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Disease and death in the ancient city of Rome

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Abstract: This paper surveys textual and physical evidence of disease and mortality in the city of Rome in the late republican and imperial periods. It emphasizes the significance of seasonal mortality data and the weaknesses of age at death records and paleodemographic analysis, considers the complex role of environmental features and public infrastructure, and highlights the very considerable promise of scientific study of skeletal evidence of stress and disease.

IN SEARCH OF FUNDAMENTALS

Despite the city's prominence in our sources, its demographic conditions are remarkably poorly known. The size of Rome's population is never properly reported and modern estimates rely on inferences from the scale of public grain distribution schemes (see Chapter 2). The geographical and social provenance of its inhabitants is likewise largely a matter of conjecture (see Chapter 5). Marriage practice and household structure may well have been peculiar to the city's exceptional environment but are difficult to derive from epigraphic documents (see Chapter 6). Overall fertility rates necessarily remain unknown. Metropolitan patterns of morbidity and mortality, however, are more amenable to empirical and even quantitative inquiry, and will therefore be the main concern of this chapter.

As always in demography, a field that is built on counting and measuring, pride of place belongs to large bodies of quantifiable data. Provided by funerary inscriptions, they record two vital features, the monthly distribution of deaths and age at death. Though very similar in character, these two datasets nevertheless lead us in opposite directions: whereas evidence of seasonal mortality has greatly improved our understanding of the impact of infectious disease on life in the city of Rome, demographic analysis of reported ages at death has remained a dead end.

SEASONAL MORTALITY AND CAUSES OF DEATH

Epitaphs that record the day or at least the month of death enable us to track variation in the mortality rate across the year. In Rome, relevant information is furnished in the first instance by early Christian commemorations from the fourth and fifth centuries CE which have survived in large numbers in the catacombs that ring the city. A sample of nearly 4,000 dates of death gathered by Brent Shaw provides a solid basis for demographic investigation. In addition, Shaw has drawn attention to a much smaller but also much earlier body of corresponding records on inscribed cinerary urns dating from the first half of the first century BCE that were found at a site on the via Appia just outside Rome. Whilst separated by approximately half a millennium, both datasets reveal a comparable concentration of mortality in the late summer and early fall (Fig.1).[1]

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[1] Shaw 1996: esp. 115-21; 2006: 93-101.

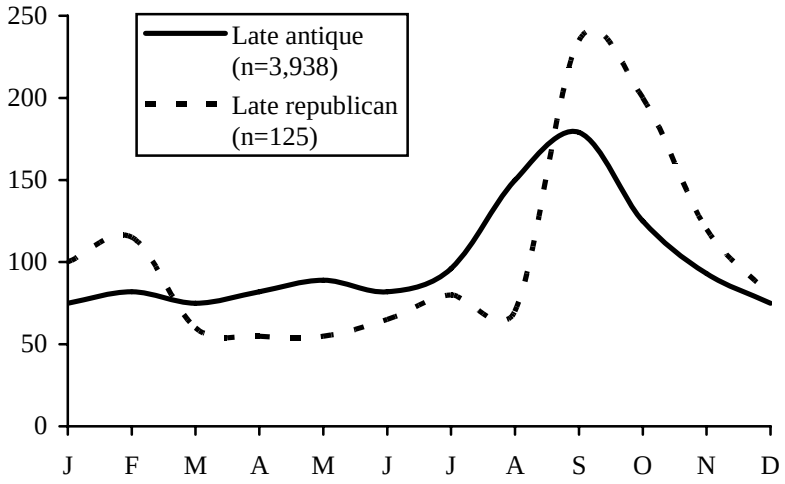

Fig.1 Seasonal mortality variation in the city of Rome
(100 = annual mean)
Source: Shaw 1996: 115 & 2006: 100 (adjusted)

There is no obvious way in which these profiles could be a mere artifact of biased recording practices. The much larger late antique sample allows differentiation according to age. It shows that the observed seasonal profile applies to most age groups: men and women up to the age of fifty, who represented the large majority of the population, disproportionately often died in August, September, and October whereas only the elderly – although they also appear to have suffered in this period – were similarly likely to die in the late fall, winter, and early spring (Fig.2).

