The Urban Penalty and the Population History of England

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This paper presents some speculations on the significance of urban mortality for the study of what McKeown christened "the modern rise of population".[1] One school of development-studies presents the links between population growth and wider social and economic changes within a model of the demographic transition. For these scholars, modernisation is associated with technological changes which in turn allow industrialisation, urbanisation, demographic transition and population growth. Technology is considered central because in raising agricultural productivity it releases people from the land. They can devote themselves to working at other things than producing food and may even move into towns to do so. At the same time, the easier availability of food may relax the positive check on population growth allowing a change from a high-pressure demographic régime of high-fertility/high-mortality to a low-pressure one of low-fertility/low-mortality. If the improvements in mortality are realised before the compensating fall in fertility, then, population growth will accompany the lagged transition.

Population growth and urbanisation over the "long" eighteenth century, 1670-1820

As a highly generalised model of the economic and demographic history of western Europe since 1700 and of more recent changes in the poor countries of today, the theory of the demographic transition is superficially attractive and promises to forge a link between economic history and development studies. Of course things were and are not quite this straigthforward and economic historians have drawn attention to a number of problems with the theory of the demographic transition. On one hand, they point out that pre-industrial Europe was never characterised by the sort of demographic systems now prevailing in many poor countries. Fertility was never persistently at what we would now recognise as high levels and, in the medium term, neither was mortality. Well before the industrial revolution, Europeans held back population growth, accumulating goods during better times and restraining fertility during worse. This control was exercised through nuptiality with Europeans

having high rates of celibacy and marrying relatively late. Pre-industrial Europe was, as Jones concludes, already a low-pressure demographic régime compared to contemporary and modern Asia.[2]

A second source of empirical embarrassment for the theory of the demographic transition follows on from this and comes with the finding that English population changes in the period 1541-1871 were fuelled more by variations in fertility than by fluctuations in levels of mortality. Indeed, the leap in the total English population from about five million in the 1670s/1680s to about eleven-and-a-half million in the 1810s/1820s, the first leg of England's modern rise of population, was produced by a rise in fertility rather than by a lagged fall in mortality and then fertility: "the fertility rise contributed about two-and-a-half times as much to the rise in growth rates as the mortality fall."[3] Wrigley and Schofield show further that this rise in fertility was brought about by falls in both the age at first marriage and in the level of celibacy. In thus stressing the preventive rather than positive check on population growth, Wrigley and Schofield conclude that the economic history of pre-industrial England must now be looked at in a more positive light than when the period was seen as one in which people were pressed against the subsistence limit, buffeted by the impersonal forces of bugs and breezes.

Wrigley and Schofield's findings change our understanding of both the behavioural and the economic context of the population dynamics of pre-industrial England. If population changes were determined by autonomous fluctuations in mortality, then, the prime moves in the demographic system appear to have been beyond human choice and the pre-industrial English population could faithfully be presented as ground down between biological urges and environmental scourges. Should the relations between economy and demography run along the tracks of preventive rather than positive checks, then, the English people chose their own adjustment to available resources rather than having one imposed upon them. In decisively shifting the weight of evidence towards the second of these sets of possibilities, Wrigley and Schofield have restored the dignity of choice to the people of pre-industrial England. Turning from behaviour to economy, the implications here are equally profound. It is clear that the sustained rise in the rate of population growth in the eighteenth century was something very special. It implies such dramatic improvements in agricultural productivity that people were repeatedly able to choose higher fertility when, as Jones suggests, they had a general predilection for chattels over children during times of plenty. It was also achieved without bringing in its train a compensating rise in the cost of living, such as might have been expected to reverse the gains of the early eighteenth century. Yet population growth and stable prices were not the only evidence of increased productivity for they were accompanied by a significant rise in the share of the population not directly engaged in producing food. This is of both economic and demographic moment. Economically, the

crucial variable is the ratio between people who produce food and those who do not. Leaving aside the question of age-specific dependence (the dependency ratio), the non-food-producers of pre-industrial England may be divided into a rural and an urban group. The rural artisans formed, suggests Wrigley, an increasingly substantial part of those living in the countryside, rising from 20% of the rural population in 1520, to 30% in 1670, to 50% in 1801.[4] In addition, practically all town-dwellers produced little or no food and to the rural non-food producers may be added the proportion of the total population living in places of 5,000 inhabitants or more, 5.25% in 1520, 13.5% in 1670 and 27.5% in 1801. In other words the food producers made up approximately four-fifths of the population in 1520, three-fifths in 1670 and one-third in 1801, leaving the rest to follow other pursuits. The demographic importance of this fall in the share of the people producing food relates to the different characteristics of urban and rural populations. This is where the debate over the role of mortality in English population growth might be re-opened.

On the face of it, the current state of scholarship in economic/demographic history appears to relegate mortality to a very lowly position in the explanation of English population growth. Of course, this directly challenges the long-held view of McKeown that declines in mortality were central to the modern rise of population. Wrigley and Schofield are in no doubt where their work leaves McKeown's arguments and note at one point: "the view that mortality played the dominant role in determining changes in population growth rates, whose most recent champion has been McKeown, must now be set aside so far as English demographic history in early modern times is concerned."[5] Certainly, McKeown's argument was rather simplistic and was spread across the whole period from 1700 to 1940.[6] Nevertheless, it is worth reconsidering the status of McKeown's conclusions in the light of the works summarised above since they have such wide currency.[7]

