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# Human–Bovine Plagues in the Early Middle Ages

This article combines written and plausible physical evidence for the human–bovine plagues (large outbreaks of acute disease) in 569–570 and 986–988 C.E. with evidence from two recent and independent molecular clock analyses (MCAs) that establish the divergence of measles (MV) from rinderpest (RPV) c. 1000 C.E. It proposes that the plagues of 569–570 and 986–988 testify to the outbreak of an MV–RPV ancestor that caused mass mortality in cattle and people. In other words, when spreading among cattle, a now-extinct morbillivirus episodically colonized and spread in human populations during the early Middle Ages.[1]

The diseases that afflicted early medieval Europeans have attracted considerable attention during the last fifteen years. Yet, most of the scholars involved, historians and bioarchaeologists alike, have rarely discussed pathogens other than *Yersinia pestis* or the occurrence and effects of diseases other than such episodic epidemics as the Justinianic Plague (otherwise known as the Early Medieval Pandemic). Economic, medical, and social historians of the early Middle Ages have occasionally devoted a few words to the nonbubonic epidemic of 569–570, the spread (or dormancy) of malaria, and the supposedly non-Yersinial plagues reported in

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1 For the idea that an MV–RPV predecessor was zoonotic in historical time, see Yuki Furuse, Akira Suzuki, and Hitoshi Oshitani, "Origin of Measles Virus: Divergence from Rinderpest Virus Between the 11th and 12th Centuries," *Virology Journal*, VII (2010), 3.

early Irish annals. Similarly, palaeomicrobiologists and palaeopathologists have drawn sporadic attention to some chronic, often nonlethal but endemic early medieval infections—such as leprosy, malaria, and tuberculosis. But, for the most part, non-Justinianic plagues, not to mention endemic diseases and epizootic diseases, have received little attention.[2]

2 For recent work on the Justinianic Plague, see Dionysios Stathakopoulos, "The Justinianic Plague Revisited," *Byzantine and Modern Greek Studies*, XXIV (2000), 256–276; Michael C. McCormick, "Rats, Communications, and Plague: Toward an Ecological History," *Journal of Interdisciplinary History*, XXXIV (2003), 1–25; Stathakopoulos, *Famine and Pestilence in the Late Roman and Early Byzantine Empire: A Systematic Survey of Subsistence Crises and Epidemics* (Aldershot, 2004), 110–154; Peregrine Horden, "Mediterranean Plague in the Age of Justinian," in Michael Maas (ed.), *The Cambridge Companion to the Age of Justinian* (New York, 2005), 134–160; Stathakopoulos, "La peste de Justinien (541–750): questions médicales et réponses sociales," in Anna-Marie Flambard Héricher and Yannick Marec (eds.), *Médecine et société de l'Antiquité à nos jours* (Harve, 2005), 31–48; *idem*, "Invisible Protagonists: The Justinianic Plague from a Zoocentric Point of View," in Ilias Anagnostakis et al. (eds.), *Animals and Environment in Byzantium (7th–12th c.)* (Athens, 2011), 87–95; Lester K. Little, "Plague Historians in Lab Coats," *Past & Present*, 213 (2011), 267–290; the twelve chapters in *idem* (ed.), *Plague and the End of Antiquity: The Pandemic of 541–750* (New York, 2007).

For comments on non-Justinianic early medieval disease, see Joel-Noël Biraben, "Disease in Europe: Equilibrium and Breakdown of the Pathocenosis," in Mirko Grmek (ed.), *Western Medical Thought from Antiquity to the Middle Ages* (Cambridge, Mass., 1998), 324, 345; McCormick, "The Imperial Edge: Italo-Byzantine Identity: Movement and Integration, A.D. 650–950," in Hélène Ahrweiler and Angeliki E. Laiou (eds.), *Studies on the Internal Diaspora of the Byzantine Empire* (Washington, D.C., 1998), 25–31; Otto S. Knotternus, "Malaria Around the North Sea: A Survey," in Gerold Wefer et al. (eds.), *Climate Development and History of the North Atlantic Realm* (Berlin, 2002), 339, 342–345; Jean-Pierre Devroey, *Economie rurale et société dan l'Europe franque (Vie-IXe siècles)* (Paris, 2003), 46; *idem*, "Catastrophe, crise et changement social: à propos des paradigmes d'interprétation du développement médiéval (500–1100)," in Luc Buchet et al. (eds.), *Actes des 9e journées anthropologiques de Valbonne* (Valbonne, 2009), 154; Donnchadh Ó Corráin, "Ireland *c.*800: Aspects of Society," in Dáibhí Ó Cróinín (ed.), *A New History of Ireland. I. Prehistoric and Early Ireland* (New York, 2005), 578–583. For more complete studies, though out of date, see Wilfrid Bonser, "Epidemics during the Anglo-Saxon Period," *Journal of the British Archaeological Association*, IX (1944), 48–71; *idem*, "Epidemics," in *idem*, *The Medical Background of Anglo-Saxon England: A Study in History, Psychology, and Folklore* (London, 1963), 51–97; William P. MacArthur, "The Identification of Some Pestilences Recorded in the Irish Annals," *Irish Historical Society*, XXIII (1949), 169–188.

For some palaeopathology, see Joel Blondiaux et al., "Epidemiology of Tuberculosis: A 4th to 12th c. AD Picture in a 2498-Skeleton Series from Northern France," in Gyorgy Pakfi et al. (eds.), *Tuberculosis: Past and Present* (Budapest, 1999), 519–30; G. Baggieri and F. Mallegni, "Morphopathology of Some Osseous Alterations of Thalassic Nature," *Palaeopathology Newsletter*, CXVI (2001), 10–16; Blondiaux et al., "Microscopic Study and X-Ray Analysis of Two 5th Century Cases of Leprosy: Palaeoepidemiological Inferences," in Charlotte A. Roberts et al. (eds.), *The Past and Present of Leprosy: Archaeological, Historical, Palaeopathological and Clinical Approaches* (New York, 2002), 105–110; M. G. Belcastro et al., "Leprosy in a Skeleton from the 7th Century Necropolis of Vicenne-Campochiaro," *International Journal of Osteoarchaeology*, XV (2005), 431–448; Jesper L. Boldsen, "Leprosy in the Early Medieval Lauchheim Community," *American Journal of Physical Anthropology*, CXXXV (2008), 301–310.

Specialists of periods richer in written sources than the early Middle Ages advise that it is a serious mistake to dismiss the baseline of endemic disease. They argue that the constant pressure of nonkilling pathogens inhibited demographic and economic growth over the long term far more than most epidemics. Nonkilling diseases also contributed significantly to excess mortality via malnutrition and secondary infections, aggravating the death toll during periods of dearth and epidemic. A growing body of work, some of it centered on the early Middle Ages, also finds that epizootic disease, of domestic bovines especially, carried considerable repercussions for human health and economy in organic agrarian economies like those of early medieval Europe, which were largely dependent on cattle for traction and fertilizer and, to a lesser extent, for dairy and meat.[3]

Additionally, the Justinianic Plague west of the Balkans after 600 may well be ill-deserving of the attention that historians and scientists have given it. In a 2007 study, Bachrach argued that the Justinianic Plague, which is now held to have occurred from fourteen to eighteen times between 541 and 750 (or 767), did not have the dramatic, long-term negative effect on Merovingian France's demographic curve that most scholars suppose; outbreaks were "too infrequent and widely dispersed." A similar argument could be advanced for neighboring European regions, as the series of charts and maps that Biraben and LeGoff attached to their seminal article about the "first bubonic plague pandemic" appears to suggest. Their essay demonstrates clearly that most seventh- and eighth-century Justinianic re-occurrences did not much affect Central, Northern, or Western Europe. Moreover, subsequent research confirms that Justinianic plagues after 600 were largely restricted to Greek-, Syriac-, and Arabic-speaking lands, and eruptions

3 Walter Scheidel, *Death on the Nile: Disease and the Demography of Roman Egypt* (Leiden, 2001), xxi; Andrew Cunningham, "Disease: Crisis or Transformation?" in Troels Dahlerup and Per Ingesman (eds.), *New Approaches to the History of Late Medieval and Early Modern Europe* (Copenhagen, 2009), 407; Carroll Gillmor, "The 791 Equine Pestilence and its Impact on Charlemagne's Army," *Journal of Medieval Military History*, III (2005), 23–45; Newfield, "A Great Carolingian Panzootic: The Probable Extent, Diagnosis and Impact of an Early Ninth-Century Cattle Pestilence," *Argos: Bulletin van het Veterinair Historisch Genootschap*, XLVI (2012), 200–210; *idem*, "Early Medieval Epizootics and Landscapes of Disease: the Origins and Triggers of European Livestock Pestilences, 400–1000 CE," in Sunhild Kleingärtner et al. (eds.), *Landscapes and Societies in Medieval Europe East of the Elbe: Interactions Between Environmental Settings and Cultural Transformations* (Toronto, 2013), 73–113.

further west were rare and regional—in England and Ireland (664–666 and 684–687), in France (c.640 and 693), and in Italy (608, 654, 746, and possibly 767).[4]

Conceivably, seventh-century northwestern insular Justinianic outbreaks could imply corresponding continental disseminations, and physical evidence for *Y. pestis* in places where written evidence for the Justinianic Plague is lacking (sixth-century Aschheim, Germany, and seventh-/ninth-century Vienne, France) might well suggest that written sources do not tell the whole story. It remains uncertain, however, whether seventh- and early eighth-century outbreaks in the West were vast and numerous enough to inhibit demographic growth until 750. Extant sources signal regular and significant population pruning in Europe only before 600, and principally in the south.[5]

THE PROMISE OF AN INTERDISCIPLINARY METHODOLOGY This article looks at a range of issues, both new and not so new, but all of them wholly non-Justinianic. Like other recent studies in the history of disease, it combines the sources, methods, and results of usually isolated disciplines—in this case, history, evolutionary biology, and zooarchaeology—offering insights unattainable otherwise. The results herein are necessarily more speculative than definitive. The temporal parameters of the written evidence and the spatial parameters of the zooarchaeological evidence are precise (at least

4 Bernard S. Bachrach, "Plague, Population, and Economy in Merovingian Gaul," *Journal of the Australian Early Medieval Association*, III (2007), 29–57 (Bachrach argues that damage before 600 was minimal, a position not supported herein); Devroey, *Economie rurale*, 47; Jean-Nöel Biraben and Jacques LeGoff, "La peste dans de haut moyen âge," *Annales: Économies, Sociétiés, Civilisations*, XXIV (1969), 1484–1510.

5 For *Y. pestis* in early medieval bones, see Ingrid Wiechmann and Gisela Grupe, "Detection of *Yersinia pestis* DNA in Two Early Medieval Skeletal Finds from Aschheim (Upper Bavaria, 6th Century A.D.)," *American Journal of Physical Anthropology*, CXXVI (2005), 48–55; Michel Drancourt et al., "*Yersinia pestis Orientalis* in Remains of Ancient Plague Patients," *Emerging Infectious Diseases*, XIII (2007), 332–333; M. Harbeck et al., "*Yersinia pestis* DNA from Skeletal Remains from the 6th Century AD Reveals Insights into Justinianic Plague," *PLOS Pathogens*, IX (2013), 1–8. Regardless of the validity of his final conclusion, Samuel Cohn's observations about the epidemiological (and, to a lesser extent, symptomological) mismatch of (modern) bubonic plague with the Justinianic Plague demonstrate that the popular diagnosis of the sixth- through eighth-century plague is not uncomplicated. See Cohn, "Epidemiology of the Black Death and Successive Waves of Plague," in Vivian Nutton (ed.), *Pestilential Complexities: Understanding Medieval Plague* (London, 2008), 74–77.

to the year in the texts), but the reverse is not true. The imprecision in the temporal and spatial parameters of the molecular data also create difficulty in marrying the different datasets. That the zooarchaeological material presented below is evidence of epizootic disease is also a matter of some dispute.[6]

The present study is also an exercise in retrospective diagnosing, a tool as much debated as practiced, which yields satisfying results only when a preliminary diagnosis, usually based on written reports of symptoms, is confirmed via the isolation of the suspected pathogen in skeletal remains. No one appears to have successfully applied the palaeomicrobiological techniques that have been employed in studies of past bubonic plague, leprosy, and tuberculosis to the diseases studied herein—the closely related morbilliviruses MV and RPV. A specific issue for this analysis, which is central to the debate about retrospective diagnosing, concerns the intended meaning and possible inconsistency of certain "medical" terminology, such as *variola*, over time and place.[7]

Notwithstanding these caveats, the collaboration between early medieval textual accounts of plagues, MCAs of the divergence time of measles and rinderpest, and recently uncovered bovine graves, allows for strong, albeit tentative, retrospective diagnoses of two non-Justinianic, early medieval mortalities. The utilization of different data helps to explain otherwise difficult-to-interpret early medieval accounts of simultaneous plague-scale deaths in people and cattle, and it allows for speculative disease identifications sturdy enough to support an assessment of the extent and consequences of two postclassical human–bovine disease events

6 For a recent interdisciplinary study in the history of disease, see Sharon DeWitte and Philip Slavin, "Between Famine and Death: England on the Eve of the Black Death—Evidence from Palaeoepidemiology and Manorial Accounts," *Journal of Interdisciplinary History*, XLIV (2013), 37–60.

