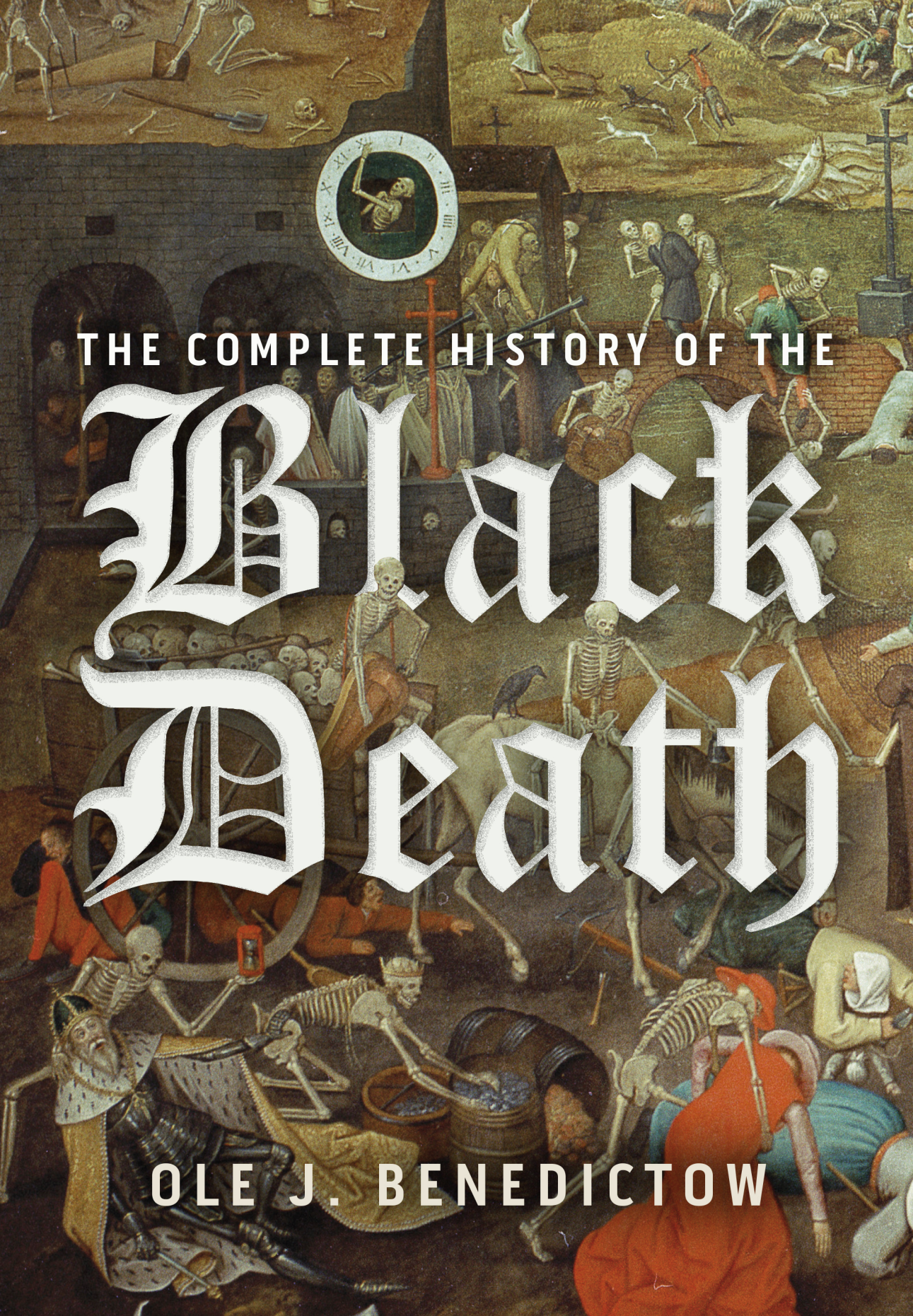
THE COMPLETE HISTORY OF THE
Black Death
OLE J. BENEDICTOW

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THE BOYDELL PRESS

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For my sons

TANCRED and ANDREAS

and in memory of

PROFESSOR SIGVALD HASUND

who discovered the late-medieval crisis

and laid the foundations of the

Norwegian School of Agricultural History


The advance of the Black Death year by year
1346
1347
1348
1349
1350
1351
1352
1353
no data
Arrival of plague preceding autumn
Direction of the advance of the Black Death
Au Augsburg
Ba Barcelona
Br Braunau
Co Colchester
Cu Cuenca
Ge Geneva
Ha Harwich
In Innsbruck
La Landshut
Li Lincoln
Ll Lleida
Ly Lynn
Ma Marseille
Mo Moldava
Ne Newark
Nu Nuremburg
Pi Piombino
Pí Písek
Po Porto Venere
Pu Pula
Sa Salzburg
Se Sebenico
Ss San Sebastian
Ta Tarragona
Tá Tábor
To Toulouse
Tr Traú
Approximate border between the Mongol Khanate, the Golden Horde, and the Russian principalities Passage prohibited for Christians
ATLANTIC OCEAN
Faroe Is.
Shetland Is.
Bergen
summer 1349
autumn 1348
1349
Nidaros
Hamar
Oslo
Stockholm
Gothenburg
Halmstad
Lund
Visby
Gotland
Copenhagen
Ribe
Kiel
Stralsund
Lübeck
Hamburg
Bremen
Danzig
Königsberg
Elbing
Thorn
Frankfurt
Poznań
Warsaw
Narva
Pskov
Novgorod
Belozersk
Rybinsk
Tver
Suzdal
Nizhniy Novgorod
Moscow
Smolensk
Ryazan
Kazan
Kama
Volga
Oka
Glukhov
Chernigov
Kiev
Dnepr
Lutsk
Lviv
Kraków
Breslau
Oder
Vistula
Elbe
Weser
Rhine
Erfurt
Mo
Prague
Pí
Tá
Brno
Nu
Passau
La
Br
Sa
Au
Munich
In
Vienna
Budapest
Oradea
Mürztal
Danube
York
Hull
Grimsby
Li
Ne
Ly
Co
Ha
Bristol
London
Weymouth
Drogheda
Dublin
Kilkenny
Calais
Ghent
Cologne
Frankfurt
Mainz
Strasbourg
Basle
Rouen
Caen
Paris
Loire
Besançon
Chalon
Lyon
Ge
Rhône
Bordeaux
Avignon
Aix
Ma
To
Narbonne
Perpignan
Ss
Gijón
La Coruña
Oviedo
Santiago de Compostela
León
Soría
Zaragoza
Ll
Ba
Ta
Cu
Teruel
Madrid
Toldedo
Valencia
Alcudia
Mallorca
Coimbra
Lisbon
Seville
Cartagena
Cadiz
Almeria
Algeciras
Oran
Fez
Algiers
Tunis
Trento
Milan
Venice
Genoa
Po
Pisa
Florence
Bastia
Perugia
Aléria
Pi
Rome
Corsica
Caprera
Sardinia
Cagliari
Naples
Messina
Trápani
Sicily
Pu
Tr
Spalato
Zara
Se
Ragusa
Belgrade
Bucharest
No data
Tiraspol
Odesa
Moncastro
Sulina
Constanta
Varna
Burgas
Constantinople
Smyrna
Bursa
Karabük
Akşehir
Konya
Karaman
Sivas
Divriği
Kayseri
Erzincan
Trebizond
Batumi
Poti
Sukhumi
Sinope
BLACK SEA
Kaffa
Soldaia
Taganrog
Tana (Azov)
Astrakhan
Dubovka
New Sarai
Old Sarai
THE GOLDEN HORDE
Don
Caspian Sea
Baku
Masalli
Tabriz
Diyarbakir
Mardin
Antioch
Aleppo
Mosul
Tigris
Euphrates
Bagdad
Basra
Persian Gulf
Homs
Damascus
Jerusalem
Gaza
Cyprus
Crete
Candia
MEDITERRANEAN SEA
Tripoli
Barqa
Alexandria
Cairo
Wadi n'Natrum
Asyut
Red Sea
N
0
800
km

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The Outbreak and Spread of the Black Death

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Preface to the First Edition

In this book, the term the Black Death is used to signify the huge wave of plague epidemics that ravaged Europe, Asia Minor, the Middle East and North Africa in the years 1346–1353.[1] Previous studies of the Black Death have mostly concentrated on the cultural, psychological and religious effects of the Black Death, and also in relation to a specific country. The central reason for this imbalance is that little was known on the spread of the Black Death in a wider perspective, and only few demographic studies on mortality were available before 1960. From about this time, many new studies on the local spread and mortality of the Black Death were published in many countries, which can be pieced together in a synthetical and holistic account. Taken together, they provide a completely new opportunity to identify the territorial spread of the Black Death in Asia Minor, the Arab world and Europe, to identify its epidemiological characteristics and make inferences on the mechanisms of transmission and dissemination. Even more importantly, it has become possible to realistically assess the level and social structures of mortality caused by this vast plague epidemic, which is highly relevant to questions of historical impact.

The main objectives of this book are to perform a complete study of the territorial spread, epidemiology and mortality of the Black Death according to all available sources and studies, and to lay the foundation for more useful discussions of its historical impact. It is in these respects that this book's ambitious goal is to be a complete history of that epic epidemic. (It should not be misunderstood to imply its final history.)

Surprisingly, these many fine studies had not been gathered together, collated, discussed and synthesized before, not even at the national level of analysis and synthesis. Ziegler's 1969 book on the Black Death in the British Isles is, in my opinion, still the best general study of the Black Death. Biraben gives a valuable but brief overview of the Black Death's spread across Europe in his 1975 study, which, however, leaves much still to be said on the subject, while his discussion of mortality is really confined to some aspects of its French history. In the early 1970s, two Spanish scholars, Sobrequés Callicó and Ubieto Arteta, published valuable overviews of the spread of the Black Death across the Iberian Peninsula; at the end of the 1980s, Dubois presented a brief, but valuable summary of the Black Death's spread in France, but passes quite lightly over the question of mortality. I would also like to mention my own discussion of the Black Death in the Nordic countries in my doctoral thesis of 1992, which was reprinted in 1993 and 1996. However, a serious attempt at producing a general synthetic study of the Black Death's epidemiology, territorial spread and mortality has not been attempted before. My book on the late-medieval plague epidemics in the Nordic countries is the only comprehensive and in-depth study of the Black Death covering national territories to have been published since Ziegler's

[1] The history and scope of the concept of the Black Death is clarified below in Chapter 1.1, the concept of pandemic in the Glossary.

study. The quite numerous new local studies and the opportunities they offer of expanded knowledge and insights have largely been neglected.

In the present book, no effort has been spared to collect, collate and synthesize the available studies on the Black Death's epidemiology, spread and demographic impact, from its origin in the lands of the Golden Horde in south-eastern Russia in 1346 until it petered out in central Russia seven years later. It had then engulfed the Caucasus, Asia Minor, the Middle East, North Africa and the whole of Europe excepting Finland and Iceland and, perhaps, a few small areas in addition. Why some areas or regions should have been spared is also an interesting question and has been allotted corresponding attention.

Most of the new studies have been undertaken by economic historians or historians of local history who have come across interesting source material that was also usable for analysis of the mortality suffered by the local population or some section of it in the Black Death. By implication, these studies quite often (but far from always) also contain information on the whereabouts of the Black Death in time and space.

The new data that have been forthcoming are usually only basic data. To develop them fully into epidemiological and demographic data requires a heavy input not only of the highly specialized knowledge of medievalists in relation to the understanding and treatment of the great diversity and intricacies of medieval source materials, but also of the insights of epidemiologists of plague, and the craft of historical demographers. There is, of course, also a very demanding requirement with respect to access to the great diversity of European tongues in which the studies containing information on the Black Death have been written. This challenging set of scholarly requirements must be the reason that so far no real attempt has been made to gather together, collate and synthesize the available data. Many of these studies have been published in local journals of history or in books on local history that can be quite difficult to identify and get hold of, especially in great numbers.

Uncertainty is inevitable in all attempts to produce demographic estimates on the basis of sources from 'pre-statistical' times – documents or rolls that never were intended to be used for this purpose. Margins of uncertainty and level of tenability are key terms. In the first half of the fourteenth century, some Italian city states developed registrations of their populations for various purposes that take on the character of censuses. However, generally the most important category of source used in these mortality studies is tax registers of various types that involve an array of source-critical issues relating to the proportion and social composition of untaxed population segments, care in registration, tax evasion, and so on. Only in England is the main type of source manorial records, which, of course, involve an array of source-critical issues of their own.

Endeavours to uncover the Black Death's pattern and pace of spread are too often dependent on notes in chronicles that are jotted down quite carelessly. Even worse, chroniclers quite often ignore the Black Death because epidemics were a peripheral subject to persons with classical education and classical models. It is often only possible to piece together a coherent basic outline of the spread of Black Death with great effort, too frequently involving substantial uncertainty in the concluding remarks.

Author's Note on the New and Revised Edition

Introduction

The first edition of this book on the Black Death was favourably received by scholars and the wider public: the fourth printing is nearly sold out, and it has acquired status of a standard work that is often referred to in plague studies to provide a general backcloth or to provide material or arguments on specific points or issues. Much has occurred in these years that has a bearing on the history of plague and the Black Death. It has, in fact, been a progressive but also quite a tumultuous period, including many valuable contributions and important new developments, but sadly also much negative and even reproachable coverage. The time has come to produce an updated second edition that includes the valuable contributions in the field of historical plague research and the Black Death in the past roughly 15–20 years and also positive developments in the author's knowledge and insights (as he sees it).

This book is a holistic study of the Black Death, the biggest and most mortal epidemic in history, and with momentous societal effects, also in terms of a long-term historical perspective. Accordingly, it was the ambition of the first edition to gather together and synthesize all useful studies and evidence on all important aspects of the epidemic.

Evidently, this approach was inspired also by some central perspectives of the French Annales School of history, the emphasis on total history, *histoire totale*, or *histoire tout court*, a study of the complete source mateiral of a historical topic with an emphasis on the methodology and techniques of demography and social science as analytical tools.

These goals appear to have been achieved insofar as no supplementary evidence on spread or mortality relating to the Black Death has been presented by other scholars. A considerable amount of new evidence will, nonetheless, be presented in this second edition.

The new availability of historical sources

An important positive development (also) for historical plague research in the years since the typescript of the first edition was finished in 2003 is the huge increase in the availability of historical sources on the Internet. This comprises the big series of editions of medieval and early-modern sources for Italy, Germany and the then German lands of Austria, Bohemia (Czeckia), and the then territory of the Polish kingdom, Russia and the Scandinavian countries. This material includes many individual editions of chronicles and early editions of historical works that often contain sources that later have been lost, and so on. This has made it possible to write a mainly source-based history of the Black Death in these countries, organized and analyzed according to contemporary economic, religious and political structures, and according to modern medical and epidemiological knowledge on plague disease. The first edition of this book contained source-based accounts of the Black Death in the three Scandinavian countries, and partly source-based for other countries. This edition also contains the first fully source-based accounts of the Black Death in Russia, Poland, Germany and Italy.

Note on historical chronology

The Black Death spread in regions that did not belong to Western civilization and which, at the time, were dominated by Muslim states, religion and culture or the Byzantine Empire and culture and Orthodox religion. In the latter case, it means that Russian chronicles and contemporary Byzantine chroniclers and historians cited in this book to write the history of the Black Death use Orthodox chronology. Arab/Muslim and Byzantine/Orthodox cultures are considered autonomous civilizations with their own historiography, historical chronology and periodization.

For practical reasons, as historians in Muslim countries using Muslim chronology constitute a tiny element in the international writing of history, and historians in Orthodox countries use Western chronology, it has been decided to use consistently Western chronology including the term medieval, which is a specific period in the Western or European civilization, throughout this book. This chronology is adapted to a non-religious practice by using the terms CE (Common Era) and BCE (Before Common Era), which are based on the same chronological point. Since (Jesus) Christ apparently was born four to seven years before AD, this akward situation strengthens the meaningfulness of a transition to a more general international historical chronology.

The term medieval refers to the first phase of the new European civilization following the breakdown of the Western Roman Empire to the emergence of early-modern society, the period from c. 500 CE to the decades around 1500. This historical period is subdivided. The historical perspective of this book includes the high Middle Ages, the period 1000–1350 CE, and the late Middle Ages, the period 1350–c. 1500 CE.

When the start of the late Middle Ages is set at 1350, this reflects the notion that the Black Death and ensuing plague epidemics shaped the last phase of the Middle Ages in a decisive way, an indication of the importance of the subject of this book. A more in-depth discussion of this perspective is performed in Part Five below.

Modern medical standard works on plague

There are several outstanding modern medical standard works on plague that are also indispensable for historical plague research. They were written by authors who had dedicated their professional lives to the combat of plague in the Third Plague Pandemic which, from 1894, spread in China and abroad from Hong Kong with modern steamships to India, Indonesia, Madagascar, Egypt, Africa and the Americas, especially USA. This was a shock that led to the establishment of several plague research teams and institutions of plague research. They produced a big corpus of primary plague research that constituted the basis for the standard works on plague where this research was synthesized. They are the works by Hirst, *The Conquest of Plague*, 1953, and Pollitzer, *Plague*, 1954, and perhaps especially the 1936 Chinese standard work co-authored by Wu L.-T., Chun, Pollitzer, and C.Y. Wu, *Plague. A Manual for Medical and Public Health Workers*. É. Brygoo's standard work, a succinct synthesis of important French plague research in Madagascar in the period c. 1921–60, should also be mentioned and, lastly, Butler's 1983 succinct presentation of modern plague that also includes the observations and studies performed by American army physicians against the background of the big plague epidemics in the Vietnam war.

Later plague research has mainly been performed in laboratories as experimental studies and relates mainly to plague reservoirs and their rodents. This research has enlarged upon and confirmed earlier research as presented in the standard works, also confirming that

epidemic plague is transmitted by so-called blocked fleas, for all practical purposes, fleas of the black rat (see below).[2] In recent years, B.J. Hinnebusch, Chief of the Plague Section of the Rocky Mountain Laboratories, has published many valuable articles on plague, alone and with various teams of colleagues, especially relating to the transmission of plague by rodent fleas.[3] Within this field of study, they confirm and develop the findings of the IPRC. Many of them are entered in the Bibliography.

An updated synthesis of plague research is given in my 2010 book *What Disease was Plague? On the Controversy over the Microbiological Identity of Plague Epidemics of the Past*, pp. 73–273, and a short version in my 2016 book *The Black Death and Later Plague Epidemics in the Scandinavian Countries: Perspectives and Controversies*, pp. 14–34.

In a time of much doubtful science and proliferation of poorly substantiated so-called alternative theories on historical plague, readers are encouraged to be sceptical of works on plague or plague history that do not have a solid basis in these standard works or the primary plague research they refer to and synthesize.

Most authors of the standard works (except Brygoo and Butler) had strong interests in the history of the disease they studied. This shows in that these works contain at least a chapter or parts where the modern plague they studied in real time was compared to historical studies, especially on the Black Death. In this adequate methodolologial way they ascertain by similarity or dissimilarity whether the plague disease they studied was the same disease or a different disease from historical plague. The methodological approach was comparative diachronic[4] analysis as historians would do. They all concluded that the modern plague they studied and historical plague was, for all practical purposes, the same disease, displaying the same clinical and epidemiological features.

The epidemiology of the Black Death as uncovered by a complete synthesis of sources and historical studies must be consistently compared with the epidemiology of bubonic plague and primary pneumonic plague as presented in the primary studies and standard works on plague. This will determine the identity of the disease, according to the historian's craft. Recently, the rise of the new scientific discipline of paleobiology has provided an independent opportunity to determine the microbiological identity of the contagion obtained in putative plague graves and to uncover the evolutionary history of historical plague (but not clinical or epidemiological aspects).

Paleobiology, a new scientific discipline of historical (plague) research

Paleobiology is a new branch of microbiological science which focuses on the study of microbiological information in biological material recovered from the remains of living organisms in the past. In paleobiological plague studies, material obtained from skeletal remains in putative plague graves is examined for remains of a(ncient)DNA or the specific F1 antigen of the protein capsule of the bacterium *Y. pestis* (or of other possible pathogens).[5] The crucial difference between these two sources of microbiological information is that DNA contains

[2] Hinnebusch, Bland, Bosio and Jarrett 2017: 1–15; Hinnebusch, Jarrett and Bland 2017: 215–32.

[3] See Bibliography below and Bibliography in Benedictow 2016: 674, 676–7, 680, 682.

[4] The comparison of social phenomena or events in different historical periods in contrast to synchronic comparison, which is between contemporary social phenomena.

[5] More accurately it is a *Y. pestis*-specific fraction 1-capsule antigen, which is a plasmid-expressed immunogenetic envelope glycoprotein.

the genetic instructions used in the development and functioning of living organisms. Antigens stimulate immune responses that leave specific detectable traces and they are also more resistant to degradation than DNA. This is more easily identifiable with technical equipment (dipstic assays for detection of antibodies). For the first time, this has allowed researchers to uncover the presence, identity and probable lethal effects of historical infectious diseases that did not produce identifying macroscopic marks on skeletal material.

By 2014, 20 paleobiological articles on the study of biological material from plague graves had been published. These 20 articles give the outcomes of 45 biomolecular studies of skeletal material obtained in putative plague graves or burial pits in 40 different localities and of hundreds of specimens of individual skeletal remains. Four of them related to the First Plague Pandemic (541–767 CE), and 36 to The Second Plague Pandemic (1346–c. 1690). In all cases, genetic evidence of *Y. pestis* was recovered but not any other serious disease. This material includes 12 studies of biological material obtained in plague graves or pits that with certainty or probability relate to the Black Death in the years 1348–50. The samples have consistently yielded positive identifications of *Y. pestis* of the Black Death in localities as far apart as London and Montpellier.[6]

The (mis)use of mathematical epidemiological models in historical plague research

In 2001, Scott and Duncan published a treatise on the Black Death and later epidemics of the Second Plague Pandemic where they introduced the use of a mathematical epidemiological model, the co-salled Reed-Frost SIR mathematical model, to identify the disease. They claimed that the model had shown that the Black Death and later plague epidemics was not bubonic plague caused by the bacterium *Y.(ersinia) pestis* transmitted and spread by rat fleas but an entirely different disease, a Filoviridae disease that was a (variant) of Ebola disease or Marburg disease. A few years later other scholars used the same model to the same effect. This was shown by paleobiological studies and by a thorough historical study to be entirely false.[7]

This ended attempts to 'prove' that the Black Death and later plague epidemics was not bubonic plague. However, this introduced a new phase when mathematical epidemic models, *the same models*, were used by a number of scholars to 'prove' that the Black Death was indeed caused by *Y. pestis* but was transmitted by human fleas and lice. This 'proved' again that mathematical (epidemiological) models can be used to 'prove' any preconceived notion depending on the selection of input of data. The long-established fact that only a minority of human plague cases develop plague bacteraemia, i.e., bacteria in the bloodstream, and will not infect any feeding insect and that almost all humans who develop bacteramiea have so low levels that far over 90 percent of feeding fleas will not contain plague bacteria at the time of the next feed,[8] has consistently been passed by in silence. The basic data will be presented below in

[6] Benedictow 2016a: 73–97; Spyrou, Tukhbatova, Feldman, et al. 2016: 874–81. All such studies published by the end of 2008 were presented and discussed in Benedictow 2010: 381–95.

[7] See previous note and Benedictow 2010: 610–63. See also pp. 1–9, 82–99, 127–30, 178–200, 298–340, 407–20, 436–46, 462–74, 482–90, 581–91, and elsewhere, cf. Index (of Names).

[8] See Benedictow 1992a/1993/1996a: 228–61; Benedictow 2016a: Chapters 7, 11 and 12, 355–94, 593–665.

Chapter 3 and will show why rats are superior sources of infection of fleas and their fleas are superior vectors of plague.

One should note that these SIR and SEIR models, also with extensions, cannot be used for analysis of bacterial diseases, diseases transmitted by insects, and diseases that have an animal reservoir, such as plague, only to mention a few central conditions that make it clear that these models would malfunction in plague studies. It is important to consider that models are intrinsically analogues, based on a perceived similarity, and as all analogues cannot be used to prove anything only to develop working hypotetheses that must be empirically tested. Models, concepts and definitions have in common that they are intellectual tools for analysis and cannot be used to prove anything but can be discussed in terms of fruitfulness or usefulness.

The misuse of mathematic epidemiological models in historical plague research is thoroughly and decisively analysed in the article 'Epidemiology of Plague: Problems with the Use of Mathematical Epidemiological Models in Plague Research and the Question of Transmission by Human Fleas and Lice'.[9]

Alternative theories on the Black Death and historical plague and the problem of 'fake science'

The Black Death, like Shakespeare, is a high-profile topic, which attracts ambitious scholars with revolutionary new theories and journalists' attention. Despite the huge corpus of primary plague research, and despite that it is synthesized in several fine standard works on plague, 13 different theories on the microbiology and or epidemiology of the Black Death and historical plague more generally have been argued in recent decades. This would not be possible within the perimeter of ordinary scholarly research. It is a development that relates to recent problems with academic standards pointed out by the prestigious and vigilant journal *The Economist*[10] a few years ago,[11] and that find various expressions also in historical plague research.

All 13 alternative theories are fundamentally incompatible. For this reason alone, at least 12 of them must be grossly erroneous and mainly arbitrary or in more popular scholarly parlance often called fake or bogus science, and *The Economist* also activates the term 'fraud' as an equally likely source of 'erroneous results' as 'incompetence'. All alternative theories on historical plague have been thoroughly analysed, making it clear that these characteristics are adequate for all of them, without exception.[12] *The Economist* pointed at the online journal *PLOS One* as particularly problematic, and this is also the case with respect to historical plague research.[13] Other journals could also by mentioned, and some of them will be pointed out below.

9 Benedictow 18 August 2019: 1–20.

10 'How science goes wrong. Scientific research has changed the world. Now it needs to change itself', *The Economist, 19* October 2013 (http://www.economist.co m/node/215880698); 'What's wrong with Science', *The Economist*, 14 December 2013 (http://www.economist.coa m/node/21591549); 'Trouble at the lab. Scientists like to think of science as self-correcting. To an alarming degree it is not', *The Economist, 19* October 2013 (http://www.economist.com/node/21588057).

11 *The Economist, 19 October 2013,* two articles.

12 Benedictow 2010 and Benedictow 2016. For the invalidation of the 13th theory, a purported epidemiological alternative, see also Hinnebusch, Bland, Bosio and Jarrett 1917: 1–15; Hinnebusch, Jarrett and Bland: 2017: 215–32.

13 See, e.g., Welford and Bossak 2009: 1–6.

Recently, a fourteenth alternative theory has been published, which is briefly but adequately presented and discussed in the following subchapter to make this discussion complete.

Independently, paleobiological research has made it clear that all alternative microbiological theories are untenable, including the theories that argue that historical plague was anthrax or a filoviridae disease (e.g., Ebola virus) or a viral disease spread by droplets much as influenza.[14] It has been decisively corroborated that the contagion of historical plague was the bacterium *Yersinia pestis* and also that the strain introduced by the Black Death took root in rat colonies (almost) all over Europe and was the source of all plague epidemics of the Second Plague Pandemic 1346–c. 1690, as it now seems. It also eventually spread to the Yunnan province of China and was the contagion also of the Third Plague Pandemic 1894–c. 1940. See also the following chapter 'Mapping the Black Death'.

The fourteenth alternative theory of the epidemiology of the Black Death and the Second Plague Pandemic: The alleged spread of plague on the (imagined) fur trade routes

Shortly before the manuscript of this book was finished, a sort of fourteenth alternative theory on the Black Death and the Second Plague Pandemic was presented.[15] It deserves a brief comment to update and complete the discussion of alternative theories. In reality, it is a variant of the twelfth theory that the Black Death and the contagion of subsequent plague epidemics were transported by caravan for more than 4,000 km along the so-called Silk Roads from East (Central) Asia to Kaffa on the Crimea where it was shipped to Europe. This theory has been thoroughly discussed and shown (as with the other alternative theories) to be invalid on many independent factual and methodological grounds.[16] Among other things, it was shown that the Black Death and later plague epidemics could not, as asserted, have been transported by caravans along the Silk roads to the Italian factory in Kaffa on the Crimea: the Khanate of the Golden Horde had converted to Islam and had, several years before the Black Death, prohibited trade with Christians and closed the so-called Silk Roads (camel tracks) for trade with Christians. This was a prohibition that the merchants of vulnerable caravans with their precious goods and wish to live longer did not defy. It was the end of the Silk Roads as a commercial link between China and Europe. This is a valid ground for invalidation independently of the fact that there is no indication that plague has ever been transported for any distance on the Silk Roads.

In this fourteenth theory, the team of authors, acknowledging tacitly that the Silk Roads were closed at the time of the Black Death and the Second Plague Pandemic, attempt to defend the basic idea of the twelfth theory by arguing that the contagion of the Black Death and later epidemics of the Second Pandemic in Europe were transported over a more southerly network of alleged 'Fur Roads'. According to the presented map (Fig. 4), these routes seem almost identical with the so-called Spice Roads, which ran via the Ilkhanate (Persia) and also along associated sea lanes.[17] However, the Ilkhanate had converted to Islam, and also these roads

[14] Benedictow 2016a: Ch. 1.5, pp. 73–99; Benedictow 2010: 381–95.

[15] Namouchi, Guellil, Kersten, et al. 2018: E11790–7, E11795. See below Chapter 16, pp. 388–9 and n. 23 for discussion with respect to this article.

[16] Benedictow 2016a: Ch. 1.4, pp. 35–72.

[17] See alleged map of the roads and sea lanes in Namouchi, Guellil, Kersten, et al. 2018: E11795.

or routes were closed for trade with Christians.[18] This decisive piece of information is not mentioned. As should be expected, the authors are unable to indicate or suggest that any plague epidemic has ever been transported over any part of these routes and therefore also not that (the contagion of) any plague epidemic in Europe in the period 1346–c. 1690 originated in Eastern (Central) Asia and was transported along these routes. This puts in perspective the entirely arbitrary nature of the basic idea of the alternative theories number twelve and fourteen, that the contagion of the Black Death and the subsequent epidemics of the Second Pandemic were all in close succession imported to Europe from East (Central) Asia by caravan and ship.

