Life Course Determinants of Mortality

How conditions in early life affect height, income, and health

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An Introduction to Inequalities in Health using Historical Causes of Death

Lectures:

1: The epidemiological transition

2: Understanding inequalities in health

3: Life course determinants of mortality

4: Intergenerational transmission of health and behaviors

5: Medical perspectives

6: Public health perspectives

Contents

1. The long-term mortality decline and underlying period and cohort factors
2. Life-course models and methods

BREAK

3. Examples using individual-level data for southern Sweden

Recommended reading

The long-term mortality decline:

- Bengtsson, T. (2015) Mortality: The Great Historical Decline. In International Encyclopedia of the Social & Behavioral Sciences, 868-783

Life-course models:

- Barker, D. J. P. (2001) Fetal and infant origins of adult disease. Monatsschrift Kinderheilkunde, 149, Supplement 1, S2-S6
- Crimmins, E. M., & Finch, C. E. (2006). Infection, inflammation, height, and longevity. Proceedings of the National Academy of Sciences of the United States of America, 103, 2, 498-503
- Kuh, D. & Ben-Shlomo, Y. (eds.) (2004) A life course approach to chronic disease epidemiology. Oxford: OUP, Ch 2, 15-37, Ch 16, 371-395

Examples of life-course analyses using individual level data:

- Bengtsson, T., & Lindström, M. (2003) Airborne infectious diseases during infancy and mortality in later life in southern Sweden, 1766-1894. International Journal of Epidemiology, 32, 2, 286-294
- Helgertz, J & Bengtsson, T (2019), The Long-Lasting Influenza: The Impact of Fetal Stress During the 1918 Influenza Pandemic on Socioeconomic Attainment and Health in Sweden, 1968–2012, Demography, 56, 4, 1389-1425
- Helgertz, J., & Nilsson, A. (2019) The effect of birth weight on hospitalizations and sickness absences: a longitudinal study of Swedish siblings. Journal of Population Economics 32, 53–178
- Lazuka, V, Quaranta, L & Bengtsson, T (2016), ‘Fighting infectious disease: Evidence from Sweden 1870-1940’, Population and Development Review, 42, 1, 27-52

1. The long-term mortality decline

- The crude death rate started to decline around 1800 in many western countries prior to the modernisation of their economies

The demographic transition: Sweden 1735-1990

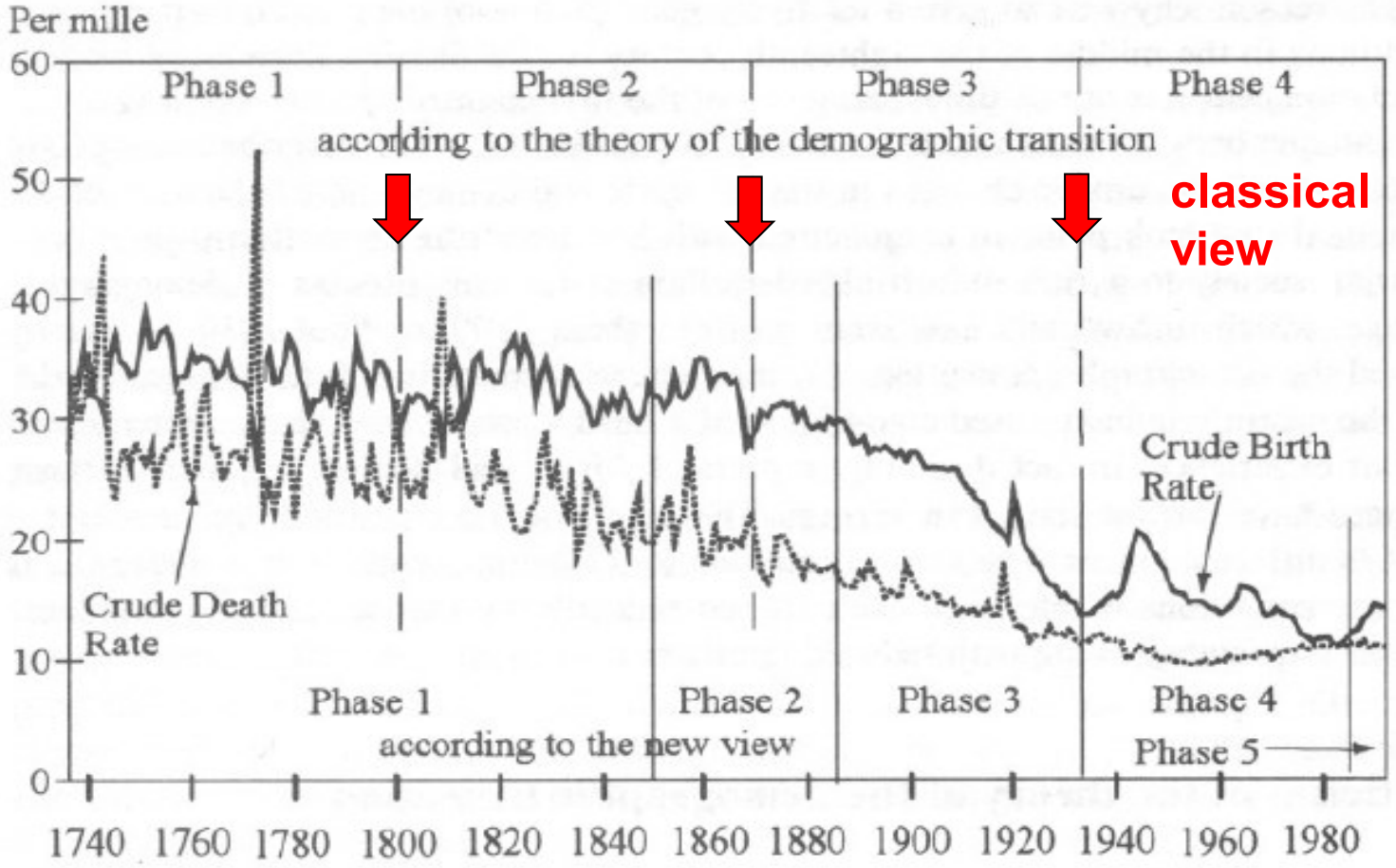


Diagram 1. Crude birth and death rates for Sweden 1736-1992.

Bengtsson & Ohlsson (1994)

Characteristics of the mortality decline

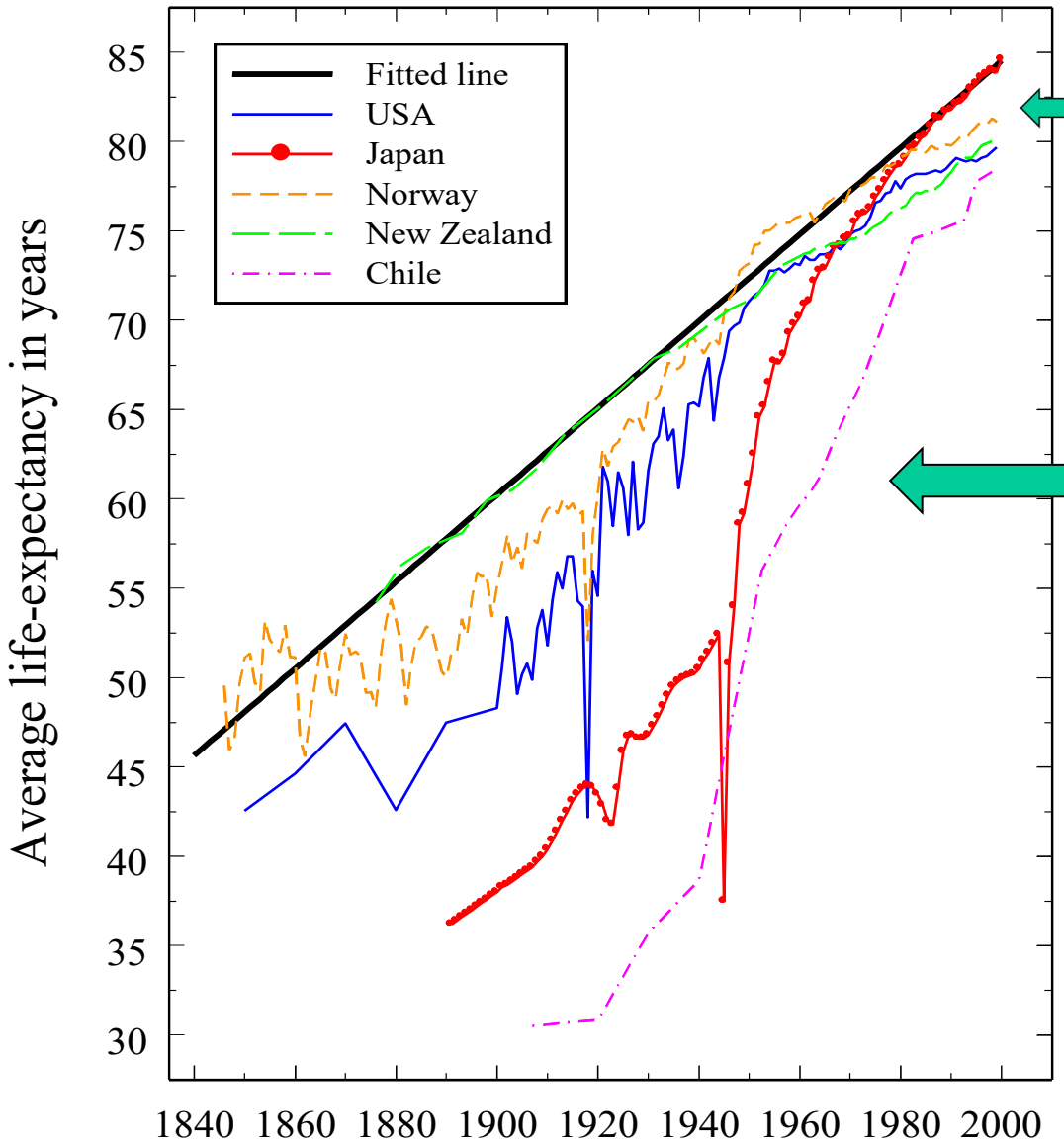
- The crude death rate started to decline around 1800 in many western countries prior to the modernisation of their economies
- Infant mortality started to decline already in the 1730s driven by a reduction in smallpox mortality
- Adult mortality started to decline later, during the second half of the 19th century (Bengtsson 2015)

