

Abstract

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Change in the fatty acid pattern of erythrocyte membrane phospholipids after oral supplementation of specific fatty acids in patients with gastrointestinal diseases.

Siener R, Alteheld B, Terjung B, Junghans B, Bitterlich N, Stehle P, Metzner C.

Department of Urology, Medical Nutrition Science, University of Bonn, Bonn, Germany.

BACKGROUND/OBJECTIVES: The fatty acid pattern of membrane phospholipids is suggested to affect membrane fluidity and epithelial barrier function as a result of membrane fatty acid unsaturation. The incorporation of n-3 polyunsaturated fatty acids (PUFAs) into membrane phospholipids may diminish inflammatory potential in patients with gastrointestinal diseases. The aim of this study was to improve the fatty acid profile of erythrocyte membrane phospholipids after oral supplementation of specific fatty acids in patients with maldigestion and/or malabsorption.

SUBJECTS/METHODS: We conducted a randomized, double-blind, controlled trial. A total of 48 patients with gastrointestinal diseases received either fat-soluble vitamins A,D,E,K (ADEK) or ADEK plus fatty acids alpha-linolenic acid (ALA), docosahexaenoic acid (DHA) and medium-chain triglycerides (FA-ADEK) for 12 weeks. The fatty acid profile of erythrocyte membrane phospholipids, dietary intake, plasma antioxidant vitamins and serum gamma-glutamyl transferase (GGT) were evaluated at baseline, 8 and 12 weeks after supplementation.

RESULTS: Supplementation with FA-ADEK increased ALA, DHA and eicosapentaenoic acid (EPA) concentrations of erythrocyte membrane phospholipids by 0.040, 1.419 and 0.159%, respectively, compared with ADEK supplementation (-0.007, 0.151 and 0.002%, respectively) after 12 weeks (all $P \leq 0.001$). Serum GGT activity decreased in patients receiving FA-ADEK compared with those receiving ADEK with a significant difference after 8 weeks.

CONCLUSIONS: The significant change in erythrocyte membrane fatty acid pattern demonstrates the incorporation of orally administered n-3 PUFA in patients with maldigestion and malabsorption. The increase in ALA and DHA, as well as the conversion of ALA to EPA is attributed to the supplementation of sufficient amounts of ALA and DHA, respectively. Serum GGT activity decreased in response to decreased oxidative stress.

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