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‘Til Poison Phosphorous Brought them Death’:
A potentially occupationally-related disease in a post-medieval skeleton from north-east England.

Charlotte A Roberts, Anwen Caffell, Kori L Filipek-Ogden, Rebecca Gowland, Tina Jakob

Department of Archaeology, Durham University, South Road, Durham, DH1 3LE

c.a.roberts@durham.ac.uk
a.c.caffell@durham.ac.uk
k.l.filipek-ogden@durham.ac.uk
rebecca.gowland@durham.ac.uk
betina.jakob@durham.ac.uk

Corresponding author: c.a.roberts@durham.ac.uk(+441913341154)
Abstract

This paper describes the pathological changes observed on the skeleton of a c.12-14 year old person buried in a north-east England Quaker cemetery dated to AD 1711-1857. Bone formation (woven and lamellar) and destruction are present mainly in the mandible, clavicles, sternum and scapulae, long bones of the right arm, left ribs, spine, ilia, and the femora and tibiae. Differential diagnoses of tuberculosis and other pulmonary disease, smallpox, actinomycosis, neoplastic disease, and “phossy jaw” are considered. While the pathological changes could represent all previously described diseases, and be associated with the insalubrious conditions in which this person lived, it is also possible that this person worked in the matchmaking industry known to be present in the region at the time. Attention is drawn to the previously overlooked condition “phossy jaw” caused by phosphorus poisoning, which was strongly associated with this industry. While matchstick making was an industry often associated with women and girls, DNA analysis of a bone sample did not successfully identify biological sex. Two dental calculus samples from this person were analysed for phosphorus, and comparisons were made with samples from the same and a different site; the levels did not indicate the person was exposed to phosphorus more than any of the other people. However, the pathological lesions described also have relevance in a clinical context, because “phossy jaw” has been observed in living populations, arising as a consequence of ingesting phosphorous contained within some pharmaceuticals used for treating neoplastic disease and osteoporosis.

Keywords: “phossy jaw”; dental; infectious; metabolic; matchmaking, Coach Lane, North Shields
1. Introduction

Work has always been part of everyday life, whether this purely involved the procurement of food for survival or working in specific industries. Ideally, employment also provides an environment conducive for the social well being and good health of the employed. The impact of work on skeletal health has been of interest to bioarchaeologists for many years, and investigated using various approaches and types of data (see Table 1). Indeed, support for such studies has been aided by “quick reference” texts (e.g. Capasso et al 1998) that facilitate identification of specific “occupations”.

<table>
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Table 1 Summary of previous work exploring “occupation” through bioarchaeology

However, there has also been a more critical appraisal of the value and rigor of such studies (e.g. see Waldron 1994, Jurmain 1999, Jurmain and Roberts 2008, Jurmain et al 2012, Meyer et al 2011, Henderson and Cardoso 2013). All of these “markers” in the skeleton can be caused by a number of variables. For example, osteoarthritis and entheseal changes are a feature of increasing age, and therefore the direct association between bone changes and specific
“occupations” is now being approached more cautiously. Using multiple markers (and controlling for age) to see if different sets of data produce the same conclusions can be used to mitigate the biases involved when only one “class” of information is studied, for example osteoarthritis. Additionally, various questions related to the work that people did in the past need to be considered, alongside the “markers” identified: for example, at what age did the person start work, was it their only job, how long each day did they perform the task, and was the work seasonal.

A more fruitful way of exploring the impact of different types of “activity” on the skeleton, would be to work from the premise of the known impact that specific occupations have on the bones and teeth of those who did the work. While this paper uses the traditional bioarchaeological approach of recording pathological changes in skeletal remains and producing differential diagnoses, it then links these skeletal lesions to a specific occupation known to have been practiced in the time and place this person lived. Therefore, the aim of this paper is to present some unusual pathological changes in a skeleton from a post-medieval Quaker burial site in north-east England, discuss potential differential diagnoses, and consider possible causes, including the impact of a specific occupation.

