The Temporal Dynamics of the Fourteenth-Century Black Death: New Evidence from English Ecclesiastical Records

JAMES W. WOOD,[1,2] REBECCA J. FERRELL,[3] AND SHARON N. DEWITTE-AVIÑA[1]

Abstract Recent research has questioned whether the European Black Death of 1347–1351 could possibly have been caused by the bubonic plague bacillus *Yersinia pestis*, as has been assumed for over a century. Central to the arguments both for and against involvement of *Y. pestis* has been a comparison of the temporal dynamics *observed* in confirmed outbreaks of bubonic plague in early-20th-century India, versus those *reconstructed* for the Black Death from English church records—specifically, from lists of institutions (appointments) to vacated benefices contained in surviving bishops' registers. This comparison is, however, based on a statistical error arising from the fact that most of the bishops' registers give only the dates of institution and not the dates of death. Failure to correct for a distributed (as opposed to constant) lag time from death to institution has made it look as if the Black Death passed slowly through specific localities. This error is compounded by a failure to disaggregate the information from the bishops' registers to a geographical level that is genuinely comparable to the modern data. A sample of 235 deaths from the bishop's register of Coventry and Lichfield, the only English register to list both date of death and date of institution, shows that the Black Death swept through local areas much more rapidly than has previously been thought. This finding is consistent with those of earlier studies showing that the Black Death spread too rapidly *between* locales to have been a zoonosis such as bubonic plague. A further analysis of the determinants of the lag between death and institution, designed to provide a basis for reexamining other bishops' registers that do not provide information on date of death, shows that the distribution of lags could vary significantly by time and space even during a single epidemic outbreak.

Frank Livingstone is justly famous for his work on malaria as a selective force affecting the human hemoglobinopathies. Perhaps less well known is his more general interest in infectious diseases of humans—both as a force of selection and as a key factor in the population ecology of preindustrial communities. As one of

[1]Department of Anthropology, Pennsylvania State University, University Park, PA 16802.
[2]Population Research Institute, Pennsylvania State University, University Park, PA 16802.
[3]Center for Population and Health, Georgetown University, Washington, D.C. 20057.

Human Biology, August 2003, v. 75, no. 4, pp. 427–448.

KEY WORDS: THE BLACK DEATH, EPIDEMIOLOGY, HISTORICAL DEMOGRAPHY, INFECTIOUS DISEASE DYNAMICS, MEDIEVAL CHURCH HISTORY

Frank's students during the 1970s, the senior author of this paper was infected (so to speak) by his enthusiasm for infection. Frank was one of very few people working in anthropology at the time with a deep understanding of mathematical models of epidemics, the biochemistry of the immune system, and pathogen evolution, topics that have become much more fashionable among biological anthropologists in recent years. In light of his early contributions, it is high time that Frank was recognized as one of the pioneers in this now-blossoming area of anthropological research.

One of the (many!) things Frank used to argue about with the senior author was the great European epidemic of 1347–1351 that we now call the Black Death.[4] Could it possibly have been as devastating as was then thought? (Recent research has shown that it was, if anything, even more devastating.) Where did it ultimately come from? (Recent research has still not answered that question with any precision.) Why were Europeans so susceptible? (Recent research has taught us a lot about epidemics in virgin-soil populations—but even in 1975 Frank knew this was the right answer.) Can we see the selective effects of the Black Death inscribed in the human genome? (Research is only now getting around to this question, and to date the CCR5-Δ32 deletion is the only serious candidate.)

One of the things that no one argued about in the 1970s—even Frank—was the cause of the Black Death. At least since the work of Alexandre Yersin (1894), it had been accepted that the Black Death was caused by the bubonic plague bacillus now known as *Yersinia pestis*. The identification of *Y. pestis* as the Black Death pathogen was originally based on a general similarity of symptoms in the medieval and modern diseases. But historical descriptions of symptoms are a poor basis for diagnosis, as recognized long ago by Macfarlane Burnet (1962:296), and epidemiological considerations are often more enlightening. Recently, several scholars have challenged the attribution of the medieval epidemic to *Y. pestis* on epidemiological grounds (Twigg 1984, 1995; Scott and Duncan 2001; Cohn 2002). Other scholars, while accepting the role of *Y. pestis*, have pointed out anomalous epidemiological characteristics of the 14th-century illness, if it was in fact the modern form of yersinial plague (Shrewsbury 1970; Ell 1980; Davis 1986; Karlsson 1996). In the past few years, there have been attempts to recover yersinial DNA from human skeletons believed to represent victims of the Black Death or later outbreaks of the same disease (Drancourt et al. 1998; Raoult et al. 2000; Voong et al. 2001). Thus far, the results have been mixed, and the suggestion has been made that the "positive" results obtained to date may reflect contamination by modern yersinial DNA (Kolman and Tuross 2000). The possibility of contamination is especially worrisome since the "ancient" DNA sequences thus far recovered have been identical to—or have differed by at most a single base-pair from—sequences found in the modern strain of *Y. pestis* used in the same laboratory as a positive control (Drancourt et al. 1998; Raoult et al. 2000).

[4]The term "Black Death" is a post-medieval invention applied retrospectively to the mid-14th-century epidemic. At the time, the epidemic was usually called by some variant of "The Great Pestilence" or "The Great Mortality" (see Wood et al. n.d.).

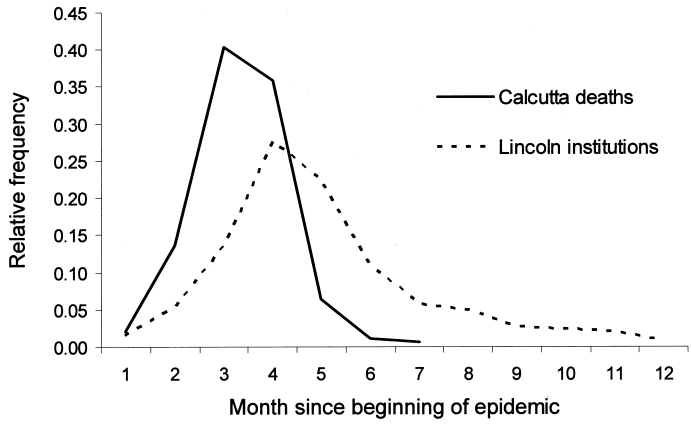

Figure 1. The temporal distribution of deaths observed during the 1903 outbreak of bubonic plague in Calcutta (*solid line*) compared to the distribution of institutions to benefices vacated by death as recorded in the bishop's register of the diocese of Lincoln from April 1349 to March 1350, the period of the Black Death (*broken line*). (Data from Simpson 1905:149; Thompson 1911.)

Thus, there is still a need for epidemiological evidence on the Black Death. One piece of epidemiological evidence that has frequently been cited, both for and against *Y. pestis* (Brownlee 1918; Russell 1948; Shrewsbury 1970; Twigg 1984; Scott and Duncan 2001), involves a comparison of the time-course of the epidemic in medieval England to that observed in either Bombay or Calcutta during the 1903 Indian epidemic of yersinial plague (Figure 1). Those who like *Y. pestis* as the Black Death pathogen think that the two distributions in Figure 1 are similar (they are not: the medieval distribution is significantly more platykurtic and right-skew). The anti–*Y. pestis* forces, in contrast, emphasize the differences between the two distributions. Unfortunately, this whole comparison, based as it is on observed deaths in the Indian case and on episcopal records of institutions to vacated benefices in the medieval case, can be challenged on both statistical and substantive grounds. New evidence from 14th-century English institutions to vacated benefices, presented here, shows that the time-course of the medieval epidemic has been seriously misrepresented by an uncritical use of the episcopal records. The episcopal records are indeed one of very few sources to tell us something about the local dynamics of the Black Death, but they must be used more cautiously than they have been in the past.