Characteristics of the mortality decline

- The crude death rate started to decline around 1800 in many western countries prior to the modernisation of their economies
- Infant mortality started to decline already in the 1730s driven by a reduction in smallpox mortality
- Adult mortality started to decline later, during the second half of the 19th century (Bengtsson 2015)
- Changes in causes of death: from epidemic, to infectious, to chronic diseases = epidemiological transition (Omran 1972)
- Linear increase in life-expectancy at birth with three months per year in best practice countries after 1840

(Oeppen & Vaupel 2002)

Best Practice Female Life-Expectancy at Birth



Today the increase is due to falling adult mortality

Catch up with one year per year due to rapid decline in infant mortality

Oeppen & Vaupel (2002)

Causes of the mortality decline

Period factors:

UN 1953 – multiple causes

1. Progress in public health
2. Medical enhancements
3. Improved personal hygiene
4. Better diets

UN 1973 as above +

5. Natural causes – decreasing virulence of diseases

Other period factors?

The role of public health interventions, personal hygiene, and medicine

1. Understanding of the mode of transmission of diseases developed during the nineteenth century making prevention by pasteurization, use of antiseptics and improvements in water and sanitary systems possible
2. Vaccines was developed from the 1890s onwards as a follow up to smallpox vaccination starting in 1800
3. Drugs to cure infectious diseases was developed in the 1930s

Table 5. *Discoveries in the control of major fatal infectious diseases sinc around 1800: vaccines and drugs.*

A. Vaccines			B. Drugs		
Date	Disease	Developer	Date	Drug	Developer
1798	Smallpox	Jenner	1908	Salvarsan	Ehrlich
1881	Anthrax	Pasteur	1935	Sulfanomides	Domagk
1885	Rabies	Pasteur	1941	Penicillin	Fleming, Florey, Chain
1892	Diphtheria	von Behring	1944	Streptomycin	Waksman
1896	Cholera	Kolle	1947 on	Broad spectrum antibiotics[a]	
1906	Pertussis	Bordet-Gengou			
1921	Tuberculosis	Calmette, Guerin			
1927	Tetanus	Ramon, Zoeller			
1930	Yellow fever	Theiler			
	Typhoid fever	Weigl			
1948	DTP	(Multiple)			
1950	Polio	Salk			
1954	Measles	Enders, Peebles			

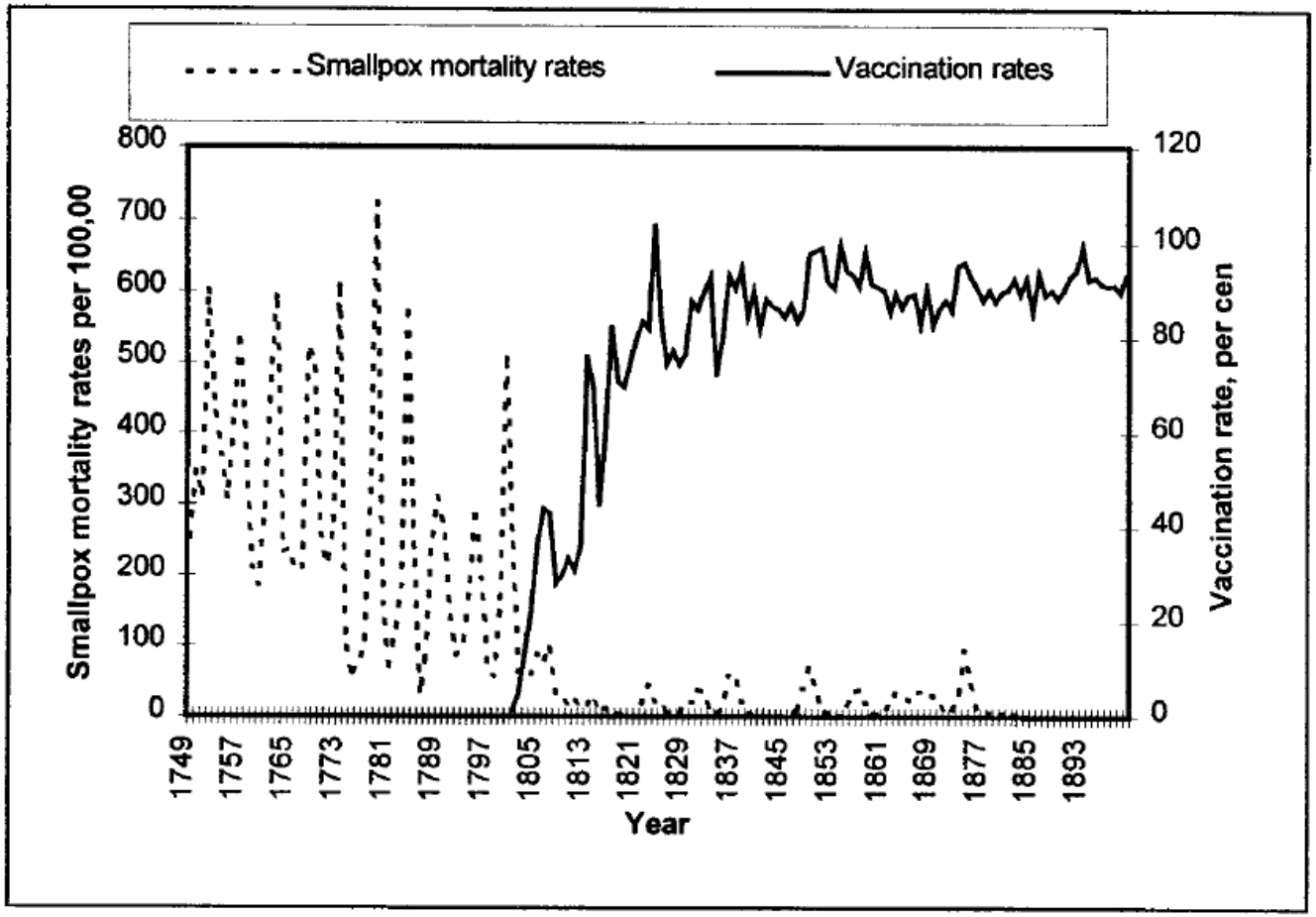
Note: [a]Lappé (1982, pp. 22–4) provides a lengthy tabulation of major antibiotics in use during 1975–81 in the United States. See also Brumfitt and Hamilton-Miller (1988).
Sources: Panel A: Parish (1965), Plotkin and Mortimer (1988).
Panel B: Baldry (1976).

Easterlin (1999)

Vaccinations

- Infant mortality decline in Sweden before 1800 was entirely due to a reduction of smallpox mortality
- Only 1 % of children were inoculated before 1800 (Sköld 1996) - minor effect on the smallpox decline.
- Vaccinations started in 1801, covered 50% of children in 1815, became compulsory in 1816

Figure I.1. Smallpox death rates per 100,000 and vaccination rates (proportion of the number of born children during the previous year, infant mortality removed)



Source: Bidrag till Sveriges officiella statistik (BiSOS), serie K), 1860–1900. Sundbärg (1905), 109–60. Compiled population records. The older archive of the Table commission. National archives.

