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EPILOGUE: A HYPOTHESIS ON THE EAST ASIAN BEGINNINGS OF THE *YERSINIA PESTIS* POLYTOMY

ROBERT HYMES

FOR THE HISTORIAN of premodern Eurasia, the 2013 study of historical variations in *Yersinia pestis* mutation rates by Yujun Cui and others signals a new departure in the cumulative study of the genetics of the bacillus over the preceding fifteen years. Whereas previous studies had been moving to define *Y. pestis*'s place of origin and dissemination only in a broad chronological framework of up to 20,000 years (Achtman et al. 2004; Morelli et al. 2010), Cui and colleagues' work makes a historical claim of much greater precision: that the evolution of *Y. pestis* since its divergence from its most recent common ancestor may have occurred within the past three to four thousand years, and more importantly that a polytomy (simultaneous or nearly simultaneous genetic divergence of multiple lineage branches) or "Big Bang" that yielded most of the current strains of *Y. pestis*, as well as the 1348 Black Death strain, took place between 1142 and 1339 (Cui et al. 2013: 580, table 1; their time interval has a confidence level of 95%). Their further finding, that the bacillus originated in or near the Qinghai-Tibet Plateau, offers historians both a period and place to look for human events that could produce a punctuated genetic divergence. I will offer as a hypothesis that the "Big Bang" can be placed in space and time in historical sources, too: that the polytomy first manifests itself historically in the long destruction, by the Mongols under Cinggis-Qan (Genghis Khan), of the Xia state of the Mi or "Tangut" people in the early 1200s, and continues with the movement of the Mongols into north China, south China, and much of Eurasia. The new genetic evidence, I argue, merits revisiting the documentary evidence of epidemics in thirteenth- and fourteenth-century Central Asia and China, epidemics that earlier authors had proposed as possible *Y. pestis* outbreaks. No piece of evidence I offer argues unambiguously for plague: in particular, the Chinese sources have little to say about clinical symptoms and nothing to say about rodents. The case I propose rests rather on how bits of evidence fit together and, as a starting point, on their fit with the Cui team's findings on the Big Bang's timing and the region of plague's origin; and I point forward to the need for new research on China and Inner Asia.

Table 7. States of “China,” 976–1644

Northwest (Gansu corridor)	North China	South China
Xia (Tangut) 1038–1227	Song (Chinese) 976–1127 except for sixteen prefectures in far north under Liao (Khitan)	
	Jin (Jurchen) 1126–1234	Song (Chinese) 1127–1279
	Yuan (Mongol) 1227/1234–1279	
Yuan (Mongol) 1279–1368		
Ming (Chinese) 1368–1644		

In 1205, the Mongols under Cinggis-Qan’s leadership began a series of incursions south into the state of Xia, founded almost two centuries before by the people whom some of their neighbors called (and historians usually call) Tangut.¹ The Mongols attacked Xia cities in separate campaigns in 1205, 1206, 1209, 1217, and 1224. In 1225 Cinggis planned a final conquest, and in 1226–27 a pincer attack by two large armies, from the west and from the north, attacked and captured almost every Xia city until the capital, Zhongxing Fu, fell after a six-month siege. (For these campaigns, see Wu 1982: 122–38.)

What may make this destruction of a now obscure state important for the history of plague is that the territory of Xia, lying in the corridor between highlands encompassed today by the Chinese province of Gansu, directly adjoined the Qinghai-Tibet Plateau pointed to by Cui and colleagues: Xia lay just north and northeast of the Qilian mountains that mark the Plateau’s northern edge.² The western pincer of the 1226 assault traced a west-east line very close to the Qilian, thus to the Plateau. Today, Gansu lies within the range of the Himalayan marmot (*Marmota himalayana*) that lives on the plateau just to the south and in which plague today is more or less stably resident, and is home to many other species of rodent that might carry or serve as intermediary transmitters of the dis-

1 They called themselves “Mi” or “Mi-nia.” The name for the state more commonly applied by modern historians, *Xi Xia* or its translation “Western Xia,” was an informal name used by Song scholars. “Great Xia” (*Da Xia*), or simply *Xia*, was its formal Chinese name. See Dunnell 1996: xiii–xiv.

2 The more recent article of Yan et al. (2014) points much more specifically to “a small region east of Qinghai Lake” as the possible origin point of *Y. pestis*. This area of Qinghai happens to be the closest part of the province to the territory of the Xia state in the early thirteenth century.

ease as well. A two-year campaign on separate routes by two large armies surely damaged the natural as well as human economy of the region, and long encampments using carried provisions and producing heaps of refuse will attract rodent scavengers. When armies moved on, such rodents and their fleas may have hitched rides in stores and feedbags. My hypothesis requires that Gansu, in the thirteenth century, lay within the bacillus's homeland posited by geneticists, and that of the rodents that carry it today (questions for future research); and my proposal is that the Mongols unwittingly carried rodent plague hosts and their fleas eastwards into Jin and Song China as passengers in their stores.³ The Chinese sources begin recording new deadly epidemics exactly in the years of the Mongols' spasmodic and then systematic Xia conquest.

William McNeill argued forty years ago that the Black Death that struck Central and Western Europe and the Mediterranean in the fourteenth century began in East Asia, riding with the Mongols (McNeill 1976: 132–75). Michael Dols showed that many Middle Eastern plague accounts at the time assumed an eastern origin (Dols 1977: 38–43; for another view see Norris 1977; and see Dols's reply in Dols 1978 and Norris's further rejoinder in Norris 1978; cf. also Sussmann 2011). In 1995, Cao Shuji, using Chinese primary sources directly, built a similar argument—though placing the plague's geographic origin in Mongolia itself rather than positing the Mongol passage through Yunnan in the 1250s as crucial, as McNeill does in one version of his hypothesis. (Cao 1995; McNeill 1976: 143, but see also 145).⁴ I owe many primary source passages I cite here to Cao's work, which has been a crucial foundation for my own, though our arguments differ in important ways.

