https://doi.org/10.1017/S0007114508020321 Published online by Cambridge University Press

British Journal of Nutrition (2009), 101, 440–445
doi:10.1017/S0007114508020321
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Intervention with flaxseed and borage oil supplements modulates skin condition in women

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(Received 9 October 2007 – Revised 16 May 2008 – Accepted 21 May 2008 – First published online 2 September 2008)

Ingestion of selected nutrients modulates dermal properties. In the present study, two groups of women ingested flaxseed or borage oil for 12 weeks. The control group received a placebo containing medium-chain fatty acids. Dose was 2·2 g total fatty acids/d with α-linolenic acid and linoleic acid as major constituents in the flaxseed oil group; in the borage oil group linoleic and γ-linolenic acid were predominant. In the flaxseed oil group, the contribution of α-linolenic acid to total fatty acids in plasma was significantly increased on weeks 6 and 12, whereas there was an increase in γ-linolenic acid in the borage oil group ($P<0·05$). Skin irritation was performed by nicotinate treatment, and changes in skin reddening and blood flow were monitored. Compared to week 0, skin reddening was diminished in both groups; blood flow was also lowered. Skin hydration was significantly increased after 12 weeks of treatment compared to week 0, with flaxseed or borage oil ($P<0·05$). Transepidermal water loss was decreased in both oil groups by about 10 % after 6 weeks of supplementation. A further decrease was determined after 12 weeks in the flaxseed oil group. Surface evaluation of living skin revealed that roughness and scaling of the skin were significantly decreased with flaxseed and borage oil comparing week 0 and week 12 ($P<0·05$). Except for hydration, none of the parameters was affected in the placebo group. The present data provide evidence that skin properties can be modulated by an intervention with dietary lipids.

Lipids: Skin: Human studies: Hydration: Blood flow

Basic skin functions like that of a shielding barrier, homeostasis of water, temperature regulation and photoprotection respond to supplementary or dietary intervention with selected nutrients[1,2]. Modulated functions are accompanied by changes in cutaneous structure and texture which affect the appearance of the skin. Studies in man have shown that an increased intake of carotenoids such as β-carotene or lycopene, carotenoid-rich food or flavanol-rich cocoa strengthens the basal protection against UV-induced erythema, increases cutaneous blood flow, and modulates skin structure and hydration[3,4].

Some PUFA are essential for the human organism and serve several biological functions. Supplementation with certain fatty acids can influence the fatty acid pattern of the skin and affect its sensitivity towards photooxidation[5] and may contribute to prevention of non-melanoma cancers[6].

PUFA, like linoleic acid (LA) or α-linolenic acid (ALA) and γ-linolenic acid (GLA), play a role in cellular signalling interfering with eicosanoid pathways or influencing the regulation of gene expression[2,7]. Both *n*-3 as well as *n*-6 long-chain PUFA are important components of cellular membranes, contributing to their fluidity, rigidity, permeability and function[8,9]. Epidermal lipids are known to play an important role in mediating normal desquamation and deficiency of essential fatty acids has been reported to be involved in cutaneous scaling disorders such as senile xerosis, psoriasis and atopic dermatitis[10].

The organism has a limited activity to generate higher PUFA from C18 fatty acids via desaturation–elongation reactions. Thus, the *n*-6 PUFA LA can be metabolized to GLA. However, it has been reported that cutaneous tissue lacks such activity[11].

Based on structural effects and signalling processes, PUFA affect macroscopic properties of the skin essential for cutaneous health. Supplementation with LA and GLA from borage oil over a 2-month period leads to decreased transepidermal water loss (TEWL) and itching. Dry skin appearance was diminished significantly, but no alteration of skin hydration was measured[12]. LA deficiency results in deformation or absence of the lamellar bodies, giving way to water passing the epidermis[13]. Consumption of flaxseed oil rich in ALA or DHA/EPA-rich fish oil is associated with lower levels of proinflammatory eicosanoids[14]. In the present study we investigated the effects of 12-week oral supplementation with flaxseed or borage oil on human skin properties.

