The Complete History of Plague in Norway, 1348-1654

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The Second Pandemic

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For my sons
ANDREAS AND TANCRED
and my lovely grandchildren

and in memory of
PROFESSOR SIGVALD HASUND
who discovered the late-medieval crisis
and laid the foundations of the
Norwegian School of Agricultural History

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PREFACE

SOME CENTRAL PERSPECTIVES AND PROBLEMS

1. Why plague can be important in the study of history

A disease can be historically important only for its mortality effects. It must be a communicable disease that combines tremendous powers of spread with huge lethality rates. Sharp and long-run reductions(s) of populations caused by recurrent epidemic disease with such properties can engender profound changes and transformations of basic structures of societies and civilizations. Such events can also change the balance of power and cultural exchange between ravaged and not ravaged societies and civilizations. Thus, epidemic disease can unleash long-run societal changes that can shape or hasten the advent of new social formations (social systems) and mould central lines and directions of historical developments; in short, make history.

Bubonic plague is the only disease studied and discussed in this perspective. The primary pneumonic droplet-spread mode of plague that may arise from cases of bacteraemic bubonic plague (with plague bacteria in the blood stream) has poor communicability and is of no consequence in this context. (Clearly, the sudden introduction of many new serious but less lethal diseases to a region may engender much the same effects, as happened in Mesoamerica in the wake of the Spanish conquest.)

This book is the history of the Second Plague Pandemic in Norway.

2. The study of the plague history of Norway

Territorially, the medieval[1] and early modern Kingdom of Norway included several areas that were lost in the later period of the Second Plague

[1] In Norwegian scholarly writing of history, the term medieval refers to the period following the Viking Period that ended around 1050 and that lasted to 1536 when the independent Norwegian kingdom became a dependency of the Kingdom of Denmark and lost national political centres such as king and Council of the Realm.

Pandemic studied in this book, 1348–1654, or a few years later. The Orkneys and Shetland formally belonged to the Realm of Norway until the end of the 1460s when the archipelagos were integrated in the Kingdom of Scotland. Continental Norway included the south-easternmost province of Bohuslēn and the very sparsely populated east-central province of Jemtland that protruded deeply into Sweden and were conquered by Sweden in the mid-1600s. Greenland, Iceland and the Faroes became dependencies of Denmark in 1814, from which Iceland gained independence in 1944. This is the territorial framework of the history of plague in Norway. Greenland and Iceland were not ravaged by plague. The disappearance of Norse settlement in Greenland and the two serious epidemics in Iceland in the 1400s that have been assumed to be caused by plague must have other causes.[2]

The source material for studies on plague epidemics in Norway must more generally be characterized as quite weak, especially in the 1400s. Comparatively, it is better than in other Scandinavian countries.

The national historical problem associated with the Norwegian state's decline and fall in the late Middle Ages made Norwegian historians eager to focus on this historical period. From the breakthrough of scholarly writing of history in the 1830s, Norwegian historians attempted to find political explanations. In 1920, there was a new breakthrough with a booklet published by Sigvald Hasund, professor of agricultural history at the then University of Agriculture (NLH). He developed source-critical and technical means that enabled him to show the onset of an abrupt and sharp fall in settlement and consequently in population size in the wake of the Black Death, and that this was an enduring situation that deepened throughout the period. He pointed out that the Black Death and the ensuing plague epidemics were likely the explanation.

This led to the formation of the Norwegian School of Agrarian History with other pioneers such as A. Steinnes, A. Holmsen, and, later, J. Sandnes. Their studies expanded on and confirmed Hasund's observations. In the wake of the Black Death, they found a huge contraction of settlement and agrarian production and a corresponding enormous fall in rents and fines payable by tenants, facts reflected in a steep fall in the value of landed property. This caused a huge fall in the incomes of the king or the Norwegian state, and of the ruling classes, the big landowners of noblemen and prelates. All these observations were consistent with the

[2] Benedictow, Ole J. 2010. Which Disease was Plague? *On the Controversy over the Microbiological Identity of Plague epidemics of the Past*. Leiden: Brills, 98–9, 502–50; Benedictow, Ole J. 2021. *The Complete History of the Black Death*. Woodbridge: The Boydell Press, 616–17.

social effects of a dramatic fall in population size and established a demographic paradigm of explanation that from the beginning was associated closely with the Black Death. The Norwegian Agrarian School of History established a radically new demographic and economic explanation of the decline and fall of the Norwegian state in the late Middle Ages.[3]

Hasund pointed at the Black Death and later plague epidemics as the cause of the dramatic changes in settlement, production, population size and the fate of the state.[4] Subsequent research was, nonetheless, performed from a one-sided agrarian perspective. In no other European country were the effects of the Black Death and later plague epidemics on the economic and social structures as energetically studied as in Norway. However, in almost no other European country were the plague epidemics so consistently neglected as a topic of historical research. While the status of plague as the causal explanation of the dramatic contraction of settlement and population in the Late Middle Ages was high, plague, the causal explanation was ignored.

