



Nineteenth-century mortality trends: A reply to Szreter and Mooney

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Abstract

In a recent article in this journal I challenged Szreter and Mooney's account of a mortality crisis in English industrial and manufacturing cities in the period c. 1830–1850. I argued, first, that there was no robust evidence for a major fall in urban life expectancies in this period; second, that there was evidence for a rise in mortality in early childhood, but that this rise occurred in rural as well as urban populations, and persisted until the 1860s; and third, that an increase in virulence of scarlet fever made a major contribution to this rise. Szreter and Mooney contested these conclusions on two main grounds: that my methodology for estimating urban life expectancies differed from theirs; and that the geography and chronology of scarlet fever patterns did not fit those of early childhood mortality. Here I demonstrate that these criticisms are invalid. Using their methodology I still find no evidence for a dramatic drop in urban life expectancies in the 1830s–40s. I also present new evidence that scarlet fever was a major cause of childhood mortality by the late 1830s and 1840s, in rural as well as urban populations, and could therefore account for the observed rise in early childhood mortality in this period.

KEYWORDS

childhood mortality, industrialization, life expectancy, scarlet fever, standard of living, urbanization

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In an influential article in the *Review* in 1998, Simon Szreter and Graham Mooney described a demographic crisis in English industrial and manufacturing cities in the 1830s and 1840s, where life expectancy in the largest non-metropolitan cities fell below 30 years, before recovering to previous levels in the 1850s.¹ According to Szreter, their evidence demonstrated ‘an epicenter of epidemiological disaster during the 1830s and 1840s in the central districts of the new industrial cities, where expectation of life at birth plummeted to levels not seen since the crisis years of the Black Death’,² and ‘a particularly deep crisis ... followed by a return to the pre-crisis levels ... in the 1850s and 1860s’.³

My recent article in this journal reviewed the empirical evidence and argued, first, that there was no robust evidence for a major fall in urban life expectancies in the 1830s and 1840s; second, that there was evidence for a rise in mortality in early childhood, but that this rise occurred in *rural* as well as urban populations, and persisted until the 1860s; and third, that an increase in the virulence of scarlet fever made a major contribution to this rise.⁴ In their reply to my article Szreter and Mooney contest these conclusions on two main grounds. First, they argue that the differences in our estimates of life expectancy in the 1830s and 1840s are a consequence of the method I used, and so my findings are in fact very similar to theirs.⁵ I have now recalculated my estimates using their methodology, and still find no evidence for a dramatic drop in urban life expectancies in the 1830s–40s. Second, they argued that scarlet fever could not have contributed to the rise and fall of mortality rates in the period c. 1830–70, because scarlet fever had little impact before the 1850s, especially in rural areas, and it did not decline before the 1880s. I present new evidence of scarlet fever mortality that demonstrates that scarlet fever was already a major cause of childhood mortality by the late 1830s and 1840s, in rural as well as urban populations. Scarlet fever mortality rates also declined after 1870. Therefore the timing, as well as the geography and age pattern, of scarlet fever mortality fits the observed mortality patterns considerably better than Szreter and Mooney’s argument regarding administrative paralysis in industrial and manufacturing towns. I discuss the question of overall trends in mortality first, and then address the contribution of scarlet fever to these patterns.

I

The key problem in tracing the early impacts of industrialization on English urban mortality patterns is the almost complete lack of robust evidence regarding mortality rates in the larger non-metropolitan towns in the century before 1837. Szreter and Mooney attempted to fill this lacuna (table 1); however, I argue that their estimates are flawed in three ways. First, their evidence for the 1820s and 1830s was derived solely from the Glasgow bills of mortality. Thus the whole phenomenon of a sudden epidemiological catastrophe in English cities in the 1830s and 1840s hangs

¹ Szreter and Mooney, ‘Urbanisation’.

² Szreter, ‘Population health approach’, p. 424. Other comparisons to the Black Death include Szreter, ‘Rapid economic growth’, p. 147; Szreter and Hardy, ‘Urban fertility’, p. 671; Szreter and Woolcock, ‘Health by association?’, p. 658.

³ Szreter, ‘Industrialization and health’, p. 80. Limitations of space preclude discussion of the advisability of comparing death rates in the 1830s–40s with the Black Death, when crude death rates averaged perhaps 30–60%, or even with other episodes of plague in the seventeenth century, when individual cities lost as much as 20–50% of their populations; Dyer, ‘Influence of bubonic plague’; Shrewsbury, *History*, p. 415; Slack, *Impact of plague*, tab. 6.1.

⁴ Davenport, ‘Urbanization’.

⁵ Szreter and Mooney, ‘Scarlet fever’.

**TABLE 1** Life expectancy in large English cities (non-metropolitan cities of 100 000+ inhabitants)

Period (1)	Szreter and Mooney (2)	No. of English cities contributing data (3)	Woods (4)	Davenport (5)	No. of English cities contributing data (6)
1801–10			32.0		
1811–20			32.5		
1821–30	35	0 ^a	32.7		
1831–40	29	0 ^a	32.6		
1838–44	29.5	<2 ^b		32.4	6
1841–50			32.0		
1851–60	34	8	32.3	34.0	7
1861–70	34	10	33.0	33.9	9
1871–80	38	13	36.6	37.1	11
1881–90	40	20	39.0	40.2	18
1891–1900	42	25	39.6	41.5	21

Notes: Estimates represent population-weighted averages for ‘administrative cities’ (see tab. A1 for details). Szreter and Mooney’s estimate for the 1840s refers to the period 1838–44, because it derives from estimates for Liverpool (1841) and Manchester (1838–44). Cols. 3 and 6 indicate the number of English ‘administrative cities’ on which Szreter and Mooney (col. 3) and Davenport (col. 6) based their estimates in each decade. See tab. A1 for estimates for individual cities contributing to col. 5.