2. Material and methods

2.1 The skeleton studied

The skeleton (Burial 69 [516]) was excavated in 2010 by Pre-Construct Archaeology Ltd (PCA 2012, Proctor et al 2014) from Coach Lane, North Shields, North Tyneside, a Quaker burial ground dated to c.1711-1857 AD (Figures 1 and 2). Two hundred and forty-four individual graves and 18 charnel features, with 236 skeletons and several hundred disarticulated bones were recovered (Figure S1). Over half the skeletons were adults of mid/older age (Langthorne 2011), and a large percentage of the non-adults were five years or less at death; there were approximately equal numbers of adult males and females. The Quaker movement was founded in 1652 in north-west England. It represented members of a family of Christian religious movements collectively known as the Religious Society of Friends. The social status of the people from this burial population is not yet known, but the Quakers were devoted to peaceful principles, and adopted plain dress, speech and living, in order to separate themselves from, what they believed to be, a corrupt world (Dandelion 2008).

2.2 Context

The period in which these people lived and died in North Shields was one of ‘massive changes in all spheres of human activity and experience, including health and disease’ (Roberts and Cox 2003:358). The post-medieval period in England heralded dramatic population growth (excess of births over deaths), migration, urbanization, industrialization and the commercialization of agriculture.
By 1850, 50% of the population lived in urban contexts (Scott and Duncan 1998), with immigration to towns and cities driven by the demand for a large work force in industrial centers. Developments in transport affected markets and commerce and the distribution of foodstuffs, the mobility of people, the introduction of new pathogens and exposure of immigrants to new diseases (Roberts and Cox 2003:287-358). Coastal towns and cities, like North Shields and Newcastle-upon-Tyne, also experienced an expansion in sea trade, transformed by the introduction of steam engines and the enlargement of existing harbors, and the establishment of new ones. However, this was not a time of economic prosperity for all; inequalities in wealth distribution increased and the real wages of unskilled laborers did not improve until the middle of the 19th century (Feinstein, 1998). The industrialized cities in the north of England tended to be focused on particular industries such as coal or cotton, with air and water often being polluted. Problems of waste disposal exacerbated the challenges of polluted and crowded living conditions. This was accompanied by poor quality housing, sanitation and working conditions, and there was often much poverty, with under- and malnutrition (1833, Chadwick 1842). The dreadful living conditions endured by the poor in many cities, including suburban areas, led to the introduction of a series of public health acts in the later 19th century (Sretzer 1988, Tulchinsky and Varavikova 2009). In particular, ‘the post-medieval (1540-1900) period in the North-East was one of radical and deep-rooted change, perhaps more so than any other period’ (Petts and Gerrard 2006:85). This encompassed rural to mainly urban living, and a move from horse, to water, to steam, and finally combustion power. Industry considerably expanded and attracted workers from elsewhere such as Scotland and Ireland. This industrial expansion both reflected and caused technological changes, in addition to meeting increases in demand for various products in Britain and elsewhere. All of these industries presented hazards to health.

2.3 Methods

A skeletal and dental inventory of Burial 69 was undertaken, and the state of preservation assessed (McKinley 2004). Age-at-death was determined using the dentition (development and eruption: Smith 1991, Moorrees et al 1963), maximum long bone lengths (Maresh 1970) and skeletal development (Scheuer and Black 2000:6-12, and references within). The skeleton was a non-adult and therefore sex estimation was not attempted because of the inaccurate nature of the methods available (Scheuer and Black 2000:15-17); aDNA analysis was attempted using a tooth from the skeleton to ascertain whether the person was male or female. This was relevant to discussions about the aetiology of the pathological lesions identified (see 4.4), but the analysis was inconclusive (Abigail Bouwman, pers. comm., February 2015). The dentition and skeletal remains were assessed for antemortem and perimortem lesions indicative of disease or trauma according to Connell (2004) and Roberts and Connell (2004), respectively. This included recording bone formation (specifying whether woven or lamellar) and destruction, and dental alterations (e.g., caries and enamel
hypoplasia). Full details of all pathological changes observed can be found in Supplementary Information S1. Radiographs were taken using a Carestream Point-of-Care digital CR reader radiograph machine set to 70kVp and 0.500mAs, but no useful features for diagnostic purposes could be identified as many bones were infiltrated with soil. In addition, elemental analysis of calculus samples from the teeth of Burial 69 was conducted, particularly focusing on phosphorus (again, see 4.4). Calculus was used because of the recent work showing how well calculus preserves many different types of “materials” i.e. they become fossilized (e.g. Warinner et al 2014). Bone was not used for this analysis because of the possibility of diagenesis; if phosphorus was a general environmental hazard in the area at the time these people lived, the cemetery soil may have be contaminated with it; additionally, calcium phosphate is a normal component of bones and teeth. Therefore it would not be possible to differentiate between phosphorus that naturally occurs in bones or teeth and phosphorus introduced from the environment. In addition, calculus should not have incorporated any natural phosphorus from the tooth with which it is associated. Calculus was also used because sampling another tooth (following the one used for aDNA analysis) was not considered appropriate. More details of this analysis and the results are included in Supplementary Information S2, including Figure S2.1 (scanning electron micrograph of a calculus sample from Skeleton 69), Table S2.1 and Figure S2.2 (phosphorus content of calculus samples from Skeleton 69, two other skeletons from Coach Lane, and control samples from a late medieval site from York).