Abbreviations: ALA, α-linolenic acid; DGLA, di-homo-γ-linolenic acid; GLA, γ-linolenic acid; LA, linoleic acid; TEWL, transepidermal water loss.
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British Journal of Nutrition

https://doi.org/10.1017/S0007114508020321 Published online by Cambridge University Press

Materials and methods

Subjects and study design

Forty-five female healthy non-smoking women, between 18 and 65 years, with sensitive and dry skin were recruited by the Institute of Experimental Dermatology, and selected according to their skin type: dry skin; Corneometer value <40 au[15]. Exclusion criteria were: pregnancy and breast-feeding, history of fat malabsorption, liver diseases, diseases regarding lipid metabolism or any photosensitizing disorder. BMI of the participants was between 18 and 25 kg/m^2, they did not take lipid or vitamin supplements or any other medication.

The study was performed as a monocentric, randomized, double-blind placebo-controlled application test, in three parallel treatment groups, consisting of fifteen persons each. Age distribution was comparable between the three groups. Four capsules with different oils were taken every day, two at breakfast and two at dinner. Daily doses amounted to 2·2 g flaxseed or borage oil; in both groups, intake of tocopherol was 10 mg/d. Daily intake of the placebo was also 2·2 g. The fatty acid pattern of flaxseed, borage oil and placebo are shown in Table 1.

Duration of the study was 12 weeks, measurements determining skin parameters and blood sampling was on day 0, week 6 and week 12. All test subjects received detailed information listing every single parameter relevant to the study. Every test subject had to submit a written declaration of consent for their participation in the study.

Fatty acid analysis

Blood samples were collected into 10 ml S-monovette tubes (Sarstedt, Nuemrecht, Germany) containing sodium EDTA as anticoagulant. After clotting, plasma was prepared by centrifugation (10 min; 4°C) at 3800 ***g*** and stored at −80°C until analysis. Lipids were extracted according to Folch & Lees[16] with slight modifications. Plasma sample (0·5 ml) was placed in a 10 ml glass tube and mixed with 5 ml chloroform–methanol (2:1), containing 200 mg/l butylated hydroxytoluene (BHT) as antioxidant. The sample was mixed by shaking at 37°C for 45 min. After addition of 1 ml sodium chloride solution (0·9 %) and centrifugation at 660 ***g*** for 10 min at 4°C, the lipophilic layer was collected and washed twice with 500 µl methanol–sodium chloride (0·9 %)–chloroform (48:47:3). The solution was dried under a stream of nitrogen. For base-catalysed transesterification to yield fatty acid methyl esters the fat extract was incubated 1 h at 60°C with 3 ml water-free methanol containing sodium methoxide (3 % m/v). Chlorogenic acid (1 ml) was added, and fatty acid methyl esters were extracted twice with 3 ml hexane, concentrated under nitrogen to a final volume of 350 µl and transferred to an injection vial sealed with a metal cap. Fatty acid methyl esters were separated by GC on a FFAP column (Crossbond Carbowax PEG for acid compounds; 30 m, 250 µm) and detected by flame ionization (Clarius 500 PE AutoSystem with built-in autosampler; Perkin Elmer, Shelton, USA). The injector temperature was 240°C, the oven temperature programme started at 150°C and increased continuously to 190°C at a rate of 20°C/min and from 190 to 235°C at a rate of 1°C/min with a final hold of 35 min. Nitrogen was used as carrier gas at a pressure of 70 kPa. Detector temperature was 260°C. Peak identification was with authentic fatty acid methyl ester standards (Supelco, Deisenhofen; Fluka; Sigma, St. Louis, MO, USA). With this procedure all esterified fatty acids are detected and referred to as 'total fatty acids'. The relative amount of each fatty acid was calculated from the ratio of the area under the signal and total area of all identified fatty acid methyl esters (100 % method).

