REPLY: OUT OF THE WEST — AND NEITHER EAST, NOR NORTH, NOR SOUTH*

‘If the fact will not fit the theory — let the theory go’.
Agatha Christie, *The Mysterious Affair at Styles*

The article below is a reply to Professor Green’s response to my recent article ‘Out of the West: Formation of a Permanent Plague Reservoir in South-Central Germany (1349–1356) and Its Implications’.¹ I should perhaps open with a caveat: I respect ideas that differ from my own views, and firmly believe that it is controversies that advance science and knowledge. Hence, I normally refrain from writing and publishing replies to colleagues’ works. However, given the conceptual and methodological importance of the topic and its relevance for the incredibly fast-growing field of plague history in general, I strongly feel Professor Green’s response merits a rebuttal. It is especially important in the context of the most recent publication of three early fourteenth-century genomes from Kara-Djigach (North Kyrgyzstan), associated with the very early history of the Second Pandemic in general and the Black Death in particular, by Spyrou *et al.*² These new findings have a

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profound impact on the substance of debate regarding the origins and early history of medieval plague; and, moreover, allow an opportunity to discuss further the taxing methodological issues that all medievalists must confront in this field, when bringing palaeogenetic evidence into dialogue with documentary historical sources.

While not dismissing (and partly accepting) my suggestion about the existence of a south-central German plague reservoir, seeded in the aftermath of the Black Death, in her response Green argues that:

1. The same reservoir was responsible only partially for the plague wave of the later 1350s and earlier 1360s (which I refer to as the *pestis secunda* of 1356–66, but which Green calls the ‘second plague’).³

2. There was at least one additional reservoir responsible for the plague outbreaks during those years — Green suggesting that the same (conjectural) reservoir in the Caucasus-Volga region, which had been seeded in the 1250s, became active in the 1330s, and sent global pandemic waves in the 1340s (the Black Death) and 1350s (this final wave being the *pestis secunda* in my terminology and the ‘second plague’ in Green’s nomenclature).

3. The ‘second plague’ wave of the later 1350s and 1360s was caused by both genetic sub-branch 1A, originating (as I have argued) in the south-central German reservoir and circulating in central and southern continental Europe, and sub-branch 1B, originating in the Caucasus-Volga focus and proliferating in northern and north-eastern Europe.⁴

4. From their Caucasus-Volga home, sub-branch 1B strains spread across northern Europe via Golden Horde and Hanseatic trade routes.

³ For Green, the term *pestis secunda* is associated only with Branch 1B; as she hypothesizes, there were at least two active reservoirs sending plague waves, caused by two concurrent branches (1A and 1B) all over Eurasia. By contrast, I believe that the 1356–66 wave (which I call *pestis secunda*) had originated in a single reservoir (south-central Germany) and was caused by a single branch (Branch 1B).

⁴ The terms sub-branch 1A and 1B refer to a phylogenetic split within Branch 1 causing the Black Death, during or shortly after the Black Death, possibly in a newly established south-central German reservoir, in which the *pestis secunda*, known to have been caused by sub-branch 1B, seems to have commenced in the summer of 1356. For a lengthy discussion of that, see Slavin, ‘Out of the West’.
5. The split within post-polytomy Branch 1,\textsuperscript{5} responsible for the Black Death, into the two sub-branches (1A and 1B) had occurred \textit{before} the Black Death arrived in Europe in 1347.

6. In addition, Green rejected my idea that the plague reached England, in early 1361, from Gascony, and challenged my assessment that late seventeenth- and early eighteenth-century plague waves seem to have been imported to Europe from the Ottoman Empire.

Each of these points requires a critical assessment, and thus the present article is structured in numbered parts that engage, in a consecutive manner, with each of Green’s arguments.

I

NO EVIDENCE FOR A CAUCASUS-VOLGA PLAGUE RESERVOIR IN THE FOURTEENTH CENTURY

In constructing her counterarguments, Green invokes her theory, as published in two articles and reiterated in her present reply, suggesting different spatio-temporal origins of the ‘Second Plague Pandemic’ in general and the Black Death in particular, from those that historians and scientists have been arguing. The main points of that theory can be summarized as follows: (i) the ‘Big Bang’ that precedes the Black Death — the polytomy/multifurcation event whereby the main plague lineage (Branch 0) split into four new plague lineages (Branches 1, 2, and another short branch, which soon would split into two branches — Branches 3 and 4) occurred in early thirteenth-century Central Asia, possibly in the Tian Shan region;\textsuperscript{6} (ii) the expansionist campaigns of the rising Mongol Empire helped to spread the emergent plague branches all over Eurasia during the thirteenth century; (iii) in the course of Hülegü Qan’s campaigns in the 1250s, Branch 1 got focalized in or around the Volga-Caucasus region, re-emerging in (or possibly before) the

\textsuperscript{5} The ‘Great Polytomy’ (aka, the Big Bang) was a multifurcation event whereby the main plague lineage (Branch 0) split into four new plague lineages (Branches 1, 2, and another short branch, which soon would split into two branches — Branches 3 and 4), discussed below.

\textsuperscript{6} Green herself spoke about ‘a fourfold divergence in \textit{Y. pestis}’s evolutionary tree’ (p. 8), even though the Big Bang involved really a ‘threelfold divergence’, with one short branch shortly splitting into two branches (Branches 3 and 4).
1330s; (iv) the Black Death of the 1340s and early 1350s originated in the same focus.7

However, most recently, and following Green’s previous publications, three aDNA specimens from East Syriac (Nestorian) burials at Kara-Djigach (the Semirech’ye/Zhetysu region of north-western Tian Shan, in North Kyrgyzstan), have been sequenced, all associated with the 1338–9 plague outbreak there. These new specimens settle centuries-old controversy about the spatio-temporal origins of the Black Death and the emergent debate about the spatio-temporal origins of the ‘Big Bang’ and the ‘Second Plague Pandemic’. In a nutshell, a palaeogenetic analysis of the three Kara-Djigach individuals revealed first, that the ‘pestilence’ found inscribed on ten tombstones from 1338–9 was indeed plague, caused by its bacterial agent *Yersinia pestis*. Second, and just as importantly, the phylogenetic position of the Kara-Djigach genomes falls right on a node (the ‘Great Polytomy Node’, also known as ‘N07’) that gave birth to the ‘Big Bang’/the Great Polytomy event, whereby the main plague lineage (Branch 0) split into four new lineages (Branches 1–4). Finally, the study found that the Great Polytomy itself followed shortly after the Kara-Djigach outbreak.8

Thus, although Green’s hypothesis regarding the geographic origins of the ‘Big Bang’ turned out to be correct, her theory regarding the timing of the ‘Big Bang’ and the spatio-temporal origins and spread of the Black Death proved to be, unfortunately, incorrect: the Kara-Djigach evidence shows that the ‘Big Bang’ took place a century later than she had thought. The ‘new-born’ post-polytomy plague strains were not spread by the Mongols in the early thirteenth century; there is no evidence that a new plague reservoir was seeded in the Caucasus-Volga

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8 Spyrou *et al.* established the phylogenetic position of the Kara-Djigach genomes and the timing of the poltomy by conducting a comparative SNP (single nucleotide polymorphism) analysis between the Kara-Djigach genomes on the one hand and previously published historical and modern *Y. pestis* genomes on the other: Spyrou *et al.*, ‘Source of the Black Death in Fourteenth-Century Central Eurasia’, suppl. tables 13–15. SNP is a 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.
regions in the 1250s, in the course of Hülegü’s campaigns; and, by extension, the Black Death itself did not originate in the same hypothesized Caucasus-Volga focus. Rather, the Black Death, commencing immediately or shortly after the Big Bang, would spread all the way from Tian Shan to Crimea between the late 1330s/early 1340s and 1346, along long-distance trans-Asian inland trade routes (often referred to as the ‘Silk Road’). The possibility that the Black Death spread, caused by Branch 1, had not started until 1341, is discussed in my forthcoming article, ‘A Rise and Fall of a Chaghadaid Community: Demographic Growth and Crisis in “Late-Medieval” Semirech’ye (Zhetysu), c.1248–1345’, in Journal of Royal Asiatic Society. As the evidence of epigraphic corpus from the Kara-Djigach cemetery suggests, the 1338–9 plague outbreak was followed by another mass mortality event in 1341–2, killing virtually all the survivors of the 1338–9 crisis. In contrast to the 1338–9 headstones, no epitaph from 1341–2 indicates the cause of the mortality crisis. The environmental context of the ‘Big Bang’ and the early spread of plague in the 1340s from Central Asia to the Azov and Crimea will be explored in detail in two further studies currently in progress.

