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# What do people die of during famines: the Great Irish Famine in comparative perspective

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The Irish Famine killed over a million people who would not have died otherwise. The nosologies published by the 1851 Irish census provide a rich source for the causes of death during these catastrophic years. This source is extremely rich and detailed, but also inaccurate and deficient to the point where many scholars have given up using it. In this article we try to make adjustments to the death-by-cause tabulations and provide more accurate ones. These tables are then used to analyse the reasons why so many people died and why modern famines tend to be less costly in terms of human life.

## 1. Introduction

Today the most resonant media images of famine are of skeletal children on the verge of starvation. Such images capture the heightened vulnerability of infants and small children to famine, but they also create the misleading impression that famine deaths are starvation deaths. Now, as in the past, most famine victims die not of literal starvation, but of infectious diseases. These diseases come in various kinds, at different times, and with differing levels of intensity. Hunger and infectious disease interact in complicated ways, some of which operate through the human body and some through the fabric of human society. The causation of death during a famine turns out to be a difficult question, with the usual philosophical undertones. Here we examine these issues in the context of the Irish famine of 1846–51. This was a real famine in the old-fashioned sense, with strong Malthusian features: a catastrophic reduction of the food supply led to major demographic readjustment. Still, every famine is to some extent *sui generis*. How different were the causes of excess mortality in Ireland in the 1840s from those operating during other famines? Why were they different? Before attempting to answer these questions we must establish with as much detail as possible the causes of excess mortality in Ireland. To be sure, the Irish case is often difficult to interpret and compare with others, partly because mid

nineteenth-century medical terminology and concepts are so different to our own, and partly because the statistical evidence is faulty or lacking. All the same, the available evidence can be utilised to draw some important historical conclusions.

Two broad classes of causes were responsible for augmented mortality during famines. The first is directly nutrition-related, and includes some cases of actual starvation. More often, however, victims of this class succumb to nutritionally sensitive diseases brought on by impaired immunity, or to poisoning from inferior foods that would have been discarded in normal times. The other is indirect: death is caused by the disruption of personal life and the normal operation of society resulting from famine, but was not the *immediate* result of a decline in nutritional status in the strict sense. Today an individual is deemed to have starved to death in the clinical sense only if he has died as the result of the attrition of protein and fatty deposits in the body causing gradual systemic atrophy, especially of the heart muscle. Pure starvation in this sense was relatively uncommon during the Irish famine. To what extent medical practitioners of the time meant something like this when they mentioned starvation remains to be seen. It seems likely, however, that at least three concepts in the medical literature of the day correspond roughly to what would be regarded today as pure starvation. First, there is actually a category called 'starvation'.<sup>1</sup> A second category is what is known today as 'oedema' or in the language of the time, 'dropsy', a swelling due to fluid accumulation often accompanying acute starvation. A third, 'marasmus', is a general term describing the death from some form of food inadequacy of infants and small children.

Yet these premodern terms also pertain to syndromes that are not famine-related. For instance, the 1841 Irish census records only 17 deaths from starvation for the entire year of 1840, out of a total of over 140,000 reported deaths for that year. In 1847 slightly over 6,000 people were reported to have died of starvation, out of nearly 250,000 reported deaths for that year. On the other hand, in 1840 dropsy and marasmus accounted for over 3,000 and over 9,000 deaths, respectively, although that year was famine-free (British Parliamentary Papers 1843, pp. 181–83, BPP 1856b, p. 663).

Most of the other diseases that killed people during the Irish famine were infectious diseases. Some were opportunistic diseases that took advantage of the fall in nutritional status and the general environmental deterioration. Specialists distinguish between *individual* immuno-suppression and *social* or

<sup>1</sup> In his introduction to the Tables of Death (on which more below), William Wilde defined 'starvation' as 'Want, Destitution, Cold and Exposure, Neglect, Want of Necessities of Life, in Irish *Gorta*'. He also suspected that some of those reported to have died of 'infirmity, debility and old age' belonged in the same category. See BPP 1856a: 518.

*collective* immuno-suppression. Individual immunity declines as the body is deprived of food, especially proteins. Recent research has questioned the widely held assumption that malnutrition *inevitably* leads to increased susceptibility to infection (Carmichael 1983, p. 53; Dirks 1993, pp. 157–63). During major famines, however, there is a threshold effect whereby a switch occurs from a regime of subnutrition or even malnutrition to one of acute deprivation, in which the immune system is severely impaired. Even then the effect is uneven. Some diseases are highly sensitive to food intake, others seem to operate entirely independent of nutritional status, and still others are in-between. In Ireland the potato blight reduced food *quality* as well as its quantity. One consequence, unsuspected by contemporaries, was that the intake of Vitamin C, now recognised as an essential element in human resistance to disease, fell precipitously. Irish diets had always been rich in Vitamin C thanks to the potato; as diets changed after the onset of the blight, scurvy made an unexpected appearance in Ireland (Crawford 1988). Few people were reported to have died of scurvy, but the accompanying weakening of immune systems must have contributed to the onset of, and increased fatality from, other diseases.

Collective or communal resistance to disease during famines in the past declined for very different reasons: as famines worsened, social structures such as formal and informal support networks and medical care broke down. Moreover, the decline in human energy output reduced labour productivity throughout the economy, leading to positive feedback effects that reinforced the initial shock. Famine begot reduced agricultural productivity which led to more famine. In addition, as Fogel and Sen have pointed out, a decline in total food supply was usually accompanied by a change in its distribution, normally to the disadvantage of the poor, people at the extremes of the age distribution, the less healthy, and possibly women (Drèze and Sen 1989, pp. 50–5; Fogel 1991, pp. 33–71; Maharatna 1996, pp. 9–10).

As resistance to disease declined, famine conditions greatly increased the ‘insults’ inflicted on the body. It is well understood today that such events produce an additional feedback effect: as disease reduces the body’s ability to absorb certain foods, it creates anorexia, while by simultaneously increasing the demand for certain nutrients, it creates synergistic effects (Carmichael 1983; Taylor 1983, pp. 285–303). These insults include:

*Digestive diseases due to decline in food quality.* As food supply declines in quantity, desperate people slid down the quality ladder, falling back on items that would normally not be eaten: seaweeds, diseased and spoiled foods, and wild plants. There is evidence that famished people in Ireland ate decomposing carrion as well as nettles, carrageen moss, and corn-weed. Such substances can mercilessly attack the digestive system and cause a variety of diseases which could become fatal in conjunction with the weakened immune systems.

