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The Effect of Nutritional Status on Historical Infectious Disease Morbidity: Evidence from the London Foundling Hospital, 1892-1919

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The Effect of Nutritional Status on Historical Infectious Disease Morbidity: Evidence from the London Foundling Hospital, 1892-1919*

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Abstract

There is a complex inter-relationship between nutrition and morbidity in human health. Many diseases reduce nutritional status, but on the other hand, having low nutritional status is also known to make individuals more susceptible to certain diseases and to more serious illness. Modern evidence on these relationships, determined after the introduction of antibiotics and vaccines, may not be applicable to historical settings before these medical technologies were available. This paper uses a historical cohort study based on records from the London Foundling Hospital to determine the causal effect of nutritional status of children, proxied by weight- and height-for-age Z-scores, on the odds of contracting five infectious diseases of childhood (measles, mumps, rubella, chicken pox and whooping cough) and on sickness duration from these diseases. I identify a causal effect by exploiting the randomisation of environmental conditions as foundling children were removed from their original homes, then fostered with families in counties nearby London and later returned to the Foundling Hospital's main site in London. I find no effect of nutritional status on the odds of contracting the five diseases, but I do find a historically important and statistically significant effect of nutritional status on sickness duration for measles and mumps. These findings have three implications. First, historical incidence of these diseases was unrelated to nutritional status, meaning that poor nutritional status during famines or during the Colombian Exchange did not affect the spread of epidemics. However, undernutrition in these events may have exacerbated measles severity. Second, improving nutritional status in the past 150 years would have reduced the severity of measles and mumps infections but not affect the decline in whooping cough mortality. Finally, selective culling effects from measles would be larger than those from whooping cough since whooping cough severity was not correlated with underlying nutritional status.

Keywords: morbidity, nutritional status, infectious diseases, health transition

JEL Codes: N01, N30

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1 Introduction

Scientists have long known that there is a complex two-way relationship between nutrition and infection in humans (Scrimshaw 2003). Infections can exacerbate nutritional status[1] by forcing the body to expend energy on fighting the disease, preventing the body from absorbing nutrients consumed and directly depleting resources in the case of parasites. These pathways have been tested with modern and historical data with economic historians showing that declines in infant mortality and its associated morbidity from diarrhoea were important causes of changes in child growth in the twentieth century (Hatton 2011, 2014; Schneider and Ogasawara 2018). However, this paper focusses on the opposite direction of the relationship: whether poor nutritional status leads individuals to be more susceptible to and/or experience more severe illness and higher case fatality rates from infectious diseases. This side of the relationship has been studied almost exclusively using modern data, and the strength of the relationship varies based on the particular disease analysed, the type of malnutrition and the context (Bellagio Conferees 1983).

Modern evidence of a link between nutrition and infectious disease has permeated historical thinking over the past six decades in critical ways. It was essential for McKeown's arguments that improving nutrition was primarily responsible for the decline of mortality in the nineteenth and twentieth centuries rather than medical innovations or public health interventions (McKeown and Record 1962; McKeown et al. 1975). It has formed an important part of the critique of the 'virgin soils' explanation of exceptionally high mortality among indigenous Americans after the Colombian Exchange. Jones (2003) argues that rather than Native Americans having inherently lower levels of immunity because of lack of historical exposure to diseases such as smallpox and measles, malnutrition exacerbated the effects of the epidemics leading to greater incidence and exceptionally high case fatality rates from these diseases. The link between nutrition and infectious disease is also important for understanding crisis mortality following famines since most deaths from famines were caused by infectious diseases rather than actual starvation (Mokyr and

[1]Nutritional status is the balance of nutrition consumed and the claims against that nutrition in fighting infection, maintaining bodily functioning and working to produce or buy the nutrition (Harris 2004).

Ó Gráda 2002). Although 'collective immunosuppression' may have been more important than 'individual immunosuppression' during famines (Mokyr and Ó Gráda 2002, p. 21), there are a number of pathways through which malnutrition caused by famines lowered individual-level immunosuppression, making people more susceptible to infections from typhus to diarrhoea to measles (Dirks 1993). Finally, the link between nutritional status and infection is fundamental to understanding how important selective mortality was in shaping the health of cohorts in the past (Bozzoli et al. 2009). If infectious diseases were more likely to kill children already suffering from malnutrition, then survival bias would lead surviving individuals to be healthier than those originally born and would mitigate the benefits of improving infectious disease mortality on cohort health.

However, all of the examples above are contested. McKeown's emphasis on nutrition in the mortality decline has been challenged on a wide number of fronts (Harris 2004; Szreter 1988). Riley (2010) disputes Jones's assertion that malnutrition increased mortality among Native Americans following the Colombian Exchange largely because there is limited evidence that smallpox responds to malnutrition, although measles was also a leading killer in the Colombian Exchange (Cliff et al. 1993, pp. 62-7). Livi-Bacci (1991) argues that malnutrition did not influence the course of mortality in premodern Europe in part because many epidemic infectious diseases are not nutrition-sensitive, and Post (1990) finds that epidemics related to famine in eighteenth-century Europe were driven more strongly by increased migration than the malnourished state of the population. Finally, Hatton (2011) finds little evidence of a selection effect of infant mortality on child heights in twentieth century Britain.

The historical relationship between nutritional status and infectious disease morbidity and mortality remains contested for four reasons. First, different diseases are more or less sensitive to the underlying nutritional state of the host. Whereas there is fairly strong evidence that measles and many respiratory diseases are sensitive to nutritional status, other infectious diseases such as plague, smallpox and typhoid are less sensitive. In fact, rough schema exist which describe the relationship between infectious diseases and nutritional status (Bellagio Conferees 1983), and there is a lot of variation across diseases

(see Table 1). Second, defining malnutrition is not an easy task. Modern studies find different effects for protein-energy malnutrition and deficiencies in certain micronutrients than for generalised undernutrition proxied via child height or weight (Solomons and Chomat 2015). There is also debate about whether only nutritional deficiency matters or whether the effect of nutritional status is continuous (Harris 2004, pp. 382-3), i.e. are there threshold effects? These subtleties require the historian to diagnose past populations with various types of malnutrition and assess the prevalence of these types of malnutrition in order to draw conclusions from the nutrition-infection interaction. While a flourishing literature has tried to capture and understand changing diets over time (Floud et al. 2011; Gazeley and Horrell 2013; Gazeley and Newell 2015; Gazeley et al. 2021), there are large margins of uncertainty in the calorie estimates (Kelly and Ó Gráda 2013), and it is still difficult to capture the effects of the changing composition of the diet and the distribution of nutrition across the population before the twentieth century (Schneider 2013a,b).

Table 1: Effect of pre-existing nutritional status on the morbidity and mortality outcomes of a number of infectious diseases

Definite	Equivocal or Variable	Minimal
Measles	Typhus	Smallpox
Whooping Cough	Diphtheria	Malaria
Tuberculosis	Staphylococcus	Plague
Most Respiratory Infections	Streptococcus	Typhoid
Most Intestinal Parasites	Influenza	Tetanus
Diarrhoeas	Syphilis	Yellow Fever
Cholera	Systemic Worm Infections	Encephalitis
Leprosy		Poliomyelitis
Herpes		

Sources: Adapted from Bellagio Conferees (1983, p. 506).

Third, despite the seeming authority with which Table 1 is presented, determining a precise causal relationship between malnutrition and infectious disease morbidity is exceedingly difficult. The data requirements are onerous since the researcher needs measures of nutritional status that pre-date sickness events. In addition, there is enormous scope for confounding bias in these estimates since malnourished children will also tend to be poorer, live in less healthy environments and face differential exposure to infectious

diseases than well-nourished children (Harris 2004). Even where researchers can control for socioeconomic status, it is highly likely that nutritional status will be correlated with investments in sanitation and hygiene and the quality of nursing care that children receive from their parents or other caretakers, and these variables are almost always unobserved (Wilson 2009, p. 26). Because most medical studies do not account for these potential sources of endogeneity, it is difficult to apply modern estimates of the relationship to the past.

Fourth, the relationship between nutritional status and infectious disease is contested because even if we take the medical research as read, it is still difficult to extrapolate from modern medical studies to the past (Watkins and Van de Walle 1983). The introduction of antibiotics and immunisations for a large number of diseases has dramatically changed who contracts infectious diseases and how severe they are. Even case fatality rates from viral diseases can fall due to antibiotics since in many viral diseases like measles and whooping cough, mortality results from secondary respiratory infections (McKeown et al. 1975). Modern understandings of hygiene and medical care have also permeated societies today in ways that were unthinkable in the past. Thus, it is not clear to what extent the relationships between nutritional status and infectious disease incidence, severity and case fatality found in modern societies would be representative of those found in the past.

This paper contributes, then, by determining the causal effect of nutritional status on morbidity from five acute infectious diseases (measles, mumps, rubella, chicken pox and whooping cough) using uniquely rich historical microdata from a London orphanage, the Foundling Hospital, from 1892 to 1915. The Foundling Hospital staff recorded the children's weight in infancy and height and weight in mid-childhood (ages 4-6), providing precise measures of the children's nutritional status. In addition, they recorded the diseases that children were treated for while they were under the Foundling Hospital's care until the age of 15, and for the life stage from age 5 to 15, sickness durations were also recorded, providing detailed medical histories for each child.Thus, I can estimate the effect of nutritional status on the probability of contracting each of the five diseases and on the sickness duration in late childhood and adolescence in a historical period pre-

dating the major technological innovations that could have altered the nutrition-infection relationship. I also contribute by taking advantage of random variation in the children's environment that can eliminate confounding bias in the relationship of interest and allow for a causal estimate for the effect of nutritional status on morbidity. The identification strategy hinges on the fact that the foundling children were removed to different locations and placed under the care of different individuals during the life stages of their upbringing. This means that the confounding factors that affected a child's nutritional status measured at the end of one life stage did not affect the child's propensity to contract an infectious disease or the severity of that disease in the next life stage. Thus, the Foundling Hospital cohort study provides both the data and research design to significantly improve our knowledge of the relationship between nutritional status and morbidity in the past.

The paper begins with a short introduction to each of the five diseases. I then discuss the data source and provide context for the main variables in the analysis: nutritional status and morbidity. Next, I present the identification strategy and statistical methods used to test the relationship between nutrition and morbidity before presenting the results. The final section concludes.

2 The Five Diseases

This paper tests whether nutritional status affected the incidence and severity of illness from five infectious diseases: measles, mumps, rubella, chicken pox and whooping cough. This section provides a concise discussion of the key features of the diseases that will matter to the analysis (see Table 2). For a more careful consideration of each of the diseases and the historical context, see Appendix C. All of the diseases are highly contagious with basic reproduction numbers (R_0) greater than five.[2] For reference, the R_0 of COVID-19 is estimated to be 2.9, making these diseases between two and six times more contagious. All of the diseases are spread by human-to-human contact via respiratory aerosols or droplets. The viral diseases provide lifelong immunity after infection, but

[2]The basic reproduction number is the number of additional people infected by one individual in a fully susceptible population.

being infected with whooping cough does not grant lifelong immunity even if subsequent whooping cough infections tend to be less severe than the first (Halperin and De Serres 2009, p. 585). Another important feature of mumps, rubella and whooping cough is that as many as a third to a half of infections may go unreported because of sub-clinical or asymptomatic cases (Halperin and De Serres 2009; Kim-Farley 1993a,b). Thus, incidence rates tend to be severely under-reported for these diseases.

Of the five diseases, measles and whooping cough were the most deadly with relatively high case fatality rates and mortality in the late nineteenth and early twentieth centuries. Both were among the leading causes of death of children under the age of five, but case fatality and mortality fell dramatically with age. In both of these diseases most deaths were caused by secondary respiratory infections, mainly pneumonia (Hardy 1993). Interestingly, whooping cough mortality began to fall much earlier in England and Wales than measles mortality (see Appendix Figures C.1 and C.2). Mortality from the other diseases was much lower but also began declining in the late nineteenth and very early twentieth century. Interestingly, these declines in mortality appear to have been driven by declines in case fatality rates rather than declines in incidence. These five diseases were highly contagious, and therefore, it is doubtful that societies could have controlled their spread in the absence of vaccination, which did not become widespread until after the Second World War. High levels of incidence are especially likely since urban and rural areas were becoming ever more integrated with improved transport infrastructure and inter-household interactions were increasing with the introduction of compulsory schooling in the late nineteenth century (Hardy 1993, p. 44).

There are various theories for the mortality declines from these diseases. Some authors have emphasised crowding and social density (Aaby et al. 1984; Cliff et al. 1998, p. 338; Wilson 1905) whereas other have emphasised improvements in nursing care (Cliff et al. 1998, pp. 328; Hardy 1993, pp. 17, 45-48). With regards to nutrition, there is a fairly well-established link between malnutrition and measles severity and case fatality (Moss and Griffin 2014). This has been challenged by Aaby in a number of papers arguing that crowding was more important since children living in close conditions would pass

Table 2: Characteristics of the five infectious diseases studied

Disease	Agent	R_0	Transmission	Lifelong Immunity?	Asympt. or Sub-clinical Cases	Historical Case Fatality Rate	Date of Decline in E&W	Mortality Rate Pre-Decline in E&W (per 1,000,000)		
								0-4	5-9	10-14
Measles	Viral	12-18	Aerosol	Yes	No	3-10%	1915	2,888	235	24
Mumps	Viral	10-12	Resp. droplets	Yes	Yes	Very low	1875	20	3	0
Rubella	Viral	6-7	Resp. droplets	Yes	Yes	Very low	1900	13	2	2
Chicken Pox	Viral	10-12	Aerosol	Yes	No	Very low	1900	30	2	0
Whooping Cough	Bacterial	5.5	Resp. droplets	No	Yes	3.6-10.5%	1892	3,609	150	8

Notes: R_0 is the basic reproduction number, the number of people infected by a single case in a fully susceptible population.

Sources: For colums 2-6: Gnann Jr (2014); Halperin and De Serres (2009); Kim-Farley (1993a,b); Moss and Griffin (2014); Orenstein and Reef (2014); Rubin (2014). For column 7, historical case fatality rates: Hardy (1993, pp. 23, 39); Kampmeier (1993); Kim-Farley (1993a,b). For columns 8-11, see sources for Appendix Figures C.1-C.5.

along a higher viral load (Aaby et al. 1983, 1984).[3] The causal link between nutrition and whooping cough morbidity is not as clear, but a number of authors have argued that improving nutrition was in part responsible for the decline of whooping cough mortality (Cliff et al. 1998, p. 338; Halperin and De Serres 2009; Hardy 1993, p. 21; McKeown et al. 1975). There are no clear links in the scientific literature between nutritional status and mumps, rubella or chicken pox (Gnann Jr 2014; Orenstein and Reef 2014; Rubin 2014).[4] I include all five diseases in the analysis because child infectious disease morbidity is relatively understudied, and it is interesting to know whether nutritional status influenced these diseases in history despite the lack of clear modern evidence.

3 Data

Historical records that contain information about nutritional status and morbidity are extremely rare. Most morbidity studies are based on the records of Friendly Societies for which information about nutritional status is not available (Harris et al. 2012; Riley 1997). Likewise, although military records provide rich information on nutritional status, they rarely contain morbidity information and where they do it is not likely to be representative since war injuries and illnesses are different from those that occur in peacetime. One interesting exception is Ogasawara et al. (2020), which correlates household-level nutrition and morbidity among low-income households in Tokyo in 1930. They find no relationship between nutrition and all-cause morbidity but do find that income is correlated with morbidity. However, their sample size is small, and they cannot measure individual-level nutritional status. Thus, this paper contributes a new, individual-level cohort dataset based on the records of the London Foundling Hospital with extremely rich information about children's nutritional status and morbidity outcomes.

[3] See Appendix C for more detail.

[4] Jones (2003, p. 735) does mention that case fatality rates from chicken pox were higher among the malnourished, but this is no longer mentioned in the latest edition of *Viral Infections of Humans: Epidemiology and Control* (Gnann Jr 2014).

3.1 The Foundling Hospital

The London Foundling Hospital was an orphanage founded in the mid eighteenth century to care for unwanted children. In its earliest days, the hospital acted similarly to other continental Foundling Hospitals in taking all children that were left at their door, and there were extremely high mortality rates among the children (Levene 2005). However, by the end of the eighteenth century, the hospital became more selective, accepting a limited number of children who fit their admission requirements and caring for these children until they were 15 or 16. After this point, the Foundling Hospital accepted first-born illegitimate children of 'respectable' mothers whose fathers had abandoned them. The hospital accepted children from a wide range of social classes and from across London. Health was not a basis for admitting or rejecting children, though the admission of children suffering from an infectious disease at the time of their acceptance was delayed until after they had recovered (Cropley Swift 1911, p. 4). Arthi and Schneider (2021) show that the birth weight of foundling children linked to the Queen Charlotte Hospital were not statistically different from the birth weights of a random sample of other children born in the Queen Charlotte Hospital, again confirming that there was not negative selection on health. They also show by comparing accepted and rejected applications that there was very little selection on characteristics beyond the Foundling Hospital's stated policies.

Figure 1 presents the four events (birth, admission, re-admission and discharge) and three life stages of children while under the hospital's care (see Appendix Table A.1 for the full information available and sources for each event and life stage). Life stage one began with the child's birth and ended when they were admitted to the Foundling Hospital. Because mothers could not petition to give up their children to the hospital until after the child was born, all children spent some time with their mothers after birth. Admission age varied between one and sixteen months with a median admission age of 3 months. All children were inspected at admission by the medical officer, who recorded information about their health both at admission and during life stage one.

