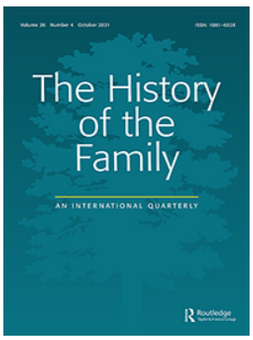

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The effect of nutritional status on historical infectious disease morbidity: evidence from the London Foundling Hospital, 1892-1919

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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.

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Supplemental data for this article can be accessed here.

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 & 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 & 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 & Ó Gráda, 2002). Although 'collective immunosuppression' may have been more important than 'individual immunosuppression' during famines (Mokyr & 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 & 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 & Horrell, 2013; Gazeley & Newell, 2015; Gazeley et al., 2021), there are large margins of uncertainty in the calorie estimates (Kelly & 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, 2013b).

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

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 Conferees (1983, p. 506).

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 & 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 1919. 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 the original strain of COVID-19 was 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 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 & 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 & De Serres, 2009; Kim-Farley, 1993a, 1993b). 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, p. 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 & 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 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 & De Serres, 2009; Hardy, 1993, p. 21; McKeown et al., 1975). There are no clear links in the scientific literature between

Table 2. Characteristics of the five infectious diseases studied.

								Mortality Rate Pre-Decline in E&W (per 1,000,000)		
Disease	Agent	R_0	Transmission	Lifelong Immunity?	Asympt. or Sub-clinical Cases	Historical Case Fatality Rate	Date of Decline in E&W	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 (2014); Halperin and De Serres (2009); Kim-Farley (Kim-Farley, 1993a,b); Moss and Griffin (2014), Orenstein and Reef (2014), and Rubin (2014). For column 7, historical case fatality rates: Hardy (1993, pp. 23, 39); Kampmeier (1993); Kim-Farley (Kim-Farley, 1993a,b). For columns 8–11, see sources for Appendix Figures C.1-C.5.

nutritional status and mumps, rubella or chicken pox (Gnann, 2014; Orenstein & 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.

3.1. 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.[5]

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.

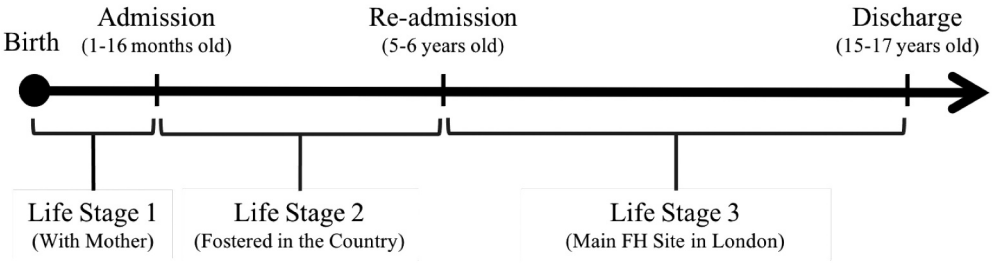

Figure 1. Life stages of a foundling child.

Because mothers could not petition to give up their childto 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 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.[6] 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.[7] 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.[8] 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

