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Chapter 6

SCURVY AS A FACTOR IN THE LOSS OF THE 1845 FRANKLIN EXPEDITION TO THE ARCTIC: A RECONSIDERATION


Abstract. In 1845, an expedition, commanded by Sir John Franklin, set out to discover the Northwest Passage. The ships entered the Canadian Arctic, and from September 1846 were beset in ice off King William Island. A note left by the expedition in May 1847 reported all was well, but by April 1848, 24 of the 129 men had died. The ice-locked ships were deserted in April 1848, but the 105 survivors were so weakened that all perished before they could reach safety. The causes of the morbidity and mortality aboard the ships have long been debated, and many commentators have argued that scurvy was an important factor. This study evaluates the historical evidence for the likely effectiveness of anti-scorbutic precautions taken on polar voyages at that time, and investigates whether the skeletal remains associated with the expedition provide evidence for scurvy. Skeletal remains available for study were carefully examined for pathological changes, and lesions potentially consistent with scurvy were subject to histological analysis. Where remains were no longer accessible, use was made of published osteological work. It is argued that the anti-scorbutic measures customarily taken on mid 19th century British naval polar voyages were such that there is no a priori reason to suppose that scurvy should have been a problem prior to the desertion of the ships. The analysis of the skeletal evidence provided little in the way of bony lesions consistent with the disease, and cannot therefore be used to support the presence of scurvy. Factors other than scurvy may have been the main causes of morbidity and mortality in the eleven months prior to the desertion of the ships.
Introduction

In May 1845 two ships, HMS Erebus and HMS Terror, set out from England on a Royal Navy voyage to the Arctic. The ships were under the command of Sir John Franklin. The purpose of the expedition was to map the route of the Northwest Passage, the seaway connecting the north Atlantic with the Pacific via the Canadian Arctic. The expedition called at Disko Bay on the west coast of Greenland in July 1845, and later that month entered the Canadian archipelago via Lancaster Sound with 129 men. None would return alive.

The expedition wintered in 1845-6 at Beechey Island in the Canadian High Arctic (Fig. 1). Three men died during this period and were buried in the permafrost (Beattie and Geiger, 1987). The following summer, the ships sailed south and west in search of the Passage. In September 1846, the ships were once more beset in ice, northwest of King William Island. The ice failed to release them the following summer. A note, left in a canister on King William Island, dated May 1847, indicated that all was well. However an addendum, dated April 1848, indicated that 24 men (nine officers, 15

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**Figure 1.**

Canadian Arctic, showing Franklin’s 1845 route (solid line), and presumed 1846 route (dashed line).
lower ranks) had died by that time, and the remainder would attempt to reach safety overland via the Back River. Surface scatters of artifacts and human remains show that many died on the march along the western and southern coasts of King William Island (Cyriax, 1939; Owen, 1978). The furthest point reached was Starvation Cove on the Canadian mainland, barely 400km along the presumed route, and still more than 1000km from the nearest Hudson Bay Company post via the Back River (Gibson, 1937).

Cut-marks on human remains (Beattie, 1983; Beattie and Savelle, 1983; Keenleyside et al., 1997) confirm Inuit accounts of cannibalism in the final throes of the expedition, and leave little doubt that those who perished attempting to make the Back River suffered from starvation. The small distance that the men had managed to travel from the ships suggested that they were already in a severely weakened state and hence that problems began before this. This is supported by the number of deaths that occurred prior to the desertion of the ships. The 24 deaths that had occurred by April 1848 represent 19% of the ships’ companies. The Franklin voyage was very well equipped, and members included men experienced in polar conditions.

Figure 2.

King William Island, showing location of finds of the material listed in Table 1. Find spots: 1, Booth Point, 2, Erebus Bay, 3, Islet in Erebus Bay, 4, Near the mouth of the Pfeffer River. X indicates the location of the ships when deserted in April 1846.
Yet the death rate up to April 1848 was markedly greater than that in any other Royal Navy polar expedition (Woodman, 1991). In eight British Naval expeditions between 1819 and 1836 the overall death rate was only 3% (Cookman, 2000). In an 1839 voyage to the Antarctic, under the command of James Clark Ross, aboard HMS Erebus and HMS Terror, the deaths after 3 years amounted to just 4% (Ross, 1994).