Table 1. Fatty acid composition of the supplements (% of total fatty acids)

Fatty acid	Flaxseed oil	Borage oil	Placebo
8 : 0	0·00	0·00	36·9
10 : 0	0·00	0·00	21·8
14 : 0	4·73	1·87	0·24
18 : 0	4·12	2·82	0·00
18 : 1*n*-9	19·6	15·0	0·60
18 : 2*n*-6	16·0	38·7	0·00
18 : 3*n*-6	0·00	21·6	0·18
18 : 3*n*-3	52·8	0·38	0·00
20 : 0	0·16	0·21	0·00
20 : 1	0·20	3·95	0·00
20 : 2	0·00	0·23	0·00
22 : 1	0·00	2·23	0·00
24 : 0	0·00	0·00	0·00
24 : 1	0·00	1·30	0·00
Non-identified fatty acids	2·39	11·7	40·3

Skin sensitivity

Nicotinate irritation was used to test the sensitivity of the skin according to Primavera & Berardesca[17]. Measurements were performed on the inner forearm of the volunteers. No treatment with any ointment on the tested areas was allowed during the whole study. Nicotinate (0·25 %; 5 µl/cm^2) was applied inducing an inflammation of the skin; reddening was measured by chromametry as *a* value (Minolta CR 300, Ahrensburg, Germany) before and after treatment. Erythema intensity is given as Δa value; *a* value after treatment minus *a* value before treatment. Additionally, capillary blood flow was determined by Laser-Doppler-Flowmetry (O2C- System; Lea Instruments, Giessen, Germany) in the irritated area.

Skin hydration and transepidermal water loss

Skin hydration (arbitrary units) was determined by corneometry (Corneometer CM 825; Courage & Khazaka Electronics, Cologne, Germany); TEWL (g/h per m^2) was measured using a TEWA-Meter TM 300 (Courage & Khazaka Electronics)[15,18].

Evaluation of skin surface

Skin surface profiles were evaluated with the surface evaluation of living skin method (Visioscan; Courage & Khazaka Electronics) in a 15 × 17 mm area. Four different parameters are applied to characterize skin surface: roughness, scaling, smoothness, wrinkles[19].

Statistics

For all parameters and all time-points (week 0, week 6, week 12) descriptive statistics (means and standard deviations) were

calculated. For all parameters pre–post differences were calculated and analysed descriptively.

Separately for each of the two pre–post differences 'week 6 − week 0' and 'week 12 − week 0' an ANOVA with the parameter treatment as the independent variable was performed. Tests for the treatment effect and the contrasts were performed. The least square means for the pre–post differences and the respective 95 % CI were calculated. These CI were used to assess whether the pre–post difference was significantly different from zero.

Results

Fatty acid analysis

Fatty acid patterns of the supplements are shown in Table 1. In flaxseed oil, ALA is predominant; also considerable amounts of LA are present. The latter is the major fatty acid in borage oil followed by GLA. Thus, in flaxseed oil *n*-3 fatty acids contribute more than 50 % to total fatty acid content, whereas more than 50 % of fatty acids in borage oil are of the *n*-6 type. Additionally, both supplements contained similar amounts of tocopherol. Medium-chain fatty acids were present in the placebo; caprylic and capric acid were dominating. Volunteers were divided into three groups, flaxseed, borage and placebo, ingesting four capsules of the supplement per day over a period of 12 weeks. Daily doses amounted to 2·2 g flaxseed or borage oil; in both groups, intake of tocopherol was 10 mg/d. Daily dose of the placebo was 2·2 g.

Blood samples were collected on day 0, week 6 and week 12 and fatty acid composition of plasma lipids (total fatty acids) was analysed by GC. Results for the different groups are shown in Tables 2–4. In the flaxseed oil group, only the contribution of ALA to total fatty acids was significantly changed. From day 0 to week 6 it increased from 0·58 to 0·83 % and remained constant until week 12. Upon supplementation with borage oil, the relative amount of GLA was significantly increased on weeks 6 and 12 compared to day 0. A marginal increase was detectable for di-homo-γ-linolenic acid (DGLA) from 2·09 % in week 0 to 2·44 % in week 6 and 2·33 % in week 12. A significant decrease of DGLA was found in the placebo group comparing week 12 to week 0. In none of the groups did vitamin E plasma levels change significantly (data not shown).