*Digestive diseases due to changes in food composition and unfamiliar emergency foods.* This was particularly important in a potato-eating country such as Ireland in which what foods could be imported from overseas, especially the notorious 'Indian corn', were mostly unfamiliar and hard to prepare in those areas where the dependence on potatoes had been the most complete and the famine most acute. The reliance on potatoes had allowed much of Ireland to dispense with the investment in basic food processing such as flour mills and bakeries. Specialisation increased the costs of switching to substitute food sources after the famine. Contemporary reports described the diseases suffered by people from consuming unfamiliar and improperly prepared foods from Indian meal.

*Infectious diseases due to population moving around.* Famine conditions frequently led panic-stricken people to quit their homes in the search for food. Mobility increases mortality in famine-stricken regions for two reasons. One is that it exposes both the famine refugees and their hosts to new disease environments and microbial regimes to which they are not immune. The other is that hygienic and sanitary needs depended on certain fixed items. As people left their homes, they left behind their laundry facilities, their cooking utensils, and sanitary arrangements, however rudimentary. The result was a decline in hygienic standards. The increase of what contemporaries referred to as 'fever' – mostly typhoid, relapsing fever, and typhus – must be in large part a consequence of this phenomenon. Indeed, the many vagrants and famine refugees on the roads produced a new term for these diseases, 'road fever'.<sup>2</sup>

*Infectious diseases due to hygiene deterioration as people become weak and despondent.* The impact of serious malnourishment is not death straightway and not even necessarily disease, but a decline in physical energy output. The first consequence of a decline in food intake may not have been a further decline in work effort and physical agricultural product (although that would follow eventually), but reduced energy spent on many of the standard household tasks such as laundry, the hauling of water, and cleaning. Fuel supplies, coming mostly from Irish peat bogs, declined as people could not muster the energy for the hard work involved. Personal care, childcare, and food preparation were neglected when energy levels declined. The purely physical effects of energy imbalances were reinforced here by the psychological effects of starvation such as indifference and lethargy. The impact of reduced food intake on the effort devoted to these activities contributed to the spread of so-called 'dirt diseases'.

*Outbreaks of seemingly unrelated epidemics such as cholera, influenza and other diseases.* Identifying to what extent these diseases are a coincidence is

<sup>2</sup> In this regard Ireland's good roads may have been a double-edged sword: although they made it possible to rush relief food supplies to starved regions, they facilitated the flows of disease-spreading famine refugees.

always a problem. The case for opportunistic disease is strong enough, but occurrences of these epidemics in the absence of food scarcity are frequent enough to allow for some coincidence.

In this article we first offer a critique of the main source on the causes of famine mortality in Ireland in the late 1840s, the 'Tables of Death' in the 1851 Irish census (Section 2). We then adjust the 'Tables of Death' to establish a plausible profile of the causes of death (Section 3). The outcome prompts a brief look at why particular diseases were so deadly. Was it because the requisite medical technologies were lacking, or would poverty have ruled out their use in any case? (Section 4). Section 5 offers some comparative perspectives on famine nosologies, while Section 6 concludes.

## **2. The Irish nosologies: a critique**

To the uninitiated, the extensive and detailed mortality tables appended to the 1851 census of Ireland may seem like an almost inexhaustible source on the causes of death during the Great Irish Famine (BPP 1856b). Although slightly less detailed in some respects than the analogous tables in the unusually rich 1841 census (BPP 1843), the 686-page volume of tables is probably unparalleled in the range of data included. Mortality-by-cause data are cross-tabulated county by county, year by year, disease by disease, and by gender. A distinction is made between rural and 'civic' areas, and there are separate entries for deaths in workhouses and hospitals. The nosology was the work of William Wilde, who modelled it closely on the tables of mortality he had created for the 1841 census. It represents the best that mid nineteenth-century medical science had to offer, and while some of the diseases do not quite correspond to something a modern coroner would recognise, much of it seems to make sense.

Unfortunately, specialists have long known that the mid nineteenth-century Irish death tables leave a lot to be desired in terms of accuracy (MacArthur 1956, pp. 308–12). Some quick calculations and comparisons confirm the serious doubts about these tables. Indeed it is easy to become so despondent about them that the best course of action would seem to be to abandon them as misleading or useless. The main reservations historians have about them are as follows.

First, the *total* numbers are clearly serious under-enumerations because most of the numbers were collected retrospectively from surviving kin. During the famine, entire families disappeared through death, migration, or a combination of the two. Hundreds of thousands of people must therefore have expired between 1841 and 1851 with no surviving household member around to report their deaths to the census enumerators in 1851. Furthermore, given the catastrophic events after 1845, it is likely that many deaths were simply forgotten by surviving relatives. There is good reason to believe that the degree of under-enumeration differed a great deal between

the pre-famine years (1842–44) and the following years. To complicate matters, the totals probably included some deaths reported by families and deaths in workhouses and hospitals, so that under-enumeration could have been offset to some extent by double-counting and in a few cases *over-reporting* cannot be ruled out, although this is the exception.<sup>3</sup> An added complication here is that the coverage of deaths in workhouses, hospitals, and prisons, which accounted for about one-quarter of all recorded deaths, is likely to be quite reliable. Most such deaths were recorded in such institutions as they happened, and these records formed the basis of the summary data reported by the relevant authorities to Wilde in 1851.

To see the extent of under-reporting, note that the 1841 census reported total Irish population at 8.18 million and the 1851 census at 6.55 million. As explained in detail in Mokyr (1980), total famine mortality may be estimated by first projecting Irish population forward from 1841 to the eve of the Famine in 1846. Births occurring in 1846–50 (adjusted for a famine-induced decline in fertility) are then added on and estimated out-migration during the Famine years subtracted. This yields a total of 1.9 million deaths in Ireland in those five years, of which slightly over one-half were due to the Famine.<sup>4</sup> The 1851 census tables report a total of 985,000 deaths, so for the country as a whole the reporting factor is about 52 per cent. This factor, moreover, varied substantially from county to county. The implications of under-reporting are serious for a nosological analysis: if there was a correlation between the probability of having survivors and the nature of the disease to which an individual succumbed, the distribution of diseases in the 1851 mortality tables would have been subject to a negative bias, that is, the diseases that increased the most would be systematically under-reported.

