Columbus was vilified by syphilis: Logical analysis of historical facts and spectrophotometric analysis of skeletal evidence

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Abstract:

The question concerning the existence of syphilis in the pre-Columbian Old World has been debated from around the 1530's when Fracastoro, Ruiz de Isla, Oviedo and Le Casas all published their thoughts on syphilis originating in the New World. However, syphilis may have existed in the Old World prior to Columbus as a mild infection under the disguise of many diseases including leprosy. Since then the debate has branched out to include other facets of evidence, including paleopathology and examination of DNA from bones. These facets have advanced our understanding of the disease and how it affects human remains but, have yet to solve its origins.

The pre- and post-Columbian literature were re-evaluated to assess an overall view of why syphilis was seen as a new disease post-Columbus. The role of the events that led the French army of Charles VIII into Italy, that decisively contributed to the Columbian thesis needs to be discussed. This thesis re-examines through both literature and mathematical calculations, the possibility of infection from the New World through Columbus' voyage, and infection through a mild form of syphilis (endemic treponematosis) which became inflamed through constant re-infection resulting in a superinfection. In addition, it suggests that mercury which was a source of medicine for syphilis can be used to support the presence of syphilis, even when there are minor pathognomonic signs of the disease in skeletal remains. Regardless, it must be taken into account that mercury was used to treat other skin diseases such as leprosy, therefore differential diagnosis is necessary to draw appropriate conclusions.

Methods included the examination of pre-Columbian skeletal remains from various countries. The collections consist of Old and Middle Kingdom Egypt, Ancient Greek Metaponto,

Oplontis (Pompeii), medieval Danish leprosarium and early medieval Polish Kolonia and Brześć Kujawski,. Small fragments were taken from bones and analysed by Laser Ablation Inductively Coupled Plasma Mass Spectrometry (LA-ICP-MS). The LA-ICP-MS is capable of measuring traces of mercury. The results suggest that the majority of skeletal remains show signs of syphilis were likely treated with mercury as mercury concentrations in bone have higher than the normal 0.1 ppm concentrations compared to those in control samples that lack pathological indicators. This suggests that people were using mercury to treat individuals with pathological signs indicative of syphilis prior to the siege of Naples in 1495. Furthermore, it adds supporting evidence that will nullify the New World as the source of condition.

Declaration:

I certify that this work contains no material which has been accepted for the award of any other degree or diploma in my name, in any university or other tertiary institution and, to the best of my knowledge and belief, contains no material previously published or written by another person, except where due reference has been made in the text. In addition, I certify that no part of this work will, in the future, be used in a submission in my name, for any other degree or diploma in any university or other tertiary institution without the prior approval of the University of Adelaide and where applicable, any partner institution responsible for the joint-award of this degree.

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Introduction:

Due to the limited evidence for the presence of syphilis in skeletal remains, difficulties associated with diagnosis via skeletal pathology and dating issues, syphilis has become one of the most controversial diseases in history. The primary question is whether this disease was introduced to the Old World following Columbus' return from American exploration, or was it an endemic Old World disease that heightened its manifestations through a more recent infection pattern. Syphilis was first reported to have appeared in Naples in 1495 (Baker et al. 1988; Waugh 1982; Crosby 1968; Naranjo 1994; Abraham 1948).

There are many theories regarding the origin of syphilis that emerge from two opposing mind-frames. One, the pre-Columbian theory, argues that syphilis existed in the Old World prior to Columbus (Holcomb 1937; Hackett 1963; Hudson 1968; Pàlfi 1992; Henneberg and Henneberg 1994; Roberts 1994; Crane-Kramer 2000; Mays et al 2003; Ioannou et al 2018). The other theory is the Columbian one which argues that syphilis was brought to Europe by Columbus from the Americas (Crosby 1969; Dennie 1962; Baker et al. 1988; Rothschild 2004; Harper et al. 2011; Zuckerman 2016). This thesis involves several distinct elements. The first which was submitted to the Journal of Interdisciplinary History, argues that it was the army of Charles VIII that was responsible for the dissemination of syphilis acquired in Naples in Italy as an epidemic. The second examines the possibility of the existence of syphilis in pre-Columbian Egypt. The third concerns the possible existence of syphilis in a leprosarium, and the fourth examines medieval Polish skeletons for evidence of venereal syphilis.

What is syphilis?

Syphilis is one of the treponemal diseases. Treponemal diseases are caused by T. *pallidum*, but depending on the mode of infection and environmental circumstances may take different forms. These different forms of treponemal disease include syphilis, bejel, yaws and pinta. These are often subdivided into congenital, endemic, venereal and some localized forms.

While there was a lot of confusion at the end of the 15th century as to what syphilis was, today the disease is well known. There are many publications now describing not only the morphology, antigenic properties, and DNA homology of pathogens but also the clinical manifestations of the disease. Some of these publications include: King and Catterall (1959); Willcox (1960); Steinbock (1976); Fitzgerald (1981); Flores (1995); Aufderheide and Rodriguez-Martin (1998); Myer et, al. (2002); Ortner (2003); Baugh and Musher (2005); Walker and Lockwood (2006); Harper et al. (2008, 2011). Harper's et al. (2008) work has been criticized by geneticists Mulligan et al. 2008 who state that no evolutionary order could be inferred of the treponemal strains. Harper et al.'s theory stating that syphilis evolved from New World's yaw is solely based on the homology of two single nucleotide polymorphisms from the degraded Guyana samples.

What is syphilis? Syphilis is a systemic disease which can be either venereal or non-venereal and is caused by the spirochete *Treponema pallidum* subsp *pallidum* that attacks multiple tissues in the human body (Ortner 2003). These organisms are spiral in shape with the appearance of a corkscrew (Piece and Katz 2011). *Treponema pallidum* has many genetic variations, responsible for the non-venereal form of disease, these are: - *T pallidum* subsp *endemicum* (bejel), *T pallidum* subsp *pertenue* (yaws), and *Treponema carateum* (pinta)

(Singh and Romanowski 1999; Giacani and Lukehart 2014). Although these genetic forms of the species *Treponema pallidum* cause various pathological symptoms and signs which differ from one another, they still have virtually identical morphology, antigenic properties, and DNA homology (Centurion-Lara et al. 1998; Lafond and Lukehart 2006; Giacani and Lukehart 2014; Štaudová et al. 2014). Most cases of venereal syphilis are acquired through direct sexual contact with the lesions of an individual, who has active primary or secondary syphilis, and transmission occurs in approximately half of such contacts. Syphilis can be transmitted from an infected mother to the fetus by transplacental passage of treponemes causing a distinct congenital form of the disease (Fenton et al. 2008).

Syphilis remains a global problem, with an estimated 12 million people infected every year (Gerbase et al. 1998; Walker and Walker 2007). In 2016 the WHO estimated that global syphilis prevalence was 6 million (WH0). In 2017 the Center for Disease Control reported that the United States of America has 30,644 cases of primary and secondary syphilis) (CDC). Congenital syphilis, a consequence of infection during pregnancy, results in serious adverse outcomes in up to 80% of cases and is estimated to affect over 1 million pregnancies annually, despite the existence of simple, validated screening tests, effective prevention measures, and cheap treatment options (Saloojee et al. 2004). In many high-income countries, successes in syphilis prevention and control were accelerated during the early and mid-1990s, with many countries approaching, or achieving elimination of endemic disease transmission (Golden et al. 2003). However, since the beginning of the 21st century, syphilis incidence has started to rise in high-income settings, in part driven by increases in cases among men who have sex with men, although more recent increases among heterosexual people have also been reported (Fenton and Imrie 2005; Fenton 2004; Fenton et al. 2008). If we understand the

history and evolution of syphilis, then it might be possible to determine how to better control such an adapting disease. (Fenton, K et al. 2008)

What is leprosy?

Syphilis is well known as the great imitator. It has the ability to imitate other diseases both clinically in living people and in the bones of the diseased. The disease that syphilis seems to imitate the most is leprosy. In the past leprosy was often confused with syphilis, with medical writers describing diseases like venereal leprosy (Gordon 1491). Even in recent times leprosy can be confused with syphilis (Fonseca et al. 1999). In addition, when assessing human skeletal remains syphilis and leprosy can often be misdiagnosed (Lefort and Bennike 2007). This is especially the case when individuals become coinfected with syphilis and leprosy (Garner et al. 1973; Murray 1982). Therefore, when investigating syphilis, leprosy must also be a part of any differential diagnosis whether clinical or paleopathological.

Leprosy, which is also caused by a bacterial infection (*Mycobacterium leprae*), is often considered when assessing the paleopathology of syphilis in skeletal remains (Crane-Kramer 2000). This is due to the many similarities that both diseases can exhibit in bones, especially if the bones are not showing distinct pathognomonic traits of the disease. Therefore, it is important to consider the cause of leprosy and how it affects the body.

Mycobacterium leprae is related to the bacteria *M. tuberculosis* and *M. bovis* (Ortner 2003; Donoghue et al. 2005). Leprosy is capable of infecting not only humans, but also armadillos and some primates (Walker and Lockwood 2006; Sasaki et al. 2001). Due to the bacterium being acid-fast and Gram-positive it results in chronic granulomatous infection (Ortner 2003; Scollard et al. 2006). It is quite easy for an infected person to transmit bacteria to another person, usually through sneezing. *M. leprae* has a low transmission rate due to its slow

growing. The incubation period of leprosy is approximately 2-12 years (Rodrigues and Lockwood 2011). The immune system is usually strong enough to keep the pathogen from spreading, therefore, it is not everyone who contracts the disease.

When the bacteria *Mycobacterium leprae* start to infect the host, the disease may appear to be mild (tuberculoid) through to intermediate, then up to severe (lepromatous) (Ortner 2003). "Tuberculoid leprosy manifests with a few well defined, hypopigmented anesthetic macules. Lesion borders are elevated and erythematous and the centres are atrophic. There is usually no loss of sensation on the face because of the abundant sensory innervation there. Patients are immunocompetent, lesions are not usually large or numerous, and this type of leprosy may resolve spontaneously if the host's immune system is strong" (Eichelmann et al 2012: 557-558).

"In the lepromatous form (also includes borderline leprosy cases) patients are characterized as having confluent papules and nodules, possibly resulting in marked, diffuse infiltration of the skin and giving rise to leonine facies and madarosis. Lesions are usually symmetrical and bilateral. This form of leprosy is characterized by greater nerve involvement and more severe disability (Eichelmann et al. 2012: 558). "In lepromatous leprosy mucous membranes, eye, bones, joints, lymph nodes, blood vessels, upper airways, teeth, and internal organs may be affected" (Lastória and Abreu 2014: 2010).

"General clinical manifestations depend more on the cellular immune response of the host to M. *leprae* than on the bacillary penetration and multiplication ability. "Patients present with skin lesions, peripheral neuropathy or the consequences of neuropathy. Skin lesions may be

hypo-pigmented, erythematous or infiltrative. These are often diagnosed as chronic common skin lesions that have responded poorly to standard treatments. Further they may report weakness, sensory loss, neuropathic pain, peripheral nerve thickening or ulcers. Less common features include arthritis, erythema nodosum leprosum, orchitis and acute uveitis" (Bharucha and Lockwood 2016: 154-155).

"Lesions may affect cutaneous peripheral nerves, primarily the posterior tibial, cubital, medial and lateral fibular nerves. Nerve involvement causes thickening, pain, and sensory and motor impairment. When small cutaneous nerve fibers become involved, the result is numbness, anhydrosis, and thermal sensory impairment. In pure neuritic leprosy the neuropathy is asymmetrical" (Eichelmann et al 2012: 559).

The musculoskeletal system is affected in 95% of cases. The most common skeletal signs are non-specific as sensory loss secondary to nerve damage leads to ulcers, deformities, and fractures. It is important to remember that osteoporosis is the second most common sign in patients with leprosy. Also patients with the lepromatous form have been reported to develop testicular compromise, mainly atrophy and acute orchitis related to erythema nodosum (Eichelmann et al 2012: 559).

Leprosy can affect the bones, although it is most noticeable in the skeleton during the lepromatous stage. It is believed that the immune system plays a major part in determining the level of severity (Ridley and Jopling 1996). At the moment there is no proof that there are subspecific differences in the pathogen responsible for the various stages of pathological manifestations.

Leprosy may be slow to infect the host; however, it would seem that the disease could survive in any climatic zone, with the exception of the artic regions. In modern societies it thrives in the sub-tropical and tropical areas of India, Thailand, Indonesia, Philippines, Africa and the Americas (Suzuki 2012). Males are twice as likely to contract the disease as females (Faget and Mayoral 1944). Leprosy affects the skin, mucous membranes, nerves, peripheral extremities, leading to loss of sensory perception. With the nerve supply to the muscles being impaired, there is a wasting away of tubular bones of both the hands and feet (Ortner 2003). Regardless of the severity of the disease, its development and progression span over decades and remain chronic.

In leprosy skeletal involvement does not play a major role, as only five percent of cases without treatment are affected by it (Resnick and Niwayama 1998). However between 1932 to 1940, there was a series of tests carried out on 483 lepers, with only 177 not portraying any signs of bone lesions (Esgurra-Gomez and Acosta 1948).

Due to the original works of Denmark doctor, Dr Vilhem Moller-Christensen; based upon the presence of leprosy in the medieval times, derives from his examination of a large number of individuals from medieval leper hospitals in Denmark, led to him establishing a series of diagnostic criteria that have continued to be invaluable for proper identification of the disease in the osseous tissue. In a paper that Dr Moller-Christensen and Faber released in 1952 coined the term “facies leprosa” this refers the particular pathologic changes that occur in the skeletal structure of the face in leprosy, on the basis of the Danish research, it was concluded that for a diagnosis

of leprosy to be made that facies leprosa must be present in the osseous tissue. The reason for this occurrence is due to the fact that bone changes in the hands and feet will often be observed with facies leprosa. 41 complete skeletons demonstrated leprosus osseous change, this occurred in virtually all of the specimens, a change in the bones of the hands and feet was observable in approximately 66% of the skeletons (Steinbock 1976:201: Moller-Christensen). The osseous change that occurs in the cranium is often restricted to the rhinomaxillary region of the face.

Keith Manchester (1984) states that "skeletal changes of leprosy are found around the oral and nasal cavities and at the limb extremities. The cranial features, the so called *facies leprosa*, consist of the progressive erosion of the alveolar process of the maxilla with the loosening and ultimate loss of the central and lateral maxillary incisor teeth. There is an associated erosion of the anterior nasal spine leading to its ultimate loss. The margins of the pyriform aperture become eroded at their lower parts. Both the nasal and the oral surfaces of the palatine process of the maxilla exhibit inflammatory changes, and there may ultimately be perforation of the hard palate" (Manchester 1984: 167).

The lower limb changes are characteristically destruction of foot bones, especially phalanges, Charcot joints in the foot or tibiotarsal joints, gross periostitis of the tibiae and fibulae, usually bilateral, and commencing at the distal ends. "There is inflammatory change in the distal foot commencing in the phalanges and metatarsals, and there may be inflammatory changes in the tarsal bones. The phalanges are lost. The metatarsals develop concentric atrophy and become pencil shaped with the loss of medullary cavity. In the hands, the inflammatory change commences in the phalanges, spreading later to the metacarpal bones.

These changes result from trauma to the anaesthetic fingers, and are sometimes associated with the claw hand deformity of leprous paralysis" (Manchester 1984:168).

According to Donald Ortner there are three different pathogenic mechanisms which can affect skeletal changes and they are: - (1) lepromatous osteomyelitis and periostitis, (2) neurotrophic bone and joint lesions, in neural leprosy and (3) ordinary osteomyelitis as well as septic arthritis because of secondary infection (Ortner 2003).

Stages of syphilis:

In order to contract syphilis, bacteria *T. pallidum* need to penetrate the mucosal surface or abraded skin during a close contact of individuals, usually sexual intercourse, where *T. pallidum* subsp *pallidum* fastens onto host cells and rapidly multiplies (Montone 2007). It only takes two to six weeks after infection for a painless indurate papule (chancre) to occur at the site of inoculation. The surface of the papule necrosis forms a hard-based chancre, teaming with treponemes (Lautenschlager 2006). A healthy immune system assists in clearing treponemes from the body, but unfortunately it does not matter how many are destroyed there are always organisms which manage to survive, enabling them to induce chronic infection. (Fenton et al 2008)

The multiplication and dissemination of treponemes must occur throughout the body before secondary syphilis can take place. *T. pallidum* subsp *pallidum* has a penchant for the lymphatic and skeletal systems, regardless of the high concentrations of anti-treponemal antibodies (Van Voorhis et al 1995). The primary stage lasts between three and four weeks; and within this timeframe the lesion will heal. The secondary stage occurs when the body is bombarded by a wide range of signs (Lautenschlager 2006; Ho and Lukehart 2011). These signs include malaise, low grade fever, headache, rash on the palms and soles of the feet, generalized lymphadenopathy, mucous patches in the oral cavity or genital tract, *condylomata lata* in moist intertriginous regions and alopecia (Goh 2005). If the secondary stage goes untreated there is a chance it will reoccur, otherwise it can last for several weeks or months. (Fenton et al 2008) Osteomyelitis may also occur in the secondary stage. The osteomyelitis may occur in early stage syphilis when the spirochetes become disseminated throughout the body infiltrating soft tissue and bone. “Spirochetes enter the deeper vascular

areas of the periosteum which results in perivascular inflammation and subsequent formation of highly cellular granulation tissue" (Dismukes et al 1976:2647). This creates a thickened, expanding, and elevated periosteum. These bone formations in early stages of the disease are comparatively rare. When the skeletal structures do become involved during early syphilis, the involvement is usually proliferative periostitis. More rarely destructive osteitis and osteomyelitis occur (Reynolds and Wasserman 1942).

The Syphilis Division of the Medical Clinic and the wards of the John Hopkins Hospital observed a series of approximately 10,000 cases of early syphilis over a twenty-two year period (1919-1940). Out of these cases 15 instances of destructive bone lesions either; osteitis, osteomyelitis or osteoperiostitis have been recognised (Reynolds and Wasserman 1942).

The third stage is the latent stage of syphilis where there is a lack of clinical symptoms, however there are still spirochetes alive in the spleen and lymph nodes (Lautenschlager 2006; LaFond and Lukehart 2006; Fenton et al 2008). This stage can last up to a year, which is followed by the late phase. During the late latent syphilis stage, the patient will find their resistance to reinfection has improved and their immunity to the active disease returning, although an immediate cure is unlikely (Fenton et al 2008).

Due to antibiotics the late stage of syphilis is now a rarity, although history reveals that one-third of untreated patients experienced the tertiary stage, around twenty to forty years after the primary stage (Gjestland 1955). Treponemes are relentless, travelling throughout the body while systematically attacking it. They congregate around the wall of the aorta causing

problems like aneurysms, aortitis, or aortic endocarditis, not to mention the CNS, blood vessels, eyes, skin, other internal organs as well as inducing inflammation (Lautenschlager 2006).

Whether it be symptomatic or asymptomatic, neurosyphilis causes meningeal, meningovascular and parenchymatous syphilis (Fenton et al 2008). One pathognomonic sign indicating syphilis is the formation of gumma, which consists of large areas of necrosis surrounded by lymphocytes, macrophages, multinucleated giant cells, plasma cells and fibroblasts (Rodriguez et al. 1988). Gummas are pink to dusky red in colour and can be up to several centimetres in size (Ficarra and Carlos 2009; Günasti and Aksungur 2014). These granulomatous lesions often are destructive of the skin, bones or viscera (Little 2005). Some individuals only develop microscopic defects, whereas others experience many large tumour like masses. If patients progress to the tertiary stage, they are usually non-contagious. The vertical transmission is a rarity and sexual transmission does not happen. (Fenton K et al 2008)

Differential diagnosis of Syphilis (venereal, congenital and non-venereal) Observable in skeletal remains

Descriptions of diseases in this chapter are limited to their impact on skeletal remains.

Congenital syphilis:

Venereal syphilis appears in two forms, congenital or acquired. Congenital syphilis occurs when treponemes invade the fetus from an infected mother's placenta (Cooper and Sanchez 2018). The treponemes wait until the third or fourth month of gestation has passed before bridging the placenta, and then advance towards the foetal bloodstream, once they have passed through, they are free to attack every part of the body (Steinbock 1976).

"An estimated million pregnancies globally are annually adversely affected by syphilis. 270,000 babies are born with congenital syphilis, 460,000 pregnancies end in abortion or prenatal death" (Walker and Walker 2007:199). In 1995 the WHO estimated the annual incidence of acquired syphilis to be around 12 million, approximately 6 million of those cases were women (Finell 1998). "Of the 6 million cases 90% are women who were of reproductive age and with the fertility rate of 20% per year, approximately 900 000 gestations occur annually among infected women. A further estimated 40% of these pregnancies (360 000) end in fetal or prenatal death and approximately 5-10% of the remaining neonates (270 000) suffer from significant physical development and sensory impairments" (Finelli et al 1998:126).

The consequences of early congenital syphilis are skeletal lesions namely osteochondritis, periostitis, and diaphyseal osteomyelitis which occur anywhere from birth to three or four

years of age, usually displaying bone involvement to some extent (King and Catterall 1959). There are major pathognomonic dental changes that occur in congenital syphilis, that will be discussed further below. As far as late congenital syphilis is concerned, it mostly occurs between five and fifteen years of age where chronic bone lesions produce abnormalities which are particularly noticeable for a number of years (Steinbock 1976).

Since early congenital syphilis produces bone lesions that for the most part heal over time, it is therefore the late congenital syphilis that is the more pathognomonic for differential diagnosis purposes (Steinbock 1976). In late congenital syphilis tibiae along with other long bones are often affected, usually bilaterally, when the tibia is affected bilaterally then saber shin occurs. Gummatous osteomyelitis or osteitis is often present in addition to the osteoperiostitis of the long bones (Ortner 2003). The major bones affected are the tibia and other long bones. The gummatous osteitis can also affect the cranium, although it is of diagnostic importance, (Hackett 1981) it is not a frequent event.

Other bone changes that can occur in late, but also can occasionally occur in early congenital syphilis, is the destruction of the bony and cartilaginous elements of the nose which may produce what is known as a ‘saddle nose’ (Steinbock 1976:106). Such nasal destruction is nonspecific and must be accompanied by other bone changes to warrant a diagnosis of congenital syphilis.

Hutchinson`s teeth are conspicuous as the central incisors of the upper jaw are narrowed, barrel shaped in appearance with convergence of both lateral margins towards the cutting surface (Steinbock 1976). Even though notching is not a regular occurrence, teeth still

possess a screw driver shape owing to the convergence of the lateral margins (Steinbock 1976; Hillson et al 1998)

Other pathognomonic changes to the teeth include the first molar, commonly known as a mulberry molar, or bud molar, dome-shaped molar, Moon`s molar or Fournier`s tooth (Steinbock 1976; Hillson et al 1998; Ioannou et al 2015). As far as the mesiodistal length of the crown is concerned this molar is quite small, with a rough and irregular occlusal surface and atrophic cusps which are represented by several small knobs (Steinbock 1976).

Acquired Syphilis:

Early in the course of acquired syphilitic infection, spirochetes are widely disseminated throughout the body tissues, including those of the bony skeleton. Despite the fact that bone, together with its periosteum and marrow cavity, is known to harbour virulent organisms, the development of observable skeletal abnormalities during the early stages of the disease is comparatively rare (Reynolds and Wasseman 1942). When the skeletal structures do become involved during early syphilis, the involvement is usually proliferative periostitis. More rarely destructive osteitis and osteomyelitis occur (Reynolds and Wasseman 1942; Park et al 2014)

The walls of medium sized arteries are affected with an intimal thickening and an accumulation of lymphoid cells associated with periostitis of syphilis. (Resnick and Niwayama 1998). Inflammations in these areas are caused by ischaemia along with damaged

blood vessels where an exuberant formation of new bone develops, and hyperemia occurs. (Cotran et al 1994). During secondary stage syphilis, spirochaetes reach deep down into the vascular areas of the periosteum where they inflame and infiltrate the perivascular tissues as well as causing highly cellular granules in tissues to form (Resnick & Niwayama, 1998). When the granulated tissue forms it continues to cause problems in the Haversian canals, resulting in osteitis. It is possible that during the secondary stage of syphilis a repaired `formation of new bone over primary osteitis sites` occurs. (Keyes, 1908), resembling congenital syphilis in its late stage (Jaffe 1972; Buckley and Dias 2002).

There are three stages of syphilis. During the second and third stage destructive gummas can occur due to osseous alterations (Gurland et al 2001). After this osteolysis ends up in the moth-eaten bones of the body, and when it appears in the cranium it is called carries sicca. It has been argued the distribution of carries sicca over most of the frontal and parietal areas of the cranium is pathognomonic for venereal syphilis, but in 1994 Skinner has expressed doubts (Skinner 1994). After syphilis has been contracted, as far as the cranium is concerned, the outer table of the cranium is first to become affected, the diploe follows while miraculously the inner table is not harmed in a number of cases.

The diagnostic criteria for carries sicca proposed by Hackett (1981: 78) are:
"*The active pathological changes start as inflammatory osteopososis at the junction of the inner surface of the outer table and the diplöe (1), and exend in all directions (2). These holes are then filled by fire-bone (3). This is next remodeled by osteoporosis followed by filling by lamellar bone (4, 5), which results in healing and sclerosis (6). Each of the two lower figures has a different pattern of multi-nodulation, types i and ii, but the internal patterns indicate*

the next to last and the last stages of remodeling (refer to Hackett's 1981 paper). The periosteal new bone goes through a similar sequence, and ends in sclerosis".

Treponematosis has a propensity to favour the tibia, causing periostitis where the destructive foci are responsible for excessive osteosclerosis. The medullary cavity of tubular bones has been known to narrow. Venereal syphilis has the ability to affect both internal organs and the nervous system causing neurosyphilis. Charcot`s joints are results of *tabes dorsalis*, producing a loss of feeling in the major joints, especially the knee (Ortner 2003). The lower thoracic spine is home to changes resulting from aortic aneurysms (Myer 2001). Hackett (1975) described long bone diagnostic criteria for syphilis. He states that expanded bones with larger than usual surface striae and pits, are also regarded as diagnostic criteria (on trial) of syphilis. Along with nodes/expansions with superficial cavitation in long bones they are a diagnostic criterion of syphilis (Hackett 1975). In advanced stages the cavities may be in a sclerotic medulla. These changes were seen in yaws in Uganda (Hackett 1951) and in bones of Aboriginal Australians suffering from yaws, and are reported in Syria (Rost 1942). Other candidates for diagnostic criteria of syphilis would be sternal or vertebral erosion from aortic aneurysm, and Charcot's joint.

In non-venereal syphilis like yaws the most focused area of bone involvement is the tibia, whereas in acquired syphilis the most common location of tertiary syphilitic lesions is in the skull, particularly in the perinasal area and the cranial vault. Furthermore, in acquired syphilis, cranial lesions represent the most pathognomonic features of the disease. (Powell 1988).

There are many different kinds of cranial involvement in syphilis, one of which is cranial periostitis, common in the earlier stages of syphilis, however, this is not a good diagnostic feature for this specific disease **(**Hackett 1975; Ortner 2003). The main interest for paleopathologists are the pathognomonic features that start in the tertiary stage of syphilis. These are gummatous, osteoperiostitic lesions of the cranial vault, the majority of which begins in the frontal bone (Hackett 1975; Ortner 2003). Other important diagnostic features are sclerotic healing and lytic lesions.

Even though syphilis is present in various postcranial areas it is the tibia which is more often than not the site of syphilitic lesions. Discovering non-gummatous lesions on the long bones could suggest treponemal infection, but then again periosteal thickening and osteoperiostitis, are common in a number of other infectious diseases. Therefore, these lesions cannot be seen as diagnostic proof for the presence of syphilis (Ortner 2003; Steinbock 1976). However, gummatous osteoperiostitis lesions could appear as an enlargement on the affected bone, as well as central gummas appearing as large lytic lesions in the medullary cavity (Ortner 2003). These lesions are encircled by a marked perifocal reactive sclerosis, all of which are indicative of syphilis.

Tabes dorsalis causes neuropathic arthropathies commonly known as Charcot`s joints, which are mainly located in the large joints of the lower extremities, like the knee. They appear in late stages of neuro-syphilis (Ortner 2003). Patients over the age of 60 usually present neuropathic arthropathy, which occurs in 5% to 10% of patients diagnosed with tabes dorsals and most commonly involves the hip, knee or spine (Alpert et all 1996) The involvement of the upper extremities is much less common but may be encountered in cases with

polyarticular disease. In the past syphilis has proved to be the most common cause of joint neuropathy.

"Radiographically, two patterns have been described: atrophic and hypertrophic. The atrophic form is characterized by massive bone resorption with virtual disintegration of the joint. This pattern is encountered most commonly in the hip, shoulder, and foot. The hypertrophic form is characterized by severe joint destruction, periarticular new bone formation, osteophytes, fractures, and osseous debris. This pattern is most commonly seen in the knee, elbow, and ankle" (Alpert et al. 1996:101).

Non-venereal (Yaws)

Yaws is found in very specific climatic conditions, primarily humid, warm environments. There are some differences between non-venereal syphilis and venereal syphilis. The main difference is that yaws attacks children and is transmitted via open skin (Hackett 1963). The paleopathology is also expressed differently. Like in venereal syphilis, in yaws the most noticeable diagnostic bone changes occur during the tertiary stage, where the skeleton undergoes chronic bone involvement (Hackett 1946; Steinbock 1976). Bone lesions and alterations occur mostly in the tibia; however, other bones like the fibula, clavicle, femur, ulna, hands and feet are affected (Ortner 2003). In yaws the cranium is also affected. In twenty cases of yaws Hackett (1946) found nodes on the skull and three cases of gangosa. Gangosa is caused by the disease destroying the hard palate and nasal cavity. A clinical study of 121 patients who were infected with yaws in Brazil 3.3% showed signs of gangosa (Furtado 1957).

Yaws also affects the metacarpals, metatarsals and phalanges which seem to be swollen by subperiosteal bone apposition, parallel to the cortex and resorption of the original cortex (Steinbock 1976; Marks et al. 2015). The tibia plays an important role in all types of syphilis, not only producing sabre shin as it undergoes subperiosteal apposition of bone along the anterior border (Rothchild and Rothchild 1995; Hong 1997). One noticeable indication of yaws are the crater like depressions found on the crania, although they do not perforate the inner table, there is a light bony thickening. A number of signs appear in tertiary yaws like gangosa which is a result of destruction of the posterior nasal cavity and gummatous osteomyelitis, which is a localised destruction of the long bones. (Steinbock 1976; Ortner 2003). The gummatous lesions appear to be irregularly oval with the long axis parallel to the shaft of the bone. The periosteal reaction in yaws is not as noticeable as it is in syphilitic osteomyelitis

Bejel

Bejel is found only in hot dry climates. This form of teponematosis affects the human population living around the middle course of the Euphrates River. Hudson (1937) did a study on bejel, which is a non-venereal form of syphilis. In his study he researched how bejel affects the Arabs in this region. Hudson examined "757 patients, male and female, 15 years and older, of whom 455 (60.1%) had bejel in childhood; 139 (18.4%) had bejel at a later specific age, making a total of 594 (78.5%) who acknowledged treponemal infection. Only 46 (6%) had the initial infection after the age of 30" (Hudson 1937:1004). Hudson had noticed signs of bejel consist of lesions similar to greyish patches with desquamation, ulcers on the mucous membrane of the oral cavity are non-existent (Hudson 1936). Examination of

saliva and scrapings from lesions indicate these lesions are full of spirochaetes with the morphology and characteristic motility of *Treponema pallidum.* Papules can be seen around the genitalia, as well as in the folds of the skin. Bejel differs from venereal syphilis as the onset of the papules around the genitalia is not determined by sexual intercourse.

Papules thrive in warm, moist areas and when they occur on the body it is usually in a circinate configuration. When they appear in the nasal cavity the nasal bones are affected which softens the nasal bridge leading to its collapse. The long bones are also affected, albeit infrequently, with tenderness and swelling of the epiphyses as well as periosteum (Hudson 1936). Within the year the lesions recede, and the child is back to normal. At the time Hudson wrote his paper, the only treatment given were injections of arsenic or bismuth that had to continue for some time. Bedouin, however, did not worry about following on-going treatment, believing that when the child ages it will not experience further symptoms. However today newer treatments are used to cure the disease like penicillin. Somewhere between the child's second and seventh decade of life it is possible an ulcer in the pharynx or on skin may appear or even a gumma of either the nasal bones or a long bone could occur, failing this they may experience aching and throbbing bones (Hudson 1936; Giacani and Lukehart 2014).

"In a clinical study in Iraq 3,507 cases of bejel were recorded. Some 30 percent of the cases showed active late lesions, and of these skin gummata (215 cases) were the commonest, followed by nasopalatal destruction (113), late periostitis (74), laryngeal involvement (59), and pigmentary changes (57). Ostealgia (621) was the prodominent symptom but is probably due to several causes, only one of which is bejel" (Csonka 1953:98).

Bejel is not known to affect the cardiovascular system and it results in fewer miscarriages.

Pinta

The Western hemisphere is home to pinta which, when contracted is mild and only causes discolouration of the skin. Pinta is caused by the bacteria *T. carateum* and anyone is prone to contracting it, although it seems to be young adults who are mainly susceptible (Giacani and Lukehart 2014).

People mainly contract pinta in their home through family members, even though the method of spreading pinta is not known, it is believed when unaffected skin touches a lesion it then becomes affected (Giacani and Lukehart 2014). Like in other forms of treponemal disease, there are three stages to pinta, a primary, secondary and tertiary stage which increases in the severity of the skin lesions as the disease progresses. Despite government attempts at eradication, the disease still exists in Latin America (Stamm 2015). Unlike other forms of treponematosis, pinta does not affect the skeletal system as it is primarily an infectious skin disease. Therefore, it is not an important form of treponemal disease to be discussed in any great detail in a paleopathologial study.

Are there sub species of *Treponema pallidum*?

To understand whether treponema has sub species, or whether it is one species that has merely adapted to changes in its environments and modes of transmission will aid in understanding the evolution of this disease. This is one of many interpretations of the evolution of treponemal species and it has been argued to be rather controversial (Mulligan et al 2008). It is subsp. *pertenue* that diverged into subsp. *endemicum* and that emerged into subsp. *pallidum*, which was the most recent addition to the treponema strains. The evolution of the earlier treponema strain *pertenue* was associated with human migration into different climatic environments and human development, that forced the earlier treponema strain *pertenue* to evolve (Harper et al 2008). If treponema strains had evolved, then there should be genetic markers to indicate if they evolved into different subspecies, or whether they remained too similar to be classified as a different subspecies.

Yaws is caused by bacteria *Treponema pallidum* ssp. *pertenue* (TPE) whereas syphilis is caused by strains of *Treponema pallidum* ssp. *pallidum* (TPA). When diagnosing yaws and syphilis epidemiological characteristics and clinical symptoms are employed to determine the difference between the two diseases. The genetic diversity of the various treponema strains shows certain connective characteristics that prove the diversity between treponemae may be limited to the changes in virulence between syphilis and yaws. The treponema strains show genetic similarities between syphilis and yaws through the closeness of the genome structure and genome sequencing of 99.8% indicating the two pathogens must be closely related (Čejková et al 2012).

Although the various treponemes share a similar morphology and antigenic profiles, their mode of transmission is different as are the symptoms they produce. These differences are caused by genetic variations in the treponema strains. Within the treponema strains differences in the genome sequencing are found in the specific regions of the chromosome, which can distinguish between species and subspecies (Centurion-Lara et al 2013). The tpr genes are responsible for the virulence variations between the various treponema species. The treponema species have their own specific tpr sequence changes, separating their classification of sub-species and species.

In a study of the *T pallidum* sub-species *endemicum* Bosnia A strain was compared with the genomes of uncultivated pathogenic treponemes. The results showed there were no major genome rearrangements in the Bosnia A strain, although they were grouped towards other yaws causing strains, while syphilis causing strains clustered separately (Štaudová et al. 2014). The study found Bosnia A genome was not just related to yaws treponemes, but also contained several sequences related to the syphilis strain.

Theories on the origin of syphilis:

Columbian theory

The Columbian hypothesis is the first of three arguments concerning the origins of syphilis. This argument suggests that syphilis began in the New World and in the 1490`s Columbus conveyed it to the Old World. As syphilis rapidly spread throughout Europe during the fifteenth and sixteenth centuries, it was so virulent, that it was believed that a new and deadly disease had surfaced for the first time (Hudson 1963; Knell 2004). George Armalagos stated that there is now a new Columbian hypothesis: one that better fits the available evidence (Armelagos et al 2012; Harper et al 2008). Skeletal evidence from many pre-Columbian sites in the New World indicates a high prevalence of treponemal disease paired with a low age of infection and an apparent absence of lesions attributable to congenital syphilis. This suggests the presence of a non-venereal form of the disease, similar to modern-day yaws or bejel and one not passed through the placenta (i.e., a congenital variant), was present (Powell and Cook 2005). Due to differences in climate, clothing, and sexual practices, Renaissance Europe would have represented a very different environment than that present in Hispaniola, the location of Columbus' first arrival in the New World. The subspecies of the bacterium responsible for syphilis, *T. pallidum* subsp. *pallidum*, would have thus encountered a very new set of selective pressures upon arrival in the Old World (Armelagos et al. 2012). Perhaps it was exposure to this unique host environment that resulted in the birth of the *T. pallidum* subspecies that causes syphilis.

The pre-Columbian theory

The pre-Columbian hypothesis differs from the Columbian hypothesis, as it supports the existence of treponemal disease in the Old World before the 1490`s, (Holcomb 1937; Hackett 1963). According to this hypothesis, syphilis in the Old World was either mild, similar to leprosy or any other bone remodeling diseases (Buret 1891; Sudhoff 1925; Hudson 1964; Hackett 1963). According to Waldron (2009) during the fifteenth century syphilis really took-off, becoming virulent (Waldron 2009). Furthermore by that time syphilis was recognized by the medical profession (Steinbock 1976), and information about the disease was disseminated widely in relation to the commencement of the printing press. (El-Najjar 1979; Kampmeier 1984). There are some researchers who believe syphilis evolved in the Old World due to changes in life-style and the environment, (Brothwell, 1981; Cockburn, 1961; Hackett, 1963).

The Unitarian theory

A third, unitarian hypothesis argues that the agent of syphilis has evolved with human populations, and was present in both the Old and the New World at the time of Columbus' explorations in the Americas. Hudson (1963,1965) maintains that pinta, yaws, endemic (nonvenereal) syphilis, and venereal syphilis are four syndromes of treponematosis, a single disease caused by *Treponema pallidum*, which evolved simultaneously with humans. The syndromes form a biological gradient in which various social and environmental factors produce different manifestations of treponematosis (Hudson 1965).

The unitarian faction believe that the four clinical syndromes are not based on aetiology but instead on epidemiology and geography due to the unavailability of tests in which the treponemal parasites can be differentiated. Endemic non-venereal syphilis was at one stage wide-spread, but now is sporadic, owing to the evolution of urbanisation and improved hygienic practices (Hudson 1964).

The Combined theory

Alexander Porro and colleagues (2009), were in a quandary as to which faction to support so they combined both the pre-Columbian and Columbian faction therefore producing a third faction who believe syphilis arrived in Italy at the end of the fifteenth century with the attack on Naples by Charles VIII and his army. According to the combined theory there was a lack of pathology that could be associated with syphilis in antiquity, probably because leprosy was often confused with syphilis. Crane-Kramer (2000; 2002) argued that there was no confusion between leprosy and syphilis because most of skeletons of people buried in leprosoria cemeteries had bony changes of leprosy, nut no syphilis-caused changes were present.. Despite this fact they believe syphilis did exist in Europe before Columbus sailed in 1492. During pre-Columbian times syphilis was present on the Asian and American continents, and after 1500 syphilis took a hold on Europe as can be seen by the descriptions written in medical journals of the time. Alexander Porro et al. (2009) also argue that Columbus and his crew introduced a virulent strain to Europe which was nothing like they had ever seen before. This in turn created a combined infection by two independent strains of treponema which were uniquely isolated from one another, causing a more severe reaction. It was not just Charles VIII and his army of mercenaries and prostitutes who spread syphilis after the siege of Naples but also the movement of displaced people like the 160,000 to 400,000 Jews who

were expelled from the Iberian Peninsula. An aggressive treponema emerged caused by the last syphilitic mutation that occurred in Europe towards the end of the fifteenth century (Porro et al. 2009). The New World on its own can not be the main cause of the syphilitic reaction seen in Italy during 1495 due to the outcome of the Oslo study. The Oslo study illiterates that the time period for secondary syphilis to develop is at least 10 months which places 99% of Columbus' crew who may have been affected during this time to have been located in Haiti, past the communicable stage by 3 months (Clark and Danbolt 1955; Gjestland 1955). However it is known that syphilis is still communicable to other people during the early tertiary stage.

Theoretical evidence that might suggest that leprosy and syphilis were being confused in Pre-Columbian times.

Until recently there have been scholarly arguments suggesting that syphilis and leprosy were being confused in pre-Columbian times. However, Crane-Kramer (2000; 2002) and Mays et al. (2003) argue that syphilis and leprosy were not being confused during this time period. According to those authors, diagnosticians were well aware of what leprosy was as evidenced by large numbers of skeletons with leprosy-related pathological changes in leper cemeteries while there are no skeletons with specific for syphilis changes in those cemeteries. Interpretation of ancient literature now presents a new line of thinking where leprosy stigma of being unclean, sexually active and morally unsound was brought on by inaccurate translation of the Hebrew word tsara'ath. Therefore, anything in literature that mentions that a person should be careful of not having sex with a leper, that leprosy is sexually transmitted or hereditary should be dismissed as religious ideology brought into medical teachings that has no bearing on the disease.

Crane-Kramer, based on modern publications interpreting old texts, argues that there was no obvious medical literature confusion of sexually transmitted treponematosis with leprosy. The only confusion is the social immoral context that was implicated onto the stigma of leprosy. These social and religious implications were known in medieval times, however medical literature like Bernard de Gordon's Lilium Medicinae might have re-alliterated some of the religious stigma like do not have sex with a menstruating woman. Bernard de Gordon's work details that what he calls "Leprosy" can be passed on through sexual intercourse, and also become congenital/hereditary. This author also says that spending much time with lepers

may result in contagion (Gordon 1491). Sexual transmission and congenital disease are both diagnostically characteristic for syphilis while spending too much time with lepers is more likely to relate to leprosy due to breathing in the baccilli that sprayed from nasal passages of the infected individual. The 13th century scholar Bartholomeus Anglicus states that leprosy was caused by "intercourse with a woman after she had been with a leprous man, heredity, and feeding a child with the milk of a leprous nurse" (Crane-Kramer 2000). Again, it is known that leprosy can be passed on to children from breast feeding. However again sex and heredity are both results of syphilis. Another example is Theodoric (thirteenth century) who said "leprous" women were venerally contagious; he successfully cured genital lesions with mercury (Hudson 1968). Mercury is ineffective in curing leprous lesions. Arguments relying on ancient/medieval texts should not be based on their translations into modern languages because those translations may be influenced by opinions or theories held by translators. Grammar, syntax and spelling of old texts are sometimes unclear and a translator may be tempted to give words and phrases interpretations closer to what seems reasonable at the time of translation, rather than at the time of writing of the original texts. Interpretations of translations by modern authors may further deviate based on those modern authors opinions.

Regardless of Crane-Kramer's argument it is known that religious beliefs influenced medical theory it does not make sense that leprosy or syphilis were spread by a menstrual blood. It appears that discussed above medical literature mixes together real ways of spreading both leprosy and syphilis with incorrect information. Hence, the medieval authors did not make a clear distinction between leprosy and syphilis. Medical reasoning based on observations may have faults given our own modern understandings of diseases.

Even if we accept that ancient medical practitioners distinguished leprosy from syphilis does not mean that syphilis and leprosy occasionally could be mistaken by the average physician. Diagnostic errors happen even today. Moreover, the ancient Greek and Latin meaning of the word “leper” or “leprosy” was simply “skin lesions”. Skin lesions occur in both treponematosis and leprosy. Even though it is evident that syphilis and leprosy are different diseases they can still mimic signs of other separate diseases (Cakmak et al. 2019). This ability creates problems for physicians to diagnose the correct disease from just clinical signs (no laboratory tests were available in Middle Ages) even in today`s society.

After all, syphilis has been repeatedly called the great imitator of other diseases. This is evidenced in modern medical literature (Fitzgerald 1981; Baum et al. 1983; Balagula et al. 2014; Domantay-Apostol et al. 2008; Kundakci and Erdem 2019). With clinical understanding and diagnostic tests for both syphilis and leprosy there are instances where physicians have reported that they have misdiagnosed the disease (Scotti et al. 1970; Nsibambi 1981; Sehgal et al. 1993; Khandelwal et al. 1994; Nwosu et al. 1994; Fonseca et al. 1999; Pandhi et al. 2005; Dupnik et al. 2012; Souza et al. 2013). If this can happen with our understanding of the disease in recent times, then why pre-Columbian physicians also could not occasionally diagnostically confuse these two diseases. In Bernard de Gordon’s work Lilium Medicine he questions his own diagnosis of leprosy. He learnt from the works by previous scholars like Galen and Avicenna who state that leprosy begins in the face.

“I wanted to absolve him, and I repeatedly asked him whether any signs had appeared in his face. He had remained quite like this for about twenty years, and he still lives with that ugliness of the extremities but without anything showing in the face. Hence I guess, with the

conjecture closest to the truth, that it was not leprosy; nor does it seem possible that he would have lasted for so long without his face being disfigured. And therefore, even though I once thought differently, now that I have labored diligently in this work, I am of another opinion and I would no longer judge him leprous. However, God knows the truth. I do not know". (Demaitre 1985:341).

Some of the crania that were taken from an ancient leprosarium on Rue de Douai and are now located at Musee Dupuytren of Paris. A syphilographer from Paris, Lancereaux (1873) diagnosed two of these syphilitic crania from the leprosarium. After being further examined and photographed by H.U. Williams he stated 'they are as surely syphilitic as it is possible for crania to be' (Holcomb 1941: 161), which illustrates the presence of syphillis in Ancient Rue de Dounai.

Møller-Christensen examined skeletal remains from Æbelholt where he found remains which he thought had leprosy. However, in one case he would change his diagnosis from leprosy to ergotism. Later Lefort and Bennike (2007) studied Møller-Christensen`s work arguing that skeleton might have treponematosis. This case is a good example of mis-diagnosis as even in skeletal remains it is hard to actually diagnose pathologies displaying similar traits.

The aforementioned information argues that both diseases are similar in traits which has led to the capacity to that from time to time confused modern physicians. Both syphilis and leprosy share similar characteristics including symptoms such as cutaneous lesions with high polymorphism that have hindered a differential diagnosis being made. Signs which frequently occur in patients of both diseases are cutaneous in nature and can manifest in practically all forms of dermatologic lesions including macules, papules, nodules, tubercles, plagues and

other infiltrations. This makes for clinically diagnosing both of these diseases to be challenging as the lesions are not specific to any one disease but can be observed in various different forms and stages throughout both diseases or sometimes can even occur in combinations (Souza et al 2013). This can lead to cases being misdiagnosed, exampling the case of a male presented with widespread nodular eruptions, which show periappendageal and perineurial granulomatous on a histopathology, causing the pathologist to diagnose the patient with leprosy instead of syphilis (Pandhi 2005). “The epithelioid cell granuloma in syphilis has been previously described as a granulomatous response in which a perineural involvement destroys the appendages has also been noted” (Pandhi 2005:257). Lepromatous leprosy is a slow and progressive disease which attacks the nervous system causing loss of sensation (Ooi and Srinivasan 2004). Neurosyphilis can occur anytime, even in the primary stage (Marra 2009).

While diagnosing indeterminate leprosy on the basis of intraneural and perineurial inflammation and even disruption of nerve parenchyma, it would be wise to keep in mind the possibility of occasional nerve involvement in secondary syphilis. Nerve changes suggestive of leprosy have been reported in dermatological conditions with no clinical evidence of leprosy such as lupus vulgaris, secondary syphilis, lichen planus and morphea (Khandelwal et al. 1994).

There are more neurological changes that occur in lepromatous leprosy. One of the signs is peripheral neuropathy. During peripheral neuropathy a patient can show many symptoms including cutaneous sensory loss appearing in the pinnae of the ears, dorsal surface of the

hands, dorsemedial forearm, dorsal feet or anterior calves, nose malar regions, breasts, abdomen and buttocks. Ulnar-innervated intrinsic hand muscles are one of the first motor deficit disorders which produces weakness, atrophy then finally claw hand deformity (Ooi and Srinivasan 2004). In comparison, if a person presents with neuro-syphilis, general paresis occurs ten to twenty years between infection and symptoms. These symptoms include dementia, irritability, confusion and delusion. with a distinct feeling of melancholy. By the time the individual reaches this stage they can show signs of cranial nerve abnormalities which are rare, they also present with other problems like intention tremors of the face, tongue and extremities which produce dysarthrias along with handwriting abnormalities, loss of tone in the facial muscles and limbs. The idea of melancholy in the past has always been associated with leprosy. In syphilis neurosyphilis is known to affect the psychological and emotional states, whereas leprosy can cause depression due to being displaced from society (Bakare et al. 2015). The clinical literature from 1868 states that a patient had become delirious and even violent. After exhibiting these symptoms the patient became listless and profoundly melancholic (Smith 1868). Avicenna, mentions in medieval literature that lepers had not only developed bad and crafty habits but they were also rather aggressive irascible, depressed, they distrusted people, and were melancholic. (Demaitre 1985). Avicenna appears to be describing syphilitic symptoms and not leprosy.

"Tabes dorsalis affected the largest group of patients with neurosyphilis in the pre-penicillin era. The early manifestations were lightning pains, paresthesiae, pupillary change, and loss of tendon reflexes. Later symptoms are ataxia, visceral crises, optic atrophy, ocular palsy, Charcot's joints, and ulcers of the feet, loss of pain sensibility also occurs following the so-called zones of Hitzig in the legs or feet, upper chest, inner side of the forearm, or in a

butterfly distribution over the face. Pain sensation is often strikingly impaired, more so than that of touch and cold" (Simon 1985:610), which can also occur in leprosy.

Skeletal changes are a good indicator of either similarities or variations particularly when the pathology changes the bone structure which can be visually seen on living people. One good example of this is in leprosy where the disease attacks the palate, alveolar process and nasal spine. Clinically this in both treponemal disease and leprosy will cause a rasping sounding voice and a collapsed nose. Paleopathologically when diagnosing syphilis and leprosy there are distinct signs which to look for. In syphilis the patient will be suffering from a destroyed hard palate, the alveolar process of the upper jaws with portions of nasal septum, the nasal bones the under surface of the sphenoid as well as the floor of the sphenoidal sinus will also be destroyed. Facies leprosa in the cranium is always a dependable sign in diagnosing leprosy. Facies leprosa is identified by atrophy appearing on the anterior nasal spine, atrophy of the maxillary alveolar process mostly in the incisor area and inflammatory changes of the superior surface of the hard palate (Møller Christensen 1978; Baker et al. 1988). In 1869 Sir Duncan Gibb donated a pathological collection with one of the cases being presented at the Pathological Society of London in 1854. This case was a 30-year-old Canadian prostitute who had been prescribed mercury for her syphilis before she died which was 5 years before her case was presented (Turk 1995). She presented with periostitis of the bones of nearly her whole skeleton with subsequent ulceration that rarely healed. There was also penetration of the dura mater with a loss of nasal bones and perforation of the roof of the mouth. Her appearance was thought to be due to the effects of syphilis as well as mercury. In 1849 Hutchinson and Gibb undertook extensive research into the effect syphilis and mercury had

on the body without any mention of exacerbation of bony syphilis (Turk 1995). The main complications of mercury referred to were increased salivation, gingivitis and tooth loss.

Syphilis and leprosy have a wide array of signs and symptoms, which are quite often seen as two distinct diseases, however occasionally these diseases will present themselves in a way that will cause diagnostic confusion.

It is known that patients can present with more than one diagnosed disease although the inter-relationships as well as the effects of multiple diseases have not been given the correct attention they deserve (Feinstein 1970; Almirall and Fortin 2013). In recent times syphilis and leprosy, with or without HIV, have been clinically misdiagnosed. Therefore, it is possible that individuals in pre-Columbian times may have been riddled with many diseases thus confusing diagnosticians.

