CHRISTMAS: DIAGNOSIS

Lead poisoning as possible cause of deaths at the Swedish House at Kapp Thordsen, Spitsbergen, winter 1872-3

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Cite this as: *BMJ* 2009;339:b5038
doi:10.1136/bmj.b5038

ABSTRACT

Objective To investigate cause of death in 17 sealers who died in the Swedish house in Kapp Thordsen, Spitsbergen, during the winter of 1872-3.

Design Analysis of skeletal samples from one sealer's grave.

Setting Field trip to Spitsbergen to exhumate skeletal remains.

Subjects One of 17 sailors who died in 1872-3.

Results No objective signs of scurvy were found. The concentration of lead in the bone samples was 102.05 µg/g.

Conclusions The high concentrations of lead indicate that this man died from lead poisoning, probably from food tins. The absence of macroscopic signs of scurvy supports this theory.

INTRODUCTION

In September 1872 six sealing vessels from Tromsø were north of Spitsbergen at the Grey Hook (Grahuken). A northern gale closed the bay with ice, trapping the six ships and 59 sealers with the prospect of "overwintering" with few provisions. At the same time, Adolf Nordenskiöld, the Swedish explorer,¹ was setting up his winter quarters, "Polhem," at Mossell Bay (Mosselbukta) (fig 1). His ships were also trapped, leaving him with a critically low food reserve.

Seven of the stranded sealers crossed the ice, some 50 km, to ask Nordenskiöld for help. He was able to help some but advised the others to spend the winter in the Swedish house at Kapp Thordsen in Isfjorden (fig 2). The house had been set up in the previous summer by a mining company that aborted their project because of poor harbour conditions and was well stocked with tinned food.^{2,3} Axel Envall, Nordenskiöld's physician, advised the sealers on how to avoid scurvy.⁴ The 17 men then rowed some 350 km and reached the house in seven days, on 14 October. Meanwhile, the ice broke up, and the remaining trapped sealers escaped in two ships. Two chose to stay with the remaining ships and died from scurvy in April 1873.⁴

The men at Kapp Thordsen hunted polar bear and reindeer until the arctic night descended and then relied on the stored food. Through a diary they

recorded all food consumed, weather conditions, sickness, and death but gave no details regarding symptoms of disease.⁵

On 2 December, two men were reported as sick; by Christmas, they were all sick. On 19 January, two men died after "hard sickness." Two more died in February, five more in March,⁵ and the last one probably just before they were found on 16 June by members of a hunting expedition from the mainland who wanted to visit them.^{6,7} The first two men who died were buried. The next five men to die were left outside under a tarpaulin,^{6,7} six were found in an adjoining room, and

Fig 1 Map of the north western part of Spitsbergen, showing the rowing route

Fig 2 |The Swedish house viewed from the common grave

the remaining in the living quarters. The rescue team buried them in a common grave.^{6,7} Their deaths were attributed to scurvy due to laziness and bad leadership in allowing scurvy to occur despite an ample food supply and the means to prevent it, and the men were left little honour.⁷

In 1845, Admiral John Franklin set out with 129 men and two ships to find the northwest passage.⁸ They took large amounts of tinned food. The expedition was equipped for four years but was never heard from again. Final evidence of the disaster came in 1859, when a written account of their ordeal was found in a crumble of stones.⁹ The ships had been wrecked by ice, and the men eventually started to walk south, dying as they walked. In the 1980s a team from Alberta University exhumed three sailors buried in permafrost at the expedition's winter quarters and found high concentrations of lead in hair samples.⁸ Bones from crew members, found scattered along the track, showed high concentrations of lead, while Inuit bones did not.¹⁰ The conclusion was that the sailors had lead poisoning, possibly leading to their death.¹⁰

Having pieced together the events in the Swedish house in 1872-3, in light of the Franklin disaster, we saw the similarities. In 2007 we discovered a previously unregistered two man grave, 540 metres from the house. Through a damaged corner, cervical vertebrae were visible and accessible for lead analysis. Several hundred empty tin cans were scattered behind the house (fig 3). The inside solder was evident, and many cans had "icicles" of solder on the sides (fig 4). The discovery of the tin cans with lead/tin solder supported our hypothesis that lead poisoning might have contributed to the deaths.

METHODS

Sample extraction

We applied for permission to open the two graves to take specimens for lead analysis and inspection of bones for signs of scurvy. In August 2008, we revisited the site and sampled a 1×1 cm piece of solder. We dug a shaft 50×50 cm in the upper part of the common grave and encountered wooden planks 60 cm down. Underneath we met an ice block. The bodies were in permafrost, and further exploration was abandoned.

Through the opening in the two man grave, we extracted one humerus, one clavicle, two cervical vertebrae, and a scapula; all from one skeleton. All bones

had smooth surfaces with no signs of subperiosteal bleeding (scaling). Two needle biopsies were taken from the head of the humerus and the body of a cervical vertebra (fig 5). From the grave and from the earth outside (as control) we took swabs for culture of *Clostridium botulinum* and stored it in a sterile transport medium (Sarstedt, D-515588 Nümbrecht). The biopsy samples were examined at the National Institute of Occupational Health, Oslo, and the bacteriological samples were cultured at the National Veterinary Institute, Oslo.