Second, some of the disease categories are rather vague. The 1851 census distinguishes between diarrhoea and dysentery, although it would have been difficult at the time to distinguish between the modern disease of *Shigellosis* and other acute forms of diarrhoea. Indeed, the 1841 census does not make the distinction. In the 1851 census the ratio of reported deaths from dysentery to deaths from diarrhoea is 5.77 in county Leitrim and only 2.07 in adjacent Sligo. The largest single cause of death reported in the 1851 census is 'fever', responsible for 222,000 (over 16 per cent of all

<sup>3</sup> The instructions given to enumerators stipulated (BPP 1856c: cxxix) that 'the enumerators will observe the period over which the inquiry extends, in order to enter with accuracy the various persons who have died since the 6th June 1841, but who would, if now alive, be reckoned among the members of the existing families as relatives, lodgers, or servants, &c'. Since the form (p. cviii) stipulated that those 'who died while residing with the family' be included, institutional deaths should not have been included. It would be surprising if none were, but we don't deem this a major problem.

<sup>4</sup> This follows from the fact that the death rate in a typical year in Ireland before the famine was about 24 per thousand. See Mokyr (1980).

reported deaths), mostly occurring between 1846 and 1850. The famine years thus reveal an enormous increase in mortality rates from causes which we would consider to be *symptoms* although at the time they were considered *diseases*.

Third, some respondents in 1851 seem to have projected some of their famine memories back to pre-famine days, reporting famine-related diseases as if they had occurred before 1845. This may be seen from a comparison of the tables for 1842 (reported in the 1851 Census) and those for 1840, the last complete year reported in the 1841 Census (see Table 1). There is no reason why the figures for these two years should differ much, as underlying conditions were similar. Yet note, for example, in Table 1 the implied increases in the death rates from dysentery and diarrhoea (from 0.25 per thousand in 1840 to 0.64 per thousand in 1842) and in starvation (from 0.0 per thousand in 1840 to 0.06 per thousand in 1842).<sup>5</sup> Some diseases that increased during the famine seem to be over-represented, others (such as marasmus) not so. How serious are these biases? A simple chi-square test using the rates per 1,000 in 1840 and 1842 fails to reject the null hypothesis of no significant differences. This suggests that the bias introduced in the 1851 census by the disappearance of hundreds of thousands of people affected the total counts, but did not bias the distribution of pre-famine diseases unduly. Nevertheless, we use 1840 weights as our guide to 'normal' or pre-famine mortality in the calculations below. The difficulty with the famine years is more serious and will be addressed below.

Fourth, we can distinguish between categories of disease that were obviously and unambiguously associated with the famine and some opportunistic diseases (such as tuberculosis and measles) which occurred at increased frequencies as a result of the immuno-depression caused by malnutrition. All the same, this still leaves unexplained seemingly odd increases recorded in some diseases that hardly seem famine-related. For example, the number of people dying of 'rheumatism' and diseases of the bones and joints was reported at 484 in 1842 and 1,145 in 1849. Diseases that should hardly be affected by the famine such as those of the 'locomotive organs' and 'diseases of uncertain seat' (tumours, phlebitis, and 'debility and old age') still show a higher level of incidence for the famine years for three of the four provinces. The exception is Leinster, where the impact of the famine was the weakest, suggesting that this effect is somehow related to the famine. As a proportion of the total number of deaths, these diseases declined, but their increased incidence remains rather puzzling.

<sup>5</sup> The same phenomenon is illustrated by the report of cholera deaths; although cholera only reached Ireland in December 1848, the census reported 1,376 cholera deaths in the years 1841–47 (plus a further 2,502 in 1848). This must be in part a reflection of faulty dating, but it is also possible that some survivors confounded the epidemic with some other disease. The 1841 census similarly reports a steady stream of cholera deaths in the 1830s.

Table 1. *Comparing pre-Famine census data.*

Disease	1840 (from 1841 census)		1842 (from 1851 census)	
	(Percentage)	(per 000, adj.)	(Percentage)	(per 000, adj.)
Smallpox	4.35	1.04	3.99	0.96
Dysentery and diarrhoea	1.04	0.25	2.67	0.64
Cholera	0.19	0.04	0.19	0.05
Fever	12.69	3.05	10.73	2.58
Others	12.36	2.97	12.97	3.12
Total Epidemic diseases	30.63	7.35	30.55	7.34
Convulsions	5.00	1.20	4.97	1.19
Others	3.13	0.75	4.29	1.03
Total Nervous System	8.13	1.95	9.26	2.22
Heart, Circulatory Organs	0.20	0.06	0.76	0.18
Consumption	11.39	2.73	14.40	3.46
Others	3.64	0.87	4.29	1.03
Total Respiratory	15.03	3.61	18.69	4.49
Dropsy	2.27	0.54	2.19	0.52
Marasmus	6.37	1.53	5.21	1.25
Others	2.80	0.67	3.16	0.76
Total Digestive System	11.44	2.75	10.56	2.54
Urin., Gen., Loc., Teg.*	2.09	0.50	2.70	0.65
Infirmity, Debility, Old Age	19.08	4.58	11.82	2.84
Others	2.82	0.68	3.22	0.77
Total Uncertain causes	21.90	5.26	15.04	3.61
Starvation	0.01	0.00	0.27	0.06
Others	3.31	0.79	3.22	0.77
Total violent & sudden	3.32	0.80	3.49	0.84
Others and unspecified	7.21	1.73	8.96	2.15
Total	100.00	24.00	100.00	24.00

Note: \* Urinary, Generative, Locomotive, Tegumentary.

Fifth, another case in which survivors' memories seem to have let them down relates to the question 'in what season did the deceased die?' The census tables reveal that for some reason the autumn was discriminated against. Only 14.1 per cent of all deaths were reported to have occurred in the autumn, against the 25 per cent expected in the absence of seasonal variation. While many individual diseases were of course seasonal, different patterns between diseases should have reduced the susceptibility of the *total* to seasonality. Moreover, some afflictions in which seasonality should not have been much of a factor (for example, 'cancer and fungus', 'burns and scalds') were also subject to the same bias.