Lastly, Roberts's (2002) generic statement concerning lepers being segregated in Later Medieval period may not be correct. It is suggested by Simpson (1842) that there may have been inaccurate diagnosis, as these methods were mostly unconventional although some learned diagnosticians would have been able to tell the difference. There is a chance the patient may not have appeared to have had leprosy so would not have been diagnosed with leprosy or even segregated. Some patients present with a low resistant form of the disease therefore their symptoms would not have been obvious. Another cause of this phenomenon

could be that the less severe cases were left to carry out their normal lives whereas the lepromatous leprosy patients presented with quite obvious symptoms (Roberts 2002).

Is syphilis Columbian or pre-Columbian?

The pre-Columbian debate rests on the archaeological interpretation of ancient skeletal remains and textual sources from individuals who were there at the time or later on. It can be shown that syphilis existed in France, Spain, Naples, England and elsewhere before Columbus returned from his voyage, and some say even before he started his first voyage (Holcombe 1937). Secondly, seeing that Columbus returned from Haiti in March 1493, it is strange that syphilis was only argued to have originated from the West Indies around 1530, many years after the event. Lastly it is strange that syphilis did not exist in a tangible form until 1495. However, syphilis was not recognized as a definite disease until it appeared in pandemic form (Garrison 1921) and even then it was not definitely diagnostically separated from a number of other diseases. There are arguments against the notion that leprosy was confused with other diseases and that physicians were well aware of how to properly diagnose leprosy (Mays et al. 2003). The lack of evidence to support the Columbian theory is quite perplexing; but then again it is abundantly clear that finding definite evidence for pre-Columbian syphilis is just as perplexing, which means the case must be decided on the balance of evidence produced (Whitwell 1940).

This tale of an unpleasant disease not being in existence locally, until introduced from some distant country is really an old and well known story: The tale of syphilis is the same as that told of leprosy, which, according to the current fable, was introduced into Europe by the Crusaders on their return from the Holy Land in 1096 (Whitwell 1940). Although there is quite clear evidence that it existed in Great Britain and Europe before the Crusaders even started out on their enterprise (with the creation of a leper hospital for women around 1137-1204 (Bennet 1891). The key to the situation in both cases is probably the same, namely,

elementary means of transport of information, and the elementary medical knowledge of those days.

In contrast the Columbian theory rests on the words of Ruiz de Isla, Oviedo and Las Casas who claimed that the voyage of Columbus was responsible for the implanting of a new and terrible disease (Holcomb 1937; Crosby 1969; Hudson 1962; Arrizabalaga 1997; Quétel 1990). These men either witnessed the disease in Spain, or heard the confessions of the Indians (Native Americans), who claimed the disease had existed in their home-land for many years. However, they wrote about this new disease many years after it became an epidemic at the battle of Fornovo, Italy.

Nonetheless, syphilis being a new disease according to the Columbian faction, would explain why it was so severe, intense and extremely virulent at the beginning of the epidemic in 1495 (Baker et al. 1988). The Columbian faction argues that if the Europeans had never been exposed to the treponema spirochete their immune systems would struggle to fight it off. They also argued that before Columbus returned to Spain there was no clear evidence to support a syphilis connection, even though there were written medical and historical documents describing various types of skin lesions, pustules, ulcers and venereal characteristics (Whitwall 1940).

If syphilis existed in the Old World prior to Columbus, then there should be some physical evidence to support the claim. According to the Columbian faction the Old World paleopathological evidence for syphilis is not as convincing as that from the New World (Harper et al. 2011). The signs of syphilis in pre-Columbian cases have not always been pathognomonic to make its diagnosis unquestionable. Furthermore, the Columbian faction

attempted to add weight to their theory by arguing that the cases which might be syphilitic, either do not have the radiocarbon dating to prove the person died before the outbreak of syphilis in 1495, or that the radiocarbon dating range was recorded as being before and after that out-break of syphilis. However, many reports of pre-Columbian syphilis in the old world have reliable archaeological dating and clear pathological signs (Stirland 1991; Henneberg et al 1992; Henneberg and Henneberg 1994; Erdal 2006; Von Hunnius et al. 2006; Cole and Waldron 2011). In order to ascertain which theory carries more weight, the whole mystery of Columbus' voyage must be assessed.

Christopher Columbus was an explorer who became famous in relation to his voyage to India that led him to the Americas. He was also blamed for bringing back a new disease from the New World that would reach epidemic proportions in Europe. However, it is clear that even that might be up for debate, as writers of the time question whether syphilis was a newly introduced disease or was always present in Europe. The propagandists of the Haitian origin of syphilis seldom go further into the testimony of Ruiz de Isla than the opening account of the first chapter. The first chapter devoted to the origins of syphilis gives two different accounts. The first attributes the disease to the voyage of Columbus, and the second identifies it with the ancient disease of the Greeks, called "lichens", and described by Pliny more than fourteen hundred years earlier, and called by him mentagra (Holcomb 1937).

Many early scholars from 1496 to the 1520's believed that syphilis was in fact mentagra and an old disease. These scholars were Sebald Clamosus (1496), Gasper Torrella (1497), Otto Raut (1501), Antonius Beneventus (1502), Wendelin Hock (1502), Jacob Grunpeck (1503), Jacob Cataneus (1517), John le Maire (1521), Jaques Bethencourt (1527), and Joseph

Struthius of Posen (1540). This was a view which was also held by Ruiz de Isla (1539) who added to it the idea of Columbus bringing syphilis back from the Americas. The aforementioned authors also used other names to describe the disease such as pudendagra and mentulagra before it was finally named syphilis in 1530 by Fracastorius. He, like Ruiz de Isla mentions both sides of the debate: an old disease linked back to Pliny's mentagra or blaming it on Columbus' voyage bringing syphilis to Spain (Holcomb 1937)

Whereas the main protagonist for the Columbian theory Ruiz de Isla may have initiated the hypothesis for an American origin he also saw similarities to an older disease - mentagra. It is not impossible to have it both ways. Hypothetically Columbus may still have brought an American strain of treponema back to Spain somewhat different from the European strain, thus causing a super-infection when both strains infect the same person.

It is important to recount the first voyage of Columbus as it will create the basis for the argument as to whether the disease was likely to have been transferred from America to Spain and Portugal.

Christopher Columbus embarked on his journey to the Americas on the 3rd of August 1492 from Palos, Spain. He left with three ships: the biggest was the Santa Maria which carried fifty–four men including Columbus and his officers (Downing 1916). The Pinta and the Nina were smaller ships and only carried eighteen men each. On the 12th of October 1492 Columbus landed on San Salvador. The crew would only stay there for a short period before they would leave to sail around the islands in that area. One of the many islands they would visit was Cuba. They may have stayed in this area from 28th of October to the 12th of November (Morison 1939).

It was recorded that on the 20th of November the Pinta commanded by Martin Pinzon deserted the other ships in order to pursue their own financial interests in Haiti. It is unclear as to what they were doing with the natives in this area and whether their time away from Columbus' fleet led to them getting infected with treponemal disease. It has been stated by Holcombe, it was believed that Martin Pinzon was infected with what was known as the New Disease (Holcomb 1937).

On the 27th of November Columbus wrote in his journal describing the health of his crew. He says up to the present time all of his crew were in good health, this included all three vessels (Morison 1939). No one had minor health problems not even a headache. He does record one man who had pain of gravel, from which he had suffered all of his life. The term gravel found in the Oxford dictionary says that it is the aggregation of crystals formed in the urinary tract. Ibn Sina stated the treatment for kidney calculi was elimination of materials with the potential to form calculi, breaking the calculi, and removing the gravel via the urinary tract (Faridi et al. 2012). On the 6th of December 1492 Columbus, with the Santa Maria and the Nina, arrived at Haiti, where on Christmas Day the Santa Maria was wrecked (Downing 1916).

With the loss of the largest ship, the Santa Maria, Columbus was forced to leave a garrison of thirty-nine men in Haiti. Those men that were left behind at Navidad were suffering from sores. A surgeon by the name of Maestre Juan was left behind with them to treat them for the sores and perform other medical tasks (Lopez 1976). It is possible these men interacted with the natives, picking up syphilis as a result of their liaisons, but even if they did they could not be held responsible for the introduction of syphilis to the Old World as they were murdered

by the natives. On the 4th of January, 1493, Columbus set sail for Spain, and two days later, on the northern coast of Haiti, they rejoined the Pinta, whose commander had been delayed trading with the natives and searching for gold (Downing 1916).

Those that advocate the American origin of syphilis all agree it originated in Hispaniola. Even though both the Pinta and Nina were separated on different areas of the island of Haiti, they both appeared to have spent equal amounts of time there and perhaps around its inhabitants. This being the case the probability of at least one ship bearing infected sailors increases (Downing 1916; Morison 1939).

According to Ellis Hudson's evolution of treponematosis theory, venereal syphilis was only found in urban areas where the people were fully clothed, the hygiene of the area is also greatly improved compared to areas with non-venereal syphilis (Hudson 1965). In relation to Haiti, the recorded birth place of syphilis, given Columbus' descriptions of the inhabitants was not at a socio-economic level where they could have been infected with venereal syphilis. Therefore, they must have suffered from yaws. Given that non-venereal syphilis has existed in the Americas for a long time, then what was in Haiti by the time Columbus and his crew arrived must have been endemic yaws. Endemic yaws would have made it easier for the crew to become infected. Non-venereal yaws is mostly a child's disease spread by skin to skin contact. However, adults can get infected with yaws if they connect with a child's lesion/ulcer against an open wound on their body. There have been recorded cases in which adults have been clinically diagnosed as having yaws (Grin 1953). When Columbus' crew arrived in the Indies they would have had to either interacted with the children in villages or had sex with villagers who had yaws.

With the Santa Maria gone, the two remaining ships the Pinta and Nina carried fifty men each not including the ten Indian prisoners being transported to Spain. On Monday 18th of February Columbus and his crew landed on the Azores. The following day half of his crew was captured by the islands Portuguese captain who administered a population of 100 Portuguese citizens. Columbus was allowed to leave the Azores on a condition that he would take 100 Portuguese of the island's population to Castile (Morison 1939). There is no written evidence that suggests that Columbus was allowed to take his crew as well. Meaning Columbus may have left his men captive until this agreement was completed. If this was so, then half of his crew, who possibly could have had treponemal disease, were left behind, reducing their chance of spreading syphilis throughout the Old World.

On the 4th of March Columbus had arrived at Lisbon, where he sent a letter to the king of Portugal. It wasn't until Friday 8th of March that the King of Portugal sent a reply to Columbus. The king invited Columbus to the Valle del Para'yso which was nine leagues from Lisbon. Columbus spent three days in the Valle del Para'yso with the King of Portugal before he left for Spain on the 12th of March (Morison 1937). This means that Columbus and his crew had spent at least eight days in Portugal giving them many opportunities to spread syphilis within Portugal. However, no literature exists that records any knowledge of an outbreak of a new virulent disease.

On the 15th of March 1493 the Nina reached Palos, with the Pinta arriving later in the afternoon. Pinzon received a chilly reception upon his arrival in Palos as he not only abandoned Columbus in Haiti, he also abandoned the Pinta during a severe storm. As Martin Pinzon died three days after his chilly reception, so close to his arrival back to Spain it has

been argued that it was due to syphilis (Morison 1939). De Isla even claimed to have treated Pinzon not long after Columbus returned. Both Holcomb and Hudson argue that it was more likely that Pinzon picked up yaws on one of their prior trips to the slave coast rather than Haiti (Holcomb 1937; Hudson 1946). Hudson also points out that Pinzon was common name back in the 15th century and Ruiz de Isla could have been treating anyone by that name.

However, this idea that Ruiz de Isla treated Martin Pinzon has been reinforced in latter works by Deborah Hayden (2003) who, through referencing Hudson`s work, incorrectly described the meaning of his words. It was Hayden who stated that Ruiz de Isla treated Martin Pinzon, as well as other sailors for a disease they contracted through being with women in the West Indies, before passing the disease onto prostitutes who worked along the water front in Barcelona (Hayden 2003). However, due to his chilly reception it could have wbeen a suicide as well as syphilis. Moreover, were Pinzon suffering from syphilis, in three days of his chilly reception coupled with poor health resulting in death, he could hardly have a chance for sexual intercourse with locals. After resting in Palos, Columbus travelled overland to Seville with several seamen and six Indians, arriving on Palm Sunday, 1493. Columbus spent several weeks in Seville until he was summoned to appear in court in Barcelona arriving around the middle of April (Downing 1916).

When Columbus attended court, he chose some of his men to accompany him to Barcelona giving them ample opportunity to spread syphilis throughout Spain. However, if the crew of the Nina and Pinta as well as the Native Americans were infected with syphilis while they were in Haiti, then they should have displayed some signs of the disease before landing in Palos, and most certainly before they reached Barcelona.

The length of the voyage from first landing in the Indies to the arrival in Barcelona calculated with the progression of syphilis could be worked out. It is unknown where the sailors became infected, however the sailors who were left on Haiti were reported to have skin ulcers (Lopez 1967). Therefore, if any sailor was infected and escaped the notice of the medical assessments, it would be logical to start the timeline of the progress of the disease from shortly before the sixteenth of January when they left Haiti until they arrived in Portugal on the fourth of March. In those seven weeks Columbus' crew should have shown signs of secondary syphilis (in the twenty first century syphilis takes two to eight weeks to reach the secondary stage) (Mattei et al 2012; Fenton et al 2008).

If this was a new disease and judging how quickly it was progressing after Fornovo, then well developed signs of the disease should have been easily seen. From their arrival in Portugal to their arrival in Barcelona in mid-April, was about five to six weeks. By this stage the crew should have displayed tertiary stage syphilis. According to the Oslo study the time period for secondary syphilis is at least 10 months. This then places 99% of Columbus's crew who may have been infected in Haiti past the infectious stage of transmission by 3 months before Charles VII recruited his army in Lyon (Clark and Danbolt 1955).There is no way Columbus would have brought his crew into the sovereignty of Spain or to meet the Royal family if they were displaying symptoms of an unknown horrific disease which could have been confused with leprosy. The small number of crew who might have been infected would have found it difficult to start an epidemic considering the probability of transmission of syphilis is 60% every time one person has sex. This means that one infected sailor needed to have sex with the same person twice to have the highest probability to pass on the infection.

The conditions on ships were appalling during this time; sailors usually suffered from scurvy or a similar condition, especially if they were at sea for a long time (Hirschmann and Raugi 1999). The ships log did not report any member of the crew being inflicted with an unusual disease. However, out of the ten Indians (Native Americans) on board one died the day they landed with three others sick, but the records made no mention of a strange, malignant disease (Downing 1916). If anyone on board the Nina and Pinta were suffering from a strange disease, their physician Maestro Alonzo of Moguer was duty bound to report it, but no such report was ever made.

The fact that no signs of any disease were picked up by the Nina's physician Maestro Alonzo makes it unlikely that syphilis was amongst the Indians or crew (Downing 1916). For when syphilis was first recorded at the Battle of Fornovo in 1495 the description was vivid. Many years later when Fractorius described the early symptoms in his book published in 1530 the virulence of the disease had not regressed. He described the early signs of the disease as thus: - foul ulcers of the genitals, sores on the lips, tonsils and nose; terrible pains in the joints, bones, muscles and nerves; swelling of the legs and face, loss of hair, fever and no desire for food (Fracastorus 1930). If syphilis infected the crew in Haiti and the disease was new to the Old World, then these symptoms should have been blatantly obvious to both physician and crew.

Even though there seems to be no immediately written document that visually describes a new and terrible disease on the crew or Indians during their stay in Spain, there are written accounts that were published many years after the outbreak of syphilis in 1495 by those who claim to have witnessed it in Columbus' crew and in the Americas from where it originated.

Oviedo was the author of one account, as he met Columbus and his crew in Barcelona, recording the information they gave him on his tablet (Quetel 1990)

Oviedo relayed this information to the King of Spain, informing him the bubas had arrived from the Indies, where the disease is common and therefore not as dangerous as it is in Europe as the Indians immune system had evolved with the disease. He also informed the King that the disease did not exist in Spain until Columbus and his crew returned from the Indies (Quetel 1990). Oviedo also claimed that King Ferdinand sent Spaniards to fight King Charles VIII of France in Naples. According to Oviedo, some of these Spanish men were already infected thus introducing syphilis to Italy. As Charles the VIII and the French army arrived in Italy at the same time the Italians decided to call the disease the French sickness. The French called it the Neapolitan sickness as it did not exist in France at the time (Williams et al 1927).

Oviedo was so self-assured of his assumptions and knowledge of the origins of syphilis that he was often amused that the Italians and French were unaware of its origins. “I laughed on many occasions in Italy when I heard the Italians call it the French disease, and the French call it the Neapolitan disease; and indeed, they would both have hit upon the right name had they called it the disease of the Indies.” (Naranjo 1994:93) Again, with that same confidence in his own knowledge of the subject of the disease he states that the wood of the guaiac tree is the only cure for this terrible disease of the great pox; “for so great is divine mercy that where our sins produce a punishment, God sends a remedy”. (Downing 1916:519; Williams et al 1927: 688). However, it is uncertain if there is any medicinal value that the wood has to cure syphilis. The general use of the wood was to treat the ulcers (Deichmann et al 1986). The oils within the guaiac wood have antiseptic and wound healing properties (Geske 2007). This

may only have healed the lesions but left the spirochete within the body keeping the stages of syphilis going.

There was some confusion concerning Oviedo's testimony, naming Columbus as the person who introduced syphilis to the Old World. When Ellis Hudson (1962) read this testimony, he noted Oviedo did not make this statement until thirty- two or even forty-two years (1535) after the return of Columbus (Hudson 1962). He also wrote that it was not until Columbus returned after his second voyage in 1496 that syphilis was introduced to Europe. However, by this time syphilis was already an epidemic which had spread into other lands. It is not impossible to assume that Oviedo made a mistake while writing or that a mistake was made in the translation. Regardless of how the error was made it does not detract from his credibility as a source.

When Columbus arrived in Barcelona, Oviedo, who was then a page at the Royal Court, did not mention anything about an epidemic sweeping the city. Even when he did mention it thirty years later, he called the disease bubas which came from the New World and did not appear in Spain until 1496, after Columbus had returned for the second time. Strangely enough Morison, a true believer in Oviedo, commented that he doctored the records in order to blame Columbus for the disease. According to Ruiz de Isla bubas was an old Spanish word used as a curse ten years prior to his arrival, and Montejo added that bubas appeared in Spanish literature before 1492. Even fifty years prior to the arrival of the disease Death appeared in the Dance of Death where he decreed his victim to die of bubas (Hudson 1946).

Ruiz de Isla finished his book classification in 1530, although his book has a license date of 1537 on it and the publishers date was 1539. Taking this information into account he must have written his book after Oviedo, and presumably he was influenced by Oviedo`s work. The title of his manuscript differs somewhat from the book's title as his manuscript title refers to bubas and his book title refers to the Serpentine disease. Both names are quite old and interesting as bubas, buvas, boas were in use many years earlier. over five hundred years before Ruiz de Isla put pen to paper, Albucasis in Cordova claimed there were four types of leprosy, one of them being the `Serpentine Disease` (Hudson 1946).

Another historian who the Columbianists also support is Le Casas who also did not see Oviedo as a credible source. He says regarding Oviedo's book the Histories of the Indies "Contains as many lies as pages" (Downing 1916:519). It does not help the Columbian theory if these early writers who they support are writing after the events of the return of Columbus. It was Le Casas who in 1502 left Spain for the Indies where he compiled his writings into a book on the Histories of the Indies which was published in 1561.

According to Le Casas, las bubas or syphilis originated in the Indies and was transported to Spain with Columbus when he returned from his voyage. He firmly believed that either, the Indians who sailed to Spain with Columbus or Spanish sailors who became infected in the Indies, were responsible for introducing the disease to Spain from 1494 to 1496 (Downing 1916). This was the time when King Charles the VIII of France and his army crossed into Italy eventually becoming infected by this contagion. This led the Italians to believe it was the French who introduced the disease to Italy, so they promptly called it the French sickness. Le Casas stated that he asked the Indians whether the disease was old and they replied, 'it

was around before the first Christians arrived' (Holcomb 1937; Harrison 1959:1; Cosby 1969:222).

Le Casas was not wrong, treponematosis existed in the Americas before Columbus' voyage. A great number of cases showing the skeletal lesions of treponematosis have been reported in pre-Columbian human remains from various sites in North America (Baker et al. 1988; Powell and Cook 2005). Nevertheless, with respect to the geographical region of the Caribbean Antilles, the presence of treponemal lesions in pre-Columbian skeletal material has not been clear or confirmed prior to Columbus' arrival.

The discovery of 138 sets of human skeletal remains, dated A.D. 600 to 1200 was made at the site of Paso del Indio, on the island of Puerto Rico. This find conveys new evidence in favour of the pre-Columbian presence of treponemal disease. This evidence comes in the form of treponematosis discovered in the skeletal remains of a woman 20 to 25 years old. There was also another woman which had shown some evidence of treponematosis in her bones (Powell and Cook 2005).

However, there have been several weaker diagnoses of skeletal remains based on limited pathology visible on the bones. Luna Calderon (1993) reported the possible presence of treponematosis. These remains were dated to pre-Columbian times and located in not one but three archaeological sites Atajadizo, La Cucama, and Narranjo Arriba (Dominican Republic of Haiti). In these sites Luna discovered inflammation on the long bones, in particular the tibia, which showed hypertrophy of the anterior crest (Powell and Cook 2005). Regrettably they failed to provide a detailed sample size, or the number of cases affected by this condition. However, when examining skeletal remains in the hopes of diagnosing any kind

of paleopathology, it is important to record any pathological signs being shown. In this case with treponematosis being a systemic disease that, like several systemic diseases can generate inflammatory responses in the long bones, any number of diseases could have caused this.

Another case was reported by Gejvall and Henschen (1971) who found five crania from Puerto Rico, with signs of extensive osteitis, a distinctive characteristic of syphilis, which unfortunately was an unconfirmed diagnosis. These crania are dated between A.D. 1400 and 1600, but these dates are barely within the pre-Columbian time frame of 1400 to 1492 (Powell and Cook 2005).

Finding evidence from the region of the Caribbean Antilles for the early presence of treponematosis in pre-Hispanic America is very important, because this is the region where the first encounters between Europeans and native Americans took place. The period between the end of the fifteenth and beginning of the sixteenth century heralded the appearance of the Spanish chronicles which described the medical treatment of potential treponemal lesions in both the native populations and their mythology. The mythological side was recorded by Ramon Pane (1498), a Spanish chronicler who wrote about a Tanio myth describing a mythological figure suffering from mal francés (The French Sickness) (Pane 1999)

Spanish chroniclers like Oviedo stated that ‘where our sins produce a punishment, God sends a remedy’. The remedy being the guaiac wood. If the Indians used guaiac wood as medicine for this disease like the chroniclers suggest then maybe that is why the Indian men and women who have the disease aren’t troubled by it, however the Spaniards are painfully afflicted (Downing 1916). This could be due to host and bacterial co-evolution where the host has been around the bacteria long enough to become more resistant to their effects.

Most Indians in the New World had O blood type, owing to little migration across Beringia during the Ice Age. Such genetic isolation from the outside world combined with lack of hygiene, may have contributed to a faster co-evolution of the disease than seen in the Old World. This would mean that there may be variation in the genetic code of the spirochetes leading to a greater virulent effect on the Old World susceptible populations than the New World where people would have been adapted to the infection (Grieco 1992). This is shown when Columbus' men returned and a lethal epidemic swept Europe within 5 years.

If the American Indian men and women were not troubled by the disease as Oviedo stated, then perhaps it was the Indians who were the only ones infected and it was not noticed by anyone on the voyage to Spain. However, Ruiz de Isla specifically stated that Columbus' sailors caught this communicable disease from the native Indians whom he treated (Cosby 1969). He also said that the disease appeared in Spain in the Year of our Lord 1493, in the city of Barcelona and as this city was infected, it followed that it would not be long before all of Europe became infected (De Ricon-Ferraz 1999). Therefore, this horrific disease should have been noticed in Spain, and it should have been historically noted the disease began in Barcelona and not Naples. However, Ruiz mentions that he did not know what the disease was at the time but later realized that he had been witness to the arrival of syphilis (Crosby 1969; Frith 2012). If Ruiz de Isla was right and he had witnessed the arrival of syphilis and treated it, then the disease could not have been as virulent as it was at the siege of Naples. Scholars such as P.S. Grigorieva supported Ruiz de Isla`s beliefs and in 1932 she added to them by stating, syphilis appeared in Barcelona in 1493 at the same time Columbus and his crew arrived in the city, arousing the doctor's interest. Doctor Nicolaus Scylatius described the symptoms of syphilis before Ruiz de Isla treated them (Stepanenko 2003). This

information would have aided the Columbian faction if someone had written about it around 1493. This information is not cited by any other source, possibly because it was not translated into English, or perhaps Nicolaus Scylatius did not exist.

One of the reasons Ruiz de Isla carried so much weight was because he had not only seen syphilis in Castile and Aragon, but by the time he was commissioned to treat patients with syphilis in the Portuguese hospital he was considered an expert in the field (De Ricon-Ferraz 1999; Naranjo 1994). At that time Lisbon attracted ships from all over the world where disembarking passengers were able to spread various diseases such as syphilis to the unsuspecting public, resulting in Lisbon becoming the centre of diseases. Syphilis was an easy disease to spread thanks to 'cosmopolitanism and widespread immorality' (De Ricon-Ferraz 1999), it is little wonder Ruiz de Isla was able to gain more knowledge of syphilis in Lisbon as he observed and treated more patients there than anywhere else.

Ruiz de Isla with the completion of his book had claimed to have treated twenty thousand patients with bubas within 20-40 years. Considering that there would have been other physicians in Spain and Portugal treating bubas, it shows that the disease was in epidemic proportions. He found that the disease was more common in port cities like Barcelona, Lisbon and Seville where the treponematosis bacteria and venereal disease were thriving. West Africa appeared to be their main supplier of yaws for more than one hundred years, while North Africa sent their infected slaves over to Seville and Barcelona that saw many treponemes from the Mediterranean arriving and leaving from their ports (Hudson 1946).

Francisco Lopez de Villalobos, like Ruiz de Isla and Fracastoro showed inconsistency hinting at a new disease but also viewed it with older diseases. Villalobos was a Spanish doctor who wrote a book called A Summary of Medicine: A Treatise concerning the Pestilential Buvas. His book was published in 1498, however, he might have started writing in 1493 as he praised the reign of then sovereigns for bringing peace (Hudson 1962).

Villalobos referred to the disease as *las buvas* which was an old word often used in Spain (Dennie 1962). The word means sore or pustule, and the plural a skin eruption. *Las buvas*, as Villalobos described it, was a contagious skin disease that began on the genitals. Villalobos said the buva on the genitals was a small, painless, hard sore. Then "after many days came a dark eruption, heat producing, with crowded pustules, blisters, burning crusts in palms and soles, yellow and reddish pustules, some ulcerating" (Hudson 1962:583). He noted that pain was also present in joints such as shoulders and knees and in bones such as the shins. He recognized firm swellings which may have been due to adenopathy as well as periostitis. He described larger buvas, not itching and not so painful, "deforming the face and hard to cure," which vaguely suggest late lesions (Hudson 1962:583).

Villalobos did not seem to regard his patients as seriously ill; he did not even mention mortality, nor discuss bad prognosis. From beginning to end *las buvas* was a contagious but short- lived eruption that started on the male genitalia and spread to the skin and joints. It was neither virulent nor lethal (Hudson 1962).

Nowhere in this picture is there any suggestion of the terrifying and swift morbidity and mortality attributed to syphilis by later writers. It is possible that Villalobos was writing and observing patients at a time when the syphilis epidemic had not spread from Naples to Spain,

and he had not witnessed the severe reactions of patients. Another possibility could be that syphilis in addition to other diseases had further lowered their immune systems reactions, allowing multiple types of diseases to invade the body, causing such severe reactions which added to the severity of signs, symptoms and the growth of mortality throughout Europe.

Villalobos's statement that *las buvas* was a new disease has carried too much weight when it is looked at in relation to how new the printing press was in Europe at the time and the fact that 'medical literature' was in its infancy (Hudson 1962). At the time referring to books meant the classics, and anything not written about by the 'authorities' of the time was considered new, even though the disease may have been present before. Medical knowledge of such epidemic diseases was often forgotten over the time that passed between each epidemic due to the lack of recorded medical information available, therefore each time the disease re-emerged it was considered to be 'new'.

In the late fifteenth and early sixteenth centuries medical knowledge and recording had improved and more 'medical books' were available to refer to, this led to an increase in the diagnosis of recurring diseases such as smallpox, typhus, syphilis and many others (Hudson 1962).

It may be difficult to prove or disprove the arrival of syphilis in Spain through Columbus' voyage, due to a lack of written accounts describing the presence of syphilis during the voyage, or while they were waiting to be seen by the Spanish royals. It is more than likely some of the Indians may have had the disease, but it was asymptomatic at the time, or they had co-evolved well enough with the disease to only show a slightly virulent form. If this is

true it would explain the reason scholars linked syphilis with an older disease and Columbus' voyage? Villalobos also does not describe the disease as a serious one, as there was no mortality described. It was probably the Old-World form of syphilis (Erdal 2006) he was treating, as he spoke of the disease as if *buvas* had been in Spain for a while. Ruiz stated he had treated some of Columbus`s crew for this new disease (Cosby 1969). He also claimed that syphilis was taking hold of Barcelona (De Ricon-Ferraz 1999). However, it has been implied that Ruiz de Isla did not know what the disease was at the time, it would have been after the siege of Naples when Ruiz de Isla realised that he had been witness to the arrival of syphilis (Crosby 1969; Frith 2012). If this is true then despite his claims of seeing and treating Columbus' men, and being witness to its spread throughout Barcelona, he was still only seeing a milder version of syphilis than that witnessed after the siege of Naples. If syphilis was a new disease, what triggered its virulent onset, and how was it related to the siege of Naples? What brought syphilis to everybody's attention?

Possible syphilitic transmission vectors for 1494-1495 Italian Wars

(separate article written for publication to the Journal of Interdisciplinary History)

Study of the literary evidence showed how the army of Charles VIII was responsible for the spread of syphilis, due to already infected mercenaries from Roussillon in Spain (Downing 1916). It stated that: Charles VIII after having failed to make a claim for the kingdom of Naples, over Prince Alphonso II, sent a force of thirty thousand mercenaries thus instigating the siege of Naples. These mercenaries included French, German, Flemish, English, Italian and Swiss. This evidence also mentioned a sizeable Spanish force of mercenaries from Roussillon, although the presence of Spaniards in the army of Charles VIII had been questioned by Karl Sudorf (Luger 1993). The mercenaries were recruited in Lyons before departing in August 1494 along with eight hundred prostitutes in toe, arriving in Rome December 31, 1494 before advancing on Naples on February 22, 1495.

It has been argued that after an easy victory, thousands of men were released from discipline into a large city that Charles VIII occupied for less than three months. Due to the loose morality of the time and the extreme contagious nature of the disease, syphilis spread like wildfire through the army of Charles VIII which had already been infected from his own Spanish mercenaries. Meanwhile fresh Spanish troops had landed in Sicily to help Alphonso Fernando, King of Naples. News of this arrival caused Charles VIII along with his weakened forces to retreat from a perilous

situation. He managed to fight his way back through Italy to Lyons, and in November 1495 he disbanded his polyglot army from whence they carried the disease all over Europe.

However, this theory so readily adopted by many academics, has failed to meet a mathematical understanding concerning the transmission timeline. By applying what we now know about the length of the various stages of syphilis, we can calculate how far the army of Charles VIII would have travelled before he should have noticed the disease (Garnett et al. 1997). The disease timeline is as follows: primary stage of a localized chancre takes one to two weeks, the secondary stage of fever and skin eruptions over the whole body takes three to five weeks, while the tertiary stage may last the lifetime, initially being contagious and manifesting with gummatous ulcerations, destruction of soft tissues and inflammation of periosteum and bones.

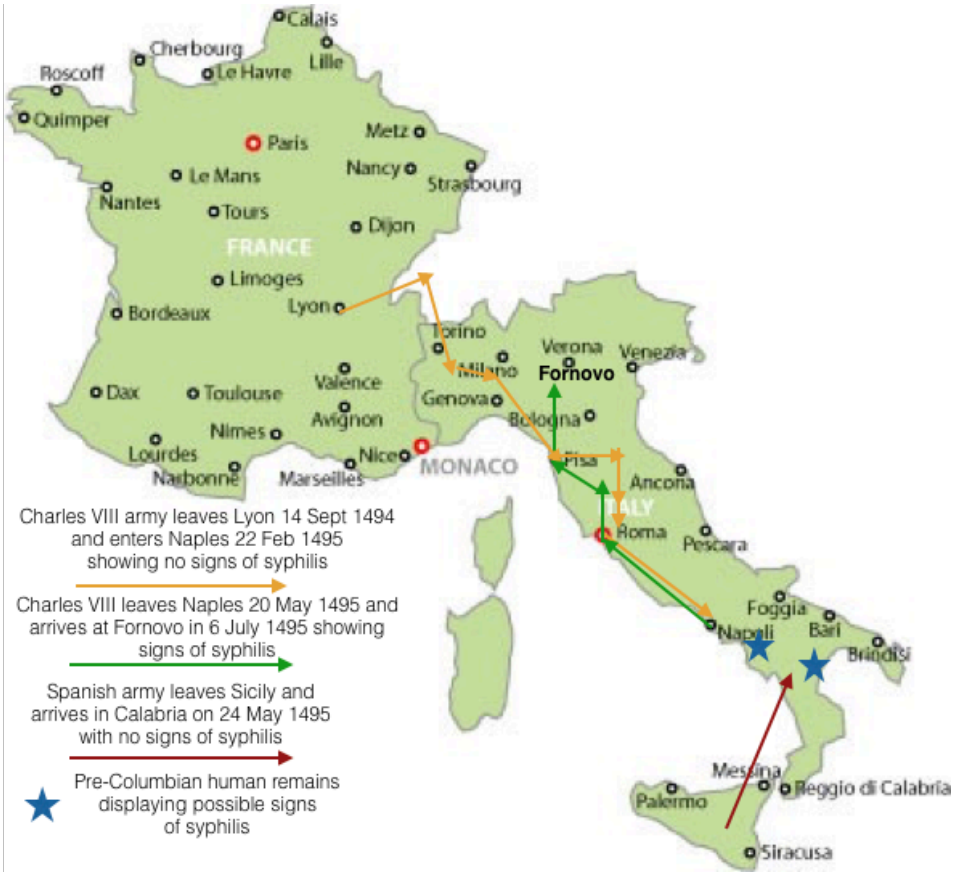

Figure 1: Map showing routes of armies with reference to possible sites of ancient cases of syphilis.

Let's assume that Spanish mercenaries brought the disease into the army assembling in Lyons. Then this transmission timeline would have commenced when the mercenaries departed Roussillon in Spain, for Lyons four hundred and fifty-one kilometres away. Assuming they travelled anywhere between ten and twenty kilometres a day, this would then equate to around twenty-five to fifty days depending on the pressure the mercenaries were under to get to Lyons in time. This means if the army of Charles VIII became infected through its hundreds of prostitutes who had contracted the disease from the Spanish mercenary force, physicians would

have noticed the soldiers displaying secondary stage syphilis before they even left Lyons.

The next leg of their journey began when they left Lyons August 1494 and crossed over the Alps arriving at Asti in Italy. Charles VIII decided to leave Louis D`Orleans in Asti with a small garrison to defend northern Italy, while he continued on with the rest of his army entering Rome on December 31, 1494 (Nicolle 1996). This journey took one hundred and five days. According to the syphilis's spread timeline the Spanish mercenaries and other soldiers who were still in the infectious stage could have passed syphilis onto the army prostitutes and those of Rome. If syphilis was rife, then within five weeks of leaving Lyons every prostitute would have been infected with the disease, which would have become endemic within the army.

This calculation is based on an assumption that each prostitute could have serviced 25 men per week. Then at least one would become infected. If in the second week this prostitute was visited by 25 men who also had contact with 10 other prostitutes, who serviced another 25 men each, and this situation was repeated for five weeks, practically all prostitutes and all men would have been infected.

On the 28 January the army of Charles VIII which was still healthy, left Rome arriving in Naples where they staged a short siege before entering Naples on February 22, 1495. The army remained in Naples for three

months and then Charles VIII army left for France on May 20, 1495. At the same time the Spanish force from Barcelona was on its way to re-claim Naples (Mallet and Shaw 2012). During this timeframe there was no mention of any severe disease affecting either the inhabitants of Naples or Charles VIII's army. There was also no evidence that the Spanish force coming from Barcelona was afflicted with any infectious disease.

Two months later at the Battle of Fornovo on 6 July 1495 a Venetian physician Mercellus Cumanus noted soldiers had small pustules on their genitals, along with some degree of itching (Quetel 1990). They were also seen to have pustules on their faces and all over their bodies. These pustules had the appearance of grains of millet and usually appeared on the surface of the foreskin. A few days later soldiers suffered from violent pains in the arms, legs, and feet, along with large pustules, or ulcers. These signs lasted for twelve months or more if un-treated. Another Venetian physician at the battle of Fornovo was Benedetto, who mentioned seeing sufferers who had lost their eyes, hands, nose and feet, facing the possibility of death. (Quetel 1990). Both descriptions of the disease suggested the army was displaying all stages of syphilis at a greater severity than what is known today. The severity was due to frequent hyper infection brought on by re-infection (Russell et al 2013).

Table 1: Possible spread of infection of Charles VIII army if Spanish mercenaries brought the disease to Lyons or caught it while in Naples.

Week	Prostitutes infected	Soldiers infected	Possible location of infection if the source was Lyons	Weeks	Possible location of infection if the source was Naples	Weeks
1	1	25	Lyons to across Alps Northern Italy	Early August 1494 to Late August (3-4 weeks	Naples to Rome	20th of May to 27th of May 13th June (1 month)
2	10	250	Northern Italy Alps to Asti	Late August to Late October (3-4 weeks)	Siena to Rome	27th of May to the 13th of June (2 weeks)
3	100	2500	Asti to Florence	Late October to 17th of November (3 weeks)	Siena to Pisa	18th of June to the 22nd of June (4 days)

4	1000	25000	Florence to Rome	25th of November to the 29th of December (1 month)	Pisa to Fornovo	22nd of June to the 6th of July (2 weeks)
5	Endemic	Endemic	Rome to Naples	28th of January to the 22nd of February 1495 (3 weeks)		

Judging by the abovementioned information, if the disease originated in Spain and travelled to Naples via the army of Charles VIII then the army physicians should have noticed the signs within five to seven weeks of departing from Lyons, well before arrival in Naples.

If the Spanish did not infect the French with syphilis, then how did the French become infected? Naples had always been a place connected with loose morality and possibly syphilis (Naranjo 1994). In the time of the Imperial Rome, Marcus Tullius Cicero described the profligate customs of the provinces of Campania and Capua, near Naples, as a *domicilium*

impudicitiae, the home of shamelessness (Naranjo 1994). Morality in Rome and Naples had not changed up to the time of Charles VIII, as prostitution was not only tolerated but encouraged.

The notion of syphilis in Naples was nothing new, as Maciej and Renata Henneberg found evidence of congenital syphilis in the cellars of the villa in Oplontis dating to the time of Vesuvius's eruption in 79 AD (Henneberg and Henneberg 2006). To the south of Naples, in the Ancient Greek colony of Metaponto evidence of endemic syphilis was found (Henneberg and Henneberg 1994, Ioannou et al. 2018). The physical evidence of syphilis is also backed up with historical accounts; Pliny the Elder, Celsus and Caticuls all wrote about a disease which affected the genitals while spreading over the body (Rackham 1962; Grieve 1818; Lee 1990). Celsus took it one step further by adding that mercury was used to cure it. Leoniceno, an Italian physician writing in 1497, claimed syphilis was present in Naples prior to the siege (Naranjo 1994).

The disease was already present in Naples in a milder form when the army of Charles VIII arrived. As the disease had been able to co-evolve with the inhabitants of southern Italy, being endemic in its communities, the strain of the treponema may have varied from those in other countries around Europe. This suggest that the army of Charles VIII had little resistance to this form of the disease and therefore their constitution would have reacted as if it was a new disease.

By the end of the 16th century a form of syphilis had become severe, spreading throughout the rest of Europe and continuing to spread throughout other countries over the ensuing centuries. One of those countries was South Africa which in 1821 suffered such a severe outbreak, that it had been compared with the epidemic which spread through Europe during the 15th and 16th centuries (Sax 1952). Although endemic treponemal disease may have been present in South Africa at least 1000 years ago (Steyn and Henneberg 1995). Guam was another country affected by syphilis, with one person in thirty five contracting the disease and the treponematoses appeared to be as severe, if not worse, than the epidemic in Europe during the time of Charles VIII (Willcox 1960).

It is difficult to ascertain the antiquity of syphilis in Scotland, but certainly the 16th century epidemic must have reached there. Irrespective of that, according to Hibbert (1926) Scotland suffered in the late 17th and early 18th century, an epidemic similar to the one which occurred elsewhere during the 15th century (Willcox 1960). Syphilis was present in the Bosnian region at least since the 16th century Turkish invasion and later French invasion in 1809, and yet its epidemic flared up twice. The first time, syphilis hit the Moslem population in 1832 during Mehmed Pasha`s military campaign, and the second time in 1941 during the German occupation where large numbers of refugees, combined with a deterioration in living conditions along with poor sanitation only served to exacerbate the problem (Grin 1953).

The occurrence of syphilis in Bosnia was similar to that which occurred in Naples. The constant flare ups of the disease were brought on by large groups of migrants and soldiers who had not adapted to this particular co-evolved local form of syphilis. Thus, a superinfection was able to give the appearance of a new disease, as the native inhabitants of the area were not accustomed to such signs of severity on the human body (Grin 1953)

Therefore, the local epidemiology aspect of the disease, which infected the army of Charles VIII in Naples, had a stronger foundation in its local origin than the theory that the disease came from Spain. If soldiers were infected with syphilis from prostitutes in Naples this process would take some time to infect the whole army. The army stayed in Naples for three months. In these three months syphilis could have become not only common, but also physicians would have noticed secondary signs in some soldiers. Fleeing with a weakened army from the incoming Spanish reinforcements, they took two months to reach Fornovo where all stages of syphilis were noted.

However, the above-mentioned theory does not explain how countries such as England escaped the epidemic, or whether they just fail to record the event? If syphilis was rife in the army of Charles VIII how did the fighting armies and their prostitutes from other countries not contract this disease?

There are two perfectly logical explanations for this phenomenon, the first being: - during the Arabian, Grecian and Roman eras of medicine people like Aetious, Aegiente, Celsus, Galen, Hippocrates and Hali Abbis continually

described pertinacious diseases as lepra from the Middle Ages to the 18th century (Campbell 1934). According to Bernard de Gordon, a medical writer in medieval times (around 1305) leprosy, a congenital disease, was sexually transmitted (Gordon 1491). This statement appeared to describe syphilis instead of leprosy. Another medieval medical writer Proksch who came from Vienna, stated that lepra was "the worst scourge of the human race" right up to 1495 (Campbell 1934:405). He also stated *malum mortar, jus parsecs* and *fornica*, although severe at the time, as soon as syphilis hit the scene, paled in comparison. The abovementioned afflictions not only completely disappeared but leprosy also became scarce (Campbell 1934). This was a strange observation, as at the time Proksch was referring to, syphilis was gaining strength. There are other theories as to why there was a decline in leprosy during this time period and that was the black death. Around about the time leprosy peaked, the black death took over not only killing lepers but decreasing a substantial portion of Europeans as well. (Richards 2000). The plague's effects on the population would have limited leprosy's ability to transmit to other individuals because carriers of Mycobacterium leprae were killed early in the progress of leprosy in their bodies. Another theory is the idea that cross immunity between tuberculosis and leprosy was responsible for the decline (Manchester and Roberts 1989; Carmichael 1993; Roberts and Manchester 1995). However, Wilbur et al. (2002) dismissed this theory as they believed cross-immunity only occurred in some areas, they found evidence. Adding weight to their theory was a finding that 20th century population of Texas experienced an increase of both tuberculosis and leprosy (Donoghue et al. 2005).

The second explanation referred to the erratic nature of syphilis as it can become asymptomatic during certain stages of the disease. An example of this would be a patient in tertiary stage of syphilis who could easily be diagnosed as having leprosy in its early stage. Though medieval diagnosticians were aware of differences between leprosy and syphilis they may have made mistakes, even if only occasionally If a patient only portrayed the primary stage of syphilis it could appear as if they were suffering from any sexually transmitted disease. A patient initially portraying syphilis in its secondary stage could be thought to have smallpox or related diseases. Cecil Hackett came up with another theory; he believed that in pre-Columbian times syphilis was a mild disease so it never really came under the physician's radar (Hackett 1963).

Conclusion:

There were thirty thousand soldiers along with a contingent of eight hundred prostitutes in the army of Charles VIII. During their expedition these prostitutes had spread syphilis acquired locally by soldiers. Many of the mercenaries who had promiscuous sexual contacts during their travels may have became re-infected. The re-infection in the tertiary stage may have caused a severe reaction with the antibodies producing a hyper infection (Russell et al 2013). This caused the signs of the disease in its many stages to show a greater severity than normal. After the spread of the

disease throughout Europe, Germany in 1495 pronounced the Edict of Worms where tighter strictures were put in place to control the disease (Fuchs 1843). In the same year France also moved to tighten control of the disease by banishing anyone who showed signs of it (Holcomb 1937). However, throughout Charles VIII's campaign against Naples no such decree or edict was ever made to control syphilis in either Spain or Portugal, which surely, would have happened if syphilis was a new disease brought in by Columbus.

The disease was seen for the first time due to its extreme virulence at Naples. The cause seems to be a superinfection brought on by constant re-infections during tertiary stage syphilis causing a hyper-infection. Superinfection of an already infected and allergic host can be the cause of an increase of severity of tertiary manifestations (Murray et al 1956). It would explain not only its sudden appearance and why it became such a virulent disease so quickly and lasted so long. Evidence for this comes from the Bakwena reserve where there is endemic treponemal disease. People of the Kalahari Desert were examined and 1:68 or 15 per 1,000 people were found to have tertiary lesions. In comparison 1:278 or 2.6 per 1,000 people in the non-Kalahari region had them. One theory for this phenomenon could be the people of the Kalahari did not have the same contact with western medicine as the people living in the non-Kalahari region. Early endemic treponemal disease was rare with the Bakwena people whereas among the Bakgalagadi people it was quite common. Grin also found evidence of this instance in Bosnia He concludes that in regions where new cases of endemic treponemal disease are prevalent

it coincided with a superinfection of latent cases in the same home as well as an increased likelihood that tertiary lesions would occur simultaneously (Grin 1953).

Literature evidence for the existence of pre-Columbian syphilis

(Written as a separate article)

One of the strongest arguments for a Columbian origin during 1494-1530 is that the disease syphilis appeared so quickly, taking everyone by surprise. The disease was new, severe, fast spreading and was able to imitate various other diseases. For this reason, the disease was given multiple names, some of them particular to the country in which you were living.

As the origin of the disease was unknown, the majority of countries that came into contact with it chose to politically defend themselves. This defense involved each country blaming its neighbours, invading explorers or merchants for the arrival of syphilis into their lands. The invasion of Italy and the attack of Naples led the French to call it the Neapolitan disease and the Italians to call it the French disease (Crosby 1969).

The disease was also given names that described the nature of the disease, Ruiz del Isla referred to it as the Serpentine disease for the disease was of an evil nature. It was also given the name the pox, like smallpox it was a reference to the pox marks/ulcers on the skin. Lastly, names were created for this disease as a link back to pre-Columbian times when similar symptoms of diseases were described, these names being pudendra and mentagra. This strengthens the argument that the disease was unlike anything anyone had seen before as there was no unified name or description. However, it may be less of a case of a new disease but rather a

case of scattered knowledge. This is because physicians treated symptoms not diseases at the time, therefore viewing the one disease as many different ones, led to not connecting the links for a proper diagnosis, which in turn led to further misunderstanding with later physicians.

Considering the passage “in the entire literature of the Old World, no description of the syphilitic syndrome anterior to 1495 is to be met with.” (Bloch 1908:8). While the afore mentioned does hold some truth in that there is no clear writing on syphilis prior to 1495, it also must be considered that there are no clear clinical writings until into the twentieth century on the matter. The knowledge of syphilis and its presentations had been growing both before and after this date, reaching its pinnacle during the twentieth century (Whitwell 1940).

Attempts have been made in the past to show evidence supporting that writers before the fifteenth century had seen and been trying to describe syphilis, although the attempts may not have been as clear and concise as would be preferred. The evidence does suggest that they may have linked earlier symptoms of syphilis to the later ones, albeit that it was frequently confused with leprosy there were still some differentiating points made. These were medical mistakes made that were common to the era, due to a lack of understanding in how the diseases worked (Whitwell 1940).

Wendelin Hock of Brackenau, who was a physician, published a medical treatise on the French Pox (1514) concerning the different names of this disease. Each country referred to the French Pox by a different name, from the time it appeared in 1494 until 1514 (Savinetskaya and Jarits 2016).

Hock was curious as to why the common people called the disease now known as syphilis, differently in various parts of the world. Hock came to the conclusion that names were given not only to diseases, but according to their symptoms as well. There were times when a disease was named based on the cause, while at other times the name was based on the effect. Therefore, the disease of the Romans called mentagra was well named, as a substitution for the disease now known as the French Pox (Savinetskaya and Jarits 2016). Mentagra was used as an euphemism for the disease which first affects the penis (Hudson 1968). Martial often uses this word in poetry in which it is described in situations with the disease being passed on by sex, however it is not certain what disease it was.

For many centuries Christopher Columbus had been blamed for bringing syphilis from the New World of Haiti when he returned to Europe in 1493. This idea was only perpetuated by later secondary writers like Ray de Isla, Oviedo, Le Casas and Fracastoro. However, no written or physical evidence had been recovered to validate these claims. In fact, the first descriptions of syphilis and names thereof came only after the siege of Naples in 1495. The disease's sudden appearance led to what is now known as the blame game, as every country participating in the siege of Naples blamed each other for

the disease (Whitwell 1940; Arrizabalaga et al. 1997). At the same time every country denied ever knowing of its prior existence.

This is one reason why a Haitian origin has been strongly supported. The fact that the disease sprang up from nowhere in Europe with such virulence, led doctors to claim it was a new disease. From this sprang a need to give the new disease a name. At first it was a matter of blaming the disease on each other, then the scholars started researching old diseases to compare it with, in order to find its origins and thus a treatment for it. After which they conveniently discovered it originated from Haiti. An alternative route treponemal disease may have taken was through the slave trade between Portugal and West Africa. Since 1442 Portuguese ships captured slaves along the west coast of Africa. Slave trade by Portugal and Spain continued by sea and overland for many centuries. This slave trade commenced 50 years before Columbus set sail for the first time (Hudson 1964).

When questioning the origins of the disease, problems arose because scholars were either inventing names which corresponded with the symptoms or links to older diseases. Therefore, it is important to find correlations between present time and ancient diseases.

The first names given to this disease were The Pox, Morbus Gallicus and Venereal lues. However, in 1530 Fracastoro composed a poem entitled `The Sinister Shepherd`, which tells of a shepherd boy named Syphilis who

insulted the powerful Greek God Apollo, who in turn bestowed a horrific disease on the boy (Fracastoro 1934). The shepherd boy's name became the common term used to describe the disease.

This caused numerous problems when associating the un-clinical terminology of syphilis to possible pre-Columbian names. Ancient and medieval physicians/writers would have recorded the external signs and complaints of patients. Therefore, there may have been many different names for the same disease.

There was a confusion of names in the Bible. The bible does not mention names of diseases but strangely enough, due to a translation error, a name for a disease was created. Mariotti et al. (2005) argued that the classical Hebrew word tsara`ath which appears in Leviticus could relate to a variety of human skin conditions, ritual un-cleanliness or moral impurity (Møller-Christensen 1967; Grmek 1983; Mariotti et al 2005). When the Bible was translated into Greek the word tsara`ath became λεπρα, which was later translated into Latin as Lepra (Andersen 1969; Roberts and Manchester 1995; Crane-Kramer 2000). Both Crane-Kramer (2000) and Zias (1991) agree that the term tsara'ath is an ambiguous terminology based on broad spectrum of meaning the Hebrews placed on the word. This broad terminology would assign religious meaning to the word lepra placing sexual connotations where none belonged from a medical standpoint.