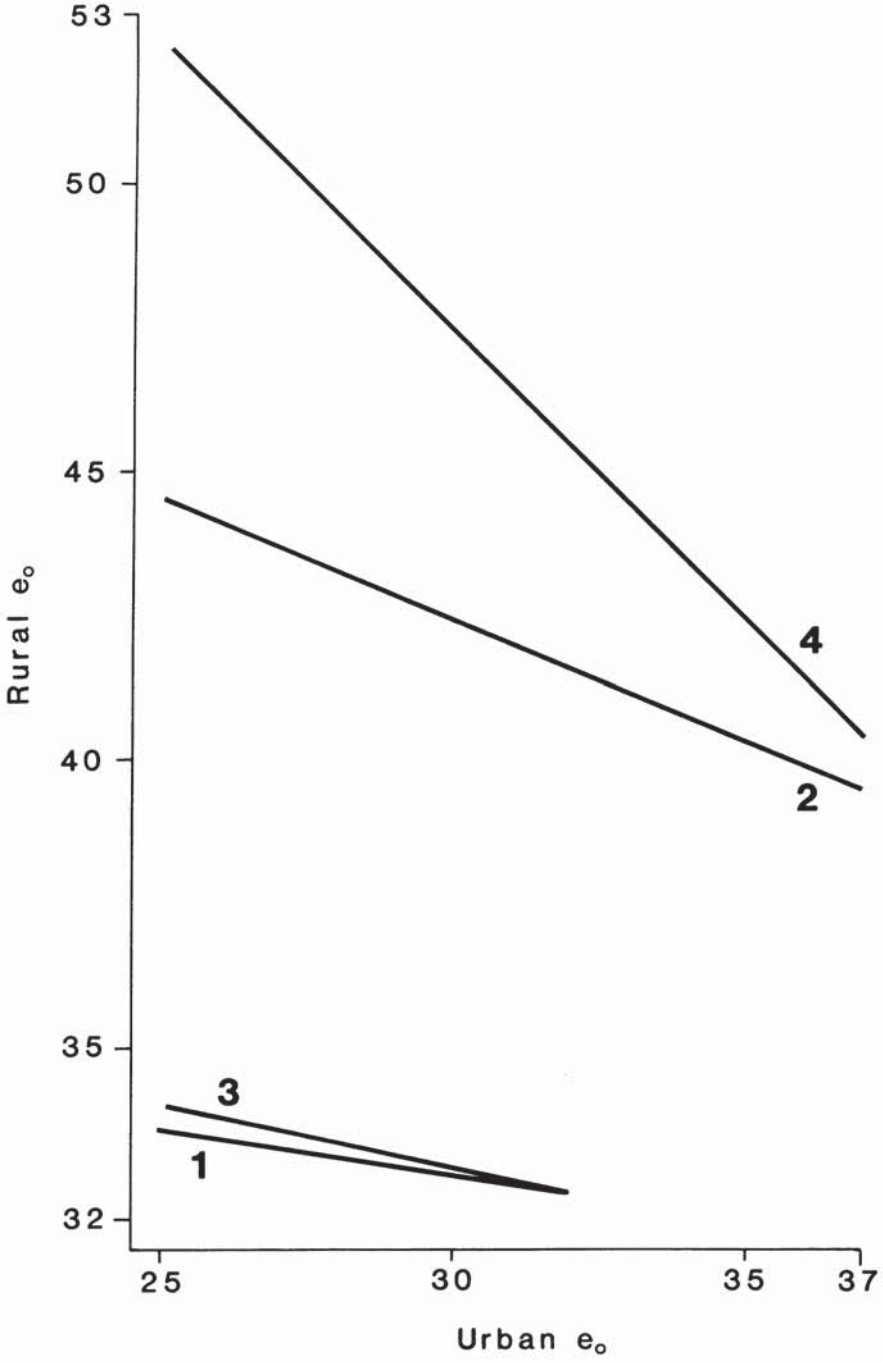
McKeown's argument is quite straightforward. In pre-industrial Europe, levels of fertility were at the biological maximum leaving no room for fertility-induced increases in population growth. Consequently, the European beginnings of the modern rise of population must have been the result of some relaxation of the mortality constraint. In other words, levels of mortality in pre-industrial Europe must have been substantially higher than those of the early nineteenth century. Yet while McKeown thus placed great emphasis on questions of health and disease in accounting for demographic development, in explaining declining mortality he laid little of the credit at the door of the medical profession. For whatever period he considered, the conclusion was the same: mortality has been more strongly influenced by diet than by medical care. McKeown said that he proceeded on the basis that "when we have eliminated the impossible, whatever remains, however improbable, must be the truth."[8] The problems for McKeown begin with the very first elimination in the argument. McKeown said nothing about marriage. Although he considered and dismissed abstention from intercourse in marriage, prolonged

lactation, mechanical birth control, abortion and infanticide as effective controls on fertility in pre-industrial England, he ignored celibacy and age at marriage altogether. Hajnal has subsequently shown these to be the axes of a distinctive European marriage pattern.[9]

There is, it seems, no basis for continuing to accept McKeown's dismissal of the possibility of the preventive check in pre-industrial England. If the case for considering mortality when explaining population growth is to be established by elimination then it must fail. On the other hand, Wrigley and Schofield have made a very strong positive case for giving pride of place to fertility for the "long" eighteenth century. A positive case can be made, however, that mortality's importance is under-estimated in their work because they model a national population which in fact consisted of at least two distinct régimes (urban and rural), the relative weights of which changed substantially over this period. The rise in English population from 2.4 millions in 1520 to 5 millions in 1670 to 8.7 millions in 1800, on Wrigley and Schofield's estimates, was accompanied by substantial urbanisation, as detailed above. It is also apparent that urban living carried with it a significant demographic penalty. Rates of mortality in towns were sometimes substantially greater than those of the countryside. As Wrigley and Schofield remark: In the past, high density frequently brought high mortality in its turn. The absolute level of the mortality rates in Hartland /a rural coastal parish in Devon/ and Gainsborough /a market town in Lincolnshire/ differed by a factor of between two and three for the most part, a remarkable example of the variable incidence of mortality in pre-industrial England. Translated into rough estimates of expectation of life at birth, the two sets of mortality rates suggest that in Hartland in 1600-1749 it may have been 50 years or more at a time when it was only 30 years in Gainsborough.[10]

This geographical variability is greater than the improvement of aggregate mortality during England's eighteenth-century growth spurt: "Expectation of life at birth, which averaged only 32.4 years in the 1670s and 1680s at the start of the "long" eighteenth century had risen to an average of 38.7 years in the 1810s and 1820s at its end."[11] The geographical variability is also persistent and in 1861 is of the same order as in 1600-1749.[12] It is the relative stability of mortality in the aggregate which is striking when set alongside internal variations. Variation across space is more significant than variation over time. In relative terms one can speak of a low-mortality rural system and a high-mortality urban system. If urbanisation was associated with no change in the severity of the urban penalty, then, other things being equal, since a greater share of the population would be living in high risk areas, aggregate mortality should rise. Whereas if urbanisation is accompanied by relatively stable levels of aggregate mortality, then, it is possible either that rural mortality is falling to compensate or that the urban penalty is decreasing in severity.

Figure 1. Some possible combinations of urban and rural life expectancies for different levels of urbanisation.



Hartland was clearly exceptional in 1600-1749 and there was certainly some scope for movement of general levels of rural mortality towards this low value over the eighteenth century. Gainsborough had a population of less than 5000 in 1600-1749 and has not even featured in the figures for urbanisation given above. If the urban penalty is paid in places of fewer than 5000 inhabitants then the proportion of the population in high risk areas obviously increases. For example, if Law's cut-off point of 2,500 is taken, then the urban share of 27.5% for places of 5000 or more in 1801 rises to 45% on this more generous definition.[13]

Given that the national life expectancy is the average of the rural and urban values weighted in proportion to their respective shares of the total population, the reasonable range of values of urban and rural life expectancies is easily estimated. Four situations are shown in Figure 1.

1. With national expectation of life at birth 32.4 years and 13.5% of the population living in towns, this illustrates the possible relations for 1670s/1680s on the assumption that the urban penalty is paid only in places with 5,000 inhabitants or more. Here the bulk of the population is rural and rural life expectancy can not depart too much from the national figure showing just how remarkable Hartland would have been at the time.

2. Should the proportion urbanised increase to 30% and the national life expectancy rise to 38.7 years (the situation by the 1810s/1820s if 5,000 is again the cut-off point), the likely combinations are dramatically changed. The rural share can reasonably be allowed to fluctuate a little further from the national average. If, for example, the urban life expectancy remained at 30 years over the "long" eighteenth century, to produce the observed rise in national life expectancy the rural value would need to have changed from 32.7 to 42.4, that is, about half as much again as the national improvement and the rural-urban differential would have gone from 2.7 years to 12.4 years. Woods gives a rural life expectancy of 42 years for 1821 for England and Wales, using 10,000 as the population defining urban areas, so that the improvement in rural mortality indicated by these assumptions is at least plausible.[14] Perhaps the rest of rural England was indeed catching up with Hartland and thus offsetting the urban penalty.

3. and 4. If, however, the urban penalty is paid in places of as few as 2,500 inhabitants, the rural improvement needs to have been even more dramatic. In the 1810s/1820s the share of the population in such towns was about 50% and if we guess at 18% for 1670s/1680s (18:13.5 = 50:30; although in fact the precise guess made makes little difference to the calculations for the earlier period), over this period a constant urban life expectancy of 30 years would have needed a rural improvement from 32.9 years to 47.4 years to secure a national change in life expectancy equal to that observed. This would have taken up all the slack between Hartland and the rest of rural England and is an almost incredible amelioration of mortality extending rural life expectancies by more than twice the national average.

Table 1. Some possible contrasts between urban and rural demographic régimes. * Assumed, □ read from graph.

