Famine Disease and Famine Mortality: Lessons from Ireland, 1845-1850

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I. *INTRODUCTION*:

It is commonplace to observe that one of the great changes of the modern age is not only that life expectancy is much longer than a century or two ago, but that there has been a radical change in the causes of death. Even in the nineteenth century, infectious disease was by far the biggest cause of death. In our own age, though these diseases have not quite disappeared and some even threaten to make a comeback, they have clearly been relegated to a secondary role in all but the poorest countries. This paper argues that this observation is central to an understanding of the nature of past famines, and of why they may differ significantly from modern famines.

There is some disagreement among scholars about what actually constitutes a >famine=. Definitions range from >the semi-starvation of many people= to >extreme and general shortage of food causing distress and death from starvation among the population=. Whether famines are mostly caused by actual food shortages or by adverse distributional shifts in what Amartya Sen has called >entitlements= is also a controversial issue. In a series of very influential publications since the late 1970s Sen has suggested that famines are not so much crises in the *total* availability of food as in its distribution. A third dispute, prompted by the context of recent famines in sub-Saharan Africa, concerns whether violence and corruption are as much to blame for famines as the forces of nature (Sen 1981, de Waal 1989).

These disputes hold few resonances for the Great Irish Famine of the late 1840s. The Irish famine was not caused by war but by a series of catastrophic crop failures. Its impact was very uneven across regions and classes, but the virtual destruction of the people=s main subsistence crop, the potato, for a number of successive years dominated >entitlement= considerations. This, then, was a real famine in the old-fashioned sense of the word and not a case in which, following Alex de Waal=s distinction, a >scarcity= was being confounded with a >famine= (de Waal 1989: 25-28). The Irish famine was a disaster with strong Malthusian features: a catastrophic reduction of the food supply led to major demographic re-adjustment. In earlier times, famines had been commonplace in Europe, but historical demographers now agree that most of them produced only temporary population adjustments. Past famines were normally followed by a period of rapid population growth which soon restored population levels to where they had been. Not so in Ireland.

What did people *really* die of during past famines? The short answer to this question is that they died mainly of infectious diseases. These diseases, however, came in various kinds, at different times, and with differing levels of intensity. The causation of death during a famine turns out to be a difficult question, raising the usual philosophical difficulties of causation. Hunger and infectious disease interact

in complicated ways, some of which operated through the human body and some through the fabric of human society. In examining such issues in the context of the Irish famine of 1846-51, we must note at the outset that this famine was in many ways sui generis, and that lessons learned from it may not apply to other cases in Europe or elsewhere. Furthermore, because mid-nineteenth century medical terminology and concepts differ so much from today=s, contemporary evidence, both statistical and qualitative, is often difficult to interpret.

Two broad classes of causes were responsible for augmented mortality during famines. The first is directly nutrition-related, and includes 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 the doctors 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=.[1] 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 Census records only 17 deaths from starvation for the entire year of 1840, out of a total reported deaths numbering over 140,000 for that year. In 1847 slightly over 6,000 people were

[1]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.

reported to have died of starvation, out of nearly one quarter of a million 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 (BPP 1843: 181-83, BPP 1856b: 663).

Most of the other diseases that killed people during the famine were, as noted, infectious diseases. Some were opportunistic diseases that took advantage of the fall in nutritional status and the general environmental deterioration. Specialists distinguish between *individual* immunosuppression and *social* or *collective* immunosuppression. Individual immunity declines as the body is deprived of food, especially proteins. It should be pointed out that recent research has questioned the widely held assumption that malnutrition *inevitably* leads to increased susceptibility to infection (Dirks 1993, Carmichael 1983:53). 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, however, 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 the *quality* of the food as well as its quantity. One consequence, unsuspected by contemporaries, was that the intake of Vitamin C, now recognized 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 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 and increased fatality of other diseases.

Community resistance to disease 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 the productivity of labor throughout the economy, leading to positive feedback effects that reinforced the initial shock. 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 (Fogel 1991, Drèze and Sen 1989: 50-5, Maharatna 1996: 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 (Taylor 1983, Carmichael

1983). These include:

X *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.

