

## THE ARMIES OF THE BLACK DEATH

Ole Benedictow, *The Black Death 1346–1353: The Complete History*

(Woodbridge: The Boydell Press, 2004)

Since it first emerged, devastating and transformative, upon an unsuspecting Europe, the Black Death has held a powerful place within the European psyche. Both historians and non-historians have long been fascinated by this monstrous epidemic that wiped out such a sizeable proportion of the European population and catapulted Europe into change. Despite this, research into details of the plague has remained fragmentary, localized and often highly suppositional. In *The Black Death, 1346–53: The Complete History*, Ole Benedictow attempts to remedy this situation.<sup>1</sup> Collating and analyzing both primary and secondary sources, he examines the nature of the disease, discusses its mode and rate of spread and estimates mortality rates to arrive at one of the most complete models of the progress of the disease over Europe.

Although Alexander Yersin identified the bacterium that caused the plague in Canton over a century ago and subsequent epidemiologists expanded upon his work to link *Yersinia pestis* to rats and rat-fleas, historians and epidemiologists still cannot reach a consensus that *Y. pestis* was the cause of the fourteenth century epidemic. Benedictow, however, firmly believes that *Y. pestis* was the culprit and supports his argument with a detailed examination of the *modus operandi* of plague, rats and rat-fleas. He explains that the rat flea *Xenopsylla cheops* ingests and then becomes “blocked” by the *Y. pestis* bacteria, regurgitating the bacteria back into both rat and human hosts when it feeds. This vector-borne mode of transmission, Benedictow avers, explains the “explosive” growth of morbidity and mortality seen in the Black Death.<sup>2</sup>

Once within the body, the bacteria spread to the lymphatic system, causing primary bubonic plague or into the blood stream, causing primary bacteraemic plague.

Primary bubonic plague can also develop into secondary bacteraemic or pneumonic plague as the bacteria spread elsewhere in the body. Secondary pneumonic plague causes coughing up of bloody sputum that can, albeit rarely, transmit bacteria directly to another’s lungs, causing primary pneumonic plague.<sup>3</sup> These three primary and three secondary forms each show different symptoms and lethality rates and go a long way to explaining the divergent descriptions of symptoms within the chronicles that Herlihy uses to suggest that plague was not the cause of the Black Death.<sup>4</sup> Moreover, understanding rat-flea interaction, the reaction of fleas and rats to temperature changes as well as the adaptation of rat fleas to light, clothing and grain can explain not only the slow-down of the plague in winter – which itself argues against the “viral” theory of Black Death – but also the ease with which the disease spread along European trade-routes and the peculiar inverse relationship between population density and mortality noted in outbreaks of plague. In these arguments, Benedictow shows a deeper understanding of epidemiology that such historians as Herlihy or Samuel Cohn. In particular, his understanding of the different forms of plague goes a long way to solidifying the validity of the role of plague in the Black Death. However, the absence of the most modern epidemiological sources and the lack of direct responses to counter arguments leave some questions remaining.

One of the most cogent questions put forth by Cohn is the disparity between the spread of bubonic plague evinced in the nineteenth century outbreaks and that seen in the Black Death.<sup>5</sup> Benedictow answers, albeit indirectly, by systematically studying the “patterns of spread in time and

---

<sup>1</sup> Ole Benedictow, *The Black Death 1346–1353: The Complete History* (Woodbridge: The Boydell Press, 2004)

<sup>2</sup> *Ibid.*, 18.

---

<sup>3</sup> *Ibid.*, 25–28.

<sup>4</sup> David Herlihy, *The Black Death and the Transformation of the West* (Cambridge: Harvard University Press, 1995), 29.

