FFAR 124S5 ACB

LAB REPORT:

Skeleton ONE94 519

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ABSTRACT

Skeleton ONE94 519 was analysed to establish sex, age, stance and possible pathological conditions. Both morphological and metric methodologies were used with results suggesting that the individual was a male who died after the approximate age of 30. Skeletal indicators suggest he was approximately 1.70 metres in height and that he suffered from poor dental health, rickets and osteoarthritis of the left wrist.

INTRODUCTION

The following report presents findings concerning skeleton ONE94 519. The skeleton was analysed to establish sex, age and possible pathological conditions. The skeleton was excavated by MOLAS between 1994 and 1996 from the site of St Benet Sherehog, London, also known as '1 Poultry'. The original church on the site was destroyed during the Great Fire of 1666 and records show that from this time the land was used as a burial site for all classes of society. The site was closed in 1853 after an act of Parliament. Skeleton ONE94 519 is believed to date from the 16th or 17th century. All post-medieval individuals retrieved from the site were buried in coffins which were roughly aligned east to west with a number of copper alloy shroud pins being recovered during the excavation. (Museum of London)

INVENTORY

BONE	PRESENT
SKULL	
Frontal	YES
Parietal	YES
Occipital	YES
Temporal	YES
Zygomatic	PARTIAL*
Maxilla	PARTIAL*
Mandible	YES
Sphenoid	YES
VERTEBRAL COLUMN	
Cervical	YES
Thoracic	YES
Lumbar	YES
Sacrum/coccyx	YES
RIBS	
Right	YES*
Left	YES*

STERNUM	YES
CLAVICLE	
Right	YES*
Left	YES
SCAPULAE	
Right	PARTIAL*
Left	PARTIAL*
HUMERI	
Right	YES
Left	YES
RADII	
Right	YES
Left	YES
ULNAE	
Right	YES
Left	YES
HANDS	
Right	YES*
Left	YES*
PELVIS	
Right	YES*
Left	YES
FEMORA	
Right	YES
Left	YES
PATELLA	
Right	YES
Left	YES
TIBIAE	
Right	YES
Left	YES
FIBULAE	
Right	YES*
Left	YES*
FEET	
Right	YES*
Left	YES*

*Table 1: Bone inventory. *See text below for specifics.*

The skeleton is almost complete with some of the bones present but in a broken form. Breakages appear to be post-mortem:

- The right zygomatic bone is not attached to the skull but is present in fragments
- The right side of the maxilla bone is not attached to the skull but is present in fragments
- The right clavicle is broken in half at the acrominal end

- The right scapula is present but the main body is broken into two pieces over the subscapular fossa and oblique ridges. The left scapula is in fragments
- 24 costal rib ends can be identified, with nine ribs complete (six left and three right) and the remaining in fragments
- Some bones are missing from the hands. Four carpals are missing from the right hand. One carpal is missing from the left along with two proximal, two intermediate and two distal phalanges
- The right pelvis is broken across the illium, while the left pelvic bone has a crack in the same region
- Both fibulae are broken halfway along the shaft
- Some bones are missing from the feet. Two of the right metatarsals are broken and three of the proximal and four of the intermediate phalanges are missing. From the left foot two proximal and two distal phalanges are missing

There is a small amount of intrusive bone which appears to be human.

PRESERVATION

The bone preservation overall is good but with post-mortem erosion evident on the edges of a number of bones. For example, around both tibial plateaus; the majority of the bones of the feet; the right ulna's olecrannon process; the right carpals; the distal ends of both femur; the transverse processes of a number of the lumbar vertebrae and above both pubic symphysis. Green and brown staining is evident in some areas with a possible cause for such stains being copper alloy shroud pins and iron coffin nails.

Skull

The skull exhibits markings. There is a black spot on the right parietal bone, bordering on the temporoparietal suture. There are what appear to be oblong soil marks at the back of the left parietal bone and ringed/circular black stains at the top and centre of the left parietal bone.

Post-mortem lesions are visible on the skull. There is a small patch in the middle of the left temporal bone; a small pit at the back of the left parietal bone above the lambdoid suture and a small patch at the front of the right parietal bone near the intersection of the coronal and sagittal sutures.

There is slight, regular pitting of the frontal, parietal and occipital bones. The right zygomatic bone and the right side of the maxilla bone are broken away from the skull. The fragments were retrieved during excavation and remain with the skeleton.

Other features

Green stains are to be seen on the anterior of the sternum, on the left along the second costal notch; on the anterior neck of the left femur; on the left triquetral carpal and on the left, lateral of the T-10 vertebrae (see figure 1).

Two rib fragments display black ringed/circular staining similar to that on the skull. The right humerus displays a brown stain, possibly from an iron coffin nail, as does the right trapezium carpal; the right pelvis at the inferior pubic ramas and the posterior of the ilium and the left hip on the posterior of the ilium. White streaks are also evident across the posterior of the left ilium. The area above the left inferior pubic ramas shows post-mortem damage along with brown staining.