Although tsara'ath may not have been a name for any specific disease, it does describe people with unclean skin like scabies, leprosy, syphilis, psoriasis, eczema and smallpox. According to Deuteronomy 28:27-8, Moses believed that people punished for disobedience would develop scabs, constant itches, madness and blindness. In Job (16, 19, 30), there was a description of him being plagued by a genital lesion, his body being covered in boils, his sight was failing and his breath corrupt (Baker et al 1988). In Psalms:1-1 1 a description is given of signs affecting King David, those being foul smelling lesions, shooting pains, failing vision and genital lesions which he attributed to his sexual relationship with Bathsheba who, as far as David was concerned, lacked cleanliness (2 Samual: 2-5). If these descriptions from the bible were accurate it would still be impossible to establish which venereal disease they were referring too.

Roman and Greek

The Romans were a society in which medical understanding was improving leading to new names being attributed to diseases. The Romans used the term Elephantiasis, Lepra and Mentagra to describe diseases that affected the skin.

Pliny the Elder mentioned a new disease which appeared during the reign of Tiberius. It was attributed to a Roman knight who had contracted the disease whilst in Asia Minor (Rackham 1947). Although contagious, it was

not in epidemic proportions in Italy or the rest of Europe. This was a painless disease and the sufferer developed a scaly face, neck, chest and hands. The Romans referred to it as Mentagra as the disease started on the chin and the Greeks called it Lichens (Rackham 1947).

Women, slaves, as well as the lower and middle classes were not plagued by this disease. Nobles were thought to contract the disease through a momentary kiss. Patients were treated by Egyptian physicians, who cauterised their wounds all the way to the bone (Rackham 1947). There was even a poem warning against kissing written by Martial, a Roman poet, who believed you should be friends with people you do not want to kiss hence avoiding ulcers, weeping sores, filthy scabs and lichen (impetigo) (Martial 1871). He could have written this poem to either warn against sexual contact resulting in treponemal disease or in fact any kind of venereal disease like herpes.

The proposition that mentagra could be venereal syphilis is pure speculation, especially since Pliny the Elder clearly stated the disease began on the chin. There were medical historians like Wilson (1846) who concurred with Pliny. According to Wilson mentagra was Sycosis a chronic inflammation of the cutaneous tissues, similar to acne, the site being the only difference. Mentagra was normally confined to the hairy parts of the body, i.e. the chin, the upper lip, eyebrows or nape of the neck. Wilson believed that the disease started in the sebaceous glands, and spread to the hair follicles, including related tissues, giving rise to conical

elevations (Erasmus 1857). These elevations then form puss at their apex and are each traversed by the shaft of the hair. However, Sycosis is a disease that only affects the face of an individual whereas mentagra also attacks the neck, chest and hands.

Grunpeck (1496), Hock (1514) as well as Hudson (1961) agreed that the word mentagra came from the Latin mentum, meaning the chin and the Greek word agra meaning something caught. Over time, mentum related to chin plus beard or just the beard. Later the pubes became known as the "little chin" or mentula, then the genitalia were included and finally reference was to the penis alone (Hudson 1948; 1961:554). Catullus (86-54 B.C.) used the word mentula for something adulterous (Lee 1990). Grunpeck (1496) and Hock (1514) came to the conclusion that the Latin words mentulagra and mentagra described a disease of the penis.

Marcellus Empiricus (410 A.D), a Roman physician stated in his book De Medicamentis that he examined two hundred cases of mentagra/lichen which developed all over the body (filthy scales) in Aquitania (Gascony) (Empiricus 1889). With the decline of the Roman Empire the name mentagra faded out.

Pliny the Elder was the first to describe a new disease that had suddenly appeared and was unknown to their forefathers (Rackham 1947). Also, Ruiz de Isla (1510) and Fracastoro (1530) were writing after the emergence of a new disease (later known as syphilis) and clearly state that this disease was

never known to their forefathers. There is a connection to these statements that relates to syphilis, as a disease syphilis can blend in with other diseases and remain hidden until it becomes epidemic. Therefore, this disease would have existed during their forefathers' time and in some ways, it was hinted upon by Ruiz and Fracastoro. In their literature they insinuate that there may be a connection between this new venereal disease and mentagra.

If mentagra was syphilis, then it is obvious the disease was not a serious strand as it did not affect the eyes and patients did not complain of pain. However, during the epidemic of 1495 and later, that disease attacked the joints (Charcot joints) and muscles causing great pain. Mentagra, was a particular generic male disease of the male genitals. Moreover, syphilis should also be infecting women as well. This does not mean that women were not being infected, it just meant their disease was recognised under a different name. In the book on medicine by Celsus, book 6 in chapter 18, on the diseases of the private parts only male genitals were noted, with the only exception being a discussion of male and female disease of the anus (Greive and Futvoye 1838). When comparing mentagra with syphilis, the only difference is syphilis does not distinguish between social classes. Slaves and lower classes would be sharing living quarters and utensils, therefore would be more susceptible to endemic syphilis which would not affect the genitals first like mentagra.

Lichens:

Authors like Hudson have discussed at length whether mentagra as a name can now be used to describe syphilis. According to Pliny the Elder the Greek word lichen was the same as mentagra which was often used by later pre-Columbian writers like Hippocrates, Arteus and Aegineta. However, the terminology for lichen is vague enough to be used for various alternative diseases like scabies, leprosy, psoriasis, eczema and elephantiasis. Lichen is defined as being very itchy, dry, rough skin containing hard pustules which creep to the nearby parts. The pustules spread out to the extremities and occur in either spring or autumn (Cook and Gibson 1700).

Even though Hippocrates may have been the first to describe lichen as a disease affecting the skin, Celsus saw a disease which exhibited two different types of papula, or perhaps a disease in which the lesions alter as the disease progresses. Paulus AEgineta believed that Celsus was describing lichens (Aegineta 1847). Celsus described the first kind of lichen as small, round, rough, red pustules which were slightly corroded with a smooth centre. The second type was worse than the first, as the skin appeared to be more corroded, red and rough and at times the hairs became lost (Greive and Futvoye 1838). The Greeks called this variety aypia. If not removed, it then progressed to impetigo. Although Celsus did not mention where the hairs were lost, the description related to the signs of syphilis as ulcerations which caused hairs to erode away.

Wilson, (1847) who was a later medical writer, strengthened the mentagra/lichen link to syphilis. He believed the papular eruption which occasionally appeared with, or became syphilis, had similar characteristics to lichen. These characteristics are comprised of small, hard pimples which are raised a little and conical in shape, being copper in colour, with a purple areola (Erasmus 1847). At times thin brownish scales can be seen over the skin and the pimples can ulcerate and are rarely extensive enough to form cicatrices (scars).

Another connection between lichens and syphilis appeared in a play written by Aeschylus, where Orestes is threatened of being inflicted with lichens by the Greek god Apollo, for neglecting to avenge his father Agamemnon. The disease attacked flesh and devoured the body, covering it with white splotches and causing madness (Grmek 1989). This had similar connections to Fracastoro's poem The Sinister Shepherd where he explained the cause of the disease, by stating that Apollo inflicted the disease on Syphilus for abandoning temples and turning away from their Gods (Fracastoro 1934).

While some writers like Celsus argued that lichens might turn into impetigo, other ancient writers like Galen and Paulus believed Lichen was a disease that could become leprous (Aegineta 1846). While these statements might seem unlikely, there might be some truth in them as syphilis and leprosy have often been confused for one another. Impetigo is not a disease but an ulceration sign in ancient times just like lichens. The belief that the

ancient Greek lichens and impetigo are the same was supported by Leonciensus (1498) in his work on the Morbo Gallicum.

However, there were some who did not support the theory that lichen, and impetigo were the same sign. Fracastoro argued that impetigo was named psora and leprosy by the Greeks as it was the result of black bile, whereas lichen or papulae was what Fracastoro called volaticae which arose from salty pituita (Fracastoro 1930). The problem with this theory stems from the fact that at that time Fracastoro was considered a modern physician who was removed from the ancient/classical notion of diseases. As such Fracastoro placed his own perspectives on what the writers were describing and substituted his own name volaticae for lichen and papulae. This then meant that later writers like Johnson Green followed these teachings and they become fact (Green 1835).

In his writings it is clear that Fracastoro was not a reliable source in this area as he has either not read or misinterpreted Pliny's work. Pliny clearly stated that mentagra and lichen of the Greeks were the same disease whereas Fracastoro seemed to view them as two different diseases. Also, Fracastoro claimed mentagra and lichen appeared at the time of Pompey, however Pliny said that leprosy arrived at the time of Pompey (Fracastoro 1930). Fracastoro also made reference to a poet Martial who used the term lichen in a wider sense which he related to a foul condition. He referred to people who suffered from lichens as dirty people. This can have many connotations to it, for instance they can be dirty as in unclean skin, diseased

or perhaps how they got the disease was dirty in a sexual impurity. Considering the poet was Martial who was writing during the Roman period about how to avoid disease by avoiding kissing people you liked, it suggests that he knew that lichens was a contagion of a sexual nature (Martial 1871).

The term Impetigo from a 7th century Latin encyclopedia compiled by Isidore of Seville in Book IV chapter 3 on medicine is a description of an eruption of rough, round dry scaly skin. The colloquial term for it is sarna (Dirckx 2007).

Celsus stated that there were four types of impetigo. He described the second type (named the red) as not only being similar to the papula but more severe, with very red scaly skin which shed scales on a regular basis (Greive and Futvoye 1838). The signs spread quickly, coming and going at a faster rate than papulae.

Villalobos`s wrote about a venereal disease, mentioning that *sarna* came from sin. This idea arose from a Spanish pun connecting *sarna* with Sarah, and a tradition that Pharaoh caught *sarna* when he seduced Abraham's wife (de Villalobos 1870). The diseases eruptions are foul, the skin was very itchy and, although dry, was prone to swell as well as producing pain of the joints and veins (Hudson 1961). Villalobos believed that *Sarna Egyptica* and *mal muerto* were identical to *las buvas* which is the Spanish name for

syphilis. Lastly, Villalobos talked about *sarna* sufferers showing signs of madness. Once again these signs were similar to those of syphilis.

Leprosy

Clinically known as Hansen's disease after Dr G. H. Armauer Hansen who discovered Mycobacterium leprae it in 1873 (Steinbock 1974: 195). Leprosy is a disease which received its name from the Bible`s chapter on Leviticus where the word tsara`ath referred to ritual uncleanness (Baker 1988). Therefore, the name leprosy would become ambiguous clashing with names of other diseases throughout history. However, none would be more entwined with leprosy than syphilis.

An early indication the name leprosy was being confused with syphilis came from Alsaharavius (Albucasis) (A.D. 936-1013) who believed four types of *lepra* existed. These four types were:- Leonina, elephantia, serpenrtina and vulpina. He wrote about the last stage of lepra which he described as being both hereditary and contagious. During this stage the nose falls in, the patient loses not only hair but also their voice, as well as a nasty outbreak of ulcers over the skin (Whitwell 1940). This disease does not relate to Leprosy but an advanced case of syphilis.

Another writer who confused leprosy with syphilis was John of Gaddesden who wrote a book entitled The Rosa Anglica (1304-1317). He believed you had to look for the tell-tale signs of leprosy which included the face turning

a black colour, with *gutta rossacea* in the nose or face. A fetid breath, constant perspiring and thinning hair were other signs (Cholmeley 1912).

In his book, John of Gaddesden (1304-1317) quoted Joannes de Sancto Amando who believed the sure signs of leprosy were a blackish tinge to the body, trouble breathing, a nasal tone to the husky voice, and continual sneezing. Loss of hair, foul smelling seat and breath, swelling of the face and limbs `rotunditas` of the eyes, greasy skin and insensibility of the calf were other signs. He also stated the fingers and toes felt cold and sleepy. The anaesthesia spreads to the skin between the fingers or toes and continues up the forearm, or in the leg up to the hip and impetiginous eruptions occur (Cholmeley 1912). Nails become distorted, the eyebrows fall out, the septum nasi becomes ulcerated, the hands and feet drop off and the lips thicken.

He was correct when he mentioned the face tended to develop a dark hue with leprosy. The *gutta rosacea* could be either leprosy or syphilis as both attack the nose and falling of the eyebrows. The signs of syphilis found within the text on leprosy are foetid breath, loss of hair and gangosa which creates the husky voice sound. What was typically leprosy was sensory deprivation in the fingers and toes as well as loss of fingers and toes. (Cholmeley 1912).

Another classic example of confusion between syphilis and leprosy was from Bernard de Gordon in his book the Practica sue Lilium Medicinae

which he wrote around the 1305. He stated that if a healthy person lies with a woman who had seeds of leprosy in her womb, he would become a leper. He also said, if a leprous woman is with child then the child would be leprous (Gordon 1491). Leprosy is not a sexually transmitted disease nor is it congenital whereas syphilis is both of these. Some authors (Crane-Kramer 200; 2002; Zias 1991) state that there is no evidence of diagnostic confusion between leprosy and syphilis. The matter of sexual transmission of leprosy is a result of religious belief that leprosy was "unclean disease" while uncleanliness is related to elicited sexual contacts.

To show the confusion between syphilis and leprosy, Holcomb used first-hand accounts from Bartholomeus Anglicus, Peter Abano and Peter Angelata who believed that leprosy was contagious and spread through contact with a menstruating woman (Cole 1951). For a long time during the Medieval period physicians believed that leprosy was caused by sin and spread by the contact with women. However, both of these statements relate to cardinal sin/relationship which most citizens would have partaken in in their lifetime. This could explain the substantial increase in the number of leper houses to 19,000 since the Crusades which was noted by Mathew of Paris. The Middle East is a hot spot for treponemal disease like Bejel (endemic syphilis). The movement of armies, is a great vehicle for carrying diseases out into different countries. When soldiers return from their campaigns they also bring new diseases to their respective home countries. The crusaders would have brought back more cases of treponemal disease and leprosy from the Middle East to Europe increasing the number of

persons infected. When they returned, they would have gone to these leper houses to be away from healthy individuals. This lasted until Pope Innocent VIII at 1490 and Pope Julius II at 1505 abolished leper houses and the Order of St. Lazarus ceased to exist due to diminished numbers of lepers. However, syphilis increased at the same time (Cole 1951).

Feodorico Borgognonia (1205-1298), who was a physician and after 1266 became bishop of Cervia, wrote a book on surgery, describing the methodical inunction with mercury salve. These inunctions, although only beneficial at the onset of leprosy, were also prescribed for scabies, cancer and *malum mortum* (Cole 1951).

Mercury has no effect on Hansen's disease which is the modern name for leprosy. It was however significantly recognised in the past as a specific treatment for what ancients called leprosy. Theodoric (1205-1296), believed mercury to be a good cure for *scabies grossa* and *mort mal* (the deadly sickness) which were considered to be forms of leprosy. By the Middle Ages, mercury was rarely used as a treatment for leprosy. However, it was routinely used to treat syphilis. Mercury actually healed the conditions that belonged to the treponemal moiety of the leprosy complex (Hudson 1961).

It is obvious that both syphilis and leprosy have been confused with each other during pre-Columbian times. The reason for this could be due to co-infections with syphilis and leprosy. It is known that syphilis can form co-infections with HIV and leprosy with tuberculosis (Massone et al 2011;

Agarwal et al 2000; Donoghue et al 2005). However, could syphilis be masked by leprosy through co-infections?

There are various models of co-infections used to determine how co-infections function. Models of the evolution of pathogen virulence have focused on computing the evolutionary stable level of virulence favoured by tradeoffs within a host and by competition for hosts, and deriving conditions under which strains with different virulence levels can coexist (Alizon and Van Baalen 2008; Alizon et al 2013). The results depend on the type of interaction between disease strains, such as single infection (immunity of infected individuals to other strains).

Co-infection (simultaneous infection by two strains), and superinfection (instantaneous take-over of hosts by the more virulent strain). Co-infection tends to favour higher virulence and support more coexistence than the single infection model (Alizon and Van Baalen 2008). Co-infection would be possible between leprosy and syphilis as they both would not be competing with each other. Both syphilis and leprosy are virulent in nature, however, only syphilis has a faster transmission and infection rate (Mosquera 1998). Therefore, only syphilis will have more of an appearance in the body than leprosy until syphilis is healed. Demaitre (1985) does not agree that leprosy was being misdiagnosed for syphilis prior to 1495. However, unless there were tradeoffs as a result of the co-infection, the progression of the disease (syphilis) would have slowed down.

It is possible to see co-infections by two or more pathogenic organisms in a person with a deficient immune system as they lack the ability to respond to disease allowing virulent organisms to infect the body (Donoghue et al. 2005). The two organisms that are capable of co-infecting are Mycobacterium leprae and Mycobacterium tuberculosis. The organisms have the capacity to affect bones and are evident in the DNA analysis in skeletal remains. Although syphilis is not caused by mycobacterium it still has the capacity to coinfect with other microorganisms like mycobacterium of tuberculosis. The skeletal remains of a 8-9 year old child were excavated from St Mary`s cemetery in Adelaide exhibiting both congenital syphilis and tuberculosis (Anson 2004). If a coinfection between syphilis and tuberculosis is possible then why not syphilis and leprosy. Research in this area is difficult as the spirochetes are not well preserved in bones making DNA analysis hard to prove the aforementioned possibility (Bouwman and Brown 2005). New research is being done in this area where there have been positive results (Guedes et al. 2018; Schuenemann et al. 2018).

Historically there have been descriptions of leprosy with both a sexual fear of transmission and the ability to pass the disease to the offspring. Both of which are characteristics of syphilis. In syphilis the bacteria cause signs in a matter of weeks not years like leprosy and the transmission happens at a quicker rate. Therefore, it can only be assumed that physicians were possibly seeing two diseases acting in the body at the same time and thought it was one disease. Therefore, when it appeared as one disease they were again confused and still called it leprosy.

There are some, but not many, ancient European/Middle Eastern skeletons from pre-Columbian times described as displaying signs of syphilis (Pàlfi et al 1992; Blondiaux 1994; Roberts 1994; Stirland 1994; Henneberg and Henneberg 2001; Mitchell 2003, Mays et al. 2003; Erdal 2006; von Hunnius et al. 2006; Cole and Waldron 2011; Roberts et al 2013; Rissech et al. 2013; Gaul et al. 2015; Ioannou 2018). The combined evidence of skeletal material and literary sources strengthens the suggestion of the disease's evolution in the Old World. It may never be proven without a doubt that mentagra is venereal or endemic syphilis.

Pre-Columbian published cases of syphilis in Old World

It stands to reason that evidence of syphilitic bones and teeth should exist from the pre-Columbian Old World, proving syphilis did exist at that time. Although skeletal remains which were syphilitic in appearance have been excavated and published, the Columbian faction dispute the fact that they were pre-Columbian since only archaeological dating was used.

There is archaeological dating using the concept *terminus ante quem* that means the stratigraphy of geological layers or human activities leading to the burial that had to occur before or at the time the burial was executed, not later. For example, an Ancient Greek sarcophagus made of stone worked with an ancient stonemasonry technique and covered by layers of soil undisturbed since 300 BCE cannot be considered a modern burial, thus its contents must be pre-Columbian. However, there is direct radiocarbon dating of human skeletal remains, which dates the deceased's bones directly, but unfortunately this form of dating also provides a broad range of possible ages due to error margins**.** As archaeological dating may involve stylistic dating (seriation) of an object found in the grave with the deceased it is difficult to ascertain whether the deceased bought the object just prior to death or whether it was inherited. This does give archaeologists a rough idea as to the date of death but when arguing if syphilis existed in pre-Columbian times this dating is not precise. However, Harper et al (2011) argue that radiocarbon dating is more scientifically accurate than other forms of dating (Harper et al. 2011). There have been an increasing amount of pre-Columbian skeletal remains that have been argued to be affected by treponemal disease that

have had radiocarbon dating (Mays et al. 2003; Siddell et al 2007; Rissech et al 2013; Roberts et al 2013). Whereas previously Harper et al (2011) had argued that there has not been enough reliably diagnosed pre-Columbian treponemal disease cases that have been radiocarbon dated.

Differences in opinion also arise between the pre-Columbian and Columbian mind-frames when assessing the severity of signs on skeletal remains. The Columbians are adept at diagnosing and reporting the existence of treponemal infection as they have witnessed treponemal infection in America, which leaves definite treponemal signs on the skeletal remains. They are under the impression that skeletal remains from the Old World should clearly show the same signs of treponemal disease as those found on American bones. However, American skeletal material only shows signs of non-venereal treponemal disease (Powell and Cook 2005; Armelagos et al 2012). In the Old World if a person progressed to the tertiary stage, they did not always have a severe enough reaction for their bones to display pathognomonic evidence of the disease. Maciej Henneberg had examined in 1984 postcranial skeletal remains from Texas, the next year he would examine skeletal remains from the Ancient Greek necropolis in Metaponto (Jackson et al 1986; Henneberg and Henneberg 2001). He noticed similarities between the Texas cases of syphilis and those which he was observing in Metaponto, only less pronounced. It was not until two years later that he made the connection that individuals at Metaponto also had suffered from endemic syphilis.

The Greek Colony of Metaponto

Pre-Columbian cases of treponematosis have been found and published. One important find came from Metaponto, an Ancient Greek Colony found in Southern Italy (580-250 BCE)

(Henneberg and Henneberg 1992, 1994) When archaeologists excavated this site they found two hundred and nineteen adult skeletons with fifty three children and adolescents out of which forty seven portrayed pathological signs of treponematosis. Bones with the appearance of being eaten by worms otherwise known as caries sicca, a sure sign of syphilis, were excavated along with crania where sclerotic healing on cranial vaults could be seen. Two males displayed sabre-shin tibiae and traces of inflammation were found on maxillae and subperiosteal bone deposits appeared on approximately 10% of long bones. Another good syphilis indicator was the mulberry molars found on the first molars of two juveniles and one of them also displayed slightly notched incisors (Henneberg and Henneberg 1994). This site was dated employing grave good seriation and burial styles which suggested that the deaths were prior to 250 BC. According to the pathological signs Metaponto was facing an epidemic of endemic syphilis, judging by the number of people affected and the severity of the disease. However, subperiosteal bone deposits and inflammation on maxillae are not pathognomonic of syphilis but indicate many systemic infections.

Table 1: Distribution of individuals with signs of treponematosis. Get percentages out of total in each age group.

Age group	N	with signs %	Total sample %
Subadults (0-19 years)	4	8.5	22.2
Young adults (20-39 years)	29	61.7	48.3
Older adults (40+ years)	14	29.8	29.5
Total	47	100	100

Michelet necropolis at Lisieux in France

Nine hundred tombs were discovered in the Michelet necropolis at Lisieux in France dating from the fourth century A.D. with one of the occupants displaying syphilitic signs. The cemetery was dated with seriation based on associated artefacts and features of the graves. This skeleton showed frontal destruction, osseous nodules on both parietal bones and periosteal appositions on both tibiae, a rhino-maxillary syndrome, a carries sicca and a tibial osteoperiostitis. In this case most of the syphilitic signs were due to bone reactions to the treponema bacteria. At least osseous nodules and caries sicca which were two pathognomonic signs of syphilis were clearly visible. (Blondiaux 1994).

The Dominican Friary of Blackfriars

Historical records of the Dominican Friary of Blackfriars (Glourcester England) provides dating evidence that the building had only been established for seven years when in 1246 they had to use their land as a cemetery due to the excessive number of deaths which occurred at that time. The friary survived for about three hundred years until 1538. Individual stratigraphy dates the cemetery to the early to mid 16th century, and radiocarbon dating dates the cemetery to AD 1438 – 1635. When the site was excavated archaeologists only recovered 140 bodies, with only one of them displaying signs of syphilis. Although they knew there were at least two thousand people buried there on the basis of ground penetrating radar (Roberts 1994). This technique however, is not always accurate at detecting human remains in soil. The cranium of the syphilitic person was intact and displayed lesions

pathognomonic of treponematosis, or caries sicca. Healed stellate lesions occurred on the occipital, right parietal and frontal bones, while destructive gummatous lesions appeared on the right side of the frontal bone. The nasal aperture was also destroyed, alveolar process of the maxilla and palate with extensive re-modelling and repair of the damage. Postcranially, the skeleton had extensive osteo-proliferative lesions on the ribs, clavicle, scapulae, sternum, both humeri, right forearm, right ilium, both femora, tibiae and fibulae (Roberts 1994: 104). Judging by the severity of the bone changes in only one person out of 140 indicates the possibility of succumbing to the disease after 1493. However, individuals have been recorded as having pathognomonic bone changes due to syphilis in pre-Columbian times.

Norwich Cemetery

In 1987 archaeologists (Stirland 1994) excavated a site in Norwich, where in 1254 a church stood for approximately two hundred and fourteen years. The cemetery was dated 11th century to late 15th century using seriation methods. Metal and mineralized fragments of cloth from the skeletons corroborate the accepted period of usage of the graveyard. Individuals were radiocarbon dated to AD 1088 – 1644, however the author notes that there is no archaeological evidence for burial post 1468. When its cemetery was excavated 80% of burials were unearthed, with the remaining 20% falling victim to previous building phases. At this site archaeologists excavated four hundred and thirty six undisturbed skeletons along with another four hundred to six hundred disarticulated remains.

Out of all of these remains only four showed signs of treponematosis. Although a young adult male with the burial number 412 had a fragmented cranium, the remainder of his skeleton was complete. The only sign that this male had syphilis was a minor postcranial

pathologic change. His post cranial changes were noticed in his long bones as in both distal ulnae and radii; both femora, tibia, fibulae the other changes appeared in his tarsals and left fifth metatarsal (Stirland 1994). New bone growth which could be seen on the tibia, whose anterior margins were remodeled and inflated, as well as the femora and forearms were caused by plaques of fluoride.

A mature adult female with the burial number 68 showed depressions and lesions on her cranium. Once again, the long bones were affected, in particular the distal left femur and tibia which showed fresh gummatous lesions, the bones were inflated and rugose (Stirland 1994). There were six bones in her upper limb which were inflated with distal plaques of new bone.

A complete adult male with burial number 129 portrayed obvious radial scars which had healed, focal superficial cavitation which had been in the process of healing and stellate scaring to his cranium (Stirland 1994). A radiograph was used which showed the deceased had lytic cranial lesions and sclerosis. There was periosteal new bone growth on his fibulae as well as signs of medullary ingress and radiolucent foci.

A mature adult male was given burial number 227 who had the remains of one lesion on his cranium which resembled focal superficial cavitation. Focal superficial cavitation is the third stage of Hackett's sequence which leads to the development of carries sicca (Stirland 1994). Once again, the long bones were involved, in particular the left clavicle and right humerus, both ulnae, right femur and both scapulae. The periosteal new bone growth on the scapulae appeared as striated nodes with lytic lesions within it, which produced superficial cavitation (Stirland 1994).

Nicaea, Anatolia

A good case of pre-Columbian congenital syphilis comes from the Byzantine burial in Nicaea Turkey. This individual ITK`9056/6 dated to the 13th century by seriation, this was achieved by finding coins and other archaeological artefacts found in situ. It was aged to be around 14-15 years old when it died. Judging by the skeletal remains this person was riddled with syphilis, as can be determined by the notch on the incisal edge of the central incisor. The upper right first permanent molar resembled a mulberry molar. This possible mulberry molar resembles a reported case from 20th century London (Ioannou et al. 2018). As far as the cranium was concerned the deceased exhibited a saddle nose. The left clavicle showed signs of osteosclerosis and gummatous osteomyelitis. The deceased`s 9th and 10th rib angles indicated localised osteomyelitis which spread to the sternal end. There were signs of gummatous osteomyelitis on the ulna of both upper limbs with sub-periosteal bone growth. There were signs of sabre tibia and on the surface of the tibia shafts, sub-periosteal new bone had been deposited. The right tibia displayed signs of four gummata having developed while the left tibia only had three gummata (Erdal 2006).

Austria

An ancient cemetery at St. Polten in Austria was home to deceased STP 7315/3045. While evidence suggests this cemetery was utilised between the 9th century AD and 1779 AD, owing to the stratigraphic sequence of this site along with the associated wall, both suggest the deceased could have been interred around the 14th century AD. The Vienna Environmental Research Accelerator at the university of Vienna conducted the radiocarbon determination, discovering the deceased who was around six years of age only displayed

signs of syphilis around the cranium. The deceased`s permanent incisors resembled Hutchinson`s incisors. This incisor matches descriptions and illustrations of how the development of a typical notched incisor occurs (Ioannou et al.2018). A deep indentation of the enamel on the permanent upper incisors was detected. The tip of the crown was encompassed by hypoplasia of enamel found on the upper and lower lateral incisors. A marked distal atrophy of the occlusal enamel was noted on the upper and lower canines, while the deceased`s upper and lower first molars resembled mulberry molars. (Gaul et al 2015)

There is a chance that these skeletal remains mentioned above were pre-Columbian. Numerous cemeteries have been excavated fitting entirely, or partly into pre-Columbian times, with many of them showing syphilitic signs with at least one of them -- Metaponto – showing signs of syphilis in epidemic proportions. This shows that treponemal was not only a moderate disease but also syphilitic palaeopathology only appeared in one-third of cases. When syphilitic bearing skeletal remains do come to light, they usually only exhibit non pathonognomic traits.

When syphilis appears, more often than not it will appear in less pathognomonic forms. When this occurs, the bones often imitate the pathology of other diseases. Another problem when diagnosing skeletal remains includes co-infections. This problem occurs when the immune system has been compromised, co-infections will occur in conjunction with syphilis. This becomes a problem when trying to prove the existence of pre-Columbian syphilis as the deceased could show signs of slight bone changes which may represent the remains of an

infectious disease or even reflect the pathological indicators of another disease instead of syphilis.

This is a reason for a lack of extreme cases of pathognomonic bone changes in pre-Columbian Old World. Even though this statement goes a long way in explaining the lack of extreme syphilitic cases it does not go far enough in explaining why syphilis was so severe after the siege of Naples. Constant re-infection could be the cause of the severe virulence, but if it was, it would have appeared in either literature or palaeopathology of the time.

Recorded post-Columbian signs of syphilis:

If Columbus introduced a new disease to Europe after his return from the Indies, then you would be excused from thinking that everyone should succumb to the disease in the similar manner. As this would have been the first time the population of Europe had been exposed to syphilis in the Old World then the severity would have been extreme, infecting people at a faster rate. Indeed, according to written records there is little diversity in the severity of syphilis. If the disease existed in the Old World in a mild form and the mercenaries in the army of Charles VIII had a reaction of the hypersensitivity due to reinfection or continual reinfections, there would be varying severity reaching in some individuals very high levels. This is seen in endemic areas like Bosnia and Bakwena Reserve where reinfections are common due to a high prevalence of infected individuals (Murray et al. 1956). Superinfection leads to greater severity of the disease where many will show more signs of tertiary stage syphilis (Grin 1952). If there is a range of mild to severe cases reported in the literature, then it would provide the evidence of whether individuals became reinfected or not.

To understand the severity of syphilis in the 15th -16th century then it is important to be able to compare it with modern signs and symptoms of syphilis. There are now three stages of syphilis. The primary stage heralds an ulcer on the penis or in the case of a female on the vulva which is known as a chancre. During the second stage a rash, fever, sore throat and ulcerations appear along with the chance of other symptoms. After this the patient experiences a period of relief as syphilis appears to be dormant for some years then they face the tertiary stage (Knell 2004). During this stage gummas and neurological damage occur which may cause insanity unless treatment is sought (Wright and Csonka 1996). In the sixteenth and seventeenth century, according to the writers of the time, the three stages of

syphilis were severe and took hold at a faster rate, today it advances at a slower pace and the symptoms are not as severe.

Early literary evidence of the severity of syphilis is very vivid and detailed as to the experiences of the victims. One early writer was Juan de Vigo, who in 1514 had a book published entitled Surgical Practice, in which he explained the order syphilitic symptoms appeared (Waugh 1982). According to him, the first sign the patient would be aware of was a black or white pustule, which appeared on their genitals and was enveloped by an induration (Frith 2012). Instead of just enduring one pustule, during the second stage the pustules spread over the body and were verruca like in appearance, accompanied by severe pain in the joints and limbs which could exude fetid sanies (Quetel 1990). Large round tumours and bone like callosities which were especially painful at night, occurred in the third stage. The bones were then destroyed leaving the patient to face life with permanently stunted or twisted bones. The tumours not only appeared in bones but also in muscles eroding cavities within them. During this stage the tumours began to ulcerate the body exposing bones and eroding the nose, lips, palate, larynx and genitals. (Quetel 1990)

Fracastoro who was another early writer, believed syphilis did not occur as soon as you contracted it, but instead was dormant before making its presence felt. He also believed the initial sign was a small ulceration around the genital region but adds that some tissue decay was noticed and even though eradicating the ulcer was quite a challenge sometimes it would disappear only to re-appear on the other side. As far as the second stage was concerned, Fracastoro agreed with Vigo, but added the pustules were covered in a rough and revolting crust which was yellowish in appearance and were scattered over the body which tended to begin at the scalp (Tognotti 2009). When these pustules began, they were small in size but

gradually grew. Even though the crust was usually yellow, sometimes it was white, black or reddish and hard. The pustules expanded for a few days before emitting a stinking and mucilaginous mucosa which continually leaked. This caused the pustules to ulcerate, twist and destroy the tissues before mutating into wide, dirty and corrosive ulcers that resisted treatment (Tognotti 2009). These ulcers also spread to the neural system before attacking the bones. Some people were more susceptible to the disease than others as some just experienced problems with their crania while others experienced problems with their upper limbs or maybe their lower extremities, while some were unfortunate enough to experience the destruction of their lips or their nose or even their genitals (Tognotti 2009).

Ulrich von Hutten was not only a writer but also a syphilis victim who, in 1519 wrote about his experiences. He said that when syphilis first took hold of him it was horrible, he had boils as large as acorns which emitted filthy stinking matter, causing people to keep their distance (Que´tel 1990; Arrizabalaga et al 1997; Tognotti 2009). His boils were dark green in appearance and very painful, accompanied by a burning sensation.

The classic early syphilitic signs include severely ulcerated genitals, pustules followed by necrosis which eroded soft tissues to the bone, as well as the sudden appearance of gummas. Gummas not only appeared in the tertiary stage of early syphilis but are still evident today (Que´tel 1990; Arrizabalaga et al. 1997).

It is easy to detect the similarities between the symptoms Ulrich von Hutten experienced and those of modern-day syphilis. Syphilitic lesions still tend to ravage the body affecting numerous systems. Lymphadenopathy is generalized and does not hurt, the rash ranges from faint to pale red and macular, although covering the entire body it is not itchy. The basic

symptoms range from non-existent, to mild consisting of aching joints and muscles, head aches usually occur at night, malaise and slight fever (Knell 2004). Syphilis tends to favour the more temperate climates where pustular and necrotic lesions are rare although they do appear in the tropics. (Wright and Csonka 1996)

That in 1495 syphilis was in its severe and contagious form, compared to the reasonably mild form seen today is probably due to a co-evolution of host and pathogen. The main reason for the high number of tertiary manifestations in geographic locations with active endemic syphilitic is due to an already infected and allergic host by treponemes becoming superinfected. (Grin 1953)

Aftermath of the battle of Fornovo produced the first descriptions of a highly virulent disease later known as syphilis. Doctor Cumano who tended the Venetian troops during the battle of Fornovo, recorded an early description of syphilis on the fifth of July, 1495. He mentioned men with an itchy pustule that when scratched produced a gnawing ulcer followed by more pustules which resembled grains of millet on their foreskin or glans which then spread to their face and body as well as a slight fever (Quetel 1990). They also experienced pains in their arms, legs and feet with large pustules that lasted a year or so if not treated.

Benedetto was also a doctor with the Venetian army at Fornovo and he witnessed a disease which he believed was worse than incurable leprosy or elephantiasis as people lost their eyes, hands, noses and feet. These people were in terrible pain especially at night and then came death. When attending an autopsy of a syphilitic woman he noticed tumours on her bones which were suppurated to the marrow with an intact periosteum. (Quetel 1990; Tognotti 2009)

It appeared as if doctor Cumano treated both primary and secondary stage syphilis while doctor Benedetto treated severe tertiary stage syphilis at Fornovo. Benedetto was describing a different disease when he mentioned the loss of hands and feet. Even though syphilis destroyed cartilage areas like the nose, it does not destroy completely bone, muscle and skin tissues to leave someone without hands and feet (Quetel 1990). It was possible Benedetto witnessed a co-infection of syphilis with some other diseases.

Eugenia Tognotti agreed with Cumano regarding the descriptions of primary and secondary stage syphilis. Tognotti (2009) wrote a paper on The Rise and Fall of Syphilis in Renaissance Europe in which she argued that victims of syphilis never made it to the tertiary stage. She noted secondary stage syphilis involved a fever, headache, sore throat, skin lesions, swollen lymph nodes and severe pains in the bones. The disease eventually culminated in death. Doctors did not observe the tertiary stage of syphilis until much later (Tognotti 2009).

When the epidemic began it is possible most of the victims did not reach tertiary stage syphilis, but there were people who survived, therefore they must have reached this stage. Many people died during the early days due to a hypersensitivity reaction to repeated infection, or disease synergy. According to the Oslo study (Clark and Danbolt 1955) even though people died during the epidemic their deaths could not be attributed to syphilis itself. Other signs like malnutrition and intercurrent illness probably exacerbated the disease. During this period, it was quite common for ancient armies to lose more men to disease than the results of battle as malnourished fifteenth century armies were not healthy and easily succumbed to disease (O`Shea 1990).

There were people who really fell victim to syphilis as it gradually destroyed their body. Bianchina of Bologna was a chronicler who noticed a person whose nose had been eroded as well as half the face. Sigismondo dei Conti noticed `the pustules and ulcers gnawed away as far as the marrow`, many individuals also experienced painful joints which caused continual screaming (Quetel 1990). There was a chronicler Francesco Matarazzo from Perugia who noted when people made it to this stage many of them committed suicide. He also noticed sores all over the swollen body, which remained quite nasty until they finally started to heal, leaving red scabs which eventually turned black. Matarazzo met a merchant whose syphilitic signs appeared between his thigh and torso which had been destroyed to such an extent you could see inside his body. According to Piero de Marco Parenti the pustules that destroy a body were putrid and smelt. In 1508 Benedictus reported that a printer in Venice who lost his penis and testicles to syphilis found out `a lesion on the penis was no laughing matter` (Quetel 1990).

The loss of the penis and testicles may not be something typically seen in syphilis in recent years, however clinical literature suggests that in the past it may have been possible. A man from Bakwena had his whole penis destroyed by ulcerations (Murray et al. 1956). The ulcerations on the penis was described as ‘circular or oval in shape, with a raised margin`. These ulcers can also appear on the scalp, chest, abdomen, shoulders, limbs.

Around the late fifteenth century syphilis was the worst disease around, it even beat leprosy and elephantiasis, as according to observers, syphilis had the capacity to disfigure and

decompose bodies (Tognotti 2009). A jurist Francesco Muralti of Como noted the disease not only eroded the nose but the penis as well. Another observer likened syphilis to small pox or leprosy. Fileno Dalle Tuade who was an annalist noted when a man succumbed to syphilis he would be bed-ridden as his body was covered in boils and extremely painful without a successful remedy in sight.

Jacques de Bethencourt wrote about the signs of syphilis he noticed in 1527 which unfortunately were still severe enough to destroy the body. He noticed that the first sign was a contagious ulcer on the genitals, followed by eruptions on the skin along with pains in the joints and bone which are short lived (Quetel 1990). After the patient has had syphilis for a while osseous lesions begin to appear with deep and destructive ulcerations, the collapse of the nose, erosion of the nasal area and cachexia along with other unpleasant symptoms. He noticed the liver, brain and nerves were also affected (Quetel 1990).

Although syphilis was not quite as virulent towards the end of the sixteenth century, Ambroise Pare maintains patients still lost their eyes, hearing, their nose or their palates became perforated or bones deteriorated (Quetel 1990).

As everyone is different, the way in which they cope with disease also differs. One man that managed to escape with a mild dose was Ser Tommasco di Silvestro, who mentioned in 1496 his body was covered in boils and scabs, with pains in his joints (Arrizabalaga et al 1997; Tongnotti 2009). Even though this seemed unpleasant his experience was better than that of other people, although writers in the sixteenth century believed their predecessors did experience periods of remission.

Writers who witnessed the disease before 1514 all agreed syphilis was contagious with the capacity to spread like wildfire, with some going one step further by classifying it as a plague (Quetel 1990). Even though it was transmitted venereally, and owing to its extreme contagiousness, the likelihood people were being contaminated in other ways was great. Due to its multiplicity of cutaneous manifestations along with the intensity of pain in the head, the bones which felt as if they were twisted and broken as well as the final sign being death all attest to their theory (Ross 2005).

Post Columbus paleopathology:

Even though archaeologists have excavated many burial sites which existed in the Old World they have found little paleopathological evidence to support the statements made by early doctors and writers.

According to Steinbock (1976) the reason for this is that only one third of patients would have had changes to their bones (Steinbock 1976), which is in stark contrast to the signs mentioned by early writers who described gummas eating away at both bone and cartilage. If there were as many people with this sign as the early writers suggest there should be more paleopathological evidence out there.

Paleopathology is a relatively new field which began around World War I, producing pioneering physicians and anthropologists such as Marc Ruffer, Elliot Smith and Frederic Wood Jones who diagnosed the pathology of many skeletal remains (Ruffer 1921; Smith and Dawson 1926; Smith and Jones 1910). Despite nearly a century of research and excavation of skeletal remains only a few cases of syphilis between the fifteenth and sixteenth century have been published (Mafart et al 1998). Even though there were many writers in the pre-Columbian Old World they mainly wrote about the signs and symptoms of syphilis but did not publish examples of syphilis.

Syphilis was not just a disease which caused a lot of grief for many people, but it was also a disease which covered its tracks by imitating other diseases, which exhibited both pathognomonic and non-pathognomonic bone changes. There are some bone changes which can only indicate syphilis while others could be the result of other diseases. Therefore, if

skeletal remains show signs of mild bone changes or there was a co-infection then diagnosis is harder to ascertain. The environment also plays a part in preserving skeletal remains, given the right conditions and soil type bodies, like those buried in a bog, or skeletal remains buried in the right soil type lead to the correct diagnosis.

Lastly the dating of the skeletal remains related to people who had syphilis between the late fifteenth century and the late sixteenth century, when syphilis was at its most virulent form has proved difficult. Dating unmarked graves of people to such a specific time comes down to relative employing seriation of grave goods, radiocarbon dating of organics grave goods or direct radiocarbon dating of skeletal remains. However, due to the error margins of radiocarbon dating this technique can a provide broad dating range. Due to these techniques, comparing palaeopathology of this time period to recorded medical literature, any theories that stem from this may not be completely accurate.

As syphilis was serious enough to have completely blanketed Europe from the early sixteenth to seventeenth century one important question arises and that is `Does palaeopathology coincide with the written evidence`? When comparing the historical literature with the paleopathological evidence there is inconsistency. However, only as little as 2-13% (Rothschild 2005) or no more than one third (Steinbock 1976) of patients suffering from syphilis develop signs on the skeleton. There also appears to be more interest in recording pre-Columbian syphilis than post-Columbian.

St Mary`s Spital

Alex Bayliss and Jane Sidell (2007) tested the four burial phases at the medieval cemetery of St Mary`s Spital, through a programme of radiocarbon dating and Bayesian modelling. These radiocarbon dates provide a chronological framework for the cemetery. Burial period fourteen commenced between AD 1040 and 1155; period fifteen which is believed to have commenced between AD 1170 and 1210; period sixteen commenced between AD 1230 and 1260 while period seventeen commenced between AD 1365 and 1410 (Connell et al. 2002). Burials at this cemetery may have slowed down around AD 1485 and ceased when the cemetery closed in AD 1539 (Bayliss and Sidell 2007).

Contained within phases fourteen to sixteen (AD 1040-1365) were twenty-five people who were diagnosed with treponemal disease. However, period seventeen (c AD 1400 to 1539) witnessed a spike in treponemal cases with `six males, seven females, two unsexed adults and three sub adults, with a crude prevalence rate of 2.8% (18/650) (Connell et al. 2002). The increased prevalence rate is obviously due to the epidemic that occurred after Naples, as well as the increase in tertiary stage syphilis with three sub adults showing signs in period 17.

The Mummy of Maria d` Aragona

The mummified remains of Maria d` Aragona, an obese sixty-five-year-old woman who lived between AD 1503 and 1568 were exhumed in Naples. Clearly she lived after the return of Columbus and the siege of Naples. Maria presented with asymmetrical swelling of the lower limbs, right inguinal tumefaction, with a cutaneous papilloma and a white yellowish

cicatricial area of 30x20mm in the right arm (Fornaciari et al 1994). There was a dressing on the left arm which covered an oval ulcer. The histological, immunological and ultra-structural findings clearly indicate treponemal infection. The macroscopic characteristics of the cutaneous ulcer are typical of third stage luetic gumma which contained numerous treponemes.

Although the mummified remains do not provide any detail as to the state of the skeleton, it does show limited ulcers on her body. This means that this individual was not suffering with the same severity as many others or she died before the disease progressed more.

Roca Vecchia necropolis

Roca Vecchia, a medieval necropolis, housed a well-preserved male skeleton who was buried between the middle of the fourteenth century and the beginning of the sixteenth century. The frontal region of the cranium showed broad destructive lesions and the left parietal bone portrayed a large injury, the result of a blow by a sharp weapon (Fornaciari et al 1994). There were a number of destructive lesions on the long bones, with a strong periosteal reaction. This is most likely a severe case occurring in the post-Columbian period.

Medical treatment of syphilis pre-and post-Columbus

The paleopathological record differs somewhat from the written record as far as syphilis peaking during the sixteenth century is concerned. As mercury was used to treat syphilis it lowered the pathological severity in skeletal remains. These conditions also applied to syphilitic patients during the Old World pre-Columbian times (It is possible that mercury may have reduced the severity of signs evidenced in patients).

Mercury was first used to treat syphilis in AD 1496 a year after the battle of Fornovo by a physician known as Giorgio Sommariva (Grimes et al 2013). Mercury was also used to treat leprosy without success. Doctors may have occasionally confused leprosy and syphilis, if a patient with leprosy was treated with mercury and improved then he obviously had syphilis.

Guaiac wood

As mercury could have lessened the severity of syphilis in the Old World, the skeletal remains excavated from that region and dated before AD 1492 may not reveal lesions on bones and other syphilitic signs. In the Americas there is no record of natives ever using mercury as a cure for syphilis or other diseases. Although it is possible the treponema strain in the Americas was more virulent than the European strain, severe pathologic bone changes found in America may be a result of no treatment with mercury that was partly effective against syphilis.

The Guaiac Wood used in Americas, known as Holy Wood, was imported into Europe from San Domingo to treat syphilis after Columbus and then during the late eighteenth century Salsapariglia, an anti-syphilitic drug, was used (Holcomb 1937; Benedek 1992; Porro et al. 2009).

The Guaiac Tree with its yoke leaf is a native of the West Indies and the north coast of South America. The Indian tribes have been using the wood of the Guaiac Tree as a traditional medicine for centuries (Geske 2009). They exported the wood to Spain at the beginning of the sixteenth century and then from there to the rest of Europe where it was used to treat syphilis.

The guaiac wood comprises 25% resin contained in the heartwood but only 2 to 3% resin is contained in the sapwood. The guaiac resin is made up of colophonic acids of the furoguaiacin, lignin, and pheolguaiacol types along with mainly guaiac wood oil, which in turn consists of sesquiterpene alcohol guaiol, alkaloids and triterpene saponins with the aglycone oleanolic acid (Geske 2009).

The guaiac resin is particularly good to use in the treatment of disease as it has a diuretic and diaphoretic effect on the body, it helps cure fungal infections due to saponins in the wood and the oil not only contains an anti-inflammatory, but antiseptic and wound healing properties as well (Geske 2009).

According to Hutten, doctors used the guaiac wood to eliminate the French Pox. It achieved this by gradually eliminating tumours, indurations, nodules and fistulae, it also removed fluxes and brought ulcers to suppuration. It cleared up hidden problems throughout the body

by purifying the blood and in so doing it eliminated the poisons therein, rendering the disease powerless to continue along its destructive path. The guaiac resin eliminated the toxins through the urine and perspiration in some people and in others through the excrements of the stool (Munger 1949). Before health was eventually restored the resin would cause excessive sweating, then the toxins would leave the body through urine where a large number of impurities are eliminated.

In his book De Cura Morbi Gallici, Pol mentioned three thousand Spaniards inflicted with syphilis were administered the resin from the guaiac tree, after which their health was restored (Munger 1949). Apparently, these Spaniards tried numerous treatments, without success before. If these Spaniards in fact had syphilis, then it can be assumed that their recovery was an attempt at propaganda. The House of Fugger grew wealthy over the monopoly of the importation of guaiac, their success depended on the support of physicians and deluded patients (Holcomb 1935:291; Crane-Kramer 2000). By the time that many of the treaties exposing the benefits of guaiac wood were published, including Von Hutten, guaiacum had generally been dismissed in favour of mercury as an effective treatment for the disease (Downing 1916).

Syphilis was quite common in Florence and around other parts of the Italian Peninsula during the early to mid 16th century. Benvenuto Cellini, (born AD 1500) was a painter and sculptor who was infected by a serving maid, mentioned his body was covered with large blotches. Doctors treated him with mercury, causing an adverse reaction, which was so bad he decided to ignore the wishes of his doctors and try the guaiac wood, which he heard was recommended by Fracastorius (Dennie 1962). Cellini believed Oviedo was on the right track when he mentioned a cure should come from the same place the disease originated from. The

belief of these two men encouraged him to infuse the wood to make a tea and drank it while going on a diet (Dennie 1962). He kept this practice up for fifty days, believing he was cured, he went off the tea only to have a relapse, so he decided to keep taking it for four more days but still he was not cured.

Ruiz de Isla was a true believer in the benefits of mercury as opposed to that of the guaiac wood. He made an ointment into which he added mercury of various strengths depending on the particular stage of syphilis the patient had reached, which was to be applied to the skin. Ruiz studied his patient, considering the type of lesion, the patient's temperament and the dimensions of the body before deciding on the maximum dose of mercury the patient could safely handle (De Ricon-Ferraz 1999). He wrote detailed instructions for doctors to help them achieve the correct dose of mercury in his treatise and he devoted the whole of chapter VIII to the subject.

Even though Ruiz de Isla was a staunch advocate of the powers of mercury, he was still interested in the guaiac wood. He believed that there were certain conditions that should be followed, such as only using it during a certain period of the year, the choice of high-quality guaiac as well as the most effective way of preparing the infusions (De Ricon-Ferraz 1999).

Finally, the opinions concerning the most effective cure for syphilis started to change, even staunch advocates of the guaiac wood like Fracastoro now either questioned or denied their beliefs. Thierry de Hery, an influential French barber surgeon, for reasons best known to himself, suddenly concluded he had never seen a patient cured by just the guaiac wood (Munger 1949). If the patient improved it must have been due to mercury with a small amount of wood. A doctor who had performed miracles by using guaiac wood was Nicolaus

Massa, who now completely denied these events and mentioned his success must be due to mercury. Gabriele Falloppio was another one who changed their tune and now solely believed in mercury. Paracelsus followed by Michael Blondus believed those treated with the guaiac wood suffered relapses leaving them in a worse condition than they were in before (Munger 1949). He also believed syphilis did not come from the New World or the Indies.

Mercury

The Ancient Egyptians were one of the first societies to use mercury and cinnabar (mercury sulphide). Evidence regarding the use of quicksilver in medicine is non-existent in dynastic times. As for mercury sulphide or cinnabar, evidence concerning that is not clear. According to the Ebers papyrus it was possible mercury was used in medicine although, due to the translation of the Egyptian terms “prst” and “mnst”, it is not certain. Mercury is believed to mean minium, red lead, red ochre or dragons blood (cinnabar or red mercuric sulphide) (Goldwater 1972). Regardless of the translation problems, there appeared to be a consensus relating to the use of mercury in medicine by a number of academics. According to Imhotep, who dabbled in ancient Egyptian medicine, they possessed the following drugs:- salts of lead like sulphate, acetate of copper, sulphate of mercury and pomegranate.