Smallpox death rates and vaccinations: Sweden 1750-1900

Sköld (1996)

Vaccinations

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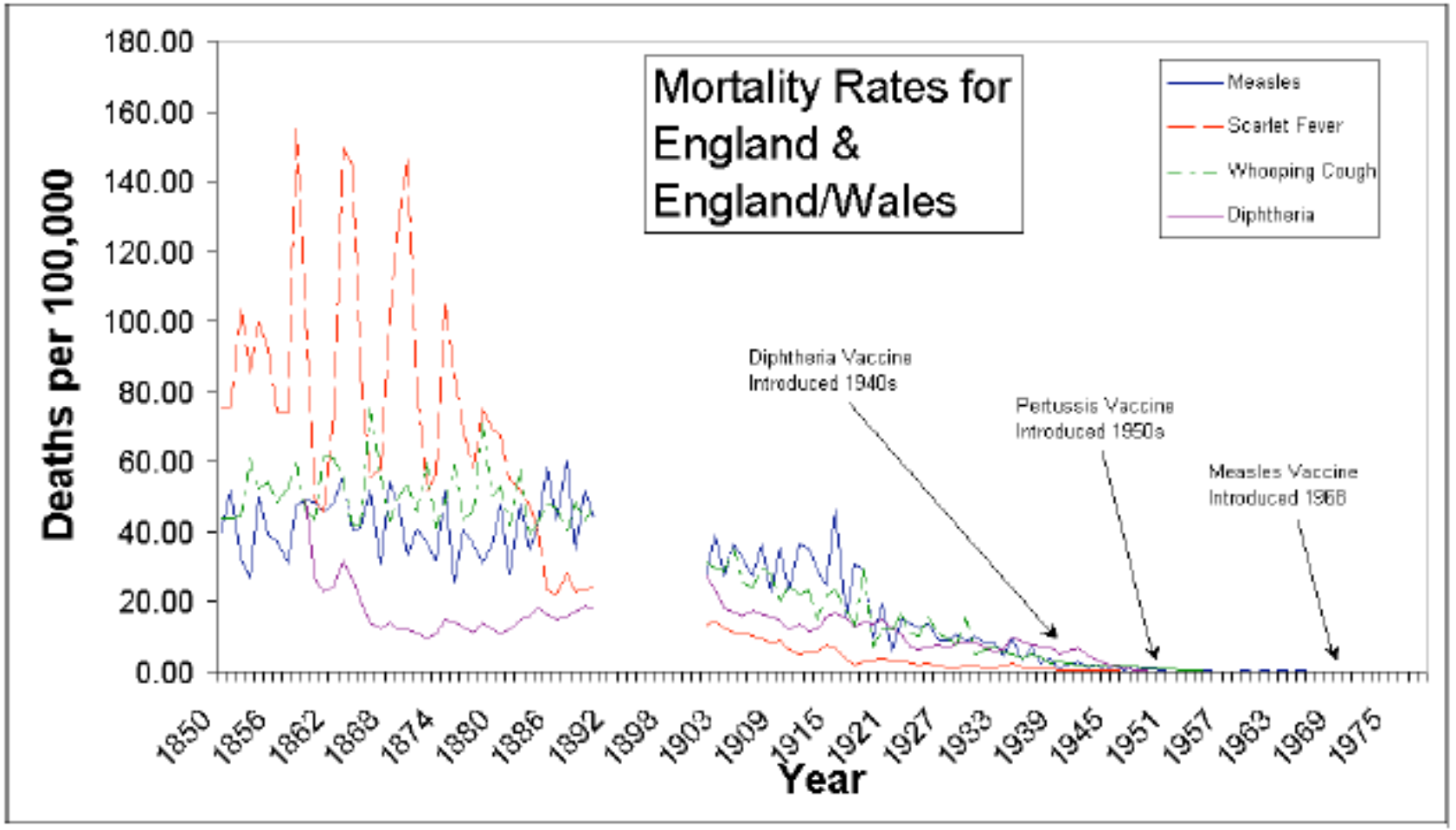
- Only 1 % of children were inoculated before 1800 (Sköld 1996) - minor effect on the smallpox decline.
- Vaccinations started in 1801, covered 50% of children in 1815, became compulsory in 1816
- Thus, the decline in smallpox mortality was not dependent on vaccinations
- Similar for several other diseases

England & Wales: mortality in measles, scarlet fever, whooping cough, and diphtheria

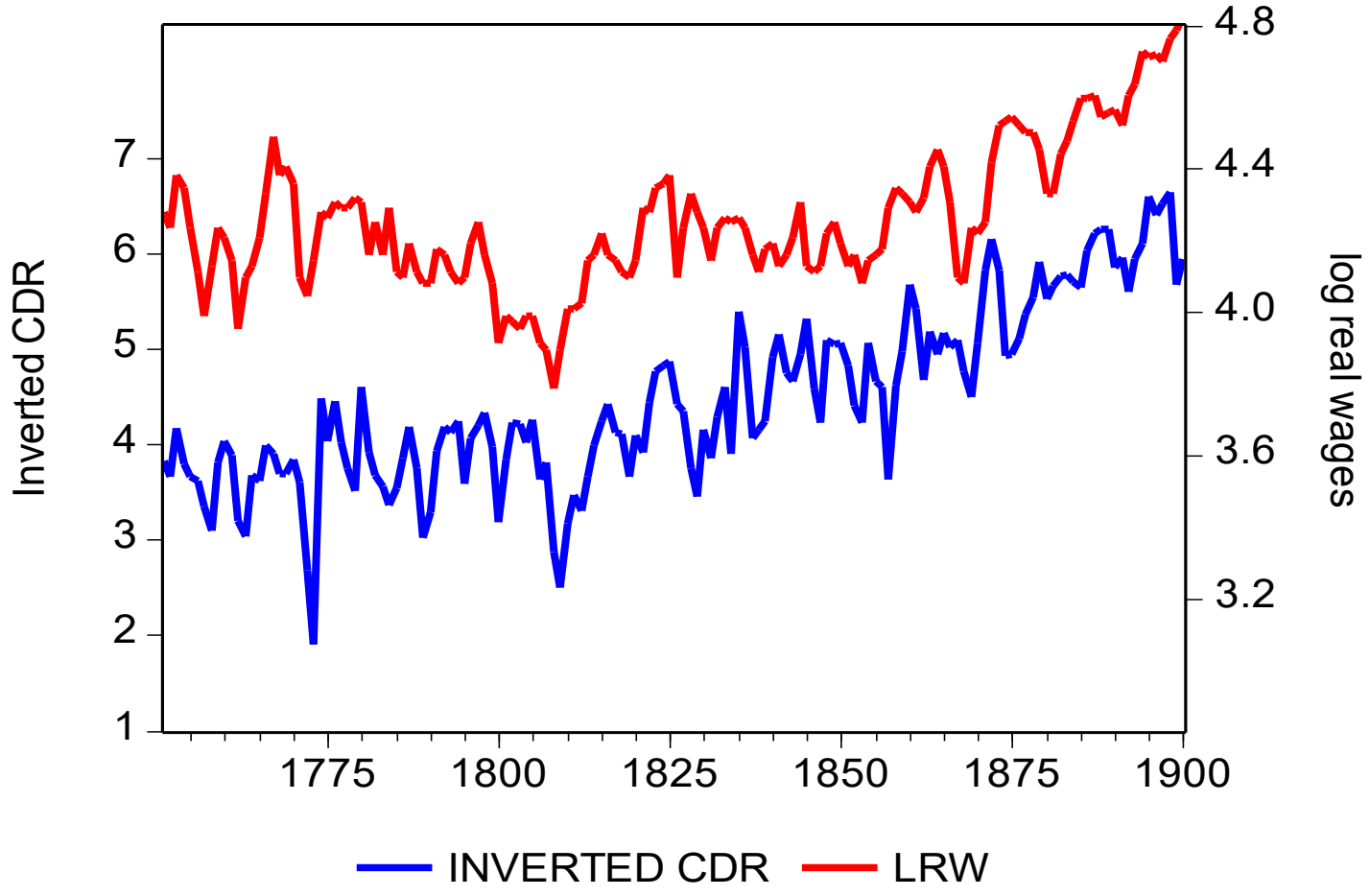


Mortality statistics for England and Wales for 95 years. Office of National Statistics (1997)

Better diets

- Nutritional influence on many diseases minimal: smallpox, malaria, plague, typhoid, tetanus, yellow fever, …
  (Rothberg and Rabb 1984)
- London Bills of Mortality: 51 out of 229,250 died from starvation over a 20-year period (Graunt 1662)
- Mortality crises – majority not related to food
  (Livi Bacci 1991)
- Late emergence of a social gradient in mortality
  (Bengtsson and Dribe 2011)
- The impact of food prices on mortality tells another story
  (Bengtsson 2000)

Log real wages and crude death rate: Sweden, 1750-1900



Bengtsson (2000)

Causes of mortality decline

Cohort factors:

1. Improved foetal and early childhood growth due to more nutritious diets

2. Decreased disease load in early childhood

   The result is lower levels of mortality throughout life

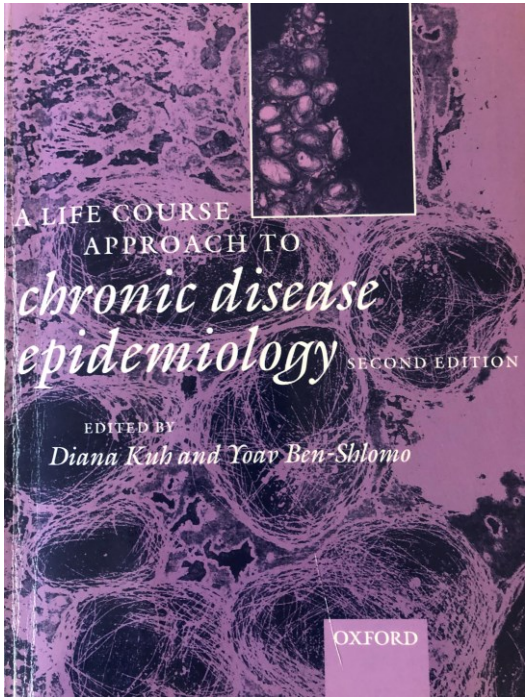
Other cohort factors?

