ARTYKUŁ ORYGINALNY / ORIGINAL ARTICLE

Influence of folic acid supplementation on coagulation, inflammatory, lipid, and kidney function parameters in subjects with low and moderate content of folic acid in the diet

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Abstract

Background: The human body requires folic acid (FA) to produce blood cells, secure cell division, and growth. Moreover, this vitamin is important in the prevention of cardiovascular disease (CVD). Because the results of studies on the use of FA in the prevention of CVD are ambiguous, it seems necessary to conduct further research, which will explain in which cases supplementation is effective.

Aim: To assess the impact of FA supplementation on the coagulation, inflammatory, lipid parameters, and kidney function in subjects with atherosclerosis risk factors, depending on the content of FA in their diet.

Methods: The study enrolled 97 young adult Caucasian individuals (34 males and 63 females) with atherosclerosis risk factors. This population was divided into two groups: A — with low content of FA in the diet (< 40% of reference daily intake) and B — with moderate content of FA in the diet (40–90% of reference daily intake). The participants were asked to take FA in the low-dose of 0.4 mg/24 h for 3 months.

Results: Low-dose FA supplementation resulted in elevation of FA concentrations (79% vs. 75.1%) in the studied groups and, concomitantly, a decrease in homocysteine concentrations (21% vs. 20.3%). Mean level of creatinine decreased after FA supplementation in both groups (0.93 ± 1.1 vs. 0.72 ± 0.15 mg/dL and 0.83 ± 0.16 vs. 0.77 ± 0.15 mg/dL). These differences were statistically significant (p < 0.0001). The difference in mean estimated glomerular filtration rate values before and after FA supplementation was statistically significant in group A (p = 0.002) and on the border of statistical significance in group B (p = 0.06).

Conclusions: FA supplementation has no influence on the coagulation, inflammatory and lipid parameters in subjects with atherosclerosis risk factors depending on the content of FA in their diet. However FA supplementation may have a beneficial effect on kidney function in subjects with low content of FA in the diet.

Key words: diet, folic acid, kidney function, supplementation

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INTRODUCTION

The human body requires folic acid (FA) to produce blood cells, secure cell division, and growth [1, 2]. Numerous studies have shown that this vitamin is important in the prevention of neurodegenerative diseases, cancer, and cardiovascular disease (CVD) [3, 4]. In 1996 Morrison et al. [5] published a study indicating that individuals with low folate concentrations (< 25th percentile) were exposed to significantly higher
risk of death from cardiac causes than were subjects with a concentration above the 75th percentile. This finding was confirmed by the European Concerted Action Project (ECAP), in which a significant relationship between low concentrations of folate in erythrocytes and coronary artery disease, peripheral vascular disease, and stroke was observed [6]. The Framingham Heart Study found that low folate concentration was correlated with a two-fold higher risk of carotid artery stenosis. This dependency was significant even after taking into account other risk factors, including concentrations of homocysteine (Hcy) [7]. A meta-analysis by Yang et al. [8] has shown that FA supplementation may be beneficial in the prevention of stroke. Huo et al. [9] pointed out that those benefits are significant in the population deprived of FA fortification, and thus with higher shortages of folate in the diet. However, there are reports that are not consistent with the results of earlier quoted studies. The authors of the Atherosclerosis Risk in Communities Study (ARIC) denied the impact of FA on the CVD and confirmed its correlation with low concentrations of vitamin B6 in the blood [10].

Moreover, results of three large-scale studies: 2004 Vitamin Intervention for Stroke Prevention (VISP) [11], Heart Outcomes Prevention Evaluation 2 (HOPE 2) [12], and the Norwegian Vitamin Trial (NORVIT) [13] published in 2006, are also contradictory. These long-follow-up trials investigated the effect of preventive FA use (optionally with other group B vitamins) on cardiovascular incidents. The administration of FA was not associated with lower risk of new cardiovascular incidents like recurrent myocardial infarction (MI), stroke, or death from it, and in the NORVIT study the number of incidents was even increased in subjects with recent MI or with stents. Because the results of studies on the use of FA in the prevention of CVD are ambiguous, it seems necessary to conduct further research that will explain in which cases supplementation is effective.