Materials and Methods

Institutions to Vacated Benefices. Since medieval records are nothing like the data sources that modern epidemiologists and demographers normally work with, it is important to note their idiosyncrasies at the outset. The data used in this paper (and in many of the earlier analyses cited above) are taken from prospective

bishops' registers listing various kinds of diocesan business, including the appointment of new clergy to local benefices vacated by the death, retirement, or movement of the previous incumbents. Although some bishops' registers survive for continental Europe (Gyug 1983; Cohn 2002), most previous statistical work has been done on a subset of the surviving institution lists from mid-14th-century England (Thompson 1911, 1914; Lunn 1930; Shrewsbury 1970; Davies 1989; Aberth 1995; Dohar 1995; Scott and Duncan 2001).[5]

The process of instituting a new beneficed priest involved several stages.[6] When the rector or vicar of a parish church died, retired, or moved to another parish, the first step was to notify the patron of the benefice, who then had responsibility for finding a suitable replacement. In all likelihood he would already have had someone in mind; he would then ask the archdeacon (who might not be immediately to hand) to carry out an enquiry as to the legality of the vacancy and the suitability of the candidate. If the candidate were indeed suitable, then the ceremonies of admission to the vacancy and of institution into the benefice would take place. Finally, the archdeacon would be given mandate to induct the new holder into the living, promising spiritual obedience to the bishop and, in the case of a vicar, personal residence within the benefice.

Clearly, this process could take some time. The speed of replacement would depend on the efficiency and goodwill of everyone involved, though no doubt the would-be incumbent would be keen to expedite matters. During the period of the Black Death, it appears that some bishops retired to their rural manors, presumably to avoid the disease; this is likely to have slowed down the process of institution by at least a few days (R.A. Davies, personal communication). Geography also played a part. In large dioceses such as Lincoln, Coventry and Lichfield, and York, it might take several days for information or personnel to travel between the local parish and the bishop's residence; in small dioceses such as London, Rochester, Ely, and Carlisle, the replacement process was presumably speedier.

At the time of institution, the vacancy and date of replacement were recorded in the bishop's official register. The more detailed registers also provide the reason for the vacancy, so that deaths of beneficed priests can be distinguished from retirements and movements.[7] By tallying such institutions, we can form an estimate of how many of the clergy died during the period of the Black Death. We can also plot the dates of institutions, which *may* tell us something about the timing of the deaths that led to them. But, as we argue below, a naïve treatment of the relationship between the timing of death and the timing of the associated institu-

[5]Registers from the Black Death period of 1348–1350 have survived for twelve of the seventeen dioceses of medieval England: Canterbury, York, Lincoln, Bath and Wells, Winchester, Rochester, Exeter, Ely, Norwich, Hereford, Coventry and Lichfield, and Worcester (Smith 1981).

[6]For this section we are grateful for the help of R.A. Davies. Aberth (1995) also provides a useful account of the institution process.

[7]Registers for four dioceses (Bath and Wells, Exeter, Winchester, and Norwich) do not distinguish clerical deaths from movements or resignations—at least not during the time of the epidemic (Smith 1981). But the registers that do distinguish deaths from other vacancies show that a large majority (more than 80%) of vacancies during the epidemic period were in fact attributable to death (Thompson 1911, 1914; Aberth 1995).

tion has been a source of serious error in previous statistical analyses of the bishops' registers.

Since cause of death is not recorded in any of the registers, deaths from the Black Death cannot be separated from deaths owing to other causes. Nonetheless, the enormous surplus mortality during the epidemic relative to normal background mortality makes it clear that the great majority of deaths during that period were indeed caused by the Black Death. Despite the limitations of the bishops' registers, several authors have suggested that they provide one of the most reliable sources of data on Black Death mortality (Coulton 1929; Hatcher 1977; Aberth 2001), at least for one segment of society.

Parish priests are not, of course, a random sample of the population at large. They are not, most obviously, representative of the general population in terms of age and sex, both of which might be expected to influence the risk of death. In addition, Hatcher (1977:23) has argued that "priests were on average better fed, better housed and better educated" than most people, all of which would presumably lower their risk—although the archaeological evidence on the parsonage at the abandoned medieval village of Wharram Percy in Yorkshire (Beresford and Hurst 1990) does not suggest a standard of living much above that of the general run of the peasantry. A counter-argument would have it that, if they actually performed their duties to the dying, priests might be exposed to the Black Death pathogen more often than would the average member of the community (Hatcher 1977:23). Either way, the results derived from the bishops' registers should not be projected uncritically onto the rest of the English population, especially the peasantry who made up perhaps 80% to 90% of the total.

Another problem, first highlighted by Thompson (1914), is that priests whose deaths are recorded may not have been residing in their parishes at the time of death. Rectors often took extended leaves, appointing vicars to cover their duties in their absence (hence the word *vicarious*). If they died while absent, their deaths could still be recorded under the parish of their benefice, thus inflating the apparent mortality there. In principle, nonresident clergy had to obtain a license from the bishop for the duration of their absence, and this license was supposed to be noted in the register. Thus, it is possible in some but not all cases to correct this bias. A similar problem has to do with plural benefices: a given priest might have benefices in two or more parishes simultaneously, and his death would open vacancies in several locations at once. A failure to correct for the resulting multiple counts would lead to an overenumeration of deaths.

By far the worst problem, at least from the perspective of infectious disease dynamics, is that the bishops' registers almost without exception list the vacancy by the date of the associated institution, not the date of the death that opened the vacancy. Following Thompson (1911) it has been assumed that there was an average lag of about a month from death to replacement. It has seemed reasonable, therefore, to subtract one month from the date of replacement to obtain the putative date of death (Figure 2). This "correction," however, would work only if the time from death to replacement were absolutely invariant. If it were not, then the

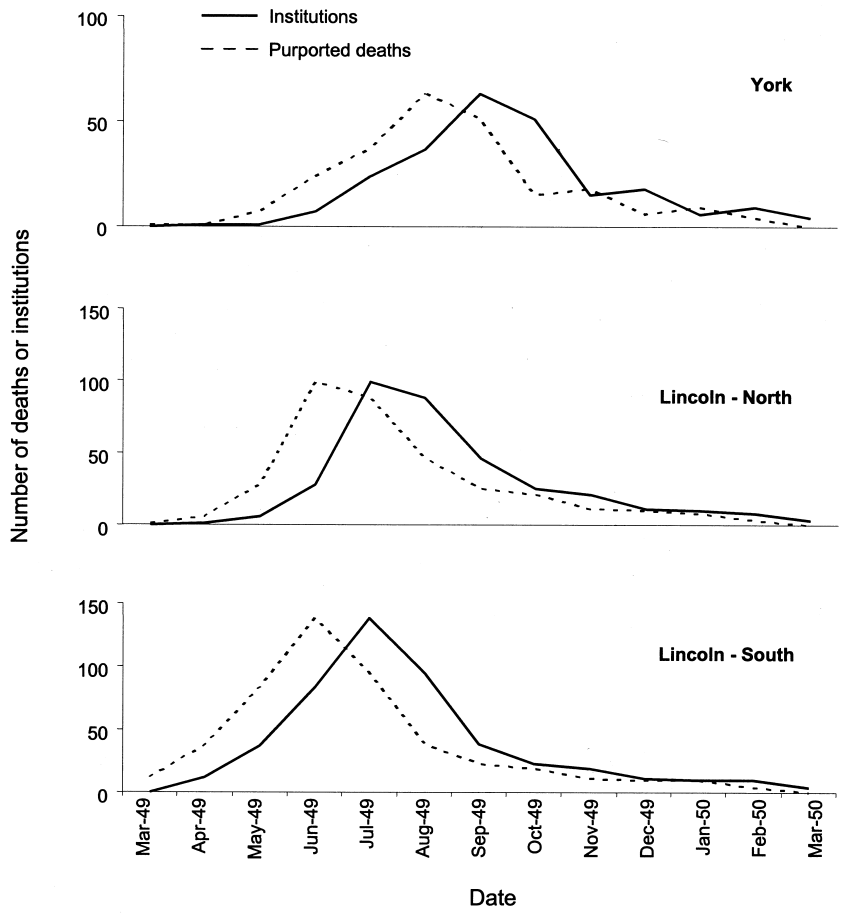

Figure 2. "Correcting" for the lag time between death and institution by assuming a constant one-month lag (redrawn from Twigg 1984:67). Note that the "corrected" distribution is identical in shape to the original one, differing only by location.