3. Results

3.1 Preservation

The skeleton was fairly well preserved with most parts of the body represented, apart from the feet (Figures S2a and 2b). However, the skull, scapulae, pelvis and spine were fragmentary and many long bone metaphyses and epiphyses were missing postmortem. In general the macroscopic bone preservation was good, graded as 2-3 (McKinley 2004).

3.2 Age-at-death

Age-at-death based on the dentition was 11.5-13.5 years. However, the left ulna measured 195mm, suggesting an age of ~10 years according to Maresh (1970, cited in Scheuer and Black 2000:308). The length of the other long bones, although too damaged for accurate measurement, appeared shorter than expected when compared to dental development. This was also the case when compared to other skeletons of the same dental age from a geographically close medieval cemetery in York (Figure S3).
3.3 Pathological changes

Pathological lesions were present in the form of bone formation and bone destruction (see Figures S2a and b). A more detailed description is provided in Supplementary Information S1.

3.3.1 Skull and mandible

On the left side of the mandible, a large lytic lesion has destroyed the medial and lateral surfaces of the posterior half of the body and inferior half of the ramus (Figure 3). Within the destructive lesion there is a large irregular rough-textured “mass” situated beneath the sockets for the second and third permanent molars and occupying the entire width and height of the body (Figure 4). Extensive woven bone deposits, partially remodeled into lamellar bone in places, coat the medial and lateral surfaces surrounding the destructive lesion. Thick woven bone surrounding irregular destructive lesions is also present on the lateral and medial sides of the right mandibular ramus, focused on the coronoid process; remodeled lamellar bone is present at the margins.

Elsewhere in the skull, new bone formation is present on the endocranial surface of the frontal and occipital bones, on the posterior surface of the zygomatic bones, and on the lateral surface of the right orbit. Fine porosity is present on the ectocranial surfaces of the occipital and right temporal bones, both zygomatic bones, and on both orbital roofs.

A total of 21 teeth have enamel hypoplasia, while dental caries occurs in two teeth (Table S1.1); dental calculus is present on 20 teeth.

3.3.2 Postcranial skeleton

Upper limb

Pathological changes are predominantly focused on the right elbow. Extensive woven bone deposits (partially remodeled) coat the antero-distal third and posterior two-thirds of the right humerus, and proximal thirds of the radius and ulna (Figure 5). Rounded lamellar bone nodules are interspersed with the woven bone on the latter two bones. The metaphyseal surfaces of the distal humerus and proximal radius also display destructive lesions. The long bones of the left arm are normal. Further new bone formation is present in both shoulders, with woven bone present on both clavicles and the right scapula.