	Quinquen. fertility mortality		Weighted average			Urban values			Rural values (Urban > 5,000)			Rural values (Urban > 2,500)		
	GRR	e_0	GRR	e_0	r	GRR	e_0	r	GRR	e_0	r	GRR	e_0	r
1676	1.906	36.37												
1681	1.939	28.47												
1686	2.170	31.77												
1674-1688			2.004	32.23	-0.18□	2.004*	30*	-0.40□	2.004*	32.57	-0.16□	2.004*	32.71	.0.14□
1816	3.056	37.86												
1821	2.981	39.24												
1826	2.885	39.92												
1814-1828			2.929	39.06	1.75□	2.959*	30*	0.90□	2.959*	42.94	1.95□	2.959*	48.12	2.20□

Figure 2. Mortality, fertility and population growth, some possible combinations 1670-1820.

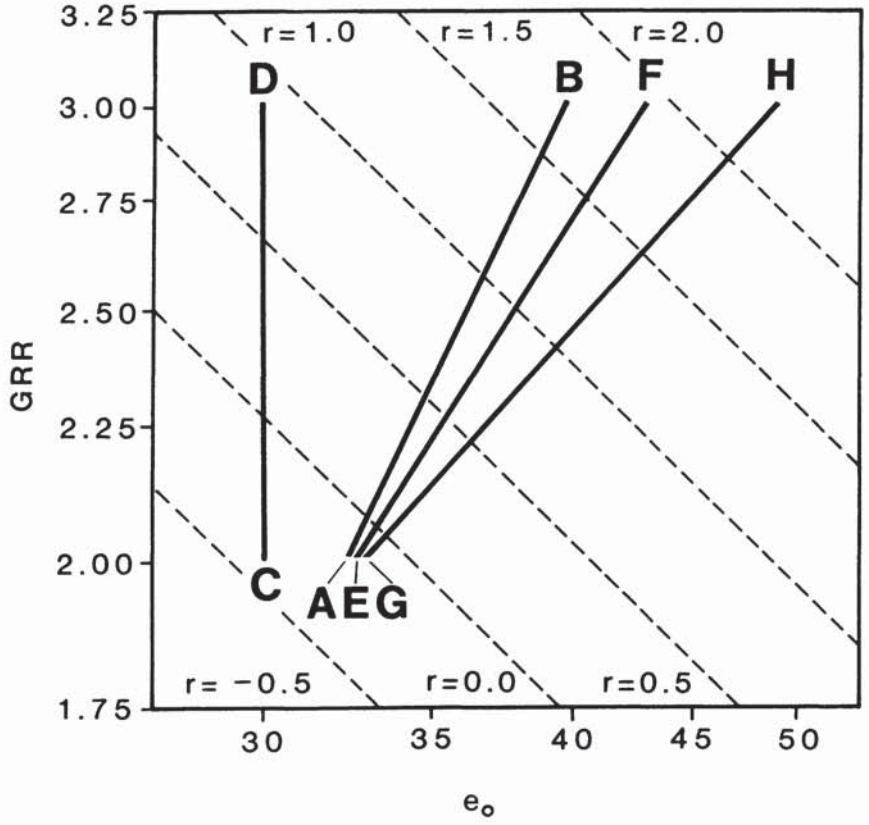


Figure 2 is a simplistic attempt to illustrate some implications of these alternatives. AB is the national change in vital rates (GRR — gross reproduction rate; eo — expectation of life at birth) over the "long" eighteenth century taking a 3-point weighted average of quinquennial rates centred on 1681 for its start and 1811 at its close, using Wrigley and Schofield's data and their ingenious graph.[15] The gradient of the line is 2 so that fertility contributed about twice as much as mortality to the observed change in growth rates (r — intrinsic rate of growth). If the same fertility rates are ascribed to urban and rural populations but we make adjustments to their respective rates of mortality, it is possible to show some of the possible consequences of the argument above. If urban life expectancy remains unchanged at 30 (CD), then, the rate of intrinsic rate of growth of this group would of course have been lower than the national total at both the beginning and the end of the period (see Table 1). Two alternative rural trajectories are shown on the basis of a cut-off point for the urbanised population of 5,000 (EF) and, more generously, 2,500

(GH). The assumption of common rates of fertility means that the higher than average rural life expectancies take the intrinsic growth rate of the rural population way past the national total by the end of the period. More intriguingly, these assumptions change the gradient of the line so that on a modest definition of the urban share, with no significant improvement in urban mortality and comparable fertility in urban and rural areas, the change in rural intrinsic rates of growth would still have been based more on fertility than mortality, but now in ratio 1.5:1. However, the broader definition of urban, with the same assumptions about general fertility and urban mortality, requires that the change in rural growth rates be the product equally of fertility and mortality improvements.

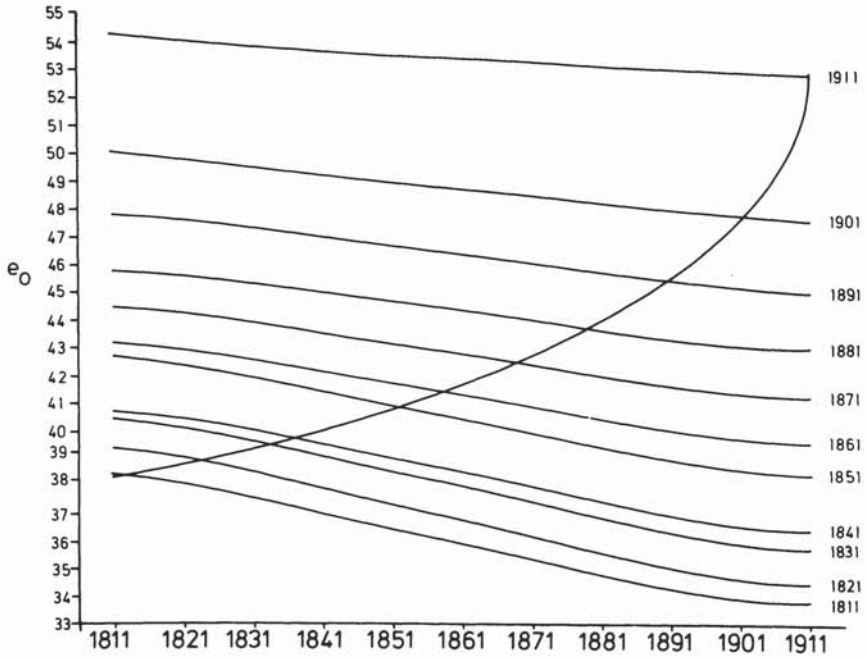
Urbanisation, therefore, poses certain problems for studies of the population changes of national aggregates. National averages are valid summary measures in synchronic studies and in many cases are the only parameters which can be reliably calibrated given the lack of information of internal migration. In diachronic studies they are at best a convenient fiction applying to a different entity at different times. It is likely that if England is modelled as one population over the period of its impressive growth spurt, the "long" eighteenth century, then, the significant changes in the mix of high- and low-risk peoples in this aggregate hides the true importance of mortality changes in relation to changes in fertility. If rural and urban fertility differ to a markedly lesser degree than do rural and urban mortality, then it will indeed be specifically the mortality variable which is passed-over too lightly in national studies. For English population history in the early modern period, the importance of changes in mortality in fuelling population growth, therefore, is perfectly compatible with significant contemporary shifts in fertility and even with such shifts being apparently more significant at the national level.