7 For the retrospective diagnosis debate, see Jon Arrizabalaga, "Problematizing Retrospective Diagnosis in the History of Disease," *Asclepio*, LIV (2002), 51–70; Andrew Cunningham, "Identifying Disease in the Past: Cutting the Gordian Knot," *idid.*, 13–34; Piers D. Mitchell, "Retrospective Diagnosis and the Use of Historical Texts for Investigating Disease in the Past," *International Journal of Paleopathology*, I (2011), 81–88. Current biomolecular techniques may be unable to identify remains of viruses in archaeological specimens and therefore MV and RPV. For cattle remains from a major French burial sent for laboratory study (discussed below), see Sylvain Renou, Cédric Beauval, and Marie Maury, "Un bilan des connaissances sur les epizooties au Moyen Âge et un episode de mort extraordinaire de bovines à Luxé (Charente) durant l'époque mérovingienne," in Ginette Auxiette and Patrice Meniel (eds.), *Les dépots d'ossements d'animaux en France, de la fouille à l'interprétation* (Montagnac, 2013), 140.

that would be impossible on the basis of the written evidence or physical evidence alone. In drawing on MCAs and advancing tentative MV–RPV diagnoses, this article not only adds flesh to theory-based molecular studies and the deep past of two pathogens of major public-health importance; it also broadens the understanding of early medieval mortality crises and the demography of mid sixth- and late tenth-century Europe.

The study provides palaeomicrobiologists with a new direction, should techniques become available for the study of viruses in archaeological specimens and heretofore overlooked farm-animal and zoonotic pathogens of the past (zoonotic pathogens being capable of causing disease in both humans and other animals). More generally, the diverse datasets expand the focus of medieval disease studies to include human-domesticate disease as well as extinct pathogens. The plagues of 569–570 and 986–988 studied below are the oldest antecedents to such present-day acute and highly infectious diseases of livestock as avian or swine influenza, capable of jumping species and colonizing human populations. The attention to extinct disease is particularly noteworthy: Retrospective diagnosing and the historiography of disease often function on the assumption that the diseases in the historical record still exist and work in familiar ways. This article, at least the molecular part of it—highlights errors in that manner of thinking. To make use of an extinct morbillivirus diagnosis, however, the paper is forced to presume that an ancestral MV–RPV would have behaved in humans like MV does and in bovines like RPV did until its 2011 eradication.[8]

EARLY MEDIEVAL HUMAN–BOVINE MORTALITY EVENTS A recent survey of early medieval written sources for epizootic disease turned up eighty-three passages, to which four others now may be added, pertaining to between thirty-two and fifty-four individual European (and some Western Asian) animal plagues. The passages are sufficiently short and vague that efforts made to match and separate them were not always successful, leading to uncertainty in the number of plagues recorded. That said, forty-four passages clearly refer to

8 On May 25, 2011, the Organisation mondiale de la santé animale declared the world free of RPV, as did the Food and Agricultural Organization on June 28, 2011. See David M. Morens et al., "Global Rinderpest Eradication: Lessons Learned and Why Humans Should Celebrate Too," *Journal of Infectious Diseases*, CCIV (2011), 1–4.

seven vast outbreaks of epizootic disease, and another fourteen passages seem to refer to an additional three major livestock mortality events. Of the total passages, twenty-nine, or 33 percent, refer to zoonotic outbreaks in humans and farm animals. Of that 33 percent, at least 86 percent identify cattle as the sick domesticate. Seemingly, acute infectious diseases common to people and cattle, though rare in the modern era, were not unusual in early medieval Europe.[9]

It is unlikely, but not impossible, that non-bovine livestock died in some early medieval bovine and human–bovine plagues. Cattle are mentioned far more often than other domesticates as suffering virulent widespread disease in the early Middle Ages, possibly because, at least in part, their deaths mattered more: In many regions, they were the most socioeconomically important species on the farm. Early medieval written sources suggest that horses and sheep may have been next-most affected by disease outbreaks; pigs sustained only rare, localized epizootics. The question of whether these animals were healthier than bovines or simply of less interest when dead to early medieval writers is uncertain, and hence significant for retrospective diagnosing. That few pathogens are known to cause large outbreaks of acute disease in multiple domesticated species, however, suggests that non-bovines were indeed less exposed to highly infectious, acute diseases.[10]

At least three of a possible nineteen early medieval human–bovine plagues were major events, spreading across parts of continental Europe and northwestern insular Europe c. 569–570, 868–870, and 986–988. There is also some indication that the vast cattle panzootic of 809–810 was zoonotic; one of the sources for it refers to concurrent mortalities in humans. Textual and potential physical

9 For the recent survey, see Newfield, "Early Medieval Epizootics," 79–88, 112. The four animal plagues not included therein are (1) the "lethal plague" c.401 that Rufinus of Aquileia describes as spreading "far and wide," carried by Alaric and the Goths "to the ruin of fields, herds and men" (Rufinus of Aquileia [trans. Philip R. Amidon], *The Church History of Rufinus of Aquileia: Books 10 and 11* [New York, 1997], 3); (2) the pestilential cloud that affected mid-sixth-century humans and bovines in Ireland (Adomnán of Iona [ed. J. T. Fowler], *Vita sancti Columbae* [Oxford, 1894], II.4, 73–75); (3) the "great food shortage and mortality of people and pestilence affecting beasts" of 869 (Byrhtferth [ed. Cyril Hart], *East Anglian Chronicle* [Lewiston, 2006], 89); and (4) the cattle plague in the *Saltair na Rann*, discussed herein.

10 For early medieval diseased horses, sheep, and pigs, see *Annales qui dicuntur Einhardi*, in Friedrich Kurze (ed.), *Monumenta Germaniae Historica Scriptores rerum Germanicarum* (Hanover, 1895), VI, 89, 91; *Annales Fuldenses*, in *ibid.* (1891), VII, 105, 127; *Annales Quedlinburgenses*, in Martin Guise (ed.), *ibid.* (2004), LXXII, 484, 489.

evidence for these plagues provides grounds for fairly good disease identifications, since multiple epidemiological (epizootiological, in the case of animal disease) properties can be teased from them. For instance, the sources indicate, significantly, that among domesticates, only bovines died in these plagues. More robust, but still tentative, text-based diagnoses, founded on knowledge of epidemiology/epizootiology and symptoms, are harder to find; only two of the eighty-seven references to animal disease unambiguously mention symptoms. Those passages pertain to the 569–570 and 986–988 plagues.[11]

THE PLAGUES OF 569–570 AND 986–988 Sometime during the late 500s (c.585?), Marius, bishop of Avenches, wrote in his chronicle, "Virulent disease greatly afflicted Italy and France with a flow of the bowels and variola, and beef animals died especially through the aforementioned places." Marius dates the passage to 570, but from the mid-560s, he is a year ahead. The second and last source for the 569–570 plague is much later, composed c.835 to 840 by Agnellus of Ravenna in his *Liber Pontificalis Ecclesiae Ravennatis*: "In the fifth year of Emperor Justin II [570] there was a pestilence of oxen and a destruction everywhere." Marius and Agnellus' accounts of die-offs in 569–570 are almost certainly independent of one another but probably refer to the same mortality, given the unlikelihood that multiple large plagues affecting cattle would erupt in successive years in roughly the same region (Marius places the outbreak in France and Italy; Agnellus addressed events primarily in northern Italy). Agnellus' known sources do not refer to the plague, and although it is unclear whether Agnellus consulted Marius' *Chroncia*, his writing shows no verbal parallels with it. Agnellus cited two nonextant works—the *Chronicon* by Maximian of Ravenna, which may have extended into the 570s, and an unknown "annalistic source" thought to have continued until 573. He may have derived his account from one of these texts.[12]

11 For a zoonotic 809–810 panzootic, see *Annales Laurissenses Minores*, in Georg Heinrich Pertz (ed.), *Monumenta Germaniae Historica Scriptores* (Hanover, 1826), I, 121; for this cattle plague, Newfield, "Carolingian Panzootic," 200–210.

12 Marius of Avenches, *Chronica*, in Theodor Mommsen (ed.), *Monumenta Germaniae Historica Auctores Antiquissimi* (Berlin, 1894), XI, 238; Agnellus of Ravenna, *Liber Pontificalis Ecclesiae Ravennatis*, in Oswald Holder-Egger (ed.), *Monumenta Germaniae Historica Scriptores rerum Langobardicarum et Italicarum* (Hanover, 1878), 337; Deborah Mauskopf Deliyannis (trans.), *The Book of Pontiffs of the Church of Ravenna* (Washington, D.C., 2004), 49, 205.

Agnellus assigned no spatial parameters to the epizootic, and those of Marius are general. The plague appears to have affected a wide region, however, as both authors claim independently. Whether the disease was prevalent throughout what is now France and Italy is less certain. Marius tended to focus on Burgundy, the region where he lived; the mortalities that he reported may have concentrated there and in neighboring northern Italy. Agnellus may have been referring to mortalities in the same area. Yet on the basis of the evidence available, it would be unwise to limit the 569–570 plague to Burgundy and the northern Italian peninsula, considering the emphasis that both authors placed on the number of deaths and the improbability of so acute and wide-occurring a disease being endemic (enzootic, in the case of animal disease) to the area in which it erupted. The plague was almost certainly introduced from another region or disease pool, possibly some distance away, where it was native. The temporal parameters, triggers, geographical origins, and paths of dissemination of the plague are all hazy, though the Lombard migration from western Hungary into Italy and briefly to parts of France in 568 might have introduced the plague to those regions.[13]

The species involved are much clearer. Although some scholars have read Marius' passage as evidence of an exclusively human epidemic and others a bovine epizootic, both authors might have been referring to people and cattle. Marius and Agnellus wrote explicitly of cattle dying (*animalia bubula* and *boves*) and implied—in Marius' case, anyway—concurrent deaths in humans. Indeed, Marius' reference to dead beef animals appears as an insertion in his report of a human disease also spreading in Italy and France. The *interitus* that Angellus mentioned, following his note about the cattle pestilence, could be read as additional emphasis on the magnitude of the cattle die-off or as a separate mortality of another species, in all likelihood human, unrelated in Angellus' mind to the *pestilentia bovum*.

13 RPV was a mild disease with low rates of morbidity and mortality in enzootic zones but a severe disease with high rates of morbidity and mortality in epizootic zones. See, J. Anderson, Thomas Barrett, and G. R. Scott, *Manual on the Diagnosis of Rinderpest* (Rome, 1996), 6–7; Anonymous, "Rinderpest," in Susan E. Aiello and Asa Mays (eds.), *The Merck Veterinary Manual 8th Edition* (Toronto, 1998), 543. Sixth-century Burgundy was centered in Geneva, spanning nearly from Arles in the south to Sens in the north and from Bourges in the west to Basle in the east. For the Lombard migration and introductions of ecdemic epizootic disease into early medieval Europe, see Newfield, "Early Medieval Epizootics," 75, 88–99, 111.

The sources for the plague from 986 to 988 are more numerous and explicit, though also brief. The *Anglo-Saxon Chronicle* observes that "the great cattle plague first came to England" in 986, and three versions of the Welsh *Brut y Tywysogion* have an animal die-off occurring in that year or in 987. The most reliable and complete rendering, the Peniarth MS 20, records that "a mortality took place upon the cattle in all the island of Britain." A longer account is encountered in the *Chronicon ex Chronicis*, slotted into the year 987: "Two plagues unknown to English people in past generations, namely, a fever of humans and a plague of animals, which in English is called 'shit' but in Latin can be called the 'flux of the bowels,' have thoroughly afflicted all England, and raged indescribably in all parts of England, affecting people with a great destruction and widely consuming the animals."[14]

The "plague and mortality, cattle plague, and disease" that Wulfstan II includes in his list of England's recent difficulties in *Sermo Lupi ad Anglos* may also be this 986–988 mortality, since he composed his homily in 1014, and no other English bovine die-off is known between the 890s and the 1040s. Wulfstan was probably alluding to the 986–988 plague in another homily commonly attributed to him (Napier 35) and written around the same time as the *Sermo Lupi ad Anglos*, which beseeches God's help against (among other disasters) "cattle plagues or human plagues through sudden diseases."[15]

Across the Irish Sea, the *Annals of Tigernach*, *Annals of Ulster*, and *Chronicon Scotorum* record pestilential deaths of people and cows in 987. Following notice of "a colic in the east of Ireland" and subsequent human mortality, the *Tigernach* observes that "the beginning of a great murrain, to wit, the unknown *máelgarb*, came for the first time"; the *Chronicon* notes "a sickness . . . in the east of Ireland which caused death among the people" and "the beginning of the cattle-plague, the *máelgarb*, such as had not occurred before";

14 Margaret Ashdown (trans.), "The Anglo-Saxon Chronicle (C), Annals 978–1017," in *English and Norse Documents Relating to the Reign of Ethelred the Unready* (Cambridge, 1930), 40–41; for the *Chronicon ex Chronicis*, Jennifer Bray and P. McGurk (trans.), *Chronicle of John of Worcester II* (Oxford, 1995), 436–437; for *Brut y Tywysogyon: Peniarth MS. 20,* Thomas Jones (trans.), *Brut y Tywysogyon, or Chronicle of Princes: Peniarth MS. 20 Version* (Cardiff, 1952), 10.

