

OUT OF THE WEST: FORMATION OF A PERMANENT PLAGUE RESERVOIR IN SOUTH-CENTRAL GERMANY (1349–1356) AND ITS IMPLICATIONS*

I

INTRODUCTION

The last ten years or so have seen unprecedented progress in the history of plague, which has brought our knowledge to new heights. Continuing work by historians, archaeologists, palaeoclimatologists, epidemiologists, molecular biologists and palaeogeneticists, based on the development and application of new methods, has made it possible to answer some pressing research questions by revealing unique facts that put an end to decades-old debates and controversies. Historians no longer doubt that the Black Death and its recurrent waves were caused by plague bacterium *Yersinia pestis*.¹ But this new plague research went much further than that, by permitting the formulation of questions and revealing more complex, yet fascinating, facts about the disease's history. Phylogenetic analyses of plague

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¹ There is a long list of plague-denying studies. Most notable ones include Graham Twigg, *The Black Death: A Biological Reappraisal* (New York, 1984) (anthrax theory); Norman F. Cantor, *In the Wake of the Plague: The Black Death and the World It Made* (New York, 2001) (combination of pandemics); Samuel K. Cohn, 'The Black Death: End of a Paradigm', *American Historical Review*, cvii, no. 3 (2002) (an unknown non-plague disease); Susan Scott and Christopher Duncan, *Return of the Black Death: The World's Greatest Serial Killer* (Chichester, 2004) (ebola-like virus). However, the sequencing of first whole genomes of *Y. pestis* from the East Smithfield cemetery (London) in 2011 put an end to these decades-old controversies: Kirsten I. Bos *et al.*, 'A Draft Genome of *Yersinia pestis* from Victims of the Black Death', *Nature*, cdlxxviii (2011).

lineages that encompass vast ranges in time and space, have revealed much about the evolutionary history of plague, whose early outbreaks in humans go back to the Neolithic period (with the earliest sequenced genomes dating to around 5000 BP).²

Other topics still await further investigation, which could permit some 'big' questions to be answered with confidence. For instance, the question of the geographic origins of both the Second Plague Pandemic, and the 'great polytomy' (the 'big bang') preceding the Second Plague Pandemic's outbreak, whereby a main plague lineage (Branch 0) split into four new plague lineages (Branches 1, 2, and another short branch, which soon split into two branches — Branches 3 and 4) at some point in the late thirteenth, but more likely in the early fourteenth century, has not yet been firmly answered.³ So far, two candidates have been suggested: the Qinghai–Tibet plateau and Central Asia (possibly, the Tien-Shan mountains).⁴ Similarly, we are still uncertain about the geographic origins of the Black Death outbreak (caused by Branch 1), which ravaged, in its human pandemic form, Western Eurasia between 1346 and 1353; as of 2019, four hypotheses have been proposed, situating the cradle of the outbreak in, respectively, China, Central Asia, Western Siberia, and the Caucasus/Caspian region.⁵ In other

² Simon Rasmussen *et al.*, 'Early Divergent Strains of *Yersinia pestis* in Eurasia 5,000 Years Ago', *Cell*, clxiii (2015); Aida Andrades Valtueña *et al.*, 'The Stone Age Plague and Its Persistence in Eurasia', *Current Biology*, xxvii, no. 23 (2017); Maria A. Spyrou *et al.*, 'Analysis of 3800-year-old *Yersinia pestis* Genomes Suggests Bronze Age Origin for Bubonic Plague', *Nature Communications*, ix (2018); Nicolás Rascovan *et al.*, 'Emergence and Spread of Basal Lineages of *Yersinia pestis* during the Neolithic Decline', *Cell*, clxxvi (2019).

³ Bos *et al.*, 'Draft Genome of *Yersinia pestis* from Victims of the Black Death'.

⁴ Yujun Cui *et al.*, 'Historical Variations in Mutation Rate in an Epidemic Pathogen, *Yersinia pestis*', *PNAS*, cx (2013); Monica H. Green, 'Taking "Pandemic" Seriously: Making the Black Death Global', *Medieval Globe*, i (2014); Robert Hymes, 'A Hypothesis on the East Asian Beginnings of the *Yersinia pestis* Polytomy', *Medieval Globe*, i (2014); Galina Eroshenko *et al.*, 'Yersinia pestis Strains of Ancient Phylogenetic Branch 0.ANT are Widely Spread in the High-Mountain Plague Foci of Kyrgyzstan', *PLoS ONE*, xii (2017); Philip Slavin, 'Death by the Lake: Mortality Crisis in Early Fourteenth-Century Central Asia', *Journal of Interdisciplinary History*, i (2019).

⁵ Francis Aidan Gasquet, *The Black Death of 1348 and 1349* (London, 1908), 2–3; Robert Pollitzer and World Health Organization, *Plague* (Geneva, 1954), 14; William H. McNeill, *Plagues and Peoples* (New York, 1976), 43; Michael W. Dols, *The Black Death in the Middle East* (Princeton, 1977), 35–8; John Norris, 'East or West? The Geographic Origin of the Black Death', *Bulletin of the History of Medicine*, li (1977); Michael W. Dols, 'Geographical Origin of the Black Death: Comment', *Bulletin of the History of Medicine*, lii (1978), 112–3; John Norris, 'Response', *Bulletin of the History of Medicine*, lii (1978); Bruce M. S. Campbell,

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words, the prevailing narrative is that of the disease originating in the east and spreading westwards.

Closely related to the question of its geographic origins is the question of the ways in which the Black Death, and indeed the subsequent plague waves, were spreading. Both historians and scientists share a conviction that plague would spread from place to place via trade routes. To use Ole Benedictow's terminology, the disease spread in 'metastatic leaps' from major port 'beachheads' into the mainland, along rivers, coasts, trade routes and eventually along inland roads, in human-driven transport, involving the transmission of both hosts (the rat) and vectors (fleas) at a distance.⁶ In recent years, both humanists and scientists have moved from this somewhat oversimplified principle of 'rats and fleas' and argue that transmission mechanisms may have involved more complex relationships between the pathogenic bacterium, its vectors (insects) and its hosts (mammals).⁷ Nevertheless, the mechanisms of physical routes of transmission are still poorly understood.

Another closely linked question is related to reservoir(s) that contributed to the post-Black Death waves in Western Eurasia and North Africa. In other words: where were those hidden homes of the plague, which would be periodically radiated out by its strains between the late fourteenth and the early nineteenth centuries, to create and spread the recurrent waves? It is now a well-established fact that plague is, first and foremost, a rodent (rather than a human) disease; the zoonotic crossover happens reluctantly, with hungry and plague-bearing vectors (fleas) switching to humans only once rodent colonies collapse. That is to say, initial plague reservoirs are found in the wild, among rodent populations.⁸ As far as the Second Plague Pandemic reservoir(s) is concerned, there are, roughly speaking,

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The Great Transition: Climate, Disease and Society in the Late-Medieval World (Cambridge, 2016), 246–7; Slavin, 'Death by the Lake'.

⁶ Ole J. Benedictow, *What Disease was Plague? On the Controversy over the Microbiological Identity of Plague Epidemics of the Past* (Leiden, 2010), 151–93.

⁷ Thus, Rebecca J. Eisen, David T. Dennis and Kenneth L. Gage, 'The Role of Early-Phase Transmission in the Spread of *Yersinia pestis*', *Journal of Medical Entomology*, lii (2015); Katharine R. Dean *et al.*, 'Human Ectoparasites and the Spread of Plague in Europe during the Second Pandemic', *PNAS*, cxv (2018).

⁸ A. P. Anisimov, L. E. Lindler and G. B. Pier, 'Intraspecific Diversity of *Yersinia pestis*', *Clinical Microbiology Reviews*, xvii (2004); Vladimir M. Dubyanskiy and Aidyn B. Yeszhanov, 'Ecology of *Yersinia pestis* and the Epidemiology of

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two main schools of thought. On the one hand, there are 'Asianists', who contend that just as in the case of the Black Death and the Third Plague Pandemic of the late nineteenth and early twentieth centuries, the recurrent waves of the Second Plague Pandemic must have originated somewhere in the east, before travelling, in each instance, westwards, via trade and warfare.⁹ On the other hand, there are 'Europeanists', both historians and palaeogeneticists, who argue for the existence of a (now defunct) European reservoir hiding in rodents and causing, from time to time, human mortality on a pandemic scale.¹⁰

With all their theoretical merits, these views lack, so far, a firm factual basis. The present article attempts to fill some gaps in our knowledge and, hopefully, to answer some of the pressing questions related to the topic, through the prism of one particular plague epidemic in late fourteenth-century Europe. The outbreak in question is the so-called *pestis secunda*, or the second wave of the Second Plague Pandemic, which, as I show below, broke out in Central Germany in late summer 1356 and then swept across Europe, the Middle East and northern Africa between 1357 and 1366.¹¹ A meticulous reconstruction of the environmental and socio-economic context, together with a dissection of palaeogenetic data of the *pestis secunda* outbreak is

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Plague', in Ruifu Yang and Andrey Anisimov (eds.), *Yersinia pestis: Retrospective and Perspective* (Dordrecht, 2016), 100–70.

⁹ Most recently, this assumption has been repeated by a group of Oslo palaeogeneticists: see Amine Namouchi *et al.*, 'Integrative Approach using *Yersinia pestis* Genomes to Revisit the Historical Landscape of Plague during the Medieval Period', *PNAS*, cxv (2018).

¹⁰ Ann G. Carmichael, 'Plague Persistence in Western Europe: A Hypothesis', *Medieval Globe*, i (2014); Kathleen Pribyl, *Farming, Famine and Plague: The Impact of Climate in Late Medieval England* (Cham, 2017); Kirsten I. Bos *et al.*, 'Eighteenth Century *Yersinia pestis* Genomes Reveal the Long-Term Persistence of an Historical Plague Focus', *eLife*, v (2016).

¹¹ The term *pestis secunda* (the 'second pestilence' in Latin) was used widely by fourteenth-century contemporaries. It has been accepted and used by both historians and palaeoscientists of plague (for instance, Monica H. Green, 'Putting Africa on the Black Death Map: Narratives from Genetics and History', *Afriques*, ix (2018), 9; Namouchi *et al.*, 'Integrative Approach using *Yersinia pestis* Genomes to Revisit the Historical Landscape of Plague during the Medieval Period', 1, 5–6). Dating of the *pestis secunda* has not been uniform in historiography: thus, Carmichael, 'Plague Persistence in Western Europe', 175–7, dates it to 1359–63; Campbell, *Great Transition*, 313–6, to 1360–3; Namouchi *et al.*, 'Integrative Approach using *Yersinia pestis* Genomes to Revisit the Historical Landscape of Plague during the Medieval Period', 1, to 1357–66.

capable, I argue, of shedding much light on the origins and perseverance of the 'hidden' European plague reservoir in Central Germany (which may have become a home to plague bacteria for centuries to come). The presence and perseverance of a plague reservoir in a landlocked region of Central Germany implies that the expansion-diffusion mechanisms of the *pestis secunda* — and possibly of later outbreaks — consisted of concentric wave radiation of the pathogen out of its reservoir via inland means. This model may not agree with the received wisdom of the 'trade routes and metastatic leaps' mode, outlined above.

II

PESTIS SECUNDA: GENERAL CONTOURS

Unlike the Black Death, which dominated the historiography of late-medieval plague, the *pestis secunda* has received disproportionately little attention. Nevertheless, a few scholars hypothesized on its geographic origins. Thus, Jean Glénisson, in his 1968/9 article on the *pestis secunda* in France contended that 'the plague *returned* [*italics mine*] to Russia in 1352', from where it spread, from 1357 onwards, westwards.¹² Lawrence Langer, in his work on the Black Death in Russia, postulated that the second wave (namely, the *pestis secunda*) 'originated in Sarai and was probably brought to Nizhnii Novgorod by one of the many caravans of Russian or Moslem merchants'.¹³ In a similar vein, Monica Green has most recently spoken of a 'postulated plague reservoir in southern Russia' from which [the phylogenetic] 'Branch 1B sent a second offshoot into Europe ... the *pestis secunda*, which may have traced the same path that the original Black Death took, or maybe took a northern route'.¹⁴ Finally, Namouchi *et al.*, in their palaeogenetic study of several fourteenth-century plague genomes, conceded (as one possibility)

¹² Jean Glénisson, 'La seconde peste: l'épidémie de 1360–1362 en France et en Europe', *Annuaire-Bulletin de la Société de l'histoire de France* (1968–9), 30–1. The term 'returned' (*repart*) is a factual error: the Black Death had not reached what Glénisson understood as 'Russia' until 1351–3; the plague had been ravaging the neighbouring territories of the Golden Horde in 1345–6, but not Russian principalities.

¹³ Lawrence N. Langer, 'The Black Death in Russia: Its Effects Upon Urban Labor', *Russian History*, ii (1975), 57.

¹⁴ Green, 'Putting Africa on the Black Death Map', 9.

that the *pestis secunda* might have been imported ‘independently from extra-Western European reservoirs in the 1360s’.¹⁵

One problem with the ‘southern Russian/Golden Horde’ hypothesis is that it is contradicted by the available evidence, both textual and (as shown later) palaeogenetic. In fact, there is no evidence at all to suggest that the *pestis secunda* originated or came from anywhere in the *east* — in contrast with the first wave of the Second Plague Pandemic, namely, the Black Death. A systematic survey of the surviving textual sources (chronicles, annals and correspondence), government papers (parliamentary records, *acta* of local assemblies, bishops’ registers) and statistical records (wills, death lists, etc.) from all over Europe, North Africa and the Middle East from the 1350s and 1360s points out that the outbreak seems to have originated somewhere in the Hesse region (Central Germany), where it first attacked the areas around Frankfurt am Main in the late summer of 1356 and, later that year, also the regions around Limburg an der Lahn and possibly Würzburg (see online [Supplementary Data 1](#)).¹⁶ According to one Frankfurt chronicle, ‘In the Year of the Lord 1356, a great pestilence prevailed in the city of Frankfurt during August and following months; and the bed-ridden ones would die in the course of a short time, [after being ill] for hardly three days. They would die having swellings around their legs and arms, as well as having swellings and pain around their heads and elsewhere in their bodies’.¹⁷ The situation was grave enough by the end of September, when local clergy and citizens conducted rogatory processions against the plague.¹⁸

¹⁵ Namouchi *et al.*, ‘Integrative Approach using *Yersinia pestis* Genomes to Revisit the Historical Landscape of Plague during the Medieval Period’, 5.

¹⁶ Biraben’s catalogue of plagues — Jean-Noël Biraben, *Les hommes et la peste en France et dans les pays européens et méditerranéens*, 2 vols. (Paris, 1976), i, 375–449 — although often helpful, has proved to contain some chronological errors and inaccuracy, and, as such, has been generally avoided. Instead, a revised geo-chronological list of plague outbreaks during the *pestis secunda*, based on the sources mentioned above, has been created and is supplied in [Supplementary Data 1](#).

