

Mawson and Mertz: a re-evaluation of their ill-fated mapping journey during the 1911–1914 Australasian Antarctic Expedition

Denise Carrington-Smith

It is now nearly a century since Douglas Mawson led the 1911–1914 Australasian Expedition to the Antarctic to map a part of the world which was then little known. In November 1912, Mawson, and two companions Belgrave Ninnis and Xavier Mertz, set out on a mapping journey. On 14 December 1912, Ninnis died when he fell into a crevasse. Mertz died on 8 January 1913. A month later, on 8 February 1913, Mawson reached Base Camp, having survived one of the most incredible journeys under conditions of extreme hardship ever recorded. Although it took some months, Mawson made a complete recovery from his ordeal.

In 1969, Cleland and Southcott suggested that both Mawson and Mertz had suffered hypervitaminosis A as a result of eating the livers of the expedition's dogs, a conclusion later supported by Shearman.^{1,2} In 2005, I re-evaluated this hypothesis and suggest that both explorers suffered not from vitamin A toxicosis but from starvation.

A mapping exercise

In 1915, Mawson published an account of the 1911–1914 expedition, including the mapping journey.³ From this we know that Mawson, Ninnis and Mertz had taken sledge dogs with them to enable rapid travel over the first part of their journey to map an area more distant than that being mapped by other members of the expedition. We also know that Mertz and Ninnis were close friends. It was they who had brought the dogs by ship from England and who cared for the dogs for the 10 months the expedition party spent in the Antarctic before they set out on their fateful mapping venture.

When Ninnis fell into a crevasse to his death on 14 December, with him was lost the sledge carrying most of the food supplies. Mawson and Mertz were faced with the daunting prospect of making their way back to Base Camp on very reduced rations. To survive, they had no option but to kill and eat the six remaining dogs.³

The tragedy occurred as Mawson's party were approaching the end of their proposed outward journey, and the dogs were already weakened from the exertion of pulling sledges against strong winds and over very rough ground. By this time, Mawson had divided the dogs into two teams — the strongest and fittest pulling the heavier load, which included all of the dog food and most of the human food, while the weaker dogs pulled the lighter sledge. It was the heavier sledge, with most of the food and all of the stronger dogs, which was lost in the crevasse.

The first of the remaining dogs died the next day. The dogs continued working until they “dropped”, as was their nature. They were then carried on the sledge in a comatose condition until shot

ABSTRACT

- During the Australasian Antarctic Expedition of 1911–1914, Douglas Mawson and two companions, Belgrave Ninnis and Xavier Mertz, undertook an ill-fated mapping journey. Ninnis died when he fell down a crevasse, together with the sledge carrying most of their food supplies, and later Mertz became ill and died. Only Mawson returned.
- In 1969, Cleland and Southcott proposed that Mertz died of vitamin A toxicity and Mawson suffered from the effects of hypervitaminosis A because, with little food left, they were forced to eat their surviving dogs, including the liver. This hypothesis was supported by Shearman in 1978.
- After re-evaluating this hypothesis, I propose that Mawson and Mertz suffered from the effects of severe food deprivation, not from hypervitaminosis A, and that Mertz died as he was unable to tolerate the change from his usual vegetarian diet to a diet of mainly dog meat. I also suggest that Mertz's condition was aggravated by the psychological stress of being forced to eat the dogs he had cared for for 18 months.

MJA 2005; 183: 638–641

and used for food for both man and dog. It is clear that the dogs were already severely malnourished — Mawson described the dog meat as “tough, stringy and without a vestige of fat”.³

In his account, Mawson made it clear that all rations were shared with the utmost impartiality.³ It may be assumed, therefore, that Mawson and Mertz shared all rations equally until 31 December, when Mertz began to feel unwell¹ and being vegetarian and understandably finding the dog meat difficult to stomach, requested an extra portion of dried milk powder, while Mawson took an extra ration of dog meat in exchange.^{1–3} This is contrary to the suggestion by Shearman that Mertz may have found the liver less repulsive and they may have struck a “bargain” on this issue.²

If the food was shared equally, then in the 3½ weeks before he died, Mertz could have eaten no more than three husky livers — about one a week or one-seventh per day. Levels of vitamin A in Antarctic husky liver vary considerably, even in healthy dogs.⁴ Although it is difficult to form an estimate of the likely vitamin A content in the livers of these emaciated dogs, it was probably low.

Several hypotheses

Vitamin A toxicosis?

In 1912, while Mawson's expedition was in the Antarctic, the term “vitamins” was first used to describe a new class of nutrients believed necessary to support life, and 1915 saw the discovery of the fat-soluble vitamin “A” in cod-liver oil and in butter. The period between the World Wars was one of great discoveries in relation to the role of vitamins and the effects of vitamin deficiencies, but it was not until after World War II that vitamin

PO Box 1143, Mossman, QLD.