To take the 1851 Tables of Death census at face value would thus be a grave mistake. Yet to abandon them altogether would leave us at the mercy of anecdotal tidbits equally, if not more, subject to biases of memory and selectivity. The value of the census lies first and foremost in its systematic organisation, which allows us to detect certain regional and temporal patterns that at least provide a rough reflection of the nosological nature of the famine as it appeared to those who had survived it. Rather than argue that these data are in any sense accurate and reliable, we adopt the more conservative strategy of (1) drawing inferences and making comparisons where the biases just noted do not present a problem, and (2) pinpointing and adjusting for some of their worst shortcomings on the basis of what is known about Irish population statistics in this period. What emerges is not an accurate picture, but a historian's approximation, based on assumptions and simplifications. This reconstruction can, however, be used to shed some more light on the quantitative dimensions of the causes of mortality during the famine, and through them on the microeconomics of death during catastrophic shocks inflicted upon vulnerable economies.

### **3. Adjusting the tables of death**

As noted, the 1851 census seriously undercounted the number of people dying both before and during the famine years. In principle there are two ways of dealing with the rate of under-enumeration across counties. The simplest ploy is to assume a constant rate. An advantage of this method is that, combined with assumptions about population growth in the absence of a famine, it generates residually-calculated independent estimates of famine-induced net emigration by county after 1845. For our present purposes, however, the assumption of constant under-reporting will not do. As noted earlier, some under-enumeration was due to the emigration of survivors, some to the deaths of entire families, some to the silence of surviving kin. Assuming constant under-enumeration across counties implies, surely implausibly, that the impact of these was the same. The most serious problem with the nosologies is the possibility that certain diseases were under-reported due to the disappearance of entire families. If the degree of under-enumeration varied from disease to disease, the result might be an under-estimate of deaths due to the most murderous of them. We thus attempt adjustments that yield estimates of under-enumeration.

The under-enumeration problem presents two types of biases in the data. Let  $R_i = \lambda_i D_i$  where  $D_i$  are total actual dead in county  $i$ ,  $R_i$  reported dead, and  $\lambda_i$  is the under-reporting factor. To assume that the  $\lambda$ 's are the same across counties would overweight the distribution of diseases in counties that under-report the least and underweight those counties where under-reporting was greatest. Since under-reporting clearly was a function of the

severity of the famine, this would bias the nationwide distribution of disease toward under-reporting famine-specific diseases. We call this *weighting* bias. Secondly, simply adjusting for under-reporting will not produce a correct estimate of the disease distribution because that still assumes that the distribution of diseases among those not reported *within each county* was identical to the distribution of diseases among those actually reported. This seems implausible. We call this *truncation* bias.

To solve the two problems, we need county-specific estimates of  $\lambda$ . We could in principle follow a procedure similar to that outlined above for Ireland as a whole, that is, arrive at total famine deaths by subtracting the population of each county in 1851 from a hypothetical population that would have been there given the population of 1846, births, and migration. The trouble with such a procedure is that while there are enough data to allow a reasonably good estimate of *total* net migration, the *county-by-county* distribution of these migrants before 1850 is not known. We estimate that distribution on the basis of three alternative assumptions: the distribution of county shares in overseas migration in 1846–50 was the same as the reported one for 1851; the same as the average of 1851–55; and the same as the weighted average between 1821–41 and 1851. In addition, we had to estimate the total population between the censuses on the eve of the Famine. There are two alternative ways of doing this, and we worked with both. We also adjust for internal migration, as reported in the 1851 census. The overall estimates of the  $\lambda$ 's are moderately sensitive to these assumptions and as there is no obvious way to choose among them, we present upper and lower bounds in Table 2.<sup>6</sup>

The province-level data in Table 2 make sense in that the  $\lambda$ 's tend to be particularly low for the worst hit regions in Connacht, and high for the Leinster counties. We also ran simple regressions of the level of the  $\lambda$ 's on crude measures of the severity of the famine such as the proportion 'starvation' of all deaths reported. These regressions (not reported here) show a consistent negative relation between the  $\lambda$ 's and the *reported* incidence of starvation (though the latter itself is of course mismeasured). The nationwide nosology resulting after adjustment for the weighting bias is provided in Table 3.

The adjustment, as might be expected, raises the proportion of famine-specific diseases like 'starvation' and dysentery and reduces the shares of more traditional causes of death such as consumption and 'infirmity'. Correcting for the weighting problem by computing county-specific under-reporting rates by itself is insufficient, however, because it assumes implicitly that the disease distribution for deaths unreported in the census was the same as that for those reported in a particular county. This seems unlikely, as the

<sup>6</sup> For a fuller account of the underlying assumptions, see Mokyr and Ó Gráda (1999).

Table 2. *Estimates of under-reporting coefficients ( $\lambda$ ) by province.*

Assumption about emigration	Ulster	Leinster	Munster	Connacht	Ireland
Shares as in 1851	0.38	0.70	0.69	0.35	0.52
Shares as in 1851–55	0.42	0.64	0.68	0.35	0.52
Mean shares (weighted) 1821–41 and 1851	0.42	0.66	0.63	0.36	0.52

Table 3. *Adjustment for weighting bias.*

Total deaths reported	Unadjusted	Adj. with 1851 emigration shares	Adj. with 1851–55 emigration shares
Dysentery	8.53	8.96	8.98
Diarrhoea	3.64	3.64	3.65
Fever	18.58	18.44	18.69
Starvation	2.02	2.31	2.35
Consumption	10.29	10.16	10.04
Dropsy	2.05	2.11	2.09
Marasmus	4.98	4.77	4.79
Cholera	3.64	3.21	3.29
Infirmity and old age	9.11	8.93	8.91
Total specified	62.83	62.52	62.79
Others	37.17	37.48	37.21
Total	100	100	100

majority of the deceased missing from the census must have been people whose relatives had either also died or had emigrated.<sup>7</sup>

To correct for this truncation bias, we applied the following weighting schemes to the ‘missing’ dead: assume that the distribution of diseases among the missing dead was as reported in the county with highest death rate (Mayo), the counties with highest out-migration rates (Clare and Tipperary, depending on the assumptions made) or the counties with the highest overall population loss (Sligo and Roscommon). These shares are

<sup>7</sup> This view is confirmed by simple regressions in which the various estimates of the degree of under-reporting are regressed on measures of the sum of mortality and outmigration and in which the coefficients were consistently significant and negative. The value of  $\lambda$  is strongly and negatively correlated with total mortality, which suggests that death was a main determinant in the incidence of under-reporting, but because of errors in measurement and the appearance of the estimated people dead in terms on both sides of the equation, these estimates are suspect.