X *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. Contemporary reports described the diseases suffered by people from consuming unfamiliar and improperly prepared foods from Indian meal.

X *Infectious diseases due to population moving around*. Famine conditions frequently led to panic-stricken people to quit their homes in the search for food. Mobility increases mortality 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=.[2]

X *Infectious diseases due to hygiene deterioration as people become weak and despondent*. The impact of serious malnourishment is not right away death 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

[2]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.

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=.

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

II: 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 analogous tables in the unusually rich and accurate 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 is 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 recognize, 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: 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 and useless as a source of information about the Great Famine. The main reservations historians have about these tables are as follows:

X The *total* numbers are clearly serious underenumerations 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 underenumeration 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 underenumeration 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.[3] 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 underreporting, note that the 1841 census reported total Irish population at 8.2 million and the 1851 census at 6.6 million. Total famine mortality can be estimated by first projecting Irish population from 1841 to the eve of the famine in 1846. We then add the births that occurred in the Famine years 1846-50 (adjusted for a famine-induced decline in fertility) and subtract out estimated out-migration during the Famine years. This yields a total of 1.9 million people dying in Ireland in those five years.[4] The 1851 census tables report a total of 985,000 people dying. For the country as a whole, thus the reporting factor is about 52 percent. This factor, moreover, varied substantially from county to county. The implications of underreporting 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 was subject to a negative bias, that is, the diseases that increased the most would be systematically underreported.

X At least some of the disease categories seem to be of a rather fuzzy nature. The 1851 census

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[3] 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.

[4] Given that the death rate in a typical year in Ireland before the famine was about 24 per thousand, this implies that *excess* mortality during the Famine (not including averted births) came to about 1.1 million. See Mokyr 1980.

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 Census is >fever=, responsible for 222,000 (over 16 percent of all reported deaths), the bulk of them 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.*

X It seems that some respondents in 1851 projected some of their famine memories back to pre-famine days, reporting famine-related diseases as if they had occurred before 1845. This can easily be inferred 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. The same phenomenon is illustrated by the report of cholera deaths; although cholera only reached Ireland in December 1848, the census reported a total of 1,376 cholera deaths in the years 1841-47 (plus a further 2,502 in 1848).[5] This projection bias should create a positive bias, in that the diseases that increased during the famine are overrepresented. How serious are these biases? A simple t-test of the hypothesis that the rates per 1,000 were the same in 1840 and 1842 does not support the rejection of the null of no significant differences. This finding suggests that the bias introduced in the 1851 census because of the disappearance of hundreds of thousands of people, while it biased the *total* counts, did not bias the distribution of *pre*-famine diseases intolerably. The difficulty with the famine years is more serious and will be addressed below.

[5]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 1000, adj.)	(Percentage)	(per 1000, 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	24.00	100.00	24.00

(*) Urinary, Generative, Locomotive, Tegumentary

X 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 immunodepression caused by malnutrition. All the same, this still leaves unexplained some 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. Disease that should hardly be affected by the Famine such as diseases of the >locomotive organs= and >diseases of uncertain seat= (tumors, 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.[6] As a proportion of the total number of deaths, these diseases declined, but their increased incidence remains rather puzzling.

X Another case in which survivors= memories seem to have let them down relates to

[6]The Wilde report itself underlined the mystery of this by arguing that adverse weather conditions in 1847 were responsible for the rise in deaths from diseases such as rheumatism. While the people in question may have suffered from the diseases in question, their deaths are more plausibly attributed to famine-related symptoms, whether from reduced immunity due to malnutrition or an undiagnosed condition.

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 were also subject to the same bias.[7]

X The number of reported starvation deaths is surprisingly low. The total for the years 1845-50 is only 20,402. Though, as noted earlier, during famines the proportion of people dying of literal starvation is usually small, in Wilde=s own words >no pen has ever recorded the numbers of the forlorn and starving who perished by the wayside or in the ditches,... whole families lay down and died= (BPP 1856a: 243.).

