CLINICAL-DIAGNOSTIC STUDIES

VITAPHOSPHOLIP® (WATER-SOLUBLE PHOSPHATIDYLCHOLINE) IN THE TREATMENT OF COMBINED HYPERLIPIDEMIA: A RANDOMIZED, PLACEBO-CONTROLLED CLINICAL TRIAL

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Vitaphospholip®, a water-soluble form of phosphatidylcholine, has been evaluated in a clinical trial aimed at reducing non-HDL-cholesterol (non-HDL-C) and triglyceride (TG) levels in patients with combined hyperlipidemia. The randomized, double-blind, placebo-controlled study included 100 patients. Vitaphospholip® or placebo was administered orally 500 mg twice a day for 12 weeks. Treatment with Vitaphospholip® resulted in a 13.2% decrease in non-HDL-C compared to 4.3% in the placebo group ($p = 0.001$). The absolute decrease of non-HDL-C was 0.6 mmol/l compared to -0.2 mmol/l in the placebo group ($p = 0.001$). The target non-HDL-C level of less than 3.4 mmol/L was achieved in 15 of 39 patients (38.5%) in the Vitaphospholip® group versus 2 of 41 patients (4.9%) in the placebo group ($p = 0.000$). The absolute decrease of TG in the group of patients treated with Vitaphospholip® was -0.7 mmol/l versus -0.1 mmol/L in the placebo group ($p = 0.001$). During therapy with Vitaphospholip®, a significant decrease in the levels of apolipoprotein B, total cholesterol, and very low-density lipoprotein cholesterol was observed. No changes in liver or kidney function, vital signs, or ECG were registered. No serious adverse events were identified. Thus, Vitaphospholip® significantly reduced the levels of non-HDL-C, TG, and atherogenic lipoprotein in patients with combined hyperlipidemia and moderate cardiovascular risk.

Keywords: Vitaphospholip®; water-soluble phosphatidylcholine; combined hyperlipidemia; non-HDL-C; triglycerides

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INTRODUCTION

Combined hyperlipidemia (earlier known as type II B hyperlipidemia or familial combined hyperlipidemia) is a disease characterized by mild to moderate hypertriglyceridemia and low levels of high-density lipoprotein (HDL) cholesterol.

Blood triglyceride (TG) levels are known to be closely associated with the development of atherosclerosis and cardiovascular disease (CVD) [1–3]. All lipoprotein particles, which transport TG, contain an apoB molecule, and non-HDL cholesterol (non-HDL-C), are currently widely used as a standard in clinical practice to assess all atherogenic lipoproteins containing apoB. A high correlation ($r = 0.90$) has been found between changes in non-HDL-C and changes in TG ($p < 0.001$) [4].

HDL, in turn, are antiatherogenic substances; their main function is to facilitate reverse cholesterol transport (RCT). The first step of RCT is the efflux of cholesterol from macrophages into HDL [5]. Animal studies have shown an inverse correlation between cholesterol efflux and the size of atherosclerotic lesions [6]. In clinical studies, cholesterol efflux was negatively correlated with the incidence of CVD [7].

At the Institute of Biomedical Chemistry (IBMC) improvement of cholesterol removal was achieved by means of a method producing ultra-small phospholipid (PL) nanoparticles (micelles) from soy phosphatidylcholine with an average size of 30 nm [8]. Mass spectrometric analysis has shown that PL nanoparticles (micelles) promote HDL phospholipidation, i.e., they enrich HDL with dilinoleoylphosphatidylcholine [9]. An *in vitro* study showed a dose-dependent 8-fold increase of PL in HDL and a two-fold increase in cholesterol efflux compared to native apoB-depleted plasma during incubation with PL nanoparticles (micelles) [10]. An *in vivo* study showed that intravenous administration of PL nanoparticles (micelles) to rabbits

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fed a cholesterol-enriched diet protected the vascular wall from the development of intimal lipid lesions and demonstrated a similar reduction in the severity of atherosclerotic lesions compared to atorvastatin and fenofibrate [10].

It is expected that phospholipidation of HDL with PL nanoparticles will reduce in the level of atherogenic lipoproteins. In this context, a water-soluble form of PL, Vitaphospholip®, has been developed at IBMC [11, 12] and results of a phase I clinical trial demonstrated the safety of two and three oral doses of 1000 mg of Vitaphospholip® for 7 days in 14 healthy volunteers [13].

The aim of this clinical trial was to evaluate the safety and efficacy of 12-week oral administration of 1000 mg of Vitaphospholip® in changing non-HDL-C, TG and atherogenic lipoproteins in patients with combined hyperlipidemia and moderate cardiovascular risk compared to placebo.

MATERIALS AND METHODS

Study Design

A multicenter, randomized, double-blind, placebo-controlled study of Vitaphospholip® was conducted at three clinical centers in Russia (Russian Cardiology Research and Production Complex, Moscow; Medical Center for Diagnostics and Prevention Plus, Yaroslavl; and Nizhny Novgorod Medical Clinic, Nizhny Novgorod) from January 1, 2016, to June 1, 2018 (ClinicalTrials ID NCT05742022).

Inclusion and Exclusion Criteria and Randomization

The study included patients aged 30–75 years with no history of coronary artery disease, acute myocardial infarction, stroke, or transient ischemic attack; peripheral arterial disease, heart failure with moderate cardiovascular risk and TG levels of 1.7–4.5 mmol/l, HDL <1.0 mmol/l for men and <1.2 mmol/l for women.

Exclusion criteria were: diseases or metabolic disorders that would increase LDL, C, and TG (secondary dyslipidemia); patients receiving high doses of statins (rosuvastatin ≥40 mg, atorvastatin ≥80 mg); type 1 diabetes mellitus (DM); patients with liver and kidney decompensation; porphyria, myopathy, central nervous system diseases; patients with exacerbation of chronic infectious diseases, with acute conditions (infectious diseases, injuries, surgeries) less than 2 months before screening; patients with a positive test for HIV, hepatitis B, C, syphilis; cancer within the last 5 years; hypothyroidism or thyroid-stimulating hormone (TSH) levels exceeding one unit above the upper limit of normal at screening; glomerular filtration rate less than 30 ml/min/1.73 m^2; alanine aminotransferase or aspartate aminotransferase levels 2-fold higher than the upper limit of norm or below the lower limits of normal in blood and urine tests; and alcohol abuse.

Randomization was performed using uniform-distributed data and the envelope method, in blocks of four envelopes (1:1) from randomly generated data. Patients were assigned to receive Vitaphospholip® (n = 50) or placebo (n = 50). Vitaphospholip® or placebo was administered orally 500 mg twice daily.

The screening period lasted 1–3 days. The treatment period was 12 weeks, with a 4-week post-treatment follow-up.