Despite McNeill and Cao, historians of China remain skeptical about an East Asian origin of plague or indeed about any plague in China as early as the Song, Jin, or Yuan dynasties.⁵ The path-breaking volume on the Song-Yuan-Ming "transition" was noncommittal on the question (Smith and von Glahn 2003: 8–9); Timothy Brook's recent general history of Yuan and Ming China is skeptical (2010: 64–66). In a more recent and thorough examination, Paul Buell (2012) has vigorously rejected the idea. Among

3 For known long-distance travel by plague-bearing rodents and/or fleas in bags of grain etc., see Benedictow 2010: 150–93.

4 On Cao's much more comprehensive 2006 treatment in collaboration with Li Yushang, see the afternote to this essay.

5 From outside the China field, see also the skeptical account in Sussman 2011, which however takes very little account of even the genetic work already known at that time.

his arguments are: that early Ming (1393) population figures that seem to show huge losses since the end of the Song dynasty (1279) cannot be taken “at face value”—true, on simple methodological grounds; that the records McNeill used as evidence for epidemics (and those used in Twitchett 1979, an inquiry into possible plague in China in the seventh through ninth centuries) are unevenly distributed across China, often vague, and faulty in other respects—true, and I attempt to improve to some degree on McNeill’s sources here; that Mongol movement (and movement of trade) from east to west in the fourteenth century was by no means as easy as some have imagined, thanks to the division of their empire into often competing domains; and that plague could not travel via the Indian ocean, as distance would burn out any shipborne outbreak. I return to each of these points (except the problem of McNeill’s sources) at least briefly below, but I plan to consider all of Buell’s arguments at length elsewhere, as there is not room to treat them fairly here. Taken together, they still seem to me to leave room for an argument for plague drawing on evidence that Buell did not consider or have available. In particular, he seems unaware of Cao Shuji’s work on Song-Yuan epidemics, though he cites Cao’s later article on plague in the Ming and Qing (Buell 2012: 139).

Buell’s assertion that “there was no demonstrable mass outbreak [of] plague in China” (2012: 129) bids to close the question prematurely: Is it “demonstrable” now? No—but this is what we want to find out. And till very recently, the same assertion would have held true for Europe, as important authors found it far from demonstrable that the Black Death itself was plague. That controversy is largely resolved after the multi-threaded genetics work of this millennium, culminating (for my purposes here) in the Cui team’s work.⁶ Monica Green (2014b, in this issue) suggests that the polytomy Cui et al. identify may have been “caused by the organism moving into new climatic environments and new hosts.” If we look for historical opportunities for such movement, the fact that these dates span the first explosive expansion of the Mongols is hard to miss.

This is the background against which to consider the materials on thirteenth-century epidemics in China that Cao Shuji compiled in 1995, along with two sources he does not cite (see points 2, 4, and 5 below). I treat these in chronological order. Note that items 1–3 emerge from exactly the period of the Mongols’ long conquest of Xia.

⁶ On the arguments against plague and the swing of the pendulum the other way in the last decade or so, see Little 2011 and Bolton 2013. The major challenges were mounted in Twigg 1984; Scott and Duncan 2001; and Cohn 2002. On the genetics work, see Green 2014b, in this issue; Bolton 2013; and Little 2011.

1. In 1211, the chief minister of the Jin state that governed the northern half of what is now China, then facing a Mongol incursion of its own, advised his ruler to submit to Cinggis to avoid costly direct conflict; and added, apparently as reassurance that Mongol dominance could not last: "I have heard that the men and horses of the Tartars [i.e., Mongols], not appropriate to the water and land [in which they find themselves], are experiencing pestilence" (*Yuanchao mishi* 1228/2005: 173; see also Cleaves 1982: 184; Cao 1995: 186). Note his clear opinion that the Mongols had not always suffered "pestilence" but had acquired it while in new country.
2. The physician Li Gao, in his account of the 1232 Kaifeng epidemic that appears in item 4 below, reported major epidemics in Dongping, Taiyuan, Fengxiang, and other cities of the Jin empire in the years between 1213 and 1222. Cao (1995: 187) notes the Kaifeng epidemic, citing Tuotuo et al. (1345b/2004, 1: 309a), but not the others, and does not cite Li Gao.
3. In 1226, the Yuan-period Chinese scholar Tao Zongyi (1316–?) tells us that the Khitan advisor Yelü Chucai (1190–1244) joined in the conquest of two cities in Chinggis Qan's Xia campaign. "The commanders competed to seize boys, girls, jades, and silks; [Yelü] alone took only several volumes of documents and two camel-loads of rhubarb. Later there was epidemic in the army, and only those who received rhubarb were curable: nearly ten thousand men were saved" ([1366]/1959: chap. 2; quoted in Cao 1995: 189, with no reference). Here, both the association of epidemic in the Mongol army with the invasion of Xia and the strange mention of rhubarb as a medicine to treat it are important. On rhubarb see item 8 below.
4. The Jin dynasty doctor Li Gao, founder of the "internal damage" school of Chinese medicine, claimed to have witnessed the Mongols' 1232 siege of the Jin's southern capital Daliang (modern Kaifeng, Henan):

At the time of the change of eras, in the *renchen* year [1232], the capital was at maximum readiness by the last ten days of the third month [April 24 to May 4].⁷ In all it had been half a month since the enemy's arrival, and after the siege was lifted,⁸ not one or two out of ten thousand of the

⁷ According to the official Jin history, the siege began on April 28 (Tuotuo et al. 1345b/2004, 1: 309a).