15 For Wulfstan's passages, see Dorothy Whitelock (ed.), *Sermo Lupi ad Anglos* (London, 1952), 39–40, 57–59; Arthur Napier (ed.), *Wulfstan: Sammlung der ihm zugeschriebenen homilien nebst untersuchungen über ihre echtheit* (Berlin, 1883), XXXV, 170; Joyce Tally Lionarons, *The Homiletic Writings of Archbishop Wulfstan* (Woodbridge, 2010), 30–32.

and *Ulster* tells of "a sudden great mortality which caused a slaughter of people and cattle in England, Wales and Ireland."[16]

The *Saltair na Rann* is thought to refer to this plague as well. In fact, the cattle mortality has long been used to date this Irish versing of biblical history to the late tenth century, unsurprisingly given the way in which the poet (or an interpolator) describes the passage of time to the present: "From Adam of the bright singing orders" to "the great slaughter of cattle" and the period "From the birth of Christ . . . " until "the hundred-fold destruction of the cattle" (which leads to a 988 dating). He then proceeds to list the kings reigning "when the pitiful vengeance came on the cattle of many countries." The Irish, Welsh, and Scottish kings mentioned were indeed ruling when the disease was spreading, but those from farther afield either may not have been (like Lothar of France *d*. March 986) or, to the best of our knowledge, were not (like Otto II of Germany *d*. 983) in power when the disease passed through their, or what were their, realms. Another king, England's Edgar I, had been dead for a decade (975). The *Saltair na Rann* clearly does not supply reliable evidence for the mortality in England or on continental soil from 986 to 988. That the first king in the list is Scottish (Cináed mac Maíl Choluim) and in power during the outbreak, however, raises the possibility that Scotland was affected.[17]

16 For the Irish annals, see The Corpus of Electronic Texts (CELT), available at http://celt.ucc.ie/publishd.html (accessed March 14–15, 2011). William M. Hennessy (trans.), *Chronicon Scotorum* (London, 1866), 231, dates the pestilence to 985, which was changed to 986 in CELT. *Idem* (trans.), *Annals of Ulster* (Dublin, 1887), 497), is preferable to Mac Airt's translation in CELT, though Hennessy dates the passage one year early. Dan McCarthy confirmed the date of 987 to be correct in a private correspondence of July 22, 2011.

17 Whitley Stokes (ed.), *Saltair na Rann* (Oxford, 1883), I.XII.2337–2372, i, v, 34 (erroneously dating the plague to 985); Benjamin T. Hudson, *Vikings Pirates and Christian Princes: Dynasty, Religion, and Empire in the North Atlantic* (New York, 2005), 62, 69, 220, n. 46. Some scholars have interpreted this entire dating passage as an interpolation, others only the kingly section. Gearóid S. Mac Eoin noted and accepted the common "plague dating" of 988 but argued that the regal part was inserted later, possibly by a Scotsman. He also proposed the *Saltair na Rann*'s Corkonian connection, although Airbertach mac Cosse of Rosscarbery is not universally accepted as having written the text: Gearóid S. Mac Eoin, "The Date and Authorship of Saltair na Rann," *Zeitschrift für celtische Philologie*, XXVIII (1960/61), 51–67; *idem*, "Observations on Saltair na Rann," *ibid*., XXXIX (1982), 2, 11, 20–21. The 988 plague date implies that the *Saltair na Rann* was not composed in central Ireland, where Irish annals document the outbreak a year earlier, unless the disease persisted there for a year and the poet/interpolator chose not to date the mortality according to its initial appearance. A composition at a distance, perhaps the far southwest, as Mac Eoin proposed, may account for the date. The

Like that of the seventh- and eighth-century Western European Justinianic plagues, knowledge of the spatial and temporal parameters of this plague, as well as the plague of 569–570, is limited. The sources indicate that the pestilence that began in 986 appeared first (among the regions known to have suffered from it) in England before spreading westward to Wales in 986 or 987 and Ireland in 987. The entry in the *Anglo-Saxon Chronicle* that it "came to England" suggests that the disease was ecdemic or foreign to northwestern insular Europe and brought to England from the continent, as does its apparent westward trajectory and alleged, or implied, wide prevalence and high mortality. Reference in the *Annals of Tigernach* and *Chronicon Scotorum* to the massive losses in Ireland, the dating of the plague in Irish texts to 987 and 988, and the implication in the *Annals of Ulster* that the disease appeared in England and Wales before Ireland reveal that this plague did not originate in Ireland. The labeling of the disease as new or unknown in England and Ireland may be a trope; such qualifying of plagues was not then atypical. Nevertheless, the plague was almost certainly imported from another disease pool, likely by living cows or people but possibly by other means as well. Although there is no record of widespread livestock disease on the continent in the late tenth century prior to 986, large concurrent human and bovine mortalities are reported in central-north Germany in 989–990 and 993. These events may be interpreted as either the continued circulation of the disease on the continent after its insular introduction or as unrelated plagues.[18]

Five independent and contemporary texts emphasize the vastness of the mortality among people and cattle. That several of these passages were composed originally at a distance from each other (in all probability Abingdon for *Anglo-Saxon Chronicle*; Worcester for the *Chronicon ex Chronicis*; St. David's for the Welsh Brut;

translation in this paragraph is from David Greene, which was unfinished at his death but available on the website of the Dublin Institute for Advanced Studies—www.dias.ie (accessed March 10, 2014).

18 The plague is dated in *Brut's* Peniarth MS. 20 to 986 (no dates are given in the *Red Book* or *Brenhinedd y Saesson* versions of the *Brut*). Based on a comparison of passages in Peniarth, the *Annals of Ulster*, and *Chronicon Scotorum*, however, Jones, the editor of the Brut texts, proposes that the dates given in the Peniarth from 974 to 1003 are a year behind. For continental human–bovine plagues in 989–990 and 993, see *Annales Quedlinburgenses*, 447; *Annales Hildesheimenses*, in Georg Heinrich Pertz (ed.), *Monumenta Germaniae Historica Scriptores* (Hanover, 1839), III, 68, 70.

Clonard or Clonmacnoise for the *Annals of Tigernach* and *Chronicon Scotorum*; Clonard, Clonmacnoise, or Armagh for the *Annals of Ulster*; and possibly Rosscarbery for the *Saltair na Rann*) supports the sources' claim of a general, rapidly developing outbreak (the *Annals of Ulster* describe the plague as "sudden," as Wulfstan II seems to do), presuming animals died not far from where the texts were composed, and manuscripts did not circulate widely during the plague years. The *Saltair na Rann*'s poetic treatment of the cattle mortality as a memorable event and Wulfstan's inclusion of people's and cattle's deaths in his homilies (assuming that he refers to the plague of 986–988) suggests that the event was by no means trifling. People and cattle in south-central England, the West Midlands, south-western Wales, and central (and possibly northeast) Ireland seem to have been afflicted within about a year. Such a fast and wide dissemination implies that the pathogen was not principally arthropod- or soil-borne but transmissible between susceptible species, like the 569–570 plague—yet another indicator that animals south of the English Channel were affected.[19]

The pestilence's trigger, geographical origin, and duration, however, remain opaque. The outbreak does not, for instance, appear to correspond spatially or temporally to a subsistence crisis, climatic anomaly, or major conflict that could have fostered its introduction into Britain or continental Europe. That Danes brought it with them to the Hebrides and Irish Sea region in 986 seems unlikely considering the dating and progression of the outbreak, as traced above. Following the methods of dissemination common to infectious airborne diseases, including MV and RPV, these plagues likely spread within and between both urban and rural communities along local and regional routes of travel and trade. Although exceptional events may have driven the causative pathogen from a distant region, once

19 RPV and MV are typically introduced to regions via the travel of their victims, either before, during, or after the incipient stages of severe disease. See T. U. Obi, P. L. Roeder, and W. A. Geering, *Manual on the Preparation of Rinderpest Contingency Plans* (Rome, 1999), 5; Anderson, Barrett, and Scott, *Diagnosis of Rinderpest*, 7; Robert T. Perry and Neal A. Halsey, "The Clinical Significance of Measles: A Review," *Journal of Infectious Diseases*, CLXXXIX (2004), S4; Gregory Hussey, "Measles," in Richard David Semba and Martin W. Bloem (eds.), *Nutrition and Health in Developing Countries* (Totowa, 2008), 216; Centers for Disease Control and Prevention, "Measles," in W. Atkinson, J. Hamborsky, and S. Wolfe (eds.), *Epidemiology and Prevention of Vaccine-Preventable Diseases* (Washington, D.C., 2012), 178. For the 989–990 and 993 mortalities, see Georg Waitz (ed.), *Annales Hildesheimenses* (Hanover, 1878), VIII, 25, 26; Guise (ed.), *Annales Quedlinburgenses*, 477.

it was introduced elsewhere the normal movements of people and cattle were likely sufficient to cause the mortalities reported. Major events, such as the Lombards' arrival in Italy, which may have itself introduced the pathogen of the 569–570 plague to Western Europe, no doubt helped, but they were not, as the 986–988 plague testifies, always necessary.[20]

The *Chronicon ex Chronicis*' dating suggests that the disease persisted for a year at least in some regions, as does the assigning of the plague to 988 in the *Saltair na Rann*. Areas especially cattle-rich may have suffered longer. The *Annals of Ulster*'s account of a "great mortality of people, cattle, and bees throughout Ireland" in 993 may signify the outbreak's persistence there for many years, similar to Ireland's experience with the 1314/25 panzootic. While the *Anglo-Saxon Chronicle*, *Brut*, and *Saltair na Rann* refer solely to the bovine mortality, the *Chronicon ex Chronicis* and Irish annals (and possibly Wulfstan) make it clear that humans died concurrently.[21]

MASS BOVINE GRAVES Until recently, the evidence for large early medieval plagues of livestock derived strictly from written sources. The dearth of identified mass burial sites appears to have had little to do with the once-popular interpretation of intact or largely intact animal skeletons that showed no traces of butchery as remnants of ritual or offering but to an actual absence of known animal "plague pits." The abandonment of animals that were victims of disease, extreme weather, or famine in fields, forests, and water may account for the lack of known mass burials, as would their interment away from excavated human sites. Early medieval

20 For the Danes, see T. M. Charles-Edwards, *Wales and the Britons, 350–1064* (New York, 2014), 541. That the Airgíalla managed to steal 2,000 head of cattle from Armagh in 996 (Ó Corráin, "Ireland c.800," 569) suggests, but does not confirm, that the 986–988 mortality spared Armagh and that this *Annals of Ulster* passage derived from the other Irish sources (a Clonmacnoise-group text). Lesser conflicts in northern Wales and on Mann in 986 and 987—involving Hiberno-Scandinavians, Welsh, and Danes—and the capture and presumed sale of a reported 2,000 Anglesey inhabitants in 987 (Charles-Edwards, *Wales and the Britons,* 549–550), may have fostered the plague's development in Britain. The Danes' ability to pillage a handful of southern Welsh monasteries with Guthfrith Haraldsson in 988 and exact a tribute from Maredudd ab Owain in 989, however, implies that they did not fall sick in large number in 986–988 but that the southern Welsh did. Hudson speculates that the Danes and Haraldsson attacked peoples "weakened" by the plague in the Hebrides, Mann, and Wales (*Vikings Pirates*, 62).
21 For the 993 mortality, see *Annals of Ulster* and *Chronicon Scotorum* in CELT (n.16). The *Chronicon Scotorum* limits the death toll to Clonmacnoise.

animal-bone assemblages thought to demonstrate large cattle mortalities, however, have now been unearthed in England, France, and Switzerland, and, like recently discovered mass animal burials from earlier and later periods, linked to epizootic disease. Such sites give some credence to the textual reports of large bovine mortalities. Indeed, these English, French, and Swiss graves lend confidence, though not confirmation, to reports of sudden and severe cattle die-offs in early medieval Europe—the plagues of 569–570 and 986–988 in particular—in much the same way that the more common human plague pits do for Justinianic plagues. The mass graves may also expand the known geographical bounds of the 569–570 and 986–988 plagues, underscoring observations made about the causative pathogens' principal mode of transmission, and probable high communicability, morbidity, and mortality.[22]