reconstructed distribution of deaths would confound variation in the timing of death with variation in the lag to replacement and therefore make the epidemic period look broader than it really was. As we discuss below, this problem must be addressed before unbiased estimates of the time-course of the epidemic can be derived from the bishops' registers.

The bishop's register from the English diocese of Coventry and Lichfield is the only one that provides actual dates of death for the Black Death period (Davies 1989). It is thus the only original source that can be used to validate the many analyses performed on timing data from *other* registers. In addition, plural benefices were uncommon in Coventry and Lichfield during the mid-13th century, and they are consistently marked in the register as being held *in commendam* (R.A. Davies, personal communication); they can thus be identified and their biasing effects removed. Here we present, for the first time, a detailed analysis of the dates of death during the Black Death and of the lag time between death and institution as reconstructed from this unique data source.

The Diocesan Records of Coventry and Lichfield. The large medieval diocese of Coventry and Lichfield, covering the counties of Chester, Derby, Stafford, and portions of Lancashire, Shropshire, and Warwickshire, was divided into the five archdeaconries of Chester, Coventry, Derby, Salop, and Stafford (Figure 3). The diocese was hit by the Black Death at a fairly early stage of the British epidemic (Shrewsbury 1970:77–79). By the late summer of 1348, the illness had entered England somewhere in the southwest, probably via the substantial port town of Melcombe Regis in Dorset (now part of Weymouth) and perhaps independently via Bristol (Horrox 1994:10). It spread rapidly to Gloucester, which acted as a center for secondary transmission into the west midlands and Welsh marches (Thompson 1889:98–100). The disease is known to have been active in the cathedral city of Coventry by April 1349 (Gasquet 1908:146) and probably spread in short order to the rest of the diocese.

A fairly complete set of late medieval bishops' registers survive for Coventry and Lichfield (starting in 1296) and are kept in the Lichfield Joint Record Office, Lichfield Public Library (Smith 1981:52 ff.). The register of Roger de Northborough, bishop from 1322 to 1358, provides excellent coverage for the Black Death period. This register has been edited and translated by Dr. R.A. Davies, who has graciously allowed us to see his unpublished material. We have examined a time-series of 235 death-related institutions running from March 1347 to September 1350. Special attention is paid here to a subset of 214 "epidemic" deaths dating from 30 March 1349 to 24 February 1350, when the Black Death was active in the diocese. There is a slight degree of arbitrariness in this periodization, but it appears to cover the entire epidemic period.

Statistical Issues and Methods. The principal methodological question addressed here is: what is the relationship between the temporal distribution of institutions (of the sort shown in Figure 1) and that of the deaths that vacated the

Figure 3. Map of the medieval diocese of Coventry and Lichfield showing its archdeaconries. (Redrawn from Beresford 1883, frontispiece.) The cross-and-orb indicates the episcopal city of Coventry. *Inset:* The medieval dioceses of England (with Coventry and Lichfield highlighted).

benefices in the first place? This question is important because, on the continent as well as in England apart from Coventry and Lichfield, we only ever observe the distribution of institutions, but what we *want* to know about is the distribution of deaths. As already noted, a simple rigid translation of the distribution of institutions backwards by a month or so would be legitimate only if there were an invariant lag between death and institution. But there is nothing in the bishops' registers to suggest any attempt to keep to such a rigid schedule of replacement. The impression, rather, is that vacancies were filled as quickly as possible given the practical constraints of time and distance—and (during the Black Death itself) the difficulty of keeping up with the sheer number of vacancies. The only effective constraint on the pace of institutions was that all vacancies were supposed to be filled within six months (R.A. Davies, personal communication). Thus, the actual time from death to replacement might be expected to vary substantially between 0 and 6 months, complicating the temporal relationship between deaths and institutions.

Figure 4 shows a hypothetical example in which the lag time between death and institution is assumed to have been an exponentially distributed random variable. This model implies that there was a constant *rate* of institution in each time interval following a death, not a constant *lag*.[8] We are not suggesting that this is a realistic model, but only that it is the simplest one allowing for a variable lag time. The distribution of institutions in Figure 4 has been rigged to look more or less like the distribution from Lincoln Diocese shown in Figure 1. The distribution of associated *deaths* is quite different: it is much more peaked and symmetrical, lacking the long upper tail of the institutions. The reason for this difference is that the distribution of institutions mixes variation in timing of death with variation in the lags to replacement. The institutions display a long upper tail purely because an exponential distribution of lags is reverse-J-shaped and itself has a long upper tail. Because the variation in lags "smears" the distribution of institutions over a wider time range—and because the number of events (deaths or institutions) is equal in the two distributions—the distribution of institutions is necessarily less concentrated around its mode and is therefore more platykurtic. If we were to ignore the variation in lags (as all previous analyses have done) and simply translate the institutions backwards along the *x*-axis by a month, we would end up concluding that the period of epidemic mortality was much longer than it really was.

It is not clear that there is any simple way to model the relationship between deaths and institutions that would be equally applicable to all dioceses. An exponential model implies that naming a replacement was rate-invariant, at least within a given diocese. But this seems unlikely. In view of the poor conditions of transport and communication in the Middle Ages, it might be expected that the bishop got around to replacing vacancies in parishes near his episcopal manor

[8]A constant lag means that all waiting-times from death to institution are exactly the same length. A constant rate, in contrast, allows for some random variation in lengths. In addition, the number of institutions expected per unit time under the constant-rate model will decline with the time since death, following a negative exponential curve, as the vacancies that still remain to be filled dwindle away.