In a mysterious way – there is no other adequate or more academic designation – the authors make Novgorod the centre of the fur trade at the time of the Black Death without any attempt at presenting corroborating evidence linking the city to the alleged Fur Roads alias Spice Roads. According to their map, the Fur Roads end in the Middle East on the south-eastern coast of the Mediterranean, surprisingly at Beirut, as it may seem, and not in the hugely more important city of Acre at the time, and allegedly also continued by land to Alexandria in Egypt, which also is a new view or idea, rather, that is also unsupported by evidence. Additionally this view/idea is surprising since ship transport would be far quicker and cheaper as the close precursor of their map confirms.[19]

The authors attribute to Novgorod an allegedly important role for the export of fur to Lübeck and Hamburg.[20] This assertion is accompanied by an arbitrary assertion, in the central meaning of not supported by evidence, that the fur could hide human fleas and lice containing plague contagion, which reveals the second central epidemiological objective of the fourteenth theory as for the twelfth theory. For support, they refer to Martin's article on the fur trade under the Mongols of the Golden Horde in the thirteenth and fourteenth centuries, in practice the period from the 1240s to the 1340s. Because this article stops in the early 1340s, before the Black Death and at the time trade with Christians was prohibited, it does not and cannot serve as support for their theory,[21] and is a bogus reference. They do not refer for support to Khoroshkevich's standard work on Novgorod's trade in the late Middle Ages. This work does not contain the (Russian) word for fur (*mʲekh*) but the words for hides (*kozha/shkury*) for production of leather, and then, not before 1435 and only in small quantities, without mentioning Lübeck or Hamburg.[22]

18 Ciocîltan 2012: 16, 27, 35–6, 55, 58–9, 68, 88, 95–6, 133, 148, 156.

19 https://upload.wikimedia.org/wikipedia/commons/7/74/Silk_route.jpg.

20 Namouchi, Guellil, Kersten, et al. 2018: E11795.

21 Martin 1978: 401–21.

22 Khoroshevich 1963: 155–8. Among the 16 authors is one historian, S.K. Cohn, who wrote a monograph on the [sixth] alternative theory to the effect that the Black Death and later plague epidemics were a viral disease spread by infected droplets by cross-infection much like influenza. This monograph and theory are really a serious matter that is commented on in over 210 pages in Benedictow 2010: 25–69, 74–7, 144–56, 169–86, 201–68, 314–17, 323–6, 340–80, 386–9, 400–2, 410–15, 664–72, and elsewhere, see Index of Names. Paleobiological studies of 36 burial sites, 12 of them relating to the Black Death, have only produced evidence of the plague bacterium *Y. pestis*. This completely supports the negative outcome of the historical analysis of his theory and the conclusion that it is not written within the perimeter of ordinary scholarly work. The reason that the team of scientists, also paleobiologists, selected a historian who represents this view is difficult to understand, and, as can be seen, he does not provide tenable historical support for their hypotheses.

Given the knowledge of the low prevalence and low levels of plague bacteria in the blood of human plague cases that will be presented in some detail below,[23] it is technically impossible that human fleas or lice have played an epidemic role. These empirical data have been long known.[24]

Recurrence is the essence of plague pandemics. It is the assertion that the contagion of each recurrence of the Second Pandemic originated in Eastern (Central) Asia and was transported by caravan and ship to Europe that is new. In my book on the history of plague in Norway, about 30 waves of plague were identified or indicated in these 300 years.[25] Biraben has shown that, on average, 28 plague epidemics broke out in Europe every year in the plague period 1356–1649, that is, in the period after the Black Death and until quarantine restrictions and other antiepidemic measures began to become effective and affect the course of the pandemic. Importantly, the epidemics broke out at about the same time in highly distant localities.[26] This shows that the Black Death's strain of plague bacteria took root in rat colonies over almost the whole of Europe, established local plague reservoirs where plague circulated, and regularly gave rise to independent contemporaneous plague epidemics in distant localities across Europe. This evidence does not stand alone any longer.

Paleobiological research has shown that the Black Death and later plague epidemics had the same basic genetic structure. In a recent article, a team of leading paleobiologists is clear on the matter. Summing up their findings in the Abstract, they state that 'Our data support an initial entry of the [plague] bacterium from Eastern Europe [the lower Volga region] and the absence of genetic diversity during the Black Death as well as low diversity during local outbreaks thereafter.' Like other paleobiologists, they have identified only one importation of plague to Europe during the Second Pandemic 1346-c. 1690, namely the Black Death. They also comment on the assertions of the authors of the twelfth and fourteenth theories on recurrent importation of plague from Eastern (Central) Asia during the Second Pandemic, and state tersely that 'genetic evidence currently favours' 'local persistence in Europe'. They add: 'This lineage [of plague contagion] descends from the strains associated with the BD […] and, therefore, likely represents plagues' legacy and persistence in or around Europe after 1353.' They also state on genetic grounds that this strain of *Y. pestis* clearly seems to have spread eastwards to China and became the precursor of the Third Plague Pandemic that spread worldwide from China from 1894.[27]

In the first edition of this book, historical evidence was presented to the effect that the Black Death and later plague epidemics of the Second Pandemic spread from the lower Volga Delta or region westwards to Asia Minor and Europe and in the opposite direction, eastwards across the Eurasian continent to China. Historical evidence also strongly indicated that it was

[23] See below, Chapter 4.1–8, pp. 24–31.

[24] For the first time, the data were gathered together from diverse scientific publications and presented in Benedictow 1992/1993/1996: 251–64. These data have been presented again in improved form and together with more recent entomological evidence in Benedictow 2016a: Ch. 12.2–.9, pp. 627–55.

[25] Benedictow 2002.

[26] With respect to the allegation that Biraben's lists of plague epidemics are construed (false) and not based on evidence, Roosen and Curtis January 2018: 103–10, see Benedictow 2019b, *Annales de démographie historique*: 213–23.

[27] Spyrou, Keller, Tukhbatova, et al. 30 November 2018: 1–25, also published in *Nature Communications* 2019: https://doi.org/10.1038/s41467-019-12154-0, pp. 1–13.

the latter wave of plague that established a plague reservoir in the Yunnan province in China, as it seems some time in the seventeenth century, but with a manifest presence from the end of the eighteenth century. This reservoir was the basis of the Third Plague Pandemic that spread from China from 1894.[28] In the present edition, this subject of the spread of plague of the Second Pandemic from the lower Volga region is enlarged upon, especially on the basis of the subsequent paleobiological studies.

Historians and paleobiologists agree. The Black Death originated in the lower Volga Delta or region whence it was spread westwards towards Asia Minor (Turkey) and Europe and eastwards across the Eurasian continent to China.

Like all other alternative theories on the microbial nature and epidemiological dynamics of plague, the fourteenth theory is entirely untenable and invalid.

'Mapping the Black Death'

The meticulous gathering together of all empirical information on the whereabouts of the Black Death in space and time also provides the best possible basis for drawing a map of its spatiotemporal spread as presented in Map 1. The outline of the map in this second edition was little affected by the greatly expanded access to contemporary sources for many countries made available on the Internet since the first edition was written (see below), while increasing the input of independent localization and pursuit of the spread of the Black Death, which comes in addition to the valuable information in local studies. The first drawing of this map was as precise as sources and local studies allowed at the time, and that has proved to be quite accurate.

It has been challenged with respect to central Europe, the then kingdoms of Bohemia (≈ Czechia) and Poland by D.C. Mengel, 'A Plague on Bohemia? Mapping the Black Death', *Past & Present*, No. 211, 2011: 3–34. His article is thoroughly examined below, in Chapters 28 and 29 on the history of the Black Death in the two kingdoms. It is shown that Mengel's challenge is based on the deletion of a crucial part of a central source and neglect or misinterpretation of many other important sources. It is based on flagrant breaches of the tenets of historical methodology, and can be appropriately designated by the most negative word in the vocabulary of the methodolology of historical science. Because I must assume that an article with such extraordinary flaws hardly can be published without the knowledge of editors and possible reviewers, however inconceivable in principle, there was no point in submitting a rebuttal, which has, then, had to wait for the publication of this new and revised edition.[29] In the meantime, the misleading information has been used by some scholars.[30]

Perspectives

In the over twenty years that have passed since the writing of the first edition of this book on the Black Death began in earnest, the study of the Black Death and historical plague epidemics more generally have seen a highly eventful period. It is important to focus on the strong positive developments, especially of palebiology and the easy availability of historical sources

[28] Benedictow 2004: 41–54. This evidence was mainly established by the great pioneer of Chinese plague research Wu Lien-Teh. Wu Lien-Teh 1936a: 10–23.

[29] See also the monographs on alternative theories, Benedictow 2010 and Benedictow 2016a.

[30] Green 2014a: 12, Green 2014b: 36; Carmichael 2014: 160, 175; Roosen and Curtis 2018: 105, 110.

and studies on the Internet. Also many substantial historical contributions have appeared and enriched this new and revised edition. The fundamental arbitrariness of all alternative theories of the microbiological nature or epidemiology of historical plague have been thoroughly disclosed, and they are in rapid retreat. When paleobiologists recognize that they are as ignorant of the study of historical society as historians are of the study of paleobiological material the ground will be cleared for a new and highly fruitful interdisciplinary cooperation that holds rich promise.

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I would also like to express my gratitude to the librarians at Oslo University Library who made such splendid efforts to provide me with copies of papers and books from libraries all over Europe, from the Soviet Union/Russia in the east of Europe to the university libraries in Pamplona and in Madrid in the south-west. They have again provided excellent help for the updating and preparation of this second edition.

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In 1997, the Association of Norwegian Non-fiction Writers and Translators gave me a grant for a three-month project-scholarship that enabled me to concentrate on planning and starting the writing of the manuscript of the first edition of the present book. In April 2003, the Norwegian Research Council gave a grant to cover some of the costs of publication.

The basis of all maps was drawn by the author and finished by the cartographers. All maps are the author's copyright and can only be reproduced with the author's permission.

In recent years, I have had much valuable scholarly contact, informative conversations and discussions with Raffaela Bianucci, the physical anthropologist and paleobiologist, at the University of Turin, and B.J. Hinnebusch, Senior Investigator and Chief of the Plague Section of the Laboratory of Zoonotic Pathogens of Rocky Mountain Laboatories, NIH, NIAID. I am most grateful.

Glossary

Abscess Local inflammation of body tissue with deep suppuration [secretion of pus]caused by bacteria that destroy the cells in the centre of the area and leave a cavity filled with pus

Bacteraemia, bacteraemic Bacteraemia refers to infectious agents or toxins that have invaded the bloodstream. This allows them to spread to almost every organ of the body. Previously, the term septicaemia was used synonymously with bacteraemia. *See also* Primary bacteraemic plague and Secondary bacteraemic plague

Biofilm *see* Blockage

Bleeding from the nose *see* Epitaxis

Blockage and regurgitative transmission Blockage of the foregut/proventriculus of fleas is the only mechanism that can make fleas infective with lethal doses of plague bacteria. Blockage can develop when fleas ingest such highly bacteraemic blood that the bacteria multiply and aggregate faster in the stomach, technically called the midgut or ventriculus, than they are moved into the lower digestive tract. This aggregate of plague bacteria grows into at least a partial block, usually of the foregut, which consists of a gelatinous hemobacillary mass mainly constituted of bacteria and haemin. When a strong stream of blood from a new feed hits a sufficiently developed blockage, it is strongly refluxed or regurgitated back into the bite wound, technically called regurgitative transmission. This reflux or regurgitant of blood takes with it not only the hugely bacteraemic blood present in the pre-blockage area of the foregut (proventriculus) but also bits torn off the blockage, often containing thousands of plague bacteria, which together can constitute lethal doses (LD) for most human beings. A blocked flea is a starving flea desperately trying to obtain a blood meal, making repeated probing attempts, each one potentially resulting in transmission. Blockage is now frequently called a biofilm, a term inherently focusing on its biological character and not on its function in the transmission of plague

Bubo, boil Hard inflamed lymph node that may suppurate; plague bubo. Boil was often used synonymously in the past

Carbuncle Localized dead body tissue (gangrene) caused by plague bacteria (or staphylococci), usually by bacteria left in the site of a flea's bite, in which case it is called a primary carbuncle

Case fatality rate *see* Lethality rate

Case mortality rate *see* Lethality rate

Cutaneous Relating to or affecting the skin. *See also* Subcutaneous, Intradermal

Ecchymosis The passage of blood from ruptured blood vessels into subcutaneous (see below) tissue, marked by a purple discoloration of the skin. *See also* Petechiae

Endemic The sporadic cases of an infectious disease in a human population, too few in number to be considered usefully as a designated epidemic but which show that a

particular type of contagion occurs in a population, are called an endemic phase or situation

Entomology The discipline of natural science that studies insects

Enzootic Sporadic incidence of contagious disease among animals, cf. endemic

Epidemic Disease that spreads rapidly through a population or community for a period

Epidemiology The science of epidemics, especially how epidemics are spread and transmitted

Epitaxis Bleeding from the nose; in plague caused by weakening of local blood vessels by the action of plague toxins

Epizootic Disease spreading among animals, i.e., a term corresponding to the term epidemic among human beings

Etiology The discipline of medicine that deals with the causes or origins of disease

Expectoration Ejection from lung airways by coughing. In cases of pneumonic plague, bloody expectoration may contain plague bacteria from ulcers in the lungs formed by plague bacteria transported there by the bloodstream. Infected droplets from expectoration can be inhaled by other persons and give rise to primary pneumonic plague. *See* Secondary pneumonic and Primary pneumonic plague

Fatality rate *see* Lethality rate

First plague pandemic occurred in the period 541–767. *See* Pandemic

Foregut *see* Proventriculus and Ventriculus

Haemorrhage/haemorrhaging Bleeding. *See also* Petechiae

Incubation period The period from infection to outbreak of disease

Infection dose A measure of virulence usually expressed as ID_{50}, i.e., the number of microorganisms or micrograms of their toxin (see this term) with which human beings (or animals) must be infected in order to cause a morbidity rate of 50%. Cf. Lethal dose

Intradermal Within or between the layers of the skin; intradermal injection of infection, for instance, by flea bite

Lethal dose A measure of virulence usually expressed as LD_{50}, i.e., the number of microorganisms or micrograms of its toxin (see this term) with which human beings (or animals) must be infected in order to kill 50% of them (cause a mortality rate of 50%). *See* Infection dose

Lethality rate The proportion of those who contract a disease and die from it = fatality rate, case mortality rate

Life table Life tables are based on a series of age-specific death rates for each gender and show, therefore, the probabilities of dying within particular age intervals according to various life expectancies at birth. Or, if focusing on the probabilities of surviving, life tables show life expectancies at each age level in societies with various life expectancies at birth

Microbiology The science of studies of microbial organisms, i.e., bacteria, viruses, prions, fungi, etc., particularly genetic material or DNA. Because immune systems generally interact with pathogenic microorganisms, microbiology also includes the study of immune systems and their responses to infections, i.e., immunology

Midgut *see* Ventriculus and Proventriculus

Mixed epidemic Epidemic of bubonic plague including substantial proportions of cases of primary pneumonic plague; occasionally (also) of primary bacteraemic plague

Morbid Diseased state or quality

Morbidity rate Proportion of a population that contracts a specific disease; the rate of incidence or prevalence of a disease; the proportion of sickness of a specific disease in a specified group, locality or community

Mortality The number of people who die within a particular period or at a particular event

Mortality rate The proportion of a population of a locality, social class, gender, age category or occupation, which dies, no matter what the causal factors. Plague mortality is the proportion of a population that dies from this disease in an epidemic

Naïve population Population without previous experience with a disease or diseases, and all members are equally susceptible

Paleobiology The study of ancient DNA (aDNA) or specific proteins reclaimed from biological material of the past taken from remains of human beings or animals. *See* Microbiology

Pandemic (1) An epidemic disease which spread over a wide geographic area, at least most of a continent, or over large parts of the world, and affecting a high proportion of the population (2) Series of wide-reaching waves of epidemics with a long temporal structure. In European history, plague has ravaged populations in two protracted series of waves of epidemics also called pandemics. The first (known) plague pandemic occurred in the period 541–767 CE; the second plague pandemic occurred in the period 1346–ab. 1690s in most of Europe, longer in the possessions of the Ottoman Empire in the Balkans, the Middle East and North Africa. In Russia, it overlapped with the third plague pandemic 1894–c. 1940 (see below). The Black Death is the first gigantic, immensely disastrous and notorious wave of plague epidemics of the second plague pandemic, spreading over almost all Europe, North Africa and the Middle East. It is, therefore, considered a pandemic also according to the first definition. A third plague pandemic broke out in 1894, but was stopped by countermeasures based on modern medicine and epidemiology and had petered out as a pandemic around 1940

Pathogen Micro-organism that can cause disease

Pathogenicity The ability of microorganisms to cause disease, cf. Virulence

Petechiae/plague spots Dark-coloured spots in the skin due to invasion by plague bacteria of the capillary vessels of the skin, i.e., consequent upon the development of bacteraemia in the bloodstream. Plague toxin weakens the walls of the blood vessels that tend to break and leak drops of blood (haemorrhages), which show through the skin as dark-coloured spots, also called plague spots. Contemporary Englishmen often knew such spots as (God's) token, because their appearance heralded imminent and certain death. *See* Ecchymosis

Plague focus/plague reservoir In many areas of the world where wild rodents live in great density, in colonies or otherwise, plague circulates continuously in the rodent population. Such a rodent population is called a plague focus or a plague reservoir

Plague pox/variola Clinical feature of plague patients who have numerous pustules or vesicles which resemble smallpox

Plague reservoir *see* Plague focus

Population mortality rate The mortality rate of an entire population (in contrast to the mortality of social subcategories like social class, gender or age, and so on). *See* mortality rate

Primary bacteraemic plague This form of plague occurs when a flea deposits plague infection directly into a blood vessel or it is passed directly into the bloodstream without stoppage in a lymphatic node (and developing a bubo). Clinically characterized by dramatic and rapid course of illness leading to certain death without the development of bubo(es), because the lymphatic system is not challenged

Primary pneumonic plague Patients with primary lung infections have been infected via the respiratory system. Droplets containing plague bacteria coughed up by persons who have plague infection in the lungs (pneumonic plague) are the source of infection (rarely also animals). *See also* Secondary pneumonic plague and Expectoration

Proventriculus or foregut of fleas, a valve positioned before the stomach proper, the midgut or ventriculus, which allows fleas to make relatively speaking huge intakes of blood, because the valve prevents the blood in the strongly distended ventriculus after a feed from forcing its way back out

Pulmonary plague fulminant type of primary pneumonic plague, i.e., caused by infection via the respiratory tract. Dissection of lungs of such cases does not show pneumonic foci of plague bacteria, which produce a cough with bloody expectoration

Pustules Pustules resemble vesicles and are due to invasion of the skin by plague bacteria through the bloodstream, i.e., they are consequent upon the development of bacteraemia. *See these concepts, and* Petechiae/plague spots

Rat fall Carcass of a rat found in the absence of rodenticide use or obvious injury and could be due to epizootic disease

Regurgitative transmission *see* blockage

Second plague pandemic occurred in the period 1346–c.1690. *See* Pandemic

Secondary bacteraemic plague In 30–40/45% of all cases of bubonic plague, bacteria at some point manage to overwhelm the lymphatic system and pass on into the bloodstream, causing a bacteraemia that is secondary to the primary bubonic condition, cf. Primary bacteraemic plague. These cases are almost invariably mortal

Secondary pneumonic plague In cases of bubonic plague in which plague bacteria pass on into the bloodstream (secondary bacteraemic plague) plague bacteria are transported to the lungs where they quite often consolidate and develop ulcers. These cause a frequent cough with bloody expectoration, a condition that is called secondary pneumonic plague, i.e., a pneumonic condition that is secondary to the primary infection of buboes. These cases are almost invariably mortal. Such cases are the origin of primary pneumonic plague

Sepsis, septicaemic In this book, the terms bacteraemia and bacteraemic are used with the same meaning according to a new consensus on terminological usage. *See* Primary bacteraemic plague, Secondary bacteraemic plague

Subcutaneous Under the skin. A subcutaneous injection is, e.g., an injection in which a needle is inserted just under the skin

Third plague pandemic occurred 1894–c. 1940. *See* Pandemic

Toxin A poison produced by micro-organisms. Each specific type of pathogenic micro-organism produces its own toxin, which causes a specific disease when present in the system of a human or animal body

Vector Carrier of disease, especially an insect that conveys pathogenic organisms from one person or animal to another person or animal

Ventriculus The stomach proper of fleas, also called the midgut. *See also* Proventriculus

Virulence This term is closely related to the term pathogenicity, i.e., the etiological ability of micro-organisms to cause disease, but introduces in addition the concept of degree. This makes it possible to differentiate between the disparate abilities of various pathogenic micro-organisms to produce disease and cause death in infected persons. Virulence is measured in terms of the number of micro-organisms or the micrograms of toxin needed to kill a given host when administered by a certain route. This is called the lethal dose. *See this term*

Part I

What Was the Black Death?

1

The Black Death

1. The name of the Black Death

In the years 1346–1353, a terrible disease swept over Western Asia, the Middle East, northern Africa and Europe, causing catastrophic losses of population everywhere, both in the countryside and in towns and cities.[1] In Florence, the great Renaissance author Francesco Petrarch wrote, dumfounded, to a friend: 'O happy posterity, who will not experience such abysmal woe and will look upon our testimony as a fable.' It wrought such havoc among the populations that it earned, it seems, eternal notoriety as the greatest-ever demographic disaster. Because it was far more mortal and terrible than anything people had heard or read about, the memory of the disaster entered folklore and the writings of the learned alike. Thus, Petrarch erred in his belief that posterity would shrug off the accounts of the havoc it wrought as tall stories.

Many centuries later, Europeans began to call it the Black Death, a name that since has become the usual frightening name of this epic epidemic. The reason for this is probably a misunderstanding, a mistranslation of the Latin expression *atra mors*, in which *atra* may mean both terrible and black.[2] It has nothing to do with clinical symptoms or features, as persons seeking a rational explanation for this graphic term often believe.

However, Simon of Couvin (Simon de Covino), a contemporary Montpellier-trained physician and astrologer working in Paris, wrote an account in Latin classical verse of this disastrous epidemic, where he calls it '*mors nigra*',[3] literally the Black Death. He does not suggest that the diseased developed black colouring of any part of the body. He characterizes the disease clinically by stating that 'burning pain is thence [from the intestines] often born in the groin',[4] evidently referring to the usual femoral–inguinal location of buboes and

1 There is no evidence of wider spread, for instance to India or China or Sub-Saharan Africa, only some speculation. Green 2014a: 13, 23; Varlik 2014: 196–7.

2 D'Irsay 1926: 328–32.

3 Littré 1840–1: 228.

4 Littré 1840–1: 232. My translation from Latin, 'Nascitur inde dolor ignitus in inguine sepe'. Littré renders the whole poem.

their extreme painfulness.[5] However, no contemporary came up with a similar graphic name or was inspired by Simon of Couvin's use, and it remained an isolated case in the late Middle Ages, and much later. It should be seen as an individual poetic inspiration of a metaphor to characterize a disastrous and gruesome disease. The name 'Black Death' emerged episodically in the seventeenth century and slowly gained more frequent usage;[6] in English historiography it was used for the first time in 1823, in Spanish historiography in 1833.[7]

If at all functional as a scholarly term, the Black Death should be used as a specific name of the gigantic outbreak and spread of bubonic plague in Europe, Asia Minor, the Middle East and North Africa in the years 1346–53. It was the most disastrous epidemic in the history of humankind with huge historical repercussions and numerous ramifications, which affected the development of western society. There is no historical evidence that it spread from China or India, but new genetic (paleobiological) findings support historical evidence that it originated in the lower Volga area. The Black Death is the subject of this book.

2. The Black Death (1346–53) and the Second Plague Pandemic (1346–c. 1690)

The use of the term 'pandemic' in historical plague research is not unproblematic, although the usage is taken for granted and the inherent problem is never pointed out and commented on. The conventional epidemiological meaning of pandemic is 'an epidemic disease spreading over a very large area' or 'an epidemic of infectious disease that spread through human populations across a large region, for example, a continent, or even worldwide'. The standard epidemiological use of the concept implies not only large territorial scale but also continuity in time and the notion of a single huge epidemic. In sharp contrast, in historical plague research the term pandemic is used to designate long periods with numerous widespread outbreaks each year that are regularly complemented by huge waves that spread over most of Europe and perhaps also to North Africa and Asia Minor (Turkey). Three historical plague pandemics with this vertical chronological organization and epidemic rhythm have been identified: the First Plague Pandemic of 541 to 767 CE, the Second Plague pandemic of 1346–c. 1690, and the Third Plague Pandemic of 1894–c. 1940. Clearly, this historical usage of the term pandemic differs sharply from general epidemiological usage.

All these three pandemics started with enormous outbreaks that each justify use of the conventional epidemiological term 'pandemic'. This means that it is useful to distinguish these conventional pandemics with a lateral chronological structure from the vertically chronologically structured plague pandemics and designate them with a readily recognizable specific name, such as the Justinianic pandemic in the case of the First Plague Pandemic. This term refers to the Byzantine emperor at the time (541–544), Justinianus I

5 Benedictow 2010: 537–50.

6 Green 2014a: 13; Bulst 1979: 45. Reichborn-Kjennerud claims that the term Black Death emerged in Scandinavian 'Monk verses' ('munkevers') in the second half of the sixteenth century. Reichbhorn-Kjennerud 1948: 359. After having studied his text and references thoroughly, I cannot confirm the tenability of his assertion. Also, unfortunately, this is not the first time, Benedictow 1992/1993/1996: Appendix, pp. 275–84.

7 Shrewsbury 1971: 37; Ruiz de Loizaga 2009: 33.

(527–565 CE).[8] It also makes it clear that it is a misnomer to call the First Plague Pandemic the Justinianic Pandemic, as is often the case, when he can evidently have no personal or even chronological relationship with the plague epidemics in the period 565–767.

The subject of this book is the Black Death, the opening pandemic of the Second Plague Pandemic in the years 1346–53, a suitably graphic name that provides a short and handy verbal reference. It serves no useful purpose to call the Second Plague Pandemic the Black Death, and it is unfortunate also because it deprives the opening pandemic of a readily recognizable specific name.

The Second Plague Pandemic includes a cyclical occurrence of huge waves of plague epidemics spreading over most of Europe and adjacent parts of the Old World breaking out at intervals of on average every 12 years. Tendentially, they are pandemics in their own right, which emphasizes the composite pandemic character of the Second Plague Pandemic.

3. Contemporary notions of contagion and epidemic causation

In the Middle Ages, people knew nothing about bacteria and viruses and other microbiological agents of disease. The great majority believed that disease was God's punishment for their sins. The few physicians and other learned men who knew classical Greek medicine attributed epidemic disease to *miasma*, but quite generally in a technical sense, without doubting that it was unleashed by the Lord to fulfil his will and was caused by his wrath. Miasma was corruption or pollution of the air by noxious vapours containing poisonous elements that were caused by rotting, putrid matter, and which were spread by wind. Miasma could enter persons by inhalation or through the pores of the skin (which revealed their presence as outlets of sweat).

This miasmatic theory of infectious disease can be illustrated by writs from the king of England, who was deeply concerned about the dangers inherent in the unsavoury conditions in London. In 1349, Edward III wrote to the mayor of London and complained about all the filth that was being thrown from the houses so that the streets and lanes through which people had to pass were foul with human faeces and the air of the city was poisoned to the great danger of men passing, especially in this time of infectious disease.[9]

> Edward also sent a writ on the same matter to the mayor and sheriffs of London in 1361. This was in the face of the second wave of plague, and, the writ contained, therefore, an analysis of the cause of plague and royal injunctions as to what should be done:
>
> Because by the killing of great beasts, from whose putrid blood running down the streets and the bowels cast in the Thames, the air in the city is very much corrupted and infected, whence abominable and most filthy stench proceeds, sickness and many other evils have happened to such as have abode in the said city, or have re-

[8] I think it is unfair and much too personal to call the first pandemic the Justinian with reference to the Byzantine (East Roman) Emperor Justinianus I (527–565), when his only relation to the pandemic was to be the reigning emperor at the time of its first outbreak in the years 541–543, and during a few subsequent epidemics. The term Justinianic is not so direct but arguably still unsatisfactory.

[9] Cited after Ziegler 1970: 161.

> sorted to it; and great dangers are feared to fall out for the time to come, unless remedy be presently made against it. We, willing to prevent such dangers, ordain, by consent of the present Parliament, that all bulls, oxen, hogs, and other gross creatures be killed at either Stratford or Knightsbridge.[10]

In the Middle Ages, this miasmatic theory of epidemic disease was supplemented by an astrological dimension. In 1348, King Philip VI of France ordered the Medical Faculty of the University of Paris to produce a report on the causes of the Black Death and to what remedies one could possibly resort. The Faculty reported that at 1 p.m. on 20 March 1345 there was a conjunction of Saturn, Jupiter and Mars in the House of Aquarius. This was extremely potent with regard to the rise of epidemic disease because the conjunction of Saturn and Jupiter gave rise to death and disaster, while the conjunction of Jupiter and Mars disseminated pestilence in the air.[11] In this astrological theory of epidemiology, Jupiter was assumed to be warm and humid and to draw malignant vapours both from the ground and from water, while Mars was assumed to be hot and dry, and, therefore, had the capacity to kindle such malignant vapours into infective fire. Consequently, the rare conjunction of Jupiter, Saturn and Mars together presaged the most terrible epidemic disaster. In Shakespeare's words:

> a planetary plague, when Jove [= Jupiter]
> Will o'er som high-vic'd city, hang his poison
> In the sick air.
>
> when the planets
> In evil mixture to disorder wander,
> What plagues, and what portents, what mutiny.[12]

In addition, medieval miasmatic theory also assumed that volcanic outbreaks and earthquakes freed noxious vapours out of the ground.