Both fibulae are broken halfway along each shaft. These breaks appear to be post mortem.

SEX

Methodology

A number of morphological sexing methods were employed to gauge the sex of ONE94 519. These methods consider the skull and pelvis. A metric method was also employed using Pearson's femur measurement system.

<u>Skull</u>

The cranium was examined and points were scored according to features specific to each method. The methods used are described below.

From Walker in Buikstra and Ulbelaker 1994: Points were scored from 1 (female) to 5 (male) depending on particular features (White & Folkens, p.390-391).

- Nuchal crest – a smooth external surface of the occipital scores 1 with a bony point or ledge protruding from the skull scoring 5

- Mastoid process – this feature's size is compared with surrounding features such as the external auditory meatus. A small mastoid process scores 1 with a large one scoring 5
- Supra-orbital margin – the thickness of the edge of the orbit is considered here. A sharp edge scores 1 and a think edge scores 5
- Supra-orbital ridge – the minimal protuberance of the glabella scores 1 and a well-developed protuberance scores 5
- Mental eminence – no projection of the mental eminence above the bone scores 1 while one that takes up most of the anterior area of the mandible scores 5

From Mays 1998 (p.37):

- Forehead and brow shape – the brow is more developed in the male and the forehead is more sloping

From Rosing et.al. 2007 (p.80):

- Orbit shape – the male is more rectangular with the female more rounded
- Mandible muscle – the male may show muscle marks and flaring while the female will be smooth

<u>Pelvis</u>

The pelvis was examined and points were scored according to features specific to each following method:

From Phenice 1969 (White & Folkens, p.397):

- Ventral arc – is only present in females
- Sub-pubic concavity – the female pelvis will display a concavity with the edge of the ramas being concave. Males show no concavity
- Ramas ridge – males will show a flat, broad surface while females have a sharper edge

From Walker in Buikstra and Ulbelaker 1994 (White & Folkens, p.393):

- Greater sciatic notch – the shape of the notch is broader (score 1) in females than males (score 5).

From White and Folkens 2005 (p394):

- Subpubic angle – the angle formed between the lower edges of the two inferior pubic rami is larger in females than males

- Preauricular sulcus – is more often visible in females than males

From Rosing et.al. 2007 (p.80):

- Obturator foramen – in males it is taller and rounded and in females it is broad and triangular

Femora

Pearson's femur measurement system was also used. This system was developed from 17th century London bones (Bass, p.229) so is appropriate for skeleton ONE94 519. Measurements (mm) were taken of each femur's vertical diameter and biocondylar width.

Results

FEATURE	NOTES/SCORE	SEX
Nuchal Crest	5	M
Mastoid Process	4-5	M
Supra-orbital Margin	5	M
Supra-orbital Ridge/Glabella	4-5	M
Mental Eminence	4-5	M
Forehead shape & Brow Ridge	Definite ridge	M
Orbit shape	Square / rectangular	M
Mandible muscle (left only)	Flaring evident	M

Table 2: Sexing the skull

FEATURE	NOTES / SCORE	SEX
Ventral Arc	No obvious ridge	M
Subpubic Concavity	Broad (some post mortem damage to the left but the right is complete)	M
Ramas Ridge	Not visible	M
Greater Sciatic Notch	3-4	M
Subpubic Angle	Tight	M
Preauricular Sulcus	Not evident, smooth surface	M
Obturator Foramen	Oval	M

Table 3: Sexing the pelvis

FEMUR	RIGHT mm	LEFT mm	SEX
Vertical diameter	50.55	49.61	M
Bicondylar width	83.57	82.88	M

Table 4: Sexing the femur

Conclusion

All results suggest that individual ONE94 519 was a male.

AGE

Methodology

A number of methods were used to attempt to determine the age-at-death for ONE94 519. These focused on the teeth, the pelvis and the ribs.

Dental Attrition (Mays, pp.57-60)

- From Brothwell 1981 – molar teeth were examined for their wear patterns
- From Miles 1963 – molar teeth were again examined for eruption and wear

Pelvis (White & Folkens, pp.379-383)

- From Suchey-Brooks 1990 – the pubic symphysis faces were examined for surface changes and categorised into one of six phases
- From Lovejoy et.al. 1985 – changes in the auricular surface of the illum were examined and categorised into one of eight phases

Ribs (Iscan et.al., pp.855-858)

- From Iscan et.al. 1985 – changes at the sternal end of the ribs were examined and categorised into one of nine phases. Five randomly chosen rib ends were chosen for this method

Results

Ageing using tooth wear methods was hampered by tooth loss (ante and post mortem) and the state of the remaining teeth.