Table 2. Fatty acid composition of plasma lipids (% total fatty acids) in the group treated with flaxseed oil (*n* 15)†

(Mean values and standard deviations)

	Time (weeks)					
	0		6		12	
Fatty acid	Mean	SD	Mean	SD	Mean	SD
18:2*n*-6	32·4	3·4	32·7	4·9	32·3	5·9
18:3*n*-6	0·59	0·27	0·53	0·19	0·55	0·19
20:3*n*-6	1·88	0·41	1·82	0·43	1·83	0·36
20:4*n*-6	7·23	1·15	6·90	1·13	6·91	1·06
22:4*n*-6	0·10	0·07	0·12	0·08	0·08	0·08
18:3*n*-3	0·58	0·21	0·83*	0·25	0·84*	0·24
20:5*n*-3	0·85	0·38	0·88	0·31	0·97	0·37
22:5*n*-3	0·44	0·12	0·42	0·08	0·42	0·16
22:6*n*-3	1·36	0·60	1·20	0·31	1·24	0·40

Mean values were significantly different from those of week 0: **P*< 0·05.
† For details of procedures and supplements, see Materials and methods section and Table 1.

Table 3. Fatty acid composition of plasma lipids (% total fatty acids) in the group treated with borage oil (*n* 15)†

(Mean values and standard deviations)

	Time (weeks)					
	0		6		12	
Fatty acid	Mean	SD	Mean	SD	Mean	SD
18:2*n*-6	31·3	4·7	28·6	9·0	31·1	5·6
18:3*n*-6	0·47	0·14	0·70*	0·21	0·64*	0·25
20:3*n*-6	2·09	0·40	2·44*	0·57	2·33*	0·59
20:4*n*-6	7·62	1·42	8·11	1·78	7·82	1·36
22:4*n*-6	0·17	0·08	0·14	0·10	0·12	0·10
18:3*n*-3	0·50	0·13	0·58*	0·11	0·52	0·12
20:5*n*-3	0·73	0·70	0·70	0·46	0·72	0·85
22:5*n*-3	0·37	0·12	0·35	0·19	0·35	0·13
22:6*n*-3	1·33	0·65	1·31	0·51	1·29	0·62

Mean values were significantly different from those of week 0: **P*< 0·05.
† For details of procedures and supplements, see Materials and methods section and Table 1.

Skin sensitivity

Exposure of skin to nicotinate leads to chemically induced inflammation. The resulting erythema was evaluated by chromametry where *a* values are a measure for skin reddening. The difference in *a* values (Δa values) before and after exposure to nicotinate is shown in Table 5. Decreasing Δa values are indicative for anti-inflammatory effects. Compared to baseline, Δa values were significantly lower on weeks 6 and 12 in the groups treated with flaxseed and borage oil; after 12 weeks the decrease was 45 and 35 %, respectively ($P < 0·05$). No changes were observed in the placebo group. However, it has to be noted that the basal value in the placebo group was lower than in the other groups.

Changes in the flaxseed oil group were signifcantly different from the placebo group (weeks 0–6, $P = 0·01$; weeks 0–12, $P = 0·006$). Also the changes in the borage oil

Table 4. Fatty acid composition of plasma lipids (% total fatty acids) in the group treated with placebo oil (*n* 15)†

(Mean values and standard deviations)

	Time (weeks)					
	0		6		12	
Fatty acid	Mean	SD	Mean	SD	Mean	SD
18:2*n*-6	32·3	4·4	31·6	5·3	31·4	4·7
18:3*n*-6	0·49	0·16	0·51	0·23	0·47	0·16
20:3*n*-6	2·03	0·30	1·92	0·32	1·81*	0·37
20:4*n*-6	7·49	1·65	7·16	1·59	7·71	2·01
22:4*n*-6	0·09	0·09	0·11	0·07	0·10	0·08
18:3*n*-3	0·64	0·20	0·62	0·17	0·54	0·12
20:5*n*-3	0·68	0·29	0·79	0·32	0·69	0·47
22:5*n*-3	0·38	0·11	0·39	0·10	0·36	0·12
22:6*n*-3	1·23	0·41	1·30	0·48	1·40	0·65

Mean values were significantly different from those of week 0: **P*< 0·05.
† For details of procedures and supplements, see Materials and methods section and Table 1.