But let us imagine for a moment that while en route from Tian Shan to Crimea in the early 1340s, Branch 1 did focalize in the Caucasus-Volga region. After all, there are, as of today, two dozen active plague foci in the Volga, Caucasus and nearby Caspian and Amu Darya regions. As genetic evidence from the twentieth and early twenty-first centuries indicates, the post-polytomy 2.MED1 is the single most prevalent branch in these foci, with the ancient pre-polytomy branch 0.PE2 (emerging around 2500 BCE and belonging to the microtus sub-group of plague lineages, which, although deadly in rodents, causes endemic rather than epidemic outbreaks in humans) the next most prevalent, and with post-polytomy 2.MED0 and 2.MED4 branches being less prevalent — but, crucially, not any sub-branch of Lineage 1, responsible for the Black Death, *pestis secunda* and subsequent plague waves. Indeed, as of May 9

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9 The possibility that the Black Death spread, caused by Branch 1, had not started until 1341, is discussed in my forthcoming article, ‘A Rise and Fall of a Chaghadaid Community: Demographic Growth and Crisis in “Late-Medieval” Semirech’ye (Zhetysu), c.1248–1345’, in Journal of Royal Asiatic Society. As the evidence of epigraphic corpus from the Kara-Djigach cemetery suggests, the 1338–9 plague outbreak was followed by another mass mortality event in 1341–2, killing virtually all the survivors of the 1338–9 crisis. In contrast to the 1338–9 headstones, no epitaph from 1341–2 indicates the cause of the mortality crisis. The environmental context of the ‘Big Bang’ and the early spread of plague in the 1340s from Central Asia to the Azov and Crimea will be explored in detail in two further studies currently in progress.


2022, of over 100 sequenced and genotyped modern strains (dated from 1917 to 2015), not a single one belongs to any of Lineage 1 sub-branches. In contrast to the Central European reservoir, which died out at some later point, the Volga, Caspian and Caucasian foci are still active. If, as Professor Green hypothesizes, Branch 1 got focalized in one of these regions in the 1250s (that is, some century before the beginning of the *pestis secunda*), why are its strains not found there today? If we were to assume that Branch 1 did become extinct there, why did not other branches — both the much older 0.PE2 and the post-polytomy Lineage 2 sub-branches?

Thus, the spatio-temporal trajectory of the Big Bang in general and the Black Death in particular, in the 1330s and 1340s, as well as the genetic evidence from the active Caucasus-Volga plague foci, when taken together, undermine Green’s arguments regarding the geography of origins and spread, as well as the epidemiological agency of the 1356–66 wave. All the available evidence does not, in any way, point to the Caucasus-Volga region as the ‘proximate’ home of either the Black Death, or the subsequent wave.

II

NO EVIDENCE FOR CONCURRENT ACTIVITY OF BRANCHES 1A AND 1B

Closely linked to the previous point is Green’s hypothesis about the concurrent activity of Branch 1A and 1B sub-branches during the plague wave of 1356–66 — another aspect, where her and my interpretations fundamentally differ. I hold that all outbreaks all over West Eurasia and North Africa associated with the wave that had commenced in south-central Germany in 1356 (the wave I refer to as the *pestis secunda*), were caused by Branch 1B strains radiating out of the south-central German reservoir. Conversely, Green assumes that the outbreaks of the same wave were caused by strains of both Branch 1A (which, as she tends to agree, seems to have originated in the south-central German reservoir and whose circulation was, according to her, found in central and southern continental Europe), and Branch 1B (radiating out of

\[n. \text{ 11 cont.}\]
the Caucasus-Volga focus and proliferating in northern and north-eastern Europe). Green’s hypothesis that at least two plague lineages were striking concurrently in these years (Branch 1A and Branch 1B), led her to refer to the 1356–66 pandemics as the ‘second plague’, rather than *pestis secunda*, as I do.\(^{12}\)

It should be noted that Green is not the first person to suggest the theory of concurrent circulation of both sub-branches 1A and 1B: it has already been put forth by Guellil *et al.*’s study (the same study that sequenced the Collalto Sabino genome discussed below).\(^{13}\) What is important, however, is that Green asserts that the south-central German reservoir spread 1A strains, while the Caucasus-Volga reservoir spread 1B strains, implying that the 1356–66 wave was spreading from two reservoirs, with at least a partial degree of a chronological overlap.\(^{14}\)

Unfortunately, this interpretation is contradicted by the chrono-geography of *pestis secunda* spread, indicated by both textual evidence and (as shown below) phylogenetic data. If we were to assume that both reservoirs were sending, concurrently or nearly concurrently, plague waves all over West Eurasia, then we would expect to see plague in the Golden Horde lands/Russian principalities at or around the beginning of the *pestis secunda* wave in the late 1350s (as was indeed the case in the German Empire), or at least in the middle of the same wave. Here, Green does not provide any detailed chronology for Golden Horde plague outbreaks. Instead, referring to T. F. Khaydarov and D. A. Dolbin,\(^{15}\) she asserts that ‘from chronicle evidence, T. F. Khaydarov and D. A. Dolbin show intense epidemic activity not simply in the well-attested outbreaks of 1346, but from 1358 continuing into the fifteenth century’.\(^{16}\)

\(^{12}\) Green (p. 4 n. 7).

\(^{13}\) ‘These two strains point toward the existence of two distinct lineages circulating in Europe during the Pestis Secunda (1357 to 1366). These strains were probably introduced in multiple waves from outside of Europe, maybe to Western and Eastern port cities, as previously hypothesized by Namouchi *et al.*: Meriam Guellil *et al.*, ‘A Genomic and Historical Synthesis of Plague in 18th Century Eurasia’, *Proceedings of the National Academy of Sciences USA*, cxvii (2020), 3–4.

\(^{14}\) Green even suggested some geographic overlap: ‘At the minimum, two different lineages of *Y. pestis* were likely to have been circulating within Europe in the 1350s and 60s, perhaps with some geographic overlap’ (p. 5).


\(^{16}\) Green (p. 33).
In reality, however, Khaydarov and Dolbin did not mention any plague outbreak in the Golden Horde territory in 1358 — not least because there has never been one.17 The only textually documented plague outbreak in the Golden Horde lands between the Black Death there (1345–7) and the 1374–5 outbreak is that from 1363–4, namely towards the end of the pestis secunda wave.18 As I make it clear in my study, by 1363–4, the pestis secunda had completed most of its vicious journey, having ravaged all of Central and Western Europe, and was approaching its devastating finishing line in Russian principalities (1364–5) and Lithuania (1366).19 In other words, textual evidence indicates that the plague came to the Jochid domains towards the end of the 1356–66 wave — and not the beginning, as Green has suggested. The geographic routes, by which the pandemic reached the Golden Horde, are discussed in the next section of this article.

To corroborate her hypothesis about the spatio-temporal contours of plague spread during the 1356–66 wave, Green refers to palaeogenetic evidence. Before going into her analysis,

17 The closest thing, chronologically, mentioned in that study, is a 1359–60 outbreak in Chobanid province of Adharbaijan, mentioned in Fasikh Khavafi’s Majmal-i Fasih (Fasikh al-Khavafi. Fasikhov Svod, trans. D. Yu. Yusupova (Tashkent, 1980), 88). The only plague-related event in the Golden Horde territory in the 1360s mentioned by Khaydarov and Dolbin is the 1364 outbreak in Volga cities: the very same outbreak I mention on p. 12 of my original article. Also, the claim that ‘Khaydarov and Dolbin, drawing on much more varied source material than Slavin identifies’ (p. 46 n. 77) is simply wrong, as just a cursory look at my Supplementary Data 1 demonstrates. One author who did suggest that there was a plague in the Volga region in 1358 is Schamiloglu: Uli Schamiloglu, ‘The End of Volga Bulgarian’, in Varias: Festschrift für Professor András Róma Tás (Szeged, 1991), 158–61; Uli Schamiloglu, ‘The Impact of the Black Death on the Golden Horde: Politics, Economy, Society, Civilization’, Zolotoordynskoye Obozreniye, v (2017), 337. Here, Schamiloglu noted that an excessive number of surviving headstone epitaphs in Volga Bulgarian language from 1357, as well as the fact that there are no Volga Bulgarian epitaphs after 1358, may indicate that local communities were ravaged by plague around those years. However, unlike the Kara-Djigach corpus, where we have 118 surviving tombstones from 1338 and 1339 (compared to just several stones in a ‘normal’, non-crisis year), Schamiloglu’s six stones from 1357 (compared to one to three surviving stones a year, not to mention years without any surviving stones) is too low a figure to reflect excessive mortality caused by plague; let alone the fact that none of these headstones mention ‘pestilence’ — unlike 10 (out of 118) Kara-Djigach inscriptions from 1338 and 1339.

18 Slavin, ‘Out of the West’, 9–12, and Supplementary Figure 1.

19 Slavin, ‘Out of the West’, 12, and Supplementary Figure 1; T. F. Khaydarov, Epoka ‘Chernoi smerti’ v Zolotoi Orde i prilegayushchikh regionakh (konets XIII — pervaya polovina XV vv.) (Kazan, 2018), 295–6.
let us first summarize our current palaeogenetic knowledge of Branch 1B circulation in the fourteenth century. As of May 2022, only four Branch 1B genomes from three sites have been published: (i) two genomes from Bergen-op-Zoom in the Netherlands, (ii) one genome from London St Mary Graces, and (iii) one genome from Bolghar city in the Republic of Tatarstan. All four genomes are positioned on Branch 1B, associated with the *pestis secunda* wave. The thin geographic coverage is, however, not the only challenge in the analysis and interpretation of Branch 1B palaeogenetic data. Another major methodological issue is the fact that the respective phylogenetic positions stated for these four genomes differ from publication to publication (see Figure 1). According to the publications by Namouchi et al. (2018), Spyrou et al. (2019) and Giffin et al. (2020), the Bergen-op-Zoom genomes are positioned first on the 1B line, followed by the London and then Bolghar. The publication by Guellil and her colleagues (2020) puts Bergen-op-Zoom first and then London and Bolghar, roughly, concurrently. Conversely, according to Seguin-Orlando et al. (2021), Bergen-op-Zoom is placed first, followed by Bolghar and then London.