The study procedures included monitoring of vital signs (blood pressure, heart rate, respiratory rate, and oxygen saturation), body mass index, waist circumference, electrocardiogram (ECG) parameters, complete blood count, blood biochemical analysis, coagulation profile, lipid metabolism parameters (total cholesterol (TC), non-HDL-C, HDL-C, LDL-C, VLDL-C, TG, ApoB, ApoA, and Lp(a)), and high-sensitivity C-reactive protein (hsCRP) levels.

Sample Size Justification

According to the results of large-scale randomized clinical trials, statin therapy reduces non-HDL-C by 17–39% from baseline [14], with a standard deviation σ not more than 17%. For $\alpha = 0.05$, study power of at least 80% ($\beta = 0.2$), an expected difference between the study and control groups for the primary efficacy endpoint of at least 15%, and a margin of superiority δ of 5%, the sample size would be 36 patients. Therefore, the planned statistical test requires results for at least 72 study subjects. Assuming a dropout rate of approximately 30%, it is recommended to enroll at least 36/(1–0.15) ~50 patients (in each group), for a total of 100 patients.

Efficacy Endpoints

The primary efficacy endpoint was the percentage change from baseline in non-HDL-C after 12 weeks of treatment with Vitaphospholip® compared with placebo.

Secondary efficacy endpoints included the percentage change from baseline in TG, TC, HDL, LDL, VLDL, apoB, apoA, and Lp(a); and hsCRP levels after 12 weeks of treatment with Vitaphospholip® compared with placebo.

Statistical Analysis

Statistical analysis and data interpretation were performed by an independent biostatistician. Statistical analysis was performed using R software (version 4.1). Continuous parameters are presented as mean and standard deviation (M±SD) and/or median and interquartile range (Med [Q1; Q3]). 95% confidence intervals were calculated for the mean. Exact 95% confidence intervals were calculated for the median. Qualitative parameters are expressed as relative frequencies in percentages with corresponding 95% confidence intervals.

Differences between two independent samples were assessed using the Mann-Whitney test for continuous parameters, and Fisher's exact test for discrete parameters. Differences between two dependent samples were assessed using the Wilcoxon signed-rank test for continuous parameters, and the McNemar exact test for discrete parameters. Quantile regression was used to assess the difference in parameter medians in two independent subgroups. The dependent variable was the parameter under study, and the independent variable was the subgroup indicator. The difference in medians was estimated using the regression coefficient for the independent variable. Differences were considered statistically significant at $p < 0.05$.

RESULTS

Patients

Among 325 patients screened, 170 did not meet the inclusion criteria: the most common of these were TG levels less than 1.7 mmol/l and HDL-C greater than 1.0 mmol/l for men and 1.2 mmol/l for women. Another 55 patients were excluded due to high-dose statin use and type 1 DM. Therefore, 100 patients were randomized to receive Vitaphospholip® or placebo (Fig. 1).

The first patient was included in the study on April 26, 2016, and the last patient was included on November 8, 2017.

Three Vitaphospholip® group patients and two placebo group patients discontinued the study early at visit 4 due to discontinuation of the study medication. Eight patients in the Vitaphospholip® group and seven patients in the placebo group were excluded by researchers due to non-compliance with the recommended diet (5 and 4, respectively) and alcohol consumption (3 and 3, respectively). Thus, 20 patients were excluded from the efficacy analysis but were included in the safety analysis (Fig. 1).

The mean compliance rate (percentage of the total planned dose received) was 78% in the Vitaphospholip® group and 82% in the placebo group.

Demographic and baseline characteristics of patients were generally similar in both groups (Table 1). In the Vitaphospholip® group, the baseline non-HDL-C level of 4.5 (4.1 – 5.3) mmol/l was basically the same as the placebo group (4.7 (4.2 – 5.2) mmol/l; $p = 0.918$).

Efficacy Assessment

Primary efficacy endpoint: percentage change in non-HDL-C. After 12 weeks of treatment, a statistically significant reduction in non-HDL-C was observed in the Vitaphospholip® group patients compared with the placebo group patients: -13.2% versus -4.3% ($p = 0.001$) (Table 2). The absolute change in non-HDL-C from baseline to week 12 in the Vitaphospholip® group patients

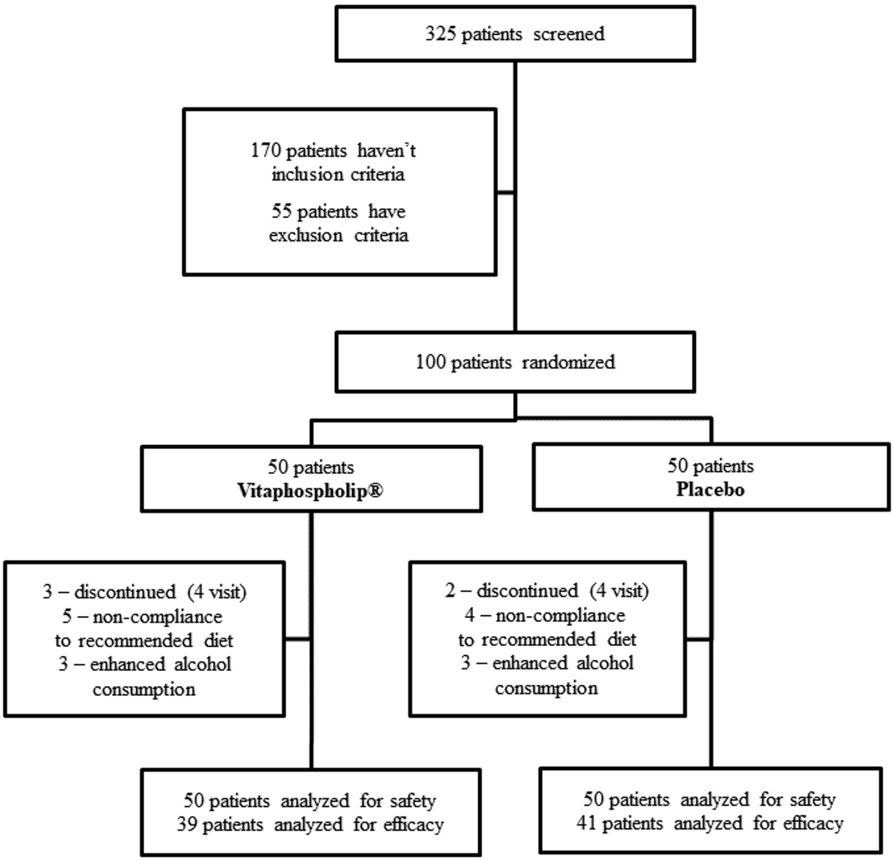

Figure 1. Study design.

