Patterns of infant mortality in rural England and Wales, 1850-1910

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Abstract: The study of nineteenth-century infant mortality in Britain has neglected the rural dimension to a surprising degree. We map the change in infant mortality rate (IMR) between the 1850s and the 1900s at Registration District (RD) level. Latent trajectory analysis, a longitudinal model based clustering method, is used to identify the clusters into which rural RDs fell, based on their IMR trajectories. Relationships between IMR and population density, fertility, female tuberculosis mortality, female illiteracy, male agricultural wages and distance from London are examined in a longitudinal study.

The tuberculosis (maternal health), illiteracy (education), and distance variables had the most effect. IMR responded most strongly to improving health and education in the east, less in the central area and least in the north and west. The eastern zone's higher than average mid-century infant mortality therefore declined faster than the national average. A central and southern zone had slightly lower IMRs in mid-century but did not keep up with the rate of decline in the east. The peripheral north and west had the lowest mid-century rates but their decline was overtaken by the other zones. The interpretation of these findings and their relevance to the wider study of infant mortality are discussed.

I

While nineteenth-century infant mortality in Britain has been extensively studied, the rural dimension has been neglected to a surprising degree.[1] Strongly influenced by Robert Woods' demonstration that urban change was the main influence on the national infant mortality rate (IMR), most work has focussed on the urban dimension.[2] The emphasis has been on the 'urban penalty', imposed chiefly by sanitation inadequate for dense populations, and

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[2] Woods and Woodward, eds., *Urban disease and mortality*; Woods, Watterson, and Woodward, 'Causes of rapid infant mortality decline part 1'; idem, 'Causes of rapid infant mortality decline part 2'.

visible above all in infant mortality from diarrhoea.[3] The idea that infant mortality was lower in rural areas was implicit in discussion of an urban 'penalty', and some studies have quantified this difference.[4] Williams and Galley also examined the evidence that diarrhoea-related infant deaths were a much smaller proportion of the total in rural areas.[5] Most work, unfortunately, has treated the rural as an undifferentiated whole and very little has been published about variation in IMR between rural places.[6] This is surprising because the evolution of rural IMR in the second half of the nineteenth century shows striking and unexpected patterns which offer insight into infant mortality more generally.

Recent developments in rural history have greatly expanded our understanding of factors closely bound up with infant mortality such as living standards, family formation and gender roles.[7] This rural literature has not yet added to our knowledge of infant mortality in a quantitative way, however. One of the greatest strengths of Reay's account of *Rural Englands* is his emphasis on diversity: in relation to IMR he notes Woods' lack of sufficient

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[3] Szreter and Hardy, 'Urban fertility and mortality patterns'; Millward and Bell, 'Infant mortality'.

[4] Millward and Bell, 'Infant mortality'; Lee, 'Regional inequalities'; Williams and Galley, 'Urban-rural differentials'.

[5] Williams and Galley, 'Urban-rural differentials', pp. 412-6.

[6] Woods and Hinde, 'Mortality in Victorian England'; Woods and Shelton, *Atlas of Victorian mortality*; Sneddon, 'Double penalty?'; Gregory, 'Different places'.

[7] See particularly Snell, *Annals of the labouring poor;* Howkins, *Reshaping rural England*; Short, ed., *The English rural community*; Verdon, *Rural women workers*; Reay, *Rural Englands*.

attention to inter-rural contrasts, citing isolated examples from primary and secondary sources.[8] These allow him to argue that local peaks in IMR could result from rural manufacturing industries or mining, but could also be found in ‘traditional rural hamlets’.[9]

The present study sets out to provide a more quantitative description for the use of future scholars. Section II presents new analyses of the spatial patterns of IMR and its evolution in rural England and Wales from the 1850s the 1900s. This includes the use of latent trajectory analysis to pick out clusters of RDs with similar IMR experience. Section III discusses possible factors affecting these rural patterns, while section IV presents the results of a longitudinal analysis, using a mixed-effects model to look at change in registration districts (RDs) over time. Section V uses the clusters of RDs to examine spatial patterns in the influences affecting IMR: section VI concludes.

II

The first maps of later nineteenth-century IMR at RD level for Victorian England and Wales were published by Woods and Shelton: their discussion concentrated on towns, but also noted rural zones in Eastern England and West Cornwall with IMR over 160 and a contrasting rural zone with IMR below 100 (when the mean was about 150), mostly in remote districts.[10]

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[8] Reay, *Rural Englands*, pp. 105-7.

[9] Ibid., p. 106.

[10] Woods and Shelton, *Atlas of Victorian mortality*, pp. 47-53.

Following Wrigley et al, Woods and Shelton felt that these lowest IMRs could have existed for centuries and might represent the best that a pre-industrial population could achieve in England.[11] Gregory used the Great Britain Historical Geographical Information System (GBHGIS) to explore rural patterns in more detail, showing that patterns of infant mortality decline in different parts of the country were more complex than had previously been described.[12] Here we extend Gregory's approach, again using the GBHGIS data to show the evolution of IMR at RD level.

The selection of RDs is a compromise between the greater definition offered by Registration Sub-Districts and the availability of data for our different variables over the chosen period. Decades were chosen because this, too, made more data available, allowing the use of cause of death as one of our independent variables. (The Registrar-General's data series on deaths by cause at RD level is only available in his Decennial Supplements.[13]) Our study considers 'rural' RDs only. To select these objectively, following Gregory, we ranked all RDs by population density into eight classes, whose boundaries were created using nested means of the populations of all RDs in all six decades.[14] The two lowest classes form our rural RDs, and have a

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[11] Wrigley, Davies, Oeppen, and Schofield, *English population history*, especially ch. 6.

[12] Gregory 'Different places'; Gregory, Bennett, Gilham and Southall, 'Great Britain historical GIS', pp. 37-49; Southall, Gilbert and Gregory, '*Census Statistics, Demography*' database.

[13] We acknowledge the work of David Gatley in transcribing this material into the GBHGIS: see Gatley, 'Computerising the 1861 census'.

[14] Population densities are calculated from the GBHGIS using totals at the end of each decade. The mean population density of all districts at every date was taken as the middle subdivision to create two classes. The averages of those above the mean and those below

population density below 183 persons per square kilometre. To be included in the study, an RD had to remain below this threshold throughout the period. (We also excluded 14 RDs for which there were missing data and 23 affected by mergers and divisions.) Our study therefore covered 363 RDs, containing 47 per cent of English and Welsh population in 1861, and 22 per cent in 1911.

Although ‘there is no agreed area which can be seen as “rural”’, our proportion of the population treated as rural accords quite closely with the views of other, including contemporary, scholars, and with contemporary labelling of places as towns and as urban or rural districts.[15] For comparison, using an arbitrary population density of 100 persons per square kilometre, the key paper by Woods et al produced a comparable set of 320 rural RDs.[16] We are confident that our set of rural places would be recognised as such by contemporaries and by other scholars. We note that analysis at RD level does not fully escape the problem of the non-homogeneity of our areal units. All rural RDs contained at least one town: larger examples include Lincoln and Aberystwyth. Where the urban proportion of a rural RD’s population grew, urban factors will have played a growing part in the RD’s IMR performance. For a study of a whole country, however, the RD is a good compromise between accuracy and data availability: not all the covariates of interest are available for areas smaller than RDs.

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the mean were then used to further subdivide these, creating four classes, and this was repeated again to create eight: Gregory, ‘Different places’, p. 778.