¹⁷ ‘Item anno domini McccLVI circa mensem augustum et sequentibus magna in opido Frankenfordensi pestilencia duravit, ac in lectis modico tempore quandoque vix tribus diebus vel circa decumbentes decesserunt. Glaucos circa crura vel brachia sua aut tumorem et dolorem circa capita sua vel alibi in corporibus habentes moriebantur’. ‘Annales Francofurtani, 1306–1364’, in *Frankfurter Chroniken und annalistische Aufzeichnungen des Mittelalters*, ed. Richard Froning (Frankfurt am Main, 1884), 3.

¹⁸ ‘Eodem anno fuit stacio generalis propter epidimiam, et sexto quinto ac quarto calendas octobris cantatum fuit in ecclesia nostra officium “recordare”, quolibet

Heinrich Taube, a Bavarian chronicler (d. 1364) and a witness to the disaster, narrated that already in 1356, the plague had spread ‘throughout all of Germany’.¹⁹ However, a closer regional analysis reveals a much more complex situation (see [Supplementary Data 1](#) and [Figure 1](#)). It was not until the following year (1357) that the plague made itself known in areas adjacent to Hesse. To the west of Hesse, it was reported to be ravaging Westphalia and east Rhineland-Palatinate. In the east, it reached Thuringia. In the north, it appears to have been decimating the population of the western parts of North Rhine and southern parts of Lower Saxony. Finally, in the south, it proliferated in the eastern part of Baden-Württemberg and Bavaria — roughly overlapping the bishoprics of Speyer, Bamberg, Augsburg, Regensburg and the western part of the diocese of Constance, all the way to Lake Constance. In 1358, the disease penetrated into north-west Germany, the eastern part of the bishopric of Constance (including the north-eastern tip of Switzerland), and the hinterland of Strasbourg. Unlike the Frankfurt chronicler, other German annalists provided no details of its physical symptoms.²⁰

In 1359, the disease spread to Pomerania in the north-east, Austria in the south-east, and the Zurich hinterland and Alsace

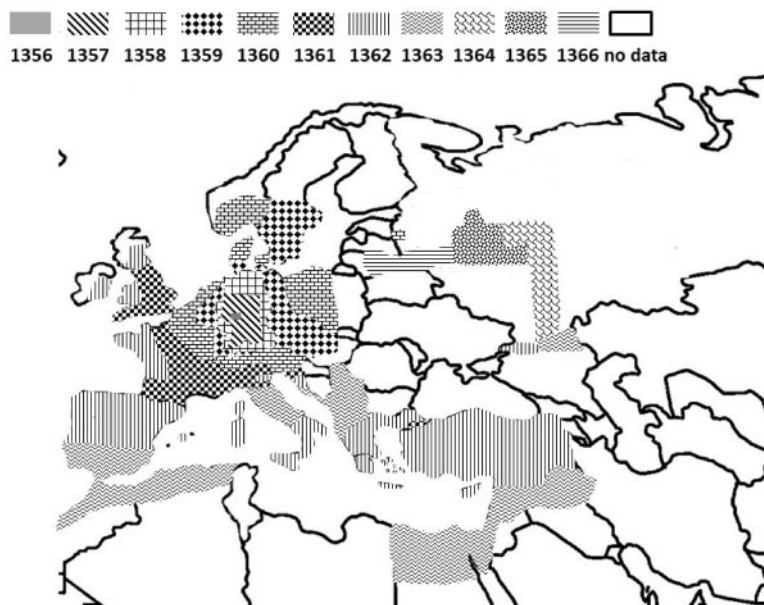
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sacerdote candelam ardentem manu tenente’ [In the same year, there was a general procession because of the epidemics, and on the sixth, fifth and fourth Kalends of October, the ‘Remember [O Lord]’ Office has been sung in our Church, with each priest holding a burning candle in his hand]. Joannes Latomus, ‘Acta’, in *Frankfurter Chroniken und annalistische Aufzeichnungen des Mittelalters*, ed. Richard Froning (Frankfurt am Main, 1884), 97.

¹⁹ *Item eodem anno insurgit pestilencia magna hominum, ut supra, per totam Alamaniam*’ [Also, in the same year, a great pestilence of people emerged, as earlier, throughout all of Germany]. ‘Die Chronik Heinrichs Taube von Selbach (Chronica Heinrici Surdi de Selbach)’, in *Monumenta Germaniae Historica. Scriptores rerum Germanicarum*, Nova series 1, ed. Harry Bresslau (Berlin, 1922), 110.

²⁰ For instance, a Mainz chronicler merely stated ‘*Anno predicto facta est magna pestilentia in multis partibus Rheni et in Hassia et Thuringia et Wedderabia*’ [In the aforesaid year, there was a great pestilence in many parts of the Rhine, as well as in Hesse, Thuringia and Wederau]. *Die Chroniken der Mittelrheinischen Städte: ainz*, 2 vols. (Leipzig, 1882), ii, 160–1. Likewise, a Zwiefalten Abbey chronicler, briefly noted that ‘*In monasterio Zwiweltun multe persone ex pestilencia hominum moriuntur*’ (In a Zwiefalten monastery, many individuals died of a human pestilence). ‘*Annales Zwifaltenses*’, in *Monumenta Germaniae Historica*, SS X, ed. D. Otto Abel and Georg Heinrich Pertz (Hannover, 1852), 62. Despite these laconic entries, however, it is clear that the 1357 and 1358 outbreaks all over Germany are simply the continuation of the 1356 outbreak in Hesse.

FIGURE 1
SPREAD OF THE PESTIS SECUNDA IN WESTERN EURASIA AND NORTH AFRICA, 1356–66*



* Source: see [Supplementary Data 1](#).

in the south-west. Concurrently, it made itself known, for the first time, in non-German lands, ravaging the northern and eastern provinces of the Low Countries (Gelderland, bishopric of Utrecht, east Brabant, east Hainaut and east Flanders), western Poland, western Bohemia and western Hungary. In the north, it also reached Scandinavia, both southern Denmark and southern Sweden (possibly from the Baltic coast of Germany via Hanseatic trade). In 1360, the disease spread further west into the western Low Countries (Holland, Frisian territories, west Brabant, west Hainaut, west Flanders and Artois), north-east France (north Burgundy, Champagne and Vermandois), and south Alpine regions around Lake Geneva and the Bugey mountains. From Austria, it marched into Slovenia, and then travelled all the way up to the Adriatic coast of Friuli. In north-eastern Europe, it penetrated further into Poland. In the north,

it ravaged all over Denmark, before reaching southern Norway, either from Denmark or Sweden. The plague was also attested in the Pskov area (north-west Russia), where it arrived either through the Finnish and Estonian lands by land, or via Hanseatic or Scandinavian trade, by sea.

In the following year (1361), the pestilence ravaged most of France (except the western regions of Poitou and Brittany, and west Normandy) and it seems that it had reached England from one of its ports (possibly Bordeaux) by sea. London was the first *locus* of outbreak in March, before it spread out in all four directions during the summer up to north Yorkshire in the north and eastern parts of Wales. Concurrently, it penetrated into the south Alpine region, all the way from Valais in the west to Trentino in the east. The plague spread further into northern Italy from three directions: from the Alps in the north, attacking Piedmont, Lombardy, most of Emilia-Romagna (except the Ferrara region) all the way to Rimini; from Provence in the west, attacking Liguria; and from the Adriatic coast in the east, penetrating into Veneto. It then spread into the south Slavic lands on the east Adriatic coast (overlapping with today's south Slovenia and north Croatia), either by sea from Venice, or by land from the eastern Alps. In the same year, the plague had also been reported in Constantinople, where it may have been spread by maritime routes (from Genoa or Venice), rather than by land (as the southern Balkans were spared until the following year).

In 1362, the plague widened its radius considerably. In the British Isles, it penetrated into north England, Wales, south Scotland and east Ireland. In France, it reached the western provinces of Poitou and Brittany, west Normandy, and in the south, it crossed the Pyrenees. There, it hit north Portugal, Catalonia, Aragon, Castile and Valencia, from where it reached, by sea, Majorca, Sardinia and Sicily, before spreading northwards, reaching Naples. In northern Italy, it was still ravaging Bologna and its hinterland, having arrived there in the previous year, as well as the Ferrara region. From Constantinople, it started spreading westwards into the Peloponnese, mainland Greece and Crete, and eastwards into Cyprus, Asia Minor and north Syria. In addition, it appeared in the Venetian colony of Tana (today's Azov, 16 km east of the

Azov Sea), most likely imported either directly from Venice or from Constantinople.

In 1363, the pathogen carried on its journey into southern parts of the Iberian Peninsula, partially in Muslim hands, before crossing into North Africa, where it spread extensively into northern parts of Morocco, Algeria and Lower Egypt. It is uncertain whether the plague came to Lower Egypt from the west (unfortunately, I was unable to find any information about the disease in Tunis and Libya), or from Mamluk Levant, where it is indeed attested in the same year. Further east, the plague was decimating the population of Baghdad. In Italy, the plague was ravaging all over the place from Naples in the south to Tuscany in the north. In the Balkans, it advanced from mainland Greece into Venetian Albania, Macedonia, Serbia and south Croatia. In the same year, the plague was also ravaging the Volga region of the Golden Horde (the cities of Beldjamen and Saray), where it most certainly arrived from the Azov area.

The years 1364–6 saw a gradual fading of the *pestis secunda*, whose geography was now confined to the northern territories of the Golden Horde, Russian principalities and the Grand Duchy of Lithuania. In 1364, it spread northwards, via the Volga, into Bolghar City, Nizhniy Novgorod, then into Kolomna and Ryazan, and then into Kostroma and Yaroslavl. In the following year, it carried on westwards, ravaging the towns of Vladimir, Suzal, Rostov, Kashin, Dmitrov, Moscow, Tver, Mozhaysk, Volog, Pereyasavl and Torzhok; in the north it reached as far as Beloozero. In 1366, it spread into Lithuania (presumably via Smolensk and Polotsk), which seems to have been the last stop of its ten-year-long quasi-circular journey.

The estimates of demographic losses are, at this point, far from being satisfactory, but a preliminary survey of probate wills from sixteen European towns for the period 1335 to 1366 indicates that the excess mortality ratio between *pestis secunda* and the preceding ten or so years was in the region of 7:1, while the comparable recorded death ratio between the Black Death and c.1335–48 stood at about 16:1.²¹ Assuming that the Black Death claimed about 50–60 per cent of the European

²¹ The dataset includes the cities of Barcelona, Lyon, Arezzo, Assisi, Florence, Perugia, Pisa, Siena, Bologna, Dubrovnik (Ragusa), Besançon, Lausanne, Tournai, London, Lübeck and Stralsund. For their references, consult Slavin, 'Death by the Lake', n. 8.

population, we may postulate that the mortality rates during the *pestis secunda* were in the area of 20 per cent, making it the second deadliest pandemic crisis of the last millennium.²² This figure agrees with scholarly estimates for England, where the average mortality rates across space and stratum seem to have been in the area of about 15–20 per cent.²³

According to a large number of written sources from different countries (from England to Croatia, from Spain to Russia), the *pestis secunda* outbreak was more discriminative than the previous one: chroniclers describe it as the plague of children, men and nobles.²⁴ Indeed, it killed almost one-fourth of the English nobility (comparable with about 27 per cent during the Black Death; however, this figure was disproportionately low compared to over 50 per cent of English peasants).²⁵ Was the *pestis secunda* sex-selective, killing more men than women, as some chroniclers suggest? At this stage, the available evidence is ambiguous. On the one hand, the sudden surge of land inheritance applications by female tenants after the deaths of their male relatives may indeed indicate that. As Mullan's study of the situation on Winchester Bishopric manors reveals, in 1361–2, female inheritance accounted for as much as 61 per

²² For Black Death mortality figures over Europe, see Ole J. Benedictow, *The Black Death, 1346–1353: The Complete History* (Woodbridge, 2004), 285–384.

²³ Jan Z. Titow, *English Rural Society, 1200–1350* (London, 1969), 70; Zvi Razi, *Life, Marriage and Death in a Medieval Parish: Economy, Society and Demography in Halesowen, 1270–1400* (Cambridge, 1980), 126–7; D. G. Watts, 'The Black Death in Dorset and Hampshire', in T. B. James (ed.), *The Black Death in Wessex* (The Hatcher Review, v, no. 46, Salisbury, 1998), 23–5; Lawrence R. Poos, *A Rural Society after the Black Death: Essex, 1350–1525* (Cambridge, 1991), 96; John Mullan, 'Mortality, Gender and the Plague of 1361–2 on the Estate of the Bishop of Winchester', *Cardiff Historical Papers* (Cardiff, 2007–8), 21–2; Sharon N. DeWitte and Maryanne Kowaleski, 'Black Death Bodies', *Fragments*, vi (2017), 1–37.

²⁴ For instance, *Black Death*, ed. and trans. Rosemary Horrox (Manchester, 1994), 86 (England); *Scotichronicon by Walter Bower in Latin and English*, ed. A. B. Scott and D. E. R. Watt, 9 vols. (Edinburgh, 1996), vii, bk xiv, 318–21 (Scotland); 'Annales Ragusini Anonymi, item Nicolai di Ragnina', ed. Natko Nodilo, in *Monumenta Spectantia Historiam Slavorum Meridionalium*, xiv (Zagreb, 1883), 233 (Dubrovnik, Croatia); *Listine o odnošajih između južnoga slavenstva i mletačke republike*, ed. Sime Ljubić, in *Monumenta Spectantia Historiam Slavorum Meridionalium* (Zagreb, 1872), iii, 22 (Hungary); *Ioannis Dlugossii Annales seu Cronicae Incliti Regni Poloniae* (Warsaw, 1975), bk 5, 302 (Poland); Rogozhskiy Letopisets, *Polnoye Sobraniye Russkikh Letopisey* (Petrograd, 1922), xv, 79 (Russian principalities).

²⁵ Josiah Cox Russell, *British Medieval Population* (Albuquerque, 1948), 217 and 224.

cent of the total inheritance, in contrast with 37 per cent during the first outbreak and 40 per cent in 1351–60.²⁶ This is partially corroborated by the skeletal evidence from St Mary Graces, where adult male skeletons accounted for 53 per cent of the total sexed adult sample (110 in total).²⁷ On the other hand, just as with the first outbreak, this does not mean that men were more susceptible than women. As DeWitte has shown, using the Gompertz model of mortality, there was no significant difference in risk of mortality between men and women during the 1361 outbreak at St Mary Graces.²⁸ In any event, comparing land inheritance evidence from multiple manors in South England recording hundreds of tenants during the plague outbreak to skeletal evidence from just one burial site in London containing just over a hundred bodies may not be the best methodological practice here.