Table 1. Demographic and clinical characteristics at baseline in the Vitaphospholip® and placebo groups in the per-protocol population

Initial characteristics	Vitaphospholip® (n = 39)	Placebo (n = 41)	*p*
Age, years, M±SD	56.9±10.4	56.2±9.5	0.732
Males, n (%)	23 (59)	19 (46)	0.273
Body mass, kg	88.4 (75.7 – 94.5)	89.0 (78.0 – 95.9)	0.711
Body mass index (BMI), kg/m^2	29.0 (26.1 – 32.8)	30.2 (28.3 – 34.0)	0.218
CVD risk factors risk factors, n (%)			
Dyslipidemia	39 (100)	41 (100)	1.000
Hypertension	37 (95)	38 (93)	1.000
History of smoking	28 (72)	29 (70)	1.000
Current smoking	12 (32)	14 (34)	1.000
Type 2 diabetes mellitus	1 (3)	1 (2)	1.000
Impaired glucose tolerance	6 (15)	5 (12)	0.753
Coronary artery disease	2 (5)	2 (5)	1.000
Acute myocardial infarction	0	0	1.000
Stroke	0	0	1.000
Transient ischemic heart attack	0	0	1.000
Peripheral arterial disease	0	0	1.000
Heart failure	0	0	1.000
Statins, n (%)	37 (95)	39 (95)	1.000
Antiplatelet agents, n (%)	39 (100)	39 (98)	1.000
β-blockers, n (%)	23 (58)	25 (62)	1.000
ACE inhibitors, n (%)	11 (28)	12 (30)	1.000
Other antihypertensive drugs: Ca^{2+} antagonists, AT-II receptor blockers, n (%)	4 (10)	3 (7)	1.000
Antidiabetic drugs, n (%)	7 (18)	6 (15)	1.000
Non-HDL-C, mmol/l	4.5 (4.1 – 5.3)	4.7 (4.2 – 5.2)	0.918
TG, mmol/l	2.5 (2.0 – 2.8)	2.3 (1.9 – 2.7)	0.382
TC, mmol/l	5.5 (5.1 – 6.3)	5.6 (5.1 – 6.3)	0.884
HDL-C, mmol/l	1.0 (0.9 – 1.0)	1.0 (0.9 – 1.2)	0.779
LDL-C, mmol/l	3.4 (3.0 – 3.9)	3.5 (2.9 – 3.9)	0.987
VLDL-C, mmol/l	1.1 (0.9 – 1.3)	1.0 (0.9 – 1.2)	0.382
Apolipoprotein B, mg/dl	111.0 (98.5 – 124.5)	115,0 (99.0 – 125.0)	0.486
Apolipoprotein A, mg/dl	134.0 (122.5 – 143.5)	137.0 (124.0 – 149.0)	0.400
Lipoprotein (a), mg/dl	11.2 (3.7 – 38.8)	8.4 (5.2 – 13.7)	0.320
hsCRP, mg/dl	0.21 (0.13 – 0.38)	0.19 (0.13 – 0.38)	0.994

Results are presented as Med (Q1;Q3), unless otherwise stated. BMI – body mass index; CVD – cardiovascular disease; HDL-C – high-density lipoprotein cholesterol; TG – triglycerides; LDL-C – low-density lipoprotein cholesterol; VLDL-C – very-low-density lipoprotein cholesterol; hsCRP – high-sensitivity C-reactive protein.

was -0.6 (-1.2 – -0.2) mmol/l compared with -0.2 (-0.4 – 0.3) mmol/l in the placebo group patients (p = 0.001). Before treatment, only one patient (in the placebo group) had a non-HDL-C level less than 3.4 mmol/l. After 12 weeks of treatment, the target non-HDL-C level of less than 3.4 mmol/l was achieved in 15 of 39 patients (38.5%) in the Vitaphospholip® group versus 2 of 41 patients (4.9%) in the placebo group (p = 0.000). The OR (odds ratio) estimate was 11.8 (2.4 – 116).

Secondary Efficacy Endpoints. Twelve weeks of treatment with Vitaphospholip® significantly reduced TG, atherogenic lipid, and lipoprotein levels. The TG percentage change in the Vitaphospholip® group patients was -23.8% versus -3.7% in the placebo group patients (p = 0.000). The percentage of patients achieving a fasting TG level of less than 1.7 mmol/l was 22 patients (56.4%) in the Vitaphospholip® group versus 9 patients (22%) in the placebo group (p = 0.003) (Table 3).

Table 2. Primary endpoint of efficacy: changes in the non-HDL-C level

Parameters	Vitaphospholip® (n = 39)		Placebo (n = 41)		*p*
	Med (Q1; Q3)	95% CI	Med (Q1; Q3)	95% CI	
Baseline non-HDL-C, mmol/l	4.5 (4.1– 5.3)	4.2 – 5.0	4.7 (4.2 – 5.2)	4.3 – 5.0	0.918
non-HDL-C after 12 weeks, mmol/l	3.8 (3.0 – 4.8)	3.1 – 4.4	4.2 (3.8 – 5.1)	3.8 – 4.8	0.008
Absolute change, mmol/l	-0.6 (-1.2 – -0.2)	-1.0 – -0.3	-0.2 (-0.4 – 0.3)	-0.3 – 0.1	0.001
Percent change from baseline	-13,2 (-29.1 – -3.9)	-23.0 – -5.9	-4.3 (-9.9 – 6.5)	-8.3 – 3.1	0.001

Here and in other tables CI is confidence interval.

Table 3. Secondary endpoint of efficacy: changes in the TG level

Parameters	Vitaphospholip® (n = 39)		Placebo (n = 41)		*p*
	Med (Q1; Q3)	95% CI	Med (Q1; Q3)	95% CI	
TG baseline, mmol/l	2.5 (2.0 – 2.8)	2.0 – 2.7	2.3 (1.9 – 2.7)	2.0 – 2.6	0.382
TG after 12 weeks, mmol/l	1.5 (1.3 – 2.3)	1.4 – 2.3	2.2 (1.7 – 2.8)	1.9 – 2.6	0.014
Absolute change, mmol/l	-0.7 (-1.2 – -0.3)	-1.0 – -0.3	-0.1 (-0.5 – 0.4)	-0.4 – 0.1	0.001
Percent change from baseline	-23,8 (-48.2 – -12.0)	-46.8 – -18.2	-3,7 (-20.0 – 15.4)	-16.9 – 5.6	0.000

