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Andrew Noymer
International Institute for Applied Systems Analysis, Laxenburg, Austria

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International Institute for Applied Systems Analysis • Schlossplatz 1 • A-2361 Laxenburg • Austria
Tel: (+43 2236) 807 • Fax: (+43 2236) 71313 • E-mail: publications@iiasa.ac.at • Web: www.iiasa.ac.at

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Contesting the Cause and Severity of the Black Death: A Review Essay*

ANDREW NOYMER

"Historians have generally paid little attention to epidemics other than the Black Death and the Great Plague of London." So wrote John Duffy (1977), referring to events in the fourteenth century and 1665, respectively. Thankfully this situation has changed somewhat since that assessment was written, but the general lack of historical interest in other epidemics has, over the years, continued to funnel scholarship into plague in general and the Black Death in particular. There is an ample supply of books, monographs, articles, and of course debates on the Black Death and its role in both social and demographic history. Among the latest arrivals is Ole Benedictow's *The Black Death*, which combines a valuable tour d'horizon of previous research with some novel mortality estimates.

The Black Death

The Black Death was an epidemic that killed close to one-third of the population of Europe between 1346 and 1353. The precise specification of the time span, particularly the end date, varies by a year or so, depending on the source. A less severe but still potent follow-on epidemic in 1361, ostensibly of the same disease, is, by convention, separate from the Black Death. A common misconception is that black refers to skin discolorations accompanying the disease. Black is meant in the metaphorical sense of terrible. In fact, the term "Black Death" was not used until the middle of the sixteenth century. Contemporaries called it the "pestilence."

The historical importance of an event that killed such a huge proportion of Europe's population requires little elaboration. Even by contemporary

*Review of Ole J. Benedictow, *The Black Death, 1346–1353: The Complete History* (Woodbridge, UK: The Boydell Press, 2004). xvi + 433 p. $55.00; $37.50 (pbk.).

standards, the Black Death was shocking in the extreme. Certainly, life in the fourteenth century was short from a modern perspective, but even the worst mortality events in the thirteenth and fourteenth centuries, up to 1346, did not compare to the Black Death. The 1290s witnessed numerous wheat failures throughout Europe, caused in the main by unfavorable weather, and the agricultural situation did not improve in the early fourteenth century. Famine mortalities reached 10 percent in some localities. There are even reports by chroniclers of cannibalism, although these are regarded as apocryphal by some historians.

Historians debate whether these stresses represented a true long-run Malthusian crisis. The counter-argument is that medieval agriculture was capable of feeding Europe, meteorological bad luck aside. In any case, the hypothesis that the Black Death itself was an inevitable consequence of population pressure—that the Black Death was endogenous, if you will—is no longer widely accepted. The intercession of some external pathogen is now regarded as a condition without which the Black Death would not have occurred. Just what that pathogen was, and from whence it came, are debated to this day.

Apart from the second plague (1361), the closest thing to a repeat of the Black Death was the Great Plague of 1665, which by some estimates killed 15–20 percent of the population in certain locales. In modern times, the 1918–19 influenza pandemic killed more people than the Black Death because it was truly worldwide and because the twentieth century had much larger populations than the fourteenth. The 1918–19 flu killed perhaps 2.5 percent of the world population: for percentage mortality it doesn't even come close to the Black Death. These comparisons are somewhat arbitrary, inasmuch as the Black Death struck Europe and western Asia, while the flu was global, but it is safe to say that the world has not experienced anything quite like the Black Death since the fourteenth century.

Although the historical importance of the Black Death is clear, its historical impact remains a matter of fierce debate. The story is that the Black Death's demographic wake relieved population pressure and caused the transition out of the late Middle Ages. David Herlihy's posthumous volume, *The Black Death and the Transformation of the West* (1997), advocates this view eloquently and succinctly. The Black Death "broke the Malthusian deadlock that [eleventh- and twelfth-century] medieval growth had created and which might have impeded further growth in different forms" (Herlihy, p. 38). With the labor–land equation turned in their favor, peasant serfs would evolve into the yeoman farmers of the fifteenth century.