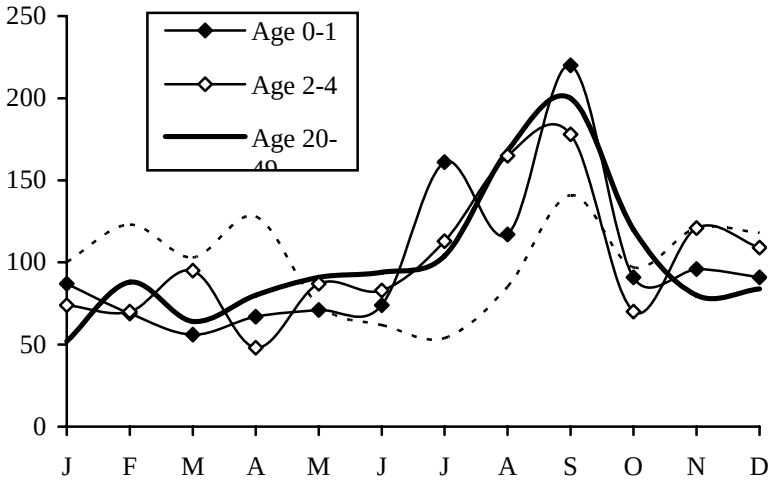

Fig.2 Seasonal mortality variation by age-group in the late antique city of Rome
(100 = annual mean)
Source: Shaw 1996: 118, 120

The latter observation in consistent with evidence from nineteenth-century Italy that shows a similar pattern at elevated ages and indicates increased susceptibility to pulmonary afflictions in the cool and wet part of the year. From a comparative demographic perspective, it is the high degree of consistency across most age cohorts that requires explanation. In the more recent past, even though mortality among children, adolescents, and young and middle-aged adults would often spike at the same time of the year, such spikes were usually much more pronounced among infants and small children who are generally more vulnerable to infection. An environment in which young adults – the most resilient element of any given population – succumbed to seasonal diseases at the same rate as the very young is therefore highly exceptional. Moreover, added mortality in the cold and wet season probably conceals the extent to which even the elderly remained at risk in the late summer and early fall. All this raises the question why adults failed to acquire some measure of immunity to seasonal diseases that carried off many children.

On a previous occasion I suggested that this unusual pattern might have been caused by a combination of two factors, a strong seasonal presence of the most pernicious strains of malaria and a high rate of immigration from healthier regions at mature ages. In the city of Rome in the nineteenth century, malaria infection peaked from August to October and overall death rates rose accordingly, a pattern that is fully consistent with that found in the late antique epitaphs. Various types of malaria, from comparatively mild benign tertian fever (caused by *Plasmodium vivax*) and quartan fever (*P. malariae*) to the more lethal malignant tertian fever (*P. falciparum*) and complications such as 'semitertian fever', are documented for the capital of the empire. As early as the first century BCE, the physician Asclepiades of Bithynia described 'quotidian' fever (typical of primary falciparian infection) as being common there. Some 250 years later his more famous colleague Galen observed that physicians practicing in Rome had no need to consult medical texts for descriptions of semitertian fever (related to the same infection) simply because its symptoms could nowhere else better be observed: 'just as other diseases thrive in other places, this one abounds in that city'. Several literary sources likewise reflect the likely impact of malaria in the late summer and early fall.[2]

If they do indeed refer to Rome proper, some of Galen's more specific references to the age-specific incidence of particular manifestations of malaria may even allow the inference that falciparian malaria had attained what is known as hyperendemicity, creating an environment in which a majority of the population carried the parasites in their blood but survivors gradually developed immunity in response to repeated infection. This suggests that persistently high seasonal death rates among adults may have been sustained by immigration from healthier – malaria-free – locales that were likely to generate net population growth for which the capital provided an attractive outlet.