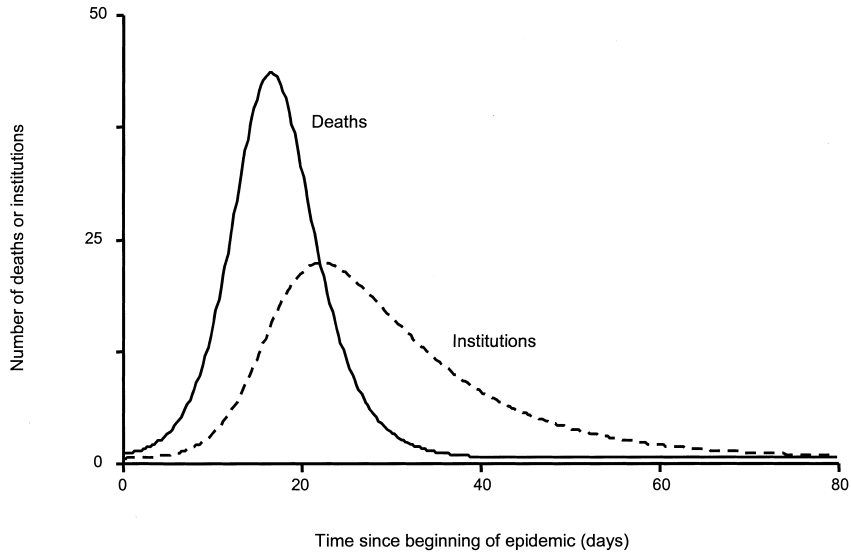

Figure 4. Hypothetical distributions of deaths (solid line) and institutions (broken line) given exponentially distributed time lags between the two events. Lags are assumed to be independent of death dates, in which case the distribution of institutions can be found as the convolution[$f_i(t)= \int_0^t f_d\ (t–x)f_l(x)dx$, where $f_i(.)$, $f_d(.)$, and $f_l(.)$ are the probability density functions for institutions, deaths, and lags respectively.

rather earlier than in more distant areas. Was the lag to institution at least independent of the time of death (if not the remoteness of the parish)? If it were, it would make the mathematics of the problem much easier, for the distribution of institutions would be a simple convolution of death dates and lag times, as is the case in Figure 4. Or did the bishop fall further and further behind as the number of vacancies accumulated during the course of the epidemic? If so, the time to institution could not have been independent of the date of death, and something more complicated than a convolution of two density functions would be needed to model the situation. In what follows, we explore these questions by applying survival analysis to the Coventry and Lichfield data.

A second statistical problem, having to do with the spatial scale at which the data are aggregated, dogs the comparison of the distribution of medieval institutions (or even deaths) with more modern data on plague deaths in cities such as Calcutta, Bombay, or Hong Kong. Despite their enormous populations, all these modern cities are concentrated on fairly small areas—small, that is, by comparison to most medieval dioceses. Once an infectious disease had been introduced into one of these cities, it would not have to travel far to infect a large number of residents, even ignoring the greater density of social contacts and potential paths of transmission in modern urban areas as opposed to the sparsely populated, overwhelmingly rural medieval world. In interpreting the distribution of institu-

tions in a geographically extensive diocese such as Coventry and Lichfield, we need to allow for the fact that settlements were widely scattered and were likely to have experienced the epidemic at different times. While the Black Death was raging in one parish, it might still be making its way to another, and might already have burned itself out in a third. If we aggregate the death dates for all these parishes over the diocese as a whole, we force several distinct, only partially overlapping epidemic curves into a single distribution, which must necessarily be wider than any of the more local distributions. Again, this problem makes the medieval epidemic period look broader than it really was. The solution, of course, is to disaggregate the data by regions much smaller than the entire diocese. But it is not easy to decide just how *local* such regions should be. The ideal would be to examine the distribution of deaths at the level of a single parish, but this is impossible because the samples are too small (only one, two, or, much less frequently, three deaths are likely to be recorded among priests of a single parish during the course of the Black Death). We need to compromise between the need to disaggregate the data and the need to retain samples large enough to be informative. For the Coventry and Lichfield data, we have opted to disaggregate deaths to the level of the archdeaconry, a unit of ecclesiastical administration that is both reasonably localized and large enough to yield usable samples (the archdeaconries of Coventry and Lichfield range in size from 75 to 106 parishes). Whether this scale is appropriate for other dioceses remains to be seen.

Results

The Distribution of Deaths versus the Distribution of Institutions. Figure 5 shows the empirical distribution of deaths and death-related institutions over a three-and-a-half-year period that straddles the time of the Black Death in Coventry and Lichfield. While the difference in the two distributions is not as extreme as in the hypothetical case shown in Figure 4, deaths still depart from institutions in the expected direction. Deaths are more peaked, less variable in their timing, and less right-skew than institutions. The variance in death dates during the epidemic period itself is less than half that of the corresponding institution dates (2340.53 versus 5118.95). Indeed, the two distributions seem to conform to entirely different families of probability density functions.[9] As expected, then, both the shape and the width of the two distributions differ. The simple trick of subtracting a month from the date of each institution would, in this case, provide a poor representation of the time-course of mortality during the Black Death.

Determinants of the Lag between Death and Institution. If the lag between death and institution was not constant, as it clearly was not, what determined its length? Figure 6 shows the empirical distribution of time lags from death to insti-

[9]Institutions are well fit by a lognormal distribution ($-2 \times$ log likelihood ratio = 5.58, $df = 3$, $p = 0.134$), whereas deaths are not ($-2 \times$ log likelihood ratio = 18.36, $df = 8$, $p = 0.019$).

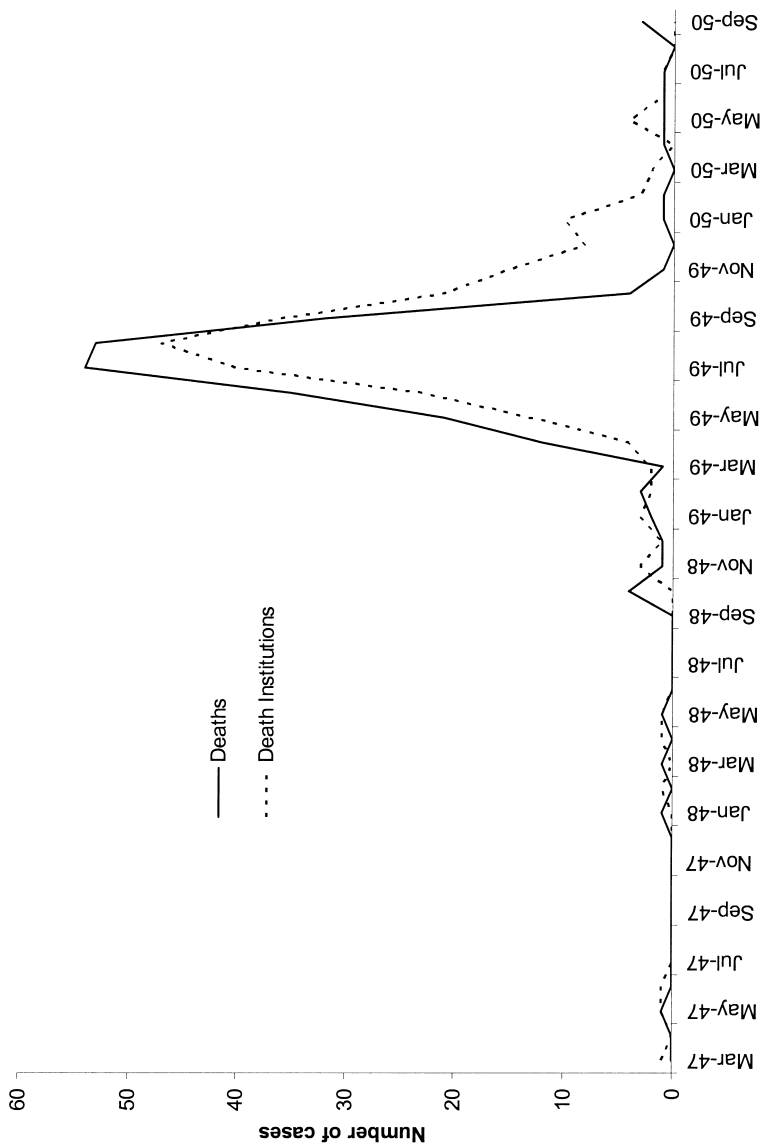

Figure 5. Empirical distribution of deaths and death-related institutions, diocese of Coventry and Lichfield, March 1347–September 1350.

