Commentary

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Fatty Liver - Insulin, Methionine, Choline, Betaine, Homocysteine, Mg, Se, Vitamin E and Pioglitazone

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About Homocysteine, Methionine and GSH

Fatty liver [1] is associated with cellular dysfunction, why GSH (glutathione) [2], its association with homocysteine (HCy) and methionine (Met) need closer assessment. Extreme form of dysfunction is necrosis [1]. HCy-Met cycle consist of two poles {look [3], Figure 1, text discussable}: Met (donor of methyl group) and HCy (recipient of methyl group) and circulation between them. The methylation of HCy is not the normal route of HCy [4]. Generally, HCy is used for Cysteine (Cys) synthesis [4]. (For methylation of HCy can be used betaine, its precursor choline or tetrahydrofolate with its cofactor, vitamin B12 [3,4]). Availability HCy for Cys synthesis can be reduced by excess of methyl groups, which turns Met-HCy balance towards Met or by lack of methyl groups via increasing catabolism of sulfur amino acids [5] (Met and HCy). The production of Cys from HCy is impeded by vitamin B6 deficiency [3], or by genetic factors [3]. This can increase HCy level because of decreased "suction", not by formation of HCy from Cys (Cys, an essential amino acid, cannot be used for HCy or Met production in humans).

Cys and serine are precursors of GSH (γ-glutamylcysteinylglycine) [6]. The synthesis of GSH intermediate (γ-glutamylcysteine) requires Mg++ (and K+) [7] and their deficiency decreases GSH level [8] and could explain a part of the role of Mg with Liver diseases [1] There is some evidence that HCy as such is not pathogenic: Mice fed methionine-rich diets had significant atheromatous pathology in the aortic arch even with normal plasma homocysteine levels, whereas mice fed B vitamin-deficient diets developed severe hyperhomocysteinemia without any increase in vascular pathology [9]. Atheroclerosis in [10] could be explained by the causes of HCy elevation (e.g. deficiencies in Mg and B vitamins), possibly even via Met effects on GSH production [5]. Explaining [11] could possibly be based on sufficient Met supply in the basal diet, why choline (methyl donor) reduction did not make any harms, but increased GSH production [5]. Effects of lecithin [1] could be possibly be explained by its obvious "unpurities", phytosterols [12]. Evaluation of Mg deficiency needs possibly its intracellular determination [13], but Its correction needs less refined food [14].

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