

## RECENT STUDIES ON VITAMINS

VITAMIN SOURCES IN ARCTIC REGIONS. By KAARE RODAHL. Oslo: Norsk Polarinstitutt, *Skrifter* 91, 1949. 10½ x 6½ inches; 64 pages; illustrations and map. Swedish kroner 6.00.

THE TOXIC EFFECT OF POLAR BEAR LIVER. By KAARE RODAHL. Oslo: Norsk Polarinstitutt, *Skrifter* 92, 1949. 10½ x 6½ inches; 90 pages; diagrams and illustrations. Swedish kroner, 12.50.

HYPERVITAMINOSIS A. By KAARE RODAHL. Oslo: Norsk Polarinstitutt, *Skrifter* 95, 1950. 10½ x 6½ inches; 206 pages; diagrams and plates. Swedish kroner, 22.50.

Three of the recent papers in the "Skrifters" published by the Norsk Polarinstitutt deal with studies on vitamins and will be of interest to all arctic scientists and explorers. These papers were written by the Norwegian scientist Kaare Rodahl, who has participated in a number of scientific expeditions to the Arctic during the past ten years and has studied many of the nutritional problems peculiar to these regions.

The first two papers contain some of the results of Dr. Rodahl's research carried out on three expeditions: in northeast Greenland on a wintering expedition in 1939-40; on the Newfoundland seal fishery in 1941; and in Peary Land, as a member of the reconnoitering party of the Danish Pearyland Expedition in 1947. The third considers the results of laboratory experiments designed to verify some of the points raised in the second paper.

The first paper deals with the vitamin sources in the arctic regions. In the first part Dr. Rodahl describes his investigations of the vitamin A content of the liver of the hooded seal (*Cystophora cristata*) and the harp seal (*Phoca groenlandica*). Over 10,000 livers from these two species were collected and assayed for vitamin A. The livers were found to have a fairly high vitamin A potency (mean value: 3,000-3,400 I.U. vitamin A per gram liver), and the values were of the same order in both species with similar variations when age and sex were taken into consideration. A seasonal variation in vitamin A content of the livers was observed; this could be explained by the feeding habits of the seal during breeding.

In the second part of the paper Dr. Rodahl gives the results of studies on the vitamin C content of the most common mammals, fishes, and birds of northeast Greenland; his work on the common plants of the same region was published in an earlier paper.<sup>1</sup> Liver, brain, kidney, and other internal organs of the musk ox and seal were found to be rich in vitamin C. The epidermis of the narwhal, the *mattaq*, which is used by the Eskimo as food, was found to be rich in vitamin C. In general, some of the internal organs (liver, brain, kidney, testicles, adrenals, thyroids, pancreas, lymph glands, etc.) of most arctic mammals proved to be excellent sources of vitamin C, as were the roe of salmon, and the brain, liver, and intestines of most arctic birds.

The author notes that some of these organs are not considered edible by the European trappers, while the Eskimo consume them all. This is probably the explanation for the fact that scurvy is unknown among the Eskimo of northeast Greenland, who must obtain all their vitamin C from the local flora and fauna, while cases of scurvy and other vitamin deficiency diseases have been reported among the white trappers even in recent years. In a dietary survey carried out among a group of Norwegian trappers the consumption of vitamin B<sub>1</sub> and vitamin C was found to be lower than accepted values for human requirements.

In his second paper Dr. Rodahl gives the results of an investigation of the toxicity of the polar bear liver. It has long been believed that illness follows the eating of polar bear liver. This belief, which is firmly established among the Eskimo, appears to be well founded. It seems, however, that certain livers are

<sup>1</sup>Rodahl, K., "Content of vitamin C (1-ascorbic acid) in arctic plants." *Trans. Bot. Soc. Edin.* Vol. 34, Pt. 1 (1944) pp. 205-10.

poisonous while others are not. Many explorers have experienced illness after eating polar bear liver, but a few have suffered no ill effects. The liver of the bearded seal was also found to be poisonous.

Prior to 1939 no attempt had been made to identify the substance responsible for the toxic effects of these livers. Certain symptoms which were reported by a number of explorers who had eaten polar bear liver, seemed to link the illness with a condition of hypervitaminosis A.