Table 4. *Irish deaths, 1846–50, by cause of death: correcting for truncation bias.*

	Mayo weights A	Mayo weights B	Clare weights A	Clare weights B	Tipp weights A	Tipp weights B	Rosc weights A	Rosc weights B	Sligo weights A	Sligo weights B
Dysentery	12.24	12.22	8.49	8.49	7.75	7.76	9.33	9.33	10.06	10.06
Diarrhoea	3.32	3.32	3.5	3.5	3.84	3.84	3.44	3.44	4.58	4.58
Fever	20.56	20.55	22.33	22.31	21.11	21.09	20.42	20.41	18.28	18.28
Starvation	4.82	4.8	2.11	2.11	1.75	1.75	3.16	3.16	2.66	2.66
Consumption	7.78	7.8	7.99	8	9.93	9.93	9.35	9.36	9.17	9.17
Dropsy	2.09	2.09	1.81	1.81	1.83	1.83	1.95	1.95	1.84	1.84
Marasmus	3.81	3.82	4.02	4.03	4.85	4.85	5	5	4.31	4.32
Cholera	3.1	3.1	4.26	4.26	3.34	3.34	2.26	2.26	3.4	3.4
Infirmity	7.1	7.11	7.17	7.18	8.88	8.88	8.54	8.55	8.44	8.44
Total specified	64.81	64.8	61.68	61.68	63.27	63.26	63.34	63.33	62.85	62.85
Others	35.19	35.2	38.32	38.32	36.73	36.74	36.66	36.67	37.15	37.15
Total	100	100	100	100	100	100	100	100	100	100

*Note:* Weights *A* use (1) the 1851 emigration shares of each county to compute the distribution of emigration rates and (2) version I of the *pre*-famine death rates. Weights *B* use the 1841 emigration share and Version II of *pre*-famine death rates.

then multiplied by the estimated number of unreported dead, and added to the reported ones. The results are provided in Table 4. Table 4 gives us a notion of how serious the biases in the data are. Starvation and dysentery, clearly, are most sensitive, being highest when we apply the Mayo weights and the lowest when we apply the two Munster county weights. All the same, the margins are not so large as to deny us an approximate decomposition of excess famine mortality.

The final step is to provide a breakdown of the contributions of the several diseases to excess famine mortality. This is done by comparing total mortality between 1845 and 1850 to normal mortality. Our estimate of normal mortality is that for 1840, as reported in the 1841 census, but adjusted for under-reporting as before. Table 5 describes the *difference* between the 'normal' pattern so defined and a range of estimates of total disease-specific famine mortality rates during the period.

Clearly the choice of weights matters. Nevertheless Table 5 implies that almost *every* disease listed by Wilde contributed something to excess mortality during the famine. The meaning of this finding seems clear: despite the problems of under- and mis-reporting, the famine's physiological impact on the population at large went beyond the direct and immediate effects of 'famine diseases'.

The link between malnutrition or famine and fever was controversial during the Irish famine itself (Ó Gráda 1997, p. 137; Geary 1997, p. 101). Further examination of Table 5 suggests that, roughly speaking, half of famine mortality was caused by diseases associated directly with bad nutrition and the other half from those resulting from the indirect effects of the famine on personal behaviour and social structure. The former status would include diarrhoea, dysentery, respiratory infections (including tuberculosis), starvation, dropsy, and a few less important diseases. Fever, cholera, and most of the diseases included in 'others' had little direct nutrition-sensitivity.

The adjusted data give us a better handle on many of the most interesting issues regarding the Famine. Table 6 gives the estimated totals dying in Ireland's four provinces of five mainly famine-related illnesses between 1846 and 1851. These are what are described in the report as dysentery, diarrhoea, dropsy, starvation, and 'fever'. 'Fever' presumably includes deaths from typhoid, typhus and relapsing fever (MacArthur 1956, pp. 265–68). The implication that the famine killed roughly twice as many people in proportion in Munster and in Connacht as it did in Ulster and Leinster is perhaps not too far off the mark.

Two plausible nosological points emerge. First, the graver the crisis, the higher the incidence of starvation and dysentery-diarrhoea, and the more likely were these to have been the proximate cause of death. Second, the proportion of famine-related deaths due to 'fever' across provinces tended

Table 5. *Decomposition of famine excess mortality, Ireland 1846–50.*

	Mayo weights	Clare weights	Tipperary weights
<i>Hunger sensitive:</i>	<b>40.82</b>	<b>28.90</b>	<b>29.05</b>
Dys. and Diarrh.	28.24	21.55	20.79
Starvation	9.06	3.97	3.30
Dropsy	1.96	1.42	1.46
Marasmus	1.56	1.96	3.50
<i>Partially sensitive:</i>	<b>29.48</b>	<b>35.75</b>	<b>36.40</b>
Consumption	4.64	5.03	8.67
Others	24.84	30.72	27.73
<i>Not very sensitive:</i>	<b>29.73</b>	<b>35.37</b>	<b>34.54</b>
Fever	27.51	30.84	28.53
Cholera	5.7	7.88	6.15
Infirmity, old age	-3.48	-3.35	-0.14
Total	<b>100</b>	<b>100</b>	<b>100</b>

to be fairly constant across provinces although the *incidence* of fever of course increased sharply in the worst-hit provinces.

#### 4. Knowledge or income?

The Great Irish Famine killed at least one million people, but between 1846 and 1851 population declined by more than that, since the famine was also responsible for emigration on a massive scale and for hundreds of thousands of averted births (Mokyr 1980, Boyle and Ó Gráda 1986). Most of the post-colonial sub-Saharan and South Asian famines seen on our television screens seem relatively mild by comparison (Ó Gráda 1997). In the twentieth century the *truly* murderous famines have tended to be man-made: the Soviet Famine of 1932–3 and Mao's 'great leap forward' famine of 1959–62 killed many more people, although the reference populations were also much larger.