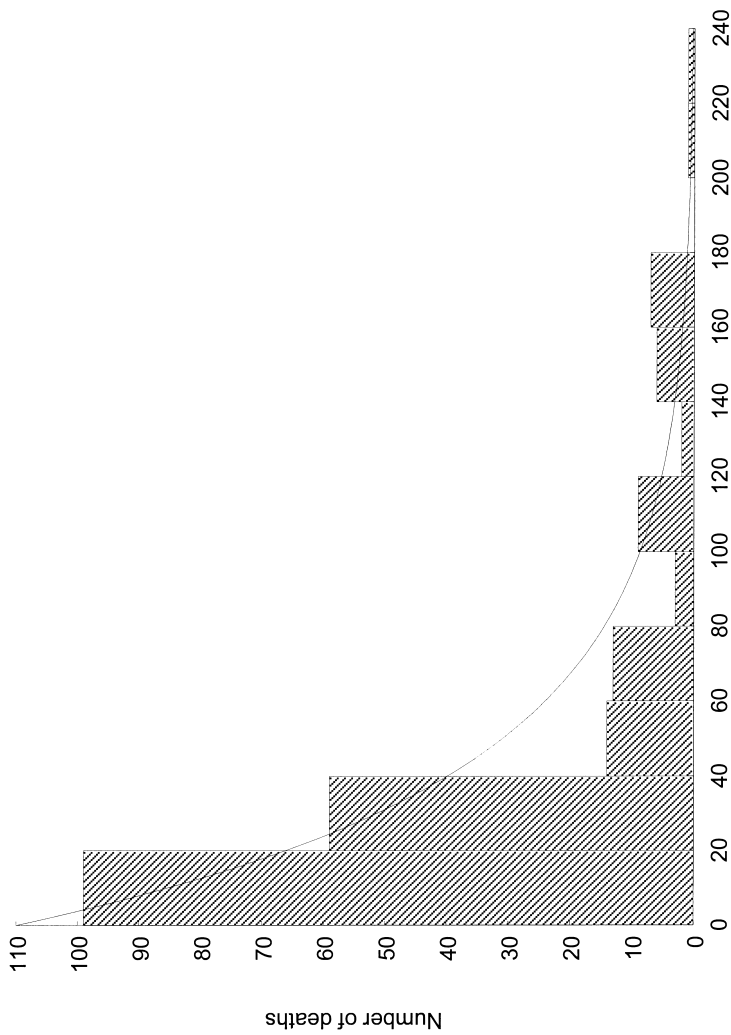

Figure 6. An exponential distribution fit to the time lags between death and institution, diocese of Coventry and Lichfield (epidemic deaths only).

tution for the 214 epidemic deaths in Coventry and Lichfield (mean lag = 39.4 ± 3.0 days, median = 22 days). As would be expected for an exponential distribution, the observed distribution is reverse-J-shaped, with a mode at zero and a long upper tail. If the observed pattern of variability could be shown to conform more exactly to what is expected from an exponential random variable, we could conclude that vacancies were filled at a constant *rate* per unit time. In actual fact, an exponential distribution can be rejected with some confidence for the Coventry and Lichfield data ($-2 \times$ log likelihood ratio = 25.62, degrees of freedom (*df*) = 5, $p < 0.001$). In particular, there are too many extreme lags, long and short, and too few of middling length in the bishop's register (Figure 6), suggesting that the lags (and the replacement rates that generated them) were heterogeneous for some reason. The heterogeneity apparently involved both time and space. With respect to time, a proportional-hazards regression of time to institution on date of death produced a significant negative coefficient ($\hat{\beta} = -0.004$, $-2 \times$ log likelihood ratio = 7.74, $df = 1$, $p < 0.01$), suggesting that, as the epidemic progressed, the bishop fell further and further behind in his efforts to replace dead priests. Some of the resulting delays, up to five months in some cases (and exceeding the statutory limit of six months in two cases), probably reflect the sheer volume of work for an administrative machine that normally had to deal with a small number of institutions each year; but another possible problem was that the pool of suitable replacements was rapidly drying up as the epidemic decimated its ranks (R.A. Davies, personal communication).[10] Whatever the reason, this deceleration of the institution process means that a simple convolution of deaths and waiting times cannot be used to model the distribution of institutions since death dates and lags were not independent.

When the waiting times were stratified by archdeaconry—that is, spatially—a borderline significant effect was found (log-rank test statistic = 7.575, $df = 4$, $p = 0.11$). In particular, the archdeaconry of Salop appears to have waited longer than other archdeaconries to have its dead priests replaced (Table 1, Figure 7). It is unclear why this should be since Salop was not especially inaccessible to the bishop. Perhaps the most plausible explanation is that Salop suffered from an unusually inefficient ecclesiastical administration, although there is no direct evidence to suggest this (R.A. Davies, personal communication). Whatever the reason, the important point is that there was variation in the process of replacement even within a single diocese. This finding should warn us that we cannot use the aggregate results for Coventry and Lichfield uncritically to correct the distributions of institutions from other dioceses.

[10]It is unclear how important this second factor was. During the decades leading up to the Black Death, an average of more than 100 priests were ordained each year, at a time when fewer than half that number of benefices became vacant (R.A. Davies, personal communication). There must, as a consequence, have been a backlog of priests biding their time as parish or chantry chaplains, and eagerly awaiting their opportunity to gain a benefice.

Table 1. Average Lag from Death to Institution by Archdeaconry (Epidemic Deaths Only), March 1349–February 1350, Diocese of Coventry and Lichfield

Archdeaconry	*Number of Benefices*[a]	*Number of Deaths*[b]	*Median (Days)*	*Mean (Days)*	*Standard Error of Mean*
Salop	41	26	31.0	61.5	11.00
Chester	90	33	22.0	39.6	8.07
Coventry	94	50	20.5	30.7	4.86
Stafford	96	37	17.0	33.4	7.08
Derby	103	68	23.0	41.4	5.37
Total	424	214	22.0	39.4	3.04

a. Based on a comprehensive search of the 14th-century registers by R.A. Davies. These counts are generally somewhat higher than those obtained from the *Taxatio Ecclesiastica Angliæ et Walliæ* (House of Commons, London, 1802), the usual source of information on the number of medieval English benefices.

b. More than one death per benefice may have occurred.

The Problem of Spatial Aggregation. The possible presence of heterogeneity in the distributions of lag-times to institution across archdeaconries highlights the need to disaggregate the data to a geographical scale smaller than the diocese as a whole if we are to get a correct picture of the epidemic. When we classify the data on death dates by archdeaconry (Figure 8), it is clear that the Black Death did not affect all portions of the diocese simultaneously. Coventry in particular was hit early—as might have been predicted given the economic ties of the town of Coventry to other market centers in the comparatively densely populated English midlands. The curves for the various archdeaconries appear to be staggered in an order that corresponds roughly to their geographical position relative to Coventry (see Figure 3). More importantly from the present perspective, it appears that the duration of the outbreak within each archdeaconry was considerably shorter than in the diocese as a whole. It took about eight months for the Black Death to sweep through all of Coventry and Lichfield, but only some 4 to 6 months to burn out in more localized subregions. And it should be borne in mind that archdeaconries themselves are not single settlements that are truly comparable to, say, Calcutta, but instead range over dozens of scattered, semi-isolated parishes. It seems probable, therefore, that the course of the epidemic in individual communities must have been very swift indeed.