20 While there is documentary evidence for the foundation of the plague burial at St Mary Graces (1361), the dating for the Bergen-op-Zoom and Bolghar burials (c.1359 and 1364, respectively) have been suggested in my paper, on the basis of textual evidence describing plague spread in their neighbouring cities/regions.

21 Amine Namouchi et al., ‘Integrative Approach using *Yersinia pestis* Genomes to Revisit the Historical Landscape of Plague during the Medieval Period’, *PNAS*, cxv (2018), fig. 3B (London with 0 SNP defining Branch 1B and 3 ‘private’ SNPs in relation to Bergen-op-Zoom; Bolghar with one SNP defining Branch 1B and an additional ‘private’ SNP in relation to Bergen-op-Zoom); Maria A. Spyrou et al. ‘Phylogeography of the Second Plague Pandemic Revealed through Analysis of Historical *Yersinia pestis* Genomes’, *Nature Communications*, x (2019), fig. 2; 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’, *Scientific Reports*, x (2020), fig. 4. Both Spyrou et al. and Giffin et al. report London with 0 SNP defining Branch 1B and one ‘private’ SNP in relation to Bergen-op-Zoom, and Bolghar with one SNP defining Branch 1B and one ‘private’ SNP in relation to Bergen-op-Zoom). In my ‘Out of the West’ (n. 71 and Figure 3), I have adopted Spyrou et al.’s figures.

22 Guellil et al., ‘Genomic and Historical Synthesis of Plague in 18th Century Eurasia’, fig. 3 (the authors do not indicate the number of SNPs, but it appears that, according to their analysis, both Bolghar and London each accumulated one SNP defining Branch 1B and one ‘private’ SNP in relation to Bergen-op-Zoom).

23 Andaine Seguin-Orlando et al., ‘No Particular Genomic Features Underpin the Dramatic Economic Consequences of 17th Century Plague Epidemics in (cont. on p. 334)
omitting the Bergen-op-Zoom genome, the London genome is placed immediately before Bolghar, while the publication by Morozova et al. (also from 2020), omitting the London genome, places Bolghar before Bergen-op-Zoom; neither publication explains the exclusion of the respective genomes. Most recently (and after Green’s reply), Spyrou et al.’s study, based on a meticulous and careful analysis of all the available genomic data, reconfirmed the original positional order of the 1B phylogeny, with the Bergen-op-Zoom genomes being first, followed by the London and then the Bolghar ones (see Figure 1.1). What can be made of these glaring phylogenetic discrepancies across different publications?

That palaeogenetic work offers the most valuable contribution to plague studies is a truism requiring no argument. Palaeogenetic publications over the last ten or so years took this field to altogether new heights, asking and answering new questions and providing new data, insights and interpretations that would otherwise have been totally hidden from our sight. But one has also to be careful and thoughtful when using and integrating this new data into historical studies. It is essential to keep in mind that the analysis, interpretations, calculations and results deriving from the same data (namely, the same genomes) can also vary across studies — as the example of the pestis secunda genomes demonstrates. Technically speaking, these discrepancies derive from the fact that different teams use different ‘variant

(n. 23 cont.)

Italy’, *iScience*, xxiv (2021), 102383, fig. 3b (the authors do not indicate the number of SNPs, but it appears that, according to their analysis, Bolghar accumulated one SNP defining Branch 1B in relation to Bergen-op-Zoom, while London accumulated two SNPs defining Branch 1B in relation to Bergen-op-Zoom and one in relation to Bolghar).

24 Julian Susat *et al.*, ‘*Yersinia pestis* Strains from Latvia Show Depletion of the *pla* Virulence Gene at the End of the Second Plague Pandemic’, *Scientific Reports*, x (2020), 14628, fig. 2 (the authors do not indicate the number of SNPs, but it appears that, according to their analysis, Bolghar accumulated one SNP defining Branch 1B in relation to London).

25 Irina Morozova *et al.*, ‘New Ancient Eastern European *Yersinia pestis* Genomes Illuminate the Dispersal of Plague in Europe’, *Philosophical Transactions of the Royal Society B: Biological Sciences*, ccclxxv (2020), fig. 2 (the authors do not indicate the number of SNPs, but it appears that, according to their analysis, the Bergen-op-Zoom genomes accumulated one SNP defining Branch 1B in relation to the Bolghar one).

26 Spyrou *et al.*, ‘Source of the Black Death in Fourteenth-Century Central Eurasia’.
lists’ (variant files) of all genomes used in their analyses; some teams’ analyses/publications exclude or include certain genomes that were included/excluded by other teams’ analyses/publications. The different positioning of *pestis secunda* genomes

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**FIGURE 1**

DIFFERENT PHYLOGENETIC POSITIONING OF FOURTEENTH-CENTURY BRANCH 1 GENOMES BY DIFFERENT STUDIES*

1.1 Spyrou *et al.* 2019 and 2022 positioning

1.2 Guellil *et al.* 2020 positioning

1.3 Susat *et al.* 2020 positioning

1.4 Morozova *et al.* 2020 positioning

1.5 Seguin-Orlando *et al.* 2021 positioning


Notes: LA = Laishevo; LC = Saint-Laurent-de-la-Cabrerisse; BA = Barcelona; SI = Siena; TL = Toulouse; LO = London East Smithfield; OS = Oslo; NA = Nabburg; BZ = Bergen-op-Zoom; LO* = London St Mary Graces; BL = Bolghar city; MP = Manching-Pichl; CS = Collalto Sabino.
is by no means the only such example.\textsuperscript{27} We return to this issue in the next section, in conjunction with the timing of the Branch 1A/1B split and the positioning of Black Death genomes.

These discrepancies can undoubtedly create some confusion for the historian, who has to acknowledge them and make their case for adhering to a particular interpretation and analysis. It is important that, where possible, that choice is backed up and corroborated by historical (textual) data. Green based her arguments on Seguin-Orlando \textit{et al.}'s study (see Figure 1.5),\textsuperscript{28} without, unfortunately, explaining why she adopted Seguin-Orlando \textit{et al.}'s analysis and results and why she found these to be more reliable than the other publications (those whose phylogenetic positionings are shown in Figure 1).

Importantly, as we have seen, Seguin-Orlando \textit{et al.} positioned the Bergen-op-Zoom genomes before both the London and Bolghar ones. This should, in fact, contradict rather than corroborate Green's hypothesis of the \textit{pestis secunda} origin in the Golden Horde reservoir, and its subsequent transmission along the Volga and North Sea routes, via Hanseatic trade. This is because Seguin-Orlando \textit{et al.}'s analysis and phylogenetic positioning indicate that the Bergen-op-Zoom outbreak came before the plague reached Bolghar — as indeed is also supported by textual evidence, as we shall see below.

In other words, the thin palaeogenetic evidence of fourteenth-century history of Branch 1B and its contradictory phylogenetic analyses between different studies, available to Green at the time of her writing, certainly does not permit any substantial claims regarding the geography of its (i) origins (a Caucasus-Volga

\textsuperscript{27} Thus, Morozova \textit{et al.}, ‘New Ancient Eastern European \textit{Yersinia pestis} Genomes’, fig. 2, is one example, in which several genomes have been repositioned into very unlikely phylogenetic positions: (i) the Laishevo genome, situated between the ‘Big Bang’ and European Black Death genomes, is positioned as ancestral on Branch 2; (ii) the Black Death genomes are placed in different phylogenetic positions, rather than in the identical ones, as in other publications (see discussion below); (iii) the Rostov2039 genome from the Rostov-on-Don fortress is positioned with other late fifteenth-/early sixteenth-century genomes around the Little Polytomy (see discussion below), despite the fact that the fortress was founded only in 1761 — and despite the fact that it allegedly came from the same context as the other Rostov-on-Don genome (Rostov2033, dated externally to 1771, and positioned at a much later phylogenetic position than Rostov2039). Most recently, Spyrou \textit{et al.}, ‘Source of the Black Death in Fourteenth-Century Central Eurasia’, reconfirmed Rostov2039’s position on the Little Polytomy.