Perhaps one of the reasons why mortality was so high in Ireland is that the mechanisms linking famine to increased mortality through infectious disease were so poorly understood at the time. The problem can be laid out starkly by noting that famine kills in large part through poverty and ignorance. At one extreme, when people fall below some absolute subsistence level, they will die no matter how much they know about the causes of disease. At the other, even well-fed individuals are at risk during famine if they are not aware that they are at increased risk of infection and do not know how to avoid contagion. In between, there is a more complex reality in which people have only a partial understanding of the modes of infection, or are too poor or too weak to avoid them. Knowledge is not enough: even today in poor countries, poor

Table 6. *Estimated disease-specific mortality rates per 1,000, by province (1846–50).*

Total death rates per 1000, 1846–50	Ulster		Leinster		Munster		Connacht	
	Mayo weights	Clare weights	Mayo weights	Clare weights	Mayo weights	Clare weights	Mayo weights	Clare weights
<i>Hunger sensitive:</i>	<b>38.79</b>	<b>29.51</b>	<b>42.98</b>	<b>34.43</b>	<b>87.14</b>	<b>66.72</b>	<b>118.56</b>	<b>86.31</b>
Dys and Diarrh.	22.51	17.30	21.88	17.07	54.05	42.58	71.92	53.81
Starvation	5.98	2.02	5.57	1.92	16.03	7.32	26.89	13.12
Dropsy	4.05	3.63	4.13	3.75	6.15	5.22	8.12	6.67
Marasmus	6.25	6.56	11.40	11.69	10.91	11.60	11.63	12.71
<i>Partially sensitive:</i>	<b>81.46</b>	<b>86.35</b>	<b>94.43</b>	<b>98.94</b>	<b>130.95</b>	<b>141.69</b>	<b>147.33</b>	<b>164.29</b>
Consumption	16.76	17.07	22.02	22.3	20.32	20.99	21.96	23.01
Others	64.70	69.28	72.41	76.64	110.63	120.70	125.37	141.28
<i>Not very sensitive:</i>	<b>48.56</b>	<b>52.95</b>	<b>63.83</b>	<b>67.70</b>	<b>98.41</b>	<b>108.08</b>	<b>120.17</b>	<b>135.44</b>
Fever	31.21	33.80	35.32	37.72	69.18	74.89	87.06	96.07
Cholera	3.01	4.71	8.00	9.57	10.90	14.63	11.43	17.33
Infirmity, old age	14.34	14.44	20.31	20.41	18.33	18.56	21.68	22.04
<b>Total</b>	<b>168.82</b>	<b>168.81</b>	<b>201.06</b>	<b>201.06</b>	<b>316.50</b>	<b>316.49</b>	<b>386.05</b>	<b>386.04</b>

water quality, overcrowding, and the prohibitive cost of medical treatment account for the continued incidence of infectious diseases, and their heightened role in time of famine. Extreme poverty is responsible for children catching deadly diseases even when their parents are familiar with the modes of transmission, simply because they cannot afford the minimal needs for prevention.<sup>8</sup>

All the same, ignorance, too, could be deadly. Neither the victims, nor the authorities, nor medical personnel understood how the diseases responsible for the bulk of Irish famine mortality did their work. How important was their ignorance in determining the demographic impact of past famines? How much difference would better knowledge have made? Table 5 suggests that more knowledge might have made a considerable difference. At least one-third of all famine mortality was caused by diseases which some people might have avoided had they better understood what exactly made them ill. Typhus and relapsing fever, endemic diseases in Ireland (MacArthur 1956, pp. 265–8), were transmitted by the human louse. The onset of famine undoubtedly made avoiding contact with lice more difficult. Yet it stands to reason that a better realisation of the dangers that lice implied would both have reduced the threat of fever in normal times and prompted efforts to slow down any epidemic during the famine itself (Geary 1996, p. 50). This holds particularly for less affected areas, as well as for the better-off.<sup>9</sup> Two telling indicators of the role of ‘spillover effects’ from the starving rural masses to others are the excess mortality in Dublin city, and the efforts made by the authorities in Belfast to keep out famine immigrants (MacArthur 1956, p. 280; Ó Gráda 1999, ch. 5). Even in the case of deaths from dysentery and diarrhoea, many deaths occurring through dehydration might have been avoided had people only known basic facts such as the need to replace fluids in patients and the importance of boiling drinking water before use. Neither patients nor doctors had such knowledge in the 1840s.

<sup>8</sup> Thus in Thane, near Bombay, an Indian woman who had already lost two children through water-borne illnesses pointed out that ‘to boil water consistently would cost the equivalent of \$4.00 in kerosene, a third of her earnings’. In Nigeria in the early 1970s (when GDP *per capita* was £100–150) the cost per patient of fluids for treating diarrhoeal diseases was £4 using locally made fluids and £20 using commercial fluids. The greatest problem was getting the fluid to the patient or the patient to the fluid. See *International Herald Tribune*, 9 January, 1997; Bryceson 1977, p. 111; World Bank 1979, Tables 1 and 2.

<sup>9</sup> It bears noting that though the Irish famine killed mainly very poor people, many who were by no means poor succumbed as well. Indeed, the poor had built up some immunity to diseases such as mild typhoid fever, so that during the Famine when fever struck the higher classes they were just as likely to succumb. At greatest risk were people such as clergymen, relief workers, and medical practitioners, whose work involved frequent contact with the diseased. In Ireland as a whole nearly two hundred doctors and medical students died in 1847, three times the pre-famine average. Catholic and Protestant clergymen also died in large numbers (MacArthur 1956, p. 311; Froggatt 1989, pp. 148–50; Kerr 1996, pp. 22–25).

Most of the worst afflicted regions of Ireland had very few trained medical personnel anyway: in 1841, Mayo, probably the worst hit county in Ireland, had one medical practitioner for every six thousand people. This compares to, say, the city of Dublin where there was a medic for every 510 people. Ireland as a whole, with one medical practitioner for every three thousand people, was relatively well endowed with doctors compared to much of the less developed world today. Whether this represented an advantage in terms of quality is dubious, however, given the low quality of mid nineteenth-century medical expertise. The inability to treat ailments that were not necessarily lethal outlasted the famine: years later, medical advice books still recommended a healthy dose of castor oil as a remedy for a child suffering from diarrhoea, without mentioning the need for rehydration. During the famine, doctors were still bleeding severely malnourished people (reportedly with 'mixed' results) and administering such medications as tartar emetic, a powerful expectorant that contributed to dehydration. Even as learned a physician as William Wilde did not really understand the basics of how to treat malnutrition and food poisoning, or how fever epidemics spread.<sup>10</sup>

Even without the full knowledge of what causes disease, certain measures could have been taken that would have reduced mortality. Medical practitioners and the authorities of the time to some extent realised the importance of cleanliness in the homes of the poor and of what they deemed to be pure water (Mokyr and Stein 1997, pp. 143–205). Yet this knowledge was neither specific nor accurate. For a large part of the population, moreover, resources may have been the binding constraint: cleanliness and hygiene were luxuries that the Irish poor could hardly afford even in normal times. Many, if not most, walked barefoot much of the time and were forced by poverty to rely on second-hand clothes. The poorest in Ireland shared their accommodation with pigs, poultry, and lice, and clustered settlements made the spread of disease more likely. Their cooking and food conservation skills were rudimentary. When famine struck, hunger made them cold and less likely to shed or change their clothes. It made them move in search of relief and work. The decline in energy meant poorer childcare, less effective care for the ill and the elderly, and probably less fuel and clean water, all of which relied on physical effort.