During the times of Hippocrates there were two different types of ointment containing cinnabar which is mercury sulfide which could be used either for burns, or ulcers and fistulae. The burn ointment consisted of old pork fat, wax, oil, incense, lotos scrapings, miltos and arum leaves cooked in wine and oil whereas the contents of the ointment used for ulcers and fistulae just consisted of miltos and honey (Goldwater 1972; Parsons and Percival 2005)

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There is a further evidence for the use of cinnabar (mercuric sulfide) as medicine by Hippocrates (460-377 BC). Hippocrates has been argued to have used cinnabar as a salve in the treatment of trachoma and presumably also for syphilis, and as a laxative it was administered orally. In the oral administration of metallic mercury, it was ingested with milk or wine (Kruse 2008).

Celsus wrote books on diseases and treatments for them. One of the treatments was the use of *minium* or *minium Sinopicum*. In book IV.22 he wrote about an enema, consisting of a mixture of minium and salt water which he used to treat intestinal cancer (Goldwater 1972). In book V devoted to oral medicine he used minium when purging was required, if treatment was needed to relieve ulcerated genitals then he had a salve containing Sinopicum minium which appeared in book VI.18 (Greive and Futvoye 1838). There is a possibility that this could be an early treatment for venereal infections. In book VI.6 there was a treatment for trachoma by using minium which probably reflected the influence of Egyptian medicine.

Galen`s dislike for mercurial medicine had an influence on doctors who followed as they too were against using mercury to treat disease. Writers like Oribasius (AD 325-403), Aetius of Amida (AD 502-575), and Paul of Aegina (AD 625-690) rarely wrote about mercury in medicine (Goldwater 1972). Mercury was known in medieval times but rarely used although, due to the influence Arab medicine had in the Latin West after the tenth century, mercury once again grew in popularity.

A 13th century alchemist began using cinnabar (mercury sulfide) as a medicinal elixir (Charlier et al. 2014). This medicinal elixir was said to make an old man young and drive out all sicknesses of the body (Mahdihassan 1986).

Leprosy was a disease which seemed to fluctuate over the years, becoming particularly noticeable during the Crusades from AD 1096 to 1270. As many Crusaders contracted this disease, they were initially treated by physicians in the Middle East who tried to cure their leprosy by using Saracen ointment which is mercury based. (Kruse 2008). It is now known that mercury only heals syphilis in the primary and tertiary stage when bacterial load is low, not leprosy. Bejel exists in the Middle East as it has for centuries and will probably continue to do so for centuries to come. To quote Oviedo ``so great is divine mercy that where our sins produce a punishment, God sends a remedy``. (Downing 1916; Williams et al 1927). The remedy used by Arabic physicians was mercury.

During the mediaeval times there was renewed faith in the healing properties of mercury. Roger of Palermo (AD 1170-1200) was a doctor who had faith in mercury for treating chronic skin disorders (Goldwater 1972). In the thirteenth century Theodoric of Cervia (AD 1205-1298) and Arnold of Villanova (AD 1240 -1311) also became known for their use of mercury. According to Neuberger (AD 1910-1925) Guy de Chauliac (AD 1300-1367) treated chronic ulcers with the use of lead coated with quicksilver (Stephens 2010)

According to O`Shea mercury is a potent diuretic and when it was used in toxic doses it induced salivation which led doctors to believe that the use of mercury would cause the syphilis virus to be excreted eliminating the illness from the body (O'Shea 1990). Contemporaries of Paracelsus stated that for the effectiveness of mercurial treatment three

pints of saliva were needed to be produced, which in turn caused the poisoning of the body. There were many ways of administering mercury to treat syphilis. Since mercury does not absorb well through the skin itself, physicians chose to administer mercury by inhalation of mercury vapours (fumigation) and by rubbing mercurial salves upon lesions (Thomann 2015).

In the hope of curing syphilis in post medieval Europe, a range of medicines were used such as plant based cures like guaiacum, arsenic and bismuth but mercury remained the favourite. (Goldwater 1972; Quetel 1990). According to Swiderski in 2008 the early medieval period marked the beginning of the use of mercury to treat skin problems, and was used to fight syphilis in the fifteenth century (Goldwater 1972). Surprisingly mercury, which was commonly mixed with arsenic or other compounds, was still in use during the nineteenth and early 20th century even though mercury was toxic (Quetel 1990).

O`Shea stated that there were several forms of mercuric compounds, one of which was calomel or sweet mercury ($Hg_2Cl_2$) which could be either taken by injection or through the mouth, in quantities of five grains (=325mg) (O`Shea 1990). Both mercuric chloride and calomel were used as salves even though mercuric chloride ($HgCl_2$) had a corrosive effect According to O`Shea, during the early 16th century doctors decided to fumigate their patients, by placing them in an enclosed area such as a tent, barrel, or over-heated room for a number of weeks or even months making the patients breathe in mercuric chloride vapours, heated cinnabar (HgS) and metallic mercury (O`Shea 1990). In 1997 Beck, who studied O`Shea`s work, agreed with him on the course of treatment then added that the patients were often rubbed down with mercury-based ointments, close to a hot fire where they remained for a long time enabling them to sweat (Frith 2012; Zuckerman 2016). If this treatment had run its

course and the patient still had the disease, then the treatment would start again. This kind of treatment could cause toxic side-effects. Physicians had known since ancient times of the toxic effects mercury has on the body (Thomann 2015).

As mercury is known for its anti-inflammatory and spirilocidal effects many people now are firm believers in its ability to heal although it is still not clear if mercury really was an effective treatment (Holmes 1984). It kills spirochetes by inhibiting the process of glycolysis which *T. pallidum* depends upon as the sole pathway for the synthesis of adenosine triphosphate (ATP) (LaFond and Lukehart 2006; Officioso et al. 2016). This process reduces the amount of energy for the bacterium to survive. According to Fabricius in 1994 and Goldwater in 1972 mercury can induce a Jarisch-Herxheimer reaction, caused by the systemic release of a lot of toxins into the body, the spirochaete bacteria then die during the treatment (Goldwater 1972; O`Shea 1990; Fabricius 1994; Zuckerman 2016). In 1990 O`Shea mentioned that mercury used in systemic or topical treatment could only have helped the patient during the primary or tertiary stage of syphilis as there are not many spirochaetes circulating around the body (O`Shea 1990). According to Holmes in 1984 mercury treatment could have helped resolve gummata, however, this is just speculation. It was possible the success doctors had with mercury treatment was due to `the fluctuating nature of untreated syphilis`. Mercury remained common treatment for syphilis until the arsenic compound arsphenaminel (1909), bismuth and later the antibiotic penicillin (1943) were introduced (McCafferty 1923; Burke 1925; Anderson et al. 1989; Ozuah 2000).

Mercury builds up in trabecular bone and is absorbed into compact bone when there is an excess of Hg in the body. (Rasmussen et al, 2013). Mercury differs from other heavy metals as the bone does not retain any significant amount, but the organs do. Garcia et al. (2001)

attested to this fact when they reported that an autopsy found less than 0.05 ppm of Hg in bone but 0.25 in the kidney and 0.14 ppm in the liver (Garcia 2001; Zuckerman 2016).

Mercury, after being ingested has a half-life in the body of about seventy days (Baselt and Cravey 1982). In 1969 Aberg et al. stated that mercury is eliminated from the body through both faeces and urine. After forty-nine days 33-35% of Hg is eliminated in the faeces and ca. 3.3 % is eliminated through the urine (Aberg et al 1969). A group of Japanese workers who had not been exposed to mercury were tested and found to contain Hg concentrations of 119 ug /L in their urine (Nakayama et al 1977). Today most human tissues contain 10-20% methylmercury, with the kidneys containing 2% and hair containing 63%. According to Rasmussen et al. (2013) Hg levels in Medieval and Renaissance Danish people who were not exposed to mercury had between 10 and 100 ng /g in samples of compact femoral bone (Rasmussen et al 2013).

The tissues in the human body constantly undergo turnover which remodels and replaces the damaged ones. The turnover rates in soft tissues are high, this is different from the turnover rate in the bone which can also differ from bone to bone. The highest turnover rate in bones can be found in the trabecular bones and the lowest rate is found in the compact long bone tissue (Rasmussen et al 2013).

For the above reasons, the mercury content of skeletal remains of syphilis patients can be elevated only if their death occurred at or shortly after active treatment was administered and may vary depending on body part. Skeletal elements overlaid with adipose tissue that accumulates mercury can capture more mercury during postmortem taphonomic processes, and will increase its content above of what is expected in the bone tissue of a living patient.

Methods:

The human skeletal collections chosen to examine signs of syphilis in bone were located at the University of Copenhagen, University of Łódź, Poland, Museum of Natural History in Vienna, the Villa Poppea in Oplontis near Pompeii and Metaponto. The mercury analyses were performed either overseas as in the case of Łódź and the Museum of Natural History, Vienna or at the University of Adelaide Microscopy.

In the Natural History Museum in Vienna the Department of Anthropology houses a couple of hundred Ancient Egyptian remains. The cranial samples were derived from Giza, El Kubanieh Nord and Ermane. Excavations of the Western Cemetery of Cheops's pyramid had begun in 1902. The skeletal collection of the Natural History Museum of Vienna was excavated by Hermann Junker in 1911/12 and 1912/13. He recovered 177 dry skulls which were sent to the Natural History Museum in Vienna. Individuals at El Kubanieh were buried in tombs that were protected from taphonomic damage during the Middle Kingdom (2050 BC and 1652 BC). The Ermane cemetery was dated to the Christian era (up to 14th century AD). These Egyptian remains consist mostly of crania with some post-cranial bones. Sex and age of individuals was determined by standard osteological methods. All crania and post-cranial bones in the collection were inspected for lesions related to treponemal infection. Five Egyptian crania showed signs that can be attributed possibly to a treponemal disease. From among those, five skulls (Figure 1) were selected since they had associated loose small fragments suitable for Laser Ablation Inductively Coupled Plasma Mass Spectrometry (LA-ICP-MS) analysis and avoided destructive sampling. Fragments of 5 other skeletons showing no pathological signs were used as controls. These were one fragment of long bone, one piece of dentine one of enamel and two from skull bones.

LA-ICP-MS measurements for mercury content were conducted employing these bone samples which weighed 2-3 g. There was no obvious surface contamination. Approximately 1 gram was subjected to the analysis. Each sample was sonicated in reagent grade 1 water before affixing to glass slides with double sided tape for mounting in the laser cell. The laser ablation analysis was conducted following Stadlbauer et al. (2007) at the University of Natural Resources and Life Sciences Vienna (BOKU). A 193 nm ArF excimer laser (NWR 193, ESI, Portland, OR, USA) was coupled to a sector field ICP-MS (Element XR, ThermoFisher Scientific, Bremen, Germany). Quantification was accomplished according to Draxler et al. (2016) using hydroxyl apatite pellets spiked with Hg. Validation was accomplished using bone meal certified reference material CRM 1486 (NIST, Gaithersburg, USA). Recoveries for Hg were between 96 % and 106 %. Laser ablation was performed using 50 µm spots. Line scans were measured in triplicates on the inner area of the bone fragments to avoid areas of exogenous contamination. The Hg signals were normalized to Ca as internal standard. The values for the lines were averaged after quantification.

In the University of Łódź in Poland two skeletal collections were used for this study. These collections of skeletal remains are known as Brześć Kujawski (BK5) and Kolonia and are pre-Columbian in origin (AD 11^{th} – 13^{th} century) (Spinek et al. 2016). After observing these remains for possible pathologies which could indicate syphilis, 8 remains from the Kolonia collection and 14 from the BK5 collection were chosen for mercury analysis.

Mercury is released from the soft tissues through decomposition and according to Rasmussen (2013) mercury is then re-absorbed into the bones. This process is caused by an excess of

mercury stored in the adipose fat and in certain organs like kidneys or liver. Bearing this in mind, samples were taken from either the ribs or femur, because they could absorb mercury post-mortem from decaying internal organs. The femur was chosen when there were no ribs to sample from the individual.

The femur and rib samples were crushed into a fine powder with a weight of 0.1g. These powdered bone samples were then stored in sample containers before being sent to the University of Warsaw, Laboratorium Biogeochemii i Ochrony Środowiska (Laboratory of Biogeochemistry and Environmental Protection) for mercury analysis by the use of a mercury analyser called a Milestone DMA 80.

In the University of Copenhagen 300 skeletons from a leprosarium called Aaderup were examined. The leprosarium is dated from AD 1300 to 1500. After surveying this collection for possible signs of syphilis, 26 skeletons were selected. Mercury concentrations were analysed from 16. All samples chosen for analysis were taken from the ribs as Rasmussen (2013) believed the ribs hold a higher concentration of mercury than the majority of other bones. The ribs were analysed by Laser Ablation inductively coupled with plasma mass spectrometry (LA-ICP-MS) at the University of Adelaide.

Results (A manuscript submitted to the Journal of Archaeological Science)

Don't Blame It on Columbus - Syphilis in Ancient Egypt suggested by presence of mercury in skeletal remains

Abstract:

The presence of syphilis in the Old World before Columbus' voyage to the Americas is debatable. Skeletal remains are the primary way of diagnosing treponemal disease as it is also very difficult to recover the test pathogen's aDNA from skeletal remains. Syphilis was likely treated with mercury in the past as indicated in the examination of six ancient skeletons from Giza, El Kubanieh and Ermenne showing skeletal lesions suggestive of treponemal disease. The mercury content, determined by the use of LA-ICP-MS[1], was much higher than the soil background levels and significantly higher than in the five control samples that lacked lesions. The widespread use of mercurial treatments in the Old World could have prevented skeletal lesions from developing to the extent that they would allow reliable diagnoses of syphilis and thus influence theories concerning its historical origins.

[1] Laser Ablation Inductively Coupled Plasma Mass Spectrometry

Introduction:

Syphilis is one of those interesting diseases which has been around for centuries and is still causing health problems that may continue for many years to come. An improved understanding of the origins of syphilis through paleopathology will contribute to knowledge concerning the ability of *Treponema* to adapt and evolve. The central question regarding the origin of syphilis is whether it existed in the Old World for millennia, or was introduced from the New World as recently as Columbus' voyage (Baker, et al., 1988, Hackett, 1963).

The debate concerning the history of syphilis, especially its introduction to the Old World by Columbus is highly controversial and is yet to be settled. The main arguments centre on the differences observed in archaeological skeletal samples. Thus far the only way to diagnose syphilis in ancient human remains has been through the observation of pathological changes on bones and teeth. DNA of the syphilitic pathogen (*Treponema pallidum*) is difficult to detect (Bouwman and Brown 2005). Besides the usual vagaries of aDNA preservation, in the longest tertiary stage of the disease, very few pathogens are present in the body (Bouwman and Brown 2005; Anastasiou and Mitchell 2013; von Hunnius et al. 2006). Consequently the diagnosis of syphilis in an adult skeleton is based on a pattern of non-specific pathological signs, since no skeletal sign is pathognomonic for the disease. Moreover, only as little as 2-13% (Rothschild 2005) or no more than one third (Steinbock 1976) of patients suffering from syphilis develop

signs on the skeleton. Patterns vary from individual to individual, and not all diagnoses are certain.

Severity of skeletal signs is highly variable. In samples from the Americas, severity of signs is commonly high which suggests treponemal disease originated in the Americas. There are, however reports of pre-Columbian skeletons showing pathological signs of syphilis in several locations in the Old World (Erdal 2006; Gaul et al. 2015; Henneberg and Henneberg 1994; Palfi et al. 1992; Roberts CA 1994; Stirland 1991; Mays et al. 2003; Von Hunnius 2006). These are disputed as in most cases the severity of signs is less than that observed in non-venereal treponemal samples from the Americans (Harper et al. 2011). In the New World skeletal signs relating to treponemal disease are prolific, while they are much less pronounced in Old World pre-Columbian skeletons. Consequently, finding a new method that strengthens detection of syphilis where there are no pathological signs on bones will be extremely valuable in relation to the testing hypotheses regarding the geographic origins of syphilis.

One main cause of differences in severity of skeletal manifestations between Old World and New World syphilis may have been the use of mercury to treat the disease. Although not completely effective against the disease, mercury is known to have reduced its signs and symptoms (Warner 1881; Walker 1869). Pathological changes occurring during the tertiary stage maybe limited by the use of the mercury. If mercury interrupts progression of carries sicca (the only pathonomic sign of syphilis), it may

not progress to the stage of stelate lesions and may not be widespread over the cranial vault. Early pathological signs can be hidden within the diploe, thus not be visible without histological study of the cranial vault.

Mercury is viewed as a powerful anti-mitotic and anti-inflammatory agent (O'Shea 1990). When mercury was locally applied it aided healing of ulcers. It has been noted to induce a Herxheimer reaction and to clear cutaneous lesions of spirochetes that may have affected bones of the cranial vault (Corey 1984). The systemic use of mercury e.g. through inhalations, is capable of reducing treponemal infection to the level of sero-negativity (O'Shea 1990). From the medical literature of the 19th century, mercury is known to have reduced and effectively controlled treponemal infection (Hutchinson 1874; TAIT 1899).

Ancient Egypt has yielded no convincing pathologies that may indicate syphilis (Smith 1908), despite having both the cultural and environmental factors that could be conducive to the presence of sexually transmitted disease (Manniche 1997). Mercury has been argued to have been used in medicine as early as the ancient Egyptian times (Dawson 1930; Goldwater 1972; Leake 1952). In order to examine the potential impact of mercury treatment on the prevention of the development of syphilitic skeletal lesions, this study seeks to identify excessive levels of mercury in some ancient Egyptian crania that have limited pathological signs suggestive of syphilis. In addition, mercury concentrations are examined in skeletal samples that do not exhibit the pathological changes suggestive of syphilis.

Materials and Methods:

In the Natural History Museum in Vienna the Department of Anthropology houses a couple of hundred ancient Egyptian remains. The cranial samples were derived from Giza, El Kubanieh Nord and Ermane. Excavations of the Western Cemetery of Cheops's pyramid had begun in 1902. The skeletal collection of the Natural History Museum of Vienna was excavated by Hermann Junker in 1911/12 and 1912/13. He recovered 177 dry skulls which were sent to the Natural History Museum in Vienna.

Individuals at El Kubanieh were buried in tombs that were protected from taphonomic damage during the Middle Kingdom. The Ermane cemetery was dated to the Christian era. These Egyptian remains consist mostly of crania with some postcranial bones. Sex and age of individuals was determined by standard osteological methods (White et al 2000). All crania and post-cranial bones in the collection were inspected for lesions related to treponemal infection.

Many Egyptian crania showed signs that can be attributed to syphilis. From among those, five skulls (Figure 1) were selected since they had associated loose small fragments suitable for LA-ICP-MS analysis that also reduced skeletal destruction associated with sampling. Fragments of 5 other skeletons showing no pathological signs were used as controls. These were one fragment of long bone, one from dentine one from enamel and two from skull bones. Laser ablation inductively coupled plasma mass

spectrometry (LA-ICP-MS) measurements for mercury were conducted. Samples weighed 2-3 g. There was no obvious surface contamination. Approximately 1 gram was subjected to the analysis. Each 1 g sample was sonicated in reagent grade 1 water before affixing to glass slides with double sided tape for mounting in the laser cell. The Laser ablation analysis was conducted following Stadlbauer et al. (2007) at the University of Natural Resources and Life Sciences Vienna (BOKU). A 193 nm ArF excimer laser (NWR 193, ESI, Portland, OR, USA) was coupled to a sector field ICP-MS (Element XR, ThermoFisher Scientific, Bremen, Germany). Quantification was accomplished according to Draxler et al. (2016) using hydroxyl apatite pellets spiked with Hg. Validation was accomplished using bone meal certified reference material CRM 1486 (NIST, Gaithersburg, USA). Recoveries for Hg were between 96 and 106 %. Laser ablation was performed using 50 µm spots. Line scans were measured in triplicates on the inner area of the bone fragments to avoid areas of exogenous contamination. The Hg signals were normalized to Ca as internal standard. The values for the lines were averaged after quantification.

Results:

Mercury Content

The results of the laser ablation (LA-ICP-MS) analysis of the mercury content in the ancient Egyptian bone samples indicated a clear relationship between pathological defects in the skulls and a higher concentration of mercury. Mercury levels in the skulls showing lesions potentially interpretable as syphilis were higher than in the controls that had no pathological signs of syphilis (Table 1). There was a significant difference of average mercury contents in skulls with and without signs of syphilis (Table. 2)

One of the small skull fragments (5230) selected as a control showed fairly high mercury content (0.174µg/g). It is impossible to determine whether this fragment belonged to a person who potentially suffered from syphilis because no pathological signs could be found either due to the fragmentary preservation or the fact that such changes do not occur in all syphilitic individuals.

Table 1: Mercury concentrations in Egyptian skulls with pathological signs of syphilis compared to those with no signs of syphilis.

Skeleton number	Sample	Site Label	Sex	Age	Hg µg/g	RSD (%)*	Signs of syphilis yes/no
5154	Skull	Giza/Pyramids	Male	Adult	0.209	50	Yes
5269	Skull	Giza/Pyramids	Female	Adult	0.212	44	Yes
5250	Skull	Giza/Pyramids	Female	Adult	0.126	43	Yes
4811	Skull	El Kubanieh	Female	Adult	0.116	50	Yes
4836	Skull	El Kubanieh	Female	Adult	0.151	50	Yes
Control sample no syphilis							
5230	Skull	Giza/Pyramids	? Fragmented Skull	Adult	0.174	25	*Observation impossible
5522	Dentin	Ermenne Nord	Male	Adult	0.045	21	No
5522	Enamel	Ermenne Nord	Male	Adult	0.016	20	No
1981	Femur	Giseh Westschacht	Male	Adult	0.008	25	No
4806	Skull	El Kubanieh	Female	Adult	0.090	50	No

Skeleton number	Sample	Site Label	Sex	Age	Hg µg/g	RSD (%)*	Signs of syphilis yes/no
4970	Femur	El Kubanieh	Male	Adult	0.008	32	No

*Fragmented remains

* The average relative standard uncertainty (RSU) of the Hg content is about 40
*RSD means precision – calculated (standard deviation / average value) * 100

Table 2: Statistical tests of mercury content in skeletons with signs of syphilis and non-syphilis (Skull 5230 included and not included) t-tests for unequal variances

Skulls	N	Mean Hg	t	Sign	Note
With syphilis	5	0.163	comparing with no syphilis		
Non Syphilis	6	0.058	3.09	0.013	Skull 5230 included
Non Syphilis	5	0.03	4.81	0.001	Skull 5230 not included

Figure 1A

B

C

D

E

F

Figure 1: Cranial pathology suggestive of syphilis

Figure 1. Adult male cranium 5154 from Giza shows signs of carries sicca (Hackett Carries sicca grade 1-2) intermixed with taphonomic changes on the parietal bone (A), and on the frontal bone (B). Mercury content 0.209µg/g. Adult female cranium 5269 from Giza, several small lesions with advanced and partly complete healing (Hackett caries sicca grade 4-5) (C), mercury content 0.212µg/g. Adult female cranium 5250 from Giza: One healed lesion on right side of the frontal squama close to the coronal suture indicated by the arrow (Hackett caries sicca grade 4-5) (D), mercury content 0.126 µg/g. Adult female cranium 4811 from El Kubanieh. Arrows indicate gummatous lesions with signs of healing (E). Other multiple circular lesions with extensive destruction of the cranial vault may be taphonomic (Hackett caries sicca grade 4-6). Mercury content 0.116µg/g. Adult Female cranium 4836 from El Kubanieh. There were partly healed lesions on the frontal bone (F) (Hackett caries sicca grade 1-3). Mercury content 0.151µg/g

Descriptions of pathological changes:

Images of skulls showing pathological signs are presented in Figure 1.

Below find an overview of the pathological changes observed in each skeletal sample:

5154: Healed carries sicca with stellate lesions and traces of nodule cavitations. Observations complicated by taphonomic damage.

5269: In the centre of the left parietal bone there are two oval lesions that have penetrated external table and affected dipole they have rounded edges while trabecular of the dipole show signs of healing. They are approximately 50mm long and 7mm wide resemble those describe for 4811. Differential diagnosis and interpretation is the same as for 4811. On the left side of the frontal squama there is a oval shot low depression whose greater access is orientated in the coronial plane and it is about 27mm while the antero-posterior width is about 80mm material border is clearly visible whereas medially the depression gently slopes out onto the surface of the frontal bone without definitive margin the floor of the lateral half of the depression is un even with small pits and elevations as if the underling diploe was covered by a fresh bone. The outline of the lateral border is uneven with a few millimetres break in the middle. The described lesion maybe the result of low impact trauma or localised infection.

5250: Early serpingenous cavitation of the forehead. On the frontal bone close to the right coronal suture there is a shallow oval depression approximately 15 by 10 mm its floor is covered by small nodules of bone. It maybe a result of low impact trauma or localised infection that has healed.

4811: On the right frontal squama, small healed gummatous lesions. On the frontal bone and both parietals, numerous round or oval lesions in various

stages of healing. These have penetrated external table and exposed diploe, diploe trabeculae are visible of the bottom of lesions, some are partly obliterated by new bone formation indicating healing, some others have still clearly distinguishable. Edges of lesions are rounded; the borders are irregular with some indentations. on the frontal bone and the left parietal several lesions coalesce into larger regular features. There are approximately 30 lesions. Exact number would depend on how to discern as seperate lesions those that partly coalesce.

Differential diagnosis should include carcinomas, multiple myeloma, Ewing's sarcoma, meningioma, Paget's disease and tuberculosis. Metastatic neoplasms of breast cancer and lung cancer are known to occur on the skull vault. These damage both internal and external tables and do not show signs of healing having sharp edges. Tuberculosis primarily damages the spine and other postcranial bones. When it occurs in the cranial vault, lesions penetrate from inside. On facial bones tuberculous lesion may take a form of porotic erosion of the external table. In the paleopathological literature we could not find descriptions of any other conditions producing changes to the cranial vault similar to what was described here. The changes on 4811 skull do not penetrate the internal table, show signs of healing including rounded borders and remodelling of diploic trabeculae, although they are not in classic forms of skull bone cavitation by treponemal infection it can be argued that their extensive number and initial healing may be related to gummatous lesions of the scalp that were healed by some medical procedure before a full penetration of the skull vault like in other cases of gentle carries sicca on the forehead.
4836: Serpiginous cavitation near the vertex

Discussion:

In skeletal remains of medieval humans, the normal range of mercury was between 10 and 100 ng-1 (0.01 and 0.1 ug/g) (Rasmussen et al. 2013). These values are much lower than our findings for bones of individuals with signs of syphilis. For bones to have high concentrations of mercury, exposure to mercury close to the time of death is needed, as Hg has a half-life in the human body of only 70 days (Baselt 2000).

Obviously, background mercury content in Egyptian soils at the sites of the cemeteries maybe different, but we do not have specific soil values for each cemetery. The mercury in Egyptian soils or sand has only been examined when areas are already suffering from heavy metal toxicity. For example, mean mercury content of 0.41ug/g was found for Allaqi Wadi Aswan (Rashed 2010), whereas mercury contamination of Mediterranean sediments around Alexandria is of a range of 0.13 to 3.0 ug/g (El-Sayed, et al., 1979). However, these cemeteries studied are a great distance from urbanized populations that cause mercury contamination to surrounding environments. Therefore, these sites should exhibit a natural level of mercury.

The natural levels of mercury in the Earth's crust are 0.02 - 0.06 ug/g (Kabata-Pendias and Mukherjee 2007). This is at best half of the level found in individuals we considered as possibly treated with mercury and equal or lower to individuals that we used as controls. It is unlikely that

mercury could have accumulated in the bones of some individual's postmortem because the only source in ancient Egypt is cinnabar, an expensive red pigment that does not seem to be used on commoners clothing or perishable decorations that could be put over the body in association with burial ceremonies.

Thus, the observed higher mercury content of the five individuals with skeletal pathologies is most likely the result of exposure to mercury shortly before they died. The analytical combination of skeletal pathologies and high levels of mercury in skeletal tissues increases the probability of diagnosis of syphilis. Since only about one third of syphilitic patients show pathological signs in bone, it is likely that some of the skeletal remains that show no signs of syphilis may also have increased levels of mercury. This may have occurred in skull 5230.

It is likely that mercury could have been used to treat other diseases producing skin lesions. Leprosy is one such disease which may leave pathological indicators on bones; however, these lesions are different from those observed on the skull vaults here (Ortner 2003). It is highly unlikely that all six individuals showing pathological indications of syphilis would have some other skin disease treated with mercury. Therefore, this study provides a strong case for the presence of syphilis amongst ancient Egyptians.

There was no diagnostic control in place to compare environmental levels of mercury. These bones were removed from its natural environment with no soil attached to be tested.

Recently the pre-Columbian existence of syphilis in the Old World, including Asia Minor has been confirmed by the study of dental signs of congenital syphilis Ioannou, et al. 2018. These authors reviewed the presence of dental signs of congenital syphilis in pre-Columbian specimens in the light of their recent findings of dental changes occurring in medically well documented cases of congenital syphilis. Specifically, the presence of congenital syphilis was confirmed in Nicaea, Turkey (Erdal et al. 2006) St. Pölten, Austria (Gaul et al 2015) Oaxaca, Mexico, (Myers et al 2009) and two cases in Metaponto, Italy (Henneber and Henneberg, 1994)

It has been argued that syphilis existed in the Mediterranean prior to 8-2 c. BCE with two cases of congenital syphilis observed in samples 320 and 306 from the Greek colony of Metaponto Italy. The case of sample 320 (Metaponto) had thickened anterior border of the fragmentary tibia suggesting possible sabre shin and dentition showed minor notches on the incisive edges of the central and lateral incisors (Henneberg and Henneberg 1994). Both samples 320 and 306 had first molars with the occlusal surface covered by pitted and crenulated enamel (Henneberg and Henneberg 1998).

Congenital syphilis was not the only form of syphilis at Metaponto. There were also signs of treponemal disease in multiple adult individuals. Of

special interest amongst those are eight cases of erosion of the skull vault with sclerotic healing of the gummatous ulcers (Henneberg and Henneberg 1998) that resemble some of the Egyptian crania studied here. However, this observation is not conclusive due to the poor preservation of the remains.

In the 13th century juvenile from Nicaea Turkey (ITK'90 56/6), the upper right first permanent molar resembles a documented 20th century case of congenital syphilis from London while the upper left central incisor resembles the Hutchinson's incisor. Both molars match descriptions and illustrations of the types of abnormalities that can occur when affected by treatments that contain mercury (Ioannou et al. 2018). The dental pathologies are further supported by Hillson et al. (1998) who state that both Moon's molars and Hutchinson's incisors are pathognomonic for congenital syphilis.

Another case that has been argued to have had syphilis and received mercury treatment is in Oplontis (near Pompeii at 79 AD). Skeletons number 2 and 41 show mulberry molars and mercuric teeth (Henneberg et al. 2006). This case has been compared with other modern cases of congenital syphilis, where patients were treated with mercury and found to be very similar (Ioannou et al. 2016). Other cases that suggest syphilis in Pompeii also exist including four skulls that exhibit minor stellate lesions (Henneberg and Henneberg 2002). These individuals are the basis of an argument that syphilis existed in the Mediterranean and that it was being

treated by mercury during the Classical Era and onwards. The current finding of increased levels of mercury in adult Egyptian skeletons showing possible skeletal signs of syphilis strengthens this argument.

The Giza Pyramid crania were dated to the fourth Dynasty c. 2650 BC. This was confirmed by examining a well-preserved skull that had a small entrance hole in the cranial vault via the cribriform plate of the ethmoid bone. This was the common practice for the removal of the brain by embalmers around this time period. The human remains in this time and area are of individuals who lived at a precise period in history, namely c. 2650 BC, and whose status in life was that of the highest in the land.

The Egyptian crania from El Kubanieh were from the Middle Kingdom. Chronology was determined via the use of relative dating of burial construction, associated artefacts (grave goods) and various historical accounts (Junker 1910). The cemetery was broken up in Early Middle and Latter period tombs. The tombs all had some sort of cover stone (differing in the type of materials used and style of tomb) and walled off areas to protect them from environmental forces (Junker 1910). This also protected the bodies from animals particularly hyenas trying to dig them up. The tombs were also positioned in sandy areas, limiting if not removing the possibility of tree roots creating taphonomic changes on the bone. The tombs may have been deep enough to protect them from insect burrowing into the tombs to eat through the bones of the individuals.

The Egyptian skull number 4811 was examined by Toldt (1919). He states that the Nr 26 (Natural History Museum 4811) skull suffered from erosion (Toldt 1919). The observed excessive erosion of this skull vault when compared with other individuals from this site is most likely the result of damage during life by gummatous lesions. These observations have not been confirmed by other authors.

Conclusion:

Prior to this study Ancient Egypt yielded no convincing pathologies that may indicate syphilis. During this study, five ancient Egyptian skulls displaying paleopathological signs possibly related to syphilis were found to have high levels of mercury, most likely originating from medical treatment during life. This evidence not only strengthens the argument that syphilis existed in ancient Egypt and the Mediterranean but also that mercury was administered as a remedy.

Syphilis in a Danish leprosarium with possible cases of co-infections

(Written as a separate article for publication)

It has been difficult to demonstrate the presence of cases of syphilis in medieval cemeteries, due to the general dating ranges provided by these cemeteries which often extends up to post Columbian times. Now there have been some cases of treponematosis that have been radiocarbon dated prior to 1493 (Mays et al. 2003). In most cases skeletal samples from the cemeteries do not provide conclusive pathological evidence to support a claim of syphilis. According to the medical literature, physicians in medieval times had problems diagnosing various skin conditions in general, leprosy and syphilis in particular.

Leprosy or Hanson's disease can manifest itself in many forms: tuberculoid, borderline and lepromatous (Bhat and Prakash 2012). Moller-Christensen examined a large number of individuals from medieval leper hospitals in Denmark, which led him to establish a series of diagnostic criteria that have continued to be invaluable for proper identification of the disease in the osseous tissue. In a paper that Dr Moller-Christensen and Faber released in 1952 coined the term "facies leprosa" this refers the particular pathologic changes that occur in the skeletal structure of the face in leprosy, on the basis of the Danish research, it was concluded that for a diagnosis of leprosy to be made that facies leprosa must be present in the osseous tissue. The reason for this occurrence is due to the fact that bone

changes in the hands and feet will often be observed with facies leprosa. 41 complete skeletons demonstrated leprosus osseous change, this occurred in virtually all of the specimens, a change in the bones of the hands and feet was observable in approximately 66% of the skeletons (Steinbock 1976:201: Moller-Christensen). The osseous change that occurs in the cranium is often restricted to the rhinomaxillary region of the face.

Keith Manchester (1984) states that "skeletal changes of leprosy are found around the oral and nasal cavities and at the limb extremities. The cranial features, the so called *facies leprosa*, consist of the progressive erosion of the alveolar process of the maxilla with the loosening and ultimate loss of the central and lateral maxillary incisor teeth. There is an associated erosion of the anterior nasal spine leading to its ultimate loss. The margins of the pyriform aperture become eroded at their lower parts. Both the nasal and the oral surfaces of the palatine process of the maxilla exhibit inflammatory changes, and there may ultimately be perforation of the hard palate" (Manchester 1984: 167).

The lower limb changes are characteristic destruction of foot bones, especially phalanges, Charcot joints in the foot or tibiotarsal joints, gross periostitis of the tibiae and fibulae, usually bilateral, and commencing at the distal ends. "There is inflammatory change in the distal foot commencing in the phalanges and metatarsals, and there may be inflammatory changes in the tarsal bones. The phalanges are lost. The metatarsals develop concentric

atrophy and become pencil shaped with the loss of medullary cavity. In the hands, the inflammatory change commences in the phalanges, spreading later to the metacarpal bones. These changes result from trauma to the anaesthetic fingers, and are sometimes associated with the claw hand deformity of leprous paralysis" (Manchester 1984:168).

As for treponemal diseases, that proves to be a real conundrum, as syphilis has not only had several names throughout history, but it also had a non-venereal strain as well as a venereal form, which caused different changes to the body. Syphilis became known as the great mimicker as it can affect human bodies both internally (bones) and externally (skin) and has similar components or characteristics as other diseases like leprosy (Rothchild 2005; Dupnik et al 2012). The historical literature also argues that during medieval times people who had leprosy and often skin conditions were administered Saracen ointment, mercury mixed with fat, to treat the disease which is quite perplexing as mercury does not cure leprosy, but it has been known to help syphilis (Steinbock 1976; Baker 1988).

Therefore, if physicians did not know whether they were treating their patients for leprosy or syphilis, then the question must be asked could syphilitic patients be interred in leprosarium cemeteries? There is evidence of this in Aaderup's Danish leprosarium cemetery where two skeletons exhibited pathological signs of syphilis.

Leprosy is a chronic bacterial infection which not only involves the skin but also nerves and other tissues. *Mycobacterium leprae* is a bacterium which can take up to 20 years to incubate without anyone knowing about it. There are only about 10% of people who are infected by someone else and those who are affected have varied clinical manifestations (Covey 2001). If treatment is not sought then the patient can become blind, with a loss of neural sensation and local paralysis.

Skeletal remains from India from approximately 2000 BC with signs of leprosy show that the disease has been known about since around that time (Robbins et al 2009). Although written documentation likely to be 'clinical leprosy' first appeared in Greece around the 3rd century BC (Anderson 1969). It has been suggested that leprosy was spread to the west by the armies of Alexander the Great in the 3rd century BC upon his return from the Indian campaign (Roberts and Manchester 1995). Further suggestions indicate that it made its way to Rome at a later date, making its way from the West Asian countries around the first century BC and then into Italy. Leprosy is then believed to have spread along the routinely travelled routes used by the armies, religious travellers and merchants of the times which spread it across the European continent becoming a common disease (Anderson 1969; Crane-Kramer 2000).

Syphilis has been argued to have existed prior to Columbus in the Old World. The oldest paleopathological evidence is from Metaponto, Pompeii

late Roman France and in Byzantine Anatolia (Erdal 2006). The existence of congenital syphilis in the pre-Columbian Old World is not in question. It has been demonstrated by Ioannou et al. (2018). However, most diseases were not well described in pre-Columbian times, whether these descriptions were made by a medical practitioner or a layman. Diseases were described by commenting on the individual signs not the disease as a whole. Therefore, it is difficult to connect a particular venereal sign with any particular venereal disease. Venereal diseases were mentioned in the Bible with some pre-Columbian researchers believing they related to syphilis (Buret 1891; Goldman 1971). However, because they are so vague it is often debated as being another type of venereal disease. The paleopathology shows a similar trend. The bones quite often reveal very little due to many factors such as healing, and only one-third of bone changes occur in syphilis and the disease needs to progress to tertiary stage for any real pathognomonic bone changes to occur for proper diagnostic purposes (Steinbock 1976). This makes it the best kind of chameleon to hide in amongst other bone changing diseases like leprosy.

It is known that syphilis can form co-infections with HIV and leprosy with tuberculosis (Agarwal et al 2000; Zetola et al 2007; Karp et al 2009). However, could syphilis be masked by leprosy through co-infections? Co-infections between treponemal disease and leprosy may be dependent on age of infection for lepers. It is more common for children to become infected with leprosy which makes it harder to contract a venereal disease. However in can take up to 20 years (with the average 3-10 years) for the

incubation of leprosy, which means that by the time symptoms appear they could already be adults able to contact venereal treponematosis (Bhat and Prakash 2012; Chaptini and Marshman 2015). In leprosy, the disease affects the testicles which eventually causes low sex drive and then impotence. In a clinical study the results showed that there was no correlation as to when this symptom begins (El-Beheiry et al. 1979). Therefore, it is possible for an individual who has leprosy to have sexual relations and become infected with syphilis. There are various models of co-infections used to determine how co-infections function (May and Nowak 1995). Models of the evolution of parasite virulence have focused on computing the evolutionary stable level of virulence favoured by tradeoffs within a host and by competition for hosts, and deriving conditions under which strains with different virulence levels can coexist. The results depend on the type of interaction between disease strains, such as single infection and co-infection.

Co-infection tends to favour higher virulence and support more coexistence than the single infection model. Co-infection would be possible between leprosy and syphilis as they both would not be competing with each other. Although syphilis is more virulent than leprosy and has different incubation rates, it is still possible for these two to coinfect if immune systems are compromised enough (Mosquera 1998). Therefore, there should be a mixture of syphilis and leprosy symptoms appearing in individuals with coinfections until syphilis becomes asymptomatic. It is known from historical sources that leprosy was mostly described accurately. However,

unless there were tradeoffs as a result of the co-infection the progression of the disease was slowed down.

Historically there have been descriptions of leprosy with both a sexual fear of transmission and the ability to pass the disease to the offspring (Whitwell 1940). Both of which are characteristics of syphilis or other sexually transmitted diseases. In syphilis the bacteria cause signs in a matter of weeks not years like leprosy and the transmission happens at a quicker rate (Newell 1966; Peeling 2006). Therefore, it can only be assumed that physicians were possibly seeing two diseases acting in the body at the same time and thought it was one disease. Therefore, when it appeared as one disease they were again confused and still called it leprosy. There is clinical evidence to support coinfections between syphilis and leprosy (Scotti et al. 1970; Nsibambi 1981; Sehgal et al. 1993; Khandelwal et al. 1994; Fonseca et al. 1999; Pandhi et al. 2005; Dupnik et al. 2012; Souza et al. 2013). There is also paleopathological evidence that supports ability for syphilis to coinfect with a mycobacterial disease like tuberculosis (Anson 2004, Ioannou et al. 2015).

Physicians before and during the medieval era diagnosed their patients based on observations and the narratives of the patients illness, they also inspected and smelled their patients excreta (Siraisi 1990). Ancient medicine relied upon a substantial emphasis of careful and detailed observations and recording of clusters of symptoms and the way these symptoms changed and developed over time as the illness progressed

(Siraisi 1990). This method worked effectively for physicians the likes of Rhazes who recognised smallpox as a disease and recorded the symptoms patients suffered. However in the cases of diseases that develop in stages such as syphilis, where symptoms appear, then disappear or change, may have made it harder for physicians to be able to recognise it as the same disease. However in modern times physicians are capable of relying on more than just observations to diagnose diseases, as now there are many clinical test that are used to ascertain what kind of bacteria or virus may be are affecting the patient.

It has been argued that leprosy was rife in medieval Europe, but it is uncertain just how common leprosy was during this period (Gussow 1989; Richards 1977; Robbins 1986). Although it is known that leprosy prevalence did not increase after the twelfth and thirteenth centuries, there was still a need to segregate lepers (Le Goff 1990; Clay 1909; Rubin 1974). The literature (Le Goff 1990; Rubin 1974) has recorded that France and Germany had around 2000 leprosaria, with England and Scotland having erected about 220 to cater for roughly 1.5 million people with leprosy (Covey 2001). Gordon (1959) acknowledged that France and Germany had approximately 10,000 leprosaria between them in 1400. However, Rogers and Muir (1946) do agree there was an increase in hospitals erected up to the thirteenth century but do not believe they all catered for patients with leprosy but instead believe people were so afraid of the disease, that history over-exaggerated its prevalence (Covey 2001).

Richards agrees with Rogers and Muir but takes it one step further by adding the hospitals were disproportionate to the number of lepers, as churches were rather inclined to erect these institutions for the sole purpose of receiving charity. Regardless of the motives for erecting these institutions the fact still remains they catered to thousands of patients throughout Europe. As physicians found it hard to distinguish between leprosy and syphilis, it stands to reason that some of the leprosy sufferers could have had syphilis. This could explain the fact that when hospitals closed, syphilis was more widely seen (Baker et al 1988).

Following the Papal proclamations in AD 1490 and 1505 leper houses were abolished due to vast decline in leprosy. This not only released lepers into communities but also syphilis patients (Holcomb 1935). With syphilis becoming better known and, on the increase, the decline of leprosy proper occurred. This statement does coincide with the time leprosy peaked between AD 1100 and 1300 when the crusades were in full swing leaving diseases in their wake (Rubin 1974). Leprosy eventually disappeared in Europe by the mid sixteenth century, with the exception of Scotland and Scandinavia. During the mid-sixteenth century syphilis was so wide-spread physicians were able to describe it in detail (Fracastoro 1930). By this time the printing press had been used widely making it easy for physicians to exchange syphilitic information among themselves. Later Fracastoro's name for the disease known as "syphilis" was generally accepted by the 19th century (Crosby 1968).

It has been noted that leprosy is not very contagious, “prevalence rates in endemic areas range from one per 1000 to 50 per 1000 (Noordeen 1998). The global incidence rates between 1985 and 1997 varying between 550,000 and 700,000 cases. Two major peaks were reported in 1998 and 2001; this last one with 763,262 new cases registered. Since 2001 there has been a steady decrease with 259,017 cases reported in 2006” (Noto and Nunzi 2008:124). Yet in approximately 200 years, hundreds of buildings had been constructed around Europe, specifically to separate and treat thousands of people who had been afflicted by this disease (Jopling 1978; CDC 2017). This scenario does not appear to be describing a low impact disease, but rather a virulent one, with the capacity to spread across Europe continuously infecting individuals and attaining high population infection rates.

There have been clinical examinations of patients that show signs of having both leprosy and syphilis. When assessing patients for either leprosy or syphilis it is important to know at times how similar they can be. In Hanson’s disease there are two kinds of complications that can arise, one is reversal reaction and the other is erythema nodosum leprosum (ENL) (Kahawita et al 2008). In Hanson’s disease only one of the conditions will occur in a patient or it is possible for both conditions to occur at seperate times in the same patient. These conditions of Hanson’s disease can develop signs and symptoms consistent with syphilis. These signs are rhinopharyngitis mutilans, skin rashes and peripheral neuropathy associated with ulcerations to the extremities (Murray 1982). As far as reactional

Hanson`s disease is concerned, its signs keratitis, uveitis and orchitis also occur in syphilis. Murray (1982) also believed both diseases could be associated with Charcot joints of the lower extremities but atrophy of the optic disc is caused only by syphilis (Murray 1982). If a person had ENL then it would be easy for extragenital syphilitic lesions to be masked by active lepromatous leprosy. However, leprosy and syphilis also may produce some very different features specific to each disease.

There are pathological similarities between both syphilis and leprosy which for a long time has often caused clinical diagnostic confusion between the two diseases. If these diseases weren't confusing enough with their own similar clinical manifestations confusing researchers and physicians alike how much harder would it be if it was possible for these diseases to coinfect? In 1982 Murray reviewed four years of serological data finding sixty patients from Carville who were true positive serological reactors (Murray 1982). These patients had both Hanson`s disease and syphilis with sero-activity to both the FTA and RPR-ABS tests. A few of these people who did not receive treatment, under-went the above-mentioned tests and were considered to be false positive reactors however, after further assessment they showed signs of tertiary syphilis. "It is clear that syphilis appears more commonly in HD [Hanson's disease] patients than in the general population, with rates higher than the 30 cases per 100,000 cited for the U.S. at large" (Murray 1982: 156).

Literature confusion:

Naturally, this debate covers a wide range of topics involving the history of diseases which encompasses: the pathocenosis, that is the distribution of diseases in the medieval world; the difficulty in obtaining compelling evidence from the often vague descriptions of diseases written in the distant past; the state of medical knowledge at the time and the confusion between syphilis and other sexually transmitted diseases such as gonorrhoea; as well as the possibility that the diseases diagnosed as syphilis may have been sometimes misdiagnosed as leprosy in previous eras.

True leprosy entered the sphere of western medical science only about 300BC., when physicians of Alexandria became acquainted with its lepromatous form and named it elephas or elephantiasis, because of the thickening and corrugation of the skin. The other forms of leprosy, particularly the tuberculoid type, were not clearly distinguished from other, nonspecific skin eruptions. Even Galen, in the second century AD, inadequately described what he called elephantiasis graecorum and lepra (Dols 1979).

True leprosy, a disease which disfigures the body, surfaced in Alexandria around 300BC. Dols (1979) states that physicians from Alexandria named

the disease Elephas (Dols 1979). Furthermore, according to MacArthur (1953) medieval leprosy came from the Greek word lepra which meant scaly skin. The Greeks used lepra as a common word for a group of diseases of the psoriasis type. As lepra became the accepted word for leprosy it then encompassed a host of skin conditions which were associated with scales or scabs but not true leprosy (MacArthur 1953; Dols 1979).

Aretaeus of Cappadocia gave a good description of the leonine look of a leper's face, which is caused by loss of eyebrows as well as toughening and swelling of the face. Elephantiasis took a long time to develop and was also known as Satyriasis owing to the red hue of the cheeks and an urge for intercourse (Dols 1979), a belief dating to medieval times. Also, Aretaeus noticed the appearance of the skin was similar to that of an elephant; the prominent signs being ulcerations and mutilations (Dols 1979). Aëtius states that men have a better chance of contracting leprosy than women and he also believed that elephantiasis had a strong venereal connection. Dols 1979).

Both Paul of Aegina and Aretaeus believed that leprosy was incurable when it was in an advanced stage. When patients were in their early stages of leprosy Paul treated them using Aretaeus method and believed they should be removed from populated areas to avoid infecting others, due to the contagious nature of the disease. It was also a popular belief that lepers

were unclean people both bodily and morally (Dols 1979). These were religious thoughts and practices that were imprinted on leprosy through incorrect translations, that have nothing to do with the disease itself (Zias 1989; Crane-Kramer 2000).

In understanding diseases like leprosy, the Arabic physicians were more advanced than the Europeans. When it came to leprosy the Arabic physicians did not agree with medieval European doctors as the Arabs regarded leprosy as a low contagious disease with no link to illicit sexual intercourse (Dols 1979).

In medieval Europe venereal syphilis is believed to be connected to sin as it is transmitted by sexual contact. A person with syphilis can not only infect their partner through sexual contact but also an unborn child. Bernard de Gordon believed sexual contact with a leprous woman should be avoided. According to Astruc Theodoric a physician in 1290, believed lepra had a venereal nature. In 1303 A.D. Bernard de Gordon, an early European writer, agreed with Theodoric and went on to report that at that time lepra was rampant and highly contagious with a short incubation period (Gordon 1491; Whitwell 1940). He also stated that children born to lepers had lepra. John of Gaddesden wrote a book entitled `Rosa Anglica` in which he mentioned people contracted leprosy from coitus and administered medicine to prevent the infection. Gaddesden stated that the symptoms initially appeared at the point of inoculation, followed by scabs and ulcers appearing all over the body. He also tried different remedies to alleviate the

condition without success until he added mercury to the mix (Cholmeley 1912). However, it is syphilis, not leprosy, which responds to mercury and his description of the disease resembled syphilis.

According to Classical and Medieval doctors, intercourse was the cause of leprosy and elephantiasis along with an increased interest in sex. However around 50% of leprosy cases relate to the occurrence of testicular atrophy, with testicular involvement rising to 90% in lepromatous cases (Achdiat et al. 2018). When there is an imbalance of testosterone and estrogen, testicular atrophy occurs which in turn causes a number of clinical manifestations including gynaecomastia, erectile dysfunction or impotence, infertility as well as female distribution of pubic hair (Achdiat et al. 2018). As some lepers were impotent, they would not have any interest in engaging in intercourse. This being the case, any form of leprosy would not be hereditary. If these doctors were describing leprosy, then it is certainly different from the leprosy known today.

One of the causes for confusion in the literature regarding the diagnosis of syphilis and leprosy could be linked to the possibility of these two diseases co-infecting individuals. Even in today's society people can at any given time be infected with multiple diseases. If this can occur in today's society then why not back then? It is known that leprosy is an immunosuppressant disease. This is caused by M. *leprae* reducing the responsiveness of the T-cells (de Souza Sales et. al 2011). Therefore, it is possible for other diseases to infiltrate the leprosy patient once the immune system is compromised.

Mercury as treatment

Mercury is viewed as a powerful anti-mitotic and anti-inflammatory agent (O'Shea 1990), when applied locally it aided healing. Mercury has been noted to induce a Herxheimer reaction and to clear cutaneous lesions of spirochetes that may have affected bones of the cranial vault. The systemic use of mercury e.g. through inhalations, is capable of reducing treponemal infection to the level of sero-negativity (O'Shea 1990).