Skeletal evidence from the 1361–2 outbreak may partially support the chroniclers' statement that it was 'the plague of the children'. At St Mary Graces, the combined proportion of infants and children aged 10 and under accounted for about 23 per cent of total mortality, which was indeed higher than the 17 per cent in the 1349–50 burial ground at East Smithfield and 13 per cent of fifteenth-century non-plague burials at St Mary Graces.²⁹ The proportion of children could, in theory, be even higher, given that child skeletons are more fragile and, thus are less likely to be recovered at excavation than the adult ones. The skeletal data, when considered alongside chronicle evidence, reflects the fact that these baby-boomers, born in the 'inter-plague' decade of the 1350s, constituted a substantial proportion within the total population and, thus, had high representation among the plague victims; certainly higher than during the Black Death. But just as with this earlier outbreak, the risk of death increased with adult age: that is, older adults were more likely to die than the younger ones.³⁰ And similar to the 1348–50 wave, frailer adults, as inferred from skeletal indicators, were more prone to death than their healthier counterparts.³¹

²⁶ Mullan, 'Mortality, Gender and the Plague', 23.

²⁷ DeWitte and Kowaleski, 'Black Death Bodies', 12–15.

²⁸ *Ibid.*, 12–15.

²⁹ *Ibid.*, 18.

³⁰ *Ibid.*, 18.

³¹ *Ibid.*, 15; Sharon N. DeWitte and James W. Wood, 'Selectivity of Black Death Mortality with Respect to Preexisting Health', *PNAS*, cv (2008).

Even though the *pestis secunda* was less destructive in terms of demographic losses compared to the Black Death, its consequences were more enduring by far. This outbreak had a huge influence on population demography, by wiping out the temporary surge in fertility of the inter-plague decade of the 1350s, and setting the stage for a new demographic regime, marked by low fertility and high mortality. With the massive number of deaths among the young and immunologically 'naïve' population born after the first outbreak, representing a disproportionately large share within the total population, as well as the Black Death survivors (who may have, in the meanwhile, lost their immunological capacity to 'remember' the immune response), population growth in West Eurasia and North Africa was depressed for many decades to come.³² All of a sudden, there was a shift from a 'bottom-heavy' to a 'top-heavy' demographic regime, characterized by a relatively large proportion of older people and low fertility. Without a sufficient share of young people, Europe was doomed to a long-term demographic stagnation and decline, which persisted for about a hundred years.³³

III

THE ENVIRONMENTAL CONTEXT OF THE 1356 OUTBREAK

At first, the identification of Hesse as a 'hot spot' for the initial outbreak of the *pestis secunda* may appear surprising. Indeed, there is no evidence — textual or statistical — that the Hesse outbreak of 1356 was preceded by any other *pestis secunda* outbreak anywhere in the German Empire, or indeed elsewhere in Western Eurasia or North Africa. The reports of a mortality crisis in Hesse in 1356 and then all over Germany in 1357–9, although often fragmentary and vague, prompt a detailed analysis of the rural environment around Frankfurt and adjacent

³² On 'immunological memory' and 'immune competence' (the ability to generate response to and destroy pathogens, via 'innate' [cellular] and 'acquired' [humoral] mechanisms), see Fabian Crespo and Matt B. Lawrenz, 'Heterogeneous Immunological Landscapes and Medieval Plague: An Invitation to a New Dialogue between Historians and Immunologists', *Medieval Globe*, i (2014).

³³ Campbell, *Great Transition*, 314–15; post-Black Death demographic trends in England have been surveyed in Stephen Broadberry *et al.*, *British Economic Growth, 1270–1870* (Cambridge, 2015), 21.

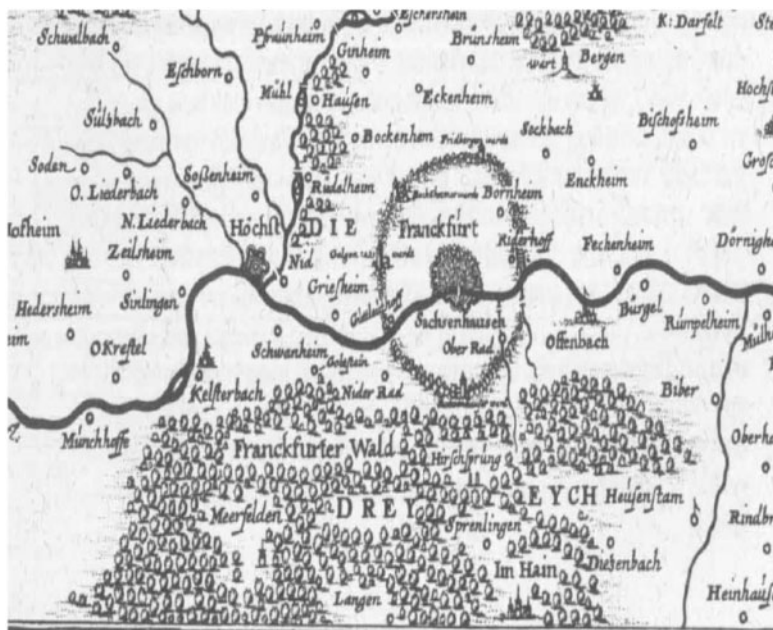
areas in mid fourteenth-century Hesse. Establishing a wider environmental and climatic context will help in appreciating the Hessian origins of the outbreak of *pestis secunda*. This approach is in line with the article's 'concentric waves' spread model — in contrast with the existing model of 'metastatic leap' spread from major port 'beachheads' into the mainland. As such, the article focuses on the interaction between humans, sylvatic rodents and their sylvatic environment, rather than on contacts between humans and rats in the context of ports and urban centres.

Ecological context

To begin with, it should be borne in mind that Hesse is the 'woodiest' region in Germany; its landscape is dominated by thick woodland, covering, as of 2019, 42 per cent of the land surface in that region.³⁴ It is unclear if Hesse, as a whole, was as woody in the later fourteenth century. Late fourteenth-century Hesse was the home of two mega-forests: the Büdinger Wald in the south-east (about 65 km north-east of Frankfurt); and the imperial chase (*wildbann*) Dreieich (immediately to the south of Frankfurt), encompassing several forests (see [Plate](#)). The Büdinger Wald was an imperial forest, leased out to the burgraves of the Imperial Palace of Gelnhausen, comprising at least fifty thousand acres. Dreieich was a gigantic and dense forest, stretching all the way from Darmstadt in the south and Frankfurt in the north, the Rhine in the west (up to the hinterlands of Mainz and Wiesbaden) and Main in the east, encompassing at least 250,000 acres of land. In 1372, a part of the Dreieich chase was acquired by the City of Frankfurt, to form the Frankfurt City Forest. Given the piecemeal clearances of the early-modern and modern periods, to make way for the growing urban landscapes of Frankfurt, Darmstadt and Mainz, it is likely that Dreieich was denser in the fourteenth century than it is today.³⁵ Apart from the Büdinger Wald and Dreieich, we have to account also for other woodland and forest areas, including the Taunus mountain range (north-west of Frankfurt),

³⁴ Hessian Ministry for Environment, Energy, Agriculture and Consumer Protection, *Forests and Forestry in Hesse*, 3–4, available at <https://english.hessen.de/sites/heu.hessen.de/files/content-downloads/Forests_and_Forestry_in_Hesse_barrier-free_geschuetzt.pdf> (accessed June 2020).

³⁵ Friedrich Scharff, *Das Recht in der Dreieich unter besonderer Berücksichtigung der Verhältnisse des Frankfurter Stadtwaldes und der umliegenden Dorfschaften* (Frankfurt, 1868), 301–8.



A Dreieich part of Hesse. Reproduced from Matthäus Merian, *Topographia Hassiae et Regionum Vicinarum* (Frankfurt am Main, 1655), 11.

Wetterau (north-east of Frankfurt) and Odenwald (south of Darmstadt).

The Dreieich chase is particularly abundant in textual evidence about its management and utilization in the later Middle Ages. This consists primarily of royal/imperial charters dealing with land privileges and disputes, as well as the 1338 *Wildbannweistum* of Dreieich, a collection of forest laws, issued by Louis IV the Bavarian (1314–47).³⁶ Although it was, at least in theory, a hunting space reserved for the emperors and higher nobility, the former were willing to allow various religious institutions and lay communities to use the forest for various purposes. This fact is reflected in a long series of thirteenth- and

³⁶ The Middle High German text of the 1338 *Wildbannweistum* has been edited in Hans-Otto Keunecke and Sigrd Schwenk, 'Das Dreieicher Wildbannweistum Kaiser Ludwigs des Bayern', *Archiv für Hessische Geschichte und Altertumskunde*, xxxvii (1979).

fourteenth-century imperial privileges. By far, most privileges were concerned with timber collection, granted to Frankfurt citizens and local religious institutions. Timber collection, however, was only one human activity appearing in late-medieval records related to Dreieich. In addition, we learn about grazing rights granted to local peasant communities in sheep, goat and cattle pastures, as well as pannage rights for local swine-owners.³⁷

Despite its arboreal density, Dreieich should not only be associated with timber collection and livestock husbandry. The sylvatic environment was also home to a series of rural communities, thriving by local groves. These included the villages of Dreieichenhain, Sprendlingen, Langen, Heusenstamm, Kelsterbach, Niederrad, Hirschsprung and Mörfelden (see [Blue](#)). Notwithstanding their situation within a sylvatic context, these communities were practising mixed husbandry, whereby both the arable and pastoral sectors were closely integrated with each other. These agricultural activities had a strong communal character, with open fields being the predominant feature of human settlement landscape.³⁸ As some documents indicate, arable fields were indeed located in the forest, cultivated by local peasants. Thus, a 1317 royal charter of Louis the Bavarian mentions arable fields in Langen producing seventy *achtel* (*octolia*) of 'forest oats' (*avena forestalia*).³⁹ The 1338 *Wildbannweistum* mentions oat cultivation in the forest.⁴⁰ Similarly, Kelsterbach had rye-fields in the forest.⁴¹ In addition, a 1439 deed of purchase in Schwanheim village refers to permanent pasture and meadows.⁴² In other words, there is enough evidence about the

³⁷ *Codex Diplomaticus Moenofrancofurtanus: Urkundenbuch der Reichsstadt Frankfurt*, ed. Johann Friedrich Boehmer (Frankfurt am Main, 1836), 137, 153, 164, 248, 256, 260, 265, 268, 303, 501; 'Dreieicher Wildbannweistum Kaiser Ludwigs der Bayern', ed. Keunecke and Schwenk, ch. 7, p. 40; ch. 10, p. 41; ch. 19, p. 43.

³⁸ Scharff, *Das Recht in der Dreieich unter besonderer Berücksichtigung der Verhältnisse des Frankfurter Stadtwaldes und der umliegenden Dorfschaften*, 104–24.

³⁹ *Codex Diplomaticus Moenofrancofurtanus*, ed. Boehmer, 438.

⁴⁰ 'Dreieicher Wildbannweistum Kaiser Ludwigs des Bayern', ed. Keunecke and Schwenk, ch. 24, p. 45.

⁴¹ As a fourteenth-century rental of Frankfurt Cathedral (Bartholomäusstift) indicates. Institut für Stadtgeschichte Frankfurt, Bartholomäusstift: Buch I.3, fo. 24^v.

⁴² Scharff, *Das Recht in der Dreieich unter besonderer Berücksichtigung der Verhältnisse des Frankfurter Stadtwaldes*, 106–8.

exploitation of diverse resources of the Dreieich forest by local communities.

Hessian woodland, including the forests within the Dreieich reserve, are marked by faunal diversity, being home to a wide variety of mammals. Different types of game included wild boar, red deer, hare and fox; to be encountered among the sylvatic rodents were stone martens (*Martes foina*), squirrels (*Sciurus vulgaris*), polecats (*Mustela putorius*), the dormouse (*Glis glis*) and the common vole (*Microtus arvalis*).⁴³ Both polecats and common voles are known as potential carriers of *Y. pestis*, and, thus, may act as transmitters of plague.⁴⁴ In her recent study of the impact of climate on famine and plague in late-medieval Norfolk, Kathleen Pribyl, who postulated the existence of (now extinct) European plague reservoirs, suggested that the common vole could have been the host during late-medieval outbreaks in England.⁴⁵ Pribyl's hypothesis can be taken further and tested in the Hessian context.

Of all voles, the common vole is the most fertile type, with females mating at as early as two weeks of age. Also, common voles are more social than other types of voles, forming large burrowing colonies, reaching densities of over a thousand rodents per hectare. Finally, in densely populated colonies, the sex ratio tends to be in favour of females, at times levelling off to 3:1 or 4:1.⁴⁶ As a result, common voles tend to be much more numerous than other vole types, and their explosive populations often translate into dangerous and costly outbreaks, causing much damage to European forests and fields. The population increase of common voles also depends on exogenous factors, primarily weather patterns and forage availability (with the

⁴³ Wilhelm Kobelt, 'Der Schwanheimer Wald. II. Die Tierwelt', *Bericht der Senckenbergischen Naturforschenden Gesellschaft in Frankfurt am Main*, xliii (1912), 168–72; Anton Krause, 'Notizen über Ratten, Mäuse und Wühlmäuse', *Archiv für Naturgeschichte*, lxxxix, no. 4 (1923).

⁴⁴ Angelina A. Kislichkina *et al.*, 'Eight Whole-Genome Assemblies of *Yersinia pestis* subsp. *microtus* bv. *Caucasica* Isolated from the Common Vole (*Microtus arvalis*) Plague Focus in Dagestan, Russia', *Genome Announcements*, v (2017); Angelina A. Kislichkina *et al.*, 'Three Genetically Different Lineages of *Yersinia pestis* subsp. *microtus* bv. *Caucasica* (0.PE2) Strains Circulate among Common Voles in Natural Plague Foci in the Caucasus', *Molecular Genetics, Microbiology and Virology*, xxxii (2017).

⁴⁵ Pribyl, *Farming, Famine and Plague*, 213–23.

⁴⁶ Jens Jacob and Emil Tkadlec, 'Rodent Outbreaks in Europe: Dynamics and Damage', in Grant Singleton *et al.* (eds.), *Rodent Outbreaks: Ecology and Impacts*, International Rice Research Institute, (Los Baños, Philippines, 2010), 208.

second variable being strongly dependent on the first one). Vole population growth responds to annual precipitation levels, with explosive population growth occurring in years following damp conditions in the previous year.⁴⁷ Likewise, snowy winters are beneficial for vole population growth, as snow insulates their burrows and keeps their colonies warm.⁴⁸ Weather would also influence the availability of forage resources for voles. Voles are omnivorous, but the most important types of forage for survival and population growth are tree mast as autumn and winter fodder and grass as spring and summer fodder. In particular, continental voles favour beech- and oak-mast. Hessian forests are particularly abundant in beech (*Fagus sylvatica*), where this genus accounts for almost one third of all the forest area, a considerably higher figure than elsewhere in Europe.⁴⁹

Climatic context

Both vole population and beech masting depend largely on climatic conditions, which need to be borne in mind when reconstructing the environmental context of the 1356 outbreak. As several scholars and palaeoscientists have shown, there seems to be a correlation between particular weather patterns and rodent plague outbreaks.⁵⁰ Both textual sources and the dendrochronological record allow a meticulous reconstruction of weather patterns in the central parts of the German Empire in the years immediately preceding the 1356 outbreak. As far as textual sources are concerned, these consist of chronicles both from the Frankfurt region and from other areas in Germany (see [Supplementary Data 2](#)). While I am aware that regional microclimates varied from place to place, I have assumed that general seasonal weather patterns are reflective on a trans-regional level. Likewise, although there is no relevant tree-ring record from the Frankfurt City Forest or in its environs,

⁴⁷ Pribyl, *Farming, Famine and Plague*, 216–19.