Denise Carrington-Smith, BA(Psych), MSocSc(Archaeology), PhD Student.

Reprints will not be available from the author. Correspondence: Ms Denise Carrington-Smith, PO Box 1143, Mossman, QLD 4873. Denise.Carrington-Smith@jcu.edu.au



Ninnis and Mawson on the deck of the SY [Steam Yacht] Aurora on the voyage down to Antarctica. (Photographer, Percy Gray. © Mitchell Library, State Library of New South Wales.)



Mertz leaving the hut [at Base Camp] by the trapdoor in the verandah roof. (Photographer, Frank Hurley. © Mitchell Library, State Library of New South Wales.)

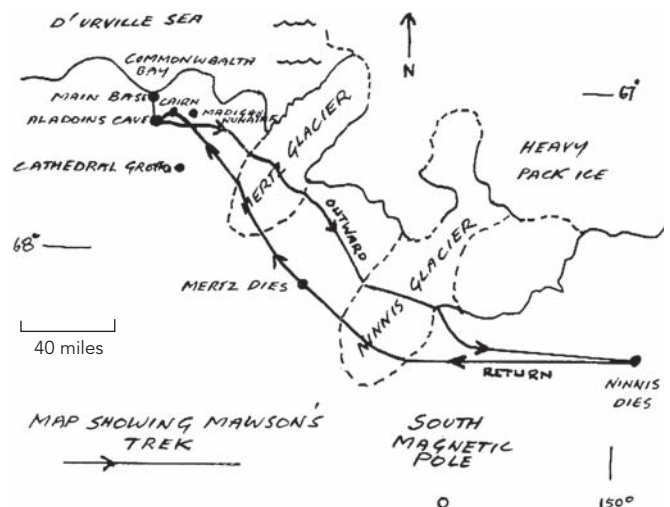
myalgia; and dizziness, blurred vision, increased intracranial pressure (causing bulging of the fontanelles in infants and severe headache in adults); and irritability and depression.^{5,6-15}

In contrast, most of the evidence for acute vitamin A poisoning remains anecdotal — the assumed acute symptoms of one case used to support the diagnosis of the next. Such anecdotal evidence of people in polar regions becoming sick soon after eating the liver of Arctic fish and sea mammals has been reported for several centuries.^{4,5,16} Symptoms included abdominal pains, nausea, vomiting, diarrhoea, headache (at times, severe), dizziness, blurred vision, irritability, sluggishness and a desire to sleep (at times, overwhelming), starting within a few hours of the meal, followed by rapid recovery. In some cases, desquamation of the skin followed.^{4,17}

Because the livers of some bears and seals have been found to be rich in vitamin A, it has been assumed that the cause of these illnesses was acute vitamin A poisoning. However, most of the cases occurred in times and/or places far removed from medical attention, so that no analysis of the patient or remains of the food consumed was possible. An exception is a report by Lonie of some Chinese fishermen living in New Zealand who consumed a shark

preparations became widely available, were consumed in large quantities by the general public, and attention began to focus on the possible effects of vitamin overdoses.

The symptoms of chronic hypervitaminosis A are well documented. Vitamin A, being fat soluble and stored in the body to some extent, is known to exhibit toxicity at very high dosages taken over long periods of time. However, most reports have been related to the ingestion of large amounts of the vitamin in tablet form over extended periods of time — usually several years rather than weeks. Symptoms have included coarseness and sparseness of hair of the scalp, eyebrows and other parts of the body; dryness of the skin, ulceration, and desquamation; hepatosplenomegaly; anorexia and diarrhoea; cessation of menstruation; haemorrhagic tendency; hyperostosis, bone tenderness or pain, especially of the distal extremities (which may be accompanied by weakness);



Sketch of the mapping journey undertaken by Mawson, Mertz and Ninnis, showing the glaciers named after the two men who died.

HISTORY

liver and soy bean paste with rice, and suffered headache and continual vomiting for some hours. Desquamation followed.¹⁸

Furman described a laboratory worker who self-medicated with 1 300 000 IU of vitamin A over a 27-hour period and suffered intense headache, blurred vision, and was unable to sit or stand because of dizziness and vertigo. Desquamation followed a few days later.¹⁷ This is one of the few cases of acute vitamin A poisoning in which there was immediate medical evaluation. However, Furman noted that this case appeared to be one of individual hyperreactivity, as many other patients have taken far higher doses over longer periods without ill effect.¹⁷ Nevertheless, similar to all accounts to date of presumed acute vitamin A toxicity, in this case there was a rapid onset and a rapid recovery.