Mortality changes and urbanisation in the nineteenth century

If the rate of urban mortality at the start of the nineteenth century was similar to the national average for rural and urban England in the late seventeenth century, then, the increase in the level of urbanisation over this period leads the national figures to understate the improvement in rural mortality. For the first half of the nineteenth century, the aggregate mortality improvement is comparable to that of the whole eighteenth century. On Woods' estimates, this now downplays a more signficant decline in urban mortality. The national life expectancy at birth for England and Wales, according to Woods, was extended from 38 years to 41 years during the period 1811 to 1861. Londoners experienced a more dramatic improvement, their life expectancy rising from 30 to 37 years. For large towns (with populations of over 100,000), the figures were 30 and 35 and other towns (populations of between 10,000 and 100,000) saw a change from 32 to 40. Only the rural (residual) areas (1811: 41; 1861: 45) appear to replicate the more sluggish national figures. The urban

leap forward is pulled back by the transfer of a larger share of the population into these high-risk areas. The urban areas have to run if the national figures are even to stand still. Figure 3 is a simple illustration of this. The line going from the lower left-hand part of the graph to the upper-right shows the progress of national life expectancy at birth from 38 to 53 over the century 1811 to 1911.[16] The other lines present national averages based on the respective mortalities of the four groups (London, large towns, other towns, rural) at different dates applied to the changing proportions of the population actually in those groups at different times. Thus if the share of the population in towns grew as it did over the nineteenth century but the urban penalty had remained constant, then, the national average life expectancy would have deteriorated in line with the transfer of people into high risk areas. By 1911 urbanisation would have reduced life expectancy at birth from the 38 of 1811 to 33, whereas in fact it actually rose to 53. Similarly, if the mortality rates of 1911 had coincided with the lower urban shares of 1811, then, eo would have been greater than 53 but, because the urban penalty was so much lower by 1911, the improvement induced by shifting people out of relatively-high risk areas is less dramatic and eo would have risen only by a further two years.

Figure 3. The interaction of urbanisation and life expectancy at birth; England and Wales, 1811-1911.



A central feature of any explanation of mortality changes over the nineteenth century must be an account of the near removal of the urban penalty. In 1811 when people in urban areas (over 10,000 in population) had a life expectancy of 31 years, rural areas were ten years better-off at 41, by 1861 the gap was seven years (38 and 45 years) and in 1911 it was only three (55 and 52). Yet when McKeown and Record consider the matter they conclude that general economic conditions as they affect diet and nutrition were primarily responsible for at least half the decline in national mortality over the second half of the nineteenth century. This implies either that the specifically urban penalty is poor diet or that the mortality changes of rural areas were so overwhelmingly diet-based that they swamped the specifically urban developments. As a prelude to exploring these issues, the rest of this paper considers how McKeown and Record establish a conclusion which is widely accepted as an axiom by sociologists, economists and historians.

McKeown and Record's approach is engagingly direct. For England and Wales in two decades (1851-1860 and 1891-1900), they provide death-rates for various causes standardised to the age structure of the population of 1901. They give the share of certain causes in the aggregate decline. They associate sets of causes with particular controlling factors and thereby establish the priority of certain factors in accounting for the decline in mortality in England and Wales over the second half of the nineteenth century. McKeown and Record compute the fall in standardised mortality to be about 3 per thousand over this period and the fall in tuberculosis rates to be equivalent to one half of this, and thus diet is set in place as primum mobile. After this comes the sanitary revolution with a third of the decline accountable to fevers and diarrhoeal diseases. A fifth is due to a spontaneous change in the virility of a disease organism (scarlet fever). Finally, medical intervention (vaccination) is credited with the one-fifteenth of the fall set down to smallpox. Doctors are relegated behind bread, brushes and bugs.[17] Have McKeown and Record correctly identified the salient features of the mortality decline and have they explained them adequately? An analysis along the broad lines they propose is certainly attractive which is why their approach is worth refining. The scope of these comments is more modest still because they are methodological rather than substantive.

Tables 2 and 3 present the age-specific death rates in England and Wales for the different causes of death identified in the Registrar General's Decennial Supplements of 1851-1860 and 1891-1900.[18] It is possible to present them separately for the two sexes but only their respective age-specific rates are given here. Male, female and total cause-specific rates were standardised to the total age structure of 1901 and these are the basis of Table 4 and Figure 4. The final column of Table 4 is what McKeown and Record base their analysis on and the table is arranged so that their key diseases rise to the top and the residual groups are arranged according to the reverse order in which they were dropped from the analysis. The table also shows the ratio between the male

Table 2. Age/cause-specific death rates (per million living), England and Wales; 1851-1860.

Cause of death	0	1-4	5-9	10-14	15-19	20-24	25-34	35-44	45-54	55-64	56-74	gt.-74	Total
a. Smallpox	1602	801	257	73	93	130	93	55	38	24	18	14	222
b. Measles	2158	2885	275	38	13	9	6	4	2	1	1	2	412
c. Scarlatina	1736	4814	1995	495	150	73	44	31	19	15	10	7	877
d. Diptheria	288	458	254	104	42	20	13	12	10	10	13	16	109
e. Whooping cough	5916	2707	174	10	2	1	1	0	0	0	1	1	504
f. Typhus	904	1523	1009	782	944	814	649	623	713	965	1413	1543	909
g. Cholera	14125	2110	229	106	11i	175	257	347	477	934	2196	5136	1082
h. Other zymotic	6334	2204	459	179	187	189	261	396	545	878	1734	3093	834
i. Cancer	28	19	9	9	17	28	104	390	862	1414	1964	2085	317
j. Scrofula	3633	1271	283	233	208	170	139	111	117	140	165	113	408
k. Phthisis	2032	1007	573	1027	2964	4182	4321	4102	3475	2848	1987	809	2683
l. Hydrocephalus	4024	1937	364	102	31	10	7	6	6	7	7	8	398
m. Brain	33404	2910	583	359	403	437	583	1024	1837	3963	9353	16180	2745
n. Heart	662	285	228	257	338	370	561	1064	1988	4364	8841	11665	1249
o. Lungs	21936	6137	597	231	339	476	673	1283	2572	5807	12149	19704	3025
p. Stomach	3368	556	252	207	288	369	520	916	1639	3007	4769	4948	1006
q. Kidneys	51	50	35	39	60	86	140	220	339	617	1396	2114	215
r. Generative	11	4	0	1	8	25	52	109	153	167	179	149	55
s. Joints	58	51	62	78	76	58	52	59	71	103	141	115	70
t. Skin	462	54	11	9	10	11	13	22	37	80	197	370	49
u. Childbirth	0	0	0	0	72	323	465	465	36	0	0	0	164
v. Violent	1760	1085	536	469	508	516	542	652	810	976	1225	2248	735
w. Other	49508	3269	291	166	184	208	270	448	845	2618	14110	89585	4132
Male	168404	36512	8523	4931	6696	8836	9578	12506	17996	30951	65474	165528	23081
Female	138930	35758	8432	5062	7394	8542	9934	12180	15240	27075	58777	155574	21360
Total	153999	36136	8477	4974	7047	8681	9764	12339	16586	28937	61869	159899	22200

Table 3. Age/cause-specific death rates (per million living), England and Wales; 1891-1900.