Neither was there, according to this theory, much that man could do to prevent or cure epidemic disease, although it contained an encouragement to improve hygienic conditions and to get rid of filth and dirt in the environment in order to prevent matter from rotting and producing miasmatic vapours in the vicinity of human settlement and habitation.

4. Contemporary epidemic notions: pestilence, plague, epidemic

Epidemic diseases were ordinary occurrences in the Middle Ages. Although they could cause significant mortality, they were often ignored by chroniclers or only episodically mentioned as unsuitable events to be noted according to their classical ideals and the traditions of education forming their minds.

Epidemics became increasingly frequent with the strong development of long-distance trade with vital offshoots in coastal, regional and local trade from the port cities of impor-

10 Cited after Gasquet 1908: 110.
11 Sticker 1908: 60–1.
12 Cited after Wilson 1963: 5–6.

tation. From the mid-thirteenth century at the latest, long-term population increase translated into increasing urbanization and rural population pressure and a general scarcity of agricultural resources. It brought about interregional trade of essential commodities but increasingly long-distance importation of the basic necessities of grain and flour.[13] This importation was not only to cover the needs of the urban populations. It also generated the growth of a vast, fine-meshed networks of distribution to small rural localities and settlements with a deficit of calorific production but with other products or produce that could be sold or bartered to balance the chronic deficit of basic food.

Nonetheless, hunger and undernutrition became increasingly usual and food supplies an increasing concern for authorities, from kings to town councils. Trade and transportation were accompanied by invisible and unrecognized enemies of humans, which caused quite frequent outbreaks of epidemic diseases and, in the present context, would function as highways for the spread of the Black Death. This topic will be repeatedly mooted and discussed in local, regional and general epidemiological perspectives below.

Bubonic plague in the form of the Black Death impressed itself on people as an unknown or new disease (some deeply read ecclesiastics quickly associated it with St Sebastian and evoked distant memories of the Justinianic pandemic). Although epidemic diseases exhibited evident different clinical features and impacted on particular sections of the population, for instance children, and obviously produced highly different mortality rates, people did not have a notional tool to explain it. Miasmatic theory was a mono-causal epidemic theory that recognized only one kind of contagion, miasma, as the basic cause of all epidemic diseases. Because they had no notion or theory that could explain that seemingly different epidemic diseases were caused by different contagions, they did not have a real terminology of epidemic disease that provided specific names for specific epidemic diseases. There was, in fact, little appreciation that individual diseases were separable entities before 1550.[14]

Physicians would use the classical Latin words *pestis* or *pestilentia*, in English pestilence or pestilential, with the general meaning of epidemic disease. The blatant specificity of the Black Death and later plague epidemics forced itself on the minds of physicians and people alike. The combination of mind-stretching mortality and several unique or unusual clinical manifestations was obvious, and plague was such an evidently specific disease that the need for a distinguishing term impressed itself upon contemporaries from the very beginning. The Greek word or medical term *epidemios*,[15] 'epidemic', was early employed for specific designation of bubonic plague, with special or implied reference to its unique mortality. The introduction of the Greek word reflected that there was formal medical education, for instance, at the famous Faculty of Medicine in Salerno (Schola Medica Salernitana), at the Faculty of Medicine in Montpellier, and at the Faculty of Medicine in Paris, where prestigious Greek medicine had long been taught according to the *Hippocratic Corpus* (of medical tracts) and Galen's writings where miasmatic theory was originally established and advanced.

13 Spufford 2002: 286–92; Pounds 1974: 394–6.

14 Benedictow 2010: 322.

15 *ἐπιδήμιος*, *epidḗmios*, composed of *ἐπί*, *epí*, meaning 'upon' and *δῆμος*, *dêmos*, meaning 'people'.

5. Bubonic plague: a defining name for plague epidemics

Alternatively, plague disease could be designated by reference to its conspicuous unique clinical feature, the ordinary occurrence of buboes on patients. Because this is a specific feature, it is distinguishing or defining, and all epidemics characterized by buboes are bubonic plague. From the beginning of the Black Death in the Crimea and Constantinople, and as it spread all through Europe, contemporary commentators who give minimal clinical detail mention buboes as the distinguishing clinical feature. This was also the case in connection with subsequent plague epidemics in the following centuries. This clinical feature impressed itself so strongly on contemporaries and distinguished this disease so sharply from all other epidemic diseases that, from the very beginning, contemporaries constructed designations that reflected the appearance of buboes as *the* conspicuous characteristic clinical manifestation.[16] This could be done either as a general reference to buboes, with descriptive specification of usual locations of buboes, or with specific reference to the most usual location of buboes, in or near the groin, in Latin *inguen*, in modern standard works on plague often called femoral–inguinal buboes.

A good example of how buboes impressed themselves on horrified contemporaries and led to the development of generalizing terminology is given by Matteo Villani, the Florentine chronicler, in his account of the Black Death in 1348: the 'mortal disease of buboes' and 'pestilence of buboes':[17]

> in most [of the infected] there were growths in the groin, and with many in the pits under the arms, under the right and the left, with others in other parts of the body, so that almost generally some single swelling manifested itself on the body of the infected.[18]

The main locations of buboes are similar to those observed in modern bubonic plague.[19] This similarity must reflect a similar process of transmission of a similar pathogen.

Villani was far from alone in making these clinical observations.[20] Subsequently, plague disease caused by the Black Death was regularly called by closely related names everywhere, for instance: 'pestis inguinaria'[21] (= inguinal pestilence, inguinal plague), 'mortalité de boces' (=mortality or pestilence of the buboes,[22] Reims 1349), 'sterffde van den droesen' (= mortality of the buboes, Cologne 1350),[23] or characterized by 'dolor ignitus in inguine sepe' (= often a fiery pain in the groin, Paris).[24]

16 Benedictow 2010: 312–80.

17 Matteo Villani, *Cronica* 1995: 1.273, 514, or alternatively Matteo Villani 1729: 622, 653. Benedictow 2010: 324–5.

18 Matteo Villani, *Cronica* 1995: 1. 9: 'e a' più ingrossava l'anguinaia, e a molti sotto le ditella delle braccia a destra e a sinistra, e altari in alter parti del corpo, che quasi generalmente alcuna enfiatura singulare nel corpo infetto si dimostrava'. My translation from contemporary Tuscan. See alternatively Matteo Villani 1729: 12.

19 Choksy 1903: 6, 43–50, 58–60; Chun 1936: 314–15.

20 Benedictow 2010: 323.

21 Sticker 1908: 51, unfootnoted.

22 Desportes 1977: 794.

23 Sticker 1908: 75.

24 Littré 1840–1: 202, 232. Cf. Gasquet 1908: 40–1.

In the subsequent plague epidemics, the use of generalized designations based on this clinical feature continued to be developed, strengthened and familiarized.[25]

Marchionne Stefani's account of the Black Death in Florence is one of the best-known and is cited also below.[26] The process of familiarization with the specificity of the clinical panorama of bubonic plague can be illustrated by his statement on the plague of 1374, which was to him the 'usual pestilence of inguinal or axillary swellings', and in 1383 his comment that the plague killed 'in the same way as the other mortalities, with that sign of great swelling under the arm and over the leg at the groin'. [27]

Another and related development was that bubonic plague was considered such a conspicuously specific disease with respect to its dynamics of spread and mortality that a distinguishing term was needed that let the communicator immediately make clear the kind of diffusive disease (s)he was referring to. From the very beginning, from early in the spread of the Black Death, the term 'epidemic' was employed for the specific designation of bubonic plague in order to distinguish it notionally and terminologically from other contagious diseases, which generally were called 'pestilence' or 'pestilential'. The need for this distinction arose immediately. In the city of Narbonne in southern France, where the Black Death faded away from the end of July 1348 after having raged for five months, the city's heralds announced in the streets that the 'mortality of the buboes that is called epidemic' was over.[28] The formulation was probably made by the city's physician(s) who were educated at the medical faculty in nearby Montpellier and understood that the term epidemic was new to many and must be pedagogically explained. In Paris, physicians called the Black Death 'épidémie'; the chronicler of the *New Normand Chronicle* relates about the outbreak of the Black Death about 24 June 1348 in Rouen: 'and is this disease called "epidemia", in which a certain highly poisonous swelling grew on the throat [neck?] and in the axillas'.[29] The term epidemic is also used by Jean de Venette in his continuation of Gillaume de Nangis' chronicle, both of them closely associated with Parisian monasteries. The Black Death was at the time designated 'epidemia' also in Trier in western Germany.[30]

This terminological development appears in strengthened form when the plague in Hesse and Westphalia in 1371 was characterized by the term *pestilentia epidemiarum*[31] (= the pestilence of epidemics), implying that that plague was considered the essence of epidemic disease, the highest and most dangerous refinement of miasmatic poison. A related term, *pestis epidemialis* (i.e., epidemic pestilence) was used in Thuringia, especially in Frankfurt and Eisenach.[32] The link becomes obvious with the expression *inguinaria pestis seu epidemiae morbus* (= inguinal pestilence or disease of epidemic) used to designate the plague that

25 Benedictow 2010: 322–34.
26 Ch. 38.1: 698.
27 Carmichael 1986: 11.
28 Cayla 1906: 79.
29 *Normanniae nova chronica* 1850: 33. My translation from Latin.
30 Sticker 1908: 60, 67.
31 Sticker 1908: 77.
32 Sticker 1908: 81.

reigned in Wittenberg in 1535.[33] The use of the term epidemic spread rapidly over Europe. The Polish chronicler Kromer stated under the years 1349–50 that 'A severe pestilence, as we say in the vernacular, that the Greeks call epidemics, spread all over Poland [.]'[34]

The English found another solution to the need for a specific designation of the new disease of bubonic plague, taking the word plague slowly into use as an Anglicized form of the Latin word *plaga* with the inherent meaning of strike or blow that originally was a general term for a dangerous disease, a development that was becoming pronounced by the late sixteenth century.[35] In a document of September 1349, Magnus Eriksson, King of Norway and Sweden, used the word *plaga* to designate the Black Death but this usage disappeared later.[36]

33 Sticker 1908: 92.

34 Kromer 1558: 315. My translation from Latin.

35 Slack 1865: 64–5.

36 Benedictow 2002: 22, 96–7. The Icelanders also used the word 'plaga' but retained its original general meaning of pestilence or epidemic disease because plague epidemics did not come to Iceland. The two waves of epidemic disease at the beginning and end of the fifteenth century cannot have been plague. Benedictow 2010: 495–552.

2

The Black Death: The Epidemic Disaster that Made History

1. Historians and the Black Death

The scholarly study of the Black Death began in Europe in the nineteenth century with the development of modern history based on source-criticism and social science. However, most of the research on the Black Death has been performed in the last four decades of the twentieth century. In the first ten to fifteen years of the twenty-first century, progress was impeded by a considerable number of rash and mutually incompatible alternative theories at variance with a huge corpus of good medical and historical research. In my numbering, they are now 14 in all, which have been thoroughly discussed and shown to be untenable for various serious empirical and methodological reasons.[1] On the other hand, the development of the microbiological study of genetic material of the past, the scientific discipline of paleobiology, has made important new contributions and also supported the results of conventional history of plague and society. Everything considered, much new real knowledge is available, and it is possible to present a far wider and more in-depth and also detailed picture than before of this greatest of all epidemic disasters and its demographic, economic, social and cultural effects. In this book, the focus is on epidemiology and demography, the pandemic's powers and patterns of spread and demographic effects.

At first, most scholars rejected the notion that any epidemic disease could have caused an enormous and lasting reduction of European populations. However, in the first half of the twentieth century, historians discovered that the late Middle Ages was actually a period of a dramatic and lasting decline in European populations both in the countryside and in towns and cities. This was first pointed out by Sigvald Hasund, the Norwegian agrarian historian, who, in 1920, published a small book, which he, using a term found in his sources, called (in translation) *On the Great Mortality*.[2] He showed that, in Norway, settlement contracted sharply, land rents plummeted, land prices were halved, and so on, and that this produced a new social scene that

1 Benedictow 2010, 2016a, and above: xxii–xxv.

2 *Ikring mannedauden*. See dedication page.

lasted throughout the late Middle Ages. Summarizing his findings, he pointed out that all these concomitant developments were interrelated and would have to reflect a huge and lasting diminution of the population, and that only the Black Death and a large number of ensuing epidemics of plague could be the cause of a contraction of the population of this order and duration.

Later, in 1935, the German scholar Wilhelm Abel published a more wide-ranging study comprising a much wider geographical perspective and more economic indicators, in which he succeeded in showing the same developments both in Germany and in several other European countries. In 1950, Michael Postan, the English economic historian, published a paper in which he managed to demonstrate definitively that this also was the case in England. Abel's and Postan's works (which were easily accessible in international languages) produced a breakthrough in the study of the late Middle Ages. In the following decades, a great number of studies from almost all countries outside Eastern Europe disclosed the same pattern of developments. For ideological reasons, the contemporary Communist regimes prevented the study of lasting demographic and economic decline and social transformation caused by disease. The new chapters below on the history of the Black Death and ensuing plague epidemics in Poland, Bohemia ('Czech and Slovak Republics') and Hungary confirm that the onslaught of the Black Death and later plague epidemics and the demographic, social and economic effects in these parts of Europe were much the same as elsewhere in Europe.

In addition, a large number of scholarly works have been published that showed that the plague epidemics also had a profound impact on religious and cultural attitudes and their artistic expressions. There was a new obsession with death and the art of dying (*ars moriendi*), a clear shift in the popularity of saints, protector saints against plague like St Rochus and St Sebastian becoming extremely popular. A vow to build a new and fine church was considered a powerful way of alleviating the wrath of the Lord and of preventing the threat of an approaching plague epidemic or reducing its ferocity. Church-building strongly increased and the building industry enjoyed a continuous boom in the late Middle Ages.

It has also been shown that the Black Death and later plague epidemics affected political developments profoundly, primarily because they reduced sharply the incomes kings and noblemen received from their manors and estates. The Hundred Years War, for instance, started in 1337 because of rivalries between the French and English kings, but lasted for more than a hundred years for other reasons: the Black Death and following epidemics caused a dramatic reduction in aristocratic and royal incomes because such a large proportion of tenants had died that rents and fines soon plummeted and land was deserted. War gave rich opportunities for substituting lost agrarian incomes through the wages of war. The knights and squires of the shires enlisted for military service and were awarded with wages, spoils, ransoms for prisoners (especially for aristocratic elite warriors), with manors and fiefs in conquered areas, and so on. The kings used the opportunity to levy new war taxes, which to a large extent ended in the pockets of the warrior class as remuneration for their services or as profits for the merchants who happily sold provisions to armies that were on a quite a constant war footing. Thus, the social elites, the political classes so to speak, had a strong interest in keeping the war going.

In Spain, the Spanish gentry, the 'hidalgoes', had lost much of their income for the same reason, and had enjoyed the alternative opportunity for earning supplementary income by waging more or less continuous warfare against the Muslim Moors on behalf of the Spanish Crown. This is the famous part of Spanish history called the 'Reconquista', the Reconquest. After the final victory in 1492, the Spanish king and queen financed Christopher Columbus'

ambitious plans, which enabled him to sail off westwards to find the sea-route to India and, thus, discovered Meso-America and South America on behalf of the Spanish Crown. Now it was the turn of the American Indians to face the greed, weaponry and martial art of the 'hidalgoes' who earned fame or notoriety under the name of 'conquistadors', i.e., conquerors.

Thus, the arrival of the Black Death heralded a new historical period in which epidemic disease affected profoundly the structure and course of European society, from Norway to Spain, from England to Russia. Later research has shown that, in demographic terms, developments were quite similar in Arab countries in North Africa and the Middle East, and this appears also to be the case in Asia Minor and the Transcaucasian countries.[3] This, then, is the reason the history of the Black Death is important: it made history.

This book has two central objectives: (1) to produce a thorough description of the spread of Black Death across Europe and adjacent parts of the Old World. This will make it possible to identify its characteristic features in interaction with contemporary society and allow inference to the mechanisms of spread. It will also allow identification of the territorial range of spread; (2) to bring together all studies of mortality suitable for estimation of mortality rates, consider them in the light of demography and estimate the mortality effects.

The usual ostensible synthetic estimates of the mortality rate in Europe or in specific countries of one-third or one-fourth or one-fifth or more recently one-half are not based on responsible gathering of source-based mortality studies and are really taken out of thin air.[4]

[3] For an interesting contrast in terms of social effects that emphasizes the historical importance of the decentralized nature of European feudal society (and feudaloid societies), see Borsch 2004: 27–44.

[4] See, e.g., DeWitte 2014: 1, 7, who assumes a Black Death mortality rate of 30–50% with reference to three works where assertions to this effect are stated and have in common that they are unsupported by any reference to mortality data and, as such, are arbitrary. These are also local studies and do not follow the spatiotemporal course of the epidemic or take a general interest in mortality data. This is the reason for the wide spread of the mortality estimate that, across the whole range, is equally free of empirical support and has an equal status of being arbitrary. They are: (1) Poos's 1991 local study of Essex 1350–1525, where mortality in the Black Death is claimed to be one-third. Poos 1991: 2. One should note that the coverage starts after the Black Death in England. (2) Cohn's 2002 book, which I have commented on over at least 210 pages in Benedictow 2010 and have shown to be in scholarly terms an exceptionally unsatisfactory matter, does not provide mortality data; the author flatly rejects that the Black Death was bubonic plague and makes some highly unusual and denigrating attacks on all who support the rat-and-rat-flea-flea basis of bubonic plague. Benedictow 2010: 25–69, 74–7, 144–52, 169–80, 211–68, 289–94, 314–26, 340–80, 386–93, 410–15, 664–72 and elsewhere (see Index, p. 741). (3) This reference is to a problematic article DeWitte co-authored with Wood and Ferrel on mortality among parish priests in an English diocese: see below Ch. 42: 867. At the end of her 2014 article DeWitte refers to the first edition of this book, and although the reference is to another topic it shows that she knows it. One should note that, in this book, the estimate of the mortality caused by the Black Death is based on a synthesis of a presumed complete collection of 190 local mortality rates from many countries, the first time all mortality data have been gathered together (and no one has pointed out missing mortality data). The only estimate of the mortality the Black Death caused that is empirically based on mortality data is studiously avoided. She also insists that the Black Death spread in the years 1347–51, although it has been long known and is well documented in the first edition of this book that it spread in the years 1346–53. See, e.g., also Gottfried 1983: xvi; Bergdolt 2003: 10.

For the first time a general mortality estimate based on the (nearly) complete gathering together of all mortality data on the Black Death was presented in the first edition of this book. Below, this material is supplemented with quite numerous new mortality estimates, especially for England, and a few data that were overlooked then.

Maximum knowledge of the territorial diffusion and level(s) of mortality of the Black Death forms the empirical basis for assessing its historical importance by social and societal impact. This requires that modern scientific knowledge of the disease must be presented, which only can be the gist of an enormous corpus of research. It is important that it gives readers the opportunity to expand their orientation by following references to the established standard works on plague and more recent efforts to supplement them by including more recent research. From amply footnoted standard works they can go on to the primary studies on bubonic plague, all depending on interest and ambition.

This book is called 'The Complete History'. This reflects the author's ambition to provide a complete presentation of its spread, from beginning to end, and a complete and demographically responsible presentation of all data on its mortality, complete, of course, in relation to extant sources and especially available scholarly studies.

2. Why the Black Death became important: some central perspectives

As shown in the first edition of this book and underscored in this edition, the Black Death is a uniquely notorious epidemic because it ravaged a much larger territory and caused much higher mortality than any other epidemic of plague or any other infectious disease in history. For these reasons, its huge social impact and historical significance have become evident. The study of the Black Death has attracted great interest for the opportunity it affords of studying historical societal change caused by an abrupt and immense population fall. Sudden and immense population contraction impacted all important social structures and their interaction in the production of social change and historical development.

The Black Death is also important in the perspective that it was the first and extremely catastrophic epidemic of the Second Plague Pandemic. The ensuing 300 years witnessed numerous, often dramatic and disastrous outbreaks, although no one of them brought about the same, mind-stretching demographic catastrophe. The capacity or dynamic powers of plague to spread had become reduced because it met increasingly efficient human resistance in the form of understanding of epidemic spread and how to prevent it but its capacity to kill its victims, its *virulence*, was unabated. The case mortality rate of plague was the same still in the big epidemic in southern France in 1720–2 and much later in India or China in the Third Plague Pandemic, 1894-c. 1940.[5] Contemporaries lived in constant fear of the next outbreak when their lives once more would be very much at risk.

This reflects that the Black Death established the realm of the Second Plague Pandemic. Almost everywhere the Black Death raged, it left plague contagion among rat colonies that were the basis of numerous contemporary and concomitant outbreaks of plague each year (see below). As underscored by W. Abel, the German agricultural historian:

> As the fourteenth century turns into the fifteenth it becomes more and more difficult to pin down outbreaks of plague. It appears that the great epidemics

[5] Benedictow 1992/1993/1996: 146–9.

> characteristic of the Black Death's early phase petered out into local outbreaks of illness that struck here and there over the whole former area of attack, but with ever less interconnection.[6]

Numerous subsequent plague epidemics gave the abrupt decline in population caused by the Black Death continuity and increasing depth and formed a historical period, the late Middle Ages. This was another crucial legacy of the Black Death that prevented demographic recuperation and formed a historical period, the late Middle Ages, with highly characteristic social, economic and cultural features.

[6] Abel 1980: 88. My bold style. This was written (in German) 10 years before Biraben published the lists of plague epidemics of the Third Pandemic. He registered 2,818 plague epidemics in the period 1346–1500, 8,068 in the period following the Black Death and to the end of continuity of plague epidemics and the Third Pandemic, the period 1356–c. 1690. This means that he registered on the average 18 epidemics each year in the late Middle Ages and 28 plague epidemics each year in the period of the Third Pandemic. Biraben 1975–1: 363–446. See Benedictow 2016a: 62. Cf. Benedictow 2019b: 213–23.

3

The Return of the Black Death and the Response

1. The outbreak of the Third Plague Pandemic in the Yunnan province in China, and early spread

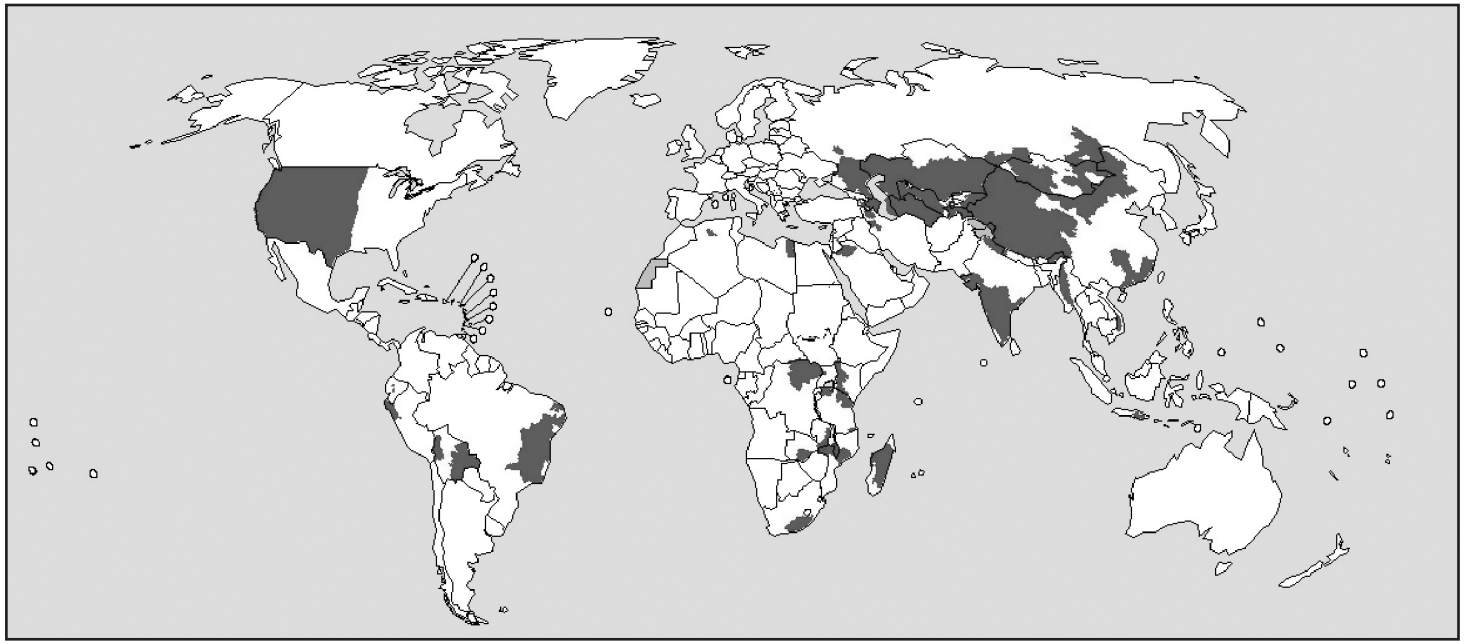
In the 1860s, missionaries and explorers reported from the Yunnan province in China that 'true' plague was observed there. The ensuing events that triggered the Third Plague Pandemic have often been misrepresented and allegedly showed that this plague had highly different properties from the Black Death and the Second Plague Pandemic. It is claimed, among other things, that the spread rates were highly different. For this reason, the events will be presented in some more detail than otherwise would have been necessary in a book on the Black Death. Because, as paleobiologists have shown, the lineage of plague bacteria in Yunnan was closely genetically related to the plague contagion of the Black Death and the Second Plague Pandemic,[1] the disparity of properties would be hard to explain also for this reason (see below).

Yunnan was a remote western province on the border of Burma and near to Tibet and Vietnam. It was 'isolated by its position and its physical features, [and] had only a limited intercourse with its neighbours and with treaty ports'. The main export products were tin and opium.[2] Plague in Yunnan is not mentioned in Chinese sources before 1792. Probably, it was not of long standing but connected with an offshoot of the Second Plague Pandemic in Europe, which is mentioned in Chinese sources in the early 1640s. It was, in fact, a genuine descendant of the Black Death that had struck roots in the Yunnan province.

The crucial event was a sudden upsurge of military activity in response to a Muslim rebellion that broke out in 1855 and entailed a strongly increased traffic of movement of troops, provisions and refugees, and with it the incidence of movement of plague contagion. In 1866, the provincial capital was ravaged by a severe plague epidemic 'in the midst of war'. The following year, returning troops transported plague a long and difficult journey of 1,600 km to the burgeoning coastal commercial hub of Pakhoi on the eastern coast of the Tonkin Gulf, and a serious plague epidemic broke out.

[1] Spyrou, Rezeda, Tukhbatova, et al. 2016: 874–81; Spyrou, Keller, Tukhbatova, et al. 30 November 2018: 1–25.

[2] See, e.g., Simpson 1905: 48–54.



Map 2. Distribution of plague in rodent reservoirs in the world today

It is a misconception that plague spread slowly and steadily from the Yunnan province further into China. Instead, the spread of plague from the Yunnan province started by a gigantic metastatic leap to a new area where, in ensuing years, a plague reservoir was established that also soon expanded eastwards into the Leizhou Peninsula and across the Qiongzhou Strait to the big Hainan Island.[3] Then followed small metastastic leaps to towns and cities as the plague spread out rather spasmodically northwards and eastwards from Pakhoi, breaking out, as it may seem, a few years apart in important urban and administrative centres when they happened to be reported or commented on in some way.[4]

However, it was not before it reached Guangzhou (Canton) in 1894 that it attracted wider attention. It killed between 40,000 and 100,000, probably around 70,000 inhabitants: the collapse of the frail and inept Chinese mandarin organization and the irrelevance of Chinese medicine in the face of epidemic disaster made more accurate estimations impossible.[5] The *lethality rate*, the proportion of those who died after having contracted plague, was 80%,[6] just as in old-time plague epidemics.[7] The Black Death was back in full strength by its true progeny and inheritor of its epidemic might.

The epidemic disaster in Guangzhou awoke the Europeans in China not only to the threat the plague posed to their commercial interests. They also realized that, in the era of steamships and efficient international transport, the plague could easily and unwittingly be spread with cargoes and passengers to Europe and the colonies, which also occurred. When plague broke out in Hong Kong in May, the Europeans were jolted into effective action to counter the threat. By applying the European anti-epidemic measures developed in the period 1500–1700, such as quarantine, isolation of diseased and contaminated areas and establishment of plague hospitals, the British colonial authorities succeeded in keeping the mortality toll down to around 3,000 inhabitants, which should be compared to the carnage in Guangzhou.[8] Shock waves spread through Europe, and the defence of Europe against the plague became a top priority for the big colonial powers that felt most threatened.

Around 1870, there was a great breakthrough in modern, scientific medicine, especially bacteriology. It was discovered that contagious diseases were caused by micro-organisms that were specific for each of them. Many such categories of organisms could be discerned under the conventional microscope at the time. Others were too small but additional techniques were developed to indicate their presence and their causal role in provoking disease (the modern electronic microscope is another matter). Most of them are viruses that cause such well-known diseases as smallpox, chickenpox, measles, mumps, influenza, the common cold, and so on. The pioneers of modern bacteriology gave the name *pathogens* to micro-organisms that caused disease. Inspired by their new capacity to identify these enemies of humankind, which killed many millions of people every year, and especially children, scientists went about their work with tremendous energy and dedication.