Discussion

The surviving bishops' registers will remain an invaluable source of information on the timing of the Black Death, but they will need to be used rather more circumspectly than they have in the past. Institutions are not deaths. Institution dates are, of course, partly determined by the corresponding death dates, but they

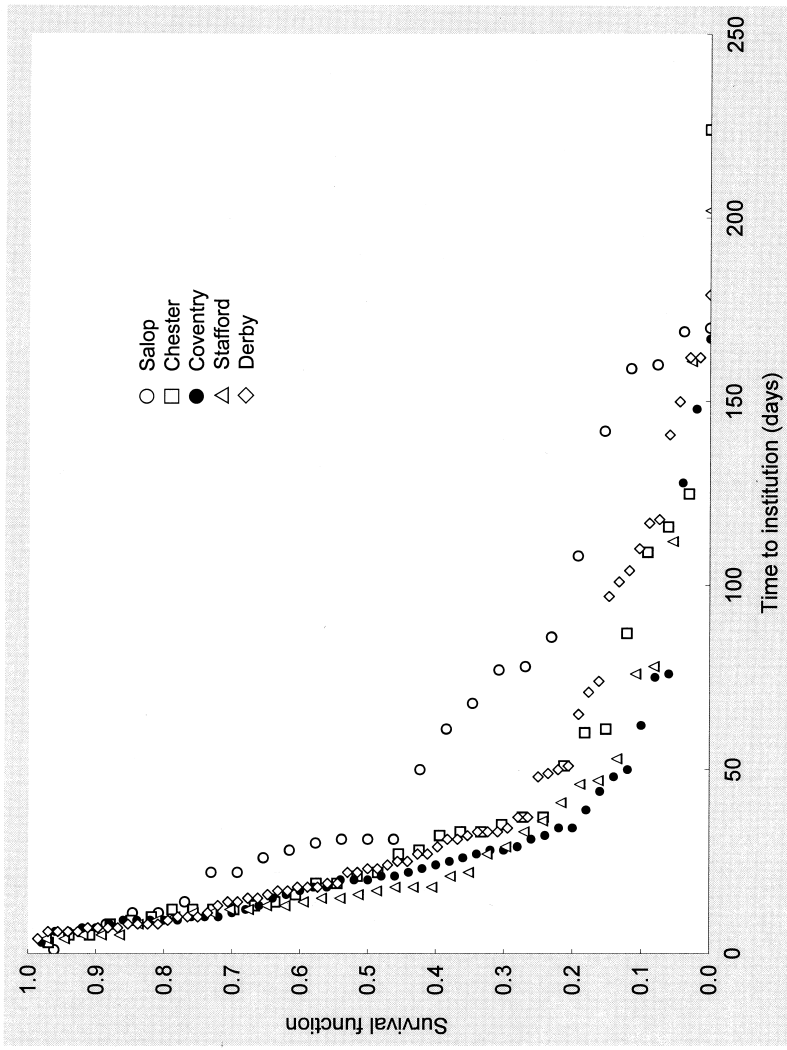

Figure 7. Kaplan-Meier (product-limit) estimates of the survival function for the waiting time from death to institution classified by archdeaconry, diocese of Coventry and Lichfield (epidemic deaths only).

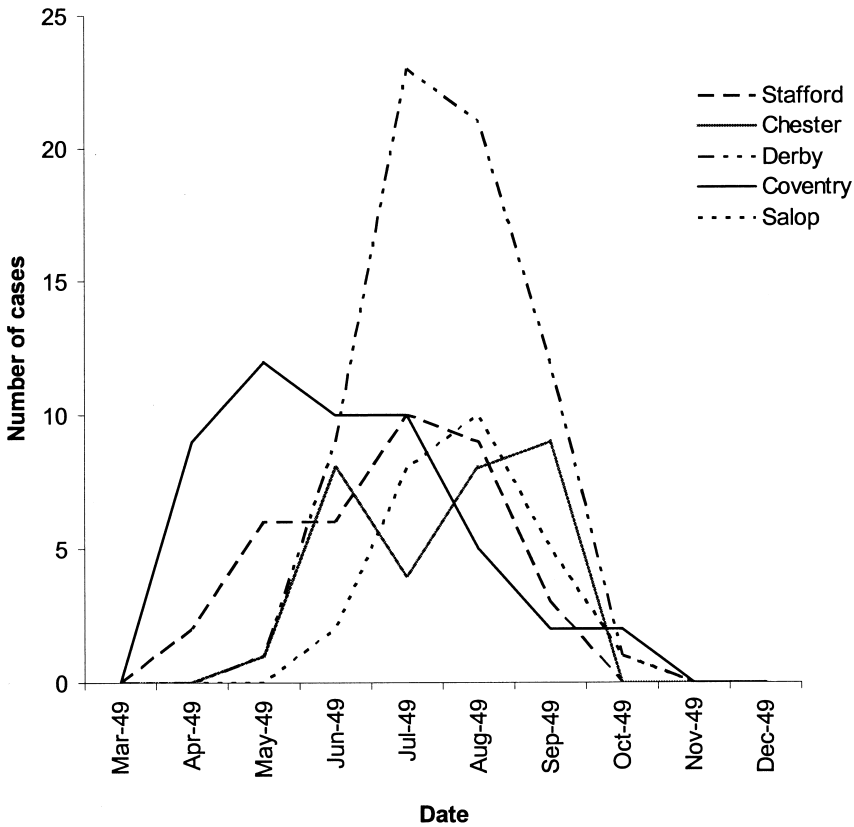

Figure 8. Distribution of death dates classified by archdeaconry, diocese of Coventry and Lichfield (epidemic deaths only).

involve additional processes that have not been examined in detail before. It is likely that the sort of temporal and spatial variation in waiting times to institution detected in Coventry and Lichfield existed elsewhere, although the details of the variation undoubtedly depended on local conditions in each diocese. It will require considerably more work before the institution process itself can be modeled in a way that can be usefully generalized.

Perhaps the most important substantive conclusion to be drawn from the analyses presented here is that the Black Death passed through local areas very rapidly, lasting only about 4 to 6 months instead of the 8 to 11 months or more inferred from previous analyses of the bishops' registers (e.g., Shrewsbury 1970:54–125). Interestingly, this finding makes it appear that the local dynamics of the Black Death were rather *more* similar to those observed in modern outbreaks of bubonic plague than believed by even the most ardent supporters of

Yersinia pestis as the Black Death pathogen. For example, if we studiously ignored all the distributions in Figure 8 *except* for the one from Stafford (which, of course, would be cheating), we might be tempted to claim that the temporal dynamics of the epidemic in the 14th century were virtually identical to those observed in 20th-century Calcutta (compare Figure 1). Does this mean that *Y. pestis* caused the Black Death after all? Any good student of Frank Livingstone's would say "no" just to be contrary. But we believe there are at least three other reasons to question the involvement of *Y. pestis*.

First, to repeat a point already made, the archdeaconries plotted in Figure 8 are not single settlements but are themselves aggregated at a fairly high geographical level. The pace of the epidemic at a genuinely local level such as the parish would almost certainly have been even more rapid, making the apparent similarity to yersinial plague in Calcutta less compelling than it might otherwise seem.

Second, the dynamics shown in Figure 8 are consistent with a wide variety of infectious agents with different combinations of transmission rates, latency periods, infective periods, and case-fatality ratios (compare, for example, the patterns based on the Reed-Frost epidemic model generated by Scott and Duncan 2001:32–35). The temporal patterns provide one important piece of the puzzle, but by themselves do not point to a single, specific disease, whether yersinial plague or anything else.