Lower limb

Both ilia display partially remodeled woven bone, while the femora and tibiae have possible lamellar bone formation along much of their shafts. The femoral shafts also displayed antero-posterior bowing (Figure S4).
Thoracic cavity

Four vertebrae (C6, C7, T5, and T6) have destructive lesions on their left apophyseal joints accompanied by woven and lamellar bone on their posterior left laminae (e.g. see Figure S5). Extensive woven and nodular lamellar bone formation is present on the neural arches of T12, L1-3, and L5, and on the anterior body of S1, extending onto the left ala (e.g. see Figure S6). A mix of woven and lamellar bone is present on the visceral surfaces (predominantly necks) of six left ribs (4-6, 8-10), and oval lytic lesions are present on the visceral surfaces of the necks and/or shafts of five left ribs (2, 4-6, and 8) – see Figure S7.

Overall the pathological changes occurred on the skull, including the maxilla, and the mandible, the teeth, clavicles, scapulae, sternum, left ribs, spine, right arm bones, and the left and right ilia and leg bones.

4. Discussion

The lesions described in the skeleton of Burial 69 suggest a number of differential diagnoses. Firstly, this person had many teeth affected by enamel hypoplasia, a condition related to stress during dental development (Hillson 1996:165f), most likely due to dietary deficiency or childhood disease. This person may have suffered both; the left ulna and both femora were small compared to dental age suggesting delayed growth.

4.1 Evidence of metabolic bone disease

Pitting and new bone formation on the endo- and ectocranium may suggest subperiosteal hemorrhage from fragile blood vessels, consistent with a diagnosis of scurvy (vitamin C deficiency) (Ratanachu-Ek et al 2003, Ortner and Ericksen 1997, Ortner et al 1999, Ortner 2003:384-7, Brickley and Ives 2006, 2008:57). The new bone formation on the femora and tibiae may also be a consequence of scurvy, as may the new bone formed on the ilia (although cited as a rare scorbutic bone change: Fraenkel 1929 in Ortner 2003). The unilateral nature of the new bone formed around the right elbow is less characteristic of scurvy, as is the extensive new bone formation and destruction on the mandible. The endocranial and ectocranial bone changes described above may be also related to vitamin D deficiency (rickets) along with the abnormal bowing of the femora (Pitt 1995:1894, Ortner and Mays 1998, Mays et al 2006). Working long hours indoors can predispose people to developing vitamin D deficiency due to the lack of ultraviolet light on their skins and subsequent lack of formation of the vitamin in the skin. Consequently, calcium and phosphorus are not absorbed and the long bones bend (Pitt 1995). In fact, Hokugo et al (2010) note that in people with osteonecrosis of the mandible there could be an interaction between
bisphosphonates and reduced vitamin D. Therefore, this person may have had rickets prior to contracting “phossy jaw”.

4.2 Evidence of specific and non-specific infections

The bone formation on the right arm bones is extensive, with woven and lamellar bone present. The focus of the lesions is on the elbow joint, which is known to be affected in a high proportion of people who have suffered smallpox, but usually bilaterally (e.g. Cockshott and MacGregor 1959; see also Andrews and Jayan 2011). The pathological changes of the ilia and the first sacral vertebra could be indicative of a psoas abscess, which can be associated with spinal involvement in tuberculosis (TB). However, there is no other evidence of spinal tuberculosis, unless the new bone formation on the neural arches and destruction of apophyseal joints of some of the vertebrae are related. The involvement of the neural arches in TB is rare, although it has been reported clinically (e.g. Kumar 1985, Arora et al 2012).

Bone formation and destruction on the visceral surfaces of the left ribs, suggests a pulmonary condition. There are many conditions that may have led to these lesions, as illustrated by the extensive bioarchaeological literature that addresses rib lesions in skeletons of people with documented causes of death (Kelley and Micozzi 1984, Roberts et al 1994, Santos and Roberts 2001, 2006) and archaeological skeletons (e.g. Molto 1990, Pfeiffer 1991, Kelley et al 1994, Sledzik and Bellantoni 1994, Roberts et al 1998, Lambert 2002, Nicklish et al 2012). While TB may be the more likely cause of rib lesions, these are not pathognomonic for TB. Therefore, anything that might be inhaled and/or cause an inflammatory reaction in the lungs and on the visceral surfaces of the ribs may be implicated (e.g. pathogens or foreign “materials”). This could include infection, such as chronic bronchitis and pneumonia, but also cancer. Urban population density during the Industrial Revolution was known to be high, including in Newcastle-upon-Tyne and its neighboring regions, such as North Shields, thus facilitating the spread of pulmonary TB. Atkins (2000) notes that between 1850 and 1950 there were many deaths from TB due to infected milk. The recognized poor air quality associated with the industries operating in North Shields could also have been responsible for the pulmonary problems they undoubtedly experienced (McCord and Thompson 1998), whether it was TB, neoplastic disease or particulate pollution.