3 This plague reservoir near Pakhoi and the subsequent spread is shown on a map, Fig. 71, in Wu Lien-Teh 1936c: 384.

4 Wu Lien-Teh 1936a: 11–19. Cf. *The Plague in India*, 1896, 1897, 1898: 68–70.

5 Wu Lien-Teh 1936a: 20–1.

6 Hirst 1953: 103.

7 See below, Ch. 3.3, pp. 21–2.

8 Hirst 1953: 103.

The discovery of pathogenic micro-organisms also meant that it had become possible to study how the pathogenic agents of diseases were transmitted and disseminated, and, thus, to design more specific, rational strategies to combat them than the old crude (but quite efficient) strategies of quarantine, isolation and cordon sanitaire[9] developed by Europeans in their combat of plague in the period 1500–1700. Bacteriological knowledge could serve as a base for strategies of action: (1) development of vaccines, especially against viral diseases; (2) development of anti-epidemic organizations and countermeasures designed to prevent diseases from being disseminated at a distance, limit its presence to local society, and combat it there with available means. Effective medication was still a distant dream, which began to come true with the development of sulphonamides in the mid-1930s, soon followed by antibiotics.

2. The beginning of scientific plague research and the discovery of the plague bacterium: why the disease was designated bubonic plague

The scientific work to disclose the true nature of the Black Death began in earnest in Hong Kong in 1894 against the backcloth of an early medical revolution in the understanding of the nature of epidemic disease, the scientific medical discipline of bacteriology. Both the Japanese government and the French government dispatched leading bacteriologists to Hong Kong, S. Kitasato and A. Yersin respectively. They arrived in the middle of June and set to work in plague hospitals. They were rewarded with almost instant success. In a few weeks, they identified the same new species of bacterium in the blood and tissues of plague patients, and they also succeeded in isolating and cultivating it in culture. They also found the same bacterium in dead rats, a fact that confirmed suspicions raised by observations of rat falls[10] in the Yunnan province, and in Guangzhou (Canton) and Hong Kong. These two men produced the first important breakthroughs in the modern efforts to combat plague: based on the new bacterial science and medical technology they succeeded in identifying the causal agent of bubonic plague and the genetic progeny of the Black Death, and also uncovered its close association with rats and other rodents.

The plague bacterium was later named after Yersin and called *Yersinia pestis*; Yersin also drew a very important conclusion from his findings on the role of rats in epidemic plague: 'It is probable that the rats constitute the principal vehicle,' and he also incriminated rat fleas as vectors of infection from plague rats to human beings.[11]

The first breakthrough in the bacteriological study of plague did not have much immediate significance for the understanding of how plague was spread and transmitted nor for the practical combating of plague. In the 1894 plague epidemics, as in the plague epidemics of the past, the great majority of the diseased developed one or sometimes two buboes, which distinguished it from all other epidemic diseases. This ordinary form of plague was therefore called bubonic plague.

9 The concept of cordon sanitaire ('sanitary cordon') refers to encirclement of a contaminated area by military forces to prevent people from moving out of it and spread the disease.

10 Rat fall refers to rat carcasses found in the absence of rodenticide use or obvious injury, and which are possibly linked to epizootic disease. For a recent survey in Uganda, see Boegler, Atiku, Enscore et al. 2018: 3.

11 Yersin 1894: 667.

Over a century later, scientists representing the new scientific discipline of paleobiology, the genetic study of biological material from the past, showed that it was actually the direct genetic descendants of the Black Death that had found their way to the Yunnan province and had unleashed the Third Plague Pandemic.[12]

3. The early combating of plague epidemics in India, 1896–1915

The implementation of conventional European anti-epidemic measures on a gigantic scale

In 1896, plague arrived by ship in India. Unsurprisingly, plague fanned out in large epidemics in this poor, populous, underdeveloped country, killing millions of people in the following four decades. Undoubtedly inspired by the success of the British colonial administration in Hong Kong, the British colonial administration in India immediately implemented on a grand scale the conventional European historical anti-plague countermeasures of quarantine regulations, isolation of infected houses or quarters, establishment of plague hospitals, and so on. Without available curative medicine, prophylactic measures to prevent infection and spread were the only efficient means to combat the epidemic and prevent disease and death.

British colonial authorities were also confronted with the very 'unmedieval' problems of mass transportation of people and merchandise by railway and steam ships. On the other hand, they enjoyed important advantages over their European predecessors: a modern mindset that immediately grasped that the new sciences of bacteriology and scientific epidemiology provided a far better framework for integration of empirical observations into a rapidly developing factual understanding of the processes of transmission and dissemination of plague. Of particular importance was the fact that they had at their disposal a vastly superior and larger organization that permitted comprehensive control of travellers and transportation of goods. They could also almost immediately, from 1897, implement mass vaccination, and millions of doses of Haffkine's anti-plague vaccine were administered. Mass vaccination had severe shortcomings, among them that antibacterial vaccines have quite generally only limited temporal protective effect and must be regularly repeated for those at a particular risk of exposure, especially medical personnel and scientists engaged in the study and combating of plague.[13]

The measures were immediately implemented on a truly gigantic scale: in February 1897, six months after plague had appeared, the Municipal Council of Mumbai employed a staff of 30,699 persons in anti-plague work. All travellers leaving infected localities by road or rail were medically inspected, and systematic medical inspection of fugitives from Mumbai by rail was initiated as early as October 1896. When Kolkata (Calcutta) was believed to be threatened, 1,800,000 travellers were medically inspected, 40,000 detained as suspects, and so on.

Mass flight out of these cities when plague broke out was partly a solution, partly a problem to be confronted.[14] This was a triumph of the administrative skills of the British colonial administration that would have been inconceivable in the late Middle Ages. The increasing administrative skills and rational understanding that epidemic diseases were contagious, which

12 Ch. 9.6.

13 Sticker 1910: 438–61. Cf. Simpson 1905: 402–9.

14 Hirst 1953: 115–18, 416–17.

developed in the ensuing first half of the early-modern period, implied that the spread of epidemic disease could be prevented by administrative measures, by preventing contact and movement of infected people or merchandise.

Mass evacuations from plague-infected villages

The new knowledge on plague contagion and the association with rats in a country with a warm climate also pointed to another solution. Strong emphasis was put on immediate mass evacuations of populations of affected villages into so-called health camps in nearby fields on indication of plague cases and occasional rat falls. The efficacy of this measure soon impressed itself on the British authorities, and there never was a single case of plague transmission in these camps.[15]

Because human fleas are nest fleas,[16] which leap onto their hosts to feed at night, they hide near the hosts' sleeping arrangements and are moved with their hosts' bedding while lice move with persons. Because human fleas and lice were moved into the makeshift accommodation in the health camps in the fields, the camps were heavily infested by human ectoparasites. Clearly, they did not play any role in the transmission and spread of epidemic plague. Only persons who slunk back into the village at night to fetch valuables contracted plague. Evidently, the transmission process was strongly associated with the habitations of villages, and for several reasons it was early suspected that it was associated with rats.[17] This was an important observation that required empirical explanation. Also in the cities, relatively poor but gainfully employed working-class families using housing units in tenement buildings called chawls immediately evacuated the buildings in the case of plague deaths or the finding of a dead rat.[18]

There are also historical examples from fifteenth-century Italy and sixteenth-century France of town populations that moved out of their towns and settled in the surrounding areas. There, they built small huts where they could stay until the epidemic could be assumed to be extinguished.[19] This must reflect the same observation of the place or area persistence of plague but without the opportunity to know, understand or explain its basis in rats and fleas.

The effects of anti-epidemic measures

These facts explain that although about 13 million Indians died from plague in the period 1896–1940, the general population mortality rate of plague in India was low, at only a couple of percent.[20] It is a misunderstanding that this shows that the Black Death would have to be a different disease from plague in India at the time because the comparison is false. The anti-epidemic measures implemented by the British colonial administration were mainly those used by early-modern European administrations to successfully eliminate plague from

15 *The Plague in India*, 1896,1897, 1898: 47.

16 Pollitzer and Meyer 1961: 469–70. See below, Ch. 6.3 (and 4.11)

17 See, e.g., Farrar and Oxon 1902: 454–6; Indian Plague Commission 1902: 1218–20; Benedictow 2010: 197–9. Cf. Lamb 1908: i.

18 IPRC, I, 1906: 480–1.

19 Roccatagliata 1976: 210; Favreau 1967: 354–5.

20 See Benedictow 2010: 195–7. Ch. 5 relates to this topic: 'Mortality in India' 2010: 194–204.

early modern Europe. In addition, came the establishment of basic modern health services, such as 2,500 hospitals, and a nationwide modern sanitary organization according to the then prevailing European model.[21]

The mortality effects of plague under the British colonial administration in India should be compared with the efforts of European authorities to combat plague in the period about 1525–1650. They succeeded in limiting the outbreaks and spread of plague epidemics and reduced their mortality effects to a level that allowed new population growth. In the second half of the seventeenth century, they succeeded in eradicating plague (except in the two non-modernized states of Russia and the Ottoman Empire). These two historical situations are comparable, while the comparison with European society at the time of the Black Death is invalid and produces erroneous notions. As stated by George Lamb, the Director of the Indian Plague Research Commission, in the early days of plague in India, most strenuous efforts were made to stamp it out by the means adopted in European countries in dealing with epidemic disease.[22]

The Indian Plague Research Commission

The British colonial authorities recognized that only a good scientific understanding of the processes of transmission and dissemination of plague could provide efficient means for combating the epidemic with administrative means. Shortly after the arrival of plague in Mumbai, the Indian Plague Commission was constituted by the parliament in London as a fact-finding commission, which presented several voluminous reports to parliament.[23]

In 1905, the Indian Plague Commission was replaced by the Indian Plague Research Commission (IPRC) with a brief to carry out a comprehensive research programme on epidemic plague. Some of Britain's best medical scholars joined it. In the meantime, the British colonial authorities had financed a contemporary state-of-the-art plague laboratory in Parel village on the outskirts of Mumbai, the first custom-built plague laboratory ever and for many years to come. The Commission's work was lavishly funded, which allowed it to organize large-scale projects. The attention was from the beginning primarily focused on three fields of study: (1) the epizootic spread of plague among rats, (2) the precise relationship of the epizootic to the epidemic, and (3) the modes by which the disease may be communicated from rat to man.[24]

In ten years of operation, IPRC produced 82 reports containing nearly 2,000 pages of primary pioneering research on plague, which included numerous important discoveries and clarifications.[25] These reports still represent the basis of plague research today and are frequently referred to in scholarly works on plague in recent years.

In the following decades, other research teams studied epidemic plague elsewhere, particularly the French research team in Madagascar and Dutch researchers in Java. These were

21 Mushtaq 2009: 6–14.

22 Lamb 1908: i.

23 E.g, in 1901: *Report of the Indian Plague Commission with Appendices and Summary, 5 volumes, Presented to both Houses of Parliament by Command of His Majesty*.

24 IPRC 1906a: 423.

25 IPRC's 82 pioneering studies or reports were published in *The Journal of Hygiene* in the years 1906–15 (comprising also two Plague Supplements in 1912), containing 1,859 pages. To this corpus of research must also be included George Lamb's, the director's, succinct digest (94 pp.) of IPRC's studies up to May 1907.

the principal outlets for the study of epidemic plague before American army physicians were confronted with large plague epidemics during the Vietnam War. These plague research teams studied the same disease and reached the same main conclusions that quite generally were concordant with the findings of the IPRC: bubonic plague mainly is a black-rat-based disease transmitted and disseminated by rat fleas.[26]

[26] Hirst 1953; Pollitzer 1954; Wu, Chun, Pollitzer, et al. 1936.

4

Transmission of Lethal Doses of Bacteria in Bubonic Plague

1. Introduction

Plague has been intensively studied. These efforts have produced a vast amount of microbial, medical and epidemiological research. Most of it is synthesized in outstanding standard works on plague. Here, only the outline and central findings of this research can be presented and updated. This presentation is amply footnoted to permit and encourage further reading.

2. Lethal doses of plague bacteria

The basic conditions of plague mortality must be considered. This study can usefully begin with the number of plague bacteria which, transmitted to humans, will constitute a lethal (mortal) dose because it clears the ground for other basic questions relating to sources of infection and mechanisms of epidemic transmission. Infectious doses of bacteria are designated ID, lethal doses LD, for, for instance, 50% of an infected normal population LD_{50}.

For evident ethical reasons, experiments cannot be performed on humans to ascertain LDs of plague infection. Because monkeys have proven unsuitable,[1] important clues must be based on rodent studies, and, unfortunately, these are few. Importantly, 90% of black rats (*Rattus rattus*) survived primary subcutaneous inoculation with 5,550 plague bacteria (= LD_{10}),[2] LD_{50} would obviously require a substantially higher infectious dose. The LD_{50} of a species of California ground squirrels that generally is considered highly susceptible to plague, is 6,070 bacteria.[3] These data have limited relevance for the question of LD_{50} for human beings, except indicating the minimum infection that could result in this mortality rate. Humans are, apparently, quite susceptible but not highly susceptible to plague infection. This is also reflected in the fact that 20% of plague diseased in normal populations in early

1 Pollitzer 1954: 187; Meyer, Quan, McCrumb, et al. 1952: 1229.
2 Chen and Meyer 1974: S664.
3 Williams, Moussa, and Cavanaugh 1979: 619.

developing countries and in historical societies survived (Benedictow 1992/1993/1996: 146–9). Arguably, the use of ~6000 *Y. pestis* as indication of LD_{50} for a normal population in developing countries or in historical societies is a cautious choice of analytical tool.

3. The size of blood meals ingested by fleas and by lice

This clarification of LDs of plague infection for humans immediately raises questions relating to humans and rats as sources of infection with plague bacteria.

1. It is often mechanically and arbitrarily assumed that (black) rats and humans function about equally as sources of infection of feeding ectoparasites.[4] In methodological principle, one must consider that the prevalence and level of plague bacteraemia, i.e., of plague bacteria in the bloodstream of plague-diseased rats and humans, could be very different, implying that plague-diseased rats and humans could function highly differently as sources of infection of feeding fleas and lice. This has long been known (see below).
2. The size of blood meals ingested by feeding fleas and lice would decide the number of plague bacteria that would follow the feed into the gut of the insects and constitute the basis of transmissions of bacterial loads.

It was early ascertained that the midgut (ventriculus) of normal female rat fleas could accommodate about 0.5 μL (microlitre = mm^3).[5] Because fleas probably did not fill up at full potential capacity a more accurate measure would be the actual size of blood meals. Recent studies have shown that the average blood meal imbibed by female rat fleas (*X. cheopis*) is 0.41 μL = mm^3 and a much smaller figure by males, at 0.18 μL. A preliminary study of blood meals of female human fleas (*P. irritans*) was smaller, at 0.32 μL.[6]

The rat-flea blood meal will be used as a standard measurement for human female fleas to ensure an adequate margin of error. Then, these data show that female fleas must ingest human blood containing ~2440 bacteria/mL (millilitre = cm^3) to become infected by 1 plague bacterium and 6,000 x 2,440 = 14.6 million or 1.46 x 10^7 bacteria/mL (millilitre = cm^3) to become infected by a (presumed) LD_{50} for humans; a male flea 33.3 million or 3.3 x 10^7 bacteria/mL. These are the infection-level divisors or conversion factors for estimation of infection doses ingested by fleas according to level of bacteraemia in hosts. These data must be compared with data on bacteraemia in plague-diseased humans and rats to furnish insights in the potential for transmission of infective doses.

The same analysis can be performed with respect to lice. Because there tend to be more females than males in a normal lice population,[7] it seems reasonable to suppose that 60% were females. An adult female louse ingests on the average 0.0001579 mL = 0.1579 μL/mm3 per blood meal, an adult male 0.0000657 ml, and lice take on the average three feeds per day.[8]

[4] Walløe 2008: 59–73; Dean 2015: i, 1–56; Bramanti, Stenseth, Walløe, et al. 2016: 13–16; Dean, Krauer, Walløe, et al. 2018: 1304–9; Dean, Krauer, and Schmid 2019: 1–6; Bolton 2013: 29–31.

[5] IPRC, XV, 1907: 396–7.

[6] Personal communications to O.J. Benedictow from B. Joseph Hinnebusch, Chief of Plague Section, Rocky Mountain Laboratories, NIAID, by emails of 9 June 2015 and 16 April 2016.

[7] http://www.cdc.gov/parasites/lice.

[8] Speare, Canyon, and Melrose 2006: 543–5.

This means that 50 adult lice, distributed as 30 females and 20 males, imbibe, on average, 0.01815 mL = ~0.018 mL or ~18 μL of blood/day. For practical or pedagogical reasons, the estimate will take as a point of departure that a level of bacteraemia of 1,000 *Y. pestis*/mL of blood implies that each μL of blood will contain, on average, 1 plague bacterium. This implies that a batch of 50 lice with the presumed distribution according to sex will, in 24 hours, altogether ingest 18 plague bacteria or that up to 18 of the 50 lice would be infected by 1 plague bacterium.

According to the feeding capacity of female and male lice, a specimen must ingest blood with levels of bacteraemia of ~6,300 and ~15,200 *Y. pestis*/mL respectively, to take in 1 bacterium, which is the infection gauge or infection-level divisor of female and male lice, respectively. To ingest a LD_{50} for humans, a female or male louse must ingest blood containing respectively 37.9 mill ~3.79 x 10^7 per mL/blood or 91.1 mill = 91.1 x 10^7 per mL/ blood.[9]

Juxtaposed with data on human plague bacteraemia, these data explain that humans cannot serve as sources of infection of feeding human fleas and lice that could give them a (significant) role in the transmission and dissemination of bubonic plague.

4. Prevalence of plague bacteraemia in humans

Types of human plague bacteraemia

The notion that human fleas can assume an important or predominant role in the transmission and dissemination of plague epidemics implies several strong assumptions with respect to the prevalence and level of plague bacteraemia in human plague cases and their efficiency as sources of infection of feeding fleas and or lice, which quite generally are ignored. In the case of humans, there are three forms of plague clinically characterized by plague bacteria in the bloodstream, technically called bacteraemia, which will decide the potential significance of inter-human cross-infection by so-called human fleas and or lice.

Primary bacteraemic plague arises when (blocked/infective) rat fleas regurgitate a bacterial load directly into a blood vessel and the bloodstream. The plague bacteria in the regurgitant will rapidly proliferate in the bloodstream and produce high levels of bacteraemia (see below). Studies on representative samples admitted at hospital in Mumbai around 1900 showed that about 3% were primary bacteraemic cases.[10]

Primary bubonic plague is the most common modality of plague. It occurs when infective fleas deposit bacterial loads into the intradermal level of the skin from where they are drained along lymphatic tracts to lymph nodes, which react by swelling into buboes. Basically, this is a non-bacteraemic form of plague but frequently translates into bacteraemic plague.

Secondary bacteraemic plague is caused by high concentration of bacterial toxins in the primary buboes associated with heavy infections, which causes attrition of the walls leading to haemorrhagic infiltration of the neighbouring veins and subsequent passage of plague bacteria into the bloodstream.[11]

Secondary pneumonic plague: In cases of secondary bacteraemic plague, bacteria are transported by the bloodstream to the lungs where they may consolidate and cause *secondary pneumonic plague*. The designation reflects that this form of plague is secondary (or tertiary

9 Benedictow 2016: 604–5.

10 Choksy 1903: 30, 58.

11 Pollitzer 1954: 205–6. See below, subch. 4.5, pp. 28–31.

rather) to the primary bubonic infection and subsequent bacteraemic developments. The consolidation of plague bacteria in the lungs may cause ulceration and a consequent cough with bloody expectoration. This occurs in 10–25% of all bubonic plague cases.[12]

Primary pneumonic plague: Cases with secondary pneumonic plague often develop a cough with bloody expectoration and droplets containing plague bacteria, which can infect by inhalation. This modality of plague is called *primary pneumonic plague*[13] because the disease originates by infection of the patients' lungs.[14] Cases are always bacteraemic because the infection is distributed from the lungs to the bloodstream. The usual proportion of such cases in epidemics of bubonic plague seems to be about 2.5%.[15] In some cultures the proportion is higher because of special social customs and conventions in relation to disease that cause much closeness and comforting intimacy.[16]

The prevalence of bacteraemia in humans

Early data on plague bacteraemia in humans are important. Because there was no medication that could be applied and affect the level of bacteraemia and the course of disease, the data show the natural prevalence and levels of bacteraemia, presumably much as in historical society. Such data were produced in plague hospitals in Mumbai around 1900.

The first and by far largest studies of the prevalence of plague bacteraemia among human cases were performed at two hospitals in Mumbai in 1901 and 1902: the blood of 411 and 1,014 native patients was examined by culture on admission, of whom 43.2% were bacteraemic.[17] A study of 433 patients showed that one-half of them were admitted to hospital on the 2nd and 3rd day of illness, in about 80% of all cases ended by death 3–5 days after the onset of disease or 1–2 days after admission to hospital. Blood was withdrawn from some of these patients later in the course of illness but unsystematically or sporadically 'on account of pressure of work'. The subsequent withdrawals and examinations of blood did not uncover cases that were non-bacteraemic on admission and later developed bacteraemia. The course of disease in these patients seems to be quite representative and therefore presumably was the prevalence of bacteraemia.[18]

Later studies of the prevalence of bacteraemia among 298 native plague cases in studies performed during the Vietnam War showed that only 26.5% were bacteraemic (see Table 2 below) (American military personnel were routinely vaccinated). Because native patients arrived at hospital late in the course of plague illness, the phase of the disease was probably much the same as in India or rather more advanced, which implies that subsequent developments of bacteraemia would not occur or only rarely. Patients were medicated immediately after withdrawal of blood and subsequent withdrawals were not performed.

12 Hirst 1953: 22. See below, Ch. 5.4: 41.

13 The indispensable main work on primary pneumonic plague is Wu Lien-Teh's 1926 monograph. It can be somewhat updated by taking into account a number of subsequent articles. Benedictow 2010: 493–552.

14 Wu Lien-Teh 1926; 168, 174–6; Pollitzer 1954: 207–8; Benedictow 1992/1993/1996: 25, and n. 34; Benedictow 2010: 8.

15 Choksy 1903: 60.

16 See below, Ch. 5.2: 40–1.

17 Choksy 1903: 165, asterisked n.

18 Choksy 1903: 15, 29, 32–4, 39, 60–1. Cf. Benedictow 2016a: 640–2.

Summing up, studies of human plague bacteraemia on huge or substantial numbers of plague cases indicate that this condition only occurred in 43% or 26.5% of all cases or, conversely, that 57% or 73.5% were non-bacteraemic during the course of illness and would not infect a feeding ectoparasite. These data show that a high proportion of bubonic plague patients would normally not develop bacteraemia before death and indicate that levels of bacteraemia in those who did would be low. The latter consideration was an important hypothesis to be tested and clarified.

5. Levels of plague bacteraemia in humans and their function as sources of infection of fleas and lice

IPRC studied bacteraemia in a sample of 28 patients from admission to a plague hospital in Mumbai to the end of illness by death or recovery.[19] Native patients tended to be admitted to hospital late in the course of illness. In this study, the first sample of blood was on the average withdrawn 42.6 hours (~1.75 days) after the onset of illness.[20]

Table 1. Prevalence and levels of bacteraemia in 28 plague patients, Mumbai, 1906

No. of patients	Survived/died	Bacteria per mL
7	0/7	0
5	5/0	0
2	0/2	1–10
3	0/3	10–100
1	0/1	>100
1	0/1	>200
4	0/4	10^2–10^3
1	0/1	10^3–10^4
3	0/3	>10^4
1	0/1	>10^6
28	5/23	

This study provided the best data on human plague bacteraemia. It is the only study of its kind performed long before medication was available but is affected by the relatively small number of cases and considerations with respect to the element of subjectivity involved in the process of selection and the representativeness of the gravity of illness of patients brought to hospital. The patients were followed through the course of illness unaffected by medication; repeated withdrawals and examination of blood from the same patient could be made, in 82% of the cases to the time of death. The slightly higher case fatality rate compared with earlier mass studies suggests that the severity of plague illness and prevalence of bacteraemia among the patients were slightly above average.

19 IPRC, IX, 1906: 524–9.
20 IPRC, IX, 1906: 527.

The observations made by IPRC are rendered in Table 1. This material shows that 12 of the patients (42.9%) did not develop bacteraemia, although a few cases had a slight transient bacteraemia that disappeared; 16 cases (57.1%) were bacteraemic, two of them barely detectable. This is a significantly higher proportion of bacteraemic cases than recorded in the mass studies of the prevalence of bacteraemia performed at Mumbai hospitals a few years earlier and tends to confirm the inference from the lethality rate that the selection process had produced a group with an above-average severity of illness and prevalence of bacteraemia. It is also considerably higher than found in American studies on plague patients during the Vietnam War. The single statistical outlier with a level of bacteraemia of $>10^6$ represents probably a case of primary bacteraemic plague, which in the Indian mass studies of patients arriving at plague hospitals represented about 3% of the cases and should accordingly be represented by one case in this material.

Twelve of the patients did not develop bacteraemia, and in 11 of the 16 bacteraemic cases levels were slight. Conspicuously, at most 5 of the patients would infect feeding fleas at all, 4 with 1–5 and 1 with > 410 bacteria.

These data show within huge margins of error that no one of these patients would be the source of a lethal dose of plague for feeding human fleas and lice.

Quite large plague epidemics raged during the Vietnam War with about 4,500–5,600 recorded cases in the three peak years of 1965–7.[21] This allowed also studies of the prevalence and levels of bacteraemia in blood withdrawn from patients on arrival at hospitals, immediately before efficient antimicrobial medication was administered. These patients strongly tended to arrive late in the course of illness.[22] According to the Indian experience, significant bacteraemic developments would not occur subsequently. The registrations should be representative with respect to prevalence and level of bacteraemia.

Table 2. Prevalence and levels of bacteraemia in human plague cases in Vietnam around 1970

Paper no.	No. of patients	Bacilli in blood	Bacteraemic %
1	161	38	23.6
2	10	5	50.0
3	40	5	12.5
4	22	6	27.0
5	23	8	34.0
6	42	17	40.5
Total	298	79	26.5

21 Marshall, Joy, Ai, et al. 1967: 604–5, 612–13; Butler, Bell, Linh, et al. 1974: S78.

22 Benedictow 2016: 644–7.

Six articles provide data on bacteraemia in 298 native patients[23] of whom 79 or 26.5% exhibited bacteraemia – see Table 2. This means that about 73.5% would be non-bacteraemic through the whole course of illness and would not infect a feeding flea by a single bacterium. As will be seen below, many bacteraemic plague patients had very low levels of bacteraemia, far below ~2440 bacteria mL of blood, and would (highly likely) not infect a feeding flea, according to the infection-level divisors. Two studies (nos. 5–6) comprising 65 cases of whom 40 were non-bacteraemic

The second study (paper no. 6) included 17 bacteraemic cases. Among these cases, 6 had <1 to 10^2 *Y. pestis*/mL of blood, 11 had >10^2. However, 4 had the relatively speaking extreme level of bacteraemia of >10^6, and 1 exceptional, extreme outlier had 4 x 10^7, about 40 times higher than the second highest level of bacteraemia recorded. The wide empty range between the two delimitations, i.e., between >10^2 and >10^6, produces gross uncertainty about the 5 cases with unspecified levels of bacteraemia. These data show that at least 7 of the bacteraemic cases would not infect feeding fleas, increasing the number of such patients to at least 32 or a proportion of 76%. Using the infection gauges differentiated according to the sex of fleas, 5 patients would infect feeding female fleas with 0->~160/>410 *Y. pestis*, while the exceptional case or extreme outlier would infect feeding fleas according to sex with 16,500/7,200 *Y. pestis*. Because bacteraemia was not determined in 5 cases, the sample size was 37 patients of whom at least 32, or 86.5%, would not infect feeding fleas before death.

A female flea feeding on the patient with a level of bacteraemia of 4 x 10^7 bacteria/mL would ingest 16,500 plague bacteria, which is much higher than the cautiously indicated LD_{50} for human beings. Also male fleas would according to the selected criteria ingest a LD_{50} for humans (if transmitted). The epidemiological significance of this single case is probably tiny or minuscule rather. The level of bacteremia in this unique case is 35–40 times higher than in any other examined case of plague bacteraemia. Among the Vietnamese cases, 219 were non-bacteraemic, and of the 79 bacteraemic cases 25 had been examined for level of bacteremia; only one of 244 Vietnamese cases would infect feeding human fleas with a LD_{50} or 0.41%.[24] If the 28 plague cases examined for level of bacteraemia in Mumbai (see Table 1) are added to form the complete material, this proportion sinks to 0.37% (1 out of 272).

The 272 cases with known level of bacteraemia including non-bacteraemic cases included 6 cases with 1.5 x 10^6–10^6 bacteria/mL and one case with 4 x 10^7 bacteria/mL. These 7 cases constituted 2.6% of the material, quite close to the about 3% of cases of primary bacteraemic plague recorded at plague hospitals in Mumbai.

The average blood meal ingested by a female human louse is 2.6 times smaller or 38.5% of the average blood meal ingested by a female human flea and would be correspondingly less infected per meal, but would have three feeding opportunities per 24 hours. Only a female louse feeding on the extreme outlier would ingest a (presumed) LD_{50} of plague bacteria for humans but only just; a male louse would ingest about 41.5% of a LD_{50} or 2,500 bacteria.

This conclusion includes primary pneumonic plague, which so far has not been represented by concrete data. Studies of the two by far largest known epidemics of primary pneumonic plague, the Manchurian epidemics of 1910–11 and 1921–2 that comprised nearly 70,000

[23] Marshall, Joy, Ai, et al. 1967: 605, 604, 610, 612; Legters, Cottingham, and Hunter 1970: 640; Butler 1972: 269; Butler, Bell, Linh, et al. 1974: S78; Cantey 1974: 281; Butler, Levin, Linh, et al. 1976: 493-9.

[24] As for bacterial growth in the foregut and midgut, see below, Ch. 4.6.

cases,[25] uncovered only one case of bubonic plague, which probably had been transmitted by a human flea.[26] Because much housing swarmed with human ectoparasites[27] this single case presumably reflects the correlation between normal high levels of primary pneumonic bacteraemia in humans and the role of human fleas. Human lice were not incriminated in any case.

