

Example of vitamin a research paper

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There are multiple forms of vitamin A and each form has a specific function. Vitamin A is retinol, once it undergoes oxidation it becomes retinaldehyde and retinoic acid, which are also biologically active compounds.

Retinaldehyde is essential for normal vision, while retinoic acid is important for morphogenesis and growth. Retinol plays a role in reproduction, iron utilization, and immunity (Harrison's, 2008, p. 446).

One person did not discover Vitamin A; its discovery was spread out over a long period of time. The earliest research began in 1816 with experiments in malnutrition on dogs. Later, in the late 19th century, it was shown that some substance found in milk was essential to nutrition. By the early twentieth century it was recognized that this substance was fat soluble and that the fats it was located in had different nutritional values than other fats. The growth supporting factor in the fat was eventually called vitamin A for "accessory factor." Karrer described the chemical structure of retinol in 1932 and it was isolated and crystalized in 1937 by Holmes and Corbet. In 1946 it was finally synthesized by Adriaan van Dorp and Ferdinand Arens (Semba, 2012).

Vitamin A comes from a wide variety of sources including meats and vegetables. Meats contain preformed vitamin A, whereas vegetables contain carotenes and pro-vitamin A. Animal sources are liver, fish, eggs, milk, and butter. Yellow or leafy vegetables like carrots, pumpkins, or spinach. The vegetable carotenoids are metabolized in the gut to active forms of vitamin A and usually form about 30% of the vitamin A in the body. B-carotene is the form that most easily converts to vitamin A (Robbins, 2007, p. 306). No specific signs of deficiency exist. Vitamin A deficiency is wide spread in poor

countries in Southern Asia, Sub-saharan Africa, parts of the Western Pacific, and even parts of Latin America. Normal ranges of vitamin A in the serum are 1.05-3 mol/L. Approximately 125 million young children have vitamin A deficiency and have a particular eye manifestation called xerophthalmia, which causes night blindness, dryness, and white patches on the sclera called Bitot's spots. Sometimes necrosis and ulcers can develop on the eye. Eventually a softening of the cornea called keratomalacia causes scarring of the cornea and blindness. At any age deficiency is associated with diarrhea, dysentery, measles, malaria, and respiratory disease, and is further implicated in immune malfunction (Harrison's, 2008, p. 447).

Signs of toxicity were first noticed when polar bear livers were eaten by arctic explorers. Toxicity is seen after more than 150mg in adults or 100mg in children. Toxicity causes increased intracranial pressure, vertigo, double vision, seizures, and dermatitis. In addition, it can make children's heads appear large. Chronic intoxication occurs in children who ingest 6mg per day every day over several months, or adults who ingest more than 15mg daily over many months. Evidence of chronicity is in dry skin, glossitis, vomiting, alopecia, bone pain, hyper calcemia, hyperlipidemia, increased intracranial pressure, and amenorrhea (Harrison's, 2008, p. 447).

Xerophthalmia should be treated with 60mg of vitamin A in oily solution in a soft-gel capsule, repeated 1 and 14 days later. Mothers with Bitot's spots or nightblindness get a 3mg or 7.5 dose of vitamin A, twice a day for three months. However, in industrialized countries there is rarely deficiency except in extremely low-birth-weight babies who are also supplemented with an oily solution of vitamin A. Diseases causing malabsorption may cause a

deficiency and treating the underlying cause is critical to correcting the deficiency, however patients are supplemented with 15mg/day, miscible in water for 1 month. (Harrison's, 2008, p. 447).

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