The mandibular bone formation and destruction predominantly affected the left side, with considerable destruction surrounding a “mass” within the body. This mass may represent the sequestrum (dead bone) of mandibular osteomyelitis. Causes of osteomyelitis in this anatomical region are many (Khullar et al 2012), but include the spread of pathogens to the alveolar bone as a result of poor dental hygiene (Resnick and Niwayama 1995a:2343), exposure to radiation (Dalinka and Haygood 1995:3287), such as in workers dealing with luminous watch dial painting (Kullar et al 2012), TB (Imamura et al 2004), actinomycosis
(Resnick and Niwayama 1995b:2503), osteopetrosis (McAlister and Herman 1995:4201), sickle-cell anemia (Resnick 1995), and the effects of phosphorus exposure (“phossy jaw”) through industrial working, or via toxicity from treatment with bisphosphonates today (Campisi et al 2012). During this period radiation was not the potential health hazard known today, while the dense appearance of bones on radiographs observed in osteopetrosis was not present in this skeleton, and nor were any other changes of sickle-cell anemia. Actinomycosis, a fungal infection, affects the ribs, spine, pelvis and the major joints, along with the mandible (Resnick and Niwayama 1995b:2504). This usually affects multiple vertebral bodies but not the neural arches, whereas in the skeleton of Burial 69 the neural arches but not the bodies are involved. Nevertheless, before dismissing actinomycosis as a possible cause for these lesions, it should be noted that recent studies implicate bacteria of the genus Actinomyces in bisphosphonate-related osteonecrosis of the jaw, discussed below (Arranz Caso et al 2012). The most likely causes for the mandibular bone changes are therefore TB and/or “phossy jaw”. It is also possible that the inflammatory changes in the mandible were transmitted hematogenously around the body, leading to the new bone formation and destruction seen elsewhere.

In summary, the bone changes suggest multiple diagnoses for this young person, and mainly, but not excluding others, TB, scurvy, rickets, and “phossy jaw”. However, it should be remembered that other possible diagnoses, including smallpox, cancer and a range of respiratory diseases, might also have been present. The most likely co-morbidities of TB, scurvy, and rickets in this person are all relevant to the diagnosis of particular interest here, “phossy jaw”. In this post-medieval context TB was common throughout Europe. Living both in high population density, and working in TB-related industries predisposed people to TB (see overview in Roberts and Buikstra 2003: 216). Working long hours in dark factories, along with particulate pollution in the air, likely also predisposed children to rickets and lung complaints (see Brickley and Ives 2008: 94-5). Alongside poor living and working environments, this person’s diet may not have contained adequate amounts of vitamin C. If the presence of “phossy jaw” in this young person was a result of being exposed to phosphorus, his or her living and working conditions could equally have led to rickets ,TB and scurvy. The implications of a diagnosis of “phossy jaw” is now discussed in more detail.

4.3 “Phossy jaw”

The bone changes in the mandible may represent osteomyelitis due to industrial exposure to phosphorus (“phossy jaw”). In this condition the lower jaw is more affected than the upper, as here. Cumulative exposure to phosphorus appears to lead to its accumulation in bone, and while people with dental disease seem more likely to develop it, ‘any factor that involves possible contact with the bone or periosteum of the oral cavity is a possible source of osteopathology….’ (Jacobsen et al 2014:35). Marx (2008) records in a report of 1862 that the sequestered bone in somebody who suffered “phossy jaw” was porous and light
in weight, and looked like pumice stone, which is similar to the “mass” seen in the lower jaw of this person, interpreted as a sequestrum. No archaeological skeletons showing “phossy jaw” have been published in the bioarchaeological literature to date, as far as the authors are aware. However, 19th century documented evidence exists at the Royal College of Surgeons of England in London in the form of two mandibles that display the effects of phosphorous necrosis, one of which belonged to a “male match-maker aged 35 years” (Figure 6). The lesions on these documented mandibles are very similar to those present in the skeleton from Coach Lane.

