | 0.0023 | -0.0023 |
| | (0.0017) | (0.0015) | (0.0017) | (0.0026) | (0.0032) | (0.0021) | (0.0043) |
| Pre-mean | 0.0607 | 0.0607 | 0.0607 | 0.0607 | 0.0607 | 0.0607 | 0.0351 |
| Individuals | 438,741 | 438,741 | 438,741 | 438,741 | 438,741 | 438,741 | 403,311 |
| Mothers | | | | | | | 326,869 |

*Source*: estimations from the *SIP*.

*Notes*: Standard errors (in parentheses) are clustered at a region-of-birth level. Pneumonia mortality rate is per 1000 mid-year population, normalized (dividing by its 95th-5th percentile range). All models include region-of-birth fixed effects (49 regions) and year-of-birth fixed effects. Model 1 is estimated according to Eq.1 Models 2-6 are estimated according to Eq.1 plus additional controls. Models 2 additionally include disease controls, such as separate interactions between *post1939* and baseline cause-specific mortality (puerperal fever, typhoid fever, diarrhoea, influenza, lung tuberculosis, heart disease, diabetes, and cancer). Models 3 additionally include interactions between *post1939* and baseline region-of-birth controls (stillbirth rate, crude birth rate, share of females, share under age 15, share above age 65, infant mortality rate, ln real regional GDP per capita, ln real wage of worker, share of employed in agriculture, share of employed in industry, ln medical personnel per 1000, ln pharmacies per 1000, ln real hospital spending per 1000, ln number of school-rooms per 1000 pupils, and ln number of teachers per 1000 pupils). Models 4 additionally include interactions between baseline pneumonic regions-of-birth (at the quartiles of baseline pneumonia mortality) and linear time trends. Models 5 additionally include region-of-birth linear time trends. Models 6 add family-level controls (mother age, mother education, father SES, and father sector of employment). Models 7 are estimated according to Eq.2. *Pre-mean* denotes mean of the outcome in 1934–1938.

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

TABLE L6 – REDUCED-FORM ESTIMATES. EFFECTS OF PNEUMONIA EXPOSURE IN INFANCY ON CAUSE-SPECIFIC MORTALITY IN AGES 34–60, BOTH SEXES, SWEDEN, COHORTS 1934–1943

| | (1) | (2) | (3) | (4) | (5) | (6) | (7) |
|---|---|---|---|---|---|---|---|
| *Infectious/Respiratory* | | | | | | | |
| post1939Xbase | 0.0004 | 0.0004 | 0.0003 | 0.0007 | 0.0009 | 0.0004 | 0.0003 |
| pneumonia | (0.0003) | (0.0004) | (0.0003) | (0.0006) | (0.0007) | (0.0003) | (0.0005) |
| Pre-mean | 0.0033 | 0.0033 | 0.0033 | 0.0033 | 0.0033 | 0.0033 | 0.0019 |
| *CVD* | | | | | | | |
| post1939Xbase | -0.0014 | -0.0013 | -0.0015 | -0.0046** | -0.0057*** | -0.0014 | 0.0002 |
| pneumonia | (0.0012) | (0.0011) | (0.0012) | (0.0017) | (0.0017) | (0.0014) | (0.0014) |
| Pre-mean | 0.0218 | 0.0218 | 0.0218 | 0.0218 | 0.0218 | 0.0218 | 0.0133 |
| *Diabetes* | | | | | | | |
| post1939Xbase | 0.0001 | -0.0001 | 0.0001 | -0.0001 | 0.0001 | 0.0001 | -0.0001 |
| pneumonia | (0.0002) | (0.0002) | (0.0002) | (0.0005) | (0.0006) | (0.0002) | (0.0003) |
| Pre-mean | 0.0015 | 0.0015 | 0.0015 | 0.0015 | 0.0015 | 0.0015 | 0.0008 |
| *Cancer* | | | | | | | |
| post1939Xbase | 0.0015 | 0.0024*** | 0.0006 | 0.0022 | 0.0017 | 0.0015 | -0.0001 |
| pneumonia | (0.0011) | (0.0009) | (0.0011) | (0.0020) | (0.0025) | (0.0012) | (0.0017) |
| Pre-mean | 0.0293 | 0.0293 | 0.0293 | 0.0293 | 0.0293 | 0.0293 | 0.0191 |
| *Degenerative* | | | | | | | |
| post1939Xbase | -0.0004 | -0.0007 | -0.0004 | -0.0013 | -0.0016 | -0.0004 | -0.0017** |
| pneumonia | (0.0005) | (0.0005) | (0.0005) | (0.0010) | (0.0011) | (0.0005) | (0.0008) |
| Pre-mean | 0.0077 | 0.0077 | 0.0077 | 0.0077 | 0.0077 | 0.0077 | 0.0040 |
| *Mental* | | | | | | | |
| post1939Xbase | -0.0002 | -0.0004 | -0.0003 | -0.0004 | -0.0005 | -0.0003 | -0.0008** |
| pneumonia | (0.0003) | (0.0003) | (0.0003) | (0.0004) | (0.0005) | (0.0003) | (0.0004) |
| Pre-mean | 0.0019 | 0.0019 | 0.0019 | 0.0019 | 0.0019 | 0.0019 | 0.0009 |
| *Other* | | | | | | | |
| post1939Xbase | 0.0003 | 0.0002 | 0.0004 | 0.0006 | 0.0001 | 0.0004 | 0.0009 |
| pneumonia | (0.0007) | (0.0009) | (0.0007) | (0.0013) | (0.0018) | (0.0009) | (0.0010) |
| Pre-mean | 0.0146 | 0.0146 | 0.0146 | 0.0146 | 0.0146 | 0.0146 | 0.0061 |
| Individuals | 895,701 | 895,701 | 895,701 | 895,701 | 895,701 | 895,701 | 818,237 |
| Mothers | | | | | | | 548,422 |

*Source*: estimations from the *SIP*.

*Notes*: Standard errors (in parentheses) are clustered at a region-of-birth level. Pneumonia mortality rate is per 1000 mid-year population, 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. Model 1 is estimated according to Eq.1 Models 2-6 are estimated according to Eq.1 plus additional controls. Models 2 additionally include disease controls, such as separate interactions between *post1939* and baseline cause-specific mortality (puerperal fever, typhoid fever, diarrhoea, influenza, lung tuberculosis, heart disease, diabetes, and cancer). Models 3 additionally include interactions between *post1939* and baseline region-of-birth controls (stillbirth rate, crude birth rate, share of females, share under age 15, share above age 65, infant mortality rate, ln real regional GDP per capita, ln real wage of worker, share of employed in agriculture, share of employed in industry, ln medical personnel per 1000, ln pharmacies per 1000, ln real hospital spending per 1000, ln number of school-rooms per 1000 pupils, and ln number of teachers per 1000 pupils). Models 4 additionally include interactions between baseline pneumonic regions-of-birth (at the quartiles of baseline pneumonia mortality) and linear time trends. Models 5 additionally include region-of-birth linear time trends. Models 6 add family-level controls (mother age, mother education, father SES, and father sector of employment). Models 7 are estimated according to Eq.2. *Pre-mean* denotes mean of the outcome in 1934–1938.

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