Nutrition 49 (2018) 74–80

Contents lists available at ScienceDirect

Nutrition

journal homepage: www.nutritionjrnl.com

Applied nutritional investigation

Effects of a hypoenergetic diet rich in α-linolenic acid on fatty acid composition of serum phospholipids in overweight and obese patients with metabolic syndrome

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ARTICLE INFO

Article history:
Received 7 April 2017
Received in revised form
5 September 2017
Accepted 1 November 2017

Keywords:
Alpha-linolenic acid
Energy restriction
Fatty acids
Conversion
Metabolic syndrome

ABSTRACT

Objectives: Plant-derived α-linolenic acid (ALA) may exert cardioprotective effects. Dietary ALA can undergo desaturation and elongation to form long-chain ω-3 polyunsaturated fatty acids, but the extent to which this occurs in humans is unclear. The aim of the study was to examine the effects of an energy-restricted diet enriched with ALA on fatty acid composition of serum phospholipids in patients with metabolic syndrome.
Methods: The present analysis compared the effects of a hypoenergetic diet high in ALA (3.4 g/d) with a control diet low in ALA (0.9 g/d) on fatty acid composition of serum phospholipids in 81 overweight or obese patients with features of metabolic syndrome.
Results: After a 26-wk intervention, concentration of ALA in serum phospholipids remained constant in both diet groups. The control group had a significant decrease in serum phospholipid eicosapentaenoic acid concentration, although no significant intergroup difference was observed. Serum phospholipid docosahexaenoic acid concentration significantly decreased to a similar extent with both interventions. Additionally, both interventions significantly decreased serum phospholipid concentrations of palmitic acid, stearic acid, total saturated fatty acids, linoleic acid, total ω-6 and ω-3 polyunsaturated fatty acids, with no effect of diet group on these changes. Compared with the ALA diet, the control diet led to a significant increase in serum phospholipid oleic acid concentration.
Conclusion: Daily intake of 3.4 g of ALA during a 26-wk energy-restricted diet did not lead to an enrichment of serum phospholipids with ALA and did not increase eicosapentaenoic acid due to conversion. Additionally, dietary ALA was unable to compensate for a decrease in serum phospholipid docosahexaenoic acid.

Introduction

Metabolic syndrome (MetS) is among the most common cause of increased cardiovascular disease (CVD) risk in otherwise healthy populations. It consists of a constellation of CVD risk factors that are heterogeneously expressed with insulin resistance as a key underlying defect, although the pathogenesis remains unclear [1,2]. Although first-line treatment for this condition should include energy restriction, primarily to promote the loss of excess abdominal fat, there is still debate over the most appropriate choice of dietary fatty acids and carbohydrate to facilitate weight loss and correct metabolic dysfunction. Recently, we presented evidence suggesting that an energy-restricted diet enriched with α-linolenic acid (ALA) from rapeseed oil has greater efficacy in lowering risk factors in MetS than an energy-restricted diet enriched with olive oil [3,4].

This study was supported by the Union for Promoting Oil and Protein Plants e.V. (UFOP) and the International Foundation for the Promotion of Nutrition Research and Nutrition Education (ISFE).
The authors have no conflicts of interest to declare.
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https://doi.org/10.1016/j.nut.2017.11.002
0899-9007/

Epidemiologic data and evidence from human randomized controlled trials demonstrate that the marine ω-3 fatty acids eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) have cardioprotective effects [5–9]. Specifically, EPA and DHA improved vascular endothelial function and reduced serum triacylglycerols (TGs), arterial blood pressure, and inflammation [10–13]. Additionally, epidemiologic studies show an inverse association between tissue or blood levels of EPA and DHA and cardiovascular events [14,15].

It is unknown whether ALA, which is abundant in certain plant foods, including various vegetable oils, such as linseed (flaxseed) oil and rapeseed (canola) oil, also has a significant preventive potential. Possible cardioprotective and antiinflammatory benefits of ALA have been reviewed [16–18]. The potential effects were examined for supplements or flaxseed oil as ALA source. For dietary practice, it is of special interest whether protective effects of ALA can be achieved without supplements but with amounts found in a long-term natural food diet. Dietary ALA can be desaturated and elongated to form long-chain ω-3 polyunsaturated fatty acids (PUFAs) in humans, but the extent to which this occurs and whether ALA has physiologic effects independent of its role as a precursor for long-chain ω-3 PUFAs are unclear [19–21].