[2] For the connection with malaria, see Scheidel 1994: 157-65; 2003: 162-3. Medical observations: Asclepiades in Caelius Aurelianus 2.63-4 ed. Drabkin; Galen 7.135, 7.465, 17A.121-2 ed. Kühn, with Sallares 2002: 220-3. The literary allusions are discussed in Scheidel 2003: 165-7; but cf. Lo Cascio 2001b: 191-2.

Comparative evidence from later periods of Italian history lends support to this conjecture.[3]

While falciparian malaria in particular was certainly capable of killing on its own, its lethality would have been greatly magnified by its synergistic interaction with other seasonal infections, such as gastro-intestinal disorders and respiratory diseases. In this connection, it is worth noting that some of these conditions – including typhoid and tuberculosis – tend to be more common among adolescents and even adults than among the very young. This, too, may help explain the striking persistence of high seasonal death rates well beyond early childhood that is documented in the epigraphic record.

It is true that seasonality profiles do not reveal either the actual scale of mortality, in the sense of the proportion of the population which died in a given year, or mean life expectancy, which is derived from that measure. It is also true that epigraphic profiles from late antique southern Italy and Roman and early Arabic Egypt echo the metropolitan Roman pattern. Even so, comparison with later historical datasets suggests that the sheer scale of the endemic seasonal mortality surge beyond childhood that we find in the ancient city of Rome far exceeded anything that can be observed in the more recent past, except in years marred by major epidemics. This alone points to extremely high levels of mortality overall.

MORTALITY RATES, AGE STRUCTURE, AND LIFE EXPECTANCY

One might think that ages recorded on Roman tombstones might be able to shed some light on this question. If the distribution of ages at death in these documents faithfully reflected actual conditions it could be used to reconstruct the age structure and hence the life expectancy of the metropolitan population. Unfortunately, as has long been recognized and frequently (and compellingly) re-iterated, this is not the case. The frequency of funerary commemoration varied hugely according to sex and age as well as other factors, even including the language in which these texts were recorded. Looking beyond the city of Rome, we encounter tremendous diversity, from locations where death in childhood was frequently commemorated to others where almost everyone seems to have lived to a ripe old age, a situation unknown before our own time. This shows that these data are not representative of actual conditions and cannot be used to reconstruct demographic profiles.[4]

More specifically, comparison between the attested distribution of ages at death in the city of Rome and that predicted by a plausible high-mortality model life table suggests that the epigraphic record underreports the loss of life at very young ages; vastly privileges deaths among older children, adolescents and young adults; and equally vastly neglects death in old age (Fig.3).

[3] Sallares 2002: 223-4 and Scheidel 2003: 164 on Galen 7.468, 11.23 ed. Kühn.

[4] Clauss 1973 is the best survey of epigraphic diversity.

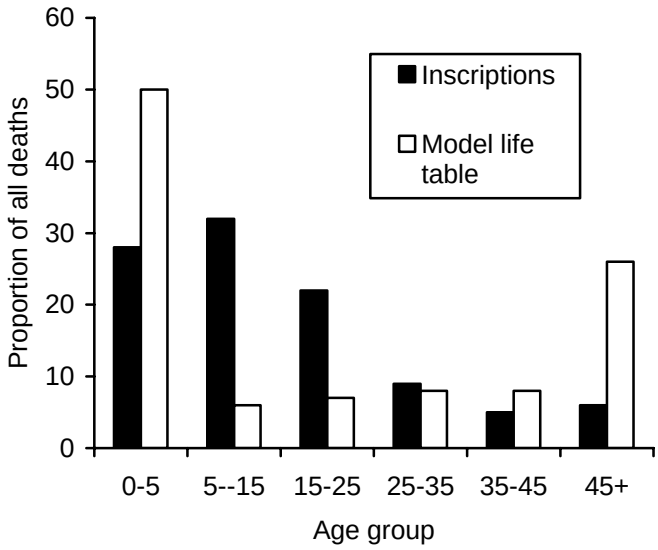

Fig.3 Age-at-death distribution in funerary inscriptions from the city of Rome and in a plausible model life table
(Source: Paine and Storey 2006: 72)