There is evidence that phosphorus was used to treat ailments in the 19th century, and white phosphorus was used as a treatment for rickets in the 19th and early 20th centuries (Hess and Weinstock 1926). It is possible that phosphorus was used to treat rickets or other ailments that this person may have suffered, with the treatment then leading to “phossy jaw”. This has parallels with the occurrence of osteochemonecrosis of the upper and lower jaws caused by the use of bisphosphonates for treating cancer metastases and osteoporosis today (Ardine et al 2006, Campisi et al 2012). Indeed, osteochemonecrosis has been likened to “phossy jaw” of the 19th and 20th centuries and since 2003 a number of people have been recorded clinically with this jaw problem (Marx 2003, Marx 2008, Khaimaisi et al 2007). These drugs inhibit bone metabolism (Otto et al 2011), for example, acting as an anti-resorption/osteoclast inhibitor drug to prevent fractures in osteoporosis and Paget’s disease (Abu-Id et al 2008), and animal experiments have shown that orally administered white phosphorus also suppresses bone destruction (Abu-Id et al 2008). Furthermore, intravenous administration of the drug, a history of dental extractions and dento-alveolar bone procedures (but also dental diseases per se), local trauma, poor oral hygiene, and anatomical abnormalities are predisposing factors to the development of this condition (Campisi et al 2012, Otto et al 2011). Its pathogenesis remains unproven, but osteopetrosis, low calcium levels and secondary hyperparathyroidism, and diabetes mellitus also appear to be risk factors. When phosphorus is combined with H₂O and CO₂ from respiration, and with amino acids, bisphosphonates are the result, with “phossy jaw” as the final outcome (Marx 2008). Of interest here is the link between actinomycosis and “phossy jaw” caused by bisphosphonate administration, as discussed above.

4.4 The matchmaking industry

A number of industries that used phosphorus in manufacturing processes were present in 19th century England, including those producing fireworks, munitions and brass (Hellstein and Marek 2005). However, one of the most likely industries in which the person represented by the skeleton of Burial 69 may have worked is that of matchmaking.

The French chemist, Dr. Charles Sauria, was the first to use yellow phosphorus (also known as white phosphorus) as a means of igniting matches (Dixon 1925;
Marx 2008), with the typical match head comprising approximately 20 percent phosphorus (Jacobsen et al 2014). “Strike anywhere” matches proved to be one of the most significant inventions of the industrial age, initiating over 260 home and major production factories in the 19th century in Britain, with the largest areas of manufacturing in the industrial centers of London, Birmingham, Gloucester, Liverpool, Leeds, and Newcastle (Figures 7 and 8, and Simon 1863; Gwyn-Smith 1978; Raw 2009; Lucifer n.d.). The labor was un-skilled, poorly paid and generally attracted the “poorest of the poor”. Half of all employees were children between the ages of six to 12 years, each of whom could be required to work a twelve hour day (Emsley 2000). Employment of women and children kept costs down and allowed British matchmaking firms to remain profitable within the competitive European market. At this time the matchmaking industry was not covered by the Factory Acts, which limited the number of hours that children could work. It was reported in 1899 (British Medical Journal 1899) that while all were at risk of “phossy jaw”, the younger individuals were most likely to succumb.

The basic processes in match manufacture consisted of setting splints of wood, twice as long as the match, which had been heat dried and dipped in melted sulfur, or paraffin, into a frame each holding several thousand sticks. These were then dipped into the lighting composition, which contained white or yellow phosphorus spread over a hot dripping table. They were then carried to a drying room and the matches, when dry, were taken from their frames, cut in half and then boxed (Harrison and Mockett 1990; Satre 1982; Marx 2008, 2012; Raw 2009). Mechanization increased from the 1860s, but dipping still tended to be done by hand. Most women worked at cutting down and boxing matches, which by all accounts was still hard and dangerous work. Rose Squire, one of the early women factory inspectors, described the scene in her autobiography:

‘I have a vivid picture in my mind of the awkward scramble of arms and hands of a crowd of girls working at feverish speed to cram the handful of matches into boxes which, when overfull flared up and were cast upon the floor, the fumes and smoke rising into one’s nostrils’ (Squire 1927:54).