^aGlasgow only.

^bCore registration districts of Liverpool and Manchester only.

Sources: See tab. A1; Szreter and Mooney, ‘Urbanisation’, tab. 6; Szreter and Mooney, ‘Scarlet fever’, p. 6 (Early View version) (life expectancy estimate for 1838–44); Woods, ‘Causes of death’; idem, *Demography*, tab. 9.4.

on a single high estimate of life expectancy in the 1820s, drawn from bills of mortality for one Scottish city.⁶ Second, contrary to their claim to have ‘evaluated all the known, most robust demographic evidence available for Britain, c.1780–1850’, Szreter and Mooney did not use all of the evidence that became available for English cities with the inception of civil registration in 1837, but relied for their life expectancy estimates for the 1840s on data for only two of the 620-odd registration districts (RDs) for which the Registrar-General published data (the central RDs of Liverpool and Manchester) (table 1, column 3).⁷ Third, their estimates of life expectancy based on these data were too low, because they relied on a single year of data for Liverpool, and because they misinterpreted the Registrar-General’s life table for Manchester.⁸ These overly low figures then biased their estimates downwards for other cities and led to too pessimistic an estimation of life expectancy in the 1840s.⁹

⁶ Woods, *Demography*, p. 368; see also Harris, ‘Public health’, p. 395.

⁷ Szreter and Mooney, ‘Scarlet fever’, p. 1 (Early View version).

⁸ Davenport, ‘Urbanization’, pp. 17–18.

⁹ Szreter and Mooney estimated figures for the outer districts of Liverpool and Manchester, and for the RDs of other large towns (Bristol, Birmingham, Sheffield, and Leeds), on the assumption that the absolute differences in life expectancies between these units and Liverpool and Manchester were the same in the 1840s as they were in the 1850s. They are therefore incorrect in claiming that their overly low estimates of life expectancy in the core districts of Liverpool and Manchester did not inform their low estimates of average life expectancy in administrative cities in the 1830s and 1840s; Szreter and Mooney, ‘Scarlet fever’, n. 12.



In my *Review* article I used the Registrar-General's published data for the central RDs of all the largest towns and demonstrated that there was relatively little difference in life expectancies in these districts between the period 1838–44 and the 1850s and 1860s.¹⁰ Szreter and Mooney argue that I should have calculated life expectancies for 'administrative cities' (their method is described in table A1). Table 1 (column 5) shows my new estimates, calculated using Szreter and Mooney's method and the published data which they overlooked. I estimate that life expectancy was 32.4 years in 1838–44, and rose by 1.6 years, to 34.0, in the 1850s. In contrast, Szreter and Mooney argued that life expectancy fell by six years between the 1820s and the 1830s, from 35 to 29 years, and then recovered by 4.5 years, from 29.5 to 34 years, between the 1840s and the 1850s (table 1, column 2). That is, Szreter and Mooney's estimate of the gap in life expectancy between the 1840s and the 1850s is almost three times larger than my estimate. My estimates are very close to those of Woods, who used a modelling approach to derive values for the decades before 1850 (table 1, column 4).

Values for individual cities are given in table A1. In the period 1838–44, life expectancy in Birmingham, Leeds, and Sheffield was in the range 35.1 to 36.1 years, very similar to levels in London (36.7 years) and the old southern city of Bristol (35.8 years), and four to five years below the national average (of 40.4 years).¹¹ Indeed, life expectancy in these rapidly growing, northern and midlands industrial and manufacturing cities was fully six to eight years higher than in Liverpool and Manchester, on which Szreter and Mooney relied so heavily for their grim picture of urban mortality rates in the 1840s. Notably, life expectancy in Glasgow was even lower than in Manchester and Liverpool in the period 1837–51, suggesting either that life expectancy has been underestimated for Glasgow in this period, or that the city provides a poor guide to levels and trends in mortality in English cities.

Do these differences matter? Szreter and Mooney describe our differences regarding levels of mortality in the mid-nineteenth century as trivial: 'The death rates prevailing in the 1830s and 1840s are agreed to have been very high and there is just a quibble over the extent of the minor improvement which occurred in the 1850s'.¹² I disagree. The extent of improvement between the 1830s–40s and the 1850s–60s is crucial to Szreter and Mooney's thesis of an epidemiological crisis in the 1830s and 1840s, because we have relatively abundant evidence for mortality levels in the 1850s and 1860s, and these indicate that mortality in large cities was, to quote Szreter and Mooney, 'moderately high', rather than extreme, in this period.¹³ The modest changes in life expectancy between 1838 and 1870 documented in table 1 (column 5) therefore provide a major challenge to their account.

II

Although there is little evidence of a specifically urban mortality crisis in the second quarter of the nineteenth century, there is clear evidence for a rise in mortality in this period, after a sustained fall over the previous seven decades (c. 1750–1820). This rise was, however, largely confined to early childhood (ages one to four years) and occurred in rural as well as urban populations. The

¹⁰ Davenport, 'Urbanization', tab. 3.

¹¹ These estimates contrast with Szreter and Mooney's estimates for the same towns in the 1840s, of 33 years for Birmingham, and 32 years for Leeds and Sheffield; Szreter and Mooney, 'Urbanization', p. 104.

¹² Szreter and Mooney, 'Scarlet fever', p. 12 (Early View version).