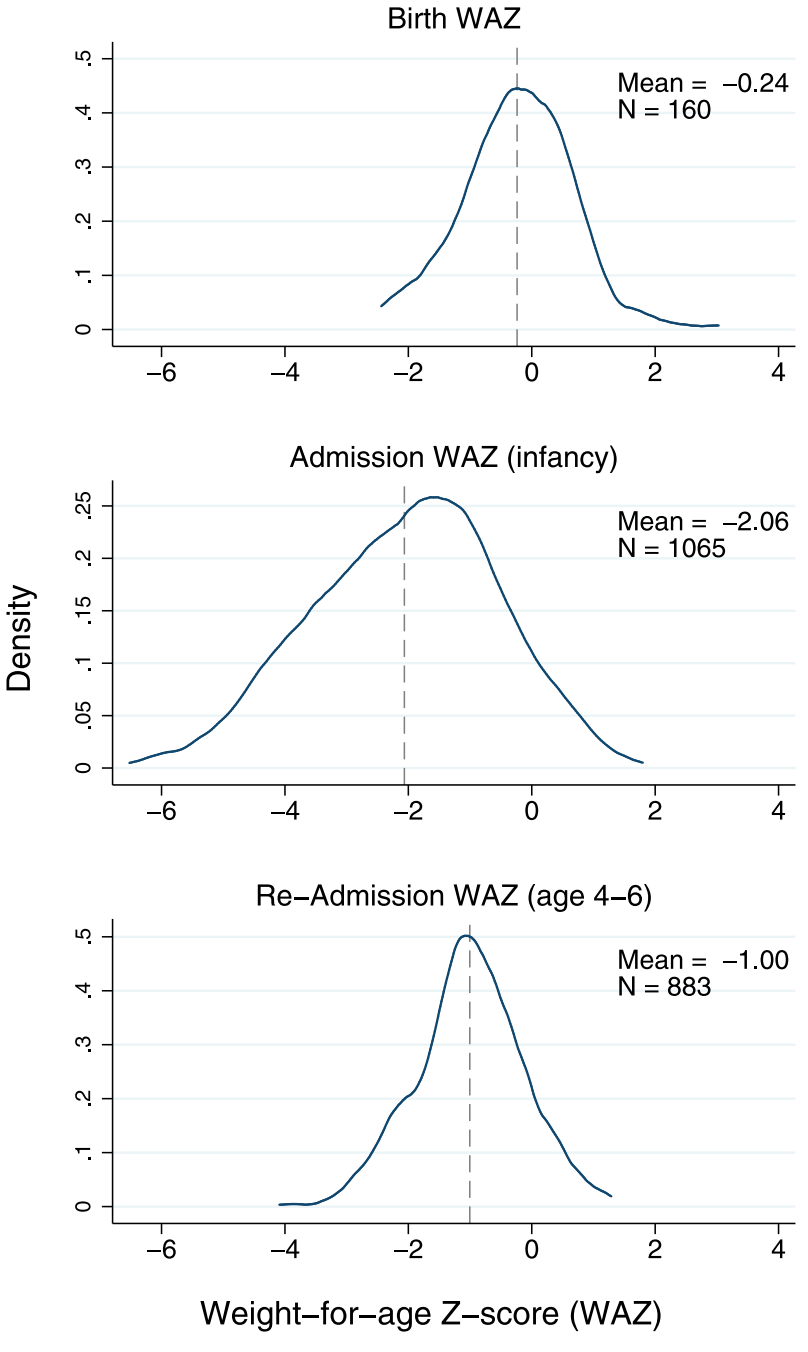

Figure 2. Distribution of weight-for-age Z-scores (WAZ) at different life stages. *Notes*: The vertical dashed line marks the mean. *Sources*: Foundling Hospital Dataset – see Appendix A for precise sources.

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.

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.[9] 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.[10]

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.

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 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.