2. Life-course models and methods:

Why are early-life conditions important for later life health?

Conditions in early life influence the development of

- cardiovascular diseases
- respiratory and allergic diseases
- diabetes, hypertension, and obesity
- breast and testicular cancers
- neuropsychiatric



(Ben-Shlomo & Kuh 2004)

Why sensitivity in early life?

Development of organs and cells are fastest during the foetal stage and in first years of life

We are therefore sensitive to disturbances caused by malnutrition, diseases and other adverse conditions during these stages

Direct effects

- Fetal stage:
  - Rubella early in pregnancy increase the risk of congenital cataract and poor childhood development (Gregg 1942)
  - Malnutrition increase the risk of biological disorders, hypertension, and arteriosclerotic heart disease

    (Forsdahl 1977; Barker 1988, 1995)
  - Spanish flu increase morbidity and mortality risks in later life (Almond 2006; Helgertz and Bengtsson 2019)
- First year(s) of life:
  - Infections/inflammations increase the risk of atherosclerosis

    (Osier 1908; Nieto 1998; Bengtsson and Lindström 2000, 2003; Finch and Crimmins 2004)
- Non-specific effects (NSE) of vaccination
  - Smallpox, BCG, OPV and MV (Aaby and Benn 2019)

Direct effects

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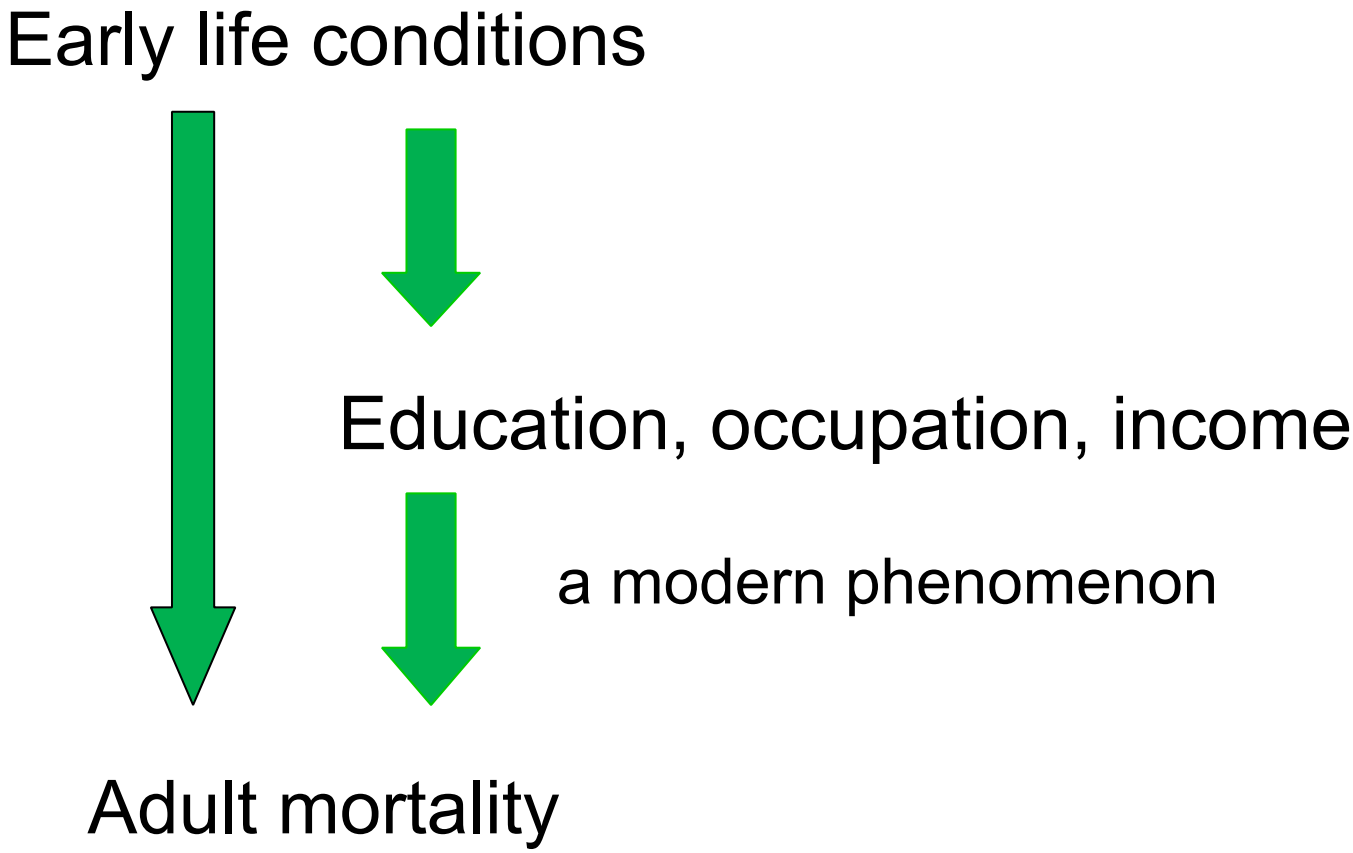
Life cycle models: Direct and indirect effects



Bengtsson and Broström (2009)

Indirect effects: The theory of technofysio evolution

Nutritional status in early life$_g$ = f (diet, disease)

Height$_g$ and output$_g$

Length of life$_g$

Nutritional status in early life$_{g+1}$

and so on for ever

Fogel and Costa (1997)

Diet or disease?

Net nutrition = the gross-nutritional intake minus the need to fight diseases

Some diseases lower our ability to take up nutrition

Fogel is taken the same position as McKeown (1976), that the mortality decline started with improved diets and continued with sanitary improvements, medical care, etc.

Cohort evidence at macro level

- Empirical evidence at macro level found in the 1930s (Kermack, McKendrick and McKinlay 1934)
- The focus in the 1950s turned to period factors
- Cohort factors gained interest again in the 1980s (Preston and de Walle 1978; Fridlizius 1989)

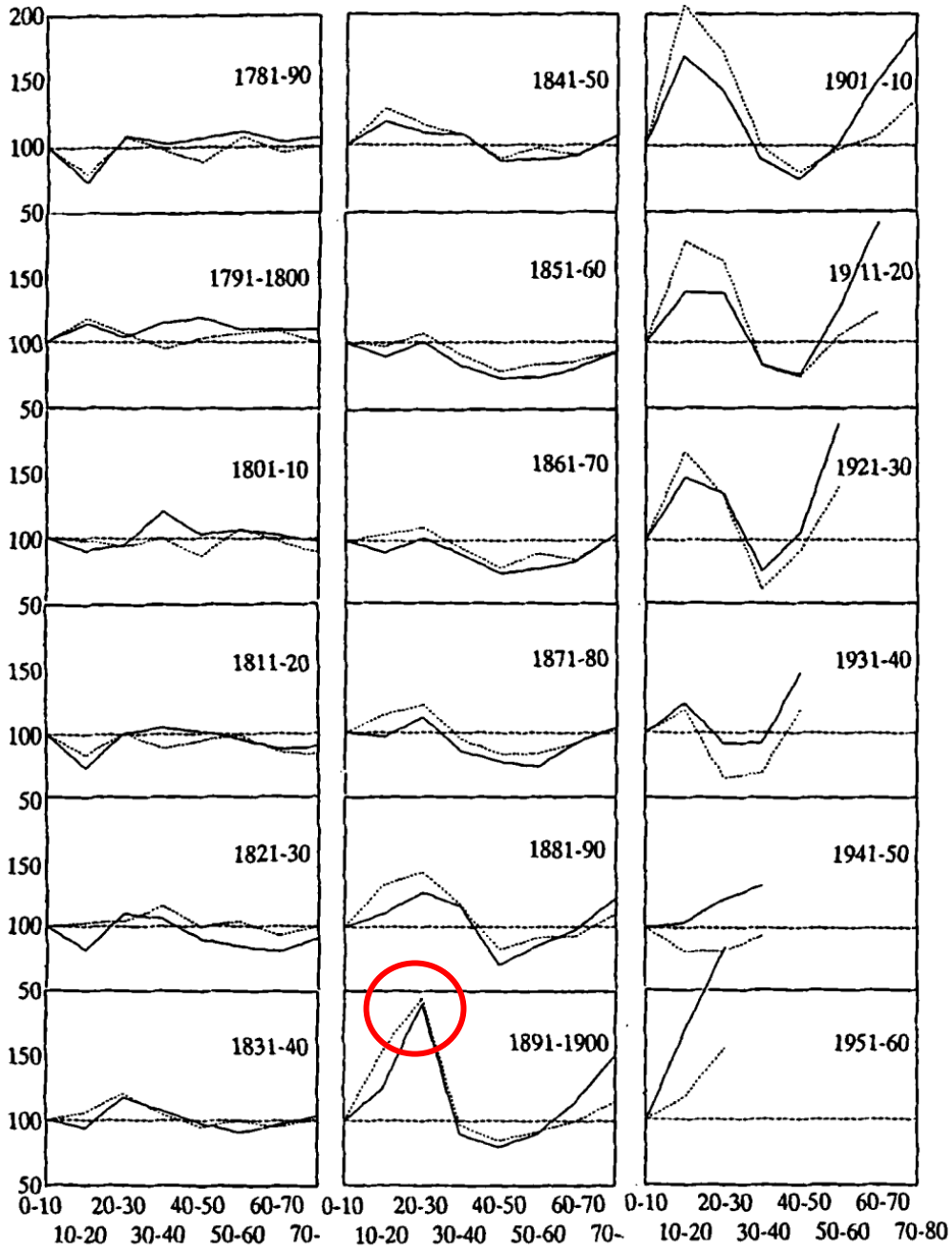
Cohort mortality in Sweden

Table 1 Sweden. Males. Age specific mortality 1781/90 - 1971/80
Index: 1751/80 = 100