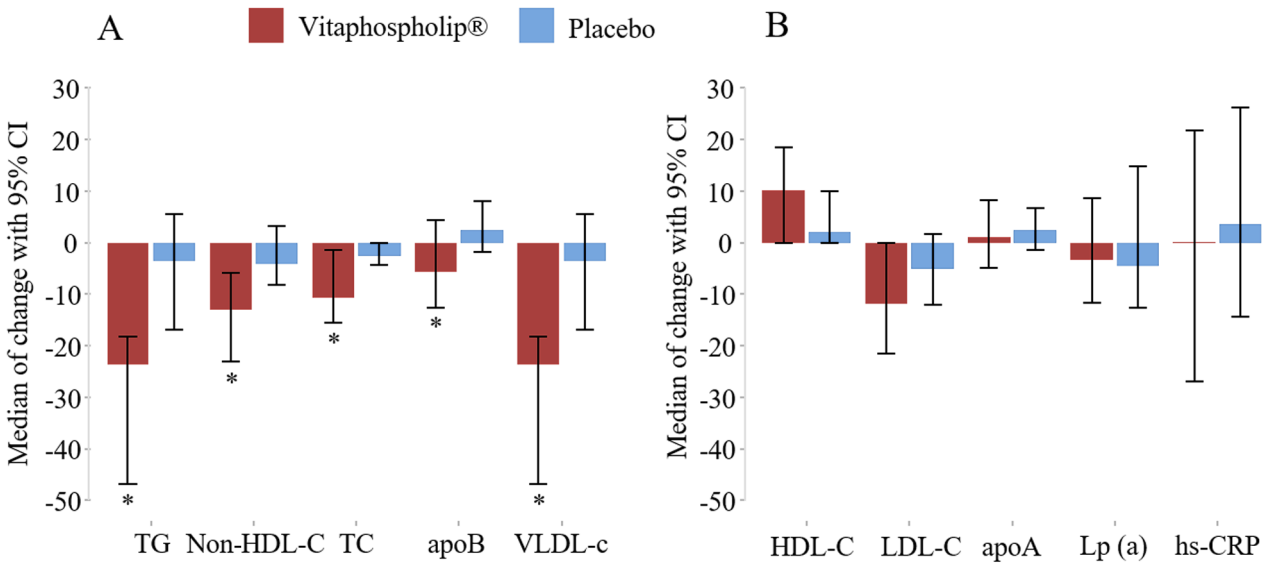

Figure 2. Statistically significant (**A**) and non-significant (**B**) changes in lipid parameters. TG – triglycerides; TC – total cholesterol; apoB – apolipoprotein B; VLDL-C – very low-density lipoprotein cholesterol; HDL-C – high-density lipoprotein cholesterol; LDL-C – low-density lipoprotein cholesterol; apoA – apolipoprotein A; Lp(a) – lipoprotein (a); Hs-SR – high-sensitivity C-reactive protein. *$p < 0.05$.

VLDL-C changes were similar to TG changes in the Vitaphospholip® and placebo group patients ($p = 0.000$) (Table 4). There were non-significant trends in changes in LDL-C in the Vitaphospholip® group: -11.9% versus -5.2% in the placebo group ($p = 0.234$) and HDL-C: +10% versus +2.0% ($p = 0.327$).

The TC decrease in the Vitaphospholip® group patients of -10.8% was more pronounced than in the placebo group patients (-2.7%, $p = 0.008$). The change in apoB levels in the Vitaphospholip® group patients was -5.8% decrease versus +2.3% increase in the placebo group patients ($p = 0.009$).

No significant changes were found in apoA ($p = 0.419$), Lp(a); ($p = 0.756$), and hsCRP ($p = 0.553$) in the Vitaphospholip® group patients compared with the placebo group patients (Fig. 2).

Safety Assessment

No serious adverse events (SAEs) were observed in either group. Adverse events (AEs) were similar in the Vitaphospholip® and placebo group patients: 14 (28%) versus 17 (34%); most of them were mild (93% and 100%, respectively). No serious AEs were

Table 4. Secondary endpoints of efficacy: changes in lipid and lipoprotein levels

Parameters	Vitaphospholip® (n = 39)		Placebo (n = 41)		*p*
	Med (Q1; Q3)	95% CI	Med (Q1; Q3)	95% CI	
Total cholesterol					
Baseline, mmol/l	5.5 (5.1 – 6.3)	5.2 – 6.1	5.6 (5.1 – 6.3)	5.3 – 6.1	0.884
After 12 weeks, mmol/l	4.8 (4.1 – 5.9)	4.2 – 5.5	5.6 (4.8 – 6.2)	5.0 – 6.1	0.019
Absolute change, mmol/l	-0.6 (-1.0 – 0.1)	-0.8 – -0.1	-0.1 (-0.4 – 0.3)	-0.3 – 0.0	0.011
Percent change from baseline	-10.8 (-18.8 – 1.4)	-15.6 – -1.4	-2.7 (-7.1 – 5.9)	-4.3 – 0.0	0.008
HDL-C					
Baseline, mmol/l	1.0 (0.9 – 1.0)	1.0 – 1.0	1.0 (0.9 – 1.2)	0.9 – 1.1	0.779
After 12 weeks, mmol/l	1.1 (1.0 – 1.3)	1,0 – 1.2	1.0 (0.9 – 1.2)	1.0 – 1.2	0.574
Absolute change, mmol/l	0.1 (0.0 – 0.2)	0.0 – 0.2	0.0 (0.0 – 0.2)	0.0 – 0.1	0.388
Percent change from baseline	10.0 (-1.5 – 21.8)	0.0 – 18.5	2.0 (-1.0 – 22.2)	0.0 – 10.0	0.327
LDL-C					
Baseline, mmol/l	3.4 (3.0 – 3.9)	3.0 – 3.7	3.5 (2.9 – 3.9)	3.1 – 3.9	0.987
After 12 weeks, mmol/l	3.0 (2.3 – 3.7)	2.4 – 3.5	3.1 (2.8 – 3.8)	3.0 – 3.6	0.206
Absolute change, mmol/l	-0.3 (-0.8 – 0.1)	-0.7 – 0.0	-0.2 (-0.6 – 0.2)	-0.5 – 0.1	0.292
Percent change from baseline	-11.9 (-23.8 – 3.9)	-21.5 – 0.0	-5.2 (-19.5 – 6.4)	-12.1 – 1.6	0.234
VLDL-C					
Baseline, mmol/l	1.1 (0.9 – 1.3)	0.9 – 1.2	1.0 (0.9 – 1.2)	0.9 – 1.2	0.382
After 12 weeks, mmol/l	0.7 (0.6 – 1.1)	0.6 – 1.0	1.0 (0.8 – 1.2)	0.8 – 1.2	0.014
Absolute change, mmol/l	-0.3 (-0.5 – -0.1)	-0.5 – -0.1	0.0 (-0.2 – 0.2)	-0.2 – 0.1	0.000
Percent change from baseline	-23.8 (-48.2 – -12.0)	-46.8 – -18.2	-3.7 (-20.0 – 15.4)	-16.9 – 5.6	0.000
Apolipoprotein B					
Basal level, mmol/l	111.0 (98.5 – 124.5)	105.0 – 117.0	115.0 (99.0 – 125.0)	108.0 – 122.0	0.486
After 12 weeks, mmol/l	103.0 (88.5 – 125.5)	92.0 – 117.0	119.0 (105.5 – 135.2)	109.0 – 129.0	0.011
Absolute change, mmol/l	-6.0 (-18.5 – 9.0)	-14.0 – 5.0	2.5 (-5.0 – 13.2)	-2.0 – 11.0	0.015
Percent change from baseline	-5.8 (-18.1 – 8.1)	-12.7 – 4.3	2.3 (-4.1 – 12.2)	-1.8 – 8.0	0.009
Apolipoprotein A					
Basal level, mmol/l	134.0 (122.5 – 143.5)	124.0 – 140.0	137.0 (124.0 – 149.0)	131.0 – 145.0	0.400
After 12 weeks, mmol/l	139.0 (120.0 – 149.0)	128.0 – 147.0	144.0 (132.0 – 157.0)	135.0 – 152.0	0.151
Absolute change, mmol/l	1.0 (-10.0 – 17.0)	-8.0 – 9.0	3.0 (-4.0 – 12.0)	-2.0 – 9.0	0.389
Percent change from baseline	0.9 (-7.3 – 12.9)	-5.0 – 8.2	2.3 (-2.8 – 9.4)	-1.5 – 6.6	0.422
Lipoprotein (a)					
Basal level, mg/dl	11.2 (3.7 – 38.8)	5.0 – 21.9	8.4 (5.2 – 13.7)	5.5 – 12.8	0.320
After 12 weeks, mg/dl	9.1 (3.9 – 22.7)	5.0 – 14.4	8.8 (5.3 – 12.0)	6.1 – 11.2	0.596
Absolute change, mg/dl	-0.2 (-3.3 – 0.5)	-1.5 – 0.3	-0.6 (-2.1 – 0.9)	-0.8 – 0.5	0.794
Percent change from baseline	-3.5 (-19.6 – 14.3)	-11.7 – 8.6	-4.6 (-21.1 – 29.3)	-12.7 – 14.7	0.756
hsCRP					
Basal level, mg/dl	0.21 (0.13 – 0.38)	0.15 – 0.30	0.19 (0.13 – 0.38)	0.15 – 0.30	0.994
After 12 weeks, mg/dl	0.21 (0.11 – 0.43)	0.14 – 0.30	0.22 (0.12 – 0.38)	0.14 – 0.30	0.746
Absolute change, mg/dl	0.00 (-0.06 – 0.06)	-0.05 – 0.00	0.00 (-0.06 – 0.08)	-0.05 – 0.00	0.910
Percent change from baseline	0.0 (-40.0 – 35.9)	-26.9 – 21.7	3.4 (-31.0 – 45.0)	-14.3 – 26.1	0.557