Reasons for the deaths in the period May 1847 - April 1848, and for the weakened state of those who deserted the ships, have been debated for the last 150 years. Starvation seems unlikely at that stage of the expedition. Franklin indicated that his supplies were sufficient for five years, and if necessary could be made to last for seven (Beattie and Geiger, 1987). In addition, firearms had been taken with the intention of taking birds or other game as the opportunity arose (Owen, 1978), and bones of wildfowl at the Franklin camp at Beechey Island testify that this occurred (Cyriax, 1939). Different theories involving health problems have been suggested. Research in the 1980s and 90s reported high lead levels in the human bones from King William Island (Beattie, 1985; Kowal et al., 1989, 1991; Keenleyside et al., 1996), and in tissue samples from the Beechey Island bodies, which were exhumed in the 1980s (Amy et al., 1986; Kowal et al., 1991). These findings have been used to support suggestions that lead poisoning, either from the expedition’s canned food supply (Beattie and Geiger, 1987; Kowal et al., 1989, 1991) or other sources (Battersby, 2008) contributed. Another suggestion was that food poisoning through inadequate canning of the tinned meat supplied to the expedition, or from shot game, was a factor (Cookman, 2000; Horowitz, 2003). However, what is perhaps the oldest theory holds that the men were suffering the effects of scurvy.

Scurvy arises due to a deficiency of vitamin C in the diet. Prime sources of vitamin C are fresh fruit and vegetables, but it is also present to a lesser extent in fresh animal products. The vitamin oxidises on exposure to air so, unless adequate precautions are taken, the vitamin C content of foods gradually diminishes during storage. Scurvy was always a potential threat on polar voyages due to the lack of locally available fresh fruit and vegetables.

In 1859, an expedition under Francis McClintock found the first traces, in the form of human remains and artifacts, relating to the final march of Franklin’s men along the western and southern shores of King William Island. Noting that the rapidity of the disaster that overtook them meant that the men must have been in a severely weakened condition when they left the ships, he ascribed this to scurvy (McClintock, 1908). Gibson (1937) felt that it was scurvy that caused the men to abandon the ships and to seek the Back River route to safety because game was known to be abundant in that area and fresh meat was understood to be beneficial for scurvy. Two years later, Cyriax, writing what has become the classic account of the expedition, felt able
to state that there had been ‘general assent’ to the scurvy theory (Cyriax, 1939). Subsequent major works on the voyage have largely concurred that scurvy was an important factor in the mortality and morbidity suffered by the expedition between May 1847 and April 1848 (e.g. Neatby, 1970; Owen, 1978; Beattie and Geiger, 1987; Berton, 1988; Woodman, 1991; Ross, 1994; Lambert, 2009; Brandt, 2011).

Despite this general consensus, there was for some time no direct evidence to support the scurvy theory. There is testimony from Inuit that expedition members they encountered on their final march along the south coast of King William Island had blackened mouths (Woodman, 1991). Darkening of the gums could be consistent with scurvy (Aschoff and Koch, 1919), but is rather non-specific – for example it may also result from chronic lead ingestion (Lockheart, 1981). The Inuit accounts were related second-hand more than 20 years later so it is difficult to know what weight to give them. In any event, they do not tell us anything about the condition of the men whilst they were still on the ships. The lack of any direct evidence for scurvy appeared to change with the examination of skeletal remains from King William Island recovered during the 1980s. Owen Beattie and his co-workers conducted the first modern archaeological surveys in this area, and recovered and studied 36 bones representing a minimum of 3 individuals.

Deficiency of vitamin C leads to weakening of blood vessel walls, so haemorrhage is a prominent and characteristic feature of scurvy. If haemorrhage occurs close to bone it may potentially lead to osteological changes, in adults principally subperiosteal deposits of new bone (Brickley and Ives 2008). Beattie reported periosteal reactions on some bones from Erebus Bay on the west coast of King William Island, and from the vicinity of Booth Point, on the south coast (Beattie, 1983; Beattie and Savelle, 1983). Periosteal reactions may arise as a response to a multitude of conditions other than scurvy (Weston, 2008). Beattie (1983) acknowledged this, but observed that their occurrence supported the presence of scurvy. In 1992, another King William Island Franklin site, on an islet in Erebus Bay, was located. More than 300 human bones were retrieved and subject to osteological study (Keenleyside et al., 1997). One bone showed periostitis, but rather than inferring scurvy, the authors felt the lesion suggested trauma or infection.