It is known that mercury can cure syphilis but not leprosy. The reason for this is that mercury interferes with the glycolysis which starves the treponema of energy because it relies on anaerobic breathing (LaFond and Lukehart 2006; Officioso et al. 2016). *Mycrobactiurum leprae*, however, relies on aerobic energy production supported partly by the host's organism. Mercury does not block aerobic breathing, using substrates other than glucose.

Mercury has been used to treat congenital syphilis during ancient Roman times. In Oplontis (near Pompeii at AD79) the skeletons number 2 and 41 showed mulberry molars and mercury teeth (Henneberg and Henneberg 2006; Ioannou et al. 2018). Sir Jonathan Hutchinson (1887) described in detail the effects of mercury in congenital syphilis on teeth. This case has

been compared with other modern cases of congenital syphilis, where patients were treated with mercury (Ioannou et al. 2015).

Rasmussen's work with mercury levels in diagnosed syphilis and leprosy bones, suggests that in syphilis the mercury was working and healing the infected patients. However, in leprosy the mercury concentrations are more constant in the range of 100 to 400ng/g (Rasmussen 2008). The reason for this is that mercury did not heal leprosy and the lepers continued to use the medicine in vain hopes they would be cured until they died. However due to mercury being an ineffective treatment for leprosy it was not widely used.

An examination of the potential variation in mercury concentrations between leprosy and syphilitic individuals was carried out at the University of Adelaide. This study sought to identify more consistent mercury concentrations within possible syphilitic individuals. The individuals who had leprosy should exhibit a greater variation, as they could have chosen to discontinue the treatment due to mercury`s inability to cure leprosy.

Materials

The Danish leprosarium at Aaderup, in Nastved was in use from AD 1300 to 1550 A.D (Møller-Christensen and Weiss 1971). In 1948 Vilhelm Moller-Christensen spent twenty years excavating a hospital, unearthing the remains of 750 people. He then examined their remains finding a number of them had leprosy. Moller-Christensen was a good palaeopathologist who would have been able to recognize signs of syphilis, however he never mentioned any cases. These remains from Naestyed can be found at the Medical Museum at the University of Copenhagen.

The discoveries at Æbelholt prompted the excavation of the cemetery at Næstved (1948–1968). Records first mention the leper hospital at Næstved during 1261 and ends in 1542 when it closed. The leper hospital only treated lepers the whole time it was open. It was Ribe Recess who ordered the closure of all leprosy hospitals in Denmark, their patients going to larger hospitals. (Moller-Christensen 1961; Anderson 1969).

Methods:

The University of Copenhagen houses a collection of 300 skeletons excavated from a leprosarium. The leprosarium is dated from 1300 to 1500 A.D. The diagnostic criteria used were presence of caries sicca on skulls and pathological changes to the long bones preferably concentrated on the midshaft. If pathological changes were seen only in the hands and feet, the diagnosis of syphilis was not made. If any possible signs on the face, likely being facies leprosa no diagnosis of syphilis was made. The primary objective was to find syphilis in a leprosarium, the criteria were designed to concentrate on differential diagnosis of syphilis over leprosy. This was done to compare mercury contents in skeletons that were possibly of syphilitic patients with hose of lepers. The assumption was that ineffective in the case of leprosy treatments with mercury were not continued in patients, while effective use of mercury against syphilis would encourage longer use of mercury, thus higher doses that may accumulate in skeletons. Mercury concentrations were compared with twenty-five leprosy samples, however only eight have been analysed. All samples chosen for analysis were taken from the ribs as Rasmussen believed the ribs hold a higher concentration of mercury than the majority of other bones. The ribs were analysed by Laser Ablation inductively coupled with plasma mass spectrometry (LA-ICP-MS) from the University of Adelaide.

The laser ablation ICP-MS system is used for micro sampling of solid material for trace elements. It consists of a 213nm Nd:YAG New Wave

pulsed solid state laser (NWR213 New Wave) coupled to an Agilent 7900x ICP-Quadrupole Mass Spectrometer. Detection limits reach into the ppb range allowing for true trace element analysis for a wide variety of solid material, including geological and biological samples. All samples were washed in alcohol and then placed onto a slide. All samples were placed in the LA-ICP-MS chamber so that the laser beam hit the surface of each bone sample. To properly calculate the amount of mercury in each sample an internal reference provided by the manufacturer was used.

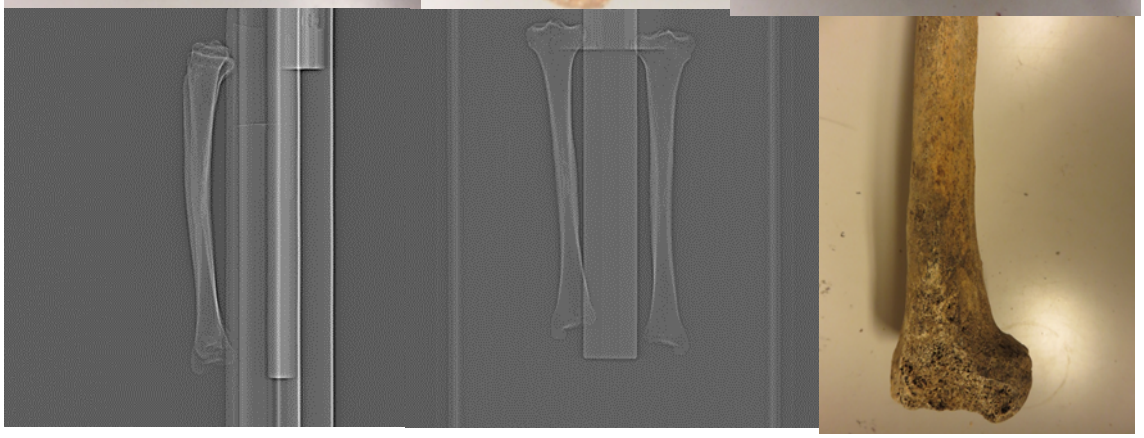
Results:

Table 1: Mercury concentrations in bones from Aaderup Leprosarium

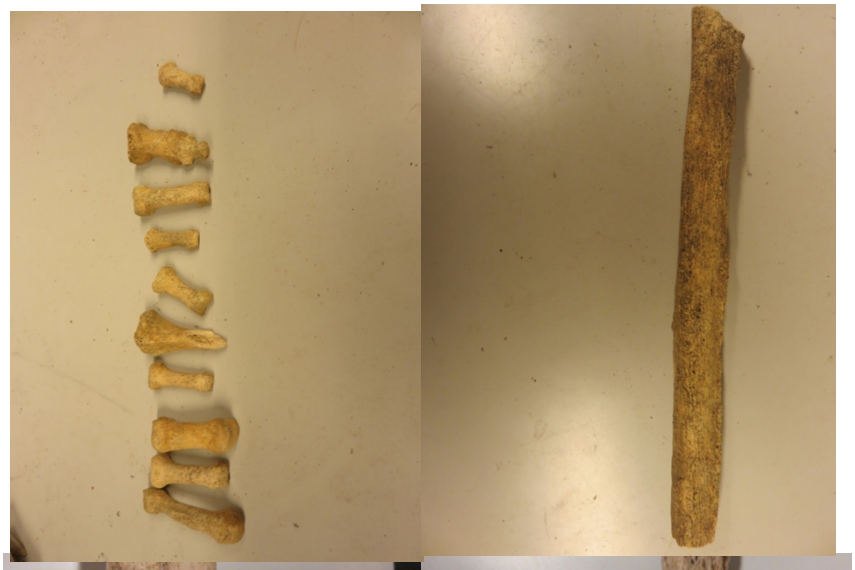
Site name/number	Age	Sex	Mercury content (ppm)	Possible Syphilis	Possible Leprosy
Aaderup 80	Adult	F	0.46	Syphilis?	
Aaderup 399	2 years	?	1.78	Syphilis?	
Aaderup 205	Adult	M	2.50	Syphilis	Leprosy
Aaderup 396	Adult	M	0.17	Syphilis	
Aaderup 314	Adult	?	0.16	?	?
Aaderup 56	Adult	?	0.14	?	?
Aaderup 42	Adult	?	0.21	Syphilis?	
Aaderup 39	Adult	?	0.03		Leprosy
Aaderup 389	Adult	?	0.03	?	?
Aaderup 392	Sub Adult	F	0.23	?	
Aaderup 287	Adult	?	1.41	?	?
Aaderup 542	Child	F	0.49	?	?
Aaderup 288	Adult	?	0.45		Leprosy
Aaderup 5B	Adult	?	0.13	?	?

Aaderup 80

Aaderup 399



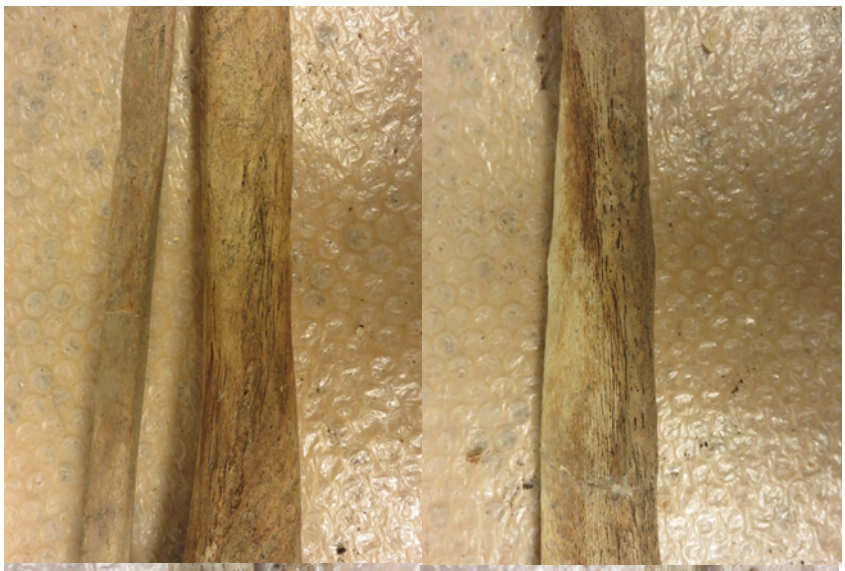
Aaderup 205



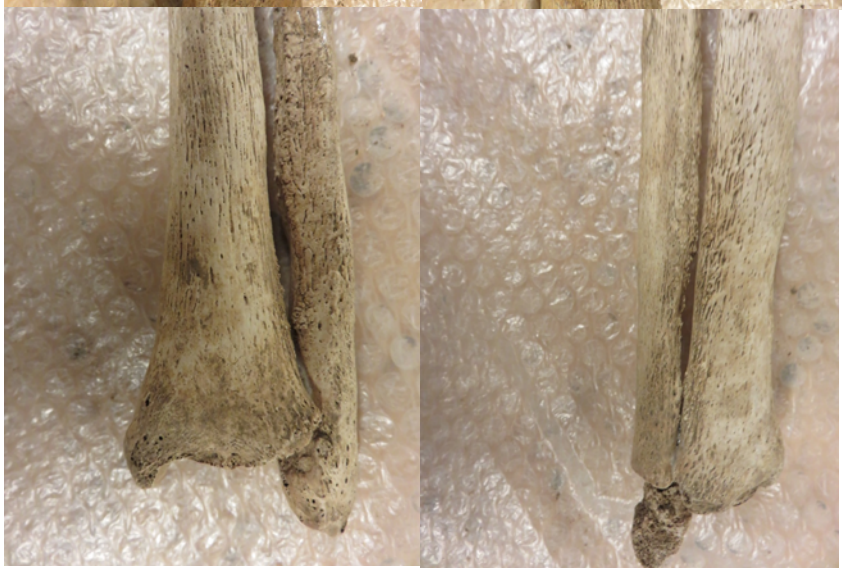
Aaderup 396

Aaderup 314

Aaderup 56

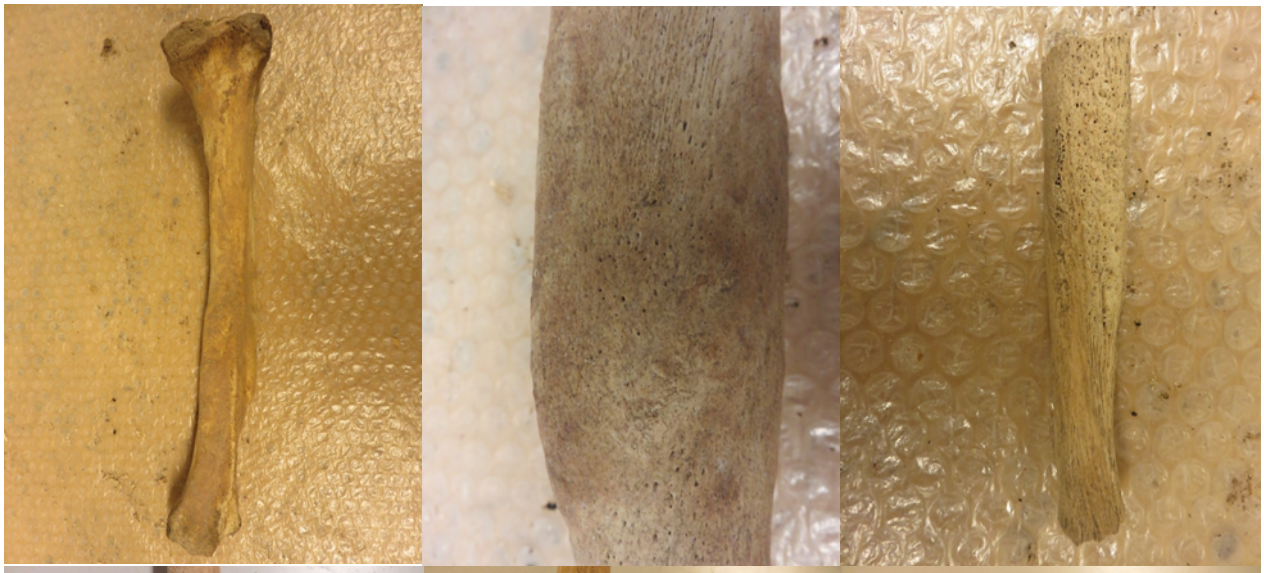


Aaderup 42



Aaderup 39

Aaderup 389



Aaderup 392

Aaderup 287


Aaderup 542



Figure 1: Leprosy and syphilis changes in skeletal remains

Aaderup 80: Left tibia has periosteal striations around mid-shaft and the right tibia has minor striations around the mid shaft together with some defects that look taphonomic. No signs of facies leprosa on the cranium.

Aaderup 399 Child's cranium shows lesions that might be periostitis or osteomyelitis, however, most possibly are taphonomic. On the midshaft of the right tibia there is an oval shaped lesion

Aaderup 205 Sabre tibia on left tibia. On the left tibia striations on the mid shaft and lesion at the head, this could be taphonomic. On the right tibia striations and periostitis on the midshaft. On the posterior end of the left femur there are lesions and some signs of healing. Signs of leprosy are noted on the tarsal bones

Aaderup 396 There is a inflammatory change with possible Sequestration at the proximal part of the right tibia with striations occurring along the shaft. The left tibia has no distinct pathological changes. The fibula at the distal end shows signs of periostitis which run up to the mid shaft. The other fibula shows signs of periosteal thickening

Aaderup 314 The right tibia has periostitis striations along the mid shaft. On the left tibia there is a severe periosteal reaction and rugose new bone.

Aaderup 56 The right tibia around the midshaft striations and periostitis. The right fibula around the lower midshaft is periostitis. On the left tibia around the midshaft there are striations and new bone growth forming distinct edging (lipping)

Aaderup 42 Right tibia shows signs of deep striations that are forming grooved cavities in the midshaft. There is also a small amount of striations at the proximal end of tibia. On the upper midshaft of the tibia there are more striations which cause a groove in the tibia. The right fibula is showing signs of periostitis. According to Hackett's criteria for syphilis this tibia also shows signs of nodes with rugose surface pattern (Hackett 1976). On the left tibia there is periosteal thickening occurring. There is also striation happening along the shaft, more concentrated severe at the top of the tibia down to the midshaft and not as bad on the distal end of the tibia. There are signs of periostitis striations.

Aaderup 39 The left tibia shows signs of striations on the distal end, which run up to the midshaft. The midshaft has extra layer of sub periosteal bone where it is trying to heal over the striations. The left fibula has extensive new bone growth on the distal end, and in the midshaft. The right tibia shows signs of striations on the distal end. In the midshaft there are signs of bone growth in response to healing. The right fibula shows signs of bone growth on the distal end and midshaft.

Aaderup 389 Right complete tibia has signs of osteitis in the middle, the left fragmentary tibia shows signs of ostitis too.

Aaderup 392 Right Tibia (from picture) is showing deep striations on the distal end and on the other side there are striations from the midshaft to the top end of the tibia. In the distal one third there is a small amount of periostitis. The other tibia not shown has a small amount of striations on the midshaft. Both fibulae have striations on the midshaft.

Aaderup 287 Both fibulae are showing signs of striations.

Aaderup 542 Left tibia has some striations from the distal end to the midshaft, there is also periostitis and reactive bone growth. The right tibia shows signs of clustered lesions on the midshaft.

Aaderup 288 On the left tibia shows signs of striations and reactive bone growth. The bone growth is more reactive in the midshaft. The left fibula shows signs of severe reactive bone growth on the distal end working its way up to the head where the bone growth is not as severe. The right tibia shows signs of striations mostly focused around the midshaft. The right fibula shows signs of striations on the distal end.

Aaderup 5B The left tibia shows signs of bone remodeling, that have distorted its shape.

Aaderup 312 this individual shows signs of knee Charcot joint on the right femur and tibia. Moller Christensen describes 312 skull as having atrophy of the anterior nasal spine (ANS). Atrophy of the alveolar process of the maxillae (APM) and inflammatory changes of the palatine process of maxilla (PPMN) and of the right inferior nasal concha. There are also inflammatory changes of the Palatine process of maxilla (PPMO) especially in the anterior part. The left tibia shows signs of periostitis changes along the midshaft. The fibula shows signs of reactive bone growth.

Discussion:

One of the main reasons why Møller Chriatiansen did not record the existence of possible cases of syphilis within the leprosarium at Aaderup may have been due to mild non pathognomonic signs of infection, but stronger signs of leprosy. One example of this is Aaderup 312. Møller Christiansen describes skull changes as having atrophy of the anterior nasal spine (ANS). Atrophy of the alveolar process of the maxillae (APM) and inflammatory changes the right inferior nasal bone and of the palatine process of maxilla especially in the anterior part (Møller-Christensen 1978). He only states that there were postcranial changes of the hands, tibiae, fibulae, and feet. However, what is not mentioned is the Charcot joint in the knee, which is more characteristic of syphilis than leprosy. In leprosy Charcot joints occur primarily in between the distal end of the tibia and the foot. There were also signs of bone changes occurring in the midshaft of the tibia which again is more characteristic of syphilis.

Another good case in this collection is Aaderup 205 who has some signs of treponematosis (Yaws or congenital). These signs include sabre tibia and striations on the left tibia, lesion on the distal end of the femur, On the right tibia striations and periostitis on the midshaft. However on one tarsal bone there is signs of leprosy thinning the bone. Since both leprosy most commonly infects children more than adults and both yaws and congenital syphilis occur in children it is possible that this individual was coinfected with these diseases.

Obviously not everyone infected with leprosy will have conclusive evidence of being coinfected with syphilis. However, given the obvious medieval confusion between syphilis and leprosy, in that leprosy could be sexually transmitted then it must be presumed that syphilis and leprosy had been coinfected enough for medical practitioners to be confused with their understanding of the disease. This means that within leprosarium the chances of finding cross of pathological signs in skeletal remains increases.

However, in lepromatous leprosy the changes of getting gynaecomastia which is the lowering of testosterone levels result in decreased libido, impotence, changes in secondary sexual characteristics, and disturbance of spermatogenesis leading to infertility (Achdiat et al 2018). Therefore, if an individual becomes coinfected then there is a chance that due to reduced libido in some cases of leprosy, the syphilis side would not be able to increase in severity of the disease due to the inability to become reinfected. This means that if any changes occur in the skeleton there will be a higher chance of it only being mild.

Within the Aaderup collection that was examined, there were cases in which there were bilateral changes on the tibia and also on the fibula. In leprosy due to the infection that occurs by damage sustained by the outer surface of the body, external bacteria eventually start causing inflammation in the bones (Andersen et al. 1994). The lesions are found starting from the distal ends of the bones and working their way up (more severe changes

always are at the distal ends). This is why in leprosy bone changes are predominantly unilateral because it is the external bacteria through the open wounds that cause the bone changes.

Due to the nature of coinfections it would be hard to distinguish between the two diseases and separate them if need be. This can be seen with Aaderup 205 who shows signs of sabre tibia, this could either be due to non-venereal syphilis or congenital syphilis. Regardless, it is not a typical trait of leprosy, also this individual was not showing any real signs of leprosy.

Some of the individuals clearly show pathological markers suggestive of syphilis, the question is what were these individuals doing in the leprosarium? Was there confusion with diagnosing these diseases as the literature seems to suggest or were individuals an example of quarantine before the Pope chose to close down the leprosaria?

The Pope was closing the leprosaria between 1490 and 1505 and people were contracting syphilis around the Siege of Naples in 1495 (Baker et al. 1988). These Leprosaria were closed due to a lack of lepers without which there was no reason to keep them open. Therefore syphilitic individuals could only seek refuge in the leprosarium for ten years. Even so, there was more than enough time for syphilitic patients to die and be buried in a lepers' cemetery. However, this does not explain the obvious misinterpretation of the progression and infectious nature of leprosy.

Medieval medical writers were under the misapprehension that leprosy was a sexually transmitted, hereditable and highly virulent disease which required mercury to alleviate the symptoms (Whitwell 1972). However these writers were describing the symptoms and medication for syphilis not leprosy.

The mercury analysis of the possible syphilitic individuals and leprosy individuals shows higher than average mercury concentrations in all samples. This means that syphilitic individuals were treated the same as leprosy patients, however, their treatments may not have lasted for as long.

In this analysis only skeletal materials were tested. All skeletal remains of each individual were individually stored in cardboard boxes where there was no original soil from the site to be tested as a control.

In Danish medieval humans the normal range of mercury was between 10 and 100 ng-1 (0.01 and 0.1 ug/g) (Rasmussen et al. 2013) . The bones analysed showed a range of 0.43 to 2.80ppm, this is well over the normal range for medieval Danish. For bones to have high concentrations of mercury, exposure to mercury close to the time of death is needed, as Hg has a half-life in the human body of only 70 days (Baselt 2000).

In the Aaderup collection the samples that were taken for analysis of LA-ICP-MS to provide scientific quantitative analysis of the concentrations of mercury within the individuals bones proved to be ineffective to separate leprosy from syphilis. The results provide a wide range of variations between the highest and lowest values. The highest was 2.50ppm and the lowest 0.03ppm. Aaderup 39 and 389 have both low mercury concentrations. They both fall under the normal range of mercury within the human body. This means that before they died these two individuals stopped using mercury as medicine for their disease or have never used mercury. In the case of Aaderup 3 signs of leprosy are shown on the skeleton. It appears this individual gave up on mercury as it was not helping. The results show that despite the fact that mercury does not cure or aid in the recovery of leprosy, the afflicted certainly kept on using mercury in the hope that they would be cured. Therefore, it is difficult to assess the diagnosis of individuals through pathological changes in order to help differentiate from leprosy by using mercury concentrations.

It is difficult at this time to ascertain whether these possible syphilitic remains are pre-Columbian in origin as their dating was a group cemetery dating, not individual. However, we can state that some of these individuals (205, 312) did carry pathological characteristics of syphilis. It can also be stated that these individuals were treated with mercury and later died with still a higher than normal trace of mercury in their bones.

This investigation into the possibility of syphilis in a leprosarium was a pilot study. Time spent investigating the pathologies on the Aaderup collection was time limited therefore a more detailed analysis should be undertaken to strengthen this argument.

Syphilis, The Hidden Disease

(Written as a separate article for publication)

Introduction:

Syphilis is a systematic disease that affects multiple tissue forms (areas of the body), except when the disease turns asymptomatic. It is known that only one third of patients will under-go bone changes (Steinbock 1976). Bone changes may not occur if the patient receives treatment prior to secondary or tertiary stage syphilis. Therefore if there are no signs of pathological changes to the bones or only limited changes, it is important to either find treatment for the disease or to locate treponemal DNA within the bones (Bouwman and Brown 2005; Von Hunnius et al 2007).

Finding evidence for DNA of treponema in bone over several hundred years is difficult due to degradation of the coding (Bouwman and Brown 2005; Von Hunnius et al 2007). The strands of DNA therefore are incomplete and only some parts can be compared to the full treponema genome. This method thus becomes inaccurate and cannot be used to support the presence of syphilis in asymptomatic cases. However, there is one way to support the evidence of syphilis existing in the Old World, and that is to locate the presence of mercury used by physicians since Roman times (Rasmussen et al 2008; Kępa et al 2012; Rasmussen et al 2013) The evidence supporting this statement occurred in the osteological remains of

congenital syphilitic twins from Oplontis Italy (Henneberg and Henneberg 2006; Ioannou et al 2018).

There were many differences in early medical practices for the treatment of syphilis. One important difference was the use of an anti-bacterial medicine which contained mercury, administered in the Old World but not in the New World. According to medical literature written in the 19th century (Goldwater 1972), mercury was known to have reduced and effectively controlled treponemal infection. It has been argued that mercury was used in medicine as early as the Ancient Egyptians.

The relative lack in Old World of human remains showing advanced signs of syphilis could be related to the medicinal use of mercury. Mercury is viewed as a powerful anti-mitotic and anti-inflammatory agent (O'Shea 1990). When mercury was locally applied it aided healing. It has been noted to induce a Herxheimer reaction and to clear cutaneous lesions of spirochetes that may have affected bones of the cranial vault (O'Shea 1990). The systemic use of mercury e.g. through inhalations, is capable of reducing treponemal infection to the level of sero-negativity. This is the reason why the severity of the disease seems to have dissipated with more people obtaining the treatment of mercury during the late 16th - 17th centuries. Mercury may have played a part in reducing the spread of pathological signs to bones as it cured ulcerations, however the host population would have been gaining some resistance to the disease itself that reduced severity of skeletal pathologies.

However, mercury was known to have treated other diseases like leprosy and other skin diseases (scabies) (Norm et al 2008). This causes greater confusion when dealing with diagnosing asymptomatic syphilis in skeletal remains, as most skin diseases leave no trace on the bones.

Methods:

The aim of this study is to examine the potential impact that mercury treatment made on the prevention of the development of syphilitic skeletal lesions. This study also seeks to identify excessive levels of mercury, in some Medieval Polish skeletal remains that possess limited pathological signs suggestive of syphilis. The lack of pathology in limited skeletal material can also indicate either a skin disease that does not leave signs on bones or accidental, possibly work related exposure to mercury in the individuals life time. A comparison will be made of the mercury levels of the patients who do not show such changes.

In the University of Łódź in Poland two skeletal collections were used for this study. These collections of skeletal remains are known as Brześć Kujawski (BK5) and Kolonia and are pre-Columbian in origin. These skeletons were unearthed in the area of Brześć Kujawski, Kujawy, north central Poland. The archaeological site is dated from 4600BCE to the early 19th century. The period the skeletons that were examined came from is dated to the 11th to the 13th century (Lorkiewicz et al. 2018). After observing these remains for possible pathology which could indicate

syphilis, 8 remains from the Kolonia collection and 14 from the BK5 collection were chosen for mercury analysis.

Mercury is released from the body through decomposition and according to Rasmunssen (2013) mercury is then re-absorbed into the ribs (Rasmussen 2013). This process is caused by an excess of mercury stored in the body fat and certain organs like kidneys or liver. Bearing this in mind, samples were taken from either the ribs or femur. The femur was chosen when there were no ribs in the individual skeletal remains.

The femur and rib samples were crushed into a fine powder with a weight of 0.1g. These powdered bone samples were then stored in sample containers before being sent to the University of Warsaw at the Laboratorium Biogeochemii i Ochrony Środowiska (Laboratory of Biogeochemistry and Environmental Protection) for mercury analysis by the use of a mercury analyser called a Milestone DMA 80.

Results

Table 1: Indicates all samples from Kolonia were of a normal range for mercury in the body as were samples from BK5, with the exception of BK5 - 18

Nr próbki	Nazwa	[Hg] µg/kg	PPM
1	KOL1 sample0	10.1	0.0101
2	KOL-36	16.8	0.0168
3	KOL-43	16.6	0.0166
4	KOL-25	58.4	0.0584
5	KOL-29	17.3	0.0173
6	KOL-30	12.3	0.0123
7	KOL-52	33.6	0.0336
8	KOL-54	12.3	0.0123
9	BK5-8 sample 0	16.4	0.0164
10	BK5-24	13.5	0.0135
11	BK5-175	43.3	0.0433
12	BK5-179	45.4	0.0454
13	BK5-152	18.5	0.0185
14	BK5-87	21.8	0.0218

15	BK5-56	16	0.016
16	BK5-18	212.1	0.2121
17	BK5-35	19	0.019
18	BK5-9	25.5	0.0255
19	BK5-163	14.9	0.0149
20	BK5-77	14.9	0.0149
21	BK5-43	18.1	0.0181
22	BK5-74	36.4	0.0364

Table 1: Soil samples taken from 2 BK5 skeletons and 2 Kolonia show that soil mercury levels are very low

Number	**Collection**	Result [mg/kg]	Error [mg/kg]
1	BK5-74	0,1051	0,0074
2	BK5-179	0,3802	0,0266
3	Kol-52	0,3409	0,0239
4	Kol-25	0,0259	0,0018

Figure 1BK5-18 severe taphonomic changes to bones

Discussion:

In the Łódź collections of BK-5 and Kolonia both dated to 11th to the 13th century which makes them early medieval cemeteries, there were no observed obvious pathology indicative of syphilis. However, one skeleton BK5 -18 had a mercury concentration of 0.221ppm which was significantly higher than any of the 22 bones sampled. As this skeleton of a 7 year old did not exhibit any clear signs of syphilis this particular individual's condition is uncertain. What is clear is that either through medication or by accident the individual at some point during life came in contact with large doses of mercury which were deposited and preserved in the bones.

Danish medieval skeletons exhibit a normal range of mercury between 10 and 100 ng-1 (0.01 and 0.1 ug/g) (Rasmussen et al 2013). For bones to have high concentrations of mercury, exposure to mercury close to the time of death is needed, as Hg has a half-life in the human body of only 70 days (Baselt 2000). Thus, it is possible that BK5- 18 may have been exposed to mercury until the time of death. Since only about one third of syphilitic patients show pathological signs in bone, it is likely that some skeletal remains that show no signs of syphilis may have increased levels of mercury due to syphilis treatment. This may have occurred in skull BK5-18.

The mercury soil levels of the four skeletons provide evidence suggesting a range of a small amount of leaching to none at all. As the mercury levels in the bones were fairly low, it is therefore unlikely there was any soil contamination of the bones.

Even though the only pathological mark on this skeleton was horizontal striations across the forehead, which could not be confused with taphonomy it could still be compared with known cases of post Columbian congenital syphilis. However, with no indication of Hutchinson`s Incisors, Mulberry Molars or any other clear indicators of the disease, it cannot be seriously considered as syphilis.

Conclusion

It is clear that BK5-18 was taking mercury as medication for some disease. The reason for this is that to retain high concentrations of mercury in the body repeated exposure is necessary. In Poland there are no mercury/cinnabar mines so it is unlikely to have been work related. If it was a disease it could not have been leprosy as leprosy attacks the nervous system first, then the skin (lesions) and last the bones. It may have been syphilis, however, it is impossible to demonstrate as it would have been asymptomatic and therefore no pathology present to diagnose. Mercury was also used for other skin diseases, most of these diseases do not leave any traces in bones.

As syphilis can be asymptomatic and does not always attack the skeletal system, any diagnosis of bones (or any conclusions drawn following an examination of bones) is not definitive, even when it is coupled with mercury analysis.

Systemic disease found in Egyptian mummy: Possible case of syphilis

(Written as a separate article)

Introduction:

According to the historical accounts syphilis is a disease that has been argued to be around for centuries (Buret 1895; Holcomb 1937; Hudson 1961; Hackett 1963). It is a systemic disease, meaning the bacteria treponema attack multiple parts of the body. Each stage of syphilis has a corresponding sign, allowing physicians to diagnose the particular stage of the disease. These signs proved rather confusing to ancient and classical medical writers who often classified each sign as a different disease. Difficulties arise when comparing the views of a modern physician in relation to the various signs of a particular disease, as opposed to the views of a classical physician.

It was well known that Egyptians suffered from a variety of skin diseases, and both Herodotus and Pliny the Elder clearly believed they were an authority on the matter. It was also Pliny the Elder who mentioned a new disease called mentagra which was introduced to Italy by a knight from Asia Minor (Rackham 1947). Mentagra has connections to both syphilis and Egypt. Martial believed the prostitutes who came to Rome from Syria and Egypt were responsible for what he referred to as 'Syrian tumours,' an expression referring to genital lesions received after sexual contact (Martial

1871). Pliny referred to mentagra as a contagion which required Rome to import skilled physicians from Egypt to treat it (Rackham 1947). According to Herodotus Egypt had many specialist physicians who each focused on one area of the body (Wilson 1962). The skin disease specialists were in great demand, leaving Egypt for other places, endeavouring to stem the spread of this rampant disease. Mentagra or the 'chin disease' originated from a euphemism for the phallus and the little chin or mentula was the pubis which referred to genitalia (Hudson 1961:554). This disease initially appeared on the penis before attacking the face, neck, chest and hands.

The continual use of mercury for skin diseases like syphilis was a popular treatment from the Egyptian period through to medieval times and even through to the early 20th century (Goldwater 1972; O'Shea 1990). According to Pliny the Elder and Celsus physicians prescribed mercury to treat ulcerations in Roman times (Grieve 1818; Rackham 1947). During the medieval times when leprosy was rife mercury was sometimes used as a treatment, however it did not aid in curing the disease. When syphilis became an epidemic throughout Europe after the siege of Naples in AD 1495 mercury was used again. By the early 20th century physicians discovered arsenic and then penicillin to treat syphilis. According to Rackham (1952) who translated Pliny the Elder's work "the Natural Histories", mercury was used externally at the time of Pliny who stated "As cinnabar and red lead are admitted to be poisons, all the current instructions on the subject of its employment for medicinal purposes are in my opinion

decidedly risky, except perhaps that its application to the head or stomach arrests haemorrhage, provided it does not find access to the vital organs or come in contact with a lesion. In any other way for my part I would not recommend its employment" (Rackham 1952:95).

The Ancient Egyptians were aware of diseases described and recorded on Papyrus, now known as the Ebers Papyrus. The Ebers Papyrus was written around 1552 B.C, which contained therein similar signs of syphilis. Some of these signs consisted of lesions of the female genitals, discharging exanthema of the scalp, pustules, lesions, itching, lesions pain and carbuncles (Fox 1915). The Ebers Papyrus indicates the possible use of mercury as medicine. However, understandings of translations are rather ambiguous. For example, the Egyptian terms prs and mnst have been argued to be minium, red lead, red ochre or dragons blood (cinnabar or red mercuric sulphide) (Goldwater 1972). Regardless, there appears to be a consensus by many writers that mercury was used in medicine. Imhotep, who was involved in ancient Egyptian medicine, stated that the Egyptians possessed the following drugs:- salts of lead such as sulphate, acetate of copper, sulphate of mercury and pomegranate.

After examining 30,000 syphilis-free bones, anatomist Eliot Smith argued against the suggestion that syphilis existed in ancient Egypt. However, he preferred to be on the side of caution by admitting a Nubian woman from the Middle Empire did display signs similar to syphilis (Smith 1908). This woman's remains appeared in Strangeways, Cambridge displaying

extensive lesions of the humerus and scapula as well as changes in the left humerus shaft which represented a syphilitic node (Moore 1912). These discoveries were good news for those advocating the presence of syphilis in Egypt but unfortunately the node could have been produced by any local inflammatory condition of the periosteum. The change found in her sternum and spinal column may have just represented a severe chronic suppuration. Even though the node on the humerus was the best evidence for syphilis in Egypt to date, as syphilis is unknown in Egyptian bones of this period, caution must be exercised before suggesting the changes were due to syphilis.

In Egypt mummifiers pressed amulets or charms against the deceased during the mummification process before tightly bandaging. The problem with this practice is that the postmortem damage it caused can be confused with skin lesions as confirmed by the mummy of Ramses IV (Smith 1924). He had an elliptic lesion on his penis which is exactly the same size and shape as a chancre.

Mummy 1775:

Despite Elliot Smiths claims there were no reliable cases of syphilis in ancient Egypt, Mummy 1775 could prove the exception. The Egyptian mummy was an adult male, about 46-48 years of age who lived during the

Roman Period, early 2nd century A.D. He is known as Artemidorous or 1775 and resides at the Manchester Museum (David 1979). As this mummy is completely wrapped it is impossible to assess him for signs of skin lesions typical of syphilis. Artemidorous has undergone both x-ray and CT scans, these were used to determine whether any bone changes indicative of a systemic disease had occurred.

Methods:

Researching at the Ancient Egyptian Tissue Bank in Manchester to identify possible signs of syphilis in mummies required the examination of preserved soft tissues of various mummies from different museums, x-rays and CT scans. Although the examination of soft tissue from mummies showed no indications of any lesions, both x-rays and CT scans performed on mummy 1775, showed bone changes indicating a systemic disease. It is now being argued that these bone changes indicate the presence of a systemic disease like syphilis.

Radiography:

Radiography is used to visualise the internal structures of mummies as it is non-invasive and minimally destructive. Radiographic analysis of mummies has observed pathological changes such as arthritis, atheroma, healed fractures as well as parasitic processes. Radiography has its

limitations, as it will only portray most of the pathological processes that reveal diseases affecting the bone. In addition, it can reveal calcified structures. It can be difficult to distinguish between the remains of various soft tissues especially when they are placed on top of each other on the x-ray film.

CT scanning

Radiography is continually advancing especially in the area of CT scanners. These scanners are more advanced than conventional x-ray images as they do not disturb superimpositions of juxtapositional structures. Whereas an image using a conventional x-ray machine is just two dimensional the CT scanner revolves around an object producing a 3D image as well as creating an image portraying a slice of the object. The item is moved through the CT scanner generating hundreds of thousands of slices, which may be viewed as serial, two-dimensional slice images (Lynnerup 2007).

When bones are viewed in a 3D form, pathological changes are easier to discover. CT scanners have not only picked up rheumatoid arthritis, bone erosions and joint subluxation but also bone tumours in two Egyptian mummies as well as indicating TB which was compared with DNA analysis (Lynnerup 2007). In addition, CT scanning can even help to locate specific organs for biopsies when precise incisions are required.

Results:

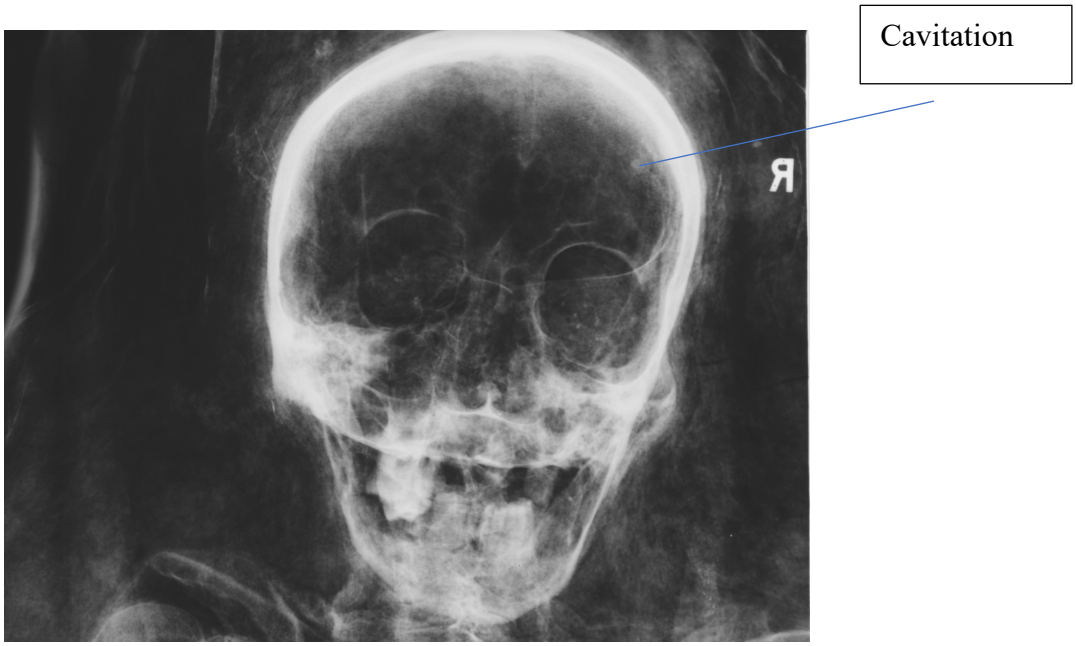


Figure :1 Frontal x-ray of cranium showing circular lesion cavitation

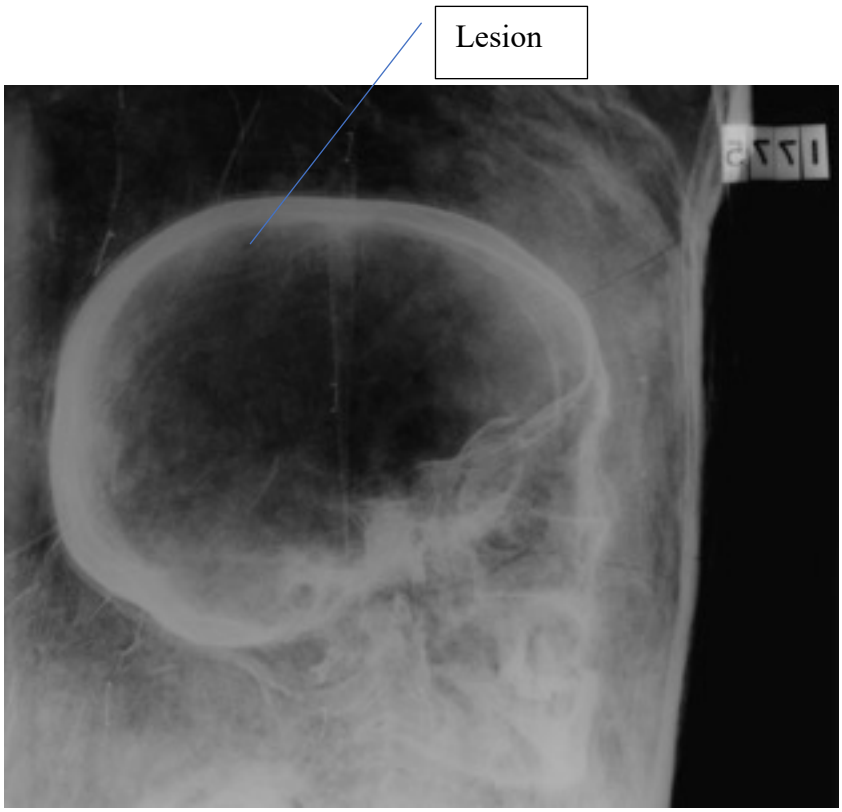


Figure 2: X-ray of Parietal view showing circular lesion

The following section is derived from a report of Mummy 1775 written by Frank Rühli (2015) as an unpublished report

Head:

"Remnants of meninges. Little rest of brain dorsally. Sinus frontalis, maxillaries, sphenoidalis, and mastoid are well pneumatised. Cellulae ethmoidales show at the right side a defect, with a fracture at the medial part up to the right orbit, up to the foramen magnum. Non-dislocated fracture at the right occipital region" (Rühli 2015).

Bone:

"The bones show partial osteoporosis. The second lumbar vertebra shows a decreased height, differential diagnoses are most likely trauma or osteoporosis related. No significant arthritis.

Peri and postmortem changes: Minimal dislocation of the scapula left, non-dislocated subcapital humerus fracture left. Proximal ulnar fracture left. Sub-luxated ileo sacral joints on both sides, luxated symphysis pubis. Multi fragmentary, not dislocated fracture of the acetabulum right. No Harris lines.

Multiple circular, lithic defects of various size partially with sclerosis of the margin can be found at the head of the skull, as well as the thoracic and lumber spine, os ileum on both sides as well as os sacrum. As a differential diagnosis: multiple myeloma, osteolytic metastasis or artefacts due to mummification can be considered" (Rühli 2015).

Dental status:

"In the upper jaw only, right first premolar, left second premolar and left third molar are present. Missing their antagonists, the teeth of the left side are extremely elongated. Left first molar has completely lost its osseous attachment. From two front teeth only the root fragments are left.

In the lower jaw only the abraded teeth from left second premolar and right canine are present, the first incisors showing large periradicular translucencies" (Rühli 2015).

Caries lesions cannot be detected.

The disastrous condition of the dentition suggests a systemic disease.

Discussion:

The research addressing the potential presence of pathological indicators of syphilis in Egyptian mummies from the Ancient Egyptian Tissue Bank in Manchester did not find anything conclusive. The X-rays and CT scans of mummy 1775 indicated that this individual suffered from a wound on the right side of the parietal bone of the skull. They also suggested that the individual possibly suffered from numerous cranial lesions.

The dentition of this individual shows signs of a systemic disease. Syphilis is a systemic disease, it attacks multiple tissues, however, there is no real indication that this individual suffered from such a disease. No other bone has been affected which would indicate syphilis.

Metaponto

Description of Metaponto and signs of pathology

The ancient Greek colony of Metaponto in southern Italy, dated to the 6th-3rd BCE displayed signs equivalent to treponematoses (Henneberg and Henneberg 1994). Excavations were carried out at Pantanello necropolis, Saldone and Sant`Angelo from 1982 to 1993 by J.C. Carter and his international team of researchers who uncovered 272 skeletons (Henneberg and Henneberg 1994).

Henneberg et al. (1992) demonstrated evidence for syphilis within the populace based on macroscopic observations and analysis of frequency distributions of pathologies in the skeletal collection. (Henneberg et al. 1992). An immunochemical test is the only way to provide evidence of syphilis. Unfortunately the success rate of this test is low due to the lack of antigen present in the bones as well as low levels of antibodies because of bone changes which occur during the tertiary stage. The fact that antigens and antibodies partially decompose after death and being buried for centuries tends to make it more difficult to produce a positive reading (Armelagos et al. 2012).

According to Maciej and Renata Henneberg the evidence syphilis existed at Metaponto takes the form of sclerotic thickening, sabre shin tibia, periostitis and cranial erosion that may have been either taphonomic or caused by gummatous ulcers, which unfortunately is not a strong

case for syphilis (Henneberg and Henneberg 1994). As the remains were poorly preserved a positive diagnosis of caries sicca was not conclusive, but on the other hand, dental stigmata were found on a number of teeth. These dental stigmata which were observed in the Metaponto skeletal remains were Hutchinson's incisors and molars with unusual occlusal surfaces full of pitts and lacking normal cusp patterns (Henneberg and Henneberg 1994)

It has been argued by Maciej and Renata Henneberg (Henneberg et al. 1992, Henneberg and Henneberg 1994, 1998) that the skeletal pathology seen in the necropolis at Metaponto indicates treponematosis not only existed during this time but was also endemic. Stella Ioannou takes the debate further and compares Metaponto cases of congenital syphilis with modern cases of congenital syphilis, finding remarkable similarities with those described by Jacobi et al (1992) (Ioannou et al. 2018). Skeletal evidence of congenital syphilis establishes syphilis was around in a particular area and time period and is a vital component of the debate concerning Old World pre-Columbian syphilis.

Even though the evidence of syphilis found at Metaponto can relate to varying diseases, there are still other methods that can be employed to strengthen the claim relating to the existence of syphilis. One such method is analyzing the bones for mercury. It is known that mercury was used to treat skin diseases like syphilis before and after AD1495 (Goldwater 1972; O'Shea 1990; Thomann 2015). Therefore, depending on the amount of time passed between mercury being used and the patient's death, there will be differences in the levels of mercury that are remaining in the individuals bones. This is due to the fact that mercury has a 70 day half-life in the human body (Ramussen et al 2013). If, after analysis, debated syphilitic bones show a higher reading of mercury than non-pathological bones, it is a sure indication of treponemal disease.

Mercury analysis

The mercury analysis was completed at the University of Adelaide Microscopy Labs. The five bone fragments from Metaponto were analysed for mercury concentrations with LA-ICP-MS. Bone fragments were taken from Henneberg's research collection. The fragments were soaked in alcohol before being placed on a microscope slide with strong double-sided sticky tape.

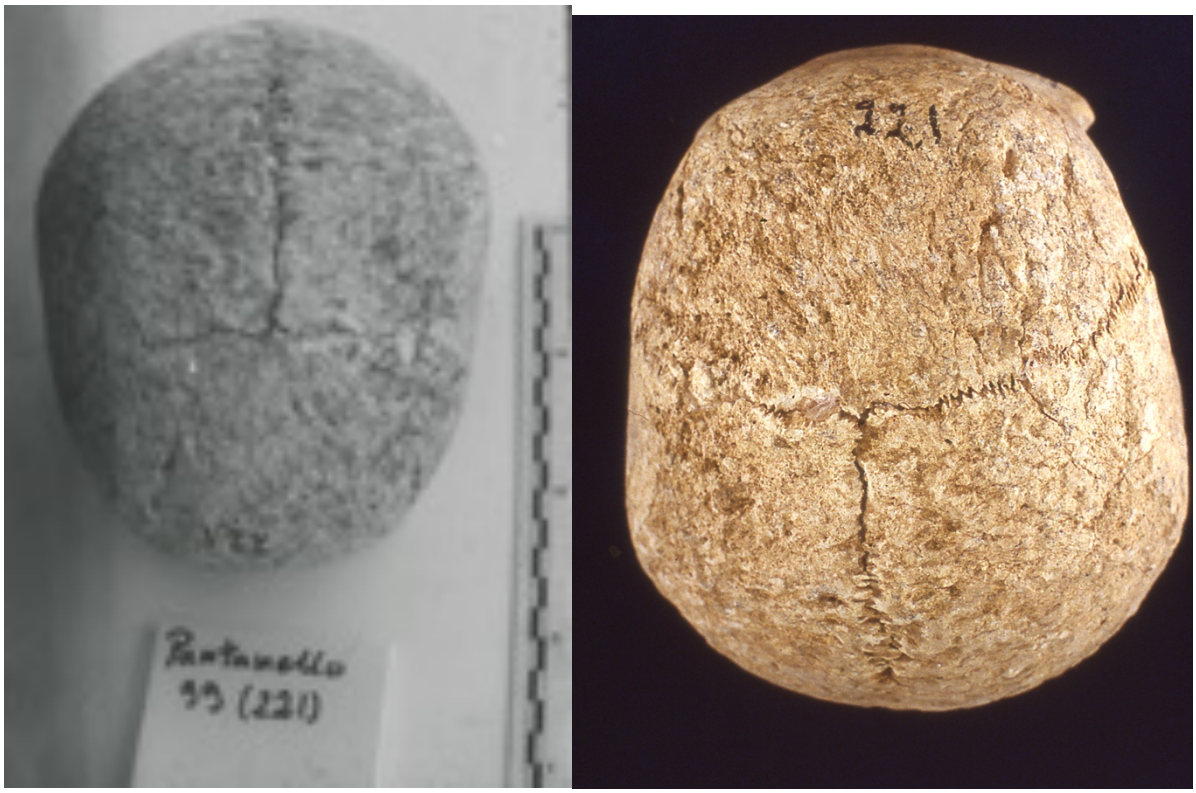


Figure 1: Metaponto 221 Sclerotic thickening, Worm eaten erosion on the cranium

Results:

Table 1: Metaponto mercury concentrations

Sample number	Pathology	Mercury concentration (PPM)
T276	Sabre Tibia	0.22
T221	Sclerotic thickening, Worm eaten erosion on the cranium	6.5
T243		0.5
T1		0.6
T246		0.4

Conclusion/Discussion

The mercury analysis of the bone fragments from Metaponto indicates that there is a strong presence of mercury. T221 had indications of syphilis through cranial changes of sclerotic thickening and possible gummatous ulcers. The mercury concentration of this individual was the highest at 6.5ppm. The lowest reading came from the T276 who had sabre tibia with a mercury concentration of 0.22. All bone fragments had a mercury concentration well above the normal range of mercury of 0.01 to 0.1ppm (Rasmussen et al. 2013). Therefore, these individuals may have suffered from syphilis during their lifetime and were being treated for it with mercury.