Somewhat desperate attempts to salvage some demographic credibility by attributing the attested profile to the effects of epidemic or otherwise 'catastrophic' mortality that might have shaped the city's population structure in unusual ways have signally failed to reconcile the observed pattern with any demographically plausible model. We must acknowledge that the real value of these texts lies in their capacity to elucidate cultural preferences such as the differential valuation of the sexes at specific ages and its change over time. Relevant as this is for the purposes of social and cultural history, it does not answer demographic questions.[5]

Skeletal remains are similarly unhelpful. Not only does paleodemographic analysis in general continue to suffer from a variety of technical and methodological problems – from difficulties in accurately ageing adult bones to the lack of information about the effect of in- and out-migration on local age structure –, this approach is even less promising in a place like imperial Rome whose population may well have been mobile and unstable in unpredictable ways (see Chapter 5 and below). This makes it impossible to apply the principles of stable population analysis – that is, the derivation of mortality and fertility rates from an observed age distribution.[6] Thus, even if the correct age at death of all available skeletons could eventually be ascertained, this would not necessarily tell us much about mortality rates and life expectancy beyond broad outlines – life was short… – that may readily be accepted on *a priori* grounds.

[5] Paine and Storey 2006: esp. 82-5 (failure to explain observed pattern); Shaw 1991 (cultural preferences).

[6] A misguided attempt to undertake stable population analysis of what must have been an even more exceptional population – at Roman Portus – illustrates the pitfalls of this approach: Sperduti 1995.

DISEASES

Faced with these forbidding uncertainties, it is particularly important to complement quantitative study with consideration of qualitative evidence. It is worth appreciating the range of diseases that were experienced in the city even if their prevalence and demographic consequences remain unknown. Galen called Rome 'this populous city, where daily ten thousand people can be discovered suffering from jaundice, and ten thousand from dropsy' (11.328 ed. Kühn). Many of the case histories he described in his works must have occurred there.[7] His own and earlier medical references to malaria in the city have already been mentioned. A century before Galen, the Elder Pliny devoted a section of his *Natural History* (26.1-9) to 'novel diseases' that had been introduced to Rome and Italy in the recent past. *Lichen* or *mentagra*, a pustulous lichen on the chin transmitted by kissing and supposedly confined to the upper classes, was one of them; *elephantiasis*, safely identifiable as lepromatous leprosy, was another. While the ubiquity of gastro-intestinal infections – much endured yet rarely mentioned – may be taken for granted, a reliable assessment of the prevalence and impact of pulmonary tuberculosis, potentially a major killer, would require empirical data that we do not currently possess. One can only hope that future analysis of skeletal remains will shed more light on this issue (see the final section below).

Epidemic outbreaks added to the disease burden but tangible information is scarce. The late Republican and imperial periods are not as well covered in our sources as earlier centuries. The annalistic tradition preserved by Livy reports numerous epidemics – usually though not always in the city of Rome – between 490 and 292 BCE and especially from 212 to 174 BCE: in the latter period, a mortality crisis is mentioned on average every 4.3 years. By contrast, authors covering the following centuries paid less attention to inauspicious events and are often unspecific as to the location of reported epidemics. Thus, from the mid-second century BCE to the end of the second century CE, epidemic outbreaks in the city of Rome itself are mentioned on only five occasions, for 142 BCE, 23-22 BCE, 65 CE, 79/80 (?) CE, and 189 CE. The record is only marginally better for late antiquity, when epidemics are expressly placed in Rome on a further ten occasions between 284 and 750 CE.[8] This paucity is undoubtedly a function of the nature of the evidence rather than a reflection of dramatic improvement: the ongoing Tiber floods alone, discussed in the next section, would have made that impossible.