- All molars on the right mandible were lost either ante or post-mortem
- Two M1s (maxillary left and mandibular right) have been lost due to ante-mortem processes and the bone has healed
- The remaining M1 (maxillary right) has been almost destroyed by disease. The roots remain along with a small section of the buccal crown

MOLAR	MAXILLARY	MANDIBLE	AGE RANGE Brothwell 1981	AGE RANGE Miles 1963
M1 right	Caries destroyed leaving only roots	Ante-mortem loss and bone healed	n/a	n/a
M2 right	Some wear	Distal half of crown destroyed by caries	25-35	32 - 36

M3 right	Some wear	Some wear	25-35	32 - 36
M1 left	Ante-mortem loss and bone healed	missing	n/a	n/a
M2 left	Some wear	missing	25-35	32 - 36
M3 left	Some wear	missing	25-35	32 - 36

Table 5: Ageing the teeth

METHOD	PHASE	AGE RANGE
Suchey-Brooks 1990	4	23-57
Lovejoy et.al. 1985	4	35-39
Iscan et.al. 1985	6	32-79

Table 6: Ageing the pelvis & ribs

Conclusion

Results to determine the age-at-death of ONE94 519 are inconclusive.

- Methods looking at the teeth suggests an age ranging from 25 – 36
- Method examining the pelvis allow for an age range from 23 – 57
- The method examining the ribs suggests anything from 32 – 79

The cross-over of these age ranges is visible in the chart below:

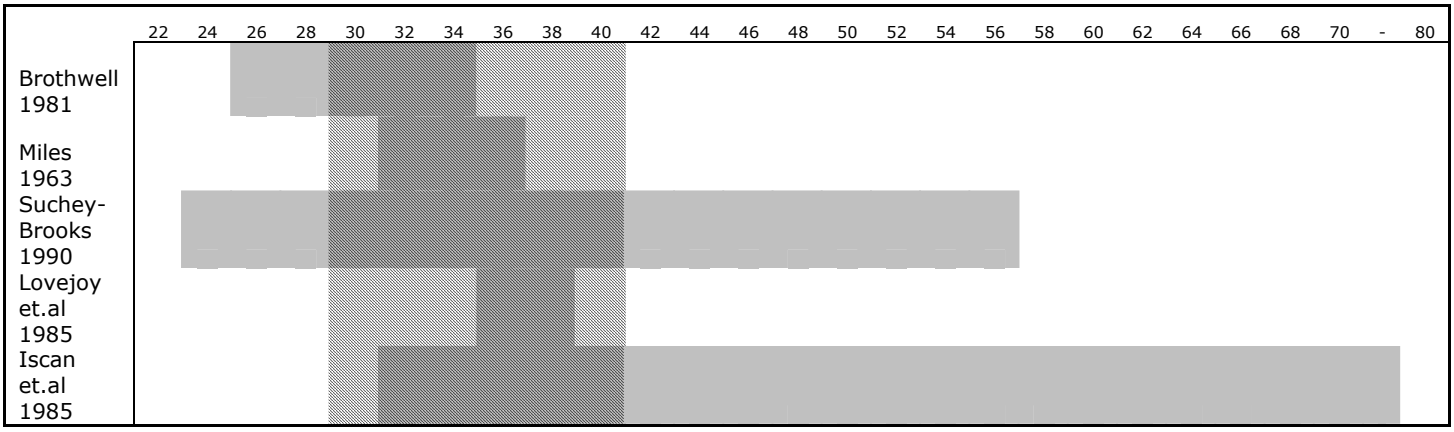


Table 7: Likely age-at-death ranges

Based on the methods used, it can be estimated that the more probable age-at-death for ONE94 519 lies between 30-40.

STATURE

Methodology

A metric method from Trotter and Gleser 1958 was used to determine individual height. This method is based on the fact that stature of the human body relates to limb bone length. Trotter and Gleser's formulae was based on racial groupings and this report uses the formulae for white males (Brothwell, p.102).

- Both fibula were broken and therefore not used
- Note that measurements from the femur, tibia, humerus and ulna are from bowed limbs

Results

BONE	LENGTH(cm)
Femur	44.3
Tibia	35.9
Humerus	31.8
Radius	24
Ulna	25.5

Table 8: Bone measurements

FORMULAE (cm)	HEIGHT METRIC cm	HEIGHT IMPERIAL inches
1.26 (Fem+Tib) + 67.09	168.142	66.197
2.32 Fem + 65.53	168.306	66.262
2.42 Tib + 81.93	168.808	66.46
1.82 (Hum+Rad) + 67.97	169.526	66.742
1.78 (Hum+Ulna) + 66.98	168.974	66.525
2.89 Hum + 78.10	170.002	66.93
3.79 Rad + 79.42	170.38	67.078
3.76 Ulna + 75.55	171.43	67.492
Average height	169.446	66.71075*

*Table 9: Formulae for stance (*conversion from diydata.com)*

Conclusion

Average results using Trotter and Gleser's 1958 method suggests that ONE94 519 was approximately 1.70 metres in height (5.5 feet).