\textsuperscript{28} Green (pp. 23–9).
reservoir), (ii) spread (east–west), and (iii) circulation (north-eastern and northern Europe). The first point is contradicted by the phylogenetic evidence from active plague foci in the Caucasus-Volga and adjacent Caspian and Amu Darya regions, as shown in the previous section. The second point is contradicted by the recent reanalysis of the 1B genomes by Spyrou et al., reaffirming the ancestral position of the Bergen-op-Zoom genomes, followed by London and then Bolghar, and thus supporting my view of the western (south-central Germanic) origin of 1B lineage in general and the *pestis secunda* in particular, as well as my chronological reconstruction of the *pestis secunda* spread, based on textual evidence.\(^\text{29}\)

But what about the third point? Surely, it cannot rest solely on the very thin phylogeographic evidence we have at the moment. To make up for that, Green, again, refers to palaeogenetic data — or to be more precise, to two undated (but clearly later fourteenth-century) genomes — one from Manching-Pichl in Upper Bavaria and the other one from Collalto Sabino in the province of Rieti, Lazio (Central Italy) (see Figure 1). While the former had been published in 2019 and was included in my discussion,\(^\text{30}\) the latter was published in late October 2020, when my study was in the typesetting stage, with no further additions possible.\(^\text{31}\) Nevertheless, the publication of the Collalto Sabino genome does not change any of my original arguments. Both Manching-Pichl and Collalto Sabino genomes are clustered together on an early position on the 1A branch — namely, on a different line from that attested in the Bergen-op-Zoom, London St Mary Graces and Bolghar city genomes (Branch 1B). Green’s interpretation of these two Branch 1A genomes is as problematic as that of Branch 1B ones.

In my own study, I have suggested that the Manching-Pichl genome is associated with one of the post-*pestis secunda* outbreaks — possibly 1367, 1373 or 1380 outbreaks that ravaged Bavaria.\(^\text{32}\)

\(^{29}\) Slavin, ‘Out of the West’, 9–12, and Supplementary Figure 1.

\(^{30}\) Ibid., 43.

\(^{31}\) Guellil et al., ‘Genomic and Historical Synthesis of Plague in 18th Century Eurasia’.

\(^{32}\) Slavin, ‘Out of the West’, 43. My subsequent research into the geographic contours of the *pestis tertia* revealed both the 1367 and 1373 dates to be incorrect; instead, it can now be established that the plague ravaged Bavaria in 1371–2 (see Hermann Schöppler, *Die Geschichte der Pest zu Regensburg* (München, 1914), 20–1).
My association of the Manching-Pichl genome (and now, by extension, also of the Collalto Sabino one) with post-pestis secunda outbreaks derives from their phylogenetic positioning, based on the genomes’ accumulation of SNPs in relation to Black Death and pestis secunda genomes. The argument is that (i) their phylogenetic positioning comes after all the sequenced pestis secunda genomes (in the case of the Manching-Pichl genome, there are a maximum of 12 SNPs separating it from the Black Death genomes — four SNPs defining Branch 1A and eight ‘private’); (ii) in the case of the evolutionary history of Branch 1, the ‘average’ mutation rate of one SNP is 4–4.5 years; and (iii) my assessment that during the pestis secunda wave, only 1B branch was epidemiologically active. By contrast, Green challenges this interpretation and suggests that both the Manching-Pichl and Collalto Sabino genomes are associated with what I refer to as the pestis secunda wave. She bases her theory entirely on the fact that the authors of the Collalto Sabino genome publication ‘felt the Collalto Sabino burial accorded with local evidence for the 1363 outbreak of plague in central Italy’.

A bit later, Green rejected my estimate of one SNP in 4–4.5 years (which, indeed, is an ‘average’ approximation; I am fully aware that the speed could vary a great deal across space and time). Instead she stated that the accumulation rate was faster (every 1.66 years), on the basis of her association of the Collalto Sabino genome with the 1363 outbreak and, hence, the assumed time lag of fifteen years between the Black Death (1348) and the Collalto Sabino genome, in the course of which nine SNPs have been acquired. But as all the available palaeogenetic and phylogenetic evidence indicates, one SNP per 4–4.5 SNPs years

33 For the definition of SNP, see n. 8 above.
34 As indeed calculated and noted by Green (p. 16). In the case of the Collalto Sabino genome, whose data was not yet published by the time of the submission and revision of my piece, nine SNPs (four defining Branch 1A and five ‘private’) separate it from the Black Death genomes.
35 Slavin, ‘Out of the West’, 27.
36 Green (p. 14). Here, Green cites a single laconic entry from (a very brief and scant) Rieti Annals (Annales Reatini, ed. L. C. Bethmann, in Monumenta Germaniae Historica, Scriptores, xix (Hannover, 1866), 267–8) that served as the base for Guellil et al.’s association of the Collalto Sabino genome with the 1363 outbreak (citing Étienne Hubert, L’ “Incastellamento” en Italie centrale: pouvoirs, territoire et peuplement dans la vallée du Turano au Moyen Âge (Rome, 2002), 407).
37 In fact, Green miscited it as ‘4.5’ rather than ‘4–4.5’ years: ‘Slavin proposed a mutation rate of 1 SNP every 4.5 years’ (p. 17 n. 33).
38 Green (p. 17 n. 33).
as an average estimate should by no means be too removed from reality: there are ninety SNPs and about 380 years separating the Big Bang and the Great Plague of Marseille, indicating about 4.2 SNPs a year.\footnote{As already stated in Slavin, ‘Out of the West’, 27. The numbers of SNPs are reported in Kirsten I. Bos \textit{et al.}, ‘Eighteenth Century \textit{Yersinia pestis} Genomes Reveal the Long-Term Persistence of an Historical Plague Focus’, \textit{eLife}, v (2016), fig. 3b.}

The dating of the Manching-Pichl and Collalto Sabino genomes, their association with the \textit{pestis secunda} and the idea that this dating reflects a much faster rate of SNP accumulation than suggested, all rest upon weak and unreliable foundations — consisting, in effect, of an arbitrary association of the Collalto Sabino genome with the 1363 outbreak mentioned in the \textit{Annales Reatini}. In reality, however, there is nothing to suggest an association between that genome and the 1363 outbreak. A closer examination of the \textit{Annales Reatini} reveals that it is a highly fragmentary and laconic source, with numerous glaring lacunae. Between 1054 and 1377 the annals contain merely 34 entries, collectively occupying a folio and a half of a printed text.\footnote{\textit{Annales Reatini}, 267–8.} Most importantly, because the \textit{Annales} lacks a 1374 entry and ends in 1377, it does not mention the later outbreaks of 1374, 1383 and 1389 that ravaged Lazio, Abruzzo and indeed other parts of Italy, as reported in other contemporary sources.\footnote{Luigi Capasso and Arnaldo Capelli, \textit{Le Epidemie di peste in Abruzzo dal 1348 al 1702} (Cerchio, 1993), 41–5; Jean-Noël Biraben, \textit{Les Hommes et la peste en France et dans les pays européens et méditerranéens} (Paris, 1975), 394–5.}

Importantly, I do indeed mention in my piece the 1363 outbreak in Central Italy, in conjunction with the \textit{pestis secunda} spread all over West Eurasia and North Africa.\footnote{Slavin, ‘Out of the West’, 12 and Supplementary Figure 1.} In contrast with Green, however, I maintain that the same 1363 outbreak in Central Italy would also have been caused by the 1B sub-branch, based on my assessment that that wave was radiated out of a single south-central German reservoir, that the same wave was spreading in concentric pulses (as Figure 1 in the original article, based on textual sources, indicates), and the fact that all so-far sequenced 1B genomes are associated with the \textit{pestis secunda}. If the Collalto Sabino genome were associated with the 1363 outbreak in Central Italy, we would expect to see it
positioned on 1B sub-branch with other *pestis secunda* genomes, rather than on 1A sub-branch — given that the same 1363 outbreak in Central Italy was a part of the *pestis secunda* wave. Not only that: phylogenetically, the Collalto Sabino genome is positioned later than *pestis secunda*-associated genomes. This hints that the Collalto Sabino genome is associated with one of the later outbreaks noted above, rather than with that of 1363. It is possible that the 1374 and 1383 outbreaks in Lazio were caused by the same waves as the 1371 and 1380 outbreaks in Bavaria. However, to confirm this point, a systematic reconstruction of plague geography spread during the subsequent late fourteenth-century waves, akin to that conducted in my *pestis secunda* study, is required — something that can certainly not be undertaken within the remits of this short article.

In light of all that, Green’s hypothesis that during the 1356–66 wave (referred to by me as the *pestis secunda*), people across Europe were succumbing to plague caused by both sub-branch 1A (originating in the south-central German reservoir and proliferating, in her view, in Central and Mediterranean Europe) and 1B (according to her hypothesis, radiating out of the Caucasus-Volga reservoir and ravaging north-eastern and northern Europe) is contradicted by palaeogenetic evidence. Because of all these shortcomings, Green, unfortunately, failed to adhere to her own dictum of ‘Doing Epidemiological History Responsibly’. It is only a question of time before new aDNA comes out to answer firmly the question of the geographic origins of the *pestis secunda* of 1356–66, and a more comprehensive phylogeography of Branch 1B strains.

