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Leprosy at the Lazaretto on St Eustatius, Netherlands Antilles

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ABSTRACT Leprosaria established in the Americas during the Colonial period bear many similarities with those found in medieval Europe. They are comparable in terms of isolation, the objectification of leprosy sufferers and their association with religious charities. The Lazaretto on St Eustatius was operated from 1866 to 1923. The site was investigated to recover palaeopathological evidence of leprosy at a leprosarium in the Americas. Five burials were excavated; three individuals showed evidence of bone modifications consistent with those caused by leprosy, including aspects of 'rhinomaxillary syndrome' and the bilaterally symmetrical post-cranial changes that have been described in leprosy examples from medieval Europe. An exceptional find was the presence of potentially leprous bone changes to the hyoid, thyroid and 3rd–6th cervical vertebrae. Copyright © 2007 John Wiley & Sons, Ltd.

Key words: leprosy; palaeopathology; Colonial Caribbean; leprosaria

Introduction

Leprosy was first described in Chinese and Indian texts dating from 600–400 BC (Merbs, 1992). In northern Europe the most abundant historical evidence of the disease coincides with the marked population expansion during the early medieval Period; this is illustrated by written and artistic sources, an increase in leprosaria, and palaeopathological evidence (Boldsen, 2001). Palaeopathologists have studied several individual cases of leprosy dating to the 4th–10th centuries from the UK, Italy, Egypt, Hungary and from medieval Poland; larger collections come from medieval leprosy hospital cemeteries excavated in England, Sweden and Denmark (Reader, 1974; Thangarat, 1983; Møller-Christensen, 1983; Palfi, 1991; Belcastro *et al.*, 2005). By the end of the 15th century there appears to have been a decrease in leprosy prevalence in Europe; it is likely that this was due to a number of factors. It has been suggested that leprosy was spread to the Americas in the 16th century via European invaders, enslaved Africans, and later Chinese labourers (Aufderheide & Rodriguez-Martin, 1998; Lechat, 2002). There is no conclusive palaeopathological evidence that leprosy existed in the Americas prior to this time. As smuggling increased during the later slave-trading years, it is likely that the number of slaves with leprosy grew because their health was not assessed before leaving Africa (Scott, 1943). The Lazaretto on St Eustatius was opened in 1866, three years after slavery was abolished in the Dutch West Indies. The site had been surveyed and recorded by archaeologists from the College of William and Mary, Virginia, USA, in 1989, but no further investigations had been conducted. Therefore, this small 'leper colony' provided an ideal opportunity to examine palaeopathological

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evidence for leprosy in the Americas during the Colonial period. Five individuals were excavated at the Lazaretto on St Eustatius; all osteoblastic and lytic lesions were recorded in these remains.

Leprosy

Leprosy or Hansen's disease (after Gerhard Armauer Hansen who discovered the bacillus in 1873) is not highly infectious, but is spread through prolonged close contact (Grange, 1996). It is the 20% of leprosy sufferers that harbour the mycobacteria in their upper respiratory tract (especially the nose) that can spread infection to others (Jopling & McDougall, 1988). Dynamics that encourage the spread of *Mycobacterium leprae* include poor housing conditions, malnourishment and overcrowding, which together reduce cell-mediated immunity and therefore make individuals more susceptible to infection (Jopling & McDougall, 1988). The biological factors that determine whether someone who has been exposed to *M. leprae* will develop leprosy and what form the leprosy will take are still poorly understood (Grange, 1996).

Upon contact with *Mycobacterium leprae* there is considerable variability in how the individual will respond to the infection. At one end of the scale is the paucibacillary form (or tuberculoid leprosy), where the immune response is high, and at the other end is the multibacillary form (or lepromatous leprosy) where there is little or no resistance to the mycobacteria (Stanford & Stanford, 2002). It is the multibacillary form where the most severe effects to the body will occur. Leprosy is the only bacterium that can infect nerves – in fact it can infect many cell types. It prefers temperatures below 37 C – hence the bacteria locate in the subcutaneous nerves, eyes, larynx and testes (Aufderheide & Rodriguez-Martin, 1998). Paucibacillary leprosy can manifest itself in a single skin blemish or a peripheral nerve; here the immune response is able to stop the infection from spreading, whereas in multibacillary leprosy the infection is able to multiply unchecked (Jopling & McDougall, 1988). The nerve damage that is caused by multibacillary leprosy begins with sensory nerve loss (Palfi, 1991), and as the disease progresses motor nerves are also infected. The flexors and extensors of the hands and feet become paralysed, which causes 'claw hand' and 'drop foot' deformities. Posterior tibial nerve function deterioration is very common in leprosy, and this in turn gives rise to the collapse of the longitudinal arch and the development of flat foot (Andersen & Manchester, 1988). The combination of sensory and motor nerve loss and paralysis of the hands and feet leads sufferers to inadvertently injure themselves. This repeated injury is often followed by chronic ulceration and secondary infection by *streptococcus* or *staphylococcus* bacteria (Andersen & Manchester, 1987).