The conditions within these factories were generally described as “deplorable” with little or no ventilation, and up to 16 hour workdays for all laborers (Simon 1863; Satre 1982: 9; Lucknow 2007; Marx 2008, 2012; Raw 2009). Indeed, the inflammatory rib lesions in the skeleton of Burial 69 could have resulted in part from inhalation of phosphorus fumes. An account of working conditions in one of the early German matchstick factories reported that the air was so badly affected by phosphorus that the walls and furnishings glowed in a bluish light at night time, and the workers often inhaled such large amounts of phosphorus vapors that their breath was luminous in the dark (Hinrichs 1957). Workers most at risk of phosphorous poisoning were those who made the compound for the match head and those who dipped the matches into it (Emsley 2000). Raw (2009:92) cites interviews with the grandchildren of those who lived near the notorious Bryant and May factory in London who still recall ‘piles of fluorescent vomit’ along
the homeward routes of the factory workers at the end of their shifts, and Myers and McGlothlin (1996:330) reference members of the Salvation Army dimming gas lamps to reveal the ‘greenish-white glow’ of the “phossy jaw” victim’s mouth, blouse, and hands.

By the 1860s, medical experts associated phosphorus necrosis (“phossy jaw” or “the phoss”) as a specific occupational hazard for matchstick factory laborers (Hunter 1978; Satre 1982; Marx 2008, 2012). In a Public Health Report presented to Parliament in 1863 compiled by John Simon, the first detailed clinical descriptions of “phossy jaw” were noted (Simon 1863). The report publicly linked “phossy jaw” to exposure to match production fumes. The effect of this process caused bone necrosis of the mandible or maxilla. Approximately 11% of those exposed to phosphorous fumes developed “phossy jaw” after an average period of five years from initial exposure (Lucknow 2007). The most commonly affected groups were said to be women, children, and adolescents, but adult men were also treated (Simon 1863; Hunter 1978; Satre 1982; Lucknow 2007; Raw 2009). The necrotic bone in the mandible had to be surgically removed if the patient was to survive. (Nilsson 1995).

Dental caries was considered to be a predisposing factor to the development of “phossy jaw” and a main route for phosphorus to enter the dental pulp cavity and subsequently the alveolar bone (British Medical Journal 1899). Attempts at limiting “phossy jaw” in people working in match factories in the later nineteenth century were focused on improving dental care, including the use of carbolic mouthwashes, and monitoring dental health (Emsley 2000). During the 19th century dental caries is known to have been highly prevalent in Britain particularly amongst the poorer classes, due to the availability of cheap sugar (e.g. Roberts and Cox 2003: 326). However, the link between caries and “phossy jaw” may have been coincidental. Others have argued that those with perfectly healthy dentitions were also affected by “phossy jaw”, further stating that the condition is a systemic rather than a localized disease, with phosphorous entering the bloodstream via inhalation and ingestion (Hughes et al. 1962). The possibility that phosphorous poisoning is not localized is worth exploring in relation to the postcranial lesions observed in the skeleton of Burial 69. A number of clinicians from the 19th century argued that phosphorous poisoning resulted in systemic compromised bone integrity, which led to greater susceptibility to spontaneous fracture (Dearden 1899). Additionally, an apparent connection was made to lung complaints in a 19th century observation: ‘Recently a match dipper in Bryant and May’s, aged 22 years, died after a lingering illness, during the course of which a swelling appeared on his right jaw which had to be lanced, the discharge being extremely offensive. Subsequently an abscess, which had to be opened, appeared in his chest; the patient had rigours, appeared delirious and died’ (British Medical Journal 1898:1346). A further link is evident in the postmortem descriptions of other people with “phossy jaw” where ‘tuberculosis of the lung’ is referred to (Stockman 1899: 9). This may be significant in light of the new bone formation on the visceral surface of the ribs of this skeleton.
Home production of matchsticks cannot be ruled out as a cause of phosphorous exposure. Due to the unregulated nature of home production, its true scale remains unknown. In France until the late 1860s the majority of matchsticks were produced at home (Weigert 1909:175). The entire family was involved in this activity and many fell ill and died. A 100mg dose of phosphorus is known to be lethal. Ingested phosphorus kills slowly by affecting the stomach lining and, after 2-6 hours the phosphorus enters the bloodstream and subsequently destroys the liver (Jansen and Ducci 2006:137), again lending possible support to the idea that hematogenous spread of the phosphorus may have led to some of the extensive postcranial bone changes seen in this skeleton.