Cause of death	0	1-4	5-9	10-14	15-19	20-24	25-34	35-44	45-54	55-64	65-74	gt.74	Total
i. Smallpox	55	20	10	3	5	11	16	18	13	10	10	8	13
ii. Measles	3319	3120	221	18	7	4	4	3	2	1	1	0	413
iii. Scarlet F.	290	979	353	81	33	22	15	8	4	2	1	0	157
iv. Diptheria	457	1584	679	125	36	20	16	14	12	14	12	9	263
v. Whooping C.	5810	2060	96	3	1	0	0	0	0	1	1	3	376
vi. Typhus F.	0	1	1	2	2	2	3	4	4	2	1	2	2
vii. Enteric F.	24	97	127	162	256	271	283	189	144	112	69	29	174
viii. Continued F.	23	17	7	4	3	3	3	3	4	5	9	13	6
ix. Diarrhoea	17521	1169	33	9	7	9	18	31	70	204	675	2035	712
x. Cholera	462	41	4	1	2	2	4	8	15	29	32	29	25
xi. Cancer	22	31	16	16	29	44	138	644	1828	3651	5594	6129	756
xii. Tabes Mes.	3721	661	103	68	57	46	38	34	30	27	21	9	216
xiii. Phthisis	561	345	206	367	1141	1726	2129	2585	2355	1876	1152	436	1388
xiv. Other T.B.	3573	1471	415	234	194	160	129	115	101	99	88	65	402
xv. Nervous	21745	2061	382	222	244	237	381	932	1910	4456	10501	19583	2166
xvi. Circulatory	502	88	169	266	334	336	541	1232	2634	6121	12958	19613	1653
xvii. Respiratory	27727	6778	587	173	277	380	647	1425	2900	6637	14476	29677	3401
xviii.Digestive	14186	1457	225	162	218	247	307	636	1226	2160	3540	5063	1190
xix. Urinary	246	145	71	56	77	118	203	438	812	1544	2936	4672	460
xx. Generative	43	9	1	2	10	22	47	87	106	104	149	176	46
xxi. Puerperal F.	0	0	0	0	24	155	220	134	5	0	0	0	68
xxii. Childbirth	0	0	0	0	25	131	240	255	16	0	0	0	84
xxiii. Violent	3029	748	357	224	326	362	447	647	875	118	1450	2560	659
xxiv. Other	50178	1364	307	307	412	422	600	1040	1644	3212	11254	61656	3523
Male	167775	24812	4309	2447	3782	5046	6738	11466	18894	34852	70286	159692	19272
Female	138360	23688	4370	2565	3658	4449	6065	9566	14702	28361	60606	146063	17101
Total	153326	24248	4340	2506	3720	4730	6384	10485	16711	31385	64931	151768	18152

and female standardised rates at each date and the proportionate change in the standardised rate for each cause over the period.[19] Figure 4 shows some of this information graphically. The height of the histograms corresponds to the standardised mortality rate in 1851-1860, broken down into some of the major causes of death on the left and into age-groups on the right.[20] The shaded area depicts the decline in mortality over the period and shows the proportionate reduction in standardised rates for each cause- and age-category.

On the same basis a deterioration in specific mortality will take any particular cause- or age-group beyond the boundary of the 1851-1860 histogram. In this way, the area shaded for each category is proportional to its contribution to the overall fall in national standardised mortality. With this figure and these tables, two comments might be made on the analysis by McKeown and Record. They make little use of sex-specific mortality, age-specific mortality and mortality from their groups of ''others'' for diagnostic purposes. Furthermore, some questions remain about their identification of the causes they do have recourse to.

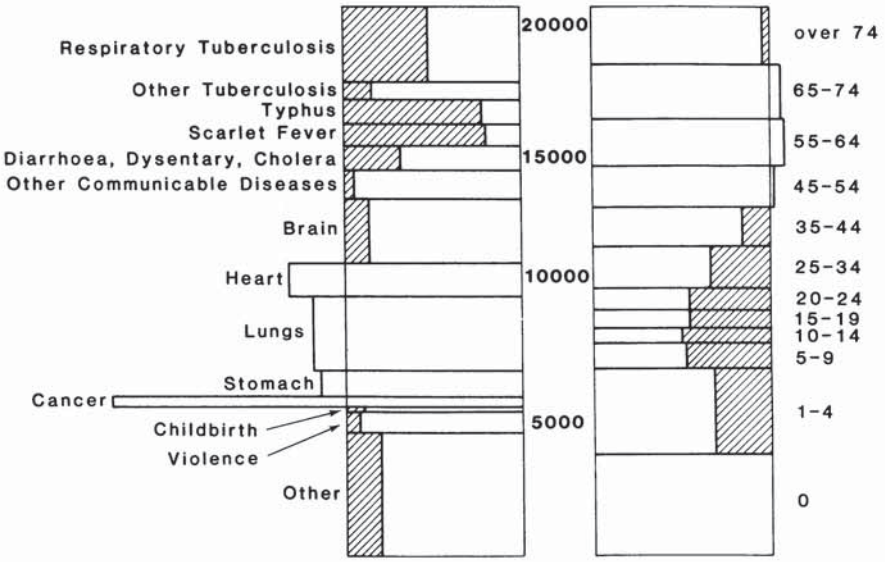
In both these decades, life was shortest for men. For 1861 Preston, Keyfitz and Schoen give an expectation of life at birth of 40.5 for males in England and Wales and 43.1 for females; for 1901 their respective figures are 45.3 years and 49.4 years.[21] The gap is increasing at a time of general improvement in mortality. In explaining mortality decline, therefore, our account must be compatible with the improvements it identifies being most particular visited upon females. Table 4 shows that male mortality was worst for most causes and that female mortality was improving faster for most causes. For some of the major causes of death, the relative sluggishness of male rates is quite marked. In the case of respiratory tuberculosis, the male standardised rate in 1891-1900 is 61% of the 1851-1860 figure while the female rate is only 42%. This ought to have some bearing on how reasons are assigned for the decline in mortality, both as a whole and as regards the contribution of particular causes.

As figure 4 shows there is no improvement in infant mortality over this period and there is a deterioration in the mortality of the elderly; if 45 for men and 55 for women is not too inappropriate a cut-off point for the use of this term. This pattern is relatively consistent between the sexes with the female improvement being significantly greater than the male in adulthood (15-44). It is clear from Tables 2 and 3 that the mortality improvements are concentrated in the middle years of life whereas the greatest mortality was found among the old and the young. In this respect the one-third fall in child mortality is especially striking. Indeed a fall in the standardised rate resulting from a decline in child mortality will have a much greater impact on the survivorship curve and thus on life expectancy than will a fall of the same magnitude caused by falling adult mortality.

Table 4. Standardised mortality rates (per million living) for various causes for England and Wales; 1851-1860 and 1891-1900.

(1)	(2)	(3)	(4)	(5)	(6)	(7)
	1851-1860		1891-1900			Change
Cause of death	s.m.r.	Male/ Female	s.m.r.	Male/ Female	(4/2)	(2-4)
A. Communicable diseases contributing greatly to mortality decline						
Resp. T.B. (k;xiii)	2760	94	1410	133	51	1350
Other T.B. (j,l;xiii,xvii)	685	123	570	116	83	115
Typhus (f;vi,vii,viii)	890	100	183	132	21	707
Scarlet F. (c;iii)	775	102	150	100	19	625
Diarrhoeal (g;ix,x)	923	106	631	111	68	292
A. Total	6033		2944			3089
B. Certain other communicable diseases						
Smallpox (a;i)	195	117	13	149	7	182
Whooping C. (e;v)	408	78	338	79	83	70
Measles (b,ii)	349	101	385	105	110	-36
Diptheria (d;iv)	98	89	251	95	256	-153
B. Total	1050		987			63
C. Certain other causes distinguished in McKeown and Record						
Brain (m;xv)	2410	118	2054	118	85	356
Heart (n;xvi)	1272	95	1665	107	131	-393
Lungs (o;xvii)	2767	123	3249	125	117	-482
Stomach (p;xviii)	992	102	1116	108	113	-124
Kidneys (q;xix)	219	286	463	179	211	-244
C. Total	7660		8547			-887
D. Other causes						
Cancer (i;xi)	328	48	764	72	232	-281
Other zymotic dis. (h)	748	124				(748)
Generative (r;xx)	57	4	47	8	82	10
Joints (s)	68	135				(68)
Skin (t)	44	113				(44)
Childbirth (u;xxi;xxii)	173	0	156	0	90	17
Violence (v;xxiii)	715	307	649	264	91	66
Other (w;xxiv)	3606	106	3270	114	91	336
D. Total	5739		4886			853
All causes	20482	108	17364	115	85	3119

Figure 4. Mortality decline in England and Wales, 1851-1860 to 1891-1900.