The number of animal skeletons necessary to indicate a large mortality event has been set at about four. These animals need not be buried in a single pit; multiple individual burials suffice. But individually interred creatures must be contemporaneous and, when carbon-14 tested, show homogenous dates. Preferably, the animals should be articulated, though bulky creatures could have been chopped into large pieces for transport. But so far as researchers are concerned, signs of a quick and deep burial; the covering of carcasses with lime (a traditional agricultural disinfectant); no or little recovery of meat, hides, hooves, or horns; and interment at an age otherwise optimal for draught, milk, and meat all serve to indicate an epizootic. Mass burials of a single species may be considered

22 For cattle abandoned in fields following the 809–910 and 878 disease outbreaks, see Poeta Saxo (ed. Paul de Winterfeld), *Annalium de Gestis Caroli Magni Imperatoris Monumenta Germaniae Historica Poetarum Latinorum* (Berlin, 1899), IV, 236–253, 51–52; Kurze (ed.), *Annales Fuldenses*, 92. Various ancient and late antique authors, perhaps reflecting common practice, recommended the isolation of sick animals. Drawing on Columella in the late fourth or early fifth century, Vegetius, for instance, stated that the graves of dead animals should be placed deep and far from human settlements. See E. Lommatzsch (ed.), *Digestorum Artis Mulomedicinae Libri* (Leipzig, 1903), I.1.3, 16; I.17.3, 34; IV.2.15, 283–284. No known mass burial dated to the early Middle Ages suggests a human–bovine plague. Domesticates were buried separately from humans; the animals (notably horses) occasionally found in human burials are generally considered to be ritual sacrifices or grave goods, see Bonnie Effros, *Merovingian Mortuary Archaeology and the Making of the Early Middle Ages* (Berkeley, 2003), 164–165; Howard Williams, *Death and Memory in Early Medieval Britain* (New York, 2006), 82, 175, 177; Pamela J. Cross, "Horse Burial in First Millennium AD Britain: Issues of Deposition," *European Journal of Archaeology*, XIV (2011), 190–209.

additional proof of a disease event, since most known virulent livestock diseases affect only one species. The English, French, and Swiss burials fit most of these criteria, though they cannot be considered definitive evidence for epizootic disease without further laboratory study for causative pathogens. These cattle might have died as a consequence of extreme weather or famine.[23]

The first of the large early medieval bovine burials was uncovered in Shapwick, England, about a 75 miles southwest of Abingdon, 80 miles south of Worcester, and 120 miles east of St. David's, across the Bristol Channel; seven intact animals were found in a limekiln, which was not fully excavated. All of the cattle were adult males, likely draft animals, articulated and devoid of skeletal stigmata, suggesting that they were healthy and died a sudden death in a disease outbreak of a "national" scale. Researchers have posited the early eleventh century as a probable date, but the radiocarbon-dated tibiae permit a wider range of 980 to 1160, meaning that these animals could have succumbed to the 986–988 plague. The grave's proximity to the location of the English and Welsh written reports for that pestilence would support this notion, though other bovine epizootics are reported for southern England within the 980 to 1160 window.[24]

The second of the multi-specimen graves was uncovered in Luxé, France, 340 miles west of Avenches, 600 miles west of Ravenna, and about 140 miles west of the western edge of early medieval Burgundy—ten largely intact bovine skeletons in close singular pits with an early medieval dating around the early seventh century, though some carbon-14 dates are as early as 570. Like the Shapwick animals, these were in the prime of their life (most of them between one and six years old), showing no signs of chronic infection or malnourishment and no or little evidence of having

23 Sophie Lefebvre, "Inhumations de bovines sur le site medieval et moderne d'Auby 'l'Îlot Béguinage' (Nord)," in Auxiette and Meniel (eds.), *Les dépots d'ossements d'animaux*, 150, 152; Annelise Binois, "Approche méthodologique des mortalités de masse ovines en archéologie," *ibid*., 278, 279–280; Renou, Beauval, and Maury, "Un bilan des connaissances," 136, 140; Olivier Putelat, "Archéologie des depots animaux et mortalité extradinaire du cheptel bovin au premier Moyen Âge: Plaidoyer pour une redynamisation et une mise en perspective de la recherché," in Auxiette and Meniel (eds.), *Les dépots d'ossements d'animaux*, 249, 252, 253, 259–262.

24 Louisa J. Gidney, "Murrain or Starvation? Catastrophic Cattle Mortality Events in Medieval and Ports Medieval England and Their Repercussions," in Richard Thomas and Theodore G. Antikas (eds.), *Animal Disease in Past Human Societies: Proceedings of the Fourth ICAZ Animal Palaeopathology Working Group* (Atlanta, forthcoming).

been used for meat or raw materials. The burials were deep and rapidly completed.[25]

Five bovines found at a third site at Bure, Switzerland, are dated to the late ninth through early eleventh century. The one bovine that was carbon-14 dated appeared to be buried slightly before the others, in the mid-tenth century. The others were interred simultaneously or near simultaneously, some of them covered with limestone blocks. It has been suggested that the animals at Bure may have died from the same cause as the seven bovines uncovered at nearby Bourogne (3) and Vellechevreux (4) in France. Regardless of whether the physical evidence in this case is sufficient to confirm a general mortality event, these animals, unlike those found at Shapwick and Luxé, died a considerable distance from the known parameters of the 986–988 plague, to which they may be associated; they very well may have died in another epizootic.[26]

HUMAN–BOVINE PLAGUE DIAGNOSING AND MORBILLIVIRUS MOLECULAR CLOCKS It is advantageous for the purposes of diagnosing both the 569–570 and 986–988 plagues that symptoms and epidemiology/epizootiology can be identified. Indeed, written and possible physical evidence for these mortalities provides good grounds for establishing their causative agents. Traditionally, historians have diagnosed pre-laboratory outbreaks of disease symptomologically, but a symptoms-based approach is not necessarily the best approach; nor is it in every case applicable. As Cohn argued forcefully in his rejection of the bubonic plague diagnosis of the Black Death, a better method, leading to a stronger, though still tentative, disease identification entails consideration of both symptoms and epidemiology/epizootiology. Although early medieval authors rarely commented directly on epizootiology, key properties are often apparent enough. A general understanding of morbidity and mortality, the species susceptible to the pathogen, the relevance of environmental conditions, and the underlying health of victims is often obvious. Rarely evident are the previous pathogen exposures (acquired immunity) or secondary infections (antagonistic or symbiotic disease interactions).[27]

25 Renou, Beauval, and Maury, "Un bilan des connaissances," 135–141.

26 Putelat, "Archéologie des depots animaux," 250–257.

27 Cohn, *The Black Death Transformed: Disease and Culture in Early Renaissance Europe* (New York, 2002).

Epizootiology has proved pivotal for the diagnosing of pre-laboratory epizootics. The tentative RPV identifications of the 809–910 and 1314–1325 bovine plagues hinged on determining the ability of their pathogens to spread rapidly and indiscriminately with regard to seasonal changes or the health of their victims, to achieve high rates of morbidity and mortality, and to attack cattle alone (or predominantly, in the case of the ninth-century outbreak). Existing comments on the 569–570 and 986–988 bovine mortalities, which played down or ignored their zoonotic nature, lean toward RPV on account of the references to diarrhea. But, excusing for a moment concurrent human mortality, the epizootiological properties of these plagues also support an RPV diagnosis: high rates of morbidity and mortality; the targeting of cattle; an ability to disseminate quickly and plausibly without regard for seasonality or environment; and, assuming the Shapwick and Luxé bovines to have died in these events, lethality among even healthy, mature animals. Had people succumbed to a different disease than did cows in 569–570 and 986–988, RPV diagnoses for these plagues would be sturdier than for those of the 809–810 and 1314–1325 panzootics, or the supposed 376–386 pan-European cattle mortality, since fourth-, ninth-, and fourteenth-century sources contain references only to RPV-like symptoms (cows with upset stomachs or cows suffering emaciation).[28]

RPV is always a good candidate for fast-moving, large-scale bovine epizootics of the past that have been attributed with the deaths of many animals. When explicit reference is made to loose bowels in such outbreaks, RPV is the only option known to modern science. Were contagious bovine pleuropneumonia more contagious and mortal, it would be a candidate for those major medieval epizootics for which diarrhea was not an explicit symptom. So would highly infectious and fast-spreading foot-and-mouth dis-

28 For the supposed late fourth-century panzootic, see A. Barton, "Plagues and Contagions in Antiquity," *Journal of the American Veterinary Medical Association*, CXXIX (1965), 505; Clive A. Spinage, *Cattle Plague: A History* (New York, 2003), 47, 82, 88; for a reappraisal, Newfield, "Epizootic Disease in Medieval Europe: Seven Cattle Pestilences and Directions for Future Research," in Thomas and Antikas (eds.), *Animal Disease in Past Human Societies* (forthcoming); for the eighth- and fourteenth-century panzootics, *idem* "Carolingian Panzootic," 200–210; Slavin, "The Great Bovine Pestilence and Its Economic and Environmental Consequences in England and Wales, 1318–50," *Economic History Review*, LXV (2012), 1239–1266; Newfield, "A Cattle Panzootic in Early Fourteenth-Century Europe," *Agricultural History Review*, LVII (2009), 155–190.

ease, were it far more acute then than it is now, though its ability to infect sheep, goats, and pigs—animals rarely identified as victims of epizootics in the early Middle Ages—would have to be acknowledged.[29]

That the 569–570 and 986–988 pathogens were common to people and cattle complicates RPV identifications considerably, since RPV is unique to bovines. The usual diagnosis for a disease that affected both humans and farm animals is anthrax, but it is a highly problematical identification for large disease outbreaks in people and cattle. Although anthrax spores cause acute disease, they are soil- and sometimes arthropod-borne and, as such, dependent on particular environmental and seasonal conditions and incapable of spreading easily between herds and regions. Nor is anthrax particular to humans and cattle: All animals are susceptible to it, though grazers like cows and sheep are the most common victims. With no real pathogenic alternative, it is not surprising that the two plagues assessed herein have been interpreted as both epidemics and epizootics. Treated separately, the bovine deaths are easier to explain. Like the pathogens killing cows, those killing people must have been highly communicable and virulent considering the short time in which they covered so much ground. They also must have been transmitted between like species; pathogens passed easily between humans account best for large rapidly developing epidemics.[30]

Several diseases fit this epidemiology; reported symptoms do little to narrow down the candidates. The 986–988 human victims had fevers, but many pathogens cause a fever. If fever were the most salient symptom, influenza might be suspected. Assuming

29 The pestivirus Bovine Viral Diarrhea Virus, as known to modern science, is a fairly contagious but not especially mortal disease with a 4 to 8% fatality rate. Although its victims suffer diarrhea, it is considered a new virus (emerging in the 1940s), which does "not behave like rinderpest." See Dirk Deregt, "Introduction and History," in Sagar M. Goyal and Julia F. Ridpath (eds.), *Bovine Viral Diarrhea Virus: Diagnosis, Management and Control* (Ames, 2005), 3–34. Foot-and-mouth strains known to modern science kill fewer than 5% of their victims. See William A. Geering and Juan Lubroth, *Preparation of Foot-and-Mouth Disease Contingency Plans* (Rome, 2002), 1, 7, 10–15. For pleuropneumonia, see Geering and William Amanfu, *Preparation of Bovine Pleuropneumonia Plans* (Rome, 2002), 5–9.

30 For anthrax, see World Organisation for Animal Health, *Anthrax in Humans and Animals* (Geneva, 2008), 1–5, 8–17; Spinage, *Cattle Plague*, 85 (though Spinage interprets the 987 Irish mortalities as anthrax [90]); Newfield, "Early Medieval Epizootics," 91–92. Bovine tuberculosis (*Mycobacterium bovis*), another well-known disease of cattle, can infect and kill cows and

that early medieval authors did not universally report every physical expression of a pathogen or even necessarily the principal sign, other diseases causing fever might be considered, such as MV and smallpox. Fever from MV can be in the range of 103 to 105 degrees Fahrenheit, a couple degrees higher than smallpox.[31]

Little else is certain about the disease expression in humans. Indeed, another factor complicating the identification of the 569–570 and 986–988 plagues' causative agents is establishing who suffered what. Did humans have diarrhea along with cows in 569–570 and 986–988? Was variola common to both species in 569–570? Marius is unclear about loose bowels and variola in the sixth century. The *Annals of Tigernach* mentions people suffering colic in 987, though the *Chronicon ex Chronicis* reserves *scitta* for cows. Tentative diagnoses of these plagues founded on the aspects

people, but it is as poor a diagnosis as anthrax. It is a chronic, debilitating disease primarily of cattle but capable of sickening humans and a number of other species, domesticated and wild. In cattle, *M. bovis* is associated principally with production losses (milk and meat), not mortality. Although it can involve the digestive tract and cause "intermittent diarrhea" in both cows and people, this disease expression is rare. The principal symptoms in humans are weight loss and fatigue. Although spread between cattle via the respiratory tract, *M. bovis* reaches humans via the consumption of contaminated dairy products and then spreads from person to person, if pulmonary. But bovine tuberculosis is slow-moving: About 5% of people develop it within two years of exposure; another 5% may develop it later. See Charles O. Thoen et al., "Tuberculosis in Animals and Humans: An Introduction," in *idem*, James H, Steele, and John B. Kaneene (eds.), *Zoonotic Tuberculosis: Mycobacterium Bovis and Other Pathogenic Mycobacteria* (Ames, 2014), 3–5; Anna Rovid Spickler et al. (eds.), *Emerging and Exotic Diseases of Animals* (Ames, 2010), 107–110.