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

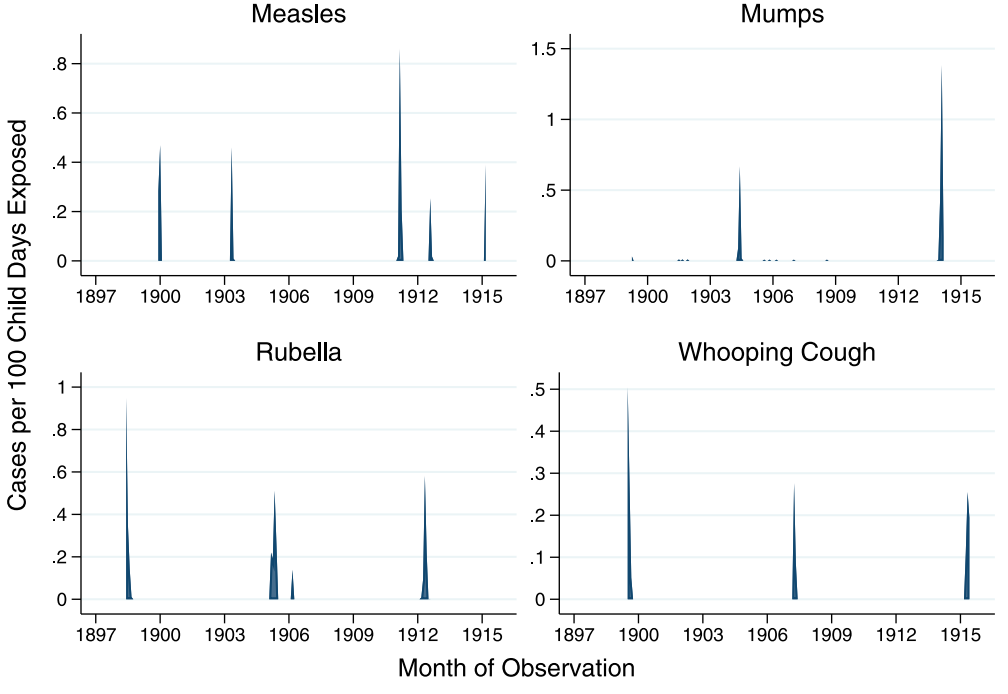

Figure 3. Monthly incidence of epidemic diseases in the foundling hospital 1897–1915. *Sources*: Foundling Hospital Dataset – see Appendix A for precise sources.

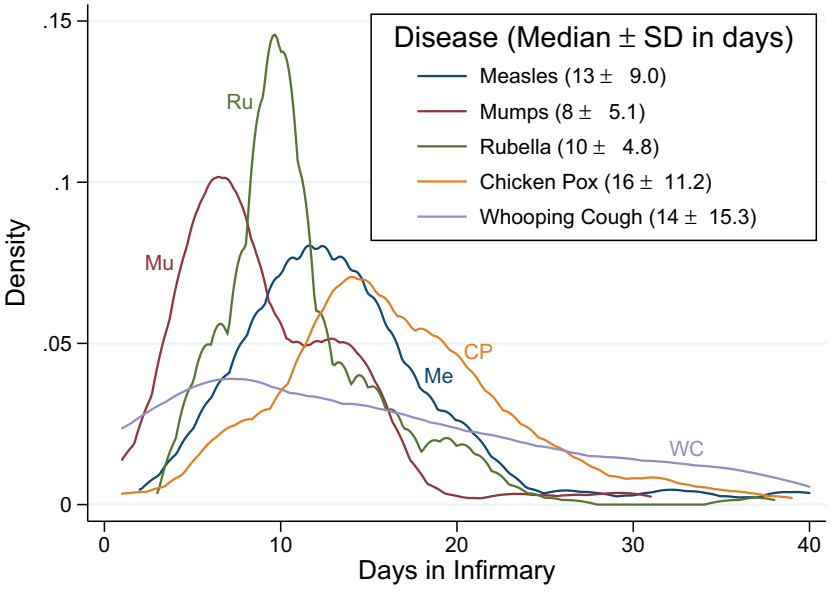

Figure 4. Sickness duration for five infectious diseases in life stage three in the foundling hospital. *Notes*: Sickness duration is truncated at 40 days so that the differences across diseases are clearer, but the full range of durations are employed in the rest of the paper. *Sources*: Foundling Hospital Dataset – see Appendix A for precise sources.

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.[11] 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 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 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

