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Catching the plague: New insights into the transmission of Early Modern plague

(working paper)

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Since Alexandre Yersin cultured the bacillus of the subtropical rodent disease bubonic plague, now called after him, *Yersinia pestis*, historians and scientists have assumed that this disease was the same as the Justinianic plague of 541 A.D. and the Black Death of 1348 with its successive waves of pestilence to the nineteenth century. Recently, however, historians and scientists have begun to question the identity of the 'three pandemics of plague'. Scholars have pointed to striking differences especially between the Black Death disease of the late Middle Ages and early modern period, on the one hand, and the pandemic of bubonic plague that spread globally at the end of the nineteenth century, on the other. While the Black Death disease could occur at any time of year, the seasonality of *Yersinia pestis* (with few exceptions) is determined by the fertility cycle of rat fleas and thus occurs predictably in warm and humid conditions, generally within temperature bands between 10º and 20º C. In Italy and other areas of the Mediterranean the Black Death and plagues into the fifteenth century peaked regularly at the lowest point in the rat flea cycle during the hottest and driest months of the year, June and July.

Secondly, mortality rates, even in India where over 95 percent of cases have occurred since 1894, are of different order of magnitude from the figures revealed by records of the late Middle Ages and early modern period. The highest death toll attained in any plague year of *Yersinia pestis* occurred in Bombay City in 1903 when 2.7 percent of the population died. By contrast, the Black Death killed over 50% in major cities across Europe, and in Florence calculation from tax records the figure was as high as 75%. Nor was the Black Death of 1348 alone in wrecking such calamitous levels. In the plague of 1629-30 Venice lost a third of its population, and in 1656-7, plague claimed about half the population of Genoa (see figure 1).

Thirdly, patterns of adaptation between pathogen and people have been different for historic plague and *Yersinia pestis*. For the latter, humans possess little, if any, natural immunity. As a consequence, the disease has recurred annually in the

same villages or towns for extended periods of seven to thirty years (as in places in the Punjab from 1896 to the 1920s); secondly, since 1894 no significant change in the age structure of victims of *Yersinia pestis* has been detected; and thirdly, before the dissemination of effective antibiotics, no downward trajectory in mortalities has been traced with increased exposure to the disease over time. The patterns of the Black Death disease, especially over its first hundred years, were entirely different. Plague rarely remained in any locale for more than two successive seasons. For its first hundred years, progressively this plague became one of childhood. In Pisa, for instance, a staggering 88% of the plague victims had become children by the fourth strike of this pestilence in 1383, while less than half were children in 1348. Furthermore, the natural immunity of humans to the late medieval plague is suggested by a steady decline in plague deaths (both absolutely and relative to the population) over the first hundred years of the Black Death disease. Finally, a detailed study of the two great Italian plagues of seventeenth century (1629-30 and 1656-57) suggests that no single community was struck by both. Instead, the areas affected match like pieces of a puzzle – which is especially the case of Liguria (see figure 2, where the weaker Sicilian epidemic of the 1620s also was a factor).

Yet the most problematic evidence for claiming that *Yersinia pestis* was the same as the bubonic disease of the medieval and early modern past regards differences in transmission. Because of its reliance on rats and fleas *Yersinia pestis* is inefficient in spreading to and through human settlements and is slow-moving. First, it must kill off most of its resident reservoir of rodents before attacking via rat-fleas human populations. Even when humans are bitten by a blocked rat-flea, transmission of the bacillus from the flea's gut to the human occurs in less than 13 percent of cases. Because of this complex and inefficient mode of transmission (as opposed to direct person-to-person transmission) mortality rates are generally low despite the extreme lethality of *Yersinia pestis*.

By contrast, contemporaries of the late Middle Ages and early modern period recognized their own plague as distinctly different from any previous disease or epidemic. They not only commented on the disease's extraordinary lethality and swiftness of death but also on the novelty of its transmission, the speed by which it spread through towns and over vast territories. Recently, a team of epidemiologists, employing sophisticated stochastic and mapping tools have calculated the speed of diffusion of the Black Death across Europe from 1347 to 1351. They have found that no human epidemic has ever shown a propensity to cover space so swiftly (including the influenza epidemic of 1918), and the difference in dissemination between the plagues of the late nineteenth and twentieth centuries and the Black Death is colossal: while the area covered by the Black Death (1347-51) was to the fourth power of time, that of the bubonic plague at its height in India, the subcontinent of the plague's (*Yerisinia pestis*) most spectacular diffusion, was to the second power of time, a difference of two orders of magnitude.