Evidence of leprosy in palaeopathology

Vilhelm Møller-Christensen (1953, 1961, 1978, 1983) conducted the earliest comprehensive leprosy studies in palaeopathology. These studies were based upon four Danish leprosaria excavations, and his leprosy case investigations in the UK and France. Møller-Christensen (1961) was the first to identify characteristic changes to the maxillae, hands, feet and lower legs that could be associated with leprosy infection. He considered the changes to the face as pathognomonic of leprosy and used the term '*facies leprosa*' to describe them. Johannes Andersen and Keith Manchester (1987, 1988, 1992; Andersen *et al.*, 1992; Manchester, 1991, 2002) redefined these rhinomaxillary changes and further explored the pathogenesis of the secondary post-cranial changes seen in the disease. The bilaterally symmetrical lesions caused by multibacillary leprosy have been studied in most detail and are described below. In contrast, palaeopathological evidence for paucibacillary leprosy is likely to be in the form of a solitary unilateral lesion that might be associated with an infected nerve; the changes associated with rhinomaxillary syndrome are not present (Aufderheide & Rodriguez-Martin, 1998).

Rhinomaxillary syndrome

The bone changes described by Møller-Christensen (1961) as '*facies leprosa*' were' identified

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in the collection from St Jørgens churchyard at Næstved, Denmark. They included:

(1) Atrophy of the anterior nasal spine.
(2) Resorption of the alveolar process of the maxillae, which begins centrally and may continue to the extent that the incisors are lost.
(3) Remodelling of the nasal aperture and destructive changes to the nasal conchae, vomer and perpendicular plate of the ethmoid (Møller-Christensen, 1983).
(4) An inflammatory reaction on the nasal and oral surfaces of the palate in the form of closely spaced pits centred on the palatine suture. The palate may be perforated (Møller-Christensen, 1961).

Møller-Christensen described leprosy pathogenesis on bone in some detail, particularly the processes of bone destruction, absorption and deposition. He also differentiated between the skeletal manifestations that might be directly caused by leproma formation and those caused by leprous neuritis and secondary infection (Møller-Christensen, 1961).

Andersen & Manchester (1992) have suggested using 'rhinomaxillary syndrome' as an appropriate term for describing bone changes to the skull, arguing that *'facies leprosa'* is associated with soft tissue lesions. Although the features of rhinomaxillary syndrome are very similar to those described earlier by Møller-Christensen, Anderson & Manchester have altered some of the terms used in describing the bone changes in order to reflect clinical and palaeopathological advances in the understanding of the processes involved in the way *M. leprae* affects bone. They describe the changes associated with rhinomaxillary syndrome as follows: the intranasal structures, particularly the nasal septum and conchae become pitted and resorbed, and the margins of the nasal aperture become rounded due to bilateral symmetrical resorption and remodelling (Andersen & Manchester, 1992). Destruction of alveolar process of the maxilla begins at prosthion, the anterior nasal spine becomes pitted and is gradually destroyed, and finally, erosive pits converge around the mid-zone along the palatine suture, and may perforate through the palate (Andersen & Manchester, 1992).

The features seen in rhinomaxillary syndrome are thought to be due to direct infection with leprosy bacteria; however, Møller-Christensen (1961) suggested that anterior nasal spine resorption might be due to a disruption in nerve supply and therefore it may be similar aetiologically to the diaphyseal remodelling of the hand and foot bones. While most of the bone changes that occur in the rhinomaxillary area are considered to be the product of *M. leprae* infection (leprous granuloma) in the nasal mucosa, pathological changes to the hands, feet and lower legs are due to a combination of factors.

Post-cranial evidence of leprosy

The factors involved in the post-cranial bone changes caused by leprosy include: repeated trauma, impaired blood and nerve supply to the bones, haematogenous spread of *M. leprae*, and secondary infection from pyeogenic bacteria in adjacent skin ulceration, which leads to osteomyelitis and septic arthritis (Jopling & McDougall, 1988). The types of post-cranial lesions caused by leprosy are not unique; however, the combination of lesion types and their bilaterally symmetrical distribution has been described as pathognomonic of leprosy.

Diaphyseal remodelling

Due to leprous neuritis and poor blood supply, the diaphyses of the phalanges of the hands and feet and the metacarpals and metatarsals are resorbed and remodelled. This remodelling may be concentric or medial and lateral, so that the diaphyses become knife-edged and gradually taper to points. Andersen *et al*. (1992) discovered that whilst the cortical bone was being absorbed, there was also endosteal new bone deposited, which would gradually reduce the medullary cavity to nothing. They also stated that this process may be the result of neurovascular dysfunction and is very different from atrophy, where changes are due to disuse and the cortex becomes thin and osteoporotic (Andersen *et al*., 1992).