¹³ *ibid.*, p. 13 (Early View version).



strongest evidence for this phenomenon is from family reconstitution of relatively small communities in England, and national evidence from Sweden.¹⁴ Following Woods, I suggested that this unusual pattern was due to a rise in the virulence of scarlet fever, which then subsided again after around 1870.¹⁵ Szreter and Mooney dismiss this possibility on three grounds: first, that scarlet fever could not have caused any rise in mortality in rural populations; second, that the chronology of scarlet fever lagged rather than led mortality trends; and third, that scarlet fever mortality patterns were not related to changes in virulence, but were a function of urban conditions.¹⁶ In the following section I demonstrate that the evidence for a rise in early childhood mortality rates (at ages one to four years: ECMR) in the 1830s is in fact strongest for relatively small communities, and I show that scarlet fever was already a major cause of childhood death in these types of communities by 1838–42, the first years for which scarlet fever was reported in RDs. I also show that scarlet fever death rates declined, nationally, after 1871, and therefore contributed substantially to falls in ECMR from that date (not 1880, as claimed by Szreter and Mooney). In addition, I argue that urbanization and fertility trends cannot explain the abruptness of the rise of scarlet fever in rural as well as urban populations nor the suddenness of its decline.

The key questions to be addressed with respect to trends in ECMR in the 1830s are, first, how large was any increase in mortality in this age group, and second, how much could scarlet fever have contributed to this increase? As discussed in my *Review* article, the first question is not straightforward to answer, because the evidence we have for a rise in ECMR derives from the knitting together of two different sources of evidence, from family reconstitutions before 1838, and from the Registrar-General's published returns thereafter. These sources dovetail remarkably well with respect to life expectancy at birth, and infant and adult mortality, but are sharply discrepant in early childhood.¹⁷ At the national level, ECMR appears to have risen from 98 per thousand in the period 1825–37 to 136 in the 1840s.¹⁸ This is a huge rise (of 28 per cent, relative to the 1840s), and its coincidence with the transition in evidence from a sample of relatively small communities to comprehensive national data that include London and the largest towns makes it likely that some of the discrepancy is due to the change in composition of the sources.¹⁹ More robust evidence derives from the comparison of family reconstitution parishes with the larger RDs in which they sat.²⁰ Among the eight parishes that were still in observation in the early 1800s and

¹⁴ Woods, 'Historical relationship'; Wrigley, Davies, Schofield, and Oeppen, *English population history*, pp. 93, 258–61. Szreter and Mooney cited the English evidence in their original paper in support of their argument, but now challenge the more detailed data I presented for the same populations. They state that only four of the eight parishes demonstrated a rise in early childhood mortality (ECMR) after 1825–37, and that in seven out of the eight districts ECMR fell after 1870, not after 1880, as they would expect from their reading of scarlet fever trends; Szreter and Mooney, 'Scarlet fever', p. 3 (Early View version). My own reading of my tab. 1 is that six of the eight parishes demonstrated a sustained rise in ECMR in the decades of the 1840s–60s (Grantham, Newton Abbot, Basford, Crediton, Hartley Wintney, and Loughborough). The issue of variations over shorter periods (including 1847–50) is addressed later in this section. In addition, the widespread falls in ECMR after 1870 are in fact consistent with the timing of the fall in scarlet fever mortality rates nationally (as discussed later; see also tab. 4).

¹⁵ Woods, *Demography*, pp. 368–71.

¹⁶ Szreter and Mooney, 'Scarlet fever'.

¹⁷ See Davenport, 'Urbanization', figs. 2a and 2b.

¹⁸ *Human mortality database*; Wrigley et al., *English population history*, p. 251.

¹⁹ I have calculated percentage change relative to the later rather than the earlier period (which is more conventional) in order to make direct comparisons with the contribution of scarlet fever, for which data are available only after 1837. Percentage change = $(136 - 98)/98 = 28\%$.

²⁰ Davenport, 'Urbanization', fig. 3 and tab. 1.



their associated RDs, ECMR was 21 per cent higher in the period 1838–44 compared with 1825–37, and only fell markedly again after 1870.²¹ There was relatively little difference in population densities between the parishes and their associated RDs, and so differences in mortality rates between the two sources were more likely to be indicative of a genuine increase in ECMR.²² Thus, at least in fairly rural areas, ECMR may have been elevated in the period c. 1838–70 by as much as 20 per cent or more above levels prevailing earlier in the nineteenth century.

Unfortunately, we have no evidence regarding trends in ECMR in English cities before 1838, except in London. In the metropolis, comparison of ECMR in Quaker families with ECMR in the registration county of London suggests (very tentatively given the potentially anomalous nature of the relatively affluent Quaker sample) a hike of around 11 per cent (from 163/1 000 among Quakers in 1825–49 to 184 in London in 1838–44).²³ Therefore it remains unclear whether ECMR rose in large towns to the same extent as in smaller settlements.