⁸ The siege was lifted on May 8 according to the official history (Tuotuo et al. 1345b/2004, 1: 310a).

people of the capital did not become sick, and the sick who died followed another without end. At each of the capital's twelve gates, [the dead] sent out each day were two thousand at most and no less than one thousand at fewest, and this was so for almost three months. Could all these million people have contracted wind-cold external damage? Needless to say, people in the besieged city had been harmed by irregular eating and drinking and by excess toil. From two or three months of hunger in the morning and gorging at night, rising and resting irregularly, and suffering cold and heat through displacement, their stomach *qi* had long been exhausted. Suddenly to eat their fill to great excess injures people, and if furthermore their [medical] treatment is inappropriate, they are certain to die. It was not only in Daliang that things were this way. Further back, during the Zhenyou and Xingding eras [1213–22], [cities] like Dongping, like Taiyuan, or like Fengxiang were all the same in the illness and death they suffered after their sieges were lifted.⁹

“Wind-cold external damage” here refers to the “cold damage” school of Chinese medical theory and therapeutic practice, which had gained dominance in the preceding two centuries precisely for its perceived value against epidemics; its therapeutic methods had naturally been tried against this one. Li Gao recounts several treatments he saw other doctors apply in accordance with the theory, noting that they elicited changes in presentation that mimicked real cold damage, though the patients all died. He thus writes an entire book to distinguish “external” cold damage from the “internal damage” he finds in the 1232 events—damage caused, as we see in his account, by poor or erratic nutrition and by “excess toil” in the siege. What is important for my argument is that he clearly perceives the phenomena he is confronting as new to medicine, requiring new methods. Further, he sees the same process at work in a number of cities of north China—he lists three, but signals with his recurrent “like . . .” that these are three of a larger number—starting about twenty years before. The three are major cities scattered widely across the north China plain:

⁹ *Neiwaishang bianhuo lun, bian yinzeng, yangzheng*, in Li Gao (1247/1993: 8–9). The Jin official history's account (Tuotuo et al. 1345b/2004, 1: 309a–311a) is independent of Li's, mentioning dates he does not and setting deaths at 900,000 instead of a million. Li Gao's account has finer-grained numbers: his “million” estimates what by his own details would lie somewhere between 600,000 and 1.2 million. I thank my student Stephen Boyanton for calling my attention to this passage. My translation has started from his but diverges on some points. I thank him also for several productive conversations on the question of plague and for other source help cited below. He is exploring the larger significance of Li Gao's shift from “cold damage” to “inner damage” in work of his own. See Boyanton 2014 and forthcoming.

even taken by themselves, let alone if typical of a larger number as he suggests, they and Kaifeng would represent mass death in north China spread across two decades. In Kaifeng itself, mortality seems to have been at least 40%, as its population when it finally surrendered to the Mongols the next year was about 1.4 million¹⁰ (Song et al. 1370/1976, 11: 3459]; Cao 1995: 187). Most important, Li Gao places the moment of outbreak in all four cities not during a process of siege but *at the lifting* of a siege (each a step in the twenty-year Mongol conquest of north China and the Jin state), thus at a point of contact by Mongol troops either with city populations or with Jin troops who then relieved the cities.¹¹ These two points—the perception of the epidemics as something new, and their association with contact with the Mongols—are critical, and will recur.

5. In 1235, the Mongol court debated the failure of already-conquered regions to submit fully to their rule. Some urged that Muslims from Central Asia (“the western regions”) be used to subdue China, and northern Chinese to subdue Central Asia. Yelü Chucai opposed this, arguing that “the water and land are of different appropriateness (*yiyi*); epidemics will arise” (Song et al. 1370/1976, 11: 3460; not cited in Cao 1995). Again, epidemic is blamed (now from experience, perhaps) on movement of peoples into lands not “appropriate” to them.
6. In 1241, when the Mongols attacked Shouchun, in Song territory just south of the Huai River, they faced long summer rain. Li Zhen, a scion of the Western Xia royal house who had gone over to Yuan, told his commander: “If we camp the army below the walls, in the summer rain an epidemic will arise, and we will fail . . . I ask that you retreat

10 I say “at least forty per cent” because of probable increase over time in the denominator. The population of Kaifeng in 1232, at the time when the Mongols began their siege, was presumably greatly swelled by refugees from the surrounding countryside, probably to a considerable distance. A million people out of the resulting population, Li tells us, died in the epidemic. But in the year between this epidemic and the Mongols’ final taking of the city, its population would presumably have been increased again by new refugees fleeing the Mongols’ second arrival. Thus one cannot treat the 1.4 million remaining in the city at its final fall as the number left over after the epidemic a year before; that number may have been considerably smaller.

11 In Kaifeng, the official Jin history makes clear that the siege was lifted (temporarily) after the Jin sent a delegation out of the city to negotiate with the Mongols (Tuotuo et al. 1345b/2004, 1: 310a). For the other three cities, the siege appears to have been lifted after battle with the Mongols by Jin troops sent to relieve them, who would then have entered the cities themselves (Tuotuo et al. 1345b/2004, 1: 253, 258, 263, 265, 270, 272, 278, 284–85, 292, 306–07). Direct contact with Mongol troops outside the city was thus common to all four cases.

several *li* [a few kilometers], and I will go myself to invite them [to yield].” The plan worked, but of interest here is the connection that Li, who had served in the Kaifeng attack in 1232, saw between sieges and epidemics (Song et al. 1370/1976, 10: 3051; Cao 1995: 187, cites Li Zhen for his death in the 1259 campaign below, but does not cite Li’s testimony on sieges).

7. Several potential references to plague center on the Mongol invasion of Sichuan in 1258–59. The official Yuan history records that, in 1258, the general Shi Yuanze “followed Xianzong [Möngke] to attack the Song, entering via western Shu [Sichuan]. In the summer of *jiwei* [1259] he camped at Mt. Diaoyu in Hezhou; there was a great epidemic in his army” (Song et al. 1370/1976, 12: 3660; Cao 1995: 187). The same year, Xianzong ordered another officer “to prepare medicated leaven [yeastcake] to treat the epidemic in the army” (Song et al. 1370/1976, 11: 3279; Cao 1995: 187). Elsewhere we read that, at this time, a Song general “was newly occupying [the Sichuanese city of] Chengdu, and his troops and horses could not tolerate the water and land and grew sick and died in great numbers” (Song et al. 1370/1976, 10: 3145; Cao 1995: 187).¹² Here it is Song troops who suffer from being in places unfit for them; but this makes sense in the text’s terms, as Sichuan for most Song people was far from home.¹³ Song accounts record the sudden death of the Song general

12 It might be asked whether this particular outbreak should be disqualified as plague because it affected horses. That would be premature, I think, though the question is worth asking. Horses do not commonly contract plague in the usual way, because fleas do not infest them. But Alexandre Yersin, after whom *Y. pestis* is named, did succeed in infecting horses (the usual host for serum manufacture at the time) in order to produce a therapeutic serum against plague. Yersin “was able to obtain a certain degree of immunity in a horse by injecting small quantities of the plague bacillus subcutaneously, and afterwards, *as this produced suppuration*, by making intravenous injections of the plague bacillus, these injections being repeated from time to time *as the temperature went down, the glandular enlargements and joint tenderness disappeared*, and the animal came to its normal condition” (emphasis mine). That is, Yersin produced *suppuration*, *fever*, and *glandular enlargements* in a horse even with carefully graduated small quantities of the bacillus: see “Plague” in *The British Medical Journal* (6 February 1897: 357). This was by direct injection, of course, and it does not necessarily follow that horses can contract plague in other ways; but the possibility that they could become sick by ingestion of the bacillus in contaminated food at least deserves further investigation.