III

DISRUPTION AND DECLINE OF JOCHID-WESTERN TRADE IN THE 1360s

Now, even if we disregard the respective phylogenetic positionings of the four Branch 1B genomes and the absence of Branch 1 strains in the Caucasus-Volga region and surrounding areas (which we should not), and if we were to accept Green’s hypothesis of the geographic origins of the 1356–66 wave in its conjectural Caucasus-Volga reservoir, one question remains: How exactly would the same plague wave spread from the Golden Horde territories around the Volga to the North Sea?
Here, Green takes up an optimistic vision of buoyant international trade connecting the Jochid realm and the Baltic–North Sea regions, with Hanseatic merchants playing a key role as intermediaries.\textsuperscript{43} Sadly, the reality was fundamentally different: the \textit{pestis secunda} was, in fact, also a period of trade disruption and hiatus between the two regions. The rosy days of long-distance trade between the Golden Horde and northern Europe, lasting from the 1260s to the late 1350s, were over.\textsuperscript{44} Following the death of Jani Beg Qan in 1357, the Ulus Juchi entered a period of political chaos, military confrontation and long-term decline. Between 1357 and 1366, that is during the \textit{pestis secunda} wave in Eurasia and North Africa, no fewer than ten rulers came and went, with some ruling for merely a few months.\textsuperscript{45} In addition to internal strife, Jochid rulers also clashed with their western neighbours — in particular, with Algirdas, Grand Duke of Lithuania, who, between 1362 and 1365, managed to conquer vast territories overlapping with today’s western and central Ukraine.\textsuperscript{46} The Jochid weakness was also taken advantage of by Novgorodian pirates known as \textit{ushkuiniks}, who raided Volga towns on numerous occasions, with the 1360s and early 1370s being a period of particularly intense activity.\textsuperscript{47} Although there is no way to establish it securely, it is possible that it was these \textit{ushkuiniks’} raids that

\textsuperscript{43} See Green, pp. 28–29, 31 and 33.

\textsuperscript{44} Archaeological excavations at the Bolghar city bazaar, conducted in 1989–91 and 2011–13, yielded, inter alia, lead-pendant seals attached to textile fabrics produced in Flemish towns, which could have been imported either by Hanseatic or Novgorodian merchants by the Volga routes via Novgorod or Italian traders via the Don route from Crimea or Azov. The seals appear to have been imported in the 1350s, but before the onset of the crisis along the Volga routes, as suggested by the chronology of unearthed coins, dated to 1352–6. See, V. Yu. Koval’, ‘“Torgovyi Inventar” iz Raskopok Bazara Serediny XIV Veka v Bolgare’, \textit{Povolzhskaya Arkheologiya}, iv (2013), 9–33.


\textsuperscript{47} Vadim Telitsyn, \textit{Russkoye Igo ili Nashestviye Ushkuinikov na Zolotuyu Ordu} (Moscow, 2013), 145–200.
helped spread the plague from the Volga basin into Russian principalities in 1364–5.48

Moreover, it should be borne in mind that even in relatively peaceful times in the late thirteenth and early fourteenth centuries, Jochid trade with Europe was usually conducted not directly by travelling Hanseatic merchants, but via east European middlemen — Russian (especially of Novgorod and Smolensk), Ruthenian (especially of Lviv) and Polish merchants.49 The ongoing conflicts with Novgorod and Lithuania ruined any prospects of such commercial brokering, and long-distance trade between the Golden Horde and Europe was thus effectively paralysed. The trade with the west resumed in 1372, with Bulaq Qan and later Arab Shah Qan (in 1379–80) granting privileges to, respectively, Krakow and Lviv merchants.50 But even then, the Polish-Lithuanian-Ruthenian–Jochid trade of the 1370s and 1380s would pass via the so-called Via Thartarica, stretching from Eastern Europe, via Galicia–Volhynia, to the Pontic-Caspian steppe, and not via the Volga route, which was still paralysed because of continuous activities

48 Indeed, the connection between plague outbreaks and ushkuiniks’ activity has been made in L. V. Vorotynstev, ‘“Chernaya Smert” i Fenomen Novgorodskogo Ushkuinichestva (XIV–pervaya polovina XV vv.)’, in Epidemii i prirodnye kataklizmy v Zolotoi Orde i na sopredel’nykh territoriyakh (XIII–XVI vv.), ed. I. M. Mirkaleyev (Kazan, 2018), 209–20.


of the ushkuiniks. In sum, the idea of buoyant riverside trade in the later 1350s and 1360s connecting the Golden Horde with northern Europe, via the Volga-Baltic route, is contradicted by textual evidence — which, in turn, further undermines the idea of the westward spread of the plague wave and the existence of a putative Caucasus-Volga reservoir allegedly sending the same wave.

How, then, did the plague reach Bolghar city? The main contours have already been discussed in my paper, but a reminder, with some additional data, would be beneficial. From Constantinople, where it broke out in 1361, the plague spread, during the course of the next year, eastwards into Anatolian beyliks, the Trebizond Empire, and northwards into the Azov sea region, where it is attested in Venetian Tana in the summer of 1362. Tana is 500 kilometres south of the Volga, and it was connected with Golden Horde cities by the Don route. Unlike the northern routes connecting the Golden Horde with the west via Russian principalities and Lithuania, the Don trade route connecting the Golden Horde with the Azov Sea and Caucasus, seems to have been functioning in the early 1360s. In particular, we hear about commercial and diplomatic voyages of Tana Venetians to New Sarai (the capital of the Ulus Juchi), including, importantly, in 1363 — the same year that the plague reached the capital (and indeed other Volga cities). It appears likely that in addition to merchandise, the Venetians also brought in bacteria on one of these voyages. From New Sarai it was only a matter of time before the plague reached Bolghar city, before spreading into Russian principalities in 1364–5 — possibly in the context of ushkuiniks’ raids, discussed above. Concurrently, the pandemic was spreading into the kingdoms of Georgia and Armenia, most likely from the Trebizond Empire.
In short, both historical and palaeogenetic evidence points to the transmission routes that were exactly the opposite of what Professor Green has suggested. Put simply, the plague did not spread westwards from the Golden Horde to Europe, but eastwards from Europe to the Golden Horde. As we have seen, both textual and palaeogenetic data points out that the plague reached the Golden Horde lands several years after it ravaged the North Sea region.

IV
BRANCH 1A–1B SPLIT DID NOT HAPPEN BEFORE THE BLACK DEATH

This brings us to the next point: the assumption that the split within Branch 1 into the two sub-branches had occurred before the Black Death arrived in Europe in 1347. Here, Green goes back to her prior hypothesis that Branch 1 had focalized in the Caucasus-Volga region in the 1250s and hence, one would expect some genetic change some 90 years later, by the time the Black Death crossed to Europe. In search of confirmation for such putative change, Green relied on a single palaeogenetic study (Seguin-Orlando et al.), which conducted computational re-analysis of all so-far published Branch 1 aDNA genomes. This study estimated that the split within Branch 1 into sub-branches 1A (represented by all so-far sequenced post-Black Death genomes, except pestis secunda ones) and 1B (represented, as of May 2022, by four pestis secunda genomes, discussed above), occurred between 1228 and 1321, with the median date of 1283. All this led Green to conclude — as a matter of fact — that ‘the split between Lineages 1A and 1B did not happen after the Black Death pandemic hit Europe; it occurred before plague was even imported into Europe’.

However, such handling of palaeogenetic data by Green is problematic on several levels. As Green herself admits in the following sentence, ‘this is only one study, of course, and its
findings remain to be confirmed by more sustained analysis.57 But in fact a much bigger issue lies within the nature of molecular dating based on probability computations, which requires some basic clarification here. As of May 2022, palaeogeneticists use BEAST2 (Bayesian Evolutionary Analysis Sampling Trees), a cross-platform programme, to conduct molecular dating — including ‘Maximum Clade Credibility’ trees, whereby the dates of different branch splits are estimated.58 Despite all of its unquestionable advantages, BEAST does not handle branch splits well — whether polytomies or bifurcations (whereby either a new line branches off the main lineage, or a split into two main branches occurs).59 Instead, it treats and displays any nodes as events leading to binary splits (bifurcations), which are assigned divergence dates, expressed as broad chronological intervals (that is, chronological intervals consisting of the earliest and latest dates, in between which a branching-off event occurs). In other words, BEAST produces ‘time-trees’ (phylogenetic tree topologies scaled to time) rather than ‘substitution-trees’ (phylogenetic tree topologies showing evolutionary changes, most often based on likelihood calculations). Therefore, the reliance on BEAST2, coupled with the fact that different teams use different ‘variant lists’ (variant files) of all genomes used in their analyses, may produce unreliable results, contradicted by other publications. Palaeogenetics is still a very young science, where unified methods and standards of practice, varying across labs and teams, are yet to be established — hopefully sooner rather than later. Establishing unified methods and standards would minimize discrepancies in phylogenetic analyses across publications and, thus, do a huge service to both palaeogenetics and historians.