Life stage two refers to the period after admission when the children were fostered with respectable married women living outside of London in the counties of Kent, Surrey and

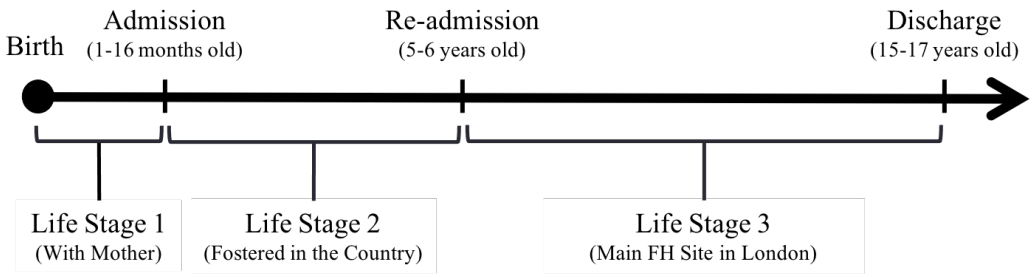


Figure 1: Life Stages of a Foundling Child

Essex: one foundling child per foster family. This fostering process appears to have been exogenous to the children's underlying health, i.e. unhealthy children were not granted 'better' foster parents, for several reasons. First, the hospital admitted approximately four children per month, and the children appeared to be fostered almost immediately after admission. There is no evidence that the infants were held on the Foundling Hospital main site for any length of time, nor is there evidence that admissions were delayed in order to place children selectively with different foster mothers.[5] Thus, it appears that children were placed wherever a foster mother was available. Second, the foster mothers were selected by country medical officers who did not participate in the admission procedure and therefore did not have information about the children's underlying health. Finally, the Foundling Hospital made substantial effort to ensure that all children were treated well. The foster mothers were compensated for raising the children but did not act as wet nurses: the children were weaned upon entry to the hospital (Cropley Swift 1911, pp. 1-4). The hospital employed several country medical officers (one for each county) who lived nearby the children and cared for the children when they were sick. The country medical officers kept medical records for each child and reported on the children's health when they returned from the countryside at the end of life stage two at age five or six.[6]

[5]The processing time between when the hospital first received the mother's petition to give up her child and admission was unrelated to breastfeeding status, admission WAZ or any other observable characteristics (not reported).

[6]There is no commentary in the Foundling Hospital records about why the children returned from the countryside at different ages. The age at return increased across the period, and boys tended to return at earlier ages than girls. There is little evidence that health was a factor in when children returned. Admission weight-for-age was not correlated with the age of return even when controlling for a number of confounding factors. Children who experienced measles in the countryside returned 1.4 months later than those who did not, but the direction of causation is not clear here since children who remained in the countryside for longer also had more time to contract measles. In any case, it is not clear that one month longer in the countryside would have mattered for their health.

Therefore, it seems reasonable to assume that the children were randomised into different foster families.

Upon re-admission to the hospital's main site in the Bloomsbury neighbourhood of Central London, the children's health was again inspected and information about their health history in life stage two was entered into the medical record. Life stage three occurred at the main London site, which reflected the typical institutional living arrangements of the time. The main building was split into two wings, one for each gender, and each wing had four dormitories of 40-50 beds. The children ate in a common area, though there were separate dietaries for children above and below the age of 8, which suggests that there was some segregation by age. There was a separate infirmary building on the main site from 1894, which had ample accommodation to care for the children. The infirmary was staffed by a team of nurses overseen by the medical officer, William J. Cropley Swift, who lived within half a mile of the Foundling Hospital and visited each morning to check-in on the patients (Cropley Swift 1911). At the end of life stage three around age 15 or 16, the children were discharged from the Foundling Hospital with boys often starting an apprenticeship and girls working as domestic servants. Again, information about their health was recorded at this point.

3.2 Key Variables

The main independent variables of interest in this analysis are the anthropometric measurements of the foundling children, which serve as a proxy for their nutritional status. The Foundling Hospital recorded the weights of children upon admission in infancy, their heights and weights at re-admission at ages 4-6 and their heights and weights again at discharge from the hospital. In addition, I was able to link a sub-sample of foundling children (n = 160) to their birth records in the Queen Charlotte Hospital, providing measures of their birth weight and length as well (see Arthi and Schneider (2021) for further explanation). To be able to compare these anthropometric measures across ages and sexes, I convert them all to Z-scores relative to the WHO 2006 child growth standard for children under age 5 and 2007 growth reference for children over age 5. Because the

main focus is on the effect of nutritional status on subsequent infectious disease morbidity, I focus on anthropometric measures at birth, admission and re-admission. Figure 2 presents the distributions of weight-for-age Z-scores (WAZ) for these age groups. Among the sub-sample of children observed at birth, birth weights were relatively similar to the birth weights of modern children with a mean WAZ score of -0.24 (Schneider 2017). However, the foundling children experienced a sharp decline in their WAZ scores during infancy with a mean WAZ of -2.06 by the time they were admitted to the Foundling Hospital. The dispersion of WAZ also increased by the time of admission with some children experiencing extremely low weight-for-age. Despite this poor start in life, the children experienced catch-up growth between admission and re-admission with their mean WAZ increasing to -1.00 at re-admission. Their height-for-age Z-scores (HAZ), first observed at re-admission, suggested greater nutritional deprivation with a mean HAZ score of -1.59 and 32.8% of children stunted with a HAZ score less than -2 (see Appendix Figure B.1).

In addition to the information collected on the children's anthropometric measures, the Foundling Hospital medical staff also carefully tracked the children's morbidity over their life course. Some diseases were listed as part of the medical inspection at admission. It is likely that these refer to both diseases that the child experienced before admission, especially infectious diseases, and diseases from which they were currently suffering at admission.[7] For life stage two, the diseases that the children were treated for in the countryside are available. Thus, I know which diseases the children contracted and can compute case fatality rates, but I do not know when the children suffered from the disease or whether their illness was severe. There was variation in the number and variety of diseases reported by the country medical officers over time, but the medical officers seemed particularly keen to report when the children had suffered from important infectious diseases, suggesting that reporting is fairly reliable at least for the diseases studied here. Since the children were fostered in different households in their rural communities, the morbidity experience of children in the countryside should be fairly representative of children in these areas at the time.

[7]This possibly adds some error to our ability to control for earlier infections from diseases that grant life-long acquired immunity since they were unlikely to have been diagnosed by a doctor.

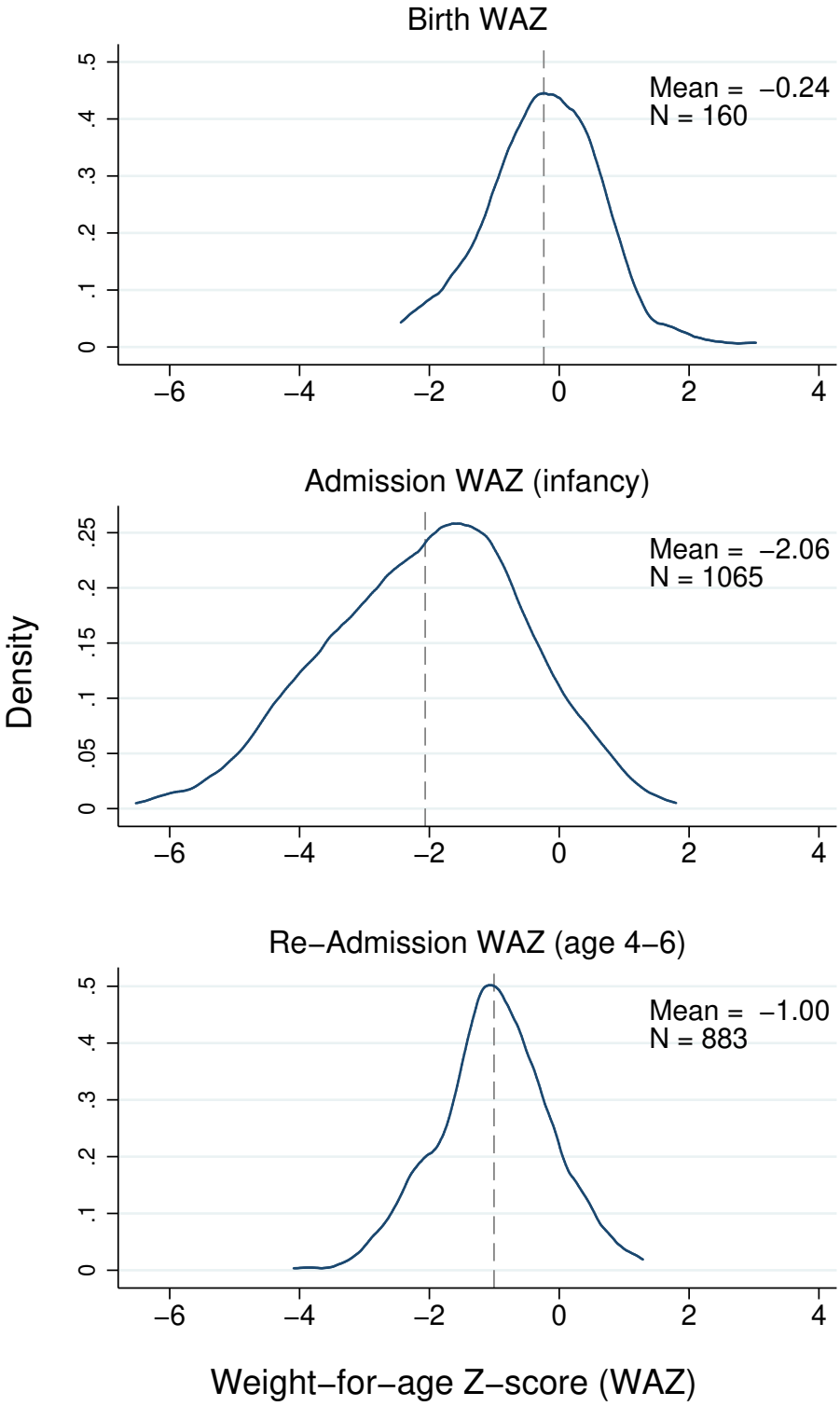


Figure 2: Distribution of weight-for-age Z-scores (WAZ) at different life stages

Notes: The vertical dashed line marks the mean.

Sources: Foundling Hospital Dataset - see Appendix A for precise sources.

Once the children returned from the countryside, information about their morbidity experiences is much more precise and comprehensive. The Foundling Hospital medical officer kept a weekly record of children being treated in the infirmary, which runs continuously for a very large number of years.[8] In this paper, I focus on the period 27 March 1897 to 5 October 1915: the start date corresponds to when the first child whose information is available in the medical record returned from the countryside and the end was imposed by a break in the surviving records. Still, this provides 17.5 years of extremely detailed information. The infirmary records state the date that a child was admitted and diseases they were being treated for upon admission. If their condition changed over time, new conditions or diseases were added to the book, showing the progress of the child's recovery. Finally, the discharge date from the infirmary was also included, providing sickness duration for each sickness event. The infirmary records include 6,409 entries to the infirmary with 695 different descriptions of diseases which have been classified into 235 ICD-10 codes. These sickness events are mapped onto a lexis diagram with the epidemics highlighted in Appendix Figure B.2. However, the focus in this paper is on the five infectious diseases that were prevalent among the foundling children in either the countryside or the London site.[9]

Table 3 presents some basic information about the incidence of these diseases. For each disease, I provide statistics for each life stage of a foundling child. The table shows the number of individuals at risk of infection at the start of each life stage, the number of cases that arose during the life stage and the probability of infection (cases divided by individuals at risk). For diseases that provided lifelong acquired immunity, I exclude those who contracted the disease in an earlier life stage from the population at risk in subsequent life stages. I also limit the calculations for life stage three and from birth to discharge to the sub-sample of children who reached discharge before the infirmary records ended so that their entire morbidity history could be observed. The incidence

[8]This record included all in-patients but excluded out-patient treatment.

[9]I exclude scarlet fever and diphtheria because these diseases were not common in life stage two affecting 3.1% and 0.5% of children respectively. In life stage three, children were removed from the Foundling Hospital and sent to the London Fever Hospital when contracting scarlet fever or diphtheria, which makes it difficult to track the children's illness in the infirmary records.

rate is the number of cases in each life stage per thousand child years lived, giving a sense of morbidity at different ages. Measles and whooping cough had greater incidence among younger children in life stage two, whereas mumps, rubella and chicken pox were more common among older children in life stage three. These diseases affected a large share of children by discharge: measles (83%), chicken pox (53%), rubella (51%), whooping cough (49%) and mumps (35%). Considering that rubella, whooping cough and mumps have many asymptomatic or sub-clinical cases, the actual infection rates from these diseases were likely much higher.

Table 3: Descriptive statistics of cases and incidence for the five main diseases

Period	Individuals at Risk	Cases	Probability of Infection (%)	Incidence (Cases per 1000 Child Years)
Measles:				
LS1: Birth to Admission	1066	14	1.3	36.0
LS2: Admission to Re-Admission	886	388	43.8	78.4
LS3: Re-Admission to Discharge	205	149	72.7	44.6
All: Birth to Discharge	342	285	83.3	54.8
Mumps:				
LS1: Birth to Admission	1066	1	0.1	2.6
LS2: Admission to Re-Admission	889	22	2.5	4.4
LS3: Re-Admission to Discharge	335	113	33.7	33.8
All: Birth to Discharge	342	119	34.8	22.9
Rubella:				
LS1: Birth to Admission	1067	0	0.0	0.0
LS2: Admission to Re-Admission	890	5	0.6	1.0
LS3: Re-Admission to Discharge	341	174	51.0	52.1
All: Birth to Discharge	342	173	50.6	33.3
Chicken Pox:				
LS1: Birth to Admission	1066	2	0.2	5.1
LS2: Admission to Re-Admission	889	163	18.3	32.9
LS3: Re-Admission to Discharge	292	130	44.5	38.9
All: Birth to Discharge	342	180	52.6	34.6
Whooping Cough:				
LS1: Birth to Admission	1066	2	0.2	5.1
LS2: Admission to Re-Admission	895	295	33.0	59.6
LS3: Re-Admission to Discharge	342	52	15.2	15.6
All: Birth to Discharge	342	168	49.1	32.3

Notes: Individuals at risk excludes individuals who contracted one of the diseases that grants lifelong immunity in an earlier life stage. For the life stage three and all categories, I restricted the sample to individuals whose complete morbidity histories to discharge were observed.

Sources: Foundling Hospital Dataset - see Appendix A for precise sources.

As infectious diseases, the five diseases did not regularly appear in the Foundling Hospital main site. Instead, they attacked the population in epidemics, infecting a large

number of children in a short period of time. Figure 3 shows that epidemics of measles, mumps, rubella and whooping cough were infrequent with two to five epidemics of each disease occurring during the 17.5-year study period. Appendix Table B.1 shows the probability of infection for each of the epidemics. Chicken pox occurred in fourteen epidemics across the period and was therefore more commonly present in the Foundling Hospital. Finally, Figure 4 displays the distributions of treatment time for each of the five diseases, combining all of the epidemics together. There were differences in median sickness duration between the five diseases with mumps and rubella involving shorter stays than measles, chicken pox or whooping cough. Interestingly, there was also considerable variation in sickness duration across children for each disease. There is relatively little evidence that children were treated for a standard length of time in the infirmary and then discharged. Chicken pox and whooping cough had especially large variation in sickness duration.

4 Methods

Having discussed the data and institutional setting above, I now present the identification strategy and methods. The Foundling Hospital provides a unique opportunity to understand the causal relationship between nutrition and infectious disease morbidity in history. To illustrate this, I present three directed acyclical graphs (DAGs), which can be used to illustrate the identification strategy used in the paper. A DAG helps to establish a causal effect between an exposure variable (nutritional status) and outcome variable (morbidity). Arrows reflect the direction of causation between variables. One can identify the causal effect of the exposure on the outcome by controlling for all joint causes (confounders) of the exposure and outcome, thus eliminating endogeneity in the form of omitted variable bias.[10] The figures use measles as an example, but the DAGs for the other infectious diseases studied would be very similar.

In a typical empirical setting, say a cohort study, it would be difficult to identify

[10]For an introduction to DAGs and their use in economic history and historical demography research, see Cunningham (2021) and Schneider (2020).

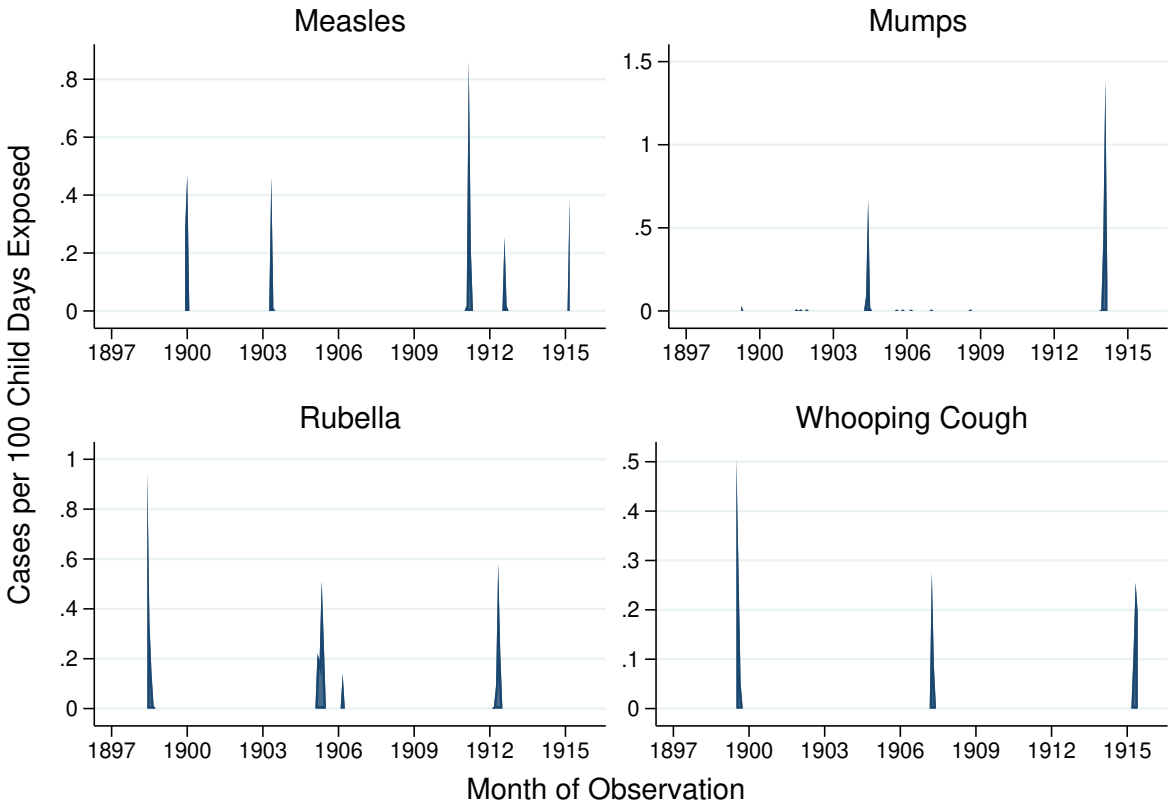


Figure 3: Monthly Incidence of Epidemic Diseases in the Foundling Hospital 1897-1915

Sources: Foundling Hospital Dataset - see Appendix A for precise sources.