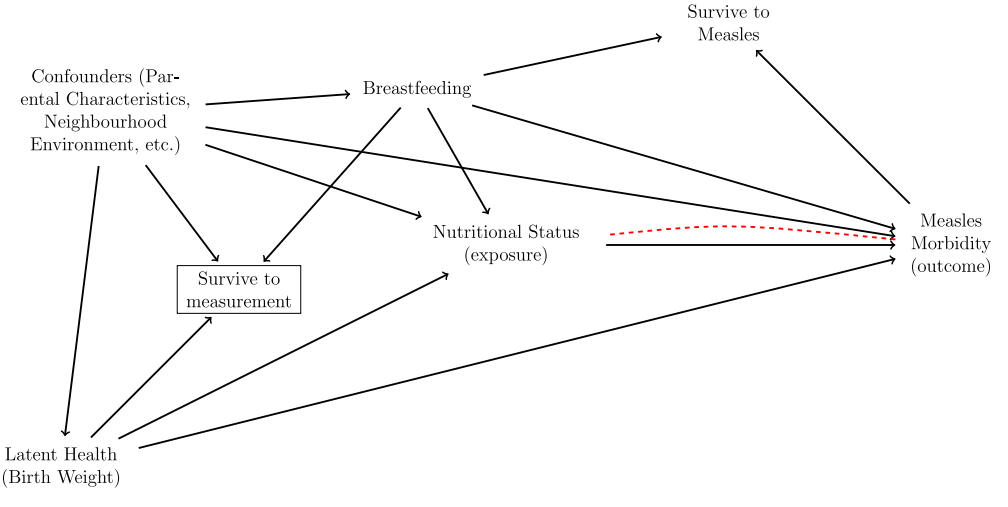

Figure 5. DAG representing the relationship between nutritional status and morbidity in a typical modern study. *Notes*: Names in each DAG represent variables and the arrows show the direction of causation between the variables in the DAG. The exposure is the main independent variable (treatment variable) 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.

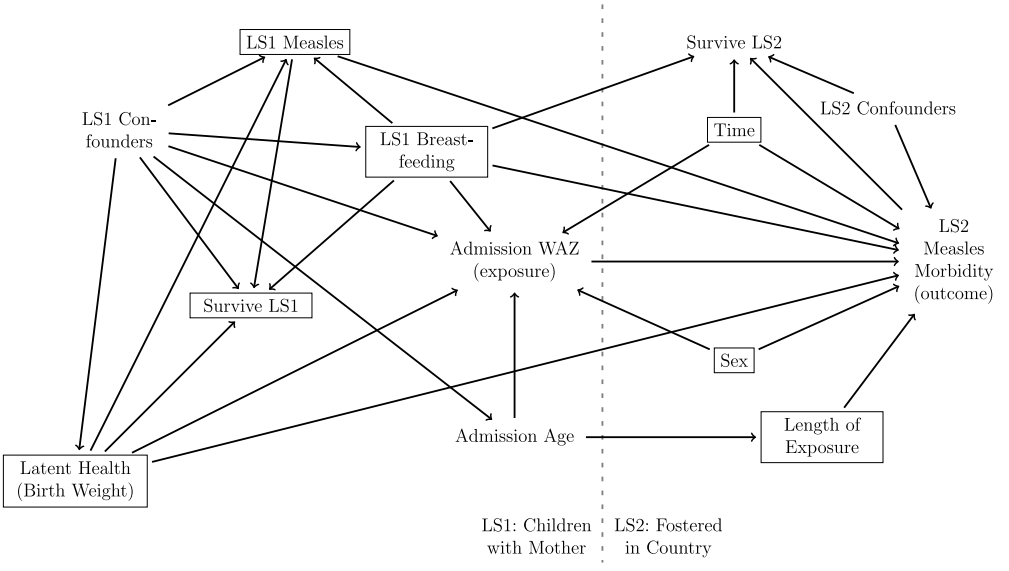

Figure 6. DAG representing the relationship between nutritional status and morbidity in the foundling hospital in life stage two. *Notes*: See Figure 5.

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:

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

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 years), 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.