<sup>5</sup> Samuel K. Cohn Jr., *The Black Death Transformed: Disease and Culture in Early Modern Europe* (London: Hodder Arnold, 2003) 111

space” to create a powerful model for plague transmission.<sup>6</sup> An important factor in this model is the concept of time. Taking what is known about plague epidemiology, he posits a six-week lag between the first arrival of plague in a location and the explosion of the epidemic and subsequent reportage in the chronicles. This time lag, he asserts, is based on the establishment of plague first in the local rat populations and only then in human populations, and is characteristic of such a vector-borne disease.<sup>7</sup> Should a model incorporating this time lag, as well as other characteristics of plague, hold for Europe, it would confirm the bubonic plague as the cause of the illness.

Rejecting William McNeill’s assertion that plague arose in Eastern Asia,<sup>8</sup> Benedictow maintains it first arose in Southern Russia, from whence it spread first to Kaffa then onwards to the Mediterranean. Studying the history of trade in the Mongol Empire, he notes that after their conversion to Islam, the Mongols halted trade with Christian nations. This turmoil would favor a Southern Russian locus as well as explain why Russia was the last rather than the first location hit by the plague.

From Kaffa and Constantinople, Benedictow follows the plague as it hit important seaports on the Mediterranean and then the Atlantic coasts of Europe, creating “beach-heads” from which the plague spread inland along rivers, coasts, trade-routes and – more slowly – along overland roads to blanket entire nations. In an exhaustive and detailed survey, he covers all of Europe, examining available primary sources and comparing them to the expected temporal progress of the disease according to his “time lag” model. Where the sources are most readily available, such as for England, Benedictow’s model holds up very well. Plague arrived in England by a “metastatic leap” along the maritime trade route from Bordeaux to Weymouth in May 1348 and spread from thence to Bristol and Ireland before the chronicles first noted the outbreak in

late June.<sup>9</sup> Using registers of clerical institutions and manorial records, he is able to trace the progress of the epidemic throughout England through 1348 and 1349. His work is meticulous and well argued throughout and the evidence cleaves so well to the model that it does lend further credence to the “bubonic plague” theory of the Black Death. However, where there is less evidence, such as in Russia, Benedictow must rely more upon supposition, positing a time-line that fits to the model rather than proving the model using a time-line. However, whilst this may weaken his argument somewhat, it does not invalidate it. The model proves more complete and compelling than any previous attempt to track the plague. Moreover, taking into account time lag and density of black rather than brown rats in fourteenth century Europe – neither of which Cohn addresses in his work – as well as human agency in the spread of disease, Benedictow goes a long way to addressing concerns about transmission rate. Taken as a whole, this serves to reinforce the bubonic plague causation theory.

Just as historians have been unable to reach consensus upon the cause of the Black Death, so too have they continued to argue about the mortality rates it caused. McNeill asserts a “best estimate” of thirty three per cent; Rosemary Horrox, surveying the historiography of plague mortality, notes that examination of sources have led to earlier estimates of between one-quarter and one third rising to a current preference for mortality rates of approximately forty to forty five percent.<sup>10</sup> Benedictow, however, rejects these figures as significant underestimates based on faulty assumptions. To gauge plague mortality, he asserts, historians must consider medieval demographic patterns, which do not necessarily correspond to demographic patterns for other eras. Moreover, primary records used to reach plague mortality figures must be subject to intense source criticism. Tax records, for example, suffer from under-registration due to evasion;

---

<sup>6</sup> Benedictow, 68.

<sup>7</sup> *Ibid.*, 58.

<sup>8</sup> William McNeill, *Plagues and Peoples* (New York: Anchor, 1977), 171.

---

<sup>9</sup> Benedictow 126.

<sup>10</sup> McNeill 179, and Rosemary Horrox, *The Black Death*, Manchester Medieval Source Series (Manchester: Manchester University Press, 1994), 231–234.

other records, including manorial rolls, do not record the poor, women or children but only heads of households. Given that the very limited sources that do include the destitute suggest a “supermortality” among the poorest in society of approximately five per cent and that as many as forty five to fifty per cent of the total population fell into the poorest economic bracket of society, ignoring this class of people has led to previous underestimations of mortality. Similarly, unconsidered supermortality among women and children can also skew the figures.