For the very poor, then, more knowledge and understanding about the causes of disease and death would have done little. Yet disease and death during Ireland's great famine were by no means confined to the very poor. For the better-off sections of the population, the benefits of the new science

<sup>10</sup> A good example of the state of medical science is provided by Wilde's analysis of scurvy. Wilde recognised the possible importance of the change in diet and the use of hard, dry grain instead of fresh vegetables, but then added immediately that the two peculiar causes that more than others contributed to induce scurvy were fluctuations in humidity and temperature and the 'moral depression coupled to inactivity'. See BPP (1856a, pp. 513–14).

would have been more tangible. A better understanding of the causes of disease would have mattered more in preventing epidemics and deaths among those, especially in the towns but also among those sections of the rural population with enough land to hold even a cow or two. Estimating the proportion of this 'slightly better-off class' is of course arbitrary, but the 1841 census classified no fewer than 63 per cent as 'labourers, smallholders and other persons without money, land, or acquired knowledge'. Not all of those, however, belonged to the 'poorest classes' since out of the 1.3 million houses in 1841 only 37 per cent belonged to the poorest quality ('mud cabins having only one room'). All in all, while the Famine doubtlessly was above all a scourge of the poorest, its nationwide impact must be explained in terms of ignorance as well as poverty.

### **5. A comparative perspective**

Medical science has advanced by leaps and bounds since the 1840s. Progress in countering infectious disease came in two distinct stages. First, in the late nineteenth century, came the identification by Robert Koch and Louis Pasteur of pathogenic agents and their mode of transmission, and the use of this knowledge for preventive care. Then, in the 1930s and 1940s, came the emergence of antibiotics. Surely one reason why some modern famines have not resulted in mortality figures on an Irish scale is the ability of modern science to prevent or contain the worst epidemics? Even the achievement of the first stage before 1846 would have made a difference in Ireland. For further insight into this issue we take a comparative look at the causes of death in some historical and modern famines.

If, as noted above, the Irish famine dwarfed most modern famines in its relative impact, how different was its nosological profile? Comparable evidence is scarce and at first sight conflicting. Wilde's data, corrected and aggregated, are compared below with cause-of-death data from some famines in nineteenth-century India and in Russia in the 1920s. The expectation of life in Ireland on the eve of the famine was higher than that in nineteenth-century India but lower than that in Russia in the 1920s (Boyle and Ó Gráda 1986, Adamets 2000). Our reworking of Stephen Wheatcroft's findings for the south Russian province of Saratov produces results uncannily similar to Wilde's (see Table 7). The other Russian nosologies are of poorer quality, with two-fifths of the excess mortality unexplained, but they too stress the overwhelming part played by infectious diseases (Wheatcroft 1981a, 1981b, 1983). The nosologies in official sources for nineteenth- and twentieth-century India are, like the Russian, far less detailed than Wilde's, though their coverage is probably better (Dyson 1991a, Table 3; Dyson 1991b, Table 7; Maharatna 1996, pp. 18–22). The comparison indicates that most of the excess mortality during the great Indian famines of the nineteenth and twentieth centuries were also due to infection (fever, diar-

rhoea/dysentery, cholera, malaria), not from literal starvation. This is also true in the case of Bengal in 1943–4, where malaria was the main killer. The main difference between India in the nineteenth and twentieth centuries or between Ireland in the 1840s and Bengal almost a century later is the smaller role of diarrhoea/dysentery in the latter. Unfortunately the role of literal starvation in India cannot be inferred from the tables.

In the relief of famines in sub-Saharan Africa today medical supplies are deemed as important as food, and several NGOs specialise in medical assistance. Undoubtedly many lives are saved by immunisation, antibiotics, and rehydration. Yet nosological data suggest that it is aggregate mortality rather than the causes of death that have changed. The major causes of death continue to be infectious diseases such as dysentery/diarrhoea, respiratory infections, malaria, and measles. Aggregate cause-of-death data are lacking, but all studies carried out in feeding camps highlight the role of infection. In the mid-1980s four-fifths of deaths in camps in Ethiopia and Sudan were due to diarrhoeal diseases and measles alone (Shears *et al.* 1987; Boss *et al.* 1994; Mercer 1994, p. 34; von Braun *et al.* 1998, p. 134).

However, a very different picture is offered by nosological evidence on a series of smaller, well-documented European famines in the 1940s. Data on the causes of death in Warsaw's Jewish ghetto before its destruction by the Nazis in July 1942 show that as the death rate there quintupled between 1940 and 1941–2, the proportion of deaths attributed to starvation rose from 1 per cent to 25 per cent. Typhus's share remained small, however: 2.4 per cent in 1940, 4.6 per cent in 1941, and 1.7 per cent in 1942. In the towns and cities of the western Netherlands famine killed about ten thousand people during the starvation-winter of 1944–5. Here also starvation accounted for a significant share of the rise, infectious diseases for relatively little. The same holds for a well-documented famine on the small island of Syros in the Aegean Sea in 1941–2, where about one-tenth of the entire population succumbed. On Syros there was almost a complete absence of epidemics, and civil registration data attribute over two deaths in three to literal starvation. Livi-Bacci's account of another twentieth-century European famine – in a part of occupied north-eastern Italy in 1918 – returns a similar verdict (Burger *et al.* 1948; Livi-Bacci 1991, pp. 43–6; Winick 1994; Hionidou, 2000). Finally, a recent nosological analysis of the Soviet famines of 1933 and 1947 suggests a reduced role for epidemic and parasitical diseases. They were responsible for about one quarter of all deaths, compared to one-half in 1922 (Adamets 2000).