Conversion of ALA has been studied using different ALA dosages and under isoenergetic and thus body weight–stable conditions. We previously demonstrated that enrichment of the habitual diet with moderate doses of ALA (3.4 and 6 g/d) for 3 to 6 wk led to a significant increase of ALA in isolated low-density lipoprotein (LDL) particles and erythrocytes and increased EPA concentrations due to increased conversion from ALA [13,22,23]. To the best of our knowledge, no previous human trials have investigated fatty acid profiles and ALA conversion during energy restriction and body weight loss. Therefore, in the present study, we examined the effects of an energy-restricted diet enriched with ALA on fatty acid composition of serum phospholipids in patients with MetS. We previously described the effects of such diets on features of MetS [3] and biomarkers of inflammation and endothelial function [4]. A range of subject baseline parameters, diet intake characteristics, and the results of body weight changes are represented here, to better relate them to the newly presented data.

Patients and methods

Patients

Details of the study design; dietary interventions; and patient recruitment, enrollment, and randomization have been described previously [3]. In brief, 150 volunteers 18 to 70 y of age and classified as overweight attended a screening that included physical assessments (height, body weight, blood pressure, waist circumference [WC], and hip circumference), clinical assessments (e.g., serum lipids, lipoproteins, and glucose), medical history, and a dietary questionnaire.

Participants with the following traits of MetS were included: central obesity (WC ≥94 cm for men and ≥80 cm for women) plus two of the following criteria:

1. fasting serum concentrations of TGs ≥1.7 mmol/L;
2. reduced serum high-density lipoprotein cholesterol (HDL-C; <1.03 mmol/L in men, <1.29 mmol/L in women);
3. elevated blood pressure (systolic ≥130 mm Hg; diastolic ≥85 mm Hg);
4. fasting plasma glucose ≥6.5 mmol/L [2,3].

Exclusion criteria included smoking; insulin-dependent diabetes mellitus; liver, gastrointestinal, or inflammatory diseases; a history of cardiovascular events; use of antiobesity medications or antiinflammatory drugs; cancer; pregnancy or lactation; alcohol abuse; or need for a medically supervised diet.

Ninety-five patients (30 men, 65 women) were included in the study. Thirteen patients dropped out due to different reasons, and one participant was excluded retrospectively due to multiple drug changes throughout the study. The final analysis included 81 patients (26 men, 55 women) [3]. Baseline characteristics are presented in Table 1.

Table 1
Baseline characteristics of participants [4]*

	ALA (n = 40)	Control (n = 41)
Age (y)	52.3 ± 10.6	50.3 ± 9.8
Body weight (kg)	97.3 ± 19.7	99.4 ± 16.2
BMI (kg/m^2)	33.4 ± 4.8	35.2 ± 5.1
Waist circumference (cm)		
Men	98.1 ± 9.6	111.1 ± 10
Women	114.1 ± 10.5	104.8 ± 12.1
Fat mass (%)		
Men	35.9 ± 4.1	34.3 ± 5.2
Women	44.8 ± 4.6	47.1 ± 4.3
Systolic blood pressure (mm Hg)	142.4 ± 18.6	140.1 ± 12.4
Diastolic blood pressure (mm Hg)	91.8 ± 11.8	90.2 ± 7.7
Fasting plasma glucose (mmol/L)	5.76 ± 1.32	5.80 ± 1.00
Fasting plasma insulin (pmol/L)	90.7 ± 41.1	98.9 ± 73.7
Fasting total cholesterol (mmol/L)	5.43 ± 0.88	5.49 ± 1.09
Fasting LDL cholesterol (mmol/L)	3.42 ± 0.82	3.49 ± 0.92
Fasting HDL cholesterol (mmol/L)		
Men	1.12 ± 0.18	1.32 ± 0.41
Women	1.50 ± 0.26	1.47 ± 0.30
Fasting TG (mmol/L)	1.94 ± 1.13	1.64 ± 1.02
Apolipoprotein B (g/L)	0.92 ± 0.21	0.93 ± 0.23
Apolipoprotein A1 (g/L)	1.57 ± 0.28	1.57 ± 0.26