DENTITION

The left maxillary bone remains with the skull. The right portion has broken away (post-mortem) and is available for examination. The mandible is also present. The teeth and associated bone show evidence of caries lesions, periodontal disease, an abscess, enamel hypoplasia and some calculus. Many crowns exhibit taphonomic dark or light brown staining. Dental inventories can be found in appendix A and B with a table indicating the percentage of teeth affected by disease as follows:

DISEASE	COUNT	PERCENT
Total teeth available	27	
Caries	8	29.63%
Periodontal disease	8	29.63%
Enamel Hypoplasia	4	14.81%
Calculus	2	7.41%

Table 10: Dental diseases evident

Caries

A high level of dental caries is evident in the teeth of ONE94 519 (figures 2 & 3). 29.63% of the available teeth show evidence of developed caries with this figure jumping to 36.6% if covering teeth exhibiting caries decay or ante-mortem loss.

Caries, the most common dental disease in archaeological populations, is the result of the fermentation of food sugars in the diet by bacteria that occur on the teeth. It is a disease in which the progressive destruction of the tooth structure is begins with microbial action on the tooth surface. There are other factors, apart from bacterial, which contribute to the development of the disease. These can include environmental, dietary, oral hygiene practices and the shape or structure of the teeth. (Robert & Manchester, pp.45-46)

Cavities generally have sharp defined edges and their diameter is often larger within the tooth than on the surface and they tend to be deeper in relation to their width than post-depositional erosions. Caries can lead to the destruction of the tooth which in turn may lead to infection of the pulp and a dental abscess, and in earlier times this could develop into a lethal complication, such as meningitis. (Mays, pp.147-148)

Most carbohydrates are sugars and studies on modern populations show a correspondence between the level of consumption of sweet food and dental caries rates. This is also evident in archaeological material. The rough diets of humans prior to recent times reduced caries by scouring the teeth clean of food debris and preventing excessive build up of dental plaque. In the UK significant amounts of sugar arrived during the 17th Century from sugar cane farmed in the New World. In the 19th Century sugar imports increased as import duties were removed and with this increase in sugar consumption, there was a rise in caries frequency. (Mays, pp.151-152)

Periodontal Disease

The roots on the teeth of ONE94 519 appear to be exposed by more than 2mm on 29.63% of the remaining teeth, indicating periodontal disease was present.

Periodontal disease is the inflammation of tissue around the tooth and causes the recession of the alveolar bone. Calculus accumulates in the spaces between teeth and soft tissue and bone and is a major predisposition to the development of periodontal disease. Roberts & Manchester suggest that periodontal disease may be over diagnosed in the past and they put this down to the lack of standardization in the recording of the disease. It is a common disease in modern populations and a major source of tooth loss in those over 40 and although it cannot be said with certainty, it is likely that periodontal disease was also a major cause of tooth loss in the past. (Roberts & Manchester, pp.56-57; White & Folkens, p.330)

Dental Abscess

Two holes sit above the first and second molars of the right maxillary bone. It is likely they are the result of an abscess related to the caries lesions evident in the first molar and pre-molars below.

Caries, calculus and periodontal disease are all forerunners to the development of dental abscess. An abscess occurs through exposure of the pulp cavity from attrition or trauma and the development of bacteria in the cavity. As bacteria accumulate in the cavity, inflammation begins and pus collects. Pressure builds up and eventually a hole develops on the surface of the jaw to allow the pus to escape. (Roberts & Manchester, p.50)

Enamel Hypoplasia

14.81% of ONE94 519's remaining teeth show evidence of enamel hypoplasia with all these being anterior teeth.

Enamel hypoplasia usually takes the form of transverse lines of depressed enamel on the sides of the tooth crown. It forms as a result of disturbance to the growth of the dental enamel. Poor nutrition and disease are considered to be the main causes. It forms during the time in childhood when crown enamel is developing and by default records episodes of disease or poor nutrition. The causes are wide ranging, and could include fevers, gut parasites, diarrhoea, rickets, or scurvy. In

permanent dentition it often shows episodes of disease or poor nutrition between the ages of one and seven. A study by Goodman & Rose in 1990 indicated that anterior teeth tend to show more hypoplastic lines than molars and pre-molars. (Mays, pp.156 – 158)

Calculus

Calculus is evident on 7.41% of the remaining teeth of ONE94 519.

Calculus is mineralised dental plaque on a tooth surface. Dental plaque can accumulate faster with sucrose in the diet. It is more common above the gum (supragingival) but can also occur below the gum (subgingival). Dental reports from archaeological material suggest that calculus was common in all periods in the UK. (Roberts & Manchester, pp.55-56)

Conclusion

Hypoplastic lines on some anterior teeth suggest ONE94 519 suffered from poor nutrition or a period of disease as a child. Rickets is evident from the state of the long bones (discussed below) which may have been a contributing factor in this case.

Ante-mortem tooth loss of three teeth may have been due to a combination of calculus, periodontal disease and series caries. The presence of these diseases (and a related abscess) in the remaining teeth and bone, and the skeleton having been excavated in a post-mediaeval context, suggest a diet potentially high in sugars.