Immediately with the plague of 1348 the word *contagio* ("contagion") became widespread in the medical literature and crossed into the common parlance of chroniclers and other non-medical writing. These writers distinguished their new disease not only by its skin disorders—a combination of buboes, carbuncles, and small bumps (*morbilli*) that covered entire bodies; they also pointed to this disease's extraordinary speed of transmission, from one household to another, through towns and villages, and over vast regions of Europe. One means of recognizing the disease and its peculiarly high contagion was through observing the clustering of cases within households.

By the second half of the fifteenth century death books such as those compiled by doctors on Milan's health board allow us now to calculate this household clustering. For seven plague years 1452 to 1524 around a quarter of all plague deaths occurred within the same household and more spectacularly on the very same day (see figure 3a). No other disease has shown such a high proportion of household clustering. Similar patterns of plague are seen elsewhere as with the remarkable combination of burial records and tax surveys ('Boccatico del Sale') at Nonantola (Modenese) during the plague of 1629-30 (see figure 3b). Here, it is also possible to compare the "real" (observed) distribution of infected people per household with what should be expected had the disease not spread person-to-person. The latter can be calculated by a binominal expansion (the probability for each person of being infected is unrelated to the health situation of other people living in the same household). The comparison between the observed and the hypothesized distribution shows that for households of any size the clustering of deaths is much greater than expected. Secondly, also the proportion of large households that were severely struck is much greater than expected (see figure 4). These findings suggest direct, human-to-human, transmission: the probability of first infection of an household member is positively correlated to household size (more than proportional). The first infection then increases the probability of other household members to be infected in their turn.

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Historians and scientists have pointed to these differences in transmission between present-day bubonic plague and the Black Death disease of the late Middle Ages and early modern period. Yet, instead of questioning the identity of these historic plagues as *Yersinia pestis*, most have sought to explain how the two could have been the same. To maintain the identity, they have taken one of three tacks. First, relying on early descriptions such as those of Guy de Chauliac that the Black Death spread first in the winter months of 1348 with symptoms of coughing and spitting of blood, they have argued that the earlier plagues were primarily pneumonic. This form, they argue, accounts for the absence of prior or concurrent descriptions of an epizootic of rats or other rodents, the person-to-person transmission of the disease, and its vastly more efficient and widespread diffusion than plagues since 1894. This tack has proven problematic: (1) pneumonic plague is a rare complication of *Yersinia pestis* which has claimed few casualties since 1894 and with only two epidemics of it (Manchuria 1911 and 1922). But even these two plagues claimed far fewer victims than those of bubonic form in India and China during the early twentieth century. The worst of the pneumonic plague, that of 1911, killed less than 0.3 per cent of the territory afflicted. (2) Doctors such as Wu Tien-Teh and more recently epidemiologists such as Steve Leach and Raymond Gani have shown that pneumonic plague spreads inefficiently person-to-person and is not highly contagious. (3) The Black Death disease, especially in the Mediterranean, spread in the hottest summer months when pulmonary diseases are generally less common. (4) By the mid-fifteenth century, descriptions of pulmonary complications with the spitting, coughing or vomiting of blood disappear completely from clinical reports such as the Milanese death records and from doctors' manuals on the signs and symptoms of their plague. Yet, without these pulmonary symptoms, the plague continued to be extremely contagious as witnessed by the uniquely high rates of household clustering and could still register massive mortality rates as with the Italian plagues of 1629-30 and 1656-7. Instead, detailed clinical reports of the early modern period described diarrhoea,

stomach pain, bloating, vomiting of food (not blood) as usual plague symptoms and suggest that their plague may have spread person-to-person via the intestinal tract. From correlations between parishes of high plague mortalities and polluted wells, sixteenth-century doctors began to conclude that the plague's transmission related to contaminated water supplies. As with other infective diseases such as cholera or typhoid, historic plague may have been water-borne.