The aim of this study was to assess the impact of FA supplementation on the coagulation, inflammatory, lipid parameters, and kidney function in subjects with atherosclerosis risk factors, depending on the content of FA in their diet.

### METHODS

The study enrolled 97 young adult Caucasian individuals (34 males and 63 females) with atherosclerosis risk factors. This population was divided into two groups: A — with low content of FA in the diet (< 40% of reference daily intake) and B — with moderate content of FA in the diet (≥ 40% and < 90% of reference daily intake). The patients’ nutrition was assessed with the use of a single 24-h dietary recall method related only to days not differing from the patients’ typical dietary regimens — a fact confirmed by cross-questioning of the dietary habits and product intake frequency. The FA contents were calculated with the use of the Polish computer program Dietetyk 2 developed by the National Food and Nutrition Institute. All subjects underwent a standard interview about atherosclerosis risk factors. The precise characteristics of the studied patients are presented in Table 1. The inclusion criteria for the study groups were: age ≥ 18 years, patient’s written informed consent, presence of atherosclerosis risk factors: family history of premature ischaemic stroke, arterial hypertension, dyslipidaemia, overweight or obesity, cigarette smoking, and low physical activity, absence of concurrent inflammation, no hypolipidaemic or metabolism-modulating agents, no administration of B-group vitamins or vitamin preparations within the six months preceding the study (use of hypotensive agents and oral contraceptives did not constitute exclusion criteria), young age of parents at the moment of ischaemic stroke (fathers younger than 55 years, mothers younger than 65 years) confirmed by means of computed tomography or magnetic resonance. The exclusion criteria were as follows: age < 18 years, lack of consent to participate in the study, non-compliance, administration of hypolipidaemic, metabolism-modulating agents, supplements, and vitamin preparations, chronic inflammation, CVD diagnosed prior to or during the study, diabetes, chronic kidney disease, and gout. The study involved an initial assessment through medical history taking, physical examination and blood analysis. Next, the participants were asked to take FA in the low-dose of 0.4 mg/24 h for three months [14], after which a follow-up examination was performed, again through medical history taking, physical examination, and blood analysis. Initially, FA supplementation was offered to 124 subjects, 27 of which (21.8%) were excluded in the course of the study due to non-compliance — taking below 80% of pills (irregular taking of FA, stopping taking it altogether, or not reporting for the follow-up examination at the end of the treatment period). The participants were given the drug in an amount sufficient until the end of the study. The study was approved by the

### Table 1. Characteristics of studied groups

<table>
<thead>
<tr>
<th></th>
<th>Group A (n = 53)</th>
<th>Group B (n = 44)</th>
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</thead>
<tbody>
<tr>
<td>FA intake [μg]</td>
<td>82.89 ± 19.73</td>
<td>199.9 ± 53.08</td>
</tr>
<tr>
<td>Age [years]</td>
<td>27.54 ± 5.4</td>
<td>28.0 ± 6.1</td>
</tr>
<tr>
<td>Sex (male/female)</td>
<td>32% male</td>
<td>39% male</td>
</tr>
<tr>
<td>Family history of PIS</td>
<td>100%</td>
<td>100%</td>
</tr>
<tr>
<td>BMI [kg/m²]</td>
<td>24.84 ± 4.3</td>
<td>23.3 ± 3.5</td>
</tr>
<tr>
<td>BMI &gt; 25 [kg/m²]</td>
<td>35%</td>
<td>34%</td>
</tr>
<tr>
<td>Dyslipidaemia</td>
<td>72%</td>
<td>70%</td>
</tr>
<tr>
<td>Cigarette smoking</td>
<td>35%</td>
<td>37%</td>
</tr>
<tr>
<td>High physical activity</td>
<td>24%</td>
<td>45%</td>
</tr>
<tr>
<td>Systolic BP [mm Hg]</td>
<td>126.0 ± 19.1</td>
<td>125.0 ± 12.3</td>
</tr>
<tr>
<td>Diastolic BP [mm Hg]</td>
<td>85.8 ± 10.8</td>
<td>83.0 ± 10.3</td>
</tr>
<tr>
<td>Arterial hypertension</td>
<td>31%</td>
<td>30%</td>
</tr>
</tbody>
</table>