PATHOLOGY

Skeletal indicators suggest that ONE94 519 suffered from rickets and osteoarthritis.

Rickets

Bowing in the femur, tibia, humerus and ulna are evident indicating that the individual suffered from rickets (figure 4).

Vitamin D deficiency is considered to be the most common cause of rickets. Vitamin D is mainly derived from the action of sunlight on the skin so deficiency is more likely to occur in individuals with darker skins or in those whose skin is

extensively covered. Vitamin D is needed for the efficient absorption of calcium and phosphorus and lack of it will lead to poor mineralization of the skeleton. If this occurs during childhood the disease is called rickets. If rickets is present the weight bearing bones of the leg become bow-legged when walking commences and arm bones may deform as crawling commences. Other bones deform under the influence of muscular contractions and the ends of the long bones will expand from excessive unmineralised cartilage causing increase in length and width in the growth plates of the bones. The growth of the pelvis and dental development may be hampered and the ribs may develop nodules. (Allgrove, p.699; Holick, p. 2063; Roberts & Manchester, p.173)

Rickets is not a new disease. It was first described by Englishman Dr Daniel Whistler in 1645 although it existed prior to this date. Osteoarchaeological evidence of the disease is not common. There is some evidence in Neolithic skeletons from Denmark and Norway. Moller-Christensen's study of medieval skeletons from the Aebelholt Monastery in Denmark showed only nine cases of rickets. Studies on the post-mediaeval Spitalfields population of London showed that at least 20 children and 15 adults had rickets. The swaddling of infants and keeping them house bound for the first years of their lives may be factors during this period. (Allgrove, p.699; Roberts & Manchester, p.175).

In the mid 1600s, most children who lived in crowded and polluted industrialized cities of northern Europe developed rickets. In the latter part of the 19th century autopsy studies performed in The Netherlands showed that 80–90% of children had rickets. In 1822 the importance of sun exposure for the prevention and cure of rickets was recognised and in the late 1800s the use of sun baths was promoted as to prevent the disease. In 1919 Huldschinski found that exposing children to radiation from a mercury or carbon arc lamp for short periods, three times a week was effective treatment. In 1918 Mellanby et al. performed experiments to show that cod liver oil could prevent rickets and soon after this nutritional factor was named 'vitamin D'. Once vitamin D was structurally identified and chemically synthesized from yeast, it was directly added to milk. (Holick, p.2062)

Following the recognition of the main causes of rickets (deficiency of sunlight and of dietary vitamin D) it was mostly eradicated from Western society however it has since returned. Large scale immigration from the West Indies and Indian subcontinent resulted in its reappearance in the UK but campaigns of vitamin D supplements, such as the Glasgow Rickets Campaign in the 1970s, were

successful in reducing incidents of the disease. Since campaigns like these have been reduced, further development of vitamin D deficiency has been noted. In 2004 Allgrove declared that "the time has come to mount a national campaign to promote awareness of the risks of vitamin D deficiency." (Allgrove, p.700)

Holick (p.2070) notes the major cause of modern rickets in the United States as a lack of appreciation that human milk contains very little vitamin D. African American women are often vitamin D deficient, and women who always wear sun protection and only take a prenatal multivitamin are also at a high risk of vitamin D insufficiency. If they provide breast milk to their infant as the only source of nutrition, the infant will become vitamin D deficient. If the infant is not exposed to sunlight or does not receive a vitamin D supplement, the infant will then develop rickets.

Other recent studies have also highlighted the return of rickets. In 2007 Ward et.al. (pp.165-166) noted the failure to prevent vitamin D deficiency in Canada. This condition was frequently observed among darker-skinned, breast-fed infants and children. They indicated the reappearance of the disease not only in regions with more limited sunshine, such as New Zealand, the United Kingdom and the United States but also from sunnier regions such as Africa, Saudi Arabia and Australia. Like Allgrove, they stressed an urgent need for heightened awareness of the disease among health care workers and the general public.

Osteoarthritis

The carpals of the left hand (specifically the articulating surface of the triquetral and trapezium) show evidence of subchondral cysts and eburnation (figure 5), suggesting that the individual suffered from osteoarthritis in the left wrist.

Osteoarthritis (OA) is a common joint disease. It is often described as 'wear and tear' and its prevalence increases with age. Symptoms can vary from minimal to severe pain and stiffness. Any synovial joint can develop OA but knees, hips and small hand joints are the sites most commonly affected. OA is defined as a complex disorder with a number of risk factors as follows:

- Genetic factors
- Constitutional factors (for example, ageing, obesity and, high bone density)
- Biomechanical factors (for example, joint injury and occupational or recreational use).