What role could scarlet fever have played in these increases in ECMR in rural areas in the 1830s? Szreter and Mooney argue that scarlet fever did not become a significant cause of death in rural areas before the 1850s, and could have accounted for ‘not more than 2–3%’ of deaths in early childhood in rural areas in the 1830s and 1840s.²⁴ Their logic is that scarlet fever mortality was related to population density (which is true), and therefore at low densities (in rural areas) scarlet fever must have accounted for a smaller *proportion* of deaths (which is false, because ECMR varied with population density). We can test their assumption straightforwardly in the 1860s, when scarlet fever was regarded as accurately reported and we have comprehensive data for cause-specific mortality in RDs.²⁵ Table 2 shows average scarlet fever and all-cause death rates, at ages one to four years, in RDs grouped by population density, in the 1860s. Scarlet fever mortality was indeed lower in more rural, low-density districts (2.7 deaths per 1 000 in the bottom quintile of population density, compared with 6.5 in the highest decile). However, *total* ECMR was also lower, because *ECMR was also correlated with population density*.²⁶ Therefore when we calculate scarlet fever deaths as a proportion of all deaths in early childhood (column 5), the impact of scarlet fever was almost as high in low-density areas (13.6 per cent of all deaths) as in the most densely populated urban districts (13.9 per cent). Clearly, contra Szreter and Mooney’s assumption, scarlet fever was a very significant cause of death in early childhood in a range of environments, from rural areas to the largest cities. Notably, the importance of measles, a more straightforwardly density-dependent disease than scarlet fever, varied more markedly with population density (column 6 of table 2), a point to which I will return.

But was scarlet fever also an important cause of death in rural populations in the decades of the 1830s or 1840s, when mortality appears to have risen markedly in childhood? This question is harder to answer, because the Registrar-General did not publish cross-tabulations of cause and age of death for small areas before the 1850s. However, in the rather experimental early years of registration, he did publish quarterly counts of deaths from scarlet fever (as well as typhus) in

²¹ This calculation is based on population-weighted estimates of average ECMR in the eight parishes, using the RD populations in 1851 as weights. These gave ECMR ($_{4q1}$) estimates of 78.9 and 100.5 per 1 000 children reaching the age of one year, for 1825–37 and 1838–44 respectively; *ibid.*, tab. 1.

²² *Ibid.*, tab. 1.

²³ Landers, *Death*, p. 136; *Fifth Annual Report of the Registrar-General* (P.P. 1843), p. 36. See n. 19 regarding the calculation of percentage change.

²⁴ Szreter and Mooney, ‘Scarlet fever’, p. 2 (Early View version).

²⁵ *Ibid.*, p. 3 (Early View version).

²⁶ Davenport, ‘Urbanization’, fig. 4; Woods and Shelton, *Atlas*, pp. 56–9.



TABLE 2 Early childhood mortality rates (ECMR), scarlet fever mortality rate (at ages 1–4 years), and measles deaths as a percentage of all deaths at ages 1–4 years in registration districts, 1861–70

Population density, RDs (percentiles) (1)	Mean persons/km ² (2)	Scarlet fever death rate (3)	ECMR (4)	Scarlet fever % of deaths (5)	Measles % of deaths (6)
1–20	36	2.7 (2.4–2.9)	19.2 (18.7–19.8)	13.6 (12.6–14.6)	5.6 (5.1–6.1)
21–40	60	2.9 (2.7–3.1)	21.2 (20.5–21.9)	13.4 (12.6–14.2)	6.5 (6.1–7.0)
41–60	84	3.4 (3.2–3.7)	24.0 (23.2–24.8)	14.0 (13.3–14.8)	7.2 (6.7–7.7)
61–80	153	4.4 (4.1–4.6)	28.6 (27.6–29.6)	15.1 (14.4–15.7)	8.4 (8.0–8.9)
81–90	637	6.3 (5.9–6.8)	40.0 (38.2–41.8)	15.8 (15.1–16.5)	9.1 (8.6–9.6)
91–100	8245	6.5 (6.0–7.0)	47.2 (44.0–50.4)	13.9 (13.2–14.6)	9.1 (8.6–9.6)

Notes: Values in parentheses are 95% confidence intervals for mean values. Mean population densities (col. 2) are geometric mean values. ECMR and scarlet fever death rates are deaths at ages 1–4 years per 1 000 population aged 1–4 years.

Source: Woods, 'Causes of death'.

RDs for the years 1838–42.²⁷ Table 3 shows reported scarlet fever deaths as a percentage of deaths from all causes in the period 1838–42, for the districts considered in my original article (that is, RDs associated with the parishes used by the Cambridge Group for family reconstitution, and the central districts of the largest towns). These districts range from relatively rural ones (including several of William Farr's 'Healthy Districts') to the largest cities, and are arranged in the table by their average population density in 1851.²⁸ Scarlet fever accounted for 3.6 per cent of deaths at *all* ages in England and Wales in the period 1838–42, ranging from less than half a per cent in urban Wolverhampton to 6.3 per cent of all deaths in relatively rural Basford, and with no apparent pattern according to population density or size.

To estimate the impact of scarlet fever among *children*, table 3 also displays scarlet fever deaths as a percentage of all deaths in the age range of one to nine years. The Registrar-General did not break down his counts of scarlet fever deaths by age, but we can assume from the age patterns of scarlet fever in later decades that most deaths occurred in this age range (the estimates in table 3 are calculated on the assumption that 90 per cent of scarlet fever deaths occurred at ages one to nine years).²⁹ On this assumption, scarlet fever accounted for 14.2 per cent of all deaths among children aged one to nine years in England and Wales in the period 1838–42. This is very similar to the estimates for the age range of one to four years in the 1850s and 1860s (tables 2 and 4).³⁰ Estimates ranged from less than 4 per cent in Bradford to over a quarter of all deaths at ages one to nine years in Basford. Importantly, scarlet fever accounted for as high a proportion of deaths

²⁷ It is not clear whether the Registrar-General included deaths attributed to diphtheria in these counts, but diphtheria was a very minor cause of death before the late 1850s; see Farr, 'Letter', pp. 183–5. Regarding diagnostic discrimination, see Hardy, *Epidemic streets*, ch. 4.