In eighteenth- and nineteenth-century catalogs of ancient and medieval disease outbreaks, the 569–570 and 986–988 plagues, however vaguely understood, were divided commonly into epidemics of people and epizootics of cattle. This tradition persisted into the twentieth century, as is especially evident with regard to the 569–570 plague and works on smallpox. Thomas Short, *A General Chronological History of the Air I* (London, 1749), treated the 986–988 human and bovine mortalities separately, choosing not to diagnosis (91). Jean-Jacques Paulet twice separated the 569–570 deaths of people and cattle. He mentions both of them but discusses only the former in his early study of past epizootics, *Recherches historiques and physiques sur les maladies épizootiques* (Paris, 1775), 73. He overlooks the sick cattle entirely in his treatise on the history of *petite vérole—Historie de la petite vérole I* (Paris, 1768), 78, 85, 87, 93. Robert Willan, *An Inquiry into the Antiquity of the Small-Pox, Measles and Scarlet Fever* (London, 1821), mentions but does not address the 569–570 bovine mortalities in his early study of smallpox (96). Wilhelm Dieckerhoff, *Geschichte der rinderpest und ihrer literatur* (Berlin, 1890), 25, 28, assigns the human and bovine deaths of both 569–570 and 986–988 to different causes. More recently, Stathakopolous, *Famine and Pestilence*, 313–314, considers the 569–570 deaths of cattle and people together but does not advance a diagnosis.

31 Perry and Halsey, "Clinical Significance of Measles," S4; Centers for Disease Control and Prevention, "Measles," 174.

of their symptoms and epidemiology/epizootiology not in doubt—the bovine diarrhea and the plagues' ability to disseminate widely and kill people and cattle rapidly—and molecular clocks recently run on the morbilliviruses MV and RPV indicate a possible solution to the problem. A joint MV–RPV diagnosis of these plagues can untangle Marius' ambiguity, accounting for both people and cows being diarrheal. First-time victims of MV are likely to have a severe reaction to the pathogen, especially if they are undernourished by modern standards (as many early medieval Europeans likely were); under these conditions, diarrhea can be acute enough to cause death. More than 63 percent of MV victims in developing countries before the introduction of the vaccine suffered diarrhea.[32]

On this point, the work of physician Abu Bakr Muhammad ibn-Zakariya Razi (Rhazes) may be significant. Rhazes penned multiple treatises c. 900 that clinically differentiated between *judari* and *hasbah*, diseases read respectively and retrospectively as smallpox and MV before and since the crystallization of either in the laboratory. Assuming the accuracy of these identifications—and a variety of MV was present in postclassical Western Asia—Rhazes' work would illuminate MV's symptoms in the early Middle Ages (a couple decades on either side of 900, and even earlier, given his debt to descriptions of judari and hasbah in the work of seventh-century Aaron of Alexandria).[33]

Though Rhazes wrote mainly about endemic judari and hasbah, observations of diarrhea form a not unimportant part of

32 For diarrhea, malnutrition, and measles in unexposed populations, see Hussey, "Measles," 218, 219, 220; Perry and Halsey, "Clinical Significance of Measles," S6–S7; Centers for Disease Control and Prevention, "Measles," 174; for undernourished early medieval people, Kathy Pearson, "Nutrition and the Early-Medieval Diet," *Speculum*, LXXII (1997), 1–32.

33 Richard Mead and Thomas Stack, who provided the first English translation of Rhazes' Arabic text—*A Discourse on the Smallpox and Measles* (London, 1748), xi–xii, 3, 100, 113–204—understood *judari* and *hasbah* to refer to smallpox and measles. So did Paulet, who first translated Rhazes' text from Latin into French—*Historie de la petite vérole* (Paris, 1768), II, 1–102—and several other eighteenth- and nineteenth-century editions of Rhazes' text (in various languages), such as William Alexander Greenhill, *A Treatise on the Small-Pox and Measles* (London, 1858), 5–9, 22–75. Many later studies agree: Willan, *Inquiry*, 1–2; Moore, *Small Pox*, 4, 56, 113; Hirsch, *Historical Pathology*, 123, 154; Edward J. Edwardes, *A Concise History of Small-Pox and Vaccination in Europe* (London, 1902), 3; Cyril William Dixon, *Smallpox* (London, 1962), 187–188; Hopkins, *Greatest Killer*, 27; Tucker, *Scourge*, 14; Wilkinson, "Virus Concept," 2–3; Ann G. Carmichael and Arthur Silverstein, "Smallpox in Europe before the Seventeenth Century: Virulent Killer or Benign Disease?" *Journal of the History of Medicine and Allied Sciences*, XLII (1987), 147, 151–152.

his work on these diseases, suggesting that loose bowels may have been common to MV during the early Middle Ages regardless of a population's familiarity with the disease. In his treatise on judari and hasbah, Rhazes reports victims of both diseases to have experienced a loosening of the bowels, identifying the diarrhea of hasbah sufferers as far more severe and debilitating than that of judari sufferers. In his *Liber ad Almansorem*, *Constinens* and *Divisio Morborum*, Rhazes repeats that hasbah diarrhea is worse, and in the *Constinens*, he observes that it could cause death. A combined MV–RPV diagnosis can also explain the tagging of the 986–988 plague with *máelgarb*, "bald-rough," in the Irish sources. The focus in Irish texts on this description of afflicted animal hides implies either that diarrhea was not understood there as the primary sign of the disease or that the annalist was concerned principally with the financial repercussions of damaged hides. Like *fluxus intraneorum*, a rough hair coat is typical of RPV, though it is not a classic or distinguishing symptom of the disease.[34]

What is known about variola? The accepted assumption is that the Avenchene bishop understood variola as affecting humans and that his variola was smallpox (*Variola major*). As far as can be told from extant sources, Marius coined the term *variola*, and although Constantinus Africanus, who wrote in the eleventh-century, is considered the first author to use *variola* in specific reference to smallpox, Marius' usage has led many authors since at least 1768 to diagnose a 569–570 epidemic as smallpox. *Variola* has been held to derive from the Latin adjective *varius*—"changing," "varying," or "different." In histories of smallpox, however, the translation is usually "blotchy" or "spotted." It can also derive from the noun *varus*, meaning "spot," "pimple," and, in histories of smallpox, "pustule." The extent to which *variola* was used in reference to a single disease in the early Middle Ages is uncertain. It may have referred to a number of acute and fast-acting illnesses that affected victims' appearance, as *lepra* is held to have been an umbrella term

34 For Rhazes and diarrhea, see Rhazes, *Kitab fi al-jadari wa-al-hasbah*, in Greenhill (trans.), *A Treatise on the Small-Pox and Measles* (London, 1858), XIII, 67–70; *idem*, *Liber ad Almansorem*, *ibid*., X.18.8, 86; *idem*, *Constinens*, *ibid*., XVIII.8.49, 114; *idem*, *Divisio Morborum*, *ibid*., CIL.3 and .7, 92, 94. Attention is also drawn to hasbah-associated diarrhea in two case reports attributed to Rhazes: Max Meyerhof, "Thirty-Three Clinical Observations by Rhazes (circa 900 A.D.)," *Isis*, XXIII (1935), 321–356; XXIV (1936), 343–344; XXVII (1939), 344–345. For RPV and rough hair coats, see Wohlsein and Salik, "Rinderpest," 73.

referring to chronic disfiguring diseases like leprosy and psoriasis. Variola does appear, however, to have been a disease of humans.[35]

As early commentators on the history of smallpox observed, several references to variola occur within a century or two of Constantinus Africanus. Most of these variola sufferers were unknown to the authors who diagnosed them, but all of them were human. The likely mid-twelfth-century *Genealogia comitum Flandriae* depicts Baldwin III dying in 962 of a *morbo variolorum*, and Ekkehard IV of St. Gall in his early eleventh-century *Casus sancti Galli* refers to a *variolam morbus* that attacked the Bavarian bishop Kaminold and was treated by Notker Physicus also in the third quarter of the tenth century. Bishop Ludger (742–809) was reputed to cure two cases of variola, one in Bramsche, north Germany, and the other in 'Ballova,' mentioned in the so-called "second vita" of the saintly Utrechter written in the second half of the ninth century in Werden. In his *Gesta Abbatum Sancti Bertini Sithiensium* of the early 960s, Folcuin reports a young noble's release from a disease that "doctors call variola" in 938 in St. Bertin, northern France. Additionally, the early eleventh-century Frulandus Murbacensis, in his *Passio Leudegarii Episcopi III*, refers to the mid-seventh-century Bishop Leodegar curing the son of a Neustrian Palace Mayor of variola in the eastern French town of Autun. Frulandus implied that variola was well known, though it is uncertain whether he meant in Leodegar's era or in his own.[36]

35 Paulet, *Historie de la petite vérole* I, I, 78, 85, 87, 93; Willan, *An Inquiry into the Antiquity of the Small-Pox, Measles and Scarlet Fever* (London, 1821), 86–105; James Moore, *The History of the Small Pox* (London, 1815), 6–8, 19, 81, 88, 146–147; August Hirsch (trans. Charles Creighton), *Handbook of Geographical and Historical Pathology* (London, 1883), 126; Jon. Hermann Baas (trans H. E. Handerson), *Outlines of the History of Medicine and the Medical Profession* (New York, 1971; orig. pub. 1889), 240; Edwardes, *Concise History*, 3; Dixon, *Smallpox*, 187; Biraben and LeGoff, "La peste," 1493–1494; Jonathan B. Tucker, *Scourge: The Once and Future Threat of Smallpox* (New York, 2001), 2; Donald R. Hopkins, *The Greatest Killer: Smallpox in History* (Chicago, 2002), 25; Devroey, *Economie rurale*, 46; Jennifer Lee Carrell, *The Speckled Monster: A Historical Tale of Battling Smallpox* (New York, 2004), xv; S. L. Kotar and J. E. Gessler, *Smallpox: A History* (Jefferson, 2013), 3–4. Constantinus Africanus' description—*De Omnium Morborum* in *Constantini Africani post Hippocratem et Galenum* (1536), VII.8, 152–153—may fail to convince everyone.

36 D. L. C. Bethmann (ed.), *Genealogia comitum Flandriae*, *MGH SS IX* (Hanover, 1851), 304, 306. Jean de Langhe refers to this work in the largely derivative early portions of his fourteenth-century *Chronicon Sancti Bertini*, but he adds that the disease is called *variola* and *pocca*. See Holder-Egger (ed.), *MGH SS XXV* (Hanover, 1880), XXVIII, 776. Ekkehard IV, *Casus sancti Galli continuatio I*, in G. H. Pertz (ed.), *MGH SS II* (Hanover, 1829), 136;

Other than associating variola with people, these iterations do little to explicate Marius' meaning. Nor is it clear whether these later authors are referring to one disease, let alone *V. major* or *V. minor*. Though Murbacensis, Ekkehard, and the author of the *Ex Vita Sancti Liudgeri* associate variola with *pustulas* (pustules or blisters), bodily *ulcera* (ulcers or sores), *inflatio* (swelling), and *pustulas*-related blindness, all of which may indicate smallpox, only the *Genealogia comitum Flandriae* describes variola as lethal. Since none of these texts suggests that variola was wide-ranging, as Marius' does, *variola* may have signified diseases with different degrees of virulence and communicability, in much the same way that the term *lepra* was not reserved for slow-moving, milder diseases but on occasion applied to plagues that modern historians associate with smallpox.[37]

Unsurprisingly, a few scholars have drifted far from a firm identification of Marius' variola as smallpox. Moore proposed it to be bubonic plague, given the timing of 569–570 plague, which occurred in the midst of several supposed outbreaks of *Y. pestis* and variola's absence from European texts for at least three centuries after Marius. Moore's singular position was that Marius' original manuscript, which, in Moore's view, must have been difficult to decipher and nearly illegible, was probably reworked with a later transcriber's interpolations. More recently, Dixon soberly observed that Marius' variola is simply unidentifiable in lieu of a clinical description of the disease. Wilkinson maintained that neither Marius' variola nor that of other writers before Rhazes can be identified as smallpox for lack of a good clinical description. Assuming that Marius' variola refers to a skin eruption or other visible effect, as the Latin suggests, smallpox remains a possibility, but MV is also a candidate for it.[38]

*Ex Vita Sancti Liudgeri II*, *ibid*., II.29, 424, II.33, 423–424; Folcuin, *Gesta Abbatum Sancti Bertini Sithiensium*, in Holder-Egger (ed.), *MGH SS XIII* (Hanover, 1881), CVI, 628. Frulandus Murbacensis, *Passio Leudegarii Episcopi III*, in B. Krusch (ed.) *MGH SrM V* (Hanover, 1910), XXXI, 360. This variola may not, in fact, belong to the second vita, written c.850, but to the third, written c.890. Altfrid's vita of Ludger, written c.840, the saint's so-called "first vita," does not mention it. Paulet diagnosed Marius' variola as *petite vérole* in 1768 (see n. 35).