In measuring the mortality decline, age- and sex-specific factors are only touched on implicitly by McKeown and Record insofar as those factors come out in the wash of aggregate rates due to the five selected causes. Taking up the question of measuring the mortality decline due to specific causes, the first point to be made is that McKeown and Record discard most causes of death from their analysis. The five sets of causes on which they focus (Group A in Table 4) make up about one-third of the standardised mortality in 1851-1860 and a sixth in 1891-1900. They argue that these causes are identified as significant by their contribution to the mortality decline. With the standardised rates for England and Wales going from 20482 per million living to 17364 per million and these causes going from 6033 to 2944, they seem to more than adequately account for the mortality decline. Table 4 attempts to show the effect of thus comparing gross falls from specific causes to the net fall from all causes. Broadly speaking, the category "other" holds a host of diverging mortalities, some improving some deteriorating. These others by no means equal the changes identified by McKeown and Record but the relative stability of the "others" category is an arithmetic fiction. We can easily illustrate this. In Groups B, C and D the final column shows eight sets of causes deteriorating in mortality and ten improving. Some of these causes are only given for one decade so the comparison is probably meaningless. Nevertheless, the sum of the positive changes is quivalent to a change in the standardised mortality rate of 1897 per million, not insignificant when set alongside the net change of 3118 per million and certainly raising questions about dismissing "other" causes as a relatively stable residual.

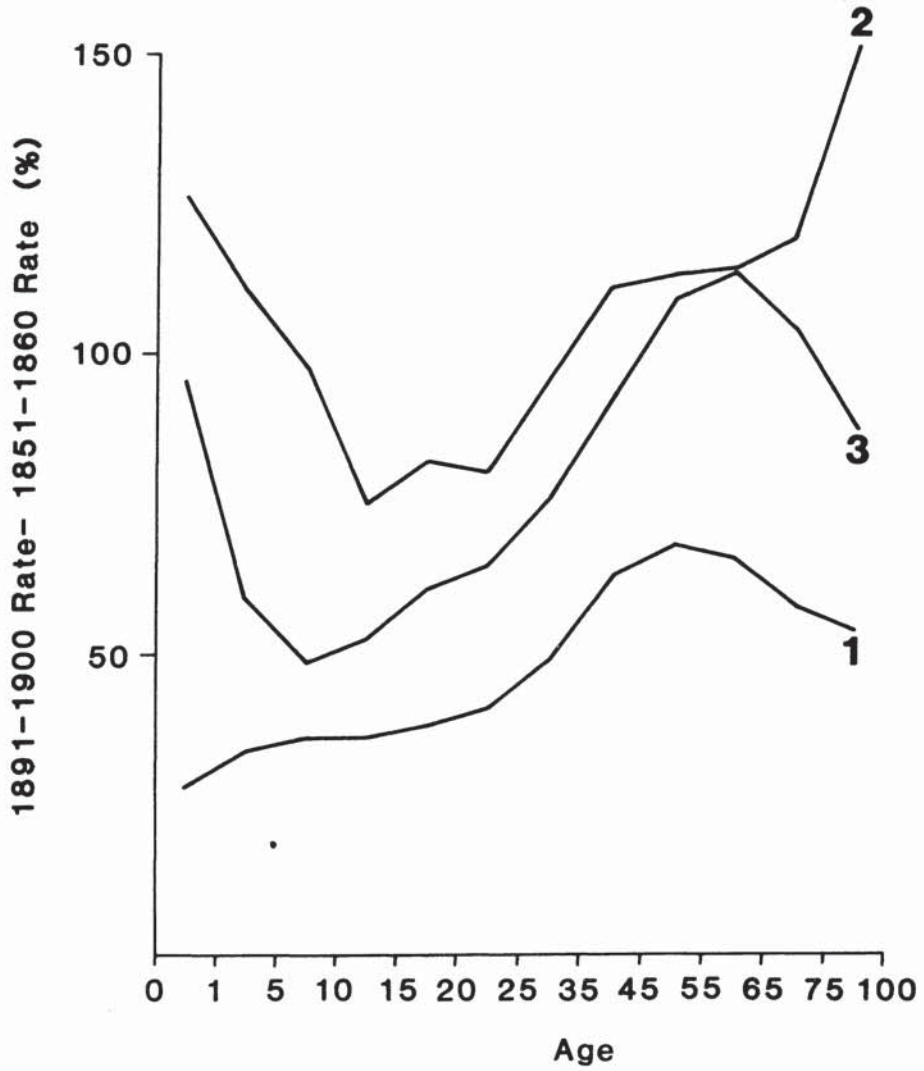
Part of the problem, as McKeown and Record recognise, arises from disease classification, misclassification and reclassification. The disease classification recognised three main categories of disease: those which "ferment" in the body quickly, feverishly dispatching it from this life; those which work slowly, wearing the body down; and those which are malfunctions of particular cogs in the mortal motor. Yet symptoms were often ambiguous guides in thus distributing deaths. McKeown and Record are right to remind modern readers that contemporaries were a good deal more familiar with these diseases than is the modern European physician. Yet if one wished to be mischevious one might begin by questioning three of the five sets of causes on which McKeown and Record hang their analysis.

First, their own discussion raises doubts about the distinction between scarlet fever and diptheria in 1851-1860 since they complain of "the confusion between scarlet fever and diptheria until 1855".[22] This would be less significant were it not for the fact that scarlet fever declines while diptheria increases. If there is some diptheria in the scarlet fever figures for 1851-1860, then, the fall in scarlet fever has been overstated. The net fall from scarlet fever and diptheria combined is 472 per million and the stated fall from scarlet fever is 625. Even on this, the least helpful assumption for their argument, their conclusion about scarlet fever seems safe.

A second disease category worth looking at is typhus. Leaving to one side the different histories of typhus and typhoid, it is clear that the fevers as a whole present problems.

McKeown and Record equate the typhus of 1851-1860 with three separate categories in 1891-1900: typhus, enteric and simple continued fevers. Yet if we are looking for that complex of fevers which contemporaries associated with environmental improvements (the purpose of isolating typhus), one might ask where the "other zymotic diseases" of 1851-1860 have gone. Certainly, contemporaries noted a great improvement in these but this is lost, firstly because they are not separately distinguished in the decennial table for 1891-1900 and secondly because McKeown and Record sweep them up with the residual "other causes" in 1851-1860. If enteric and simple continued fevers are to be added to typhus as a fevers-complex in 1891-1900, a reasonable case might be heard for adding some of the "other zymotic diseases" in 1851-1860. The consequences are of moment. Typhus alone (if the bulk of the 1851-1860 figure refers to this) falls from 890 to 2 per million, adding in enteric and simple continued fevers for 1891-1900 reduces the fall by 181 but adding in other zymotic diseases increases it by 748. We have a fall in the fevers-complex which ranges from 707 to 1455 per million. The latter figure is greater than that set down to respiratory tuberculosis and if the sanitary revolution were measured by the diarrhoeal and fevers sets of causes, it would only require three-fifths of the "other zymotic" category to be added to the fevers before the environmental factor would give a fall equal to that of McKeown and Record's tubercular diet and nutrition factor.

Figure 5. Change in age-specific mortality rates for respiratory tuberculosis, chest diseases and other causes.