The most dramatic mortality crisis in imperial Rome may well have been the so-called 'Antonine Plague', probably smallpox, that swept across the empire from 165 CE and continued into the 170s and perhaps into the late 180s CE or even later. If this epidemic was indeed a case of smallpox striking a 'virgin' (i.e., previously unexposed) population, it could in theory have killed a sizeable proportion of the metropolitan population (a third?), with a death toll in the hundreds of thousands. Yet we will never know, and sporadically proffered numbers are notoriously unreliable. Cassius Dio (72.14.3) refers to 2,000 fatalities a day during an epidemic in Rome in 189 CE, possibly a resurgence of the 'Antonine Plague', a figure that has the dubious merit of being at least

[7] Mattern 2008: 173-202 lists all cases.

[8] For the earlier periods, see Duncan-Jones 1996: 111. Epidemics in Rome: Oros. 5.4.8; Dio 53.33.4, 54.1.3; Tac. *Ann*. 16.13; Suet. *Nero* 39; Hieron. *Chron*. 188 ed. Helm (with Suet. *Tit.* 8.3 for the correct date); Dio 72.14.3. For late antiquity, see Stathakopoulos 2004: 175-386 (*pace* 30).

theoretically possible for the very large capital. Other tallies give the appearance of rounding or stylization and are probably merely symbolic, such as the 30,000 residents supposedly killed by an epidemic under Nero or, far less credibly, 10,000 per day under Titus or 5,000 per day under Gallienus.[9]

In any event, the size, density, and connectedness of the city must have invited microbial onslaughts. This combination of risk factors was not lost on ancient observers: as Herodian noted, when an epidemic struck Italy in the late 180s CE – probably the same event mentioned by Dio –, "it was most severe in Rome, which, apart from being normally overcrowded, was still getting immigrants from all over the world" (1.12.1). We cannot tell if the thousands of bodies deposited in the ditch of the 'Servian' *agger* on the Esquiline that were reportedly found in 1876 were at least in part hastily discarded victims of epidemics. Yet the recent discovery (atop the later catacombs of St Peter and Marcellinus 3km south-east of Rome) of a series of rooms filled with large numbers of tightly stacked corpses that had been deposited at the same time and clad in garments decorated with gold threads and amber points to a sudden event that required the unceremonious disposal of numerous deceased individuals of not inconsiderable standing, quite possibly an epidemic in the late second or early third century CE that might even have been a resurgence of the 'Antonine Plague'.[10]

ENVIRONMENT AND INFRASTRUCTURE

Throughout the Roman period the Tiber remained prone to flooding. A new estimate based on over 2,000 years of records reckons with significant flooding every few years and massive inundations a few times per century. The relationship between floods and spikes in infectious disease is well established; malaria in particular would have been exacerbated by these incidents. In this respect but also more generally, topography played an important role in determining health conditions: Livy once called the hills of Rome 'most healthy' (5.54.4), and data from later periods suggest that they were indeed healthier than low-lying areas. Among other things, locations even at moderately elevated altitudes are less exposed to malaria infection.[11]

Even as the size of the city's population and hence its settlement density remain uncertain, there can be little doubt that many of its residents lived in crowded conditions that were conducive to unsanitary practices and infection (see Chapter 9). As already noted, Rome's nodal position encouraged the introduction of new strains of disease while its exceptionally large population would have made it easier for such arrivals to become endemic and contribute permanently to the metropolitan disease pool. At the same time, we must ask which factors that were peculiar to Rome might have mitigated health hazards.

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[9] Duncan-Jones 1996 (Antonine Plague); Scheidel 2003: 171-2 (outbreaks in Rome, on Suet. *Ner.* 39.1; Hieron. *Chron.* 188 ed. Helm [misdated to 77 CE]; *HA Gall.* 5.5).

[10] Graham 2006: 67, 73, 80-1 (ditch); Blanchard et al. 2007 (catacomb). Here and in the following, all distances are from the Aurelian Wall.

[11] For flooding and its health consequences, see Aldrete 2007: 81, 141-54; and cf. also Chapter 14. Malaria and altitude: Sallares 2002: 206-9, and cf. 331 s.v. 'altitude'.