The crucial point is that the historians neglected to study and evaluate the (level of) tenability of the explanatory hypothesis. In the decades before the arrival of the Black Death, around 350,000 inhabitants lived within Norway's then borders, of whom around 95% lived in the countryside. This raises immediately several crucial questions, also called problems. Is it possible that an epidemic disease could move with enormous powers of spread through the sparsely populated Norwegian countryside? And also highly problematical: could an epidemic disease combine enormous powers of spread with enormous mortality and rapid death? It is a basic epidemiological rule that the more dangerous and rapidly mortal a disease is for infected humans, the weaker will be the powers of spread of the disease. The reason is that the time of infectiousness will be short and many diseased will die before they have had time and opportunity to infect at least one other person. If the likelihood of transmission of infection falls below an average infection rate of 1, the disease will die out.

This was the central problem in my 1992 doctoral dissertation *Plague in the Late Medieval Nordic Countries. Epidemiological studies*. My work

[3] Quite a detailed presentation of the history of plague studies in Norway, from P.A. Munch (1862) to J. Sandnes in the 1970s is given in my doctoral dissertation, Benedictow, Ole J. 1992/1993/1996. *Plague in the Late Medieval Nordic Countries. Epidemiological Studies*. Oslo: The M Press, 34–44.

[4] Hasund, S. 1920. *Ikring Mannedauden. Ei liti sogestudie*. Krisitiania: Grøndahl & Søns Boktrykkeri, 62–7; Hasund, S. 1934. *Det norske folks liv og historie gjennem tidene. Tidsrummet 1280 til omkring 1500*, Vol. 3. Oslo: H. Aschehoug & Co., 136–42.

showed, somewhat to my own surprise, that it was possible to coordinate the central information in medical and epidemiological standard works and primary studies on plague with the requirements for giving plague strong powers of spread in rural districts and a short course of illness. The explanatory hypothesis was tested and found tenable. It was also solid proof that the plague epidemics had, in fact, played a highly important part in Norwegian demographic history, economic history, social history and political history through three centuries.

As can be seen from the title of the dissertation, the focus was on the epidemiological and demographic explanatory potential. Although the plague epidemics' status as causal explanation of the pervasive societal processes in the late Middle Ages won wide acceptance, the numerous plague epidemics in the period about 1500–1654 were, at the time of the first edition of this book, almost unexplored. The ambition was to clarify as far as the sources would allow when and where plague entered Norway, the frequency of plague epidemics, the spread in the country, patterns of spread, seasonality and effects of climate, and types of plague. All information on these topics would be discussed in the light of modern knowledge of plague epidemiology and plague medicine. Several of the topics should also be considered in the light of trade, international and national, and other conditions that might explain conveyance of plague contagion to Norway and epidemic spread. The perspective of research was to produce a complete history of plague in Norway, a study of all plague epidemics that spread in Norway during the Second Plague Pandemic, from 1348 to 1654.

It should be expected that the transition of the social formation of the Middle Ages to the social formation of the early Modern Period would be reflected in substantial changes in the epidemiological pattern and social effects of plague. The reason is that the transition included a great change in the pattern and volume of international demand for Norwegian products, great intensification of shipping and trade, and the introduction of new industries, in Norway, for instance, the introduction of the water-driven sawmills and mining.

In Norway, as elsewhere, there was a certain fascination with alternative ideas of plague, especially epidemiological alternatives such as interhuman cross-infection by human fleas or human lice and fleas or by arbitrary notions of mutations or alternative rat fleas. It has been shown that they, like all other alternative ideas, are untenable, and it will again be shown below.[5]

[5] Benedictow, O.J. 2016a. *The Black Death and Later Plague Epidemics in the Scandinavian Countries. Perspectives and Controversies*. Berlin: De Greuter.

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 blood stream. This allows 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 so 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 consisting mainly of bacteria and haemin. When the 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 lymph node inflamed by plague bacteria, 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 plague bacteria left in the site of

a flea 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 and Intradermal.