⁴⁸ C. Imholt *et al.*, 'Identification of Weather Parameters Related to Regional Population Outbreak Risk of Common Voles (*Microtus arvalis*) in Eastern Germany', *Wildlife Research*, xxxviii (2011).

⁴⁹ Hessian Ministry for Environment, Energy, Agriculture and Consumer Protection. *Forests and Forestry in Hesse*, 3–4.

⁵⁰ K. L. Kausrud *et al.*, 'Modeling the Epidemiological History of Plague in Central Asia: Palaeoclimatic Forcing on a Disease System over the Past Millennium', *BMC Biology*, viii (2010); Campbell, *Great Transition*, 228–30; Pribyl, *Farming, Famine and Plague*, 213–23; Slavin, 'Death by the Lake'.

dendrochronology of oak (*Quercus* spp.) from Kassel (198 km north of Frankfurt) and Göttingen (236 km north of Frankfurt), recording the annual precipitation levels of June-to-September (JJAS), can provide many clues about summer weather patterns in the Frankfurt area (see Figure 2). To see if the two available series are reliable and reflective, I have juxtaposed the Kassel and Göttingen samples against the 'Old World Drought Atlas' (OWDA) dataset, which is based on hundreds of regional tree-ring series from across Europe, including Germany.⁵¹ The OWDA dataset corroborates the Kassel-Göttingen dendrochronological series.

As both the Kassel and Göttingen tree-rings, the OWDA dataset and a number of chronicles indicate, the summers of 1353 and 1354 were dry and hot, while those of 1355 and 1356 were cold and excessively damp, and were in fact among the dampest on record in the fourteenth century (Figure 2).⁵² Textual sources also indicate the weather in other seasons. The winters of 1353/4, 1354/5 and 1355/6 seem to have been exceptionally cold, with major rivers, such as the Rhine and Main, freezing, in each instance.⁵³ One Austrian chronicle reports an abundance of snow in February 1356.⁵⁴ The three consecutive springs were wet, with river inundations reported.⁵⁵ The autumn seasons did not receive as much attention in chronicles, but the available evidence indicates a hot and dry autumn in 1354 and, by contrast, a cold and damp one in 1355, with hail events recorded in September, all over the place.⁵⁶

How do the regional weather patterns of 1353–6 translate into the environmental settings of the plague outbreak in the area in summer 1356? Here, we have to take into account the following variables: (1) the impact of weather on masting; (2) the impact of masting on the vole population; and (3) the impact of weather on the vole population. As far as the first variable is concerned, it

⁵¹ Source: Edward R. Cook *et al.*, 'Old World Megadroughts and Pluvials during the Common Era', *Science Advances*, i, no. 10 (2015), at <<https://www.ncdc.noaa.gov/paleo-search/study/19419>> (accessed June 2020).

⁵² Supplementary Data 2; see also, Rüdiger Glaser, *Klimageschichte Mitteleuropas: 1000 Jahre Wetter, Klima, Katastrophen* (Darmstadt, 2001), 67.

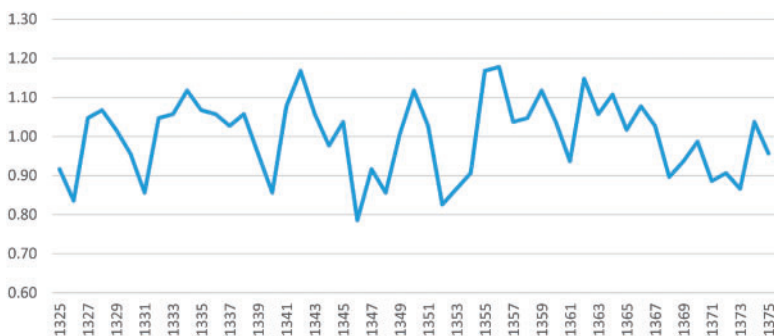
⁵³ Supplementary Data 2; Glaser, *Klimageschichte Mitteleuropas*, 77.

⁵⁴ 'Die kleine Klosterneuburger Chronik', ed. H. J. Zeibig, in *Archiv für Kunde österreichischer Geschichtsquellen*, vii (1851), 8.

⁵⁵ Supplementary Data 2; Glaser, *Klimageschichte Mitteleuropas*, 85.

⁵⁶ Supplementary Data 2; Glaser, *Klimageschichte Mitteleuropas*, 89.

FIGURE 2
PRECIPITATION INDEX IN FOURTEENTH-CENTURY CENTRAL
GERMANY, AS INDICATED IN TREE-RING WIDTH FROM KASSEL AND
GÖTTINGEN, LOGGED ON 1325–1375 (1325–75 = 1.00)*



* Source: Ulf Büntgen *et al.*, 'Tree-Ring Indicators of German Summer Drought over the Last Millennium', *Quaternary Science Reviews*, xxix, no. 7–8 (2010); Barbara Leuschner, data privately supplied. (I thank Dr Barbara Leuschner, Dendrochronologisches Labor Göttingen, for making the Göttingen data available for the study.)

has long been established that masting depends on weather conditions in the previous year (with July being the most crucial month), with hot, sunny and dry summers in the previous year assuring good masting in the following year.⁵⁷ On average, full masting in European beech occurs every 4–6 years, while moderate to good masting can occur, approximately every 2–3 years.⁵⁸ It appears that the hot and dry summers of 1353 and 1354 were highly favourable for good or even full masting in 1354 and 1355. This meant that more than enough fodder was available for voles, and this fact may have boosted their population during these years. Their growing population may have been sustained further by the weather conditions of 1354–6. In particular, the wet springs of 1354, 1355 and 1356, as well as the wet summers of 1355 and 1356, would have ensured abundant grass resources and perennial crops that are

⁵⁷ J. D. Matthews, 'The Influence of Weather on the Frequency of Beech Mast Years in England', *Forestry: An International Journal of Forest Research*, xxviii, no. 2 (1955).

⁵⁸ *Ibid.*, 108; Rafał Zwolak *et al.*, 'Advantages of Masting in European Beech: Timing of Granivore Satiation and Benefits of Seed Caching Support the Predator Dispersal Hypothesis', *Oecologia*, clxxx (2016).

vital for maintaining vole populations. Also, the cold and snowy winters of 1353/4, 1354/5 and 1355/6 may have had a positive impact on vole population growth. Prolonged periods of winter snow cover insulate vole burrows, thus protecting them from both predators and cold weather, and allowing older animals (the latest offspring of the last reproduction cycle in October) to commence reproduction activity with the melting and disappearance of snow in early spring. The unique combination of seasonal weather variances between 1353 and 1356 may have encouraged the growth of the vole population virtually unchecked — to the point where its levels reached those required for an ‘outbreak’ in 1356. Nomadic communities in Central Asia have long considered population outbreaks in rodents as a forewarning of plague outbreak in humans, as data from early Soviet Kazakhstan and Kyrgyzstan suggests.⁵⁹ Moreover, wet years present optimal conditions for the hatching and survival of flea larvae, which are equally crucial for the outbreak and spread of plague, first in rodents and later in humans.⁶⁰

The weather context for the summer of 1356 is hardly surprising. As several studies have shown, plague outbreaks in rodents tend to occur in wet years, as was the case in Kazakhstan between 1904 and 1945, and as seems to have been the case in the Chu Valley region in northern Kyrgyzstan, shortly before the 1338–9 outbreak in local Christian (East Syriac) communities.⁶¹ It was in the context of the wet summer of 1356 that we hear about the commencement of the outbreak of human plague.

IV

FORMATION OF A SOUTH-CENTRAL GERMAN PLAGUE RESERVOIR

Before it broke in humans, however, the pathogen had to cross over from a receding rodent population. The 1356 outbreak in humans was, in effect, the climax of a long and complex process, which lasted some seven years, commencing with the arrival of

⁵⁹ Pr. Nikanoroff, ‘Rapport URSS’, in *Les faunes régionales des rongeurs et des puces dans leurs rapports avec la peste: Résultats de l’enquête du Comité permanent de l’Office International d’Hygiène publique*, ed. R. Jorge (Paris, 1928), 126–7.

⁶⁰ Rebecca J. Eisen and Kenneth L. Gage, ‘Transmission of Flea-Borne Zoonotic Agents’, *Annual Review of Entomology*, lvii (2012), 69.

⁶¹ Kausrud *et al.*, ‘Modeling the Epidemiological History of Plague in Central Asia’; Slavin, ‘Death by the Lake’.

Branch 1 of *Y. pestis* bacterium in Hesse during the Black Death outbreak, developing with the split of Branch 1 into two sub-branches (1A and 1B) and the formation of a local plague reservoir, and culminating with the *pestis secunda* outbreak. As we shall see later, *pestis secunda* was caused by Branch 1B, in contrast with subsequent late-medieval and early-modern plague outbreaks, which were caused by Branch 1A — thus, the two ‘sister’ branches caused human plague sequentially in time, rather than concurrently. To appreciate how Branch 1B caused the sudden plague outbreak in humans, beginning in late summer 1356, it is necessary to reconstruct — in a hypothetical manner — each stage of this process.

Palaeogenetic context: emergence of Branch 1B

The period between the arrival of the Black Death in Hesse in summer 1349 until the 1356 outbreak seems to be crucial not only in setting the stage for the *pestis secunda* outbreak, but also for the history of plague evolution, in general. To begin with, it is necessary to contextualize the *pestis secunda* outbreak, in palaeogenetic terms, considering both space and time. As noted in the Introduction, the Black Death was preceded by the so-called ‘great polytomy’ (the ‘big bang’), whereby a main plague lineage (Branch 0) split into four new plague lineages (Branches 1, 2, and another short branch, which soon split into two branches — Branches 3 and 4). Sometime after the polytomy, Branch 1 erupted into what seems to be the deadliest mortality crisis in recorded history — the Black Death of 1346–53. Without analysing a rather complex route of the pathogen’s dissemination across Europe, it will suffice to say that by late June 1347, the plague had been imported into Sicily (through the port of Messina), from where it reached Genoa by the end of the same summer, before carrying on to northern Italy and south-eastern France in 1348. It appears that the plague reached the German Empire via north-eastern Italy in the east and via the Basel region in the west. It had definitely reached Hesse by the summer of 1349, and by 22 July the pandemic was reported in Frankfurt.⁶²

By the time the Black Death was decimating Hessian communities, there were uncountable multitudes of *Y. pestis*

⁶² Benedictow, *Black Death*, 187–8.

bacteria thriving all over Western Eurasia and North Africa. It was by pure chance (or by some reason still unknown) that one bacterium, out of the uncountable multitude of bacteria, happened to have mutated and become a founding father of a new branch, Branch 1B. This was a watershed event in the evolutionary history of *Y. pestis*, whereby Branch 1 split into two sub-branches: 1A and 1B.⁶³ Both sub-branches seem to have seeded their reservoirs in Europe (most likely in Hesse, as argued below), but their subsequent history seems to have differed. As of 2020, the presence of Branch 1A is attested in twenty-eight genomes from ten Second Plague Pandemic contexts in Europe and more palaeogenetic evidence will be published in the near future.⁶⁴ Hence, as far as palaeogenetic evidence goes, it appears

⁶³ The split into two sub-branches was first noted in Bos *et al.*, 'Eighteenth Century *Yersinia pestis* Genomes Reveal the Long-Term Persistence of an Historical Plague Focus', 5. The terms Branch 1A and 1B were coined by Monica Green in 2015, who detected that one skeletal sample from the Museum of London (Sample 6330), originally thought to be from the East Smithfield burial ground of 1349 (and reported in Bos *et al.*, 'Eighteenth Century *Yersinia pestis* Genomes Reveal the Long-Term Persistence of an Historical Plague Focus') comes, in fact, from St Mary Graces burials of 1361. This finding was subsequently announced in 'Plague Dialogues: Monica Green and Boris Schmid on Plague Phylogeny (II)', available online at <<https://contagions.wordpress.com/2016/06/29/plague-dialogues-monica-green-and-boris-schmid-on-plague-phylogeny-ii/>> (accessed 18 June 2020). This nomenclature was also used in Green, 'Putting Africa on the Black Death Map'.

⁶⁴ Bos *et al.*, 'Draft Genome of *Yersinia pestis* from Victims of the Black Death' (London East Smithfield, 1349 and London St Mary Graces, 1361); Bos *et al.*, 'Eighteenth Century *Yersinia pestis* Genomes Reveal the Long-Term Persistence of an Historical Plague Focus' (Marseille, 1720–2); Maria A. Spyrou *et al.*, 'Historical *Y. pestis* Genomes Reveal the European Black Death as the Source of Ancient and Modern Plague Pandemics', *Cell Host and Microbe*, xix, no. 6 (2016) (the genomes from Barcelona, 1348; Bolghar City, Tatarstan, c.1364; Ellwangen, Baden-Württemberg, c.1498–1640); Namouchi *et al.*, 'Integrative Approach using *Yersinia pestis* Genomes to Revisit the Historical Landscape of Plague during the Medieval Period' (the genomes from Saint-Laurent-de-la-Cabrerisse, 1348; Oslo, 1349/50; Sienna, 1348; Bergen-op-Zoom, Netherlands, 1359); Maria A. Spyrou *et al.* 'Phylogeography of the Second Plague Pandemic Revealed through Analysis of Historical *Yersinia pestis* Genomes', *Nature Communications*, x (2019) (the genomes from Laishevo, Tatarstan, c.1345; Toulouse, 1348; Nabburg, Bavaria, 1350; Manching-Pichl, Bavaria, late fourteenth century; Starnberg, Bavaria, c.1433–1523; Cambridge, c.1475–1536; Landsberg, Bavaria, c.1455–1632; Stans, Switzerland, c.1485–1635; London Liverpool St — Crossrail, c.1560–1635 [1603 or 1625?]; Brandenburg, c.1618–48 [1625–6?]); Karen Giffin *et al.*, 'A Treponemal Genome from an Historic Plague Victim Supports a Recent Emergence of Yaws and its Presence in 15th Century Europe', *Nature Scientific Reports*, x (2020) (the genomes from Vilnius, c.1453–85). The respective dates for the Black Death and *pestis secunda* outbreaks have been assigned by the present article's author, on the basis of information deriving from textual sources.

that Branch 1A was responsible for most of the European outbreaks during the Second Plague Pandemic, until becoming extinct at some point in the early nineteenth century. Conversely, Branch 1B has been attested so far only in four genomes from three later fourteenth-century contexts, all related to the *pestis secunda* outbreak — Bergen-op-Zoom, the Netherlands (1359), St Mary Graces, London (1361) and Bolghar City, Tatarstan (c.1364). Thus, contrary to Branch 1A, which seems to have seeded a *permanent* reservoir in Europe, Branch 1B seems to have seeded a *temporary* reservoir and caused a one-off outbreak in Europe, before leaving the continent. It is unclear why Branch 1B, unlike Branch 1A, left Central Germany and subsequently the continent, having sent (as presently available evidence suggests) only one plague wave. In the course of its evolutionary history outside Europe, Branch 1B gave birth to several new phylogenetic subgroups. Geographically, those subgroups are attested globally in Africa (strain 1.ANT in East Africa and strain 1.ORI around the Great Lakes District in Kenya, Congo, Uganda and Zambia, as well as in Madagascar), Asia (strain 1.IN in China and strain 1.ORI in Turkey, India, Myanmar and China), and America (western United States and Brazil).⁶⁵

Recently, Monica Green has drawn our attention to both the *pestis secunda* and the split within the Branch 1 lineage as a historical and archaeogenetic problem.⁶⁶ One big question to consider here is where and when this branching-off event, splitting Branch 1 into two sub-branches (Branches 1A and 1B), happened. The available palaeogenetic evidence hints that the divergence between the two sub-branches occurred at some point between the Black Death's arrival in Hesse in summer 1349 and the *pestis secunda* outbreak there in 1356. This is suggested by the differences in genetic signature between the Black Death genomes and those of the *pestis secunda*. Perhaps the single most important indicator of change in non-recombining pathogens, such as *Y. pestis*, is SNPs (single nucleotide polymorphisms). SNP is a

⁶⁵ The arrival and spread of 1.ANT strain in Africa has been most recently studied by Green, 'Putting Africa on the Black Death Map', 1–45. For other strains, see Cui *et al.*, 'Historical Variations in Mutation Rate in an Epidemic Pathogen, *Yersinia pestis*', and [Supplementary Data 1](#); Amy J. Vogler *et al.*, 'A Single Introduction of *Yersinia pestis* to Brazil during the 3rd Plague Pandemic', *PLoS ONE*, xiv (2019), and [Supplementary Data 1](#).