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Other changes to the phalanges of the hands and feet include the proximal articular surfaces of the phalanges. These become cup-shaped due to subluxation and circumferential osteophyte formation. The circumferential osteophyte formation at the proximal ends and resorption of the distal ends of the distal phalanges of both the hands and feet also creates a 'shark-tooth' shaped deformity. The interphalangeal joints of the hands may also become fused due to pathological fracture and sepsis. Additionally, volar grooves (visible as delineated depressions) may be found on the proximal phalanges of the hands adjacent to the proximal interphalangeal joint. Andersen & Manchester (1987) suggested bony changes will occur at this location as a result of pressure caused by interphalangeal joint hyperflexion, due to flexor and extensor paralysis.

Tarsal bars

With longitudinal arch collapse, the mechanical stresses placed upon the foot change, triggering an osteoblastic reaction to alleviate pressure on certain areas of the foot (Andersen & Manchester, 1988). Tarsal bars can be identified as a ridge of new bone that extends transversely across the dorsal surface of the tarsus as a response to intertarsal subluxation (Andersen & Manchester, 1988). In their suspected leprosy case from 7th century Italy, Belcastro *et al*. (2005) found exostoses at the sites of ligamentus attachment to the navicular and calcaneous, which was probably due to chronic stress caused by motor paralysis and proprioception loss.

Tibial and fibular changes

Møller-Christensen (1961) found that 74% of the tibiae and fibulae examined from excavations at Næstved, Denmark, exhibited extensive changes including protruding osseous deposits, vascular grooves and punctuate depressions. Subperiosteal bone formation often occurs in the distal tibiae and fibulae in leprosy, and is bilaterally symmetrical, with the most prolific changes usually along the interosseous border. According to Manchester (2002) this is due to secondary infection in the foot and is frequently associated with longitudinal arch collapse and midfoot plantar ulceration.

Summary of pathological changes

The pathological lesions observed in three (2, 4 and 5) of the five skeletons uncovered at the Lazaretto are described here. In recording and analysing the five skeletons, notes were made on the number of bones, teeth and joints present, and overall preservation and completeness. Assessment of sex was possible using standard pelvic and cranial morphological features as outlined in Buikstra & Ubelaker (1994). Estimation of age-at-death was primarily based upon changes to the pubic symphyses and auricular surfaces using Suchey-Brooks (Brooks & Suchey, 1990) and Lovejoy *et al*. (1985). All bony changes were recorded individually according to their location and lesion type; terminology is descriptive, not diagnostic.

Skeleton 2 was 90% complete, although the long bones were fragmentary, particularly around the joints such as the knees. This individual is probably female, using the criteria mentioned earlier. Ageing this specimen was based upon the methods described above, as well as epiphyseal fusion and tooth eruption. The medial epiphyses of the clavicles had not fused, which is usually complete by age 25 (Bass, 1995). However, the third molars were fully erupted. Age-at-death was estimated to have been between 20–25.

Skeleton 4 was in a variable state of preservation, as some bones were almost completely intact, while others were very fragile and disintegrated readily. The skeleton was probably about 80% complete. The sex of skeleton 4 was recorded as indeterminate due to post-mortem damage to the skull and pelvis. Estimation of the age-at-death of this specimen was possible using the pubic symphyses; surface changes correlated with a 40–50 age range. Despite the poor preservation of the skull and pelvis, the hands, feet and lower legs were in good condition.

Skeleton 5 was 95% complete, and despite some fragmentary bones the cortical surfaces were in good condition for examination. The sex of this specimen is indeterminate because only

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the sciatic notch was preserved out of the pelvic features. This was quite wide; however, the cranial features gave a mixture of male and female characteristics. Age-at-death was estimated to have been 40–50 using the features noted on one auricular surface.

Cranial pathological changes

Bone changes to the cranium of skeleton 2 included slight remodelling of the nasal aperture and porosity on the interior surface of the nasal aperture. The palatine process of this specimen was destroyed post-mortem, and the anterior nasal spine also appeared to have sustained post-mortem damage, so it was impossible to examine these areas for changes. Examination for pathological changes to the skull of skeleton 4 was not possible due to post-mortem damage. The skull of skeleton 5 also sustained some post-mortem damage, but pathological changes could be observed. Changes to skeleton 5 included an enlarged nasal aperture that was smooth and rounded. The labial wall of the maxillary alveolar process above the central incisors was pitted and thinned, which created a fenestration, exposing the central incisor roots. The anterior nasal spine was present, but appeared to have been reduced. There was also slight pitting in the joint surface of the left mandibular notch, on the left inferior nasal concha and on the oral surface of the palate, which was particularly porous around the anterior midline suture. Medium-coarse pitting was also present on the posterior hyoid body surface and on the bodies of the 3rd–6th cervical vertebrae. The vertebral bodies also had abnormal bone nodules on their surfaces and the thyroid had loose woven bone formation.