Group A — low content of FA in the diet; Group B — moderate content of FA in the diet; FA — folic acid; PIS — premature ischaemic stroke; BMI — body mass index; BP — blood pressure
Bioethics Committee of the Pomeranian Medical University in Szczecin, Poland, according to the provisions of the Declaration of Helsinki (as revised in Edinburgh 2008), and written consent was obtained from all participants.

**Laboratory methods**

Fasting blood for biochemistry was collected. Blood tests comprised the following: measurements of FA and Hcy concentrations, lipid profile — total cholesterol (T-C), high-density lipoprotein-cholesterol (HDL-C), low-density lipoprotein-cholesterol (LDL-C), triglycerides (TG), apolipoprotein A1 (ApoAI), apolipoprotein B (ApoB), lipoprotein(a) (Lp(a)), coagulation, inflammatory parameters, and creatinine (Cr) concentrations. The FA level was determined by an Abbott test kit (Abbott Laboratories, Chicago, Illinois, USA) using the ion capture method on an IMX immunochromatography system (by Abbott). Total Hcy was determined by high performance liquid chromatography using test kits from Bio-Rad, on a Hewlett-Packard analyser with a fluorescence detector. TG and T-C levels were determined by enzymatic methods. LDL-C and HDL-C were obtained using the precipitation method and cholesterol concentrations in each fraction were measured. Apolipoproteins B and A1 were measured using a photometric method utilising antigen-antibody binding. Measurements were taken using reagents from the commercial test kits from Roche, and a Clinilab analyser from bioMérieux. Lp(a) was measured using a photometric method utilising antigen-antibody binding with a latex carrier, with the use of test kits from Diahor, and a Clinilab analyser. The von Willebrand factor (VWF) was measured by means of a miniVidas analyser (bioMérieux) and enzyme-linked immunosorbent assay. Fibrinogen (Fb) level was determined by the Clauss method on a Hemolab analyser, using bioMérieux test kits. High-sensitivity C-reactive protein (hsCRP) concentration was measured by the immunonephelometric method using a Dade-Behring BN-100 analyser (Dade Behring Holding GmbH, Liederbach, Germany). Creatinine concentration evaluation was performed with a Reflotron analyser using strip tests by Roche. The estimated glomerular filtration rate (eGFR) was calculated with the Modification of Diet in Renal Disease (MDRD) Study Group formula.

**Statistical analysis**

Statistical analysis was performed using the STATISTICA StatSoft Polska v.9.0 package (StatSoft Inc., Tulsa, Oklahoma, USA), and the examined parameters were first evaluated for normal distribution (Shapiro-Wilk test). The Lp(a) and hsCRP values were subjected to logarithmic transformation. All continuous variables are presented as mean ± standard deviation. Differences between biochemical parameters among the studied groups were compared using unequal variance Student’s t-test for the two samples (heteroscedastic variance). The significance level was set at p ≤ 0.05.