Others hold that the medieval European social-demographic system rebounded from the Black Death. It was not the wake of an epidemic, according to this view, but the cultural stirrings of the Renaissance and other factors (themselves not a direct result of the Black Death) that led to the transition from the late medieval period to early modern times. Norman Cantor (2001)

deems the causal relationship between the Italian Renaissance and the Black Death "tenuous." Economic demographers David Bloom and Ajay Mahal (1997) provide a quantitative argument against the Black Death as a pivot. They estimate that post-pestilence changes in real (wheat-price-deflated) wages in England and France do not deviate significantly from the long-term trend. According to Bloom and Mahal, various changes (population, wages, prices) acted in compensatory ways, as best as can be discerned from the scant available evidence. The debate on the impact of the pestilence will continue because the relevant counterfactual—no Black Death—introduces a host of alternate universes, with diverse factors such as Malthusian pressure, crop failures, shifting fortunes in the Hundred Years War, and so on, all coming into play.

Was it plague?

The historiography of the Black Death is chock full of debates, none more heated of late than the question of etiology. Plague has been used as a general term for any great epidemic, but it is also a specific disease caused by the bacterium *Yersinia pestis*, named after the French bacteriologist Alexandre Yersin (previously called *Pasteurella pestis*, after Yersin's employer, the Institut Pasteur). Like many diseases, plague is a zoonosis: it comes to humans from animals. Plague has a natural reservoir among wild rodents, and a vector in fleas. Plague persists to this day. Plague foci, as enzootic regions are called, exist throughout the world, including in Asia, Africa, South America, and the southwestern United States.

Plague ecology is complex, but a thumbnail sketch indicates that when humans become inserted into the rodent–flea–rodent cycle of *Yersinia pestis* transmission, an outbreak occurs. Although plague exists in a wide variety of rodents, including squirrels and marmots, rats are indicted in the Black Death because of their tendency to nest around humans and to stow away on ships. The black rat (*Rattus rattus*) in particular was the culprit. The brown rat (*Rattus norvegicus*), now prevalent throughout much of the world, was by most accounts not present in Europe in medieval times. The rat flea, *Xenopsylla cheopis*, is the disease vector. When the rats die of plague, hungry fleas go looking for blood meals elsewhere, and they find humans. On longer voyages, provided the ambient humidity is not too low, the rat flea can survive for a time after the rats have died, persisting on grain dust. There is, again, a debate about whether the human flea, *Pulex irritans*, also transmitted the plague from person to person during the Black Death.

The epidemiology of plague is inexorably linked to rodent and human ecology. Normally, plague bacteria live in small concentrations in the blood of rodent hosts and in the guts of fleas who feed upon them. It is not known exactly what triggers an epizootic, but it happens when a chain of biological

events is set in train. First, a higher concentration of bacteria accumulates in a rodent. This causes a flea to ingest more bacteria, which multiply in the gut of the flea to such an extent that they subsequently form a solid mass in the flea's stomach. The flea in this state is said to be "blocked." The blockage causes a flea to regurgitate while it feeds, and the regurgitation includes bits of the blockage and therefore has an ultra-high concentration of bacteria. At these high concentrations, rodents succumb to the plague. When the rodent dies, the flea moves on to a new rodent. When an entire rat colony has died, fleas move on to other animals, including farm animals (except horses, whose smell repels even fleas) and humans. Plague epidemics are thus preceded by epizootics.

The most common form of plague in humans is bubonic plague, characterized by buboes, or enlargements of the lymph nodes, usually in the groin, armpits, or neck, depending upon the location of the flea bite. There is one bubo in most cases, arising three to five days after the bite. Today plague is treatable with antibiotics, but if left untreated, plague case-fatality in modern populations is 50–60 percent. In fatal cases, plague bacteria escape the bubo and enter the bloodstream, killing the victim on average eight days after the bite. A second type of clinical classification is pneumonic plague, which occurs when the bacteria spread to the lungs and cause pneumonia. Primary pneumonic plague refers to infections acquired from other cases of pneumonic plague (i.e., person-to-person spread without fleas). Yet another debate about the Black Death surrounds the relative importance of pneumonic versus bubonic plague. A third type of plague, septicemic, occurs when the flea hits a vein and injects plague bacteria directly into the bloodstream. Septicemic plague essentially skips the step with the buboes, and without prompt treatment it is almost always fatal.