⁶⁶ Green and Schmid, 'Plague Dialogues'; Green, 'Putting Africa on the Black Death Map', 9 and 42.

variation (or, mutation) in a single nucleobase (A for adenine, C for cytosine, G for guanine, or T for thymine) at a certain genomic position. SNP occurs when a mutation in a single cell replicates itself many millions of times and, thus, becomes a genomic signature of a lineage.⁶⁷ Just as with other living organisms, the genomes of *Y. pestis* lineages evolve over time, with each lineage accumulating new SNPs. SNPs occur periodically, but there is neither ‘absolute’ nor ‘approximate’ time lag between two SNP events between two nodes on the same genetic lineage: in the case of *Y. pestis*, two SNPs could be several months, or several decades apart.⁶⁸

If we take the history of Branch 1A, it appears that during the course of approximately 375 years or so between the ‘big bang’ and the Marseille plague of 1720–2, the branch acquired one SNP every 4 to 4.5 years (Figure 3).⁶⁹ Such a rate seems to be, more or less, consistent with the frequency of SNP accumulation between the Black Death and the *pestis secunda*. There is one SNP between the Laishevo (Tatarstan) genome (c.1345) and the eight genomes from five European Black Death contexts — three from London (1348/9), one from Barcelona (1348), one from Saint-Laurent-de-la-Cabrerisse in south France (1348), one from Oslo (1349/50), one from Toulouse (1348), and one from Nabburg in Bavaria (1350).⁷⁰ Then, there

⁶⁷ For the discussion on genetic mutation in *Y. pestis*, its chronological and geographic significance, and application to historical studies of plague, see Green, ‘Putting Africa on the Black Death Map’, 11–12.

⁶⁸ *Ibid.*, 11–12 and nn. 43 and 47; Spyrou *et al.*, ‘Historical *Y. pestis* Genomes Reveal the European Black Death as the Source of Ancient and Modern Plague Pandemics’ (fig. 2B).

⁶⁹ *Ibid.*; Green, ‘Putting Africa on the Black Death Map’, n. 47.

⁷⁰ Spyrou *et al.*, ‘Historical *Y. pestis* Genomes Reveal the European Black Death as the Source of Ancient and Modern Plague Pandemics’ (fig. 2B); Spyrou *et al.*, ‘Phylogeography of the Second Plague Pandemic Revealed through Analysis of Historical *Yersinia pestis* Genomes’; Namouchi *et al.*, ‘Integrative Approach using *Yersinia pestis* Genomes to Revisit the Historical Landscape of Plague during the Medieval Period’ (fig. 3B). The genomes are dated ‘externally’ by the present article’s author, on the basis of textual information deriving from historical sources. It should be pointed out that the Laishevo genome (c.1345) is removed from the ‘big bang’ by just one SNP, which, in turn, may indicate that the polytomy had happened much later than the previous estimates (thus, Cui *et al.*, ‘Historical Variations in Mutation Rate in an Epidemic Pathogen, *Yersinia pestis*’, p. 580 dated the event to c.1142–1339, with the median date of c.1268, while Spyrou *et al.*, ‘Analysis of 3800-year-old *Yersinia pestis* Genomes Suggests Bronze Age Origin for Bubonic Plague’, and Supplementary Data Table 9, recalibrated the dating of the divergence of the Black Death strain from Branch 1 to the median date of c.1238 [+/- c.230 years]; Green, ‘Putting Africa on the Black Death Map’, 9 dates it to the thirteenth century — perhaps as late as the 1330s.

are two SNPs between the European Black Death genomes and the earliest sequenced *pestis secunda* genome from Bergen-op-Zoom in the Netherlands (1359), as well as the London St Mary genome (1361) (see Figure 3).⁷¹ Given the rate of SNP accumulation, as well as the fact that all of the so-far sequenced Black Death genomes share the same SNP profile and do not share the derived positions identified in the *pestis secunda* genomes, it appears that the split must have occurred at some point between the arrival of the Black Death in the Frankfurt region in summer 1349 and sometime before the outbreak of the *pestis secunda* in humans in summer 1356.⁷² In other words, the process of branching-off was happening concurrently with the process of establishing a new plague reservoir in the Frankfurt hinterland.

Zoological context: plague establishes a rodent reservoir

By 22 July 1349, the Black Death was decimating the human population of Frankfurt, implying that the disease may have invaded the town by early June (allowing about forty days between the pathogen's introduction into the commensal rodent population and the outbreak in humans).⁷³ At some point around that time or shortly thereafter, the Black Death strain seems to have seeded a reservoir somewhere in rural Hesse, which, thus, became the 'ultimate' origin of the *pestis secunda* (and possibly of later medieval outbreaks, as suggested below).⁷⁴

⁷¹ Spyrou *et al.*, 'Historical *Y. pestis* Genomes Reveal the European Black Death as the Source of Ancient and Modern Plague Pandemics' (fig. 2B), and four by Namouchi *et al.*, 'Integrative Approach using *Yersinia pestis* Genomes to Revisit the Historical Landscape of Plague during the Medieval Period' (fig. 3B).

⁷² It should be noted that one of the genomes recently sequenced by Namouchi *et al.* (from Abbadia San Salvatore in Siena, genome ID BSS31) consists of two additional SNPs — Namouchi *et al.*, 'Integrative Approach using *Yersinia pestis* Genomes to Revisit the Historical Landscape of Plague during the Medieval Period', 5. This anomaly may be to do with either (1) the possibility that the Sienese genome is, in fact, from a later fourteenth-century context rather than the Black Death (the authors did not justify their dating); or (2) the two additional SNPs are, in fact, environmental signals that can dominate in low-coverage genomes, such as BSS31 (44 per cent coverage at 3×).

⁷³ Benedictow, *Black Death*, 187; Benedictow, *What Disease was Plague?*, 6. It should be noted that the 22 July date is given by a sixteenth-century Frankfurter chronicler: 'Annalen eines Anonymus', in *Frankfurter Chroniken und annalistische Aufzeichnungen des Mittelalters*, ed. Richard Froning (Frankfurt am Main, 1884), 144–5. By contrast, contemporary sources do not provide an exact date in 1349.

⁷⁴ The nomenclature distinguishing between 'ultimate' and 'proximate' origins of plague outbreaks was introduced into the Second Plague Pandemic

It is unclear how exactly the pathogen seeded a new reservoir: in theory, at least, humans are regarded as a 'dead end', after which a plague outbreak burns itself out and dies.⁷⁵ To seed a reservoir in the wild, sylvatic rodents had to be exposed to bacteria, which would then need to proliferate among them for some time.

In theory, several speculative scenarios are possible. First, a process of 'anthroponosis' may have taken place, whereby the Black Death bacteria would find its way to cross over from humans back to rodents. 'Reverse zoonosis' is in itself an intriguing and understudied phenomenon. I was unable to find any confirmed documented case of such a crossover in the context of plague, despite some claims made by Russian doctors and French Pasteurians regarding the consumption of infected human flesh by Siberian marmots and rats, in the contexts of the Manchurian plague of 1910–11 and Madagascar outbreaks in the 1920s, respectively.⁷⁶ In the case of plague, it could be that it was *Xenopsylla cheopis* (oriental rat flea), rather than human ectoparasites that travelled with humans to a new rodent reservoir in Hesse (such as the Dreieich forest) and thus introduced the bacteria to their new putative home. As we have seen, there is abundant evidence about the exploitation of Hessian woodland resources by humans, not only via hunting, grazing and timber collection, but also via settlement and farming, which implies that humans were indeed in close contact with other plague hosts. Such a scenario, however, may not be very likely, and aside from occasional cases, the transmission pathway of plague from humans to rodents is not one that could have led to a sustained infection pattern. In

(n. 74 cont.)

historiography in Norris, 'East or West?', and subsequently used by Campbell, *Great Transition*, 242–3 and Spyrou *et al.*, 'Historical *Y. pestis* Genomes Reveal the European Black Death as the Source of Ancient and Modern Plague Pandemics'. The historiographic controversy relating to the 'ultimate' and 'proximate' origins of the Black Death has been surveyed in Nühket Varlik, *Plague and Empire in the Early Modern Mediterranean World: The Ottoman Experience, 1347–1600* (Cambridge, 2015), 94–7.

⁷⁵ As seems to have been the case in the instance of the 1613–4 outbreak in Freiberg, Saxony: see Stefan Monecke, Hannelore Monecke and Jochen Monecke, 'Modelling the Black Death: A Historical Case Study and Implications for the Epidemiology of Bubonic Plague', *International Journal of Medical Microbiology*, cxcix (2009).

⁷⁶ Paul Preble, 'The Tarbagan (*Arctomys bobac*) and Plague', *Public Health Reports*, xxvii (1912); Branwyn Poleykett, 'Ethnohistory and the Dead: Cultures of Colonial Epidemiology', *Medical Anthropology*, xxxvii (2018), 477–8.

rodents (sylvatic or commensal) that, while alive, had been roaming around the countryside, shortly before their crossover to humans. Ingesting the flesh of plague-killed rodents would ensure that the bacteria would have entered the system of naïve rodents. Although still an understudied phenomenon, the ability of *Y. pestis* to survive in a post-mortem host is a well-known fact, thanks to the enzymatic activity of pesticin (a protein toxin, produced by *Y. pestis*, while a host is alive) within a decomposing corpse.⁷⁸ Plague crossover via consumption of flesh of infected marmots and camels by humans has been documented in twentieth-century Central Asia.⁷⁹ Alternatively, both mechanisms could be involved in introducing the plague into a new reservoir of naïve rodents. In any event, at the present time, the discussion cannot move beyond a speculative framework.

At some point after the introduction of *Y. pestis* to its new putative home in Hesse in summer 1349, a random bacterium underwent a process of mutation in a single nucleobase, and having replicated itself millions of times, a new reservoir, represented by a new phylogenetic sub-branch Branch 1B, has emerged. It is uncertain at what stage between the two outbreaks this process occurred. What is certain, however, is that before crossing over to humans in the summer of 1356, the pathogen had to go through a complex and potentially lengthy process, referred to by some as the 'plague cycle'. The cycle in question consists of five stages: (1) an enzootic phase, whereby plague coexists with sylvatic ground-burrowing rodents, causing low mortality rates among these animals; (2) an excess mortality phase, in which mortality levels among rodents increase due to higher concentrations of bacteria, higher transmission rates by fleas and higher population densities of hosts; (3) an epizootic phase, with entire sylvatic rodent colonies being decimated and with a possible spillover of fleas from sylvatic rodents to commensal rodents (primarily, black rats); (4) a zoonotic phase, when a crossover from rodents (sylvatic or commensal) to humans occurs, with hungry rodent fleas utilizing humans and other non-rodent mammals as temporary hosts (in particular,

⁷⁸ W. R. Easterday *et al.*, 'An Additional Step in the Transmission of *Yersinia pestis*', *ISME Journal*, vi (2012).

⁷⁹ Yu. Z. Rivkus and A. G. Blyummer, *Endemiya chumy v pustynyakh Sredney Azii i Kazakhstana* (Voronezh, 2016), *passim*.

domesticates), and humans dying in large numbers; and (5) an epidemic (in some instances transforming into a pandemic) phase, when either rat fleas continue transmitting the disease in humans, or when human fleas and/or lice take over from rodent fleas as the main vector of the spread of the disease, and it is then that mortality levels in humans attain catastrophic levels.⁸⁰

During the enzootic stage, the newly introduced bacteria would make their home in Hessian woodland, presumably near to or in rodent burrows. As modern experimental research has established, *Y. pestis* is capable of thriving in contaminated soil, without hosts, for at least a few months, and it is likely that it is capable of surviving for particularly long periods in salty soils, thanks to its ability to coexist and replicate once ingested by soil amoebae on the one hand, and to develop halo-tolerance, on the other.⁸¹ The Hessian woodland is dominated primarily by brown earth, but also by gleysol.⁸² While the former has a low salt content, the latter is characterized by salinity, and as such it could host the newly introduced bacteria for a few months. This hypothetical introductory stage may have corresponded to the later stages of the human pandemic in late 1349. In theory, the enzootic stage, during which the plague reservoir would have been securely established, may have lasted for some five years, until the period of rapid growth in both the host and vector populations, between 1354 and 1356. As we have seen, the vole population growth may have been facilitated by the increased availability of fodder (mast and grass) and burrow insulation in

⁸⁰ The cycle is described in Campbell, *Great Transition*, 235–40. Please note that Campbell used a different terminology in reference to stages 2 and 3, calling them ‘epizootic’ and ‘panzootic’, respectively. However, the difficulty with this terminology is that the term ‘panzootic’ implies epizootic on a trans-regional/international level. This was, clearly, not the case in the plague cycle, whereby the zoonotic stage would occur after the decimation of local rodent colonies, namely after the epizootic stage.