**RESULTS**

Table 2 shows a comparison of the analysed biochemical parameters between the groups with low and moderate content of FA in the diet. Low-dose FA supplementation resulted in elevation of FA concentrations (79% vs. 75.1%) in the studied groups and, concomitantly, a decrease in Hcy concentrations (21% vs. 20.3%). However, these differences were not statistically significant. A slight reduction in mean concentration of T-C was observed in both groups (5.3% vs. 5.04%) after FA supplementation. These differences were not statistically significant. The HDL-C values increased after FA supplementation in group A (2.05%), but decreased in group B (1.09%), and LDL-C values decreased in both groups (7.4% vs. 5.67%). Mean level of TG decreased in group A (7.8%) but increased in group B (1.71%). However, these differences did not reach the border of statistical significance. After FA supplementation, concentrations of apolipoproteins (ApoAI, ApoB) and Lp(a) did not change significantly between the groups. There was a decrease in VWF levels in group A (7.5%) and group B (4.97%), an increase in Fb levels in both groups (0.98% vs. 1.8%), and a decrease in CRP concentrations in group A (3.77%) followed by a decrease in group B (13.9%). These differences were not statistically significant. Mean level of Cr decreased after FA supplementation in both groups (0.93 ± 1.1 vs. 0.72 ± 0.15 mg/dl in group A and 0.83 ± 0.16 vs. 0.77 ± 0.15 mg/dl in group B). These differences between the groups were statistically significant (p < 0.0001). Mean eGFR values increased after FA supplementation in both groups (109.94 ± 26.8 vs. 120.92 ± 30.6 mL/min/1.73 m2 in group A and 109.45 ± 1.41 vs. 117.68 ± 4.94 mL/min/1.73 m2 in group B). These differences were not statistically significant between the groups; however, the difference in mean eGFR values before and after FA supplementation was statistically significant in group A (p = 0.002) and on the border of statistical significance in group B (p = 0.06).

**DISCUSSION**

In our study, the starting point was the analysis of FA content in the diet. We evaluated the change between the baseline FA levels and after supplementation, to verify the thesis that individuals with low FA content in the diet would benefit more. For both groups, FA intake was below daily reference, but significantly much lower in group A than in group B (82.89 ± 19.73 μg vs. 199.9 ± 53.08 μg; p < 0.00001). This was reflected in slightly lower levels of FA, although the difference was not statistically significant. Perhaps the reason for the lack of stronger correlation between consumption and blood levels of folate is a high sensitivity to thermal processing of food. Although FA supplementation affected the coagulation, inflammatory, and lipid parameters in the studied individuals, the comparison between groups divided depending on folate content in the diet revealed no significant differences. However, FA supplementation resulted in...
Table 2. Comparison of analysed biochemical parameters between groups with low and moderate content of folic acid in the diet