13 The text has earlier referred to large-scale *nueli* among Niulin’s own troops, a term that in modern discourse means “malaria,” and in the testimony of the Persian historian Rashid-al-din the epidemics of this campaign are called variously

Wang Deng: “He had agreed [with the Mongols] on a day for battle, and the night before was arranging army affairs when he suddenly collapsed, emitting blood from all five viscera.”¹⁴ Within moments he died (Tuotuo et al. 1345a/1977, 35: 12385; Cao 1995: 188 notes that Wang Deng’s troops had already been suffering epidemic disease, but offers no citation).

8. In 1273, as the Song state fell, a certain Liu Fu joined the loyalist minister Chen Yizhong for a last-ditch campaign against the Yuan, but died on reaching Luofu in Guangdong.

Before this, Chen dreamed someone told him: ‘This year a Heaven-sent disaster will spread, and nearly half the people will die; those who take rhubarb will live’. Afterward a great epidemic arose, and those who took [rhubarb] indeed did not die. When Liu was sick, [Chen] had him take it, but he could not be saved. (Tuotuo et al. 1345a/1977, 35: 12249; Cao 1995: 188)

This is the second mention of rhubarb as a treatment for these epidemics we have seen. It may be that this was simply a regional folk remedy of which Yelü Chucai gained knowledge in the conquest of Xia and which he brought with him from Gansu, and that knowledge of it had spread to the Song side by the 1270s. Rhubarb is a commonplace item in Chinese traditional pharmacology, but almost always as one element in a compound: to use rhubarb alone is quite unusual. In the great imperially sponsored Song compendium of prescriptions, the *Prescriptions by Imperial Grace of the Taiping Era* of 992, I have found just seven listings for rhubarb alone out of the many thousands of prescriptions the work collects. Only one of these appears in a section directly concerned with epidemic diseases, in this instance called literally “seasonal ethers” (*shi qi*). There rhubarb is the sole component of a prescription specifically for “pea sores in epidemics.”¹⁵

“dysentery” and “cholera” (Boyle 1971: 228, 248). Cao Shuji quotes the latter passage in Chinese, and his term is *huolan*, the word for cholera in modern Chinese; but as he points out, none of these terms can necessarily be taken as equivalent to any modern biomedical disease entity, and the diarrhea suggested by Rashid al-din’s “dysentery” and “cholera” as well as the high fever suggested by the Yuan history’s *nueli* are consistent with plague (Cao 1995: 187).

14 “Blood from the five viscera” in this context probably means that Wang Deng bled from all bodily orifices.

15 Wang 992/1958: 428; the description of “pea sores” appears on p. 427; another description in connection with “cold damage” disorders (also epidemics) appears on p. 285. Interestingly, three other prescriptions in the compendium that specify rhubarb

“Pea sores” (*wandou chuang*) are described as raised sores, white or red, that are shaped like a pea (or “emerge like a pea”) and that are probably meant to be understood as pea-sized. Given their size, I think there is virtually no chance that the “pea sores” described here are buboes, and I would certainly not argue for plague as something widely enough known in China in 992 to make its way into an imperial compendium. If, however, one imagines that an official faced with an epidemic in 1226 or 1273 might consult this rather old but still quite authoritative volume, then the only entry for epidemic he would have found that specified rhubarb alone as a treatment was *one intended specifically for an epidemic involving raised sores*. To put it differently, if an official consulted this compendium about an epidemic and came away with rhubarb as a sole treatment, he must have been looking for help with a disease that involved raised sores. He would have been generalizing somewhat from a prescription originally meant for an epidemic involving a different *kind* of raised sores; but surely this would be a rational procedure in the face of a new disease. Again, we cannot know that the rhubarb treatment came from consulting this work, as opposed to from local knowledge in 1226 Gansu and/or 1273 south China, or from sheer improvisation; but it is possible that the mentions of rhubarb in items 3 and 8 may carry, for us, a tantalizing though indirect hint that the epidemics of this period involved raised sores.

Cao Shuji judges the episodes he cites to be plague based on several factors: their mortality levels, clearest for 1232 Kaifeng (and the others that Li Gao likens to it, which Cao does not cite); their timing, as he goes on to trace epidemic records in the Yuan official history through the fourteenth century, a series that he appears to see as leading up to the spread of plague to Europe; and their association, in two sources, with rhubarb—to which I return below. My own reason for urging that we take these scattered records as possible evidence of plague is also timing, but from a different angle: that the encounters with epidemic by Yuan armies, and then by those they invade, begin just when Mongol troops pass in force, and with great destruction, through a region that abuts the Qinghai-Tibet Plateau and where plague is now endemic in marmots and other mamma-

as the sole treatment also involve raised sores: these are a prescription against “fluid-filled vesicles” on the face (p. 1213); one against “vermillion toxin sores,” i.e., large raised swellings on the skin (p. 2990); and one against “pox-like rash” in children: the section that includes this one mentions “cold damage” in its introductory matter, so we must take it as intended for a context of epidemic as well, though specific to children. The remaining three rhubarb-only prescriptions, none for epidemic contexts, are for two kinds of jaundice and for delirious speech in fever (pp. 480, 1685, and 1686).

lian hosts. But one must also note (with Cao) the extraordinary mortality reported by the Jin history for Kaifeng, and by Li Gao for Kaifeng and three other cities: 40% or more is a level attested in Eurasia, to my knowledge, for no pre-nineteenth-century infectious disease but plague.