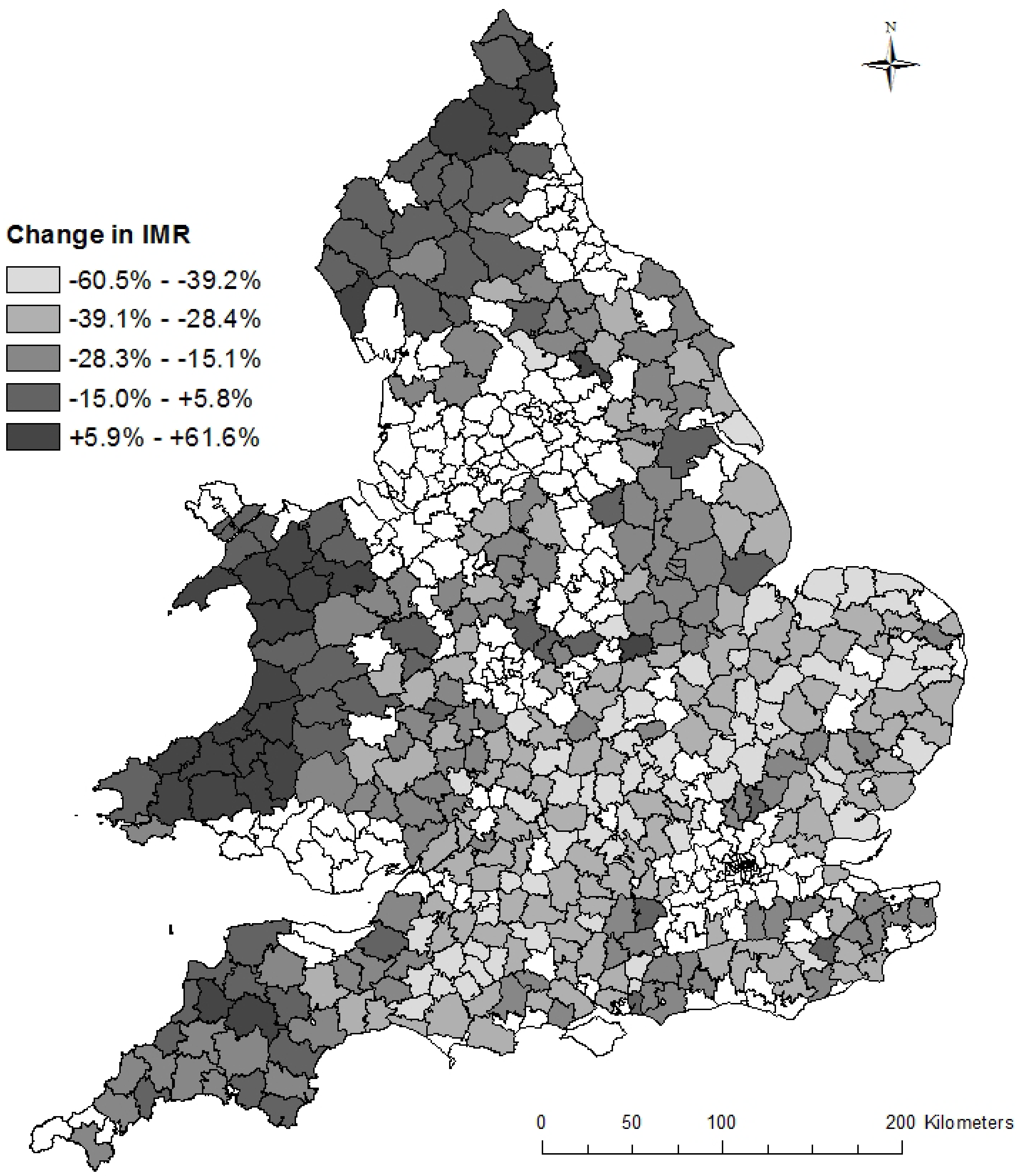
[15] Short, ‘Rural demography’, p. 1236.

[16] Woods et al, ‘Causes of rapid infant mortality decline part 2’, p. 127.

To set the scene for the discussion which follows, figure 1 presents the change in IMR for each rural RD between the 1850s and the 1900s. In the broadest terms, IMR fell fastest to the south of a line from the Humber to the Severn, but the picture is more nuanced than this. The biggest improvements are, unsurprisingly, to be seen in areas which had relatively high IMR in the 1850s, simply because they had more room for improvement: these include the Fenland districts noted by Woods and Shelton but also extend south and west through Cambridgeshire and parts of Bedfordshire, Buckinghamshire, and Northamptonshire. In the Fens, typical IMRs fell from the 180s to the 100s: in these other places from the 160s to the 80s. The Fenlands were characterised in mid-century by the rapid reclamation of land for arable cultivation such as vegetables: this called for large amount s of female labour of a particularly intense type. We believe this may have been an obstacle to adequate child care, resulting in higher infant mortality.[17] Elsewhere in the south and east, women were increasingly excluded from the agricultural labour market, which was dominated by male wage-labour on arable farms . While this avoided the problem of child care, it left them in a particularly low status within their households. We believe this limited their access to resources, resulting in poorer diets, with adverse consequences for maternal health and infant mortality.

[17] Sneddon, ‘Double penalty?’

Figure 1: Per cent change in IMR, 1850s-1900s (rural Registration Districts: quintiles)



Greatest reduction: -60.5, greatest increase: +61.6, mean change: -14.2, SD 18.2, N=363.
Unshaded RDs are not rural, or data for them unavailable
Source: derived from GBHGIS

By contrast, hardly anywhere north of York, in Wales or in Devon or Cornwall (places whose 1850s IMRs were typically in the 100s or 110s) achieved improvements of more than 30 deaths per thousand. If this was the whole story we could conclude with Wrigley et al that they had long ago achieved the best that could be done in pre-industrial circumstances. The question would then be why these districts did not advance further after the 1850s once others to the south began to do so. In this northern and western zone, pastoral agriculture played a much larger role. This preserved an eighteenth-century and older 'family economy' longer than it lasted in the more capitalised farming of the arable zone, as Reay's calculations of the 'family labour' share of the agricultural workforce confirm.[18] Snell saw this family economy as creating a more equal sexual division of labour because it included more access to commons, more owner-occupiers and small tenants, and less dependence on wage labour away from home:[19] Verdon's studies of farm records tell the same story.[20] It is our view that this resulted in better female diets and maternal health than experienced in the lowland zone, resulting in the relatively low mid-century IMRs. There is a problematic subset of these remote RDs, where IMR *increased* between the 1850s and the 1900s. Places in North Wales such as Ffestiniog and Corwen, where IMR was in the low 100s and rose by 12 or more points, are typical of this group. Forming a significant exception to the simple north-south divide just proposed, 21 RDs in Hampshire, Surrey, Sussex, and Kent improved by less than 30: these had more in common with the north and west than with their Home Counties neighbours.

[18] Reay, *Rural Englands*, p. 24.

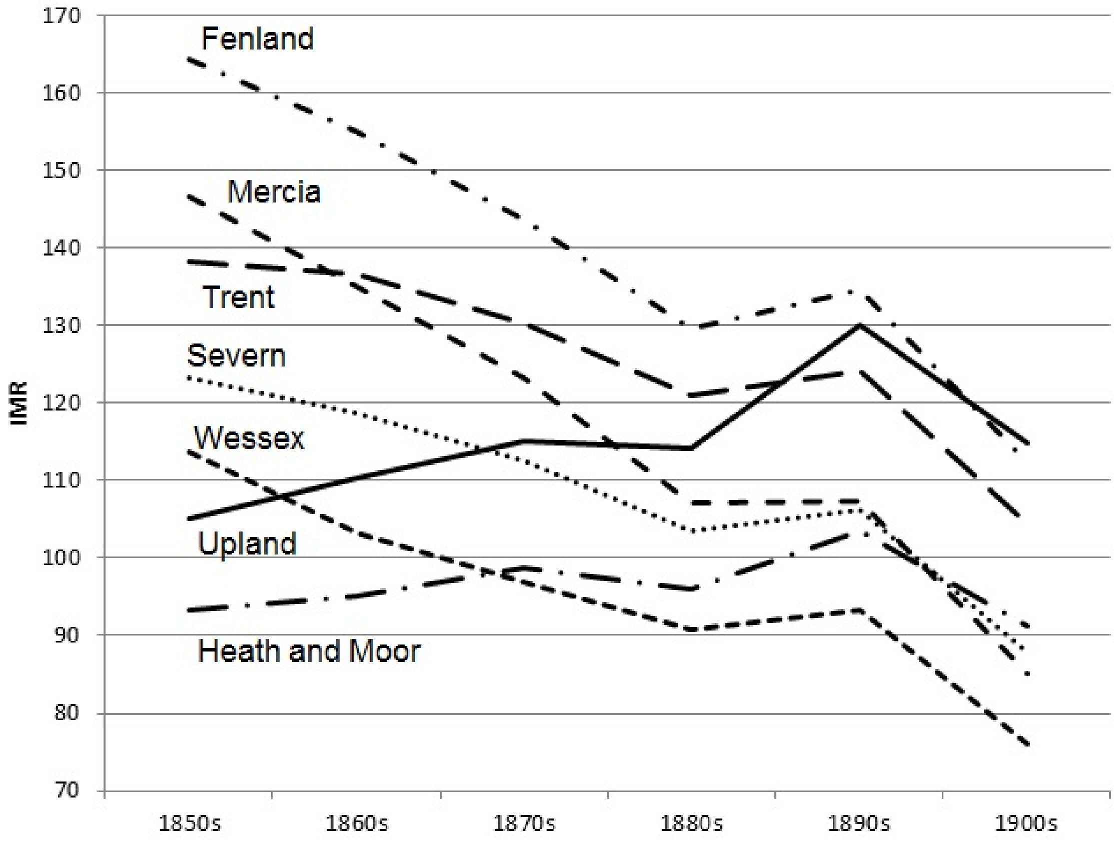
[19] Snell, *Annals of the labouring poor*, p. 62.

[20] Verdon, *Rural women workers*, p. 123.

It is also informative to use a statistical method to group the RDs whose IMR trajectories were most similar. We used latent trajectory analysis, a longitudinal model based clustering method, to do this.[21] The number of clusters selected is a trade-off between the Bayesian Information Criterion (BIC – a measure of the heterogeneity of the cluster, which declines as more clusters are used) and ease of visualisation. Our data show a strong decrease in BIC up to six classes, then slower decline: we accordingly chose a seven-class model. Figure 2 shows the mean IMR trajectory of each of these clusters: Figure 3 the locations of the RDs of each cluster.