BMI, body mass index; HDL, high-density lipoprotein; LDL, low-density lipoprotein; TG, triacylglycerol.
* Values are mean ± standard deviation. Variables were not significantly different between the groups in independent sample *t* tests ($P > 0.05$ for all).

The study was conducted according to the guidelines laid down in the 1964 Declaration of Helsinki, and its later amendments and all procedures involving human participants were approved by the ethical committee of the Ruhr-University Bochum, Germany located at the Herz- und Diabeteszentrum NRW. Written informed consent was obtained from all participants. The study was registered at www.germanctr.de/ and http://apps.who.int/trialsearch/ as DRKS00006232.

Study design and dietary intervention

This was a randomized controlled dietary intervention and was conducted under outpatient conditions over a 26-wk period [3]. In a parallel design, patients were randomized to an energy-restricted diet (see below) enriched with ALA (ALA diet; ~ 3.4 g/d ALA) or an energy-restricted control diet (~0.9 g/d ALA; Table 2). The

Table 2
Composition of the habitual and study diets [4]*

	Habitual diet (n = 81)	ALA (n = 40)	Control (n = 41)
Energy intake (MJ/d)	9.1 ± 2.8	6.8 ± 1.5	6.7 ± 1.2
Protein (% of energy)	17.5 ± 3.3	19.5 ± 3.8	19.0 ± 2.6
Protein (g/d)	91.6 ± 29.8	78.6 ± 30.5	75.4 ± 19.9
Carbohydrates (% of energy)	43.9 ± 6.9	41.3 ± 4.3	42.4 ± 3.6
Fat (% of energy)	37.3 ± 6.2	37.9 ± 4.5	37.8 ± 3.5
SFAs (% of energy)	15.4 ± 3.5	9.8 ± 2.2	10.7 ± 1.5
SFAs (g/d)	37.8 ± 18.1	17.8 ± 5.6	19.3 ± 4.8
MUFAs (% of energy)	13.6 ± 2.7	18.1 ± 2.6	19.3 ± 2.4
MUFAs (g/d)	33.4 ± 14.7	32.2 ± 5	34.1 ± 6
PUFAs (% of energy)	5.2 ± 1.6	7.8 ± 1.5	5.5 ± 0.7
PUFAs (g/d)	12.3 ± 4.5	13.7 ± 2.7	9.6 ± 1.6
LA (g/d)	10.1 ± 3.6	10.2 ± 2.2	8.6 ± 1.4
AA (g/d)	0.16 ± 0.12	0.07 ± 0.05	0.07 ± 0.06
ALA (g/d)	1.61 ± 0.77	3.35 ± 0.65	0.85 ± 0.16
EPA (g/d)	0.08 ± 0.14	0.02 ± 0.05	0.01 ± 0.03
DHA (g/d)	0.18 ± 0.25	0.03 ± 0.06	0.03 ± 0.05
Dietary fiber (g/MJ)	2.6 ± 0.9	4.1 ± 0.9	4.1 ± 0.9
Energy density (kJ/g)	5.9 ± 1.2	4.5 ± 0.7	4.6 ± 0.8

AA, arachidonic acid; ALA, α-linolenic acid; DHA, docosahexaenoic acid; EPA, eicosapentaenoic acid; LA, linoleic acid; MUFAs, monounsaturated fatty acids; PUFAs, polyunsaturated fatty acids; SFAs, saturated fatty acids.
Total fat contains ~95% fatty acids; the other ~5% is made up of glycerol and other lipids. Energy density was calculated for solid foods and energy-containing beverages.
* Values are mean ± standard deviation. Data were calculated from 3-d food diaries; composition data of the study diets were calculated from the protocols of weeks 12 and 26.