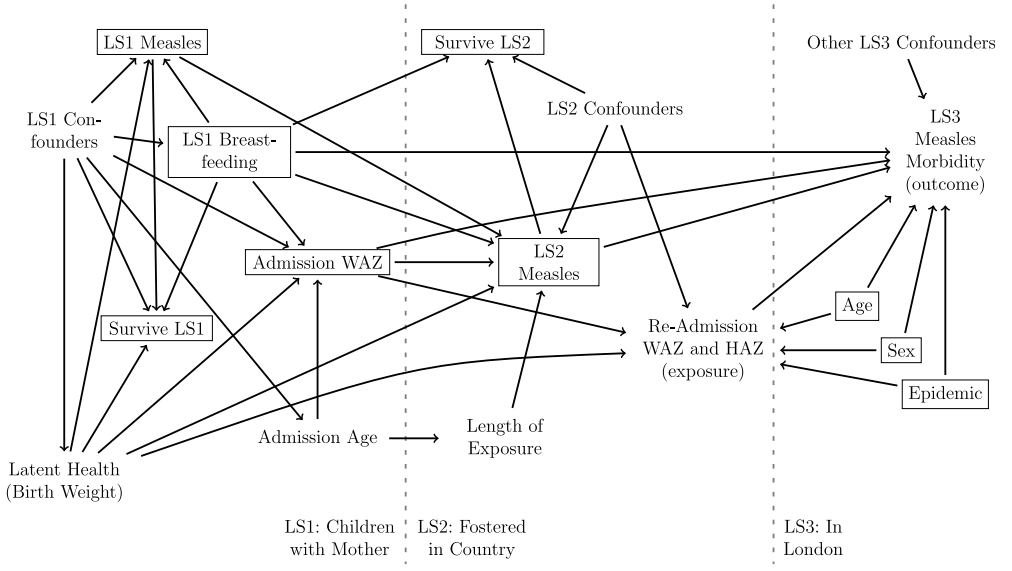

Figure 7. DAG representing the relationship between nutritional status and morbidity in the foundling hospital in life stage three. *Notes*: See Figure 4.

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.[12] 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 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 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.[13]

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

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.[14] The age break down within the wards is not clear nor is precisely which children were assigned to each ward at each point in time, so I assume that children in three age 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.[15] 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.

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.[16] 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 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.

Table 4. Effect of nutritional status on the probability of contracting each disease while in the countryside (life stage two.

	(1) Measles	(2) Measles	(3) Chicken Pox	(4) Chicken Pox	(5) Whooping Cough	(6) 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:						
B	0.244	−0.728	0.169	0.150	−0.024	0.397
	(0.239)	(0.730)	(0.305)	(0.852)	(0.250)	(0.734)
B+	0.264*	0.660	0.246	0.547	−0.067	0.587
	(0.149)	(0.433)	(0.190)	(0.563)	(0.154)	(0.457)
NoB	(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. B means exclusively breastfed; B+ means breastfed with supplementation; and NoB means never breastfed. 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.

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

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.

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 from measles 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