Importantly, the data implies that transmission of IDs of 2–3 x 10^3 plague bacteria would be a subinfective dose of bacteria, too small to cause disease or only cause light disease and be successfully combated by the immune apparatus. This would produce immunization effects that attenuate epidemic transmission.[28] Almost all fleas feeding on the registered cases of human bacteraemia would be infected by numbers of bacteria that would, if transmitted to other humans, serve to increase temporarily the prevalence of immunity against plague infection in a population.[29]

That would probably not happen. Fleas that have fed immediately begin to digest the blood meal. Within 24 hours, the meal is processed by passing into the hindgut.[30] This part of the digestive process has also the function of autopurification of plague infection. With the exception of the extreme outlier, the fleas that had ingested >10^6 would only have a small fraction of the originally ingested bacteria in the midgut at the time of the next blood meal while, probably, all other fleas that had ingested some plague bacteria would be infection-free. This material indicates that around 90–95% of all individually examined Vietnamese plague cases would not infect feeding fleas, nearly all which became infected would ingest a low number of *Y. pestis* and probably have freed themselves at the time of the next feed while those that remained infected would only contain tiny numbers of bacteria.

Taken together, all data on human plague bacteraemia show that, within huge margins of error, the prevalence and levels are far too low to enable feeding human fleas or lice to transmit plague, except exceedingly rarely. Human plague cases do not have a role in the epidemiology of plague. The basis of epidemic transmission of plague must be entirely different. This is the reason that studies of the latency in plague epidemics consistently show a duration of 19–27 days, which reflects the temporal structure of rat-and-rat-flea-borne plague.

6. Prevalence and levels of plague bacteraemia in rats: rats as sources of infection of feeding fleas and lice

IPRC is the only research team to have studied plague bacteraemia in black rats.[31] Among a selection of 32 healthy rats, 23 were inoculated cutaneously (in the skin) with materials from fresh spleens of rats dead from acute plague, and 9 were inoculated subcutaneously (under the skin) with cultures of virulent plague bacteria. The IPRC wished to ascertain the level of bacteraemia before death to avoid unforeseeable changes produced by death. For this reason, as a control measure, 13 and 7 respectively of these rats were euthanized in an apparently moribund state, in the terminal or agonal phase of the disease, 2–4 days after inoculation, on average after 3.25 days, while 10 and 2 rats, respectively, were allowed to die naturally, which

25 Wu Lien-Teh 1926: 71, 81.
26 Wu Lien-Teh 1926: 185–7.
27 Wu Lien-Teh 1913–14: 248–50.
28 Legters, Cottingham, and Hunter 1970: 649–50.
29 See above, Ch. 3.4.
30 Hinnebusch, Jarrett, and Bland 2017: 216, 218.
31 IPRC, VIII, 1906: 519–23.

occurred, on average, 3.66 days after inoculation or 9.8 hours later than the euthanized specimens. This uncovered the important fact that short periods of incubation and illness are crucial factors in the rapid development of plague epizootics in local rat populations.[32]

Table 3. Prevalence and levels of plague bacteremia in black rats, Mumbai, 1906

No. of rats	Killed/died	Bacteria/mL of blood
3	3/1	<10
3	3/0	10–10^2
3	2/1	10^2–10^3
1	0/1	10^3–10^4
7	4/3	10^4–10^5
4	3/1	10^5–10^6
1	0/1	10^6–10^7
4	2/2	10^7–10^8
6	4/2	10^8–10^9
32	20/12	

Table 3 shows that, quite likely all but at least 29 of the rats, or at least 90.6%, exhibited bacteraemia, frequently at very high or extreme levels.[33] On average, 2.9 days after inoculation, 4 rats exhibited 10^7–10^8 Y. pestis/mL of blood, another 4 at least 10^8, and 2 cases 10^8–10^9. Tendentially, bacteraemia was highest in the rats inoculated subcutaneously, despite that 7 of 9 were killed, on average, 2.6 days after inoculation; bacteraemia was similar for the 2 specimens of this group that died naturally from the infection.

Plague bacteraemia in this cohort of rats was nearly 1,000 times higher than in the cohort of humans, that is, as a comparison between the rats and humans that exhibited bacteraemia and thus are comparable. Because a majority or a large majority of humans was non-bacteraemic, bacteraemia among rats was, in reality, relatively even much higher. Extreme levels of bacteraemia developed early in the course of illness of the rats.[34]

Clearly, IPRC held the opinion that they had observed a natural representation of bacteraemia in rats: the experiment showed that fleas ingesting the blood of most rats shortly before death would be highly or grossly infected and would have ample opportunity of doing that in the course of a plague epizootic among rats. Comparatively, these two sets of data show, as mentioned, that rats are hugely better sources of plague infection of fleas than human beings.

7. Mechanism of transmission by fleas: the function of blockage by biofilm

IPRC had clarified that human plague cases only exceptionally could be the source of lethal doses or heavy infection of feeding ectoparasites. This was a sufficient condition or reason for concluding that human fleas and lice did not have a role in the transmission and dissemination

32 Benedictow 2010: Appendix 3.
33 IPRC, VIII, 1906: 521–2, and n. 2.
34 Benedictow 2010: 652–3.

of plague in epidemics. They had also clarified that black rats were the animals living in close connection with humans, in human habitation or environments, that generally developed plague bacteraemia and frequently at very high or extreme levels per mL of blood. They had also observed that black-rat fleas (*X. cheopis*) were efficient transmitters of plague and, in the absence of their preferred host, also leapt onto humans to feed.

However, a crucial question remained unresolved: how were lethal doses of plague bacteria transmitted? IPRC had early made preliminary evaluations of several possible mechanisms including contaminated flea faeces deposited on the skin and rubbed into the bite wound but the solutions were not supported by experiments, which was confirmed by other researchers later.[35] The option that plague infection could stream back into the bite wound from the gut against the stream of blood of the next feed was considered but could not be explained.

The conundrum was resolved by entomologists of IPRC at the end of its operation.[36] Transmission of lethal doses of plague bacteria requires the development of a peculiar condition called blockage caused by the growth of a biofilm. This condition is closely related to a special feature of fleas' anatomy. The stomach/midgut of fleas is equipped with a foregut with a valve function that allows fleas to ingest relatively large blood meals because it prevents blood from the strongly distended midgut forcing its way back. Biofilm can develop when fleas ingest such highly bacteraemic blood that the bacteria multiply faster than they are digested and moved into the hindgut (lower digestive tract). The foregut is the primary site of colonization by plague bacteria. Bacterial growth and the digestive processing of blood produce a biofilm[37] consisting of a gelatinous haemobacillary mass (of blood and bacteria)[38] forming a partial or complete blockage of the foregut, which impairs the valve function and prevents new feeds from entering the midgut. In rat fleas, formation of biofilm starts with ingestion of about 3,800 plague bacteria, which is the minimum threshold level of infection. It requires that the ingested blood contains at least 9.3 million or 9.3 x 10^6 bacteria/mL of blood, which for all practical purposes excludes human plague cases. This development occurs with increasing frequency and in shorter time with increasing levels of bacteraemia in the ingested blood.[39] A stable transmission situation is established when the aggregates of biofilm become too large to be defecated or adhere to the foregut and will function continually at feeding.

When the forceful stream of blood from a new feed hits a sufficiently developed blockage, it recoils, a process supported by the recoil of the esophageal[40] wall, producing regurgitation: the blood is forced to stream back into the bite wound, taking with it fragments torn off the blockage with a large number or even thousands of plague bacteria. These extraordinary facts readily explain how infectious doses of immotile[41] plague bacteria are moved backward out of the mouth parts and back into the bite wound when fleas attempt to feed: they move with the blood as it flows back, with the regurgitants.

35 IPRC, XV, 1907: 405, 418–19; IPRC, XXIV, 1907: 876; Eskey and Haas 1940: 51–2; Eskey and Haas 1939: 1476; Benedictow 2016: 609–10.

36 IPRC, LVII, Bacot and Martin 1914: 423–39; IPRC, LXIX, Bacot 1914: 447–655.

37 Bland, Jarrett, Bosio, and Hinnebusch 2018: 14.

38 Haemobacillary means consisting of blood material (haemo-) and bacteria/bacilli.

39 Lorange, Race, Sebbane, and Hinnebusch 2005: 1909.

40 The esophagus is the muscular tube that conveys food from the pharynx at the back of the mouth to the stomach. It is also called the gullet. Cf. Bland, Jarrett, Bosio, and Hinnebusch January 2018: 2.

41 Lacking the ability to move.

A blocked flea is a starving flea and will spend the last few days of its life trying to obtain a blood meal, making repeated probing attempts, each usually resulting in a transmission. Often the flea will die with its mouthparts still inserted in the host.[42] The extreme lethality in plague is also the effect of heavy infections due to multiple transmission by the same blocked flea. This explains that blockage is a necessary and efficient condition for transmission of LDs of plague bacteria to humans. There is not any empirically observed or realistically conceivable alternative of transmission. The larger proportion of a species that becomes blocked and the faster it becomes blocked the higher will be its potential for transmission of plague, called vector efficiency (see below, Ch. 4.8).

Fleas begin digestion of a blood meal, as mentioned, immediately after a feed and it is usually complete within 24 hours when the digested meal has been passed into the hindgut. Because fleas process blood meals rapidly, ingested plague bacteria are in immediate danger of being passed from the midgut into the hindgut before having the opportunity to convey their genetic properties by transmission to a new host. By evolutionary selection they have developed the ability to cause the formation of biofilm within a few hours after an infectious blood meal if the ingested bacterial load is sufficiently large.[43] This ability is closely associated with virulence.[44] It also explains the rapid development of a plague epizootic in a rat colony and transition to endemic and epidemic plague (see Glossary, p. xxviii).

8. Biological condition of plague bacteria for formation of biofilm and blockage in fleas

A particular property of plague bacteria functions as a necessary condition for formation of biofilm and blockage in fleas and regurgitative transmission: that is, high virulence produced

[42] Jarrett, Deak, Isherwood, et al. 2004: 783–92; Hinnebusch 2012: 1–9; Hinnebusch, Jarrett, and Bland 2017: 216–20. This crucial point has been pointed out by several scientists: Bacot and Martin 437; Eskey and Haas 1939: 1474; Perry and Fetherston 1997: 51. Cf. Benedictow 2016a; 607, 662–3.

In an email of 27 July 2018, I asked B.J. Hinnebusch to enlarge on this decisive point of plague transmission, especially in relation to an article where experiments pertinent to this question was published: Lorange, Race, Sabbane, and Hinnebusch 2005: 1907–12. He responded in an email of 9 August 2018:

> 'We determined the number of bacteria transmitted by *X. cheopis* fleas by allowing individual blocked fleas a single chance to feed for only 1h. By this protocol, we found (JID paper) and subsequent work that the number transmitted per feeding attempt is very variable, from 0 to several thousand. In the natural setting, blocked fleas will make continuous feeding attempts over a period of several hours to a few days (until they die of starvation). It is reasonable to assume that there is a high probability that, cumulatively, a substantial number of bacteria will be transmitted. For example, in at least one of the many feeding attempts thousands of bacteria will be transmitted. I have observed starving blocked fleas make continual feeding attempts for hours and sometimes will die with their mouthparts still inserted. Thus, there may even be posthumous transmission!' Hinnebusch, Joe (NIH/NIAID)

[43] Hinnebusch, Jarrett, and Bland 2017: 216, 218. See below, Ch. 8.1.

[44] The ability of plague bacteria to produce blockage and biological transmission depends on pigmented hemin (hms), storage locus, and only fleas infected with hms$^+$ *Y. pestis* develop blockage. The unpigmented hms-mutants are unable to block and transmit LDs to humans. Hinnebusch, Perry, and Schwan 1996: 367–70; Lorange, Race, Sebbane, and Hinnebusch 2005: 1907–12.

by pigmented haemin storage, or hms$^+$. This property is evolutionarily conditioned or pro-selected because it ensures that virulent strains of plague bacteria with the property of hms$^+$ (or they would not be virulent) have the ability to engender biofilm and blockage in fleas and become transmitted. This is crucial because transmission ensures the continuity or survival of the bacterial strains and their genetic properties, including for high virulence.

Mutant low-virulent strains and non-pigmented (low-pigmented) hms$^-$ strains are often observed in laboratories but will be deselected and disappear because they lack the crucial property that produces formation of blockage in fleas with consequent inability to engender transmission and genetic continuity of this property. The effects of these evolutionary processes and the crucial role of virulence and hms$^+$ also show in paleobiological studies of the DNA of plague bacteria of the past that have identified mutated variants but not any strain that lacks this property for high virulence.[45] This also explains the stability of high virulence and lethality rates among human plague cases over many centuries.[46]

9. The crucial role of rats as sources of infection

The finding that ingestion of 3,800 plague bacteria represents the minimum threshold level for development of blockage by biofilm and infectivity of rat fleas is important. In the material on individual measurements of levels of human plague bacteraemia only one of 272 human plague cases or 0.37% would meet this criterium and infect feeding fleas with this number of bacteria, albeit with a huge margin. Crucially, no other human bacteraemic case would infect a feeding flea with anywhere near this minimum level of infection, and the other cases of primary bacteraemic plague would infect feeding fleas with small fractions of this number of plague bacteria. This is another proof that human fleas (and lice) do not play a part in the transmission and dissemination of plague among humans.

The IPRC's study of plague bacteraemia in rats showed that about one-third of them, a proportion a hundred times higher than among human plague cases, would infect feeding fleas with 4,100–41,000 plague bacteria. This was the case in the experiment, although a large majority of the rats were killed nearly 10 hours before death and may not have developed the full potential level of bacteraemia.

Late in a rat epizootic, when a colony is sharply reduced, large numbers of fleas from dead hosts have gathered together on the remaining rats, which will be exposed to multiple infective doses conducive to high bacteraemia and heavy infections of feeding fleas. This is an important aspect of the rat epizootic that the IPRC's experiments were not designed to study and has remained overlooked.

10. The crucial role of plague-infected rat blood to make rat fleas rapidly infective by blockage

Rat fleas are adapted to living conditions in the rat coat, with a warm microclimate, a sheltered life in the fur, the smell of rat, the opportunity of feeding at will, and so on. Laboratory experiments on plague transmission expose fleas to alien living conditions, often in vitro,

[45] Tiflov 1964: 181–98; Bibikova and Klassovskiy 1974: 115–23; Bibikova 1977: 28; Lorange, Race, Sebbane, and Hinnebusch 2005: 1907–12. See also Hinnebusch, Perry, and Schwan 1996: 367–70; Benedictow 1992a/1993/1996a: 241–2; Benedictow 2016a: Ch. 1.5.

[46] Benedictow 1992/1993/1996a: 146–9.

feeding at the will of researchers, in the cool environment of the laboratory. Often fleas are fed on bacteraemic mouse blood because it is convenient for the scientists. This practice contains the risk that the unnatural laboratory conditions will affect the physiological processes and behavioural pattern of rat fleas. I have several times pointed out that this was the reason that the development of blockage in fleas and transmission took much longer in laboratories than the IPRC observed in their experimental godowns where the rhythm and spread rates of plague in 'rat colonies' were studied under more natural conditions.

In a recent experiment, scientists studied the effect of host blood on rat fleas' transmission capacity. They were fed bacteraemic mouse, rat, guinea pig or gerbil blood. The experiment showed that the type of infectious blood affected the process of transmission. Two of the rodent flea species fed bacteraemic rat or guinea pig blood refluxed a portion of the infected blood meal into the esophagus within 24 hours of feeding (PIER), while this was rarely observed in rodent fleas fed on bacteraemic mouse or gerbil blood. At their next feeding, the fleas with PIER were 3–25 times more likely to appear partially blocked by biofilm, and this condition was much strengthened after three days when these fleas transmitted far more plague bacteria and by regurgitation of *Y. pestis* from a foregut partially blocked by biofilm.[47] The results of these experiments go far in explaining the difference between IPRC's observations on the development of rat epizootics and later laboratory experiments.

The scientists claimed that the relative insolubility of the hemoglobin of rats seems to promote regurgitative transmission by blockage/biofilm and may be one important reason that rats are crucial in plague epidemics. It implies another sufficient condition for why human fleas do not play any (significant) role in plague epidemics.

11. Vector efficiency of fleas of plague contagion

For all practical purposes, transmission of plague is performed by fleas (partially) blocked by biofilm: 'transmission from unblocked fleas is exceedingly rare',[48] which, according to the references, seems to imply not observed. IPRC noted that heavily infected cat fleas did not transmit plague to healthy guinea pigs, rodents that are extremely susceptible to plague infection. In large-scale laboratory experiments, extremely infected human fleas hardly transmitted plague to healthy guinea pigs while, in the presence of rat fleas, epizootics soon flared up but they could not explain the difference.[49] Later research has uncovered that there are indeed great differences between species of fleas with respect to the prevalence and velocity with which they develop blockage and become vectors of plague infection, also when they are fed on the same bacteraemic blood. Technically, this is called *vector efficiency* or *vector capacity*. The larger proportion of a species that becomes blocked and the faster it becomes blocked the higher will be its potential epidemic powers of transmission of plague.

Burroughs, for instance, studied the vector efficiency of ten species of fleas, among them the black-rat flea (*X. cheopis*), the human flea (*P. irritans*), the brown-rat flea (*Nosopsyllus fasciatus*) and several species of fleas associated with wild rodents in American plague reservoirs. Some of the wild rodent fleas were good vectors but because the chance that specimens of these species in a transmissive condition of plague without a rodent alternative will meet

47 Bland, Jarrett, Bosio, and Hinnebusch 2018: 1–27.Innebusch

48 Perry and Fetherston 1997: 51.

49 IPRC, I, 1906: 435–67; IPRC, XV, 1907: 395–419; IPRC, XVI, 1907: 421–35. Cf. above, Ch. 3.2.

a human is in the range of microprobabilities, this is without epidemic significance. Burroughs concluded that '*X. cheopis* […] proved to be the vector *par excellence* that it has long been claimed to be',[50] with a larger proportion becoming blocked and in a shorter time than other species. No one of 55 *P. irritans* transmitted plague, one of them blocked but died before it had performed a transmission. This is the only observed case of a blocked human flea.[51] The negative outcome of this experiment with respect to the vector efficiency of the human flea is a consistent finding.[52]

N. fasciatus, the brown-rat flea, was found to be quite a good vector (which is not a general observation).[53] However, because it is a nest flea that does not ride with its host (see below), its host avoids people and, within human settlements, prefers to live in sewers and cellars, it cannot perform a significant epidemic role[54] Although dogs rarely and cats not uncommonly can contract plague and die from the disease,[55] they are relatively few in relation to the human population, often live at a social distance from humans and their fleas are inefficient vectors.[56]

Only a species of fleas which easily blocks from plague infection and that is widely and densely present in human environments, habitations, workplaces, peridomestic surroundings, and in means of transportation, can be an important or predominant vector of epidemic plague. So far, only the flea of the black rat has been found to satisfy these conditions for performing the role as a vector of transmission of plague on an epidemic scale. Clearly, the panorama of plague bacteraemia among black rats explains that there is a close connection between the presence of black rats in human settlements and the spread of plague epidemics. Black rats are the only source of plague infection of fleas in the vicinity of human beings that will make efficient vectors infective and able to perform an epidemic role.

50 C.Y. Wu 1936: 250, 274; Eskey and Haas 1940: 43–4; Girard 1943: 32; Brygoo 1966: 39.

51 Burroughs 1947: 379–81, 387.

52 Perry and Fetherston 1997: 53. Cf. Butler 1984: 51.

53 Burroughs 1947: 383–4, 393. This is not a general finding. Cf. Benedictow 2016a: 521, 525–6.

54 Benedictow 2016a: Ch. 10.4, pp. 503–34.

55 Hirst 1953: 151.

56 IPRC, XV, 1907: 412–13; Pollitzer 1954: 44, 376; Perry and Fetherston 1997: 53; Bland and Hinnebusch 2016: 1–25; Hinnebusch, Jarrett, and Bland 2017: 225.

5

Medical and Clinical Features of Bubonic Plague

1. Buboes and bubonic plague

The designation of bubonic plague arises from the fact that it is the only disease where buboes are a normal clinical feature. This unique feature has a unique causation. The skin is the human body's first line of defence against infection. Bubonic plague is transmitted by blocked rat fleas, which pierce the skin and, attempting to feed, often regurgitate a lethal dose of plague bacteria and release it at an intradermal level in the catchment area of the lymphatic system. In most cases, the regurgitant will be drained from the bite site through a lymphatic tract to a lymph node that, upon infection by plague bacteria, begins to swell and develop into an intensely painful bubo.

For these reasons, buboes mainly arise in anatomical locations with concentrations of lymph nodes, the groin and adjacent part of the thigh (inguinal–femoral area), the armpits (axillas) and the neck (cervix). This is corroborated by a huge amount of clinical descriptions in historical sources; modern and historical specifications of the usual sites of plague buboes are identical.[1] Because the legs are the largest surface area of human beings and because legs can attract fleas both when people are asleep and standing up or walking about, most bites occur there. The contagion will then usually be drained to a lymph node in the inguinal–femoral area. Sleeping humans will often be bitten on the upper parts of the body and develop buboes in the armpits or quite frequently on the neck or nearby shoulder.[2]

The specificity of transmission of infective bacterial loads of plague bacteria can explain that bubonic plague is the only epidemic disease clinically characterized by the clinical manifestation of buboes and is, therefore, also a defining feature. The designation bubonic plague is appropriate and contains an identifying function.

The infection of a lymph node is characterized by excruciating pain from the moment it begins to swell and before a bubo appears, which usually is on the second or third day.[3]

1 Benedictow, 2010: Ch. 9: 312–80.
2 Pollitzer 1954: 419–23.
3 de Langen and Lichtenstein 1936: 192–3.

The sudden onset of stabbing pain was often the first indication of plague disease.[4] In a parish in western Norway, an account was told and recorded much later about a dairy maid on a peasant holding who, in the plague epidemic in 1600, returned from the shieling[5] one night, put down the milk bucket and said: 'Now the plague stung me!' The next day she was dead.[6]

Piercing pain is frequently mentioned in historical descriptions of plague cases. Shyness in relation to the sexual connotations of the groin and nearby parts of the thigh that is the most common site may keep some commentators from mentioning bubo(es) there and instead refer to the intense pain.[7] Gabriel de Mussis, the chronicler, gives a typical clinical description of the Black Death in his home town of Piacenza (Italy) in 1348:

> completely sound persons suddenly were hit by piercing pains. Next, they were overwhelmed by great frost that made them shiver with fever and they felt stinging spikes, as if they were pierced by arrowheads. Some were cruelly hit under the collarbone [i.e., in the armpits], others where the belly and thigh meet [notice the euphemism]. Here grew a smaller or larger lump […] Those who had hardened glands [= lymph nodes] died unless they did not turn soft [i.e., begin to suppurate] […].[8]

The Icelandic *Lawmaster's Annals*' clinical description of the Black Death in Bergen (Norway) in 1349 is also a case in point and is, despite the name that refers to a later owner, written by a contemporary ecclesiastic. His good informant was one of Iceland's two bishops who had stayed in Bergen during the Black Death: 'The disease was such that men did not live for more than a day or two with *sharp stings of pain*, then they began to vomit blood, and then they expired.' The chronicler conveys the most conspicuous clinical features of bubonic plague, the excruciating pain from nascent and growing buboes the first two days followed by the dramatic development of secondary pneumonic plague characterized by a cough with bloody expectoration that portended certain death (see below).

In the French city of Reims, a list was kept during the Black Death in order to register by name, approximate age and social status of persons who had been miraculously healed from plague disease by intercessory prayer to St Remi, the city's protector saint. Some of the entries included a basic clinical description as proof that the disease really was plague. A mother declared to have seen her small boy be healed from three buboes, two buboes in the groin, and one in an armpit; at the time of Ascension Day (21 May), a young girl first had a bubo 'under her arm', then, a second bubo on her neck; a married man had an enormous bubo on his neck on 17 July; Jehan de Blanzy, a man of some learning, related that his wife's disease began with strong fever, next buboes appeared in the groin but after intercessory prayer to St Remi had been said, her condition began to improve on 29 July.[9]

4 Benedictow 2010: 542–6.

5 A shieling is (the English word for) a shelter for domestic animals associated with the use of mountain pastures in the summer and early autumn.

6 Benedictow 2016a: 138.

7 Benedictow 2010: 536–50.

8 Original Latin text rendered by Henschel 1842: 55–6. Cf. German translation by Sticker 1908: 52–3.

9 Desportes 1977–2: 797–8.

Historical clinical presentation of plague buboes can be completed by citing the proto-scientific description given by the M.G. Block, the physician, in a plague tract written after his observations of the plague epidemic in a Swedish town in 1710–11 where these pieces of information are joined:

> it pricks like needles everywhere in the glands and particularly under the shoulders and in the groin. Swellings in those places, swellings that eventually, though quite slowly mature into buboes […].[10]

The descriptions of buboes, their anatomical sites and general appearance on the diseased are consistent features mentioned by contemporaries of all three known pandemics of plague.[11] Not only the general terminology but also all individual pieces of clinical information provided by contemporaries are consistent with observations of modern bubonic plague.

There are two forms or modalities of plague in which the infection is not released at an intradermal level that challenges the lymphatic system but directly into the bloodstream or via the lungs and are not, therefore, characterized by buboes.[12]

2. Secondary bacteraemic plague: plague spots, haemorrhages, pustules and plague pox, nosebleed, carbuncles

When fleas deposit large bacterial loads and cause heavy infections of lymph nodes, high concentrations of bacterial toxins will develop in the primary buboes. They consist of exotoxins that are produced and secreted by living bacteria into its surroundings and endotoxins that are present within bacteria and released when bacteria die and disintegrate. Generally, microbial toxins promote infection and disease by directly damaging host tissues and by disabling the immune system. Plague exotoxins cause attrition of the walls of buboes leading to haemorrhagic infiltration of the neighbouring vessels and subsequent passage of plague bacteria into the bloodstream where they will travel to almost any part of the body, including the peripheral capillary vessels.[13] Plague endotoxins cause coagulation and the formation of tiny blood clots in blood vessels throughout the body. Together with the attrition of the walls of blood vessels, clots will cause capillary vessels to burst so that blood seeps into the skin and forms what looks like blue or purple blotches or spots, technically called petechiae.[14] This development is also associated with comprehensive internal haemorrhages that are uncovered by autopsies.[15]

Because plague spots are associated with bacteria in the bloodstream, they also are symptoms heralding the imminent demise of the diseased, almost without exception. This was understood by contemporaries. During the Black Death, they are described, for instance, by Boccaccio in the introduction ('First Day') of *The Decameron*:

10 Block 1711: 21. My translation from contemporary Swedish.
11 Benedictow 2010: 312–80.
12 See below, subch. 5.5.
13 Pollitzer 1954: 205–6.
14 Block 1711: 22–3; Simpson 1905: 226–7, 284; Hirst 1953: 29; Pollitzer 1954: 424–6; Benedictow 2010: 373–8.
15 Simpson 1905: 226–48; Pollitzer 1954: 203–17; Benedictow 2010: 372–80, 653–61.

> after a while, the fashion of the contagion began to change into black or livid blotches, which showed themselves in many on the arms and about the thighs and every other part of the person, in some large and sparse and in others small and thick-sown; and just as the plague-boils [buboes] had been first a very certain token of coming death, even so were these for everyone to whom they came.[16]

In England, the plague spots were called 'God's token', or, as a reflection of familiarity, only 'token' because those who developed plague spots were considered as being called home by God. Shakespeare used the expression in its dramatic meaning: 'The tokened pestilence where death is sure.'[17] These tokens were so generally dreaded that they gave plague the name of 'the spotted death'.[18] In Norway, the expression 'the spots are counted' meant that a person was dying, and it was in use long after the plague period when the original meaning was lost.[19]

Because plague spots often will contain infected blood, they will tend to increase rapidly in size and then rise in the form of blisters or vesicles that may develop into pustules, by contemporaries called blains. Pustules often are widespread over the body and can be so numerous and like the bumps of smallpox that even, at the turn of 1900, physicians who were familiar with real smallpox, misdiagnosed such cases.[20] This form is designated *plague pox/variola*.[21]

Because plague toxins wear down the walls of blood vessels and produce blood clots, and the widespread clotting also will deplete the body's clotting resources and reduce its ability to control bleeding, nosebleed (epitaxis) is a usual clinical feature of bacteraemic bubonic plague.[22]

Carbuncles are quite a usual clinical feature, consisting of localized dead body tissue (gangrene) caused by plague bacteria left in the upper part of the bite site of a flea, in which case it is called a primary carbuncle.

Because pustules arise from infected blood that seeps into the skin and tend to grow, they may coalesce and form areas of necrosis that also (on factual grounds) are called carbuncles, definitionally secondary carbuncles.[23]

3. Severe headache

Severe or excruciating headache is also a usual clinical feature of the onset of bubonic plague disease. From the beginning of the Black Death, this clinical feature is mentioned by many chroniclers, including Michaele da Piazza, Gabriele de Mussis, John Clyn, among others.[24]

16 Boccaccio 1982-1: 9. Boccaccio's assertion that all plague cases who displayed buboes died is not correct because about 20% survive. Plague without buboes, i.e., primary bacteraemic plague and primary pneumonic plague, is (for all practical purposes) mortal.

17 McArthur 1925–6: 358; Wilson 1963: 102.

18 Bell 1951: 127–8.

19 Benedictow 2016a: 104.

20 Simpson 1905: 227, 246, 284; Chun 1936: 311, 313, 316, 329; Benedictow 2010: 376–7.

21 Simpson 1905: 373; Hirst 1953: 30; Pollitzer 1954: 425–7; Butler 1983: 79.

22 Block 1711: 21, 23; Simpson 1905: 247, 265; Chun 1936: 313; *Manson's Tropical Diseases* 1921: 268.

23 Block 1711: 21–2; Simpson 1905: 284–6; Pollitzer 1954: 206, 424–5.

24 Michaelis Platiensis 1791: 567; Gabriele de Mussis 1842: 55, English translation in Horrox 1994: 24; *Fratris John Clyn Annales Hiberniæ* [*Annals of Ireland*] 1849: 36; Cayla 1906: 46–7.