Oplontis

Description of Oplontis

The Villa of Poppaea is located near the modern town of Torre Annunziata near Naples. In 1964 excavations began to unearth the Villa which exposed a well-preserved building and within the cellar numerous skeletal remains related to people that died there during the eruption of Mount Vesuvius. Due the eruption of Mount Vesuvius the skeletal materials were sealed in an underground cellar which left them well-preserved. However, since excavations at the villa have taken place the bones were left to slowly decay. Maciej and Renata Henneberg examined the skeletal material at the Villa of Poppaea and found two cases of congenital syphilis (Henneberg and Henneberg 2006).

Description of pathology

The two skeletons that have been argued to have had congenital syphilis were twins aged around 12 to 14 years. The pathology of the skeleton known as no 41 shows signs of severe hypoplasia with horizontal grooves and irregular pitting on the incisors, canine and first molar in the right maxilla (Ioannou et al. 2018). The first molar on the right maxilla also had Fournier's teeth and mulberry molar. The crown of the first incisor on the right maxilla appears to have Hutchinson's incisors or screwdriver incisor. On the right mandible the first molar shows signs of a mulberry molar (Henneberg et al 2006).

The skeleton known as No 2 shows signs of hypoplasia on the left maxilla's second incisor and canine (Ioannou et al. 2018). The second incisor showed signs of Hutchinson's teeth. The right maxilla had signs of a mulberry molar on the first molar. The right maxilla had enamel hypoplasia on the second right incisor. The first premolar showed signs of Fournier's tooth and Hutchinson's tooth. The right maxilla on the first permanent first molar showed signs of a mulberry molar. The fragmented right mandible showed signs of a mulberry molar (Henneberg and Henneberg 2006).

There is no differential diagnosis for these individuals as no other disease/toxin affects teeth this way.

Methods:

At the Villa Poppaea in the cellar small bone fragments were collected from the skeletons. The sub-adult argued to have congenital syphilis labeled 2 was removed from the ground and was placed in a crate. The skeleton number 41 was found over a skeleton that was still in the ground. This posed a problem due to the layout of the skeletons being comingled. Fragments were taken from the left of skeleton 41 and Right of 41 to get variation of mercury concentrations and as a precautionary measure due to the comingled setting.

Mercury analysis

The mercury analysis was conducted at the University of Adelaide Microscopy Labs. The five bone fragments were analysed for mercury concentrations with LA-ICP-MS. Bone fragments were taken from Henneberg's research collection. The fragments were soaked in alcohol before being placed on a microscope slide with strong double-sided sticky tape.

Table 1: Pompeii/Oplontis mercury concentrations

Sample number	Pathology	Mercury concentrations (PPM)
Pompeii2	Congenital syphilis	0.18
Pompeii41R		0.11
Pompeii41M	Congenital syphilis	0.22
Pompeii41L		0.33

Conclusion/Discussion

The twins found in the cellar of the villa of Poppaea were described and argued by Maciej and Renata Henneberg as having congenital syphilis. Ioannou et al. (2018) take the research of the twins further and argue that they show signs of mercurial teeth (Ioannou et al 2018). The use of LA-ICP-MS on bone fragments of selected skeletons from the cellar of the Villa Poppaea has shown that these individuals had traces of mercury preserved in their bones. Skeleton 2 one of the congenital syphilis twins had a mercury reading of 0.18ppm, which is above normal concentrations, whereas 41R has a lower reading of 0.11ppm, that is only just

above the upper reading of normal concentrations. The mercury concentration for the other twin was 0.22ppm which shows that this individual had mercury well above normal concentrations. The highest mercury concentration came from 41L of 0.33pmm which could still have come from 41M as they were comingled.

The cause of the high mercury concentration could be a result of either treatment of the disease or due to the natural high mercury content in the soil due to the Mount Vesuvius volcano (Cicchella et al 2005). As no soil from the site was ever tested this cause for the skeletons high mercury concentrations cannot be ruled out. However mercury soil pollution studies show evidence that the highest Hg baseline values (0.20–0.90 ppm) exactly coincide with the mostly urbanized Napoli area. The Sorrento Peninsula soils of the vast majority of suburban provincial areas are characterized by baseline values in the range 0.05–0.20 ppm, which we identify as natural background values (Cicchella et al 2005).

Although the number of congenital syphilis cases from the Mediterranean is limited, their close match to pathognomonic dental traits well documented in the 19th century is sufficient to strengthen the view advocating the presence of the disease in the Old World.

Discussion:

This study having re-evaluated the literary evidence pertaining to the origins of syphilis, has found there is not enough evidence to blame Columbus, for bringing a virulent strand of treponematosis from Haiti to the Old World. In fact, this study suggests that syphilis existed in the Old World prior to Columbus, but does not preclude the introduction of a new strain of syphilis from the New World upon Columbus' return. Through examining skeletal collections in universities and museums around Europe, and using LA-ICP-MS to determine the concentrations of mercury within the selected individuals, we have found that high mercury concentrations can support limited skeletal pathology that might indicate syphilis.

This study was important in understanding the origins of syphilis and improving the methods employed for the diagnosis of syphilis in skeletal remains. The use of analytical techniques which can be used to support the existence of mild cases of syphilis, with limited to no pathognomonic signs and high concentrations of mercury, which was often used as a remedy for syphilis in pre and post Columbian times, expands the range of evidence.

Why Columbus was not to blame?

Syphilis is a disease that has caused great confusion clinically in the past due to its mimicking abilities (Rothchild 2005; Dupnik et al. 2012). The origin of the disease has also caused confusion historically, as each country blamed another for introducing syphilis to their country (Whitwell 1940). Lastly its evolving genetic nature has also caused confusion, because the close genetic connections syphilis has with its non-venereal strains are not well

known (Harper et al 2008; Čejková et al. 2012; Centurion-Lara et al 2013; Štaudová et al. 2014). Expanded knowledge regarding the origins of syphilis will improve the understanding of its evolutionary future. The high prevalence of syphilis causes the disease to evolve, becoming more resistant to certain antibiotics, however penicillin is still effective against syphilis (Stamm 2010; Tipple et al 2011).

Christopher Columbus has been a complication in understanding the origins and epidemiology of syphilis within a world view. Due to the emphasis on Columbus' contacts with Native American populations, the Columbian theory has received strong support among scholars in Americas. This has led to American paleopathologists using it as a template for all possible cases of syphilis around the world (Harper et al. 2011). Due to significant variations in medical knowledge, living conditions and severity of the disease between the Old and New World in paleopathology, assessing skeletal remains should be based on what is seen in the typical pathognomonic bone changes in the Old World not the New. The assessment of the more serious and prevalent pathognomonic skeletal changes, indicative of syphilis in the Old-World cases provides an improved understanding of less severe cases that may have been associated with a milder strain of the disease or involved treatment with mercury.

The historical evidence provides a detailed picture of the role Columbus played in the spread of syphilis in the Old World. Columbus' ships log books were read and transcribed by La Casas who supported the American origin theory (Morrison 1939). He did not mention in his journal that Columbus' crew or the Native Americans who returned with him, were infected with any new disease. In fact, Columbus' log speaks of no serious ill health on board his ships. During this era of seafaring it was required for any sickness, disease, or injury to be recorded by the Admiral. In the journal of Vasco da Gama (AD 1497-1499) he recorded both

sickness (scurvy) and the amount deaths that took place. However, all that Columbus mentions is that there were some men with ulcers who were left behind in Haiti with a ships doctor (Morrison 1939). The presence of virulent diseases was never mentioned in the Azores or Portugal where they had landed before arriving in Spain. Even then it was not until the AD 1530's that any mention or blame would befall Columbus' part in the spread of syphilis. The reliability of Oviedo, Ruiz de Isla, Le Casas and Fracastoro needs to be examined critically. Although the writers could have observed symptoms potentially associated with an outbreak of syphilis, the length of time before writing and publishing skews their perceptions regarding the nature of the disease. On the basis of a number of historical accounts, it may be argued that the focus on the American origins of syphilis was associated with propaganda to shift the blame to a supposedly uncivilized, immoral part of the world.

Columbus' voyage was not the main cause of the spread of syphilis in Europe. In fact, the paleopathological evidence in Europe prior to Columbus suggests that not only did Europe suffer from syphilis but that it was treated it with mercury (Rasmussen et al 2008; Rasmussen et al 2013). At Pompeii, the evidence shows signs of congenital syphilis but also the presence of mercuric teeth existed in Pompeii (Ioannou et al. 2018). Mercuric teeth show a distinct colouration associated with exposure to mercury during the period of tooth formation. These cases of congenital syphilis indicate syphilis had a presence in the Old World. Consequently, the idea of Columbus bringing back non-venereal syphilis from Haiti becomes a moot-point. Syphilis may have been a mild disease in the Old World prior to Columbus, but there is evidence that occasionally it flared up becoming endemic. This evidence is derived from Renata and Maciej Henneberg's papers on Metaponto. The number of individuals showing signs of non-specific infectious disease, and more distinctive pathognomonic signs of syphilis like sabre tibia, sclerotic thickening of the crania and Hutchinson's incisors, all indicate a

large part of the population were continuously being infected through the generations (Henneberg and Henneberg 1994).

There were many reasons why syphilis became rife during AD 1495. One reason for this disease catastrophe was the presence of mercenaries and prostitutes from many European countries in the army of Charles the VIII which invaded Italy leaving a severe epidemic in their wake (Abraham 1944; Naranjo 1994). Men in Charles the VIII's army would have had sex with many prostitutes along the way to Naples. There is only a 60% chance of being to get infected with syphilis in a single sexual intercourse (Garnett 1997). Therefore, in many cases, one person needs to have sex with an infected individual at least twice to become infected themselves. The more infected the army becomes, the quicker the rate of infection. The more infected the army is, the greater chance individuals have of reinfection, thus creating a hyper sensitivity which in turn caused a superinfection. This is seen in endemic areas like Bosnia and Bakwena Reserve where reinfections are common due to a high prevalence of infected individuals (Murray et al. 1956). Superinfection leads to greater severity of the disease where many will show more signs of tertiary stage syphilis (Grin 1952).

There is evidence explaining why the abovementioned cause of a syphilis epidemic, is a more reliable argument than the alternative hypothesis of Columbus bringing a new disease to the Old World. This evidence is found in the paleopathology of post-Columbian syphilis and the historical literature. Given what has been published the congenital syphilis cases from Spitalfields, Roca Vecchia, Cambrai France and the Italian mummy (Maria d` Aragona) all show varying severity of syphilis (Connell et al 2012; Fornaciari et al 1994; Fornaciari 1994). If it was a new disease from Haiti then theoretically like smallpox it should attack everyone

the same way. However, if individuals did not get reinfected with syphilis then they should only have suffered from endemic syphilis. Grin (1953) described how endemic syphilis affected outsiders more severely than people living in the area (Grin 1953).

Another cause was a vast population movement around AD 1495. Hudson argues that the expulsion of Jews from Spain, and the closing down of leper houses caused an influx of movement to urbanized areas (Holcomb 1937; Hudson 1964; Hudson 1968). There were also bad harvests which drew more people to urbanized areas for employment. All of these events would have facilitated a rapid rate of the spread of syphilis throughout Europe. Moreover, it would have been the cause for the disease keeping its virulence, due to the presence of prostitutes in urbanized areas allowing for reinfection to occur.

How mercury has supported the identification of syphilis in skeletal remains within this study?

According to Armelagos, who supported the Columbian theory, the physical remains of people who died around the AD 1500`s, should reveal the reason for the French Disease arriving in the Old World.

The literature also states that syphilis may have only been a mild disease when present in the Old World (Cockburn 1961; Hacket 1963; Wood 1978), whereas in America the evidence is more prominent, due to the fact that many regions of the Americas were suffering from endemic treponematosis (Powell and Cook 2005). Because there was no cure for treponematosis, therefore bone changes would not be hindered by any form of artificial healing. Consequently, it is important to find analytical methods to support the diagnosis of syphilis in pre-Columbian Old World remains.

Elliot Smith, a palaeopathologist, examined around 30,000 ancient Egyptian and Nubian skeletons excavated from various parts of the country, as well as different eras over the past sixty centuries, without even finding one bone to denote syphilis before modern times (Smith 1908; Barrack 1956).

Since Elliot Smith made this comment, no one has yet found a strong case for syphilis in pre-Columbian Egypt. It has been a recent development to use analytical techniques like LA-ICP-MS to test the concentrations of mercury in bones. Rasmussen has been a leader in this development and has been using it on individuals with either leprosy or syphilis (Rasmussen et al 2008; Rusmussen et al 2015). He has published a paper indicating that the amount of normal traces of mercury in Danish individuals is 10-100ng-1 which is 0.0l-0.1ppm (Rasmussen et al 2008). This has been used to compare with Egyptian individuals from El Kubanieh and Giza, who showed signs of bone changes to the crania that might indicate syphilis. These bone changes are carries sicca, lesions and serpigineous cavitation (Hackett 1975, 1981). The mercury concentrations in these Egyptian samples range from 0.116 to 0.212. The ancient Egyptian mercury concentrations are significantly higher than those reported for both the Danish normal population and the normal range of Egyptian mercury concentrations of 0.008-0.090ppm. Consequently, ancient Egyptian mercury concentrations appear to be high enough to support an argument for the use of mercury as a treatment for syphilis.

The paleopathologist Møller Christiansen excavated leprosaria and examined cases of leprosy. He stated that through all of the leprosaria he has excavated that he has not examined any cases showing syphilitic signs (Møller-Christiansen 1967). However, he did find one skeleton from Æbelholt Monastery that he at first thought was leprosy but over time changed his diagnosis to ergotism. This skeleton has been re-examined since Møller Christiansen with

the most probable revised diagnosis of treponematosis, but neither smallpox nor sarcoidosis could be excluded (Lefort and Bennike 2007).

Medical papers have also eluded that syphilis and leprosy have been known in modern time to form co-infections (Murray 1982). Therefore, it is reasonable to suggest that in one of Møller Christiansen's leprosy collections, there might be an individual showing signs of both leprosy and syphilis. The Danish leprosarium at Aaderup was excavated and examined by Møller Christiansen, who wrote a paper describing the crania that had leprosy (Møller Christensen 1978). In one case number 312 he describes leprosy changes in the cranium and only mentions leprosy changes in the post-crania. However, this individual diagnosed with leprosy also exhibited bone changes in the post-crania showing signs of syphilis. These changes were Charcot joints on the knee, with both the tibia and femur being affected. Other bone changes include periostitis on the midshaft of the other tibia. Leprosy affects bone on the distal ends of the long bones, hands and feet, working its way up, whereas syphilis starts in the midshaft and spreads out. In syphilis also Charcot joints are also more common in the knee while leprosy typically is associated with charcot joints in the ankle (Ortner 2003).

In the Aaderup collection there were also other individuals showing pathological signs of syphilis. These include sabre tibia, symmetrical signs on both tibiae or fibulae, along the midshafts and pitting on child crania, which is also associated with leprosy. Considering that this is a leprosarium, the majority of samples taken had high concentrations of mercury. The mercury concentrations ranged from 0.03- 2.50ppm. In this case using mercury to support the diagnoses of syphilis is difficult as lepers also used mercury, despite the fact it did not help fight the disease. It also does not help when there are possibilities of co-infections. There could be obvious signs of leprosy displayed in the paleopathology of the individuals;

however, there may be still traces of pathology left in the bones indicating syphilis due to the co-infections. This could be the reason why lepers continued to use mercury as some symptoms may have disappeared if mercury was used to treat syphilis, whereas if it was used to treat leprosy it would not have helped.

In pre-Columbian Poland there was not much in the way of urbanized areas which may have prevented outbreaks of syphilis, as little paleopathological evidence of syphilis exists in pre-Columbian Poland. At the University of Łódź two medieval collections BK5 and Kolonia did not yield any diagnostic evidence of syphilis. BK5 -18 had a mercury concentration of 0.221 which was significantly higher than any of the 22 bones sampled. As this skeleton did not exhibit any clear signs of syphilis, the identity of the condition associated with this particular person before death at around seven years of age is uncertain. However, it is evident that this individual either through medication or by accident, came in contact with large traces of mercury which his bones were still storing. Thus, it is possible that BK5- 18 could have been exposed to mercury until the time he died. Since only about one third of syphilitic patients show pathological signs in bone, it is likely that the skeletal remains of some individuals inflicted with syphilis will not show any signs of syphilis. However, if they were treated with mercury they may still exhibit increased levels of mercury in their bones. This could have occurred in cranium BK5-18.

Even though the only pathological mark on this skeleton was horizontal striations across the forehead, which could not be confused with taphonomy, it could still be compared with known cases of post-Columbian congenital syphilis. However, with no indication of Hutchinson`s Incisors, Mulberry molars or any other clear indicators of the disease, it cannot be seriously considered as syphilis.

Conclusion:

This thesis has demonstrated that historical evidence does not support the introduction of a virulent form of syphilis to the Old World following the return of Columbus from the Americas. There is not enough evidence to suggest that any sailors were infected with a new disease on board the Nina or Pinta. There is also a lack of first-hand accounts of the disease starting in Barcelona. Considering the nature of the disease, there is more evidence to suggest that the Europeans did not want to be blamed for such a disease, so they used Columbus' recent voyage to an uncivilized part of the world to cast blame. This explains why writers like Fracastoro and Ruiz de Isla started comparing syphilis to old diseases and then later stating that it came from the Americas Holcomb 1937).

The reason that historical accounts from the Old World stated they had never witnessed such a terrible disease before was because syphilis had been a mild disease that imitated other diseases like leprosy (Cockburn 1961; Hacket 1963; Wood 1978). The two diseases have been confused with one another throughout antiquity, often being described as venereal leprosy (Gordon 1491). It was only when Charles the VIII's army invaded Italy that syphilis was regularly exported due to its high virulence from constant reinfection that created a superinfection.

The paleopathological evidence that was examined as part of this thesis has supported the claim that syphilis existed in pre-Columbian times. The Egyptian crania that were examined from the Natural History Museum in Vienna showed a range of signs of carries sicca, skeletal lesions and serpigineous cavitation. The University of Copenhagen had part of the Aaderup

leprosarium collection, which also showed signs of syphilis through sabre tibia and Charcot joints.

Skeletal remains from both Egypt and Denmark show signs of syphilis, all with above normal mercury concentration in their bones. This suggests that not only did they have skeletal pathologies that were linked to syphilis but they also employed mercury treatments to cure the disease. This provides strong evidence for the existence of syphilis existed in pre-Columbian times in the Old World.

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Appendices:

Mercury Concentrations:

University of Adelaide LA-ICP-MS results

GLITTER!: Trace Element Concentrations MDL filtered.							
Element	stdMAPS4-1	stdMAPS4-2	stdMAPS4-3	stdMAPS4-4	N612-1	N612-2	stdMAPS4-5
P31	177,818.47	168,788.81	168,343.14	159,967.95	54.77	52.16	158,052.84
Ca43	338,300.06	338,300.06	338,300	338,300	85,042.27	85,042.27	338,300.03
Sr88	3,154.64	3,112.31	3,085.23	3,042.45	84	83.65	3,072.54
Cd111	23.19	21.28	23.52	24.25	22.52	21.15	23.97
Hg200	3.65	2.77	3.07	2.67	0.208	0.198	2.78
Hg201	3.64	2.8	3	2.71	0.2	0.188	2.76
Hg202	3.65	2.77	3	2.74	0.215	0.203	2.79
Pb208	217.69	210.16	222.74	228.13	35.02	34.1	229.93
GLITTER!: 1 sigma error.							
Element	stdMAPS4-1	stdMAPS4-2	stdMAPS4-3	stdMAPS4-4	N612-1	N612-2	stdMAPS4-5
P31	54,661.11	52,043.75	52,069.31	49,638.41	17.13	16.37	49,545.65
Ca43	10,710.01	10,708.79	10,711.26	10,712.49	2,701.14	2,701.22	10,713.08
Sr88	110.84	109.32	108.45	107.1	2.97	2.97	109.1
Cd111	1.61	1.48	1.64	1.7	1.59	1.5	1.72
Hg200	0.52	0.4	0.45	0.4	0.032	0.031	0.44
Hg201	0.5	0.39	0.42	0.39	0.03	0.029	0.42
Hg202	0.49	0.38	0.42	0.39	0.031	0.03	0.42
Pb208	11.89	11.46	12.14	12.47	1.92	1.89	12.8

GLITTER!: Trace Element Concentrations MDL filtered.						
Element	Polish-1	Polish-2	Polish-3	Polish-4	Polish-5	Polish-6
P31	163,655.09	146,755.27	163,260.91	158,218.59	160,974.88	156,708.59
Ca43	399,603.63	399,603.66	399,603.66	399,603.66	399,603.63	399,603.63
Sr88	325.54	292.15	428.25	318.49	392.46	402.44
Cd111	2.23	1.6	1.36	1.99	2.19	1.8
Hg200	0.527	0.497	0.462	0.71	0.468	0.69
Hg201	0.506	0.494	0.471	0.67	0.45	0.66
Hg202	0.491	0.496	0.455	0.7	0.462	0.68
Pb208	277.48	175.02	549.96	327.37	536.27	836.34
GLITTER!: 1 sigma error.						

Element	Polish-1	Polish-2	Polish-3	Polish-4	Polish-5	Polish-6
P31	51,484.7	46,336.9	51,741.19	50,335.2	51,412.16	50,249.58
Ca43	12,645.54	12,647.21	12,645.16	12,654.72	12,646.51	12,646.85
Sr88	11.61	10.47	15.43	11.55	14.33	14.8
Cd111	0.17	0.13	0.11	0.17	0.18	0.15
Hg200	0.086	0.083	0.08	0.13	0.087	0.13
Hg201	0.08	0.08	0.079	0.12	0.08	0.12
Hg202	0.076	0.079	0.075	0.12	0.081	0.12
Pb208	15.6	9.95	31.67	19.12	31.79	50.39

GLITTER!: Trace Element Concentrations MDL filtered.						
Element	Pompeii2-1	Pompeii2-2	Pompeii2-3	Pompeii2-4	Pompeii2-5	Pompeii2-6
P31	170,504.84	156,024.8	163,181.83	156,712.48	151,151.48	161,673.59
Ca43	399,603.66	399,603.66	399,603.66	399,603.66	399,603.66	399,603.69
Sr88	549.63	517.02	484.77	472.61	499.23	455.48
Cd111	1.27	1.22	1.09	0.916	0.809	1.09
Hg200	0.18	0.226	0.152	0.143	0.219	0.206
Hg201	0.173	0.229	0.148	0.135	0.215	0.2
Hg202	0.184	0.223	0.149	0.142	0.233	0.203
Pb208	113.44	46.39	101.38	93.34	83.77	71.52
GLITTER!: 1 sigma error.						
Element	Pompeii2-1	Pompeii2-2	Pompeii2-3	Pompeii2-4	Pompeii2-5	Pompeii2-6
P31	54,896.45	50,443.61	52,981.65	51,101.58	49,505.78	53,190.18
Ca43	12,644.81	12,646.18	12,647.01	12,647.07	12,646.49	12,647.85
Sr88	20.37	19.32	18.28	17.99	19.18	17.68
Cd111	0.11	0.11	0.1	0.088	0.08	0.11
Hg200	0.036	0.047	0.033	0.032	0.051	0.05
Hg201	0.033	0.046	0.031	0.029	0.048	0.047
Hg202	0.035	0.044	0.031	0.03	0.051	0.046
Pb208	6.95	2.89	6.44	6.04	5.52	4.8

GLITTER!: Trace Element Concentrations MDL filtered.						
Element	Pompeii41R-1	Pompeii41R-2	Pompeii41R-3	Pompeii41R-4	Pompeii41R-5	Pompeii41R-6
P31	160,928.34	142,612.11	146,474.53	150,469.7	152,323.39	153,508.69
Ca43	399,603.66	399,603.69	399,603.69	399,603.69	399,603.66	399,603.66
Sr88	840.91	827.44	866.93	838.38	793.74	812.34
Cd111	0.488	0.477	0.402	0.532	0.505	0.618
Hg200	0.101	0.107	0.105	0.091	0.134	0.16
Hg201	0.089	0.1	0.114	0.102	0.139	0.145
Hg202	0.094	0.109	0.113	0.099	0.135	0.155

Pb208	64.87	22.69	53.13	43.84	35.69	47.32
GLITTER!: 1 sigma error.						
Element	Pompeii41R-1	Pompeii41R-2	Pompeii41R-3	Pompeii41R-4	Pompeii41R-5	Pompeii41R-6
P31	53,187.4	47,353.5	48,866.66	50,441.59	51,313.27	51,970.13
Ca43	12,646.72	12,647.4	12,650.55	12,651.71	12,650.79	12,650.69
Sr88	32.96	32.78	34.72	33.95	32.5	33.65
Cd111	0.053	0.054	0.049	0.064	0.061	0.074
Hg200	0.026	0.028	0.029	0.026	0.04	0.049
Hg201	0.022	0.026	0.03	0.028	0.04	0.043
Hg202	0.023	0.027	0.029	0.027	0.038	0.045
Pb208	4.44	1.59	3.78	3.18	2.64	3.57

GLITTER!: Trace Element Concentrations MDL filtered.							
Element	stdMAPS4-6	stdMAPS4-7	stdMAPS4-8	stdMAPS4-9	N612-3	N612-4	stdMAPS4-10
P31	170,606.67	159,710.55	168,629.78	166,391.36	51.14	55.68	172,311.09
Ca43	338,300.06	338,300.09	338,300.09	338,300.06	85,042.29	85,042.3	338,300.09
Sr88	3,135.17	3,090.11	3,150.9	3,108.23	84.3	83.69	3,046.29
Cd111	22.55	23.26	21.76	23.85	21.96	21.65	23.34
Hg200	3.34	3.3	2.76	2.89	0.245	0.22	2.95
Hg201	3.31	3.33	2.77	2.92	0.231	0.213	2.93
Hg202	3.29	3.28	2.8	2.93	0.234	0.215	2.93
Pb208	216.49	229.51	212.17	223.39	33.95	33.85	217.88
GLITTER!: 1 sigma error.							
Element	stdMAPS4-6	stdMAPS4-7	stdMAPS4-8	stdMAPS4-9	N612-3	N612-4	stdMAPS4-10
P31	58,050.96	54,622.59	57,973.74	57,506.87	17.86	19.54	60,530.23
Ca43	10,715.1	10,714.8	10,713.07	10,714.01	2,703.43	2,703.07	10,713.26
Sr88	131.38	131.01	135.17	134.94	3.71	3.73	137.11
Cd111	2.38	2.5	2.4	2.68	2.52	2.54	2.8
Hg200	1.06	1.09	0.96	1.04	0.092	0.087	1.21
Hg201	1.02	1.06	0.92	1.01	0.084	0.081	1.15
Hg202	0.99	1.03	0.91	1	0.083	0.08	1.13
Pb208	16.65	17.99	16.95	18.18	2.81	2.86	18.72

GLITTER!: Trace Element Concentrations MDL filtered.							
Element	stdMAPS4-6	stdMAPS4-7	stdMAPS4-8	stdMAPS4-9	N612-3	N612-4	stdMAPS4-10
P31	171,494.02	160,006.53	168,377.77	165,585.7	50.72	55.03	169,746.81
Ca43	338,299.97	338,299.97	338,299.97	338,299.94	85,042.25	85,042.26	338,299.94
Sr88	3,125.44	3,080.94	3,141.99	3,099.87	84.08	83.49	3,039.34

Cd111	22.69	23.4	21.89	23.98	22.08	21.76	23.45
Hg200	3.37	3.29	2.73	2.83	0.238	0.211	2.81
Hg201	3.33	3.32	2.74	2.86	0.224	0.205	2.79
Hg202	3.31	3.28	2.77	2.88	0.228	0.207	2.8
Pb208	216.31	229.7	212.69	224.3	34.14	34.1	219.82
GLITTER!: 1 sigma error.							
Element	stdMAPS4-6	stdMAPS4-7	stdMAPS4-8	stdMAPS4-9	N612-3	N612-4	stdMAPS4-10
P31	56,848.91	53,039.41	55,814.94	54,892.25	16.91	18.34	56,293.66
Ca43	10,715.1	10,714.8	10,713.07	10,714.01	2,703.43	2,703.06	10,713.26
Sr88	104.81	103.31	105.4	104.11	2.84	2.82	102.76
Cd111	1.25	1.28	1.2	1.32	1.22	1.22	1.32
Hg200	0.46	0.45	0.38	0.4	0.035	0.032	0.42
Hg201	0.46	0.46	0.39	0.41	0.034	0.032	0.43
Hg202	0.43	0.43	0.37	0.39	0.032	0.03	0.4
Pb208	11.26	11.95	11.08	11.73	1.8	1.81	11.76

GLITTER!: Trace Element Concentrations MDL filtered.						
Element	Pompeii41M-1	Pompeii41M-2	Pompeii41M-3	Pompeii41M-4	Pompeii41M-5	Pompeii41M-6
P31	159,008.5	146,328.8	147,341.47	145,534.8	161,663.17	146,891.98
Ca43	399,603.56	399,603.47	399,603.5	399,603.5	399,603.53	399,603.5
Sr88	691.8	734.08	696.98	721.28	708.75	728.49
Cd111	0.453	0.535	0.489	0.572	0.563	0.519
Hg200	0.204	0.232	0.239	0.283	0.163	0.196
Hg201	0.211	0.23	0.23	0.265	0.174	0.186
Hg202	0.216	0.24	0.233	0.271	0.166	0.206
Pb208	245.79	249.08	193.47	269.74	352.96	352.55
GLITTER!: 1 sigma error.						
Element	Pompeii41M-1	Pompeii41M-2	Pompeii41M-3	Pompeii41M-4	Pompeii41M-5	Pompeii41M-6
P31	52,743.36	48,549.59	48,899.64	48,315.85	53,690	48,804.07
Ca43	12,646.54	12,648.32	12,647.7	12,647.18	12,649.85	12,647.68
Sr88	23.46	25	23.84	24.79	24.49	25.31
Cd111	0.037	0.045	0.041	0.046	0.049	0.044
Hg200	0.032	0.037	0.039	0.047	0.029	0.035
Hg201	0.034	0.038	0.039	0.046	0.032	0.034
Hg202	0.032	0.037	0.036	0.044	0.028	0.035
Pb208	13.29	13.64	10.74	15.2	20.22	20.54

GLITTER!: Trace Element Concentrations MDL filtered.						
Element	Pompeii41L-1	Pompeii41L-2	Pompeii41L-3	Pompeii41L-4	Pompeii41L-5	Pompeii41L-6
P31	129,306.51	142,575.52	141,739.42	142,579.53	137,744.42	141,232.63
Ca43	399,603.53	399,603.5	399,603.53	399,603.5	399,603.5	399,603.5
Sr88	924.7	957.55	976.95	793.32	1,024.53	965.41
Cd111	0.6	0.51	0.578	0.622	0.535	0.693
Hg200	0.27	0.322	0.369	0.196	0.366	0.5
Hg201	0.269	0.351	0.353	0.213	0.35	0.44
Hg202	0.277	0.334	0.359	0.214	0.353	0.457
Pb208	72.51	203.89	109.36	14.4	189.38	132.96
GLITTER!: 1 sigma error.						
Element	Pompeii41L-1	Pompeii41L-2	Pompeii41L-3	Pompeii41L-4	Pompeii41L-5	Pompeii41L-6
P31	42,980.5	47,413.91	47,160.5	47,466.66	45,884.4	47,076.55
Ca43	12,648.15	12,648.47	12,649.8	12,652.17	12,649.36	12,659.88
Sr88	32.33	33.7	34.62	28.32	36.85	35.02
Cd111	0.05	0.045	0.051	0.057	0.049	0.07
Hg200	0.05	0.061	0.072	0.04	0.077	0.11
Hg201	0.051	0.069	0.071	0.045	0.076	0.099
Hg202	0.049	0.061	0.068	0.042	0.071	0.096
Pb208	4.3	12.33	6.74	0.91	12.15	8.71

GLITTER!: Trace Element Concentrations MDL filtered.						
Element	MetapontoT276-1	MetapontoT276-2	MetapontoT276-3	MetapontoT276-4	MetapontoT276-5	MetapontoT276-6
P31	78,151.91	143,375.52	163,102.13	96,011.84	73,345.82	107,839.3
Ca43	399,603.5	399,603.5	399,603.5	399,603.5	399,603.5	399,603.5
Sr88	178.29	224.73	306.95	224.91	170.48	239.48
Cd111	0.197	0.304	0.213	0.179	0.312	0.309
Hg200	0.152	0.174	0.194	0.338	0.192	0.282
Hg201	0.158	0.142	0.212	0.344	0.175	0.266
Hg202	0.159	0.177	0.213	0.351	0.192	0.26
Pb208	4.89	6.73	4.81	8.21	4.97	5.33
GLITTER!: 1 sigma error.						
Element	MetapontoT276-1	MetapontoT276-2	MetapontoT276-3	MetapontoT276-4	MetapontoT276-5	MetapontoT276-6
P31	26,067.54	47,857.3	54,482.65	32,097.05	24,539.93	36,111.89
Ca43	12,645.74	12,664.03	12,660.27	12,654.88	12,648.84	12,652.03
Sr88	6.52	8.29	11.42	8.44	6.46	9.16
Cd111	0.023	0.042	0.034	0.03	0.035	0.037
Hg200	0.034	0.041	0.047	0.085	0.05	0.076
Hg201	0.037	0.035	0.054	0.09	0.048	0.075
Hg202	0.035	0.04	0.05	0.085	0.048	0.068
Pb208	0.33	0.46	0.34	0.59	0.36	0.4

GLITTER!: Trace Element Concentrations MDL filtered.							
Element	stdMAPS4-11	stdMAPS4-12	stdMAPS4-13	stdMAPS4-14	N612-5	N612-6	stdMAPS4-15
P31	167,439.31	174,842.14	162,618.28	161,124.19	48.64	50.32	164,559
Ca43	338,299.88	338,299.88	338,299.88	338,299.88	85,042.23	85,042.23	338,299.88
Sr88	3,135.45	3,105.67	3,097.28	3,094.27	85.17	83.58	3,081.32
Cd111	22.91	22.3	23.65	22.12	22.34	22.06	23.72
Hg200	3.51	2.65	3.14	2.73	0.255	0.224	3.1
Hg201	3.54	2.63	3.07	2.7	0.285	0.232	3.15
Hg202	3.49	2.6	3.11	2.77	0.276	0.258	3.17
Pb208	225.42	212.28	220.35	210.25	35.62	35.61	227.34
GLITTER!: 1 sigma error.							
Element	stdMAPS4-11	stdMAPS4-12	stdMAPS4-13	stdMAPS4-14	N612-5	N612-6	stdMAPS4-15
P31	56,120.57	58,656.68	54,609.38	54,162.38	16.48	17.05	55,496.25
Ca43	10,714.6	10,714.2	10,732.98	10,736.27	2,705.06	2,704.67	10,715.17
Sr88	121.03	121.04	121.98	123.1	3.43	3.4	126.29
Cd111	1.83	1.82	1.99	1.9	1.94	1.95	2.14
Hg200	0.97	0.76	0.93	0.84	0.082	0.075	1.06
Hg201	1.02	0.78	0.95	0.87	0.096	0.081	1.13
Hg202	0.94	0.73	0.9	0.83	0.086	0.084	1.06
Pb208	17.06	16.39	17.37	16.91	2.92	2.98	19.39

GLITTER!: Trace Element Concentrations MDL filtered.							
Element	stdMAPS4-11	stdMAPS4-12	stdMAPS4-13	stdMAPS4-14	N612-5	N612-6	stdMAPS4-15
P31	169,637.63	177,146.16	164,769.13	163,263.11	49.29	50.99	166,767.55
Ca43	338,299.97	338,299.94	338,299.97	338,299.94	85,042.25	85,042.26	338,299.97
Sr88	3,071.6	3,048.04	3,045.4	3,048.02	84.05	82.63	3,051.95
Cd111	22.46	21.9	23.27	21.79	22.05	21.8	23.49
Hg200	3.6	2.69	3.15	2.72	0.251	0.218	2.98
Hg201	3.63	2.66	3.07	2.67	0.278	0.223	2.99
Hg202	3.57	2.63	3.1	2.73	0.269	0.249	3.01
Pb208	217.53	205.35	213.67	204.36	34.71	34.77	222.56
GLITTER!: 1 sigma error.							
Element	stdMAPS4-11	stdMAPS4-12	stdMAPS4-13	stdMAPS4-14	N612-5	N612-6	stdMAPS4-15
P31	56,722.41	59,233.05	55,094.98	54,591.48	16.59	17.16	55,762.74
Ca43	10,714.6	10,714.21	10,732.99	10,736.28	2,705.06	2,704.67	10,715.17
Sr88	106.74	105.92	105.91	106.01	2.93	2.88	106.06
Cd111	1.39	1.36	1.46	1.38	1.37	1.35	1.46
Hg200	0.55	0.41	0.48	0.41	0.039	0.034	0.45
Hg201	0.6	0.44	0.51	0.44	0.048	0.039	0.49
Hg202	0.55	0.4	0.48	0.42	0.042	0.039	0.46
Pb208	13.55	12.8	13.32	12.74	2.17	2.17	13.87

GLITTER!: Trace Element Concentrations MDL filtered.						
Element	MetapontoT221-1	MetapontoT221-2	MetapontoT221-3	MetapontoT221-4	MetapontoT221-5	MetapontoT221-6
P31	253,000.13	151,239.06	154,426.55	164,014.39	199,822.61	203,577.09
Ca43	399,603.53	399,603.56	399,603.53	399,603.53	399,603.56	399,603.56
Sr88	357.71	344.53	332.47	332.62	346.09	347.02
Cd111	0.327	0.265	0.207	0.213	0.242	0.328
Hg200	3.11	13.5	4.57	17.97	4.63	2.47
Hg201	3.01	13.35	4.43	18.33	4.73	2.51
Hg202	3.25	13.46	4.46	18.18	4.73	2.52
Pb208	110.24	80.04	90.81	71.75	57.58	1,702.83
GLITTER!: 1 sigma error.						
Element	MetapontoT221-1	MetapontoT221-2	MetapontoT221-3	MetapontoT221-4	MetapontoT221-5	MetapontoT221-6
P31	84,596.6	50,571.13	51,636.69	54,844.65	66,815.83	68,070.91
Ca43	12,654.83	12,691.59	12,678.73	12,770.61	12,669.72	12,657.34
Sr88	12.43	12	11.57	11.64	12.04	12.06
Cd111	0.036	0.053	0.046	0.083	0.039	0.039
Hg200	0.47	2.05	0.69	2.73	0.7	0.38
Hg201	0.5	2.2	0.73	3.03	0.78	0.41
Hg202	0.5	2.07	0.69	2.8	0.73	0.39
Pb208	6.87	4.99	5.66	4.49	3.59	106.09

GLITTER!: Trace Element Concentrations MDL filtered.						
Element	Aaderup80-1	Aaderup80-2	Aaderup80-3	Aaderup80-4	Aaderup80-5	Aaderup80-6
P31	200,682.45	268,507.94	158,196.17	200,883.3	172,069.73	151,755.7
Ca43	399,603.53	399,603.56	399,603.53	399,603.56	399,603.56	399,603.56
Sr88	410.69	494.44	395.35	457.39	502.05	375.99
Cd111	2.46	2.26	1.43	2	2.86	1.69
Hg200	0.429	0.336	0.494	0.352	0.642	0.488
Hg201	0.53	0.33	0.446	0.405	0.61	0.466
Hg202	0.491	0.348	0.444	0.34	0.6	0.457
Pb208	12.95	12.52	24.73	13.47	10.62	16.43
GLITTER!: 1 sigma error.						
Element	Aaderup80-1	Aaderup80-2	Aaderup80-3	Aaderup80-4	Aaderup80-5	Aaderup80-6
P31	67,102.95	89,782.81	52,896.95	67,170.25	57,535.8	50,743.32
Ca43	12,654.85	12,678.55	12,668.54	12,660.32	12,662.87	12,663.97
Sr88	14.27	17.2	13.75	15.9	17.46	13.08

Cd111	0.17	0.18	0.12	0.15	0.2	0.13
Hg200	0.066	0.052	0.076	0.054	0.099	0.075
Hg201	0.089	0.057	0.076	0.068	0.1	0.079
Hg202	0.076	0.055	0.069	0.053	0.093	0.071
Pb208	0.81	0.78	1.54	0.84	0.66	1.03

GLITTER!: Trace Element Concentrations MDL filtered.						
Element	Aaderup399-1	Aaderup399-2	Aaderup399-3	Aaderup399-4	Aaderup399-5	Aaderup399-6
P31	224,882.72	178,970.81	272,985.16	176,513.53	172,707.23	228,700.92
Ca43	399,603.56	399,603.56	399,603.53	399,603.56	399,603.53	399,603.56
Sr88	720.13	792.34	685.23	768.19	590.85	612.72
Cd111	6.9	5.57	7.16	5.15	2.39	8.75
Hg200	2.17	0.55	1.29	1.42	0.554	4.72
Hg201	2.08	0.526	1.41	1.39	0.536	4.66
Hg202	2.33	0.534	1.32	1.49	0.536	4.68
Pb208	98.12	19.6	83.88	53.92	56.18	112.34
GLITTER!: 1 sigma error.						
Element	Aaderup399-1	Aaderup399-2	Aaderup399-3	Aaderup399-4	Aaderup399-5	Aaderup399-6
P31	75,195.19	59,843.18	91,281.93	59,022.32	57,748.61	76,472.14
Ca43	12,665.35	12,656.23	12,737.28	12,690	12,647.82	12,673.76
Sr88	25.04	27.54	23.9	26.74	20.53	21.31
Cd111	0.46	0.36	0.55	0.37	0.16	0.58
Hg200	0.33	0.084	0.2	0.22	0.084	0.72
Hg201	0.34	0.088	0.24	0.23	0.089	0.77
Hg202	0.36	0.083	0.21	0.23	0.083	0.72
Pb208	6.12	1.22	5.24	3.37	3.5	7

GLITTER!: Trace Element Concentrations MDL filtered.							
Element	stdMAPS4-16	stdMAPS4-17	stdMAPS4-18	stdMAPS4-19	N612-7	N612-8	stdMAPS4-20
P31	162,952.17	169,813.38	166,615.77	165,640.11	51.49	47.48	160,301.19
Ca43	338,299.94	338,299.94	338,299.97	338,299.97	85,042.25	85,042.26	338,299.97
Sr88	3,166.1	3,187.68	3,181.12	3,125.67	86.17	86.03	3,110.02
Cd111	25.88	24.19	22.94	21.75	23.46	24.11	22.99
Hg200	3.52	2.98	3.07	2.19	0.264	0.23	3.43
Hg201	3.66	2.87	2.98	2.18	0.265	0.221	3.59
Hg202	3.61	2.95	3.02	2.23	0.259	0.201	3.5
Pb208	245.1	214.23	228.55	215.38	37.29	37.14	233.36
GLITTER!: 1 sigma error.							

Element	stdMAPS4-16	stdMAPS4-17	stdMAPS4-18	stdMAPS4-19	N612-7	N612-8	stdMAPS4-20
P31	54,487	56,781.85	55,712.05	55,385.86	17.35	16	53,600.78
Ca43	10,716.03	10,740.63	10,717.63	10,719.42	2,706.34	2,705.08	10,724.14
Sr88	110.03	110.89	110.56	108.64	3	3	108.12
Cd111	1.6	1.53	1.43	1.36	1.46	1.49	1.44
Hg200	0.53	0.45	0.47	0.33	0.041	0.036	0.52
Hg201	0.6	0.48	0.49	0.36	0.046	0.039	0.59
Hg202	0.55	0.46	0.46	0.34	0.041	0.032	0.54
Pb208	15.27	13.35	14.24	13.42	2.33	2.32	14.54

GLITTER!: Trace Element Concentrations MDL filtered.							
Element	stdMAPS4-16	stdMAPS4-17	stdMAPS4-18	stdMAPS4-19	N612-7	N612-8	stdMAPS4-20
P31	168,310.06	175,396.88	172,094.13	171,086.39	53.18	49.04	165,571.91
Ca43	338,299.91	338,299.91	338,299.91	338,299.91	85,042.25	85,042.24	338,299.91
Sr88	3,063.15	3,084.02	3,077.67	3,024.03	83.37	83.23	3,008.89
Cd111	25.06	23.42	22.21	21.06	22.71	23.34	22.26
Hg200	3.57	3.03	3.12	2.22	0.269	0.234	3.48
Hg201	3.69	2.9	3.01	2.2	0.267	0.223	3.62
Hg202	3.63	2.97	3.03	2.24	0.26	0.202	3.52
Pb208	228.91	200.08	213.44	201.14	34.82	34.68	217.93
GLITTER!: 1 sigma error.							
Element	stdMAPS4-16	stdMAPS4-17	stdMAPS4-18	stdMAPS4-19	N612-7	N612-8	stdMAPS4-20
P31	58,144.04	60,592.87	59,451.31	59,103.22	18.5	17.06	57,198.32
Ca43	10,716.03	10,740.62	10,717.62	10,719.42	2,706.34	2,705.08	10,724.14
Sr88	106.29	107.12	106.8	104.95	2.9	2.9	104.44
Cd111	1.87	1.77	1.66	1.57	1.69	1.74	1.67
Hg200	0.57	0.49	0.5	0.36	0.044	0.039	0.56
Hg201	0.62	0.49	0.51	0.37	0.047	0.04	0.61
Hg202	0.59	0.48	0.49	0.36	0.043	0.034	0.57
Pb208	13.58	11.87	12.66	11.93	2.07	2.06	12.93

GLITTER!: Trace Element Concentrations MDL filtered.						
Element	Aaderup258-1	Aaderup258-2	Aaderup258-3	Aaderup258-4	Aaderup258-5	Aaderup258-6
P31	325,386.53	202,105.47	188,817.2	217,182.89	288,385.91	186,905.53
Ca43	399,603.5	399,603.5	399,603.53	399,603.5	399,603.5	399,603.5
Sr88	638.56	609.88	588.66	631.03	641.26	583.92
Cd111	7.21	4.6	4.58	5.46	5.99	5.87
Hg200	1.28	0.599	0.602	3.63	3.08	0.84
Hg201	1.46	0.62	0.571	3.46	3.04	0.85
Hg202	1.4	0.596	0.595	3.37	3.14	1

Pb208	99.56	131.9	84.96	127.09	157.64	106.99
GLITTER!: 1 sigma error.						
Element	Aaderup258-1	Aaderup258-2	Aaderup258-3	Aaderup258-4	Aaderup258-5	Aaderup258-6
P31	112,406.84	69,818.92	65,228.54	75,027.75	99,625.75	64,569.12
Ca43	12,646.17	12,657.66	12,664.04	12,665.71	12,674.52	12,705.5
Sr88	22.15	21.17	20.44	21.91	22.27	20.31
Cd111	0.54	0.36	0.36	0.43	0.48	0.5
Hg200	0.21	0.097	0.098	0.58	0.5	0.14
Hg201	0.25	0.11	0.098	0.59	0.51	0.15
Hg202	0.23	0.097	0.097	0.54	0.51	0.16
Pb208	5.91	7.83	5.04	7.54	9.36	6.36

GLITTER!: Trace Element Concentrations MDL filtered.						
Element	Aaderup205-1	Aaderup205-2	Aaderup205-3	Aaderup205-4	Aaderup205-5	Aaderup205-6
P31	249,727.66	283,311.63	198,382.47	231,465.78	247,346.84	179,813.73
Ca43	399,603.5	399,603.5	399,603.5	399,603.47	399,603.5	399,603.53
Sr88	728.86	733.79	632.75	647.87	660.55	649.36
Cd111	2.12	3.3	1.46	2.37	1.66	1.137
Hg200	4.27	86.04	2.39	1.79	2.73	1.3
Hg201	2.53	88.17	2.84	1.66	3.83	1.45
Hg202	5.51	87.65	2.37	1.56	2.59	2.78
Pb208	20.61	53.99	23.48	13.44	24.39	17.2
GLITTER!: 1 sigma error.						
Element	Aaderup205-1	Aaderup205-2	Aaderup205-3	Aaderup205-4	Aaderup205-5	Aaderup205-6
P31	86,270.85	97,872.33	68,533.05	79,962.01	85,449.33	62,117.96
Ca43	12,673.22	12,661.1	12,668.2	12,669.69	12,704.49	12,653.07
Sr88	25.31	25.47	21.97	22.5	22.97	22.53
Cd111	0.19	0.27	0.13	0.2	0.18	0.099
Hg200	0.69	13.81	0.38	0.29	0.44	0.21
Hg201	0.43	14.86	0.48	0.28	0.65	0.25
Hg202	0.89	14.15	0.38	0.25	0.42	0.45
Pb208	1.23	3.2	1.4	0.8	1.45	1.02

GLITTER!: Trace Element Concentrations MDL filtered.						
Element	Aaderup396-1	Aaderup396-2	Aaderup396-3	Aaderup396-4	Aaderup396-5	Aaderup396-6
P31	174,801.28	180,691.13	159,704.78	161,680.63	162,638.89	155,014.72
Ca43	399,603.5	399,603.47	399,603.5	399,603.47	399,603.5	399,603.53
Sr88	526.42	667.63	610.14	578.68	702.93	528.5

Cd111	4.72	4.97	4.52	4.69	4.33	3.98
Hg200	0.93	1.21	1.3	1.04	7.83	0.63
Hg201	1.15	1.53	1.18	0.97	7.57	0.63
Hg202	0.98	1.54	1.23	0.95	7.43	0.62
Pb208	150.84	194.19	132.93	152.11	243.39	90.72
GLITTER!: 1 sigma error.						
Element	Aaderup396-1	Aaderup396-2	Aaderup396-3	Aaderup396-4	Aaderup396-5	Aaderup396-6
P31	60,386.29	62,422.12	55,171.26	55,854.12	56,190.68	53,551.03
Ca43	12,649.27	12,698.94	12,656.62	12,670.47	12,931.3	12,655.97
Sr88	18.26	23.21	21.17	20.1	24.68	18.34
Cd111	0.36	0.42	0.35	0.38	0.54	0.31
Hg200	0.15	0.2	0.21	0.17	1.27	0.1
Hg201	0.19	0.26	0.2	0.17	1.29	0.11
Hg202	0.16	0.25	0.2	0.16	1.21	0.1
Pb208	8.95	11.53	7.89	9.03	14.51	5.38

GLITTER!: Trace Element Concentrations MDL filtered.							
Element	stdMAPS4-21	stdMAPS4-22	stdMAPS4-23	stdMAPS4-24	N612-9	N612-10	stdMAPS4-25
P31	173,079.77	164,897.23	158,368.2	168,526.33	46.87	48.18	160,469.69
Ca43	338,299.91	338,299.94	338,299.91	338,299.91	85,042.25	85,042.24	338,299.91
Sr88	3,143.23	3,169.11	3,161.52	3,121.91	86.01	85.07	3,123.14
Cd111	25.85	21.02	22.74	24.23	24.62	24.33	23.21
Hg200	3.72	2.99	2.47	2.89	0.258	0.212	3.19
Hg201	3.67	2.93	2.47	2.98	0.245	0.202	3.22
Hg202	3.75	2.95	2.49	2.92	0.225	0.2	3.17
Pb208	241.1	220.25	220.83	227.39	37.63	37.15	226.5
GLITTER!: 1 sigma error.							
Element							
P31	stdMAPS4-21	stdMAPS4-22	stdMAPS4-23	stdMAPS4-24	N612-9	N612-10	stdMAPS4-25
Ca43	59,791.85	56,965.06	54,709.54	58,218.78	16.37	16.81	55,435.64
Sr88	10,719.3	10,716.5	10,715.53	10,717.34	2,707.69	2,707.37	10,721.06
Cd111	109.08	109.97	109.7	108.33	3	2.96	108.39
Hg200	1.93	1.57	1.69	1.81	1.83	1.81	1.73
Hg201	0.6	0.48	0.4	0.47	0.043	0.036	0.51
Hg202	0.62	0.5	0.42	0.5	0.044	0.037	0.54
Pb208	0.61	0.48	0.4	0.47	0.038	0.034	0.51
	14.3	13.07	13.1	13.49	2.24	2.21	13.44