Linking historical disease outbreaks to established modern syndromes is a preoccupation of historical epidemiologists. A recurrent exercise involves assigning a disease to the Plague of Athens (430 BCE), to which Thucydides famously attributed a panoply of symptoms. Medieval accounts of symptoms were more often than not vague, and linkage with modern diseases is full of potential pitfalls. The nineteenth-century medical historian Charles Creighton (1891) was dubious about the prevalence estimates of leprosy in medieval Britain—it is not clear whether most inmates in Europe's numerous leprosaria actually had leprosy as defined by modern medicine (i.e., *Mycobacterium leprae* infection). Another example is that the diagnostic distinction between smallpox and measles—two major diseases—was not made by Western medicine until the sixteenth century.

The classic account linking yersinial plague and the Black Death is as follows. The symptoms described by chroniclers agree, *grosso modo*, with the symptoms of modern yersinial plague, particularly the presence and location of boils, interpreted as buboes. The chroniclers were not physicians, however,

and as noted most accounts were vague. Boccaccio's novel *Decameron* also offers a description of symptoms, and such is the state of Black Death nosography that we are dependent upon a work of fiction as much as anything else. The apparent spread of the Black Death along shipping routes is congruent with plague, because the black rat is a good climber and would have gained access to ships in harbor via mooring ropes. But many other diseases tend to spread along trade routes. Gaps in the story include absence of comments about dead rodents in the chronicles, even though there must have been plenty of these if the Black Death were yersinial plague. It is possible that dead rats were not deemed worthy of record. The Black Death also appears sometimes to have traveled faster than one would expect from modern plague epidemiology.

A recent argument in favor of the classic, yersinial account surrounds detection of plague DNA in dental pulp from teeth in a medieval mass grave in France, ostensibly of Black Death victims (Raoult et al. 2000). The modern DNA test (PCR, polymerase chain reaction) is highly sensitive, a fact that has raised objections about the possibility of false positives. While no single piece of evidence is conclusive, it seems to me the DNA evidence deserves a lot of weight. Even though the grave is not dated precisely to the Black Death, it still puts *Yersinia pestis* relatively close temporally to the scene of the crime. Also, without placing blind faith in lab tests, it seems to me that the measurement error of PCR is likely to be far smaller than what might be called "speculative error" when there is a free-for-all among hypotheses competing to displace *Yersinia pestis* in the record books.

The opening salvo in the revisionist, non-yersinial account of the Black Death was Graham Twigg's 1984 monograph, *The Black Death: A Biological Reappraisal*. Twigg suggests anthrax, caused by the spore-forming bacterium *Bacillus anthracis*, as the etiologic agent, while the ensuing debate has also suggested hemorrhagic viral fever—a fourteenth-century ebola—as a possibility. J. F. D. Shrewsbury (1970) argues that a co-epidemic of yersinial plague and louse-borne typhus was responsible for the great mortality of the fourteenth century. The debate is spirited. Susan Scott and Christopher Duncan (2001) list a twenty-item bill of particulars against *Yersinia pestis* (pp. 356–362). One of their contentions is that the dynamics of the Black Death are not plague-like, based on reasoning from mathematical models.

Twigg (1984) makes use of John Brownlee's modeling of plague outbreaks over four centuries. Brownlee was a key figure in the development of epidemiology, but I take his models with a grain of salt. As Paul Fine (1979) discusses in great detail, Brownlee's notions of the shape of epidemic curves were underpinned by the incorrect view that all epidemics decline as a result of diminished virulence. Scott and Duncan note that the Black Death did not behave according to Reed–Frost dynamics, as they contend a plague epidemic ought to. On the other hand, George Christakos et al. (2005), who likewise provide a strident argument against yersinial plague, fit a series of modified

Reed–Frost models to the Black Death. The key lies, evidently, in the modification of the models. These are treacherous waters to enter, and despite my own interest in disease modeling more generally, I find the Black Death modeling exercises to be the least persuasive. Models need data for parameter estimation and for validation. The Black Death data are of especially dubious quality, a fact that permits a wide latitude of possibilities and interpretations when it comes to model fitting.