It is a dynamic process that involves all joint tissues. Key pathological changes include local loss of articular cartilage and the remodelling of adjacent bone with new bone formation (osteophytes). The specific targeting of OA for certain joints remains unexplained, but one hypothesis suggests an evolutionary fault where joints that have most recently altered are biomechanically under-designed and so fail more often. (The National Collaborating Centre for Chronic Conditions, pp.viii, 3)

OA is the most common disease seen in archaeological populations. It cannot be recognised by the formation of bony outgrowths alone. Eburnation, where the bone affected takes on a polished, ivory-like appearance (White & Folkens, p.327), is a definite sign of OA, but if this is not present, osteophytes must be accompanied by a porous joint surface. Comparison of the prevalence of OA between archaeological and modern populations is difficult as many diagnostic methods differ. For example, the occurrence of symptoms and changes on radiography seen in a modern patient is not possible for assessing an archaeological joint. (Roberts & Manchester, p.105)

Modern data indicates that people practising particular occupations suffer OA at certain joints. In archaeology, the study of activity related skeletal changes has been used to identify differences in lifestyle with subsistence change. Waldron warned of stretching the evidence too far and noted the lack of correlation of bone degradation with specific conditions in modern studies. Many factors can influence the development OA and these may determine if it is identifiable to a specific activity. Even so, is accepted that for occupational related changes to show on a skeleton, the individual must have started the activity at a young age.

Archaeological studies have used OA as a means to answer questions on activity in contrasting economies. In 1991 Bridges suggested an examination of bone strength & OA might help explain activity patterns in past populations, as these changes may be responses to different kinds of forces. Her studies showed that agricultural populations had stronger bones than their hunter-gather predecessors, suggesting an increased workload. These findings are supported by OA in the same populations. In 1984 Goodman et.al. suggested significantly increased OA in their Dickson Mound population with the transition to agriculture and in 1989 Walker & Hollimon noted increased mechanical stress with a shift to the intensive exploitation of a marine environment, from a hunter-gather lifestyle, by Californian Indians. (Roberts & Manchester, pp.109, 111 – 112,)

Relatively little work has been done on OA of the hand and foot in archaeological groups, perhaps because these small bones do not always survive burial and excavation. In 1989 Waldron & Cox hoped to explore the occupational link to OA and examined hand bones of skeletons from Spitalfields dated between 1729 and 1869. Here an individual's occupation was known from historical sources. The hypothesis, which did not pan out, was that a weaver's occupation could induce OA. Thirteen males had OA in their hands yet only 3 of these were weavers. The main correlation observed was between OA and increased age. (Roberts & Manchester, p.114)

Today osteoarthritis is still one of the most widespread of joint diseases and is a leading cause of chronic pain and disability worldwide. By retirement age it can be observed in over half the population. Eighty percent of people with the condition have some degree of limitation of movement and 25% cannot perform major activities of daily life. In small joints such as the hands and fingers OA makes many ordinary tasks very difficult. Increases in life expectancy and ageing populations are expected to make OA the fourth leading cause of disability by the year 2020. It has a significant negative impact on the UK economy, with its total cost estimated as equivalent of 1% of GNP per year. (The National Collaborating Centre for Chronic Conditions, pp.viii, 3, 7-8)

FIGURES

Figure 1: Green staining on the thoracic region

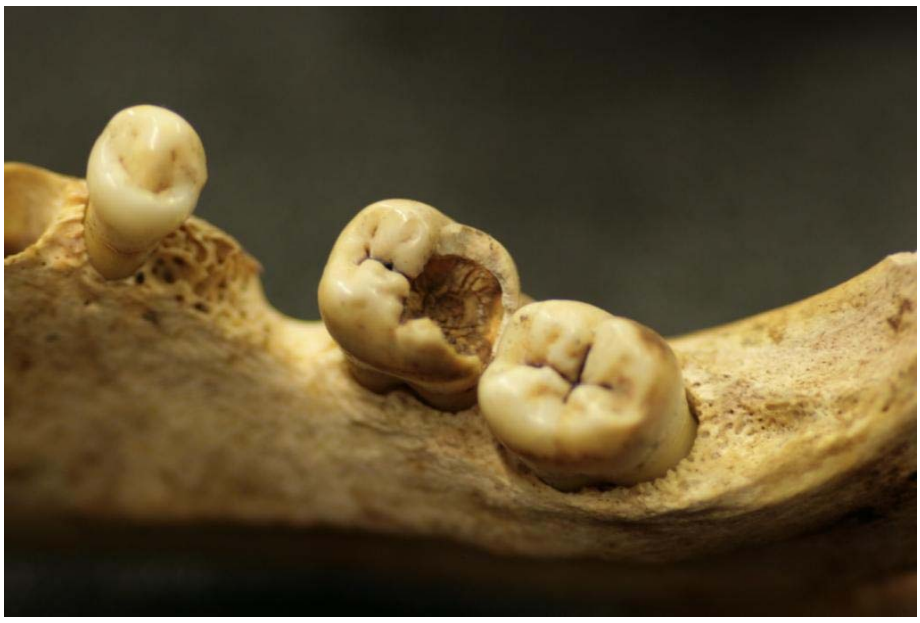


Figure 2: Example of dental caries on ONE94 519 (right maxillary)