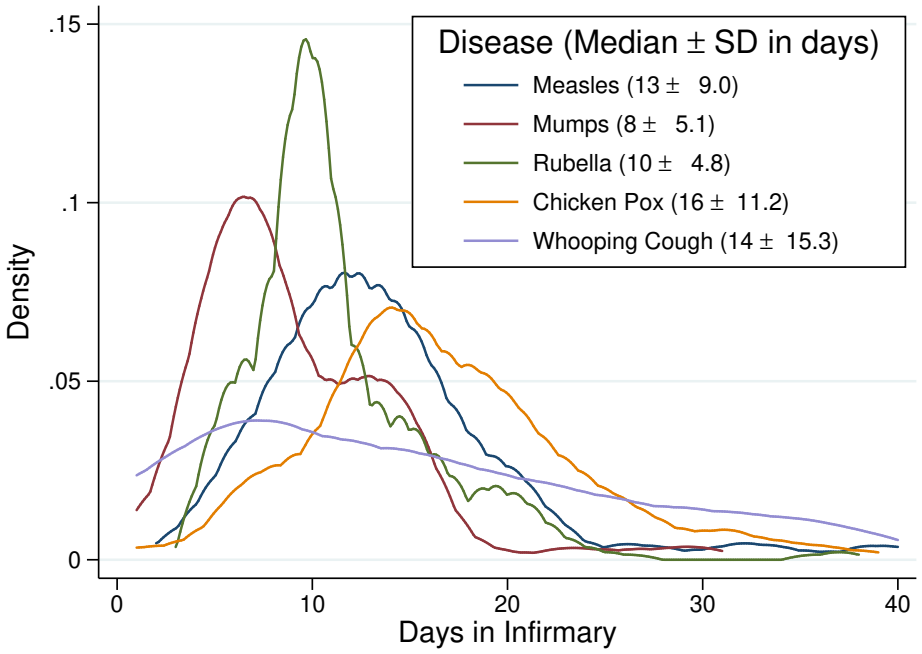


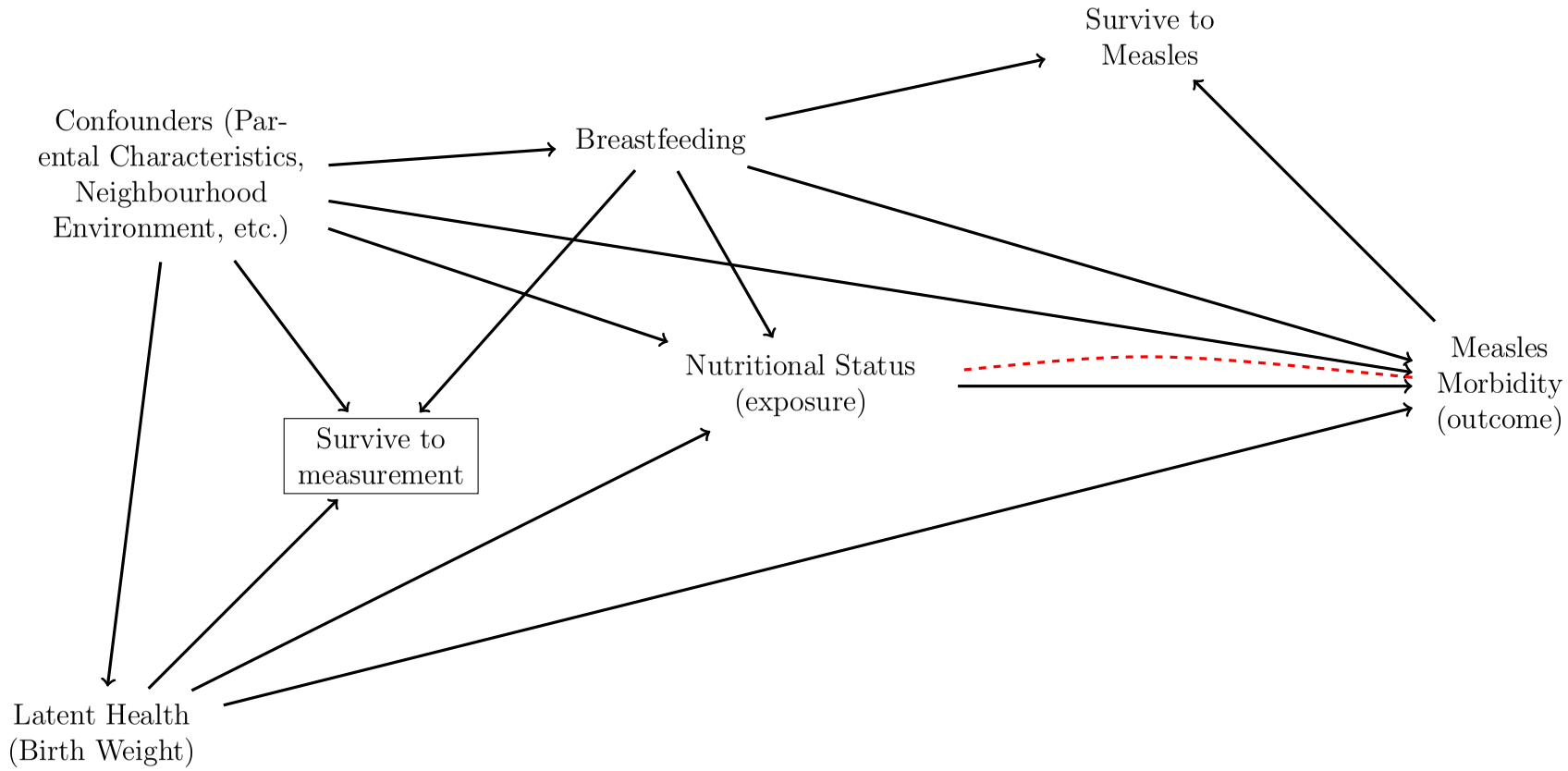
Figure 4: Sickness Duration for Five Infectious Diseases in the Foundling Hospital

Notes: Sickness duration is truncated at 40 days so that the differences across diseases are clearer, but the full range of durations are employed in the rest of the paper.

Sources: Foundling Hospital Dataset - see Appendix A for precise sources.

the effect of nutritional status on infectious disease morbidity because there would likely be large numbers of omitted confounder variables like parental socio-economic status, housing quality, crowding, infant feeding practices, investments in hygiene and sanitation, nursing care, etc. that would affect both nutritional status (the exposure variable of interest) and measles morbidity (the outcome) (see Figure 5). The omitted variables introduce confounding bias in the estimated effect of nutritional status on measles morbidity, represented by the red-dashed line. The only way to eliminate this bias is to control for all of the potential confounding factors, which is often impossible in a non-experimental setting. However, because children in the Foundling Hospital were taken from their original environments and fostered with new families in the countryside, many of the problematic confounding variables in life stage one, like the ones raised above, were unrelated to the children's exposure to infectious disease in life stage two (Figure 6). Likewise, the health conditions in life stage two that might affect a child's propensity to contract an infectious disease were unrelated to the child's nutritional status at admission (the end

Figure 5: DAG representing the relationship between nutritional status and morbidity in a typical modern study



Notes: Names in each DAG represent variables and the arrows show the direction of causation between the variables in the DAG. The exposure is the main independent variable (treatment variable) of interest, and the outcome is the dependent variable. Variables in a box have been controlled for or conditioned on in the DAG. This could mean that they are included as a control in a regression or that they are conditioned on by design since the data is only collected for a subset of the variable or sample selection is correlated with the variable. The red, dashed line shows the confounding bias that is induced by not controlling for all confounders.

of life stage one) since the children were more or less randomised across foster families and were given the same access to medical care and stipend for their room and board. The children's health conditions were randomised yet again when they returned from the countryside to the Foundling Hospital's main site in London around the age of five or six, ensuring that the conditions during life stage two in the countryside would not influence infectious disease morbidity in life stage three and health conditions in life stage three would be unrelated to the children's anthropometric measurements upon re-admission to the Foundling Hospital (Figure 7). The two changes of environment for the children, therefore, provide an excellent opportunity to test the influence of nutritional status at the outset of a life stage on the subsequent risk of contracting infectious diseases and on the severity of each infection.

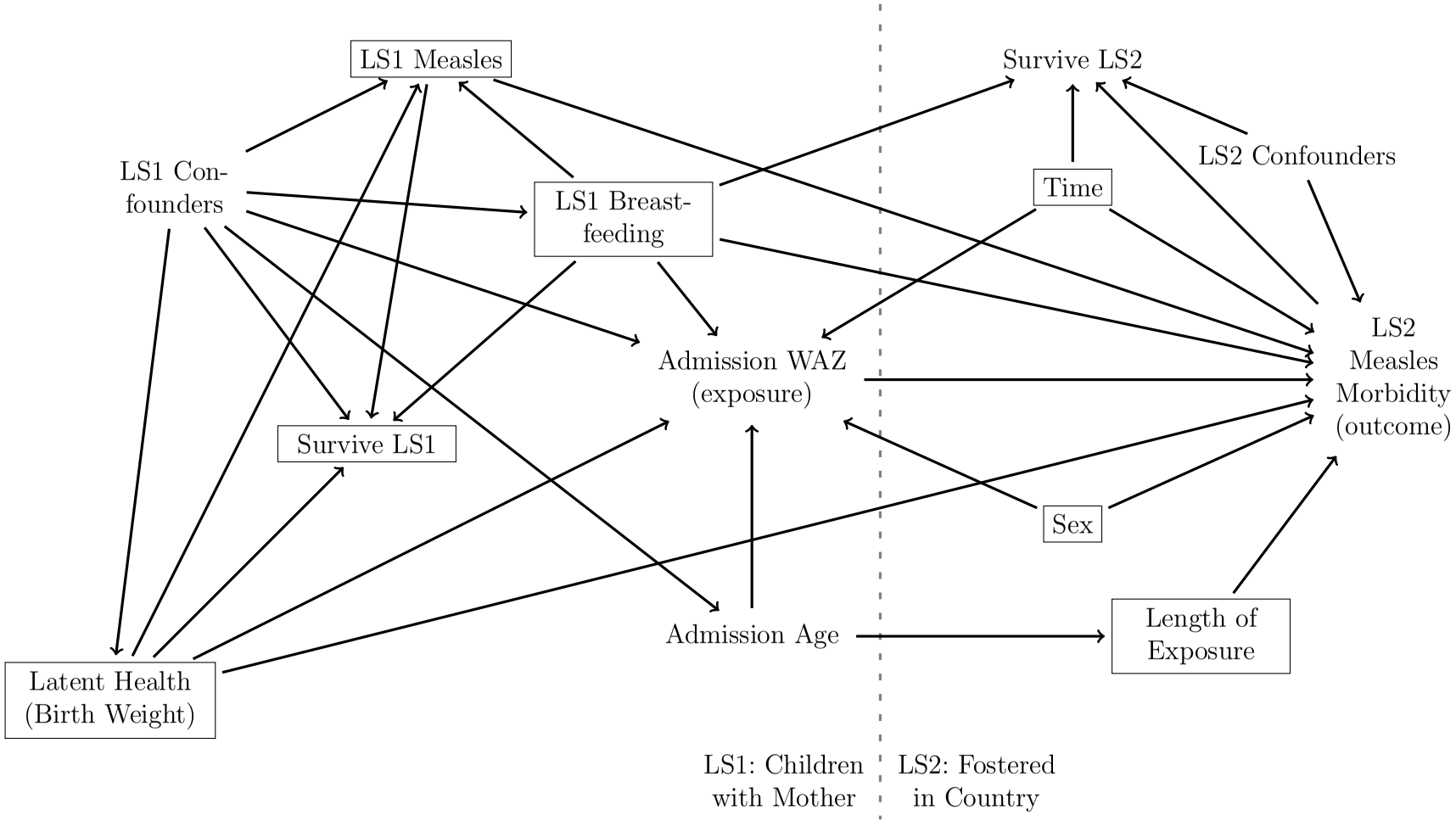
4.1 Identification and Empirical Strategy for Life Stage Two Outcomes

Figure 6 presents a DAG identifying the relationship between WAZ at admission in infancy on the propensity for individuals to contract measles (as an example) while fostered in the countryside in life stage two. This DAG can be represented by the following empirical equation estimated by logistic regression:

$$Dis_i = \alpha + \beta_1 N_i^{ad} + \beta_2 N_i^{birth} + \beta_3 B_i + \beta_4 Exp_i + \beta_5 year + \beta_6 \text{sex}_i + \epsilon_i \qquad (1)$$

where Dis_i is a binary outcome variable equal to zero if child i was at risk of contracting a disease in life stage two and one if child i contracted the disease. For life stage two, I focus on three infectious diseases which had high incidence rates among the children (see Table 3): measles (78.4 cases per 1,000 child year), whooping cough (59.6) and chicken pox (32.9). The other diseases that I focus on in life stage three were relatively rare among the children at this life stage and therefore it makes little sense to try to predict incidence for these diseases: mumps (4.4) and rubella (1.0). N_i^{ad} is child i's WAZ at admission to the Foundling Hospital in infancy and is the main independent variable of interest.

Figure 6: DAG representing the relationship between nutritional status and morbidity in the Foundling Hospital in Life Stage Two



Notes: See Figure 5.

Since the foundling children were removed from their initial environment in London, there is limited possibility for confounding bias. Life stage one variables that could potentially lead to confounding bias are variables that influence infectious disease morbidity in life stage two directly. Breastfeeding status during life stage one may be a confounder since breastfeeding has been found to influence susceptibility to measles long after breastfeeding ceases (Silfverdal et al. 2009). Therefore, B_i is a set of dummy variables controlling for infant feeding practices before admission to the Foundling Hospital. The three categories are children who were exclusively breastfed, breastfed with other supplemental foods or never breastfed. There was no preferential treatment for mothers who claimed to have breastfed their children, so we do not believe there will be bias in the mother's reporting of the child's breastfeeding status: see Arthi and Schneider (2021) for further detail. A second potential life stage one confounder is latent health at birth assuming that it may have lasting effects on children's nutritional status and susceptibility to disease. To control for this, in some specifications, I also include N_i^{birth}, the birth weight (kg) of children born in the Queen Charlotte Hospital, but this is only necessary if we believe that there is a direct causal relationship between latent health and subsequent infection. Finally, since children who contracted measles in life stage one are no longer at risk in life stage two, I implicitly condition on the variable LS1 Measles in the DAG by excluding children who had previously contracted diseases that granted lifelong immunity.

In addition to these potential life stage one confounders, there are three confounders in life stage two that could affect nutritional status (directly or indirectly) and morbidity outcomes. The first, Exp_i, is the total length of time in life stage two for each child since this would affect their chances of contracting a disease and admission age was strongly related to admission WAZ.[11] Second, I include the birth year trend *year* to capture potential confounding from trends over time in admission WAZ and measles case fatality rates or incidence. Finally, sex_i captures any differences in WAZ and morbidity outcomes that vary by sex. These regressions, then, by eliminating confounding bias, provide a

[11]The results were qualitatively similar when including admission age in months dummies into Equation 1 (not reported). Thus, admission age is not an important confounder, likely because although it influences WAZ at admission, it does not affect the propensity of children to contract one of the diseases (the outcome).

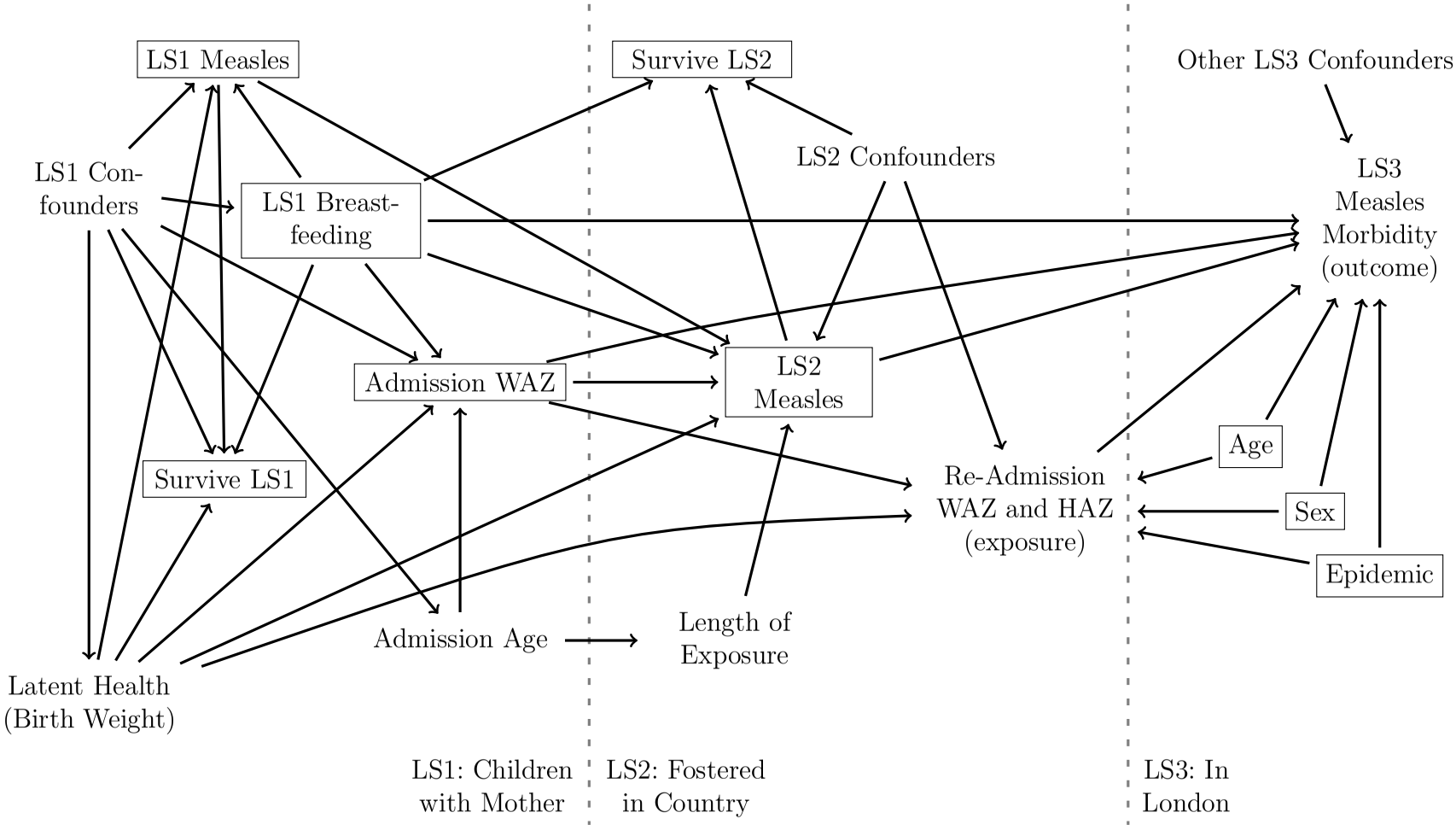
causal estimate of the influence of admission WAZ on the likelihood of contracting measles in life stage two.