Major legislative reforms in Britain helped to mitigate the occurrence of “phossy jaw”, especially in children, but it was not until the 1888 “London matchgirls strike” at the Bryant and May factory in the east end of London that members of the public became embroiled in this occupational disease controversy (Satre 1982; Harrison 1995; Raw 2009). Despite the well-documented association between matchstick production and “phossy jaw”, the production of yellow phosphorous matches continued in Britain until its eventual outlaw in 1910 (Myers and McGlothin 1996).

4.5 The impact of “phossy jaw” on social identity

“Phossy jaw” was and is potentially very painful, and it could and can lead to septicemia, meningitis and even death. Not only would this person have appeared facially disfigured, with swelling and suppurations of the affected side of the face, the foul discharge from their mouth as a result of osteomyelitis would have been odorous, thus potentially resulting in social stigma (Dangerous Industries 1893; Satre 1982; Moss 1994). In one account from a “phossy jaw” sufferer, it was contended that the accompanying smell from the necrotic and infected tissue was so great that her husband and children were unable to attend to her (Satre 1982:16). Lücke (1973) advocated a prosthesis made of gutta-percha (a latex from Malaysian trees) to avoid shrinking of the facial features that particularly caused distress amongst female patients. Nineteenth century journalists and medical practitioners likened the sufferers to “lepers” and, even when the sufferer survived, the resulting disfigurement created a high degree of social stigma and made it difficult for them to secure further employment (Dangerous Industries 1893; Malone 1999). The person represented by Skeleton 69 would also likely have suffered respiratory problems, such as shortness of breath and chest pain, due to pulmonary involvement. As the disease progressed it is possible that this person, still a child, would have experienced ostracism within their local community. However, if “phossy jaw” was a common complaint of locals who worked in the matchmaking industry then this person may have been more accepted amongst friends and relatives.

5. Conclusions
The bone changes identified in the skeleton of this person from the Coach Lane Quaker cemetery in North Shields suggest that this person most likely suffered from “phossy jaw”, in addition to tuberculosis, scurvy and rickets. It is likely that this person similarly suffered from poor health during their growth as evidenced by their diminished height and enamel hypoplasia. While it is not certain that the mandibular osteomyelitis present reflected exposure to phosphorus during match making, the historical data for the region suggest that such factories did exist in the region at the time this person was living. The skeletal changes are also very similar to clinically documented people with “phossy jaw”, and this is a condition that has previously been overlooked in the bioarchaeological literature. If this is indeed “phossy jaw” then the sufferer was already in a poor state of health as evidenced by the range of skeletal lesions expressed. However, one cannot completely reject other differential diagnoses, and synergistic relationships between rickets, actinomycosis, neoplastic disease and osteonecrosis of the mandible; all these conditions are described in the clinical literature, and have been described historically. Furthermore, evidence for poor health in the form of rickets, scurvy and rib lesions has also been observed in a number of non-adult skeletons within the Coach Lane skeletal population (e.g. Ostrander 2013, Roderick 2011). Nevertheless, the range of pathological lesions expressed in the skeleton of this person reflects the challenging environment in which he or she lived and worked during their short life.

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Supplementary Information S1: Detailed description of pathological changes of the skeleton from Burial 69
Supplementary Information S2: Elemental analysis of calculus samples from the teeth of the skeleton from Burial 69

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