

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

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Editorial

This article is assessing “fatty liver”, its etiology and its responses to several treatments and makes a question to specialists: Is there a time to prove or to disprove (some of) the treatments of fatty liver beneficial for steatohepatitis? PubMed Search: “magnesium liver” gave 6 492 and “selenium liver” 5 584 results (in April 2024). Magnesium has been shown to decrease serum ASAT, ALAT and gamma-GT values and to increase muscle power of alcoholics, although they did not reduce alcohol consumption [1]. Often it is written that alcoholics suffer from Mg deficiency, as is observed in an intravenous (30 mmol) Mg loading test: 2 SD variation was by healthy controls 3 (-2 - 8) % and by alcoholics 33 (29 - 36) % [2]. Panresectomized dogs, although treated adequately with insulin, develop severe fatty livers. This disease could be prevented or cured by supplementation diets with lecithin or choline [3].

Choline is a constituent of lecithin and the capacity of liver to generate lecithin is dependent on a supply of choline or a supply of methyl groups from S-adenosylmethionine (sulfur compound) [3]. Fatty livers could be produced in rats by administration a diet poor in choline and low in protein. This fatty liver could be prevented and cured by choline [3]. Interesting that betaine, a metabolite of choline can reduce homocysteine content. Betaine can donate a methyl group to homocysteine - as tetrahydrofolate - to form methionine [4]. PubMed search: “betaine homocysteine” gave 1 100 results. In a human experiment of 96 days Vitamin E (800 IU, natural form, once a day) and pioglitazone (30 mg, once a day) improved/relieved similarly (and

significantly better than placebo) histological features and serum inflammation markers in patients with nonalcoholic steatohepatitis [5]. The main form of selenium in plant and animal origin feed materials is organic protein bound selenomethionine (in which selenium has replaced sulfur), together with small amounts of selenocysteine and selenite (Underwood and Suttle, 1999) [6].

The benefit of methionine [3] can (partially) be explained by its content of selenium, because pure selenium salts can prevent liver necrosis [7]. The disadvantage of low protein diets could have partially been dependent on low selenium content of (soil and food) [8]. The success of (low protein) Hindhede diet: “bread, potatoes and cabbage in sufficient quantity, and then some milk” [9] seems not to have been well understood, e.g. [10] (missing the importance of cabbage, which contains calcium ca 19-fold to potato, per DW [11] and is rich in sulphur [11], which is a structural component of methionine [12]). Nutritional survey (NHANES III, with over 13 000 participants) showed that every 100 mg increase in intake of magnesium was associated with a 49% reduction in the risk for mortality due to liver diseases [13].

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