The role of Roman urban infrastructure in particular merits consideration. From a public health perspective, this infrastructure's capacity to provide clean water and dispose of human and animal waste must be regarded as its most significant features (see Chapter 18). The city's sewer system's potential to prevent disease was diminished to the extent to which it served to provide street drainage rather than to remove sewage from the source. Rome's costly network of aqueducts delivered clean water on a scale that would have been the envy of other premodern cities and could in principle have made a massive contribution to public health. However, once again the system was set up in a way that is likely to have reduced its actual benefits. Most residents obtained water from public basins and fountains, a practice that created several problems. The accumulation of garbage and excrement (and the occasional corpse and carcass) in the streets combined with the overflow from these water outlets to contaminate street surfaces, creating an environment that was conducive to the spread of gastroenteritis, typhoid, diarrhea, salmonellosis, and worm infection.[12]

It is true that the continual flow of water would have flushed out contaminants that reached these outlets, preventing them from being compromised for longer than in the very short term. Nevertheless, the availability of fresh water at these facilities, in relative abundance and free of charge, did not automatically translate to sanitary storage and consumption. Studies conducted in contemporary developing countries highlight the great importance of contamination between source and point-of-use: safe water drawn from improved sources frequently shows unsafe levels of *E. coli* upon consumption. A variety of factors are responsible, from bacterial re-growth in stored water and re-contamination through dipping with unclean hands and cups to the presence of biofilms on the inner surfaces of containers. This is a phenomenon that historians need to keep in mind whenever they consider the actual health benefits of the generous public provision of clean water in the imperial city of Rome.[13]

The Roman culture of public bathing is another example of unexpected consequences. Immersion in unchlorinated water posed its own health hazards, especially when it was heated and thus triggered bacterial growth. Authorities such as Celsus and Pliny the Elder leave no doubt that 'medicinal bathing' was supposed to take place in public baths: as a matter of fact, the former advises patients with bowel troubles to bathe their anuses in the hot pools located at these venues but (not unreasonably) warns those with infected wounds not to expose them to the filthy contents to these facilities.[14]

A 'POPULATION SINK'?

What this all means for the demographic 'balance sheet' of the city remains profoundly unclear. Large premodern cities in general were unhealthy places that tended to suffer from excess mortality or even an excess of deaths over births. The scale and especially the causes of these phenomena continue to be debated: a variety of factors from elevated levels of density-dependent morbidity and mortality to the effects of

[12] Scobie 1986: 407-22; Aldrete 2007: 142-5. Cf. also Raventos and Remolà 2000: 63-73, 95-105, 123-7.

[13] Aldrete 2007: 152-4 (overflow); Clasen and Bastable 2003; Gundry, Wright and Conroy 2004; Gundry et al. 2006 (secondary contamination).

[14] Scobie 1986: 425-7; Fagan 2006: 191-4; Celsus, *Med.* 4.25.3, 5.26.28c.

immigrants' age distribution and of urban labor markets on marriage and reproduction appear to have played a role. A very rough guesstimate for the early modern city of London envisioning an annual excess of deaths over births equivalent to 1% of the total size of the population has repeatedly been acknowledged by Roman historians, yet its relevance is doubtful: it might equally well be too pessimistic (because imperial Rome enjoyed better infrastructure and welfare provisions) or too optimistic (because London was free from falciparian malaria).[15]

It is important to remember that not all premodern metropolises were equally deadly, and that in so far as they *were* deadly this was not necessarily so for the same reasons. On the one hand, the medical and demographic impact of the aqueducts and free food distributions of imperial Rome must not be overrated: while the benefits produced by the former would in part have been undone by the adverse consequences of overflow and secondary contamination, the significance of the latter is called into question by the observation that in premodern societies location and population density were generally more potent determinants of health and life expectancy than food intake. On the other hand, it would surely be unwise to throw the baby out with Celsus's soiled bathwater: a counterfactual city of Rome that had to rely on wells and the water of the Tiber, or one that had lacked state-backed food supply, would almost certainly have been a worse place than the historical city actually was – smaller, perhaps, but also (even) less healthy.