To take the Tables of Death of the 1851 census at face value would thus be a grievous mistake. Yet to abandon them altogether as a source of information 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 organization, 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 (a) drawing inferences and making comparisons where the biases just noted do not present a problem, and (b) pinpointing and adjusting for some of their worst shortcomings on the basis of what is known about Irish population statistics in this

[7]Thus the proportions of people dying of >cancer and fungus= and of >burns and scalds= in the autumn were only 19 per cent and 17 per cent, respectively, of the annual total.

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.

III: *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 underreporting 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 underreported due to the disappearance of entire families. If the degree of underenumeration varied from disease to disease, the result might be an underestimate of deaths due to the most murderous of them. We thus attempt adjustments that yield estimates of underenumeration.

The underenumeration 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 λ is the underreporting factor. To assume that the λ's are the same across counties would overweight the distribution of diseases in counties that underreport the least and underweight those counties where underreporting was the worst. Since underreporting clearly was a function of the severity of the famine, this would bias the nation-wide distribution of disease toward underreporting famine-specific diseases. We call this *weighting* bias. Secondly, simply adjusting for underreporting 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 λ. We could in principle follow a procedure similar to the one 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.[8] 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.[9] We also adjust for internal migration, as reported in the 1851 census. The overall estimates of the λ's are moderately sensitive to these assumptions and as there is no obvious way to choose among them, we

[8]The distribution 1821-41 was estimated by Cousens (1965) using cohort analysis. The total outmigration in Ireland to North America in 1846-50 is reported to have been about 925,000 (Vaughan and Fitzpatrick 1978: 260). To that we added we added migration to England at about 40,000 people a year based on the British census of 1851.

[9]The two alternatives are laid out in Mokyr (1985: 34-35, 68-69). Version I adjusts the prefamine under-reporting of death rates by a single nation-wide adjustment factor and then computes net out-migration residually. The alternative (Version II) uses the Cousens procedure to estimate the county-by-county outmigration rates and computes the death rate distribution residually.

present upper and lower bounds in Table 2.

Table 2: ESTIMATES OF UNDERREPORTING COEFFICIENTS (λ) BY PROVINCE

Assumption about emigration	Ulster	Leinster	Munster	Connacht	Ireland
Shares as in 1851	.38	.70	.69	.35	.52
Shares as in 1851-55	.42	.64	.68	.35	.52
Mean shares, (weighted) 1821-41 and 1851	.42	.66	.63	.36	.52

The province-level data make sense in that the λ'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 show a consistent negative relation between the λ's and the *reported* incidence of starvation (though the latter itself is of course mismeasured). The nationwide-wide nosology resulting after adjustment for the weighting bias is provided in Table 3.

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

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 weighting problem by computing county-specific underreporting by itself is insufficient, however, because it assumes implicitly that the disease distribution for those whose deaths were not reported in the census was the same as for those who were reported in that county. This seems unlikely, as the majority of those missing from the census must have been those deceased whose relatives had either died as well or had emigrated.[10] We therefore 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 then multiplied by the estimated number of unreported dead, and added to the reported ones. The results are provided in Table 4.

[10]This view is confirmed by simple regressions in which the various estimates of the degree of underreporting are regressed on measures of the sum of mortality and outmigration and in which the coefficients were consistently significant and negative. The values of λ is strongly and negatively correlated with total mortality, which suggests that death was a main determinant in the incidence of underreporting, 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 gives us a notion of how serious the biases in the data are. Starvation and dysentery, clearly, are most sensitive and tend to be high 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.

Table 4: CORRECTING FOR UNDERREPORTING

	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 (see footnote 21). Weights *B* use the 1841 emigration share and Version II of prefamine death rates.

The central finding of this research is the decomposition of famine mortality into the contributions of the several diseases. A distribution by cause of the famine mortality can be carried out by comparing total mortality between 1845 and 1850 to normal mortality. As noted, the reported mortality from famine-diseases for the pre-famine years 1842-44 in the