British Journal of Nutrition

https://doi.org/10.1017/S0007114508020321 Published online by Cambridge University Press

https://doi.org/10.1017/S0007114508020321 Published online by Cambridge University Press

Table 5. Sensitivity of skin to nicotinate-induced irritation in women supplemented with flaxseed or borage oil: intensity of erythema and cutaneous blood flow (arbitrary units; *n* 15)†

(Mean values and standard deviations)

	Time (weeks)					
	0		6		12	
	Mean	SD	Mean	SD	Mean	SD
Δ*a* value						
Flaxseed oil	4·3	1·3	2·9*	0·9	2·4*	1·0
Borage oil	4·2	1·5	3·0*	1·6	2·7*	1·2
Placebo	3·4	1·0	3·4	1·3	3·3	1·2
Cutaneous blood flow						
Flaxseed oil	75·0	22·0	50·0*	23·0	14·0*	12·0
Borage oil	71·0	20·0	51·0*	30·0	47·0*	20·0
Placebo	70·0	22·0	63·0	26·0	64·0	29·0

Mean values were significantly different from those of week 0: $*P<0·05$.
† For details of procedures and supplements, see Materials and methods section and Table 1.

group significantly differed compared to the placebo group (weeks 0–6, $P=0·03$; weeks 0–12, $P=0·03$). There was no statistically significant difference between both groups treated with plant oils.

Inflammation of the skin is characterized by an increased cutaneous blood flow which is at least in part responsible for reddening. In parallel to decreasing Δa values, cutaneous blood flow, compared to baseline, was lower in the groups receiving flaxseed and borage oil after 6 and 12 weeks of supplementation (Table 5). No change was observed in the placebo group. After 12 weeks the effect was more pronounced in the group that ingested flaxseed oil (−82 %) compared to the borage oil group (−34 %) ($P<0·05$). The changes in the flaxseed oil group were significantly different from the borage oil and the placebo group (weeks 0–12, $P=0·00\,001$ and $P=0·002$, respectively).

Skin hydration and transepidermal water loss; skin structure

In women supplemented with flaxseed or borage oil, skin hydration was significantly increased after 12 weeks of treatment compared to day 0 (Table 6). In the flaxseed oil group the effect was already seen after 6 weeks. A significant increase in skin hydration was also observed in the placebo group but only after 12 weeks. The increases observed for the plant oils were not significantly different from the placebo group.

Table 6. Effects on skin hydration and transepidermal water loss (TEWL) following supplementation with flaxseed or borage oil (*n* 15)†

(Mean values and standard deviations)

	Time (weeks)					
	0		6		12	
	Mean	SD	Mean	SD	Mean	SD
Hydration (arbitrary units)						
Flaxseed oil	32·0	8·0	35·0*	10·0	38·0*	7·0
Borage oil	30·0	7·0	32·0	6·0	35·0*	7·0
Placebo	29·0	5·0	30·0	5·0	32·0*	5·0
TEWL (g/h per m^2)						
Flaxseed oil	9·7	1·1	9·0*	1·1	7·2*	1·8
Borage oil	9·9	0·8	9·0*	1·1	8·8*	1·2
Placebo	9·7	1·2	9·7	1·5	9·6	1·6

Mean values were significantly different from those of week 0: $*P<0·05$.
† For details of procedures and supplements, see Materials and methods section and Table 1.

TEWL was decreased in both plant oil groups by about 10 % after 6 weeks. A further decrease was determined after 12 weeks in the flaxseed oil group, whereas there was only small difference in TEWL values between weeks 6 and 12 in the borage oil group. No statistically significant differences between day 0 and weeks 6 and 12 were measured in the placebo group. The change in the flaxseed oil group from weeks 0 to 12 was significantly different to the placebo group ($P=0·00\,006$) and to the borage oil group ($P=0·01$).