Others, for instance Jean Noel Biraben, have speculated that the high person-to-person contagion and rapid diffusion of the Black Death and early modern plagues can be explained by the human flea, *Pulex irritans*. With this vector, however, they fail to explain (1) why late medieval and early modern plagues in the Mediterranean spread usually during the summer months and rarely during the winter when victims wore more clothing, used more bedding, and when problems with human fleas occur? (2) Why this flea, which in laboratory tests is one of the least efficient in transmitting plague (*Yersinia pestis*), should have been in the Middle Ages and early modern period more powerful by two orders of magnitude in spreading plague than the most efficient plague vector, *X. cheopis*? And (3) why there have been few, if any, clear examples of *Yersinia pestis* being spread by *Pulex irritans* since Yersin's discovery? Moreover, when this flea has been suspected as the vector (as in mountain villages of Iran in 1967), plague has been extremely limited in its spread, restricted to individual villages.

Thirdly, historians and scientists have speculated that the strain of the supposed medieval and early modern plagues derived from a more lethal or virulent 'biovar'. But what difference would enhanced virulence of the pathogen have meant for these vast differences in the transmission? In fact, both *Yersinia pestis* and historical plague were extremely lethal and killed rapidly. At Hong Kong in 1894 *Yersinia pestis* killed 90 percent of victims; yet the diffusion of the disease remained low in comparison with many other less lethal infectious diseases. Indeed, the Black Death disease, despite its much higher rates of mortality and wider dissemination may have often been less lethal than *Yersinia pestis*. Medical observers of the sixteenth century, for instance, claimed that if patients were well nourished and attended they could survive the plague. During the plague of 1576-7 the Venetian doctor Annibale Raimondo claimed that nine of ten of those receiving proper care recovered. Instead of increasing the diffusion of a disease, heightened virulence of a pathogen can spell the opposite (as seen with the limited diffusion of Ebola): a virus can be too deadly for its own good.

Finally, by the late sixteenth century, European patterns of plague diffusion began to change with Italy becoming distinctive from the rest of Europe. Lazzaretti, hospitals for convalescents, public health boards, the use of printed broadsheets for the daily posting of plague ordinances, military enforcement of quarantine, and new forms of plague intelligence with reports as far a field as Africa and the Near East became increasingly common throughout Italian towns and regions particularly with the plague of 1576-8. As a result, the intervals between plagues in Italy widened. Between 1449 and 1524, for instance, at least five waves of plague afflicted Milan. Between 1524 and 1630, it experienced only two. The differences between Italy and the rest of Europe were more impressive: between 1540 and 1666 devastating plagues struck London, for example, nine times and, between 1550 and 1660, hit Denmark (which did not institute state-wide plague regulations until 1625) eleven times. Between 1560 and 1660 regions in central Europe were invaded by eleven maritime plagues in addition to a number of internal ones. By contrast, over the same period only two plagues afflicted Milan and Venice; and only one Florence. However, when

plague broke through Italian regional defences as in 1629-33 and again in 1656-7, the devastation was disastrous with far higher casualties than experienced north of the Alps: the Great Plague of London in 1665, for example, felled less than a fifth of its population, while Genoa, which had not known plague since 1579, lost half its numbers in 1656 (or even two-thirds according to another estimate). Did Italy profit demographically or economically from its greater plague surveillance? The *prima facie* evidence from early modern population and economic trends suggests that it did not. This does not suggest that Italian health boards were wasting money, as Carlo Cipolla had it. According to him, contemporaries had been misled by their erroneous theories about plague: quarantine, instead of preventing the spread of 'peste' had the opposite effect. It supposedly locked up the healthy together with dangerous plague rats thus assisting the spread of the disease. On the other hand, Cipolla thought that sanitary cordons could be beneficial because they blocked the transit of men (fairly innocuous) and the very dangerous rat-infested merchandise. If, however, we accept the hypothesis that historical plague spread person to person, then health boards were applying useful sanitary regulations that limited the frequency of plague in Early Modern Italy compared to the rest of Europe. Such success, however, came with a poison bullet—a diminution in immunity— resulting in the much more devastating effects of the two seventeenth-century plagues in Italy.