GLITTER!: Trace Element Concentrations MDL filtered.							
Element	stdMAPS4-21	stdMAPS4-22	stdMAPS4-23	stdMAPS4-24	N612-9	N612-10	stdMAPS4-25
P31	173,681.55	165,470.58	158,918.83	169,112.28	47.03	48.35	161,027.64
Ca43	338,300.03	338,300.03	338,300.03	338,300	85,042.27	85,042.27	338,300.03
Sr88	3,048.55	3,073.65	3,066.29	3,027.87	83.42	82.51	3,029.07
Cd111	25.62	20.83	22.54	24.01	24.4	24.11	23
Hg200	3.92	3.15	2.6	3.05	0.272	0.224	3.36
Hg201	3.85	3.07	2.59	3.12	0.257	0.212	3.38
Hg202	3.94	3.09	2.61	3.06	0.237	0.209	3.33
Pb208	233	212.85	213.41	219.76	36.36	35.9	218.89
GLITTER!: 1 sigma error.							
Element	stdMAPS4-21	stdMAPS4-22	stdMAPS4-23	stdMAPS4-24	N612-9	N612-10	stdMAPS4-25
P31	60,249.17	57,400.76	55,127.97	58,664.07	16.49	16.94	55,859.64
Ca43	10,719.3	10,716.5	10,715.54	10,717.34	2,707.69	2,707.37	10,721.06
Sr88	109.36	110.25	109.98	108.61	3	2.97	108.67
Cd111	2.2	1.79	1.93	2.06	2.09	2.07	1.98
Hg200	0.59	0.48	0.39	0.46	0.043	0.036	0.51
Hg201	0.57	0.46	0.39	0.47	0.042	0.035	0.5
Hg202	0.6	0.47	0.4	0.46	0.037	0.033	0.5
Pb208	12.46	11.38	11.41	11.75	1.95	1.92	11.71

GLITTER!: Trace Element Concentrations MDL filtered.						
Element	Aaderup314-1	Aaderup314-2	Aaderup314-3	Aaderup314-4	Aaderup314-5	Aaderup314-6
P31	263,905.5	236,644.42	216,602.75	176,369.44	181,890.59	175,476.41
Ca43	399,603.63	399,603.66	399,603.63	399,603.63	399,603.63	399,603.59
Sr88	788.52	613.9	658.31	641.79	571.17	713.49
Cd111	3.77	2.4	2.15	1.11	0.676	1.05
Hg200	7.46	0.84	6.21	0.428	0.477	1.31
Hg201	6.87	0.75	6.17	0.47	0.351	1.38
Hg202	6.69	0.78	5.74	0.402	0.381	1.33
Pb208	47.69	6.22	36.5	2.14	1.679	3.48
GLITTER!: 1 sigma error.						
Element	Aaderup314-1	Aaderup314-2	Aaderup314-3	Aaderup314-4	Aaderup314-5	Aaderup314-6
P31	91,547.98	82,090.27	75,138.6	61,181.41	63,096.59	60,871.58
Ca43	12,681.47	12,649.34	12,673.2	12,653.8	12,650.65	12,651.91
Sr88	28.31	22.02	23.63	23.02	20.49	25.59
Cd111	0.36	0.21	0.21	0.11	0.069	0.1

Hg200	1.13	0.13	0.94	0.066	0.073	0.2
Hg201	1.03	0.11	0.92	0.072	0.054	0.21
Hg202	1.01	0.12	0.87	0.062	0.058	0.2
Pb208	2.55	0.33	1.96	0.12	0.091	0.19

GLITTER!: Trace Element Concentrations MDL filtered.						
Element	Aaderup56-1	Aaderup56-2	Aaderup56-3	Aaderup56-4	Aaderup56-5	Aaderup56-6
P31	246,337.81	226,295.17	183,257.89	159,790.88	175,334.41	191,997.86
Ca43	399,603.63	399,603.63	399,603.63	399,603.66	399,603.66	399,603.63
Sr88	795.64	679.76	718.52	709.16	727.32	566.51
Cd111	9.37	11.2	4.82	4.72	4.3	5.73
Hg200	3.22	1.86	0.536	0.6	0.635	1.79
Hg201	3.38	2.03	0.503	0.579	0.627	1.87
Hg202	3.18	2.04	0.526	0.573	0.66	1.9
Pb208	67.56	23.76	6.22	11.82	5.57	7.69
GLITTER!: 1 sigma error.						
Element	Aaderup56-1	Aaderup56-2	Aaderup56-3	Aaderup56-4	Aaderup56-5	Aaderup56-6
P31	85,453.59	78,500.79	63,571.23	55,430.52	60,822.9	66,602.91
Ca43	12,674.26	12,670.78	12,665.04	12,658.9	12,677.58	12,658.32
Sr88	28.56	24.4	25.78	25.44	26.11	20.32
Cd111	0.83	0.98	0.43	0.42	0.4	0.51
Hg200	0.49	0.28	0.082	0.092	0.098	0.27
Hg201	0.51	0.31	0.077	0.089	0.097	0.28
Hg202	0.48	0.31	0.081	0.088	0.1	0.29
Pb208	3.62	1.27	0.34	0.63	0.3	0.41

GLITTER!: Trace Element Concentrations MDL filtered.						
Element	Aaderup550-1	Aaderup550-2	Aaderup550-3	Aaderup550-4	Aaderup550-5	Aaderup550-6
P31	286,729.06	320,780.22	184,304.47	168,418.58	213,308.92	192,757.78
Ca43	399,603.63	399,603.63	399,603.63	399,603.66	399,603.63	338,300.03
Sr88	500.51	490.33	390.56	400.15	399.16	356.8
Cd111	1.54	1.43	1.18	0.85	1.48	0.58
Hg200	0.51	0.352	0.455	0.438	0.528	0.198
Hg201	0.505	0.387	0.44	0.47	0.588	0.209
Hg202	0.497	0.345	0.455	0.44	0.494	0.204
Pb208	16.59	8.6	6.43	5.65	17.8	3.62
GLITTER!: 1 sigma error.						
Element	Aaderup550-1	Aaderup550-2	Aaderup550-3	Aaderup550-4	Aaderup550-5	Aaderup550-6
P31	99,464.45	111,276.4	63,934.02	58,423.36	73,995.96	66,866.64
Ca43	12,654.83	12,650.32	12,653.71	12,656.33	12,672.2	10,720.54
Sr88	17.95	17.59	14.01	14.36	14.33	12.81
Cd111	0.15	0.13	0.11	0.088	0.15	0.066

Hg200	0.078	0.054	0.07	0.067	0.082	0.031
Hg201	0.077	0.059	0.068	0.072	0.091	0.033
Hg202	0.076	0.053	0.07	0.068	0.076	0.032
Pb208	0.89	0.46	0.35	0.3	0.96	0.2

GLITTER!: Trace Element Concentrations MDL filtered.							
Element	stdMAPS4-26	stdMAPS4-27	stdMAPS4-28	stdMAPS4-29	N612-11	N612-12	stdMAPS4-30
P31	160,799.86	165,421.05	172,102.14	173,129.14	44.55	49.25	175,590.61
Ca43	338,300.03	338,300	338,300.03	338,300.03	85,042.27	85,042.27	338,300.03
Sr88	3,134.11	3,199.1	3,111.36	3,162.28	85.92	85.35	3,179.07
Cd111	28.15	22.48	23.18	21.56	25.68	24.66	21.7
Hg200	3.77	2.59	3.12	2.6	0.265	0.222	2.66
Hg201	3.86	2.67	3.07	2.62	0.267	0.218	2.58
Hg202	3.82	2.59	3.15	2.59	0.22	0.227	2.69
Pb208	243.08	225.79	228.13	210.78	37.79	37.16	206.7
GLITTER!: 1 sigma error.							
Element	stdMAPS4-26	stdMAPS4-27	stdMAPS4-28	stdMAPS4-29	N612-11	N612-12	stdMAPS4-30
P31	55,780.74	57,383.54	59,701.3	60,057.48	15.64	17.24	60,911.58
Ca43	10,725.83	10,715.19	10,720.05	10,716.82	2,708.68	2,708.4	10,725.78
Sr88	112.46	114.75	111.62	113.43	3.09	3.07	114.07
Cd111	2.42	1.93	1.99	1.85	2.2	2.12	1.87
Hg200	0.57	0.39	0.47	0.39	0.042	0.036	0.4
Hg201	0.58	0.4	0.46	0.39	0.043	0.036	0.39
Hg202	0.58	0.39	0.48	0.39	0.035	0.036	0.41
Pb208	13	12.08	12.2	11.27	2.02	1.99	11.06

GLITTER4.4.4: University of Adelaide Laser Ablation Analysis Results

All values are reported in ppm

GLITTER!: Trace Element Concentrations MDL filtered.							
Element	stdMAPS-1	stdMAPS-2	stdMAPS-3	stdMAPS-4	N612-1	N612-2	stdMaps-1a
P31	163089.64	162193.59	169682.66	173472.5	53.49	45.71	169077.72
Ca43	338300.09	338300.09	338300.09	338300.09	85042.28	85042.28	338300.06
Sr88	3083.34	3076.86	3091.82	3124.99	81.1	81.38	3128.35
Cd111	21.98	22.16	22.99	23.93	23.01	22.29	24.94
Hg200	4.16	3.07	2.6	2.48	0.54	0.55	2.93
Hg201	4.03	3.09	2.62	2.45	0.54	0.54	3.05

Hg202	4.1	3.12	2.63	2.43	0.56	0.51	2.96
Pb208	214.22	216.81	216.43	222.67	32.38	32.34	234.08
GLITTER!: 1 sigma error.							
Element	stdMAPS-1	stdMAPS-2	stdMAPS-3	stdMAPS-4	N612-1	N612-2	stdMaps-1a
P31	16166.77	16102.08	16875.84	17289.46	5.52	4.77	16992.31
Ca43	10763.6	10765.09	10762.17	10761.74	2742.15	2742.44	10773.43
Sr88	102.8	102.6	103.13	104.3	2.77	2.78	104.86
Cd111	1.67	1.69	1.74	1.82	1.74	1.7	1.97
Hg200	0.91	0.68	0.59	0.57	0.13	0.14	0.73
Hg201	0.82	0.64	0.56	0.53	0.13	0.13	0.7
Hg202	0.88	0.68	0.58	0.55	0.14	0.13	0.72
Pb208	10.58	10.72	10.73	11.08	1.65	1.66	11.91
GLITTER!: Minimum detection limits (99% confidence).							
Element	stdMAPS-1	stdMAPS-2	stdMAPS-3	stdMAPS-4	N612-1	N612-2	stdMaps-1a
P31	2.34	2.41	2.35	2.3	1.86	1.88	2.62
Ca43	37.03	37.35	37.51	33.79	27.9	29.34	37.97
Sr88	0.00562	0.00574	<0.00000	<0.00000	<0.00000	0.00454	<0.00000
Cd111	0.0409	0.0419	0.0416	0.0414	0.0469	0.0472	0.0951
Hg200	0.0812	0.101	0.104	0.105	0.0617	0.0597	0.111
Hg201	0.103	0.136	0.141	0.137	0.0814	0.0811	0.141
Hg202	0.0689	0.0905	0.0907	0.0928	0.0507	0.0522	0.0945
Pb208	0.0152	0.012	0.0109	0.0108	0.00387	0.00871	0.0146
GLITTER!: Trace element concentrations normalised to chondrite.							
Element	stdMAPS-1	stdMAPS-2	stdMAPS-3	stdMAPS-4	N612-1	N612-2	stdMaps-1a
P31	70.91	70.52	73.78	75.42	0.0233	0.0199	73.51
Ca43	25.06	25.06	25.06	25.06	6.3	6.3	25.06
Sr88	259.1	258.56	259.82	262.6	6.81	6.84	262.89
Cd111	21.76	21.94	22.76	23.69	22.78	22.07	24.69
Hg200	7.11	5.24	4.45	4.24	0.92	0.94	5.01
Hg201	6.9	5.28	4.47	4.18	0.92	0.93	5.21
Hg202	7.01	5.34	4.49	4.15	0.96	0.87	5.07
Pb208	58.69	59.4	59.3	61.01	8.87	8.86	64.13
GLITTER!: Mean Raw CPS background subtracted.							

Element	stdMAPS-1	stdMAPS-2	stdMAPS-3	stdMAPS-4	N612-1	N612-2	stdMaps-1a
P31	8543215	8295273	9061229	9311410	3585	3042	7631923
Ca43	225847	220829	230912	232446	73074	72674	196341
Sr88	1255479	1225220	1287631	1310328	42532	42455	1108597
Cd111	1098	1081	1170	1225	1470	1414	1073
Hg200	1889	1345	1179	1114	298	298	1069
Hg201	1046	773	676	628	169	169	635
Hg202	2450	1800	1560	1431	409	364	1415
Pb208	91182	90217	94152	97489	17725	17604	86512

GLITTER!: Trace Element Concentrations MDL filtered.						
Element	B70-1	B70-2	B70-3	B70-4	B70-5	B70-6
P31	162263.64	181981.92	175478.55	144378.64	158930.63	159951.17
Ca43	399603.69	399603.69	399603.63	399603.66	399603.63	399603.63
Sr88	2825.85	3179.41	2908.53	2618.32	3007.66	2938.94
Cd111	0.99	1.11	0.94	0.48	0.94	0.86
Hg200	3.32	4.92	1.9	1.64	3.29	4.42
Hg201	3.28	4.91	4.36	1.63	3.42	4.26
Hg202	3.23	4.55	1.95	1.63	3.49	4.23
Pb208	338.31	456.83	319.51	145.26	206.93	373.77
GLITTER!: 1 sigma error.						
Element	B70-1	B70-2	B70-3	B70-4	B70-5	B70-6
P31	16360.59	18419.85	17828.86	14732.11	16294.47	16477.12
Ca43	12693.81	12740.47	12689.25	12690.71	12740.71	12712.02
Sr88	94.78	107.06	97.96	88.43	102.07	99.93
Cd111	0.17	0.24	0.16	0.11	0.22	0.18
Hg200	0.84	1.28	0.51	0.46	0.95	1.31
Hg201	0.77	1.19	1.08	0.42	0.91	1.17
Hg202	0.8	1.17	0.52	0.45	0.99	1.24
Pb208	17.36	23.74	16.79	7.75	11.23	20.54
GLITTER!: Minimum detection limits (99% confidence).						
Element	B70-1	B70-2	B70-3	B70-4	B70-5	B70-6
P31	2.15	2.37	2.32	2.35	2.67	3.03
Ca43	33.85	26.91	32.79	27.68	24.18	29.26

Sr88	0.0072	0.00815	0.00937	0.00462	<0.00000	0.00884
Cd111	0.0376	0.0301	0.0347	0.0485	0.0276	<0.00000
Hg200	0.074	0.0581	0.0677	0.0674	0.0547	0.0714
Hg201	0.105	0.0817	0.0903	0.0893	0.0693	0.101
Hg202	0.0655	0.0523	0.0623	0.0581	0.0477	0.0658
Pb208	0.0131	0.00926	0.0156	0.00973	0.0106	0.00981
GLITTER!: Trace element concentrations normalised to chondrite.						
Element	B70-1	B70-2	B70-3	B70-4	B70-5	B70-6
P31	70.55	79.12	76.3	62.77	69.1	69.54
Ca43	29.6	29.6	29.6	29.6	29.6	29.6
Sr88	237.47	267.18	244.41	220.03	252.74	246.97
Cd111	0.98	1.1	0.93	0.47	0.93	0.85
Hg200	5.68	8.41	3.26	2.8	5.62	7.55
Hg201	5.6	8.4	7.46	2.78	5.84	7.28
Hg202	5.53	7.78	3.34	2.78	5.96	7.23
Pb208	92.69	125.16	87.54	39.8	56.69	102.4
GLITTER!: Mean Raw CPS background subtracted.						
Element	B70-1	B70-2	B70-3	B70-4	B70-5	B70-6
P31	9266438	12964846	10857135	9039960	12369877	9042220
Ca43	293851	367130	319314	323622	402884	293061
Sr88	1269034	1784197	1419860	1295668	1853192	1317467
Cd111	54	75	55	28	69	46
Hg200	1513	2758	915	787	1936	1865
Hg201	853	1578	1201	447	1153	1030
Hg202	1926	3339	1227	1019	2679	2326
Pb208	158390	267162	162484	74851	132720	174340

GLITTER!: Trace Element Concentrations MDL filtered.						
Element	T243-1	T243-2	T243-3	T243-4	T243-5	T243-6
P31	195547.52	168361.06	78164.5	124323.84	118621.07	165123.59
Ca43	399603.66	399603.63	399603.59	399603.59	399603.59	399603.56

Sr88	244.47	221.36	146.12	195.58	191.76	230.49
Cd111	0.13	0.238	0.112	0.091	0.034	0.071
Hg200	0.6	0.58	0.34	0.2	0.43	0.88
Hg201	0.6	0.38	0.32	0.205	0.38	0.88
Hg202	0.58	0.43	0.39	0.181	0.4	0.86
Pb208	22.85	33.15	11.95	16.05	16.84	22.92
GLITTER!: 1 sigma error.						
Element	T243-1	T243-2	T243-3	T243-4	T243-5	T243-6
P31	20248.33	17525.13	8183.05	13093.65	12569.75	17612.73
Ca43	12719.18	12677.3	12686.64	12699.84	12679.32	12714.55
Sr88	8.4	7.6	5.05	6.79	6.67	8.08
Cd111	0.072	0.068	0.052	0.051	0.029	0.05
Hg200	0.19	0.19	0.12	0.079	0.16	0.33
Hg201	0.19	0.12	0.11	0.083	0.14	0.31
Hg202	0.18	0.14	0.13	0.071	0.14	0.32
Pb208	1.31	1.9	0.71	0.97	1.03	1.43
GLITTER!: Minimum detection limits (99% confidence).						
Element	T243-1	T243-2	T243-3	T243-4	T243-5	T243-6
P31	2.74	2.8	2.41	2.27	2.06	2.26
Ca43	21.53	25.79	24.28	26.25	29.16	30.09
Sr88	0.0127	0.00434	<0.00000	0.00604	<0.00000	0.00471
Cd111	0.0403	<0.00000	0.032	<0.00000	0.0318	<0.00000
Hg200	0.058	0.0614	0.062	0.0577	0.0533	0.0705
Hg201	0.074	0.0818	0.0771	0.0769	0.0781	0.0901
Hg202	0.0475	0.0531	0.0518	0.0534	0.0492	0.0605
Pb208	0.00986	0.0112	0.00822	0.00901	0.00966	0.00812
GLITTER!: Trace element concentrations normalised to chondrite.						
Element	T243-1	T243-2	T243-3	T243-4	T243-5	T243-6
P31	85.02	73.2	33.98	54.05	51.57	71.79
Ca43	29.6	29.6	29.6	29.6	29.6	29.6
Sr88	20.54	18.6	12.28	16.44	16.11	19.37
Cd111	0.128	0.235	0.111	0.09	0.033	0.071
Hg200	1.02	0.99	0.58	0.34	0.74	1.5
Hg201	1.02	0.65	0.55	0.35	0.65	1.5
Hg202	0.99	0.74	0.67	0.31	0.68	1.47

Pb208	6.26	9.08	3.27	4.4	4.61	6.28
GLITTER!: Mean Raw CPS background subtracted.						
Element	T243-1	T243-2	T243-3	T243-4	T243-5	T243-6
P31	14728565	11149658	5252272	8334488	8596449	10002226
Ca43	391047	344343	349914	349623	378518	316863
Sr88	146262	116636	78252	104674	111127	111838
Cd111	9	15	7	5	2	4
Hg200	332	279	161	94	218	364
Hg201	190	104	88	55	110	209
Hg202	419	269	243	111	258	460
Pb208	14217	18162	6652	8924	10136	11543

GLITTER!: Trace Element Concentrations MDL filtered.						
Element	T1-1	T1-2	T1-3	T1-4	T1-5	T1-6
P31	133146.89	148635.75	130763.41	147726.06	151001.45	145639.61
Ca43	399603.59	399603.53	399603.56	399603.53	399603.53	399603.56
Sr88	234.92	248.41	236	260.85	264.02	246.74
Cd111	0.44	0.118	0.221	0.227	0.36	0.098
Hg200	1.69	0.41	0.37	0.69	0.47	0.33
Hg201	1.6	0.46	0.37	0.68	0.49	0.39
Hg202	1.59	0.39	0.39	0.67	0.49	0.35
Pb208	11.44	3.18	3.6	13.59	6.65	3.32
GLITTER!: 1 sigma error.						
Element	T1-1	T1-2	T1-3	T1-4	T1-5	T1-6
P31	14296.49	16069.32	14238.7	16205.21	16690.1	16222.26
Ca43	12700.58	12683.59	12690.25	12704.4	12703.03	12692.76
Sr88	8.26	8.76	8.37	9.31	9.47	8.9
Cd111	0.12	0.052	0.08	0.086	0.11	0.053
Hg200	0.66	0.17	0.16	0.31	0.22	0.16
Hg201	0.58	0.18	0.15	0.28	0.22	0.18
Hg202	0.61	0.16	0.17	0.3	0.23	0.17
Pb208	0.74	0.22	0.25	0.93	0.47	0.25

GLITTER!: Minimum detection limits (99% confidence).						
Element	T1-1	T1-2	T1-3	T1-4	T1-5	T1-6
P31	2.96	1.91	2.27	1.77	2.8	2.22
Ca43	40.58	22.16	32.8	19.59	37.69	22.76
Sr88	<0.00000	<0.00000	<0.00000	<0.00000	<0.00000	0.00459
Cd111	<0.00000	0.0303	0.0625	0.0292	0.0448	0.0349
Hg200	0.0785	0.0587	0.0659	0.0567	0.0925	0.0692
Hg201	0.114	0.0723	0.0922	0.0752	0.116	0.0863
Hg202	0.0731	0.051	0.0588	0.0497	0.0765	0.0605
Pb208	0.017	0.00846	0.00822	0.011	0.0125	0.0112
GLITTER!: Trace element concentrations normalised to chondrite.						
Element	T1-1	T1-2	T1-3	T1-4	T1-5	T1-6
P31	57.89	64.62	56.85	64.23	65.65	63.32
Ca43	29.6	29.6	29.6	29.6	29.6	29.6
Sr88	19.74	20.87	19.83	21.92	22.19	20.73
Cd111	0.43	0.117	0.219	0.225	0.36	0.097
Hg200	2.89	0.7	0.63	1.18	0.81	0.56
Hg201	2.74	0.79	0.63	1.16	0.84	0.66
Hg202	2.71	0.67	0.67	1.14	0.84	0.6
Pb208	3.13	0.87	0.987	3.72	1.82	0.909
GLITTER!: Mean Raw CPS background subtracted.						
Element	T1-1	T1-2	T1-3	T1-4	T1-5	T1-6
P31	6676663	10556060	7796579	10867082	7247296	8974376
Ca43	262705	372628	313309	387141	252969	325281
Sr88	94522	141795	113287	154751	102366	123038
Cd111	20	7	12	15	16	5
Hg200	572	192	142	326	143	126
Hg201	311	124	82	184	85	85
Hg202	694	237	195	407	192	172
Pb208	4775	1880	1793	8358	2672	1713

GLITTER!: Trace Element Concentrations MDL filtered.							
Element	stdMAPS-5	stdMAPS-6	stdMAPS-7	stdMAPS-8	N612-3	N612-4	stdMaps-2a
P31	163751.3	163081.98	172002.5	167083.47	42.37	43.05	167635.92
Ca43	338299.94	338299.91	338299.91	338299.94	85042.25	85042.24	338299.94
Sr88	3129.48	3079.4	3114.12	3111.06	81.14	81.29	3067.34
Cd111	22.81	21.79	23.58	22.77	23.1	22.77	23.53
Hg200	3.43	3.19	2.61	2.98	0.56	0.45	3.05
Hg201	3.34	3.03	2.75	3.01	0.5	0.54	3.04
Hg202	3.38	3.17	2.64	2.98	0.52	0.51	3.06
Pb208	223.28	221.75	219.66	222.58	33.11	32.6	212.28
GLITTER!: 1 sigma error.							
Element	stdMAPS-5	stdMAPS-6	stdMAPS-7	stdMAPS-8	N612-3	N612-4	stdMaps-2a
P31	18387.08	18461.1	19633.03	19234.31	5.1	5.22	19812.52
Ca43	10772.55	10768.18	10764.92	10772.35	2743.06	2742.29	10768.89
Sr88	113.14	111.99	113.94	114.58	3.06	3.08	115.23
Cd111	2.55	2.48	2.73	2.71	2.77	2.79	2.97
Hg200	1.7	1.65	1.41	1.68	0.33	0.28	1.96
Hg201	1.51	1.43	1.36	1.55	0.28	0.31	1.77
Hg202	1.68	1.64	1.43	1.68	0.31	0.32	1.98
Pb208	15.75	15.96	16.13	16.68	2.55	2.56	16.9
GLITTER!: Minimum detection limits (99% confidence).							
Element	stdMAPS-5	stdMAPS-6	stdMAPS-7	stdMAPS-8	N612-3	N612-4	stdMaps-2a
P31	2.8	2.8	2.45	2.58	1.96	1.92	2.44
Ca43	33.89	30.42	34.56	39.69	25.59	23.47	34.49
Sr88	<0.00000	<0.00000	0.0113	0.00607	0.00793	<0.00000	<0.00000
Cd111	0.0481	<0.00000	0.0433	0.0806	0.0351	0.0347	0.08
Hg200	0.102	0.139	0.141	0.158	0.0866	0.0882	0.143
Hg201	0.14	0.175	0.18	0.205	0.114	0.113	0.197
Hg202	0.0959	0.123	0.126	0.141	0.0824	0.0799	0.131
Pb208	0.0122	0.0116	0.0109	0.0105	0.0119	0.00676	0.0104
GLITTER!: Trace element concentrations normalised to chondrite.							
Element	stdMAPS-5	stdMAPS-6	stdMAPS-7	stdMAPS-8	N612-3	N612-4	stdMaps-2a
P31	71.2	70.91	74.78	72.64	0.0184	0.0187	72.89
Ca43	25.06	25.06	25.06	25.06	6.3	6.3	25.06

Sr88	262.98	258.77	261.69	261.43	6.82	6.83	257.76
Cd111	22.59	21.58	23.34	22.54	22.87	22.54	23.3
Hg200	5.86	5.45	4.46	5.09	0.95	0.77	5.21
Hg201	5.71	5.18	4.71	5.14	0.85	0.92	5.2
Hg202	5.78	5.42	4.51	5.09	0.89	0.88	5.24
Pb208	61.17	60.75	60.18	60.98	9.07	8.93	58.16
GLITTER!: Mean Raw CPS background subtracted.							
Element	stdMAPS-5	stdMAPS-6	stdMAPS-7	stdMAPS-8	N612-3	N612-4	stdMaps-2a
P31	7582990	7677555	8849869	7723551	2687	2765	8090127
Ca43	207264	211031	230992	207846	71817	72846	217995
Sr88	1174726	1177154	1303256	1171730	42014	42699	1212331
Cd111	1004	975	1153	1000	1393	1390	1079
Hg200	968	899	790	796	200	161	802
Hg201	545	495	483	466	103	111	466
Hg202	1228	1148	1024	1017	240	234	1027
Pb208	86772	87726	95096	86688	17719	17692	86662

GLITTER!: Trace Element Concentrations MDL filtered.						
Element	T246-1	T246-2	T246-3	T246-4	T246-5	T246-6
P31	166631.03	81278.49	145158.72	78222.74	142644.23	183787.86
Ca43	399603.5	399603.44	399603.44	399603.44	399603.44	399603.47
Sr88	260.03	210.63	319.63	161.92	341.91	369.14
Cd111	0.87	0.27	0.25	0.31	0.091	0.26
Hg200	0.14	0.37	0.42	0.79	0.43	0.29
Hg201	0.24	0.39	0.29	4.43	0.35	0.2
Hg202	0.6	0.43	0.35	2.56	0.42	0.28
Pb208	174.75	5.83	19.84	80.77	2.71	6.89
GLITTER!: 1 sigma error.						
Element	T246-1	T246-2	T246-3	T246-4	T246-5	T246-6
P31	19896.67	9782.85	17641.42	9598.29	17660.22	22976.68
Ca43	13127.08	12713.44	12846.15	12906.74	12702.18	12745.43
Sr88	10.28	8.07	12.44	6.46	13.34	14.55
Cd111	0.45	0.1	0.16	0.21	0.056	0.12
Hg200	0.16	0.27	0.32	0.62	0.35	0.25
Hg201	0.25	0.26	0.23	3.08	0.27	0.17

Hg202	0.44	0.31	0.28	2	0.35	0.25
Pb208	14.36	0.51	1.74	7.03	0.26	0.65
GLITTER!: Minimum detection limits (99% confidence).						
Element	T246-1	T246-2	T246-3	T246-4	T246-5	T246-6
P31	2.35	1.88	1.54	1.44	2.15	2.89
Ca43	31.89	28.73	23.56	16.52	28.58	28.58
Sr88	0.013	0.00436	<0.00000	0.0029	<0.00000	<0.00000
Cd111	<0.00000	<0.00000	0.029	0.0503	0.0393	<0.00000
Hg200	0.113	0.091	0.0777	0.0607	0.1	0.0902
Hg201	0.152	0.112	0.1	0.0752	0.13	0.122
Hg202	0.103	0.08	0.0683	0.0529	0.0905	0.0816
Pb208	0.0103	0.01	0.00916	0.00502	0.00877	0.00825
GLITTER!: Trace element concentrations normalised to chondrite.						
Element	T246-1	T246-2	T246-3	T246-4	T246-5	T246-6
P31	72.45	35.34	63.11	34.01	62.02	79.91
Ca43	29.6	29.6	29.6	29.6	29.6	29.6
Sr88	21.85	17.7	26.86	13.61	28.73	31.02
Cd111	0.86	0.27	0.25	0.31	0.09	0.26
Hg200	0.24	0.64	0.72	1.36	0.74	0.49
Hg201	0.41	0.66	0.49	7.58	0.6	0.34
Hg202	1.02	0.74	0.61	4.38	0.72	0.47
Pb208	47.88	1.6	5.44	22.13	0.742	1.89
GLITTER!: Mean Raw CPS background subtracted.						
Element	T246-1	T246-2	T246-3	T246-4	T246-5	T246-6
P31	8757230	5186305	10797246	7492503	8091512	11955581
Ca43	280845	341514	398719	514236	305012	350324
Sr88	112112	110453	195718	127899	160216	198706
Cd111	43	16	17	28	4	16
Hg200	39	124	159	381	120	89
Hg201	38	75	63	1245	57	35
Hg202	212	183	171	1555	148	108
Pb208	77793	3155	12532	65792	1308	3819

GLITTER!: Trace Element Concentrations MDL filtered.						
Element	Montegine2001-1	Montegine2001-2	Montegine2001-3	Montegine2001-4	Montegine2001-5	Montegine2001-6
P31	159802.86	147567.66	140798.67	149681.5	150706.58	146349.38
Ca43	399603.47	399603.41	399603.38	399603.38	399603.44	399603.44
Sr88	592.05	586.59	565.75	586.43	592.65	596
Cd111	0.53	1.04	0.94	0.43	0.93	0.48
Hg200	0.66	0.68	0.52	0.75	0.58	0.51
Hg201	0.68	0.59	0.48	0.72	0.56	0.49
Hg202	0.73	0.55	0.49	0.84	0.62	0.54
Pb208	125.88	73.62	48.07	46.28	75.04	63.89
GLITTER!: 1 sigma error.						
Element	Montegine2001-1	Montegine2001-2	Montegine2001-3	Montegine2001-4	Montegine2001-5	Montegine2001-6
P31	20173.12	18819.15	18127.04	19462.31	19795.14	19415.34
Ca43	12744.3	12891.32	12736.51	12712.75	12757.69	12706.57
Sr88	23.47	23.61	22.76	23.74	24.24	24.5
Cd111	0.18	0.38	0.25	0.14	0.27	0.14
Hg200	0.59	0.64	0.51	0.75	0.62	0.56
Hg201	0.54	0.51	0.43	0.66	0.54	0.5
Hg202	0.66	0.53	0.49	0.87	0.68	0.62
Pb208	11.49	6.9	4.57	4.47	7.38	6.39
GLITTER!: Minimum detection limits (99% confidence).						
Element	Montegine2001-1	Montegine2001-2	Montegine2001-3	Montegine2001-4	Montegine2001-5	Montegine2001-6
P31	2.39	1.92	2.04	1.95	2.03	2.1
Ca43	22.87	20.95	24.73	24.37	24.77	25.95
Sr88	<0.00000	<0.00000	0.00601	<0.00000	<0.00000	0.0118
Cd111	0.0328	0.0284	0.0333	<0.00000	<0.00000	<0.00000
Hg200	0.0909	0.0756	0.0876	0.0868	0.101	0.0982
Hg201	0.112	0.1	0.114	0.117	0.129	0.126
Hg202	0.0821	0.0743	0.0796	0.0796	0.0915	0.0943
Pb208	0.0103	0.00632	0.00978	0.00787	0.00915	0.0095

GLITTER!: Trace element concentrations normalised to chondrite.						
Element	Montegine2001-1	Montegine2001-2	Montegine2001-3	Montegine2001-4	Montegine2001-5	Montegine2001-6
P31	69.48	64.16	61.22	65.08	65.52	63.63
Ca43	29.6	29.6	29.6	29.6	29.6	29.6
Sr88	49.75	49.29	47.54	49.28	49.8	50.08
Cd111	0.53	1.03	0.94	0.43	0.92	0.47
Hg200	1.13	1.16	0.89	1.28	0.99	0.87
Hg201	1.16	1	0.82	1.24	0.95	0.84
Hg202	1.25	0.94	0.83	1.44	1.05	0.92
Pb208	34.49	20.17	13.17	12.68	20.56	17.5
GLITTER!: Mean Raw CPS background subtracted.						
Element	Montegine2001-1	Montegine2001-2	Montegine2001-3	Montegine2001-4	Montegine2001-5	Montegine2001-6
P31	10487625	11180164	9104684	10152142	9612107	8976335
Ca43	353984	409283	349873	367546	346169	333419
Sr88	322090	369038	304315	331434	315525	305674
Cd111	33	74	57	28	56	27
Hg200	203	235	150	220	156	128
Hg201	122	119	81	125	89	73
Hg202	282	239	175	309	206	169
Pb208	70540	47690	26613	26912	41089	33687

GLITTER!: Trace Element Concentrations MDL filtered.						
Element	Pompeii4-1	Pompeii4-2	Pompeii4-3	Pompeii4-4	Pompeii4-5	Pompeii4-6
P31	113695.64	148346.13	152671.97	137070.16	137981.22	107106.26
Ca43	399603.44	399603.38	399603.34	399603.38	399603.38	399603.34
Sr88	614.25	770.03	751.17	752.93	749.01	560.64
Cd111	0.34	0.195	0.155	<0.040	0.173	0.88
Hg200	0.69	0.74	0.46	0.33	0.46	0.42
Hg201	0.34	0.68	0.39	0.37	0.47	0.67
Hg202	0.49	1.09	0.5	0.33	0.51	0.99
Pb208	109.87	49.23	85.51	42.15	119.17	231.24

GLITTER!: 1 sigma error.						
Element	Pompeii4-1	Pompeii4-2	Pompeii4-3	Pompeii4-4	Pompeii4-5	Pompeii4-6
P31	15236.53	20084.06	20881.76	18944.92	19268.23	15144.08
Ca43	12688.38	12693.52	12676.65	12755.76	12744.6	13703.18
Sr88	25.43	32.13	31.57	32.01	32.08	25.31
Cd111	0.1	0.078	0.06	0.044	0.099	0.7
Hg200	0.8	0.9	0.6	0.45	0.66	0.75
Hg201	0.36	0.74	0.45	0.46	0.59	1.04
Hg202	0.6	1.38	0.67	0.48	0.77	1.62
Pb208	11.17	5.1	8.99	4.53	12.98	25.89
GLITTER!: Minimum detection limits (99% confidence).						
Element	Pompeii4-1	Pompeii4-2	Pompeii4-3	Pompeii4-4	Pompeii4-5	Pompeii4-6
P31	2.08	2.14	1.5	2.5	1.73	5.7
Ca43	27.57	28.42	18.51	30.81	24.01	69.7
Sr88	0.0044	0.00831	0.0034	<0.00000	0.00383	<0.00000
Cd111	<0.00000	<0.00000	0.0269	0.0403	0.0304	0.133
Hg200	0.108	0.113	0.0805	0.131	0.102	0.315
Hg201	0.144	0.151	0.106	0.171	0.12	0.389
Hg202	0.0942	0.101	0.0772	0.115	0.0961	0.295
Pb208	0.00856	0.0102	0.00783	0.0125	0.0105	0.0229
GLITTER!: Trace element concentrations normalised to chondrite.						
Element	Pompeii4-1	Pompeii4-2	Pompeii4-3	Pompeii4-4	Pompeii4-5	Pompeii4-6
P31	49.43	64.5	66.38	59.6	59.99	46.57
Ca43	29.6	29.6	29.6	29.6	29.6	29.6
Sr88	51.62	64.71	63.12	63.27	62.94	47.11
Cd111	0.34	0.193	0.153	0	0.172	0.88
Hg200	1.18	1.27	0.79	0.56	0.79	0.73
Hg201	0.58	1.17	0.67	0.63	0.8	1.15
Hg202	0.84	1.86	0.86	0.57	0.87	1.69
Pb208	30.1	13.49	23.43	11.55	32.65	63.35
GLITTER!: Mean Raw CPS background subtracted.						
Element	Pompeii4-1	Pompeii4-2	Pompeii4-3	Pompeii4-4	Pompeii4-5	Pompeii4-6
P31	7061780	8729626	12222035	7345750	9778384	2466074

Ca43	338169	320897	437234	293162	388282	126350
Sr88	319579	380238	505488	339779	447764	109082
Cd111	20	10	11	1	11	19
Hg200	171	169	140	64	116	33
Hg201	50	93	70	43	70	31
Hg202	151	307	185	79	156	94
Pb208	58748	24973	59090	19523	73102	46149

GLITTER!: Trace Element Concentrations MDL filtered.							
Element	stdMAPS-9	stdMAPS-10	stdMAPS-11	stdMAPS-12	N612-5	N612-6	stdMaps-3a
P31	178468.61	178011.5	177109.14	171528.84	40.96	40.55	184463.06
Ca43	338299.78	338299.78	338299.75	338299.78	85042.21	85042.21	338299.75
Sr88	3093.68	3111.55	3092.86	3070.09	81.94	81.26	3065.89
Cd111	22.14	23.8	26.79	23.65	24.63	24.33	24
Hg200	5.08	4.71	4.72	4.73	0.84	0.74	5.8
Hg201	4.69	4.49	4.55	4.34	0.86	0.78	5.4
Hg202	5.5	5.18	4.98	4.96	0.92	1.01	6.83
Pb208	224.2	228.63	229.01	217.18	33.68	33.82	222.26
GLITTER!: 1 sigma error.							
Element	stdMAPS-9	stdMAPS-10	stdMAPS-11	stdMAPS-12	N612-5	N612-6	stdMaps-3a
P31	25442.51	25643.38	25780.88	25230.79	6.24	6.24	27999.22
Ca43	10761.56	10769.54	10769.14	10768.32	2744.54	2744.32	10767.56
Sr88	134.35	136.27	136.56	136.67	3.72	3.72	139.9
Cd111	4.05	4.43	5.07	4.56	4.81	4.84	4.87
Hg200	7.96	7.8	8.24	8.74	1.64	1.55	12.83
Hg201	6.45	6.5	6.93	6.98	1.47	1.4	10.23
Hg202	9.2	9.19	9.39	9.97	1.97	2.32	16.79
Pb208	25.2	26.12	26.59	25.61	4.05	4.13	27.45
GLITTER!: Minimum detection limits (99% confidence).							
Element	stdMAPS-9	stdMAPS-10	stdMAPS-11	stdMAPS-12	N612-5	N612-6	stdMaps-3a
P31	2.46	3.01	2.8	2.58	2	1.95	2.34
Ca43	27.52	34.1	38.2	37.19	23.64	25.69	31.78
Sr88	0.00559	0.00859	0.00604	0.0132	0.00662	<0.00000	<0.00000

Cd111	0.0893	<0.00000	<0.00000	0.0476	0.0377	0.0532	<0.00000
Hg200	0.178	0.255	0.276	0.299	0.18	0.192	0.29
Hg201	0.212	0.318	0.355	0.367	0.213	0.222	0.365
Hg202	0.17	0.25	0.275	0.278	0.174	0.176	0.282
Pb208	0.0119	0.014	0.0105	0.0116	0.00915	0.00998	0.0139
GLITTER!: Trace element concentrations normalised to chondrite.							
Element	stdMAPS-9	stdMAPS-10	stdMAPS-11	stdMAPS-12	N612-5	N612-6	stdMaps-3a
P31	77.6	77.4	77	74.58	0.0178	0.0176	80.2
Ca43	25.06	25.06	25.06	25.06	6.3	6.3	25.06
Sr88	259.97	261.47	259.9	257.99	6.89	6.83	257.64
Cd111	21.92	23.56	26.53	23.42	24.38	24.09	23.76
Hg200	8.68	8.06	8.06	8.08	1.43	1.27	9.92
Hg201	8.02	7.68	7.77	7.43	1.48	1.33	9.22
Hg202	9.41	8.85	8.51	8.48	1.57	1.73	11.68
Pb208	61.42	62.64	62.74	59.5	9.23	9.27	60.89
GLITTER!: Mean Raw CPS background subtracted.							
Element	stdMAPS-9	stdMAPS-10	stdMAPS-11	stdMAPS-12	N612-5	N612-6	stdMaps-3a
P31	8938277	7906506	7898910	8062647	2430	2412	9119928
Ca43	233044	207001	208186	219764	69857	70152	232263
Sr88	1311640	1172001	1171844	1228128	41457	41294	1296896
Cd111	1051	1002	1132	1053	1384	1371	1124
Hg200	845	671	650	661	141	120	753
Hg201	469	386	379	369	89	77	430
Hg202	1101	882	816	819	182	191	1023
Pb208	97461	88263	88894	88973	17445	17585	96172

GLITTER!: Trace Element Concentrations MDL filtered.						
Element	Pompeii8-1	Pompeii8-2	Pompeii8-3	Pompeii8-4	Pompeii8-5	Pompeii8-6
P31	160042.55	159460.14	158677.48	154103.63	148247.09	176496.7
Ca43	399603.34	399603.25	399603.25	399603.25	399603.28	399603.25
Sr88	491.7	494.85	489	462.13	497.56	472.34

Cd111	0.54	0.191	0.28	0.32	0.266	0.44
Hg200	0.54	0.71	0.72	0.63	0.56	0.93
Hg201	0.49	0.68	0.84	0.59	0.5	0.55
Hg202	0.59	0.76	0.82	0.78	0.81	0.91
Pb208	74	38.63	16.43	21.07	39.19	21.41
GLITTER!: 1 sigma error.						
Element	Pompeii8-1	Pompeii8-2	Pompeii8-3	Pompeii8-4	Pompeii8-5	Pompeii8-6
P31	24549.73	24716.51	24854.19	24392.6	23711.59	28530.74
Ca43	12745.7	12704.96	12698.28	12709.47	12679.54	12760.04
Sr88	22.69	22.98	22.89	21.83	23.66	22.73
Cd111	0.2	0.088	0.11	0.12	0.093	0.19
Hg200	1.28	1.79	1.94	1.81	1.75	3.13
Hg201	1	1.45	1.9	1.45	1.3	1.53
Hg202	1.56	2.17	2.52	2.6	2.96	3.66
Pb208	9.3	4.93	2.13	2.77	5.22	2.91
GLITTER!: Minimum detection limits (99% confidence).						
Element	Pompeii8-1	Pompeii8-2	Pompeii8-3	Pompeii8-4	Pompeii8-5	Pompeii8-6
P31	1.52	1.85	1.81	2.12	2.98	2.71
Ca43	21.49	19.72	18.82	21.35	21.78	18.88
Sr88	<0.00000	<0.00000	<0.00000	0.00946	<0.00000	0.00605
Cd111	0.0281	<0.00000	0.0283	<0.00000	<0.00000	<0.00000
Hg200	0.147	0.144	0.164	0.166	0.171	0.179
Hg201	0.195	0.176	0.187	0.194	0.223	0.232
Hg202	0.152	0.15	0.164	0.172	0.196	0.193
Pb208	0.00912	0.00783	0.00863	0.00879	0.00552	0.0075
GLITTER!: Trace element concentrations normalised to chondrite.						
Element	Pompeii8-1	Pompeii8-2	Pompeii8-3	Pompeii8-4	Pompeii8-5	Pompeii8-6
P31	69.58	69.33	68.99	67	64.46	76.74
Ca43	29.6	29.6	29.6	29.6	29.6	29.6
Sr88	41.32	41.58	41.09	38.83	41.81	39.69
Cd111	0.54	0.189	0.28	0.31	0.263	0.43
Hg200	0.93	1.21	1.23	1.07	0.96	1.59
Hg201	0.84	1.16	1.43	1.02	0.86	0.93
Hg202	1.01	1.3	1.4	1.33	1.38	1.55
Pb208	20.27	10.58	4.5	5.77	10.74	5.87

GLITTER!: Mean Raw CPS background subtracted.						
Element	Pompeii8-1	Pompeii8-2	Pompeii8-3	Pompeii8-4	Pompeii8-5	Pompeii8-6
P31	12296934	12567455	12110968	12229687	10805919	13368186
Ca43	427053	438745	425578	443217	407746	424377
Sr88	323818	334877	321042	316034	313091	309400
Cd111	39	14	20	23	18	31
Hg200	104	133	124	106	83	134
Hg201	58	79	90	63	46	50
Hg202	129	161	158	145	129	140
Pb208	49829	26723	11023	14713	25179	14312

GLITTER!: Trace Element Concentrations MDL filtered.						
Element	Pompeii26-1	Pompeii26-2	Pompeii26-3	Pompeii26-4	Pompeii26-5	Pompeii26-6
P31	149951.06	139694.05	142916.48	165356.89	136447.47	138872.66
Ca43	399603.28	399603.25	399603.25	399603.22	399603.19	399603.25
Sr88	600.59	575.94	593.31	597.48	578.66	645.82
Cd111	0.42	0.36	0.39	0.55	0.53	0.35
Hg200	1.08	1.42	1.34	1.93	2.58	2.25
Hg201	1.06	1.4	1.06	1.85	2.1	2
Hg202	1.61	1.85	1.8	2.73	3.52	3.17
Pb208	122.19	158.21	152.15	139.48	209.12	248.49
GLITTER!: 1 sigma error.						
Element	Pompeii26-1	Pompeii26-2	Pompeii26-3	Pompeii26-4	Pompeii26-5	Pompeii26-6
P31	24494.01	23056.21	23835.47	27868.06	23235.27	23894.52
Ca43	12748.31	12692.55	12693.27	12723.77	12694.02	12679.98
Sr88	29.1	28.09	29.17	29.65	28.92	32.53
Cd111	0.18	0.13	0.14	0.2	0.18	0.12
Hg200	3.95	5.64	5.84	9.22	13.72	13.41
Hg201	3.18	4.5	3.69	7	8.68	9.07
Hg202	7.16	9.21	10.06	17.4	26.05	27.68
Pb208	16.73	21.94	21.39	19.89	30.2	36.36

GLITTER!: Minimum detection limits (99% confidence).						
Element	Pompeii26-1	Pompeii26-2	Pompeii26-3	Pompeii26-4	Pompeii26-5	Pompeii26-6
P31	1.73	3.92	5.05	3.54	4.15	4.57
Ca43	18.04	21.7	26.01	18.11	20.37	22.4
Sr88	<0.00000	0.00649	0.00673	<0.00000	0.00666	0.0038
Cd111	0.032	<0.00000	<0.00000	0.0557	0.0449	<0.00000
Hg200	0.152	0.222	0.299	0.238	0.285	0.316
Hg201	0.177	0.251	0.355	0.275	0.335	0.329
Hg202	0.171	0.261	0.351	0.283	0.35	0.42
Pb208	0.00542	0.00658	0.0111	0.00664	0.00675	0.00817
GLITTER!: Trace element concentrations normalised to chondrite.						
Element	Pompeii26-1	Pompeii26-2	Pompeii26-3	Pompeii26-4	Pompeii26-5	Pompeii26-6
P31	65.2	60.74	62.14	71.89	59.32	60.38
Ca43	29.6	29.6	29.6	29.6	29.6	29.6
Sr88	50.47	48.4	49.86	50.21	48.63	54.27
Cd111	0.41	0.36	0.38	0.55	0.52	0.34
Hg200	1.85	2.43	2.3	3.29	4.42	3.85
Hg201	1.82	2.39	1.81	3.17	3.6	3.42
Hg202	2.75	3.17	3.07	4.66	6.02	5.42
Pb208	33.48	43.35	41.68	38.21	57.29	68.08
GLITTER!: Mean Raw CPS background subtracted.						
Element	Pompeii26-1	Pompeii26-2	Pompeii26-3	Pompeii26-4	Pompeii26-5	Pompeii26-6
P31	14326223	9820368	8180494	12831696	9300515	9907941
Ca43	536167	395158	322272	437616	385017	403658
Sr88	497129	351409	295291	403869	344194	402811
Cd111	37	24	20	40	34	23
Hg200	185	166	119	213	229	190
Hg201	117	107	61	136	126	116
Hg202	287	222	158	290	288	233
Pb208	103176	98443	77189	96073	126699	157810

GLITTER!: Trace Element Concentrations MDL filtered.						
Element	Pompeii27-1	Pompeii27-2	Pompeii27-3	Pompeii27-4	Pompeii27-5	Pompeii27-6
P31	151214.48	139353.61	129589.26	137086.59	133992.58	142315.83
Ca43	399603.22	399603.22	399603.16	399603.16	399603.16	399603.16
Sr88	461.82	475.97	430.08	496.49	508.94	507.08
Cd111	0.21	0.44	0.26	0.29	0.47	0.181
Hg200	0.73	1.1	2.39	3.45	2.37	2.15
Hg201	0.47	0.73	1.8	2.51	1.76	1.17
Hg202	0.92	1.4	4.66	9.69	11.34	181.85
Pb208	86.66	2921.79	84.1	74.74	113.92	54.84
GLITTER!: 1 sigma error.						
Element	Pompeii27-1	Pompeii27-2	Pompeii27-3	Pompeii27-4	Pompeii27-5	Pompeii27-6
P31	26291.27	24481.91	23000.5	24581.8	24274.99	26047.89
Ca43	12743.1	12777.26	12712.42	12680.89	12684.79	12675.88
Sr88	23.51	24.46	22.23	25.84	26.7	26.81
Cd111	0.12	0.21	0.12	0.11	0.16	0.075
Hg200	4.96	8.55	21.62	37.35	31.71	37.47
Hg201	2.35	4.1	11.38	18.08	14.74	11.57
Hg202	9.77	18.8	84.11	263.96	614.44	266569.28
Pb208	12.87	438.89	12.81	11.52	17.78	8.67
GLITTER!: Minimum detection limits (99% confidence).						
Element	Pompeii27-1	Pompeii27-2	Pompeii27-3	Pompeii27-4	Pompeii27-5	Pompeii27-6
P31	2.88	2.47	3.86	3.19	4.52	3.8
Ca43	13.42	11.97	19.31	21.53	18.96	17.78
Sr88	0.00451	0.00249	<0.00000	0.00643	0.00404	0.00332
Cd111	<0.00000	<0.00000	0.0333	0.0438	0.0585	<0.00000
Hg200	0.209	0.247	0.478	0.507	0.662	0.714
Hg201	0.218	0.274	0.481	0.485	0.61	0.548
Hg202	0.306	0.401	0.838	1.11	2.26	55.06
Pb208	0.00396	0.00437	0.00705	0.00731	0.00871	0.00716
GLITTER!: Trace element concentrations normalised to chondrite.						
Element	Pompeii27-1	Pompeii27-2	Pompeii27-3	Pompeii27-4	Pompeii27-5	Pompeii27-6
P31	65.75	60.59	56.34	59.6	58.26	61.88
Ca43	29.6	29.6	29.6	29.6	29.6	29.6