Moreover, Seguin-Orlando et al. were not the first to suggest a pre-1347 date to the 1A–1B split: in 2018, Spyrou et al. estimated the event to have occurred c.1309.60 It is crucial to

57 Green (p. 21).
60 Maria A. Spyrou et al., ‘Analysis of 3800-Year-Old Yersinia pestis Genomes Suggests Bronze Age Origin for Bubonic Plague’, Nature Communications, ix (2018), suppl. fig. 6.
understand that both studies were published before the analysis and publication of the Kara-Djigach genomes, and hence, they neither did, nor could include these in their probability computations. Now, thanks to the analysis and publication of those more recent genomes, we know that the pre-Black Death polytomy happened shortly before the Black Death and not in the early thirteenth century; while the dating of the 1A–1B split has been positioned, in a firm manner, shortly after the Black Death’s arrival in Europe. This confirms my assumption regarding the timing of the split and refutes Green’s interpretation.

This brings us to another point related to the handling of palaeogenetic data. In discussing and making use of Seguin-Orlando et al.’s publication, Green notes the authors’ phylogenetic repositioning of previously published Black Death genomes. By the time that Green wrote her response (that is, before the publication of the three Kara-Djigach genomes most recently published by Spyrou et al. and shown to be positioned right on the node immediately preceding the Great Polytomy), there were ten published genomes from eight sites associated with the first wave of the Second Pandemic — namely, with what became known as the Black Death of the 1340s and early 1350s. These are: one from Laishevo (on the Volga–Kama confluence in the Republic of Tatarstan), one from Barcelona, one from Saint-Laurent-de-la-Cabrerisse in southern France, one from Toulouse, one from Siena, three from London East Smithfield, one from Oslo, and one from Nabburg in Bavaria.

Just as with the 1B genomes, the phylogenetic positioning of Black Death-associated genomes varies across publications, reflecting the same methodological and dataset discrepancies discussed in section II (see Figure 1). As noted, these discrepancies derive from the fact that different teams use different ‘variant lists’ of all genomes used in their analyses, with some teams choosing to exclude/include certain genomes that were included/excluded by other teams. Thus, several publications place the Laishevo genome ancestral to all nine European Black Death genomes, all positioned together after the Laishevo genome, having accumulated one SNP in relation to

61 Seguin-Orlando et al., ‘No Particular Genomic Features Underpin the Dramatic Economic Consequences of 17th Century Plague Epidemics in Italy’, fig. 3.
the Laishevo one, and two SNPs in relation to the Great Polytomy Node. One publication (Guellil et al.) places all ten genomes (nine European and the Laishevo one) together. Another (Susat et al.) groups the Barcelona, Laishevo and Nabburg genomes roughly together after the London East Smithfield ones (omitting the Saint-Laurent-de-la-Cabrerisse, Toulouse, Siena and Oslo genomes). Another one (Morozova et al.) establishes a highly complex and suspicious topology, with Laishevo positioned on Branch 2, London East Smithfield genomes placed as ancestral to all other Branch 1 Black Death genomes, followed by Barcelona, then Nabburg and Toulouse, and then Oslo.

Conversely, according to Seguin-Orlando et al.’s study: (i) a genome from Laishevo (Tatarstan), originally positioned on the phylogenetic tree after the Great Polytomy Node (one SNP away from the Great Polytomy Node) but before European Black Death genomes (two SNPs away from the Great Polytomy Node), has been repositioned together with two Black Death genomes (one from Nabburg in north-eastern Bavaria of today and one from Oslo), in a single cluster; (ii) the Laishevo, Nabburg and Oslo genomes either share an immediate common ancestor, or that from Nabburg was actually more basal than Laishevo and Oslo. Thus, Seguin-Orlando et al.’s analysis suggests a much higher rate

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63 Spyrou et al. ‘Phylogeography of the Second Plague Pandemic Revealed through Analysis of Historical *Yersinia pestis* Genomes’, fig. 2; Giffin et al., ‘Treponemal Genome from an Historic Plague Victim Supports a Recent Emergence of Yaws’, fig. 4. Similarly, Namouchi et al., ‘Integrative Approach using *Yersinia pestis* Genomes to Revisit the Historical Landscape of Plague during the Medieval Period’, fig. 3B, published before the publication of the Laishevo genome, places all the Black Death genomes together, albeit calculating two ‘private’ SNPs to the Siena genome (which may, in fact, be environmental signals dominating in low-coverage genomes, rather than ‘real’ SNPs — as I have suggested in ‘Out of the West’, 28 n. 72).

64 Guellil et al., ‘Genomic and Historical Synthesis of Plague in 18th Century Eurasia’, fig. 3.

65 Susat et al., ‘*Yersinia pestis* Strains from Latvia Show Depletion of the *pla* Virulence Gene at the End of the Second Plague Pandemic’, fig. 2.

66 Morozova et al., ‘New Ancient Eastern European *Yersinia pestis* Genomes’, fig. 2.

67 Spyrou et al., ‘Phylogeography of the Second Plague Pandemic Revealed through Analysis of Historical *Yersinia pestis* Genomes’, figs. 2 and 3.

68 Seguin-Orlando et al., ‘No Particular Genomic Features Underpin the Dramatic Economic Consequences of 17th Century Plague Epidemics in Italy’, fig. 3A.

69 Ibid., fig. 3B. According to the same study, the second cluster of Black Death genomes (complementing the Laishevo–Nabburg–Oslo one) included these from Barcelona, Toulouse and London.
of mutation during the Black Death years. Most recently (and after Green published her response), the fact that the Laishevo genome does indeed precede all other Black Death genomes by one SNP (with all Black Death genomes positioned together), and that there is no evidence of such a high rate of concurrent mutation among different Black Death genomes, has been reconfirmed by Spyrou et al., in their recent analysis and publication of the Kara-Djigach genomes.70

Just as with the pestis secunda genomes, Green accepted the new repositioning of the Black Death genomes by Seguin-Orlando et al. without questioning its accuracy, and took it as an indication of the early plague dispersal from the Black Sea up the Danube: in particular on the basis that the Nabburg genome was ancestral to the Laishevo (and Oslo) ones.71 The spread of the Black Death by the Danube is nothing new: it already appears clearly on Benedictow’s map of the plague spread in Europe in 1346–53, based entirely on textual evidence, in both the first (2004) and second (2021) editions of his Black Death.72 What is more important here, however, is that the Nabburg genome cannot be either ancestral to or clustered together with the Laishevo genome — as indicated by both textual and palaeogenetic evidence. The position of the Nabburg genome together with all other European Black Death genomes has been reconfirmed by Spyrou et al., in their recent analysis and publication of the Kara-Djigach genomes (see Figure 1.1). Green, quoting Benedictow, was correct to note that ‘in Bavaria, plague may have been arriving from at least two directions’.73 What she did not note is the spatio-temporal aspects of the plague entrance and dispersal in Bavaria. Grossly oversimplifying, we may summarize that the southern parts of today’s Bavaria (overlapping with Swabia, Upper Bavaria and western parts of Lower Bavaria) were invaded by the plague in summer of 1348, while northern Bavaria (overlapping with most of Lower Bavaria, Upper Palatinate and Franconia) and neighbouring Bohemia were spared until 1349, or 1350.74

70 Spyrou et al., ‘Source of the Black Death in Fourteenth-Century Central Eurasia’.
71 Green (pp. 24–26).
73 Green (p. 26).
Although we do not have direct textual evidence from Nabburg proper, its situation in the Upper Palatinate region hints that it was hit by the plague in 1349–50 — namely some 3–5 years after the plague’s arrival in the Volga region (late 1345/early 1346).\(^n\)

V

THE *PESTIS SECUNDA* INTRODUCED INTO ENGLAND EITHER FROM GASCONY OR SOUTH-WESTERN LOW COUNTRIES, EARLY 1361

Firmly believing in co-circulation of the two sub-branches, Green puts forth, as an example, the possibility that London, where 1B genomes are attested in its 1361 burial at St Mary Graces, could be concurrently hit by 1A strains.\(^n\) Leaving aside the fact that this view is not based on any evidence, let us examine her arguments. Green states that ‘English sources claim that plague arrived in London or the south of England either in late September 1360 or around March or April of 1361’, citing vaguely four pages from Rosemary Horrox’s classic (and incredibly useful) *Black Death* textbook — but not specifying what these sources are.\(^n\) In fact, the four pages in question contain eight short-ish excerpts from English chronicles dealing with the *pestis secunda*. Seven out of eight chronicles give ‘1361’ (with only one, the Continuator of Ralph Higden’s *Polychronicon*, specifying the seasonality — ‘around Easter’), and only one (the *Annals of Great Friars of Lynn*) note ‘at about Michaelmas 1360’.\(^n\) The Lynn annalist, living some 170 kilometres north of London, while providing much valuable information about the events in King’s Lynn and its hinterland,\(^n\) clearly supplied a wrong date for the beginnings of the pandemic in London. This is corroborated by the evidence of London wills, probated at the Court of Husting, which indicate that the rise in mortality did not start until April 1361 (see Figure 2) — as the continuator of Ralph Higden’s *Polychronicon* indeed stated.