1851 Census is probably upward biased. We have therefore compared the disease-specific famine mortality rates with those of 1840, as reported in the 1841 census and adjusted for underreporting as before. The results, reported in Table 5, are thus based on the comparison of an Ireland with an 1840 nosological structure with the actual deaths reported in the 1851 census for the famine years. Table 5 decomposes total mortality into a >normal= component that would have occurred in any event and a >famine= component. To carry out this decomposition, we utilize the 1840 census to approximate what >normal= mortality patterns would have looked like in the absence of the famine. We construct the number of people that would have died of each disease by multiplying the per thousand rates of 1840 by the mean population between 1846-50. These are the numbers that would have died of each disease had the prefamine *pattern* of diseases dominated the actual population of 1846-50. We subtract from these numbers the actual estimated death figures on various assumptions. This table can be expanded to account for other diseases whose contribution to famine mortality is of interest such as smallpox and >convulsions= and we plan to expand our research into that direction. The tentative results are presented in Table 5:

Table 5: DECOMPOSITION OF FAMINE MORTALITY

	Mayo weights	Clare weights	Tipperary weights
Hunger Sensitive:	**40.82**	**28.90**	**29.05**
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
Partially Sensitive:	**29.48**	**35.75**	**36.40**

Consumption	4.64	5.03	8.67
Others	24.84	30.72	27.73
Not very sensitive:	**29.73**	**35.37**	**34.54**
Fever	27.51	30.84	28.53
Cholera	5.7	7.88	6.15
Infirmity, old age	-3.48	-3.35	-0.14
Total	**100**	**100**	**100**

It is interesting to note 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: 137; Geary 1997:101) .[11] Further examination of Table 5 suggests that, roughly speaking, one half of famine mortality was caused by diseases that were the result of bad nutrition and the other half from those resulting from the indirect effects of the famine on personal behavior and social structure. The former status would include diarrhoea, dysentery, respiratory infections (including tuberculosis), starvation, dropsy, and a few smaller diseases. Fever, cholera, and most of the diseases included in >others= had little direct nutrition-sensitivity.

IV: FURTHER ANALYSIS OF THE FAMINE NOSOLOGIES

The adjusted data can give us a better handle on many of the most interesting issues

[11] It should be remembered that >fever=encompassed a whole range of diseases, including some like typhoid which were clearly famine-sensitive. Wilde pointed out that those who recovered from the immediate effects of starvation, contracted a peculiar and generally fatal form of typhus which no administration of food could avert (BPP 1856a: 734).

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: 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.

Table 6: ESTIMATED DISEASE-SPECIFIC MORTALITY RATES PER 1000 (1846-50)

	Ulster		Leinster		Munster		Connacht	
Total death rates per 1000, 1846-50	Mayo weights	Clare weights	Mayo weights	Clare weights	Mayo weights	Clare weights	Mayo weights	Clare weights
Hunger Sensitive:	**38.79**	**29.51**	**42.98**	**34.43**	**87.14**	**66.72**	**118.56**	**86.31**
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
Partially Sensitive:	**81.46**	**86.35**	**94.43**	**98.94**	**130.95**	**141.69**	**147.33**	**164.29**
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

Not very sensitive:	**48.56**	**52.95**	**63.83**	**67.70**	**98.41**	**108.08**	**120.17**	**135.44**
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
Total	**168.82**	**168.81**	**201.06**	**201.06**	**316.50**	**316.49**	**386.05**	**386.04**

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

The figures over time as well as the breakdown by province show, as one would expect, that the incidence of these diseases roughly corresponded with the severity of the Famine. The proportion and numbers of those dying of fever and digestive-tract diseases rose in 1845, accelerated in the next year, then peaks in the horror year 1847. The increase in the two diseases is very sharp, of course, but whereas the incidence of dysentery and diarrhoea increased by a factor of eight to twelve, as opposed to a factor of four to five for fever, they start from a much lower basis and thus the contribution of the two diseases to increased famine mortality is comparable in absolute terms. The two measures do not move tightly together, indicating the difficulty of measuring the >impact= of a disease. To some extent famine-related diseases must have >crowded out= others, that is, people who succumbed to famine-related diseases would have died of other diseases in its absence. At the same time, people who were relatively well-off and did not starve, still were infected by contagious diseases contracted by their less fortunate neighbours.

VI: KNOWLEDGE OR INCOME?