Preparation and analysis of bone samples

Air dried bone samples were added to 1 ml of 65% ultrapure nitric acid (Chemscan, Elverum, Norway) in a polypropylene digestion tube. The sample was digested by heating the tube to 95°C for one hour. The digest was cooled to room temperature and 200 µl of an internal standard solution containing thallium-90 for various lead isotopes was added and diluted to a final volume of 5 ml with ultrapure water.

We analysed the digested bone sample with inductively coupled plasma sector field mass spectrometry (ICP-SF-MS) with an Element 2 mass spectrometer (Thermo Electron, Bremen, Germany) calibrated with nitric acid matched aqueous standard solution. The instrument was programmed to determine lead by use of the various isotopes at low mass resolution. The detection limit (3 SD of all blank samples) for lead in bone was 1 ng/mg if a sample mass of 1 mg was used.¹¹

RESULTS

We found no macroscopic evidence of scurvy. All bone surfaces were smooth with no scaling or enhanced ridges. The bone samples weighed 33 mg and contained 65 µg/g of lead. As this is dry bone, the figure should be multiplied by a factor of 1.57, giving the actual level of lead content of 102.05 µg/g.¹² The lead content of the solder was 40%. The culture for *C botulinum* was unsuccessful because of overgrowth of other bacteria.

Fig 3 |The heap of tin cans behind the house

Fig 4 | Detail of one of the tins, showing “icicles” of solder on the inside

DISCUSSION

Though the deaths of the sealers in Kapp Thordsen have been attributed to scurvy, our research indicates that lead poisoning was a more likely cause. Scurvy, caused by lack of vitamin C, was a plague of seafarers until its prevention was discovered in 1753, and intake of lime juice was implemented in the British navy in 1795.¹³ The disease causes disintegration of connective tissue and can be visualised macroscopically in skeletons of people who have died from scurvy. The subperiosteal bleeding leads to diagnostic scaling of the bone surfaces and enhanced ridges of the muscle attachments.¹⁴ The prevention of scurvy had been documented as early as 1816 in arctic Norway, where sealers and fishermen used “scurvy grass” (*Cochlearia officinalis*).¹⁵

The sealers at Kapp Thordsen had received instructions on how to avoid scurvy, so we think it an unlikely cause of their deaths, particularly we found no signs of previous subperiosteal bleeding. As it has been postulated that botulism might have caused the deaths of those in the Franklin expedition¹⁶ we tested for this but did not succeed in culturing the bacteria.

Lead poisoning

Lead is a poisonous heavy metal that has acute and chronic effects on the body. Metallic lead is poorly dissolved in an alkaline environment but is readily

Fig 5 | Extracting samples from the grave; note the smooth surface of the bones

WHAT IS ALREADY KNOWN ON THIS TOPIC

Early tinned food contained up to 50% of lead in the solder. This has been linked with serious disease and death caused by lead poisoning.

Arctic expeditions have been particularly susceptible.

WHAT THIS STUDY ADDS

Lead poisoning might be the cause of unexplained deaths in the arctic areas.

dissolved in acid. After absorption it is deposited in the skeleton.^{17,18} According to the inventory list, the men at Kapp Thordsen consumed large amounts of tinned food. They probably reheated the tins on the stove, exposing the lead alloy to acid and dissolving the lead in the food.¹⁹

Lead poisoning typically leads to stomach pains and cramps, anorexia, weight loss, a blue line of the gingival margin, neuropathy particularly affecting the extensor muscles, joint pain, and a curious condition called “debility”—inertia and complete loss of incentive and interest.²⁰

The content of lead in our samples was 102.05 µg/g. Samples of soil from outside the burial grounds showed 16 µg/g of lead. The lead content in the bones from the Franklin expedition ranged from 97.6 to 188.6 µg/g, accumulated over about three years.¹⁰ In our case, the lead accumulated over a few weeks. The first men were sick by 2 December. As the accumulation of lead in bone occurs slowly, at a rate of 1 mg a year if the blood concentration is 1.8 µmol/l in cases of chronic lead poisoning,²¹ our victim must have had high blood concentrations of lead. To our knowledge, there are no cases in the literature examining simultaneous blood and bone concentrations of lead. The blood concentrations must therefore be extrapolated.

In the Franklin expedition, if half the bone lead content had been accumulated during the expedition, the cumulative blood lead concentration over this time must have been 29.0–75.5 µmol/l, giving a mean concentration of 29.0–24.2 µmol/l.¹⁰ This is three to 10 times higher the recommended upper concentrations of lead in the blood to prevent neurological damage.²¹ When our “patient” had accumulated 102.6 µg/g in 13–14 weeks, he must have been suffering from acute as well as subacute lead poisoning.

Interpretation

The evidence gives strong indications that our “patient” died of lead poisoning. The other man in the grave also probably suffered the same fate. Possibly lead poisoning, because of its “debilitating” effect and anorexia, worked with other factors, contributing to the men’s deaths.

We thank Tora Hultgreen of Svalbard Museum, Einar Johansen of the Polar Institute, Yngvar Thomassen of the National Institute of Environmental Health, and the National Veterinary Institute for their contributions towards this expedition. A more extensive version of this paper is in press with *Polar Record*.

Contributors: Both authors took part in the planning, excavation, and preparing the manuscript. UA is guarantor.

Funding: This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Competing interests: None declared.

Ethical approval: The study was approved by the National Ethics Committee for Medical Research and the Governor of Cultural Heritage.

Data sharing: No additional data available.

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