The third set of causes we might reconsider is respiratory tuberculosis. The fall in respiratory tuberculosis was accompanied by a rise in diseases of the lungs. The first point to make is that, as can be seen from Table 4, the fall in standardised rates due to respiratory tuberculosis (1350 per million) far outstrips the deterioration in diseases of the lungs (482 per million). Secondly, from Tables 2 and 3, it is clear that diseases of the lungs are afflictions of the young and old but respiratory tuberculosis stalks the relatively healthy years of adulthood, being responsible for 41% of all deaths between the ages of 15 and 45 in 1851-1860. There is little evidence here that the two disease categories are equivalent. Over time, their distinctiveness becomes even more pro-

nounced which may suggest some confusion in the earlier period. Figure 5 shows the proportionate change in mortality at the different ages for respiratory tuberculosis (1) and diseases of the lungs (2); equivalent data for all other causes (3) is given for comparison.

Between ages 5 and 55, the two diseases share the general pattern of improvements in mortality being concentrated in early adulthood but the changes for the young and old are divergent for the two causes. Although the disease was primarily an adult-killer, one-fifteenth of all phthisis deaths in 1851-1860 were registered to those under five years of age, whereas in 1891-1900 the figure was one in twenty-eight. The changes in mortality due to respiratory tuberculosis at these ages are commensurate with the increases assigned to diseases of the chest, and similarly for the oldest age groups. The worst case would be to suggest that the bulk of the improvement in respiratory tuberculosis among the under-fives and the over-sixty-fives can be explained by a greater tendency in the later period for deaths in these age-groups to receive the less-specific label "diseases of the respiratory system" rather than the more precise term "phthisis". This would wipe-out one-sixth of the fall in standardised rates displayed by respiratory tuberculosis. If we confine our skepticism to the younger age group the effect is less and about one-fourteenth of the improvement is removed. In terms of the balance between the factors explaining the mortality decline, any adjustment along these lines will narrow the gap between the nutrition factor and the sanitary revolution. There is at least a prima facie case for looking again at the statistics of respiratory tuberculosis.

When all is said and done, considerations such as those above will only put wider margins of error around any conclusions we may draw on the basis of cause of death statistics, we can obviously never replace a historical and messy set with a modern and tidy one. We have to work with what we have got. The main attraction of McKeown and Record's analysis has been that they used cause of death statistics to say something definite about the factors controlling mortality. It has been hinted above that age and sex might also be powerful diagnostic tools and question marks have been placed against the relative sizes of the falls associated with their environmental and nutrition factors. Their measurement of mortality change needs its possible margins of error spelling out. When that is done there is still the matter of interpretation. In at least two respects their equation of causes and factors may be queried. First, there is their claim that the primary control on tuberculosis was exerted by nutrition. Secondly, there is the question of the interaction between causes of death.

So much depends on McKeown and Record's interpretation of the decline in tuberculosis mortality that it is worth looking at this factor again. Tuberculosis, they suggest, was primarily responsive to changes in the quality of diet: "incomplete as it is, the evidence for the nineteenth century is at least consistent with the view that diet was the most significant environmental in-

fluence in relation to the trend of mortality from tuberculosis.''[23] By this, they do not mean that they have found evidence that the quality of diet advanced faster in places where tuberculosis fell dramatically, nor do they mean that the chronology of mortality decline follows that of certain indicators of real income. Rather, they fail to turn up evidence for other hypotheses and are left holding this one. Livi-Bacci has recently commented on the simplistic way historical demographers turn to nutrition in explaining long term trends in mortality and there is certainly a body of evidence to suggest that respiratory tuberculosis is not one of those diseases where nutrition exerts a strong influences on rates of fatality.[24] With a respiratory disease, one might expect a primary control to be exerted by contact with infected persons and among other things one would might pay close attention to overcrowding. In the nineteenth century, McKeown and Record inform us, ''new building of houses did little more than keep pace with the increase in the size of the population, and the number of persons per house decreased only slightly (from 5.6 in 1801 to 5.3 in 1871).''[25] Despite these statistics, it is hard to believe that in certain cities the problem of overcrowding did not fluctuate in ways which might allow one to re-examine the claim that diet was the primary determinant of receding rates of respiratory tuberculosis. Certainly, the development of new bye-law housing in cities such as Liverpool over the second half of the nineteenth century may have diluted the problem of overcrowding for the working class. McKeown and Record also countenance no improvement in the quality of milk before the 1922 Ministry of Health Order on the pasteurisation of milk yet city authorities were aware of the problem of infected milk and at least towards the close of our period made efforts to control the milk supply drawing particularly on districts which they felt to be relatively free from infection. In addition the vigorous campaign against urban cowkeepers conducted throughout this period may have improved the salubrity of dairies offering some prospect of falls in bovine tuberculosis. With this, as with other diseases, a multiplicity of factors may be at work and the aim of the analysis should be to explore these through comparative analysis of different places and periods. McKeown and Record's residual reliance on nutrition indicates that the issue is still open.They have not proved that general economic improvement rather than specific medical or sanitary interventions was the primary motor of progress.

If it is difficult to assign a unique factor to a single disease it is also hard to confine a particular factor to a solitary disease. McKeown and Record proceed as if the effectiveness of any intervention may be limited to falls in the disease it immediately touches upon. One needs to distinguish here between using changes in the mortality from certain diseases as indicative of certain improvements and using those changes as measurements of the contribution of those improvements. McKeown and Record have followed the latter and more difficult path. There are certain problems involved with thus considering diseases in isolation. If we consider the consequences of the sanitary

revolution and of medical intervention which they associate with fevers plus diarrhoeal diseases and with smallpox respectively, we can easily appreciate the problem. Cleanliness obviously reduces exposure to certain of the infections producing both respiratory and bowel complaints; it is difficult to understate the importance of clean hands. McKeown and Record look to fever and diarrhoeal diseases to register the sanitary revolution in water supply, drains and sewers. As indicators that improvements have been made, this seems helpful and a comparative study of different cities along these lines might be instructive. However, as a measurement of the consequences of the sanitary revolution this is potentially misleading. Diseases obviously interact and a generally lower level of infection might save individuals from debilitating complaints which lay them low before the depredations of some other, unrelated killer. The same goes for smallpox and their medical factor. The practical eradication of smallpox over this period points to the success of a variety of control strategies and by investigating the geography of this conquest one might begin to evaluate those strategies. Yet the importance of smallpox extended beyond the immediate deaths it claimed to the scores more who bore its disfiguring marks for the rest of their lives and to those whose constitutions were permanently impaired by an attack of this vigorous disease at a time when their young bodies should have been developing apace. In other words, McKeown and Record's hypotheses and methods are suggestive rather than conclusive and their air of precise accounting is deceptive. The causes of death which capitalised upon victims weakened by early smallpox or repeated stomach or chest infections may well appear in boxes far removed from the "sanitary revolution" or "medical intervention" but it is these two latter developments which have deprived them of their mortality tribute all the same.

A lot of the issues raised in this consideration of McKeown and Record's account of the decline in mortality in nineteenth-century England and Wales are almost impossible to explore with aggregate data at a national level. Data on individual life histories would be ideal but there is a halfway post which is worth exploring further. Given the importance of the geographical redistribution of people between rural and urban areas and given the relations which may exist between labour markets and sex- and age-specific mortality, there may be a lot to be gained in following a comparative approach in comparing rural and urban mortality and in looking at the histories of individual cities.[26]

Urban mortality and the context of population change

This paper has considered two issues. First is the claim that the population history of England in the early modern period resonates to an economic rhythm heard mainly through the preventive check. In this respect, urbanisation and urban mortality pose some difficulties for attempts to isolate mor-

tality and fertility effects at a purely aggregate level. Some playful alternatives have been proposed all of which rely on urban mortality being more sluggish in improving than rural mortality in eighteenth century England. The second claim which this paper has reconsidered is that mortality changes in the nineteenth century can largely be set down to changes in diet and that, as such, they are an almost unconscious benefit of economic development in general rather than a tribute to specific interventionist measures. Here, it has been suggested that the matter is far from closed because the identification, apportionment and interpretation of the mortality decline might still be subject to contention. In particular, the sorts of measures one associates with better urban management may have contributed to the decline in respiratory tuberculosis, their effect on mortality from fevers may have been understated and the effects of changes in employment patterns have not yet been explored.