Vilhjalmur Stefansson made it his life work to study the life and diet of the Eskimo.¹⁹ Some of his companions had experienced headache, nausea and weakness after eating bear liver, although they recovered the next day. On one occasion, Stefansson and three companions experimented by dividing up a bear liver between them. One man felt very nauseous, Stefansson suffered loss of appetite, the other two suffered no ill effects.¹⁹

While some of the symptoms suffered by Mawson and Mertz occur in hypervitaminosis A, none were exclusively those of vitamin A toxicity. However, these symptoms may also be attributed to severe food deprivation and the effects of the cold and wet conditions which the pair were forced to endure. Mawson and Mertz had no change of clothing and wore their damp clothing for weeks on end, to say nothing of sleeping in damp sleeping bags.³

Further, no mention is made by Mawson, in his meticulous account, of the symptoms which would have been expected in

acute vitamin A toxicity — headache, nausea, vomiting, dizziness, weakness of the legs, excessive tiredness or haemorrhaging.^{3,20} If there was sufficient vitamin A present in the husky livers to cause acute toxicity, more of these acute symptoms should have been apparent. Indeed, it is difficult to comprehend how Mawson could have completed his journey, involving as it did extreme physical exertion, had he been suffering vitamin A toxicity.

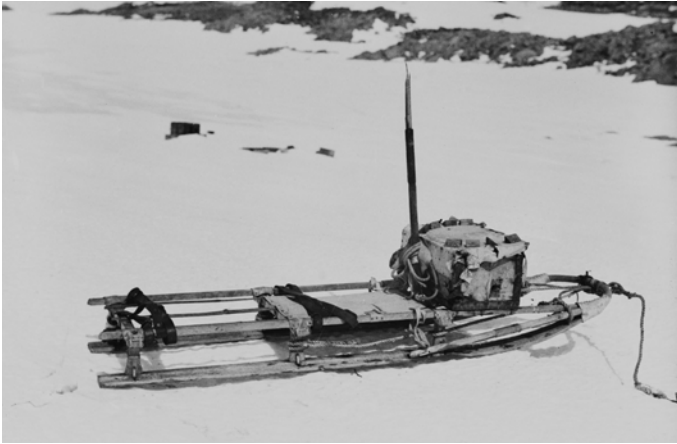
Scurvy?

In their differential diagnosis between hypervitaminosis A and scurvy, Cleland and Southcott pointed out that the symptoms of scurvy include swelling of the gums, ecchymoses and petechiae due to fragility of the capillaries.¹ Capillary fragility results in easy bruising which would have been expected to follow the heavy labour of sledge pulling and the falls experienced.

Mawson, in particular, had several falls down crevasses, and records being saved by the rope attached to his sledge, the latter having jammed across the top of the crevasse. It took a considerable struggle for Mawson to haul himself out of the crevasse. Almost at the top, he slipped back and was once again left dangling. Only a superhuman effort enabled him to struggle back up his rope a second time. After that, Mawson made himself a rope ladder. Another time, he cut one of the sledges in half using a pocket knife. It is doubtful that he would have been able to do all these things without considerable bruising or haemorrhaging had he been suffering from scurvy. This was pointed out by Cleland and Southcott and used as an argument against the presence of scurvy.¹ However, the same argument could also be made in respect of vitamin A poisoning, as easy bruising is a symptom common to both a deficiency of vitamin C and an excess of vitamin A.

Sledging across the Adelie Land plateau. (Photographer, Douglas Mawson. © Mitchell Library, State Library of New South Wales.)





Half sledge brought back by Mawson on returning from the fateful sledge journey. (Photographer, Frank Hurley. © Mitchell Library, State Library of New South Wales.)

Cleland and Southcott further claimed that scurvy could not have been present as “No other member of the expedition was reported as having any symptoms which could be suggestive of scurvy”; however, the condition of the other members of the expedition could not be considered as relevant here, as they suffered no shortage of food.¹

Starvation?

No evaluation of Mawson and Mertz's experiences would be complete without taking into account possible vitamin deficiencies resulting from food deprivation. For example, the vitamin B group is known to be of special value in times of stress, and lack of this group leads to dermatitis (especially of body parts exposed either to sunlight or friction), loss of hair and sloughing of the skin, symptoms suffered by Mawson and Mertz.^{19,21,22}

I suggest that their symptoms may be explained by generalised malnutrition, including vitamin deficiencies, as a result of extreme food deprivation. The desquamation noted most likely resulted from the contact of their emaciated skin with damp clothing under conditions of extreme cold accompanied by blizzard-like biting winds.

Why did Mertz die?

Why did Mertz, and not Mawson, die? Mertz was “near vegetarian”.^{2,23} He accepted the need to eat pemmican biscuits, made from dried, powdered beef, as part of the sledging rations, but this is a far cry from being forced to eat the flesh of his beloved dogs. When Ninnis died, Mawson suffered the loss of a companion and a member of the party for whom he was ultimately responsible, but Mertz had lost a close friend. Indeed, he had lost seven friends, one human and six animals. Not only did Mertz lose these friends, but the remaining dogs were dying, one by one. In addition to witnessing their suffering, he then had to assist in their final killing and to eat their flesh.