4.2 Identification and Empirical Strategy for Life Stage Three Outcomes

Once the children returned to the Foundling Hospital's main site in Bloomsbury, London, the rich morbidity histories in the infirmary books allow for the study of the influence of nutritional status on both an individual's propensity to contract an infectious disease and the time they were treated for the disease in the infirmary, a proxy for illness severity. Figure 7 presents an updated DAG for the infectious disease outcomes in life stage three. Again, because the children were removed from the health environment they faced in life stage two and brought back to the London site, most of the health conditions in life stage two will not affect a child's propensity to contract measles or the severity of their illness if they did contract measles. However, there are a few confounding variables that could still influence the outcome despite the change in environment. From the earlier periods, a child's breastfeeding status may affect their risk of contracting measles or another infectious disease. Their nutritional status in infancy may still have a direct affect on their health status as well. Finally, for diseases with lifelong acquired immunity, whether the child had contracted the disease before would affect their disease status, so I limit the analysis to those children still at risk of contracting a disease. Thus, the regressions are not dissimilar to those performed for life stage two.

However, there are some additional complications. Because all of the children were living together in a relatively close space, it is no longer reasonable to assume that each individual's propensity to contract a disease was independent of whether other children had contracted the disease. In fact, looking at the period incidence of infectious diseases, it is clear that children contracted infectious diseases in epidemics that were relatively infrequent occurrences (see Figure 3). In addition, there is evidence that infectious disease epidemics spread in a systematic way within the Foundling Hospital. The children were separated into different wings based on sex and likely slept in different wards depending

Figure 7: DAG representing the relationship between nutritional status and morbidity in the Foundling Hospital in Life Stage Three



Notes: See Figure 5.

on their age. Children of the same age were also likely to interact more frequently with one another in school classes and in their work to maintain the hospital. Thus, epidemics tended to spread among children of similar age and sex first before spreading to other groups (see Appendix Figure B.3 for an example). In some cases, the hospital was even able to prevent infectious diseases from spreading to all children at risk (see Appendix Figure B.4 for an example).

The fact that children contracted infectious diseases in epidemics and that the spread of disease in the Foundling Hospital was mediated by age and sex unfortunately prevents a hazard model approach to modelling the relationship between nutritional status and susceptibility to disease. However, I can still analyse whether nutritional status affected the propensity to contract an infectious disease by estimating the following empirical equation with logistic regressions for each epidemic separately.[12]

$$Dis_i = \alpha + \beta_1 N_i^{ad} + \beta_2 N_i^{re-ad} + \beta_3 B_i + \phi \text{sex}_i + \sum_{a=1}^{A} \theta \text{age}_i + \rho \left(\text{sex}_i \times \sum_{a=1}^{A} \text{age}_i \right) + \epsilon_i \tag{2}$$

As above, Dis_i is a binary outcome variable equal to zero if child i was at risk of contracting a disease in each epidemic and one if child i contracted the disease. N_i^{ad} and B_i are the same as in Equation 1. N_i^{re-ad} is child i's WAZ and/or HAZ score at re-admission to the Foundling Hospital's main London site at ages 4-6. To account for the differential spread of diseases within the Foundling Hospital, I also control for sex and age categories interacted so that individual propensity to contract the disease varies across these categories.[13] The age break down within the wards is not clear no is precisely which children were assigned to each ward at each point in time, so I assume that children in three age

[12] I do observe children who were treated for measles more than once during their time in the hospital. In this case, I treat the child as at risk of contracting measles in all epidemics where they were exposed and count their treatment for measles as indicating that they had measles. There is no good way of picking which measles case was the true one.

[13] Note that it is not clear whether these variables would confound the relationship between re-admission nutritional status and contracting measles in life stage three. They certainly affect an individual's propensity to contract measles, but it is less clear how sex and the age of contracting measles would affect re-admission nutritional status. However, I cautiously include these controls in order to reduce potential bias.

categories were more likely to interact: children aged 7.99 and younger, children aged 8 to 11.99 and children aged 12 and older.

In addition to understanding the propensity to contract a disease, I use the sickness duration reported in the infirmary reports as a second dependent variable to test whether nutritional status affected the severity of each infectious disease. I exclude a small number of cases where the child contracted the disease in the infirmary, and thus, their sickness duration would include their initial treatment plus the treatment for the infectious disease. The DAG for this empirical setup is virtually identical to Figure 7. Consider the following equation:

$$Dur_i = \alpha + \beta_1 N_i^{ad} + \beta_2 N_i^{re-ad} + \beta_3 B_i + \sum_{n=1}^{N} \gamma \text{epidemic} + \phi \text{sex}_i + \sum_{a=1}^{A} \theta \text{age}_i + \rho \left(\text{sex}_i \times \sum_{a=1}^{A} \text{age}_i \right) + \epsilon_i \quad (3)$$

where Dur_i, the number of days child i spent in the infirmary for each disease, is the outcome and N_i^{ad} and N_i^{re-ad}, defined above, are the main independent variables of interest. In addition, in separate specifications, I specify N_i^{ad} and N_i^{re-ad} as binned dummy variables to capture non-linear effects of nutritional status on sickness duration. B_i and the age and sex interactions are the same as in Equation 2. γ are a series of dummy variables denoting the epidemic in which child i contracted the disease. This controls for changes in policy affecting duration of stay in the infirmary over time which might be correlated with secular trends in nutritional status and confound the main causal path of interest.[14] I estimate Equation 3 using zero-truncated negative binomial models because the number of days spent in the infirmary is a count variable, never takes a value of zero and is over-dispersed.

[14]One might also worry that the sickness duration might vary across an epidemic either because the infirmary became overcrowded leading children to be discharged earlier or because the viral load would be higher for individuals contracting the disease when many other children were infected extending their sickness duration. In the data, there is no relationship between when a child contracted the disease within an epidemic and their sickness duration (not reported), which suggests that either neither of these forces were at play or the two were counteracting one another.

5 Results

5.1 Morbidity in Life Stage Two

Beginning with the results for life stage two, Table 4 shows that nutritional status at admission and birth weight did not have a statistically significant or strong effect on the propensity to contract measles, chicken pox or whooping cough: the coefficients on admission WAZ were very close to zero and the small standard errors confirm a precise null. These results held when specifying the nutrition variables non-linearly as integer bins (not reported). There is a statistically significant positive coefficient of birth weight on whooping cough incidence, which is puzzling, but the sample for which I observe birth weight is relatively small, so I have chosen not to over-emphasise this result. It is also interesting that breastfeeding does not appear to offer any long-run protective effects in preventing children from contracting these infectious diseases. Arthi and Schneider (2021) found that ever breastfed children did have a lower mortality risk in infancy in life stage two, but this protective effect did not appear to extend beyond infancy. This challenges earlier research that has found protective effects for breastfeeding on measles incidence (Silfverdal et al. 2009). Taken together, though, these results suggest that individual-level immunosuppression due to poor nutritional status did not affect individuals' propensity to contract these diseases.

Unfortunately, the only information about sickness severity in life stage two is mortality, and there were too few deaths, 9 from measles and 5 from whooping cough, for statistical analysis on case fatality rates.[15] However, if we compare the admission WAZ of those who died from measles and whooping cough to those who survived these diseases in life stage two, there are some interesting indicative findings. The mean WAZ of children who died of measles in life stage two was 0.69 standard deviations lower than those who survived measles, suggesting that nutritional status could have been a factor in explaining measles mortality. However, this difference was not statistically significant (two-tailed t-test p-value = 0.17). For whooping cough, the mean WAZ of deaths was

[15]These deaths yield case fatality rates of 2.3% and 1.7% for measles and whooping cough respectively.

Table 4: Effect of nutritional status on the probability of contracting each disease while in the countryside (Life Stage Two

	(1)	(2)	(3)	(4)	(5)	(6)
	Measles	Measles	Chicken Pox	Chicken Pox	Whooping Cough	Whooping Cough
Nutritional Status:						
Birth Weight (kg)		-0.597		-0.914		1.235**
		(0.502)		(0.633)		(0.530)
Admission WAZ	-0.003	0.149	-0.044	0.154	-0.017	-0.080
	(0.048)	(0.155)	(0.060)	(0.189)	(0.050)	(0.157)
Infant Feeding in LS1:						
Exclusively Breastfed	0.244	-0.728	0.169	0.150	-0.024	0.397
	(0.239)	(0.730)	(0.305)	(0.852)	(0.250)	(0.734)
Breastfed with Supplementation	0.264*	0.660	0.246	0.547	-0.067	0.587
	(0.149)	(0.433)	(0.190)	(0.563)	(0.154)	(0.457)
Never Breastfed	(ref)	(ref)	(ref)	(ref)	(ref)	(ref)
Additional Controls:						
Male	Yes	Yes	Yes	Yes	Yes	Yes
Birth Year Trend	Yes	Yes	Yes	Yes	Yes	Yes
Length of Life Stage 2	Yes	Yes	Yes	Yes	Yes	Yes
Cases	388	60	163	26	295	49
N	871	137	883	136	884	137

Notes: Coefficients with standard errors in parentheses: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Models are estimated with a logistic regression. I also ran all specifications with birth year fixed effects and the results were qualitatively the same.

Sources: Foundling Hospital Dataset - see Appendix A for precise sources.

only 0.17 standard deviations lower than that of survivors and this difference was highly insignificant (two-tailed t-test p-value = 0.78). Thus, there is tentative evidence to support a role for nutritional status in measles illness severity proxied by case fatality rates but not for whooping cough.

5.2 Morbidity in Life Stage Three

Moving to life stage three, there is again very little evidence that nutritional status affected the propensity to contract the five diseases (see Appendix Table B.2). Only one coefficient is statistically significant at the 5% level, and the signs are more or less evenly split between positive and negative effects of WAZ on contracting the diseases: 15 of 28 coefficients are negative. The negative coefficients are somewhat larger in magnitude than the positive ones, and the standard errors are large, but taken as a whole, there is not strong support that nutritional status affected who contracted these diseases. I also estimated specifications that included admission WAZ and re-admission HAZ and specifications that used the underweight and stunting thresholds for each of the nutrition measures, but these did not produce any clear cut results either (not reported). Thus, there do not appear to be threshold effects for nutrition either.

Shifting to sickness severity by estimating Equation 3, Table 5 shows strong relationships between nutritional status and sickness duration for measles. We see that across the specifications, there is a negative and statistically significant relationship between WAZ at re-admission and measles sickness duration. Interestingly, there is no equivalent effect for HAZ at re-admission, which is puzzling since weight-for-age is often considered a less persistent measure of nutritional deprivation than height-for-age. This may suggest that short-run energy stores were more important when fighting measles than the child's longer-term nutritional status proxied by HAZ. There is also a moderately significant effect of WAZ at admission on measles sickness duration. This result bolsters the existing evidence of long-run scarring effects of nutritional deprivation in infancy on later health, establishing that morbidity could be affected as well as mortality and height (Hatton 2011; Quaranta 2014).

This relationship holds when relaxing linearity assumptions by specifying the nutrition variables as integer binned dummy variables as reported in Figure 8. Predicted sickness duration for children with re-admission WAZ below -2, underweight individuals, was roughly similar. Then there was a decline in sickness duration in the next two bins with children with re-admission WAZ above -1 experiencing roughly similar sickness duration. The difference between underweight children and children with WAZ above -1 was statistically significant and roughly equal to five days, which is large compared to the standard deviation of sickness duration of 9.0 days (see Figure 4). This evidence suggests that the underweight threshold is salient, but the higher sickness duration for children with WAZ scores between -2 and -1 suggests that marginally undernourished children also had more severe measles illness. This evidence, then, confirms the effect of nutritional status on measles morbidity outcomes (Moss and Griffin 2014).

As mentioned above, measles case fatality rates were highest for young children, so one might wonder to what extent these findings for children aged *c.* 5 and older can be applied to younger children. Since most measles deaths for young children were caused by secondary respiratory complications, analysing the relationship between nutritional status and complications from measles infections in life stage three may help to extrapolate the findings to younger children. Ten percent of children experienced complications from measles in life stage three. Half of those experiencing complications had some form of pneumonia whereas the other half had complications including enlarged lymph nodes, stomatitis, bronchitis and otorrhoea. A number were simply listed as convalescent, but since these children were singled out for a longer stay in the hospital, it seems that they had more severe illness. Appendix Table B.3 reports logistic regressions replacing the outcome variable in Equation 3 with a binary variable being equal to one if a child experienced measles complications and zero if they contracted measles in life stage three. These results confirm that not only was nutritional status related to sickness duration, higher WAZ at readmission also reduced the probability of having a complication from measles as well: a one-unit increase in re-admission WAZ led to a 84.8% decrease in the odds of developing complications. Thus, we can assume that these results would hold

Table 5: Effect of nutritional status on measles sickness severity (duration of stay in the infirmary)

Dep Var: Measles Sickness Duration	(1)	(2)	(3)	(4)
Nutritional Status:				
Admission WAZ (infancy)				-0.038*
				(0.021)
Re-Admission WAZ (age 4-6)	-0.116***		-0.143***	-0.139***
	(0.031)		(0.039)	(0.039)
Re-Admission HAZ (age 4-6)		-0.038	0.038	0.046
		(0.027)	(0.033)	(0.034)
Infant Feeding in LS1:				
Exclusively Breastfed	0.006	0.004	0.011	0.034
	(0.104)	(0.106)	(0.104)	(0.104)
Breastfed with Supplementation	0.060	0.046	0.061	0.076
	(0.061)	(0.062)	(0.061)	(0.061)
Never Breastfed	(ref)	(ref)	(ref)	(ref)
Sex (male = 1)	0.128*	0.130*	0.133*	0.135*
	(0.072)	(0.073)	(0.072)	(0.072)
Age at Infection:				
Age 7.99 and under	(ref)	(ref)	(ref)	(ref)
Age 8 to 11.99	-0.105	-0.074	-0.099	-0.089
	(0.104)	(0.105)	(0.104)	(0.103)
Age 12 and over	0.073	0.065	0.074	0.094
	(0.169)	(0.172)	(0.169)	(0.169)
Additional Controls:				
Epidemic Dummies	Yes	Yes	Yes	Yes
Sex × Age Categories	Yes	Yes	Yes	Yes
N	281	281	281	280

Notes: Coefficients with standard errors in parentheses: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Models are estimated with a zero-truncated negative binomial regression: the over-dispersion statistic is significant for all specifications suggesting that the negative binomial model is more appropriate than poisson. The age category dummies are the age when the child contracted the disease.

Sources: Foundling Hospital Dataset - see Appendix A for precise sources.

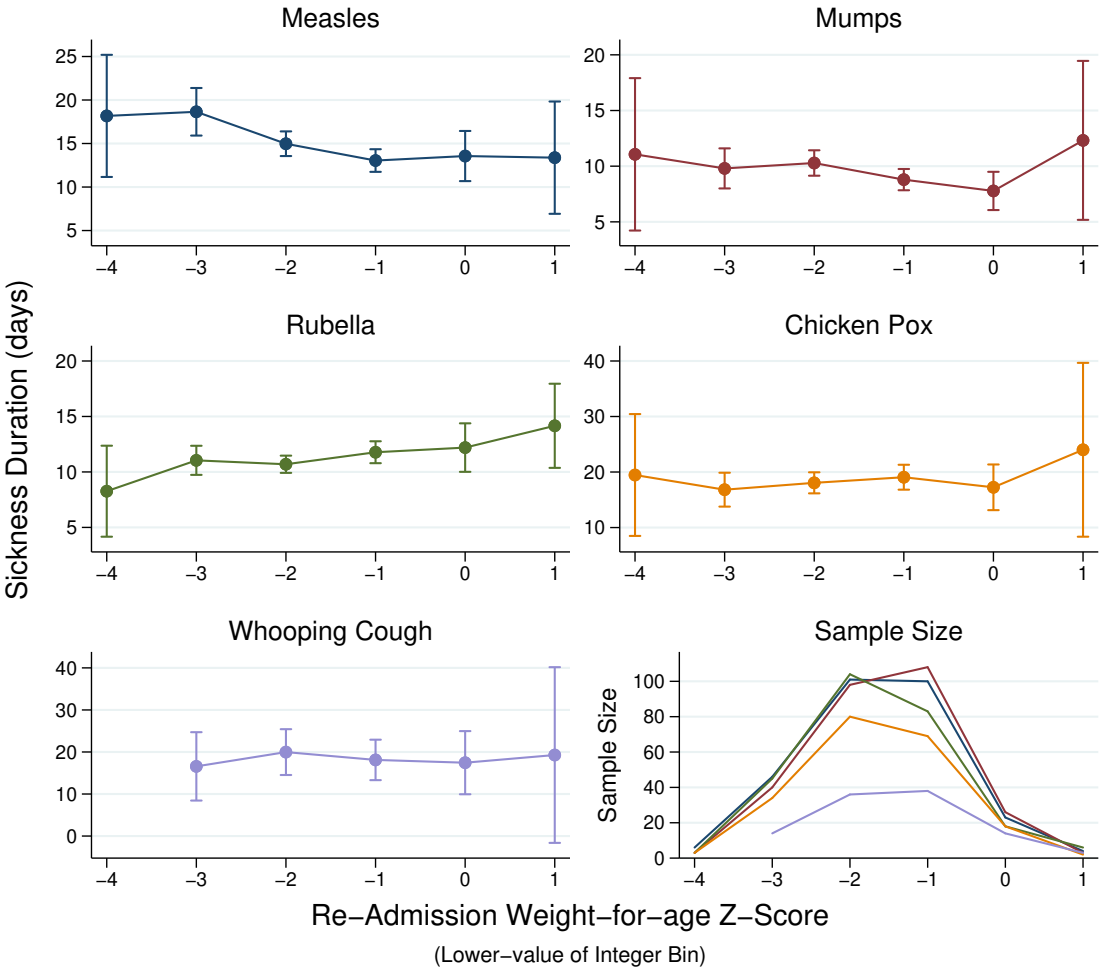


Figure 8: Predicted values of sickness duration by re-admission weight-for-age bins for each disease

Notes: Point estimates and 95% confidence intervals are predicted from zero-truncated negative binomial regressions estimating Equation 3 where WAZ is specified as integer-binned dummy variables. The final graph shows the sample size used in the regressions for each WAZ bin for each disease.