Skin profiles were analysed using the surface evaluation of living skin method which provides parameters associated with roughness, scaling, smoothness and wrinkling of the skin. Upon intervention for 12 weeks with flaxseed oil, roughness, scaling and smoothness were improved (Table 7). Supplementation with borage oil caused modifications in the same parameters, but only the decrease in scaling from week 0 to week 6 was statistically significant. No changes were determined in the placebo group except for a small but statistically significant decrease in the parameter smoothness. None of the treatments affected wrinkling.

Comparing groups, there was a significant difference between flaxseed oil and placebo for the parameter roughness (weeks 0–12, $P=0·006$) and smoothness (weeks 0–6, $P=0·002$; weeks 0–12, $P=0·02$).

Discussion

Flaxseed oil, also known as linseed oil, derived from the seeds of the flax plant (*Linum usitatissimum*), is one of richest

Table 7. Parameters related to skin structure determined by surface evaluation of the skin at weeks 0, 6 and 12 of the study (arbitrary units; *n* 15)†

(Mean values and standard deviations)

	Time (weeks)					
	0		6		12	
	Mean	SD	Mean	SD	Mean	SD
Roughness						
Flaxseed oil	1·27	0·40	1·07*	0·43	0·84*	0·40
Borage oil	1·43	0·61	1·32	0·56	1·22	0·42
Placebo	1·19	0·25	1·20	0·21	1·20	0·35
Scaling						
Flaxseed oil	0·69	0·33	0·49*	0·28	0·45*	0·29
Borage oil	0·59	0·33	0·44*	0·11	0·43	0·11
Placebo	0·59	0·37	0·57	0·33	0·52	0·24
Wrinkles						
Flaxseed oil	35·0	2·1	34·6	2·3	35·0	2·2
Borage oil	35·2	2·9	35·5	2·7	35·6	2·8
Placebo	35·2	3·6	35·8	2·6	35·8	2·7
Smoothness						
Flaxseed oil	37·2	6·1	39·1*	5·5	38·9*	5·7
Borage oil	38·2	7·7	38·1	6·4	39·2	6·7
Placebo	35·8	6·4	34·3*	6·2	35·0	5·8

Mean values were significantly different from those of week 0: $*P<0·05$.
† For details of procedures and supplements, see Materials and methods section and Table 1.

https://doi.org/10.1017/S0007114508020321 Published online by Cambridge University Press

British Journal of Nutrition

sources of *n*-3 fatty acid. With more than 50 % of total fatty acid, ALA is predominant but also the *n*-6 LA (16 %) and the monounsaturated oleic acid (20 %) are major constituents (Table 1). The flaxseed oil supplement used in the present study is typical with respect to its composition of fatty acids[20]. Borage oil is produced from the seeds of the borage plant (*Borago officinalis*) and is rich in the *n*-6 fatty acids GLA (22 %) and LA (39 %); oleic acid (15 %) is also present in quite high amounts (Table 1). Also this supplement is typical regarding its fatty acid pattern[12].

Ingestion of ALA-rich flaxseed oil or GLA-rich borage oil led to increases in the contribution of the respective fatty acid to total plasma lipids (Tables 2–4). Changes in other fatty acids were not observed, apart from increases in DGLA in the group supplemented with borage oil. DGLA is likely formed from GLA by an elongase-catalysed two-carbon chain elongation. In addition to elongases, desaturases are required for the synthesis of arachidonic acid and higher homologues of the *n*-6 family as well as for C20 PUFA of the *n*-3 family. Increases of C20 PUFA in plasma and different lipid fraction in the blood have been shown after application of high doses of ALA[21]. In the present study the total dose of ALA and GLA was quite low and thus changes in the levels of longer-chain fatty acids may have been too small for detection. It should be further noted that the pattern of plasma lipids does not necessarily reflect the lipid composition of tissues such as skin. Some major effects observed here are likely due to more significant changes in skin lipids. However, dermal punch biopsy would be required to analyse the fatty acid pattern at the target site.