Since the publication of Beattie’s work, major works on the Franklin tragedy have used his skeletal evidence to support the scurvy hypothesis. Some have greatly exaggerated the evidence, or else attributed to the findings an unwarranted degree of diagnostic certainty. For example, Woodman states that changes attributable to scurvy ‘were universally found in the remains from King William Island’ (Woodman, 1991), and for Lambert, the presence of scurvy has been ‘confirmed by forensic science’ (Lambert, 2009). The purpose of the current work is firstly to review the anti-
scurbutic precautions followed on British naval Arctic expeditions at the time of the Franklin voyage, and secondly to evaluate the osteological evidence from remains associated with the expedition to determine whether, as has been claimed, they provide evidence for the presence of scurvy.

**Scurvy and the Franklin expedition**

Following the pioneering experiments of 18th century British naval surgeon James Lind that demonstrated the value of citrus fruit in combating scurvy, and the advocacy of Sir Gilbert Blane, another naval physician, orders were issued in 1795 that lemon juice be carried on board every ship in the Royal Navy (Sauberlich, 1997). The use of lemon juice virtually eliminated the problem of scurvy in the British navy for the next 50 years (Carpenter, 1986; Harvie, 2002), and it was routinely used at the time the Franklin expedition set sail.

Victualling records indicate that the Franklin expedition was supplied with 9300lbs of lemon juice (Cyriax, 1939). When it was introduced in 1795, the daily lemon juice ration was three-quarters of an ounce per man (Brown, 2003). However, in view of the special threat of scurvy in polar regions, this was increased to one ounce on Arctic voyages (Cyriax, 1939). On this basis, there would have been sufficient lemon juice on board for full Arctic rations for at least three years and two months, i.e. until July 1848, three months after the date at which the ships were deserted.

Although the cause was not understood, it was well-recognised by the 19th century that lemon juice gradually lost its antiscorbutic properties upon exposure to air. The usual way to preserve it was in containers with olive oil poured into the top, and which were then corked and sealed, a system first devised in the 17th century (Carpenter, 1986). It is not known exactly how the lemon juice was stored aboard the *Erebus* and *Terror* (Beattie, 1985), but given the care with which the expedition was equipped and victualled (Cyriax, 1939; Beattie and Geiger, 1987) it seems unlikely that these routine and elementary precautions against its deterioration, which were used on other mid 19th century polar expeditions (e.g. Armstrong, 1858), would have been neglected.

That men could be kept healthy on prolonged naval voyages under polar conditions is illustrated by contemporary accounts. John Ross’s 1818 Arctic expedition spend four winters on the ice without suffering scurvy (Houston, 1990). In the same commander’s 1829 expedition, his men were ‘fresh and good humoured’ after three winters on the ice (Neatby, 1970), and only one man died of scurvy during the whole four-year voyage (Cookman, 2000). In Parry’s 1821 Arctic expedition, slight symptoms of scurvy were reported after 27 months but no-one succumbed (Houston,
In 1854, the crew of Collinson’s *Enterprise* emerged from three winters in the Arctic in excellent health (Neatby, 1970). Aboard McClure’s *Investigator*, in a three year Arctic expedition beginning in 1850, only three men were lost to scurvy, and that was toward the end of the expedition, some months after the lemon juice ration had been halved (Armstrong, 1858; Harvie, 2002). However, scurvy was a greater problem on some other polar voyages. The 1848 expedition in search of Franklin under James Clark Ross suffered from scurvy in their first winter (Owen, 1978). George Back’s 1836 Arctic voyage was afflicted by scurvy after only 6 months (although, as he noted, other voyages were by that time usually exempt from the disease). The narrative of the expedition (Back, 1838) suggests that provision of lemon juice was inadequate in quantity, and anti-scorbutics did not appear to have been taken daily.

It would seem that although polar expeditions were not always free of scurvy, the disease could be effectively combatted for prolonged periods with proper dosage of lemon juice. There is therefore no *a priori* reason to assume that scurvy was a problem for the Franklin expedition in the 11 months between May 1847 and April 1848. In addition, the scurvy hypothesis fails to account for the disproportionate numbers of deaths that befell the officers during that period (9 out of 24 officers (38%) died compared with only 15 of 105 (14%) lower ranks). The remainder of this paper attempts to investigate the extent to which skeletal remains from the voyage can be used to support the scurvy hypothesis.