Sources: Foundling Hospital Dataset - see Appendix A for precise sources.

with younger children.

Table 6 shows similar negative effects for nutritional status on mumps sickness duration. For mumps, re-admission WAZ has a consistently negative effect on sickness duration, but there is no effect of re-admission HAZ. If we again replace the linear nutrition variables with binned integer dummies, there do appear to be some threshold effects for mumps (Figure 8). Children with WAZ scores under -1 had relatively similar sickness duration around 10 days, but sickness duration declined with nutritional status with children with WAZ scores between 0 and 1 having sickness durations of 7.75 days. This difference of around 2.25 days (statistically significant at the 5% level) is large relative to the standard deviation of sickness duration for mumps of 5.1 days. Thus, for mumps, the underweight threshold was less important than for measles, although there do appear to be threshold effects for both measles and mumps.

The results for chicken pox, whooping cough and rubella are reported in Appendix Tables B.4-B.6, but in all cases show neither statistically significant nor demographically meaningful relationships between nutritional status and sickness duration. These linear results are confirmed when allowing for non-linearities in the relationship between nutritional status and sickness duration (Figure 8). The relationship for rubella almost seems positive, but none of the predicted values are statistically different from one another. There were also no effects of nutritional status on the probability of developing complications from these diseases, though the complication rates were very low (not reported). The results are perhaps unsurprising for rubella and chicken pox, since there is relatively limited evidence that their severity is linked to nutritional status. However, many scholars have argued that nutrition affects whooping cough severity, so the results challenge this consensus (Bellagio Conferees 1983; Cliff et al. 1998; Halperin and De Serres 2009; Hardy 1993).

The fact that many whooping cough cases are asymptomatic or sub-clinical would only bias this finding related to sickness severity if the propensity to have a symptomatic case was related to nutritional status, i.e. low nutritional status children were more likely to experience symptomatic infection than high nutritional status children. If this were true,

Table 6: Effect of nutritional status on mumps sickness severity (duration of stay in the infirmary)

Dep Var: Mumps Sickness Duration	(1)	(2)	(3)	(4)
Nutritional Status:				
Admission WAZ (infancy)				0.001
				(0.024)
Re-Admission WAZ (age 4-6)	-0.070*		-0.097**	-0.097**
	(0.038)		(0.046)	(0.047)
Re-Admission HAZ (age 4-6)		-0.006	0.039	0.039
		(0.029)	(0.036)	(0.036)
Infant Feeding in LS1:				
Exclusively Breastfed	-0.108	-0.091	-0.095	-0.096
	(0.110)	(0.111)	(0.111)	(0.112)
Breastfed with Supplementation	-0.095	-0.099	-0.096	-0.097
	(0.068)	(0.068)	(0.069)	(0.069)
Never Breastfed	(ref)	(ref)	(ref)	(ref)
Sex (male = 1)	0.194	0.176	0.206*	0.207*
	(0.122)	(0.122)	(0.123)	(0.123)
Age at Infection:				
Age 7.99 and under	(ref)	(ref)	(ref)	(ref)
Age 8 to 11.99	-0.195*	-0.194*	-0.187	-0.186
	(0.117)	(0.117)	(0.117)	(0.118)
Age 12 and over	-0.564***	-0.568***	-0.567***	-0.567***
	(0.148)	(0.149)	(0.148)	(0.149)
Additional Controls:				
Epidemic Dummies	Yes	Yes	Yes	Yes
Sex × Age Categories	Yes	Yes	Yes	Yes
N	279	280	278	278

Notes: Coefficients with standard errors in parentheses: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Models are estimated with a zero-truncated negative binomial regression: the over-dispersion statistic is significant for all specifications suggesting that the negative binomial model is more appropriate than poisson. The regression excludes one influential outlier where a child remained in the infirmary for 259 days following a mumps infection. This single observation nearly tripled the coefficient on re-admission WAZ.

Sources: Foundling Hospital Dataset - see Appendix A for precise sources.

counterfactually including asymptomatic cases at higher levels of nutritional status would reduce sickness severity among children with high nutritional status and create a negative relationship between nutritional status and whooping cough sickness severity. However, the results from life stage two suggest that nutritional status did not affect the propensity of an individual to contract a symptomatic case of whooping cough: symptomatic cases are the only cases that can be observed in the Foundling Hospital data. Thus, it seems unlikely that the influence of nutritional status on having a symptomatic case is large enough to create a negative relationship between nutritional status and whooping cough severity.

Although the main variables of interest are the nutritional status variables, it is also interesting that the breastfeeding variables did not have a strong effect on sickness duration for measles and mumps. This again suggests that breastfeeding may not have provided the long-run protective effect against measles and other diseases that Silfverdal et al. (2009) found. Boys had slightly longer measles sickness durations than girls, but the difference was only borderline significant (Table 5). Likewise, children over age 12 had substantially shorter sickness durations for mumps and chicken pox (Table 6 and B.5), suggesting that the illness may have been less severe for older children. Although I do not report the epidemic dummies and sex by age category interactions, a few of the epidemics had longer or shorter durations, highlighting the importance of including these variables, but the sex by age category interactions were very rarely statistically significant.

6 Conclusion

This paper uses a unique historical cohort study to analyse the effect of nutritional status on the probability of contracting measles, mumps, rubella, chicken pox and whooping cough and on sickness duration and severity once infected. Nutritional status, proxied by anthropometric status before infection, did not strongly influence children's susceptibility to the five diseases studied here. This is perhaps unsurprising given how contagious these diseases were (Post 1990, p. 243). However, there is substantial evidence that low

nutritional status exacerbated measles infections by increasing sickness duration and the probability of developing potentially fatal complications. These effects were worst for underweight individuals, indicating that there were threshold effects in the relationship. Nutritional status also influenced mumps sickness duration, but the effect size was somewhat smaller. Finally, there were no effects of nutritional status on rubella, chicken pox or whooping cough sickness duration, which contradicts earlier literature that had argued for an effect for whooping cough (Bellagio Conferees 1983; Cliff et al. 1998; Halperin and De Serres 2009; Hardy 1993; Post 1990).

Interestingly, weight-for-age was more important for measles and mumps sickness duration than height-for-age, and these results were statistically significant even when the weight-for-age measurements were taken many years before the sickness event. While this may lead to some attenuation bias in my estimates, it also emphasises the difference between weight-for-age and height-for-age as proxies for nutritional status. Wells (2010) describes weight as 'liquid' capital which can be used by the body over relatively short periods when needed whereas height is 'illiquid' capital that reflects longer-term health conditions but cannot be changed or adapted in the short run. This framework seems to apply here since heavier children were able to draw on their nutrient stores to help fight measles or mumps. Therefore, the availability of these stores may be more important than the child's underlying long-term health proxied by their height. In any case, most of the historical evidence on child growth has focused on trends in height and child stunting (Gao and Schneider 2021), and this evidence suggests that finding trends in weight-for-age for children under age 10, where the WHO WAZ standard applies, could be fruitful in understanding trends in children's liquid capital.

The evidence from this paper also helps to settle some of the larger debates discussed in the introduction. First, at least for these five, highly infectious diseases, nutritional status did not affect susceptibility to infection even when considering life stage two where the children were living in a non-institutional setting. This means that individual-level immunosuppression caused by poor nutritional status for instance during famines or among indigenous peoples following the Colombian Exchange was likely not an important factor

in promoting the spread of diseases. The breakdown of collective immunosuppression may have been far more important (Mokyr and Ó Gráda 2002; Post 1990). However, the results do suggest that mortality from measles epidemics would have been greater among malnourished populations, which means that poor nutritional status could have exacerbated measles mortality following famines or during the Colombian Exchange.

These results also speak to the causes of mortality decline in the nineteenth and twentieth centuries. Measles and whooping cough were leading killers of children in the mid-nineteenth century, and whereas whooping cough mortality began to decline in England and Wales before 1900, measles mortality did not begin to fall until the First World War (see Appendix Figures C.1 and C.2). The highly contagious nature of whooping cough and measles along with the limited historical evidence on incidence suggests that incidence of these diseases was not falling over time, so the decline in mortality was driven by a decline in the case fatality rates (Amirthalingam et al. 2013; Cliff et al. 1998; Fine and Clarkson 1982; Gordon and Hood 1951; Hedrich 1930; London and Yorke 1973). The evidence from this study suggests that improvements in nutrition in the late nineteenth and first half of the twentieth century would not have influenced whooping cough case fatality rates, so declines in whooping cough mortality must have been driven by other factors such as improved quality of nursing care (Hardy 1993, p. 27).

The late timing of measles mortality decline presents a conundrum for understanding the influence of nutritional status on measles mortality decline. The current historical consensus is that nutrition and nutritional status of British people had improved dramatically by the First World War. Calories and protein were plentiful and even micronutrient deficiencies were far less common by 1914 (Floud et al. 2011; Gazeley and Horrell 2013; Gazeley and Newell 2015). Adult and child heights had been increasing since the 1870s (Gao and Schneider 2021; Hatton 2011, 2014). Thus, why did measles mortality only fall after 1915? This question is beyond the scope of the current paper, but one possible explanation is that the introduction of compulsory schooling led to greater social mixing and increased the frequency of measles epidemics. While this would probably not have affected the share of the population contracting measles before adulthood, more frequent

epidemics would have increased measles incidence among young children, who had higher mortality risk from the disease, and increased case fatality rates. However, improvements in nutrition during the same period would have had a downward effect on measles case fatality rates. Thus, stagnant measles mortality rates from 1870 to 1915 may have been the result of these two countervailing forces. Why measles mortality began to decline from 1915 is less clear. Hardy (1993, pp. 54-55) emphasises the importance of the First World War in changing consumption patterns because of rationing and granting women greater control over household resources to the benefit of children. Nutritional conditions also continued to improve after 1915 (Gazeley et al. 2021), so it is possible that nutritional conditions reached a threshold where measles case fatality rates were much lower. This highlights again that further research on trends in child weight might be useful for clarifying measles mortality trends.

Finally, the results from this paper affect interpretations of scarring and selective culling and their impact on population health. Quaranta (2013, chapter 3) analyses how exposure to measles and whooping cough epidemics in infancy shaped mortality up to age 70. She finds that whooping cough had a strong scarring effect on mortality at older ages, whereas the pattern was less clear for measles with no statistically significant differences in later life mortality between cohorts experiencing measles epidemics in infancy and those who did not. According to the results presented above, while whooping cough morbidity and perhaps mortality was not related to nutritional status, measles was more likely to kill children who already had low nutritional status and thus low health status. This means that the survival bias of children surviving measles is likely larger than for whooping cough. Since health outcomes in later life are always a balance of the scarring and selection effects across the life course, it is perhaps not surprising that the scarring effect is more dominant for whooping cough as the associated survival bias (selection effect) would be smaller than for measles.

These insights have important implications for understanding changes in cohort health over time. As mentioned above, it is unlikely that the incidence of measles or whooping cough fell before widespread vaccination, which means that there might not have been

reductions in the penalties to health of contracting these diseases as mortality fell. Because of data constraints, Quaranta was not able to determine whether these penalties changed as case fatality and mortality rates fell. If anything, the older-age scarring effects of whooping cough became more important across the nineteenth century in her data (Quaranta 2013, p. 145). Unless the decline in case fatality rates from these diseases was also associated with reduced later-life health penalties, then reductions in mortality rates would not necessarily reflect improvements in cohort health. In addition, the strong selection effects from measles mortality would have diminished as measles mortality fell, perhaps making the later-life scarring effect more important for cohort health as measles mortality fell. In any case, the effects of changing incidence and case fatality rates of measles and whooping cough on cohort health are ambiguous and would certainly not have had a straightforwardly positive influence on cohort health. Thus, improvements in cohort health related to reductions in childhood disease were more likely driven by declines in diarrhoeal and/or respiratory diseases, diseases that influenced height, instead of the diseases important for child mortality highlighted here (Schellekens and van Poppel 2016).

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Online Appendices

A Data Sources and Structure of the Dataset

Records relating to the Foundling Hospital and held at the London Metropolitan Archives (LMA). Although the records are held by the LMA, which is a public body, the records are still strictly controlled by the Coram Foundation, the institution that succeeded the Foundling Hospital. All records that contain personal information about children are subject to a 110-year embargo to the general public, and researchers can only access the documents by gaining permission from Coram. Table A.1 reports the specific sources for various pieces of information about the children. Of these, the medical record and register of applications are closed access, and certain parts of the petitions and weekly infirmary reports are also closed. I am grateful to the Coram Foundation for allowing me to access the records and create the linked cohort study upon which this paper is based.

Table A.1: Structure and sources of the Foundling Hospital Dataset

Event/Life Stage	Years	Source (LMA Reference)	Socioeconomic/Administrative Information	Health Information
Birth	1892-1908	FH Petitions (A/FH/A/8/1/2/102-117)	Mother's approximate address Mother's age Where child was born Father's occupation When mother last saw father What became of father	Child's birthday Child's sex
	1909-1914	Register of applications (A/FH/A/8/5/1)	Mother's approximate address Mother's age Father's occupation	Child's birthday Child's sex
	1892-1908	Registers of In-Patients Queen Charlotte Hospital (H27/QC/B/1/8-13)	Mother's marital status Mother's age	Child's birthday Child's sex Child's parity Birth weight Birth length
LS1: Pre-admission to FH (0-1 year old)	1892-1914	Medical Record (A/FH/A/18/15/1)		Infant feeding practice (breast, milk or food) Duration of breastfeeding
Admission to FH (around 0.37 years old)	1893-1914	Medical Record (A/FH/A/18/15/1)	Admission date Hospital number Admission age	Child's birthday Child's sex Weight Subjective nutritional assesment Vaccinated Diseases present at entry
LS2: Time Fostered in Country (1-6 years old)	1893-1919	Medical Record (A/FH/A/18/15/1)	County child was fostered in	Diseases child was treated for in country
Return from Country to FH (4-6 years old)	1897-1919	Medical Record (A/FH/A/18/15/1)	Re-admission date Re-admission age	Weight Height Subjective nutritional assesment Eye exam Ear exam
LS3: Time Resident in FH (6-17 years old)	1897-1919	Medical Record (A/FH/A/18/15/1)	School standard	Diseases child was treated for in hospital Re-vaccinated
	1897-1915	Weekly Infirmary Reports (A/FH/A/18/5/30-35)		All diseases child was treated for in infirmary Complications from diseases Dates of entry to and exit from the infirmary Duration of each sickness event
Discharge from FH (around 15-17 years old)	1907-1919	Medical Record (A/FH/A/18/15/1)	Discharge date Discharge age Employment after discharge	Weight Height Subjective state of health
Other Life Events				
Restored to Parents (any age)	1892-1919	Medical Record (A/FH/A/18/15/1)	Date of restoration Who child was restored to	
Deaths (any age)	1892-1919	Medical Record (A/FH/A/18/15/1)	Date of death Place of death	Cause of death

B Additional Results

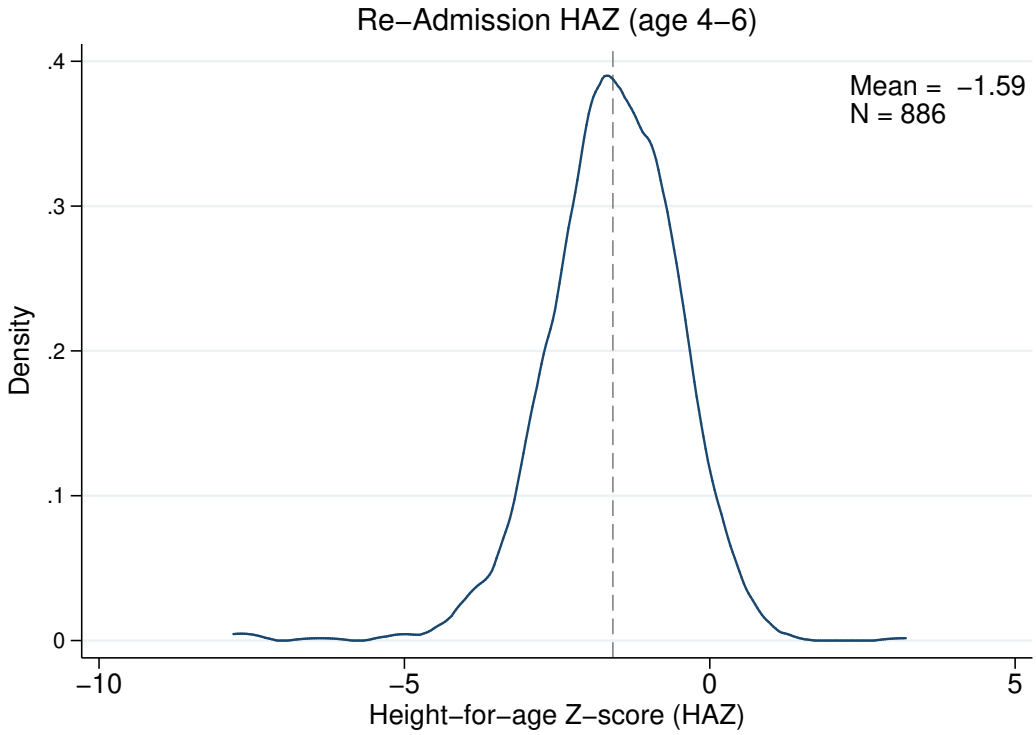

Figure B.1: Distribution of height-for-age Z-scores (HAZ) at re-admission

Notes: The vertical dashed line marks the mean.