Was one of the main reasons why the Irish famine was so much more deadly than modern events that the actual mechanisms linking famine through infectious disease to increased mortality were poorly understood? The problem can be laid out starkly by noting that Famine kills 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 do not understand that they are at increased risk of infection and how to avoid contagion. In between, there is a more complex area in which people are aware to some extent of the modes of infection but do not get it quite right, or are too poor or too weak to avoid them. In poor countries, even today, water quality, over-crowding, and the prohibitive cost of medical cures 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.[12]

[12] 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 diarhoeal 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*, Jan. 9, 1997; Bryceson 1977: 111, World Bank 1979: tables 1 and 2.

It bears repeating that most victims of past famines, including the Irish famine, were not killed directly by hunger and exposure but by micro-organisms. Neither the victims, nor the authorities, nor medical people understood this basic fact until the 1880s. Their ignorance of the exact nature of what it was that was killing most famine victims is a crucial element in determining the demographic impact of past famines. It certainly is true that even without the full knowledge of what causes disease, certain measures could have been taken that would have averted the worst. Nonetheless, even with such knowledge high peaks of mortality can occur. Especially during a period of crisis, even a rudimentary understanding of the mechanisms of infection can be of the greatest importance in preventing or limiting massive mortality crises. Such understanding was sadly missing in the 1840s. 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.[13]

How much difference would better knowledge have made? The evidence provided above would suggest that more knowledge might have made a considerable difference even if it would not have prevented mass mortality. Table 5 suggests that at least one-third of all Famine mortality was caused by diseases such as fever which might have been avoided had people understood better what exactly made them ill. Both typhus and relapsing fever were transmitted by the human louse, and while avoiding lice would have become more difficult in the desperate uprooted populations gathering in poor houses, relief works and food depots, it stands to reason that had people only known how dangerous lice were, that efforts could have been made to slow down the epidemic (Geary 1996: 50). This holds particularly for urban areas, as well as for deaths among the better-off in the countryside. 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: 280; Ó Gráda 1999: Ch. 5). Moreover, even among

[13] A good example of the state of medical science is provided by Wilde's analysis of scurvy. Wilde recognized the possible importance of the change in diet and the use of hard, dry rain 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: 513-14).

those dying of dysentery and diarrhoea, death occurred through dehydration which might have been avoided in many cases 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.

Though the famine killed mainly very poor people, many who were by no means poor succumbed as well. Indeed, the Irish 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: 311; Froggatt 1989: 148-50; Kerr 1996: 22-25). A better understanding of the underlying disease transmission mechanisms and the consequent need for cleaner water and greater hygiene might have saved not only some of them, but surely many others too.

Most of the worst afflicted regions of Ireland had very few trained doctors: in 1841 Mayo, probably the worst hit area of Ireland, counted a total of 37 physicians and 28 surgeons, or one medical practitioner for every six thousand people. This compares to, say, the town of Dublin where there was a medic for every 510 people. Yet the problem was not really one of the lack of medical personnel. More important was the low quality of medical expertise and that the people themselves did not know what made them ill nor, once they were ill, how to treat ailments that were not necessarily lethal. This was hardly specific to the remote rural areas of Connacht: years after the Famine, many 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, medical people were still bleeding severely malnourished people (reportedly with >mixed= results) and administering such medications as tartar emetic, a powerful expectorant that contributed to dehydration. The Irish misfortune was that such ignorance had to confront an unprecedented shock to the food supply system in a poor and comparatively primitive rural economy.

The belief that infectious disease >like the ague, owes its origin to terrestrial

miasms=, was commonplace among medical men at the time of the Irish famine. As noted earlier, the role played by social conditions was controversial, yet most doctors and officials realized the importance of cleanliness in the homes of the poor and of what they deemed to be pure water (Mokyr and Stein, 1997). Yet famine made hygiene and pure water more difficult to maintain and obtain. Moreover, when diseases struck, the medical profession was as powerless to cure the sick as it was to prevent the spreading of the epidemic.

Medical science has advanced by leaps and bounds since the 1840s. 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? The decline in infectious disease consisted of two distinct stages. First, in the late nineteenth century, came the identification 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. Even the achievement of the first stage before 1846 would have saved many victims of the Irish famine. For further insight into this issue we take a comparative look at the causes of death in some historical and modern famines.