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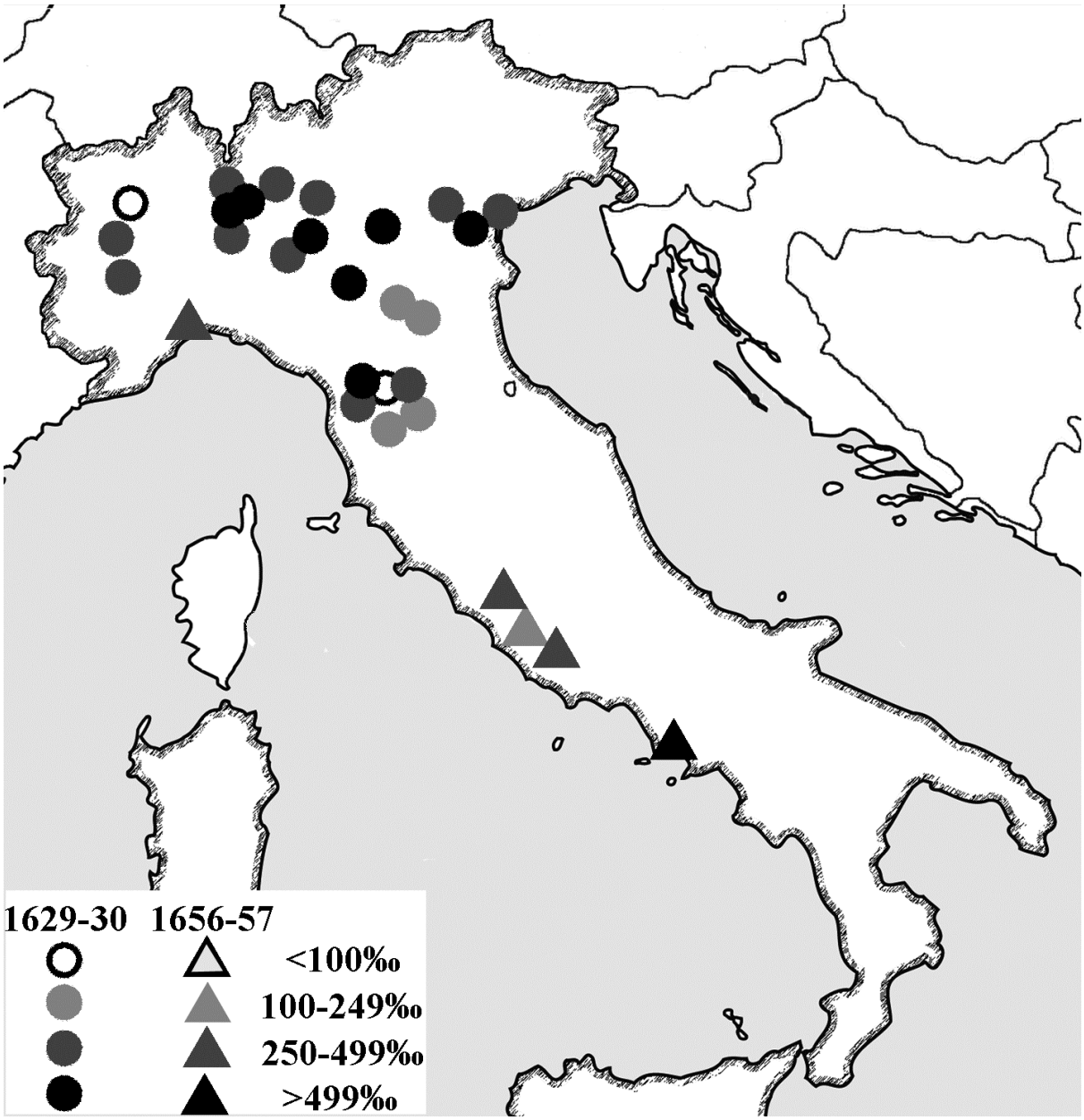
In conclusion, historical plague had epidemiological characteristics that were strikingly different from *Yersinia Pestis*, suggesting two possibilities: (1) 'peste' (historic plague) was an entirely different illness, an hypothesis that cannot be ruled out nor be developed further by us (at least at present); (2) it was related to *Yersinia pestis* with similar DNA, perhaps a separate biovar of it. But either case the key differences between historic plague and *Yersinia pestis* did not turn on differences in the virulence of the pathogen but on modalities of transmission.