A difficulty with interpreting skeletal evidence for scurvy is that signs of the disease in the adult skeleton are rather subtle, and certainly less pronounced than in the growing skeleton, and none are pathognomonic (Joffe, 1961; Ortner, 2003; Brickley and Ives, 2008; Mays, 2008). The most important skeletal changes are connected with osteological responses elicited by haemorrhage. The bleeding and swelling of the gums that is such a prominent soft tissue feature of scurvy may potentially lead to changes in the alveolar bone (Ortner, 2003; Van der Merwe *et al.*, 2010a). However, alveolar bone changes arise through a great many other causes which have nothing to do with vitamin C deficiency (Hillson, 1986), and some alveolar bone pathology is almost universal in adults in British pre-modern skeletal populations (Kerr, 1998). Alterations to alveolar bone are therefore of little value in the identification of scurvy. Diffuse osteopaenia may occur in scurvy (Joffe, 1961) and may predispose to fracture, especially of the ribs (Aschoff and Koch, 1919) and vertebrae (Grusin and Kincaid-Smith, 1954). However, a great many diseases other than scurvy can predispose to fracture, and, obviously, fracture may also occur without a predisposing skeletal condition. Subperiosteal haemorrhages occur in scurvy, often in response to minor trauma. In infants and young children, bony
alterations due to haemorrhage characteristically occur in the craniofacial skeleton (Ortner, 2003). These may sometimes be seen in adults (Geber and Murphy, 2012), but the more typical lesions occur postcranially. In the scorbutic adult, subperiosteal haemorrhages occur most often in the lower limb (Hess, 1920) where they may provoke an osteoblastic response resulting in deposition of new bone upon the normal bone surface (Hess, 1920; Wolbach and Howe, 1926; Grusin and Kincaid-Smith, 1954; Joffe, 1961; Aufderheide and Rodríguez-Martín, 1998; Weinstein et al., 2001; Fain, 2005). In the absence of vitamin C intake, haemorrhage, indicated by ecchymoses (darkening caused by escape of blood into subcutaneous tissue) may appear on the lower limb in as little as 1-2 months, even in individuals who were replete with the vitamin at the time the vitamin C-free diet was begun (Hodges et al., 1971).

The current work re-evaluates the skeletal evidence for scurvy by, as far as is possible, re-examination of the skeletal material from King William Island. The focus will be principally on the evidence for periosteal new bone in the remains.

Materials

The human remains recovered from King William Island since 1980, and upon which there is published scientific work, are listed in Table 1, and the find spots are shown in Fig. 2. Remains from three of the four locations were scatters of disarticulated remains partially exposed upon the ground surface. They principally comprise bones from the lower limb, upper limb long-bones and cranial vault fragments. The remains interred beneath the Franklin Memorial in Greenwich were recovered from King William Island in 1869 as an articulated burial in which most major elements were present. All remains were recovered from locations lying on the presumed route of the final march of Franklin’s men, so it is likely that they mainly or entirely represent remains of men who perished after the desertion of the ships in April 1848. Of the remains in Table 1, those described by Keenleyside et al. (1997) are no longer available for study, but they were subject to detailed osteological analysis by Keenleyside’s team. The other material in Table 1 has been examined for the present work.

Methods

The remains recovered by Beattie’s team in 1981/2, and the skeleton interred beneath the Franklin memorial, were carefully examined under strong light for
evidence of pathology, paying particular attention to evidence of periostitis. Any instances of subperiosteal new bone thus identified were subject to histological study with the aim of distinguishing periosteal new bone formation as a response to haemorrhage (and hence potentially due to scurvy) from that arising from other causes (van der Merwe et al., 2010b).

For histological study, a transverse section of bone was removed by making two, closely spaced parallel cuts of sufficient depth to penetrate the full thickness of the cortical bone. The saw blade was twisted to break the section free; care was taken not to cause any flaking of the bone surface.