Upper extremities

Volar grooves, which are visible as delineated depressions, were found on the proximal phalanges adjacent to the proximal-interphalangeal joint (right hands of skeletons 2 and 4, and both hands of skeleton 5). The phalanges from all of the skeletons also exhibited cup-shaped proximal joint surfaces. This was particularly pronounced on skeleton 5, where the intermediate and distal phalanges of both hands had very narrow shafts and osteophytes around the joint surfaces (see Figure 1). The diaphyses of the metacarpals

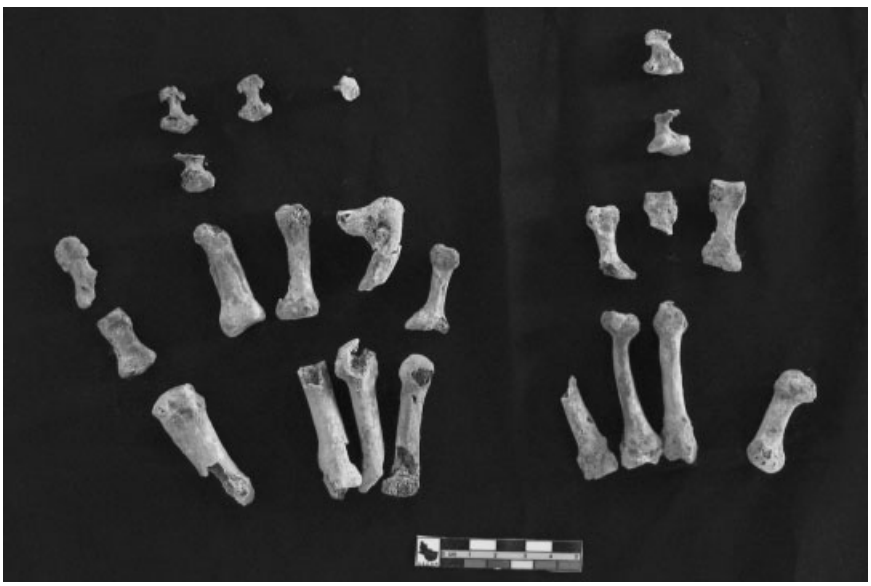

Figure 1. Hands of skeleton 5 (palmar view), showing the fused 4th left proximal interphalangeal joint, volar grooves (4th right proximal phalanx), narrowed diaphyses and circumferential osteophyte formation.

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(skeleton 4), and the proximal, intermediate and distal phalanges (of skeletons 2, 4 and 5), had been resorbed and concentrically remodelled. The right 5th intermediate and distal phalanges of skeleton 4 had fused straight at the joint and the left 4th proximal-interphalangeal joint of skeleton 5 had fused at 90°.

Pathological changes to the radii and ulnae of skeletons 4 and 5 were also recorded. Coarsely porous areas of bone surface were observed on the left humerus between the trochlea and the medial epipcondyle, and in the olecranon fossa of skeleton 4. This specimen also had a smooth-walled depression or pressure erosion (7.2 mm) in the semilunar notch of the left ulna. Skeleton 5 also had porosity and a smooth-walled depression (1.5 mm diameter) in the radial notch of the right ulna. There was also coarse porosity (1 mm diameter pores covering 50% of the joint surface) on the head of the right radius of this specimen. The left ulna had similar changes beneath the radial notch, although they were less marked. Nodules of sub-periosteal bone growth were present beneath the radial notch and extending down the interosseous crest of skeleton 5. Plaques of white, smooth, but slightly porous compact bone were observed on the visceral surface of the ribs of skeleton 2.

Lower extremities

Abnormal sub-periosteal porous, striated and nodular woven and compact bone growth was present on the distal surfaces of the tibiae and fibulae of skeletons 2, 4 and 5, particularly along the interosseous border. The right tibia and fibula of skeleton 4 in particular had prominent nodules of abnormal bone formation along its entire length (see Figure 2). Similar bone changes were present along the length of the left fibula and the anterior crest of the left tibia, although they were less marked.