Sources: Foundling Hospital Dataset - see Appendix A for precise sources.

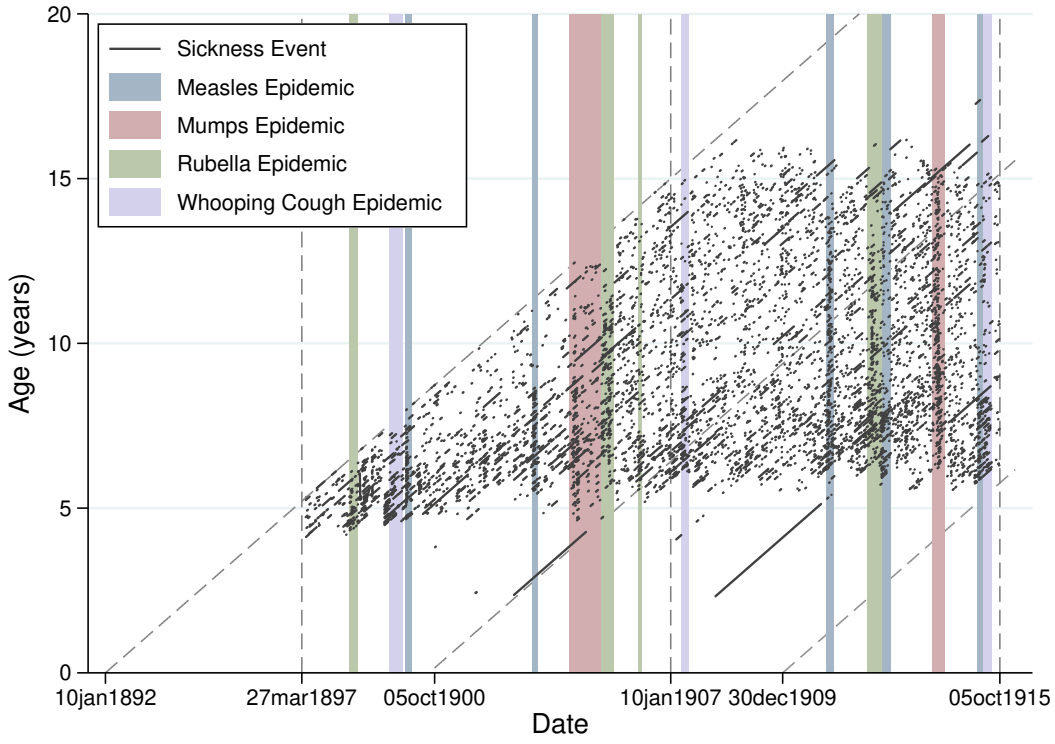

Figure B.2: Lexis diagram showing all sickness events recorded in the infirmary book and epidemics from four of the five diseases

Notes: Chicken pox epidemics are excluded because there were too many to easily highlight on the graph.

Sources: Foundling Hospital Dataset - see Appendix A for precise sources.

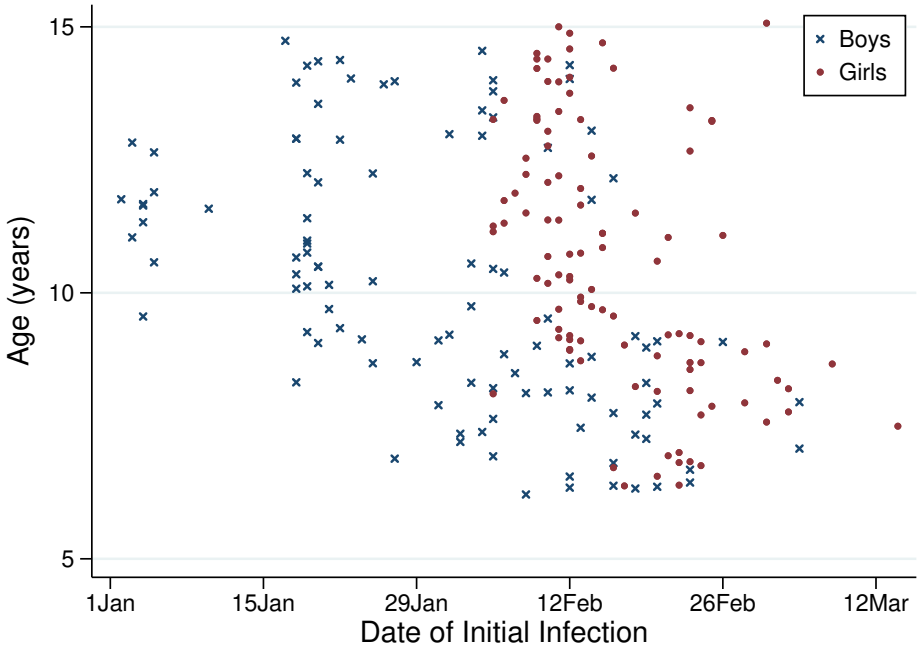

Figure B.3: Spread of Mumps in the Foundling Hospital Population in 1914

Sources: Foundling Hospital Dataset - see Appendix A for precise sources.

Table B.1: Descriptive statistics of cases and incidence for epidemics of the five main diseases in the Foundling Hospital

Epidemics	Individuals at Risk	Cases	Probability of Infection (%)
Measles Epidemics:			
20dec1899	68	60	88.2
07may1903	76	56	73.7
25feb1911	198	127	64.1
01aug1912	111	32	28.8
04mar1915	100	31	31.0
Mumps Epidemics:			
06may1904	262	82	31.3
16dec1913	356	212	59.6
Rubella Epidemics:			
30jun1898	61	31	50.8
12mar1905	253	116	45.8
02mar1906	167	16	9.6
27mar1912	303	108	35.6
Chicken Pox Epidemics:			
22oct1898	56	24	42.9
10apr1900	59	18	30.5
06jan1902	90	28	31.1
17jun1904	167	48	28.7
31aug1905	156	3	1.9
16mar1906	152	8	5.3
21jan1907	179	16	8.9
19aug1907	177	13	7.3
09apr1910	181	15	8.3
27jan1911	195	16	8.2
09sep1912	220	12	5.5
27aug1913	214	4	1.9
05oct1914	222	12	5.4
02feb1915	219	2	0.9
Whooping Cough Epidemics:			
21jul1899	88	15	17.0
18apr1907	334	39	11.7
24apr1915	370	56	15.1

Notes: Individuals at risk were present in the Foundling Hospital at the start of the epidemic, had not contracted the disease before it the disease granted lifelong immunity and could be linked to the medical record (see Figure B.2). Epidemics are defined as distinctive appearances of each disease separated by months with no incidence.

Sources: Foundling Hospital Dataset - see Appendix A for precise sources.

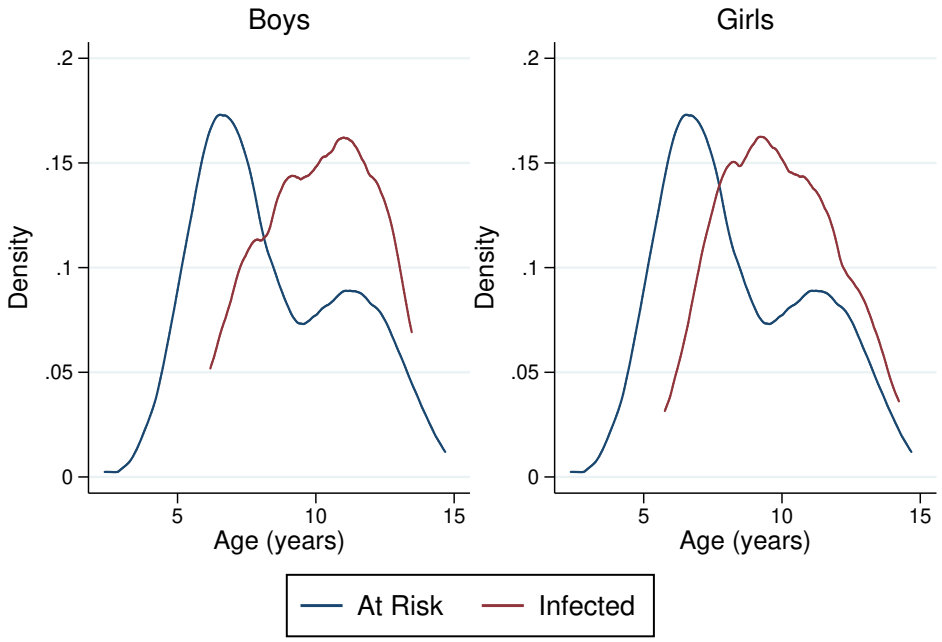

Figure B.4: Age Distribution of those at risk of contracting measles versus those who were infected (February-April 1911)

Notes: Individuals at risk were present in the Foundling Hospital at the start of the epidemic, had not contracted the disease before it the disease granted lifelong immunity and could be linked to the medical record (see Figure B.2).

Sources: Foundling Hospital Dataset - see Appendix A for precise sources.

Table B.2: Logistic regressions showing the effect of nutritional status on the propensity to contract a disease in the epidemics of life stage three

	Admission WAZ				
Epidemic	Coefficient	Standard Error	Cases	N	Pseudo R-square
Measles					
Epidemic 1	-0.301	0.399	58	66	0.018
Epidemic 2	-0.050	0.367	56	76	0.189
Epidemic 3	0.179	0.167	125	196	0.085
Epidemic 4	0.477	0.392	32	69	0.181
Epidemic 5	-1.011*	0.557	31	55	0.454
Mumps					
Epidemic 1	0.190	0.157	82	259	0.035
Epidemic 2	-0.093	0.138	207	349	0.025
Rubella					
Epidemic 1	-0.311	0.322	31	60	0.017
Epidemic 2	0.002	0.150	115	244	0.082
Epidemic 3	0.062	0.450	16	108	0.199
Epidemic 4	-0.150	0.166	105	264	0.073
Chicken Pox					
Epidemic 1	-0.523	0.327	24	55	0.054
Epidemic 2	0.400	0.329	18	57	0.032
Epidemic 3	-0.020	0.290	28	59	0.026
Epidemic 4	0.061	0.210	48	163	0.166
Epidemic 5	-0.417	0.879	3	30	0.197
Epidemic 6	0.719	0.547	8	83	0.183
Epidemic 7	-0.974**	0.421	16	59	0.100
Epidemic 8	-0.703*	0.417	12	53	0.120
Epidemic 9	-0.381	0.354	15	108	0.232
Epidemic 10	-0.588	0.382	14	84	0.168
Epidemic 11	0.183	0.499	12	112	0.078
Epidemic 12	0.065	0.349	4	125	0.041
Epidemic 13	-0.663	0.421	12	98	0.197
Epidemic 14	0.020	0.907	2	52	0.008
Whooping Cough					
Epidemic 1	0.568*	0.333	15	87	0.057
Epidemic 2	-0.119	0.214	39	331	0.091
Epidemic 3	0.114	0.193	56	360	0.100

Notes: Coefficients and standard errors reported: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Models are estimated with a logistic regression following Equation 2. Models are presented as rows and contain the following controls as stated in Equation 2: breastfeeding status, sex, age category, and sex by age category interactions. Models including admission WAZ and re-admission HAZ were qualitatively similar (not reported).

Sources: Foundling Hospital Dataset - see Appendix A for precise sources.

Table B.3: Effect of nutritional status on complications arising from measles

Dep Var: Measles Complications	(1)	(2)	(3)	(4)
Nutritional Status:				
Admission WAZ (infancy)				-0.239
				(0.150)
Re-Admission WAZ (age 4-6)	-0.614**		-0.574**	-0.545*
	(0.247)		(0.283)	(0.279)
Re-Admission HAZ (age 4-6)		-0.269	-0.067	-0.022
		(0.175)	(0.207)	(0.201)
Infant Feeding in LS1:				
Exclusively Breastfed	1.187*	1.026	1.186*	1.276*
	(0.666)	(0.678)	(0.664)	(0.680)
Breastfed with Supplementation	-0.094	-0.216	-0.103	-0.001
	(0.460)	(0.448)	(0.460)	(0.463)
Never Breastfed	(ref)	(ref)	(ref)	(ref)
Sex (male = 1)	0.601	0.631	0.593	0.544
	(0.538)	(0.524)	(0.534)	(0.545)
Age at Infection:				
Age 7.99 and under	(ref)	(ref)	(ref)	(ref)
Age 8 to 11.99	-0.393	-0.201	-0.400	-0.349
	(0.756)	(0.784)	(0.756)	(0.761)
Age 12 and over	1.721*	1.680*	1.746*	1.869*
	(1.005)	(1.015)	(1.024)	(1.035)
Additional Controls:				
Epidemic Dummies	Yes	Yes	Yes	Yes
Sex × Age Categories	Yes	Yes	Yes	Yes
N	233	233	233	232

Notes: Coefficients with standard errors in parentheses: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Models are estimated with a logistic regression. The age category dummies are the age when the child contracted the disease.

Sources: Foundling Hospital Dataset - see Appendix A for precise sources.

Table B.4: Effect of nutritional status on rubella sickness severity (duration of stay in the infirmary)

Dep Var: Rubella Sickness Duration	(1)	(2)	(3)	(4)
Nutritional Status:				
Admission WAZ (infancy)				-0.003
				(0.016)
Re-Admission WAZ (age 4-6)	0.024		0.057*	0.058*
	(0.025)		(0.032)	(0.032)
Re-Admission HAZ (age 4-6)		-0.015	-0.048	-0.046
		(0.023)	(0.029)	(0.030)
Infant Feeding in LS1:				
Exclusively Breastfed	0.021	0.009	0.028	0.030
	(0.077)	(0.076)	(0.076)	(0.077)
Breastfed with Supplementation	-0.039	-0.042	-0.044	-0.043
	(0.047)	(0.048)	(0.047)	(0.048)
Never Breastfed	(ref)	(ref)	(ref)	(ref)
Sex (male = 1)	0.104	0.101	0.105	0.104
	(0.070)	(0.070)	(0.070)	(0.070)
Age at Infection:				
Age 7.99 and under	(ref)	(ref)	(ref)	(ref)
Age 8 to 11.99	0.043	0.039	0.044	0.046
	(0.071)	(0.071)	(0.071)	(0.071)
Age 12 and over	-0.075	-0.073	-0.066	-0.070
	(0.170)	(0.171)	(0.170)	(0.171)
Additional Controls:				
Epidemic Dummies	Yes	Yes	Yes	Yes
Sex × Age Categories	Yes	Yes	Yes	Yes
N	261	260	260	260

Notes: Coefficients with standard errors in parentheses: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Models are estimated with a zero-truncated negative binomial regression: the over-dispersion statistic is significant for all specifications suggesting that the negative binomial model is more appropriate than poisson.

Sources: Foundling Hospital Dataset - see Appendix A for precise sources.

Table B.5: Effect of nutritional status on chicken pox sickness severity (duration of stay in the infirmary)

Dep Var: Chicken Pox Sickness Duration	(1)	(2)	(3)	(4)
Nutritional Status:				
Admission WAZ (infancy)				0.017
				(0.026)
Re-Admission WAZ (age 4-6)	0.061		0.039	0.033
	(0.039)		(0.049)	(0.050)
Re-Admission HAZ (age 4-6)		0.044	0.028	0.025
		(0.031)	(0.038)	(0.038)
Infant Feeding in LS1:				
Exclusively Breastfed	0.041	0.046	0.039	0.034
	(0.126)	(0.126)	(0.126)	(0.126)
Breastfed with Supplementation	-0.087	-0.082	-0.089	-0.096
	(0.069)	(0.069)	(0.069)	(0.070)
Never Breastfed	(ref)	(ref)	(ref)	(ref)
Sex (male = 1)	-0.043	-0.036	-0.035	-0.035
	(0.069)	(0.070)	(0.070)	(0.071)
Age at Infection:				
Age 7.99 and under	(ref)	(ref)	(ref)	(ref)
Age 8 to 11.99	-0.083	-0.071	-0.081	-0.079
	(0.150)	(0.150)	(0.150)	(0.150)
Age 12 and over	-0.826**	-0.849**	-0.827**	-0.825**
	(0.374)	(0.374)	(0.374)	(0.374)
Additional Controls:				
Epidemic Dummies	Yes	Yes	Yes	Yes
Sex × Age Categories	Yes	Yes	Yes	Yes
N	206	207	206	205

Notes: Coefficients with standard errors in parentheses: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Models are estimated with a zero-truncated negative binomial regression: the over-dispersion statistic is significant for all specifications suggesting that the negative binomial model is more appropriate than poisson.

Sources: Foundling Hospital Dataset - see Appendix A for precise sources.