In 1939 Dr. Rodahl collected samples of polar bear livers in northeast Greenland which, when assayed, proved to be very rich in vitamin A (18,000 I.U. vitamin A per gram liver). Preliminary experiments carried out on rats fed with the liver indicated that the ill effects produced by the ingestion of polar bear liver were due to hypervitaminosis A. In 1947, during the Danish expedition to Peary Land, more samples were collected and again proved to be very rich in vitamin A (21,900 and 26,700 I.U. vitamin A per gram liver). Samples of bearded seal, arctic fox, walrus, and arctic hare livers were also assayed. The bearded seal and the arctic fox livers were found to be very rich in vitamin A (15,000 and 12,000 I.U. vitamin A per gram liver), while the walrus and arctic hare were relatively poor (1,200 I.U. vitamin A per gram liver). The Eskimo appear to recognize this difference as they eat the walrus and arctic hare livers but not those of the polar bear, bearded seal, and arctic fox. Dr. Rodahl's further experiments have shown that polar bear liver is toxic to rats (average: 0.5-0.7 gram liver per day corresponding to 13,350-18,690 I.U. vitamin A). The following symptoms were observed: reduced weight increase, haemorrhages, oedema of the palpebrae, soreness around the mouth, alopecia, limping, fractures, and death. Polar bear-liver oil in average daily doses of less than 30,000 I.U. vitamin A produced the same symptoms, but fat-free polar bear liver fed to rats caused no ill effect nor did vitamin A-free polar bear-liver oil. Rats given purified whale-liver oil (31,600 I.U. vitamin A per day) showed the same symptoms as those produced by the ingestion of polar bear-liver oil containing a similar quantity of vitamin A.

Dr. Rodahl concludes that polar bear liver is toxic to rats and that the toxic substance is vitamin A in excess, the illness produced being a condition of hypervitaminosis. He also showed that the symptoms in rats are proportional to the doses of vitamin A or the quantity of liver ingested. It is natural to assume that in the liver of bearded seal and arctic fox, known to be toxic and rich in vitamin A, the toxic factor is also vitamin A in excess. The absence of toxicity of certain livers found by some explorers, can be explained by the variation in vitamin A content of the liver, resulting from factors such as age and hibernating and feeding habits of the polar bear.

The condition of hypervitaminosis A has been known for 25 years but many aspects need further investigation. Dr. Rodahl reports the case of a man who ingested vitamin A in the form of halibut-liver oil. After smaller daily doses than may have been absorbed by explorers eating a meal of polar bear liver, he developed symptoms of the same kind. The studies on the toxicity of polar bear liver, which identified the toxic factor of the liver as vitamin A in excess, led to further work on the nature of the condition known as hypervitaminosis A. The results of these studies which are presented in his third paper are of interest to the physiologist and the nutritionist. Dr. Rodahl investigated in detail the following problems:

- 1) the possibility that toxic substances other than excess vitamin A were contained in liver oils
- 2) the influence of the method of administration of vitamin A
- 3) the effect of a massive single dose of vitamin A compared with the effect of prolonged administration of excessive amounts of vitamin A
- 4) the dose which produces toxic effects at different stages of development
- 5) the clinical manifestations and the pathological changes
- 6) the relation between hypervitaminosis A and the other vitamins.

The results of experiments have shown clearly that vitamin A is the toxic factor in the liver oils. Highly purified vitamin A given in excess produced the same symptoms on rats as vitamin A given in equivalent amounts in the form of whale-liver oil. Whale-liver oil in which vitamin A was destroyed caused no intoxication.

Oral, local, or subcutaneous administration of excessive amounts of vitamin A over a long period of time produced the same symptoms, although subcutaneous injection, due to a lower rate of absorption, produced the symptoms to a lesser degree.

Oral administration of a single massive dose produced acute intoxication with no detectable pathological changes and no lethal effect, while prolonged administration of the vitamin in excess produced a condition of hypervitaminosis A with clinical manifestations and pathological changes. The sex and age did not alter the symptoms, although the condition appeared later in older animals.

Doses over 50-100 I.U. vitamin A per gram body weight daily caused slight manifestations of hypervitaminosis A in rats. Doses between 200-500 I.U. vitamin A per gram body weight were particularly toxic while doses over 800 I.U. were lethal.

The acute intoxication produced by single massive doses of the vitamin was characterized by changes in the pelts, drowsiness, muscular weakness, and reduced activity. Chronic intoxication caused by prolonged administration of great quantities of vitamin A, produced symptoms similar to those of the rats fed 0.5-0.7 gram of bear liver per day. No pathological findings were significant except emaciation, arterial hyperemia, subcutaneous, muscular and visceral haemorrhages, thinning of the bone shaft which caused spontaneous fractures, enlarged adrenals, swelling of the visceral lymph glands, and fatty liver.

Additional vitamin B<sub>1</sub> had no influence on any of the symptoms of hypervitaminosis A. The condition of hypervitaminosis A did not prevent the development of rickets in rats fed a rachitic diet, though excessive doses of vitamin A proved to be more injurious to them than to normally fed rats. Hypervitaminosis A did not counteract the ricket-curing effect of vitamin D. Although in a condition of hypervitaminosis A the prothrombin time is prolonged, additional supply of vitamin K did not influence the appearance of haemorrhages or any of the other symptoms.

In the condition of hypervitaminosis A the liver and the serum had a very low ascorbic acid content. Large doses of vitamin C did not give any significant protection against the injurious effects of massive doses of vitamin A although the enlargement of the adrenals was prevented. However vitamin C had a beneficial effect against moderate excess of vitamin A. A great similarity exists between the symptoms of hypervitaminosis A as described above and the symptoms of scurvy. The complex clinical picture of hypervitaminosis A indicates an interrelation between vitamin A and vitamin C.

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