37 For smallpox as lepra, see Donnchadh Ó Corráin, "Ireland c.800," 581.

38 Moore, *Small Pox*, 6–8, 19, 81, 88, 146–147; Dixon, *Smallpox*, 190; Hirsch, *Historical Pathology*, 126–127; Lise Wilkinson, "The Development of the Virus Concept as Reflected in Corpora of Studies on Individual Pathogens: 5. Smallpox and the Evolution of Ideas on Acute (Viral) Infections," *Medical History*, XXIII (1979), 2, n. 6.

If Rhazes' judari and hasbah are smallpox and measles, respectively, his guidelines for differentiating between the two may be important. For Rhazes, the difference lay in the severity of nausea (worse in hasbah) and back pain (worse in judari), not the color, degree, or location of any blotchiness, rash, or other skin eruption. In his treatise on the diseases, he addressed judari pustules frequently and depicted hasbah sufferers as pustular (contrary to modern experience with MV), and in his *Liber ad Almansorem*, he identified victims of both diseases as appearing reddish and pustular. In his *Divisio Morborum*, he echoed that judari and hasbah cause pustules but, in accord with modern knowledge, he also noted that hasbah's fever is more severe. This observation occurs again in Rhazes' largely derivative *Constinens*, in which he wrote that that the skin of those who suffered from these diseases turned red, except that the hasbah pustules came "out all at once," "on the surface of the skin," whereas those of judari emerged "gradually" in the form of round eminences. These distinctions concerning the skin are compatible with what is known about smallpox and MV. Though Rhazes thought that physicians could differentiate between the two through the careful study of symptoms, his work portrays these diseases as more similar than distinct. Familiarity with the nuances was clearly required to distinguish between them. Non-professionals, like Marius, with or without previous experience may have confused them, employing a single term for both.[39]

39 Rhazes, *Kitab fi al-jadari wa-al-hasbah*, III.1–2, 34–35; VIII.1–2, 56; IX.1–2, 57–58; XIV.2–4; .7, 7–10, 71–73; *idem*, *Constinens*, XVIII.8.6, 102–103; XVIII.8.19, 105; XVIII.8.47, 113; XVIII.8.62, 119; XVIII.8.65, 119–120; XVIII.8.71, 121; XVIII.8.80, 126; XVIII.8.81, 127; XVIII.8.82, 127; *idem*, *Liber ad Almansorem*, X.18.1, 84; X.18.7, .9, 86; *idem*, *Divisio Morborum*, CIL.1, 90. The distinctions that Rhazes makes concerning the effects of hasbah and judari on the skin seem compatible with smallpox and measles in that their rashes are red and appear facially before spreading out over the body, but a smallpox rash gradually blisters and becomes pustular whereas a measles rash remains flat. That said, measles' rash is often characterized as maculopapular, and the papules emanating from the macules are very occasionally referred to as pustules. See, for example, Alpay Azap and Filiz Pehlivanoglu, "Measles," in Onger Ergonul et al. (eds.), *Emerging Infectious Diseases* (London, 2014), 347, 351, 353; Ludvig Hektoen, "Experimental Measles," *Journal of Infectious Disease*, II (1905), 239, 241. Some have had no difficultly accommodating Rhazes' description of hasbah (measles) as pustular. See S. C. Ashtiyani and A. Amoozandeh, "Rhazes Diagnostic Differentiation of Smallpox and Measles," *Iranian Red Crescent Medical Journal*, XII (2010), 481. Closer attention may be required regarding Rhazes' pustule terminology in the original Arabic.

Neither measles nor smallpox are zoonotic. The relationship between smallpox and cowpox, closely related orthopoxviruses, was essential to Edward Jenner's late eighteenth-century smallpox vaccine, but cowpox, though zoonotic and transferable between humans and cattle, is neither a rapidly spreading disease nor an acute disease in humans or cattle. Cowpox also affects numerous other animals, from rats to rabbits, cats, and horses. A joint smallpox–cowpox ancestor is therefore a bad fit with the epidemiology/epizootiology of the 569–570 and 986–988 plagues. In any case, recent molecular clocks determined that smallpox colonized humans many thousands of years ago, and comparisons made between gene sequences in several studies now show that smallpox has little in common with cowpox. Its similarity to taterapox and camelpox indicates that it evolved from one of those viruses or emerged independently from an ancestral poxvirus with them. Had an ancestral cowpox–smallpox orthopoxvirus ever existed—a proposition that is highly unlikely on firm biological grounds—it would have been a bad fit for the 569–570 and 986–988 plagues symptomologically as well. The only orthopoxvirus known to cause diarrhea in its victims is monkeypox, diarrhea being a secondary complication of that mild and rare disease, which also afflicts rodents and humans.[40]

Like smallpox, MV spreads between humans and requires a minimum population in the hundreds of thousands to become endemic and survive. Contact between a human population with the disease and another without it (in other words, between disease pools) results in an epidemic and, in a sufficiently large new population, a new endemic focus. Scholars have long surmised

40 Sergei N. Shchelkunov et al., *Orthopoxviruses Pathogenic for Humans* (New York, 2005), 2, 160, 162, 193–248; Yu Li et al., "On the Origins of Smallpox: Correlating Variola Phylogenics with Historical Smallpox Records," *Proceedings of the National Academy of Sciences*, CIV (2007), 15787–15792; Austin L. Hughes et al., "The Evolutionary Biology of Poxviruses," *Infection, Genetics and Evolution*, X (2010), 50–59; Jessica M.C. Pearce-Duvet, "The Origin of Human Pathogens: Evaluating the Role of Agriculture and Domestic Animals in the Evolution of Disease," *Biology Review*, LXXXI (2006), 371–373. William H. McNeill, *Plagues and Peoples* (New York, 1976), 45, and Jared Diamond, *Guns, Germs, and Steele: The Fates of Human Societies* (New York, 1997), 204–207, among others, speculated that smallpox diverged from cowpox. Although smallpox is the notable exception, many poxviruses exhibit "highly diverse host ranges" and "infect a broad spectrum of animals." See Sherry L. Haller et al., "Poxviruses and the Evolution of Host Range and Virulence," *Infection, Genetics and Evolution*, XXI (2014), 15.

that MV and its morbillivirus cousin RPV diverged in antiquity c. 3000 B.C.E. on the western steppes and that post-divergence MV established an endemic focus in Western Asia (ancient Sumer), where a human population could support its survival. The supposed morbillivirus "archaevirus" RPV was thought to have evolved c. 8,000 to 10,000 years ago on the central or eastern steppes in large herds of bovines. MCAs run recently on MV and RPV, however, dispute this theory.[41]

That a diagnosis of a MV–RPV predecessor can be considered for the 569–570 and 986–988 plagues is based on two independent morbillivirus MCAs conducted in Sendai and San Diego to determine when measles diverged from rinderpest. Divergence dates refer to the time of the most recent common ancestor, the latest point at which, in this case, the current genetic diversity of MV and of RPV were united as one genotype. The morbillivirus MCAs employed the rate of MV and RPV's recent evolutions, their known molecular distancing, to estimate their date of separation, proceeding on the assumption, following from the neutrality theory of molecular evolution, that the rate of evolution is constant. Because rates of evolution vary, however, relaxed clock models, used in both morbillivirus studies, have been developed, allowing varying rates of evolutionary progression along different phylogenetic branches. Nonetheless, these analyses provide only rough diversification times, since they are restricted to modeling recent evolutionary history that may not be entirely applicable to the distant past in the case of rapidly evolving viruses. The first MCA assigned MV and RPV's most recent common ancestor to the eleventh

41 For the old paradigm, see Barrett and Paul B. Rossiter, "Rinderpest: Impact on Humans and Animals," *Advances in Virus Research*, LIII (1999), 93–94; Barrett, "Rinderpest and Distemper Viruses (*Paramyxoviridae*)," in Allan Granoff and Robert G. Webster (eds.), *Encyclopedia of Virology* (San Diego, 1999), III, 1559, 1563–1564; Diane E. Griffin, "Measles Virus," in David M. Knipe et al. (eds.), *Fields Virology* (Philadelphia, 2001), 1401; Peter L. Roeder and William P. Taylor, "Rinderpest," *Veterinary Clinics of North America: Food Animal Practice*, XVIII (2002), 516; Andrew Cliff, Peter Haggett, and Matthew Smallman-Raynor, *World Atlas of Epidemic Diseases* (Boca Raton, 2004), 42–43 (with maps); Ashley C. Banyard, Bertus K. Rima, and Barrett, "The Morbilliviruses," in Barrett, Paul-Pierre Pastoret, and Taylor (eds.), *Rinderpest and Peste des Petits Ruminants: Virus Plagues of Large and Small Ruminants* (Amsterdam, 2006), 23; Stephen C. Stearns and Jacob C. Koella, *Evolution in Health and Disease* (New York, 2008), 169; Peter Roeder, Jeffrey Mariner, and Richard Kock, "Rinderpest: The Veterinary Perspective on Eradication," *Philosophical Transactions of the Royal Society*, CCCLXVIII (2013), 1; Azap and Pehlivanoglu, "Measles," 348.

and twelfth centuries C.E. The second one pushed it back to the ninth or tenth centuries C.E., estimating the morbilliviruses' rate of evolution to be faster.[42]

This difference of a century or two in ancestry may be notable on historical grounds, but the general agreement between the two independent studies that MV and RPV separated postclassically is more significant. Without the independent confirmation of the second MCA, however, this conclusion, which contradicts the firmly held theory that MV first appeared millennia before the Common Era, might have been suspect. This article, which proposes that the 986–988 plague was an outbreak of an MV–RPV forerunner, prefers a separation no earlier than the late tenth century, roughly in agreement with both molecular studies, though it does not suppose that historical evidence for disease be given precedence over molecular data.

It is not known where the divergence occurred. The first molecular study stipulates only that MV evolved from RPV "in an environment where cattle and humans lived in close proximity." Were the 569–570 and 986–988 plagues an MV-RPV ancestor, the separation probably would not have happened in Europe, since these plagues caused considerable mortality in European cattle populations; before divergence, the ancestral morbillivirus was first confined to bovines; and pathogens do not typically cause large sudden die-offs in the same population within which they evolved, spent considerable time, and were endemic/enzootic. This article holds the origins of the 569–570 and 986–988 plagues, and the divergence, to have been extra-European. Considering the sizable human and bovine populations required for endemic MV

42 For MV–RPV MCAs, see Furuse, Suzuki, and Oshitani, "Origin of Measles Virus," 1–4; Joel O. Wertheim and Sergei L. Kosakovsky Pond, "Purifying Selection Can Obscure the Ancient Age of Viral Lineages," *Molecular Biology and Evolution*, XXVIII (2011), 3355–3365; for molecular clocks, Francisco Rodriguez-Trelles, Rosa Tarrio, and Francisco Ayala, "Molecular Clocks: Whence and Whither?" in Philip C. J. Donoghue and M. Paul Smith (eds.), *Telling the Evolutionary Time: Molecular Clocks and the Fossil Record* (London, 2005), 5–26; Philippe Lemey and David Posada, "Molecular Clock Analysis," in Lemey, Marco Salemi, and Anne-Mieke Vandamme (eds.), *The Phylogenetic Handbook: A Practical Approach to Phylogenetic Analysis and Hypothesis Testing* (2009), 362–372. If the second morbillivirus MCA underestimated evolutionary rates, and the divergence were pushed slightly deeper in time, the 986–988 plague may be evidence of the temporary co-existence of an ancestral morbillivirus with distinct measles and rinderpest viruses.

and enzootic RPV, respectively, the separation probably took place in Asia, as has long been suspected.[43]

POSSIBLE CONSEQUENCES OF THE PLAGUES Like most of the early medieval accounts of epidemics and epizootics in Europe, the sources for the 569–570 and 986–988 plagues say nothing about short- or long-term consequences beyond a brief mention of inflated dairy prices after 987 in what is likely a late version of the Welsh *Brut*. There are also no means, direct or indirect, to measure sudden or short-term changes in human or bovine population levels across the areas affected. Nonetheless, the vast spatial parameters, high mortality, and concurrent attack on people and cattle of the 569–570 and 986–988 outbreaks probably translated into considerable short-term, and weaker long-term, demographic and economic consequences.[44]

If an ancestor to MV and RPV were indeed to blame, outbreak parameters larger than those that the written sources and bovine graves suggest would be likely, due not only to the highly contagious nature of these viruses (75 to 90 percent of those in contact with MV victims become infected) and the possibility of an ancestral virus passing between species (therefore more easily disseminated) but also to the pathogen's extra-European origin. MV and RPV are density-dependent diseases requiring large numbers for endemicity/enzooticity. MV demands an urban population of at least 250,000 or a series of well-connected smaller centers to become endemic. It could have become established in Rhazes' populous Western Asia, rather than in early medieval Europe, which was predominantly rural, with few towns surpassing a population of 10,000.