VII: A COMPARATIVE LOOK

In his ghost-written introduction to one of the classics of Irish famine historiography, the historian Kevin Nowlan wrote that >perhaps all that matters is that many, many died=(in Edwards and Williams 1956: vii). Modern accounts dispute this, insisting that it does matter *how many* died, as well as *who* died and *from what cause*. Two estimates, independently-derived but employing a very similar methodology, have by now firmly established the number of *excess* deaths (that is, deaths that would not have occurred in the absence of the Famine) at over one million (Mokyr 1980, Boyle and Ó Gráda 1986). The actual population deficit is larger, as the Famine was also responsible for emigration on a massive scale and for hundreds of thousands of averted births (children who would have been born were it not for the Famine). Such numbers led the eminent economist Amartya Sen to declare in an unguarded aside that >he knew of no other famine in the world in which the proportion of people killed was as large as in the Irish famines in the 1840s=(Sen 1995). This may exaggerate, yet it underlines the dimensions of the Irish famine in terms of sheer demographic effect. By comparison, most of the post-colonial sub-Saharan and South-Asian

famines that we see on our television screens seem relatively mild (Ó Gráda 1997). In the twentieth century the *truly* murderous famines have tended to be man-made: the Ukrainian Famine of 1932 and Mao's >great leap forward= famine of 1959-62 killed many more people although the reference populations were also much larger. In comparative terms, a noteworthy feature of the Irish famine is that it occurred in a country basically at peace. This tranquillity contrasts sharply with post-colonial famines in Biafra, Somalia, and Ethiopia.

If 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. Below Wilde=s data, corrected and aggregated, are compared with cause-of-death data from some famines in nineteenth-century India and in Russia in the 1920s. Our reworking of Wheatcroft=s data on the south Russian gubernaia of Saratov produces results uncannily similar to Wilde=s (see Table 7). The other Russian nosologies are of poor 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 1997:18-22). In Table 7 the outcome is summarized and compared with our picture for Ireland in the 1840s. It indicates that most of the excess mortality during the great Indian famines of the nineteenth and twentieth centuries were also due to infection (fever, diarrhoea/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. Perhaps this is a sign that some of the messages of modern medicine had got through to India by the 1940s; any stronger claim is complicated by the significant role of diarrhoeal diseases in Berar in 1897-1900.[14] Unfortunately the role of literal starvation in India cannot be inferred from the tables.

In famine relief in Africa today medical supplies are deemed as important as food,

[14] Perhaps this was a product of the >unwholesome water and food= mentioned by the local sanitary commissioner. See Maharatna (1997: 57).

and undoubtedly many lives are saved by antibiotics and rehydration. Unfortunately, cause-of-death data are very scarce. Data collected in refugee camps in Sudan in the mid 1980s make for disheartening reading, however: 17 per cent of deaths were attributed to malaria, 27 per cent to diarrhoeal disease, and another 23 per cent to respiratory disease (Mercer 1992: 34).

TABLE 7: CAUSES OF EXCESS DEATHS IN IRELAND, RUSSIA, AND INDIA

Cause of Death	Ireland 1840s	Saratov 1918-22	Petrograd 1918-22	Moscow 1918-22	Bombay 1877	Berar 1897	Berar 1900	Punjab 1900	Ut. Prad. 1908
D.D.G.[a]	23.4	22.8	9.8	12.5	9.7	30.4	37.0	3.0	-1.2
Cholera	7.0	8.5	2.0	0.8	16.5	12.1	9.6	7.6	4.6
Fever	24.5	31.9	15.0	13.3	45.9	29.0	23.9	72.2	90.9
Respiratory	12.8	13.5	20.6	10.5	n.a.	n.a.	n.a.	n.a.	n.a.
Starv./Scurvy	4.1	6.8	12.8	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.
Other, Unknown	28.2	16.5	39.8	43.3	5.7	28.5	17.2	17.2	5.7
Total	100	100	100	100	100	100	100	100	100

a - Diarrhoea, Dysentery, Gastroenteritis.