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Group A: Low content of FA in the diet (n = 53)</th>
<th>Group B: Moderate content of FA in the diet (n = 44)</th>
<th>p*</th>
<th>p**</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before supplementation</td>
<td>After supplementation</td>
<td>Change (95% CI); %</td>
<td>Before supplementation</td>
</tr>
<tr>
<td>Folic acid [ng/dL]</td>
<td>6.7 ± 3.14</td>
<td>12.0 ± 4.1</td>
<td>5.3 (4.29--6.28); 79.0%</td>
<td>6.85 ± 2.8</td>
</tr>
<tr>
<td>Homocysteine [μmol/L]</td>
<td>11.1 ± 3.6</td>
<td>8.7 ± 2.11</td>
<td>-2.4 [-2.98--(-1.74)]; 21.0%</td>
<td>11.3 ± 5.0</td>
</tr>
<tr>
<td>Total cholesterol [mg/dl]</td>
<td>195.6 ± 41.7</td>
<td>185.1 ± 38.6</td>
<td>-10.43 [-14.05--(-4.8)]; 5.3%</td>
<td>214.0 ± 49.3</td>
</tr>
<tr>
<td>HDL-C [mg/dl]</td>
<td>53.6 ± 12.1</td>
<td>54.7 ± 12.2</td>
<td>1.1 (-0.78--3.02); 2.05%</td>
<td>55.0 ± 10.7</td>
</tr>
<tr>
<td>LDL-C [mg/dl]</td>
<td>105.5 ± 35.2</td>
<td>97.6 ± 31.9</td>
<td>-7.9 [-11.93--(-3.38)]; 7.4%</td>
<td>119.9 ± 41.6</td>
</tr>
<tr>
<td>Triglycerides [mg/dl]</td>
<td>98.5 ± 44.7</td>
<td>90.8 ± 35.8</td>
<td>-7.7 [-16.0--0.77]; 7.8%</td>
<td>116.3 ± 95.0</td>
</tr>
<tr>
<td>Apol [mg/dl]</td>
<td>146.2 ± 19.4</td>
<td>148.4 ± 21.5</td>
<td>2.2 [-0.53--(-4.18)]; 1.5%</td>
<td>146.7 ± 19.6</td>
</tr>
<tr>
<td>ApoB [mg/dl]</td>
<td>91.1 ± 20.6</td>
<td>90.5 ± 18.3</td>
<td>-0.6 (-3.56--2.44); 0.65%</td>
<td>94.5 ± 18.5</td>
</tr>
<tr>
<td>Lp(a) [mg/dl]</td>
<td>30.5 ± 25.2</td>
<td>31.3 ± 23.7</td>
<td>0.8 (-1.02--2.40); 2.6%</td>
<td>23.2 ± 22.5</td>
</tr>
<tr>
<td>VWF [%]</td>
<td>77.2 ± 22.1</td>
<td>71.4 ± 22.1</td>
<td>-5.8 [-9.72--(-1.67)]; 7.5%</td>
<td>74.3 ± 22.7</td>
</tr>
<tr>
<td>Fibrinogen [g/L]</td>
<td>3.04 ± 0.68</td>
<td>3.07 ± 0.76</td>
<td>0.03 (-0.19--0.13); 0.98%</td>
<td>2.65 ± 0.6</td>
</tr>
<tr>
<td>C-reactive protein [mg/L]</td>
<td>1.06 ± 1.11</td>
<td>1.02 ± 1.1</td>
<td>-0.04 (-0.27--0.20); 3.77%</td>
<td>0.86 ± 1.0</td>
</tr>
<tr>
<td>Creatinine [mg/dl]</td>
<td>0.93 ± 1.1</td>
<td>0.72 ± 0.15</td>
<td>-0.21 (-0.59--0.12); 22.5%</td>
<td>0.83 ± 0.16</td>
</tr>
<tr>
<td>eGFR [ml/min/1.73 m²]</td>
<td>109.94 ± 26.8</td>
<td>120.92 ± 30.6</td>
<td>10.98 (2.9--18.9); 9.91%</td>
<td>109.45 ± 1.41</td>
</tr>
</tbody>
</table>

*Differences between baseline values of the parameters in the studied groups.
**Differences between the changes in the mean concentrations of the parameters in the studied groups.
CI — confidence interval; HDL-C — high-density lipoprotein cholesterol; LDL-C — low density lipoprotein cholesterol; Lp(a) — lipoprotein(a); ApoB — apolipoprotein B; Apol — apolipoprotein I; VWF — von Willebrand factor; eGFR — estimated glomerular filtration rate
significant kidney function improvement in the group with low folate content in the diet.