Only three phalanges of skeleton 4 survived post-mortem, but these and all of the tarsals and metatarsals were in good condition. The feet of skeleton 4 showed especially striking bone changes, including cyst-like depressions (such as on the right 2nd metatarsal (7.3 mm)), extensive bone destruction and remodelling. Most of the surfaces of the tarsals in particular were strongly deformed; the changes were so severe that the orientation and architecture of the tarsal joints had altered. The non-articular surfaces of the tarsals of skeleton 4 and 5 were porous and there was marked abnormal bone growth around all the joint surfaces. On skeleton 5 there were abnormal woven bone growths on the posterior and plantar surfaces of the left calcaneus. Both cuboids had reactive woven bone formation on the medial surfaces, and porosity on the superior surface. There was a ridge of remodelled bone along the plantar surface of all cuneiforms in skeleton 5. The metatarsal diaphyses and the proximal phalanges of skeletons 2, 4 and 5 were narrowed, particularly medially and laterally, which gave the proximal joint surfaces of the phalanges a cup-shaped appearance (see Figure 3). The fifth right metatarsal of skeleton 4 had narrowed so much that the distal diaphysis and joint surface had completely disappeared. Smooth-walled cyst-like cavities were also

Figure 2. Nodules and striated sub-periosteal bone formation along the entire length of both the tibia and fibula of skeleton 4 (posterior view), particularly along the interosseous border.

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Figure 3. Feet of skeleton 5 (dorsal view), showing medially and laterally narrowed diaphyses of the metatarsals and the proximal phalanges, and cup-shaped proximal joint surfaces.

observed on tarsals, metatarsals and phalanges of both feet of skeleton 5.

Differential diagnosis

The pathological lesions observed in skeletons 2, 4 and 5 at the Lazaretto may be due to a number of different causes. Historical documents, such as from the Leprosarium in Spanish Town, Jamaica, suggest that the poor, mentally disabled, and people suffering from syphilis, yaws, tuberculosis, psoriasis and elephantiasis were also housed in these colonial asylums (Abraham, 1889). Therefore, it is possible that evidence of other conditions can be found in human remains uncovered at 'leper colonies' at least throughout the Caribbean. In some cases, there is no evidence of the illnesses that patients suffered from during life present in the osteoarchaeological remains.

Since bone is limited in its response to disease or trauma, diagnosis of the cause(s) of pathological changes should be founded upon an accurate description of lesion types (whether they are destructive or prolific, active or remodelled, for example) and their precise location on individual bones as well as their distribution throughout the entire skeleton. For a differential diagnosis, diseases that cause similar lesions to those observed in these three specimens are examined.

Changes to the rhinomaxillary area in skeletons 2 and 5 included bilaterally symmetrical pitting and remodelling of the nasal aperture, and pitting of the labial wall of the maxillary alveolar process. These osteolytic lesions are likely to have been chronic because they present both bone destruction and remodelling. Fungal infections, such as *Murcormycosis*, *Cryptococcus* and *Actinomycosis*, often cause bone destruction; however, there is often little or no marginal repair or remodelling. These infections can affect many bones including the cranial bones (Belcastro *et al.*, 2005). *Murcormycosis* in particular can affect the rhinomaxillary area, including the nasal cavities, maxillary sinus and palate (Ortner & Putschar, 1981). However, in contrast to the changes displayed in skeletons 2 and 5, lesions caused by *Murcormycosis* (and all fungal infections) are unilateral. Therefore, the remodelled lesions observed in the rhinomaxillary area in skeletons 2 and 5 and the bilaterally symmetrical post-cranial lesions observed in all three skeletons are not likely to have been caused by a fungal infection.

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Sarcoidosis is a granulomatous disease that can affect the nasal bones. This disease causes lytic lesions, with little or no reactive bone formation (Ortner & Putschar, 1981). It is not a likely explanation for the bone changes found in these three specimens, both because of the type of lesions found and their distribution. The bone changes found in the rhinomaxillary area of skeletons 2 and 5 (and throughout all three skeletons) are indicative of recurrent disease. In contrast to sarcoidosis, bone remodelling was present around the nasal aperture, and in the diaphyses of the hand and foot phalanges, tarsals, metatarsals and metacarpals. Moreover, sarcoidosis cases often exhibit lesions located in multiple vertebral bodies. No destructive lesions were found in the vertebrae of these three skeletons.

Diabetes is an endocrine disorder that affects the bones of the feet and lower legs. Diabetes causes osteolysis of the distal metatarsals and proximal phalanges, so that they become tapered (Belcastro *et al.*, 2005). While these changes are similar to the concentric remodelling found in these three specimens, the combination of lesions identified is different from those caused by diabetes, both in type and distribution throughout the skeleton. For example, diabetes does not affect the hands or the facial bones. Moreover, reactive new bone formation and evidence of secondary infection is not likely to be found in association with the osteolytic lesions found in diabetes cases.

Septic arthritis causes the destruction of bone and fusion of joints; however, as it is caused by a localised infection, pathological changes are unilateral. In both skeletons 4 and 5 there was evidence of fusion of the phalanges of the hand. This may be caused by septic arthritis, although as the post-cranial pathological changes in both specimens are bilaterally symmetrical, septic arthritis is likely to have been *secondary* to paralysis and injury caused by another condition.