observed in either group. The most common AEs in the Vitaphospholip® and placebo groups were upper respiratory tract infections: 5/14 (12%) versus 5/17 (10%) and gastrointestinal AEs (dyspepsia): 5/14 (10%) versus 4/17 (8%), respectively (Table 5).

No patient experienced a decrease in platelet count below 150,000 mm^3 or changes in other blood parameters; no differences in liver biochemistry tests, renal function, or significant changes in other laboratory parameters were observed; no changes in vital signs or electrocardiographic measurements were detected.

DISCUSSION

This study has shown that Vitaphospholip® (a water-soluble form of phosphatidylcholine), significantly reduced compared with placebo non-HDL-C (p = 0.001) and TG (p = 0.000) after 12 weeks of therapy at a daily dose of 1 g in patients with combined hyperlipidemia and moderate cardiovascular risk. According to the PubMed and ClinicalTrials.gov databases, this is the first randomized, placebo-controlled clinical trial of a water-soluble phosphatidylcholine preparation on non-HDL-C and TG levels.

The reduction in non-HDL-C after 12 weeks of treatment in the Vitaphospholip® group (0.6 mmol/l) was 2-fold higher compared to the results obtained in a total of 24 clinical trials of non-statins (fibrates, niacin, omega-3) (0.3 mmol/l). In 49 studies of non-statins and statins, the average reduction in non-HDL-C was 0.5 mmol/l and was similar to the average reduction in non-HDL-C of 0.6 mmol/l detected in the Vitaphospholip® group patients (Table 6) [4].

The 0.7 mmol/l reduction in TG after 12 weeks of treatment with Vitaphospholip® was 2-fold higher than in 24 non-statin studies (0.35 mmol/l), including the Reduce IT (Icosapent Ethyl omega 3) study, and 3.5-fold higher than in 25 statin studies (0.2 mmol/l). Overall, the average TG reduction in 49 non-statin and statin studies was 0.3 mmol/l [4] versus 0.7 mmol/l observed in this study in the Vitaphospholip® treated patients.

The hazard ratio of TG reduction to the risk of major cardiovascular events (CVE) in the 49 statin and non-statin studies was 0.84 per 1 mmol/l (p = 0.0026) [4]. Thus, Vitaphospholip® is 3.5 times more effective in reducing TG levels than statins and reduces the residual risk of developing cardiovascular events by 4 times as monotherapy or in combination with statins (Table 7) [15].

Table 5. Adverse events

Parameters, n (%)	Vitaphospholip® (n = 50)	Placebo (n = 50)	*p*
Serious adverse events (SAEs)	0	0	—
Adverse events (AEs)	14 (28)	17 (34)	—
Mild AEs Moderate AEs Severe AEs	13 (93) 1 (7) 0	17 (100) 0 0	0.452 0.452 —
Platelets count < 150000 mm^3	0	0	—
ALT ≤2 ULN AST ≤2 ULN	0 0	0 0	— —
Increase in creatine by more than 1 mmol/l	0	0	—
GFR decrease by more than 10% from baseline	0	0	—

ALT – alanine aminotransferase, AST – aspartate aminotransferase, GFR – glomerular filtration rate, ULN – upper limit of normal.

Table 6. Comparison of the effectiveness of Vitaphospholip® with other lipid-lowering drugs

Parameter	Vitaphospholip®	Omega-3	Niacin	Fibrates	Non-statins	Statins	Non-statins + statins
Number of studies	1	12	3	9	24	25	49
Number of patients	100	125544	32995	41520	200059	177088	377147
Baseline non-HDL-C, mmol/l	4.5	3.6	2.3	4.0	3.3	4.3	3.8
Baseline TG, mmol/l	2.5	1.8	2.4	2.1	2.1	1.7	2.0
Non-HDL-C decrease, mmol/l	0.6	0.1	0.4	0.5	0.3	1.0	0.5
TG decrease, mmol/l	0.7	0.2	0.4	0.5	0.35	0.2	0.3

Data on Omega-3, niacin, fibrates, non-statins, statins and their combinations have been taken from [4].

Table 7. Association of lipid-lowering therapy with a reduction in residual CVD risk

Parameter	Vitaphospholip®	Statins
Non-HDL-C decrease during therapy	0.6 mmol/l	1.0 mmol/l
Percent of CVD risk decrease	15%	25%
TG decrease during therapy	0.7 mmol/l	0.2 mmol/l
Percent of CVD risk decrease	10%	2.5%

Data on statins have been taken from [4].