Figure 3: Example of dental disease on ONE94 519

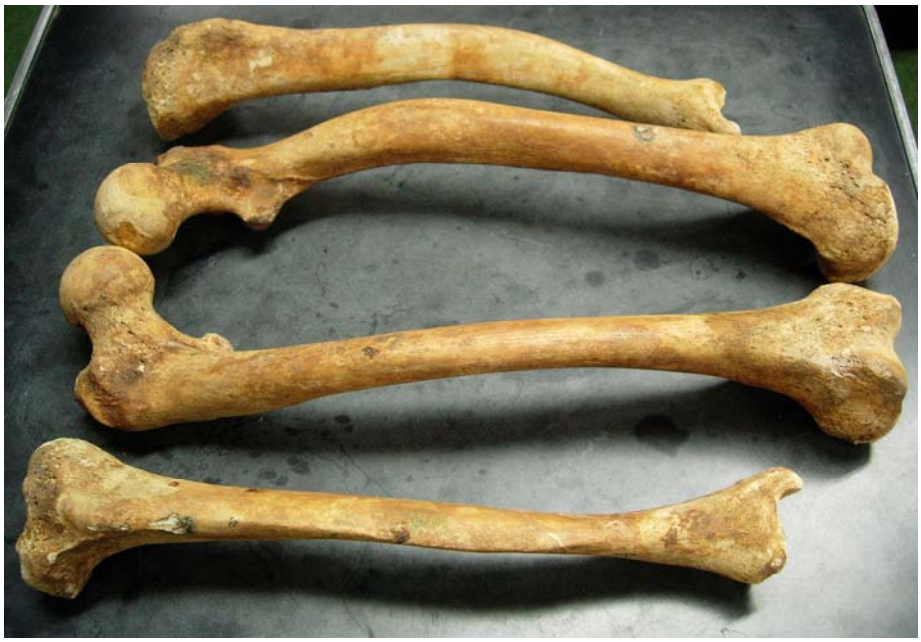


Figure 4: Bowed long bones of ONE94 519

Figure 5: Eburnation of the left carpals

APPENDIX A: Dental description

REF	BONE	TOOTH	POSITION	DESCRIPTION
1	Maxillary	3rd right molar	In jaw	little wear, evidence perio disease, brown stains buccal surface
2	Maxillary	2nd right molar	In jaw	little wear, evidence perio disease, brown stains buccal surface
3	Maxillary	1st right molar	In jaw	serious caries, only root & some of buccal crown remains
4	Maxillary	4th right pre molar	In jaw	serious caries, roots only remain
5	Maxillary	3rd right pre molar	In jaw	serious caries, roots only remain
6	Maxillary	right canine	Out of jaw	post-mortem loss, evidence dental enamel hypoplasia, labial surface. Some wear on medial corner
7	Maxillary	right lateral incisor	Out of jaw	post-mortem loss, series caries holes on labial surface of crown
8	Maxillary	right incisor	Out of jaw	post-mortem loss, brown staining on labial surface of crown, evidence dental enamel hypoplasia, some wear with dentine showing
9	Maxillary	left incisor	Out of jaw	post-mortem loss, brown staining on labial surface of crown, some wear with dentine showing
10	Maxillary	left lateral incisor	Out of jaw	post-mortem loss, enamel hypoplasia, distal corner showing wear
11	Maxillary	left canine	In jaw	evidence of perio disease
12	Maxillary	3rd left pre molar	In jaw	serious caries, roots only remain, evidence of perio disease
13	Maxillary	1st left molar	In jaw	calculus deposition on buccal side , evidence of perio disease
14	Maxillary	1st left molar	n/a	*Not available, ante-mortem*
15	Maxillary	2nd left molar	In jaw	little wear, dark stains in occlusal surface fissures & buccal of crown
16	Maxillary	3rd left molar	In jaw	little wear, dark stains in occlusal surface fissures & buccal of crown, caries in occlusal & buccal surface
17	Mandible	3rd left molar	Out of jaw	post-mortem, roots only, diseased
18	Mandible	2nd left molar	n/a	ante-mortem loss
19	Mandible	1st left molar	n/a	post-mortem loss
20	Mandible	4th left pre molar	n/a	post-mortem loss
21	Mandible	3rd left pre molar	Out of jaw	post-mortem loss, some brown staining on crown
22	Mandible	left canine	Out of jaw	post-mortem loss, some brown staining on crown
23	Mandible	left lateral incisor	Out of jaw	post-mortem loss, some calculus on medial edge
24	Mandible	left incisor	In jaw	some wear with dentine showing
25	Mandible	right incisor	In jaw	some wear with dentine showing
26	Mandible	right lateral incisor	Out of jaw	post-mortem loss, small brown spot distal side
27	Mandible	right canine	In jaw	perio disease, evidence of enamel hypoplasia
28	Mandible	3rd right pre molar	Out of jaw	post-mortem, small brown spot distal side
29	Mandible	4th right pre molar	In jaw	little wear, perio disease
30	Mandible	1st right molar	n/a	ante-mortem loss
31	Mandible	2nd right molar	In jaw	series caries, hole in distal half of crown, little wear on remaining half, dark stains in lingual surface
32	Mandible	3rd right molar	In jaw	perio disease, some black staining on lingual and buccal surface of crown, a little wear, dark stains in occlusal fissures

