Contagion in the Capital: Exploring the Impact of Urbanisation and Infectious Disease Risk on Child Health in Nineteenth-Century London, England

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ABSTRACT

Nineteenth-century London was notorious for overcrowding, poor housing, and heavy air pollution. With a large proportion of its population living in conditions of poverty, diseases flourished as people were increasingly drawn to the industrialising centres of England in search of employment opportunities. Utilising historical documentary and skeletal evidence, this paper explores the impact of increasing urbanisation on non-adult (those aged 0–17 years) health, particularly in relation to exposure to a multitude of infectious diseases in circulation during this time. Focusing on the community of St Bride’s Church, London, it highlights the greater susceptibility of infants and children to risk of severe morbidity and mortality from infectious diseases, particularly amongst the lower classes. When considered against the socio-political, cultural and economic milieu of nineteenth-century London, this reveals how the multi-faceted process of urbanisation exacerbated ill-health, increased susceptibility to deadly infectious pathogens, and ultimately further marginalised its poorest inhabitants.

KEYWORDS

Infant; child; industrialisation; infectious disease; poverty; inequality; St Bride’s Church

Introduction

Urban environments have long been entities of disparity, with lived experiences of such landscapes often dependent on social and cultural positioning, as well as access to resources and amenities. Indeed, cities can provide unrivalled opportunities for advancement, acting as economic, technological and knowledge hubs. Yet, urban centres also often give rise to marked social inequalities between their inhabitants, with a disproportionate number living in conditions of poverty, in turn leading to substantial health inequality (Borrell et al. 2013). London in the nineteenth century is perhaps an archetype of such divided urban spaces; the Industrial Revolution heralded a significant population expansion, with a burgeoning middle class able to take advantage of the new technological age (Beier 1978; Storey 1992). Yet, concurrently, social inequalities between rich and poor continued to widen (Storey 1992; Lindert 1994; Newman and Gowland 2017).
Furthermore, overcrowding, lack of access to sufficient health care, poor sanitation, deleterious working conditions and inadequate nutrition, resulted in a large swathe of London’s population, including infants, children and adolescents, exposed to significant health risks (Newman and Gowland 2017).

With a well-established correlation between health status, disease risk, and the social, cultural and environmental systems in which individuals function (e.g. Goodman, Arme-lagos, and Rose 1984; 1988; Bush 1991; Steckel and Rose 2002), poor communities of nineteenth-century London provided the optimum environment for infectious diseases to propagate (DeWitte et al. 2016; Forbes 1972). Overcrowding and insanitary living conditions were conducive to rapid transmission, and the already poverty-burdened population was particularly susceptible to the heightened disease risks (Beier 1978). Consequently, London residents were subject to numerous epidemic and endemic outbreaks of infectious disease throughout the century, causing contemporary burial grounds to rapidly become overcrowded, as both the general population and mortality rates expanded.

Utilising both historical documentary and skeletal evidence from the burial grounds for St Bride’s Church, Fleet Street, London, this paper explores how increasing urbanisation impacted on non-adult health and survival in nineteenth-century London, specifically addressing how life in the city exacerbated their exposure to infectious diseases.

Growing up on the epidemic streets

Children of the eighteenth and nineteenth centuries, like today, had to face a multitude of illnesses as both their physical form and immune system developed. They were continually exposed to the endemic (often epidemic) diseases of childhood including whooping cough, scarlet fever, measles, diphtheria, and infantile diarrhoea, but also numerous environmentally borne threats to health (Hardy 1992; 1993; Lewis 2002).