²⁸ Farr, 'Construction of life-tables'; Woods, *Demography*, pp. 181–4.

²⁹ 92.8% of all the deaths attributed to scarlet fever occurred at ages 1–9 years in England and Wales in the years 1848–70; Davenport, 'Annual deaths by cause'.

³⁰ In the decade 1851–60 scarlet fever accounted for 13.7% of all deaths at ages 1–4 years nationally, and 15.9% at ages 1–9 years. Corresponding figures for 1861–70 are 14.8 and 17.3%, suggesting that the use of the age range 1–9 to estimate the impact of scarlet fever at ages 1–4 would produce an overestimation of perhaps 16–17% of the true value at ages 1–4 years.

**TABLE 3** Deaths attributed to scarlet fever as a percentage of deaths from all causes, 1838–42

Registration districts	County	Population density, 1851	% of all deaths	% of deaths at ages 1–9 years
Crediton,* Moulton, Torrington,* Barnstaple*	Devon	54	4.4	20.8
Hartley Wintney, Alresford,* Petersfield, Alton, Basingstoke	Hampshire	55	1.5	8.5
Grantham, Sleaford	Lincolnshire	57	2.4	12.6
Banbury, Woodstock, Bicester	Oxfordshire	79	2.8	14.3
Newton Abbot	Devon	110	2.9	12.8
Loughborough, Barrow on Soar	Leicestershire	120	4.3	19.6
Eastrey, Isle of Thanet	Kent	175	2.0	10.8
Basford	Nottinghamshire	182	6.3	25.6
Wolverhampton and Seisdon	Staffordshire	478	0.4	7.3
Bradford	West Riding	1 115	1.0	3.7
Sunderland	Durham	1 460	4.5	15.8
Portsea, Alverstoke	Hampshire	1 689	2.9	12.5
Sheffield	West Riding	2 418	5.1	16.0
Newcastle	Northumberland	3 102	2.6	9.2
Leicester	Leicestershire	3 784	3.0	11.1
Norwich	Norfolk	3 896	3.8	16.0
Manchester	Lancashire	4 470	3.3	10.0
Salford	Lancashire	4 478	3.6	10.9
Hull	East Riding	6 853	5.8	21.4
Nottingham	Nottinghamshire	7 720	3.8	13.5
Bristol	Gloucestershire	8 825	2.5	8.6
Leeds	West Riding	11 925	3.5	11.2
Birmingham	Warwickshire	16 159	3.4	11.1
Liverpool	Lancashire	28 744	5.3	12.0
England and Wales			3.61	14.2
London			3.29	11.7

Notes: Asterisks indicate William Farr's 'Healthy Districts'. Values in the final column include 90% of all scarlet fever deaths, to adjust for deaths from the disease outside the age range 1–9 years. Scarlet fever deaths were reported quarterly for RDs for the years 1838–42. Total deaths were reported by age in RDs as aggregated totals for the seven years 1838–44. These were therefore deflated by a factor of 5/7 to make them comparable with scarlet fever deaths (this adjustment will tend to underestimate the contribution of scarlet fever to all deaths, because the population was increasing over this period). Deaths for England and Wales, and for London, were reported for single years, and refer to the years 1838–42. Population densities refer to acreages and populations of RDs in the census of 1851 (because the acreages of RDs were not reported in the 1841 census). The areas may be slightly inaccurate.

Sources: *Census of Great Britain, 1851* (P.P. 1852/3 LXXXVI); *Appendix to 3rd Annual Report of the Registrar-General* (P.P. 1841, VI), pp. 144–57; *4th Annual Report of the Registrar-General* (P.P. 1842 XIX), pp. 200–5; *5th Annual Report of the Registrar-General* (P.P. 1843, XXI), pp. 324–38; *6th Annual Report of the Registrar-General* (P.P. 1844, XIX), pp. 196–209.



TABLE 4 Quinquennial death rates from all causes, scarlet fever, and measles in early childhood, England and Wales, 1851–1900

Period (1)	ECMR (2)	Scarlet fever (3)	Measles (4)	Scarlet fever % of all deaths (5)
1851–5	36.9	5.0	2.8	13.7
1856–60	36.2	5.0	3.0	13.7
1861–5	36.2	5.0	3.0	13.7
1866–70	34.9	5.4	3.0	15.4
1871–5	31.5	4.3	2.5	13.6
1876–80	30.6	3.8	2.5	12.5
1881–5	27.2	2.4	2.8	8.8
1886–90	26.1	1.4	3.3	5.5
1891–5	25.2	1.1	3.0	4.4
1896–1900	23.3	0.9	3.2	3.7

Notes: Cols. 2–4 refer to deaths per 1000 population aged 1–4 years. Col. 5 refers to scarlet fever deaths as a percentage of all deaths at ages 1–4 years.

Sources: Davenport, ‘Annual deaths by cause’ (deaths); *Human mortality database* (annual population aged 1–4 years).

in relatively rural districts in Hampshire and Devon as in the largest cities, including London, Bristol, Liverpool, and Manchester.³¹

We can also use the information in table 3 to estimate the average proportion of childhood deaths due to scarlet fever in the eight reconstitution parishes and associated RDs in the period 1838–44. Scarlet fever accounted for an average of 17 per cent of deaths in the age range of one to nine years in these RDs.³² That is, scarlet fever was a very important cause of childhood death even in relatively rural parishes by the late 1830s.