Different diseases spread differently, and knowing the cause of the Black Death affects many aspects of its investigation. For one thing, estimates of the speed of the epidemic's spread depend in part on the incubation time of the disease in animal reservoirs, in humans, or in both. The arrival of the Black Death in a given locale dates from the onset of symptoms minus the incubation period(s). Without an estimate of the duration of incubation, the dynamics become considerably fuzzier. Nonetheless, a point worth emphasizing is that one may study the Black Death and its social and demographic upheavals without committing to linking the causative pathogen or pathogens with any specific germ or germs as we know them today. Clearly, some infectious disease roiled Europe in the mid-fourteenth century, and it would be a pity if scholarship on the Black Death should become too distracted by debates over the cause. Many historians of the Black Death have, quite reasonably, adopted an agnostic stance vis-à-vis the etiologic agent. Readers interested in the debate should consult the recent assessment by John Theilmann and Frances Cate (2007).

The complete history

This brings us to *The Black Death, 1346–1353*, one of the most demographically oriented of all the recent histories of the pestilence. Ole Benedictow is a historical demographer and professor of history emeritus at the University of Oslo. The subtitle of the book, *The Complete History*, is sure to make Black Death specialists everywhere bridle, but he assures the reader that the book is not intended to be the last word on the subject, but rather "a general synthetic study of the Black Death's epidemiology, territorial spread and mortality" (p. xi).

The book is divided into five parts. Part One, "What was the Black Death?," gives a series of reviews of plague and the Black Death. Part Two, "Spread of the Black Death," is a valuable tour of the existing literature, with chapters devoted to individual countries and regions. This is the strongest section of the book. It distills literature from diverse sources on the geographic spread of the Black Death. Part Three, "Patterns and dynamics of the Black Death," contains a single chapter that builds upon Part Two by considering several conundrums of Black Death epidemiology. Benedictow attributes the sometimes seemingly impossibly rapid spread of the Black Death to "metastat-

ic leaps" of fleas moving on ships. Part Four, "Mortality in the Black Death," gives another region-by-region assessment, this time of death rather than spread. Part Five, "The Black Death: Its impact on history," is another single chapter giving some broader context. Benedictow shares the assessment that the Black Death was a pivot, and it is somewhat curious that he does not cite Herlihy's 1997 book. Social-historical topics, such as the Black Death's role in provoking persecution of Jews, are also discussed.

As noted, *The Black Death* has a demographic orientation, with the caveat that medieval population studies are a data-starved endeavor, and quantitative studies of this period necessarily involve numerous assumptions. To his credit, Benedictow discusses the assumptions he uses, both throughout the book and in specifically methodological sections. Readers with a strictly historical interest will thus likely find chapter 26, "The medieval demographic system," somewhat esoteric, with its discussion of the level 4 Model West life table (life expectancy at 27.5 years for women and 25.26 for men, though Benedictow suggests [p. 256] using the male table for both sexes) and so on. Those with no background in demography will likely not find enough information to appreciate fully the intricacies of the life table. But it is hard to fault Benedictow for keeping the formal demography exposition to a minimum.

The book has some small inconsistencies. For example, on p. 5, Benedictow writes that "the Black Death was the *first* and particularly violent outbreak [of plague]" (emphasis added). He, evidently, does not believe that the so-called Plague of Justinian (541 CE) was caused by plague. Clarification comes four chapters later: "The Black Death was not the first clearly identified great wave of plague epidemics..." (p. 39), and he goes on to discuss the Justinianic outbreak. The former assertion thus apparently refers to the Black Death as the epidemic that ushered in the second plague pandemic, while the latter deals with all of history. The misunderstanding could have been avoided if the various overviews that open the book were better organized.

I did not care for the many military metaphors, with plague having "armies" and "strategy," and making "campaigns," "invasions," "bridgeheads," and so forth, though this is a question of taste. For the most part, Benedictow writes clearly and cogently. The book's regional organization gives it the feel of a stamp collection. Regional specialists will doubtless have their cavils, but the breadth of stamp collections makes such volumes extremely useful as encyclopedia-like tomes. When I want to know what work has been done on the Black Death in a given region of Europe, I certainly will pull Benedictow's book off the shelf.

A more serious problem with the book has to do with the way it presents various perspectives. Benedictow firmly believes that the Black Death was yersinial plague. Some will disagree; so be it. But I think Benedictow has done his readers a disservice by not giving the other side a fairer hearing. Right or wrong, Twigg's 1984 monograph is a serious work, not a conspiracy

theory. Certainly, Black Death experts are currently divided into camps. Twigg deserves a footnote, at least.