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

Endemic 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, is 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 human 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. This term corresponds 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 blood stream. 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.

First Plague Pandemic occurred in the period 541-767. *See* Pandemic.

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%. *See* Lethal dose.

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

Lethal dose Measure of virulence. It is 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, i.e., cause a mortality rate of 50%. *See* Infection dose.

Lethality rate The proportion of those who contract a disease who dies 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 shows, 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 Microbiology is 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 comprising 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 which 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 rate is the proportion of a population which dies from this disease in an epidemic.

Naïve population Population without previous experience with a disease or diseases.

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 spreading 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 three 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 causes 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 blood stream. 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 called such spots (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 category, 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 (causing growth of 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 with 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 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. *See also* petechiae/plague spots.

Regurgitative transmission *See* blockage.

Secondary bacteraemic plague In about half of all cases of bubonic plague, bacteria at some point manage to overwhelm the lymphatic system and pass on into the bloodstream. They cause 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. The ulcer causes 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 former meaning according to a new consensus on terminological usage. *See* primary and secondary bacteraemic plague.

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

Third plague pandemic occurred in the period 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 midgut. *See* also Proventriculus.

Virulence This term is closely related to the term pathogenicity, 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 hosts of a normal population sample when administered by a certain route. This is called lethal dose, *see* this term.

PART ONE:

WHAT DISEASE WAS PLAGUE?

1. The Rise of a Disease that made History: Bubonic Plague

1. The evolutionary history of the plague bacterium and the agent of plague: from *Yersinia pseudotuberculosis* to *Yersinia pestis*

Plague is an acute infectious disease caused by the bacterium *Y. pestis*, named after Alexandre Yersin, the Swiss naturalized French bacteriologist, who discovered it and described it in Hong Kong in 1894. Yersinia is a genus of bacteria in the family of *Yersiniaceae*. Yersinia species have a number of basic features in common but are also highly diverse with respect to virulence, method of transmission, and mechanism(s) of spread.

All alternative theories on the microbiological identity and/or dynamics of the plague epidemics of the past have been invalidated by the conventional work of historians, historians of the history of medicine, and by scientists of the new scientific discipline of paleobiology.[1] Paleobiologists are the representatives of a new branch of microbial science which studies a(ncient)DNA obtained in putative plague graves or plague pits to decide the microbiological agent of death.

Paleobiologists have also studied the origin and evolutionary history of *Y. pestis* by analysis of mutations and genetic change. This has led to the construction of a phylogenetic tree, the genealogical tree or the pedigree, so to speak, of the disease. It shows that *Y. pestis* originally began to evolve from mutations of the bacterium *Yersinia pseudotuberculosis* somewhere in the distant reaches of East Asia.[2]

Bubonic plague is a flea-borne disease, predominantly transmitted by the black rat flea, *Xenopsylla cheopis*, occasionally by other rodent fleas, rarely by other fleas, which reflects that plague fundamentally is a rodent disease. *Y. pseudotuberculosis*, *Y. pestis*' evolutionary progenitor, was/is mainly a water-borne disease and is not transmitted by fleas. Central in the history of plague contagion is the evolvement of the transmissibility of

[1] Benedictow 2010: Ch. 10; Benedictow 2016a: Ch. 1.5; Benedictow 2021: xix–xx.
[2] Morelli, G., Song, Y., Mazzoni, C.J., et al. December 2010. "*Yersinia pestis* Genome Sequencing Identifies Patterns of Global Phylogenetic: *Nature Genetics*, 42, 1141–2; published online 10.31.2010, 2–3.

contagion by fleas that constitute the crucial property of the fully-fledged bacterial genus of *Y. pestis*.

In 2015, a team of paleobiologists reported finds of early *Y. pestis* in human skeletal remains obtained in several archaeological sites spread over a vast Eurasian area in the period dating back to 5,000–3,600 years BP or correspondingly 3,000–1,600 years BCE.[3] One of these finds, the youngest, obtained in a grave in Armenia datable to around 950 BCE or about 3,000–2,900 BP, possessed the genetic adaptation called *Yersinia* murine toxin or *ymt*, which represents the necessary properties to be carried and transmitted by fleas and produce bubonic plague. The scientists suggested that this lineage had a long history in the Middle East.[4]