Because the Black Death and later plague epidemics of the Second Pandemic show great genetical consistency it is the case, as expected, that headache is a normal part of later clinical descriptions of plague. In his diary, Absalon Pederssøn Beyer mentions two cases in the epidemic in Bergen in 1565–6: 'A man was ill with pestilence, he raged, he stood up in his rage and ran to L. Lundegaard where he drowned himself.' A clergyman of one of the Hanseatic congregations in Bergen died in the plague 'and raged somewhat in his head in his illness'.[25] In his book on the plague in Norrköping in 1710–11, M.G. Block, the Swedish physician, gives a detailed description of the clinical features of plague, among them 'headache and confusion'.[26]

Because an offshoot of the Black Death or rather another epidemic of the Second Plague Pandemic to the Yunnan province in China was the point of departure of the Third Plague Pandemic, it is as expected that the standard works on plague mention splitting headache as a usual clinical feature, including the last standard work based on personal experience during the large plague epidemics under the Vietnam War where 85% of a group of 40 plague patients presented with headache.[27]

4. Secondary pneumonic plague

In cases of secondary bacteraemic plague, bacteria are transported by the bloodstream to the lungs. Most plague patients with this condition die fast from severe bacteraemia. In those who hang on to life for some days, plague bacteria quite often attach to lung tissue, and proliferate and consolidate in a manner producing an ulcer. Accumulation of blood, pus and suppuration means that the diseased will tend to produce a cough with bloody expectoration. A condition has arisen from the original bubonic plague infection, which is called *secondary pneumonic plague* because it is a secondary (or tertiary rather) development from the original primary bubonic condition. While 30–40/45% of primary bubonic plague cases develop bacteraemia, secondary pneumonic plague arises in 10–25% of all bubonic plague patients or in 33–55/62% of all cases with secondary bacteraemia.[28]

5. Primary pneumonic plague and pulmonary plague

Cough by persons with secondary pneumonic plague can expel plague bacteria out of the lungs in droplets that may be inhaled by another person in the vicinity. A person infected by droplets inhaled into the lungs contracts *primary pneumonic plague* because infection of the lungs is the original cause of the disease. Primary pneumonic plague is an extremely lethal disease and form of plague, and survivors are exceptional.

Primary pneumonic plague is not a highly contagious disease, and for several reasons. Plague bacteria are much larger than viruses. This means that they need much larger and heavier droplets for aerial transportation to be transferred. Big droplets are moved over much shorter distances by air currents in the rooms of human housing than small ones. Studies of cough by pneumonic plague patients have shown that 'a surprisingly small number of bacterial colonies develop on culture plates placed only a foot directly opposite

25 My translation. Benedictow 2002: 208, cf. English updated edition 2012[1]b: Ch. 18.
26 Block 1711: 21, 23. My translation from contemporary Swedish.
27 Simpson 1905: 263; Chun 1936a: 310; Pollitzer 1954: 411; Butler 1972: 271–2; Butler 1983: 73.
28 Hirst 1953: 22.

the mouth'. Physicians emphasize that to be infected in this way normally requires that one is almost in the direct spray from the cough of a person with pneumonic plague. Most cases of primary pneumonic plague give a history of close association 'with a previous case for a period of hours, or even days'.[29] It is mostly persons engaged in nursing care who contract this disease: in modern times, quite often women and medical personnel; in the past, undoubtedly women were most exposed. Our knowledge of the basic epidemiological pattern of pneumonic plague is precisely summarized by J.D. Poland, the American plague researcher:

> Primary plague pneumonia occurs primarily in persons in close and prolonged contact with another person with pneumonic plague. Hence, respiratory transmission occurs most frequently to medical personnel or household contacts who are directly involved with the care of the patient.[30]

Because droplets that can carry plague bacteria are relatively large, they tend upon being inhaled to impinge on the upper parts of the respiratory tract where they infect the tonsillar region. This will cause pharyngeal and primary bacteraemic plague (see Glossary). This development, the conversion of the effects of inhaled plague-infected droplets from causing primary pneumonic plague to causing primary bacteraemic plague that is acutely lethal and non-infectious, depletes and weakens the powers of spread of pneumonic plague.

Wu Lien-Teh, the leading authority on primary pneumonic plague, emphasizes the importance of a third type of fulminant cases called *pulmonary* plague. Dissection of the lungs of such cases did not show the usual pneumonic foci that produce the cough with bloody expectoration. These types of cases that do not develop a bloody contagious cough are quite usual and also undermine the powers of spread of pneumonic plague.[31]

These are the main reasons that primary pneumonic plague is not strongly contagious and does not easily 'recruit' new infective agents who assure the continuation and dynamic development of the epidemic process. These are also the reasons that cause 'autolimitation' and 'spontaneous decline' of epidemics of primary pneumonic plague,[32] and that most outbreaks are tiny or small, at the social levels of family and neighbours.[33]

It also explains the usual tiny prevalence of cases of primary pneumonic plague in bubonic-plague epidemics. Studies of 480 and 411 plague cases admitted to the Arthur Road Hospital in Mumbai in 1899 and 1901 showed that about 2.5% of them suffered from primary pneumonic plague.[34] This was about the same percentage as later recorded by IPRC,[35] hence it seems to be about the normal prevalence of cases of primary pneumonic plague in epidemics of bubonic plague, which is the usual context of most such cases.

29 Hirst 1953: 226–7.

30 Poland 1983: 1230.

31 Wu Lien-Teh 1926: 189–92; Wu Lien-Teh, Chun, and Pollitzer 1934: 86; Wu Lien-Teh 1936b: 420–1; Benedictow 2010: 525–7.

32 Wu Lien-Teh 1926: 187–95; Benedictow 2010: 518–30.

33 Klimenko 1910: 659–62; Wu Lien-Teh 1913–14: 243–7; Wu Lien-Teh 1926: 69–97; Benedictow 1992/1993/1996: 220–3.

34 Choksy 1903: 60.

35 Lamb 1908: 1.

In Madagascar, Upper Egypt and Java, cases of primary pneumonic plague were more frequent because of special cultural or social reasons that produced frequent close personal contact between family members, relatives or neighbours and the diseased.[36]

No epidemic of primary pneumonic plague has been identified within the multitude of (known) epidemics constituting the Black Death. This was made clear in the first edition of this book and nothing has changed this conclusion. The epidemic form of primary pneumonic plague is, therefore, not a subject of this book. One likely mixed epidemic of bubonic plague and primary pneumonic plague occurred in Avignon and will be presented below.

Sticker made efforts to identify a historical epidemic of primary pneumonic plague but only succeeded in identifying six mixed plague epidemics consisting of proportions of bubonic plague and primary pneumonic plague, a list that was supplemented by Wu Lien-Teh.[37]

In his fine study of plague and other epidemic diseases in early-modern Central Europe, Eckert discusses some evidence on plague epidemics based on parish registers relating to plague epidemics in 17 communities in the years 1563–4. The clinical evidence provided by contemporaries does not discern between secondary and primary pneumonic plague but the fact that 11 of 17 communal epidemics reached their peaks of burials in the winter months merits attention. However, the author concludes his discussion by stating that these and other outbreaks of winter epidemics probably were mixed epidemics, pointing to the Madagascan epidemics for an analogous model.[38] Despite much effort, no certain or likely historical epidemic of primary pneumonic plague has been identified before 1850.[39]

6. Two modalities of plague without buboes

There are two modalities of plague where the infection is produced in ways that do not challenge the lymphatic system and cause the growth of buboes. Primary bacteraemic plague arises when a blocked rat flea regurgitates a bacterial load directly into a blood vessel and the bloodstream. The plague bacteria in the regurgitant will rapidly proliferate in the bloodstream and produce high levels of bacteraemia. Primary bacteraemia is easily recognizable by the absence of bubo(es) and a fulminating course of illness lasting, on average, about 15 hours before certain death.[40] This modality of plague was observed by early-modern physicians who remark that plague without buboes is the most dangerous form.[41] Studies on representative samples of 480 and 411 plague cases admitted at the Arthur Road Hospital in 1899 and 1901 showed that primary bacteraemic cases constituted about 3% of all cases.[42]

36 Wu Lien-Teh 1926: 34, 62, 165, 180–1, 301–2; Wu Lien-Teh 1936b: 413, 416; Wakil 1932: 98, 102; Brygoo 1966: 64–71.

37 Sticker 1910: 246; Wu Lien-Teh 1926: 7.

38 Eckert 1996: 83, 85–6. For the Madagascan plague epidemics, see the fine and comprehensive overview by Brygoo 1966.

39 The two Manchurian epidemics of primary pneumonic plague of 1910–11 and 1920–1 have an extreme social and economic background. Wu Lien-Teh 1913–14, 1922–3, 1926, 1936b; Benedictow 2010: 493–552.

40 Philips and Hirst 1917: 529–30, 534–5.

41 E.g., M.G. Block, the Swedish physician, who wrote a remarkable proto-scientific study of a plague epidemic in his home town of Norrköping in 1710–11: 'That plague is the most dangerous which leaves no external signs, attacking directly the spirits of life in the heart itself.' Block 1711: 21, 24–5: My translation from contemporary Swedish.

42 Choksy 1903: 30, 58.

Furthermore, primary pneumonic plague that is caused by infection via the lungs does not challenge the lymphatic system and does not exhibit the development of buboes. The mass registrations and categorization of plague patients brought to plague hospitals during the plague in Mumbai around 1900 registered a prevalence of primary bacteraemic plague of about 2.5%.

All other forms of plague are derived from bubonic plague and bacterial loads that are discharged at an intradermal level and challenge the lymphatic system.

6

Basic Aspects of the Epidemiology of Bubonic Plague

1. The temporal rhythm of rat epizootics and the role of rat fleas

IPRC started with basic experiments on the rat-and-rat-flea-borne theory of plague, using locally trapped black rats and laboratory rodents. These experiments were carried out in especially designed godowns constructed after the manner of Indian houses, which allowed experimental animals to roam quite freely, much as in a normal rat colony in a usual human environment.[1] This permitted basic experiments, examining, for instance, the option that plague was spread by contact: 50 healthy guinea pigs were placed together with 10 guinea pigs inoculated with a virulent culture of *Y. pestis* from which all fleas had been removed. A week later, all inoculated animals had died of plague, and none of the healthy animals had died from plague. On 25 November 1905, this experiment was repeated with a normal presence of fleas: all inoculated animals were dead after a week and the epizootic flared up among the healthy. The first death among the healthy guinea pigs occurred eight days later, a period that would include the death of at least one inoculated animal, the transfer of at least one of its infective fleas to one of the healthy animals, infection, incubation and the time of illness. Later studies showed that the average time from infection to death of black rats is 3.66 days,[2] which fits well, even perfectly, assuming the lapse of a few hours from the infective flea leaving the dead or dying guinea pig until it finds a new host among healthy animals. By 17 days later, all originally healthy animals were dead.[3]

Significantly, 400 fleas were recovered from the last two guinea pigs, 326 from one which was moribund, and 74 from the other which was dead and had been deserted by many of its fleas on the hunt for a new host.[4] This showed that, as the animals were dying or died, their fleas gathered together on the remaining live hosts, which meant that when these hosts died,

1 Liston 1924: 953; Lamb 1908: 2, 38.
2 IPRC, VIII, 1906: 521.
3 IPRC, I, 1906: 435–67; IPRC, XVI, 1907: 421–35.
4 IPRC, I, 456–9, 472–5, 479–82.

hundreds of infective rat fleas would be released much at the same time. Numerous such observations were also made on wild rats dying or recently dead from plague: they carried on average about 80–100 fleas, which proved infective when introduced among healthy rodents.[5] After some involuntary fasting, these fleas would presumably be ready to leap onto a usable host in the proximity to take a blood meal, for instance, from a human (which was corroborated by a later experiment, see below). This explained how several members of a household fell ill by plague at about the same time.

Experiments were also performed in a tenement building (chawl) inhabited by poor workers in Mumbai that had been evacuated after finds of dead rats and human plague cases. On several occasions, guinea pigs were allowed to roam freely in a room where they attracted a substantial number of fleas. These were predominantly rat fleas because the rats were now dead and their fleas did not find new rat hosts and could not be choosy over hosts for feeds. These fleas proved fatal to these guinea pigs and other rodents. This also explained why members of the same household often were infected at much the same time.

A long series of experiments also showed that fleas were unaffected by the presence of lice on both healthy and inoculated animals. IPRC made a succinct summary of the initial findings:

1. Close contact of plague-infected animals with healthy animals, if fleas are excluded, does not give rise to an epizootic among the healthy.
2. In comprehensive and varied transmission experiments where lice were allowed to remain in the fur but all fleas were removed no case of transmission occurred, which allowed the IPRC to state unreservedly that 'fleas, and fleas alone, were the transmitting agents of infection'.[6]
3. If fleas are present, the epizootic, once started, spreads from animal to animal, the rate of progress being in direct proportion to the number of present fleas.[7]

2. The latency period: from the infection of a rat colony to the first human death: a defining feature of bubonic plague

Progressing along this line of research, IPRC studied the temporal rhythm of phases following the introduction of plague contagion into a rat colony in a locality to the first human case or cases:

1a. When an infective black-rat flea (*X. cheopis*) has been introduced into a rat colony, it takes 10–14 days before the rat colony has become so reduced that it is difficult for the great numbers of fleas that have gathered on the remaining but soon-to-die rats to find new hosts.[8]

1b. After about three days of fasting, hungry rat fleas will turn on human beings in their immediate surroundings.[9]

[5] IPRC, I, 1906: 454, 456–7, 459–61, 466–7, 474–5, 479–82; IPRC, XVI, 1907: 431–3; IPRC, XXIV, 1907: 884–5. Cf. Petrie, Todd, Skander, et al. 1924–5: 130.

[6] IPRC, XVI, 1907: 421–35.

[7] IPRC, I, 1906: 466–7; IPRC, XVI, 1907: 422–31.

[8] IPRC, XXII, 1907: 764; Lamb 1908: 21, 51; Wu Lien-Teh 1936b: 222.

[9] IPRC, XXII, 1907g: 765; IPRC, XXV, 1907: 943; Lamb 1908: 52; Wu Lien-Teh 1936c: 387; Pollitzer 1954: 485. Cf. IPRC, I, 431–2.

This initial epizootic phase of a bubonic-plague-epidemic-to-be, before rat fleas turn on humans, lasts 13–17 days. This is the basic latency period of bubonic plague when plague imperceptibly is spreading in a black-rat colony without spilling over into disease among human beings.

2a. When a human has been infected with bubonic plague, the infection takes on the average 3–5 days to incubate before the disease breaks out.

2b. The course of illness takes on the average another 3–5 days before the diseased in most cases die.[10]

The development of a human plague case from infection to death usually takes 6–10 days, on average 8 days. This completes the latency period.

3. Normally, it takes 19–27 days, usually about 23 days, from the introduction of plague contagion among rats in a human community before the first person dies from plague. If the first death is the carrier of an infective rat flea, the next death follows at about the same temporal distance.

This time lag or latent period between the introduction of contagion into a rat colony and the first human death is one of the defining features of bubonic plague: no other epidemic disease exhibits a similar temporal pattern of early development. New research has fully clarified the biological premises.[11] This period of around 23 days has been identified quite often in historical plague research[12] and will repeatedly be demonstrated in analyses of plague epidemics of the Black Death below, also consistently in the three extant parish registers from the Black Death.

3. Fur fleas and nest fleas: role in plague epidemics

The study of the latency period established a crucial role for infected rat fleas in the development of a plague epizootic and its translation into human plague. Rat fleas also have specific properties that give them a crucial role in plague epidemics.

There are two main categories of fleas: (1) *fur fleas*, which normally ride in the coat of their hosts, and (2) *nest fleas*, which normally live in the nests of their hosts. Evidently, this difference is crucial for the role they can play in the spread of epidemic plague.

Most species of fleas are nest fleas, which live almost their entire life in the nest of their hosts. This ensures ready access to food, the larvae feeding on the blood in the droppings of the adult fleas and would succumb in the absence of adult fleas. This largely excludes a role in plague epidemics for this category of fleas. This is, for instance, the case with the species *Nopsyllus fasciatus*, the ordinary flea parasite of the brown rat (*Rattus norvegicuse*). Also the so-called human flea, *P. irritans*, is a typical nest flea. Predominantly they live in or around beds and other sleeping arrangements, which function as their nest. They come out at night, take their fill and hurry off to find a suitable hiding place for digesting the meal. Being normally active at night, they dislike exposure to daylight and travel only incidentally with a host. Examination of clothing in use did not yield many human fleas, even if human housing was

10 IPRC, XXII, 1907: 765; IPRC, XXV, 1907: 943; Lamb 1908: 52; Chun 1936: 309–10, 313; Wu Lien-Teh 1936c: 387; Pollitzer 1954: 409–11, 418 (485). Cf. Benedictow 2010: 276, n. 3.

11 Benedictow 2010: Appendix 3: 441–5.

12 Benedictow 2010: 281–8.

greatly infested, as was the case in India, Indonesia and North Africa. As expected, they did not find any *N. fasciatus*, although the brown rat was densely present in human environments. As a nest flea, human fleas typically are spread with the host's belongings, especially furniture and bedding, i.e., passive transport with the nest.[13]

Although the brown-rat flea and the human flea were widely and densely present in human habitation or peridomestic environments, their properties as nest fleas exclude a significant epidemic role in the epidemic dissemination of plague.[14] In the case of human fleas, this negative condition comes in addition to the decisive conditions springing from the low prevalence and levels of bacteraemia in humans and minuscule vector efficiency of human fleas.[15]

This stands out in contrast to *X. cheopis* which, being a fur flea, is adapted to riding with its host in the habitations and peridomestic surroundings of humans. The process of evolutionary selection has also adapted it to cope with the quite usual contingency for fur fleas to fall off or be scratched off a host as they wander about searching for food. This flea has developed quite versatile feeding alternatives in human environments such as humans, dogs and cats, and tolerates light well. In the absence of its natural host, *X. cheopis* readily leaps onto humans for feeds at any time of the day, finds riding in human clothing natural and may easily and inadvertently be spread quite widely, transported in the clothing or luggage of a person or in merchandise.[16]

4. Dissemination of plague contagion and the development of a plague epidemic

Spread by leaps or metastatic spread

Plague could only be effectively combated following a thorough knowledge of how it was spread. This required that the mechanisms and vehicles of dissemination were identified so that effective administrative countermeasures could be designed. The IPRC launched a series of comprehensive research programmes to study how plague was introduced into communities, and how plague was spread within and between communities and at various distances.

It had been shown that the black-rat flea, *X. cheopis*, had a high vector efficiency of plague (see Ch. 4.8) and also was adapted to being readily spread over distance by riding in human clothing or luggage. This indicated an epidemic process in which plague infection could be widely spread in the gut of infected fleas at the pace of movement of their human carriers. They could bite their carrier but might also hesitate and wait for an opportunity to feed on the natural host, which would be questions of time and opportunity.

IPRC performed a suitable experiment in a tenement building in Mumbai that had been evacuated after finds of dead rats and cases of plague. The houses were densely populated chawls, typically inhabited by relatively poor but gainfully employed working-class families crowding together in each small room.[17] For four consecutive days, a (vaccinated) man with

13 Benedictow 2010: 168–9; Bacot, Petrie, and Todd 1914: 505; Petrie, Todd, Skander, et al. 1924: 131.

14 Pollitzer and Meyer 1961: 461–3; Benedictow 2016: 28–9, 504–34.

15 See Ch. 4.3–4.8.

16 Busvine 1976: 17; IPRC, XXII, 1907: 775; IPRC, XXV, 1907: 891; IPRC, XXIX, 1908: 245–6; Karimi, Eftekhari, and de Almeida 1974: 587.

17 IPRC, XXII, 1907: 769–72.

bare legs[18] entered 'for a short time' a room in the chawl. On the first day, 40 human fleas (*P. irritans*) were found on the legs of the man; on the next day, 113 fleas, of which were 55 human fleas, 51 black-rat fleas (*X. cheopis*) and 7 cat fleas; on the third day, 76 fleas, of which 40 were human fleas, 34 black-rat fleas and 2 cat fleas; and on the fourth day 80 fleas, of which 18 were human fleas, 60 black-rat fleas and 2 cat fleas.[19]

On the first day, the man only picked up human fleas, while from the second day he also picked up a large number of rat fleas.[20] This reflected a process that IPRC had observed in the experimental epizootics constructed in the godowns at the laboratories in Parel. As plague spread in the local rat colony and rats died, their fleas gathered together on the remaining rats. When the last rats began to die,[21] hundreds of infective rat fleas left them and spread out in search of a host and, after three days of fasting, eagerly also leapt onto humans for feeding.

One must consider that the rooms were evacuated, that all fleas were badly starved, and that many persons would live in each room normally inhabited by a family whose members would share the flea load between them. Rat fleas are fur fleas that normally stay in the coat of the host and are not, therefore, caught by scientists until such a high proportion of rats has died from plague that their fleas do not find new hosts and after about three days of starvation leap onto humans to feed. The human flea is a nest flea that usually does not ride with its hosts but stays in the nest, in this case in the proximity of human sleeping arrangements, emerging at night to take a feed and find a place to digest the meal (and lay eggs).[22] This explains that on the first day only human fleas were caught, and the subsequent data show that the rat epizootic had reached a critical level two days before the experiment began, when large numbers of rat fleas left dying or dead rats without finding new hosts and, on the third day of starvation, readily leapt onto humans.

Clearly, as pointed out by the scientists, if the fleas had not been removed from the man's legs (or, as would normally have been the case, from his lower clothing), they would have been carried or transported to locations that the man visited.[23] There, some fleas would have left

18 IPRC, XX, 474, nos. 1–2.

19 IPRC, XX, 1907: 474–5.

20 Cf. above, Ch. 3.2 in fine.

21 A research project on the sensitivity of fleas to temperature showed that plague-infected *X. cheopis* preferred an ambient temperature up to 1.6 °C cooler than non-infected fleas. This change in thermal preference apparently is important in the transmission of plague from rat to human by *X. cheopis* for two main reasons: (1) rats have core temperatures approximately 1.5 °C higher than human beings, and (2) infected rats would react with fever to the plague infection, which would produce a further increase in temperature. Thomas, Karstens, and Schwann 1993: 209–13. 'In view of the preference for cooler temperatures in plague-infected rat fleas, it is possible that the fleas would actually prefer the human host. This being the case, temperature becomes an important factor in the transfer of *Y. pestis* from rat to man.' Brown 1995: 931; Perry and Fetherston 1997: 55. In short, infected rat fleas (*X. cheopis*) would tend to leave the host in an early phase of the febrile process in rats, which starts after the end of incubation period and would leave the rat about two days before it normally will have died. This would significantly reduce the duration of the epizootic and explains the relative rapidity of the epizootic process leading to near exhaustion of rat colonies, release of rat fleas, and transition to endemic and epidemic developments.

22 See Ch. 3.3, p. 21, and 6.3, pp. 48–9

23 Liston 1924: 997, 999, 1001.

him to search for their natural host and triggered plague epizootics in the rat colonies. The tendentially multiplicative dynamics of this structure of events is evident and explains that the typical insidious start of a plague epidemic was soon followed by widespread almost simultaneous territorial outbreaks, a fulminating rise in the number of plague cases and a dramatic increase in mortality rates. It also explains that contemporary people did not understand the process of spread characterized by the seemingly random structure of often simultaneous outbreaks in different parts of villages or urban society or how it leapt to neighbouring local societies or parts of a city. They could also not understand the seasonality of the plague epidemics, which often started with widespread and quite simultaneous outbreaks in the spring (see below). These features were rather more explicable by miasma spread by the wind than contagiousness and cross-infection. There will be many opportunities below to show this pattern of dispersed quite simultaneous outbreaks of spread in the presentation of epidemics of the Black Death.

This study also provided important scenarios explaining the social dynamics of local spread in historical plague epidemics: neighbours, relatives (inheritors), priests, and other persons who visited houses where people were sick and dying or had recently died from plague (e.g., to collect inherited objects, clothing or bedding), exposed themselves to grave danger. They would quite likely be bitten by infective and starving rat fleas that swarmed in the houses and/or pick up fleas in their clothing and bring them to their own home. There, the fleas would seek out the rats and trigger a new plague epizootic followed by an outbreak of plague elsewhere in local society or in a neighbouring local society. The connection would, however, be concealed because of the delay of approximately three weeks before the rat epizootic had run its deadly course and the incubation period and course of illness of the first plague case(s) were ended.

This study accounted for intra-local spread and indicated the mechanism of short-distance inter-local spread associated with social or work-related activities.

The spread of plague to a distance was called spread by leap (*per saltum*)[24] or, by Dutch scholars, metastatic spread.

Inter-local spread of plague

Logically, IPRC next launched a large project to study how plague arose in villages on the outskirts of Mumbai. They chose four remote villages where scientific personnel were deployed to wait for plague cases to appear in a socially and territorially transparent situation and with good contact with the village population. This would allow researchers to immediately register the first plague cases and investigate their origin, whether it was indigenous or arrived from the outside and, in the latter case whence, how and by whom plague contagion had arrived and had been introduced into specific households, and whence the person(s) came. This strategy enabled them to ascertain that plague arrived in the clothing or luggage of persons who could be identified. They were often natives of the villages who had found work at the cotton mills in the big city and now fled from the plague there or were persons who had travelled to Mumbai for pressing social reasons. Dead rats had been found before the first human plague cases, which indicated that these persons had introduced plague-infected rat fleas that first had sought out their natural host and unleashed a rat epizootic that preceded

[24] IPRC, XXIII–XXV, 1907; van Loghem und Swellengrebel 1914a: 460–81.

the first human plague cases, but in two cases without having infected the carriers. This view was reinforced when guinea pigs placed in a house with plague cases contracted plague and died.[25] This showed that plague consistently was spread by leaps with human carriers of infected rat fleas.

Contiguous spread between rat colonies

Because of their huge fertility, rats and rat colonies always live on the brink of starvation and death; a weakened rat colony will be invaded by rats from neighbouring rat colonies that use the opportunity of feeding on its food resources, including the carcasses of dead rats, and will there pick up infective rat fleas and introduce them into their own colonies.

IPRC also had the unforeseen opportunity of studying contiguous spread between adjacent rat colonies. The observation of the spread rate was made in a part of one of the villages[26] after it had been evacuated by the inhabitants following the discovery of a dead rat and the occurrence of a case of human plague.[27] Because the area had been evacuated, plague contagion could not be spread by the dynamics of human agency, by persons picking up rat fleas in their clothing at home or elsewhere and redistributing them as they went about their business or socialized with neighbours, as would have been the case in historical plague epidemics when people stayed in their homes. Under these circumstances, the pace of contiguous spread of plague between adjacent colonies of black rats could be measured, namely 300 feet or 91.4 m in six weeks, corresponding to the duration of the serial epizootic processes in three rat colonies and to 792 m in a year.[28]

This unplanned case study is included because advocates of alternative theories of plague have used it to claim that it was a study of the normal spread and standard spread rate of plague and showed that bubonic plague only spread contiguously between adjacent rat colonies. In the words of Twigg, for example: 'An important aspect of plague spread is the continuity across a rat population'; he also refers explicitly and erroneously to the Black Death's spread from Marseilles to Paris.[29] The fact that it was a tiny and incidental part of a large research project that proved exactly the opposite is consistently passed over. This allows advocates of alternative theories to deny or overlook the huge amount of primary research on spread by leaps or metastatic spread by transportation of rat fleas[30] synthesized in quite some detail in all standard works of plague,[31] which here only can be exemplified and equipped with references for further reading.

25 IPRC, XXIII, 1907: 799–873.

26 A village in India might often contain several thousands inhabitants.

27 IPRC, XXIII, 1907: 805–28, 835.

28 IPRC, XXIII, 1907: 827; Lamb 1908: 19.

29 Twigg 1984: 57, 100; the reference is given on pp. 131–2, and 209. Paris was infected from Rouen.

30 Twigg 1984: 57, 100, 131–2, with unpaged reference on p. 209; Scott and Duncan 2001: 79–80; Cohn 2005: 1354–5; Cohn and Alfani 2007: 178; Dean, Krauer, Schmid, Walløe, et al. 2018: 1304–8. Twigg is the original user of this argument, giving a general reference to the report in the last of the three page references (p. 131) but not the specific page reference, which is IPRC, XXIII, 1907: 827. See Benedictow 2010: 180–8; Benedictow 2016a: Ch. 11.4. Other advocates of alternative theories refer to Twigg's book as evidence or present it as an evident fact. Other arguments are used, which are commented on in Chs. 2–4 above.

31 C.Y. Wu 1936: 249–308 and Wu Lien-Teh 1936c: 383–423; Hirst 1953: 152–88; Pollitzer 1954: 315–408, 483–521; Brygoo 1966: 39–40, 71–4.

Because the Black Death and later plague epidemics evidently moved much faster, these observations showed, according to advocates of alternative theories, that historical plague was a different disease from bubonic plague. This is the basic but bogus argument for the construing of all alternative theories of historical plague, that it was, for instance, anthrax, a filovirida disease (e.g., Ebola disease), a viral disease spread by cross-infection by droplets, bubonic plague spread by human fleas and lice and several other mutually incompatible theories,[32] which implies that at least all but one must be completely wrong.

Contiguous spread of plague between rat colonies is a feature of plague epidemiology. It can be important for the duration of plague in streets or small densely settled areas.[33] However, the crucial factor in the spread of epidemic plague is the tendentially multiplicative spread rate of plague with infected rat fleas within the community by the movement of people and the corresponding establishment rate of new local centres of spread. This explains the insidious beginning of a plague epidemic followed after a couple of weeks by a fulminating rise in the number of local outbreaks, number of plague cases and mortality.

5. Long-distance spread of plague and grain-eating fleas

Another crucial epidemiological question remained unanswered: namely the mechanisms of long-distance metastatic spread. How did plague leap over long distances, hundreds and even thousands of kilometres, to another region or country or other part of Europe or another continent? Clearly, this had occurred with the Black Death and also with later plague epidemics long before the age of steamships and railways. Scientists had noticed that the present plague pandemic had been transported over huge distances, from Hong Kong to Kolkata (Calcutta) and Mumbai, and from Mumbai to Madagascar and to Jeddah on the Red Sea, to South America, and from Hong Kong to Honolulu whence plague probably was transported to San Franscico (USA), and so on.[34] Rats could, of course, travel in cargoes over long distances but rats infected with plague would normally die in a few days and finish any possible role as agents of spread.

