PUFA rich Plant Oils and Oxidative Stress in Type 2 Diabetics

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Increasing evidence suggests that oxidative stress plays a major role in the pathogenesis of type 2 diabetes. A contemporary deficiency in the antioxidative endogenous defense system can cause oxidative stress, which contributes to the raising number of diabetic complications. The aim of this study was to investigate the influence of plant oils on different parameters of the antioxidative defense system and oxidation products of type 2 diabetics.

92 subjects (34 IDDM and 58 NIDDM) were randomised into a single-oil-group (SO) or the mixed-oil-group (MO) and instructed to consume 3 teaspoons of oil/day (9 g/day) for 10 weeks. Blood samples were taken before intervention (T0), after 4 (T1) and 10 (T2) weeks of intervention, and 8 weeks after finishing (T3). Plasma was analyzed for tocopherols, β-carotene, vitamin C, fatty acids, oxidized low-density lipoprotein (ox. LDL), advanced oxidation protein products (AOPP) and total antioxidative capacity (TAC). Activities of superoxide dismutase (SOD), glutathione peroxidase (GSH-Px), and catalase (CAT) was measured in erythrocytes.

Compliance was confirmed by a significant increase of γ-tocopherol, α- and γ- linolenic acid in both intervention groups. At baseline, SOD activities were significantly higher in NIDDM (2013±162 I.U./g Hb) than in IDDM subjects (1807±221 I.U./g Hb). Impairment of blood antioxidant potential was reflected by a significant increase of SOD activity in all groups from T0 to T1 and significant decrease in CAT activity in all groups from T0 to T1 and T2. GSH-Px remained unchanged during the oil intervention. No differences were observed for β-carotene and vitamin C. TAC and oxidation products as AOPP and ox. LDL showed no significant differences compared to baseline values within the intervention period.

These results indicate that an additional amount of 9 g PUFA rich plant oil influences the endogenous antioxidative defence system of type 2 diabetics and do not increase oxidative stress.