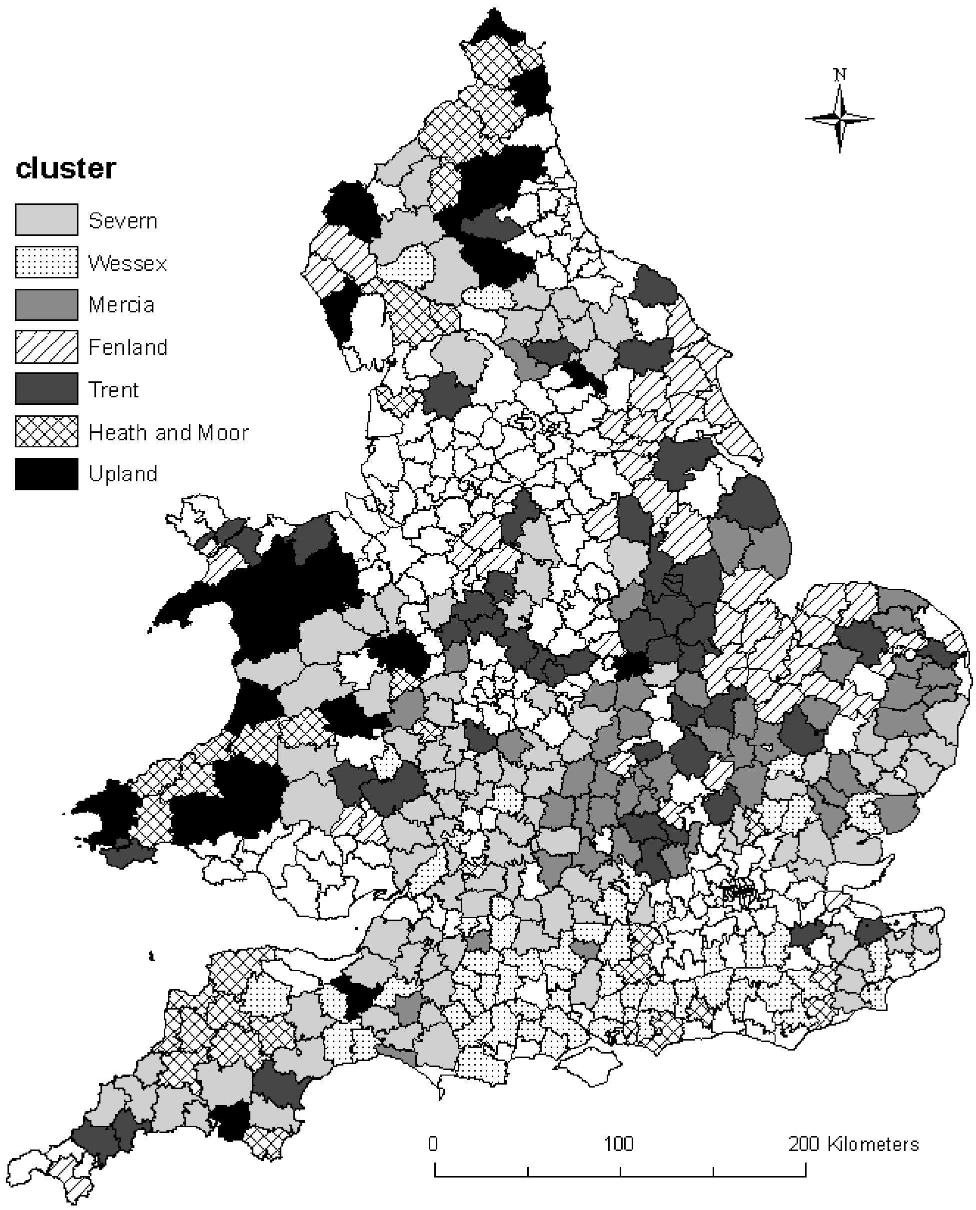
[21] Nagin, 'Analyzing developmental trajectories'.

Figure 2: Clusters of RDs with similar IMR performance: results of latent trajectory analysis



Source: derived from GBHGIS

Figure 3: Locations of RDs in the different clusters



Source: derived from GBHGIS
Unshaded RDs are not rural, or data for them unavailable

To a striking degree, the RDs which we clustered for their statistical similarity also cluster spatially: rural England and Wales had identifiable IMR zones. Further discussion of the significance of these clusters is postponed to section V, where IMR and its covariates are considered together.

III

In identifying possible covariates of IMR, our study design builds on earlier models. Woods, Watterson and Woodward discussed the influence of population density, fertility and female literacy and concluded that all three were relevant for towns and for the national aggregate: they did not report separate results for rural areas.[22] Williams and Galley drew attention to the advantages of separating the influences on IMR into maternal, home, and the wider ‘public environment’.[23] Our design is particularly influenced by Millward and Bell, who looked at fertility, maternal health, and the factors which influenced these.[24] None of these studies examined the differences between rural areas of different kinds. Since population density, then, has normally been discussed in examinations of IMR, it forms our first covariate. Previous studies suggest it was a key influence in towns and cities: we test here whether density mattered below the threshold represented by rural levels of population.[25] Our data are decennial averages of population density taken from the GBHGIS.

Scholars are agreed that the impact of fertility on infant mortality was both important and complex,[26] and we include a measure of fertility as our second

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[22] Woods et al, ‘Causes of rapid infant mortality decline part 2’, pp. 127-9.

[23] Williams and Galley, ‘Urban-rural differentials‘, p. 417.

[24] Millward and Bell, ‘Infant mortality’, pp. 705-6.

[25] Woods et al, ‘Causes of rapid infant mortality decline part 2’; Williams and Galley, ‘Urban-rural differentials’; Lee, ‘Regional inequalities’.

[26] Reher, ‘Back to the basics’; Woods et al, ‘Causes of rapid infant mortality decline part 2’, pp. 121-6.

covariate. In addition to its indirect effect via improving maternal health, falling fertility reduced infant mortality directly by allowing each infant a bigger share of household food and more time for maternal care, and by reducing the number of siblings, each susceptible to contracting and passing on infectious disease.[27] The example of fertility is a reminder of the endogeneity of the variables discussed here: it is also reported that falling infant mortality could be associated with higher fertility, when improving maternal health led to fewer complications of childbirth and so to higher continuing fertility among existing mothers.[28] In the absence of contemporary records of fertility, we follow Woods in relying on Coale's indices of marital fertility I_g and proportion marrying I_m, which have the attraction that they can be calculated from census data.[29] Our fertility indices are taken from Friedlander and Okun, who calculated them by RD for each Census from 1851 to 1911: we use the 1851 data for our 1850s time point, and so on.[30] Following Woods, we have relied on the low ratio of illegitimate births in the later nineteenth century to make the simplifying assumption that the product of I_g and I_m provides us with Coale's index of overall fertility I_f.[31] (Inspection of the Registrar-General's reports provided reassurance that there were not marked regional differences in illegitimacy ratios. For example in 1862, the ratio in Malling, Kent was 8.2%: in 1882 it was 6.6%. In the same two years,

[27] Millward and Bell, 'Infant mortality', pp. 709-10; Reves, 'Declining fertility'.

[28] Millward and Bell, 'Infant mortality', p. 722.

[29] Woods, *Demography of Victorian England and Wales*, pp. 8-9.

[30] Friedlander and Okun, 'Pretransition marital fertility'; Friedlander and Okun, '*Demographic processes*' database.

[31] Woods, *The Demography of Victorian England and Wales*, p. 9.

ratios in Alnwick, Northumberland were 8.8% and 4%.) These RD-level decennial estimates of the index of overall fertility provide our fertility measure.

We then turn to maternal health. Millward and Bell suggest that for infants under the age of one year, influences mediated through the mother affected mortality most.[32] Millward and Bell's model emphasises the impact of maternal health on the infant via routes such as birth weight, immune system, and the quality and duration of breastfeeding, which can be summed up in the statement 'what makes mothers ill makes babies ill'. Millward and Bell make a persuasive case that the tuberculosis mortality of females of child-bearing age is a good, and measurable, proxy for maternal health.[33] They also show that tuberculosis mortality does not proxy the quality of the household environment so closely, making it a 'purer' indicator of maternal health alone, and that the key influence on it is rising real household incomes. Indeed, their conclusion is that rising real incomes and falling fertility rates explain much of the pattern of IMR decline.[34] Here we reproduce their approach to the measurement of female tuberculosis mortality to see whether it is as good a predictor of IMR across all 363 rural places as it was for their mixed sample of 36 locations.

The Registrar-General provided the age-sex breakdown we require for the 1850s, 1860s, and 1900s: for the three intervening decades he reported tuberculosis deaths of females (and males) undifferentiated by age, and

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[32] Millward and Bell, 'Infant mortality', pp. 705-9.