A phase II clinical trial of olersazen (apo CIII inhibitor) showed a 2.08 mmol/l (65%) reduction in TG levels from baseline after 6–12 months of treatment [16]. In the STRENGTH trial of omega-3 fatty acids (eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA)), a 19% reduction in TG levels in patients taking statins was not associated with a reduction in the primary endpoint (major cardiovascular events), and the STRENGTH trial was therefore stopped [17]. Despite established TG-lowering strategies, a therapy that modifies HDL particle function that would reduce CVD risk remains unknown [18]. Trials aimed at increasing HDL levels by using HDL nanoparticles have mostly been stopped due to adverse events and low efficacy [19–22]. In 2025, a placebo-controlled study of intravenous apo A-I (CSL112) (NCT03473223) was completed; however, at the time of writing, its results have not been published yet.

Nevertheless, the main conclusion by Day, that "only one substance has demonstrated the ability to prevent the development of experimental atherosclerosis: lecithin (phosphatidylcholine)" [23], still remains relevant. An *in vitro* study showed that superphospholipidation with phosphatidylcholine increased RCT [24]. The authors suggest that increased HDL PL may contribute to increased RCT in patients with metabolic syndrome and early stages of coronary artery disease. These patients were found to have low RCT, low HDL, and high TG levels. The low HDL PL content was positively correlated ($r = 0.62$) with low TCR levels ($p < 0.001$) [25].

Recently, a PL nanoparticle, miNano (15±1 nm), has been developed; it directly binds to cholesterol crystals (C), dissolves them, and increases RCT. Experimental data indicate, that miNano prevented foam cell formation and stabilized atherosclerotic plaques. The authors suggest that these results may provide a new approach to treating atherosclerosis using miNano phospholipid nanoparticles [26].

These data are consistent with the results of a large number of studies on the effects of various PL classes on HDL in patients with stable coronary artery disease and acute coronary syndrome (ACS). In ACS patients, the phosphatidylcholine/phosphatidylethanolamine (PC/PE) ratio in HDL was lower than in patients with stable coronary artery disease. The authors suggest that phospholipidation by PC in ACS patients stabilizes atherosclerotic plaques and prevents their rupture and the development of myocardial infarction [27].

In this context Vitaphospholip® has certain advantages over the miNano phospholipid particles discussed above. The water-soluble PC form was obtained without detergents and apoproteins by homogenization [11]. The developed water-soluble dosage form of PC, Vitaphospholip®, demonstrated safety and efficacy over 12 weeks of oral administration and convenience for long-term use.

Our clinical study has certain limitations, most of which highlight prospects for future research, including the use of coronary angiography or computer-aided monitoring of treatment effectiveness. This study did not include patients with clinical evidence of cardiovascular disease, but only those with risk factors and had an age limit of 30–75 years. Treatment duration was short-term, just 12 weeks. Future studies will include patients aged 18 years and older, with and without cardiovascular disease, who will receive Vitaphospholip® for at least 1 year with angiographic monitoring of efficacy.

Thus, the developed original water-soluble form of PC for oral administration, Vitaphospholip®, demonstrated safety, good tolerability, and efficacy at a daily dose of 1 g during the 12 week-course to reduce non-HDL-C and TG in patients with combined dyslipidemia and moderate cardiovascular risk.

CONCLUSIONS

Vitaphospholip® significantly reduces non-HDL-C, TG, and atherogenic lipoprotein levels in patients with combined hyperlipidemia and moderate cardiovascular risk. Its mechanism of action, targeting HDL phospholipidation, is a unique and effective approach that leads to a reduction in non-HDL-C, TG, and atherogenic lipoproteins [28]. Vitaphospholip® may be used for the treatment of patients with combined hyperlipidemia as monotherapy or in combination with statins and fibrates.

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COMPLIANCE WITH ETHICAL STANDARDS

The study was conducted in accordance with the principles of the Declaration of Helsinki. The study protocol was approved by the Ethics Council of the Russian Ministry of Health (no. 8414 dated July 10, 2015), as well as by the local ethics committees of the study centers: the Ethics Committee of the Russian Cardiology Research and Production Complex (Protocol no. 08 dated August 12, 2015), the Ethics Committee of the Medical Center for Diagnostics and Prevention Plus (Protocol no. 04 dated September 8, 2015), and the Ethics Committee of the Nizhny Novgorod Medical Clinic (Protocol no. 06 dated August 1, 2015). Informed consent was obtained from all subjects participated in this study.

CONFLICT OF INTEREST

The authors declare no conflicts of interest.