Age group	1781-90	91-1800	01-10	11-20	21-30	31-40	41-50	51-60	61-70	71-80	81-90	91-1900	01-10	11-20	21-30	31-40	41-50	51-60	61-70	71/80
0-10	98	89	89	86	81	66	64	71	64	58	49	40	31	24	17	13	9	5	4	2
10-20	108	72	102	80	64	67	62	76	63	58	56	54	50	52	33	25	16	9	8	8
20-30	113	79	107	92	85	87	74	78	70	71	65	65	62	76	44	33	23	12	11	11
30-40	113	82	105	101	102	108	90	86	71	69	58	57	50	57	36	28	20	13	12	12
40-50	104	89	103	103	105	106	92	88	73	65	56	52	48	45	35	32	23	18	18	19
50-60	100	93	117	110	108	110	98	95	83	69	60	57	52	50	43	42	36	31	30	32
60-70	110	103	118	115	110	109	102	98	92	76	66	64	59	57	54	54	48	46	46	46
70-80	108	114	122	115	110	114	107	105	98	87	77	74	69	69	65	67	61	60	60	58

Fridlizius (1989)

Figure 2 Sweden. The deformation of the different cohorts 1781/90 - 1951/60. Index: The age group 0-10 years = 100. Male (——) Female (----)



Fridlizius (1989)

Cohort evidence at meso level

- Norwegian counties showed considerable variations in mortality from heart disease in the 1960s
- Mortality from heart disease in ages 40 and 69 years were associated with infant mortality around the time of birth
- Suggests that poverty in childhood is a risk factor for heart disease (Forsdahl 1977)
- Follow up for England and Wales (Barker and Osmond 1986, 1987)

Are poor living conditions in childhood and adolescence an important risk factor for arteriosclerotic heart disease? (Forsdahl 1977)

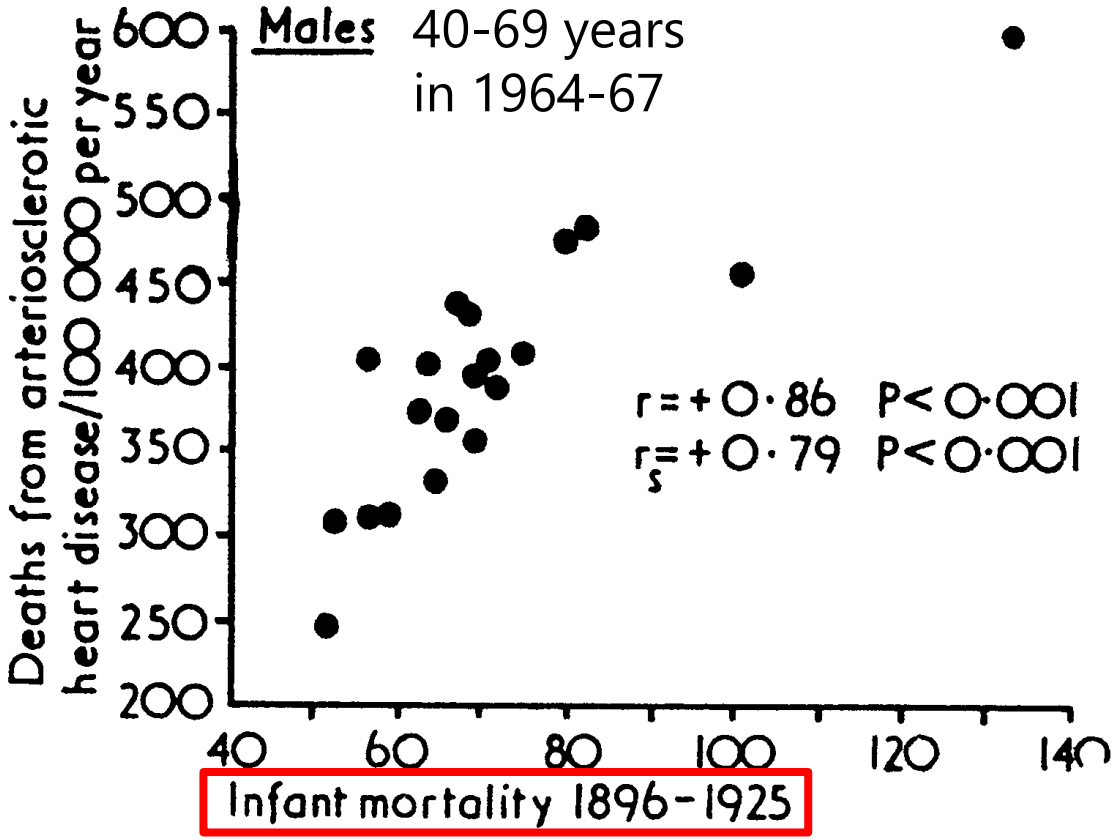


Fig. 2 *Correlation between mortality from arteriosclerotic heart disease, 1964-67, in men aged 40 to 69 years (standardised rates/100 000 population) and infant mortality rates 1896-1925.*

Data

- Macro: Synthetic birth cohorts (Kermach et al 1934; Preston and de Walle 1978; Fridlizius 1989; Finch & Crimmins 2006)
- Meso: Regional data on adult mortality (CVD, etc) linked with data for the same region at the time around birth (IMR, maternal mortality) (Forsdahl 1976; Barker and Osmond 1988)
- Micro: Individual level data in adulthood (education, income, health, mortality) linked with macro data on **a single nutritional or disease shock** at time of birth (Almond 2006; Lindeboom et al 2008; Helgertz and Bengtsson 2019)

Any methodological problems? Solutions?

BREAK

Data

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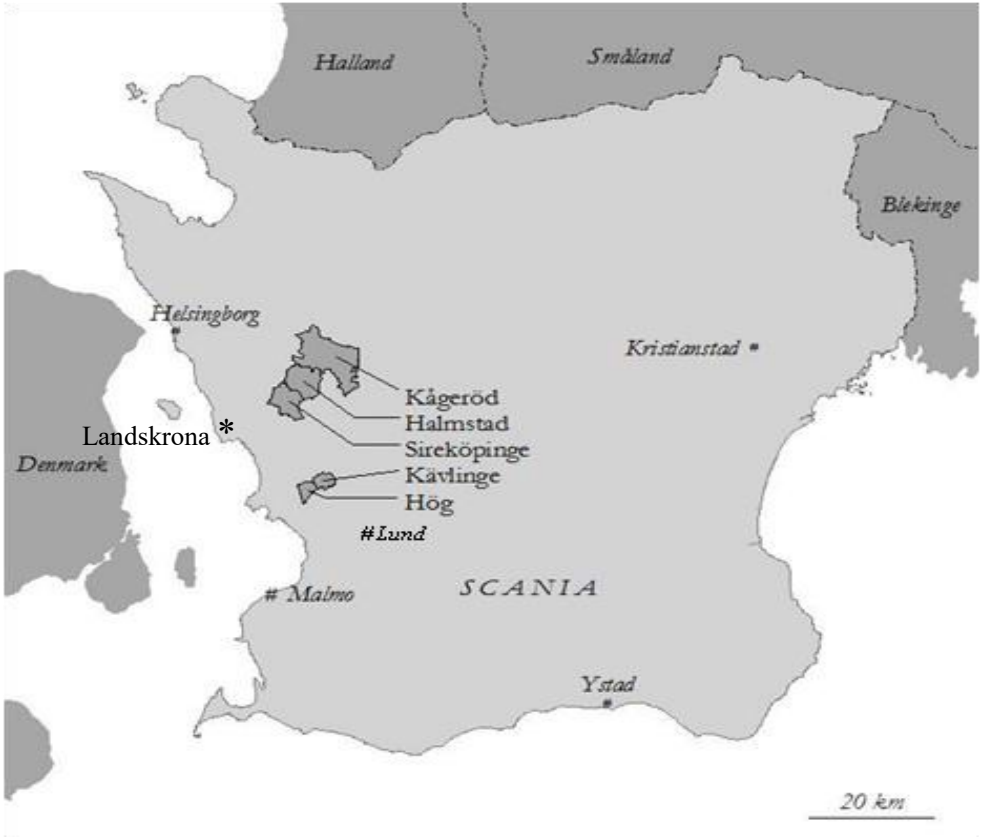
- Micro: Individual level data over the life course combined with macro data **on repeated nutritional or disease shocks** at time of birth

3. Examples using individual-level data for Sweden:

The Scanian Economic Demographic Database (SEDD)