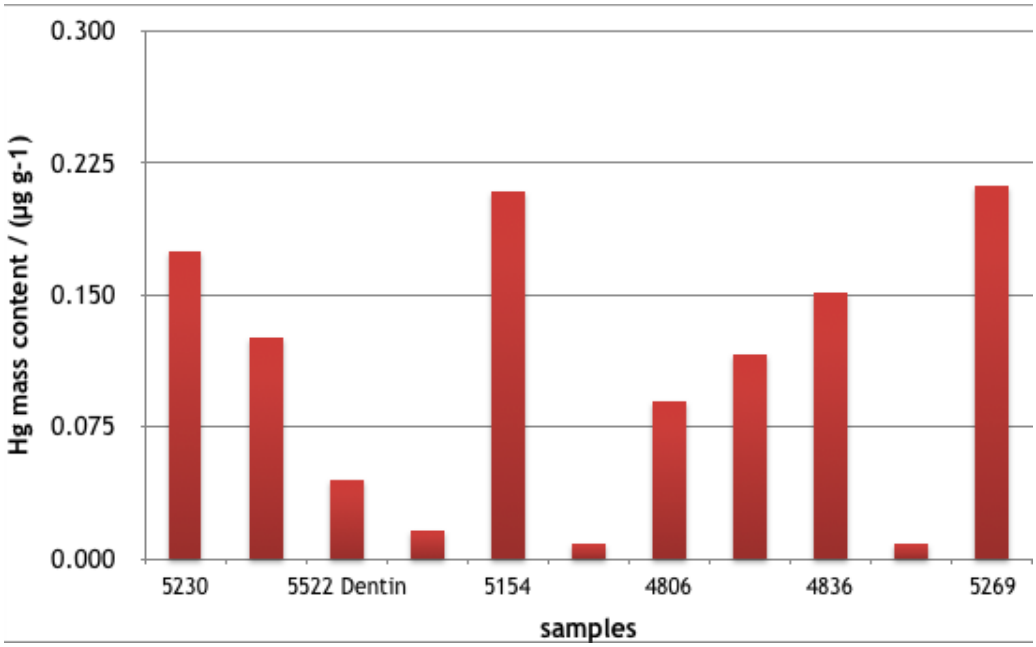
Sr88	38.81	40	36.14	41.72	42.77	42.61
Cd111	0.21	0.44	0.25	0.29	0.46	0.179
Hg200	1.26	1.89	4.09	5.9	4.05	3.68
Hg201	0.8	1.25	3.08	4.29	3.02	2
Hg202	1.57	2.4	7.96	16.56	19.39	310.85
Pb208	23.74	800.49	23.04	20.48	31.21	15.02
GLITTER!: Mean Raw CPS background subtracted.						
Element	Pompeii27-1	Pompeii27-2	Pompeii27-3	Pompeii27-4	Pompeii27-5	Pompeii27-6
P31	17511234	14619041	8413028	9929521	8902767	11102386
Ca43	656266	595480	369115	412500	379009	445741
Sr88	468395	438110	245426	316684	298319	349627
Cd111	23	44	15	19	29	13
Hg200	90	108	126	172	88	73
Hg201	40	52	71	99	56	37
Hg202	92	102	158	246	135	95
Pb208	89454	2736166	48808	48462	67859	38407

GLITTER!: Trace Element Concentrations MDL filtered.							
Element	stdMAPS-13	stdMAPS-14	stdMAPS-15	stdMAPS-16	N612-7	N612-8	stdMaps-4a
P31	186012.58	182025.77	185911.86	178658.56	40.25	43.18	179661.89
Ca43	338299.75	338299.63	338299.63	338299.59	85042.16	85042.17	338299.59
Sr88	3111.8	3092.1	3127.16	3092.31	82.23	80.77	3080.81
Cd111	25.62	24.25	27.55	25.2	26.78	25.18	24.73
Hg200	48.96	79.42	225.82	<****	<****	<****	<****
Hg201	26.78	31.96	41.41	58.37	18.56	<****	<****
Hg202	<****	<****	<****	<****	<****	<****	<****
Pb208	231.3	228.05	242.64	223.56	34.32	34.67	225.49
GLITTER!: 1 sigma error.							
Element	stdMAPS-13	stdMAPS-14	stdMAPS-15	stdMAPS-16	N612-7	N612-8	stdMaps-4a
P31	34396.89	34004.08	35084.34	34058.01	7.95	8.56	35299.19
Ca43	10770.63	10771.3	10771.24	10761.42	2748.65	2745.89	10780.75
Sr88	165.91	166.18	169.41	168.83	4.56	4.52	172.3
Cd111	6.93	6.65	7.66	7.09	7.64	7.27	7.26
Hg200	1208.32	3301.29	28249.02	26973.91	468.97	151.09	615.07
Hg201	323.24	491.17	867.39	1894.8	1300.01	65268.64	6160.69

Hg202	7338.29	1645.19	703.73	367.59	38.31	21.75	134.32
Pb208	37.01	36.93	39.77	37.08	5.77	5.9	38.74
GLITTER!: Minimum detection limits (99% confidence).							
Element	stdMAPS-13	stdMAPS-14	stdMAPS-15	stdMAPS-16	N612-7	N612-8	stdMaps-4a
P31	5.67	4.82	4.54	3.6	3.02	2.8	3.68
Ca43	31.55	37.26	30.24	28.12	25.45	24.23	36.18
Sr88	<0.00000	0.0087	0.00619	<0.00000	<0.00000	<0.00000	0.00906
Cd111	<0.00000	<0.00000	<0.00000	<0.00000	0.0405	<0.00000	0.0769
Hg200	2.14	4.33	13.97	<0.00000	<0.00000	<0.00000	<0.00000
Hg201	1.43	2.38	3.39	4.6	6.82	<0.00000	<0.00000
Hg202	<0.00000	<0.00000	<0.00000	<0.00000	<0.00000	<0.00000	<0.00000
Pb208	0.0137	0.0143	0.0181	0.00945	0.0104	0.0073	0.00566
GLITTER!: Trace element concentrations normalised to chondrite.							
Element	stdMAPS-13	stdMAPS-14	stdMAPS-15	stdMAPS-16	N612-7	N612-8	stdMaps-4a
P31	80.88	79.14	80.83	77.68	0.0175	0.0188	78.11
Ca43	25.06	25.06	25.06	25.06	6.3	6.3	25.06
Sr88	261.5	259.84	262.79	259.86	6.91	6.79	258.89
Cd111	25.36	24.01	27.28	24.95	26.52	24.93	24.49
Hg200	83.69	135.76	386.01	0	0	0	0
Hg201	45.78	54.64	70.78	99.78	31.73	0	0
Hg202	0	0	0	0	0	0	0
Pb208	63.37	62.48	66.48	61.25	9.4	9.5	61.78
GLITTER!: Mean Raw CPS background subtracted.							
Element	stdMAPS-13	stdMAPS-14	stdMAPS-15	stdMAPS-16	N612-7	N612-8	stdMaps-4a
P31	8167338	7905922	7730902	8568655	2230	2396	7163391
Ca43	212739	210788	202147	233536	67933	68167	195116
Sr88	1209791	1191321	1155645	1320447	40636	40063	1099706
Cd111	1062	994	1082	1141	1401	1319	930
Hg200	678	655	600	620	113	100	572
Hg201	400	376	346	369	63	55	340
Hg202	893	815	753	804	150	122	732
Pb208	91310	89185	90980	96821	17198	17426	81542

Vienna Egyptian data:

		Sr	RSD (%)	Hg	RSD (%)	Pb	RSD (%)
concentration		µg/g		µg/g		µg/g	
5230		413	3	0.174	25	4.7	60
5250		1011	4	0.126	43	39.6	43
5522 Dentin		317	21	0.045	21	19.3	23
5522 Enamel		104	5	0.016	20	1.3	37
5154		327	8	0.209	50	48.3	50
1981		1491	10	0.008	25	0.5	37
4806		1168	5	0.090	50	43.6	63
4811		1237	5	0.116	50	50.2	21
4836		675	4	0.151	50	320.9	41
4970		189	3	0.008	32	0.3	43
5269		653	5	0.212	44	16.0	43
relative to maximum							
		Sr		Hg		Pb	
5230		0		0.821		0.0	
5250		1		0.592		0.1	
5522 Dentin		0		0.213		0.1	
5522 Enamel		0		0.074		0.0	
5154		0		0.984		0.2	
1981		1		0.038		0.0	
4806		1		0.423		0.1	
4811		1		0.547		0.2	
4836		0		0.712		1.0	
4970		0		0.040		0.0	
5269		0		1.000		0.0	


0.300
0.225
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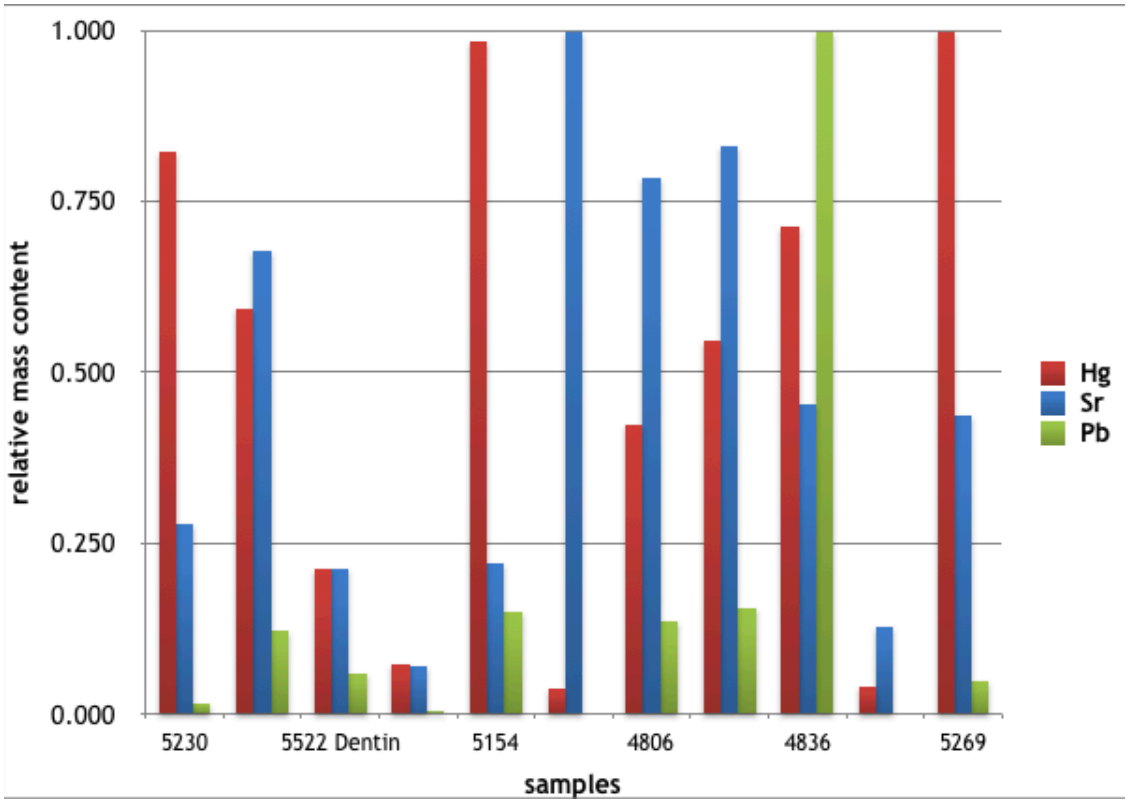
0.075
0.000
Hg mass content / (µg g-1)
5230
5522 Dentin
5154
4806
4836
5269
samples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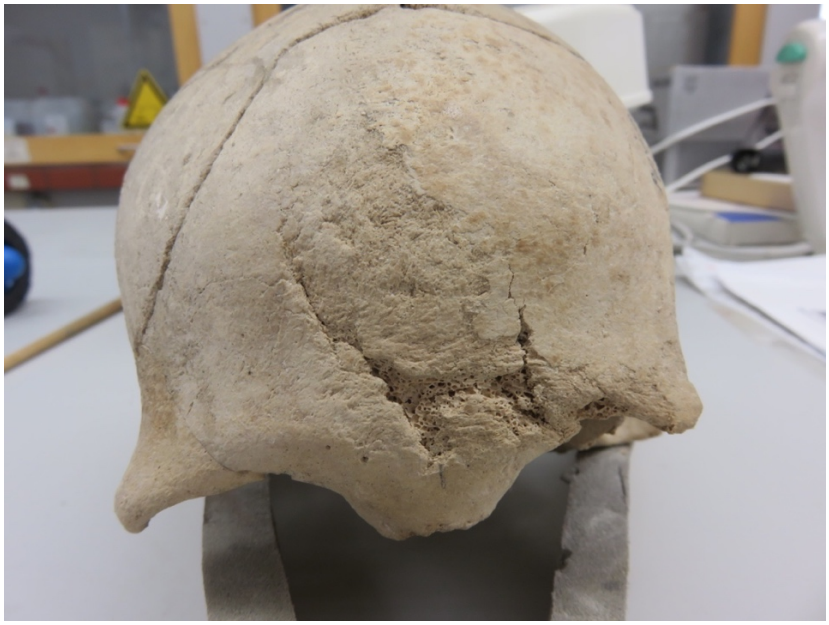
0.750
0.500
0.250
0.000
relative mass content
5230
5522 Dentin
5154
4806
4836
5269
samples
Hg
Sr
Pb

Lódz BK5 and Kolonia mercury concentrations

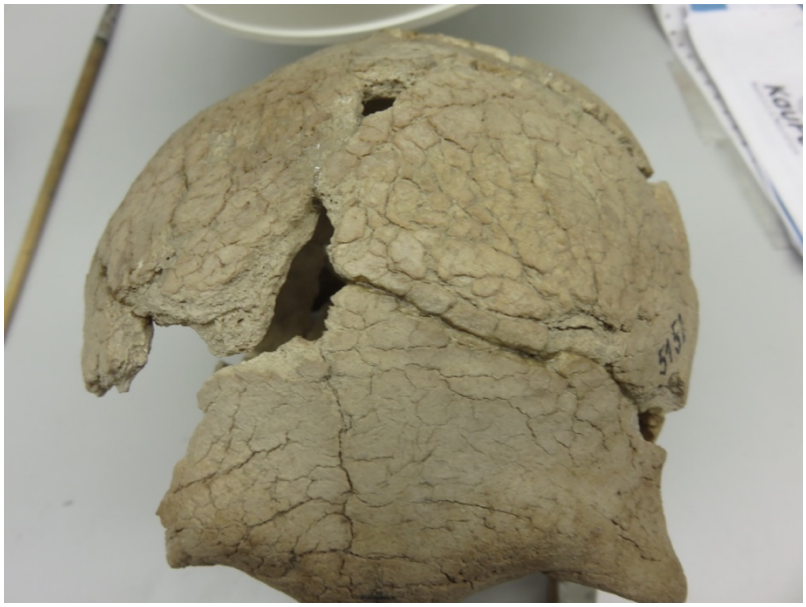
Nr próbki	Nazwa	[Hg] µg/kg	PPM	
1	KOL1 sample0	10.1	0.0101	
2	KOL-36	16.8	0.0168	
3	KOL-43	16.6	0.0166	
4	KOL-25	58.4	0.0584	
5	KOL-29	17.3	0.0173	
6	KOL-30	12.3	0.0123	
7	KOL-52	33.6	0.0336	
8	KOL-54	12.3	0.0123	
9	BK5-8 sample 0	16.4	0.0164	
10	BK5-24	13.5	0.0135	
11	BK5-175	43.3	0.0433	
12	BK5-179	45.4	0.0454	
13	BK5-152	18.5	0.0185	
14	BK5-87	21.8	0.0218	
15	BK5-56	16	0.016	
16	BK5-18	212.1	0.2121	
17	BK5-35	19	0.019	
18	BK5-9	25.5	0.0255	
19	BK5-163	14.9	0.0149	
20	BK5-77	14.9	0.0149	
21	BK5-43	18.1	0.0181	
22	BK5-74	36.4	0.0364	

Natural History Museum Vienna Egyptian skull photos:

GIZA 5154



Giza 5151

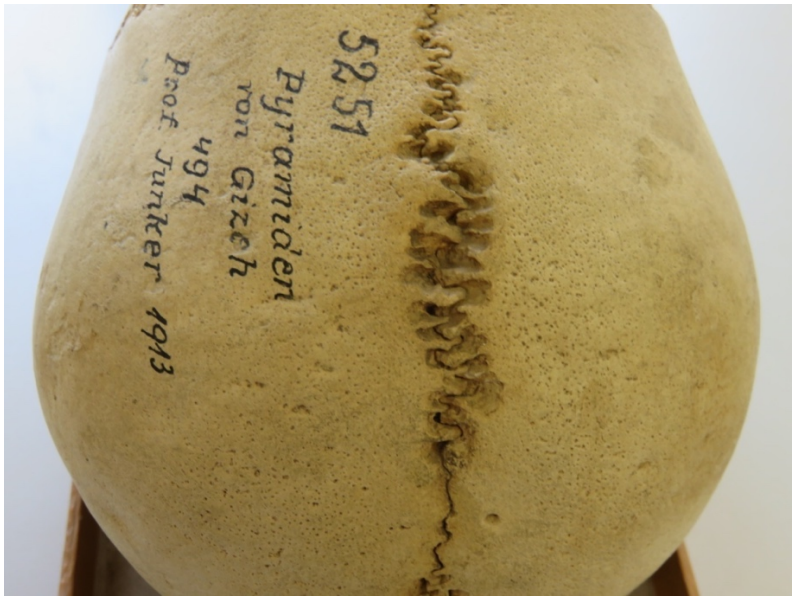


Giza 5171

Giza 5171 side view

Giza 5174

Giza 5251

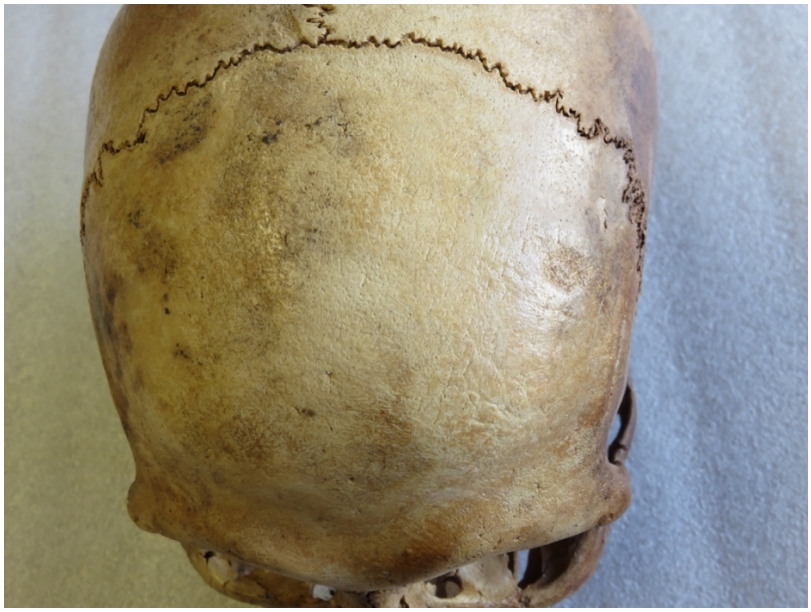


Giza 5262

Giza 5319

El Kubanieh 4970

El Kubanieh 4970



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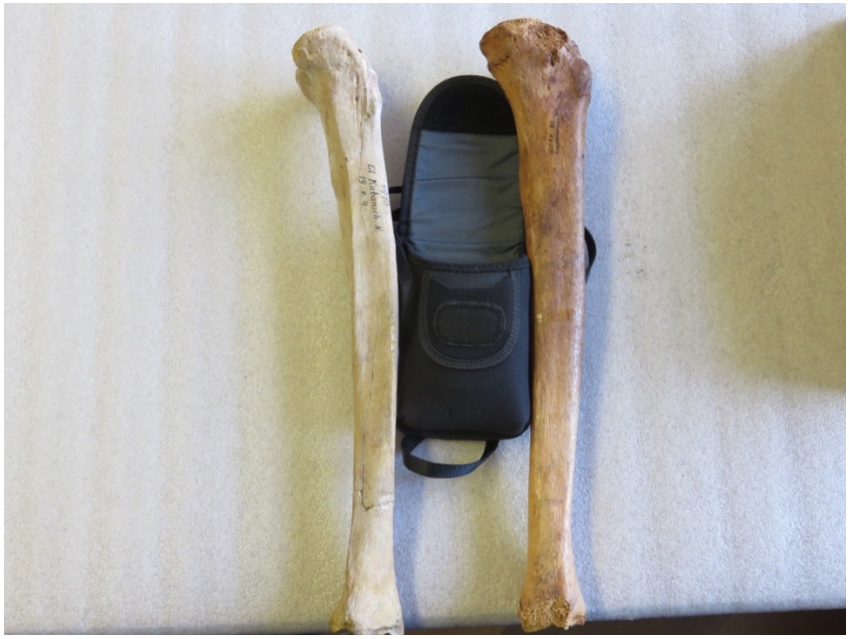

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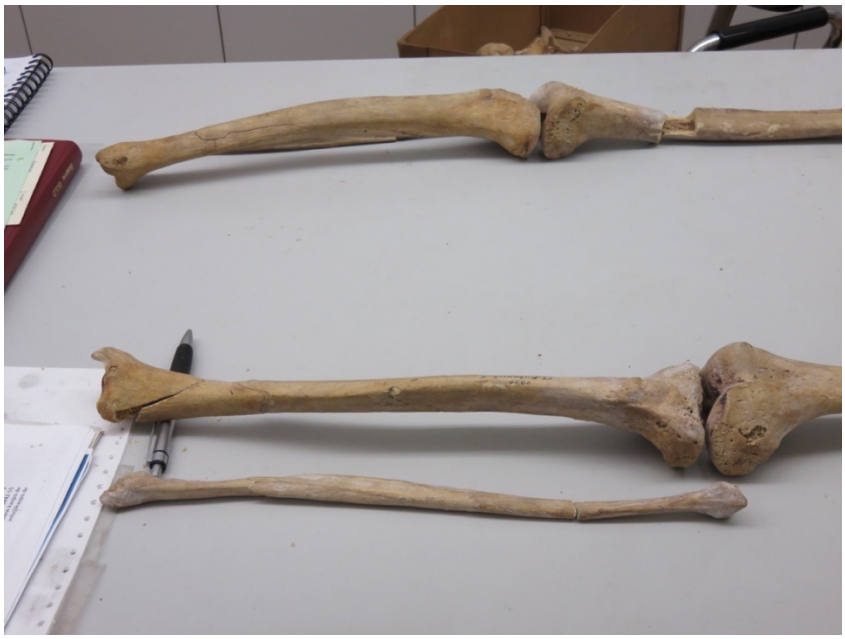

El Kubanieh 4816

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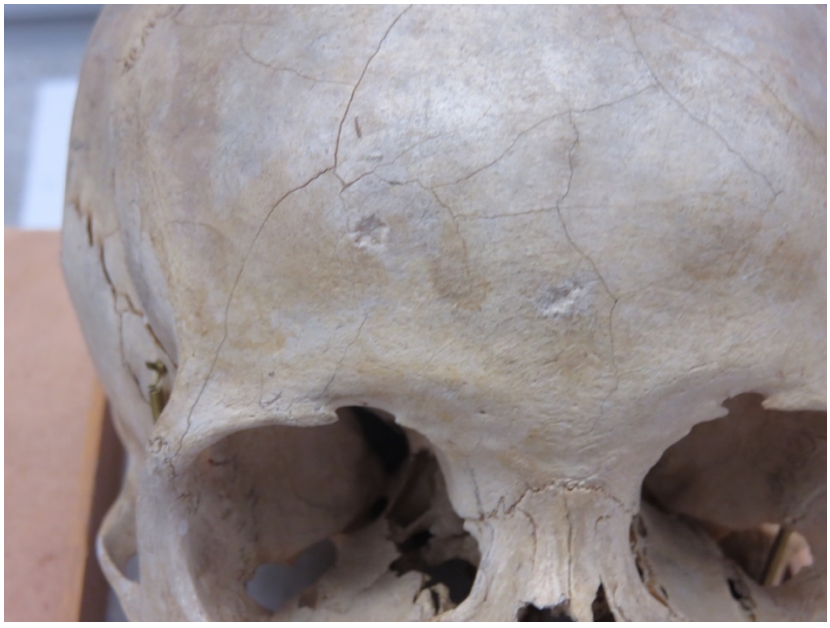


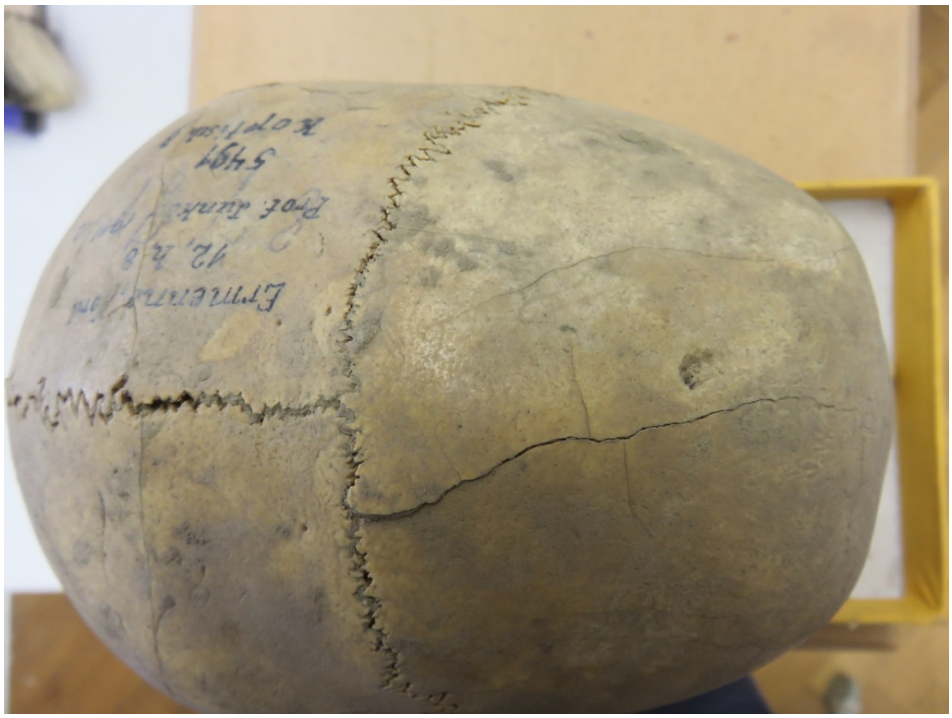
Ermenne Nord 5669

Ermenne Nord 5669

Ermenne Nord 5502

Ermenne Nord 5502

Ermenne Nord 5491



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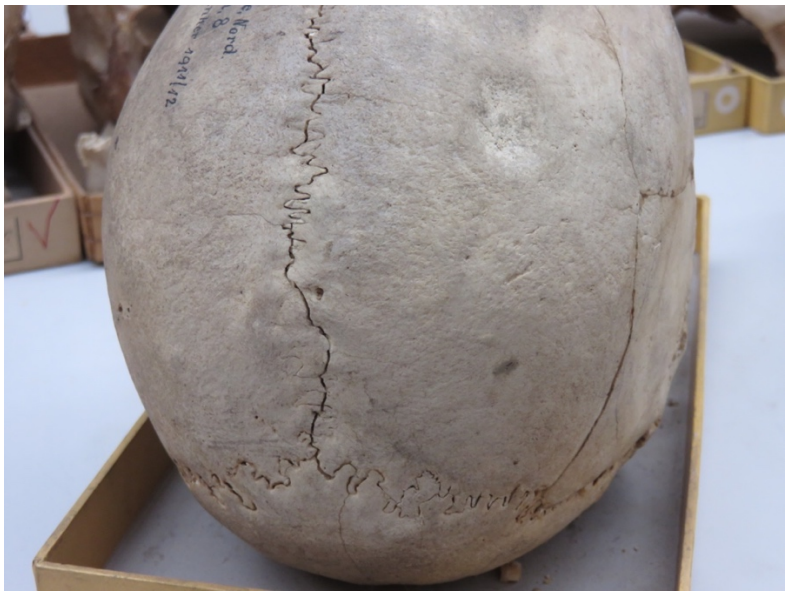

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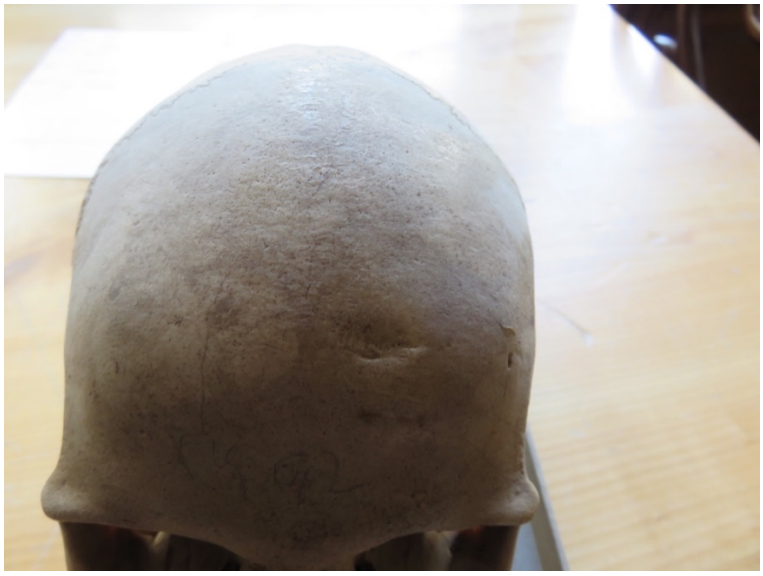


Ermanne Aegypten 7174

Ermenne Aegypten 7800

Ermenne Aegypten 7800

University of Lódź BK5 and Kolonia Medieval collection

BK5- 22

BK5 6/5

BK5 32/5

BK5 32/5

BK5 32/5

BK5 43/5

Bk5 179/5

BK5 76/5

BK5 98/5

Kolonia 36

Kolonia 6

BK5 18

BK5 18

BK5 18

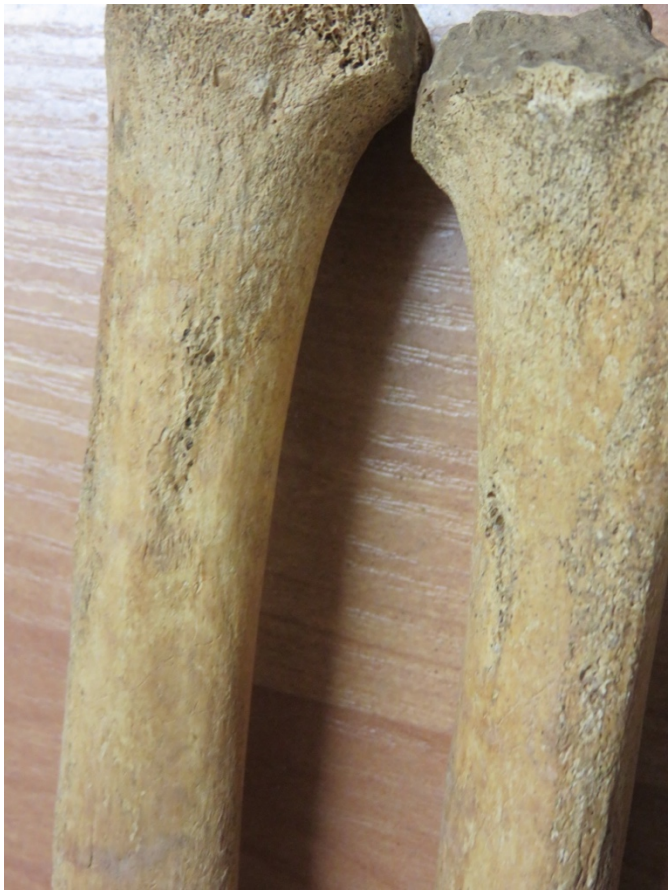
BK5 18

BK5 64/5

BK5 87

BK5 96

BK5 96



BK5 161

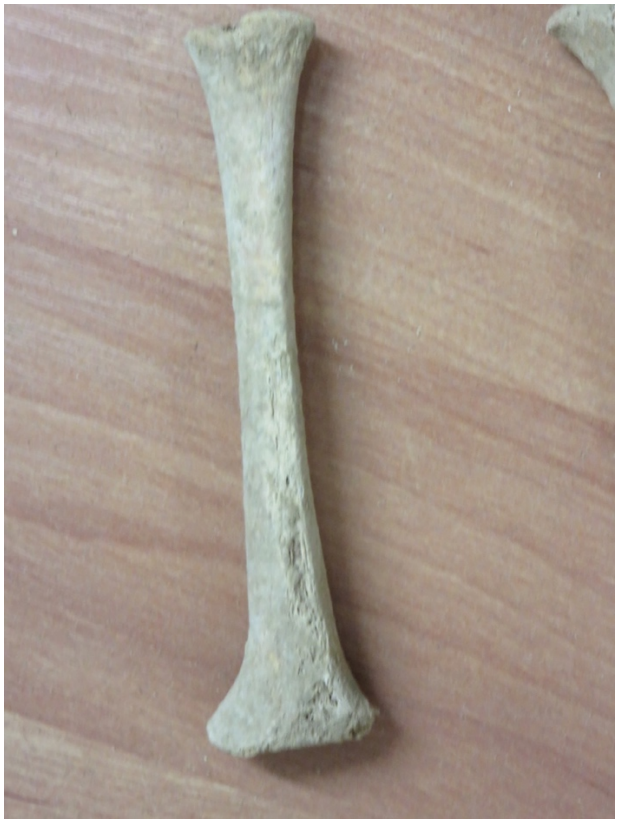
BK5 152

BK5 152

Kolonia 30

Kolonia 30

Kolonia 18



Kolonia 18

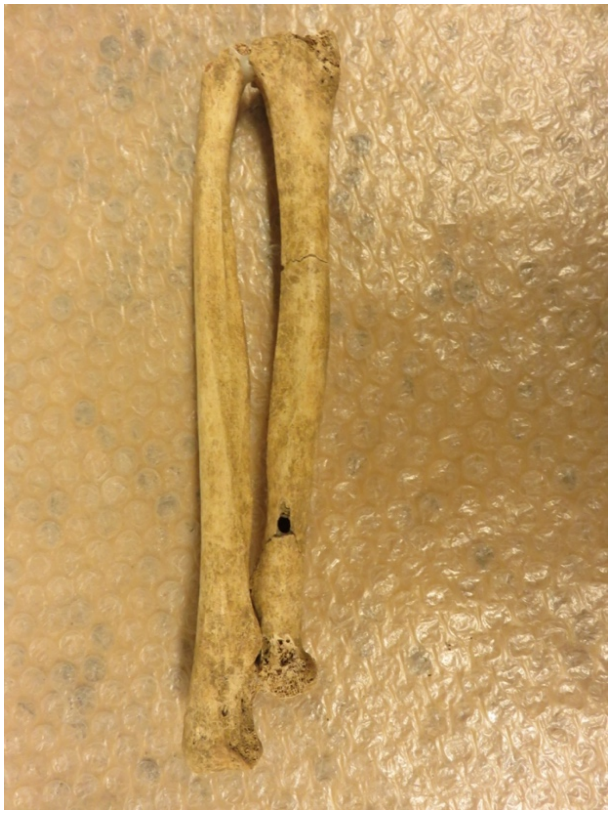
Kolonia 54

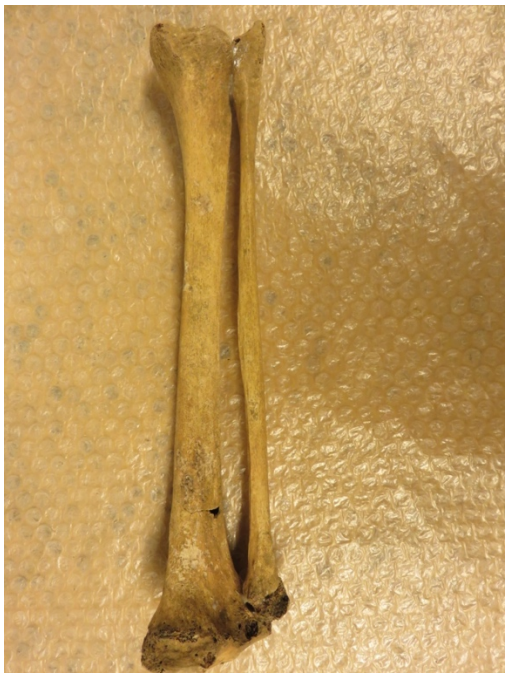
Kolonia 54

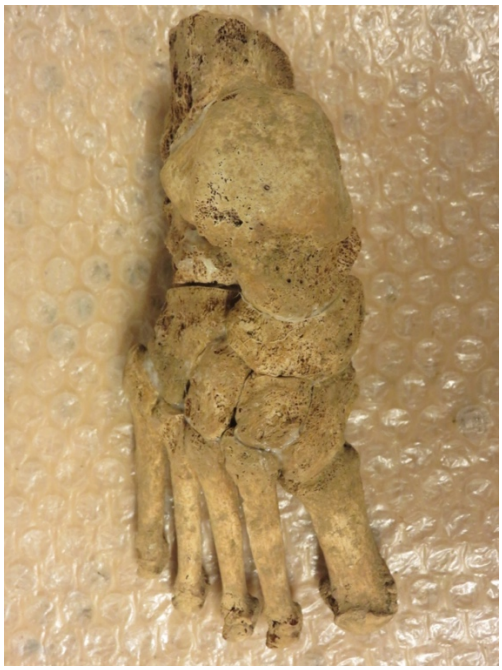
Kolonia 54

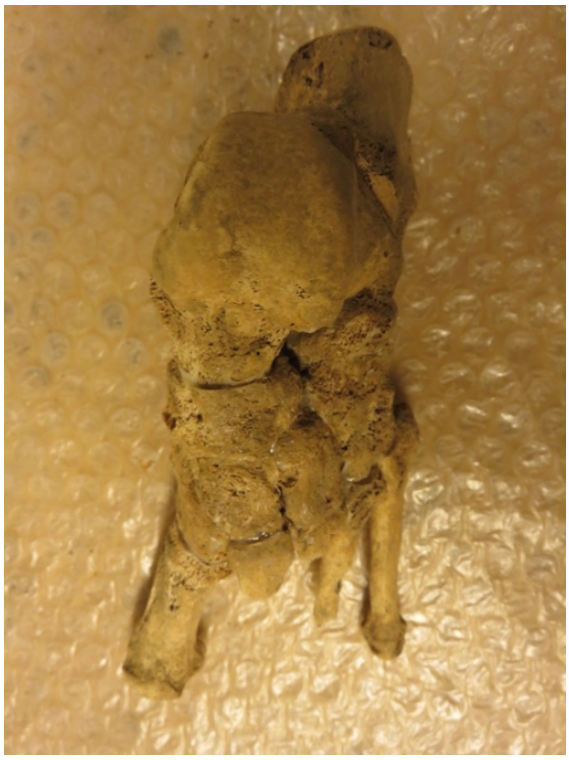
University of Copenhargen Leprosy Collection

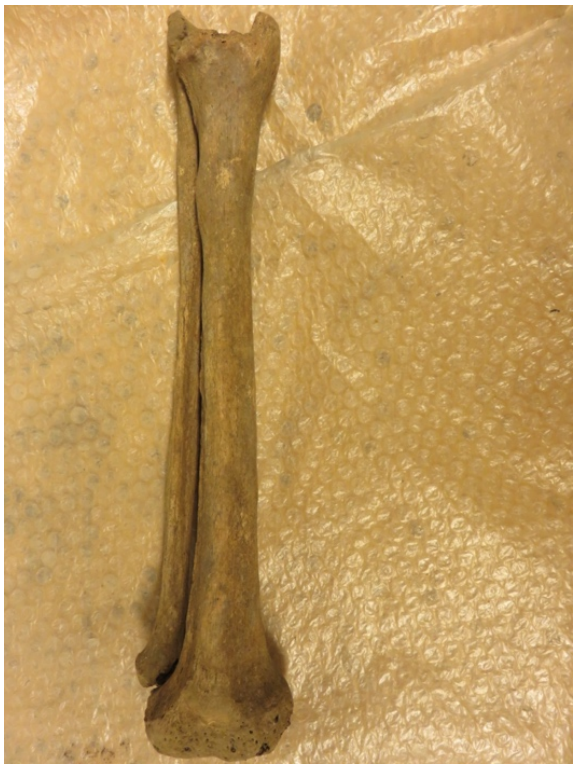
Aaderup 80



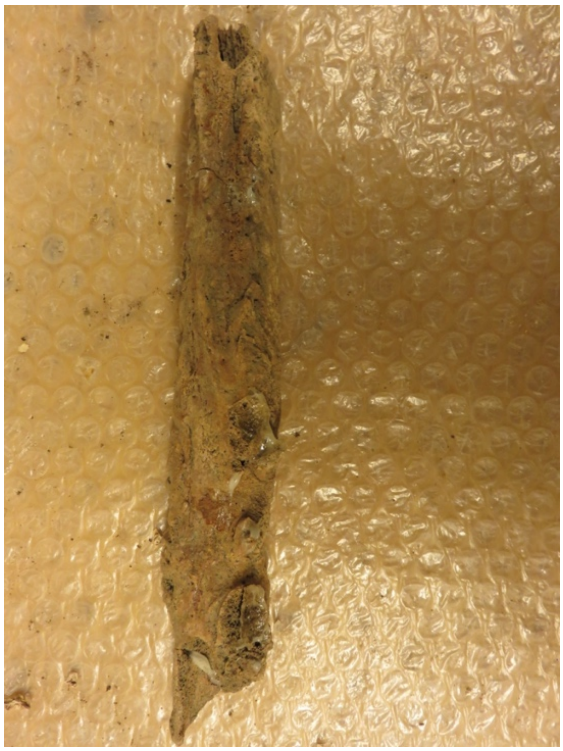




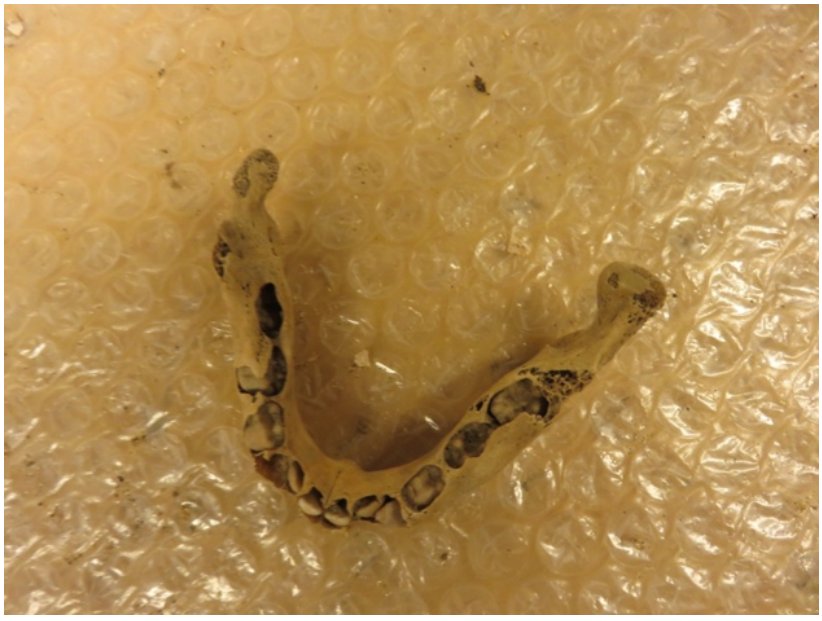


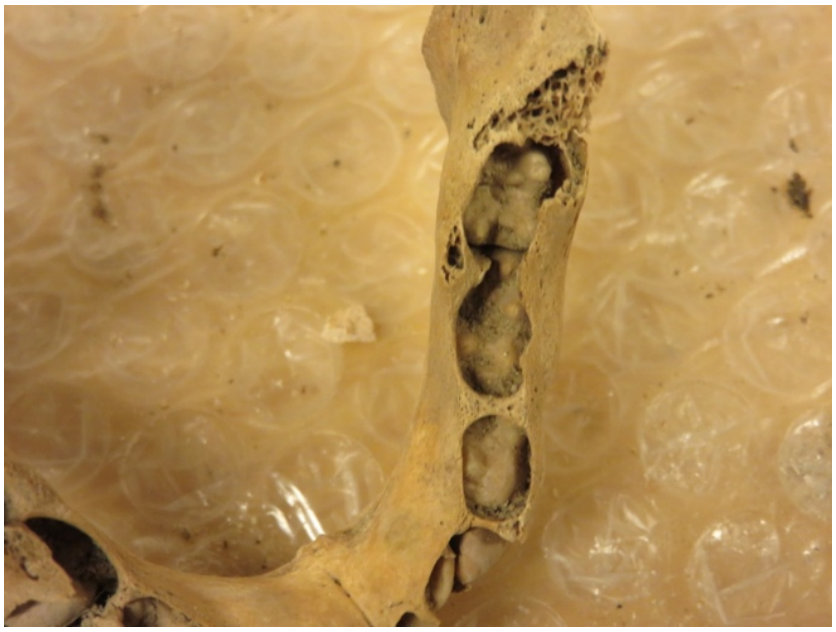


Aaderup 49



Aaderup 611-612

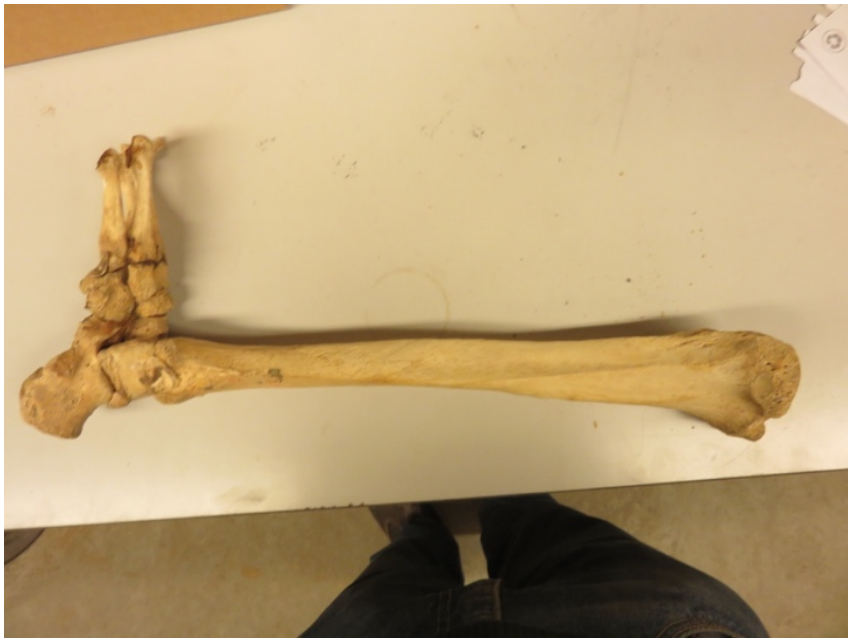


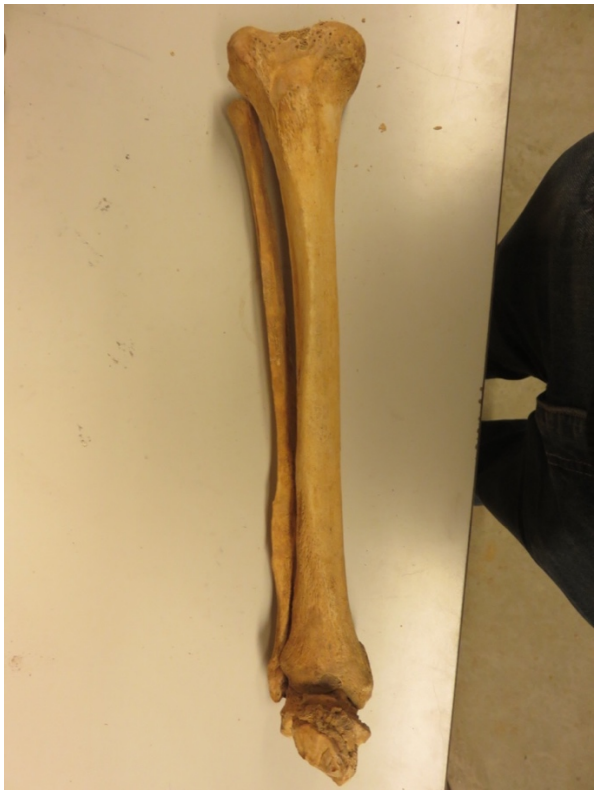


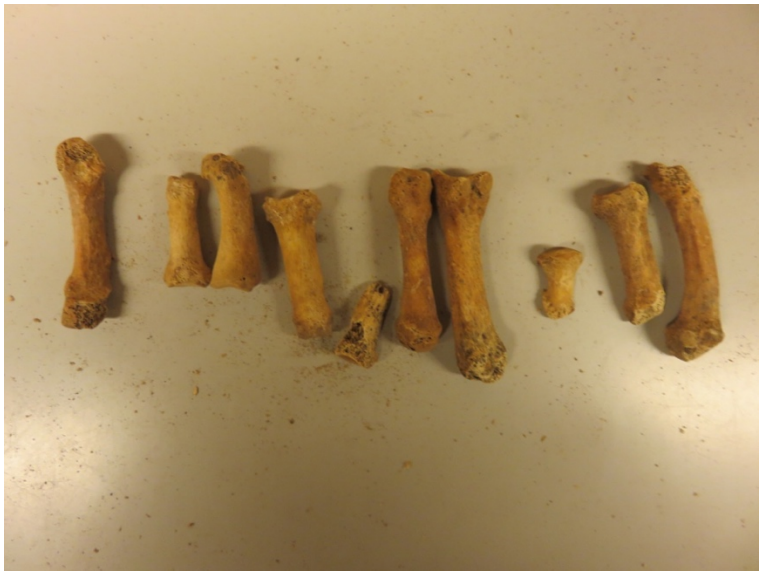
Aaderup 393


0899
Khm J.nr
25:
0899 Tjørby
KHM J.nr
25:33
Tjørby
Grav
Løsfund
AS
Mus.nr.
mand kvinde ukendt
bemærk.
0-1 1-18 >18 ukendt

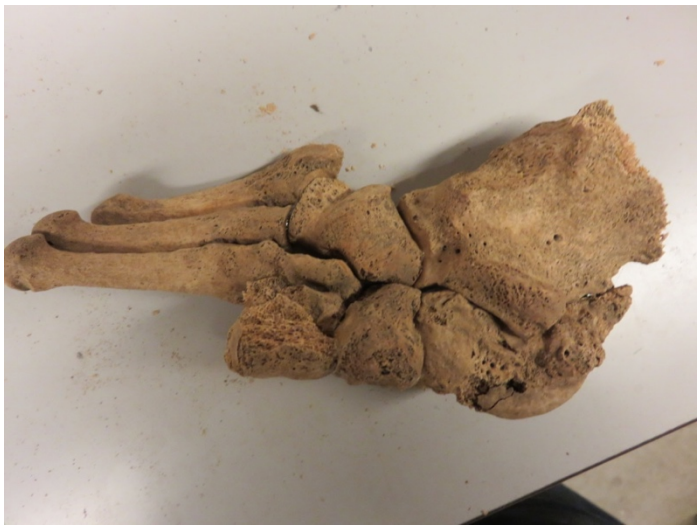
Aaderup 25

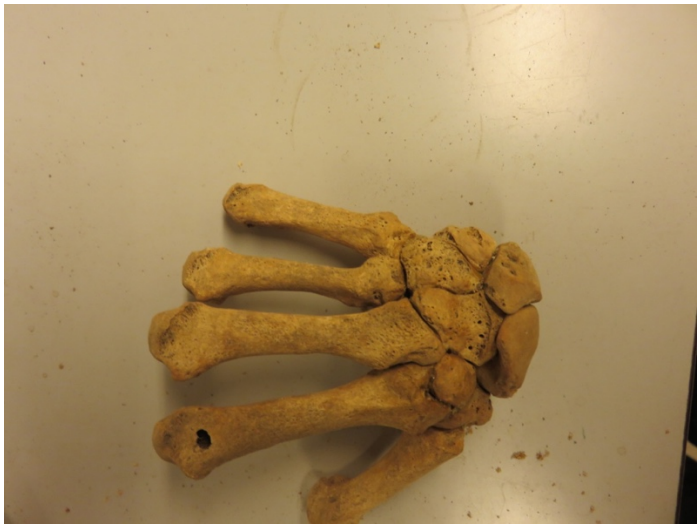






Aaderup 312





Aaderup 69