The extensive air pollution from smoke and chemicals, by-products of the coal burning and manufacturing processes, promoted respiratory infections such as bronchitis and pneumonia (Hardy 1993; Kirby 2013). The poor sanitation, damp housing, and severe overcrowding of the cities also favoured the spread of a range of infectious ‘filth diseases’ such as typhoid, typhus, and cholera (Hardy 1993; Crawford 2010). In the fifth report of the Poor Law Commissioners from 1839, the spread of ‘fever’ was a problematic epidemic that caused great suffering to the population, and was attributed to the “… filthy, close and crowded state of the houses …’ and ‘… the total want of drainage …’ (Young and Hand-cock 1956, 769). For the inhabitants of London, a large proportion would have been reliant on the river Thames for both their water supply, and a means for disposal of sewage (Hardy 1993). As filtration systems were not sufficiently implemented by water companies before 1840, contamination of a water supply was a threat faced by all, and as such the spread of typhoid fever was endemic within local communities in the nineteenth century (Hardy 1993; Lane 2001; Steere-Williams 2010). Infection could also occur through food, and the significance of milk as a source of transmission was recognised in the 1870s for not only typhoid, but also scarlet fever, tuberculosis (Mycobacterium bovis), and diphtheria (Atkins 1992; Steere-Williams 2010). This form of transmission was of particular risk to infants, as they were more likely to consume milk during the weaning process, and due to the influence of fashionable child care practices that may have driven...
preference for hand-rearing over breastfeeding during this time (Steere-Williams 2010; Newman and Gowland 2017). Typhus was staunchly a disease of poverty, and thrived in conditions of uncleanness, and poor ventilation and personal hygiene within the unsanitary housing of the city dweller (Hardy 1988; Lane 2001). Engels stated ‘It is to be found in the working-people’s quarters of all great towns and cities, and in single ill-built, ill-kept streets of smaller places, though it naturally seeks out single victims in better districts also …’ (1950, 99).

Whooping cough, measles, scarlet fever, smallpox (Variola major), tuberculosis (Mycobacterium tuberculosis) and diseases of the respiratory system are all passed from person to person via airborne droplet infection (e.g. saliva expelled from coughing and sneezing). The efficient transmission of these diseases would require close contact, and the combination of the ever-expanding industrial city, poor hygienic conditions, and insufficient diets exacerbated their rapid spread amongst the urban population. As such, infectious diseases occurred at a higher frequency within the poorer classes due to the severe overcrowding and poor ventilation within households, and the inability to keep afflicted siblings apart (Hardy 1993). For those that survived the initial, potentially fatal, effects of these infectious diseases, secondary complications could also lead to severe morbidity and further risk of mortality. Smallpox was a particularly debilitating and stigmatising disease; marked scarring, particularly to the face, could often lead to ostracism, physical mobility and eyesight could be affected, and many were also vulnerable to further ill health (Oxley 2003). Scarlet fever too is associated with complications such as anaemia, meningitis, kidney disease, and rheumatic fever (Hardy 1993; CDC 2015a). Measles and whooping cough could leave the individual weakened and vulnerable to further infectious diseases or malnutrition (Ashby and Wright 1896; Hardy 1993; CDC 2015b; WHO 2015), and tuberculosis (also referred to as phthisis, ‘wasting’, and ‘consumption’) was a chronic condition that could lead to a slow and debilitating decline in health. Scrofula, referring to a tubercular infection of the lymph nodes of the neck causing them to swell, was also a common form of tuberculosis. However, it was the pulmonary manifestation of this disease that caused the highest fatality rates (Lane 2001).

Thus, the multitude of infectious diseases that ravaged urban centres such as London produced a melting pot of morbidity and mortality risk for the entire population. Reported population mortality rates of selected diseases from 1844 to 1853 (in order of highest frequency first) revealed that tuberculosis, pneumonia, bronchitis, typhus, convulsions, scarlatina, and whooping cough were the most frequent causes of death in London (Smith 1854). Tuberculosis averaged 60,000 deaths a year between 1839 and 1843 in the industrial centres of this time (Lane 2001), and many who contracted smallpox would not survive, with 755 fatalities per million in London alone in 1838–1842 (Oxley 2003). Fatalities from infectious disease occurred up to four times more frequently in towns than in the country (Engels 1950) and it was observed by Mayhew in 1849 that ‘As season follows season, so does disease follow disease in the quarters that may be more literally than metaphorically styled the plague-spots of London’ (1849, p. 4). So at no point in the year were urban inhabitants free from the unremitting risk of infectious disease.