REFERENCES

1. *Triglyceride Coronary Disease Genetics Consortium and Emerging Risk Factors Collaboration; Sarwar N., Sandhu M.S., Ricketts S.L., Butterworth A.S., di Angelantonio E., Boekholdt S.M., Ouwehand W., Watkins H., Samani N.J., Saleheen D., Lawlor D., Reilly M.P., Hingorani A.D., Talmud P.J., Danesh J.* (2010) Triglyceride-mediated pathways and coronary disease: collaborative analysis of 101 studies. Lancet, **375**(9726), 1634–1639. DOI: 10.1016/S0140-6736(10)60545-4
2. *Do R., Willer C.J., Schmidt E.M., Sengupta S., Gao C., Peloso G.M., Gustafsson S., Kanoni S., Ganna A., Chen J., Buchkovich M.L., Mora S., Beckmann J.S., Bragg-Gresham J.L., Chang H.-Y., Demirkan A., den Hertog H.M., Donnelly L.A., Ehret G.B., Esko T., et al.* (2013) Common variants associated with plasma triglycerides and risk for coronary artery disease. Nat. Genet., **45**(11), 1345–1352. DOI: 10.1038/ng.2795
3. *Miller M., Stone N.J., Ballantyne C., Bittner V., Criqui M., Ginsberg H.N., Goldberg A.C., Howard W.J., Jacobson M.S., Kris-Etherton P.M., Lennie T.A., Levi M., Mazzone T., Pennathur S.; American Heart Association Clinical Lipidology, Thrombosis, and Prevention Committee of the Council on Nutrition, Physical Activity, and Metabolism; Council on Arteriosclerosis, Thrombosis and Vascular Biology, Council on Cardiovascular Nursing; Council on the Kidney in Cardiovascular Disease* (2011) Triglycerides and cardiovascular disease: a scientific statement from the American Heart Association. Circulation, **123**(20), 2292–2333. DOI: 10.1161/CIR.0b013e3182160726
4. *Marston N.A., Giugliano R.P., Im K., Silverman M.G., O'Donoghue M.L., Wiviott S.D., Ference B.A., Sabatine M.S.* (2019) Association between triglyceride lowering and reduction of cardiovascular risk across multiple lipid-lowering therapeutic classes: a systematic review and meta-regression analysis of randomized controlled trials. Circulation, **140**(16), 1308–1317. DOI: 10.1161/CIRCULATIONAHA.119.041998
5. *Ouimet M., Barrett T.J., Fisher E.A.* (2019) HDL and reverse cholesterol transport. Circ. Res., **124**(10), 1505–1518. DOI: 10.1161/CIRCRESAHA.119.312617
6. *Rader D.J., Alexander E.T., Weibel G.L., Billheimer J., Rothblat G.H.* (2009) The role of reverse cholesterol transport in animals and humans and relationship to atherosclerosis. J. Lipid Res., **50**(Suppl), S189–S194. DOI: 10.1194/jlr.R800088-JLR200
7. *Rohatgi A., Khera A., Berry J.D., Givens E.G., Ayers C.R., Wedin K.E., Neeland I.J., Yuhanna I.S., Rader D.R., de Lemos J.A., Shaul P.W.* (2014) HDL cholesterol efflux capacity and incident cardiovascular events. N. Engl. J. Med., **371**(25), 2383–2393. DOI: 10.1056/NEJMoa1409065
8. *Archakov A.I., Guseva M.K., Uchaikin V.F., Tikhonova E.G., Ipatova O.M.* (2014) Medical forms of phospholipid preparations and methods for their preparation. United States Patent No. US8680061B2. Retrieved from https://patents.google.com/patent/US8680061B2/en
9. *Lokhov P.G., Maslov D.L., Balashova E.E., Trifonova O.P., Medvedeva N.V., Torkhovskaya T.I., Ipatova O.M., Archakov A.I., Malyshev P.P., Kukharchuk V.V., Shestakova E.A., Shestakova M.V., Dedov I.I.* (2015) Mass spectrometry analysis of blood plasma lipidome as method of disease diagnostics, evuation of effectiveness and optimization of drug therapy. Biomeditsinskaya Khimiya, **61**(1), 7–18. DOI: 10.18097/PBMC20156101007
10. *Kudinov V.A., Torkhovskaya T.I., Zakharova T.S., Morozevich G.E., Artyushev R.I., Zubareva M.Yu., Markin S.S.* (2021) High-density lipoprotein remodeling by phospholipid nanoparticles improves cholesterol efflux capacity and protects from atherosclerosis. Biomed. Pharmacother., **141**, 111900. DOI: 10.1016/j.biopha.2021.111900
11. *Archakov A.I., Guseva M.K., Uchaikin V.F., Ipatova O.M., Tikhonova E.G., Shironin A.V.* (2010) Nanoform of phospholipid preparation for oral use (sashet) and method of its obtaining (versions). Russian Patent No. RU2463057C2. Retrieved from: https://patents.google.com/patent/RU2463057C2/ru
12. *Tikhonova E.G., Sanzhakov M.A., Tereshkina Yu.A., Kostryukova L.V., Khudoklinova Yu.Yu., Orlova N.A., Bobrova D.V., Ipatova O.M.* (2022) Drug transport system based on phospholipid nanoparticles: production technology and characteristics. Pharmaceutics, **14**(11), 2522. DOI: 10.3390/pharmaceutics14112522
13. *Zubareva M., Kudinov V., Malyshev P., Rozhkova T., Markin S., Kukharchuk V.* (2020) Usage of a new pharmacological agent of ultra-small phospholipid nanoparticles (micelles) in the treatment of combined hyperlipidemia. Eur. Heart J., **41**(Suppl.2), ehaa946.3007. DOI: 10.1093/ehjci/ehaa946.3007
14. *Robinson J.G., Wang S., Smith B.J., Jacobson T.A.* (2009) Meta-analysis of the relationship between non-high-density lipoprotein cholesterol reduction and coronary heart disease risk. J. Am. Coll. Cardiol., **53**(4), 316–322. DOI: 10.1016/j.jacc.2008.10.024
15. *Markin S.S., Kuharchuk V.V., Lisitsa A.V., Ponomarenko E.A., Romashova Y.A., Pleshakova T.O., Ipatova O.M., Tikhonova E.G., Guseva M.K., Kutsenko V.A., Ivanov S.V., Yarovaya E.B., Zubareva M.Y., Beregovykh V.V., Archakov A.I.* (2025) Water soluble peroral pharmaceutical form of phosphatidylcholine Vitaphospholip®: experimental and clinical data in combined hyperlipidemia. Annals of the Russian Academy of Medical Sciences, **80**(1), 42–48. DOI: 10.15690/vramn18026

