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Editorial

Flavin Cofactors FMN and FAD, the Biologically Active Forms of Riboflavin and Healthy Life

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Natural flavin cofactors FMN -Flavin mononucleotide or Riboflavin 5'-phosphate - and FAD -flavin adenine dinucleotide- differ from Riboflavin (otherwise known as vitamin B2) in their ribityl side chains, which contain a phosphate residue or an ADP moiety in ester linkage with its terminal hydroxyl group, respectively [1]. FMN and FAD, the biologically active forms of Riboflavin, play a lead role in a diverse array of biological processes, which is a reflection of their structural and chemical versatility [2]. FMN and FAD are mainly located in mitochondria, where they act as redox cofactors of a number of dehydrogenases and oxidases that play a crucial function in both bioenergetics and cellular regulation, in protein folding, apoptosis, epigenetics and mitochondrial terminal metabolism. The homeostasis of Riboflavin and flavin prosthetic groups may be altered by some factors, such as defective FMN and/or FAD synthesis, increased FMN and/or FAD catabolism, by different susceptibility of holo- and apo-flavoproteins to proteolytic digestion and altered mitochondrial metabolism and transport [3,4]. A problem in Riboflavin metabolism or a low intake of this vitamin will have consequences on the level of FAD and FMN in the cell, resulting in disorders associated with Riboflavin deficiency [5]. Based on erythrocyte glutathione reductase activation coefficient test, 54% of British non-elderly adult population was at least having borderline Vitamin B2/Riboflavin deficiency [6]. Indeed, Riboflavin deficiency across European countries ranges between 7 and 20% [7] while 10 - 15% of global population have an inherited condition of limited Riboflavin absorption and utilization [6]. Riboflavin is a potential neuroprotective agent given that has demonstrated its ability to tackle significant pathogenesis-related mechanisms in neurological disorders, exemplified by the ones attributed to the pathogenesis of Parkinson's disease, migraine headache and multiple sclerosis [8]. The antioxidant properties of Riboflavin and its effect on oxidative stress reduction have been reported. Recent studies confirm the antioxidant nature of Riboflavin and indicate that this vitamin can protect the body against oxidative stress, especially lipid peroxidation and reperfusion oxidative injury. The mechanisms by which Riboflavin protects the body against oxidative stress may be attributed to the glutathione redox cycle and also to other possible mechanisms such as the conversion of reduced Riboflavin to the oxidised form [9]. Riboflavin is found in a wide variety of animal and plant foods. Meat and dairy products are the major contributors of Riboflavin dietary intake [10]. The Riboflavin deficiency has profound effect on iron absorption, metabolism of tryptophan, mitochondrial dysfunction, gastrointestinal tract, brain dysfunction, and metabolism of other vitamins as well as is associated with skin disorders. Toxicological and photosensitizing properties of Riboflavin make it suitable for biological use, such as virus inactivation, excellent photosensitizer, and promising adjuvant in chemo radiotherapy in cancer treatment. A number of recent studies have indicated and highlighted the cellular processes and biological effects associated with Riboflavin supplementation in metabolic diseases. Overall, a deeper understanding of these emerging roles of Riboflavin intake is essential to design better therapies for future [11].

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