Ultimately, it was the poorest members of society that fared worst alongside declining urban conditions, and it was their plight that became both highly politicised and often vilified in the nineteenth century (Shields Wilford 2018). In his report on the sanitary condition of the labouring population in 1842, Chadwick commented that ‘Immediately
behind rows of the best-constructed houses in the fashionable districts of London are some of the worst dwellings, into which the working classes are crowded; and these dwellings, by the noxious influences described, are the foci of disease.’ Albeit often guided by flawed concepts regarding the root causes of specific diseases, the impact of urbanisation on population health of London at this time was frequently the subject of satirical political messages as published in Punch magazine. Notably ‘A Court for King Cholera’ in 1852, and the image of Father Thames introducing his offspring – diphtheria, scrofula, and cholera – to the fair city of London in 1858.

The extent to which the above conditions of urbanisation and rampant infectious disease transmission impacted specifically on child health and mortality will now be explored via historical documentation and skeletal evidence from the St Bride’s burial grounds.

**Documentary evidence**

Located within Central London, St Bride’s Church on Fleet Street has a long history of ministering the population of the ‘Farringdon without’ ward. Dating back to Roman times the site has seen seven successive churches built on the site, the first of which was built in the 7th century (Huda and Bowman 1995, 135). The modern church standing on the site today is a replica of Wren’s original design, which was destroyed by an air raid in 1940 (Scheuer 1998, 100). Subsequent excavations at the site following bomb damage to the church, and later urban redevelopment, have provided skeletal evidence of population health from the seventeenth to nineteenth centuries revealing the experiences of those living in the parish at this time (Kausmally 2008). As such, individuals buried in St Bride’s Parish are split between the crypt and the two external cemetery grounds – the Upper Ground and Lower Ground cemeteries (Scheuer 1998, 103), with individuals from across the social strata represented (Shields Wilford 2018).

Thus, St Bride’s was selected as a focal point for this study based on the availability of skeletal data following excavation of its lower burial ground, and contemporaneous burial records (see below) from its lower and upper burial grounds, and vaults. The documentary evidence from burial records will first be explored to determine the reported impact of infectious diseases on child mortality within the parish during the early-mid nineteenth century.

Name, age at death, date of death, abode, burial location, and cause of death for all interments at St Bride’s Church, Fleet Street between 1820 and 1850 were transcribed from burial records available on Ancestry (2019 – see Table 1; n = 4,228). Approximately 40% of the total sample were classed as non-adults (less than 17 years of age, n =

| Area       | Burial Fees | Status  | Non-adult sample size | Adult sample size | Total  |
|------------|-------------|---------|-----------------------|-------------------|--------|
| Lower Ground | ~1s to 19s | Low     | 1112                  | 1236              | 2348   |
| Upper Ground | ~£1 to £2 | Low/middle? | 496                     | 1093              | 1589   |
| Vaults     | ~£2 to £7  | High    | 73                     | 218               | 291    |
| All        | –           | Mixed   | 1681                   | 2530              | 4228   |