Benedictow places the geographic origin of the Black Death in the Golden Horde (one of four successor Khanates of the Mongol Empire, territorially comprising parts of present-day Ukraine, Kazakhstan, and southeastern Russia), dismissing theories of a Far Eastern origin. This is not a settled matter, either. William H. McNeill's influential (and controversial) book *Plagues and Peoples* (1976) places the origins of the Black Death much farther east; he does not pinpoint an epicenter but offers several eastern scenarios. Most studies of the Black Death in the yersinial mold are in concordance that it came from the east. The outbreak at Caffa (or Kaffa, modern-day Feodosiya) in the Crimea indicates that the Black Death was in the Golden Horde by 1346.

The Caffa outbreak has become the stuff of legend. The Genoese established a trading colony at Caffa. A street brawl escalated into a small war, with a Tatar army eventually laying siege to the city. Then the Black Death erupted among the Tatars, devastating the besiegers. Desperate, the Tatars catapulted dead bodies into the citadel, spreading the Black Death to the Genoese, who then beat a retreat to Genoa via Constantinople and Messina, thus spreading the Black Death to Asia Minor, Sicily, and continental Europe. Or so the story goes. Clearly, the Black Death passed through the Golden Horde—but did it start there? The uninitiated reader is not given a sufficiently distinct notion that the Golden Horde theory is not a consensus. Interested readers should also consult a classic debate between John Norris (1977, 1978) and Michael Dols (1978) (cf. also Uli Schamiloglu 1993).

The most original contribution of the book is Part Four, a series of mortality estimates, including, in chapter 33, an extrapolation of these estimates to a novel synthetic estimate for Europe as as whole. Benedictow is suitably alert throughout to "Problems of source criticism, methodology and demography" (the title of chapter 27). I am concerned, though, that he seems to be especially source-critical when the data do not fit his picture of high mortality. Benedictow correctly asserts that wills may be used to date the arrival of the Black Death in a locale, but are less useful in judging mortality, since wills were composed as a fearful reaction to the sudden increase in deaths, even in cases where those writing the wills would ultimately survive. However, he notes changes in intervals between dating and probate (p. 137), which would seem to indicate that, at least in some jurisdictions, probate data are available. This is especially relevant for London, where no contemporary ecclesiastical registers survive (p. 135).

To me, the most striking example of Benedictow eschewing data that do not fit his story is his consideration of Black Death mortality in Mallorca in the Balearic Isles. His treatment provides a compact example of his overall approach. Benedictow reviews the available evidence, which points to 16 percent island-wide mortality (p. 280). He notes that this mortality is low in

comparative perspective. Then he comments, "so far no scholar has come up with any epidemiologically relevant reason(s)" for Mallorca's good fortune (p. 281). He observes that the discrepancy between rural and urban mortality does not fit the typical pattern. Then he summarily sets the 16 percent figure aside: "These Mallorcan data are, therefore, infested with major problems of demography, sociology and source criticism..." (ibid.). That there should be regional differences in mortality strikes me as completely normal for any epidemic. We see this in disease outbreaks down to this day. Sixteen percent mortality may indeed be an exception, but many distributions have thick tails, and I fail to see why exceptions are unexpected—all the more so since Mallorca is an island. The rural–urban difference may be viewed as the result of data problems or as consistent with outlier status, depending on how one looks at it. My fear is that the baby has gone out with the bathwater.

After sorting and sifting the available evidence, Benedictow produces a synthetic estimate for Black Death mortality in Europe as a whole of 60 percent (p. 383). This figure is based on "the remarkable similarity of the levels of mortality in...widespread and diverse regions..." (p. 381). Here, he duly notes that previous estimates are in the 25–33 percent range. If Benedictow is correct, and assuming a European population of circa 80 million, then the Black Death would about equal the 1918–19 flu in absolute die-off. Crisis mortality is intrinsically hard to ascertain—the exact number dead in the twentieth-century event is by no means settled either (cf. Johnson and Mueller 2002). Benedictow's estimates for mortality from the Black Death alone are on par with what previous experts have come up with for the Black Death and the 1361 plague combined, or, in some cases, also including the unrelated famine mortality earlier in the fourteenth century.