43 Furuse, Suzuki, and Oshitani, "Origin of Measles Virus," 1. Griffin, "Measles Virus," previously located the MV–RPV divergence "where cattle and humans lived in close proximity" (1401). For an Asiatic divergence, see "old paradigm" works cited in n. 41. That the eighteenth-century bovine panzootics, thought to be RPV, spread westward into Europe also indicates that MV and RPV did not separate in Western Europe. For the comparatively vast scholarship on these cattle plagues, see Spinage, *Cattle Plague*, 103–150, 241–262. The Shapwick and Luxé bovines may provide further evidence for an extra-European origin, since in enzootic zones, RPV typically affects young cattle after their maternal immunity dissipates, not mature cattle. See Anderson, Barrett, and Scott, *Diagnosis of Rinderpest*, 6–7, 10; Anonymous, "Rinderpest," 543.

44 *Brut y Tywysogion* in Cambrian Archaeological Association, *Archaeologia Cambrensis* (London, 1864), X, 36–37.

Foci of the ancestral virus may be suspected on the steppes or possibly in Western Asia but not further west.[45]

A zoonotic ancestor to MV and RPV, given what is known about each of them now, would also have caused significant population loss in naïve or unexposed human and bovine populations; 20 to 30 percent of infected people and 80 to 95 percent of infected cattle may have died. Marius' particular emphasis on the magnitude of the bovine losses in 569–570 may be interpreted as evidence of such uneven mortality between the species. The qualification of the 986–988 cattle mortality but not the corresponding human mortality as "great" in the *Annals of Tigernach* also suggests that bovine losses surpassed human losses at that time, as does the silence in the *Anglo-Saxon Chronicle*, *Brut,* and *Saltair na Rann* regarding human deaths. The simultaneous loss of people and cattle would have had significant repercussions—deaths in one population compounding the demographic and economic effects of deaths in the other. Though human fatalities might have diminished the impact of concurrent bovine fatalities (reducing apace the demand for the products and services that cattle provided), the unequal mortality of a combined morbillivirus might have offset any collapse in demand for draft, fertilizer, milk, meat, and hides.[46]

45 Populations of 250,000, 300,000, and 500,000 are commonly cited for endemic measles. See Andrew D. Cliff and Peter Haggett, "Global Trends in Communicable Disease Control," in Norman Noah and Mary O'Mahony (eds.), *Communicable Disease Epidemiology and Control* (Chichester, 1998), 24; Lauren Sompayrac, *How Pathogenic Viruses Work* (Mississauga, 2002) 31; Cliff and Matthew Smallman-Raynor, *Oxford Textbook of Infectious Disease Control: A Geographical Analysis from Medieval Quarantine to Global Eradication* (New York, 2013), 14. In the early Middle Ages, Rome, Europe's largest city, possessed c. 25,000 people. See Adriaan Verhulst, *The Carolingian Economy* (New York, 2002), 106.

46 For measles' communicability, see Perry and Halsey, "Clinical Significance of Measles," S4; Hussey, "Measles," 216; for mortality rates, D. Nanche, "Immunology of Measles Virus Infection," in Griffin and Michael B. A. Oldstone (eds.), *Measles: Pathogenesis and Control* (Berlin, 2009), 153, 163; Peter Wohlsein and Jeremiah Salik, "Rinderpest and Peste des Petits Ruminants—The Disease: Clinical Signs and Pathology," in Barrett, Pastoret, and Taylor (eds.), *Rinderpest and Peste des Petits Ruminants*, 69. Endemicity and vaccination programs have made measles a childhood disease. As Robert Sallares, *The Ecology of the Ancient Greek World* (Ithaca, 1991), 249–252, pointed out, without endemic foci, severe attacks of MV would not have been restricted to children in postclassical Europe. Large mortalities have been attributed to the introduction of measles into previously unexposed populations. For example, in 1875 the virus killed 25 to 30% of Fiji's 150,000 inhabitants within six months. See R. A. Derrick, "Fiji's Darkest Hour: An Account of the Measles Epidemic of 1875," *Transactions and Proceedings of the Fiji Society*, VI (1959), 3–16; Cliff, Haggett, and Smallman-Raynor, *Measles: An Historical Geography of a Major Human Viral Disease from Global Expansion to Local Retreat, 1840–1990* (New York, 1993). Since pathogen virulence is not fixed, an ancestral RPV could

Outbreaks of disease common to people and cattle on this scale could have affected early medieval populations and their organic agricultural economies for a generation, especially in more densely populated areas. The 569–570 plague, alongside the mid-sixth-century Justinianic plagues, would have contributed to the contraction of southern European settlement and arable land already then underway. The 986–988 event, like other ninth- and tenth-century mortality crises, would have temporarily undone or stalled settlement and arable expansion initiated in many northwestern European regions long before the 980s. Compared to bubonic plague, which can kill more than 50 percent of its victims, a MV–RPV predecessor may have been mild in humans, with a mortality rate like that of MV alone. Yet more people would have contracted it than any rodent-based *Y. pestis* had it been, like MV or RPV, airborne, easily spread between species, and infectious days before severe disease expression.[47]

High human morbidity may be expected in small but congested early medieval settlements. A rough idea of the possible mortality in affected towns is obtainable by superimposing measles' virulence on virgin populations. If 25 percent of a settlement with a population of 5,000, roughly the size of mid-sixth-century Ravenna and mid-tenth-century London, were to come into contact with a pathogen as virulent as that proposed herein, 250 to 375 deaths would ensue. If 75 percent were infected, the number of fatalities would be in the range of 750 to 1,125. Considering the high infectivity of MV and RPV, morbidity rates less than 50 percent seem unlikely for a pathogen that disseminated efficiently among people.[48]

have been more acute in human populations than measles is in pristine populations now. Increases in virulence are known to accompany the spread to new species. See Andrew D. Morgan and Britt Koskella, "Coevolution of Host and Pathogen," in Michel Tibayrenc (ed.), *Genetics and Evolution of Infectious Diseases* (London, 2011) 149. A source for the continental 989–990 human–bovine plague reported that the bovine mortality was greater than the human mortality, further implying that this die-off was tied to the insular plague of 986–988. See *Annales Quedlinburgenses*, 447.

47 World Health Organization, "Plague," Fact Sheet, 267, available at www.who.int/mediacentre/factsheets/fs267/en/ (accessed November 1, 2014).

48 For Ravenna and London's postclassical populations, see Deborah M. Deliyannis, *Ravenna in Late Antiquity* (New York, 2010), 322, n. 42; John McDonald, *Production Efficiency in Domesday England, 1086* (London, 1998), 52.

Using the same incident percentages and mortality rates, smaller, more representative towns of 1,000 may have seen between 50 deaths (25 percent infected, 20 percent killed) and 225 deaths (75 percent infected, 30 percent killed). If 90 percent were exposed, 270 deaths might be expected. Town industries and markets would have suffered correspondingly. Lower morbidity rates and fewer deaths in humans would have accrued in the countryside, though a decline in agricultural activity in the wake of a combined human–bovine plague is nevertheless certain. Labor-intensive sectors, such as arable farming, possibly would have suffered considerably in the short-term. Since fixed inputs of human and animal labor were required to keep agrarian systems running, mortality could have depleted such inputs sufficiently to disrupt production. At least in some areas hit by the 569–570 pestilence, viticulture, with its heavy labor requirements, would also have been susceptible. Additionally, an ancestral morbillivirus may have blinded some human survivors. Severe keratitis and corneal scarring from MV is a common source of blindness in untreated and malnourished populations.[49]

Because animal rearing was more prominent than arable agriculture during the sixth century than during the tenth, even in Ireland where cattle raising very much dominated before the ninth century, the 569–570 and 986–988 cattle mortalities did not affect food supplies uniformly. Specialized, large-scale, late Roman cattle rearing, primarily north of the Alps (in areas of northern France, Belgium, Germany, the Netherlands, and Britain), as well as in some cisalpine and southerly transalpine regions (see, for example, Cassiodorus' remarks on the *Calabri peculiosi* and his mentions of cattle droves emanating from Italy's south, and some late Roman bovine-dominated Provençal animal-bone assemblages), generally gave away in the sixth and seventh centuries to mixed animal husbandry. But bovines, although smaller, remained prominent in most early medieval agrarian regimes, essential as they were for draft power and fertilizer. Zooarchaeology suggests that the number of cattle increased in late rather than early Anglo-Saxon

49 For measles and blindness, see Perry and Halsey, "Clinical Significance of Measles," S7–S8; for the Justinianic plague of 571, see Gregory of Tours, *Liber de passione et virtutibus sancti Iuliani martyris*, in Bruno Krusch (ed.), *Monumenta Germaniae Historica Scriptores rerum Merovingicarum* (Hanover, 1885), I.2,XLVIa, 131–132; Marius, *Chronica*, 238.

England in part because of an extension of cultivation and greater demand for traction as the amount of arable land multiplied.[50]

Some regions were more bovine-centric than others, but the 569–570 and 986–988 plagues seem to have spread whether or not a region was primarily given to animal rearing or to arable land. Severity of impact, however, would have fluctuated regionally, even locally, corresponding to the degree of reliance on cattle for traction (arable agriculture, transportation, and warfare); for fertilizer (arable agriculture); for milk and meat (diet, trade, and warfare); and for bone, hide, and horn (industry and trade); as well as on the ability to replace or replenish cattle via the market. In general, bovine fatalities likely had worse repercussions for human health in the ninth century because of a generally less varied diet and a greater reliance on cattle for draft and fertilizer. Where animal rearing was dominant and diversified, cattle losses were easier to repair and stocks of sheep and pigs easier to augment as compensation for lost bovine milk and meat, despite presumably higher morbidity and mortality rates. DeWitte and Slavin argued that dramatic declines in milk, meat, and grain following the 1314–1325

50 For Roman and early medieval cattle, see Anthony King, "Diet in the Roman World: A Regional Inter-Site Comparison of the Mammal Bones," *Journal of Roman Archaeology*, XII (1999), 169–171, 174, 176–180, 182, 190; Geoffrey Kron, "Archaeozoological Evidence for the Productivity of Roman Livestock Farming," *Münstersche Beiträge zur Antiken Handelsgeschichte*, XXI (2002), 55, 57, 59–64; Annie Grant, "Domestic Animals and Their Uses," in Malcolm Todd (ed.), *A Companion to Roman Britain* (London, 2004), 374–377, 381, 382, 384, 387; Timothy Howe, "Domestication and Breeding of Livestock: Horses, Mules, Asses, Cattle, Sheep, Goats, and Swine," in Gordon Lindsay Campbell (ed.), *The Oxford Handbook of Animals in Classical Thought and Life* (New York, 2014), 104; Vianney Forest and Isabelle Rodet-Belarbi, "À propos de la corpulence des bovines en France Durant les périodes historiques," *Gallia*, LIX (2002), 291–295, 298; Tamara Lewit, "Pigs, Presses and Pastoralism: Farming in the Fifth to Sixth Centuries AD," *Early Medieval Europe*, XVII (2009), 79, 80; Benoît Clavel and Jean-Hervé Yvinec, "L'archéozoologie du moyen âge au début de la période moderne dans la moitié nord de la France," in Jean Chapelot (ed.), *Trente ans d'archéologie médiévale en France: Un bilan pour l'avenir* (Caen, 2010), 76–77, 80; N. J. Sykes, "From *Cu* to *Sceap* to *Beffe* and *Motton*: The Management, Distribution, and Consumption of Cattle and Sheep in Medieval England," in Christopher M. Woolgar, Dale Serjeantson, and Tony Waldron (eds.), *Food in Medieval England: Diet and Nutrition* (New York, 2006), 57–58; Terry O'Connor, "Livestock and Deadstock in Early Medieval Europe from the North Sea to the Baltic," *Environmental Archaeology*, XV (2010), 1, 2, 6–11, 13; *idem*, "Livestock and Animal Husbandry in Early Medieval England," *Quaternary International*, CCCXLVI (2014), 109–115, 109–118; Finbar McCormick, "The Decline of the Cow: Agricultural and Settlement Change in Early Medieval Ireland," *Peritia*, XX (2008), 210, 214–217, 219–221; *idem*, "Agriculture, Settlement and Society in Early Medieval Ireland," *Quaternary International*, CCCXLVI (2014), 119–130; Ó Corráin, "Ireland *c.*800," 568–574; Cassiodorus (trans. Thomas Hodgkin), *Variae* (London, 1886), III.50, 225; VIII.33, 382; XI.39, 484; XII.12, 499.

panzootic resulted in shortages of calcium, protein, and vitamin B12, heightening the vulnerability of people to disease for more than a decade. A similar situation may have prevailed after 569 and 986.[51]

In arable areas, the loss of grain must have been severe. Slavin's assessment of fourteenth-century English manorial data reveals that equine-rich landlords were able to maintain their arable acreage after the 1314–1325 panzootic by increasing the number of horses employed as draft animals by about 40 percent. Other land owners were less fortunate. Winchester's estates, for example, tilled about 18,000 acres before that bovine plague crossed the Channel in 1319 but only 9,000 in 1321 after it had claimed roughly 62 percent of the national herd (more than 1 million cattle). Oxen were the draft animals of choice in the areas hit by the 569–570 and 986–988 plagues. The relative unavailability of equines as a traction substitute implies arable contractions more severe and long-lasting than in the 1300s.[52]

The evidence does not indicate that early medieval bovine mortalities triggered severe food shortages by themselves. Cattle plagues contributed most often to production failures and famines generated by extreme weather and short-term climatic anomalies. In lieu of additional environmental shocks, the threat of widespread food shortage that the loss of draft animals and fertilizer, and the corresponding contraction of arable land, posed does not appear to have materialized in 569–570 or 986–988. Only Wales recorded a subsistence crisis that could correspond to the tenth-century pestilence. The simultaneous deaths of people and cattle in many early medieval plagues is another probable explanation. Indeed, the excess human mortalty may have curbed demand for grain enough to prevent famine, even though bovines were possibly 75 percent less likely than humans to survive infection.