\(^{75}\) Ibid., 529–31.
\(^{76}\) Green (p. 37).
\(^{77}\) Green (p. 37).
\(^{78}\) Rosemary Horrox, *The Black Death* (Manchester, 1994), 85–8, nos. 25 (a)–(i).
\(^{79}\) The Latin text, together with commentary, is printed in Antonia Gransden, ‘A Fourteenth-Century Chronicle from the Grey Friars at Lynn’, *English Historical Review*, lxxii (1957), 270–8.
How did the plague come to London? Here, Green adheres to her theory connecting the Volga littoral and the North Sea via the ‘Hanseatic axis’, arguing that the plague arrived in London via the northern route.80 In dismissing my suggestion that, just like the Black Death in 1348 ‘it appears that the *pestis secunda*, too, reached London in March 1361 from Gascony’, she pushed the envelope by stating that ‘Slavin claims, for example, that an alleged plague outbreak in 1361 in the northern Pyrenees — which was then under English control in the midst of the Hundred Years’ War — is not only testimony to the fact that plague had spread by then from Germany to Gascony, but that it was transported from there to England, following the same Atlantic route plague seems to have taken in 1348’.81 There are two issues with this statement: (i) the 1361 outbreak in the northern Pyrenees is not alleged, but real;82 (ii) I did not see this outbreak as a testimony for the plague’s travel from Gascony to London, but merely suggested that the plague may have arrived in London from Gascony. A bit later, Green states that ‘the only source Slavin has for plague’s presence in English-controlled Gascony testifies to plague’s presence there in the summer of 1361’.83 She then refers to an article by Capra and Bériac, dealing with English-occupied Bigorre in 1361–2.84 While the article in question indeed stated that ‘la peste rôde certainement en Bigorre, à partir de l’été 1361’, a source they refer to — an account book of Gerald de Menta, treasurer of Bigorre for 1361–2 — makes no mention of the plague there in the summer.85 A close reading of the account book reveals that all it has on plague is a short entry regarding 60 arpents of land (presumably of vineyard) whose tenants did not pay their annual dues, collectable on the All Saints’ Day (1 November), ‘because of mortality’ *(propter mortalitatem)*.86

80 Green (pp. 29–31).
81 Green (p. 37).
82 As clearly indicated in sources mentioned in nn. 86–88 below.
83 Green (p. 37).
85 *Ibid.*, 152. I have consulted the original document in the National Archives, London (hereafter TNA), E 101/176/3, and cite it, alongside with Capra and Bériac-Lainé’s paper, in my Supplementary Data 1.
86 TNA, E 101/176/3, fo. 2r.
There is not a single surviving source, to my knowledge — be it an administrative document or a chronicle — to reveal when exactly in 1361 the plague broke out in Gascony. All we know is that the plague was there in 1361 — as I indeed specified in the Supplementary Data 1, without speculating about the seasonality. There is, however, one important piece of information (of which I was unaware at the time of writing the article): an eighteenth-century catalogue of the archives of


87 I have gone, in a systematic manner, through all the surviving English administrative documents related to Gascony for the years 1361–3: TNA, E 101/176/2–8, 12–13, 19–20; E 30/1277; and C 61/73–76 — the latter four documents, the so-called Gascon Rolls, are available, in both a digital and transcribed/translated forms, online at <http://www.gasconrolls.org/en/edition/index.html> (accessed May 2022) — but could not find any additional reference to the plague outbreak. Likewise, I have communicated with the archivists of both the Archives de Bordeaux Métropole and Archives départementales de la Gironde, both in Bordeaux, who could not find any documents regarding the 1361 outbreak in Gascony (I am thankful to Frédéric Laux, director of the Archives de Bordeaux Métropole and Béatrice Olive, director of the Archives départementales de la Gironde for being very helpful in this regard). The Chronique de Guyenne, written in a Gascon dialect of Occitan, reports the outbreak misdating it to 1344 (under the ‘1343’ entry), corrected by its editor Lefèvre-Pontalis to ‘1363’ — Germain Lefèvre-Pontalis, ‘Petite chronique de Guyenne jusqu’à l’an 1442’, Bibliothèque de l’École des chartes, xlvii (1886), 63 and 74. In the recently published comprehensive study on the history of plague in late-medieval and early-modern Bordeaux, the authors (dating the plague in Bordeaux to 1362) refer only to the Chronique de Guyenne, and no other sources: Stéphane Barry et Marie Faure, Préservez-nous du mal! Les Bordelais face à la peste, XIV–XVIIIe siècles (Bordeaux, 2021), 47. I am indebted to Frédéric Laux for alerting me to this publication.
Puymirol (in the Garonne region, roughly halfway between Bordeaux and Toulouse) lists a patent letter, now unfortunately lost, dated 29 October 1360. This letter states that the inhabitants of that town were excused any levies, because of plague that ravaged there in the same year. 88 Unless the catalogue supplied an incorrect date (say, mistaking 1360 for 1361), it reveals that the plague may have invaded Gascony in later 1360; in which case, it could have well reached Bordeaux in early 1361, before arriving in London in March of the same year.

Let us return to the question of plague importation into England. Given the insular position of England and the rest of the British Isles, the only way for plague to enter was from the sea, most likely via one of her ports. A close analysis of vast manorial documentation allows a meticulous reconstruction of the regional spread of the plague in England (a topic to be addressed elsewhere), and it becomes apparent that London was the same entry port. 89 The three most likely candidates are: northern France, the Low Countries (a scenario along the line of Green’s vision) and Gascony. The first possibility should be discarded right away: Normandy and Brittany were spared any plague until 1362. The Low Countries remains a possibility, if we assume that it arrived from one of the Flemish or Zeeland ports or inlets — the Zwin or Terneuzen, for instance — given a high mortality in late 1360 and early 1361 recorded in nearby Ghent. 90 But Gascony remains an equal possibility, in light of the discussion above. 91 In any event, I do concede that I should have considered the Low Countries as another possibility alongside Gascony; but there is no evidence to reject the latter scenario.

89 The contours and impact of pestis secunda spread in England and elsewhere in the British Isles will be explored in a separate study I am currently working on.
91 The distance from Bordeaux to London could be easily covered in three weeks (conservatively assuming a slow speed of only one knot an hour), or somewhat faster. As Craig Lambert estimated, it would take seventeen days to travel from Portsmouth (a Hampshire port) to Bordeaux in the fourteenth century: Craig L. Lambert, Shipping the Medieval Military: English Maritime Logistics in the Fourteenth Century (Woodbridge, 2011), 197 n. 176.
Towards the end of her response, Green rejects my idea that at some point the south-central German reservoir may have died out and given way to other West Eurasian reservoirs — one of which, as I suggest, seems to be situated in the Ottoman Empire, sending plague waves in the early modern period (including the infamous plagues of 1663–6 ravaging northwestern Europe and the 1720–2 plague of Marseille).  

The routes by which the 1663–6 plague travelled as it ravaged the Netherlands, parts of Germany and the British Isles, cannot be securely established within the remits of the present article. Obviously, statements of some contemporaries — such as Samuel Pepys of London, whose knowledge was based on coffee house rumours, claiming that plague arrived from ‘Argier’ (Algiers) — can by no means be seen as secure testimony to the North African origin of that outbreak. But we do know that there was a plague outbreak in 1663 in both Algeria and Izmir; Dutch ships were arriving from North Africa and Turkey in that year; and the plague hit Amsterdam in late 1663, before
spreading to other parts of Holland (and north-western Europe).96 Was there a connection between three variables? Only a thorough analysis of all the surviving documents (including ship logs) can answer this question. As for now, this remains an open question.

The plague of Marseille (1720–2) is an altogether different story. Leaving Izmir in autumn 1719, the infamous Grand Saint-Antoine, passed through and called at Tyre, Sidon, Tripoli, Cyprus and Toulon, before arriving in Marseille on 25 May 1720.97 Quite remarkably, the news of the infected ship making its way from the east would spread across the west Mediterranean before its final arrival in Marseille.98 Although the plague is not attested in Izmir in 1719–20,99 it certainly is in the Lebanese ports and Cyprus in the same years.100 The infection may have been transmitted via one infected passenger boarding the ship in Tripoli on 3 April 1720 and dying two days later, or/and in contaminated cotton bales, laden in Tyre on 5 February of the same year.101 To the best of my knowledge, there was no region in France, or indeed anywhere in west Mediterranean Europe, affected by plague in the preceding months or years, which could thus have served as a potential source of infection importation into Marseille. All the facts above point to one of the Ottoman ports as the likely source of infection.102