Burial fees for each burial area from Cauch (1840, 33).
1,681), and approximately 60% were classed as adults (more than 18 years of age, \( n = 2,530 \)). Burial location (lower ground, upper ground, and vaults) was used to establish crude status groups based on reported burial fees for St Bride’s, Fleet Street (Table 1; Cauch 1840, 33). Vault burials are therefore suggestive of wealthier individuals, whereas those in the Upper Ground could be representative of more variable financial status. Conversely, the Lower Ground was formed due to the congestion and overcrowding of the original churchyard (Upper Ground) (Miles and Conheeney 2005). It was the cheapest burial place in the parish, with densely packed coffins, indicating that the majority of the population buried there were likely of lower status (Kausmally 2008). In addition, there are multiple entries in the St Bride’s Burial Records of individuals from Bridewell workhouse and Fleet Prison, both of which were in the locality (Miles and Conheeney 2005; Kausmally 2008). Using information provided by entries for the cause of death, individuals dying from infectious diseases (tubercular diseases, measles, whooping cough, scarlet fever, typhus, typhoid, dysentery, influenza, cholera, thrush, smallpox, croup, rheumatic fever, and erysipelas) were identified in the burial records.

**Figure 1** shows that non-adults were more vulnerable to infectious diseases, with 34.8% of the total non-adult sample dying from an infectious disease, compared to just
16.8% of the total adult sample. This may be due to children being more susceptible to contracting infectious diseases, or may simply be representative of children being more likely than adults to die as a result of complications from them.

Children appeared to be at greatest risk of morbidity and mortality from infectious disease from birth to approximately 7 years of age (Figure 1(a)). This peaks between 1 and 3 years of age, where infectious diseases in this age category accounted for approximately 17% of all non-adult deaths (Figure 1(a)). In contrast, there appears to be no increased risk of death from infectious disease across the adult age categories (Figure 1(b)).

The human body is highly labile, able to change in response to environmental factors (Bogin and Rios 2003; Clukay et al. 2018). Furthermore, during the early years of growth and development (infanthood and childhood) the human body is going through rapid physiological changes (Lewis 2007, 60). Consequently, due to this expeditious growth trajectory and immature immune system, non-adults are particularly susceptible to adverse onslaughts (Goodman and Armelagos 1989; Perry 2006). Widely held as sensitive barometers of overall population health, non-adults, especially those in infanthood and early childhood, are thus typically the most vulnerable to infectious diseases (Eisenberg et al. 2017; Degani 2006). Identifying an increased morbidity rate in those aged 1–3 years for the St Bride’s individuals is therefore not unexpected. During the nineteenth-century, many infectious diseases were found to disproportionately affect the very young, with those less than two years of age particularly susceptible to whooping cough (Smith 1854), those between 1 and 5 years of age to scarlet fever (Hardy 1993), and those aged 1–2 years of age to measles (Hardy 1992; 1993).

A higher risk of death from infectious disease is also evident in non-adults from lower status groups. Nearly a quarter (24%) of all non-adult deaths in the St Bride’s Burial Records can be ascribed to children dying from infectious disease and being buried in the Lower Ground, suggesting they came from poorer backgrounds (Figure 1(a)). However, a Chi-square test revealed no statistically significant differences between the three burial groups ($X^2 = 4.109, p = 0.128$). Again, this disparity between the three burial groups is most evident from birth to 7 years of age, and particularly between 1 and 3 years of age (Figure 1(a)), although this too was not found to be statistically significant ($X^2 = 2.224, p = 0.329$). Given the inherent vulnerability of non-adults to disease, acting alongside factors such as overcrowded housing, poor nutrition and sanitation, and a fundamental lack of healthcare (Newman and Gowland 2017), an increased exposure to infectious agents and/or reduced immune status of the individuals within lower status groups is not surprising. Social and health inequalities rendered poorer families at greater risk of the burden of contraction of infectious diseases, and resultant child morbidity and mortality (Newman 2021), further fuelling the cycle of poverty in these communities.