The cut sections were processed according to earlier published methods (Maat et al., 2001, De Boer et al., 2013). For reasons of readability the procedures will be summarized here. Prior to further processing, the sections were embedded in LX-112 epoxy resin to prevent any damage to the fragile subperiosteal bone. After overnight curing, the sections were cut by means of a hacksaw and ground down by hand to a thickness of approximately 80µm. The sections were cover-slipped using the same resin as was used for embedding. Histological analysis was done by means of a standard light microscope, using bright light and polarized light.
Results

Gross observations

The Greenwich Memorial skeleton was examined prior to its reburial in 2009. The specimens collected by Beattie from Booth Point and Erebus Bay were studied at the Canadian Museum of Civilization in Ottawa. All were present in the collection, save for a partial left scapula and a small cranial fragment, both from Erebus Bay, which had been destroyed for lead analysis (Beattie, 1985; Kowal et al., 1991); neither had been identified by Beattie (1983) as pathological. Some small samples had also been cut from a few other elements for lead content analysis. The condition of the material in general was good. Most subperiosteal surfaces were undamaged, but a few showed flaking and/or cracking due to weathering.

In general, there was little evidence for pathological changes. None of the specimens examined showed in vivo fractures or gross suggestions (in the form of thinned cortices or rarified trabecular bone) of osteopaenia, nor were there any potentially haemorrhagic changes in the cranial bones. Similarly, none of these indications was reported by Keenleyside et al. (1997) in the material they examined, but one specimen, a right tibia, was reported by them as showing periosteal new bone. None of the bones from the skeleton from the Franklin Memorial in Greenwich show evidence of periosteal new bone, but three postcranial specimens from Beattie’s fieldwork appeared to show minor, well remodelled periosteal reactions:

Booth Point, Beattie specimen number 81-22. The Booth Point remains comprise parts of at least eight skeletal elements - cranium, left fibula, left tibia, right femur, left and right radii, left and right humeri - probably from a single individual. Specimen 81-22 is the left tibia. It is the diaphysis in two parts. The proximal part is 14cm long, the distal part 17cm; both ends are missing (Beattie, 1983: Fig. 3). This bone has had a small piece removed from the midshaft for lead analysis. At the midshaft there is a slightly porous area on the postero-medial angle of the bone (Fig. 3a). It extends along the middle third of the bone shaft. It appears slightly raised above the surrounding bone.

Erebus Bay, specimen 82-1. This specimen was found as part of a scatter on the south side of the bay. It is a right tibia (Beattie, 1983: Fig. 10), complete but for three samples that have been removed for lead analysis at the proximal and distal metaphyses and at the midshaft. Its maximum length is 34.2cm. The specimen bears a small, raised area of bone on the medial surface of the proximal metaphysis (Fig. 4a). This area shows a little pitting, and its posterior border is fairly distinct and slightly raised above the surrounding bone.
Erebus Bay, specimen 82-12. This specimen was an isolated find on the south side of the bay. It is a left tibial shaft (Beattie, 1983: Figs 7 and 10), both ends missing, 22.2cm long. It is not from the same individual as 82-1. There is a pitted, slightly roughened area extending from the midshaft to the broken distal end of the bone on the posterior part of the medial surface (Fig. 5a).

In arctic burial conditions, remains of haematomas, even if unossified, may survive as dark staining on the bones (Maat, 1982, 2004). Neither the Greenwich skeleton, nor the material collected by Beattie, showed evidence for such staining. Keenleyside et al. (1997) noted no such alterations in the material they studied.

**Histological observations**

Transverse sections of the periosteal lesions identified on specimens 81-22, 82-1 and 82-12 were removed and histological slides prepared. Each specimen showed excellent preservation of microstructural features.

The microscopic section from specimen 81-22 (Fig. 3b, 3c) appears to show a very well consolidated, localised deposit of new bone, with a maximum thickness in the cut section of about 2mm. The original subperiosteal surface is discernible, particularly under polarised light (Fig. 3c), but the circumferential lamellae are no longer continuous, having been partly obliterated by remodelling. There is no evidence for any lytic changes nor of any alterations to the original periosteal surface that might suggest infection. There is no sign of any pathological process active at time of death. The appearance of the lesion is suggestive of an extensively remodelled ossified subperiosteal haematoma (phase III remodelling on the scale of van der Merwe et al. (2010b)).

Specimen 82-1 (Fig. 4b, 4c). There are sinuous cavities beneath the intact periosteal surface which may be relics of an earlier infectious process. There is no sign of haematoma formation and no evidence of any abnormal process active at time of death. The evidence suggests an old remodelled lesion which arose as a result of a non-haemorrhagic process.