Sources:

IRELAND: see text

SARATOV: S. Wheatcroft (1983: 340). The percentages are weighted averages of the annual totals for 1918-1922, where the weights are Wheatcroft=s estimates of annual excess mortality. Respiratory includes tuberculosis and pneumonia.

MOSCOW and PETROGRAD: Wheatcroft (1981b: 17-8).

INDIA: Maharatna (1997: 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. Also has data on smallpox but its contribution is small (as in Ireland).

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 one per cent to one-quarter. 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 the Greek famine of 1941-2. Livi-Bacci=s account of another twentieth-century European famine -- in a part of occupied north-eastern Italy in 1918 -- returns a similar verdict (Livi-Bacci, 1991: 43-6, Winick, 1994, Burger, Drummond, and Stanstead, 1948, Hionidou, 1995: 293). These famines occurred before the discoveries of Pasteur and Koch had been translated into an effective and widely-available new medicine. The outcome underlines the importance of an understanding of the modes of transmission of infectious disease and of preventive measures. In these places the people at risk clearly knew the importance of keeping clean, of disinfectants, and so on. But these were relatively advanced places in economic terms, with universal literacy, a good supply of medical personnel, clean running water for drinking and washing, lots of changes of clothes, housing that was easier to keep clean (no thatch, no pigs, no mud floors), less overcrowding, adequate cooking facilities for what little food there was.

The nosologies of the Indian and Russian famines reported above have much more in common with Wilde=s than with those of famine-affected regions of Europe in the 1940s. Why? The answer must be in part that while the knowledge may have existed, behavioral patterns and consumption were subject to a great deal of inertia. It is not enough for people in some sense to Aknow@ what causes disease, they have to be *persuaded* to change their behavior. In part, the answer is that the associated remedies must have been difficult to put into practice in the crisis conditions obtaining. The outcome suggests a variation of a pattern associated with the medical historian Thomas McKeown. In a series of publications dating back to the 1960s McKeown maintained, controversially, that medical science contributed little to life expectancy before the end of the nineteenth century, meaning that the decline in mortality

from several infectious diseases in the developed world preceded effective medical treatment. In today=s less developed world, health lags rather than leads medical science, with the result that the decline in mortality from specific causes such as gastroenteritis, malaria, and tuberculosis has tended to lag behind technology. Economic and political progress are a precondition for modern health technologies playing their part in improving the health of the masses.

Ireland on the eve of the Famine, with one medical practitioner for every three thousand people, was well endowed with doctors compared to much of the less developed world today, but whether this represented into an advantage in terms of quality is highly dubious. More important, the Irish poor knew or cared little about hygiene. Many, if not most of them, walked barefoot much of the time and were forced by poverty to rely on second-hand clothes. Most of the poor 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 the Famine struck, hunger made them cold and less likely to shed or change their clothes. The decline in energy meant poorer childcare, less effective care for the ill and the elderly, and possibly less fuel and clean water, all of which relied on physical effort.

The gap between such conditions and those implied by the new science was enormous. Cleanliness and hygiene were luxuries that the Irish poor could hardly afford even in normal times. For the very poor, then, more knowledge and understanding would have done little. For the better-off sections of the population, however, the benefits of the new science would have been more tangible. A better understanding of the causes of disease would have mattered more in preventing epidemics and deaths among the somewhat better off, especially in the towns.

VIII: 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 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 Deaths=.

It bears repeating that in past famines, including the Great Irish Famine, most victims were not killed directly by hunger and exposure but by micro-organisms. Neither the victims, nor the authorities, nor medical experts understood this basic fact until the 1880s. Their ignorance of the exact nature of what it was that was killing most victims is a crucial element in determining the demographic impact of past famines. The main reason why modern famines differ from past famines is that today we understand the role that infectious disease plays during nutritional crises. A careful analysis of epidemics during past famines can therefore 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. Moreover, even in the presence of severe food scarcities, the complete collapse of hygiene and personal care can be prevented. In this respect, the timing of the Irish famine was as tragic as its dimensions: had *Phytophthora Infestans* attacked only a few decades later, better understanding of the basic mechanisms of death thanks to the scientific advances following the work of Pasteur and Koch might have saved many thousands of lives.

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