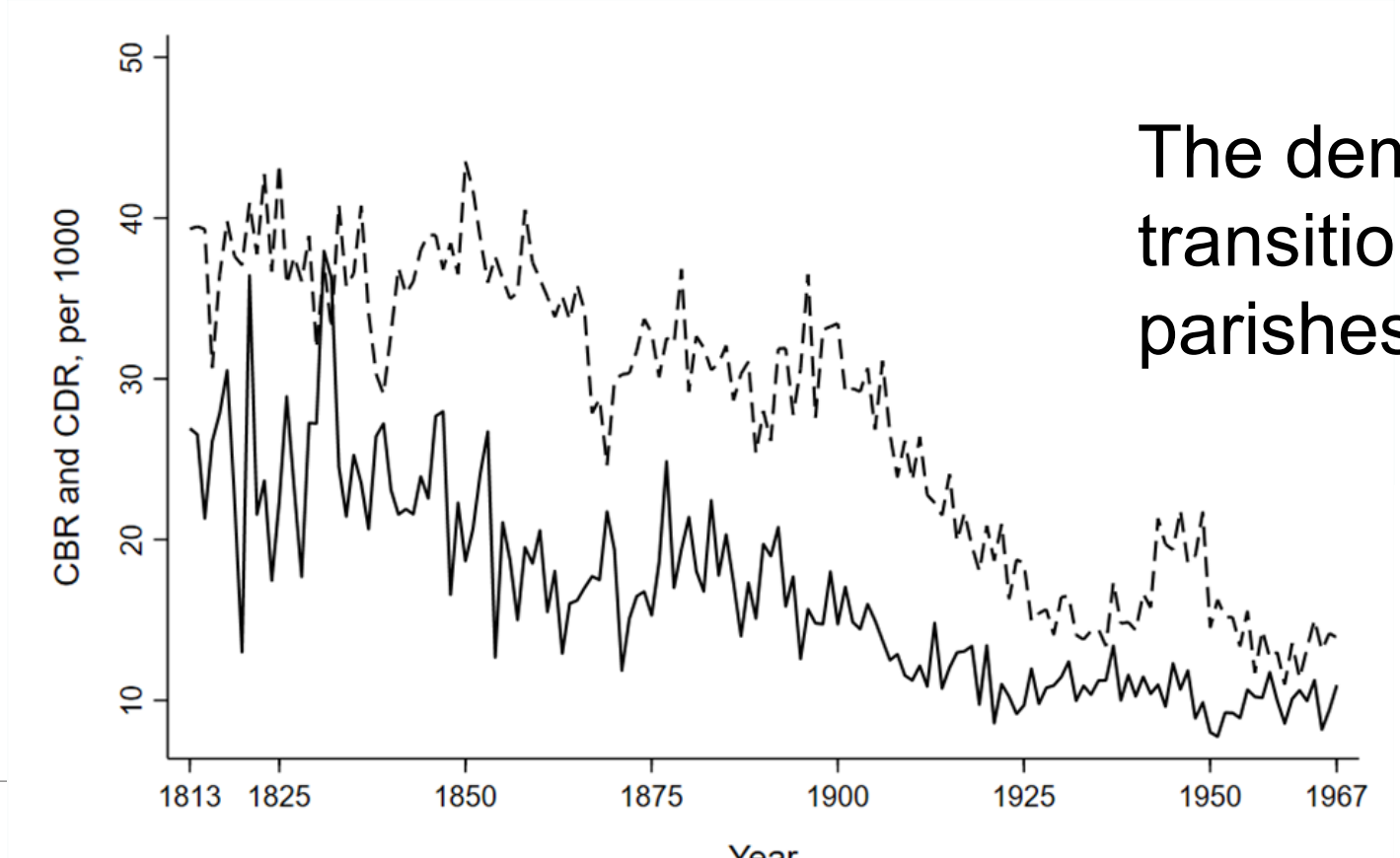
- Vital events 1646-2015 including causes of death
- Tax register 1875-2015
- Household registers and migration records 1813-
- Income records 1902-
- Midwife reports, school records, military records, hospital records, etc.
- Total population ~ 250,000

The SEDD area in the south of Sweden



Area characteristics

- Rural area (plains, woodland) and Landskrona (port town) 1905-
- Urbanization and industrial development after 1890
- Life expectancy show similar trends and is about one year above the national average



The demographic transition in the rural parishes

Early life indicators, macro and micro

	Fetal stage	Infancy
Disease	CDR 20–50-year $_{t-9\ months}$	IMR $_{t}$
Diet	Food prices $_{t-9\ months}$	Food prices $_{t}$
Diet	SES $_{t-9\ months}$	SES $_{t}$

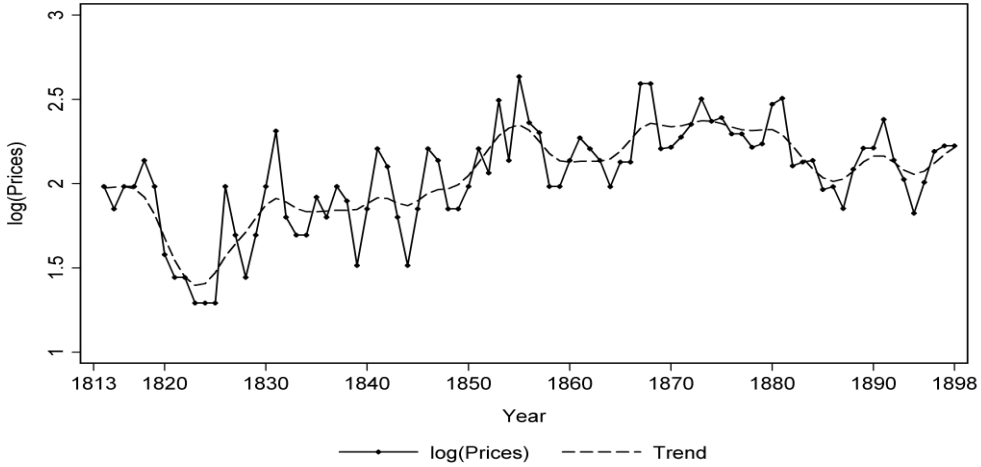
The diet indicator: Price of food

- Macro level studies for Sweden show that food prices have a strong impact on adult and but not infant mortality until the mid-nineteenth century (Bengtsson and Ohlsson 1985)

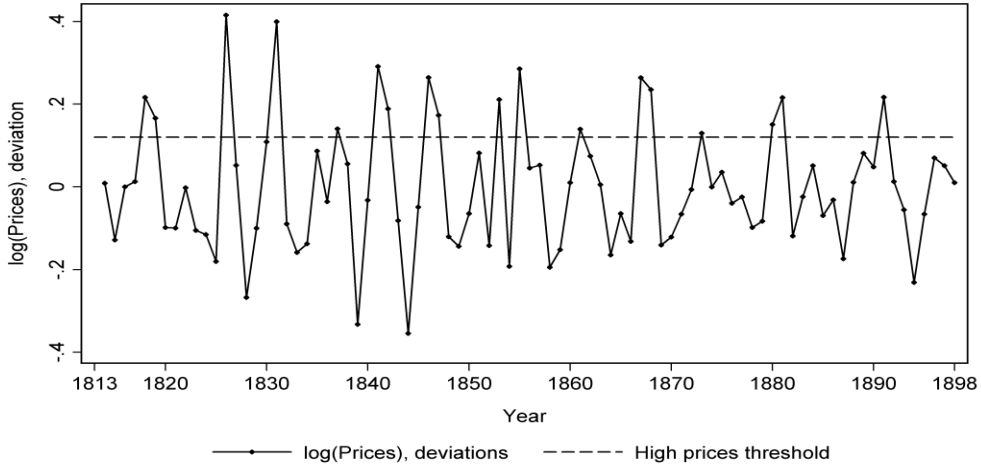
- Micro level studies show that it was families of workers but not farmers and well-off groups that suffered (Bengtsson 2004)