All of these European famines occurred before the discoveries of Pasteur and Koch had been translated into effective and widely-available medical treatments or cures. The outcome underlines how an understanding of the modes of transmission of infectious disease can prompt the necessary preventive measures. Those at risk clearly knew the importance of keeping clean, of washing clothes, of disinfectants, and so on. A crucial consequence

Table 7. *Causes of excess deaths in Ireland, Russia, and India.*

Cause of Death	Ireland 1840s	Saratov 1918-22	Petrograd 1918-22	Moscow <sup>c</sup> 1918-22	Bombay 1877	Berar 1897	Berar 1900	Punjab 1900	Ut. Prad. 1908
D.D.G. <sup>a</sup>	24.9	19.7	10.4	16.0	9.7	30.4	37.0	3.0	-1.2
Cholera	6.8	5.1	2.0	0.7	16.5	12.1	9.6	7.6	4.6
Fever	29.2	24.1	19.3	24.6	45.9	29.0	23.9	72.2	90.9
Respiratory <sup>b</sup>	4.8	9.8	19.3	20.2	na	na	na	na	na
Starvation/Scurvy	10.0	5.5	12.8	na	na	na	na	na	na
Other, unknown	24.3	35.8	36.2	38.5	27.9	28.5	29.5	17.2	5.7
Total	100	100	100	100	100	100	100	100	100

*Notes:* <sup>a</sup> Diarrhoea, Dysentery, Gastroenteritis; <sup>b</sup> Includes normal tuberculosis and pneumonia. Defined as 'consumption' for Ireland; <sup>c</sup> Excess deaths in comparison with 1914 levels.

*Sources:* Ireland: Table 5, average of Clare and Mayo weights.

Saratov, Moscow, and Petrograd: derived from Wheatcroft (1983, p. 340; 1981b, pp. 17-8) and Wheatcroft (personal communication, April 1998). The percentages are 1918-22 averages calculated for the entire five year period.

India: Maharatna (1996, pp. 46-7, Table 2.6). We subtracted cause-specific death rates in baseline years from rates during famine years to get excess mortality by cause. We then calculated the percentages of the totals explained by the different causes. Maharatna's D.D.G totals are for diarrhoea and dysentery.

is that the infectious diseases which bulked so large during famines elsewhere were not endemic (compare Seaman *et al.* 1984, pp. 50–1). However, these were all relatively advanced places in economic terms, with high literacy levels, a good supply of medical personnel, clean running water for drinking and washing, changes of clothes, housing that was easier to keep clean, less overcrowding, and adequate cooking facilities for what little food there was.

The nosologies of the famines reported in Table 7 and of modern famines in sub-Saharan Africa have much more in common with Ireland in the 1840s than with famine-affected regions in Europe in the 1940s. Why? Part of the answer must be that while the knowledge may have spread at least as far as medical personnel and officials, behavioural patterns and consumption were subject to a great deal of inertia. It is not enough for people in some sense to ‘know’ what causes disease, they have to be *persuaded* to change their behaviour. More important, the associated remedies must have been difficult to put into practice in the crisis conditions obtaining. Even in ‘normal’ times in sub-Saharan Africa, the world’s main famine-prone region, infectious and parasitic diseases alone are still responsible for nearly half of all deaths, with diarrhoeal diseases accounting for nearly one-quarter of those. Another 13 per cent of deaths are due to respiratory diseases. In Asia (excluding China) the same categories account for about one-third of all deaths (Murray and Lopez 1994). In other words, such diseases are endemic in these places: little wonder, then, that they still dominate during famines. Much more mortality in both Africa and Asia – both crisis and non-crisis – could be prevented by low-cost primary health care such as immunisation, prophylactics, and rehydration. In these underdeveloped areas, however, public health lags rather than leads medical science.

A final comparative point links what we know of the causes of death with the well-known gender gap in famine mortality. Famines, with few well-attested exceptions, are more likely to kill males than females. The male disadvantage, which is present throughout the life cycle except at very young ages, is usually ascribed to physiological factors. This prompts the hypothesis that the more important literal starvation as a cause of excess mortality, the greater the female advantage. A comparison of the rather scarce data on the gender gap supports the prediction. In the Irish Famine the gender gap was very small. In the case of the Great Finnish Famine of 1868 there was a slight female disadvantage, while in Russia in 1892 excess death rates were practically the same for males and females across the life cycle (Adamets 2002; Mokyr and Ó Gráda 2002; Pitkänen 2002). However, in Syros in 1941–2 male deaths accounted for 59 per cent of all deaths and in neighbouring Mykonos for 67 per cent (Hionidou 1995, 2000). Comparing famines in Russia in 1892, 1933, and 1947 is also consistent with the hypothesis; the gender gap was much greater in both 1933 and 1947 than in 1892 (Adamets 2002).

## 6. Conclusion

The dimensions of a disaster depend on the size of the impact and the vulnerability of the society upon which it is inflicted. The functional relation between outcome and the two determinants is, however, additive rather than multiplicative. Even seemingly invulnerable societies can be devastated if the impact is large enough. Conversely, weak and vulnerable societies may survive for long periods if they are lucky enough to avoid major challenges. Sadly, Ireland was not lucky. Ireland's vulnerability was in terms of its overall poverty, the physical impossibility of storing potatoes, and the thinness of markets in basic subsistence goods due to the prevalence of the potato. But there is a second dimension to the vulnerability which compounds the first one, and that is that all populations of the time were vulnerable to an increase in the incidence of infectious diseases in the case of outside shocks. The absence of a clear understanding of the nature of disease meant that the privations and disruptions of the Famine quickly translated themselves into the horror-filled statistics of Wilde's 1851 'Tables of Death'.

A careful analysis of epidemics during past famines can help us toward a better understanding of precisely what happened in the past. The understanding of the epidemiology and etiology of infectious diseases and the physiology of their symptoms, and the knowledge of how to treat patients suffering from basic ailments such as fever and diarrhoea, will remain with us even if antibiotics lose some of their effectiveness with the proliferation of drug-resistant strains. Equally, an analysis of the role of epidemics in twentieth-century famines offers a better insight into famine mortality in a counterfactual mid-nineteenth century Ireland, where the potato failed but where the scientific advances following the work of Pasteur and Koch had already been absorbed. It suggests that had *Phytophthora Infestans* attacked only a few decades later, a better understanding of the basic mechanisms of death would have influenced public health policy and, in particular, would have saved many middle-class lives. However, the analysis of modern third-world famines suggests that many of the poor would still have died. Economic and political progress is a precondition for modern health technologies playing their part in improving the health of the masses.

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