When the infectious disease category is broken down into more specific causes of death, it is clear that non-adults faced severe morbidity and mortality from a greater array of pathogens (Figure 2(a)), with the top five diseases bringing the highest risk of premature mortality being whooping cough (25.3%), tuberculosis (19.5%), measles (16.9%), smallpox (13.2%), and scarlet fever (12.3%). Whereas an overwhelming proportion of adults died from tubercular diseases (namely consumption, likely referring to pulmonary tuberculosis)
and to a lesser degree cholera (Figure 2(b); 75% and 11.7% of all adult deaths due to infectious diseases, respectively). This makes sense, as the diseases faced by children were compounded, a result of both exposure to diseases usually associated with childhood (e.g. scarlet fever and measles) and the epidemic diseases exacerbated by the urban environments in which they resided. Conversely, the adult population represented in this burial sample had likely survived through these infectious diseases in circulation during their own childhood, so were perhaps more likely to succumb to environmentally borne and socially propagated infectious agents such as tuberculosis, typhus, typhoid, and cholera.

Skeletal evidence

The infectious diseases described above were all propagated and extensively spread through the detrimental urban environment so that ‘Hour after hour, day after day, year after year, the negative effects of these disamenities accumulated, carving their influence on the human body’ (Oxley 2003, 9). However, despite their bodily impact, detection of specific infectious diseases on human skeletal remains can be problematic; therefore, their presence within skeletal collections from 18th-19th century urban centres, and archaeological populations in general, is not easily visually assessed.

Assessing pathological changes in non-adults is considered to be particularly challenging (Lewis 2018), once again a result of their immature immune systems and a higher degree of bone turnover (Lewis 2000, 2002, 2018; Satterlee Blake 2018). As the most sensitive members of past societies (Goodman and Armelagos 1989) non-adults can rapidly express skeletal changes as a result of pathological conditions. Macroscopic assessment of non-adult pathology is most commonly employed (Satterlee Blake 2018) and some conditions/diseases are clearly identifiable and diagnosable despite the young age and immature development of these individuals (e.g. tuberculosis, leprosy and congenital syphilis. For a full discussion of the skeletal manifestations of these conditions in non-adults see Lewis 2017). When a specific condition is suspected, diagnosis is more feasible if distinctive skeletal changes follow a clear pattern in line with modern clinical knowledge.
of the disease process (e.g. Hutchinson’s incisors and mulberry molars in cases of congenital syphilis). However, the majority of disease processes (including most infectious diseases, metabolic disturbances, psychosocial stress, and sometimes even trauma) are more liable to producing ‘non-specific’ skeletal changes, more commonly presenting as periosteal new bone formation (NBF) (Lewis 2018). Indeed, many conditions leave no, or very few skeletal traces, instead primarily affecting soft tissues; e.g. plague, cholera, smallpox and rubella (Ortner 2008). Furthermore, acute conditions may result in pathological lesions being quick to heal, or result in rapid death, meaning no skeletal lesions were able to manifest (Ortner 2008). As can be seen in Table 2, of the five most common causes of non-adult mortality seen in the St Bride’s burial records, only two may result in potentially diagnostic skeletal manifestations, and even for these in a very small percentage of the overall number of individuals affected.

Therefore, many of the pathological interpretations derived from osteological analysis of non-adults rely on distinct patterning of NBF. However, differentiating between pathological NBF and NBF associated with normal growth is highly problematic (Lewis 2017); healthy NBF, associated with normal growth, is known to appear in an almost indistinguishable way to pathological NBF (be it caused by infectious, metabolic or traumatic onslaughts), as the process by which the bone forms is identical (Lewis 2000). Thus, the presence of NBF remains non-specific in nature and multiple causes could be attested (Nade 1983), such as meningitis, rubella, measles, smallpox, puerperal fever and diarrhoeal diseases (Anderson and Gonik 2011; Lewis 2017).