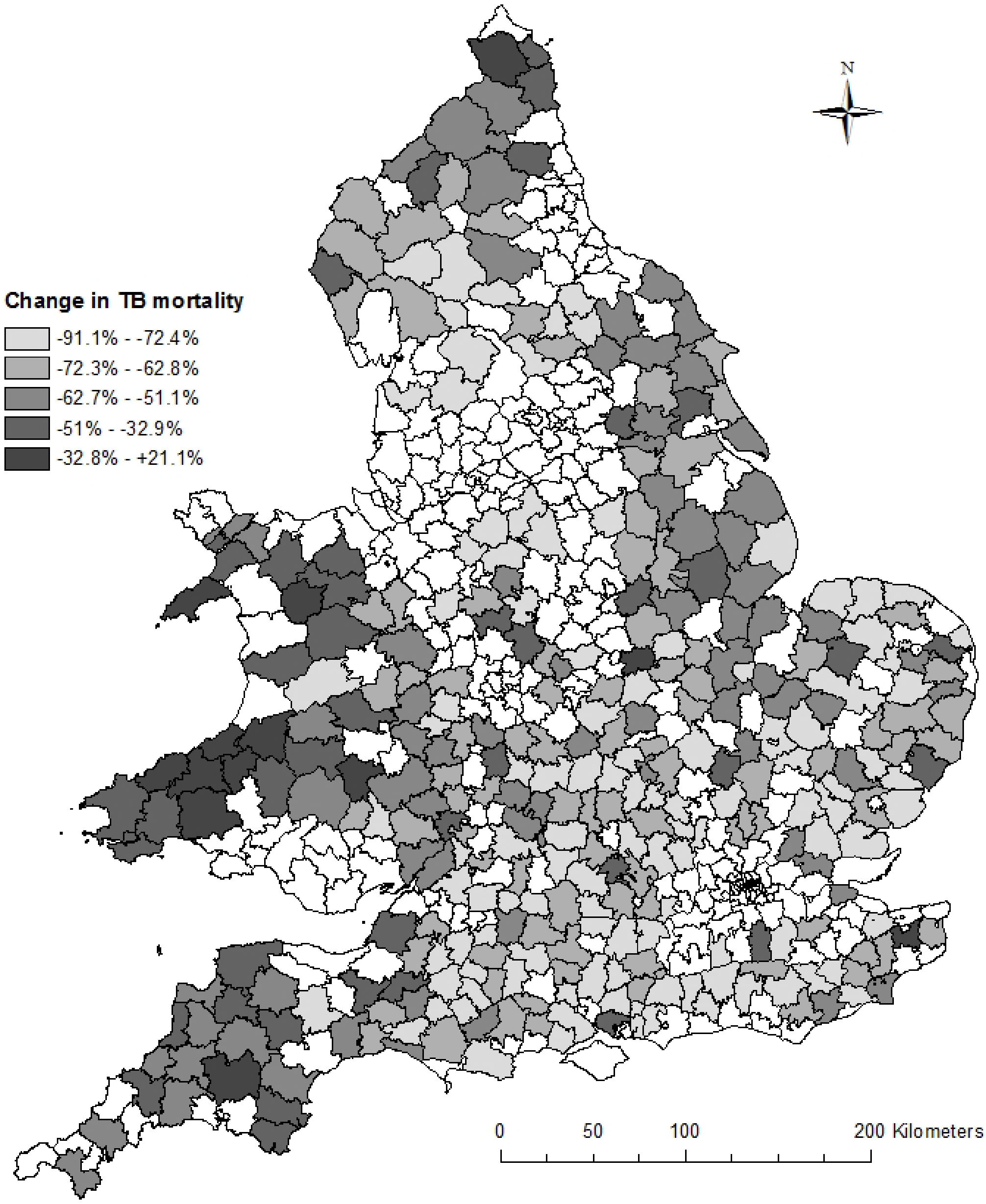
[33] Ibid., pp. 714-6.

[34] Ibid., p. 728.

deaths by age band undifferentiated by sex. Following Millward and Bell's approach, we interpolated values for the 1870s, 1880s and 1890s by two alternative methods and averaged the result: the first method assumes that shares of female tuberculosis deaths accounted for by the 15-44 age band fell smoothly between the known data points of the 1860s and 1900s: it then applies these shares to each place's total female tuberculosis deaths. The second method assumes that shares of age 15-44 tuberculosis deaths accounted for by females fell smoothly between these two dates, and applies these shares to each place's total deaths at age 15-44.[35] We then use an average of the two results as our estimate of female tuberculosis mortality at age 15-44. The need to interpolate data is of course a weakness, but the approach taken by Millward and Bell, which assumes only that a RD behaves consistently with itself over time (rather than assuming, say, that all RDs experienced some aggregate trend) is the best way to use the available data for a spatial analysis: it avoids introducing any spatial artefacts. The two methods had results within 20 per cent of each other in five-sixths of our RDs.

[35] Ibid., p. 713.

Figure 4: Per cent change in female mortality from tuberculosis at ages 15-44, per thousand living, 1850s to 1900s (rural registration districts: quintiles)



Greatest reduction: -91.1, greatest increase: +21.1, mean change: -62.5, SD 16.0, N=328
Unshaded RDs are not rural, or data for them unavailable
Source: derived from GBHGIS

Cronje analyses TB trends and their possible causes by county, using the Registrar-General's classification of counties as 'mainly urban' and 'mainly rural'.[36] She reports that in the mainly rural counties, female (all-ages) death rates fell from 2.7 per thousand in the 1850s to 0.9 in the 1900s. For females, rural tuberculosis death rates were at all times little better than urban ones (in one decade actually slightly worse): a distinct contrast with the male picture, where a rural advantage was clear. She suggests this pattern resulted from unhealthier urban male lifestyles (especially their greater exposure to infection in workplaces and places of entertainment).[37] Cronje notes that rural tuberculosis death rates are inflated to some degree by the phenomenon of young adults travelling to urban areas to find work, especially females to domestic service, developing tuberculosis, and returning to their rural family homes to be nursed and die: she estimates that this increased rural female tuberculosis mortality in the 15-34 age group by 11-15 per cent.[38]

Figure 4 shows the spatial pattern of the decline in tuberculosis mortality among females of fertile age (15-44). Clearly the spatial distribution of this mortality decline was even more complex than Cronje's county-level data showed. Slower improvement can be discerned in the north and west than in the south: it was particularly slow in rural Northumberland, south and west Wales, Devon and Cornwall, much of Lincolnshire, and east Yorkshire. A typical example of the best quintile is Newmarket's improvement from 5.8 deaths per thousand to 1.6: at the opposite end of the distribution,

[36] Cronje, 'Tuberculosis and mortality decline', pp. 89-97.

[37] Ibid., pp. 93-94.

[38] Ibid., p. 95.

Carmarthen typifies the slow improvers with values of 4.4 in the 1850s and 3.2 in the 1900s.

Millward and Bell's 'mother as medium' model is solely about the impact of maternal health. It is possible to extend this model to capture some of the impact of mothers' education. Literature on recent infant mortality in less developed countries is relevant here. Caldwell and McDonald, for example, show that 'age and sex differentiations in power, decision-making and benefits within the larger family are reduced when schooling … [means] … women and children are allocated higher priorities in terms of care and allocation of food and in which parents can make decisions about health and child care without reference to their elders.'[39] Despite its gendered curriculum, nineteenth-century education, thanks to equal access for boys and girls, raised female status within households and communities.

Millward and Bell did not attempt to measure the effects of maternal education. It is, however, possible to draw on research by Horrell and Oxley, Humphries, and others into female status within the household to generate some testable hypotheses.[40] This work cautions against simplistic accounts of the effects of income on social conditions which ignore its changing distribution within the household. These researchers showed that a disadvantaged status for women could produce excess female mortality or

[39] Caldwell and McDonald, 'Influence of maternal education'; see also, for example, Jain, 'Regional variations in infant mortality'.

[40] Horrell and Oxley, 'Bringing home the bacon?'; Horrell and Oxley, 'Bargaining for basics?'; Humphries, "'Bread and a pennyworth of treacle"'; McNay, Humphries, and Klasen, 'Excess female mortality'.

shorter stature: our hypothesis is that it also affected the infant. Education made itself felt in mothers' ability to secure more resources for feeding and childcare, and could have given access to improved childcare knowledge, though sources for the reception of contemporary texts on this are scarce. If Millward and Bell's case is that what makes mothers ill makes babies ill, we wish to add that what makes mothers vulnerable makes babies vulnerable.

Education is proxied here, as it was by Woods et al, using female illiteracy, captured from the Registrar-General's reports of proportions of brides making a cross in the marriage register rather than signing it.[41] Most of the illiteracy data used here were taken from Friedlander and Okun's dataset, picking mid-decade data points of 1856, 1866, and 1876.[42] To extend their series, we produced an 1880s data point using the last instance when the Registrar-General reported on the literacy of spouses, in 1884.[43] While there are issues about the validity of this measure of literacy,[44] using it has the merit of producing findings comparable to Woods et al's results, which only reported on urban places and a national aggregate.[45]

Figure 5 shows the spatial distribution of the decline in female illiteracy. Places in the best quintile combined high starting points with a rapid fall. Blything, in Suffolk, typifies these with a reduction in illiteracy from 67 per cent to 12 per cent. In the worst quintile, the starting point could be either

[41] Woods et al, 'Causes of rapid infant mortality decline part 2', p. 127.