⁸¹ Saravanan Ayyadurai *et al.*, ‘Long-Term Persistence of Virulent *Yersinia pestis* in Soil’, *Microbiology*, cliv (2008); Maliya Alia Malek *et al.*, ‘*Yersinia pestis* Halotolerance Illuminates Plague Reservoirs’, *Scientific Reports*, vii (2017); Javier A. Benavides-Montaña and Viveka Vadyvaloo, ‘*Yersinia pestis* Resists Predation by *Acanthamoeba castellanii* and Exhibits Prolonged Intracellular Survival’, *Applied and Environmental Microbiology*, lxxxi, no. 13 (2017); David W. Markman *et al.*, ‘*Yersinia pestis* Survival and Replication in Potential Ameba Reservoir’, *Emerging Infectious Diseases*, xxiv (2018).

⁸² HLNUG (Hessisches Landesamt für Naturschutz, Umwelt und Geologie) Database, available at <<http://bodenviewer.hessen.de/mapapps/resources/apps/bodenviewer/index.html?lang=en>> (accessed March 2019).

the winter, while the increase in flea population may have been encouraged by higher hatching rates, because of damp conditions. At this point, there could have been the first isolated instances of human infection, as a result of direct contact between humans and voles in Hessian woodland. The epizootic stage must have kicked in relatively close to the pandemic: by the late spring or early summer of 1356, the vole population may have reached its peak, sustaining high concentrations of bacteria and rapid transmission by fleas, thus, succumbing in large numbers.

V

FROM WOODS INTO THE CITY

There is no doubt that both the zoonotic crossover and the initial spread of the plague in late summer 1356 were facilitated by the presence of humans in the forest area and their close interaction with a wider ecosystem. As we have seen, Dreieich forest was frequented by Frankfurters and inhabitants of local villages in the city's hinterland, in order to collect timber and graze their livestock on sylvatic pastures and pannages. In addition, we have to take into account the network of villages, situated in the Frankfurt hinterland, both in Dreieich groves and other wooded regions. These rural communities practised mixed husbandry, where arable fields were an integral part of a local landscape, as we have seen.⁸³ Late summer/early autumn was a particularly busy time for local inhabitants and it would be around that time that their chances to interact with infected rodents would have been greater than ever. Around late July/early August local communities would start gathering their grain harvest, a process lasting over a month or so; of course, the wet conditions in summer 1356 might imply that harvest took longer than usual.⁸⁴

Harvested crops would then be carted from the fields to local storage facilities (barns and granaries, often attached to peasant

⁸³ 'Dreieicher Wildbannweistum Kaiser Ludwigs des Bayern', ed. Keunecke and Schwenk, ch. 25, p. 45; Scharff, *Das Recht in der Dreieich unter besonderer Berücksichtigung der Verhältnisse des Frankfurter Stadtwaldes*, 106–8.

⁸⁴ The impact of wet weather on harvest length has been pointed out (in a late-medieval English context) in Pribyl, *Farming, Famine and Plague*, 143–60 and Philip Slavin, *Experiencing Famine in Fourteenth-Century Britain* (Turnhout, 2019), 37–8.

houses). The 1338 *Wildbannweistum* expected each homestead in Dreieich to have a bakery and a barn.⁸⁵ Rodents, both sylvatic and commensal are known to be notorious consumers of arable crops, growing in fields or stocked at local barns and granaries. In particular, they tend to be active gleaners of post-harvest grain leftovers.⁸⁶ Likewise, rats can cause much damage to crop storage, in particular during their outbreaks. Although I was unable to find direct evidence about the damage done to stored crops by rats in late-medieval Germany, there are references to depredation by 'rats and mice' (which, of course, could also stand for voles) in some English agricultural documents from that period.⁸⁷ Finally, despite the conspicuous silence of plague witnesses about dying rats, there is enough zooarchaeological evidence indicating that rats were thriving in fourteenth-century Germany. In particular, rat bones have been uncovered from high-medieval contexts in Hattingen, Stuttgart and Nuremberg.⁸⁸ It must have been precisely in this context of close interaction between rodents and humans that the zoonotic stage took place, whereby the plague spread from sylvatic rodents' habitat in the forest to human habitat in nearby villages. It is unclear whether the zoonosis occurred as a direct spillover from sylvatic rodents to humans, or via an intermediate phase, involving a crossover from sylvatic rodents to commensal rodents, and then to humans. Both scenarios are possible.

The next stage consisted of plague transmission from the sylvatic countryside into the city of Frankfurt around early/mid July 1356. The pattern of country-to-city transmission of plague during the Second Plague Pandemic has already been noted by Ann Carmichael in the context of the 1560s outbreak in the

⁸⁵ 'Dreieicher Wildbannweistum Kaiser Ludwigs des Bayern', ed. Keunecke and Schwenk, ch. 17, p. 42.

⁸⁶ R. Tertilt, 'Impact of the Common Vole, *Microtus arvalis* (Pallas) on Winter Wheat and Alfalfa Crops', *Bulletin OEPP*, vii, no. 2 (1977).

⁸⁷ Christopher Dyer, 'The Material World of English Peasants, 1200–1540: Archaeological Perspectives on Rural Economy and Welfare', *Agricultural History Review*, lxii (2014), 12; see also, Jan C. Zadocs, *Crop Protection in Medieval Agriculture* (Leiden, 2013), 77–8.

⁸⁸ Frédérique Audoin-Rouzeau and Jean-Denis Vigne, 'La colonisation de l'Europe par le rat noir (*Rattus rattus*)', *Revue de Paléobiologie*, xiii (1994), see esp. 129–32; Frédérique Audoin-Rouzeau and Jean-Denis Vigne, 'Le rat noir (*Rattus rattus*) en Europe antique et médiévale: les voies du commerce et l'expansion de la peste', *Anthropozoologica*, xxv–xxvi (1998), see esp. 401.

southern Alps.⁸⁹ There can be little doubt that the commercial contacts between the city of Frankfurt and its rural hinterland inhabitants would have facilitated the process. Given the non-agricultural occupations of the inhabitants of Frankfurt, the latter depended strongly on agricultural supply of its rural hinterland, first and foremost foodstuffs, but also timber, hay, hides, wool, wax and tallow. In particular, Dreieichenhain and Mörfelden were important market villages, attracting buyers of agricultural produce from both nearby villages and the city.⁹⁰ Of all commercial activities, grain trade played an exceptionally important role, given that the diet of an average fourteenth-century German commoner consisted primarily of cereal-based foods.⁹¹ As in other large urban settlements, grain was extensively traded at local markets in Frankfurt, and in particular, the 'wheat market' (*forum frumenti*, *Kornmarkt*), situated next to St Leonard's church.⁹² Grain was carted into the city by both Hessian peasants, and by Frankfurt cornmongers, serving as middlemen between rural producers and urban consumers.⁹³

By the time the harvested grain was carted to Frankfurt, the city would have been exceptionally crowded and grain traffic seems to have been busier than usual. Apart from being home to the population of some fifteen- to twenty thousand burgers, the city would have been hosting its international autumn fair, lasting from 15 August to 8 September (not including eight days of imperial safe conduct before and after the fair, granted to its visitors).⁹⁴ After the decline of the Champagne Fairs in the early fourteenth century, the Frankfurt Fair became one of the most

⁸⁹ Carmichael, 'Plague Persistence in Western Europe', 162–74.

⁹⁰ Horst Wolfgang Böhme, 'Kritische Bemerkungen zur salischen Turmburg von Dreieichenhain und ihrer Vorgängerbauten', *Hessisches Jahrbuch für Landesgeschichte*, lv (2005). In 1256, Dreieichenhain was mentioned as a 'town', while in the early fifteenth century, Mörfelden imposed and collected its own customs.

⁹¹ Werner Rösener, *Peasants in the Middle Ages*, trans. Alexander Stützer (Champaign-Urbana, 1992), 97, 102, 104; Ernst Schubert, *Essen und Trinken im Mittelalter* (Darmstadt, 2006), 71–95.

⁹² Michael Rothmann, *Die Frankfurter Messen im Mittelalter* (Stuttgart, 1988), 119.

⁹³ Friedrich Bothe, 'Frankfurts Wirtschaftsleben im Mittelalter', *Zeitschrift für die gesamte Staatswissenschaft*, xciii (1932). See also a 1265 document on grain imports into the city in *Codex Diplomaticus Moenofrancofurtanus*, ed. Boehmer, 136.

⁹⁴ Rothmann, *Die Frankfurter Messen im Mittelalter*, 38 and 56.

important international trade hubs. During the fair, a multitude of merchants, both German and foreign (primarily from the Low Countries, northern Italy, France, as well as Bohemia, Poland and Hungary) would have gathered to conduct trade business, revolving primarily around wine and wool, but also clothes, yarns, leather, flax, spices, fish, cattle, glass, metal and dyestuffs.⁹⁵

Some wool traded at the fair was presumably produced in the Frankfurt hinterland, and this might have facilitated the spread of the pathogen into the city. In theory, sheep (and other domesticates) could serve as temporary plague hosts, and rodent–sheep/goat plague zoonosis is not unheard of.⁹⁶ Also, infected fleas could find a hiding place in wool sacks, to jump and bite humans found nearby.⁹⁷ Finally, the bacillus could, in theory, survive in contaminated wool, without live fleas, for a long period: dead fleas can sustain *Y. pestis* for up to 427 days.⁹⁸ That plague can be transmitted via wool is indicated in some plague accounts, from different chronological and geographic settings. Thus, it is hinted in one passage in Gabriele de' Mussi's *Historia de Morbo*, which narrates how four soldiers camping outside Genoa in 1348 stole a fleece and used it as their overnight bedding, only to die the morning after.⁹⁹ Equally, grain-laden carts, driven by horses, would have attracted infected rodents (potentially both sylvatic and commensal), and thus facilitated transmission of the pathogen from the villages to the city. Both the citizens and foreign merchants had to be provisioned with food and drink and hence there can be little doubt that the volume of grain carted from Hessian villages to the city was higher than usual during that time. But apart from potentially contaminated goods, the carts were also transporting another dangerous plague host: humans. Poor hygienic conditions of local rustics could always encourage infestation of lice. As some recent studies have shown, human ectoparasites are important

⁹⁵ *Ibid.*, 137–8.

⁹⁶ Ruixia Dai *et al.*, 'Human Plague Associated with Tibetan Sheep Originates in Marmots', *PLoS Neglected Tropical Diseases*, xii, no. 8 (2018); A. B. Christie, T. H. Chen and S. S. Elberg, 'Plague in Camels and Goats: Their Role in Human Epidemics', *Journal of Infectious Diseases*, cxli, no. 6 (1980).

⁹⁷ L. Fabian Hirst, *The Conquest of Plague* (Oxford, 1953), 303–455.

⁹⁸ Jeffrey Wimsatt and Dean E. Biggins, 'A Review of Plague Persistence with Special Emphasis on Fleas', *Journal of Vector Borne Diseases*, xlii (2009), 91.

⁹⁹ *Black Death*, trans. and ed. Horrox, 20.

vectors of plague transmission and, as such, they are to be taken seriously.¹⁰⁰

It was in the festive context of the autumn fair of 1356 that the pathogen reached the city of Frankfurt, with the busy traffic of goods and human congestion contributing to the arrival of the plague, its spread within and eventually outside the city. The role of transportation and trade of goods in spreading plague has been highlighted, time and again, by historians. Thus, as Michael McCormick contended, the spread of plague during the active period of the First Pandemic in the Middle East and Mediterranean Europe from 541 onwards was undoubtedly facilitated by grain trade, along both maritime and inland routes.¹⁰¹ Likewise, the movement of goods played a major role in spreading the pathogen during the Black Death. For instance, it was merchant vessels carrying English grain that imported the disease to Bergen in Norway in autumn 1349, before it made it to the north of Scotland, Shetland and the Faroe Islands.¹⁰² Likewise, the Black Death arrived on the Dorset coast in late June 1348, on board wine-carrying ships from Gascony (and it appears that the *pestis secunda*, too, reached London in March 1361 from Gascony).¹⁰³ One big difference between the spread of the *Y. pestis* from its proximate origins around Pelusium in 541, or Caffa in 1346, and that from its ultimate origin in Hesse in 1356 is in their geographic patterns. This topic deserves to be given full consideration here in the context of wider historiographical debates.

VI

PLAGUE SPREAD: METASTATIC OR CONTIGUOUS?

Roughly speaking, there are two schools of thought related to the geographic patterns of plague spread. First, there is a 'metastatic' approach, which, as noted earlier, seems to have reached strong consensus among most historians and some scientists. According to this view, the plague enters into the

¹⁰⁰ Dean *et al.*, 'Human Ectoparasites and the Spread of Plague in Europe during the Second Pandemic'.

¹⁰¹ Michael McCormick, 'Rats, Communications, and Plague: Toward an Ecological History', *Journal of Interdisciplinary History*, xxxiv (2003).

¹⁰² Ole J. Benedictow, *The Black Death and Later Plague Epidemics in the Scandinavian Countries: Perspectives and Controversies* (Berlin, 2016), 136–46.

¹⁰³ *Black Death*, trans. and ed. Horrox, 63.

mainland from major port 'beachheads', into which it has been imported via sea routes via 'metastatic leaps', usually in conjunction with long-distance trade. Once new beachheads are established, the plague spreads in an exponential manner along rivers, coasts, trade routes and eventually along inland roads via the transmission of both commensal hosts (the rat) and vectors (fleas), again in metastatic leaps, facilitated by the speed of human-driven transport. This model was advanced by Hirst and Pollitzer and more famously by William McNeill in his 1976 *Plagues and Peoples*, and then by Ole Benedictow on several occasions.¹⁰⁴ Thus, Benedictow identified the ports of Constantinople, Alexandria, Messina, Dubrovnik, Split, Genoa, Venice and Marseille as such beachheads, where the plague arrived at different points in 1347 and from which it would spread metastatically all over Europe, the Middle East and North Africa over the course of the subsequent five years.¹⁰⁵ According to Zlata Blažina Tomić and Vesna Blažina, in Dubrovnik 'all major plague onslaughts had arrived on an infected ship'.¹⁰⁶ Likewise, Amsterdam and London acted as similar beachheads in 1664 and 1665, respectively, while Marseilles was the epicentre, from which the plague spread across Provence in 1720–2.¹⁰⁷ The same pattern can be discerned also in the course of the (global) Third Plague Pandemic, and especially between 1894 and 1901, with the ports of Hong Kong (1894), Mumbai (1896), Alexandria, Porto and Honolulu (1899), Glasgow, Rio de Janeiro, Buenos Aires, San Francisco and Sydney (1900), and Cape Town (1901) becoming local beachheads.¹⁰⁸ Most recently, Namouchi and his colleagues, in their palaeogenetic analysis of several fourteenth-century plague genomes

¹⁰⁴ Hirst, *Conquest of Plague*, 306–23; Pollitzer, *Plague*, 385–90; William H. McNeill, *Plagues and Peoples* (New York, 1976), 114, 135, 139, 145; Benedictow, *Black Death*, 227–35; Benedictow, *What Disease was Plague?*, 151–93; Benedictow, *Black Death and Later Plague Epidemics*, *passim*.

¹⁰⁵ Benedictow, *Black Death* (map 1, after p. xvi).