Macro indicators of early life conditions

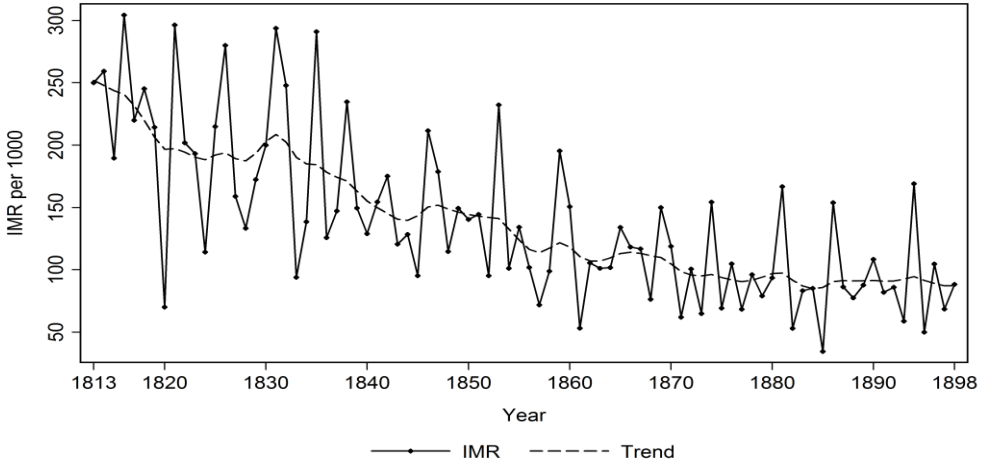
Log rye prices



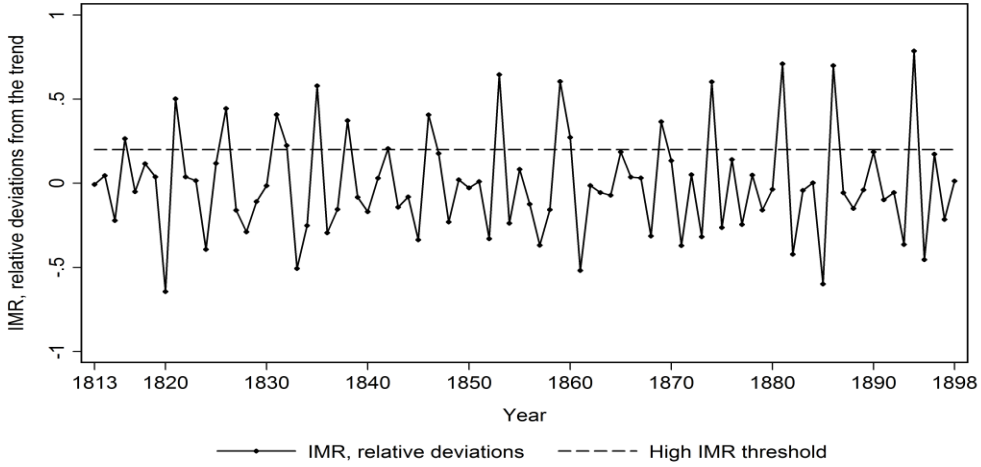
Log rye prices, deviation



IMR



IMR, deviation



Quaranta (2014)

The first step: Effects of early-life conditions on mortality in ages 55-80 years, 1766-1894

	Rel risk	P-value
Disease load		
IMR, at birth	**2.93**	**0.00**
CDR, ages 20—50,		
at conception	0.67	0.91
Diet		
Food prices, 10 % increase		
at birth	0.19	0.86
at conception	0.65	0.60

Effects of a one-unit change in IMR. Controls for birth year, etc.
No effects of SES!

Bengtsson and Lindström (2003)

Effects of IMR on causes of death in ages 55-80 years, 1760-1894

Cause of death	Re risk	P-value
Airborne infectious	**4.65**	**0.00**
Non-infectious[1]	**3.41**	**0.05**
Old age mortality	1.46	0.45

[1] Congenital heart disease, stomach, and kidney diseases

Effects of a one-unit change in IMR. Controls for birth year, SES, etc.

- Both IMR trend and IMR cycle affect mortality
- Years with smallpox and whooping cough have strong impact

Bengtsson and Lindström (2000)

The Inflammation Hypothesis

- Exposure to smallpox and whooping cough cause premature mortality in airborne infectious diseases and certain chronic disease like CVD, an indication of atherosclerosis

  (Bengtsson and Lindström 2000, 2003)
- Infections and inflammations in early life cause atherosclerosis

  (Osier 1908; Nieto 1998; Liuba 2003)
- Testing the infancy inflammation hypothesis using macro data for Sweden without separating annual variations in IMR from the trend

  (Finch and Crimmins 2004)

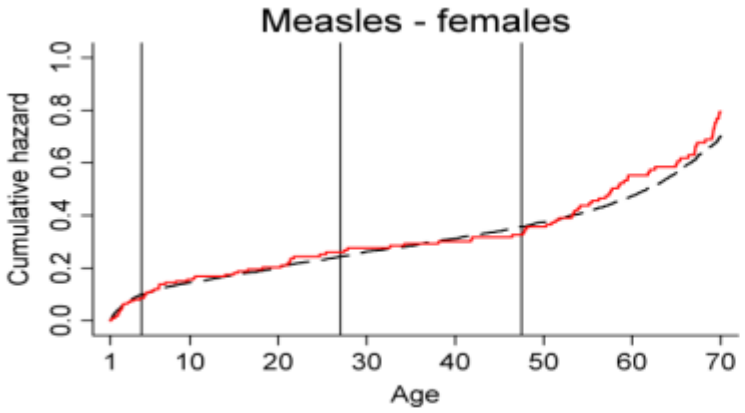
The second step: Specific early-life conditions and mortality in ages 1-80 years, 1813-1968

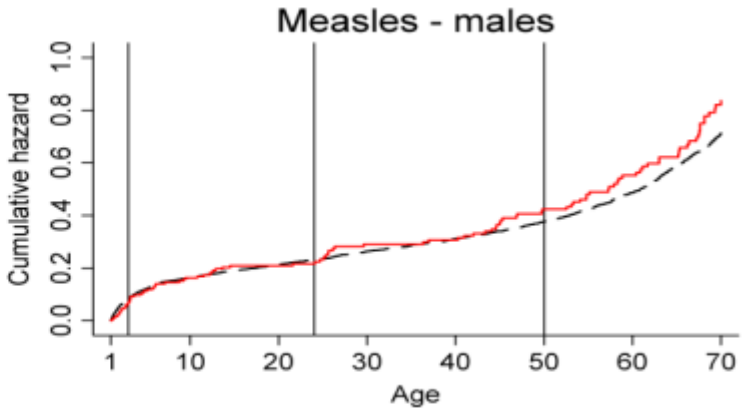
Disease load during the year of birth:

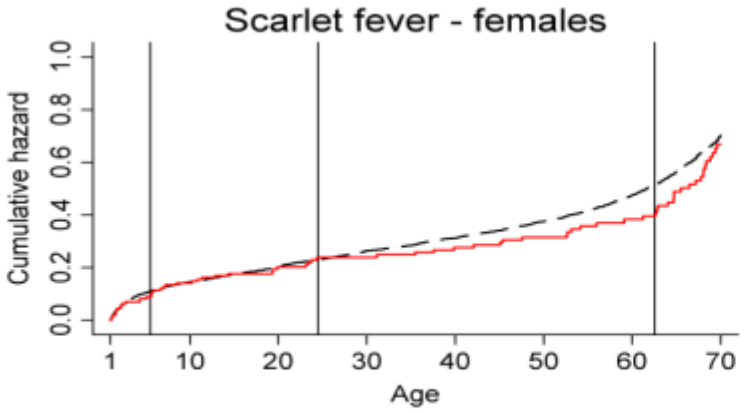
- Low-medium vs. high infant mortality rate
- Low-medium infant mortality rate vs. measles, scarlet fever or whooping cough epidemics

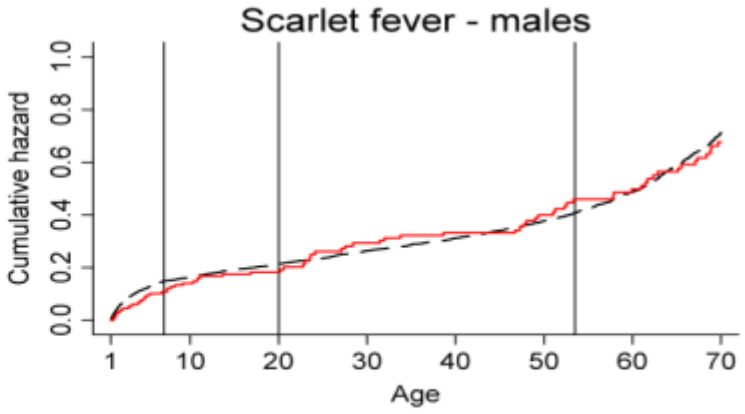
  - Measles: 1821, 1846, 1862, 1874
  - Scarlet fever: 1860, 1869, 1877
  - Whooping cough: 1816, 1826, 1831, 1835, 1853, 1859, 1894

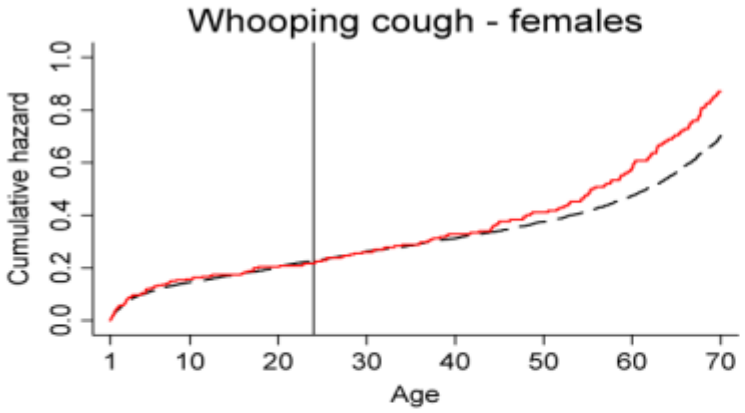
Quaranta (2013)