The role and mechanism of action of FA in the prevention of CVD is not fully known. Some studies link the FA metabolism with the Hcy level, as the main anti-atherosclerotic effect [4, 7, 15]. FA must be reduced and methylated to 5-methyltetrahydrofolate (5-MeTHF), which serves as a cofactor for Hcy methylation and reduction of dihydrobiopterin (BH$_2$) to tetrahydrobiopterin (BH$_4$), to become a metabolically active form that can be utilised by tissues [16]. Therefore, when content of FA in the diet is low, these reactions are insufficient. As a consequence, Hcy levels inside the cells increase and become a distress to the inner layer of arteries. In the presence of Hcy, free radicals inactivate the nitric oxide (NO) and damage the endothelium. This mechanism is the basis of atherosclerosis pathogenesis [17]. Many authors suggest that the potential beneficial effects of FA on vascular endothelium may be associated with pathways other than through Hcy. Verhaar et al. [18] first demonstrated that 5-MeTHF in higher concentration reduces free radicals originating from different reactions in the body. Moreover, it has been shown that 5-MeTHF can directly affect the activity of nitric oxide synthase (NOS), although this mechanism was not observed when the level of NOS was not associated with a shortage of BH$_4$. The beneficial effect of 5-MeTHF on NOS was noted also in healthy individuals, and some reports indicate that 5-MeTHF may stabilise BH$_4$ and prevent its oxidation to inactive BH$_3$. Many works have assessed the influence of FA on availability of NO and endothelium function; however, to explain its detailed mechanism of action, further studies are needed.

Beneficial impact of FA in regard to reducing the risk of CVD may also result from effects on the clotting mechanism. Liem et al. [20] found no association of low-dose FA supplementation with Fb concentration. Nevertheless, other reports state that FA supplementation results in a decrease of Fb concentration and an increase in plasminogen and antithrombin III levels [21]. The ARIC study confirmed that VWF is a risk factor of ischaemic stroke [10]. It was shown that FA supplementation decreases VWF concentrations. In our previous study the effect of low-dose FA supplementation (0.4 mg/24 h) on Fb, VWF, and CRP concentrations was evaluated. After FA supplementation a significant decrease in VWF concentrations in the analysed individuals was observed. Moreover, this effect was especially marked in smoking and dyslipidaemic women and in smoking, dyslipidaemic, and overweight men. FA supplementation had no effect on Fb and CRP concentrations in the studied group [22]. In this study we observed that FA supplementation resulted in significant Cr decrease in subjects with low content of FA in the diet. There are reports that administration of FA, pyridoxine, and cyanocobalamin in patients with chronic renal failure had a favourable effect on risk factors of atherosclerosis. However, authors of this report did not evaluate Cr concentrations [13]. Similar observations were made by Qin et al. [23]. The conclusion of their meta-analysis is that FA therapy can reduce CVD risk in patients with end stage renal disease by 15%. In another study, glomerular function was assessed among rats exposed to ethanol. The supplementation of FA had no effect on the Cr levels in the studied animals [24]. The results of Anti-Oxidant Therapy in Chronic Renal Insufficiency (ATIC) study indicate that Hcy-lowering therapy with use of FA, pyridoxine, and cyanocobalamin has no effect on renal function [25]. However, that study was conducted on patients with chronic kidney disease.

In the present study, there were no significant changes in the VWF serum concentrations; however, there were differences on the border of statistical significance in the group with low content of FA in the diet. Moreover, in our earlier study we found that VWF serum concentrations decreased significantly after FA supplementation, regardless of the FA content in the diet [22]. VWF is considered to be a marker of endothelial dysfunction in vascular disorders. Administration of FA improves endothelial cell function through Hcy level lowering and oxidative stress reduction. The kidneys play a crucial role in plasma Hcy clearance. Patients with kidney disease present hyperhomocysteinaemia, and markers of GFR (like serum Cr) correlate with Hcy, even in subjects with normal renal function. The association between Hcy and GFR seems linear and is present even in the hyperfiltrating range. The precise mechanism by which GFR is related to Hcy concentration is not definitively established. Nevertheless, FA supplementation through Hcy level lowering affects the serum Cr concentrations [26]. We assume that this effect can be more intensive in subjects without established kidney disease than in patients with already existing kidney failure.

CONCLUSIONS

In view of our study, FA supplementation has no impact on the coagulation, inflammatory, and lipid parameters in subjects with atherosclerosis risk factors, depending on the content of FA in their diet. However, FA supplementation may have a beneficial effect on kidney function in subjects with low content of FA in the diet.