The section from specimen 82-12 (Fig. 5b, 5c) presents a similar appearance to that from 81-22. There appears to be a well-remodelled deposit of subperiosteal new bone upon an intact cortical surface. The original circumferential lamellae have been partially removed by remodelling. There is no evidence for any abnormal process active at time of death. The lesion appears to represent an extensively remodelled ossified haematoma.
Figure 3.

Booth point, specimen 81-22. (3a) Left tibia diaphysis, medial view. The arrows denote the raised, slightly pitted area towards the posterior margin of the medial surface. (3b and c) Transverse section of the lesions, under plain (b) and polarized light (c). Scale bar 2 mm. Arrows (c) indicate the line of the original subperiosteal surface.

Figure 4.

Erebus bay, specimen 82-1. (4a) Proximal part of right tibia medial surface. The arrows indicate the raised, slightly pitted area of bone. A sample of bone had been cut for lead content analysis. (4b and c) Transverse section of the lesion, under plain (b) and polarized light (c). Scale bar 2 mm. Arrows (b) indicate two large sinous cavities.

Figure 5.

Erebus Bay, specimen 82-12. (5a) Distal part of the left tibia shaft, medial view. There is an area of slightly roughened bone at the posterior part of the medial surface, toward the distal end of the bone (arrows). (5b and c) Transverse section of the lesion, under plain (b) and polarized light (c). Scale bar 2mm. Arrows in image (c) indicate the line in of the original subperiosteal surface.
Discussion

In two of the three specimens showing gross signs of abnormality, histological study suggested the presence of very well remodelled ossified haematomas. Ossified haematomas may arise due to trauma of the periosteum in undiseased individuals (Resnick and Niwayama, 1988), as well as in scurvy. Lesions due to the former cause are usually isolated and often occur on bones such as the tibia where there is only a thin covering of soft tissue (Zimmerman and Kelley, 1982; Aufderheide and Rodríguez-Martín, 1998). In scurvy, the potential for widespread haemorrhage upon minimal trauma means that ossifying haemorrhagic lesions are often multiple, and may involve more than one bone. Given the nature of the remains, the question of whether multiple bones were affected in these individuals cannot be answered conclusively, but a few points can be made. None of the other bones available from the Booth Point individual show periosteal new bone (a femur did show slight porosity over its general surface, some of which at least was antemortem, but there appeared to be no new bone deposition and the porosity appeared within the bounds of normal variation). It is not known whether left tibia 82-12 and the right tibia from the site on the small islet in Erebus Bay, described by Keenleyside et al. (1997) as showing periosteal new bone, come from the same individual but given the difference in location it seems unlikely. The lesions in 81-22 and 82-12 are located on subcutaneous surfaces vulnerable to trauma. Trauma in a healthy individual would appear to be as likely an interpretation as haemorrhage associated with scurvy. Both the ossified haematomas examined here were very well remodelled. Whatever the cause, these haemorrhagic lesions occurred long before death, potentially prior to the expedition rather than on it.

It appears that some vitamin C is needed for individuals to be capable of mounting a significant osteoblastic response, and hence for ossification of subperiosteal haematomas. It might therefore be suggested that we would not expect to see periostitis in those who failed to recover from scurvy. Experimental work with guinea pigs, which, like humans, are unable to synthesise vitamin C and hence require a dietary intake, indicates that in the absence of vitamin C bone formation is severely retarded, but when some vitamin C is restored to diets bone deposition does occur. Ossification of subperiosteal haemorrhages occurs in partially vitamin C deficient guinea pigs, and only small amounts of vitamin C are needed to enable an osteoblastic response – only about 2-5% of the dose required to maintain vitamin C saturation in a healthy animal (Bourne, 1942, 1943; Murray and Kodicek, 1949a, b). Observations in man show that radiographically visible periosteal new bone deposition in association with haemorrhage adjacent to bone occurs in acute as well
as in chronic cases of scurvy (Joffe, 1961), presumably because, even though insufficient to prevent acute scurvy, enough vitamin C was present to enable an osteoblastic response.

Given the effectiveness of lemon juice for preventing scurvy in 19th century sea voyages, for scurvy to have been a problem, either the anti-scorbutic properties of Franklin’s supplies of lemon juice must have deteriorated due to breakdown of the vitamin during storage, or the men were put on short rations to try and eke out the supply. In the first case, the juice would still have contained some vitamin C even if it eventually became insufficient to prevent scurvy. If the men were put on short rations and the juice administered in higher quantities only to the sick (as for example occurred aboard Investigator when supplies of lemon juice ran low (Armstrong, 1858)), some vitamin C would still have been ingested by those with scurvy. In addition, such fresh meat as could be hunted would also have supplied some vitamin C.