Third, the Black Death exhibited a pattern of *geographical diffusion* very different from that of modern yersinial plague. Simply stated, not only did the Black Death sweep through given localities very rapidly, it also moved from place to place with extraordinary speed (as hinted in Figure 8)—and indeed it swept across all of Europe in less than four years (Carpentier 1962). This point has been made by previous authors, most notably Twigg (1984:54–58), but it needs to be reemphasized. A pattern of rapid geographical spread is consistent with the swift movement of the Black Death through local communities suggested by the Coventry and Lichfield data—and it is profoundly inconsistent with what happened in southern China during the 18th and 19th centuries, when it took bubonic plague more than a hundred years to move from Yunnan Province to Hong Kong, a distance roughly the same as from one end of Europe to the other (Benedict 1996:30–70). The subsequent worldwide pandemic of bubonic plague in the early 20th century could not have happened without steamships and railways.

The main reason that yersinial plague moves so slowly is that it is primarily a zoonosis, and it must first be established in a local rodent population before it can spread to humans (Figure 9). And most wild rodent species are philopatric and thus unlikely to move long distances on their own, limiting their capacity to act as efficient transmitters of disease across localities (Twigg 1984:75–112). In view of its rapid spread between neighboring human populations, it is almost certain that the Black Death was not a rodent-based zoonosis such as bubonic plague. Could it then have been the *pneumonic* form of yersinial plague with its potential for direct person-to-person transmission? Many researchers have

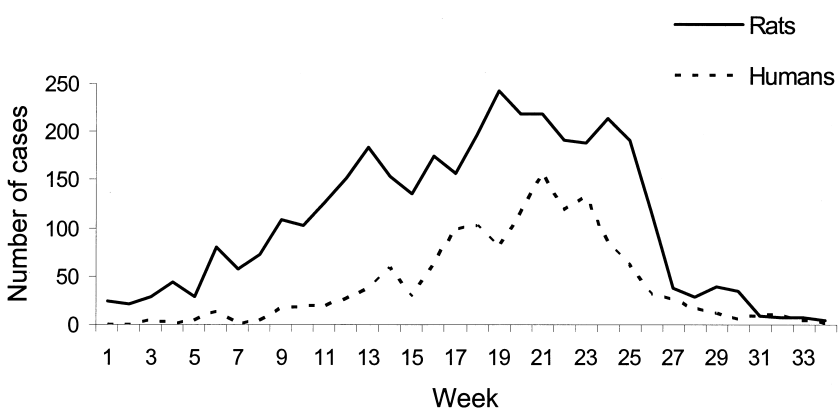

Figure 9. The distribution of cases of yersinial plague detected in live-trapped rats (solid line) and diagnosed in humans (broken line), Hong Kong, January 1–August 31, 1903. (From Simpson 1905:102.)

thought so (e.g., Morris 1971; Ell 1980; Smith 1989; Horrox 1994:4–5). But this belief appears to be founded on two widespread misconceptions about pneumonic plague: first, that pneumonic plague is a fairly common outcome of infection by *Y. pestis* and, second, that it is highly communicable. It is often claimed that the pneumonic form makes up 20% or more of human plague cases and may sometimes reach far higher frequencies. In fact, as clinical observations from the early-20th-century pandemic show, pneumonic plague cases rarely make up more than 5% of all human cases, and the more usual figures fall between 0% and 3% (Wu Lien-teh 1922; Petrie and Todd 1923; Seal 1969). The second point is even more telling. *Y. pestis* seems to be ill adapted to transmission by respiratory aerosols (Bannerman 1906), perhaps because loss-of-function mutations in two genes (*yadA* and *inv*) limit its ability to adhere to and cross the alveolar epithelium of the lungs (Simonet et al. 1996; Perry and Fetherston 1997). Certainly direct person-to-person transmission by aerosols can occur, but there is no evidence that it is anything but rare. Clusters of pneumonic plague cases always start with an outbreak of bubonic plague, and the pneumonic form is almost never transmitted more than two or three steps away from the initial pneumonic case (Wu Lien-teh 1922; Seal 1969; Twigg 1984:147–170). As shown by several classic epidemiological studies of pneumonic plague in Manchuria, Mongolia, and India (Brownlee 1918; Wu Lien-teh 1922, 1926; Seal 1969), the resulting geographical pattern is one of small, scattered, highly localized, and self-limiting outbreaks, not of a widespread, fast-moving epidemic involving pneumonic transmission alone.

Pure pneumonic plague appears to be an example of an infectious disease whose basic reproductive number is too low to spark widespread outbreaks. The basic reproductive number, R_0, is the expected number of cases of the disease transmitted by a single infective individual in a population that is otherwise made

up exclusively of susceptibles. For pure pneumonic plague spread exclusively by person-to-person contact, R_0 would be equal to ρ nt, where ρ is the probability of transmission during a single contact between an infective and a susceptible, n is the daily number of contacts by an infective, and t is the total duration (in days) of the infective period.[11] If $R_0 < 1$, then introduction of the disease into a previously unexposed population will rapidly end in "fade-out" without causing a widespread epidemic. Indeed, estimated values of R_0 for diseases such as measles and pertussis, which often cause large-scale epidemics, are generally well in excess of ten (see Anderson and May 1991:70). The evidence suggests that t is small in pneumonic plague—on the order of 1–3 days (Seal 1969). And ρ is almost certainly much smaller than most historians claim (i.e., the disease is not highly contagious). Because the severity of symptoms quickly immobilizes the victim of pneumonic plague, the value of n (the number of contacts per day) is also likely to be small (Seal 1969). Thus, pure pneumonic plague almost certainly fails to meet the condition $R_0 > 1$ and is therefore unlikely to be the cause of any epidemic as widespread and fast moving as the Black Death.

We accept as a theoretical possibility that the Black Death may have been caused by something closely related and perhaps ancestral to *Y. pestis*. But if so, the pathogen must have been sufficiently different from any known modern strain of *Y. pestis* to have had quite different metapopulational dynamics (Scott and Duncan 2001:86–88), arising perhaps from differences in infectivity, mode of transmission, or virulence. In principle, recovery of pathogen DNA from known Black Death victims is the only way to settle the issue conclusively. But given the vagaries of ancient DNA analysis (Kolman and Tuross 2000; MacHugh et al. 2000; O'Rourke et al. 1996; Stone 2000; Hofreiter et al. 2001), epidemiological evidence on the Black Death still has an important role to play in the debate.

Acknowledgments The authors owe an enormous debt of gratitude to Dr. Richmond A. Davies, who generously gave us access to his unpublished translation of Bishop Roger de Northborough's register, and who uncomplainingly helped us unravel the mysteries of medieval church organization. We also thank Jesper Boldsen, Darryl Holman, Hans Christian Petersen, Stephen Matthews, and Graham Twigg for comments and suggestions. Figure 3 was prepared by the GIS Unit of the Pennsylvania State University's Social Science Research Institute, whose assistance we gratefully acknowledge. This paper is dedicated to Frank Livingstone with gratitude and affection, though he'll hate us for saying so.

Received 4 December 2002; revision received 30 April 2003.

[11]In the literature on mathematical epidemiology, R_0 is more commonly written as β/γ, where β is the total daily transmission rate, equal to ρn. The parameter γ is the daily removal rate, which is approximately equal to $1/t$ if the infective and symptomatic periods largely overlap.

Literature Cited

Aberth, J. 1995. The Black Death in the diocese of Ely: The evidence of the bishop's register. *J. Medieval Hist.* 21:275–287.

Aberth, J. 2001. *From the Brink of the Apocalypse: Confronting Famine, War, Plague, and Death in the Later Middle Ages.* London, UK: Routledge.

Achtman, M., K. Zurth, G. Morell et al. 1999. *Yersinia pestis*, the cause of plague, is a recently emerged clone of *Yersinia pseudotuberculosis. Proc. Natl. Acad. Sci. USA* 96:14043–14048.