In terms of general economic history restating the importance of the urban penalty might have a number of consequences. First, it may reinforce the view of the rapid development of British agriculture in the eighteenth century. Ironically, it appears to leave open the door for an exploration of dietary improvements in rural England, operating as a long term trend rather than through short-term crises. Alternatively, it may return our attention to the towns as ecological gatekeepers with rural areas sharing in the benefits of a more effective exclusion of infectious or epidemic disease from the country. This is a theme which could extend over the whole period 1500-1940 and which requires that we explore the correlations between cause-specific mortality declines across countries with very different economic histories.

Acknowledgements

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Notes

1. As McKeown defines the term, "it will be taken to refer to the growth of population which began in the late seventeenth or early eighteenth century and has continued to the present day"; T. McKeown, *The Modern Rise of Population* (London 1976), p.1.

2. E.L. Jones, *The European Miracle: Environments, Economies and Geopolitics in the History of Europe and Asia* (Cambridge 1981).

3. E.A. Wrigley, 'The Growth of Population in Eighteenth-Century England: a Conundrum Resolved', *Past and Present*, 98 (1983), pp. 121-150, p. 131. See also R.S. Schofield, 'The Impact of Scarcity and Plenty on Population Change in England, 1541-1871', *Journal of Interdisciplinary History,* 14 (1983), pp. 265-291. These two articles present some of the main findings of their book, E.A. Wrigley and R.S. Schofield, *The Population History of England, 1541-1871: a Reconstruction* (London 1981).

4. These and the other statistics in this paragraph are taken from E.A. Wrigley, 'Urban Growth and Agricultural Change: England and the Continent in the Early Modern Period', *Journal of Interdisciplinary History,* 15 (1985), pp. 683-728.

5. Wrigley and Schofield (1983), p. 484.

6. The main works are as follows: T. McKeown and R.G. Brown, 'Medical Evidence Related to English Population Changes in the Eighteenth Century', *Population Studies,* 9 (1955), pp. 119-141; T. McKeown and R.G. Record, 'Reasons for the Decline of Mortality in England and Wales during the Nineteenth Century', *Population Studies,* 16 (1962), pp. 94-122; T. McKeown, R.G. Record and R.D. Turner, 'An Interpretation of the Decline in Mortality in England and Wales during the Twentieth Century', *Population Studies,* 29 (1975), pp. 391-422; T. McKeown, R.G. Brown and R.G. Record, 'An Interpretation of the Modern Rise of Population in Europe', *Population Studies,* 26 (1972), pp. 94-122; T. McKeown, 'Food, Infection and Population', *Journal of Interdisciplinary History,* 14 (1983), pp. 227-247.

7. For a recent review of McKeown's arguments see R.Woods and J. Woodward (Eds.), *Urban Disease and Mortality in the Nineteenth Century* (London 1984).

8. McKeown (1983), p. 227; and elsewhere.

9. J. Hajnal, 'The European Marriage Pattern in Perspective', D.V. Glass and D.E.C. Eversley (Eds.), *Population in History* (London 1965), pp. 104-106.

10. E.A. Wrigley and R.S. Schofield, 'English Population History from Family Reconstitution: Summary Results', *Population Studies*, 37 (1983), pp. 157-184, pp.178-179.

11. Wrigley (1983), p.129.

12. This is clear from the maps presented in R. Woods, 'The Structure of Mortality in Mid-Nineteenth Century England and Wales', *Journal of Historical Geography,* 8 (1982).

13. C.M. Law, 'The Growth of the Urban Population of England and Wales, 1801-1911', *Transactions of the Institute of British Geographers,* 41 (1967), pp. 125-143.

14. R. Woods, 'The Effect of Population Redistribution on the Level of Mortality in Nineteenth Century England and Wales', *Journal of Economic History,* 45 (1985), pp. 645-651.

15. Based on figure 7.12 (p.243) and Table A3.1 (pp. 528-529) in Wrigley and Schofield (1981).

16. All the data are taken from Woods (1985).

17. McKeown and Record (1962).

18. The population at risk for age 0 is the total number of births in each ten year period. The other ages are estimated as the geometric mean of the values given for that age in the decennial censuses bordering the period, 1851, 1861, 1891, 1901. This estimate of the population at risk is not the best possible and could be improved on. In 1851, the age group 0-4 is not broken down into its component years and here it is assumed that those in ages 1-4 made up the same proportion of the 0-4 group in 1851 that we know they did in 1861. This approximation depends on the infant mortality rate being roughly constant between the two dates and is certainly capable of refinement.

19. The numbers and letters in the tables refer to the following lists of causes of death, abbreviated in tables 2 and 3. '1851-1860: a.smallpox, b.measles, c.scarlatina, d.diptheria, e.whooping cough, f.typhus, g.cholera,diarrhoea,dysentery, h.other zymotic diseases, i.cancer, j.scrofula, tabes mesenterica, k.phthisis, l.hydrocephalus, m.diseases of the brain, n.heart disease and dropsy, o.diseases of the lungs, p.diseases of the stomach and liver, q.diseases of the kidneys, r.diseases of the generative organs, s.diseases of the joints, t.diseases of the skin, u.childbirth and metria, v.violence, w.other. 1891-1900: i.smallpox, ii.measles, iii.diptheria, iv.whooping cough, v.typhus, vi.enteric fever, vii.simple continued fever, viii.diarrhoea and dysentery, ix.cholera, x.cancer, xi.tabes mesenterica, xii.phthisis, xiii.other tubercular and scrofulous diseases, xiv.diseases of the nervous system, xv.diseases of the circulatory system, xvi.diseases of the respiratory system, xvii.diseases of the digestive system, xviii.diseases of the urinary system, xix.diseases of the generative system, xx.puerperal fever, xxi.childbirth, xxii.violence, xxiii.other.

20. The age-specific mortality rates are standardised to the population distribution of 1901. Since both the 1851-1860 and the 1891-1900 rates are standardised to the same age structure, the ratio of any given age-specific rate between the two dates will be the same as the ratio of the unstandardised age-specific rates. The statistics on tuberculosis used by McKeown and Record come from a 1949 Ministry of Health Report but inspection of the data suggests that the definition adopted here gives similar figures and has the virtue of being compatible with the rest of the tabulated data.

21. S.H. Preston, N. Keyfitz, and R. Schoen, *Causes of Death. Life Tables for National Populations* (New York and London 1972), pp. 224, 226, 240, 242.

22. McKeown and Record (1962), p.95.

23. McKeown and Record (1962), p.115.

24. M. Livi-Bacci, 'The Nutrition-Mortality Link in Past Times: a Comment', *Journal of Interdisciplinary History,* 14 (1983), pp. 293-298. See the review essay, D.N. McMurray, 'Cell-Mediated Immunity in Nutritional Deficiency', *Progress in Food and Nutrition Science,* 8 (1984), pp. 193-228. On the basis of the literature cited therein one can only conclude that there may be some diseases where the replication of the disease in the body and thus its ability to overwhelm the system is prejudiced by under-nutrition. Respiratory tuberculosis could be one such disease and thus the well-documented lack of immune response with under-nourished children is an unreliable guide to their prospects of surviving an attack.

25. McKeown and Record (1962), p.113.

26. At the conference some very early results were presented for the twenty-two largest English cities, to which Hans Norman makes brief reference in his commentary. This discussion has been excluded from the present article and I hope to develop the analysis of the individual cities in a later paper.