[42] Friedlander and Okun, *Demographic Processes* database.

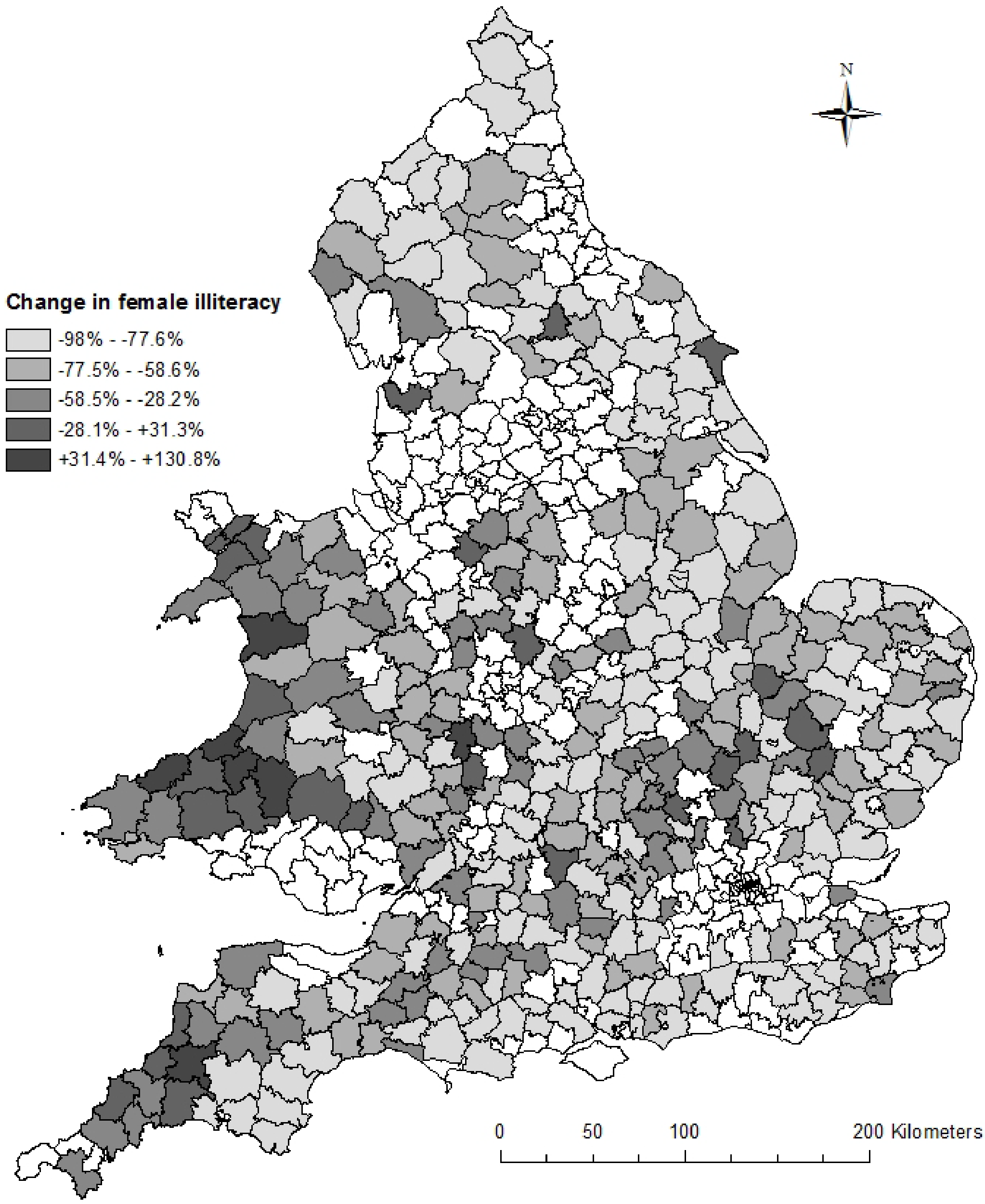
[43] *Forty-Seventh Annual Report of the Registrar General* (P.P. 1886, XVII), pp. 2-25.

[44] Schofield, 'Dimensions of illiteracy'.

[45] Woods et al, 'Causes of rapid infant mortality decline part 2', pp. 127-9.

high or low. In south and west Wales illiteracy began high and remained so: Newcastle Emlyn is a representative example, with rates of 40 per cent and then 35 per cent. Other places in the worst quintile had lower starting points, as in Launceston (Cornwall), where illiteracy actually rose from10 to 13 per cent, or in Ely where it fell from 20 to 14 per cent. This variety of starting levels is a warning that mapping the difference between two snapshots in time does not tell the whole story: the longitudinal regression analysis, of course, captures all the time points.

Figure 5: Change in per cent of brides signing with a mark, 1850s to 1880s (rural registration districts: quintiles)



Greatest reduction: -98.0, greatest increase: +130.8, mean change: -63.7, SD 32.2, N=356.
Unshaded RDs are not rural, or data for them unavailable
Sources: Friedlander and Okun, '*Demographic Processes*' database; *Forty-Seventh Annual Report of the Registrar General* (P.P. 1886, XVII), pp. 2-25.

The next covariate to consider is income. A link between rising real income and falling IMR is intuitively likely, though we have already noted that the distribution of income within the household might be more important than its level. Measurement of household income has proved elusive. Woods et al used the amount of employment in domestic service as a proxy but found their results unconvincing.[46] Millward and Bell's estimation of income was thorough but the availability of data confined them to 36 mainly urban sites: they showed the importance of rising income for better maternal health.[47] We tried to test the impact of income across all rural RDs. In the absence of RD-level datasets for nineteenth-century incomes we used E. H. Hunt's series of wage rates by county for agricultural labourers, and deflated these by Crafts and Mills' cost of living index to produce real wage levels.[48] The model assumes that the wage rate in each RD was the prevailing one for its county. While clearly an approximation, this is the best income data available and for such a potentially important variable it was judged necessary to include it. We used all of Hunt's data, fitting his rates for 1867/70, 1898, and 1907 into our relevant decades and interpolating values for the 1870s and 1880s by assuming constant rates of change for each RD (as with tuberculosis mortality). Figure 6 shows the spatial distribution of the rise in real agricultural wages.

Since the wage data, unlike those for TB and illiteracy, were available only at county level, the map shows less detail. At mid-century, the southeast had

[46] Ibid., pp. 126-9, especially footnote 78.

[47] Millward and Bell, 'Infant mortality', pp. 718-19.

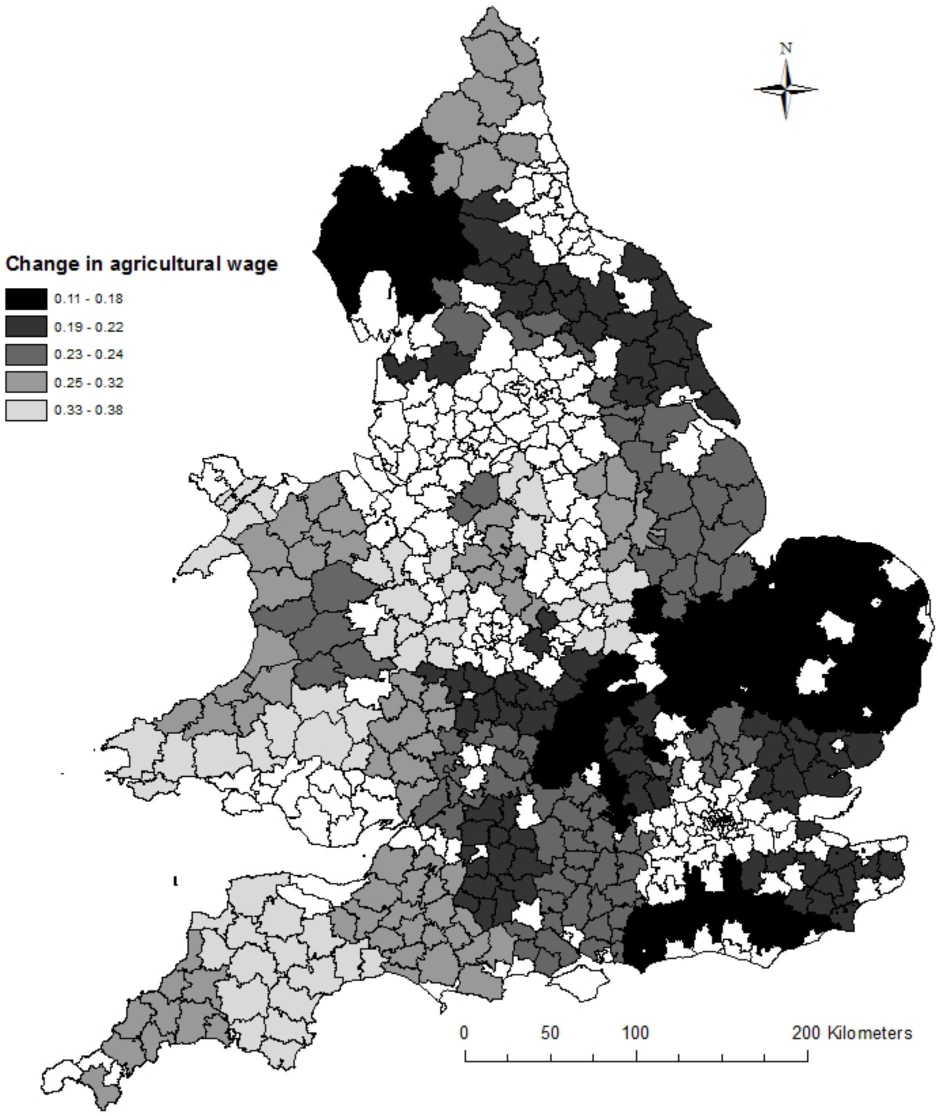
[48] Hunt, *Regional wage variations*, pp. 61-3. Hunt's main sources were Bowley, *Wages in the UK in the Nineteenth Century* (Cambridge, 1900), endtable; *Reports on Wages and Earnings of Agricultural Labourers,* P.P. 1900, LXXXII and *Earnings and Hours Enquiry. V. Agriculture*, P.P. 1910, LXXXIV; Crafts and Mills, 'Trends in real wages'.