Tuberculosis, caused by *Mycobacteria tuberculosis* or *M. bovis*, produces both bone destruction and formation, but predominantly destruction (Roberts & Buikstra, 2003). The facial bones can be affected in tuberculosis as a result of soft tissue infection (known as *lupus vulgaris*). However, lesions in this location are unlikely to be bilateral and nor would they involve the alveolar process and the palate, as seen in skeletons 2 and 5. The most frequent sites for cranial lesions caused by tuberculosis are the frontal and parietal bones, where lytic lesions initiate on the inner table of the skull. No lesions were found in this location in the three skeletons from the Lazaretto. Tuberculosis also primarily affects the spine, with 40% of lesions found in this location (Aufderheide & Rodriguez-Martin, 1998). The weight-bearing joints such as the hips and the knees are also affected (Roberts & Buikstra, 2003). Destruction of these joints (or of joints such as the ankle or wrist) was not present in these skeletons and bone changes were not unilateral, as found in tuberculosis. Additionally, the changes to the phalanges of the hands and feet in skeletons 2, 4 and 5 are not likely to have been caused by tuberculosis, as tubercular lesions are rarely found in these locations in adults.

Treponemal diseases are caused by spirochetes of the genus *Treponema*. The bone changes to the rhinomaxillary area, lower limbs and feet in the third stage of treponemal infections can be confused with those caused by leprosy. According to Steinbock (1976) the majority of skeletal pathology in treponemas is non-specific; only lesion types such as *caries sicca* can be considered pathognomonic. These shallow, pitted and remodelled cortical lesions on the cranial vault were not found in skeletons 2, 4 or 5. Rhinomaxillary changes can occur in venereal and endemic syphilis and yaws. The changes include remodelling of the nasal aperture and can include considerable destruction of the nasal and palatal areas. Cook (2002) suggested that some of the features of 'rhinomaxillary syndrome' are not specific to leprosy and can be found in treponemal infections, particularly the destruction of the anterior nasal spine. However, Andersen & Manchester (1992) argued that *all* the aspects of 'rhinomaxillary syndrome' are unlikely to be present in a case of treponemal infection. Skull vault lesions are rarely found in leprosy, and treponemal infections do not have the same distribution and type of post-cranial lesions that are caused by leprous neuritis and secondary infection in leprosy. When comparing leprosy with the treponemal syndromes, the distribution of lesions throughout the entire skeleton must be considered, as this is unique.

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In both yaws and endemic syphilis, gummatous lesions can be found on the long bone shafts, particularly on the tibia, fibula, radius, ulna and femur (Powell & Cook, 2005). These destructive focal areas of necrosis were not found in the long bones of skeletons 2, 4 or 5. Venereal syphilis can be identified by the presence of 'Charcot's joints', which is the destruction of the weight-bearing joints such as the hips, knees and ankles, with the production of new bone. There was no destruction of the weight-bearing joints in these three specimens.

The only post-cranial bone changes that were found in skeletons 2, 4 and 5 that match the distribution found in treponemal infections is the deposition of sub-periosteal new bone on the tibia and fibula. In yaws in particular, distinctive bone deposition and remodelling occurs along the anterior crest of the tibia, producing a 'saber skin' deformity (Powell & Cook, 2005). In contrast, the concentration of sub-periosteal new bone on the tibiae and fibulae of skeletons 2, 4 and 5 was located along the interosseous border and there was no anterior bowing.

Psoriatic arthritis is an erosive arthropathy. Diagnosis of psoriatic arthritis is possible with the presence of sacro-iliac and intervertebral joint fusion (Zias & Mitchell, 1996). There is also no involvement of the cranial bones. Psoriatic arthritis could be the cause of lesions found in the hands and feet of the Lazaretto skeletons. Often the distal and proximal interphalangeal and metatarsophalangeal joints are involved (Rogers & Waldron, 1995), where lytic lesions can lead to joint deformation. However, psoriatic arthritis is not considered to be the cause of the bone changes in these skeletons because there is no evidence of sacro-iliac or intervertebral joint fusion, changes to the phalanges are not limited to the joints, and the diaphyses were concentrically remodelled. Moreover, changes in psoriatic arthritis are asymmetrically distributed, which was not the case in these specimens.

Discussion of diagnosis

The three skeletons described here display a range of pathological changes that are consistent, in both nature and distribution, with a chronic infectious disease such as leprosy. The changes to the rhinomaxillary area in skeleton 5, combined with the bilaterally symmetrical post-cranial bone changes found in both skeletons 4 and 5, are strongly suggestive of the presence of multibacillary leprosy. Skeleton 2 also showed changes that are characteristic of leprosy; however, they were less dramatic, and rhinomaxillary changes were minimal.