These important findings were followed by a breakthrough. Paleobiologists made two positive findings of *Y. pestis* in dental pulp recovered from nine individuals buried together about 3,800 BP (the late Bronze Age) in the Samara region on the Volga in south-eastern Russia. They succeeded in reconstructing the genome, the complete set of genetic material, the DNA, of this ancient *Y. pestis*. It displayed a full *ymt* gene position (on a chromosome) that represents the property that protects bacteria from being rapidly lysed[5] in a flea's midgut where they are exposed to digestive juices and ferments. This version of *Y. pestis* had acquired a key "innovation", the crucial property that enables plague bacteria to colonize the midgut of fleas, develop blockage by biofilm, and, hence to be carried and transmitted by fleas and cause genuine bubonic plague. This was a fully-fledged *Y. pestis* of bubonic plague that was much older than the Armenian case presented above. Nonetheless, it would be highly improbable that the scientists had come across and studied the first victims of this new lineage. Molecular dating analysis (which is quite inaccurate) suggested that it was probably present at least 200 years earlier, about 4,000 years BP. The scientists considered it, nonetheless, "tempting to hypothesize" that it was some 1,000 years older and had originated before 5,000 BP (or 3,000 BCE).[6]

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[3] Rasmussen, S., Allentoft, M.E., Nielsen, K., et al. 2015. "Early Divergent Strains of *Yersinia pestis* in Eurasia 5,000 Years Ago", *Cell*, 163: 571–82.

[4] Rasmussen, Allentoft, Nielsen, et al. 2015: 575.

[5] Cause dissolution or destruction of a cell membrane with lysin (an antibody or other substance able to cause lysis of cells, especially bacteria).

[6] Spyrou, M., Tukhbatova, R.I., Wang C.-C., et al. June 8, 2018. "Analysis of 3,800-year-old *Yersinia pestis* genome suggests Bronze Age Origin for Bubonic Plague", *Nature communications*, 2.

This shows that plague had a long evolutionary history of development from its earliest evolvement from *Y. pseudotuberculosis* to fully-fledged *Y. pestis* which, according to inaccurate microbiological clocking techniques, occurred between 4,000 and 7,000 years BP. Since its movement westwards across the Eurasian continent started, it had slowly acquired mutations for genetic development towards *Y. pestis*, a development that seems to be attained between 5,000–4,000 BP.

Particularly important is the fact that this new lineage of *Y. pestis* of bubonic plague was obtained in the Samara region that much later was part of the lands of the Kipchak Khanate of the Golden Horde including the Caspian-Pontic Steppe north of the Caspian Sea and the Black Sea. The territory of this khanate contained a vast plague reservoir of long standing which explains the fact that the Black Death broke out there nearly 3150 years later. The scientists pointed out that plague reservoirs in Central and East Asia and "most notably those of the Caspian Sea region harbour *Y. pestis* strains" that "support the notion of these foci having persisted for millennia".[7]

2. Earliest plague history

i. Earliest historical accounts of plague epidemics

In the case of plague, written information goes further back in time than could reasonably be expected. The exceptional ferocity of plague epidemics is, of course, one important reason. Another reason is that plague is the only epidemic disease that exhibits buboes as a standard conspicuous clinical feature. Bloody cough or expectoration is also quite a characteristic clinical feature of plague that caught people's imagination and will quite often be mentioned. Most scholars accept the Biblical account of the epidemics that ravaged the towns of the Philistines during a war with the Israelites as evidence of epidemic bubonic plague. This is the oldest written (probable) description of bubonic plague and dates its history in the Middle East to 1057 BCE (3075 BP).[8]

Unquestionable references to bubonic plague are found in classical Greek medicine.[9] Plague is mentioned in the so-called *Hippocratic corpus*

[7] Spyrou, Tukhbatova, Wang et al. 2018: 2.

[8] Benedictow, O. J. 2013. "*Yersinia pestis*, the Bacterium of Plague, arose in East Asia. Did it Spread Westwards via the Silk Roads, the Chinese Maritime Expeditions of Zheng He or over the Vast Eurasian Populations of Sylvatic (Wild) Rodents?", *Journal of Asian Studies*, 47, 12–14.

[9] In Greek history of the antiquity, the Classical Period runs from 500 to 323 BCE, the Hellenistic Period from 323 to 32 BCE.

of medical books and writings.[10] The leading Greek physicians of antiquity came from Greek colonies along the coasts of Asia Minor, the Middle East, and North Africa. The famous Ancient Medical School of Alexandria was established in the third century BCE where medicine was taught and developed mainly on the basis of the *Hippocratic corpus* for 500 years. The references to evident cases of bubonic plague must be assumed to reflect observations in areas where Greek physicians were active and the discussion of these observations in the learned medical circles associated with the medical school.