Again, the solution was found with the rat fleas and the immense creativity of force of evolution by selection according to the basic principle of the *survival of the fittest*. Because rat fleas were fur fleas adapted to riding with their hosts, they have for thousands of years fallen off, been scratched off or have left their dead hosts in the favourite environments of black rats. Because grain is the favourite food of black rats and they are not hostile to humans, their fleas have tended to be on their own in human environments with grain or cereal debris. It turned out that the fleas of the black rat had acquired the ability to live off grain and cereal debris, the females being dependent on blood only for laying eggs.

The huge fertility that characterizes fleas and the rapid turnover of generations produce continuously a correspondingly huge number of mutant specimens, and therefore also at some point specimens that had this special property. The specimens that had acquired this property and their offspring would survive more often than rat fleas that did not possess this property. In time, this species of flea would be genetically adapted to this situation and have developed the general ability to live off cereal debris. Larvae of *X. cheopis* have also acquired

[32] All thirteen alternative theories published by 2015 are thoroughly presented and discussed in Benedictow 2010 and Benedictow 2016.

[33] Slack 1977: 53–7; Hult 1915: 100, 106–20; Benedictow 2010: 567–8.

[34] Hirst 1953: 314; https://en.wikipedia.org/wiki/San_Francisco_plague_of_1900%E2%80%931904.

this property, cereal debris providing a most suitable nourishment and making them independent of availability of blood in the droppings of adult fleas, on which the larvae of nest fleas are dependent.[35] Estrade, the entomologist who worked with the French plague research team in Madagascar in the 1930s, studied the prolonged survival of *X. cheopis* in cereal dust in Madagascar because they were found living free in or near the huts and hovels of the natives. In environments characterized by temperatures of 15–20°C and high humidity corresponding to the climate of the Madagascan highlands this flea can 'live far from every host on the condition that it can stay in various types of dust but on the condition that it contains cereal debris'.[36] Young rat fleas were found to develop by the hundreds in the dark corners of huts in Madagascar. Clearly, such free-living fleas were apt to obtain their first blood meal from humans rather than from rats and would be adapted to this contingency.[37] In the Madagascar highlands, rat fleas survived for several months in human habitations where grain was ground. Cases of plague often occurred in the seeming absence of a rat epizootic owing to the prolonged survival of rat fleas long after the rats had died from plague. According to Hirst,

> The infection survives in the gut of fleas in dusty corners of the living rooms where the family corn is ground. Not infrequently, such infected fleas were transported considerable distances by human agency and gave rise to sporadic outbreaks of plague at destination.[38]

The ability of *X. cheopis* to lead an independent existence as free-living fleas has also been observed in India, and *X. brasiliensis*, their close genetic relative, fellow rat flea and efficient plague vector, found widely in South America, India and Africa,[39] has been found living on cereal debris accumulated in dark and unswept corners of straw huts in the present-day Democratic Republic of Congo.[40] Clearly, this is quite a general property for this species of flea. Querns or hand mills were also widely used for grinding corn for preparation of coarse bread or porridge and suchlike in medieval households all over medieval Europe, and cereal dust was common outside and inside houses.

Access to cereal debris is not an absolute condition for allowing infected rat fleas to live long and travel far in the absence of hosts. At temperatures of 10–15 °C in humid environments such as prevail at sea, *X. cheopis*, the most usual vector of plague, can survive in a very heavily infected state for at least 50 days. Also rat burrows will often have high humidity. Unfed black-rat fleas have been found alive in a deserted rat burrow after six months,[41] and, if infected by plague, would have infected rats that repopulated the burrow. It is highly improbable that the maximum longevity of unfed, infected rodent fleas is known.[42]

35 Pollitzer 1953: 323, 335.

36 Estrade 1935: 293–8. My translation from French. Hirst 1953: 322–3.

37 Pollitzer and Meyer 1961: 460–1.

38 Hirst 1953: 322–3, 331.

39 Miarinjara, Rogier, Harimalala, et al. 2016: 2207–8; https://en.wikipedia.org/wiki/Xenopsylla_brasiliensis.

40 Hirst 1953: 182–3; Pollitzer and Meyer 1961: 461.

41 The original research was performed by A. Machiavello. For the reference, see Pollitzer and Meyer 1961: 486 and 586, n. 74; Hirst 1953: 322.

42 IPRC, LXVII, 1914: 437; Hirst 1953: 322–3, 330; Benedictow 2010: 180.

At the time of the Black Death, there were two main types of ships engaged in international trade, ordinary round ships and galleys. Ordinary squarerigged round ships appear to have sailed on the average around 40 km/day in the Middle Ages, hence a voyage of 50 days corresponded to a distance of 2,000 km. At a temperature of 27°C, *X. cheopis* lived for 23 days and died infected; in another experiment specimens of *X. cheopis* kept under natural conditions transmitted plague after 29 days of starvation,[43] corresponding to transportation over distances of 920–1,160 km by round ships. In lower temperatures at sea, infected rat fleas can live much longer: at temperatures below 15°C, they can live in a very heavily infected state for at least 50 days.

Galleys moved much faster, over twice as fast, at an average sailing rate of about or more than 85 km/day and covered correspondingly longer stretches within these time horizons.[44] This also meant that infected rat fleas could make long voyages at sea after their rat hosts had died from plague. Galleys were huge ships with a usual crew of about 150 sailors and rowers. This meant that they carried large stores of food and had areas for the preparation and consumption of food. A single Italian galley in Southampton required 35,000 lb (15,740 kg) of biscuit for the homeward voyage,[45] also to the unintended satisfaction of the rat-flea stowaways. Galleys also had the space to accommodate several rat colonies. This would extend the process of rat mortality in a plague epizootic on board, before the rat fleas leaving dying or dead rats would have difficulties in finding new hosts, and expanded the period infected rat fleas could be under transportation. Storages of grain and cereal debris from preparation of food or crumbs of biscuits on the decks would allow infected rat fleas to travel at almost any distance at the time.

Modern plague researchers have made numerous observations of the long-distance spread of plague with shipments of grain or flour or rice by railway or by ship, which constitutes a large and compelling source of empirical material. The first plague cases in, for instance, Mumbai, Surabaya (Java) and Cairo were observed among grain merchants or bakers.[46] The importance of starving rat fleas' ability to sail long distances without access to cereal debris was supported by a huge amount of evidence and is also emphasized in all standards works on plague.[47] Textiles, raw cotton and raw wool have proved to be highly suitable environments for the long-distance transportation of starving infected rat fleas.[48]

Britons were alerted to the connection between the grain trade and the spread of plague at home: in 1910, a plague epidemic broke out in Latimer Cottages, a row of houses in the parish of Freston, five miles south of Ipswich, on the Shotley peninsula. Suddenly, people began to fall seriously ill and die, and it was thanks to an alert local family doctor that the epidemic was confined to a few cases.

Medical personnel have taken a keen interest in two other outbreaks of dramatic illness in the same area that they now suspect could have been plague cases. Again, long-distance

43 IPRC, LXVII, 1914a: 437; Hirst 1953: 324, 330–1; Pollitzer and Meyer 1961: 468.

44 See below, Ch. 11, pp. 154–7.

45 Rance 1896: 56.

46 Hirst 1953: 312–13, Wakil 1932: 24–30.

47 Benedictow 2010: 160–81; Liston 1924: 950–1, 997, 1001; C.Y. Wu 1936: 285–90; Hirst 1953: 311–16, 321–7, 330–1; Pollitzer 1954: 320–3, 334–5; Pollitzer and Meyer 1961: 469–70.

48 Hirst 1953: 310–20, 366–8.

transport of grain came to attention as the vehicle of spread of plague. Oceangoing ships on their way up the estuary leading to Ipswich would pause at anchor at Butterman's Bay to lighten their loads by transferring some of their cargo onto barges before proceeding upstream to Ipswich docks. Many of these ships were grain ships coming from distant foreign ports. This provided work for local labour and, hence, infected rat fleas could be brought ashore and trigger an epizootic in the local rat populations.[49]

However, the epidemiological point had been underscored, that plague travelled easily with shipments of grain at long distances. The importance of grain transport was again noted by American physicians in connection with the large plague epidemics during the Vietnam War when plague arrived with shipments of rice.[50]

6. The dating of outbreaks of the Black Death

The transition from the endemic phase of bubonic plague to the epidemic phase

The study of the spread of the Black Death is dependent on information in contemporary sources on the time of outbreaks. The sources do not provide exact dating of outbreaks but instead the time the presence of the Black Death was recognized by contemporaries and written down by, for instance, chroniclers, or the time an unusual number of deaths was registered in the records of manorial courts, which must be seen in relation to the time of the preceding court session, and so on. The approximate dating of the real beginning of an outbreak of plague requires a composite retrogressive analysis with the source-based dating as the time of departure.

It has been shown above that an outbreak of plague is preceded by a latency period running from the introduction of plague contagion in a rat colony to the first human death about 23 days later, followed by an endemic phase with a sprinkling of cases with increasing incidence, which translates into the early epidemic phase.

To develop into an epidemic, this process must be repeated by the spread of infective rat fleas to other rat colonies where the disease will develop in a similar way or rhythm. When a rat colony is seriously weakened by disease and death it will no longer be able to defend the food resources within its territory. Because of their huge fertility, rats and rat colonies always live on the brink of starvation and death, and fight tooth and nail to defend the food resources within their territory. For the same reason, a seriously weakened rat colony will be invaded by rats from neighbouring colonies, which will fight for the opportunity of feeding on their resources, and also on the carcasses of dead rats. Crucially in the present context, they will pick up infective rat fleas and introduce them in their own colonies. At the end of each epizootic process in a rat colony settled in a close human environment, infective and soon-starving rat fleas will swarm into the human habitat and leap onto humans who will be bitten but also carry the fleas in their clothing, luggage or merchandise to other houses and localities as they socialize or go about their business. Although the beginning of this process is insidious and slow, with only a sprinkling of human cases, it has has an inherently multiplicative struc-

[49] Zwanenberg 1970: 62–74; Howell and Ford 1986: 191–209. Because the house rat, i.e., the black rat, had disappeared and the brown rat, which lived at some distance from people, in sewers, cellars and as wood rats had supplanted it, contact between people and rat fleas was much reduced since medieval times, and plague took on an episodic and highly localized character.

[50] Marshall, Joy, Ai, et al. 1967: 604–5, 610; Butler 1984: 37.

ture of dynamics. After a few or some weeks, depending on the size of the community, it will present as sudden fulminating epidemic developments that would attract alarmed attention.

The significance of size of community

In the terminology of the sociology of medieval society, various types of human communities are defined according to size of population. Villages or townships have under 1,000 inhabitants, and hamlets some tens of inhabitants up to (perhaps) about 200 inhabitants; towns have between 1,000 and 10,000 inhabitants, cities between 10,000 and 100,000 inhabitants, and metropolises 100,000 inhabitants or more. The number of metropolises at the time of medieval maximum population around 1300 can probably be counted on one hand. One important reason is the staggering problem of providing predictable sufficient food provisions for population concentrations of this size under medieval conditions of supply and means of transportation.

Towns, cities and metropolises have a social structure and a social geography. Usually, plague broke out among the poor and, therefore, in the parts of urban communities where poor people crowded together in huts and hovels. In seaport centres on the coast or along big rivers that would usually be in the harbour area where the contagion also would usually arrive by ship. Elsewhere poor people usually lived in the northern or eastern areas of the urban geography, in big cities or metropolises, that might also be in outparishes, liberties or suburbs outside the walls. This is reflected in the time elapsing from the first human plague case to the epidemic developments that began to unfold in such dramatic form that it attracted the attention of the chronicle-producing social classes. Only then, information on the outbreak would be recorded, which might survive to posterity and be used by historians.

This lapse of time from introduction of plague to social recognition of a plague outbreak is not a constant but increases with the size of the urban community because it increases the territorial and social distance between the destitute and poor and the upper classes. These urban structures slow down and weaken the exchange of information between these social classes, including on epidemic outbreaks. Outbreaks of epidemic diseases among the poor would be quite usual events to which the upper classes would not pay much attention unless it developed a frightening form and until the spread made them feel threatened. Generalizing significantly, it will take 6 weeks in towns, 7–8 weeks in cities and 8–9 weeks in metropolises before the epidemic presence is acknowledged by the chronicle-producing social classes and produces evidence that is useful to historians. These are standard assumptions that in many cases are empirically testable by the sources, as will frequently be shown below in the presentation of the spread of the Black Death. This is, therefore, an important analytical tool applicable according to the rule of regularity inherent in standard assumptions.

7

Historical Presence and Role of Black Rats in the Black Death (and Later Plague Epidemics)

1. Denials of a normal presence and crucial role of rats in plague epidemics

The black rat was the only rat in Europe before the early 1700s. The conventional view on historical plague epidemics is that they were rat-and-rat-flea-borne. In the first edition of this book, the epidemiological analysis of the data reflecting the spread of the Black Death across Europe and adjacent regions of the Old World showed patterns of the epidemiological process that only were compatible with rat-and-rat-flea-borne bubonic plague. The analysis of all available material on the spread of the Black Death at the time was concordant with the conventional view, also as presented in the standard works on plague. The data on the spread of the Black Death available twenty years later and presented below provide maximum empirical evidence for inferences to the epidemiology of the Black Death, the mechanisms and dynamics of the transmission and dissemination of plague contagion.

The highly disparate and mutually incompatible alternative theories to the conventional rat-and-rat-flea-borne epidemiology of historical plague are quite consistently based on the same denial of the wide and usual presence of rats in medieval Europe.[1] It is disappointing that the thorough discussion of their counter-arguments[2] and the competent syntheses of finds of skeletal remains of black rats can be ignored or misrepresented. This also means that another endeavour to present the full evidence and discuss their arguments will not serve any purpose and this book is not the right forum for it. Readers who wish to consider for themselves all the skeletal evidence on the historical presence of rats and the inferences it supports can relate to the syntheses of the skeletal material mentioned below and referred to in notes 65–71. In this chapter, only the deniers' main arguments, some main features of the skeletal material, and some new evidence and understanding of the evidence will be considered or presented to formulate an updated status of research in the perspective of the Black Death. The crucial role of rats and their fleas will also be demonstrated by the peculiar epidemiological features it produces:

1 See above, Author's Note, pp. 6–10.

2 Benedictow 2010: 73–150; Benedictow 2016a: 395–443.

1. Some scholars point out that dead rats are not mentioned in contemporary sources in connection with historical plague epidemics. They assert that this is strong or decisive proof that rats were, if not absent, only weakly present and could not have played a significant role in the spread of the Black Death and subsequent plague epidemics.[3]
2. These scholars also assert, also as an explanation of point (1), that black rats cannot live in cold climates and could not have had a wide and dense presence in Europe for this reason. They could only have had a tiny presence in urban centres, especially seaports towns on the Mediterranean littoral where they could benefit from the warm climate and heating in human habitation during winters, and their purportedly tendentially dwindling numbers or extinction could only be reversed by importation by ship.[4] Almost all over Europe, about 95–85% of all people lived in the countryside. Clearly, without rats (and rat fleas) widely distributed in rural settlements of all kinds as peridomestic rats and commensal rats in people's houses, there could not be large-scale epidemics of bubonic plague. Therefore, the epidemic dynamics of the Black Death and later plague epidemics must have been based on a different mode of spread.
3. For the same basic reason, it is claimed that black rats do not burrow and do not have a separate existence as wild rats in woods and fields and elsewhere in nature.[5]
4. It is also claimed that black rats were not outcompeted by the stronger and more aggressive brown rats when they arrived in Europe in the early 1700s. It is purportedly wrong that brown rats combat, expel and kill black rats in the competition for food resources and suitable places for burrowing. Instead, the crucial point is that black rats purportedly had only a tiny presence.[6]
5. Only few skeletal remains of black rats have been found by archaeologists and almost exclusively in urban environments.[7]

It appears, therefore, useful to present a short outline of the history of the black rat in Europe that also addresses these arguments and paves the ground for a tenable analysis of the epidemiology of the Black Death and the plague epidemics of the past.

[3] Twigg 1984: 83, 75–112; Scott and Duncan 2001: 54–7, 359; Cohn 2002: 1, 8–54, 82, 133–4, cf. Benedictow 2010: 25–69, 74–7, 85–6, 93–8, 135–6, 144–50; Walløe 2008: 59–60; Hufthammer and Walløe 2010: 29–43; Hufthammer and Walløe 2013: 1753–6; Dean, Krauer, Walløe, et al. 2018: 1304; Dean, Krauer, and Schmid 2019: 1–3, cf. 'Great Britain: Report from Glasgow – Verification of Plague Cases reported in October 1907 –Examination of Rats for Plague-Infection', *Public Health Reports*, 23 (12), 20 March 1908: 357–9; Bramanti, Dean, Walløe, et al. 2019: 1–8.

[4] Shrewsbury 1971: 8; Twigg 1984: 57, 86–8, 99–100, 112, 218; Scott and Duncan 2001: 54–5, 359; Lodal 2007: 157–8; Hufthammer and Walløe 2010: 40–1; Hufthammer and and Walløe 2013: 1753. Although influential, the article by D.E. Davis 1986: 455–70, is not specifically referred to on any point for serious reasons, see Benedictow 2010: 98–116. These works have been thoroughly discussed in Benedictow 2010: 73–150, and Benedictow 2016a: 395–451.

[5] Hufthammer and Walløe 2013: 1753–4.

[6] Hufthammer and Walløe 2013: 1753–9.

[7] Hufthammer and Walløe 2013: 1752–9. This article has been thoroughly and critically discussed in Benedictow 2016a: 395–451. Some points made later will be addressed in this chapter.

2. Where have all the dead rats gone? The behavioural strategy of dying rats[8]

All standard works on plague and many individual studies of plague point out the scarcity or absence of rat falls found in association with modern plague epidemics (from 1894). This is concordant with the observation that historical sources do not mention rat falls in connection with plague epidemics. Some scholars and generally all advocates of alternative theories use, as mentioned, this observation as decisive proof that rats were absent or only slightly or passingly present in urban centres, mainly on the coast of the Mediterranean littoral. This inference is not made in the standard works on plague and in specific studies of the behavioural strategies of rats. The reason is already explained in the earliest modern plague research and presented in the standard works on plague and confirms the central methodological principle that absence of evidence is not evidence of absence.

In 1906, J.A. Thompson published a paper summarizing Australian research after bubonic plague appeared in Sydney in 1900 and the insights gained by the health authorities under his leadership as President of the Board of Health and Chief Medical Officer of the Government of New South Wales. He states that the reason some scholars thought that there were plague epidemics without rat-plague was that a 'proper search had not been performed'. 'Systematic detection of rat-plague is in reality a difficult business.' It was not until a rat intelligence staff had been trained and acquired practical experience in the search for plague rats that they were regularly found in the individual houses in which plague cases had occurred.

The crucial reason is that 'Rats eat each other in nature.' When rats become seriously ill or dying and cannot defend themselves, they try to hide away as best they can in order not to be eaten alive by their fellow rats. They tend to die out of sight in obscure and quite inaccessible places. Therefore, finds or reports of sick rats that had come into the open 'could be counted on the fingers'. Thompson goes on to enlarge on the observations:

> They do die in unusual places, and so regularly that we feel justified in regarding the discovery of three or four carcases at a similar stage of decomposition under floors, or in, or on the tops of cupboards, etc., during known presence of an epizoötic as probable evidence of death from plague. In ordinary, as is very well known, rats generally die out of sight […]. Even when poison has been laid discovery of several dead bodies in such situations should arouse suspicion.[9]

In their experimental godowns, IPRC often observed that rats dying or dead from plague were eaten.[10] The IPRC also studied rat-plague epizootics in selected localities in Mumbai and the Punjab. Again, their findings were similar to those reported by J.A. Thompson: 'In proportion to the severity of the epidemic the number of plague-rats found was very small, notwithstanding the very thorough and extensive search made.' The experience made in sever-

[8] This is only a brief summary of a thorough presentation of the topic in Benedictow 2010: 85–116; Benedictow 2016a: 411–18. Cf. McCormick 2003: 4–5, and n. 5.

[9] J.A. Thompson 1906: 548, 550–1. Cf. Hirst 1953: 147–8.

[10] IPRC 1910a: 316–17, 318, 321, 324, 326, 331; IPRC 1907a: 373–81; IPRC 1910c: 453–4, 456, 469; Lamb 1908: 33–6.

al areas 'points to the danger of concluding that plague-rats are absent from an infected locality unless a very thorough search is carried out'.[11]

When plague broke out in Colombo, Sri Lanka, in 1914, W.M. Philip and L.F. Hirst were sent to investigate the epidemic. Keeping J.A. Thompson's advice in mind, they organized a rat intelligence staff and set out to uncover the underlying plague epizootic. 'To begin with they found few dead rats in the open and conspicuous evidence of a rat epizootic was lacking.' To reveal the presence of dead plague rats it was usually necessary to open rat burrows and thoroughly dissect the tiled roofs of houses where nesting rats had hidden to die. As the rat intelligence staff 'gained experience, the correlation between rat and human plague in space and time became closer and closer'.[12]

Dutch scientists in Java met the same curious absence of rat falls and almost had to completely tear down native huts in order to reveal the rat nests and the presence of dead rats.[13] The results of research on rats in plague epidemics in Java in the early twentieth century are summarized by C.D. de Langen and A. Lichtenstein, the Dutch medical scholars:

> The most important argument adduced by the opponents of the 'Rat-Flea-Man' theory of the distribution of plague is that in various epidemics no rat plague has been observed, or the epizootic has only developed after the epidemic has started. A really critical examination of the data in these cases shows in every instance that no sufficient search was made for the preceding rat plague.[14]

Similarly, Wu Lien-Teh, the prominent Chinese plague researcher, pointed out: 'Modern examples could be quoted where not only the population at large but even the medical men believed rat plague to be absent and yet the presence of an epizootic was demonstrated by a proper search.' He rightly goes on to emphasize: 'In old Europe many factors militated against such discoveries.'[15]

Where rats nest in roofs/ceilings, they may occasionally fall down on the floor when they abruptly become severely ill from plague and attempt to find a hiding place; a few plague-diseased rats may not have time to hide before being overtaken by severe illness and die in the open. These are consistently only a few cases. Also when their classical ideals are disregarded, there is no reason that contemporaries should consider such odd observations highly peculiar or extraordinary, associated with the plague epidemic, and a topic for chronicles.

Against this backcloth, it must be concluded that the lack of mentioning of dead rats in chronicles and other narrative sources written during the Black Death and subsequent plague epidemics is what should be expected and does not at all prove or indicate the absence of rats. The crucial role of rats and rat fleas in the epidemiology historical plague epidemics must be shown on the basis of epidemiological features that cannot be explained by other mechanisms of transmission and dissemination, and can be supported by numerous and widespread finds of rat bones. Much epidemiological and material evidence will be presented below. One should also note the importance of specially trained personnel and the careful search for dead rats.

11 Lamb 1908: 18, cf. p. 24.
12 Hirst 1953: 148; Philip and Hirst 1917: 542–5.
13 van Loghem and Swellengrebel 1914: 467, 473. See also the photographs in the appendix.
14 de Langen and Lichtenstein 1936: 185–6.
15 Wu Lien-Teh 1936a: 8–9.

Appendix to Chapter 7.2

The reason that this topic is presented in such detail in a synthetic work on the Black Death is the continued endeavours of advocates of alternative theories to deny the wide presence and crucial role of rats and their fleas in the epidemiology of plague. It is an unpleasant duty to point out again that this view cannot be argued within the perimeter of the standards of scholarly work. For the sake of brevity and the unpleasantness of the task, only a couple of comments will be made to the recent endeavour by Bramanti, Dean, Walløe, and Stenseth which, nonetheless, should suffice. Referring initially to J.A. Thompson's article, they maintain that

> some researchers have argued that the authorities were unlikely to find plague-infected rats because they would go into hiding [51], thus differing in their behaviour from the rats in Hong Kong during the outbreak of 1894, which were described as dead 'in abundance on the streets and in the houses' [48, p. 311].[16]

This is a serious matter. The part of the citation, here rendered in bold type, is allegedly supported (reference no. 48, p. 311) by an 1894 article by 'G.F. Treille, A. Yersin', and decisively supported by an alleged quotation of a crucial part: 'the rats in Hong Kong during the outbreak of 1984 […] were described as dead 'in abundance on the streets and in the houses [48, p. 311]'. This is a bogus or concocted reference, is not stated in the paper and nothing like it, which on one and a half pages (= 310–11) renders a brief talk by Treille at a conference after consultation with Yersin. On the contrary, Treille's talk that reflects his and Yersin's observations in Hong Kong in that year agrees entirely with the rat-and-rat-flea-based theory of plague.[17]

3. To smell a plague rat or pestilential miasma

When severely ill rats die in obscure, unusual and quite inaccessible places they will after a few days begin to decompose and send off a telling fetid smell, and in the case of a huge epizootic among rats a pervasive putrid smell.

Plague cases were registered in Glasgow in the early 1900s. Inquiry had been maintained to discover plague-diseased rats but had failed to discover any since 1902. In August and October 1907, a girl and a boy fell ill by plague. About mid-October, offensive smells began to be perceived in the buildings of a police station, and on 29 October work began to ascertain the cause of the smell. This resulted in the discovery of 31 dead rats in various stages of decomposition in various rooms, also in the muster hall. Against the gable of the muster hall were built the stables of an adjoining bakery, and in a loft over the stables it was customary to keep grain and other feed for the horses. About a week before the cause of the smells in the police buildings began to be inquired into, about 20 dead rats had been found underneath the grain bags. They had the appearance of dying about the same time, and at a date apparently more recent than those found in the police buildings.[18]

16 Bramanti, Dean, Walløe, and Stenseth 2019: 5 and 7, col. 3.

17 https://hal-pasteur.archivews-ouvertes.fr/pasteur-00442093.

18 'Great Britain: Report from Glasgow – Verification of Plague Cases Reported in October 1907 – Examination of Rats for Plague-Infection, 1900–1907', *Public Health Reports*, 23 (1908): 357–9. Recently, a team of researchers published a paper on the outbreak of plague in Glasgow in 1900, arguing

Around 1900, the new microbiological science and associated new understanding of the spread of disease was quite widely accepted and had largely replaced the time-honoured classical Hippocratic–Galenic miasmatic epidemiology. The fetid smell of rotting flesh was recognized, evoked concrete suspicion of causation and directed relevant action for inquiry into the origin, which led to the findings of decomposing rats.

During the centuries of the Second Plague Pandemic the fetid smell of decomposing rats during a plague epidemic would be understood according to miasmatic epidemiology, as the smell of an especially poisonous and foul-smelling miasma from rotting matter that caused plague. Because people contracted plague from fleas that had left dead rats and after some days of starving would leap onto humans for feeds, the rooms, surroundings and environments of plague cases would coincide with well-hidden, dead and decomposing rats. Miasma was, as mentioned, understood as noxious vapours, which had emanated from decomposing organic matter or rotting flesh in the ground, was spread by air, and was the cause of epidemic disease by inhalation or personal contact.

Within the contemporary miasmatic framework of epidemiological understanding, fetid or putrid smell corroborated miasmatic assumptions. It gave no specific rational reason to suspect that the cause was decomposing rats that had died by plague and that rats had anything to do with plague disease and plague epidemics. Also the diseased could be assumed to smell badly because they contained plague-inducing miasma in their body and corrupted the surrounding air with foul miasmatic breath, vomit, bloody expectoration or body fluids, although the obnoxious smell came from the immediate surroundings. These logical inferences from miasmatic epidemiology and medicine are often found in local chroniclers' contemporary comments on local plague epidemics and were formulated by such words as putrid, putrefaction or fetid or foul-smelling, unbearable stench or corrupted air.

Michaele da Piazza, the anonymous author of a much-used (problematic) account on the spread of the Black Death in Sicily in the summer and autumn of 1347, related from Catania, quite likely his hometown, the clinical main features of the plague diseased as understood within the framework of miasmatic understanding of epidemic disease. He describes the growth of buboes,

> that became intensely painful, and by putrefying the humours[19] they forced the said human body to cough up blood and, in passing from the infected lungs into the throat, corrupted the whole body [and showed that] the whole human body was putrified. After this putrefaction […] the diseased died.[20]

The key words are 'putrefying', 'putrified' and 'putrefaction', referring to the effects of miasmatic invasion of the body as reflected in the pungent putrid smell associated with the diseased and their surroundings.

that plague was transmitted by human fleas and lice because no plague rats had been found. Clearly, the chronological delimitation serves a purpose. Dean, Krauer, and Schmid 2019: 1–10.

19 The term 'humours' refers to the classical humoral theory of Hippocratic–Galenic medicine and epidemiology that the body was composed of four humours (fluids) – blood, phlegm, yellow bile and black bile – that must be in balance if the body should stay healthy. This is also the background of bloodletting or bleeding to cure illness and disease.

20 Michele da Piazza 1791: 567. My translation from Latin, see also Horrox 1994: 40; Aberth 2005: 30.

In Narbonne, where the Black Death broke out in early March 1348, the chronicler described the 'unendurable smell that reigned around the diseased, especially among those who had an intense lung inflammation with sudden and lethal expectoration of blood'.[21] Clearly, the chronicler associated the unbearable smell with miasmatic emanations from the breath and bloody expectoration of terribly ill and dying persons.

Gabriele di Mussis described plague disease in his hometown of Piacenza about the same time: it was characterized by the growth of painful buboes and 'acute and putrid fever', referring to a fetid smell similar to rotting flesh, assuming by implication that it was due to miasmatic emanations from the diseased body.[22]

Likewise, Abbot Gilles le Muisit of Tournai stated in his chronicle that 'of those who drank wine, avoided corrupted air and visiting the diseased, few or none died'.[23] He clearly implies a close link between corrupted air ('malo aere') and the visiting of the diseased and contraction of plague. His advice was good, for entirely different reasons, since by avoiding fetid miasmatic smells people unknowingly avoided environments with rotting rats and, more importantly, their fleas on the hunt for a feed.