Table 2. Overview of potential skeletal manifestations of specific infectious diseases.

| Infectious disease | Diagnostic skeletal manifestation in non-adults |
|--------------------|-----------------------------------------------|
| Measles            | None reported.                                |
| Scarlet Fever      | None reported.                                |
| Smallpox           | *Skeletal changes associated with Osteomyelitis variolosa:*  
|                    | - Periosteal new bone formation/destructive osteomyelitic changes to skeletal elements due to inflammation of joints.  
|                    | - Can be unilateral or bilateral.  
|                    | - Ankylosis of affected joint(s) common.  
|                    | - Commonly seen in elbows, wrists, and ankles, often multiple sites.  
|                    | % of infected individuals developing skeletal changes:  
|                    | - 2–5% of children aged 9 mths-14 years with smallpox (Davidson and Palmer 1963)  
| Tuberculosis       | *Skeletal changes associated with destructive lytic lesions:*  
|                    | - Destruction of vertebral bodies leading to spinal collapse (Pott’s disease).  
|                    | - Destruction of ends of bones associated with joints. May be unilateral or bilateral. Commonly affects the hip, knee, and ankle joints.  
|                    | - Rarely causes destructive lesions of the bones of the cranial vault.  
|                    | % of infected individuals developing skeletal changes:  
|                    | - Various reports, from 1% to 3%, and 3% to 5%. Figure may be higher in children, potentially ~12% of cases (Lewis 2017).  
| Whooping cough     | None reported.                                |

Based on the infectious diseases most commonly seen in the documentary sample. Information compiled from Ortner (2003) and Lewis (2017) unless otherwise stated.
Furthermore, NBF is often correlated with metabolic disorders, such as vitamin C and D deficiency (Schultz 2001; Lewis 2007; Dawson 2017), hypervitaminosis A (Fujita, Lo, and Brindle 2017), and anaemia (Wapler, Crubézy, and Schultz 2004; Rivera and Lahr 2017). Whilst many osteological studies have considered the diagnosis and skeletal manifestations of metabolic disturbances (e.g. Brickley and Ives 2006, 2008; Besbes et al. 2010; Lewis 2010; Ortner and Ericksen 1997; Ortner and Mays 1998), it is evident that these conditions can often be a product of pre-existing conditions, such as parasitic infections, and/or lead to a reduction in the individual’s immune function, leaving them susceptible to other infectious agents. For example, it is suggested that vitamin D plays an important immunoregulatory role, with reports that children affected by rickets often experience repeated episodes of infectious disease, including an increased susceptibility to the common cold virus (Holick 2006; Shin et al. 2010). The synergistic and compounding effects of metabolic disturbances and infectious disease thus often result in non-specific changes to the skeleton which are unable to be precisely identified, but their presence may, in turn, indicate populations that were more susceptible to severe morbidity and mortality in response to infectious diseases agents.

Thus, while we know based on documentary evidence that approximately 36.3% of non-adults interred in the lower burial ground of St Bride’s, Fleet Street, London between 1820 and 1850 died from infectious diseases, it is unlikely that these high rates of infectious disease would be reflected within macroscopic skeletal analysis alone. Excavations of the Lower Ground at St Bride’s resulted in the recovery of 606 individuals, 544 of which were analysed, and data made available by the Museum of London (WORD database 2020). A total of 175 individuals were identified as non-adults, with data pertaining to skeletal pathology available for 173 of them, accessible via the Wellcome Osteological Research Database (WORD database 2020). As can be seen in Table 3, only a very small number (6/173: 3.47%) exhibited skeletal evidence for a possible specific infection, being tuberculosis for the vast majority of cases. Yet, approximately 18% showed evidence of non-specific NBF, 7% for vitamin C deficiency, 8% for active vitamin D deficiency, and 27% for cribra orbitalia. Cribra orbitalia is a condition often associated with anaemia, particularly in reference to unhygienic environments, dietary deficiencies, and/or pathogen load (Stuart-Macadam 1991; Walker et al. 2009; Oxenham and Cavill 2010). It has also recently been suggested to have a potential association in some cases with respiratory infections (O’Donnell et al. 2020). While a high prevalence of all of these conditions is frequently seen in eighteenth to nineteenth-century skeletal

Table 3. Prevalence of pathology seen in the St Bride’s Lower, London, skeletal assemblage.