Acknowledgements

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Conflict of interest: none declared

References


Streszczenie
Wstęp: Ludzki organizm wymaga kwasu foliowego (FA) do produkcji krwinek oraz zabezpieczenia podziału komórek i ich wzrostu. Ponadto ta witamina jest ważna w prewencji chorób sercowo-naczyniowych (CVD). Przeprowadzono wiele szeroko zakrojonych badań klinicznych obejmujących duże populacje, w których oceniano użycie FA w prewencji przede wszystkim udaru mózgu i zawału serca. Większość tych badań dotyczyła jednak profilaktyki drugorzędowej. Ponieważ wyniki stosowania FA w profilaktyce CVD są niejednoznaczne, konieczne wydaje się prowadzenie dalszych badań, które wyjaśnią, w jakich przypadkach suplementacja jest korzystna.

Cel: Celem badania była ocena wpływu suplementacji FA na parametry krzepnięcia, zapalne, lipidowe i funkcję nerek u osób z czynnikami ryzyka miażdżyca, w zależności od wyjściowego stężenia FA w ich diecie.

Metody: Do badania włączono 97 młodych osób dorosłych (34 mężczyzn i 63 kobiety) z czynnikami ryzyka miażdżyca, takimi jak dodatni wywiad rodzinny w kierunku przedwczesnego udaru niedokrwiennego mózgu, nadciśnienie tętnicze, dyslipidemia, nadwaga i otyłość, palenie tytoniu, niska aktywność fizyczna. Populacja została podzielona na dwie grupy: A — z niską zawartością FA w diecie (< 40% zalecanego spożycia) i B — ze średnią zawartością FA w diecie (40–90% zalecanego spożycia). Uczestników badania poproszono o przyjmowanie FA w niskiej dawce 0,4 mg/24 h przez 3 miesiące. Zaproponowana dawka została ustalona na podstawie doniesień z piśmiennictwa i wcześniejszych badań autorów. We krwi badanych oznaczono stężenia: FA, homocysteiny, profilu lipidowego — cholesterolu całkowitego, frakcji HDL, frakcji LDL i triglicerydów, apolipoproteiny A1, apoliporoteiny B, lipoproteiny (a), czynnika von Willebranda, fibrynogenu, białka C-reaktywnego i kreatyniny.

Wyniki: Suplementacja niskimi dawkami FA spowodowała wzrost jego stężeń (79% vs 75,1%) w obu grupach ze współtowarzyszącym obniżeniem stężeń homocysteiny (21% vs 20,3%). Jednak różnice te nie były istotne statystycznie. Różnice w średnich stężeniach parametrów krzepnięcia, zapalnych i lipidowych nie były znamienne po suplementacji FA. Średnie stężenie kreatyniny obniżyło się po suplementacji FA w obu grupach badanych (odpowiednio 0,93 ± 1,1 vs 0,72 ± 0,15 mg/dl i 0,83 ± 0,16 vs. 0,77 ± 0,15 mg/dl). Różnica te były istotne statystycznie (p < 0,0001). Średnie wartości estymowanego wskaźnika filtracji kłębuszkowej przed suplementacją i po suplementacji FA różniły się znaminnie w grupie A (p = 0,002) i były na granicy istotności statystycznej w grupie B (p = 0,06).

Wnioski: Suplementacja FA nie wpływa na parametry krzepnięcia, zapalne i lipidowe u osób z czynnikami ryzyka miażdżyca w zależności od wyjściowego stężenia FA w ich diecie. Jednak podaż FA może mieć korzystny wpływ na funkcję nerek wśród osób z niską zawartością FA w diecie. Poprawa w zakresie funkcji nerek może się wiązać z korzystnym wpływem FA na śródbłonke naczyń.

Słowa kluczowe: dieta, funkcja nerek, kwas foliowy, suplementacja

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