Table B.6: Effect of nutritional status on whooping cough sickness severity (duration of stay in the infirmary)

Dep Var: Whooping Cough Sickness Duration	(1)	(2)	(3)	(4)
Nutritional Status:				
Admission WAZ (infancy)				0.005
				(0.055)
Re-Admission WAZ (age 4-6)	0.062		0.061	0.060
	(0.081)		(0.092)	(0.093)
Re-Admission HAZ (age 4-6)		0.026	0.002	0.000
		(0.069)	(0.079)	(0.081)
Infant Feeding in LS1:				
Exclusively Breastfed	-0.001	-0.002	-0.001	-0.004
	(0.243)	(0.246)	(0.245)	(0.248)
Breastfed with Supplementation	-0.264	-0.276	-0.264	-0.265
	(0.173)	(0.172)	(0.173)	(0.174)
Never Breastfed	(ref)	(ref)	(ref)	(ref)
Sex (male = 1)	0.280	0.264	0.279	0.280
	(0.206)	(0.208)	(0.209)	(0.209)
Age at Infection:				
Age 7.99 and under	(ref)	(ref)	(ref)	(ref)
Age 8 to 11.99	0.004	-0.015	0.003	-0.000
	(0.257)	(0.259)	(0.259)	(0.262)
Age 12 and over	0.150	0.117	0.149	0.149
	(0.314)	(0.312)	(0.315)	(0.315)
Additional Controls:				
Epidemic Dummies	Yes	Yes	Yes	Yes
Sex × Age Categories	Yes	Yes	Yes	Yes
N	105	105	105	105

Notes: Coefficients with standard errors in parentheses: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Models are estimated with a zero-truncated negative binomial regression: the over-dispersion statistic is significant for all specifications suggesting that the negative binomial model is more appropriate than poisson.

Sources: Foundling Hospital Dataset - see Appendix A for precise sources.

C The Five Diseases: Descriptions and Historical Context

C.1 Measles

Measles was a very deadly disease in the late nineteenth and early twentieth century. Measles is a viral infection spread by respiratory droplets. The incubation period lasts ten days when typically a fever develops followed by a distinctive rash after 14 days. Patients are most infectious a few days before and after the rash appears, ensuring that the disease is able to spread before it can be definitively diagnosed. Measles is one of the most contagious diseases known to scientists with a basic reproduction number (R_0) of 12 to 18. Most measles deaths arise from respiratory complications such as pneumonia following the disease, and these complications (and fatalities) are most common among younger children. Infants are somewhat protected from measles in the first six to nine months of life from antibodies passed from mother to child *in utero* (Moss and Griffin 2014). There is some evidence that breastfeeding is also protective against contracting measles, though the Silfverdal et al. (2009) study that found this link is based on the 1970 British Cohort Study after vaccination against measles became routine in the UK in 1968 (Fine and Clarkson 1982) and may not be representative of the pre-vaccine era.[16] Measles case fatality rates vary depending on the historical period, socio-economic context and development of health systems with measles case fatality rates having fallen from nearly 4% in 1990 to close to 1% in 2015 (Portnoy et al. 2019). Nutritional status is also an important contributor to case fatality rates, with malnourished children facing much higher mortality (Moss and Griffin 2014), though this consensus has been challenged (Aaby et al. 1983).[17]

[16]The identification in this study is based on looking at breastfeeding on having contracted measles by age 10. The authors control for socio-economic status and a host of other confounder variables, but there are still serious questions about whether children not vaccinated and not breastfed in this period are negatively selected on unobserved characteristics relative to children breastfed and not vaccinated.

[17]Aaby et al. (1983) challenged the notion that malnutrition was important for case fatality rates using based on a measle outbreak in Guinea-Bissau in the late 1970s. They found no difference in height-for-age, weight-for-age or weight-for-height between children who survived or died in the epidemic. Instead, they argue that case fatality was related to previous respiratory infections, comorbidities during measles infection and overcrowding, which increased the initial viral load in the infected (Aaby et al. 1984). This

Before widespread vaccination, measles incidence and mortality followed a relatively uniform cyclical pattern. Incidence and deaths tended to peak in late winter and early spring annually, and major outbreaks occurred every two to five years in larger populations. The annual cycle was likely driven by increased social interaction among children during the school year and the increased transmission of the measles virus in cooler and less sunny conditions. The longer term cycle was produced by the growing number of susceptible children born each year, eventually leading to a larger outbreak (Moss and Griffin 2014).

Information about measles incidence rates over time is difficult to find because measles was not a notifiable disease in the UK until 1915 (Hardy 1993, p. 53). Early evidence of incidence was collected for Aberdeen in the late nineteenth century and shows increasing measles incidence between the 1880s and 1890s (Wilson 1905). Evidence for New York City and Baltimore do not show clear declines in incidence before vaccination in the early twentieth century (Fine and Clarkson 1982; Hedrich 1930; London and Yorke 1973), nor does a national series produced by Cliff et al. (1998, p. 326). In addition, four urban surveys in the early twentieth century (1913-23) from the United States, Canada and the UK suggest that over 90% of children had contracted measles by the age of 15 (Hedrich 1930).[18] Measles epidemics among US soldiers during the Civil War and among US and Australian soldiers during the First World War suggest that in these countries, measles

evidence is interesting, but it is based on a relatively small sample size and related to an epidemic with a case fatality rate of 25%, well above case fatality rates from measles in other community and historical studies. There is thus a question about whether the cases are accurately measured. Perhaps Aaby et al. only observe relatively severe measles cases and are missing less severe cases in healthier individuals. If this were not the case, then the epidemic would not necessarily be representative of a typical measles epidemic. Guinea-Bissau has high levels of polygamy, which may increase the number of children being infected by measles in the same household concurrently whereas greater spacing between children in other contexts may ensure that only one child is vulnerable at a time. Aaby et al. do not use multiple regression to control for observable confounders, which may mean that their mean differences are biased. However, their emphasis on overcrowding may help to explain declining measles mortality in the first half of the twentieth century.

[18]Woods (2000, p. 321-322) uses Wilson (1905)'s data on Aberdeen to estimate that only 42% of children would have been infected by measles by their tenth birthday. This figure is obviously in stark contrast to those produced by (Hedrich 1930), which suggests that notification may have been incomplete for measles in Aberdeen or that the surveys that Hedrich utilised overstated measles incidence. Given that there were essentially no interventions attempted to control the spread of measles before immunisation, I tend to believe the higher measles incidence figures. These are confirmed by the Foundling Hospital data, despite the institutional context, where 83.3% of children had been infected with measles by age 15-16.

infection before adulthood was not as common in rural, isolated areas (Cliff et al. 1993, pp. 146-57; Black 1964), but the only comparable measles epidemic among British troops in the First World War occurred in the Highland Territorial Division with men from isolated areas in Scotland (Kinnear 1923). Thus, Britain likely had greater measles prevalence due to its small size and integration. Measles cases among soldiers declined dramatically in the United States, Australia and Britain between the First and Second World Wars, which again suggests increasing measles incidence before vaccination (Cliff et al. 1993, pp. 158-9). These numbers are confirmed by serological testing of US army recruits in the early 1960s, which showed very high levels of acquired immunity (Black 1964). As Hardy (1993, p. 49) notes, 'without vaccination there is no hope of controlling the incidence of measles'. Thus, incidence rates appear to have remained high (at least in Britain) until vaccination.

These high incidence rates do not match the pattern of measles mortality rates from the mid-nineteenth century onwards. Figure C.1 shows the national decline in measles mortality in England and Wales from 1841 to 1949 by each five-year age group aged 0 to 15. Measles mortality was more or less stagnant at ages 0-4 and 5-9 before 1915: measles mortality actually increased somewhat in the second half of the nineteenth century in London (Hardy 1993, p. 29). After 1915, measles mortality began to decline. This decline accelerated after 1935 when the first chemotherapies were available to treat respiratory complications from measles such as pneumonia McKeown et al. (1975). These falling measles mortality rates were also present in the United States and other Western countries (Cliff et al. 1998, pp. 326-327). Falling mortality rates before vaccination when incidence was unlikely to be falling suggests that case fatality rates must have been falling to make this change. Back-of-the-envelope calculations of the change in the case fatality rate in England and Wales before 1935 when chemotherapies for measles became available suggests that the case fatality rate fell from 3-4% to 1% during this period, which seems plausible given the reduction in measles case fatality in recent years (Portnoy et al. 2019).

Given the relatively high and stagnant mortality rates from measles in the nineteenth and early twentieth centuries, it is worth briefly discussing the social demographic

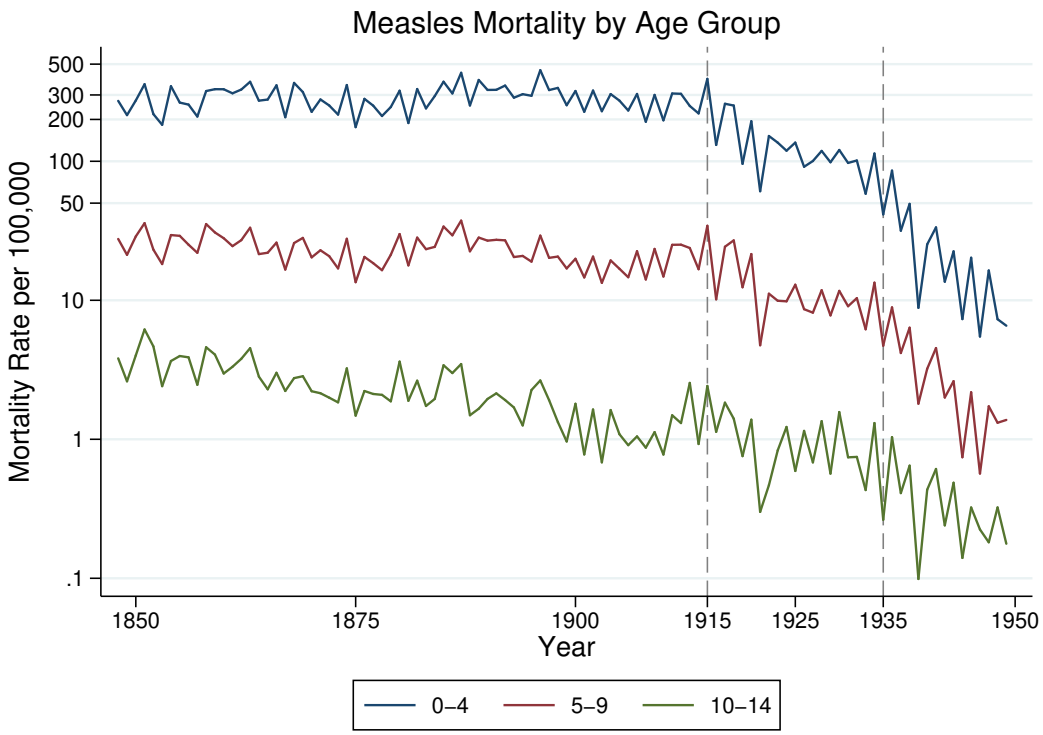

Figure C.1: Measles Mortality in England and Wales, 1848-1949

Notes and Sources: Population data by age group were taken from the Human Mortality Database and interpolated using geometric interpolation (Human Mortality Database 2021). Mortality data by cause up to 1900 were drawn from Davenport (2019). Mortality data from 1901-49 were drawn from the ONS Twentieth Century Mortality Files (Office of National Statistics 2021).

factors that maintained these high mortality rates. Hardy (1993, pp.43-44) argues that poor nursing by working class families contributed to high case fatality rates of measles. Measles was seen as inevitable by most parents, and they often ignored the disease until the symptoms became severe at which point it was more difficult to manage respiratory complications. In addition, high underlying levels of diarrhoeal and respiratory morbidity likely increased the risk of measles mortality (Hardy 1993, pp. 40-41; Aaby et al. 1984). The diarrhoea epidemics of the 1890s likely made the situation worse. Atmospheric pollution could also increase the severity of measles complications and thus increase the death rate. Hanlon (2019) shows that in London fog events, which trapped coal pollution close to the ground, substantially increased the mortality risk from measles, and Hardy (1993, p. 45) suggests that the different seasonal pattern of mortality in London and the Lancashire industrial towns was likely driven by atmospheric pollution. Compulsory primary education was also introduced in 1870 in England, and this likely increased the spread of measles between different households. Evidence from the late nineteenth century suggests that most index (first) cases in a household were school-aged children even if mortality risk was highest for their younger siblings (Hardy 1993, p. 44). Finally, there was relatively little intervention from medical officers of health and other public health officials. Measles was not a notifiable disease under the Infectious Disease Notification Act 1889, and while a few towns made measles a notifiable disease, there is mixed evidence that this influence case fatality rates in any meaningful way. There were debates in the late nineteenth and early twentieth centuries over school closures during measles epidemics and on isolating measles cases in hospitals, but neither of these policies were implemented with enough rigour to have substantially altered measles incidence or case fatality (Hardy 1993, pp. 48-51).

Several factors likely drove down the case fatality rates of measles in England and Wales after 1915. First, McKeown et al. (1975) and others have highlighted the importance of improving nutritional standards in the measles mortality decline. Nutrition did improve during the interwar period with increases in child and adult mean height as well as the sudden change in the speed of maturation, another indicator of improving

nutritional status (Gao and Schneider 2021; Hatton 2014). In addition, to changing nutrition, Cliff et al. (1998, p. 328) and Hardy (1993, pp. 45-48) emphasise an improvement in the quality of nursing care to prevent respiratory complications from measles. Hardy (1993) in particular shows that where disease notification was coupled with information about preventing measles and care for measles patients, case fatality rates were lower. Hardy (1993, pp. 54-55) also highlights the potential importance of social changes following the First World War that could have led to a reduction in measles.[19] Finally, Aaby et al. (1984) emphasise previous respiratory infections and household-level overcrowding, which increases the viral load if multiple children are infected at the same time, as important factors influencing the measles case fatality rate. Previous respiratory infections may be related to nutritional conditions, but household-level overcrowding will be related to housing quality and especially the number of rooms shared by a single household and the number of children in each household. Wilson (1905) also found that the case fatality rate from measles in late-nineteenth century Aberdeen was substantially higher when households shared a single room rather than having more than one room. Of course households sharing a single room will also be poorer and be disadvantaged in other respects, but this relationship still suggests that a reduction in household-level crowding over time could have reduced case fatality rates from measles.

However, despite the factors discussed above, a few puzzles remain. First, if nutrition is related to the case fatality rates, it is surprising that measles mortality rates did not begin to fall until after 1915 since adult stature and child height were increasing long before the interwar period (Gao and Schneider 2021; Hatton 2014) and other studies of nutrition show that nutritional standards had improved dramatically in England by the early twentieth century (Floud et al. 2011; Gazeley and Newell 2015). This includes fat consumption, which Hardy (1993, p. 54) speculates may have reached a threshold around the time measles mortality began to decline. If nutrition were most important in driving the trend in measles mortality, one would have expected measles mortality to fall at an earlier date. In addition, the relationship between overcrowding and measles case

[19]She emphasises the improved diet during the war due to rationing and the new found power of women as they entered the labour force during the war to allocate household resources more fairly.

fatality rates is also less than clear. As Hardy notes, measles mortality did not move in close relation with overcrowding in London (Hardy 1993, p. 55), and it seems likely that improvements in housing quality began long before the First World War. Thus, there is much left to explain in the history of the decline of measles mortality before vaccination. A large scale data project that could look at annual and spatial variation in measles mortality rates and incidence rates after 1915 is needed to answer these questions.

C.2 Whooping cough

Whooping cough or pertussis was a leading cause of death among children in the late nineteenth and early twentieth century. Whooping cough arises from a bacterial infection spread through droplets at close contact. In the initial phase of the disease, it presents very similarly to a common cold, and the eponymous whooping cough only arises in the second stage of the disease after a couple of weeks. Unfortunately, individuals are highly contagious during the first stage of the disease, which means that whooping cough spreads to others before the disease can be accurately diagnosed and the patient isolated.[20] This means that even today antibiotics are largely ineffective against whooping cough with vaccination being the key modern method for controlling the disease: at present there are no treatments to cure whooping cough symptoms. Whooping cough is most fatal in young children with less severe cases in older children, and mortality is often driven by pulmonary complications. Whooping cough does not grant acquired immunity so it is possible for the same individual to be infected multiple times (Halperin and De Serres 2009).

Historically, whooping cough mortality rates began to decline from the 1870s in Britain (McKeown et al. 1975). At its peak in the 1860s, it killed 377 children under five per 100,000 in England and Wales and had declined modestly to 309 deaths per 100,000 children under five by the 1890s. Whooping cough mortality rates were even higher in London at 715 deaths per 100,000 under five in the 1850s, but declined steadily to 421 deaths per 100,000 children under five by the 1890s (Hardy 1993, p. 10-11). The decline in

[20]Modern studies place the basic reproduction number (R_0) for whooping cough at 5.5 (Kretzschmar et al. 2010).

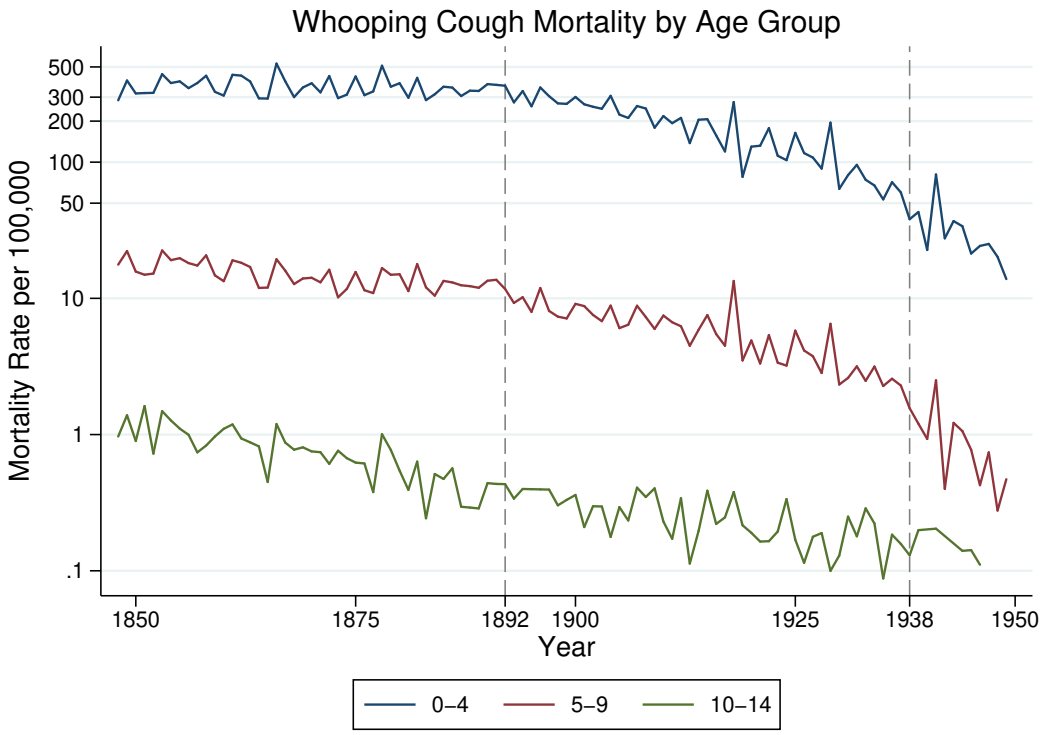

Figure C.2: Whooping Cough Mortality in England and Wales, 1848-1949

Notes and Sources: See Figure C.1.

whooping cough deaths continued into the twentieth century with McKeown et al. (1975) showing that 86% of the decline in age-standardised whooping cough death rates occurred before 1938 when sulfa drugs were introduced to treat complications from whooping cough such as pneumonia. Figure C.2 presents the national trend for whooping cough mortality in England and Wales from 1841 to 1949. This pattern is similar in other Western countries (Chow et al. 2016; Cliff et al. 1998, pp. 336-337).