Szreter and Mooney’s objection regarding timing also ignores the inevitably episodic nature of epidemic diseases. In the nineteenth century scarlet fever caused large autumnal epidemics with relatively long inter-epidemic intervals (of five to six years at the national level, with shorter intervals in the larger cities).³³ Before the 1850s epidemics were not synchronized between cities or counties.³⁴ Therefore when measured in windows of less than decadal length (as in table 3, of necessity), then there was considerable geographical heterogeneity in the impact of scarlet fever that probably reflected the varying probability that one or more epidemics occurred within the window of observation. However, when an epidemic did occur, then it caused huge local surges in

³¹ Szreter and Mooney misinterpreted fig. 9 in Davenport, ‘Urbanization’, to mean that scarlet fever mortality in early childhood (1830–44) was attributable to a single ‘moderate’ epidemic (in 1833). However, fig. 9 referred to mortality at all ages. Scarlet fever accounted for 30% of ECMR in 1833, and 7% in other years from 1830 to 44.

³² Averages are population-weighted as described in n. 21. Corresponding figures for ages 1–4 years in the decades 1851–60 and 1861–70 are 13.5 and 13.5%.

³³ Duncan, Scott, and Duncan estimated an epidemic cycle of 3.7 years in Liverpool, 1848–80, compared with an estimated 2.4 years for measles; Duncan et al., ‘Dynamics of measles’; eisdem, ‘Scarlet fever’; eisdem, ‘Demographic model’.

³⁴ Charles Creighton drew attention to the evidence of very large scarlet fever epidemics in the scant sources for the period pre-1837, but dated the emergence of *national* outbreaks to 1840, contrary to Szreter and Mooney’s reading of his comments as implying no major outbreaks before the 1840s; Creighton, *History*, pp. 724–7; Szreter and Mooney, ‘Scarlet fever’, p. 2 (Early View version).



child mortality, and therefore even episodic epidemics had a large effect on *average* mortality rates. When regional epidemics were out of phase, then this dampened epidemic peaks at the national level, and produced a high plateau of excess mortality. By the late 1850s national epidemics had become more synchronized, and the huge spikes caused by scarlet fever outbreaks were clearly visible in annual series of childhood mortality rates at the national level.³⁵

How much could scarlet fever have contributed to the rise in ECMR in small and rural populations in the 1830s and 1840s? The contribution of scarlet fever, at 13 per cent (in 1851–60) and perhaps 15 per cent (in 1838–44) of deaths in early childhood, appears at first glance to comprise a very considerable portion of the roughly 20 per cent increase in ECMR between 1825–37 and 1838–44.³⁶ There are, however, several problems in evaluating the absolute additive (or multiplicative) contribution of scarlet fever to ECMR. Apart from questions of the completeness and accuracy of cause-of-death recording (which may have led to either an over- or an under-estimation of deaths directly attributable to scarlet fever), it is also the case that the share of deaths due to a particular cause, even if accurately measured, does not provide a direct measure of the independent contribution of that cause to overall mortality rates. Children who died from scarlet fever would not necessarily have survived in the absence of scarlet fever—they may well have died from measles or some other disease if scarlet fever had not claimed them first. On the other hand, it should also be borne in mind that individual diseases can have *multiplicative* effects on mortality from other causes. In the case of measles, very longstanding observations of high levels of excess mortality from respiratory and diarrhoeal diseases following a bout of measles have been confirmed by recent evidence that the measles virus produces a generalized suppression of immune system function for up to two years after acute infection.³⁷ Scarlet fever was associated with a number of complications including pneumonia (which could lead to alternative diagnoses of cause of death), and with immune-mediated sequelae, most notably acute rheumatic fever.³⁸ It is difficult with the data to hand to evaluate the extent to which scarlet fever mortality was independent of other causes. Hinde and Harris's careful multiple-decrement analysis of the contribution of scarlet fever to life expectancy indicated that reductions in scarlet fever after 1870 contributed around a quarter of the total gains in life expectancy in each of the decades 1871–1900. There was no comparable rise in mortality from other childhood diseases, as would be expected if scarlet fever had simply been picking off children who were otherwise highly likely to die.³⁹ This apparently fairly additive effect of scarlet fever is consistent with contemporary observations that the disease was less socially selective than measles, and with the older age distribution of scarlet fever deaths compared with other childhood diseases.⁴⁰

To summarize, by the 1840s scarlet fever was a very significant cause of death almost everywhere in England. The fragmentary data available for the decades before 1838, summarized in my previous article, indicate that major scarlet fever epidemics began in the 1830s in England.⁴¹ Therefore the emergence of scarlet fever as a major childhood disease seems to have

³⁵ Mercer, *Infections*, fig. 8.1.

³⁶ The figure for 1838–44 is estimated for ages 1–4 years from the figure of 17% for ages 1–9 years (see n. 30).

³⁷ Mina, Metcalf, de Swart, Osterhaus, and Grenfell, 'Immunomodulation'; Petrova et al., 'Incomplete genetic reconstitution'.

³⁸ Sanyahumbi, Colquhoun, Wyber, and Carapetis, 'Global disease burden'.

³⁹ Mortality from measles, diphtheria, and diarrhoeal diseases rose markedly in the 1890s, but not in the decades of the 1870s and 1880s, when the decline of scarlet fever was most dramatic; Hinde and Harris, 'Mortality decline', tab. 3.

⁴⁰ Creighton, *History*, p. 731; Hardy, *Epidemic streets*, ch. 3.

⁴¹ Davenport, 'Urbanization', fig. 9. See also Mercer, *Infections*, p. 103.



coincided with, rather than lagged, the rise in childhood mortality in England in the 1830s and 1840s.