Anderson, R.M., and R.M. May. 1991. *Infectious Diseases of Humans: Dynamics and Control.* Oxford, UK: Oxford University Press.

Bannerman, W.B. 1906. The spread of plague in India. *J. Hygiene* 6:179–211.

Benedict, C. 1996. *Bubonic Plague in Nineteenth-Century China.* Stanford, CA: Stanford University Press.

Beresford, M., and J. Hurst. 1990. *Wharram Percy, Deserted Medieval Village.* New Haven, CT: Yale University Press.

Beresford, W. 1883. *Diocesan Histories: Lichfield.* London, UK: Society for Promoting Christian Knowledge.

Brownlee, J. 1918. Certain aspects of the theory of epidemiology in special relation to plague. *Proc. Roy. Soc. Med.* 11:85–132.

Burnet, M. 1962. *Natural History of Infectious Diseases.* 3rd ed. Cambridge, UK: Cambridge University Press.

Carpentier, É. 1962. Famines et épidémies dans l'histoire du XIVe siècle. *Ann. Écon. Soc. Civil.* 17:1062–1092.

Cohn, S.K. 2002. *The Black Death Transformed: Disease and Culture in Early Renaissance Europe.* London, UK: Edward Arnold.

Coulton, G.G. 1929. *The Black Death.* London, UK: Ernest Benn.

Davies, R.A. 1989. The effect of the Black Death on the parish priests of the medieval diocese of Coventry and Lichfield. *Hist. Res.* 62:85–90.

Davis, D.E. 1986. The scarcity of rats and the Black Death: An ecological history. *J. Interdisc. Hist.* 16:455–470.

Dohar, W.J. 1995. *The Black Death and Pastoral Leadership: The Diocese of Hereford in the Fourteenth Century.* Philadelphia, PA: University of Pennsylvania Press.

Drancourt, M., G. Aboudharam, M. Signoli et al. 1998. Detection of 400-year-old *Yersinia pestis* DNA in human dental pulp: An approach to the diagnosis of ancient septicemia. *Proc. Natl. Acad. Sci. USA* 95:12637–12640.

Ell, S.R. 1980. Interhuman transmission of medieval plague. *Bull. Hist. Med.* 54:497–510.

Gasquet, F.A. 1908. *The Black Death of 1348 and 1349.* 2nd ed. London, UK: George Bell.

Gyug, R. 1983. The effects and extent of the Black Death of 1348: New evidence for clerical mortality in Barcelona. *Mediev. Stud.* 45:385–398.

Hatcher, J. 1977. *Plague, Population and the English Economy 1348–1530.* London, UK: Macmillan.

Herlihy, D. 1997. *The Black Death and the Transformation of the West*, ed. S.K. Cohn. Cambridge, MA: Harvard University Press.

Hofreiter, M., D. Serre, H.N. Poinar et al. 2001. Ancient DNA. *Nature Rev. Gen*. 2:353–359.

Horrox, R. 1994. *The Black Death.* Manchester, UK: Manchester University Press.

Karlsson, G. 1996. Plague without rats: The case of fifteenth-century Iceland. *J. Mediev. Hist.* 3:263–284.

Kolman, C.J., and N. Tuross. 2000. Ancient DNA analysis of human populations. *Am. J. Phys. Anthrop.* 111:5–23.

Lunn, J. 1930. The Black Death in the Bishops' Registers. Ph.D. diss. Cambridge, UK: St. John's College, University of Cambridge.

MacHugh, D.E., C.J. Edwards, J.F. Bailey et al. 2000. The extraction and analysis of ancient DNA from bone and teeth: A survey of current methodologies. *Ancient Biomols.* 3:81–102.

Morris, C. 1971. The plague in Britain. *Hist. J.* 14:205–215.

O'Rourke, D.H., S.W. Carlyle, and R.L. Parr. 1996. Ancient DNA: Methods, progress, and perspectives. *Am. J. Hum. Biol.* 8:557–571.

Perry, R.D. and J.D. Fetherston. 1997. *Yersinia pestis*—Etiologic agent of plague. *Clin. Microbiol. Rev.* 10:35–66.

Petrie, G.F., and R.E. Todd. 1923. *A Report on Plague Investigations in Egypt.* Cairo, Egypt: Public Health Laboratories.

Pollitzer, R. 1954. *Plague.* Geneva, Switzerland: World Health Organization.

Raoult, D., G. Aboudharam, E. Crubézy et al. 2000. Molecular identification by 'suicide PCR' of *Yersinia pestis* as the agent of the medieval Black Death. *Proc. Natl. Acad. Sci. USA* 97:12800–12803.

Russell, J.C. 1948. *British Medieval Population.* Albuquerque, NM: University of New Mexico Press.

Scott, S., and C.J. Duncan. 2001. *Biology of Plagues: Evidence from Historical Populations.* Cambridge, UK: Cambridge University Press.

Seal, S.C. 1969. Epidemiological studies of the plague in India: I. The present position. *Bull. W.H.O.* 23:283–292.

Shrewsbury, J.F.D. 1970. *A History of Bubonic Plague in the British Isles.* Cambridge, UK: Cambridge University Press.

Simonet, M., B. Riot, N. Fortineau et al. 1996. Invasin production by *Yersinia pestis* is abolished by insertion of an IS*100*-like element within the *inv* gene. *Infect. Immun.* 64:375–379.

Simpson, W.J. 1905. *A Treatise on Plague*. Cambridge, UK: Cambridge University Press.

Smith, D.M. 1981. *Guide to Bishops' Registers of England and Wales: A Survey from the Middle Ages to the Abolition of Episcopacy in 1646.* London, UK: Offices of the Royal Historical Society.

Smith, R.M. 1988. Human resources. In *The Countryside of Medieval England,* G. Astill and A. Grant, eds. Oxford, UK: Blackwell, 189–212.

Stone, A.C. 2000. Ancient DNA from skeletal remains. In *Skeletal Biology of Past Peoples: Research Methods,* S. Saunders and M. Katzenberg, eds. New York, NY: Wiley-Liss, 351–371.

Thompson, A.H. 1911. Registers of John Gynewell, Bishop of Lincoln, for the years 1347–50. *Archaeol. J.* 68:300–360.

Thompson, A.H. 1914. The pestilences of the fourteenth century in the Diocese of York. *Archaeol. J.* 71:97–154.

Thompson, E.M., ed. 1889. *Chronicon Galfridi le Baker de Swynebroke.* Oxford, UK: Oxford University Press.

Twigg, G. 1984. *The Black Death: A Biological Reappraisal.* London, UK: Batsford.

Twigg, G. 1995. Bubonic plague: Doubts and diagnoses. *J. Med. Microbiol.* 42:383–385.

Voong, K., W.J. White, and M.B. Prentice. 2001. Attempted PCR detection of *Y. pestis*-specific DNA from dental pulp of Black Death victims. Paper presented at the Fourth International Congress on the Evolution and Paleoepidemiology of Infectious Diseases, Marseilles, France.

Wood, J.W., S.N. DeWitte-Aviña, R.J. Ferrell et al. N.d. *The Biology of the Black Death 1347–1350.* Cambridge, UK: Cambridge University Press, in preparation.

Wu Lien-teh. 1922. Plague in the Orient with special reference to the Manchurian outbreaks. *J. Hygiene* 26:62–76.

Wu Lien-teh. 1926. *A Treatise on Pneumonic Plague.* Geneva, Switzerland: League of Nations.

Yersin, A. 1894. La peste bubonique à Hong-Kong. *Ann. Inst. Pasteur* 8:662–667.