Benedictow's mortality estimates may eventually come to be regarded as the standard, in spite of readers' doubts that the remarkably similar die-off across regions is due in part to rejecting data indicating lower figures through source criticism. The estimates are internally consistent with his assessments of plague case-fatality (circa 80 percent, p. 350) and prevalence. If plague lethality is over 50 percent in modern populations, then 80 percent is not implausible for medieval times, considering the nutritional stresses of the fourteenth century. The intervening 600 years is also enough time for *Yersinia pestis* (or another pathogen) to have evolved to lower virulence. The higher the case-fatality, the lower the prevalence need be (*ceteris paribus*) to produce a given total mortality. So Benedictow's estimates do not require that everyone still surviving at the end of the day was a plague survivor in the literal sense (which would seem unrealistic). What is more, one of the ironies of the Middle Ages is that even though most of the population was rural, people lived at very close quarters. Benedictow's estimates are consistent with this, since if one person in an abode got plague, the likelihood of everyone getting it was high. I retain my doubts that the Black Death by itself killed more than 50 percent of Europe's population, but I do not dismiss it entirely.

Benedictow describes plague as a "hydra-headed monster," and I hope I have not understated the Herculean task Benedictow faced in taming this beast for the printed page. This is a demographic history of the Black Death, not a general or social history. Historical context is important, but plague is also a biological and demographic topic, and we need books like this that focus strictly on the historical epidemiology. For one thing, it's hard for a single author to balance everything. The late Norman F. Cantor, a medieval historian at New York University, clearly was not ignorant of the socio-historical milieu of the late Middle Ages. But Cantor's *In the Wake of the Plague: The Black Death and the World It Made* (2001) unfortunately is poorly researched in its treatment of epidemiological aspects. One example is that Cantor gives serious consideration to astronomer Fred Hoyle's preposterous notion that the Black Death came from outer space. Benedictow has stuck mostly to historical demography, giving a round-up of the available quantitative evidence for all of Europe, and by putting all of this in one place he has performed a valuable service.

Scholars looking for an introduction to the Black Death could consult Benedictow's book as a country-by-country field guide to the demographic history. For a general introduction to the Black Death, I think Robert Gottfried's (1983) study remains a better overview, despite its publication date. It gives more emphasis to the Black Death's place in medieval history; for better or for worse it has less demography. As with Benedictow's book, some will dispute the yersinial outlook in Gottfried, but so far at least, I think the book has weathered well. Gottfried's is an environmental history, and the influence of McNeill and Alfred Crosby (1972) is palpable. A more standard treatment by Philip Ziegler (1969) is older but still in-print.

Every library that covers population studies, epidemiology, or medieval history should have a copy of Benedictow's book, and any historical demographer whose interest extends back before early modern history will want a copy as well. The book is too specialized to be used as a textbook, except in advanced graduate seminars on the Black Death, but to be fair it does not seem to have been written with the classroom in mind.

Despite all the existing scholarship on the Black Death, there is still room for more work on the great mortality crises of the fourteenth century. The second plague epidemic, beginning in 1361, is not covered by Benedictow, and although mortality was lower than in the Black Death, it still killed some 10 percent of Europe's remaining population by standard estimates. I have no quarrel with Benedictow sticking to the Black Death proper—the second epidemic merits a volume of its own. There are a number of opportunities for research on the two outbreaks in comparative perspective: the later epidemic appears to have skewed toward mortality at younger ages, which may have been a cohort effect of acquired immunity. If so, this would have implications for Black Death attack rates and hence for case-fatality. Iceland was affected by the second epidemic (though not until the early fifteenth

century) but escaped the Black Death, which may provide a natural experiment of sorts, especially since the second epidemic struck Iceland severely (Tomasson 1977).

In sum, Benedictow's *The Black Death* is a useful compendium of the demography of the most calamitous epidemic in documented history. With its regional organization, it is an excellent go-to guide of the demography of the Black Death. It would have been improved by giving a fuller account of opposing views, and many of Benedictow's estimates, particularly of mortality, will need to be digested more fully by other scholars before they may be regarded as authoritative. The book's value will be greatest for experts who are able to weigh the arguments critically. At the same time, historical demographers and epidemiologists will doubtless continue to look afresh at the available data.

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