We would not expect to find skeletal evidence in any cases of scurvy beginning during the final throes of the expedition on the march along the shores of King William Island. At that time the men were suffering from starvation, so there would presumably have been a complete absence of vitamin C. If scurvy did break out prior to this, aboard the ships, it is likely to have been due to insufficient vitamin C rather than its total absence, so had this occurred we would expect to see skeletal evidence.

There are a number of archaeological assemblages representing populations specifically known from documentary sources to be suffering from scurvy in the years immediately prior to death. In those from 19th century prisoners from Quebec (Cybulski, 1988), early 17th century French pioneers in Maine (Crist et al., 2005) and a 19th century Great Famine population from Ireland (Geber and Murphy, 2012) bony signs of the disease were present. However this is not invariably the case. Among 17th century Dutch whalers buried at Spitsbergen (Maat, 1982), although soft tissue signs of scurvy were present, bony changes on the whole were not, presumably suggesting a diet for most individuals totally lacking vitamin C. If the three cases of subperiosteal new bone described here are added to the tibia described by Keenleyside et al. (1997), they give an overall prevalence of periosteal reactions of 1% for the 409 bones listed in Table 1. Unfortunately, the changes in the known scurvy populations described above are not quantified in ways which permit direct comparison with the disarticulated remains from the Franklin expedition. However a few studies of 19th century skeletal remains from England report periostitis prevalences calculated by bone rather than by individual. Among remains of retired Royal Navy personnel interred in the 18th and 19th centuries at Royal Hospital Greenwich, London, about 4% of total bones examined showed periosteal reactions
(Boston et al., 2008). At least some of these men probably suffered (and recovered) from scurvy at some point in their lives, probably many years before their deaths. The rate at which periosteal reactions are removed by remodelling is not known, and this complicates comparison with the Franklin remains. If scurvy was present on the Franklin voyage it would clearly have affected in men in the last years and months of their lives. Hence there would be less opportunity for lesions to be removed by remodelling, so if scurvy was present we might expect a high prevalence of lesions, but in fact, their prevalence among the Franklin remains is lower than at Greenwich. Among osteological studies of 19th century skeletal remains from England from burial grounds where there is no reason to believe scurvy was an especial problem, studies of larger assemblages (N=1458-18970 bones), give figures in the range 2.3-7.5% (Boulter et al., 1998; Boston et al., 2009; Western and Kausmally, 2010). Although comparisons are not straightforward, if scurvy was a widespread problem on the Franklin expedition, we might expect an elevated prevalence of periosteal reactions. In fact, the King William Island remains show little periostitis compared with contemporaneous English skeletal series.

The paucity of periosteal reactions or other bony signs of scurvy means that the skeletal remains provide little support for the idea that scurvy was a serious problem for the expedition prior to the desertion of the ships. Absence of evidence does not of course equate to evidence of absence. For one thing, the evidence is based on a small amount of skeletal remains – the material listed in Table 1 represents the very partial remains of a minimum of 15 out of the probable 126 expedition members surviving when the ships were beset in September 1846. Nevertheless, it does show that, contrary to claims in the literature, the remains do not provide support for the scurvy hypothesis.

**Conclusion**

The Franklin expedition set sail at a time when the Royal Navy had, in the form of lemon juice, an effective means of combatting scurvy. The Franklin expedition was provided with adequate quantities for the voyage. Effective procedures to prevent deterioration of anti-scorbutic properties of lemon juice were routine at that time. There is therefore no *a priori* reason to assume that scurvy should have been a problem. Gross and microscopic examination of extant skeletal remains from King William Island showed only two bones with changes consistent with subperiosteal haematoma. Both lesions were well remodelled and so may have formed prior to, rather than on, the expedition. In addition, they may well represent old traumatic injuries unconnected with scurvy.
The skeletal remains produced little evidence for scurvy. Although this does not provide evidence of its absence, it may be that other factors, for example lead poisoning, for which there is significant skeletal evidence (Beattie, 1985; Kowal et al., 1989, 1991; Keenleyside et al., 1996), may have been more important as causes of morbidity and mortality aboard Franklin’s ships between May 1847 and April 1848.

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