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Perspective

Source and Benefits of Vitamin B_{12} in Human Body

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DESCRIPTION

Vitamin B_{12} is a critical component for methylation activities that are vital in DNA and cell metabolism responses, a deficit can cause DNA and cell metabolism to be disrupted, which can have major clinical repercussions. For the homeostasis of methylmalonic acid and homocysteine, intracellular conversion of vitamin B_{12} to two active coenzymes, adenosylcobalamin in mitochondria and methylcobalamin in the cytoplasm is required. Vitamin B_{12} serves as a cofactor in the conversion of methylmalonic acid to succinyl-CoA [1]. Methionine is biosynthesized into homocysteine, which is then resynthesized or transformed into the amino acid cysteine. Vitamin B_{12} deficiency (also known as cobalamin deficiency) is rather prevalent, with significant and varied clinical implications.

Vitamin B_{12} is only found in animal products such as meat, fish and dairy. Vitamin B_{12} is found in the Western diet in amounts of 5 g to 30 g per day, of which 1 g to 5 g is absorbed. The UK government recommends 1.5 g of vitamin B_{12} per day, whereas the European Union recommends 1 g and the US recommends 2.4 g [2]. The amount of storage in the body is relatively high, ranging from 1 mg to 5 mg. As a result, insufficiency due to decreased intake or absorption may not appear for several years after stocks have been depleted. Deficiency can occur in a variety of populations as a result of increasing requirements, such as during growth in children and adolescence or during pregnancy.

Certain groups, such as those with poor nutrition, the elderly or those who follow a vegan or vegetarian diet may have lower intake. Vitamin B_{12} deficiency is prevalent in the United Kingdom and the United States with a prevalence of roughly 6% in those under the age of 60 years and closer to 20% in those over 60 years. Approximately 40% of children and adults in Latin America have clinical or subclinical deficiency. Deficiency is far more common in African and Asian countries with 70% of Kenyan schoolchildren, 80% of Indian preschool children and 70% of Indian adults suffering from it [3]. In the United Kingdom, 11% of vegans are low in vitamin B_{12}, but in Ethiopia 62% of vegetarian pregnant women are deficient.

Vitamin B_{12} is coupled to haptocorrin as holohaptocorrin (officially transcobalamin III) and transcobalamin as holotranscobalamin in the bloodstream. 80% to 94% of natural plasma vitamin B_{12} is holohaptocorrin. On the other hand, holotranscobalamin contributes for 6% to 20% of bound vitamin B_{12}. It is produced by enterocytes and is responsible for the uptake of vitamin B_{12} from the ileum into the bloodstream and other cells *via* receptor-mediated endocytosis. Only holotranscobalamin bound vitamin B_{12} is presented for cellular absorption. Vitamin B_{12} insufficiency is caused by the malabsorption of this holotranscobalamin protein bound vitamin B_{12} [4]. The parietal cells of the cardiac and fundic mucosa of the stomach create intrinsic factor, which is a protein. It binds vitamin B_{12} to facilitate absorption *via* the gastrointestinal system *via* an intrinsic factor receptor found only in cells of the terminal ileum. As a result of malabsorption, resection or illness of the gastric mucosa or terminal ileum causes vitamin B_{12} insufficiency. Pernicious anaemia is an autoimmune disease characterised by atrophy of the body's gastric mucosa and stomach fundus. This decreases the amount of parietal cells that create the intrinsic factor required for vitamin B_{12} absorption. Intrinsic factor secretion is analogous to stomach acid secretion consequently, in an alkaline environment established by long term use of high dose proton pump inhibitors and related medications, there will be reduced secretion.

Mild deficiency is characterized by fatigue and anaemia, with indicators indicating B_{12} deficiency but no neurological symptoms. An evident macrocytic anaemia with for example, glossitis and some mild or subtle neurological symptoms, such as distal sensory impairment, are all signs of moderate insufficiency [5]. Severe deficiency is characterized by bone marrow suppression, neurological manifestations and the risk of cardiomyopathy. It's crucial to remember however, that clinical signs of deficiency can appear without anaemia or low serum vitamin B_{12} levels. Treatment should still be delivered as soon as possible in these circumstances.

Vitamin B_{12} deficiency caused by pernicious anaemia cannot currently be prevented. Deficiencies caused by gastric and

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terminal ileum disease should be predicted and supplemented before symptoms appear. As a non-animal dietary source of vitamin B_{12}, breakfast cereals are fortified. This may be beneficial to persons over the age of 65 years and those who have a restricted diet. Each serving contains about a quarter of the recommended daily vitamin B_{12} requirement. Oral cyanocobalamin, as well as heightened screening and surveillance for vitamin B_{12} deficiency, might be considered for a person using long term metformin and proton pump inhibitors.

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