¹⁰⁶ Zlata Blažina Tomić and Vesna Blažina, *Expelling the Plague: The Health Office and the Implementation of Quarantine in Dubrovnik, 1377–1533* (Montreal, 2015), 42.

¹⁰⁷ Stephen Porter, *The Great Plague* (Stroud, 2009), 108–9; P. E. Lemontey, *La peste de Marseille et de la Provence* (Laville, 2013).

¹⁰⁸ 'The Report of The Indian Plague Commission', *British Medical Journal*, i (1902); Myron Echenberg, 'Pestis Redux: The Initial Years of the Third Bubonic Plague Pandemic, 1894–1901', *Journal of World History*, xiii, no. 2 (2002).

(including one from the *pestis secunda*), concluded that their findings 'could support the hypothesis of an entry of plague into Western European ports through distinct waves of introduction during the Medieval Period'.¹⁰⁹

Then there is a 'contiguous' approach, adopted by some historians, most notably those who denied that the Black Death was caused by plague. This view argues that plague spreads in a contiguous fashion, resembling concentric waves, rather than leaps.¹¹⁰ Thus, Twigg, in theorizing that the Black Death was caused by anthrax, conceded that bubonic plague can spread fast only by contacts between adjacent rat colonies, while discontinuity between the colonies would slow down its spread.¹¹¹ Similarly, Samuel Cohn, who thought that the Black Death was a non-plague disease, pointed out the very slow speed of plague transmission during the Third Pandemic (13 to 19 km a year), which did not square up with late-medieval pandemics, which traversed at a much faster pace.¹¹² It should be noted, however, that 'metastatic leaps' are not to be equated with 'fast speed', as mentioned below. Scott and Duncan portray the plague spread in Provence during the 1720–2 outbreak as slow and gradual — in contrast with the fast speed of the Black Death, thought to have been caused by an ebola-like virus.¹¹³ The contiguous view is reflected in Élisabeth Carpentier's famous map of the Black Death spread, published in conjunction with her 1962 article.¹¹⁴

The geographic spread of the *pestis secunda* appears to be at odds with both schools of thought. Here, it has originated in a landlocked, woody region; it spread into Frankfurt and other

¹⁰⁹ Namouchi *et al.*, 'Integrative Approach using *Yersinia pestis* Genomes to Revisit the Historical Landscape of Plague during the Medieval Period'; here they provide imprecise/incorrect chronology of the appearance of the *pestis secunda* in the ports of Genoa, Venice, Ragusa, Constantinople and Alexandria, relying on the otherwise excellent Varlik, *Plague and Empire in the Early Modern Mediterranean World*, 119.

¹¹⁰ The arguments of this school of thought are analysed, in a very detailed manner, in Benedictow, *What Disease was Plague?*, 151–93.

¹¹¹ Twigg, *Black Death*, 100.

¹¹² Cohn, 'Black Death', 711–12. According to Benedictow's reckoning, the Black Death was spreading, on average, at the speed of 0.5–2.5 km per day over land, covering about 1–2 km a day, if traversing along main roads (see Benedictow, 2010, 170–3).

¹¹³ Susan Scott and Christopher Duncan, *Biology of Plagues: Evidence from Historical Populations* (Cambridge, 2001), 346–51.

¹¹⁴ Carpentier, 'Autour de la peste noire' (between pp. 1070 and 1071).

Hessian towns via inland routes; and its subsequent diffusion all over Europe, the Middle East and North Africa seems to have occurred, for the most part, via inland (surface and waterway) routes (with the exception of Byzantium, England, Ireland, Sicily, and the Azov region, where the pathogen was imported via sea). There were no beachheads similar to those during the Black Death; and the overall contours of spread appear to be contiguous, rather than metastatic. The pathogen seems to have radiated out of a single epicentre in Hesse, progressing in concentric waves, without creating additional local epicentres of the disease (see Figure 1). At the same time, however, the pathogen seems to have spread over land at a remarkably fast speed, largely comparable with that of the Black Death, depending on road types and conditions and seasons. Thus, it took almost two years for the plague to reach Lübeck from Frankfurt (550 km, at a speed of 0.8 km per day), and five months from London to Lincoln (210 km, at a speed of 1.4 km per day). In other words, there is no correlation between a mode of transmission and speed. 'Contiguous spread' is not to be equated with low speed, while 'metastatic leaps' do not preclude fast pace of transmission, as indeed evidenced in the Third Plague Pandemic.

This, in turn, is related to the question of the vector-host mode of the pathogen transmission. Obviously, this cannot be answered within the scope of the present paper, but a few hypotheses may be offered. The non-existence of port beachheads and metastatic leaps on the one hand and the contiguous, yet fast speed of spread on the other, implies either (1) density of continuous rat colonies, situated in proximity to each other, and hence, the rat-flea-man mode of transmission, or (2) man-lice-man (bubonic) and man-man (pneumonic) modes of transmission. But even if the pathogen did spread directly man to man, namely as a pneumonic plague, then it was only the *secondary* form of transmission, as there are more than enough contemporary descriptions of bubonic swellings during the *pestis secunda*.¹¹⁵ Whether the plague was spreading zoonotically (namely, from rats to humans) or directly between humans, it was doing so in a contiguous, rather than metastatic manner. This, of course, does not negate the metastatic spread during the Black

¹¹⁵ Cohn, 'Black Death', 716–17.

Death and the Third Pandemic. Instead, it indicates that more than one geographic fashion of transmission existed; certainly, metastatic leaps was one mode, but not the only one. These would vary from outbreak to outbreak, largely depending on their ultimate and proximate origins. Given that both Pelusium in 541 and Caffa in 1346 were *maritime* proximate origins of, respectively, the First Pandemic and the Black Death, the subsequent spread of the pathogen was bound to be in metastatic leaps. But in the case of the *pestis secunda*, its ultimate origin was a landlocked area in Hesse, and hence, its subsequent diffusion was contiguous and primarily via inland routes. Thus, whether subsequent Second Pandemic outbreaks would spread metastatically or contiguously would depend on the geographic origins of their reservoir/s. This point will be dealt with in the following section of the article.

VII

AFTER THE *PESTIS SECUNDA*: PERSEVERANCE OF THE EUROPEAN RESERVOIR(S)?

In 1364, when the *pestis secunda* was at the late phase of its journey, and eight years after its initial outbreak in Hesse, a new plague outbreak hit Central Germany. In the same year, the outbreak was reported in the region of Mainz, namely some 37 km to the west of Frankfurt, and right outside the western limits of the Dreieich forest.¹¹⁶ Just as the *pestis secunda* before, it was initially confined to various regions in Germany (1365–7), before spreading out in all four directions and visiting, roughly speaking, the same Western Eurasian and North African terrain between 1367 and 1376, as the *pestis secunda*.¹¹⁷ The *pestis tertia* of 1364–76 was followed by at least *eight* subsequent quasi-global plague waves, in the course of the late fourteenth and the fifteenth centuries. Because of the vast and complex nature of the topic, and its documentation, it was impossible, at this stage, to reconstruct the geographic contours of each wave in a detailed manner. However, a cursory look, based (at this stage) on

¹¹⁶ *Chroniken der Mittelrheinischen Städte*, ii, 168.

¹¹⁷ I am in the process of putting together a wider dataset of plague outbreaks and spread routes during the late fourteenth and fifteenth centuries. At this point, its chronological resolution is far from being high, but all the available evidence points to the Central German origins of the *pestis tertia*'s outbreak. One gets the same impression after a cursory look at Biraben's catalogue of late-medieval plagues — Biraben, *Les hommes et la peste*, i, 375–449.

Biraben's catalogue of plague outbreaks, points back to Central Germany, as their point of origin (in 1371, 1380, 1387, 1395, 1401, 1450, 1463 and 1482). At this point in time, we cannot yet establish the exact region in which each wave would have originated; but in some instances (1371, 1380, 1395), we can identify the terrain between the Rhine in the west and the Main in the east (overlapping today's eastern Rhineland-Palatinate and western Hesse regions, and situated in close vicinity to Dreieich), as their geographic origin.¹¹⁸ In each instance, the geographic and chronological trajectory of the plague waves would be remarkably similar to the *pestis secunda*: after a year or two of being confined to Central Germany, it would spread into the northern and southern German territories, before spreading out in all four directions. A close examination of the detailed chronology and geography of each wave will shed much light not only on the existence and perseverance of the European plague reservoir, but also on the dynamics and modes of transmission of recurrent plague waves during the Second Plague Pandemic.

As of 2020, only four out of thirty-six sequenced and published Second Plague Pandemic genomes are Branch 1B strains; all four genomes are from three *pestis secunda* contexts (Bergen-op-Zoom, London St Mary Graces and Bolghar City). The remaining genomes are all either Black Death strains, or from post-split Branch 1A strains (see Figure 3). In other words, Branch 1B may have left its Hessian reservoir in particular and Western Eurasia in general, to migrate, at a much later point, into sub-Saharan Africa, east Asia and the west coast of America. Yet, post-*pestis secunda* outbreaks, caused by Branch 1A, would still originate in Central Germany (and as far as the available evidence hints, in or in the vicinity of Hesse). This reveals not only that Central Germany served as a temporary Branch 1B homestead, but also its potential suitability as the focus for the subsequent and prolific Branch 1A outbreaks, at least until the sixteenth century (at which point, plague outbreaks seem to start being imported from the Ottoman Empire, rather than from the domestic reservoir/s in Germany).¹¹⁹ Indeed, a late fourteenth-century isolate from

¹¹⁸ *Ibid.*

¹¹⁹ It should be noted that the Marseille plague of 1720–2, even though it was the last major plague outbreak in Western Europe, was imported on a ship from plague-infected Cyprus, rather than from the Central German reservoir. By the

(cont. on p. 43)

Manching-Pichl (Bavaria), the earliest palaeogenetic evidence of post-*pestis secunda* outbreak, belongs to Branch 1A (Figure 3).¹²⁰ In theory, the strain could be related to the 1367 (*pestis tertia*), 1373 or 1380 outbreaks in Bavaria. Whichever outbreak it was, a permanent reservoir that would send recurrent plague waves all over West Eurasia and North Africa had been firmly established in Central Germany by that point.

It is unclear at what stage the Branch 1A established a permanent reservoir in Central Germany. Was it after the departure of Branch 1B from its temporary home in Hessian woodland, around Frankfurt? Or, did the two sub-lineages share the same reservoir, once it was established shortly after the Black Death's arrival in Hesse in summer 1349? The latter possibility is by far the most likely. This is given the fact that the recurrent outbreaks of the late fourteenth and the fifteenth centuries (including the *pestis tertia* of 1364–76, immediately following the *pestis secunda*) would have, as I contend, originated in Central Germany, most likely in or in the vicinity of Hessian woodland — in other words, in the same focus that was a home to *pestis secunda*. After all, the 'choice' of Central German woodland as its new 'home' by migrating bacteria in 1349 was purely coincidental, and it is highly improbable that the same coincidence would occur more than once. There were a multitude of sylvatic landscapes inhabited by wild rodents all over Western Eurasia, and any of these forests could, at least in theory, have become a plague *focus*. Yet, both the Branch 1B outbreak (*pestis secunda*) and the subsequent Branch 1A waves seem to have shared the same *focus*. In theory, both strains could have coexisted in the same reservoir, as is the case in some modern plague *foci*. For instance, the Tien-Shan *focus* of eastern Kyrgyzstan and south-eastern Kazakhstan is home to no fewer than four plague strains: 0.ANT5, 0.ANT2, 0.ANT3 (all three are strains older than the polytomy giving rise to Branches 1, 2,

(n. 119 cont.)

late seventeenth century, the Central German reservoir may have exhausted itself and died out; the late outbreaks, such as the 1664–6 outbreak in the Netherlands, Germany, Austria and England, and the 1679–80 outbreak in Germany and Austria, seem to have been imported via trade routes from the Ottoman Empire, where Branch 1 may have similarly established a reservoir, as attested in five sequenced genomes from the Marseille outbreak.

¹²⁰ Spyrou *et al.*, 'Phylogeography' (table 1).

3 and 4), and 2.MED1.¹²¹ In any event, establishing the 'proximate' origins, and hence the reservoir(s) of subsequent late-medieval and early modern times would require a separate study, reconstructing the geography of plague spread for each major trans-regional outbreak, deploying the same methodology as in this paper. Since such a study cannot be undertaken within the scope of the present article, the putative existence of a Central German reservoir of Branch 1A remains, at the present stage, a hypothesis.

VIII

PLAGUE: OUT OF THE WEST?

The geographic origins and movement of both the *pestis secunda* and the subsequent late-medieval plague outbreaks is at odds with the prevailing scholarly view. Because of the disproportionate place that the Black Death occupies in historiography of plague (and plague studies in general), the association of plague origins with the 'east' has become a kind of scientific mantra. This association with the east has been perpetuated, in fact, since the very days of the Black Death, as indicated by numerous authors, including Gabriele de' Mussi, Boccaccio and Guglielmo Cortusi.¹²² The scholarly preoccupation with the first wave of the Second Pandemic, namely the Black Death on the one hand, and relative inattention towards subsequent waves on the other, results in the creation of some sort of 'mental map' of plague transmission that consists of the following scheme: 'Eastern origins + international trade (sea) routes + rats (added at a much later stage) = plague in the West'.

This perpetuated idea of 'eastern origins and westward transmission' is reflected in historical discussion of other plague waves. Thus, in his 1976 *Les hommes et la peste*, Jean Biraben has no fewer than eight maps of plague transmission routes during the First Pandemic (dated by him to 541–750), all depicting the westward spread from their proximate origins in the Middle East or Asia Minor.¹²³ As we have seen, several historians, including Glénisson, Langer and Green argued in favour of south-Russian/Golden Horde origins and the westward spread of the *pestis*

¹²¹ Eroshenko *et al.*, 'Yersinia pestis Strains'.

¹²² *Black Death*, trans. and ed. Horrox, 17–19, 26–7, 34.

¹²³ Biraben, *Les hommes et la peste*, i, 34–41.

secunda.¹²⁴ In their work on plague control in late-medieval and early-modern Dubrovnik, Blažina Tomić and Blažina suggested that the subsequent waves of plague came 'either directly from the Ottoman-controlled territories, or indirectly, via an Italian port city'.¹²⁵

The idea of the recurrent reintroduction of plague from the east via maritime trade routes has been advanced by some palaeogeneticists as well. In their 2010 article on phylogenetic diversity and geographic diffusion of *Y. pestis*, Morelli and her colleagues, ascribed the mysterious appearance of branch 1.ANT1 (which itself branched-off from Branch 1B) in East Africa to Chinese maritime expeditions led by Muslim admiral Zheng He between 1409 and 1433.¹²⁶ In their 2015 study about the connection between climate and plague, Schmid and his colleagues advanced a theory-based model that both the Black Death and the successive plague waves were climate-driven reintroductions into Europe from Asia.¹²⁷ Similarly, Namouchi and his colleagues have most recently suggested that the *pestis secunda* might have been imported into Western Europe from the 'Land of Darkness' (which, although it is known to have been in Western Siberia near the Arctic Circle, was mistakenly identified here as 'a region speculated to be situated on the Kama river').¹²⁸

In contrast to the 'Asian origin-westward spread' view, there is another school of thought asserting the existence of (the now-defunct) European plague reservoir, responsible for post-Black Death outbreaks. The idea of plague persistence in Europe has long been hinted at by scholars such as Biraben (who divided

¹²⁴ Glénisson, 'La seconde peste', 30–1; Langer, 'The Black Death in Russia', 57, and Green, 'Putting Africa on the Black Death Map', 9.