As leprosy is a chronic disease, where there are periods of severe reaction and quiescence, it is possible that this individual (skeleton 2) died before bone changes had become more pronounced (age-at-death was estimated to have been 20–25). Thin plaques of new bone on the visceral surface of the ribs suggest that perhaps this individual had a pulmonary infection or immune response to *M. leprae* infection in the lungs. Tuberculosis is one of the main causes of death for leprosy sufferers today (McDougall, 2002). Pleural rib lesions have been correlated with tuberculosis in archaeological remains; however, they may actually be due to other pulmonary infections (Mays *et al.*, 2002). Alternatively, the bilaterally symmetrical post-cranial changes combined with slight modifications in the rhinomaxillary area may be indicative of the individual's greater immune response. If this individual had slightly higher resistance to *M. leprae*, clinically they may have been classed as having borderline multibacillary leprosy today.

Rhinomaxillary changes to skeleton 2 included slight remodelling of the nasal aperture and porosity of the interior surface of the nasal aperture. The pathological changes seen in skeleton 5 that are part of the 'rhinomaxillary syndrome', as described by Andersen & Manchester (1992), include remodelling of the nasal aperture and the anterior nasal spine, and thinning of the maxillary alveolar process. Osteolytic bone changes and osteoblastic remodelling in this location are caused by the presence of leprous granuloma in the nasal cavity.

The post-cranial bone changes exhibited by skeletons 2, 4 and 5 include volar grooves. These were described by Andersen & Manchester (1987) as being the result of motor nerve paralysis of the flexors and extensors of the hand. Interphalangeal joint hyperflexion puts pressure on the bone adjacent to the joint, which is remodelled to

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alleviate this pressure. Interphalangeal joint fusion observed in skeletons 4 and 5 may have been caused by septic arthritis due to dorsal trauma and infection resulting from motor and sensory nerve damage caused by *M. leprae* infection. Both septic arthritis and smooth-walled cysts (observed in the radii and ulnae and feet of skeletons 4 and 5) can be found in leprosy cases (Jopling & McDougall, 1988). These are caused by pathological fracture and invasion by pyogenic bacteria as a result of sensory nerve loss in leprosy.

Concentric remodelling, cupping of joint surfaces and erosion of the ends of the distal phalanges was found in the metacarpals and phalanges of skeletons 2, 4 and 5. The diaphyses of the metatarsals and proximal phalanges of the feet in these three skeletons were also remodelled (mainly on the medial and lateral surfaces, so that the diaphyses became narrower). Diaphyseal remodelling of the bones of the hands and feet in leprosy sufferers is caused by a number of factors. Jopling & McDougall (1988) identified these factors as follows: repeated trauma; impaired blood supply due to endarteritis of nutrient vessels; impaired nerve function; haematogenous spread of *M. leprae* to bone; osteoporosis in males due to testicular atrophy; reduced osteoblastic activity; and osteomyelitis due to chronic ulceration of the skin. These processes also lead to erosion of the ends of the distal phalanges and circumferential osteophyte development, creating the characteristic 'shark-tooth' shaped deformity observable in skeletons 4 and 5.

'Tarsal bars' (visible in skeletons 4 and 5) are an osteoblastic reaction to longitudinal arch collapse. The effects of longitudinal arch collapse and changes in foot architecture in leprosy are especially noticeable at sites of ligamentus attachment; this has not been noted in other aetiologies of flat foot (Jopling & McDougall, 1988). The tarsals of skeleton 4 in particular were completely deformed by reactive bone growth due to changes in the pressure placed upon the joint surfaces and intertarsal subluxation. Inflammatory pitting and dorsal bar formation on the tarsals is caused by muscle paralysis and soft tissue infection found in leprosy sufferers. According to Ortner & Putschar (1981), the feet are often more severely affected than the hands in leprosy sufferers. This was the case in skeletons 4 and 5, as the feet are more likely to have suffered from repeated trauma and chronic plantar ulceration.

Finally, secondary periostitis on the tibiae and fibulae is associated with chronic infection of the foot (Manchester, 2002). Periosteal new bone formation in leprosy is bilaterally symmetrical and the most prolific changes are usually along the interosseous border. Belcastro *et al*. (2005) found sub-periosteal bone changes on the tibia and fibula in their specimen from 7th century Molise, Italy. They stated that the changes were most pronounced distally and were in the form of pitting and longitudinally striated deposits. All three skeletons uncovered at the Lazaretto had striated and nodular remodelled bone deposition along the distal fibulae and tibiae, particularly along the interosseous border.

A feature not previously noted in palaeopathological literature was pitting of the posterior of the body of the hyoid and the bodies of the 3rd–6th cervical vertebrae in skeleton 5. In adult males the hyoid is adjacent to the epiglottis, just above the larynx, which is opposite the 3rd–6th cervical vertebrae (Williams, 1995). The posterior surface of the hyoid is normally smooth and is separated from the epiglottis by the thyrohyoid membrane and loose areolar tissue (Williams, 1995). Osteoarthritis is often diagnosed by the presence of pitting, eburnation and osteophytic outgrowths. This specimen did not have any eburnation in this area and had minimal osteophytic outgrowths around the vertebral bodies. Therefore, it is possible that osteolytic lesions and loose woven bone formation found in the hyoid, 3rd–6th vertebral bodies and on the thyroid cannot simply be explained by osteoarthritis, but were caused by leprosy.