98 Thus, the news was spread by Maltese Hospitallers anchoring in Alicante three weeks before the Grand Saint-Antoine’s arrival in Marseille (Giuseppe Restifo, I porti della peste (Messina, 2005), 3). Similarly, the port authorities of Livorno did not allow the ship to enter and anchor there, because of the onboard epidemic (Signoli and Tzortzis, ‘La Peste à Marseille et dans le sud-est de la France’, 5).
100 Daniel Panzac, La Peste dans l’Empire Ottoman, 1700–1850 (Leuven, 1985), 31 and 606.
101 Signoli and Tzortzis, ‘La Peste à Marseille et dans le sud-est de la France’, 5.
102 This interpretation is different from the recent suggestion by Nükhet Varlık that the Marseille outbreak may have come from a reservoir ‘in or around Marseille itself, rather than from the eastern Mediterranean’, conceding, however, that ‘the latter possibility cannot be definitively ruled out, given the lacunae of ancient DNA (aDNA) evidence from that region’. Nükhet Varlık, ‘Rethinking the History of Plague in the Time of COVID-19’, Centaurus, lxii (2020), 288. As I argue in the following paragraphs, the available aDNA evidence (as of late 2021) indeed points in the direction of the Ottoman Empire.
The case for the Ottoman origin of both the 1663–6 and 1720–2 outbreaks is strengthened further by palaeogenetic evidence. In the course of the evolutionary history of Branch 1A — the same branch detected in all post-*pestis secunda* sequenced genomes in Europe — there were at least two further splits (see Figure 3). The first split occurring towards the end of the first half of the fifteenth century gave birth to a new lineage, here tentatively called Branch 1A0, documented, as of May 2022, in only two sixteenth-century isolates, one from Cambridge and the other one from Riga.\(^{103}\) The other split, occurring around the mid/early second half of the fifteenth century, was another multifurcation event (or, the ‘Little Polytomy’), whereby the main Branch 1A got divided into two internal sub-branches (tentatively called Branch 1A1 and Branch 1A2) and (presently known) three short terminal sub-branches, each represented by one genome (Figure 3).\(^ {104}\) As of May 2022, Branch 1A1 is represented by thirteen genomes from six seventeenth- and eighteenth-century contexts — London Bedlam (c.1560–1635), Azov in south Russia (very generally dated to the fifteenth to seventeenth centuries, but its recently recalibrated phylogenetic position hints at the early seventeenth century),\(^ {105}\) Pestbacken in

\(^{103}\) The Cambridge genome is published in Spyrou *et al.* ‘Phylogeography of the Second Plague Pandemic Revealed through Analysis of Historical *Yersinia pestis* Genomes’ (see fig. 2); the Riga genome is published in Susat *et al.*, ‘*Yersinia pestis* Strains from Latvia Show Depletion of the *pla* Virulence Gene at the End of the Second Plague Pandemic’ (see fig. 2). The nomenclature of 1A0 is mine. The approximate dating of its divergence is based upon the fact that it shortly preceded the ‘Little Polytomy’ (see the next footnote). Discussing possible phylogenetic and environmental contexts of the 1A0 split lies outside the scope of the present study.

\(^{104}\) The approximate dating of the ‘Little Polytomy’ is based on Giffin *et al.*’s radiocarbon dating of two Vilnius genomes (AGU007 and 010) to 1453–85 (median date = 1469), Sigma-1 range (Giffin *et al.*, ‘Treponemal Genome from an Historic Plague Victim Supports a Recent Emergence of Yaws’, table 3 and fig. 5) and the fact that both genomes are positioned immediately after (one SNP ahead of) the Little Polytomy (*ibid.*, fig. 4). The estimated dating is further corroborated by the fact that there are 29 SNPs separating European Black Death genomes (1348–50) and the Little Polytomy. Assuming, with all the caveats listed above, the approximation of one SNP every 4.2 years, we arrive at the estimate that the Little Polytomy may have occurred some 120 years after the Black Death, that is, in the 1460s–70s. The nomenclature of the ‘Little Polytomy’ and branches 1A1 and 1A2 has been coined and agreed upon by Marcel Keller and myself, in conjunction with our ongoing collaboration.

\(^{105}\) Morozova *et al.*, ‘New Ancient Eastern European *Yersinia pestis* Genomes’; in the recent recalibration in Spyrou *et al.*, ‘Source of the Black Death in
south Sweden (1710–11), Marseille (the 1720–2 outbreak discussed above), Maist on the Chechen–Georgian border in the Caucasus (c.1613–1836) and Rostov-on-Don in south Russia (1771) (see Figure 3).

Despite such a wide geographic dispersal, one seemingly common element can be established for most (if not all) of these genomes: an association, direct or indirect, with some Ottoman context or connection. The case of Marseille has been discussed above. The south Swedish genome comes from a Great Northern War context, where warfare and plague were acting in tandem. The geographic origins and spread routes of this plague wave are yet to be studied in meticulous detail, but all the available information points, again, into Ottoman territories. Indeed, we know that the plague broke out in Constantinople in 1698–1700, spread into Edirne (Adrianople), then into western Bulgaria, as well as eastern and central Serbia in autumn 1700. Concurrently, the plague is reported in Transylvania in 1698, from where it spread into south Hungary in 1700.106 ‘Traditionally’, the story of the Great Northern War plague begins with a putative outbreak in a Swedish lazaret at Pińczów (south-eastern Poland), in the aftermath of the Battle of Kliszów (19 July 1702).107 However, the outbreak in question may not, in fact, have been plague at all, but rather a short-lived and isolated episode of some other disease.108 We do know,
However, that the plague reached the south-eastern limits of Lesser Poland and Lviv in late 1704 (most likely, by way of the Carpathians). From there, it spread all over southern

(n. 108 cont.)


Ukraine, into Poland, the Baltic region, northern Germany and southern Scandinavia. Arguing for an alternative importation scenario is hardly feasible: there were no outbreaks anywhere in north-western Europe since the 1660s and in Central and south-western Europe since the early 1680s (and the last outbreak of 1677–82 in Central Europe and south Spain seems, again, to be imported from the Ottoman Empire). Conversely, the territories of North Africa, the Middle East, Anatolia and the southern Balkans were continuously ravaged by plague outbreaks in the 1680s, 1690s, 1700s and 1710s.

Both Azov — at that point Ottoman Azak (formerly Venetian Tana), briefly seized and controlled by Don Cossacks in 1637–42 and then conquered and annexed by Peter the Great in 1696 — and Rostov-on-Don (founded as a fortress by Empress Elizabeth of Russia in 1749) were situated on the Russian–Ottoman frontier around the Sea of Azov, overlapping, roughly, with the Rostov–Krasnodar–Stavropol regions axis of present-day Russia. During an outbreak associated with the Azov genome, the city was, most likely, under Ottoman rule (unless it happened during the five-year Cossack control). The Rostov-on-Don specimen comes from the context of the Russo-Turkish War of 1768–74, in which the Rostov fortress played an instrumental role. Just as in the case of the Great Northern War, the Russo-Turkish War went hand in hand with plague spread. The plague was ravaging Anatolia and the Balkans continuously in the 1760s, and it entered the Russian and

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Ukrainian territories via Wallachia and Moldova in the west and Crimea in the east, arriving on the Black Sea and Azov Sea littorals in autumn 1771.115 By contrast, there were no plague outbreaks anywhere in Central or Western Europe in the preceding 30 years — since the 1738–40 wave in Central Europe and the 1742–4 outbreak in Messina.116

In light of all that, all the current knowledge points to the Ottoman origin not only of the Marseille plague, but also of some other late plague outbreaks in Europe in the seventeenth and eighteenth centuries — or to be more precise, those associated with the 1A1 sub-branch. This does not, of course, preclude the existence of a ‘native’ European reservoir in the early modern era. As we have seen, there was a multifurcation event in the late fifteenth or early sixteenth century, whereby Branch 1A split into 1A1 and 1A2 sub-branches as well as (presently known) an additional three short terminal branches, each represented by a single genome. While 1A1 genomes may have been associated with a putative ‘Ottoman’ reservoir, 1A2 genomes point in a different direction. So far, all sequenced 1A2 genomes (eighteen in total, as of May 2022) come from five Central European contexts, three of which are associated with the Thirty Years’ War (see Figure 3).117 Could it be that 1A2 sub-branch seeded its reservoir(s) somewhere in Central Europe — for instance, in the Alps, whose early-modern focus had been already hypothesized by Ann Carmichael?118 New palaeogenetic evidence, coupled with a meticulous reconstruction of early-modern plague waves in Central Europe will undoubtedly answer this question.

In her response to my original article, Green characterized my suggestions regarding an early modern Ottoman plague reservoir as ‘epidemiological orientalism’ and ‘nothing more than the impressionistic thinking that has dominated historical epidemiology in Europe since the sixteenth century’.  But does the textual and palaeogenetic evidence set out in detail above, justify such objectionable labelling? It is important — as Professor Green herself has demonstrated — that plague historians understand the technical details and ongoing scientific discoveries that aDNA sequencing and genomic analysis can provide. In this response, I have pointed to new discoveries in this field that further the current debate, and have sought to point out some continuing pitfalls that the plague historian must try to avoid, as we draw upon the flow of scientific publication. As we continue, collectively, to engage in these areas, I strongly believe that any scientific debate, aimed at advancing any field, should be based not only on firmly grounded evidence, but also on mutual respect.

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119 Green (p. 38). There, Green also states that I cite ‘a study from 2001 (well before the techniques of palaeogenetics had earned consensus among biologists)’. This is untrue: I cite Stephen Porter’s 2009 Great Plague. The term ‘epidemiological orientalism’ had been coined by Nükhet Varlık in her ‘“Oriental Plague” or Epidemiological Orientalism?: Revisiting the Plague Episteme of the Early Modern Mediterranean’, in Varlık (ed.), Plague and Contagion in the Islamic Mediterranean (Kalamazoo, 2017), 57–87.