51 DeWitte and Slavin, "Between Famine and Death," 37–60.

52 For the consequences of the 1314–1325 panzootic, see Slavin, "Great Bovine Pestilence," 1241–1266; *idem*, "The Fifth Rider of the Apocalypse: The Great Cattle Plague in England and Wales and Its Economic Consequences, 1319–1350," in Simonetta Cavaciocchi (ed.), *Le interazioni fra economia e ambiente biologico nell'Europa preindustriale, secc. XIII–XVIII: Proceedings of the 41st Study Week of the Fondazione Istituto Internazionale di Storia Economica "F. Datini"* (Florence, 2010), 171–181; for the importance of oxen in early medieval agrarian regimes, Verhulst, *Carolingian Economy*, 67; Banham and Faith, *Anglo-Saxon Farms*, 86, 108–110.

Nevertheless, the loss of essential human and animal labor would have heightened vulnerability to dearth in arable regions.[53]

The value of milk, meat, and hides surely inflated after the 569–570 and 986–988 plagues, as the possibly corrupt version of the *Brut* reports and as they did in the early fourteenth century. Meat and hides of diseased animals might have been salvaged, although we do not know for sure. The Irish annals suggest that hides were damaged, and the available zooarchaeology implies that hides and meat were not taken from plague cattle. If they were, they may have served to spread the disease while fresh. The recovery of urban and rural communities, agricultural production, and especially bovine stocks would have taken considerable time. As Gregory of Tours noted in his account of a bovine mortality in 591, cattle plagues severely impeded breeding and replenishment. Five years after the 1314–1325 panzootic, only 9 percent of English manors had restocked. Not until the Black Death was the seigniorial herd in England restored, largely via farm transfers, markets (which were less numerous and interconnected in most regions affected in 569–570 and 986–988), and natural regeneration.[54]

53 For cattle plagues and food shortage in the early Middle Ages, see Newfield, "The Contours, Frequency and Causation of Subsistence Crises in Carolingian Europe (750–950)," in Benito I. Monclús, *Crisis Alimentarias en la Edad Media: Modelos, Explicaciones y Representaciones* (Lleida, 2013), 133, 166. Subsistence crises reported in Wales in 988/89 and 993/94 may attest to heightened susceptibility to shortage following the 986–988 plague (*Brut y Tywysogyon:* Peniarth MS. 20, 10), though the pestilence may have caused the first of these two crises.

54 Meat was plentiful after plagues for those willing to eat it. Early medieval annalists and chroniclers are quiet on the consumption of plague cattle, but the zooarchaeology addressed above suggests that sick animals were not always eaten. For biblical restrictions on eating carrion in the early Middle Ages, possibly indicating that people ate diseased domesticates, see Rob Meens, "Pollution in the Early Middle Ages: The Case of Food Regulations in Penitentials," *Early Medieval Europe* IV (1995), 3–19. High medieval sources suggest that the meat of bovine victims of acute disease was often salvaged. See Newfield, "Epizootics and the Consumption of Diseased Meat in the Middle Ages," in Francesco Ammannati (ed.), *Religione e istituzioni religiose nell'economia Europea, 1000–1800: Proceedings of the 43rd Study Week of the Fondazione Istituto Internazionale di Storia Economica "F. Datini"* (Florence, 2012), 619–639, n. 11; also see Putelat, "Archéologie des depots animaux," 266. RPV could spread for a few days postmortem via trade in fresh hides (untreated and undried) and fresh meat. The movement of these goods is considered to possess considerably less potential for virus dissemination than the movement of live cattle. See Spinage, *Cattle Plague*, 16–17; Obi, Roeder, and Geering, *Rinderpest Contingency Plans*, 5; Anderson, Barrett, and Scott, *Diagnosis of Rinderpest*, 7; R. G. Bengis, "Animal Health Risks Associated with the Transportation and Utilisation of Wildlife Products," *Revue scientifique et technique de l'OIE*, XVI (1997), 104, 106. For Gregory of Tours' remarks, see his *Libri Historiarum X*, in Krusch (ed.), *Monumenta Germaniae Historica Scriptores rerum Merovingicarum* (Hanover, 1937), I, X.30, 525.

Natural regeneration was the most common and the most prolonged method of herd replenishment during the early Middle Ages. Heifers gestate for nine-and-a-half months and rarely deliver multiple births; sexual maturation takes at least fourteen months. The wait for optimum milk production is about twenty-four months, and raising an ox and training it to plow can take years. Moreover, cattle surviving acute disease would have been weak, malnourished, and, in the short run, unlikely to become pregnant. Pregnant heifers with an ancestral MV–RPV may have aborted, as those suffering RPV often do. Subsequent mortality events would have slowed recovery further, especially in the sixth century, for bovine and human populations (during the cattle plagues of 583–584 and 591 in France and with the Justinianic plague of 571 in France and Italy, among later sixth-century visitations). Survivors of the 569–570 and 986–988 plagues possibly benefited from immunity to re-infection, however, as morbilliviruses are known to confer life-long protection. A low level of temporary immunization against MV and RPV is passed maternally as well. Yet, without endemic foci or regular exposure to the ancestral virus, populations would have been left vulnerable to re-infection and similar deadly events caused by the same disease within two generations.[55]

REMAINING QUESTIONS There are molecular, textual, and possibly zooarchaeological grounds for suspecting that a now-extinct MV-RPV morbillivirus occasionally erupted in early medieval Europe, taking a considerable demographic toll in the human and bovine populations before burning out. The plagues of 569–570 and 986–988 may provide historical substance to the laboratory-detected postclassical divergence of MV and RPV, but many questions remain regarding the occurrence and epidemiology/epizootiology of that virus before divergence. Whether the virus was spread directly and easily among people and among cows, as well as across species, matters a great deal for the estimation of its plausible impact, as does the virus' mortality rate in humans and bovines. Does the emphasis in the sources for the

55 For RPV and aborted pregnancies, see Wohlsein and Salik, "Rinderpest," 71; for MV, RPV, and immunity, Nanche, "Immunology of Measles," 151, 160, 162–164; S. Louise Crosby, Chieko Kai, and Kazuya Yamanouchi, "Immunology of Rinderpest—An Immunosuppression but a Lifelong Vaccine Protection," in Barrett, Pastoret, and Taylor (eds.), *Rinderpest and Peste des Petits Ruminants*, 196–197; Wohlsein and Salik, "Rinderpest," 69.

569–570 and 986–988 events on bovine deaths indicate that the pathogen was neither as virulent nor as widespread in people as it was in cattle? If the virus were much more lethal for cattle than people, as proposed herein, we would have an explanation for why certain early medieval plagues, like those discussed herein and that of 809–810, appear in some sources as zoonotic but in most sources as epizootic.

A virus capable of jumping species would explain why some early medieval bovine plagues—that of 809–810 especially—could disseminate over a large area of Europe more quickly than could the fourteenth- and eighteenth-century (post-divergence) European cattle panzootics thought to be RPV. That the best candidate for the 809–810 panzootic is RPV raises another crucial issue—the temporary co-existence of an ancestral MV–RPV with distinct MV and RPV varieties. Does a c.1000 C.E. MV–RPV divergence negate MV diagnoses of ancient Anthenian, Antonine, and Cyprian plagues (however unpopular those disease identifications may now be), the reading of Rhazes' hasbah as MV, or interpretation of early medieval Japanese epidemics as MV? Was a distinct MV virus endemic in postclassical Western Asia, as historians have long argued, and was an MV–RPV forerunner located somewhere else from where it erupted into Europe? Could MV have diverged before 1000, spread into Europe, but failed to establish endemicity there or elsewhere?[56]

56 Possibly of note, Orosius (trans. A. T. Fear), *Seven Books of History against the Pagans* (Liverpool, 2010), VII.27.10, 367, reports that the Cyprian plague claimed people and cows (*jumenta* and *pecuda*); Carl Zangemeister (ed.), *Historiarum adversus Paganos Libri VII* (Leipzig, 1889), VII.27.10, 269. An earlier passage of interest comes from the Roman historian Livy. In the context of a drought in Italy in 428 B.C.E, which was purportedly severe enough that cattle died of thirst, diseases—in particular, one identified as *scabies*—began in bovines and spread to humans via interspecies contact (*volgatique contactu in homines morbi*). "Country people and slaves" were infected first, but the disease eventually spread to "the city." See Livy (trans. B. O. Foster), *Ab Urbe Condita Libri II* (Cambridge, Mass., 1967), IV.30.7, 354–355. Dionysus of Halicarnassus' account differs slightly. He reported cattle and sheep dying on account of the drought, but he connects the mortality in domesticates to the mortality in humans less explicitly. He specifies that the disease in humans caused "dreadful pains in the skin with its itchings and in case of any ulcerations raged more violently than ever, a most pitiable affliction and the cause of the speediest of deaths." See Dionysus of Halicarnassus (trans Earnest Cary), *Roman Antiquities VII* (London, 1950), XII.6, 216–217. In light of the morbillivirus MCAS, Jennifer Manley, "Measles and Ancient Plagues: A Note on New Scientific Evidence," *Classical World* 107 (2014), proposes that measles can no longer be considered the cause of the Athenian or Antonine plagues (393–397), though she does not speculate about what the new scientific evidence could mean for the history of pre-divergence zoonotic plagues.

Many intricacies in MV and RPV's history still need to be untangled. As scientists attempt to unravel the molecular history, they would do well to work with historians when suspected divergences are set in historical time. Overlooked written sources for disease or re-appraisals of well-known ones can lend support to estimated divergence dates, as they do in this article. For their part, historians of postclassical and earlier Europe would do well to engage scientific studies to understand the pathological realities underlying the written accounts and the archaeology of acute disease. Such interdisciplinary work will lead to a better understanding of the evolution and geography of pathogens. Integral to all this activity, and so far missing, is palaeomicrobiology for the ancestral species of modern pathogens. Extracting and analyzing the necessary material from potential specimens in archaeological contexts may require the further development of biomolecular techniques. However, without palaeomicrobiological study, connections made between molecular clocks and written and archaeological evidence for acute disease remain speculative.

The tentative connections advanced in this article represent a new approach to the history of disease and the impact of disease on people and their animals. For periods not endowed with copious written sources, like the early Middle Ages, molecular clocks are particularly useful, providing grounds for more detailed assessments of the origins, parameters, and consequences of plagues. MCAs allow for dynamic histories of disease and for the inclusion of past inter-species disease outbreaks—events of special interest given today's "emerging diseases." Indeed, this article suggests that an ancestral MV–RPV morbillivirus episodically colonized and spread in human populations, as have the SARS Coronavirus and Ebola Disease Virus in recent years. MCAs not only reveal that people and animals in the distant past faced a different assortment of pathogens than people and animals do now, such as an MV–RPV ancestor, but also that people long ago were confronted by new diseases, such as MV, that are now familiar.[57]

57 The emergence of the ebola disease virus may have been early medieval. See Y. H. Li and S. P. Chen, "Evolutionary History of Ebola Virus," *Epidemiology and Infection*, CXLII (2014), 1140, 1142.