Leprosy causes lesions of the larynx, as the mycobacteria infiltrate the epiglottis and the vocal cords. It also causes acute laryngeal oedema during leprous reactions (Thangarat, 1983). In their study of 1111 nerve trunks from 93 autopsies, Tze-Chun & Ju-Shi (1984) found that the distribution and incidence of leprous lesions in the upper respiratory tract was as follows: nose 45.5%, nasopharynx 36.4%, larynx 55%, and the trachea and bronchi 12.1%. In Soni's (1992) ear, nose and throat examination of 30 patients with untreated multibacillary leprosy, laryngeal

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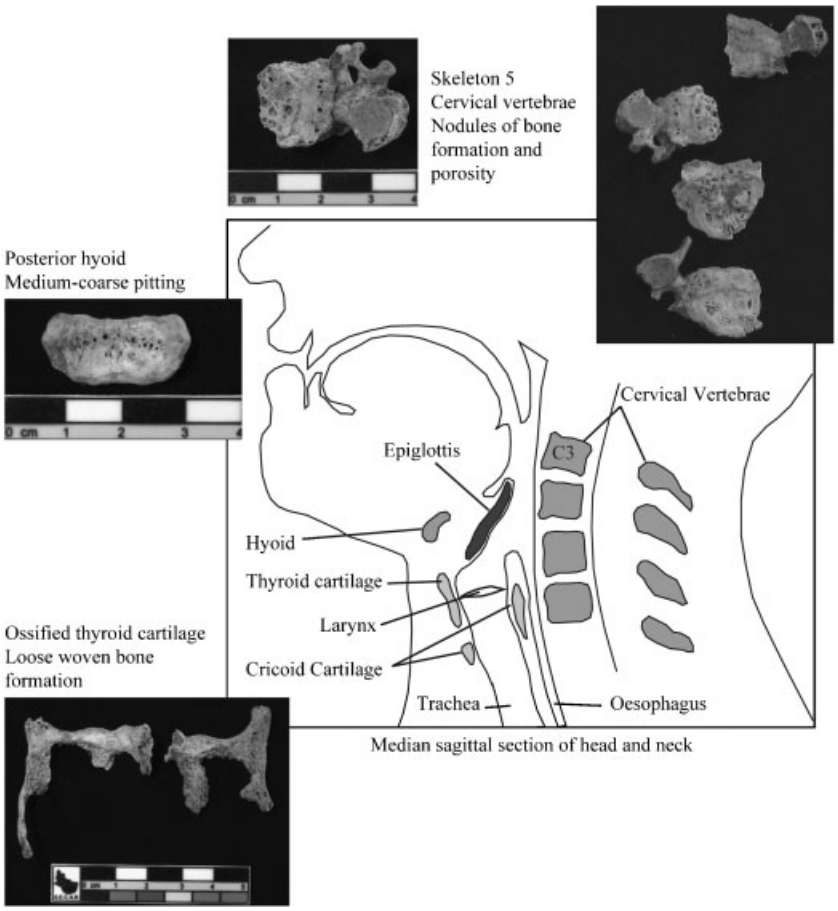

Figure 4. Pathological changes to the hyoid, thyroid and cervical vertebrae, showing their location in relation to the larynx and epiglottis.

pathology was found in 11 cases (36.6%), with the epiglottis being the most frequent site of involvement. The epiglottis was found to be thickened, nodular, ulcerated, and in some cases was deformed and destroyed due to infiltration of *M. leprae* (Soni, 1992). It is significant that all the bones (hyoid, cervical vertebrae and thyroid) in proximity to the larynx and epiglottis were affected by pathological changes (see Figure 4). The changes to the hyoid, thyroid and 3rd–6th cervical vertebrae are suggestive of leprous infection in the respiratory tract of skeleton 5.

Conclusion

Skeletal changes that have been documented from medieval examples diagnosed as leprosy in Europe were also found in three skeletons found at the Lazaretto on St Eustatius. These were identified on the basis of the *characteristic distribution* and specific nature of the *different types* of pathological lesions that are consistent with those caused by leprosy. The inflammatory lesions found in the post-cranial skeleton and in the rhinomaxillary area are similar to those found in other diseases; however, their distribution is pathognomonic of leprosy (Manchester, 2002). Bone changes that result from the infection of the larynx (as seen in skeleton 5) have not been described before in palaeopathology. Tze-Chun & Ju-Shi (1984) illustrated that in the upper respiratory tract, most lesions are found in the larynx, and therefore the hyoid may also be a significant area to examine for the presence of leprosy in palaeopathology, as well as the 3rd–6th cervical vertebrae and ossified thyroid cartilage.

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