The history of medicine, some time ago, moved away from “retrospective diagnosis” toward attending to the constructions that historical actors themselves placed on the phenomena of illness or health they experienced and recognized. This approach has illuminated much, though as a sole lens it may also obscure. The present volume, of course, is precisely focused on redefining “retrospective diagnosis” by incorporating the diagnostic capabilities of a new type of microbiological analysis. Yet one would still hope to know just what construction(s) contemporary actors placed on the thirteenth-century epidemics in China. Since all our sources (except perhaps Li Gao’s eyewitness account) lie at a considerable temporal distance from events,¹⁶ and (as works of official history) are the products of the state and of literati elites, it is very unlikely that they even come close to exhausting the constructions that contemporaries placed on these illnesses; we can only ask what *our sources* “construct.” But that is still worth asking. First, they see simply *epidemic*: *yi* or *wen*. For Chinese medicine at the time it was arguably “epidemicity” itself, and not specific symptoms, that most dictated classification: “cold damage” theory had arisen to treat epidemics as a single category. More importantly, our sources—brief mentions in chronicle-style accounts or in the biographies of officials or military officers taking part in campaigns or in debates over policy *versus* the enemy—are the kinds least likely to be specific about symptoms. Second, very importantly, they see something *new*: for Li Gao this required a remaking of the medicine of his time because it was an epidemic that *resisted usual epidemic treatment*; for others, it needed explanation, significantly, in the movement of people into spaces not customary for them. Third, they see something connected to *the lifting of siege*, thus to contact with the besieging Mongols. And fourth, in two cases (nos. 3 and 8), they see something treatable with rhubarb, which as I have suggested above may convey indirectly our only hint about symptoms.

16 One diary of the Kaifeng siege has survived, as far as I know, written by Liu Qi (1203–50) and included in his miscellany *Guqian zhi*. Surprisingly, his more or less month-by-month and sometimes day-by-day record jumps from the fourth month of 1232 straight to the seventh month, simply skipping the entire period that our other sources tell us encompassed the epidemic. It seems to me very likely that he had left the city during that period. I am grateful to Stephen West for letting me know of Liu Qi’s diary.

I would argue that, rhubarb aside, these are just the terms in which a Chinese-literate audience might well construct bubonic plague that they recognized as coincident with Mongol armies moving through their states and sacking their cities. They had no exact terms for it because, as they saw, it was new, and older exact terms were (according to Li Gao) failing. Most striking were its mortality (which should strike us too) and its speed, but perhaps these simply made it a particularly dangerous *yi*. Finally, as Cao suggests, the epidemics presented themselves to all involved as concomitants of war and dynastic change, thus as just one of the forms of death and destruction prominent across China (Cao 1995: 190). If Chinese records do not proclaim a “Black Death” as European records do, this does not tell us that demographically damaging plague was absent: the Chinese could construct such damage in other terms.

But is there, in any case, evidence of the demographic damage one would expect from plague? Were there large population losses in the Song-Yuan or Yuan-Ming transitions? Buell says no, but other serious scholars have thought so. In their book on Chinese population history, not (in Buell’s words) “taking at face value” official Ming totals but critically mining local sources as well, Zhao Wenlin and Xie Shujun place the combined population of Song and Jin (ruling south and north respectively) in 1210 at just over 108 million; the total population under Yuan (ruling both north and south) at a bit more than 75 million in 1292 and about 87 million in 1351; and the total under Ming (also ruling both north and south) in 1381 at about 67 million (Zhao and Xie 1984: 310, 333, and 364). Significantly, the population losses have different distributions: massive loss in the north from Song to Yuan, large loss in the south from Yuan to Ming. It is interesting to compare this distribution to Cao Shuji’s material. After his discursive treatment of thirteenth-century outbreaks, Cao simply lists epidemics from the Yuan official history for 1297 through 1362 (Cao 1995: 190–01). These impressively number thirty-one for the sixty-five years and cover many prefectures and all regions, but offer little detail,¹⁷ most simply noting “great epidemic” for each place, with few numbers or percentages of dead. If we add Cao’s earlier cases to his post-1297 list, six cases of recorded epidemic emerge between 1211 and 1292 in the north, only two in the south (the 1259 Sichuan campaign and the 1273 Guangdong case), matching the higher northern population loss that Chao and Hsieh find across just those decades. At the other end the match is less

17 Cao does not provide citations for this list, but the relevant passages I have been able to trace are in Song (1370/1976, 2: 413, 416, and 559; 3: 603, 620, 625, 740, 764, 784, 800, 820, 912, 914, 918, 944; and 4: 1080 and 1111).

clear: between 1351 and 1381 (without Ming data for 1369 on), we find eight epidemics recorded in the north to five in the south. The difficulty here is that by the 1350s China was in chaos, with civil war in the south, so that the Yuan court in the far north was in a poor position to record southern epidemics. Nor should we expect plague, if it was there, to do all the demographic work in a region constantly at war. We may never sort out the relative weight of war and disease here; yet no evidence we have excludes a role for plague in southern population loss.

Gaps in this picture cry out for research. As epidemic records after the Yuan conquest are (even) less circumstantial than those of the Sung-Yuan transition, Cao Shuji (1995) simply assumes that many of the epidemics in his post-1297 list are plague because he believes he has shown that earlier ones are. This is hardly sufficient and badly needs to be reinforced by further evidence. Some obstacles that seemed serious to earlier plague researchers, however, no longer seem so. No particular speed of local spread is too fast, any longer, to represent plague, as the potentially quite slow “blocked flea” mechanism of older theories is, we now know, not the only one available mode: newer research on early-phase transmission by “unblocked” ground-squirrel fleas, prairie-dog fleas, and even rat fleas has settled that problem and made rapid person-to-person spread via fleas plausible.¹⁸ Thus, for plague to ravage a city the size of Kaifeng (swollen, as we have seen, to something perhaps near 2.4 million by refugees from the countryside) and to kill nearly a million within fifty days presents no serious epidemiological problem as far as speed is concerned. All this also means that we do not need to find reports of mass rat death in our sources to find plague plausible: even if the sources were likely to attend to such things, neither rat epizootics nor rats themselves are necessary to spread plague.¹⁹ The apparent patchy character of plague in China (if many of these epidemics are plague)—now here, then silent for a while, then over there—need not be a problem either, as we now know that plague can be carried great distances even without rodents sick enough to die quickly: in food or feed contaminated by rodent or flea feces, or by fleas themselves

¹⁸ Eisen et al. 2006; Eisen et al. 2007; Wilder et al. 2008; for a general review of this point, see Gage 2012: 86–91. Note that the ground-squirrel and prairie-dog fleas investigated are of the same genus, *Oropsylla*, as the marmot flea.