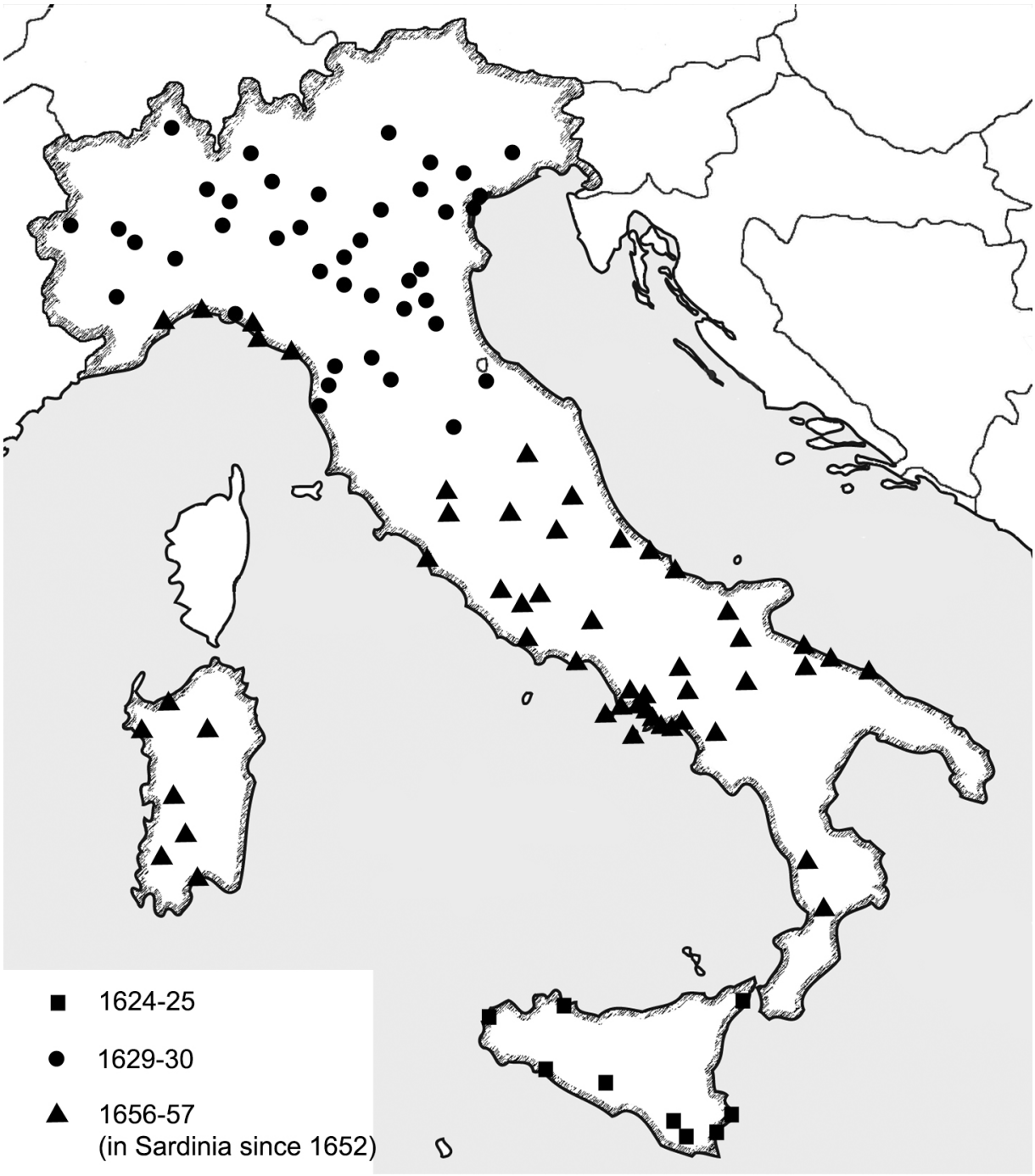
Perhaps in the near future paleo-demographers and geneticists who analyze the dental pulp of ancient victims of the disease will discover new pathogenic relations between historic plague and *Yersinia pestis*. Despite early enthusiasm and confidence given by the new tools of DNA research into the historic past, the results of this research on plague remains inconclusive and debate continues between teams of scientists who claim to have found it and others who find no evidence for it and have been highly critical of the scientific methods of the rival groups. Nonetheless, even if it is established that historical plague was *Yersinia pestis* or a close relative of it, scientists would still have to confront the question of the vast and fundamental differences in transmission of historic plague and any form of *Yersinia* known today. We hope our study makes clear that not all the answers can be found from the new genetic studies of the past. Still continued historical research using "traditional" methods of historical demography will be essential for discovering how people in the past caught plague and why this disease spread so rapidly over vast areas as efficiently as any disease now known to medical science .