| Pathology                        | N | n present | CPR (%) |
|----------------------------------|---|-----------|---------|
| Periosteal new bone formation    | 173| 26        | 17.69   |
| Cribra Orbitalia                 | 114a| 31b       | 27.19   |
| Vitamin D deficiency             |    |           |         |
| Active                           | 173| 14        | 8.09    |
| Healed                           | 173| 11        | 6.36    |
| Total                            | 173| 25        | 14.45   |
| Vitamin C deficiency             |    |           |         |
| Total                            | 173| 12        | 6.94    |
| Possible specific infection      | 173| 6         | 3.47    |

Data retrieved from the Wellcome Osteological Research Database (WORD 2020). CPR – crude prevalence rate, calculated as percentage of the sample of non-adults with skeletal elements present for analysis.

aBased on number of individuals with at least one orbit present for analysis.
bBased on the number of orbits exhibiting a score of more than 2 (using Stuart-Macadam 1991).
assemblages from London, it is impossible to suggest whether the non-specific lesions had any association with infectious diseases in the absence of specific skeletal evidence in these cases. However, their high prevalence can by proxy identify populations that may have increased susceptibility to severe morbidity or mortality from infectious diseases due to a weakening of the immune system and under nutrition. Prior analysis of multiple low status skeletal assemblages of infants from post-Medieval London showed comparable results, with limited evidence of specific infection, but a high prevalence of general non-specific skeletal pathology, indicative of overall poor health (Hodson and Gowland 2020). Thus, when combined with historical documentation, such as the St Bride’s burial records, skeletal data becomes an essential component to the overall assessment of the bodily impact of urbanisation on non-adults. Here, seemingly small percentages of discrete skeletal conditions become signals of wider population health issues.

**Urban poverty: past and present**

Children growing up in nineteenth-century urban centres were at an extremely high risk of mortality from infectious diseases, particularly whooping cough, tuberculosis, measles, smallpox, and scarlet fever for those interred in the burial grounds of St Bride’s Church. The fear surrounding childhood diseases at this time was not reserved for the poor, ill health struck regardless of class, and in the pre-antibiotic era its course was unpredictable and indiscriminate (Newman 2021), and would only be curtailed by sanitary reform and scientific advancement.

It was not until the epidemic of 1853–1854 that the link between the deadly cholera and contaminated water supplies was made by John Snow when he identified the Broad Street pump in Soho, London, as the root of 344 deaths in its locality in the short space of four days (Lane 2001; Chan, Tuite, and Fisman 2013). With the growing awareness of the detrimental effects of faulty sewerage systems and poor water supplies, improvements to public health systems meant that cholera could be controlled more efficiently in the later outbreak in 1865. The clearing of cesspools, repairs to drains and improvements to sewerage, and general improvement in water supplies restricted the type of environment in which water borne disease could flourish (Hardy 1993; Lane 2001). As such, typhoid began to decrease in prevalence by the twentieth century, and typhus too diminished due to improvements to sanitation brought about by sanitary reform from the mid-nineteenth century (Hardy 1988; Lane 2001).

Despite the early promise of controlling smallpox by the introduction of an inoculation in 1720 by Lady Mary Montagu, many were wary of this process (Porter 1995; Fine 2014). The requirement for the child to be infected with smallpox (via the pus from a smallpox pustule from a sufferer of a milder strain of the disease) to bring immunity, and the prospect of disfigurement or fatality that accompanied this procedure, was considered to be risky. Instead, many parents preferred to follow their own methods of amateur home inoculation (Bayne-Powell 1939; Burnett 1984; Lane 2001), and anti-vaccination movements hampered advancement up until the late 1800s. Consequently, the elimination of smallpox did not make substantial progress until 1797, with the introduction of the smallpox vaccination by Dr Jenner, and following the Vaccination Acts that first made it freely available in 1840, then compulsory in 1853 (Oxley 2003).
Therefore, while the nineteenth century saw rampant outbreaks of infectious disease in London, and pervasive morbidity and mortality amongst its population, this century also wrought significant advancements in public health. This led to improvements in working and living conditions felt long into the twentieth century. Chadwick (1842) believed that improvements to sanitation, drainage, and clean water supplies would improve health and would be of benefit to all of society. However, there existed an aversion by the upper classes to the plight of the lower classes, with the belief that many existed in this position due to deficiencies in their moral character (Booth 1889; Gowland 2018; Shields Wilford and Gowland 2019). This inequality and class aversion meant that issues of sanitation did not readily improve. Over 100 years after Booth first produced his poverty maps of London, many areas still remain marked by social and health inequality (Dorling et al. 2000).