¹⁹ Katherine Royer shows brilliantly the process by which an awareness of a variety of mechanisms for plague transmission among early twentieth-century scientific plague researchers was transmuted into a collective and very long-lasting conviction that rats, the rat flea, and mass rat death were essential features of plague (Royer 2014).

(cf. especially Benedictow 2010: 150–93). I argue that it may also travel in (transported) rodents from populations with a history of coping with the disease: Gansu rodents—like those that I am suggesting hitched rides with the Mongols—having lived for a very long time in a region where the plague bacillus had long been present too, may offer this possibility.²⁰ Finally, where other populations of potential host rodents are available, the bacillus may settle down in a new (or old) reservoir for a considerable time without new epizootic or epidemic activity. Many such reservoirs, or more accurately regional assemblages of reservoirs, are known in the world today.

This is a central point. We know that the reservoirs of plague-bearing rodents in contemporary North America, for instance, are the result of movements of people, rodents, and fleas from Asia in the late nineteenth and early twentieth centuries during the Indian and Chinese plague outbreaks that stimulated the first scientific work on plague and *Y. pestis*. It is reasonable to assume (in fact it is the direct implication of Cui and colleagues' work), and we should investigate the likelihood, that some of the other reservoirs known to exist in China and Eurasia were created by similar processes of movement in much earlier periods.²¹ Ann Carmichael (2014, in this issue) proposes that the formation of such reservoirs in the Alpine marmot population can account for the periodic recurrence of plague in Europe after 1348 without necessarily new imports of rats,

20 Wang et al. (2009) have shown the reservoir status of Himalayan marmots in Sichuan. Zhang et al. (2012) demonstrate actual plague tolerance and thus reservoir capacity in Central Asian great gerbils. Eisen and Gage (2009: 2) are skeptical of the idea of “truly independent epizootic and enzootic cycles” that they note “has dominated the literature.” Yet there seems no doubt that plague is resident long-term in certain rodent reservoirs, Himalayan marmots (as in the Wang et al. 2009 above) and California ground squirrels among them, and Eisen and colleagues' explanation by difference of “rates of transmission and numbers of hosts infected,” though replacing a dichotomy with a possible continuum, may offer an explanation for the phenomenon more than deny it. The question would then be what factors can affect these transmission rates and numbers of infected.

21 Cao Shuji (1995: 186) appears to assume that the Mongolian reservoir pre-dates the Mongol conquests, and McNeill (1976: 145) appears to make the same assumption for both Mongolia and Yunnan. Yet Julia Riehm and her team (2012) argue from genetic data that the Second Pandemic strain did not come to Mongolia before about six hundred years ago, and my hypothesis here implies that the Mongols brought plague from Gansu both to Yunnan in their incursion in the 1250s and back to Mongolia at some later date. Cf. also Varlık (2014, in this issue), dealing with a late Ottoman context: “It may be erroneous to assume that the active foci of the eighteenth and nineteenth centuries also existed before that time.”

fleas, or bacilli from port cities or by major trade routes. Part of the burden of my hypothesis is that at least some of the existing rodent reservoirs in China and the rest of Eurasia were established by the movements of Mongol armies and are thus historical, human (not merely biological or zoological) artifacts with specific moments of origin that new research, both genetic and historical, may establish. Significantly, the work of Julia Riehm and colleagues shows that Mongolia itself seems to have become a reservoir for post-polytomy strains only after the Mongol conquests in the south.²² Are local rodent populations too, or their attendant flea populations, historical human imports in some cases? On the human side we need archeological work combined with genetic ancient-DNA analysis to determine whether *Y. pestis* can be found in thirteenth- and fourteenth-century Chinese and Central Asian bones or teeth as it has been found in Black Death victims in Europe. The hypothesis I offer here may not be finally confirmable except by such evidence.

I offer no specific solution for how the plague gets from East (or Inner or Central) Asia to the specific Mongols at Caffa in 1346 (if that story stands), but if the Mongol expansions of the previous century or more did indeed establish new rodent reservoirs for plague in other parts of Eurasia, there may be no reason to assume an immediate Chinese origin for the events of 1347 and after in the Mediterranean basin and Western Europe; and in this respect Buell could be right. Alternatively, and I think more probably, his point about the Mongol empire's division could explain plague's presence in China and Central Asia for more than a century (1211–1340s?) before it goes west to the Golden Horde, to produce the Caffa incident. But we should also consider the possibilities of a second route. In the south there are major epidemics in 1331, 1333, 1344, and 1345, suggesting continuous presence by that time; and merchant ships sailing to the Indian Ocean, which since mid-Song had carried an extensive Chinese trade with both south Asia and the Middle East, departed from the south (Sen 2006; Chaffee 2006; Clark 1991; So 2000). Especially interesting in this connection are the epidemics recorded for 1333 and 1344. In 1333, the prefectures affected included Songjiang, Jiaying, and Hangzhou. The three lie in a northeast-to-southwest arc along the southeast China coast in what is now Zhejiang; more importantly, the major Shanghai and Ganpu ports where the emperor Khubilai had established official Offices of Maritime Affairs to tax but also encourage trade with the south seas lay within Songjiang and Hangzhou respectively: here, then, were major epidemics in 1333