an overstocked labour market with low (often very low) wages, while higher agricultural wages were earned in northern districts where farms had to compete for labour with nearby industry.[49] Hunt's data show that, whether or not there was a 'Great Depression' in British agriculture in the last quarter of the nineteenth century, labourers everywhere were able to secure gradually rising incomes (at least in the long term). No county shows a real decrease: the south and east do least well, generally achieving small gains on a low base (for example in Norfolk rising from 12s.7d. per week to 14s.9½d.).[50] The remotest parts of the northwest show a similarly small gain but on a much higher base, as in Westmoreland (16s.to 18s.5d.). The greatest progress in wages was enjoyed by a western and west Midlands group where wages began very low: Devon is typical with a progression from 10s.9½d. to 17s.2½d.

[49] Thompson, 'Anatomy of English agriculture', pp. 235-9.

[50] The examples of wages in this paragraph are deflated to 1900 prices

Figure 6: Change in real agricultural wage (£ per week), 1860s – 1900s (rural registration districts: quintiles)



The classes shown are quintiles. Minimum: +0.11, maximum: +0.38, mean: +0.24, SD: 0.07, N=346.
Unshaded RDs are not rural, or data for them unavailable
Source: Hunt, *Regional wage variations*, pp. 62-3.

Population density, fertility, female tuberculosis mortality, female illiteracy, and (male) agricultural wages thus form our within-subjects variables. We also used two between-subjects variables to model time-invariant factors.

The first of these is an RD's peripherality, defined as the distance in kilometres from London of its centroid. This is readily calculated using GIS software such as ArcGIS from the geographical data about RDs in the GBHGIS. Gregory drew attention in earlier work to a possible relationship between increasing peripherality and slower improvement in IMR.[51]

Our final variable is average elevation of the RD above sea level. Historical geographers divide England and Wales into a 'highland' north and west with predominantly pastoral agriculture and a lowland south and east, mainly arable.[52] Including average elevation of each RD allows a more fine-grained analysis. We included average elevation to investigate whether it could be used as a proxy for type of agriculture. Digital Elevation Model (DEM) data were downloaded from the Consultative Group for International Agricultural Research, which provides digital elevation at 90 metre intervals globally.[53] From the elevation points contained in each RD, the average elevation was calculated in ArcGIS. This variable failed to give statistically significant results. Either the unknown rural influences on IMR did not correlate well with average elevation at RD level, or they correlate well enough with the other variables used here for elevation not to have an important independent effect.

IV

[51] Gregory, 'Different places', pp. 780-4.

[52] Holderness, 'Farming regions', p. 363.

[53] http://srtm.csi.cgiar.org/ (accessed 20/10/2015)

We use a mixed-effects longitudinal model in which RDs are the subjects (cases) and decades are the time points. Such a design provides the time perspective which Woods acknowledged to be lacking in his own impressive snapshot studies of single time periods.[54] We aimed for the simplest credible model specification: refinements to it could improve the fit. There were two levels: level one was the within-subject effects (variations within an RD over time) and level two was the between-subjects (time-invariant) effects. As we found no gain in model accuracy by specifying a more complex level one covariance structure, the model takes the covariance of level one effects as unstructured. Nor did we model any cross-level interactions.

Table 1 shows the availability of data by decade. This shows that it is not possible to run the model with all the data for all six decades. Confining analysis to the period 1860s-1880s when all the data are available is not attractive as it excludes a period of the most rapid decline in IMR after 1890. Another option, extrapolation of our data series for illiteracy and wages, would lack rigour. We therefore undertook three model runs to capture all the data in stages: the first three variables for 1850s-1900s; then adding illiteracy and looking at the 1850s-1880s, and finally replacing illiteracy with wages and looking at the 1860s-1900s.

[54] Woods et al, 'Causes of rapid infant mortality decline part 2', 129.

Table 1: Data availability for covariates of infant mortality

	1850s	1860s	1870s	1880s	1890s	1900s
Population density	✓	✓	✓	✓	✓	✓
Fertility	✓	✓	✓	✓	✓	✓
Female TB	✓	✓	✓	✓	✓	✓
Female illiteracy	✓	✓	✓	✓		
Agricultural wage		✓	✓	✓	✓	✓

The results we present in Tables 2 and 3 below show the estimates of effects on IMR per unit change in each covariate (column *(b)*), along with standard errors and significance levels (columns *(c)* and *(d)*), calculated in SPSS. Following the approach taken by Millward and Bell, we multiply the coefficient *(b)* by the aggregate change in each covariate *(f)* for our rural RDs to show the change in IMR which it contributed *(g)*. Finally we divide this result by the total change in IMR over the period in question to show what proportion was due to each covariate *(h)*. We now review the results of each model run in turn. In each case the 'Decade' row captures the change in IMR which the model cannot attribute to one of the other variables and by default is linked to the passage of time.

Table 2: Estimates of Effects on IMR: population density, fertility and maternal health: 1850s-1900s

(a)	*(b)*	*(c)*	*(d)*		*(f)*	*(g)*	*(h)*
Parameter	Estimate	Std. Error	Sig.		change in variable	effect on IMR *(b*f)*	% of total change in IMR
Intercept	89.77	4.52	0.000				
Popden	0.12	0.02	0.000		7.34	0.89	-2.6
Fertil	26.46	6.72	0.000		-0.06	-1.71	5.0
femTB	3.02	0.46	0.000		-3.01	-9.08	26.4
Decade	-4.22	0.40	0.000		5.00	-21.11	61.3
distance	0.07	0.01	0.000				

Table 2 shows the estimated effects of population density, fertility, and maternal health on infant mortality. The figures in column *(g)* are divided by the total change in the aggregate IMR for our rural districts between the 1850s and 1900s of -34.44 to give the percentages in column *(h)*, the proportion of change in IMR associated with each covariate. We first note that the effect of change in population density over time is not only very small but also has an unexpected sign (-2.6 per cent). For practical purposes it can be ruled out as an influence on change in IMR over time, at least for areas remaining rural, which are the subject of this study. Ecological studies (of variations between places at one point in time) have previously found an important role for population density:[55] however, Williams and Galley caution

[55] Ibid., pp. 127-8.

against imagining that the factors influencing these spatial variations must also be the ones governing temporal change.[56]

It is likely that, beyond a certain threshold, rising population density would indeed raise IMR (above all, by exposing infants to greater risk of diarrhoeal disease) unless the quality of sanitation kept up with population growth. Our findings suggest, however, that this threshold was higher than the one we used to define rural, 183 persons per square kilometre. This is consistent with Williams and Galley's demonstration that infant mortality to diarrhoeal disease was very much lower in rural than urban RDs.[57] Rural sanitation was often as bad as urban, or worse.[58] Yet our findings support Williams' and Galley's conclusion that, at rural population densities, bad sanitation did not translate into high mortality from diarrhoeal disease. Focussing on rural areas suggests that RD-level population density is an imprecise measure for the real cause of the urban harm, crowding at street and neighbourhood level, which raised the proportion of infants living in dangerous proximity to human waste.

Turning to the effects of fertility, we find that the impact on IMR of its change over time was, again, small (5 per cent). This is another interesting result, given the significance of fertility in ecological studies of IMR determinants such as Woods et al's.[59] Again, a predictor of IMR variation between places does not appear to be a strong predictor of change in one location over time.

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[56] Williams and Galley, 'Urban-rural differentials', 417.

[57] Ibid, 419-20

[58] Waddington, '"It might not be a nuisance"', p. 192; Reay, *Rural Englands*, p. 106.

[59] Woods et al, 'Causes of rapid infant mortality decline part 2', pp. 121-26.