Fig. 1. Urban mortality rates during the plagues of 1629-30 and 1656-57



Source: Alfani 2009, *Plague in seventeenth century Europe*

Fig. 2. Territorial integration of Italian plagues of Seventeenth Century
(dots = places known to be infected)



Source: Alfani 2009, "Pestilenze e 'crisi di sistema' "

Fig. 3a. Intervals between deaths within households:

the Lazzaretto of San Gregorio, 1485

0 days	1 day	2 days	3 days	4 days	5 days	6 days	7 days	8 days	9 days
30	7	5	5	1	5	3	4	2	4
10days	13	14	15	17	19	20	21	29	34
2	2	3	2	1	2	1	2	2	1
51days	76								
1	1								

Fig. 3b. Intervals between deaths within households:

Nonantola, 1630

0 Days	1	2	3	4	5	6	7	8	9
46	35	28	23	9	16	15	9	15	6
10 Days	11	12	13	14	15	16	17	18	19
10	9	10	11	4	4	7	5	5	5
20 Days	21	22	23	24	25	26	27	28	29
5	3	3	4	3	1	2	3	0	3
30 Days	31-40	41-50	51-60	61-70	71-80	81-90	91-100	101-110	>110
1	11	6	5	4	5	3	0	1	2

Source for both figures: Cohn and Alfani 2007, “Households and Plague”

Fig. 4. Observed and Casual distribution of burials per head in Nonantola, 1630 (per household size and number of burials)

		Deaths per Household															
		0		1		2		3		4		5		6		>6	
Household Size	N. Households	Observed	Casual	Observed	Casual	Observed	Casual	Observed	Casual	Observed	Casual	Observed	Casual	Observed	Casual	Observed	Casual
1	38	32	32	6	6	-	-	-	-	-	-	-	-	-	-	-	-
2	111	86	78	18	30	7	3	-	-	-	-	-	-	-	-	-	-
3	83	59	49	15	28	6	5	3	0	-	-	-	-	-	-	-	-
4	98	56	48	23	37	15	11	3	1	1	0	-	-	-	-	-	-
5	99	67	41	17	40	7	15	3	3	2	0	3	0	-	-	-	-
6	57	38	20	7	23	9	11	1	3	2	0	0	0	0	0	-	-
7	41	21	12	7	16	4	9	3	3	4	1	1	0	1	0	0	0
8	27	11	7	6	10	3	7	2	3	1	1	1	0	0	0	3	0
9	18	10	4	1	6	1	5	2	2	2	1	0	0	2	0	0	0
10	13	8	2	2	4	0	4	2	2	0	1	1	0	0	0	0	0
11	9	6	1	1	3	0	3	1	2	1	1	0	0	0	0	0	0
12	8	2	1	2	2	0	2	1	2	0	1	1	0	0	0	2	0
13	7	3	1	0	2	1	2	0	1	1	1	0	0	0	0	2	0
14	7	1	1	0	2	3	2	0	2	2	1	0	0	1	0	0	0
>14	7	2	0	1	1	1	2	0	2	0	1	0	1	0	0	3	0
Total	623	402	296	106	211	57	81	21	25	16	7	7	2	4	1	10	0
N. Burials		0	0	106	211	114	162	63	75	64	29	35	10	24	3	87	0
% Burials		0	0	21,5	42,9	23,1	33,1	12,8	15,3	13,0	5,9	7,1	2,1	4,9	0,7	17,6	0

Source: Alfani and Cohn 2007, “Nonantola 1630. Anatomia di una pestilenza”

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