22 Riehm et al. 2012; see n. 21 above.

at or near two of the four officially sponsored ports through which most Yuan trade with the Indian Ocean passed. The most important of those four ports, however, was Quanzhou in Fujian.²³ The epidemics in 1344 were concentrated in Fujian and affected Fuzhou, Yanping, Shaowu, and Tingzhou prefectures. Quanzhou is not specifically listed, but both Fuzhou and Yanping bordered it directly, while Shaowu and Tingzhou were mountain prefectures joined by overland trade and downriver traffic to the prefecture in which Quanzhou port lay. Goods infested with rodents, fleas, or their feces could have found their way from any of these epidemic prefectures to the Quanzhou port and onto South Sea trading ships. The considerations on distant transmission that I have already touched on seem to me to leave it possible that plague moved from China to, for example, the Persian Gulf by sea and thus contributed, if not to the European Black Death (it may be difficult to posit plague's passage north from the Gulf to the Golden Horde's territories), perhaps to its Middle Eastern equivalent.²⁴ There is also the problem of siege conditions, and why plague would seem so strongly associated with them in China. This may go back to the identity of the rodent: if (for instance) marmots, or other rodents from the plague foci in Gansu and the Qinghai-Tibet Plateau, are unlike rats in being able to live for extended times as stable carrier-populations for the bacillus, then perhaps it is siege that turns stable carriers into unstable ones. If so—and this is speculation apart from my main hypothesis—perhaps nutrition is key. Perhaps in sieges the rodents brought from far away, like humans, begin to starve, or switch to new foods, and this changes their relation to the bacilli that some of them carry. We still know too little about how enzootic turns to epidemic; and a key element that my hypothesis glosses over for now is the identity of the plague-carrying rodents I am proposing came with the Mongols from the territory of Xia. Marmots seem to be the main, though not necessarily the only, carriers of plague in that region today; but does *Marmota himalayana* descend low enough from the

23 For Khubilai's decree establishing Quanzhou, Shanghai, Ganpu, and Qingjiang as sites of Offices of Maritime Affairs, see Song et al. 1370/1976, 94: 2401.

24 On the practicality of sea-borne transmission, cf. Benedictow (2010: 185–87); on the possibility of Indian Ocean transport of plague, though not necessarily from China, see Green (2014b, in this issue). See also her introduction, in which she suggests that the genetic evidence may support the notion of two sources for plague in Anatolia and North Africa (Green 2014a, in this issue). This possibility is strengthened and extended to Europe by the work of Stephanie Haensch's team (Haensch et al. 2010), showing that distinct strains seeming to belong to separate points on the polytomy later identified by Cui's team are both present in European Black Death victims. This may support the possibility of separate transmissions both overland and by sea.

mountain slopes of the region to have had significant enough encounters with the Mongols and their encampments? Are marmots capable of entering something like a commensal relation with human beings, at least for long enough to be transported into other regions of China in their store-wagons and feedbags?²⁵ If not, could other rodents in the Gansu corridor have played this role? Studies of the distribution of plague among rodents in the larger region have concentrated more on Qinghai than on Gansu,²⁶ so new work may be needed there. And ultimately we need to know about Gansu rodents in the thirteenth century more than now, which argues a need for bio-archeological work in the old territory of Xia.

As a final point for further research there is the question of rhubarb. We have seen it used twice against the epidemics I have traced here, and I have traced how those mentions might constitute a hint as to symptoms. But the story of rhubarb and plague in China may not end there. Cao Shuji cites the traditional Chinese physician Li Jianshi who, in 1935, claimed success in treating bubonic plague with very high rhubarb doses (Cao 1995: 189; I have not so far been able to obtain Li's book). Biomedically trained physicians in China are currently undertaking research on *why* rhubarb is effective against *Y. pestis*, apparently having already concluded that it is (Bai et al. 2008). We need not take our Song-Yuan sources' word when they tell us that everybody who took rhubarb survived in order to believe that some ameliorative effect may have been at work. Where did Li Jianshi and current researches get the idea of trying rhubarb against plague in

25 Voluntary stowing-away need not be the only plausible means for marmots to move with the Mongol armies into China. The *Secret History of the Mongols*, Marco Polo, and contemporary Chinese observers all report that the Mongols, in their native territory, hunted marmots for food (Cleaves 1982: 29; Smith 1984: 223; Buell and Anderson 2000: 45). It is likely that they made use of the pelts as well. John Masson Smith (1984: 227) has considered the possibility that marmots were an important food for Mongol armies on the move, and concluded that they "must have been a supplementary rather than a basic component of Mongol rations." But a supplementary role would suffice for the purpose of my hypothesis if it should motivate Mongol troops in Xia territory to capture local marmots and transport them for future use, either as food or as furs. If Mongols were already eating or clothing themselves in marmot in Xia territory, a plague epidemic in their army might have begun there; but as this epidemic was likely also either to burn itself out or destroy the army outright on the spot, it is probably easier to account for the pattern of siege-related epidemics in China if we suppose that marmots carrying the plague bacillus, but not yet sick and transmissible, were transported alive to China and transmitted the disease to humans later under siege conditions.

26 See for instance Yang et al. 2014. I am grateful to Monica Green for making me aware of this article.

the first place? Did a belief in its effectiveness descend historically from Yelü Chucai and others in the thirteenth century? from the *Prescriptions by Imperial Grace* of 992? from local knowledge in Gansu? Does rhubarb actually inhibit *Y. pestis*, and if so, how?²⁷ There is historical, medical-historical, and perhaps even medical investigation to be undertaken here.