Fertility's raw coefficient is quite large (26.5) but it needs to be recalled that Coale's I_f is an index of observed fertility divided by his estimate of 'natural' fertility, so its values run from zero to one. Thus the impact of any plausible change in fertility is not so large as this coefficient implies. For the 363 rural RDs in the study, I_f was 0.364 in the 1850s, actually increased marginally in the 1860s and 1870s, was still above its 1850s value in the 1880s, and declined by only 22 per cent from its 1870s peak to its 1900s value of 0.299, a reduction rounded in Table 2 to 0.06. The message of Table 2 is that the impact of fertility change in rural areas was so small because not only did IMR respond only rather weakly to fertility change, but also the fertility change occurring was rather small. It should be noted here that Szreter has identified multiple fertility declines and shown that the reduction in many types of urban setting, especially textile towns, will have been faster and begun earlier.[60] These urban-rural fertility differences contributed to urban-rural differences in the behaviour of IMR.

About a quarter (26.4 per cent) of changing IMR was associated with female tuberculosis mortality during the fertile period, a proxy measure of maternal health. This striking result provides strong support for Millward and Bell, confirming their results with a much larger study, and showing that these held good in rural places (which provided only about a sixth of their data). Millward and Bell argued that improving maternal health could account for almost half of the IMR decline between the 1870s and the 1900s: our findings are consistent with a change of this magnitude, the difference

[60] Szreter, *Fertilty, class and gender*, ch. 7.

between our estimates and theirs explained in part by the use of different sets of places as well as a different period.[61]

We now consider the contribution of falling female illiteracy to IMR decline, using this as a proxy measure of female education. The restricted availability of literacy data, noted above, compels us to run the model for a shorter period, the 1850s to the 1880s: Table 3 shows the results. The change in aggregate IMR used to calculate column *(h)* is -20.02 for this period.

Table 3: Estimates of Effects on IMR: population density, fertility, maternal health, and female illiteracy: 1850s-1880s

(a)	*(b)*	*(c)*	*(d)*		*(f)*	*(g)*	*(h)*
Parameter	Estimate	Std. Error	Sig.		change in variable	effect on IMR *(b*f)*	% of total change in IMR
Intercept	86.59	5.38	0.000				
Popden	0.17	0.02	0.000		2.48	0.43	-2.2
Fertil	2.16	9.74	0.824				
femTB	4.11	0.46	0.000		-1.81	-7.44	37.2
Femillit	0.13	0.03	0.000		-26.77	-3.41	17.0
Decade	-3.28	0.52	0.000		3.00	-9.85	49.2
Distance	0.05	0.01	0.000				

[61] Millward and Bell, ‘Infant mortality’, tab. A1, p. 729.

The estimated effects of population density and tuberculosis are fairly robust, and in addition we now see that about a sixth of changing IMR (17 per cent) was associated with changes in female literacy. This is the first study of which we are aware which has been able to give a weight to the effect of improving education. Adding maternal education to maternal health allows us to account for about half of all change in IMR over time within an RD. Adding female literacy to the model causes fertility to cease to be statistically significant. This suggests, as Woods et al and much of the literature on modern less developed countries have also thought, that fertility is mediating the effect of literacy on IMR. In other words, literate women with their higher status achieved greater agency and were more successful in securing their fertility goals, in this case family limitation.

A third model run (not reported here) was necessary to investigate our final level one variable, agricultural wages. The coefficient produced showed IMR tending to rise with labourers' wages. No plausible causal mechanism would link the two directly in this way: we conclude that some underlying factor(s) contributed to both rising wages and rising IMR. We also investigated the possible impact of improving communications with a model run which included a measure of the density of railway coverage each decade: the results were unconvincing.

We tested inter-relationships between these time-dependent variables. Population density had a significant but small effect on female tuberculosis, explaining 5% of its decrease. The effect of fertility on female tuberculosis was significant at the .1 level but not the .01 level: its magnitude would account for 24% of the reduction in female tuberculosis: thus fertility probably affected IMR indirectly by this route, as well as directly. The effect of falling tuberculosis on fertility was significant, and would have caused it to rise if other factors affecting fertility had not outweighed this. Literacy did not have a significant effect on tuberculosis, or on fertility.

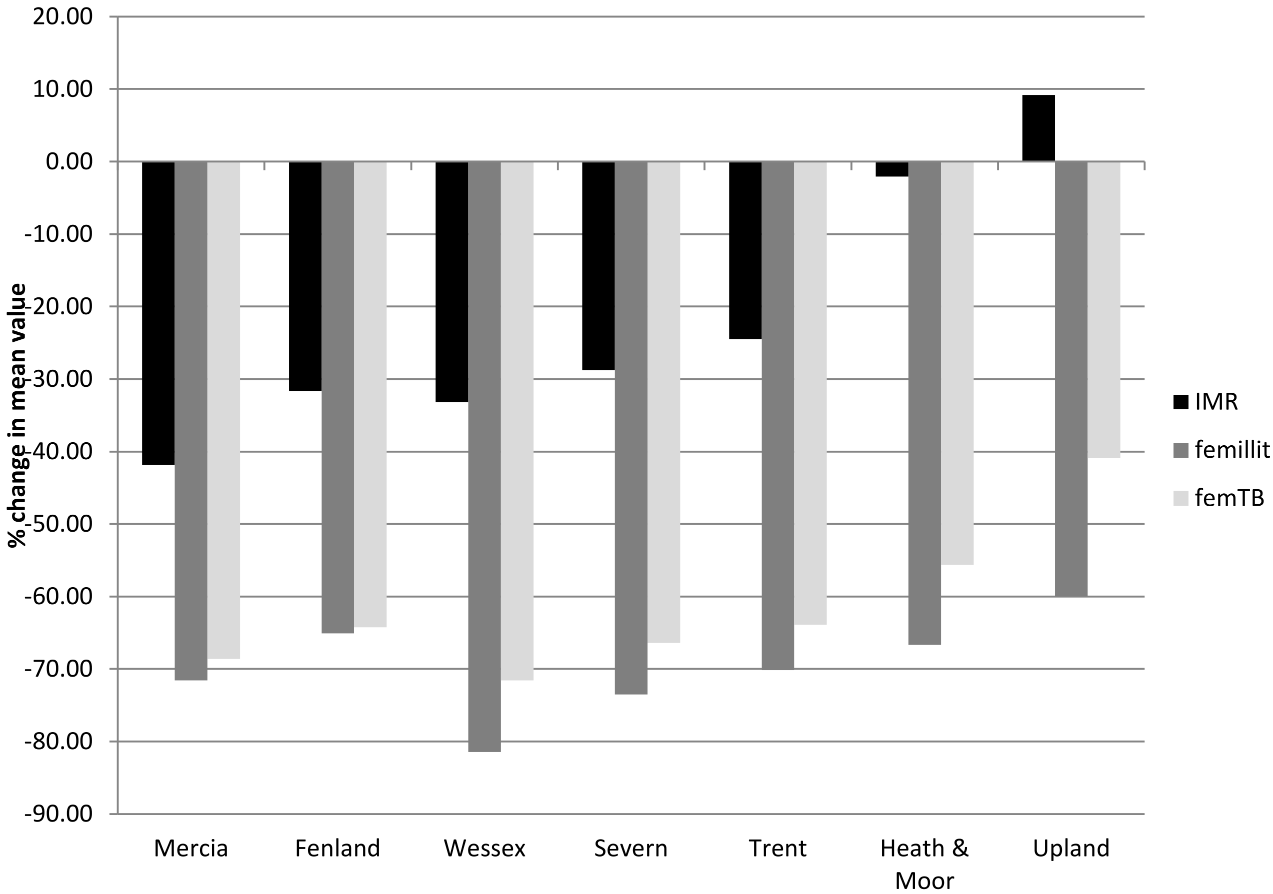
The remaining variable to report is distance from London, which achieved high significance in every model run. The 'distance' coefficient in the model is the contribution of distance to change in IMR over the period after taking account of the effects of the other covariates. Taking the value from Table 2, its effect size was an increase in IMR of seven deaths per thousand births for each hundred kilometres from London. We examined whether the distance effect applied only to the most peripheral places, turning distance into a category variable by splitting distance from London into 50-kilometre bands. IMR increased with distance from London from one band to the next at all distances over 100 kilometres, a finding consistent with Gregory's 2008 results.[62]

V

We noted in section II that rural England and Wales had identifiable IMR clusters. It is now possible to discuss how IMR interacted at the cluster level with the covariates discussed here. Figure 7 shows the movements in IMR along with the two covariates found to be most important in the previous discussion, female tuberculosis (health) and female illiteracy (education).

[62] Gregory, 'Different places', pp. 781-4.

Figure 7: Change in IMR, female tuberculosis, and female illiteracy by cluster, 1850s-1900s



Sources: Derived from GHBGIS, Friedlander and Okun, '*Demographic Processes*' database and *Forty-Seventh Annual Report of the Registrar General* (P.P. 1886, XVII), pp. 2-25.