I do, however, agree with Szreter and Mooney that the decline in scarlet fever was not the only driver of falling mortality in early childhood after the mid-1860s. A secular fall in ECMR began in the late 1860s, before any fall in scarlet fever (table 4). Szreter and Mooney are wrong, though, to date the fall of scarlet fever mortality to the 1880s. At the national level (where we can calculate annual rates), scarlet fever mortality rates fell across the 1870s (from 5.0–5.4 deaths per 1000 of the population aged one to four years in the 1850s and 1860s, to 4.3 and 3.8 in successive quinquennia of the 1870s) (table 4, column 3).⁴² Despite this fall in absolute rates, scarlet fever did not fall as a *proportion* of all deaths in early childhood until the 1880s, because deaths from certain other causes fell faster (table 4, column 5).⁴³ That is, falling scarlet fever mortality in the 1870s was preceded and accompanied by falling death rates from other causes. I also agree with Szreter and Mooney, and with Hinde and Harris, that these early falls were driven in part by declines in mortality from waterborne and diarrhoeal diseases.⁴⁴

In the third prong of their argument, Szreter and Mooney argued that the rise of scarlet fever mortality in nineteenth-century cities reflected the growing opportunities for disease transmission in increasingly overcrowded and unhealthy populations. Conversely, the marked decline in scarlet fever mortality in the last decades of the nineteenth century could be explained by reductions in crowding and childhood infections caused by improvements in housing and by falling fertility and reductions in gastrointestinal infections.⁴⁵

These are valid *a priori* hypotheses. However, it is difficult to see why these factors should have operated with such force on scarlet fever patterns and not on other childhood diseases that also depended on person-to-person transmission. The abrupt surge in scarlet fever in the 1830s and its rapid decline after 1871 (table 4) were not accompanied by similar surges and recessions of other ‘crowd’ diseases such as measles. Measles transmission and lethality were much more sensitive than scarlet fever to effects of population density, family size, domestic overcrowding, and possibly nutritional status.⁴⁶ Yet measles mortality did not fall before the early 1900s. Both Reves and Mercer, who have emphasized the importance of fertility declines for mortality improvements, date these effects to the first decade of the twentieth century (when the absolute number of births began to fall), not the 1870s or 1880s.⁴⁷ Therefore while Szreter and Mooney may dispute the contribution of scarlet fever to urban mortality patterns, they offer little reason to reject the more

⁴² As tab. 4 indicates, falls in scarlet fever mortality in the 1870s were not simply a consequence of a rise in the previous decade, as Szreter and Mooney (‘Scarlet fever’, n. 9) appear to suggest. Moreover, in claiming that falls in respiratory tuberculosis and ‘waterborne’ diseases were more important than scarlet fever to improving life expectancy in the 1870s, they fail to distinguish between life expectancy (which was affected by mortality at all ages) and ECMR, where scarlet fever was the leading cause of death, and where respiratory tuberculosis was of minimal importance.

⁴³ Tab. 4 in Davenport, ‘Urbanization’, contains a serious error in the figures for scarlet fever as a percentage of all deaths in the decades 1881–1910. These should read ‘7.2%’ for 1881–90, and ‘4.0%’ for 1891–1900 and 1901–10. This error did not affect the argument regarding the timing of the decline in scarlet fever mortality. Corrected figures on a quinquennial basis are given in tab. 4 in this article.

⁴⁴ See Davenport, Satchell, and Shaw-Taylor, ‘Cholera as a “sanitary test”’.

⁴⁵ Szreter and Mooney, ‘Scarlet fever’, pp. 10–11 (Early View version).

⁴⁶ Aaby, ‘Malnutrition’; Aaby, Bukh, Lisse, and Smits, ‘Severe measles’; Woods, *Demography*, pp. 321–3. See also tab. 2.

⁴⁷ Mercer, *Infections*, p. 112; Reves, ‘Declining fertility’.



widely held view that scarlet fever underwent important changes in virulence in the nineteenth century.⁴⁸

I also argued that an autonomous rise in the virulence of scarlet fever could explain rises in early childhood mortality in other European and American populations in the middle decades of the nineteenth century. I set out all of the evidence that I could find that related to urban and rural (rather than national) populations in this period. Szreter and Mooney draw attention to some of the deficiencies of this evidence. I agree that crude death rates are a weak indicator of underlying trends. However, most of the evidence I presented related to age-specific mortality rates, and this showed a rise in mortality specifically in early childhood.⁴⁹ Szreter and Mooney's objections to the most robust evidence I adduced (for rural and urban communities in northern Italy, Sweden, the Netherlands, and Belgium) focus on the fact that the timing of rises in early childhood in these populations was later than in England, occurring in the 1840s or 1850s rather than the 1830s. However, as I have argued above, geographical variations in the timing of mortality impacts are quite consistent with the spread of a new disease. Given the long inter-epidemic intervals that were characteristic of scarlet fever, and the stochastic nature of disease transmission, then we might expect any new variant to have spread within Europe with a relatively complex pattern and some large element of randomness.⁵⁰ Importantly, these data indicate that trends in early childhood mortality were similar in rural as well as urban populations, something that is very difficult to explain as a consequence of political paralysis in new industrial cities.