16. *Tardif J.-C., Karwatowska-Prokopczuk E., Amour E.S., Ballantyne C.M., Shapiro M.D., Moriarty P.M., Baum S.J., Hurh E., Bartlett V.J., Kingsbury J., Figueroa A.L., Alexander V.J., Tami J., Witztum J.L., Geary R.S., O'Dea L.S.L., Tsimikas S., Gaudet D.* (2022) Apolipoprotein C-III reduction in subjects with moderate hypertriglyceridaemia and at high cardiovascular risk. Eur. Heart J., **43**(14), 1401–1412. DOI: 10.1093/eurheartj/ehab820
17. *Nicholls S.J., Lincoff A.M., Garcia M., Bash D., Ballantyne C.M., Barter P.J., Davidson M.H., Kastelein J.J.P., Koenig W., McGuire D.K., Mozaffarian D., Ridker P.M., Ray K.K., Katona B.G., Himmelmann A., Loss L.E., Rensfeldt M., Lundström T., Agrawal R., Menon V., Wolski K., Nissen S.E.* (2020) Effect of high-dose omega-3 fatty acids vs corn oil on major adverse cardiovascular events in patients at high cardiovascular risk: the STRENGTH randomized clinical trial. JAMA, **324**(22), 2268–2280. DOI: 10.1001/jama.2020.22258
18. *Duan Y., Gong K., Xu S., Zhang F., Meng X., Han J.* (2022) Regulation of cholesterol homeostasis in health and diseases: from mechanisms to targeted therapeutics. Signal Transduct. Target. Ther., **7**, 265. DOI: 10.1038/s41392-022-01125-5
19. *Nicholls S.J., Puri R., Ballantyne C.M., Jukema J.W., Kastelein J.J.P., Koenig W., Wright R.S., Kallend D., Wijngaard P., Borgman M., Wolski K., Nissen S.E.* (2018) Effect of infusion of high-density lipoprotein mimetic containing recombinant apolipoprotein A-I Milano on coronary disease in patients with an acute coronary syndrome in the MILANO-PILOT trial: a randomized clinical trial. JAMA Cardiology, **3**(9), 806–814. DOI: 10.1001/jamacardio.2018.2112
20. *Nicholls S.J., Andrews J., Kastelein J.J.P., Merkely B., Nissen S.E., Ray K.K., Schwartz G.G., Worthley S.G., Keyserling C., Dasseux J.-L., Griffith L., Kim S.W., Janssan A., di Giovanni G., Pisaniello A.D., Scherer D.J., Psaltis P.J., Butters J.* (2018) Effect of serial infusions of CER-001, a pre-β high-density lipoprotein mimetic, on coronary atherosclerosis in patients following acute coronary syndromes in the CER-001 atherosclerosis regression a cute coronary syndrome trial: a randomized clinical trial. JAMA Cardiology, **3**(9), 815–822. DOI: 10.1001/jamacardio.2018.2121
21. *Tardif J.-C., Grégoire J., L'Allier P.L., Ibrahim R., Lespérance J., Heinonen T.M., Kouz S., Berry C., Basser R., Lavoie M.-A., Guertin M.-C., Rodés-Cabau J.; Effect of rHDL on Atherosclerosis-Safety and Efficacy (ERASE) Investigators* (2007) Effects of reconstituted high-density lipoprotein infusions on coronary atherosclerosis: a randomized controlled trial. JAMA, **297**(15), 1675–1682. DOI: 10.1001/jama.297.15.jpc70004
22. *Nissen S.E., Tsunoda T., Tuzcu E.M., Schoenhagen P., Cooper C.J., Yasin M., Eaton G.M., Lauer M.A., Sheldon W.S., Grines C.L., Halpern S., Crowe T., Blankenship J.C., Kerensky R.* (2003) Effect of recombinant ApoA-I Milano on coronary atherosclerosis in patients with acute coronary syndromes: a randomized controlled trial. JAMA, **290**(17), 2292–2300. DOI: 10.1001/jama.290.17.2292
23. *Day C.* (1975) Obstacles to the clinical investigation of phospholipids. Artery, **1**, 1–2.
24. *Tchoua U., Gillard B.K., Pownall H.J.* (2010) HDL superphospholipidation enhances key steps in reverse cholesterol transport. Atherosclerosis, **209**(2), 430–435. DOI: 10.1016/j.atherosclerosis.2009.10.002
25. *Paavola T., Kuusisto S., Jauhiainen M., Kakko S., Kangas-Kontio T., Metso J., Soininen P., Ala-Korpela M., Bloigu R., Hannuksela M.L., Savolainen M.J., Salonurmi T.* (2017) Impaired HDL2-mediated cholesterol efflux is associated with metabolic syndrome in families with early onset coronary heart disease and low HDL-cholesterol level. PLOS One, **12**(2), e0171993. DOI: 10.1371/journal.pone.0171993
26. *Luo Y., Guo Y., Wang H., Yu M., Hong K., Li D., Li R., Wen B., Hu D., Chang L., Zhang J., Yang B., Sun D., Schwendeman A.S., Chen E.Y.* (2021) Phospholipid nanoparticles: therapeutic potentials against atherosclerosis via reducing cholesterol crystals and inhibiting inflammation. EBioMedicine, **74**, 103725. DOI: 10.1016/j.ebiom.2021.103725
27. *Meikle P.J., Formosa M.F., Mellett N.A., Jayawardana K.S., Giles C., Bertovic D.A., Jennings G.L., Childs W., Reddy M., Carey A.L., Baradi A., Nanayakkara S., Wilson A.M., Duffy S.J., Kingwell B.A.* (2019) HDL phospholipids, but not cholesterol distinguish acute coronary syndrome from stable coronary artery disease. J. Am. Heart Assoc., **8**(11), e011792. DOI: 10.1161/JAHA.118.011792
28. *Archakov A.I., Kukharchuk V.V., Lisitsa A.V., Ponomarenko E.A., Romashova Yu.A., Pleshakova T.O., Yarovaya E.B., Kutsenko V.A., Guseva M.K., Beregovykh V.V., Ipatova O.M., Zubareva M.K., Tikhonova E.G, Ivanov S.V., Bedretdinov F.N., Markin S.S.* (2024) Ultra-small phospholipid nanoparticles in the treatment of combined hyperlipidemia: a randomized placebo-controlled clinical trial. Res. Pharm. Sci., **19**(6), 656–668. DOI: 10.4103/RPS.RPS_274_23

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ЛЕКАРСТВЕННЫЙ ПРЕПАРАТ ВИТАФОСФОЛИП® (ВОДОРАСТВОРИМАЯ ЛЕКАРСТВЕННАЯ ФОРМА ФОСФАТИДИЛХОЛИНА) В ЛЕЧЕНИИ КОМБИНИРОВАННОЙ ГИПЕРЛИПИДЕМИИ: РАНДОМИЗИРОВАННОЕ ПЛАЦЕБО-КОНТРОЛИРУЕМОЕ КЛИНИЧЕСКОЕ ИССЛЕДОВАНИЕ

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Представлены результаты клинического исследования лекарственного препарата Витафосфолип® (водорастворимая форма фосфатидилхолина), направленного на снижение уровней холестерина не-ЛПВП (ХС не-ЛПВП) и триглицеридов (ТГ) у пациентов с комбинированной гиперлипидемией. В рандомизированное двойное слепое плацебо-контролируемое исследование было включено 100 пациентов. Витафосфолип® или плацебо вводили перорально по 500 мг 2 раза в день в течение 12 недель. Лечение препаратом Витафосфолип® привело к снижению ХС не-ЛПВП на 13,2% по сравнению с 4,3% в группе плацебо (p = 0,001). Абсолютное снижение ХС не-ЛПВП составило -0,6 ммоль/л по сравнению с -0,2 ммоль/л в группе плацебо (p = 0,001). Целевой уровень ХС не-ЛПВП менее 3,4 ммоль/л был достигнут у 15 из 39 пациентов (38,5%) в группе Витафосфолип® против 2 из 41 пациентов (4,9%) в группе плацебо (p = 0,000). Абсолютное снижение ТГ в группе пациентов, получавших Витафосфолип® составило -0,7 ммоль/л в сравнении с -0,1 ммоль/л в группе плацебо (p = 0,001). На фоне терапии препаратом Витафосфолип® отмечено значительное снижение уровней аполипопротеина В, общего ХС, холестерина липопротеинов очень низкой плотности. Изменений в функции печени, почек, жизненно важных показателях и ЭКГ не зарегистрировано. Серьёзных нежелательных явлений не выявлено. Таким образом, было установлено, что Витафосфолип® значительно снижает уровень ХС не-ЛПВП, ТГ и атерогенных липопротеинов у пациентов с комбинированной гиперлипидемией и умеренным сердечно-сосудистым риском.

Полный текст статьи на русском языке доступен на сайте журнала (http://pbmc.ibmc.msk.ru).

Ключевые слова: Витафосфолип®; водорастворимая форма фосфатидилхолина; комбинированная гиперлипидемия; холестерин не-ЛПВП; триглицериды

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