Accounts of serious plague epidemics in the Hellenistic Period (323–32 BCE) are found in classical medical writings. Rufus of Ephesus who lived around 100 CE rendered comments by the pupils of Dionysius (the Hunchback) on severe, clearly identified, outbreaks of plague reported in North Africa and in the Middle East around 300 BCE:

> The buboes that are called pestilential, are very acute and very fatal, especially those which one may encounter unexpectedly in Libya, Egypt, and Syria, and which they say were accompanied by high fever, agonizing pain, severe constitutional disturbance, delirium, and the appearance of large, hard buboes that did not suppurate [secrete pus: my insertion], not only in the usual regions of the body, but also at the back of the knee and in the bend of the elbow, where, as a rule, similar fevers do not cause their formation.

In antiquity, the geographical term Libya meant North Africa and the geographical term Syria comprised also most of present-day Israel, Palestine and Jordan. Rufus also mentions subsequent plague epidemics in Libya about 50 BCE.[11] Around year 100 CE, Aretaeus of Cappadocia, a Greek physician living in Alexandria, described epidemic plague in no uncertain terms: “the epidemic buboes in the groin are caused from the liver; they are very malign.”[12]

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[10] Sticker, G. 1908. Abhandlungen aus der Seuchengeschichte und Seuchenlehre, Vol. 1, Part 1: Die Geschichte der Pest. Giessen: Alfred Töpelmann, 19–20.

[11] Dioscorides was a Greek army surgeon in the service of Emperor Nero (54–68 CE)

[12] Sticker 1908, 21–2. The view that the growth of malignant buboes in the groin originated in the liver is a usual medical opinion in Hippocratic medicine. It is inferred from its humoral theory of human physiology which is based on a notion to the effect that the human body contained four chief fluids, the so-called *cardinal humours*, namely blood, phlegm, choler and, lastly melancholy or black choler.

ii. The First Plague Pandemic, the Justinianic, 541–767CE

The term pandemic has two central meanings: (1) an epidemic disease that spreads over a wide area, at least most of a continent, and affecting a significant proportion of the population; (2) recurrent, often widespread waves of epidemics with a long temporal structure. Plague pandemics satisfy both meanings, they start with an explosive, chronologically continuous wave of epidemics that spread over vast territories with huge demographic effects and lasts for several years, and they continue to recur for 200–350 years. These two dimensions of pandemicity can usefully be analytically differentiated by being designated synchronic pandemic and diachronic pandemic, with the provision that a diachronic plague pandemic also includes an initial synchronic plague pandemic.

The *first (known) plague pandemic* that included both pandemic criteria broke out in in 541 CE and developed into a huge synchronic pandemic in the following years, resembling, as it may seem, the Black Death in severity, scope and duration. In the following 200 years, it was followed by numerous waves of plague epidemics, constituting the diachronic plague pandemic.

In the years 541–767 CE, epidemics of the First Plague Pandemic spread in the Byzantine Empire and beyond, including Asia Minor, much of North Africa, the Middle East, and, in addition, large parts of other countries and regions surrounding the Mediterranean littoral, southern Spain (for most of the period), Greece, Italy and long also south-eastern France. Ireland and England were ravaged in 543–4, 664–7 probably in 683–6.[13] Northern parts of Frankland (France), most parts of Germania, the Slavic and Baltic regions and the Nordic countries appear to have avoided the scourge altogether. In the Nordic countries and northern Germania, this may have been due to absence of black rats that seem not to have arrived before the Early Viking Age, around 800, and continued to arrive by ship and to spread in the following centuries.[14]

In recent research, scientists underscore the increasingly strong genetic evidence that there was only one introduction of plague in the First Plague Pandemic, namely with the initial Justinianic wave of plague in the years 541–4 that established a plague reservoir (or plague reservoirs) among the

[13] MacArthur, W.P. 1949. "The Identification of Some Pestilences Recorded in the Irish Annals", *Irish Historical Studies*, 6: 176–81; MacArthur, W.P. 1959. "The Medical Identification of some Pestilences of the Past", *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 53, 423–9.

[14] Benedictow 2010: 126–9, 131–41; Benedictow, O. J. 2016a. *The Black Death and Later Plague Epidemics in the Scandinavian Countries: Perspectives and Controversies*. Berlin: De Greuter, 395–451.