Poverty is a complex concept, and though primarily associated with financial wealth, or lack thereof, it is experienced in a variety of ways due to insufficient resources to meet basic needs of living and access to adequate health care (JRF; What is Poverty 2020). It is, therefore, also the most predominant factor regulating morbidity and mortality today (Feinstein 1993). For those of nineteenth-century London, infectious diseases perhaps posed the greatest urban health risk, yet the interplay of this high pathogen burden with overcrowding, poor sanitation, and compromised or limited nutrition led to further unmanageable hardship, poverty and ultimately ill-health. Present-day urban landscapes still typically maintain inflated rates of poverty, overcrowding and infectious disease; though markedly improved since nineteenth-century London, death as a result of factors such as air pollution, tuberculosis, and cancer are still highest within the capital (Public Health England: TB in the UK 2013; NHS London – A Call to Action 2013; Western and Bekvalac 2020).

Indeed, whilst infectious disease is in itself a health and/or mortality risk, these are often intensified, enhanced and perpetuated by a cycle whereby housing, nutrition, health care and other underlying health conditions all play a part in exacerbating or minimising further disease risk (Katona and Katona-Apte 2008). For those where the risks are highest, often the poorest members of communities, it is often a result of a paucity or limited access to one of these factors, resulting in an intrinsic correlation between socio-economic and health disparities (Prior and Manley 2018; Adler and Newman 2002). In terms of child health within urban centres, early life exposure to adverse conditions can impact on immunity and the regulation of bodily responses to infection (Babones 2008), forming an integral link between immune function and social position. While the risk of endemic and epidemic diseases may have been ubiquitous in nineteenth-century London, the data from the St Bride’s Church burial records and Lower Ground skeletal collection serve as a reminder that the burden of child morbidity and mortality was not equally felt by all.

Conclusion

Historical documentary and skeletal data from the burial grounds of St Bride’s Church, Fleet Street, London, reveal the pervasive impact of epidemic and endemic infectious diseases on non-adult health in nineteenth-century London. Documentary evidence of high mortality rates from diseases (such as whooping cough, scarlet fever, smallpox,
tuberculosis, and measles) alongside skeletal evidence for generalised stress infer wider population health risks within London at this time, particularly amongst poorer communities. Social disparities widened by economic growth within the metropolis, crowded and deficient domestic and occupational conditions, and slow public health reform precipitated inherent vulnerability to uncontrolled disease spread. While skeletal evidence alone cannot currently reveal the true extent of specific infectious disease on morbidity and mortality within past populations, it can reveal bodily evidence for general health status, and by proxy potential susceptibility to environmental risks identified by historical documentation. This synergistic relationship allows us to consider the magnitude of the impact that ill-health, particularly infectious diseases, had on urban communities, and the socio-political and environmental vectors driving them.

The narrative generated concerning detrimental nineteenth-century urban conditions, poverty, and child health mirrors contemporary concerns surrounding infectious disease, and the inequalities experienced in the face of these. Today, as systemic inequalities within our urban communities remain ever-present, it is imperative that we understand the complexities of sociocultural factors and their impact on long-term health and well-being.

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