I am proposing, in sum, that we may need to place the “beginnings of the Black Death” more than a century earlier than we have been accustomed to place them. My suggestion that the expanding Mongols were the

27 Here I might risk an even more speculative expansion on my speculative suggestion above, about nutrition in rodents. Oddly enough, the Qinghai-Tibet-Gansu region that is the original home of plague is a natural botanical home of undomesticated rhubarb as well (Wang et al. 2010; Foust 1992). Marmots—again, the most frequent rodent carrier of plague in that region—are reported by contemporary travelers to eat large quantities of rhubarb leaves (Anon. 2013: 2). Awareness of a connection between marmots and rhubarb in this region is much older, however: the first European hunters of rhubarb for medical purposes noted how commonly marmot burrows were to be found surrounded and shaded by rhubarb plants, and argued that marmots were enabling the growth of rhubarb by aerating the soil with their burrowing activities: see Burke (1767: 84–85, quoting John Bell’s memoirs of his travels, in this instance with regard to Mongolian marmots and rhubarb). Rhubarb leaves are toxic to humans because of their high oxalic acid content; the content of the stems and roots, though lower, is significant as well. (Traditional Chinese medicine always used the root, dried; it should be noted however that drying vegetables typically doubles the proportion of oxalic acid; cf. Paul et al. 2012: 313, especially table I.) Oxalic acid compounds (oxalates) have been used by *Y. pestis* researchers to breed non-virulent strains *in vitro* (Higuchi and Smith 1961) or to detect virulent strains by their inhibited reproduction on oxalate-rich agar (Bhaduri, Chaney-Pope, and Smith 2011). This works, it seems, because oxalic acid binds calcium, which the bacillus needs for its virulence plasmid: batches raised in calcium-poor conditions develop either without the plasmid or with drastically reduced reproductive rates. Rhubarb is not the only plant food high in oxalic acid: two more are oats and buckwheat, which a horse-borne army might carry with it in grain bags, just the way rats and fleas hitched train rides during the Indian and Chinese plagues of the last century (Benedictow 2010: 160–61). Alongside rhubarb we have seen “medicinal leaven” or yeastcake as a treatment for the disease in our sources (no. 7, above); and after the 1232 siege and epidemic the Jin government, needing money to repair the city wall, put a special tax on tea-leaf sellers—along with coffin-makers, clergy, and physicians—because all these had “arrogated great profit to themselves since the epidemic began” (Tuotuo et al. 1345b/2004, 1: 311a). Coffin-makers for the dead, doctors for the sick, clergy for prayers and funerals, of course; but tea-leaf sellers? Tea-leaves, it turns out, are extremely high in oxalic acid, and so are many yeasts. Does *in vitro* modulation of *Yersinia pestis* virulence or reproduction via oxalates work to some extent *in vivo* too? Had Central Asian marmots benefited from this for millennia, and did some Mongols, Jurchen, and Chinese benefit from it, though of course theorizing its benefit in quite other ways, in a thirteenth century crisis?

agents of plague's spread is hardly new, but my hypothesis places the critical moment neither in the Mongols' emergence from their native steppe, in itself, nor in their passage through Yunnan into Sichuan fifty years later, but in their repeated assaults on and eventual highly destructive conquest of a state that sat cheek-by-jowl with what the new genetic evidence is telling us was probably the first home of plague. It has been crucial for the framing of the hypothesis that the genetic evidence points not only to a region but to a well-defined period. Most people in the world today have never heard of the state of Xia, or of the Mi (Tangut) people who founded it and successfully defied or coexisted with powerful neighboring Chinese and then Jurchen states for almost two hundred years. But if the hypothesis I articulate here is correct, the Mongols' choice to make Xia their first conquest outside of Mongolia has had enormous consequences for the history of the medieval and modern worlds.

Afternote

When Monica Green kindly invited me to contribute to the present issue, I had been away from the direct study of plague in Song-Yuan China for many years and had not followed all new developments. Thus, it was only when this article had reached its final stages that I was able to acquire Cao Shuji's more recent book on the history of plague in China and its association with war, coauthored with Li Yushang (Cao and Li 2006). I have been able to undertake only a quick review of the historical sections so far, and have not yet tracked down all the source passages that Cao and Li cite. On the Mongol-associated epidemics I discuss here, their evidence is essentially the same as in Cao's 1995 article (Cao and Li 2006: 79–99). More potentially problematic is their position, new for Cao, on certain epidemics of the early Jin (1139–48), which they also believe to be plague (Cao and Li 2006: 60–65).

Space does not allow a thorough examination of their evidence, but on my examination so far I find it very hard to agree that the *datou wen*, literally the “big-head epidemic” that their sources describe for the early Jin epidemics, is plague. Among the sources they cite, only retrospective accounts of Yuan or even Ming date mention sores of any kind (by which time, on my hypothesis, plague was known from recent experience in north China and later in south China, and retrospection could conceivably have conflated two different epidemic-associated diseases); and all their sources seem to describe a condition of general swelling of the throat-and-neck and often of the face, which they further describe as regularly involving “the throat closing” and an inability to open the eyes, a “filling”

of the eyes and ears obstructing sight and hearing, and a “head like an ox.” I have not encountered any accounts elsewhere of blindness, deafness, inability to open the eyes, throat-closing, or generalized swelling of the neck-and-face area as standard symptoms of plague. A Ming source (cited by Cao and Li 2006: 57–67 but especially 60–65) that associates “big-head epidemic” with the popular disease names “frog epidemic” and “cormorant epidemic” similarly seems to point to a generalized swelling of the throat-and-neck, since a frog puffs out its whole throat and a cormorant famously expands its throat when it swallows a large food item. Neither seems a likely simile for a discrete bubo on the neck.

I am currently undertaking deeper research into the medical sources Cao and Li cite on these issues, as well as other medical sources of the same period, and will publish my findings on them separately elsewhere. The scholarly discourse on plague among non-Chinese scholars has yet to take any notice, as far as I know, of this book. Nor has work in that discourse (that I am aware of, other than my own) paid proper attention to Cao’s 1995 article and his first heroic assembly of sources there. Such notice is well overdue.

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Abstract The work of Cui et al. (2013)—in both dating the polytomy that produced most existing strains of *Yersinia pestis* and locating its original home to the Qinghai-Tibet Plateau—offers a genetically derived specific historical proposition for historians of East and Central Asia to investigate from their own sources. The present article offers the hypothesis that the polytomy manifests itself in the Mongol invasion of the Xia state in the Gansu corridor in the early thirteenth century and continues in the Mongols’ expansion into China and other parts of Eurasia. The hypothesis relies to a considerable extent on work of Cao Shuji (1995), but argues for a different means and direction for the spread of plague than either Cao or William McNeill have previously posited.

Keywords Plague, Mongols, China, Black Death, Song Dynasty, Yuan Dynasty, Jin Dynasty, Xia, Gansu, rhubarb, oxalic acid.