To complicate matters further, Rome's inhabitants faced problems that were uncommon in other very large premodern cities, most notably endemic malaria. Comparative historians are left speculating that they might be dealing with a zero-sum game: although in the case of Rome, certain hazards that were more common elsewhere (primarily water contamination and food shortages) were mitigated by public provisions, their place may have been taken by others (such as malaria) that were less common elsewhere. The scope of modern guesswork is therefore only feebly constrained by comparative historical reasoning and the weighing of different factors. This leaves margins of uncertainty that are wide enough to accommodate quite diverse scenarios of metropolitan demography, from a city with a stable core population that was able to reproduce itself to one that relied on continuous massive immigration merely to maintain its size.[16]

THE WAY FORWARD

These uncertainties do not mean that there is no hope of progress. Science holds very considerable promise in expanding our empirical knowledge of Roman health. The best source of information on this topic is necessarily the Roman body itself, represented by large numbers of skeletons that have come to light near the city or are yet to be discovered. However, although it has long been possible to study dental, cranial, and other skeletal anomalies that are indicative of stress and disease, and analyses of ancient

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[15] See Woods 2003 for the debate. For London, see Wrigley 1987: 135.

[16] While Morley 1996: 39-54 as well as Scheidel 2003: 175-6 and 2004: 15-19 argue for the need for massive immigration, Lo Cascio 2001a, 2001b and 2006 assumes greater stability. Skeletal isotope analysis can be expected finally to shed light on the actual scale of immigration: for now, see Prowse et al. 2007, on Portus near Rome; cf. also Killgrove 2008b and more generally the following section.

DNA that reveal the presence of a variety of pathogens have now also begun to appear, relevant work that focuses on the city of Rome and its *suburbium* has remained relatively rare. Skeletal material retrieved from the suburban catacombs has suffered the largest degree of neglect. Excavations prompted by construction work continue to expand the available evidence: by now over 5,000 Roman skeletons are said to have come to light in this way and mostly still await detailed study.[17]

The most telling skeletal features are two kinds of lesions that are often described as porotic hyperostosis but may actually have different aitiologies – namely orbital lesions (*cribra orbitalia*) that are associated with chronic iron-deficiency anemia and other disorders, and porotic lesions of the cranial vault (*cribra cranii*) – as well as enamel hypoplasia, a dental condition resulting from the temporary arrest of enamel matrix growth caused by infection, parasitism, or vitamin D deficiency. While these markers do not enable us to identify specific diseases, their prevalence and distribution are indicative of the overall health status of affected populations.

Preliminary reports on a large imperial cemetery near the ancient via Collatina, about 2km east-north-east of the Porta Praenestina, refer to extremely high levels of enamel hypoplasia (80-92%) and porotic hyperostosis (50-77%). This is consistent with findings from more fully published suburban sites. In a sample from Lucus Feroniae, some 30km north-east of Rome, at least 80% suffered from enamel hypoplasia and a third – and two-thirds of pre-adults – exhibited advanced *cribra orbitalia*. At Vallerano, about 10km south of the ancient city, 69% had (mostly advanced) *cribra orbitalia* and the incidence of enamel hypoplasia was also very high. In a sample from the large cemetery on the Isola Sacra near Portus, the main port of imperial Rome, 80% suffered from enamel hypoplasia. Signs of poor health have likewise been reported for a cemetery at Casale Capobianco near the via Nomentana about 8km north-east of Rome. All these datasets document high levels of ill-health and stress. However, preliminary investigation has revealed comparatively fine health among skeletons unearthed at Casal Bertone, a Roman industrial site less than 2km east of the Porta Maggiore: hardly anyone suffered from enamel hypoplasia, and only one in seven from *cribra orbitalia*. In a sample from yet at another suburban site, Castellaccio Europarco 8km south of Rome, dental health is similarly good but orbital lesions were more frequent albeit less so than at other sites, peaking at 20%. This points to a considerable amount of local variation and should caution us against sweeping generalizations. Above all, it shows how much work remains to be done.[18]