APPENDIX B: Visual dental inventory

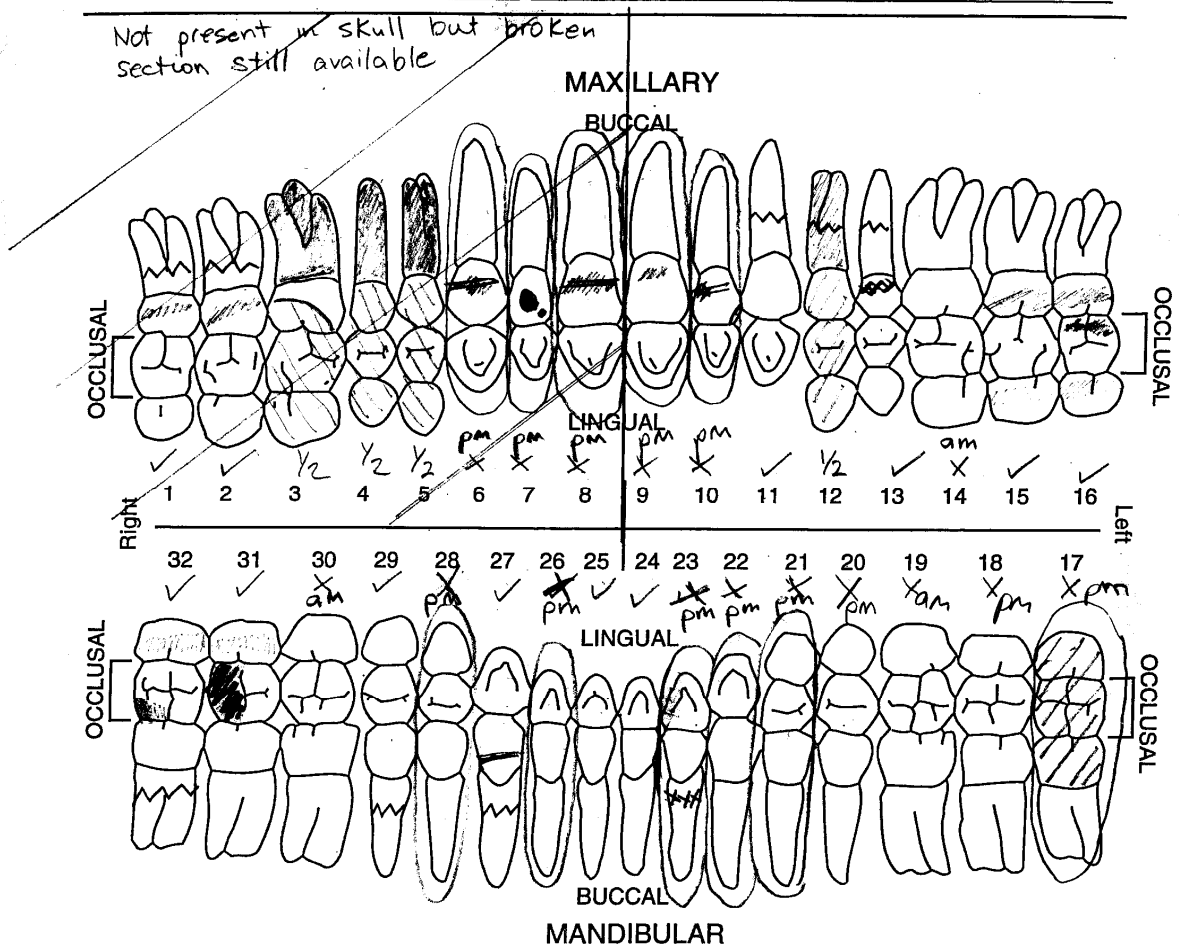
DENTAL INVENTORY
VISUAL RECORDING FORM: PERMANENT DENTITION

Site Name/Number _____/_____ Observer _____

Feature/Burial Number _____/_____ Date _____

Burial/Skeleton Number _____/_____

Present Location of Collection _____



✓ – in jaw
X – missing
½ – roots only
X ○ circled – post mort loss; tooth available for examination
//// – this part of teeth missing
pm – post mort. loss
am – ante mort. loss

■ black shading – caries
XXXX hatching – calculus
light shading – black staining possibly post-mortem
lines – evidence hypoplasia
waves – evidence perio. dis.

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