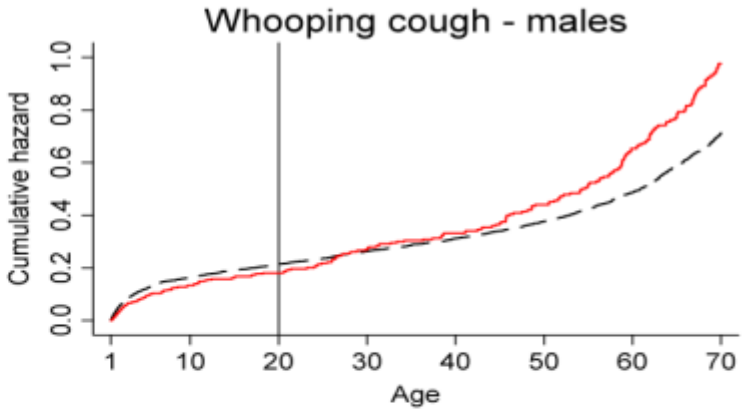




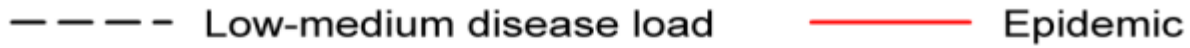






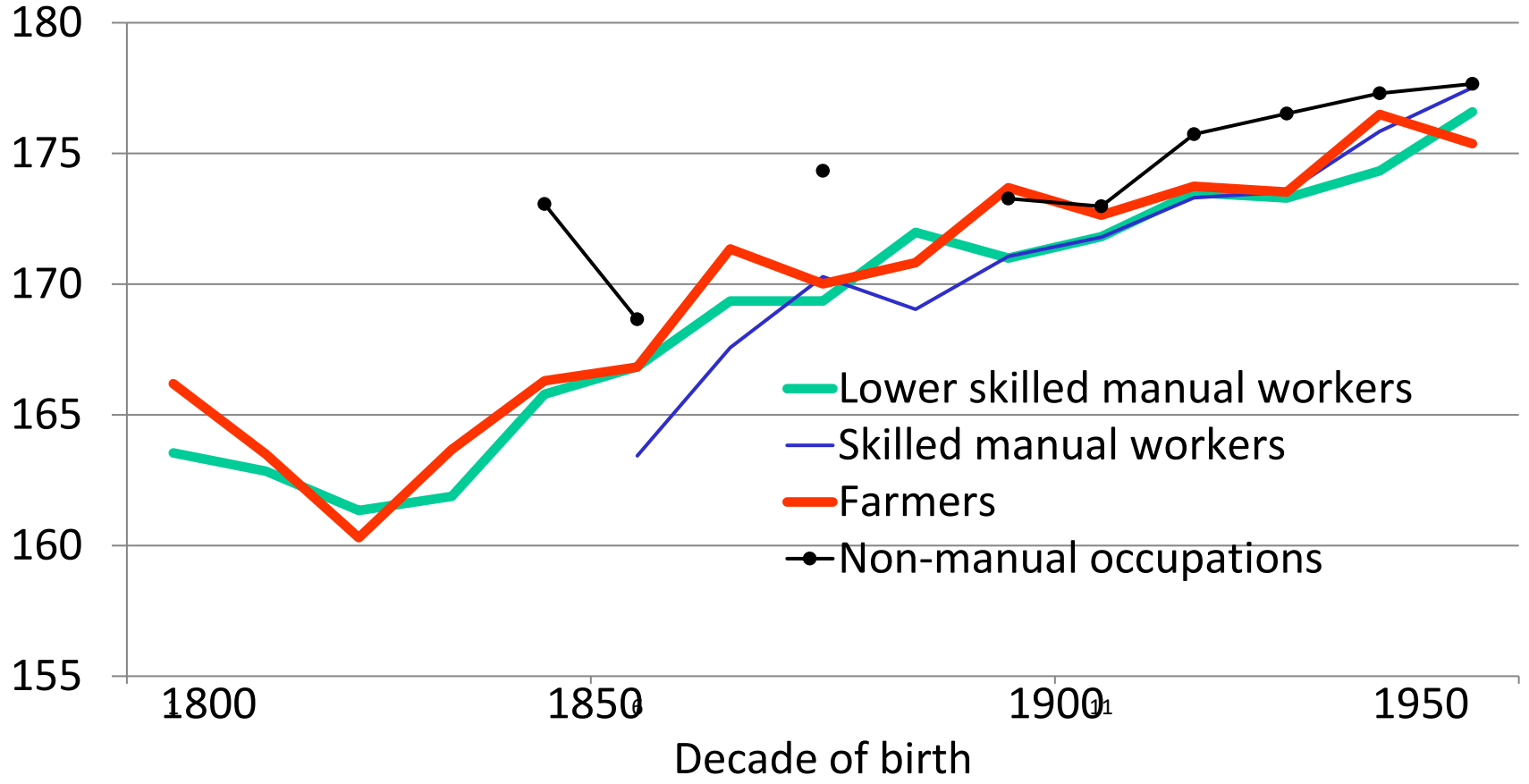


Exposure to specific epidemics in the year of birth and later life mortality, 1813-1968



Quaranta (2013)

Effects of early life conditions on heights 1797-1950?



- No effects of IMR at birth on heights despite using various definitions of IMR

(Öberg 2014)

The third step: expansion to the present

- Does disease exposure, as measured by IMR in year at birth, have an impact on adult- and old-age mortality also for individuals born in the 20th century despite the public health interventions taking place and the decline in IMR?
- Do economic factors, such as GDP/capita (van den Berg et al 2006) and SES at birth become of greater importance over time?

Bengtsson, T. & Quaranta, L. (2019) Where you live, or who you are? The role of childhood for adult health, southern Sweden 1881-2015. Working Paper.

Mortality in ages 50-69

	Women			Men		
	1881-1899	1900-1919	1920-1939	1881-1899	1900-1919	1920-1939
High IMR year at birth	0.90	0.94	**1.38****	0.98	**1.18****	0.92
High IMR year aft. birth	1.12	0.89	1.13	0.93	1.02	0.94
Low GDP year bef. birth	1.08	0.89	1.01	**1.18***	1.03	**1.21****
Low GDP year at birth	1.10	0.92	1.09	1.03	1.01	1.00
SES at birth						
Low	0.94	1.05	**1.26****	1.02	1.06	1.04
Unknown	***2.15*****	*1.15*	***1.44****	***2.20*****	*0.98*	***1.26****
Individuals	4019	5296	3751	3706	5256	3434
Deaths	724	673	452	836	1247	699

Results

- Women born 1880-1899 in years with high IMR have 16-17 % higher risks of dying in ages 70-95 than others
- Similar in ages 50-69 for women born 1920-40 and men born 1900-1919
- Exposure to diseases still important into the 20th century, but less than before
- SES is becoming increasingly important in the 20th century, so does economic cycles

The fourth step: Effects of public health interventions on mortality, 1870-1940

Public health interventions:

- Isolation hospitals in 1893 and 1895
- Improved midwifery in 1903 and 1915

Outcomes:

- All-cause mortality
- Cause-specific mortality
- Income

Method:

- Difference-in-difference

Short-term effects of public health interventions on mortality, hazard ratios

Isolation hospitals	<1 year	1-4 years	5-14 years
Non-treated (ref)	1.00	1.00	1.00
treated parish * post-introduction	0.52*	0.55	0.86

Improved midwifery	<28d	>28d	<1 year
Non-treated (ref.)	1.00	1.00	1.00
treated parish * post-increase	0.36***	0.53**	0.46***

Lazuka, Quaranta and Bengtsson (2016)

Long-term effects of improved midwifery on mortality, hazard ratios

	All-cause		Infectious	CVD	Cancer
	ages 15-39	ages 40-80	ages 15-39	ages 40-80	
Traditional (ref)	1.00	1.00	1.00	1.00	1.00
Qualified midwife	0.58*	0.94	0.57	0.65**	0.97

- Control-for-observables based on individual-level data on qualified midwifery
- Social rate of return 11:1

Lazuka (2018)

Long-term effects of vaccinations on health and socio-economic outcomes in later life

Polio

- No effects of vaccinations on education, income and health among natives experiencing the outbreak in the 1950s

  (Serratos-Sotelo, Bengtsson and Nilsson 2019)

- Similar findings for immigrants to Sweden

  (Serratos-Sotelo 2020)

Smallpox

- Hugh positive non-specific effects of vaccinations

  (Lazuka and Sandholt Jensen 2024)

Summary: Long-term effects in Scania

- No or small effects of food prices and SES on later life mortality at least up to 1970
- Disease environment in year of birth have a strong influence on mortality in later life. Years of smallpox and whooping caught particularly harmful

  (Bengtsson and Lindström 2000, 2003)
- Scarring effect of whooping cough in first year of life outpace selection at ages above 25 years

  (Quaranta 2013)
- Small effects on heights

  (Öberg 2015)
- Direct effects on adult SES

  (Bengtsson and Broström 2009)

Indirect effects: The theory of technofysio evolution

Nutritional status in early life$_g$ = f (diet, disease)

Height$_g$ and output$_g$

Length of life$_g$

Nutritional status in early life$_{g+1}$

and so on for ever

Fogel and Costa (1997)

The fifth step?

In honour of Robert Fogel

- Director for Center for Population Economics, University of Chicago
- Born: July 1, 1926, New York
- Died: June 11, 2013, Chicago
- Nobel Prize winner in 1993 (with Douglas North)
- Honorary Doctor at LU 2005