Although malaria may induce anemias that lead to porotic lesions, many other causes produce the same outcome; the presence of malaria must therefore be established in other ways. DNA of *Plasmodium falciparum*, responsible for the deadliest form of malaria, has been extracted from a late Roman child skeleton in Umbria, but comparable genetic evidence from the region of Rome itself is still lacking. The identification of tuberculosis poses its own problems. Only a very small proportion of modern tuberculosis sufferers develop visible bone lesions. For this reason alone, evidence of a healing tuberculous spinal infection in a skeleton from an early imperial suburban

[17] Van der Linde 2009 (catacombs); Catalano 2008 (number of recent finds). Donoghue and Spigelman 2006 summarize references to pertinent DNA studies.

[18] Buccellato et al. 2003: 346-8; Buccellato et al. 2008; Manzi et al. 1999: 338-9; Salvadei, Ricci and Manzi 2001: 711; Cucina et al. 2006: 107; Nencioni, Canci and Catalano 2001; Killgrove 2008a and 2008b.

necropolis close to the via Nomentana contributes little to our understanding of the weight of this disease. Its actual prevalence can only be traced through biomolecular analysis. It is encouraging that DNA of *Mycobacterium tuberculosis* (as well as *M. leprae*, responsible for leprosy) has repeatedly been found in (provincial) Roman-period skeletons, and that the same is now also true of the *Yersinia pestis* bacterium that causes bubonic, pneumonic and septicemic plague. This suggests that similar discoveries may eventually be made on the outskirts of Rome.[19]

Weaning represented a considerable health hazard for small children and it is therefore important to be able to derive its timing from the study of dental remains: this task has already been accomplished for skeletons from Portus. Metropolitan air pollution also contributed to ill-health: the mummified body of a child from the second century CE found at Grottarossa, 7km north of Rome, exhibits severe anthracosis, a lung disease caused by the inhalation of particulate pollutants. However, further investigation of this phenomenon may not be feasible for the city of Rome. For instance, the detection of pleurisy in several bodies from Herculaneum, a disorder associated with indoor pollution from lighting, cooking, and heating, cannot readily be replicated elsewhere because it depends on exceptionally good skeletal preservation. Moreover, the DNA of pathogens that trigger intestinal diseases, which must have accounted for a large share of all deaths in Rome, cannot be recovered unless soft colonic tissue survives, which is not normally the case. To give another example of unique circumstances, carbonized foodstuffs from Herculaneum show high rates of microbial contamination, something that presumably also happened in Rome but cannot be empirically established. The same is true of lesser conditions such as pediculosis (lice infestation) that has been shown to have been common in Herculaneum.[20]

Despite these and other limitations, this line of research can hardly fail to put our understanding of Roman living conditions on a much more solid footing: we will know much more about what people ate, how healthy they were, and even where they came from. This information will reduce at least some of the uncertainties that I have highlighted throughout this chapter. In this respect, after generations of scholarship, we have the good fortune to be standing very much at the beginning.

FURTHER READING

Scobie 1986 remains the classic survey of living conditions in Rome (and other Roman cities). While deliberately focusing on the bleak side and thereby inviting allegations of bias, it has generally weathered well. Lo Cascio 2006 seeks to make the case for a more optimistic perspective, arguably with rather limited success. Scheidel 2003 surveys the disease environment in the imperial city of Rome. The epigraphic evidence for seasonal mortality is most fully set out by Shaw 1996 and 2006 and was first associated with malaria by Scheidel 1994. Sallares 2002: 201-34 gives the best account of malaria in ancient Rome. Scheidel 2001 surveys the general demographic background. The invaluable scientific work introduced in the final section still awaits synoptic treatment.

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[19] Sallares 2002: 67-8 (malaria); Canci et al. 2005 (osseous TB); Donoghue and Spigelman 2008 (DNA).

[20] FitzGerald et al. 2005; Prowse et al. 2008 (weaning); Ascensi et al. 1996 (mummy); Capasso 2000 and 2007 (Herculaneum). For soot from cooking in the capital, cf. Sen. *Epist.* 104.6.

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