III

My hypothesis regarding the contribution of scarlet fever to the hump of childhood mortality in rural and urban populations in the period c. 1830–70 is supported by the evidence that scarlet fever was a very important cause of childhood death throughout the period 1838–71 and across the settlement hierarchy. Importantly, this hypothesis does not entail a denial that public health measures were important to mortality levels and trends. Clearly they were. Nor does the relative stability of mortality levels in cities between the 1830s and the mid-1860s imply that cities were investing adequately in public health-related measures. Clearly they were not.⁵¹ How we judge these patterns probably depends on our *a priori* expectations. By the standards of the late nineteenth or twentieth century, early Victorian cities performed very poorly with respect to mortality levels and public health. However, from the vantage point of pre-industrial urban demography, what is most remarkable about mortality patterns during the British industrial revolution is that life expectancy did *not* fall catastrophically. The English population almost tripled, from 5.7 to 16.7 million, between 1740 and 1851, and the urban share doubled from c. 25 to 50 per cent, an absolute increase of over seven million.⁵² As Wrigley has pointed out, such unprecedented rates of growth

⁴⁸ Proponents of this view include Hardy, *Epidemic streets*, p. 59; Katz and Morens, 'Streptococcal infections'; McKeown, *Modern rise*, p. 83; Mercer, *Infections*, pp. 103–6 (with respect to the rise in scarlet fever as a cause of death); Swedlund and Donta, 'Scarlet fever'; Woods, *Demography*, p. 323.

⁴⁹ Davenport, 'Urbanization', pp. 19–22 and figs. 6 and 7.

⁵⁰ Katz and Morens, 'Streptococcal infections'.

⁵¹ Harris and Hinde, 'Sanitary investment'.

⁵² 'Urban' is defined here as settlements with populations of 2500 inhabitants or more. On the measurement of urbanization in Britain, see Langton, 'Urban growth'.



would have spelled disaster in any previous period.⁵³ Szreter is no doubt right to argue that economic growth brings disruption. However, in the case of nineteenth-century England the puzzle may be why such unprecedented demographic and urban growth was not *more* disruptive.

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⁵³ Wrigley, 'Coping with rapid population growth'; see also [Crafts and Mills](#), 'Race between population and technology'.



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APPENDIX

TABLE A1 Life expectancy in administrative cities with populations of 100 000 or more

City	1838–44	1851–60	1861–70	1871–80	1881–90	1891–1900
Liverpool	28.3	30.3	28.7	33.4	35.4	37.5
Manchester	29.8	32.2	32.3	34.9	37.3	38.3
Birmingham	35.5	37.1	37.5	39.0	42.0	41.9
Bristol	35.8	39.1	39.4	41.5	45.7	48.1
Leeds	35.1	35.3	34.6	37.3	40.3	41.7
Sheffield	36.1	35.2	35.5	37.3	40.2	41.6
Bradford		35.3	36.5	38.2	41.9	39.5
Newcastle			34.1	36.8	40.3	41.5
Salford			34.9	34.6	36.6	36.5
Hull				40.3	42.6	44.0
Portsmouth				43.5	45.3	45.7
Blackburn					39.2	41.1
Bolton					41.4	41.9
Brighton					45.6	46.4
Leicester					42.6	44.0
Nottingham					41.8	44.1
Oldham					38.6	39.5
Sunderland					40.7	41.1
Cardiff						45.2
Norwich						46.4
Preston						38.6
Weighted mean	32.4	34.0	33.9	37.1	40.2	41.5

(Continues)



TABLE A1 (Continued)

City	1838–44	1851–60	1861–70	1871–80	1881–90	1891–1900
England and Wales	40.4	41.1	41.2	43.0	45.3	46.1
London	36.7	38.0	37.7	40.4	42.6	43.7
Glasgow	27.3 ^a	29.6 ^b	32.1 ^c			

Notes: Life expectancies at birth (e_0) were calculated using abridged life tables based on single years of life at ages 0–4 and five-year age groups for ages 5–99 (for 1838–44) and on five-year age groups for ages 5–24 and 10-year age groups for ages 25–75 or 25–85 (1851–1900). Those dying in infancy were assumed to have lived on average 0.3 of a year, and years lived in the final open age interval were calculated as (number surviving to the age opening the last interval)/(age-specific death rate). Estimates refer to ‘administrative cities’ (defined as the municipal borough or urban sanitary district) and are calculated as population-weighted averages of life expectancies in RDs associated with each city. Cities were included in the sample when the population of the administrative city reached 100 000 in the census year that opened the decade. The contribution of RDs to administrative city e_0 values was weighted by the proportion contributed by each RD to the population of the city as specified in the census taken at the beginning of each decade (to avoid problems otherwise caused by boundary changes). The mean e_0 for all large cities combined was weighted by the estimated population of each city in the middle of each decade (or 1841, for 1838–44). Estimates of e_0 for England and Wales and London in the period 1838–44 refer to Woods’s figures for the decade 1841–50. This selection of cities differs slightly from that of Szepter and Mooney, because Plymouth–Devonport and Newcastle–Gateshead were treated separately (because they formed separate municipal boroughs).

^aValues refer to the years 1837–41.

^bValues refer to the year 1851.

^cValues refer to the year 1861.

Sources: 8th and 9th Annual Reports of the Registrar-General (P.P. 1847/8, XXV) (values for English cities 1838–44); *Census of Great Britain, 1851* (P.P. 1852/3, LXXXV); *Census of England and Wales, 1861* (P.P. 1862, L); *Census of England and Wales, 1871* (P.P. 1872, LXVI); *Census of England and Wales, 1881* (P.P. 1883, LXXVIII, LXXIX); *Census of England and Wales, 1891* (P.P. 1893–4, CIV, CV); Woods, ‘Causes of death’ (1851–1900); Szepter and Mooney, ‘Urbanisation’, tab. 5 (values for Glasgow); Woods, *Demography*, tab. 9.3 (England and Wales and London).