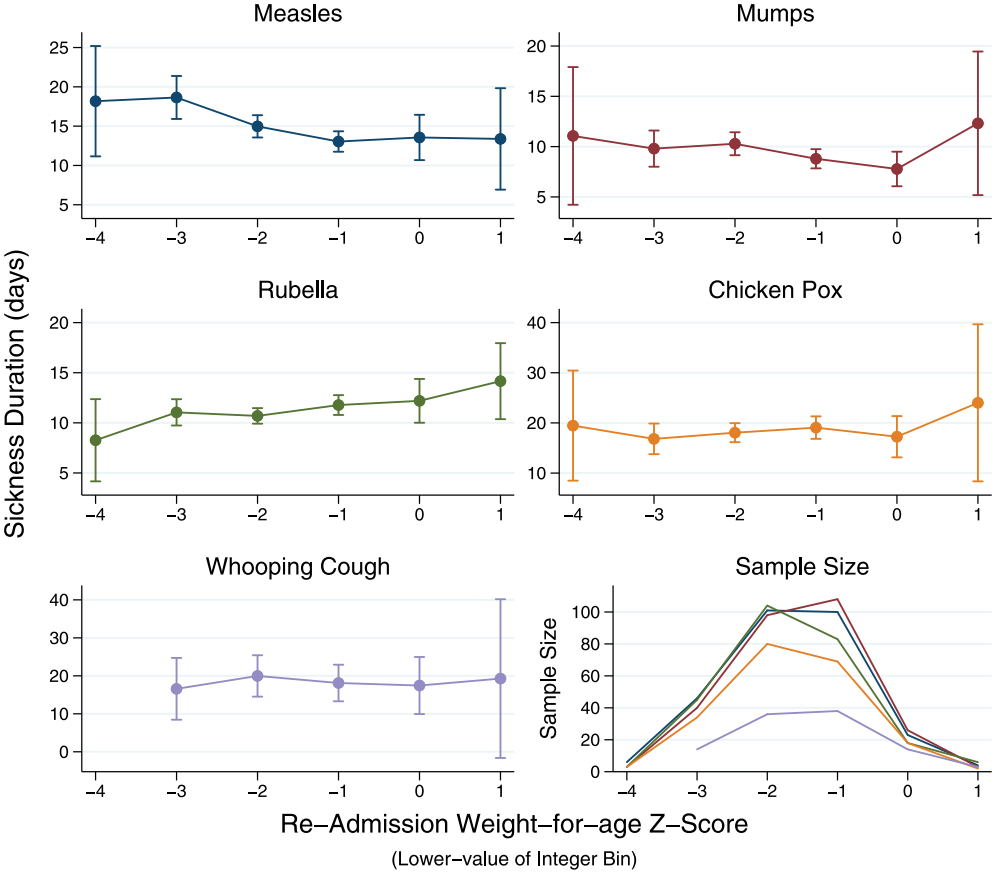

Figure 8. Predicted values of sickness duration by re-admission weight-for-age bins for each disease. *Notes*: Point estimates and 95% confidence intervals are predicted from zero-truncated negative binomial regressions estimating Equation 3 where WAZ is specified as integer-binned dummy variables. The 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.

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 & 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 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 Cliff et al., 1998; Conferees, 1983; Halperin & De Serres, 2009; Hardy, 1993).

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.

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, 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.

Another potential concern is that the sickness events analysed in this study could occur years after the measures of nutritional status were taken. This is true in both life stage two and three, but in life stage three, it is possible to test whether measurement error from the long gap between anthropometric measurement and sickness is attenuating the effect. Appendix Table B.7 re-estimates specification 3 from Tables 5–6 and Appendix Tables B.4, B.5 and B.6, but limits the data to sickness events that started within two years of the child being measured at re-admission. There is very limited evidence of attenuation bias in

these coefficients. The measles, rubella and chicken pox coefficients are slightly larger in the limited sample, but the coefficients for mumps and whooping cough are smaller. The measles coefficient is still highly signficant, but the mumps coefficient lost signficance, likely because the sample size fell so dramatically. The coefficients for the other diseases are still highly insignificant, suggesting that attenuation bias cannot explain the null result in the full sample. Appendix Table B.8 shows the coefficient for measles using different time windows after measurement and confirms that the attenuation bias is likely very small.

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 (Cliff et al., 1998; Bellagio Conferees, 1983; Halperin & 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 & 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 & 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 & Clarkson, 1982; Gordon & Hood, 1951; Hedrich, 1930; London & 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 & Horrell, 2013; Gazeley & Newell, 2015). Adult and child heights had been increasing since the 1870s (Gao & 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 & van Poppel, 2016).

Notes

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).
2. The basic reproduction number is the number of additional people infected by one individual in a fully susceptible population.
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, 2014).
5. However, one should note that all foundling children experienced an adverse childhood experience when they were given up to the hospital. This would only limit the interpretation of my findings if the relationship between nutritional status and disease were different for children who experienced or not adverse childhood experiences. It is impossible to test whether the relationship differed with historical data, and as stated in the introduction, extrapolating from modern studies to history is very difficult. Thus, rather than speculating about potential effects here, readers should consider this potential limitation when interpreting the findings.
6. 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).
7. 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.
8. 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.
9. This record included all in-patients but excluded out-patient treatment.
10. 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.
11. For an introduction to DAGs and their use in economic history and historical demography research, see Cunningham (2021) and Schneider (2020).
12. 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).
13. 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.

14. 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.
15. 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.
16. These deaths yield case fatality rates of 2.3% and 1.7% for measles and whooping cough respectively.

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