The irritating effects of nicotinate were ameliorated by long-term intake of either flaxseed or borage oil (Table 5). Reddening of the skin after the chemical challenge was lower in both groups after 6 and 12 weeks of intervention. Similar effects have been described after intake of long-chain *n*-3 fatty acids[5]. Supplementation with dietary fish oil diminished UV-B erythemal sensitivity in man. Although the mechanism of protection is not clear, it has been speculated that interferences with inflammatory pathways are important. In this context it is interesting to note that in the present study both supplements were active, although cutaneous blood flow was more affected by flaxseed oil. Therefore, it might be speculated that the contribution of PUFA on membrane and cell structure is at least in part responsible for the prevention of exogenous damage.

The function of selected C20 PUFA as precursors of eicosanoids for the synthesis of leukotrienes and PG may also play a role. This is in concordance with the effects of borage oil. GLA may be elongated to DGLA, which is metabolized to PG of the series 1 and to 15-hydroxyeicosatrienoic acid. Products of these eicosanoids mediate anti-inflammatory and antiproliferative effects[11]. However, ALA cannot be metabolized to anti-inflammatory eicosanoids in tissues lacking desaturases[11]. Thus, other mechanisms are likely operative. Long-chain *n*-3 fatty acids decrease the generation of inflammatory cytokines, the expression of adhesion molecules and probably the generation of reactive oxygen species[22]. The influence of ALA in these pathways is unclear. A study with murine macrophages has shown that ALA down-regulates inflammatory inducible nitric oxide synthase, cyclooxygenase-2 and TNF-α gene expression interfering with NF-κB and mitogen-activated protein kinase[23]. DGLA lowers TNF-α and IL-10 levels in isolated human peripheral blood mononuclear cells[24].

Stabilizing effects and improvements of cellular membranes may be responsible for changes of macroscopic skin properties related to water homeostasis. Again, with both supplements skin hydration was increased after 12 weeks of intervention, whereas TEWL was lower than control. Improvement of skin moisture and TEWL as well as firmness, indications for an improved barrier function, have been described after the intake of GLA-rich primrose and borage oil[25]. Skin hydration slightly increased in the placebo group. However, this was not accompanied by a decrease in TEWL. External factors maybe responsible for this minor effect.

There have been several approaches to modulate skin parameters with respect to influencing skin appearance under cosmetic aspects. With the surface evaluation of living skin method one can evaluate the skin structure and obtain information on roughness, scaling and wrinkling. Intervention with flaxseed or borage oil led to changes in roughness and scaling, which might be in part explained by the observed modulation of skin hydration and firmness. However, a parameter such as wrinkling, which results mainly from changes in the molecular structure of extracellular components, was not affected by any of the treatments.

It has been shown that under conditions of LA deficiency, LA is substituted by oleic acid in epidermal acylceramides[26], which has been associated with an increased TEWL, perturbations of the intercellular membranes and an altered epidermal homeostasis[27]. Thus, an increased LA supply may contribute to improved skin parameters. ALA and GLA may also affect skin homeostasis along this line.

Modulation of skin properties has been shown for several other nutrients[3,4], indicating that dietary intervention is a way of improving skin conditions and providing protection. It should be stated that effects are moderate and develop over a long-term period.

Acknowledgements

H. S. is a Fellow of the National Foundation for Cancer Research (NFCR), Bethesda, MD.

S. D. S. holds a scholarship of the GK 1033 (DFG). We thank A. Grieger for statistical analyses and M. Wiebusch for technical assistance. S. D. S. was responsible for the blood analyses, evaluation and interpretation of the data, statistics and was involved in drafting and writing the article. W. S. was responsible for the development of the study design, interpretation of the data, supervision of the analytical work, drafting and writing of the manuscript. H. T. and H. S. were involved in the conception of the study design, supervison of skin analyses, writing and final approval of the article. M. B. and J.-M. M. were responsible for the development of the study design, involved in the interpretation of the data, conception of the article and final approval of the manuscript. U. H. was responsible for the development of the study design, performance and control of skin measurements, interpretation and evaluation of the data, drafting and writing the manuscript. None of the authors had a personal or financial conflict of interest.

https://doi.org/10.1017/S0007114508020321 Published online by Cambridge University Press

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