¹²⁵ Blažina Tomić and Blažina, *Expelling the Plague*, 42.

¹²⁶ Giovanna Morelli *et al.*, 'Phylogenetic Diversity and Historical Patterns of Pandemic Spread of *Yersinia pestis*', *Nature Genetics*, xlii, no. 12 (2010). This claim has been refuted in Ole J. Benedictow, '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 History*, xlvii (2013), 2–5 and 20–8. The appearance and perseverance of branch 1.ANT1 in Africa has been analysed in Green, 'Putting Africa on the Black Death Map'.

¹²⁷ Boris V. Schmid *et al.*, 'Climate-Driven Introduction of the Black Death and Successive Plague Reintroductions into Europe', *PNAS*, cxii, no. 10 (2015).

¹²⁸ Namouchi *et al.*, 'Integrative Approach using *Yersinia pestis* Genomes to Revisit the Historical Landscape of Plague during the Medieval Period', 6; for the discussion of the 'Land of Darkness' as one possible ultimate origin of the Black Death, see Slavin, 'Death by the Lake', 86–7.

Western Eurasia into the 'north-western' and 'south-eastern' regions, and contended that the two regions had their own separate plague regimes and chronologies, unconnected among themselves),¹²⁹ but it was not until more recently that hypothesized locations of these plague *foci* were advanced. In particular, Ann Carmichael postulated, in discussing Swiss outbreaks in the 1560s, the existence of such a reservoir in Alpine Switzerland, in colonies of Alpine marmots.¹³⁰ Similarly, Kathleen Pribyl, in her work on the interaction between climate, famine and plague in late-medieval East Anglia, postulated the existence of (now extinct) European plague reservoirs, including an English reservoir.¹³¹ Likewise, Timur Khaydarov, in his recent work on plague pandemics in late-medieval Russian and Golden Horde territories, hypothesized, on the basis of earlier assumptions, the existence of three *foci* in Eurasia, namely: 'North-Western Russia', the 'Great Russian Plain', and 'Great Eurasia'.¹³² Finally, a team led by palaeoscientists currently at the Max Planck Institute for the Science of Human History in Jena, has been sequencing and analysing aDNA of historical plague victims and expanding our knowledge immensely over the last few years, and argues strongly, on the basis of a phylogenetic continuity across different Second Plague Pandemic outbreaks, for the perseverance of a 'domestic' plague reservoir in Europe.¹³³

One aspect that the 'Europeanist' school of thought does not account for is the geographic movements of post-Black Death plague outbreaks. In particular, there is no engagement with the spread of the pathogen from Europe to the Middle East, North

¹²⁹ Biraben, *Les hommes et la peste*, i, 105–29; this bipartite model was adopted by Blažina Tomić and Blažina, who claimed that '[the plague] became endemic in the Mediterranean and Western Europe, remaining a constant reality of life of the early modern world for over 350 years' (*Expelling the Plague*, p. 42).

¹³⁰ Carmichael, 'Plague Persistence in Western Europe', 157–91.

¹³¹ Pribyl, *Farming, Famine and Plague*, 213–23.

¹³² Timur Khaydarov, *Epokha 'Chernoy Smerti' v Zolotoy Orde i Prilegayushchikh Regionakh, Konets XIII–Pervaya Polovina XV vv.* (The Era of the 'Black Death' in the Golden Horde and Adjacent Regions, Late Thirteenth–Early Fifteenth Centuries) (Kazan, 2018), 84, 162, 214.

¹³³ Bos *et al.*, 'Eighteenth Century *Yersinia pestis* Genomes Reveal the Long-Term Persistence of an Historical Plague Focus'; Spyrou *et al.*, 'Historical *Y. pestis* Genomes', 874–81. In addition, the existence of the European reservoir has been postulated by Seifert *et al.*'s Munich team: Lisa Seifert *et al.*, 'Genotyping *Yersinia pestis* in Historical Plague: Evidence for Long-Term Persistence of *Y. pestis* in Europe from the 14th to the 17th Century', *PLoS ONE*, xi (2016).

Africa and beyond. Up until now, all maps of ‘global’ spread of plague during the Second Plague Pandemic related to its first wave, namely the Black Death, while no subsequent wave has ever been mapped, with the exception of some valuable regional studies.¹³⁴ As the map of the ‘global’ spread of *pestis secunda* in 1356–66 (see Figure 1) indicates, the movement was not ‘linear’ or ‘horizontal’ (west–east), but rather ‘concentric’ or ‘radiant’, from the Hessen reservoir into all four directions. The present study managed to track the spread of the *pestis secunda* all the way from its home to as far east as the Golden Horde and Iraq, and as far south as North Africa and Egypt. But it does not mean that its journey ended there; all it means is that we have reached a point where we cannot trace it further, because of the lack of written evidence. Could it be that the pathogen carried on from Egypt (where it is attested in 1363) into the Nile Valley and eventually into sub-Saharan East Africa, where it has been attested at an early stage by the presence of 1.ANT1 strain — one possibility that Monica Green has most recently suggested?¹³⁵ Or could it be that the Branch 1B continued its journey from the Golden Horde not only *northwards* into the Russian principalities and Lithuania (1364–6), but also *eastwards* through the vast steppe of Central Asia, eventually into the Far East, where it finally re-emerged in 1855 with the beginning of the Third Plague Pandemic in Yunnan? To traverse all the way to Yunnan, the pathogen would need to pass first through the Central Asian steppe, via Xinjiang (Uyghuristan) and then through the mountainous Qinghai–Tibet corridor. Given the nomadic or semi-migratory pastoral lifestyle, under-urbanization and thin population densities of local human communities (be it Turkic, Mongolic or Tibetan), as well as large and diverse sylvatic rodent populations, that characterize all these regions, one could expect a slow spread of the pathogen there — in

¹³⁴ Ernst Kirsten, Ernst Wolfgang Buchholz and Wolfgang Köllmann, *Raum und Bevölkerung in der Weltgeschichte: Bevölkerungs-Ploetz*, 3 vols. (Würzburg, 1965), iii, 94; Carpentier, ‘Autour de la peste’ (between pp. 1070 and 1071); Biraben, *Les hommes*, 88–9; Benedictow, *Black Death* (map 1, after p. xvi). The shortcomings and advantages of those maps have been discussed in David C. Mengel, ‘A Plague on Bohemia? Mapping the Black Death’, *Past and Present*, no. 211 (May 2011). One good example of regional maps of post-Black Death waves is Varlik, *Plague and Empire in the Early Modern Mediterranean World*, 134, 137, 161 and 188.

¹³⁵ Green, ‘Putting Africa on the Black Death Map’, 22–3, 25.

theory, taking several centuries. As of today, the Qinghai–Tibet Plateau is one of the most genetically diverse plague reservoirs, attesting the presence of several Branch 1B spin-offs, including 1.IN1 and 1.IN2, both preceding chronologically the 1.ORI strain, responsible for the Third Pandemic outbreak.¹³⁶ The contribution of the *pestis secunda* eastbound migration to the diverse pool of *Y. pestis* in both the Qinghai–Tibet Plateau and Yunnan province is yet to be established, but the question of the eastward migration of plague strains from Europe into China has already been raised by Yujun Cui in 2016.¹³⁷

IX

CONCLUSIONS

As the present article hoped to show, the geographic origins and spread of the *pestis secunda* differed substantially from the Black Death and some later seventeenth- and eighteenth-century plague outbreaks. The picture painted here is totally at odds with the perpetuated concepts of plague spread, and it supports a highly complex mechanism of plague outbreaks and modes of transmission. A meticulous analysis of a wide array of surviving evidence from all over Western Eurasia and North Africa allowed the reconstruction of the chronology and geography of the *pestis secunda*'s spread out from its original home, which I argue to be somewhere in Hesse, in all four directions. Hesse was a landlocked region, with access to central rivers (the Main and Rhine), but not the sea. International trade, conducted via downstream river routes, consisted of the famous international Frankfurt Fair, bringing together merchants from all over Europe. But it was not the traders who *brought* the plague to Hesse; rather, they *took* it with them. This, coupled with the fact that we have established Hesse as the original reservoir of the *pestis secunda*, should preclude any assumption that the latter was imported into Europe from the 'east' via maritime trade, as has been often suggested.¹³⁸ With this in mind, we should be encouraged to abandon the misconceived perception of the

¹³⁶ Cui *et al.*, 'Historical Variations in Mutation Rate in an Epidemic Pathogen, *Yersinia pestis*', 578, and [Supplementary Data 1](#).

¹³⁷ Yujun Cui, 'East to West or West to East: Plague Spread after the Black Death', *Infectious Diseases and Translational Medicine*, ii (2016), 58–9.

¹³⁸ Namouchi *et al.*, 'Integrative Approach using *Yersinia pestis* Genomes to Revisit the Historical Landscape of Plague during the Medieval Period', 5–6.

plague as 'an east-born westbound force, spreading via sea routes with the assistance of rats' (the 'metastatic' model). Rather, it was, at least in the case of the *pestis secunda*, a 'radiant' disaster, spreading in all four directions from its landlocked home in Hesse, primarily via inland routes (the 'contiguous' model). In other words, there is no such thing as a 'universal' model of plague diffusion.

Naturally, this does not suggest that the geographic origins of plague are located in Europe. After all, the emergence of the four new branches as the result of the pre-Black Death 'big bang' (presumably in the early fourteenth century, closer to the Black Death outbreak itself) must have occurred most certainly somewhere in the east, and it was only with the migration of Branch 1 closer to the western limits of Western Eurasia during the Black Death outbreak that the bacterium underwent mutation and seeded a reservoir in Hesse. Both genetic mutation and the establishment of the reservoir in Central Germany were coincidences, with far-reaching consequences in the sense that Branch 1 split into two sub-branches, one of which still persists today globally. To understand how these two almost concurrent processes happened, it was necessary to rely on a wide array of both textual sources and palaeoscientific data, consisting of palaeoclimatic and palaeogenetic information. A meticulous reconstruction of year-to-year climatic variations in the Hesse region in the 1350s sheds light on the interaction between weather, biomass availability (grass and mast fodder), and population dynamics of both sylvatic rodent hosts (most likely, common voles) and insect vectors (rodent fleas), which, in turn, played an enormous role in the process of establishing a new plague reservoir. After a few years of warm and dry weather and seemingly good masting (1353–4), wet weather kicked in (1355–6), producing grass in abundance. These were the optimal conditions for the growth of rodent populations as well as the population of fleas, and the eventual outbreak of plague.

Palaeoclimatic data are complemented by palaeogenetic data. At some point between the introduction of the Black Death to Hesse in summer 1349 and the eventual outbreak of the *pestis secunda* seven years later, I argue that the bacterium mutated and seeded a reservoir (or, the other way around) in that region.

Between the Black Death and the *pestis secunda*, the bacterium had also acquired two additional SNPs, most likely while proliferating among Hessian rodents. What is, perhaps, more intriguing is the fact that while Branch 1B, responsible for the *pestis secunda*, seems to have seeded a *temporary* plague reservoir, only to disappear and migrate southwards and eastwards (perhaps all the way into sub-Saharan East Africa and, through the Central Asian steppe and the Qinghai-Tibet plateau, into Yunnan province), Branch 1A, responsible for the recurrent plague outbreaks in Europe, seems to have created a *permanent* plague reservoir, also seemingly in Central Germany. This hypothesis is reflected in the fact that the subsequent late-medieval plagues of the late fourteenth and the fifteenth centuries all seem to have originated in the same region in Central Germany. As we have suggested, both branches shared the same reservoir: a phenomenon known from other plague *foci*.

Although Branch 1A seeded a permanent reservoir in Central Germany, sending recurrent waves all over Western Eurasia and North Africa in the late fourteenth- and early-fifteenth centuries, it appears that the same branch managed to seed some additional reservoirs in Western Eurasia and North Africa. As noted above, some later outbreaks are known to have been imported into Europe from elsewhere.¹³⁹ Thus, the 1663–6 outbreak that devastated Holland, and then England and Germany, seems to have arrived from Algeria; the plague of Marseille arrived from plague-infected Cyprus, while the Russian plague of 1770 and the Romanian plague of 1813 were both imported from Turkey. What is remarkable is the fact that the (basically identical) genomes of the Marseille plague, which originated somewhere in the Ottoman Empire, all belong to the Branch 1A family.¹⁴⁰ In other words, at some point an additional *focus/foci* must have been established somewhere in the Ottoman Empire. It will be up to historians and geneticists to work together to establish *where* and *when* Branch 1A established a permanent reservoir in the Ottoman Empire, and what anthropogenic forces (long-distance trade, facilitated by behemoths such as the Dutch and English East India companies, or military campaigns) were responsible for

¹³⁹ See n. 113 above.

¹⁴⁰ Bos *et al.*, 'Eighteenth Century *Yersinia pestis* Genomes Reveal the Long-Term Persistence of an Historical Plague Focus'.

importing it back into Europe. It will also be up to the same humanists and scientists to determine when the Central German reservoir died out, thus making the hypothetical ‘Ottoman’ reservoir take its place as the geographic origin of late plague outbreaks in Western Eurasia and North Africa. More than anything, it underlines the quasi-global nature of plague pandemics, which have to be approached from a broad and comparative perspective, as has been voiced more and more in recent years.¹⁴¹

This study’s findings prove — time and again — that plague is a highly complex topic that can only be fully appreciated and understood through the prism of multi-disciplinary research. The paucity of written sources on the *pestis secunda* outbreak in Central Germany in 1356 can be compensated by the growing corpus of palaeogenetic data, providing some invaluable hints about the appearance and spread of Branch 1B, responsible for the *pestis secunda* and indeed, much later plague outbreaks, in the course of the so-called ‘Third Pandemic’ of the late-nineteenth/early-twentieth centuries. But to understand in what circumstances Branch 1B, and consequently the *pestis secunda*, originated, it is necessary to reconstruct their immediate natural and climatic environment. Palaeoclimatic data comes to the rescue here, by supplying some important information about year-to-year fluctuations in vegetation growth and weather patterns. For one, the future of plague history belongs to multi-disciplinary collaboration between humanists and scientists working in a consilient manner to unearth, analyse, share and advance new intellectually unified knowledge of the past. Such collaborative and trans-disciplinary research has great potential to take our exponentially growing knowledge to altogether new heights.

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¹⁴¹ Green, ‘Taking “Pandemic” Seriously’; Green, ‘Putting Africa on the Black Death Map’; Slavin, ‘Death by the Lake’.