In Bohemia, Francis of Prague (Franciscus Pragensis) stated about the Black Death that 'In many places, too, the air was more infected and more deadly than poisoned food, from the corruption of the corpses because there was no one left to bury them.'[24] Here the chronicler clearly implies that the air was poisoned and foul-smelling from the corruption of unburied human corpses, the most dangerous source of miasma.

Konrad von Megenberg (1309–1374), the German philosopher, theologian and naturalist, included in his pioneering *Book of Nature* a chapter on the nature of epidemics in a miasmatic perspective. This included the usual contemporary view that miasma could be released from the earth and mountains by earthquakes, volcanic activity and specific constellations of planets, which were medieval additions to the original Hippocratic–Galenic miasmatic epidemiology. He performs a concrete miasmatic analysis of the spread of the Black Death on the basis of its ravages in Austria and Vienna, and states (wrongly) that it spread from there to Bavaria and Passau and that the disastrous mortality was due to the 'poisoned air'.[25]

In modern Norwegian, the idiomatic expression 'it smells pestilenced (here)'[26] is still frequently used about fetid smells, although the original meaning is lost.

4. Black rats in cold climates

The presence of black rats in the fossil record and in the Neolithic period

It is usually overlooked that the fossil remains of a 'very similar species' to black rats have been found in Pliocene strata (5.3–2.58 mya) in Lombardy, in Quaternary strata (2.59–1.8 mya) near Pisa and, in Crete, Bohemia and near Geneva in Pleistocene strata (1.8 mya–11,000 BP),[27]

21 Cayla 1906: 46. Cf. Sticker 1908: 64.

22 Gabriele de Mussis 1842: 55. English translation in Horrox 1994: 24.

23 *Annales de Gilles le Muisit* 1906: 254–8. Translation from another edition but similar text by Horrox 1994: 51–4.

24 *Continuatio Francisci Pragensis*, 1875: 597. Translation from Latin by Gasquet 1908: 37–8.

25 Konrad von Megenbert 1861: 107–13.

26 'Det lukter forpesta her'.

27 Donaldson 1924: 7, n. 1; Hirst 1953: 126; Rackham 1979: 112.

a period covering several ice ages.[28] Although dating of the fossils within these long periods is not given, the time horizon is so long that the black rat or a closely related species apparently has a long history in Europe, retreating with glaciations and returning in the wake of their withdrawals, much like reindeer, bears, wolves and many small mammals.

Hence, the evidence indicates that a species similar to the black rat was an ordinary part of European fauna long before humans turned up. Evidently, they were well adapted to northerly climates, and they did not have any choice: they had to be burrowing and also to thrive in cold climate in the absence of human housing and heating. This perspective can usefully be linked to Tchernov's view that the black rat was present in the coastal region of present-day northern Israel in the late Mesolithic period about 9,500–7,500 BCE and that it was a native species of the southern Levant where it had lived since the end of the Pleistocene.[29] This indicates that the black rat was part of the original late Pleistocene fauna over large parts of Europe and adjacent regions in North Africa and the Middle East, and had adapted to the climate of the glacial periods, retreated with the expansion of glaciers and returned when they retreated.

The notion of a long presence in Northern Europe is supported by the fact that numerous bones of black rats have been found in the remains of the Neolithic pile dwellings in the north-eastern German region of Mecklenburg from about 4,000 to 2,500 BP and in western Germany.[30] Skeletal remains of black rats have also been found at late Bronze Age pile dwellings at Lake Neuchâtel in eastern Switzerland,[31] and in the territory of modern Hungary.[32] It seems that the black rat (quite) generally was present at the Neolithic/Bronze Age pile dwellings in Central and North-eastern Europe. The finds of bones of the black rat from the Bronze Age in caves in central Italy should not come as a surprise and be deeply mistrusted as much later incursions into the deposits, although they look exactly like other skeletal material at the site from this period.[33]

The shinbone of a black rat dated to the Hallstatt period, 3,200–2,470 BP, was found in Henneberg[34] in southern Thuringia. Highly interestingly, in Henneberg finds of skeletal remains of the black rat were continuous from the Hallstatt period to the late Middle Ages.[35] The apparent identity of the skeletal finds at Henneberg as remains of the black rat over this period of at least 2,000–3,000 years is clear,[36] and serves as evidence of a continuous presence of the black rat in Europe.

28 On the presence and development of rats in this period, see Aplin, Suzuki, Chinen, et al. 2011: 2, 9–10, 14.

29 Tchernov 1984: 91–115. Cf. Armitage 1994: 232 where uneasy unsubstantiated doubt is indicated.

30 Donaldson 1924: 7, n. 1; Hirst 1953: 126; Sharp and Villano 2013: 1.

31 Roguin and Studer 1991: 79–83.

32 Stampfli 1965–6: 454.

33 Salari 2014: 89–90, 93. There is a tendency to consider the earliest finds of rat bones in Southern Europe as the earliest arrivals. See, e.g., Audoin-Rouzeau et Vigne 1994: 127, 132–5; Pascal and Vigne 2003: 320–3. Methodologically they are only evidence of the (preliminarily) earliest presence in the find area.

34 Bartel 2017a: 72; Hans-Volker 2017: 227–8, 239–40.

35 Bartel 2017a: 72, 76; Bartel 2017b: 83–4, 88; Spazier 2017b: 97, 99, 101, 104–5, 119; Hans-Volker 2017: 228, 239–40.

36 Hans-Volker 2017: 240.

This is supported by the fact that rat bones from about 2,500 BP was found in the Slavic village of Smuszewo in north-western central Poland and also in the then Baltic village of Tolkmicko in north-eastern Poland on the Vistula Lagoon (Zalew Wiślany) and the Baltic Sea. In Poland, finds of the bones of the black rat were made in several locations from the early Middle Ages and continuously later.[37] The find of rat bones from 2,500 BP in Tolkmicko on the Vistula Lagoon in the south-eastern corner of the Baltic Sea can usefully be seen in connection with finds of Stone Age rat bones in Gotland I.,[38] 275 km north-west of Tolkmicko.

This material indicates the presence of the black rat over a huge area from eastern Central Europe to the Baltic area in the Neolithic period and puts the Pliocene Quaternary and Pleistocene fossil finds in a highly interesting perspective. The view that black rats are dependent on a warm climate and close association with heated human habitation is evidently not correct.

Neolithic finds of the remains of black rats have also been made in Southern Europe, for instance, from 5,500 BP (3,500 BCE) in Sardinia, and from the late Bronze Age in north-eastern Andalusia in Spain.[39] One could also include finds of rat bones from ab. 2400–2170 BP made in the western Mediterranean islands of Corsica and Minorca and in Pompeii[40] on the coast of the Italian mainland near Naples. Taken together, these data indicate a widespread and continuous European presence of the black rat dating back to the Pleistocene period, with continuity into the Neolithic period and the Bronze Age and further into subsequent periods of European history. These data suggest that the history of the black rat in Europe will be quite radically rewritten and this is only the first harbinger.

The evidence also indicates that this species of black rat rapidly found a new and attractive ecosystem with the arrival of humans and their settlements, housing and outbuildings, and arable and animal husbandry with storage of produce and the opportunity for sheltered nesting. The close association with the pile-dwelling culture is symptomatic. As pointed out by Hirst, this indicates that 'rats were associated with man since his first cultural beginnings, since the time when he first began to store large quantities of cereals in granaries'. This also means that rats did not follow in the wake of humans, they were present and used the opportunities that the arrival of humans offered. Zoomicrobiological research on the biogeographic and phylogeographic[41] history of black rats corroborates this information.[42]

Quite likely, the find of bones of the black rat in Magdalensberg near Klagenfurt in southern Austria from around the turn of BCE and CE should be seen in this context. Magdalensberg was (probably) the royal capital of the local Celtic kingdom of Noricum, which was an ally of Rome from the time of the Roman Republic. Magdalensberg also had strong commercial functions and traded intensively with Rome, especially with local iron, gold and agricultural produce.

37 Teichert 1985: 265–7; Sorge 1996: 389, 391.

38 Personal communication in email of 20 June 2006 from Gustav Malmborg, the Swedish zoo-paleoosteologist at University of Uppsala, Campus Gotland.

39 Audoin-Rouzeau 1994: 129.

40 Vigne and Valladas 1996: 199–215; Audoin-Rouzeau 1999: 423.

41 Phylogeography is the study of the historical processes that may be responsible for the contemporary geographic distributions of individuals. This is mainly performed by considering the geographic distribution of individuals in light of population genetics.

42 Aplin, Suzuki, Chinenet, et al. 2011: 14.

The trading centre of Magdalensberg consisted, therefore, of two parts, a native Celtic town on the hilltop and a Roman trading colony that had grown up at the foot of the mountain from early in the first century BCE. Significantly, the find of a rat bone was made in the Celtic town and not in the Roman settlement.[43] Also because agricultural produce was exported from Noricum to Italy or to feed Roman legions and not imported, the likelihood is that the rats were of local origin and fauna and rather would be exported from Magdalensberg than imported. With the development of political and commercial functions the resultant growth of urban structures produced a new and attractive ecosystem for the local rats. Significantly, the find in Magdalensberg is at least 200 years older than the find of rat bones in the big Roman camp for Legion II at Lorch (Lauriacum) on the Danube, which was established about 200 CE.

In this perspective should, presumably, also be seen the finds of rat bones in Germanic and Slavic settlements in eastern Germany, from 200 to 400 CE in a Germanic settlement in Waltersdorf near modern Berlin and, from the late 300s, in Magdeburg, 125 km west of Berlin.[44] A find in Szczecin (Stettin) is dated to the 4th–8th centuries while it still was a Slavic peasant village.[45]

This view is supported by the fact finds of rat bones from the Roman or medieval period have not been found in a wide swathe of land of 350–400 km across stretching south-eastwards roughly from the rivers Elbe and Oder with the towns of Wrocław and Opole in Silesia in modern south-western Poland to about the Roman defensive line along the rivers Rhine and Danube.[46] This is said with the reservation that a local stock of rats may seem to have been present also in Switzerland and Austria.

This evidence indicates that black rats have been evolutionarily adapted to a wild life in northerly climates and cold winters, and this should be innate properties of the species.

Black rats in Macquarie Island

With respect to the purported delicacy of black rats in relation to climate and their dependence on heated human housing in chilly climates it is a highly relevant fact that burrowing black rats long were thriving in severe subantarctic conditions on the uninhabited Macquarie Island (latitude 54.30°S, longitude 159°E), not far from Antarctica.[47] For geophysical reasons, the temperatures in this region are several degrees Centrigrade lower than at equivalent latitude(s) in the northern hemisphere,[48] and there is no equivalent of the Gulf Stream. According to systematic meteorological measurements of temperatures in the period 1948–2005, the average temperatures are below 0° C for 8–9 months of the year, and in the months of April 1948–2005 average temperatures varied between +6 and -8° C.[49]

43 Teichert 1985: 265; https://en.wikipedia.org/wiki/Magdalensberg.

44 Teichert 1985: 263, 265–6.

45 Teichert 1985: 266–7.

46 Teichert 1985: 266; Sorge 1995: 389.

47 Pye, Swain, and Seppelt 1999: 269–87.

48 The Earth's orbit is elliptical, five million km closer at the periapsis (3 January) in the northern hemisphere than at the apoapsis (3 July) in the southern hemisphere. https://en.wikipedia.org/wiki/Earth%27s_orbit.

49 http://www.environment, gov.au/node 22358/Macquarie Island. Source: Australian Government, Department of the Environment and Heritage 2006, Indicator 1 – Monthly mean air temperatures at Australian Antarctic Stations. See http://aadc-maps.aad.gov.au/aadc/soe/display_indicator.cfm?soe_id=1.

The zoologists who studied these rats summarized these black rats' ability to colonize this environment successfully thus:

> The species was introduced to this subantarctic island by sealers during the 19th century. The rats are now widespread and abundant in coastal areas all around the island. The distribution of rat populations is divided into discrete units by the availability of suitable habitat which, in turn, is a consequence of the rugged topography, particularly on the west coast. Rats are found from almost sea level to 200–250 m a.s.l.[50] and up to 1 km inland. They have adapted successfully to the rigorous climate and firmly occupy a habitat niche in an environment where food is plentiful, predators are few and interspecific competition minimal.[51]

Norway is rightly considered a cold northern country with long winters, much snow and ice and cold winds, which should be suitable for comparison, especially with respect to coastal climates. In the period 1971–2000, the average monthly temperature in Macquarie Island was +0.1° C. This is significantly lower than in the town of Tromsø in the far north of northern Norway, latitude 69°40´N where the average monthly temperature in the period 1870–2014 was +2.54° C.[52] Hammerfest is the northernmost town in Norway, situated on the north-western coast of Norway's northernmost county of Finnmark, latitude 70°70´N. There, the average monthly temperature in the chronologically comparable period 1961–1990 was +1.9° C, and in the period 1985–2104 + 0.7° C.[53] There is no significant locality with a meteorological record along the northern coasts of Norway with an equally low or cold annual average temperature as in Macquarie Island. Nonetheless, black rats burrowed, thrived and multiplied and spread across this cold, often ice-cold, windswept island with much and frequent rain and snow. Because these rats were considered pests engendering much environmental damage, among other things, predation on chicks and eggs of sea birds, a huge eradication programme was implemented in 2011 and declared successful in 2014.

These data make it inexplicable that black rats could not burrow all over Norway, for instance, much in the same way as in Macquarie Island. These data can explain that, according to an Icelandic chronicle and the few contemporary sources extant from medieval northern Norway and the 1500s, plague epidemics raged there in 1391 and 1600–1.[54]

The fact that a few specimens of black rats that had slunk ashore from a ship visiting Macquarie Island succeeded in establishing a numerous and continuous presence, demonstrates the black rat's remarkable resilience and hardiness. The rats' establishment in the island would have to be immediate without time for evolutionary adaptation. It shows that black rats have, in fact, an innate ability to tolerate cold climates that must be physically deeply embedded by

50 above sea level.

51 Pye, Swain, and Seppelt 1999: 269. Cf. McCormick 2003: 22–3, n. 36.

52 http://www.yr.no/sted/Norge/Troms/Troms%C3%B8/Troms%C3%B8/klima.måned02.html. In the last decade, global warming (also recorded in Macquarie I.) has increased the average monthly temperature to + 3.2°C.

53 http://www.yr.no/sted/Norge/Finnmark/Hammerfest; http://www.wikipedia.org/wiki/Hammer-fest.

54 Benedictow 2002: 116–17, 234, 240–3; *Lawmaster's Annal* 1888/1978: 284.

a long evolutionary history, as has been indicated above for European black rats (from which also black rats in the Americas are descended).

Except, as it seems, for parts of North-eastern Europe and the Baltic area and some other areas, black rats were, as will be shown below, reintroduced and spread over large parts of Europe by Roman legions and their huge trains to military settlements whence they were spread further by ship and trade across Europe. This took place over a period of over a thousand years, which also introduces a long-term evolutionary perspective. In this period, black rats with their enormous fertility and short generational cycles would have the opportunity to adapt by degree to chillier or colder climates as they were transported northwards in Europe over the centuries.[55] But then again, this evolutionary perspective may not be important: the rats that slunk ashore on Macquarie Island were able to deal spontaneously with the climate.

The same would be the case with their consort of fleas, with even much higher fertility and faster generational change. 'As shown by its wide geographical distribution, *X. cheopis* is able to adapt itself to a considerable range of climatic conditions. It is […] also the common rat-flea in Manchuria.'[56] As fur fleas, they enjoy the microclimate of the coat and easy access to feeding. This evolutionary perspective explains that, 'in Peru, *X. cheopis* could remain [plague] infected in empty rat burrows for periods of up to six months or even longer'.[57]

5. General comments on the presence of black rats in the Nordic countries[58]

The history of black rats in the Nordic countries[59] demonstrates the strong tolerance in this species for cold climates and long, harsh winters and their quite general presence, also as a wild rat in sparsely populated countries. One should consider that winters in Sweden and Finland generally are significantly colder than even in Norway because the Gulf Stream's flow along the coasts of Norway raises the average temperature there about 10°C above that in regions situated elsewhere at the same northerly latitudes.[60] Nonetheless, black rats must have been widespread also in northerly counties of Sweden and in Finland, making it clear that the climate would not prevent black rats from burrowing all over Norway.

The Swedish Species Information Centre presents, on its website 'Artfakta – Rattus rattus', a map, see Map 3, showing the historical presence of the black rat in Sweden up to the 1790s and with a text making it clear that the arrival of the brown rat at this time was the cause of its sudden and rapid decline and replacement:

55 Benedictow 2010: 98–122.

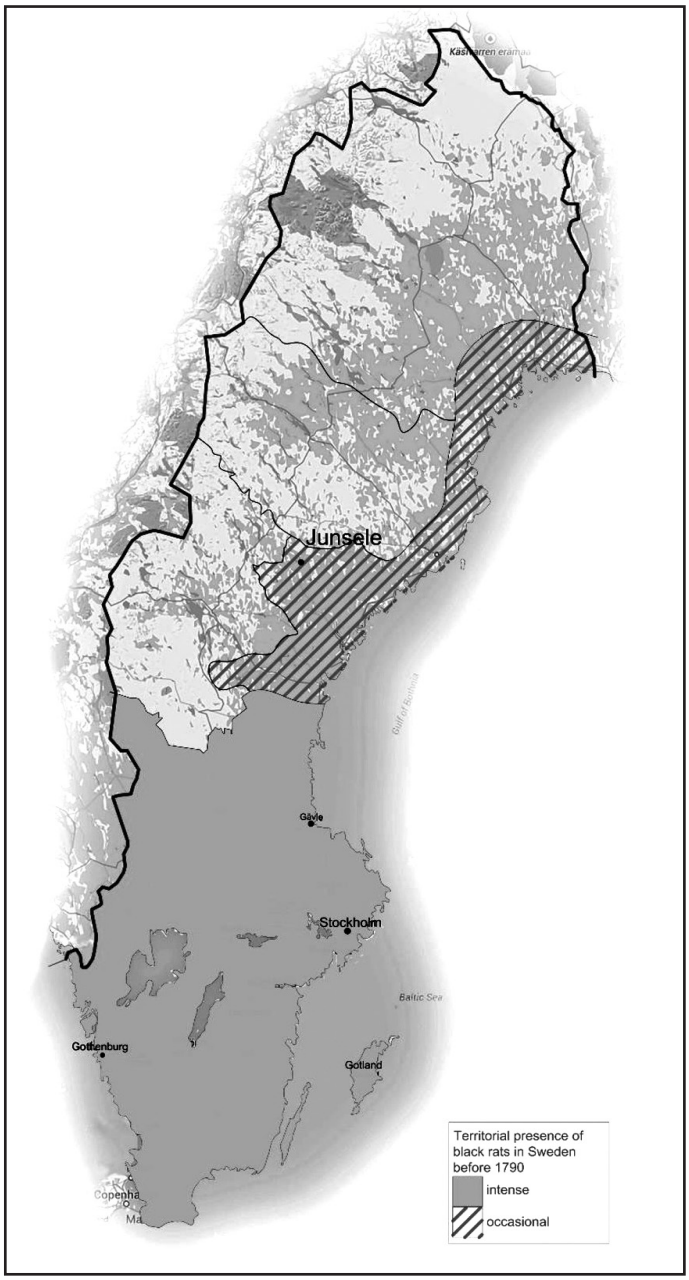
56 Pollitzer 1954: 327.

57 Pollitzer and Meyer 1961: 468.

58 With some additions and reflections, this short chapter gives the gist of Benedictow 2016a: Ch. 8, pp. 395–451.

59 Finland is formally geographically not a Scandinavian country (Sweden, Norway, and Denmark) but is included in the term Nordic countries.

60 Eldevik 2006: 48. Eldevik is research leader at the G.C. Rieber Climate Institute at the Nansen Environmental and Remote Sensing Centre and Bjerknes Centre for Climate Research. Mean temperature in these parts of Norway is 10 °C higher than in other regions situated at the same northerly latitudes. Benedictow 2010: 139 n. 213, 418.



Map 3. The presence of black rats in Sweden before the 1790s. Based on a map on Swedish Species Information Centre's website, supplemented by comments by M. Tjärnberg at the Centre (who updated the site in 2010) and the author's corrections and updates.

> The black rat has formerly been generally present over southern and central Sweden up to [i.e., including the historical provinces of] Hälsingland and Ångermanland [in northern Sweden]. Only 30 years after the arrival and spread in the country of the brown rat, *Rattus norvegicus*, from the 1790s, the black rat was said to become all the more rare. Around 1890 there were only scattered populations left of the species' formerly contiguous territorial presence, remainder populations in parts of Hälsingland, and in northern Halland with adjacent parts of Västergötland (West Gothland). The black rat disappeared from the latter area around 1940, while it was still present in Arbrå parish in Hälsingland in 1951.[61]

Map 3 shows widespread registrations of local populations of the black rat in northern Sweden: in Medelpad, a region of Västernorrland County about 400 km north of Stockholm at 62°24'N, and – as it seems – far further north along the western coast of the Gulf of Bothnia. There was not only a coastal connection by ship but also a main trackway running along the coast all the way via Luleå (Gammelstaden) and Piteå to the modern border with Finland.[62]

The associated footnoted Factsheet[63] enlarges on this information. Among other things, a mummified black rat was found in the remote rural settlement of Junsele, deep in the north-western part of the county of Västernorrland (Ångermanland), quite near the border with the (then Norwegian) county of Jämtland, very sparsely populated regions of vast forests and widely dispersed small settlements. It is hard to envisage that this was an incidental find of a rat that was on its own in this vast area.

The zoologists at the Swedish Species Information Centre emphasize these points: (1) that up to the 1790s, there was a contiguous territorial rural presence of the black rat all over the modern territory of Sweden including Hälsingland and Ångermanland in northern Sweden;[64] (2) that the rapid retreat and disappearance of the black rat was caused by the arrival of the brown rat in the 1790s, which outcompeted the black rat. On this point, they agree with Nybelin and Bernström, the pioneering Swedish zoologists, and with Swedish zoo-paleoosteologists;[65] (3) that the process was fast: thirty years after the brown rat's arrival, around 1825, it was noticed that black rats had become quite scarce and were in retreat. In the late 1800s, black rats were still quite numerous in a few rural areas in northern, central and southern Sweden[66] but in continuous decline. The skeletal evidence on the presence of black rats in medieval Sweden is presented below as case history II in subchapter 7.

In Finland, rats had arrived at some time in the high Middle Ages with Swedish migrant peasants who travelled via Stockholm and the archipelago, which stretches (almost) continuously over to the south-western coast of Finland.[67] Around 1300, Finland had only one small

61 http://www.artdatabanken.se, version in English.

62 See map showing Swedish medieval trackways, Schück 1934B: 240b, and below, p. 475, Map 10.

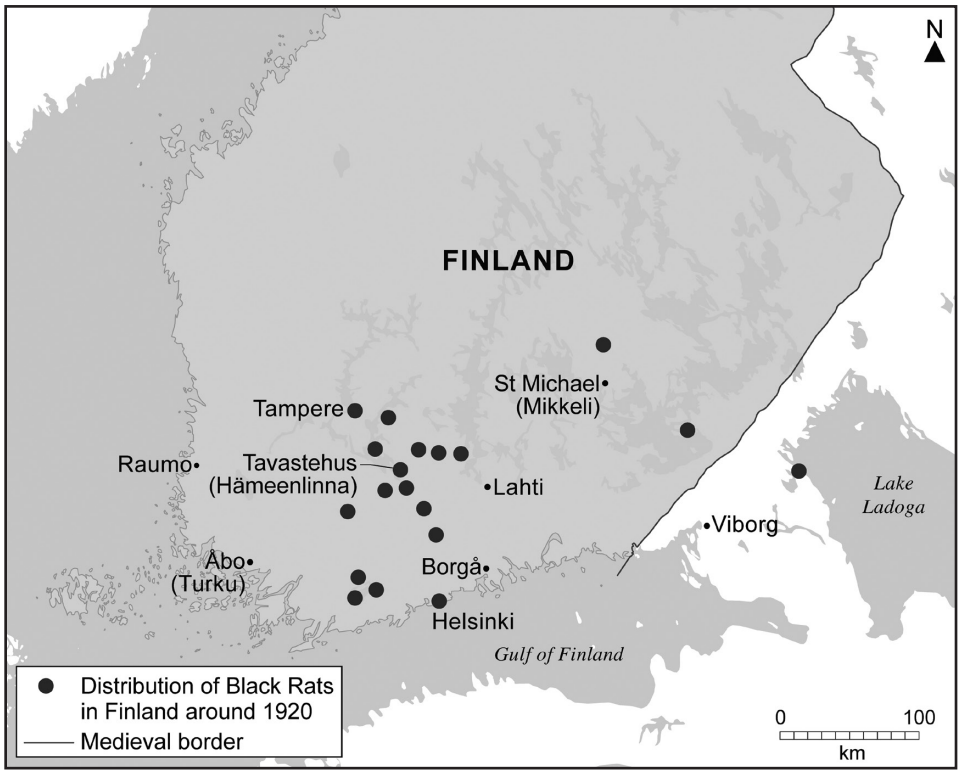
63 The references in the footnotes are rendered in Benedictow 2016a: 441, n. 1252.

64 Cf. Bernström 1969: 581.

65 Nybelin 1928: 853–5; Bernström 1969: 578–9; Vretemark 1982: 294; Vretemark 2009: 5; Vretemark 2010: 10, 31; Pettersson: 31; Malmborg, personal communication by email, 20 June 2006; Benedictow 2016a: 405–7.

66 See Benedictow 2016a: Map 8.1 on p. 437: 'Distribution of black rats in Sweden in the late 1800s.'

67 Vilkuna 1969: 583–4.



Map 4. The presence of black rats in Finland around 1920

town and a total population of some 65,000 inhabitants,[68] mainly settled in the southerly parts. The presence of rats in the densely forested and extremely sparsely settled country must have been almost entirely rural, which also shows in early-modern registrations. The medieval ancestors of these black rats must have been comfortable with an existence as wild burrowing wood rats and in widely scattered rural settlements in regions with very cold winters, even colder than in Sweden. In a country with almost no urban structures, they had readily spread inland from the south-western coast and northwards and eastwards into the very sparsely settled deep forests of Finland, all the way to and across the present border with Russia.[69]

The brown rat arrived in Finland at about the same time as in Sweden, in the 1790s, which suggests that it occurred from Stockholm. Even in the 1920s, there was a significant presence of black rats in the densely forested and sparsely settled rural parts of south-central Finland between 60 and 62°N, see Map 4. These areas can certainly be adequately designated 'remotely situated districts'.[70] They must represent the remaining populations of a formerly widespread and quite continuous presence of black rats across Finland, well adapted to a burrowing life

68 See below, Ch. 32.4, and n. 7.

69 Nybelin 1928: 854.

70 Vilkuna 1969: 584. My translation from Swedish.

in a harsh winter climate. At these outposts of settlement, they would be confronted by brown rats with a considerable delay.

In Norway, medieval skeletal remains of black rats have been found in all major towns including Trondheim (Nidaros) at 63°25′N (about 8 latitude degrees more northerly than Moscow). Skeletal remains have also been found at two rural settlements, an uninhabited subsidiary farm of a monastery and in a medieval peasant holding (but the find dates probably from about 1600).[71] Finds of rat bones in agricultural holdings require total archaeological excavations of the farmsteads in order to recover skeletal remains where rats hid to die. Such excavations are few and the history of zoo-paleoosteology is quite short in Norway.[72] In the present context, it is significant to note that one of the rural finds was made on an uninhabited subsidiary holding on an uninhabited island in the inner Oslofjord. There were only simple unheated sheds for keeping of agricultural tools and storing of some agricultural produce. The other rural find was made in the rural heartland of Vestfold County.

6. Do black rats burrow?

In the previous chapters, it has been shown that a species of rats similar to black rats lived in cold areas of Europe long before humans arrived and that burrowing black rats were thriving in severe subantarctic conditions in the uninhabited Macquarie Island not far from Antarctica but far from humans. Black rats were also burrowing and thriving far away from human housing in the chilly and windy north Atlantic climate of the virtually uninhabited Shiant Isles in the Outer Hebrides (Scotland) until recently when an eradication programme for the protection of seabirds was successfully implemented.[73] However, black rats are still living on the chilly, windswept and uninhabited Westray Island, outermost in the Orkney archipelago.[74] 'Here, without competition from its brown relative, […] they have adapted to habitats which are probably essentially little different to those of the post-Roman period.' Skeletal remains of black rats from 800–1100 CE have been found on the Brough of Birsay, a small island outside the northern tip of the Mainland Island of Orkney (Scotland).[75] Black rats were not only present in warm urban housing but as wild burrowing animals on the chilly and windswept northern outskirts of Britain.

The fact that black rats like to live in burrows is also reflected in that 'the available evidence' shows that its usual flea consort, *X. cheopis*, has an 'affinity for rats living underground rather than for those sheltering on higher levels of the houses'.[76]

The notion that black rats could only live in heated housing and in close association with humans and (nearly) exclusively in urban centres reflects the environments and sites where archaeologists often perform excavations and make their finds. True, to look for medieval rat burrows in forests and other uninhabited areas would be a well-nigh impossible task. At the heart of the problem is a methodological principle: that absence of evidence is not evidence of

71 Benedictow 2016a: 430–3.

72 It has also been misrepresented: Hufthammer and Walløe 2010: 29–43; Hufthammer and Walløe 2013: 1752–9. See Benedictow 2016a: Ch. 8, pp. 395–451.

73 Key, Fielding, Goulding, et al. 1998: 228–33; McCormick 1998: 223.

74 Dobney and Harwood 1998: 377.

75 Audoin-Rouzeau and Vigne 1994: 130.

76 Pollitzer 1954: 336.