Figure 7 illustrates the strong relationship between these three variables which has just been demonstrated. More than this, though, it allows us to see some variation between clusters in the strength of this relationship. In the clusters we label 'Mercia' and 'Fenland', IMR reduced by somewhat more than improvements in health and education predicted. We have already seen, and Figure 2 shows, that the IMR reduction could be so great here because the starting figure was so high: the 'Fenland penalty' discussed by

Sneddon and by Hinde & Fairhurst.[63] This penalty gradually dissipated, not lasting long beyond the 1870s in Sneddon's view. Reay points out that the link between a marshy place (or a recently drained one with labour-intensive agriculture) and high IMR applied wherever such places were found in southeast England, not only the Fens:[64] on the strength of our analysis we suggest it occurred in low-lying parts of East Yorkshire too. The special factors at work at the start of our period, according to Hinde and Fairhurst, were a combination of damp environment plus two transitory factors: the intensive agricultural employment of women in circumstances where they could not make adequate provision for the care of their infants, and a statistical artefact whereby in-migration of young women with infants inflated the number of infant deaths but not the denominator of IMR, the number of births in the area.[65] This migration, Sneddon added, could have created worse overcrowding within households than in other rural areas.[66] The evolution of Fenland agriculture after the 1870s to less distinctive forms produced a strong improvement in IMR by diminishing the two transitory factors just noted.

The three clusters of Wessex, Severn, and Trent occupied the middle of the IMR distribution in the 1850s. The other characteristic they shared was an IMR performance closest to that predicted by maternal health and education. This broad region of central and southern England was the area where the

[63] Sneddon, 'Double penalty?'; Hinde and Fairhurst, 'Why was infant mortality so high?'.

[64] Reay, *Rural Englands*, pp. 108-9.

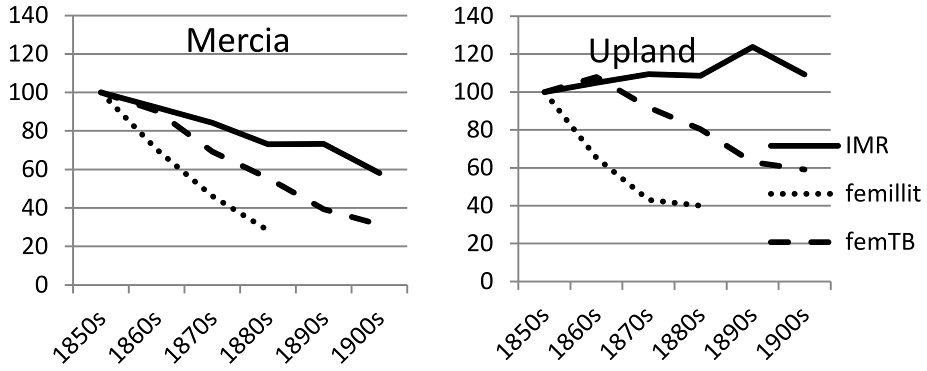
[65] Hinde and Fairhurst, 'Why was infant mortality so high?', pp. 65-66.

[66] Sneddon, 'Double penalty?', p. 96.

rural IMR trajectory was closest to that of rural England and Wales as a whole. It is time to supersede the binary comparison of north and west versus south and east with a threefold scheme, in which rural IMR, and its responsiveness to maternal health and education, performed best in the east, moderately in the centre, and worst in the north and west. Within the central zone itself, Figures 2 and 3 show that there was a rising south-north IMR gradient, persistent over time, with the RDs of Wessex averaging IMRs about 10 per thousand lower than Severn, which in turn came about 15 per thousand below Trent. One component of this gradient was the distance from London effect: the consistently good performance of Dorset, north Hampshire and much of Wiltshire was another.

The final two clusters, Heath & Moor and Upland, represent the opposite end of the scale from Mercia and Fenland, since their IMR performance was the worst, and also worse than the development of their health and education status predicted, as Figure 8 shows by contrasting the trajectories of Mercia and Upland. Situated very largely in the pastoral zone, in Wales, the far north of England, and Devon, the final two clusters began with the best IMRs. Heath and Moor's stagnated, but ended up still quite near the bottom of the IMR distribution: Upland's deteriorated and finished at the top (Figure 2). One reason for these two clusters' relatively small improvement in tuberculosis mortality (Figures 7 and 8) may be that they started the period closer to the lowest achievable level than the other clusters.

Figure 8: Relative change in IMR and its covariates in Mercia and Upland: 1850s-1900s



All variables indexed so that 1850s = 100
Source: as for figure 7

It appears that the relative advantage enjoyed by Upland and Heath & Moor in mid-nineteenth century was linked to their pastoral agriculture. This preserved an eighteenth-century and older 'family economy' longer than it lasted in the more capitalised farming of the arable zone, as Reay's calculations of the 'family labour' share of the agricultural workforce confirm.[67] Snell saw this family economy as creating a more equal sexual division of labour because it included more access to commons, more owner-occupiers and small tenants, and less dependence on wage labour away from home:[68] Verdon's studies of farm records tell the same story.[69] Our examination of maternal health and education suggests that this social structure gave mothers higher status and safeguarded their infants better.

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[67] Reay, *Rural Englands*, p. 24.

[68] Snell, *Annals of the labouring poor*, p. 62.

[69] Verdon, *Rural women workers*, p. 123.

This leaves the challenge of why these northern and western zones did so poorly after mid-century. Distance from London was relevant. Rural out-migration, generally heaviest in the most peripheral places,[70] will have exerted upward pressure on infant mortality there, as out-migrants (male and female) were better off, better educated, and better informed than stayers, [71] and must therefore have been healthier too. Nineteenth-century developments in the cultures of domesticity and respectability could also have played a part.[72] These changes reduced women's access to the labour market, with results for infant welfare which have been controversial,[73] but within the household they improved mothers' status, with unequivocally positive outcomes for infants. We speculate that there was a spatial gradient to these changes, diffusing from socially fluid urban settings, especially London to the most peripheral rural areas, the delay causing infant mortality decline to fall behind there.

VI

This first longitudinal analysis of rural IMR has incorporated features of earlier work such as Woods et al's choice of fertility and literacy measures and Millward and Bell's use of female tuberculosis mortality to proxy

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[70] Lawton, 'Rural depopulation', p. 243; Hunt, *Regional wage variations*, pp. 247-51; Baines, *Migration in a mature economy*, pp. 283-298.

[71] Short, 'Rural demography', pp. 1285-7.

[72] Verdon, *Rural women workers*, p. 199.

[73] Dyhouse, 'Working Class Mothers'; Garrett and Reid, 'Satanic mills'.

maternal health.[74] By replicating these elements of their models we allow some comparisons with their findings. While Woods et al showed that fertility was a significant predictor of IMR between places at one point in time, we found it a poor predictor of change over time in one place. On the other hand we confirmed and extended the findings that maternal health and literacy were important predictors of IMR change.

This work has demonstrated the feasibility of using the data in the GBHGIS for longitudinal statistical analysis. This resource is just as valuable to scholars studying small geographical areas as to those looking at the national picture. Our findings suggest fruitful areas for further work. These include the contributions of the status of women and migration. We lack direct evidence on how out-migration affected the health of rural areas. Local studies of different kinds have the most to tell us about the varying social and political conditions which shaped women’s status, and with it, fertility and infant mortality. As well as benefitting from the demographic material in the GBHGIS and the new wealth of raw Census resources available through I-CeM, [75] local studies could use the forthcoming Atlas of Fertility Decline in England and Wales and the quantitative parish-level data in the Agricultural Returns.[76]

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[74] Woods et al, ‘Causes of rapid infant mortality decline part 2’, p. 127; Woods, *Demography of Victorian England and Wales*, p. 9; Millward and Bell, ‘Infant mortality’, pp. 714-16.

[75] Schürer and Higgs, ‘*Integrated Census Microdata*’ database.

[76] http://www.geog.cam.ac.uk/research/projects/victorianfertilitydecline/ (accessed 6 November 2015).

Infant mortality varied systematically across rural England and Wales in the later nineteenth century. Its most important covariates over time were maternal health, responsible for about a quarter of IMR change, and maternal education, contributing a further sixth. Overall, using all the leading variables suggested by earlier work, we account for only half of the change in IMR over time. The remaining influences are more resistant to quantitative study, but our findings about the importance of peripherality offer clues. The speed of improvement in IMR reduced with distance from London at all ranges of more than about 100 kilometres. IMR responded most strongly to improving health and education in the east, less in the central area, and least in the north and west. The eastern zone's higher than average mid-century infant mortality therefore declined faster than the national average, due to the erosion of earlier disadvantages created by the opening up of labour-intensive agriculture on reclaimed wetland. A central and southern zone had slightly lower IMRs than the eastern zone in mid-century but did not keep up with the rate of decline in the east. The peripheral north and west, where the longer survival of the family economy gave women a stronger position in the household, had the lowest mid-century rates. Thereafter, the diffusion of cultural and social change from centre to periphery eroded this advantage as the status of mothers elsewhere caught up with that in the north and west.

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