As he did when considering the spread of the Black Death, Benedictow proceeds to examine the available records of various regions of Europe in turn, reconsidering the mortality figures in light of detailed and forceful source criticism. His results are remarkably consistent. In England, for example, examination of manorial court rolls, lists of frankpledge and head tax records each yield mortality figures of between sixty and sixty five per cent.<sup>11</sup> Only registers of clerical institutions yield lower figures, yet Benedictow explains this by analyzing a number of factors that could have led to lower rather than (the more commonly assumed) higher mortality figures for the clergy. Moreover, in each of the regions where sufficient records exist, similar figures emerge. Thus, although many regions do not have adequate records to gauge local mortality, the stability of the results elsewhere leads him to conclude that the mortality rate of Europe in the Black Death was a “mind boggling, horrifying and unnerving” sixty percent. Of a general population of approximately eighty million, some fifty million perished. While certainly these figures are based upon a series of assumptions and extrapolations, something that Benedictow does acknowledge, he maintains his assumptions are “cautious.”<sup>12</sup> Certainly, he explains each assumption he makes and the consequence of such an assumption in some detail. None of the assumptions appear particularly irrational or egregious. Furthermore, his conclusions fit with local studies whose “remarkable and even startling results” previous historians

have ignored as too unimaginable.<sup>13</sup> This all leads to the depressing but persuasive conclusion that estimates of mortality should be significantly raised above the previous levels.

Such mortality on a massive scale, Benedictow asserts, had to have a profound impact upon late medieval society. Rejecting the Malthusian model, he portrays the societal changes that emerged out of the Black Death to be intensified continuations of existing trends, given new “dynamic powers” by the demographic collapse.<sup>14</sup> Unfortunately, in this, the weakest section of the book, Benedictow’s detailed analysis of sources disappears. His conclusions are neither well argued nor well presented. Mirroring Herlihy, for example, he notes a growth in laborsaving technology post-Black Death; a growth that Cohn maintains is dubious at best.<sup>15</sup> His assertion of universal religious panic, as evinced through the flagellant movement, contradicts the more rational and varied reactions seen in Horrox’s collection of chronicles.<sup>16</sup> Where he does agree with other sources or historians, he makes sweeping generalizations. In this respect, he most resembles William McNeill, whose *Plagues and Peoples* covered such a broad swathe of history that deep analysis of regional variations was impossible. Also, like McNeill, he tends to focus upon the agency of the disease itself: what it was, how it spread, what its immediate effect was on mortality. In doing so, to a great extent he omits the human element that was so striking in the chronicles. While one might argue that this is an inevitable result of the demands of the analysis of spread and mortality, of the sheer amounts of data Benedictow processed, William Jordan’s *The Great Famine* shows that a historian can include compelling data, make conclusions about demographics and mortality, as well as examine individual, corporate and societal reactions to disaster in one work.<sup>17</sup> This de-emphasis upon human agency, together with the weakness of Benedictow’s

---

<sup>11</sup> Ibid., 377.

<sup>12</sup> Ibid., 382.

---

<sup>13</sup> Ibid., 380.

<sup>14</sup> Ibid., 388.

<sup>15</sup> Herlihy, 49 and Samuel Cohn, “Introduction” in Herlihy, 10.

<sup>16</sup> Horrox, *passim*

<sup>17</sup> William Jordan, *The Great Famine* (Princeton: Princeton University Press, 1997)

conclusions on the impact of the Black Death are, in a text purporting to be a “Complete History,” regrettable shortcomings. In spite of this, Benedictow’s arguments and conclusions regarding the cause, transmission and mortality

are so concomitantly strong that *The Black Death, 1346–53: The Complete History* is an essential work for those interested in the Black Death, the history of medicine or in demographic history.