Also, a sudden change of diet to one consisting mainly of meat would have added to the difficulties that he and Mawson faced. Draper has reported that a sudden change from a mixed diet to a primarily meat-based diet leads to asymptomatic ketosis and ketonuria.²⁴ This being the case, the change by Mertz, not from a

“mixed” diet but from a vegetarian one, to a diet based primarily on meat may have resulted in problems which have not yet been considered.

These two additional factors, the psychological stresses related to the death of a close friend and the deaths of the dogs he had cared for, as well as the need to kill and eat his remaining dogs, and the physiological stress caused by a change in diet, may have contributed to Mertz's death. Paradoxically, Mertz' death probably saved Mawson's life, as it made available a double ration of the remaining food. Although the nutritional value of the dog meat would have been low, such little as there was may have contributed to Mawson's survival.

References

- 1 Cleland J, Southcott RV. Hypervitaminosis A in the Antarctic in the Australasian Antarctic Expedition of 1911-1914: a possible explanation of the illness of Mertz and Mawson. *Med J Aust* 1969; 1: 1337-1342.
- 2 Shearman DJ. Vitamin A and Sir Douglas Mawson. *BMJ* 1978; 1: 283-285.
- 3 Mawson D. The home of the blizzard. London: William Heinemann, 1915.
- 4 Southcott R, Chesterfield N, Lugg D. Vitamin A content in the livers of huskies and some seals from Antarctic and subantarctic regions. *Med J Aust* 1971; 1: 311-313.
- 5 Rodahl K, Moore T. The vitamin A content and toxicity of bear and seal liver. *Biochem J* 1943; 37: 166-168.
- 6 Di Benedetto RJ. Chronic hypervitaminosis A in an adult. *JAMA* 1967; 201: 700-702.
- 7 Feldman MH, Scheizinger NS. Benign intracranial hypertension associated with hypervitaminosis A. *Arch Neurol* 1970; 22: 1-7.
- 8 Frame B, Jackson CE, Reynolds WA, Umphrey JE. Hypercalcemia and skeletal effects in chronic hypervitaminosis A. *Ann Intern Med* 1974; 80: 44-48.
- 9 Gerber A, Raab AP, Sobel AE. Vitamin A poisoning in adults; with description of a case. *Am J Med* 1954; 16: 729-745.
- 10 Hillman RW. Hypervitaminosis A: experimental induction in the human subject. *Am J Clin Nutr* 1956; 4: 603-608.
- 11 Morrice G Jr, Havener WH, Kapetansky F. Vitamin A intoxication as a cause of pseudotumor cerebri. *JAMA* 1960; 173: 1802-1805.
- 12 Muentner MD, Perry HO, Ludwig J. Chronic vitamin A intoxication in adults. *Am J Med* 1971; 50: 129-136.
- 13 Pease CN. Focal retardation and arrestment of growth of bones due to vitamin A intoxication. *JAMA* 1962; 182: 980-985.
- 14 Stimson WH. Vitamin A intoxication in adults: report of a case with a summary of the literature. *N Engl J Med* 1961; 265: 369-373.
- 15 Sulzberger MB, Lazar P. Hypervitaminosis A: report of a case in an adult. *JAMA* 1951; 146: 788-793.
- 16 Kane EK. Arctic explorations: the second Grinnell expedition in search of Sir John Franklin, 1853, '54, '55 (Vol 1). Philadelphia: Childs & Peterson, 1856.
- 17 Furman KI. Acute hypervitaminosis A in an adult. *Am J Clin Nutr* 1973; 26: 575-577.
- 18 Lonie TC. Excess vitamin A as a cause of food poisoning. *N Z Med J* 1950; 49: 680-685.
- 19 Feeney RE. Polar journeys: the role of food and nutrition in early exploration. Washington, DC: American Chemical Society, and Fairbanks: University of Alaska Press, 1997.
- 20 Jacka F, Jacka E, editors. Mawson's Antarctic diaries. Sydney: Allen & Unwin, 1988.
- 21 Best CH, Taylor NB. The physiological basis of medical practice. Baltimore: Williams and Wilkins, 1961.
- 22 Rosenberg HR. Chemistry and physiology of the vitamins. New York: Interscience Publishers Inc, 1945.
- 23 Bickel L. This accursed land. Melbourne: Macmillan, 1977.
- 24 Draper HH. The aboriginal Eskimo diet in modern perspective. *Am Anthropol* 1977; 79: 309-316.

(Received 20 Apr 2005, accepted 31 Oct 2005)

□