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Dietary Sulfur Requirement

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Short Communication

There has been a growing account for a vegan, not vegetarian, based diet. What this requires is that all food nutrients are derived from vegetarian sources. While an exclusive vegetarian sourced diet is possible (excluding meats, fish, milk, and eggs), it is unnecessary and is not without risk. The main risk to consider is obtaining a sufficient essential amino acid L-methionine. This can be achieved by eating a sufficient amount of nuts, soy, and several beans, any of which has less methionine than an animal sourced protein. However, a sufficient quantity is necessary.

At the heart of protein-energy malnutrition lies a deficiency of sulfur amino acids (methionine cysteine glutathione) which explain most of the edematous malnutrition [1]. This condition is particularly important in children, and occurs in sulfur poor regions and those of extreme poverty. A typical serum blood level would be less than 2.8g/dl. More importantly, the serum transthyretin level is extremely low, less than 12mg/dl. Methionine is only obtained by food intake, and unlike other amino acids, it is essential for the tissue synthesis of protein.

Methionine and other sulfur amino acids such as cysteine [2] govern circulating glutathione levels which are very low in kwashiorkor. Circulating glutathione clears free radicals and also nitric oxide produced by activated immune cells. In addition, experimental evidence indicates that antioxidant control did not prevent the onset of kwashiorkor [3]. Kwashiorkor may hopefully be substantially eliminated by provision of adequate protein to malnourished infants.

Cachexia causes weight loss and increased mortality. It affects more than 5 million persons in the United States. Other causes of weight loss include anorexia, sarcopenia, and dehydration [4]. Cytokines play a major role in immunomodulation and have been implicated in the etiology of anorexia, weight loss, cognitive dysfunction, anemia, and frailty. Excessive elaboration of proinflammatory cytokines such as interleukin (IL) 1, IL-2, interferon γ , and tumor necrosis factor (TNF- α) is probably the most common cause of cachexia observed in acutely ill patients [4]. This is not an inference of protein energy malnutrition, per se, but it is a result of induced negative nitrogen balance with breakdown of lean body mass without sufficient protein renewal. In addition, prolonged exposure to oxygen in patients induced a steady state of hypoalbuminemia [5] compared to zero time. The groups differed with regard to growth characteristics. Recurrent infection rates and duration of ventilation were consistently worse among patients with indicators of malnutrition.

According to R Fisher [6], in the majority of cases, chronic gastrointestinal disease is synonymous with wasting, at least until the disease is treated. This is related to any of the following: obstruction to the delivery of nutrients; intestinal mucosal diseases leading to loss of mucosal absorptive surface; intraluminal maldigestion; pancreatic insufficiency; and hepatobiliary diseases. Accordingly, intestinal mucosal diseases constitute the largest proportion of the scenarios when wasting occurs as a result of or in the presence of gastrointestinal diseases.

Proteins are primarily synthesized in the endoplasmic reticulum (ER). This organelle is equipped with a variety of molecular chaperones and folding enzymes that are engaged in the folding process. A major posttranslational modification of ER-synthesized proteins is disulfide bridge formation, which is catalyzed by the family of protein disulfide isomerases [7].

Sulfur (S)-deficiency is a significant factor in the sizeable proportion of hyperhomocysteinemic patients experiencing varying degrees of nitrogen (N)-depletion. Decreasing LBM resulting from insufficient dietary protein and/or excessive hypercatabolic losses induces proportionate S-losses. Elevation of homocysteine plasma levels is negatively correlated with LBM reduction and declining TTR plasma levels [8]. In states of inadequate caloric intake or catabolic disease, the overall breakdown of cell proteins, especially in skeletal muscle, increases to provide the organism with amino acids essential for gluconeogenesis, new protein synthesis and energy production [9]. While many physiological and disease processes can stimulate the breakdown of proteins and amino acids

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in muscle, mammals also have adaptive mechanisms that conserve cell protein and essential amino acids. Clinically, individuals fed a very-low protein diet (but adequate calories) exhibit a reduction in T3 levels, and there is a decrease in overall protein degradation [10]. Furthermore, it has been shown that in the acutely malnourished and infected state, children with edematous severe childhood under nutrition have slower methionine production than do children with nonedematous sulfur under nutrition because of a slower rate of release from protein breakdown [11]. Moreover, there is a shortage in cysteine availability for GSH synthesis in children with edematous severe childhood under nutrition [12].

Only 2 of the 20 amino acids present in proteins contains sulfur, even though sulfur is the third most abundant element in the human body. Methionine cannot be synthesized by our bodies, is therefore essential, and therefore is necessary for the synthesis of cysteine and other sulfur amino acids by our bodies [11]. It has been suggested that nitrogen balance should be evaluated as a study methionine for S balance, which is not done. A study of dietary intake of sulfur (as sulfur amino acids) in a random population and perform sulfur balance studies was carried out in a limited number of human volunteers.

The results suggested that a significant proportion of the population that included disproportionately the aged, may not be receiving sufficient sulfur [13].

It is known that sulfur amino acids can be used to increase synthesis of S-adenosylmethionine (SAME), glutathione (GSH), taurine, and N-acetylcysteine (NAC) [14]. These include methionine, cysteine, cystine, homocysteine, homocystine and taurine. Moreover, dietary SAA analysis and protein supplementation is suggested for vegan athletes, children, or patients with HIV, because of an increased risk for SAA deficiency in these groups.

A study examined whether a modification of protein source in the diet could lower the diabetes risk. The result of the study indicated that a plant based sulfur amino acid source combined with fish lowers the branched chain amino acids in the circulation of the study participants [15]. It should be noted that the fish in the diet is also a very good source of omega-3 fatty acids.

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