Estimating precise historical incidence and case fatality rates for whooping cough is difficult for a number of reasons. Whooping cough is variable in its severity, at times similar to the common cold and other times far more deadly, which means reported cases are likely an understatement of the true incidence. In addition, whooping cough was not a nationally notifiable disease in England and Wales until the mid-twentieth century, so reported cases before that time only come from a few localities that required whooping cough notification earlier. Halperin and De Serres (2009, p. 582) argue that in the absence of immunisation, nearly all children contract whooping cough, suggesting that incidence

would have been very high in the past. Where good data on incidence exist in parts of the United States and England and Wales, there is no suggestion that incidence was falling before immunisation even though the case fatality rate was falling dramatically (Amirthalingam et al. 2013; Gordon and Hood 1951). Historical estimates of case fatality rates prior to immunisation present a wide range. In the nineteenth century, case fatality rates in Aberdeen vary between 10.5% to 3.6% in the 1880s and 1890s (Hardy 1993, p. 23). However, the twentieth century saw large declines in case fatality rates. Gordon and Hood (1951) shows falling mortality despite stagnant incidence in the first half of the twentieth century in Massachusetts and New York. More precisely, in Providence, Rhode Island, case fatality rates fell from 1.36% in 1930-34 to 0.24% in 1945-49 (**?**, p. 340). There were also declines in case fatality rates in England and Wales at all ages under 10 between 1944-45 and 1950-53 (Amirthalingam et al. 2013, p. 3). Taken together this evidence suggests that the decline in whooping cough mortality was mostly driven by falling case fatality rather than a reduction in incidence.

In nineteenth-century Britain, whooping cough was understood as a disease of poverty driven by overcrowding and poor medical care and nutrition. For instance, contemporary observers noted that whooping cough in Aberdeen was 'three-and-a-half times more fatal to children living in one-roomed houses than to children living in houses of five or more rooms' (Hardy 1993, p. 17). Crowding would have made it more difficult to isolate and nurse children suffering from whooping cough and would have facilitated the spread of the disease within a household Cliff et al. (1998, p. 338). However, there were not clear associations between declines in whooping cough mortality and improved housing quality across London districts (Hardy 1993, pp. 25-26). Contemporaries were also aware of the importance of good nursing care for whooping cough cases recommending 'a warm and well-ventilated room with a nourishing and easily digested diet' (Hardy 1993, p. 16). Indeed, Hardy (1993, p. 27) does suggest that improvements in nursing care could have contributed to the modest decline in whooping cough mortality in the nineteenth century, but she argues that preventive services 'bore little, if any, direct responsibility for the city's reduced whooping cough mortality in the years before 1900' because whooping

cough was not a notifiable disease and public health officials largely ignored it. One might wonder whether pollution could have mattered, but Hanlon (2019) shows that fog events associated with high levels of atmospheric pollution in London did not affect whooping cough mortality as they did for measles. McKeown et al. (1975, p. 416) note in passing that there may have been a shift in the nature of the whooping cough pathogen over time, and Hardy (1993, p. 27) notes that the different timing of mortality decline from measles and whooping cough may suggest growing resistance to whooping cough over time.

Interestingly, many observers argue that there was a place for nutrition in explaining the decline in whooping cough mortality rates before effective medical interventions. Hardy (1993) notes that children with rickets were more likely to suffer severe complications than other children and given rickets's association with sunlight, crowded and polluted urban environments may have led to greater prevalence of rickets. Any link to whooping cough should be treated sceptically though since the poor were more likely to develop rickets and rickets was also most prevalent between nine and eighteenth months of age, when whooping cough is also naturally most deadly. McKeown et al. (1975), Hardy (1993), Cliff et al. (1998, p. 338) and Halperin and De Serres (2009) all argue that nutrition may have been important for declines in whooping cough mortality, but they are not very precise about exactly what the causal mechanism was. Hardy (1993, p. 21) states that 'although the disease does not seem to be more severe in malnourished children, such children will not withstand a severe attack of it so well'. Cliff et al. (1998, p. 338) argue that improved nutrition may have reduced case fatality associated with complications such as broncho-pneumonia. There is also an emphasis on comorbidities, i.e. children who had recently contracted measles or who were also suffering from chronic diarrhoea may have had higher case fatality rates from whooping cough. Improvements in nutrition may influence these comorbidities and thus influence the case fatality rate of whooping cough indirectly.

C.3 Mumps

Mumps was another common acute childhood disease in the past, though it was far less fatal than measles or whooping cough. Mumps is spread via respiratory droplets between people and is highly contagious with an estimated basic reproduction number between 10 and 12 (Public Health Laboratory Network 2015). Perhaps one-third of mumps infections are asymptomatic, and hosts are contagious prior to the onset of symptoms, making it particularly difficult to manage the spread of the disease with isolation measures. The most common symptom of mumps is parotitis (swelling of the salivary glands), but mumps is also associated with orchitis (inflammation of the testicles), mastitis (inflammation of breast tissue) and temporary hearing loss with orchitis and mastitis being more frequent and serious in post-pubertal children. Before widespread vaccination, mumps was most common among children aged 6 to 7 (Rubin 2014). Infection from mumps grants life-long immunity (Kim-Farley 1993a), which means that mumps showed the same pattern of 2-3 year cyclical epidemics as measles (London and Yorke 1973).

Before vaccination, there is relatively strong evidence that most children in well-connected places had contracted mumps before adulthood (Rubin 2014). Approximately 75% of US military recruits in the early 1960s (pre-vaccination) had positive serum test results for mumps, lower than the rate for measles reported above but still high enough to suggest spread across the United States. There were not statistically significant differences between regions or between urban and rural areas. However, incidence of mumps among US soldiers during the First World War was relatively high at 55.8 per thousand soldiers per year, indicating that in earlier periods the spread of mumps may not have been as complete, especially in the sparsely populated United States (Black 1964). In Britain, mumps mortality was first reported in 1875. Figure C.3 shows that mortality rates from mumps were extremely low, and although there was some decline in the mumps mortality rate for children age 0 to 4 and 5 to 9 from 1875 to 1949, this decline was very small. I have not found any earlier literature that discusses this decline in mumps mortality rates. Finally, it is useful to note that in a recent review chapter on mumps, Rubin (2014) does not mention any nutritional or dietary influence on mumps morbidity or mortality or for

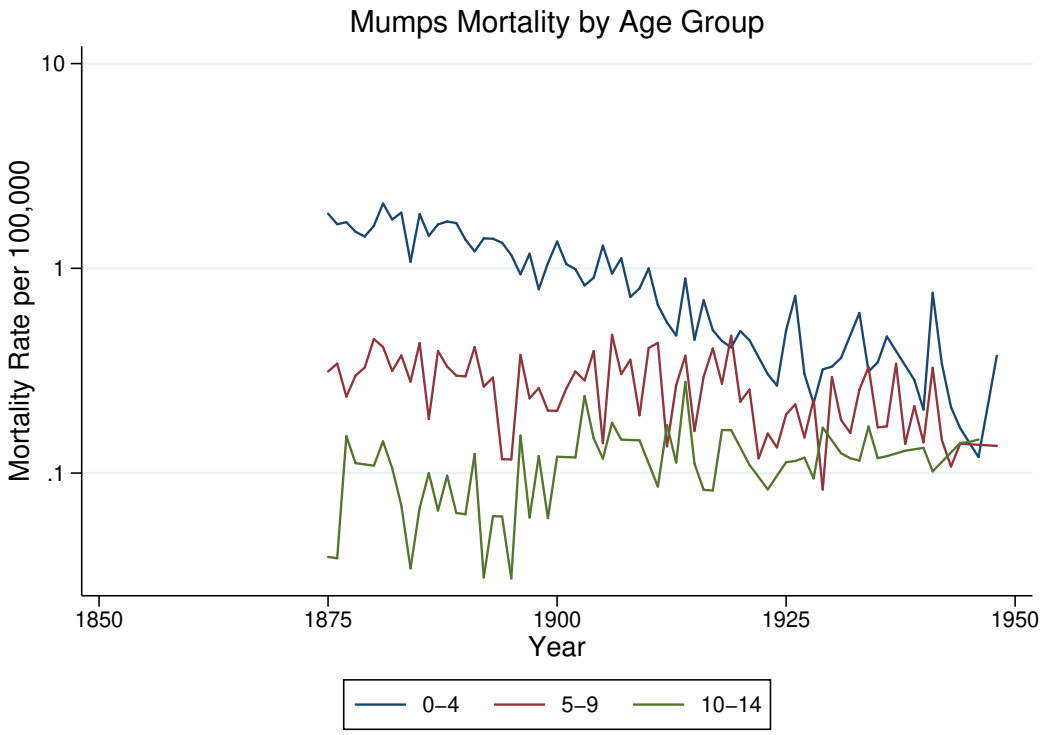

Figure C.3: Mumps Mortality in England and Wales, 1875-1949

Notes and Sources: See Figure C.1.

the complications that arise from mumps infections.

C.4 Rubella

Rubella is a mild childhood infection leading to a rash that is rarely fatal. It is spread via respiratory droplets and is highly infectious with a basic reproduction number of six to seven. Serological surveys in the pre-vaccination era in the United States showed that 80-92% of the adult population had contracted rubella, but the vast majority of cases occurred in school-aged children (Orenstein and Reef 2014). Up to half of rubella cases may be sub-clinical or asymptomatic, but all cases of rubella grant life-long immunity (Kim-Farley 1993b). As is clear, rubella infections among children are rarely problematic. However, rubella infection of women during the first sixteen weeks of pregnancy can lead to miscarriages, stillbirths and children born with congenital malformations. A rubella epidemic in the United States from 1962 to 1965 led to very high levels of fetal and neonatal death. Thus, rubella immunisation was initiated largely to protect other children

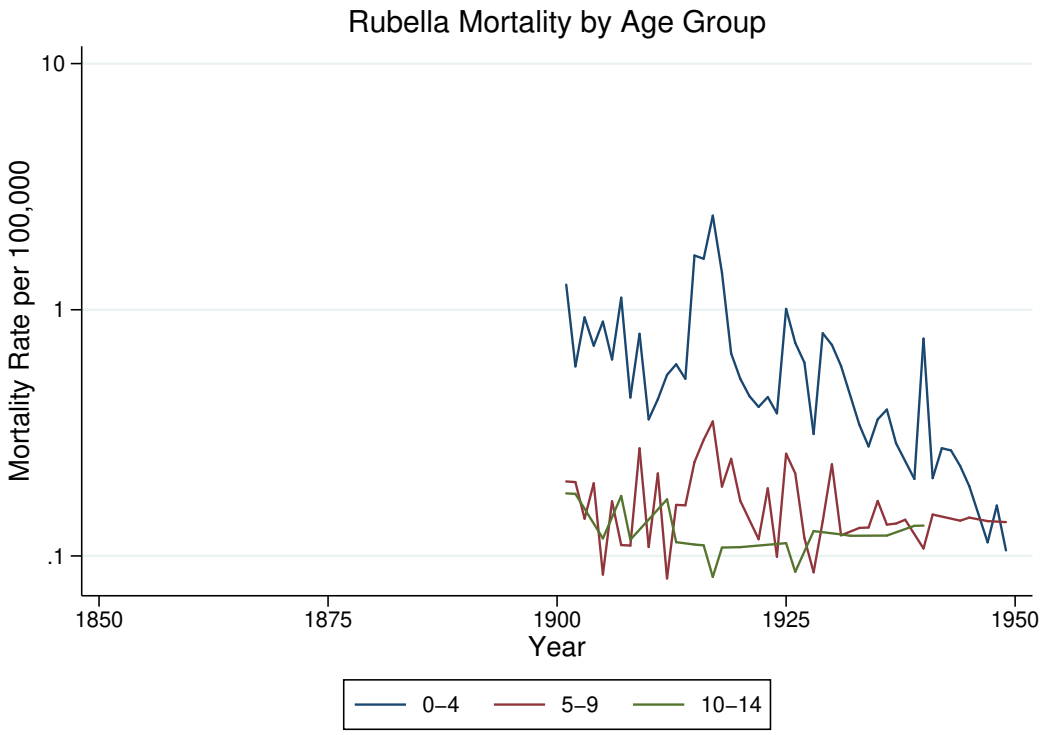

Figure C.4: Rubella Mortality in England and Wales, 1900-1949

Notes and Sources: See Figure C.1.

in utero or in early life.

Figure C.4 reports mortality rates from rubella from 1900 to 1949. Rubella clearly was not an important source of mortality in the early twentieth century with mortality rates below 1 per 100,000 even in the 0-4 age group. While rubella mortality rates for the 5-9 and 10-14 age group remained largely constant in the first half of the twentieth century, there was a decline in deaths caused by rubella in the 0-4 age category. To my knowledge, there is no explanation for this small but dramatic decline in rubella mortality.

C.5 Chicken Pox

Chicken pox (or varicella) is a mild, acute infectious disease of childhood. Chicken pox is spread through respiratory droplets and is highly contagious with a basic reproduction number between 10 and 12. Individuals are contagious before the appearance of the characteristic rash and lesions, which appears after an average of 14 days of incubation. Very few cases are sub-clinical or asympotmatic and the symptoms are striking enough

that it is relatively easy to diagnose. The disease is most common among children aged five to nine, and adolescents and adults are known to have more severe cases and more complications than children. Modern evidence suggests that over 90% of the population is seropositive by age 20. The virus grants life-long immunity to further varicella infections, but the virus can reappear as shingles (herpes zoster) later in life (Gnann Jr 2014; Kampmeier 1993).

Chicken pox mortality was registered in England and Wales from 1875 onwards (Figure C.5). Mortality from chicken pox was very low, although not as low as mumps and rubella. There was a decline in mortality in the age group 0 to 4 beginning in 1900. The higher mortality in this age group contrasts with the modern findings that mortality and morbidity are more severe for adolescent and adult cases. Although some historians have cited evidence that chicken pox case fatality rates were worse among those with low nutritional status (Jones 2003, p. 735), this relationship is no longer reported in the latest edition of *Viral Infections of Humans: Epidemiology and Control* (Gnann Jr 2014). There is, to my knowledge, no explanation in the secondary literature for the decline in chicken pox mortality in the 0 to 4 age group. The most serious complication is pneumonia, but Gnann Jr (2014, p.978) argues that this is most severe for adolescents and adults. Neonatal varicella resulted in death in 30% of cases in the mid twentieth century United States, so perhaps the decline in the 0 to 4 age group is related to changes in exposure of pregnant women to the virus as fertility declined and women were less likely to be pregnant at any given point in time. It may also be a product of the improvement in the classification of causes of deaths as the twentieth century progressed.

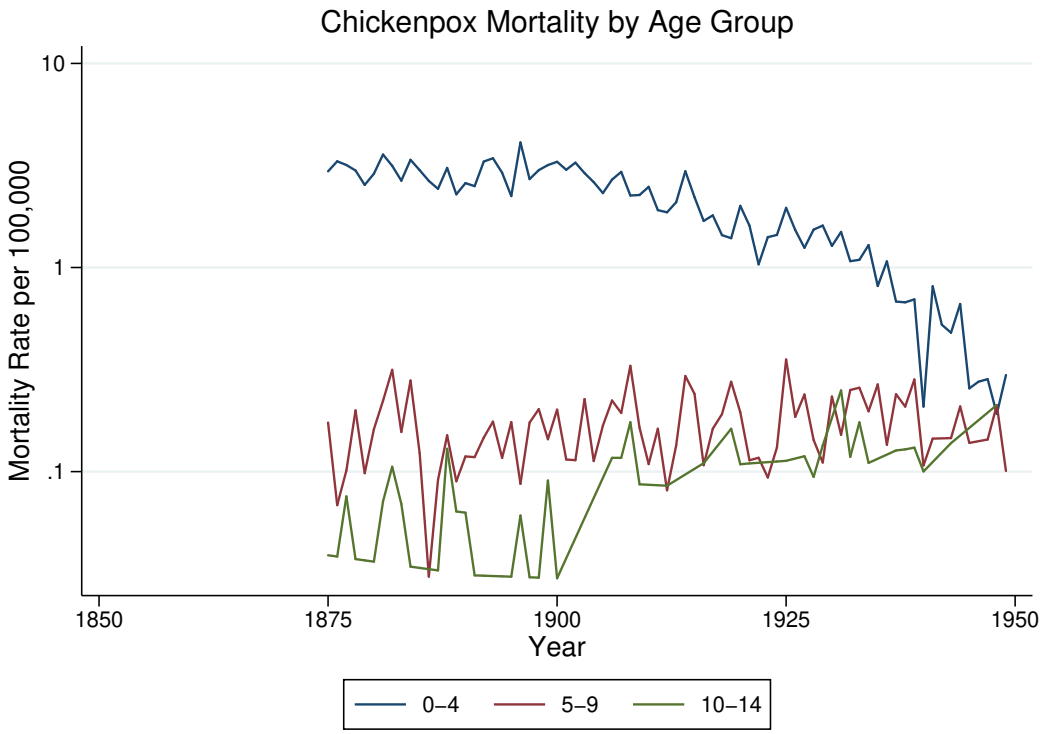

Figure C.5: Chicken Pox Mortality in England and Wales, 1875-1949

Notes and Sources: See Figure C.1.