

# Impact of phototherapy on selected lipid metabolism indices and oxidation markers in patients with psoriasis vulgaris

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Post Dermatol Alergol 2011; XXVIII, 2: 83–91

## Abstract

**Introduction:** Dyslipidaemia occurring concomitantly with psoriasis and proatherogenic serum lipid profile have been observed in multiple studies of middle-aged subjects. Modifications of lipid concentration indices are associated with increased risk of atherosclerosis observed in the course of psoriasis.

**Aim:** To evaluate the impact of phototherapy, taking into consideration its types, on concentrations of selected lipid profile indices, oxidation markers and antioxidant defence of patients with psoriasis vulgaris.

**Material and methods:** The studies included a group of 60 patients with diagnosed psoriasis vulgaris, which for statistical purposes was provisionally divided into three groups of 20 patients each, depending on the type of phototherapy used. All patients underwent 10 therapies using one of three phototherapy methods (UVA, UVB, UVB 311).

**Results:** No significant impact of any of the phototherapy types on concentrations of studied lipid indices was observed. Within the entire study group, phototherapy led to increase of total cholesterol and LDL cholesterol concentrations, but no intensification of oxidation processes was observed after its completion.

**Conclusions:** On the basis of the obtained results it can be concluded that a treatment cycle consisting of ten UV radiation therapies is safe for patients and it does not increase the risk of atherosclerosis in middle-aged psoriatic patients.

**Key words:** psoriasis, phototherapy, oxidatively modified low-density lipoprotein, autoantibodies against oxLDL, ferric reducing ability of plasma.

## Introduction

Psoriasis is an inflammatory skin disease characterized by significant keratinocyte proliferation, combined with increased angiogenesis of skin vessels, activation of fibroblasts, leucocyte infiltration, changes in eicosanoid metabolism and increased production of some cytokines [1, 2].

It is believed that lipid metabolism irregularities play an important role in pathogenesis of psoriasis, and psoriatic patients may have increased risk of developing ischaemic heart disease [3]. Simultaneously, studies demonstrate that male psoriatic patients are predis-

posed to developing atherosclerosis and arterial embolism [3, 4].

Modifications of lipid concentrations observed in psoriasis may be associated with increased risk of atherosclerosis observed in middle-aged patients [3, 5]. This predisposition appears to be associated with intensification of disease symptoms [4-7]. Compared to the healthy population, components of metabolic syndrome occur more frequently in psoriatic patients, increasing the risk of developing cardiovascular diseases in those patients. Chronic inflammation is the common base for these disorders [8, 9]. For many years there have been observed

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links of psoriasis with cardiovascular diseases, such as atherosclerosis, hypertension and irregularities of the heart valves [10-13]. It has been well known for many years that the risk of atherosclerosis and coronary heart disease is associated with increased serum total cholesterol and LDL cholesterol concentrations, and is negatively correlated with HDL cholesterol concentrations [5, 11, 12, 14]. The results of lipid profile assessment are quite frequently ambiguous and indicate increased, decreased or normal concentrations of individual indices [3-6, 11, 12, 15-18]. Due to the chronic inflammatory condition, changes in lipid profile become proatherogenic [3].

Oxidatively modified LDLs (oxLDLs) are produced as a result of LDL exposure to oxidative stress [19], because inflammatory processes and sustained oxidative stress in psoriasis induce production of free radicals [6, 20, 21]. Oxidatively modified LDLs are capable of inducing an inflammatory condition which affects endothelial cell adhesion and oxidative potential of vascular wall cells, thus affecting development of early atherosclerosis [15, 22]. According to recent studies, oxidative modification of lipoproteins, in particular low-density lipoproteins (LDLs), induces development and progress of atherosclerosis more effectively than native lipoproteins [23].

Oxidatively modified lipoproteins (oxLDLs) are immunogenic in humans. After oxidation changes their physiochemical properties and not being recognized by receptors for native LDL they become recognized as non-self antigens [24]. It is believed that in atherosclerosis, oxLDLs and heat shock proteins are the main antigens presented by macrophages to T cells, thus being stimulators of the immune response, which is confirmed by the fact that anti-oxLDL antibodies are detected in atherosclerosis [25, 26]. The concentration of anti-oxLDL antibodies provides indirect evidence of the importance of oxLDLs in development of atherosclerosis-based cardiovascular diseases [15], and may also be used as a useful indicator of in-vivo oxidation [27-29].

Long-term exposure to oxidative stress leads to depletion of immune mechanisms. Due to reduction, and in consequence of insufficient antioxidant potential of the organisms, the balance between oxidative and antioxidative processes tends to shift towards a pro-oxidant state and development of atherosclerosis [22]. The current antioxidative state of the organism can be assessed by determination of ferric reducing ability of plasma (FRAP) [30, 31].

Phototherapy has been used as a treatment of psoriasis for many years. UVA radiation absorbed by chromophores stimulates photo-oxidation reactions, leading to production of reactive oxygen species, which in turn oxidize proteins and lipids [32]. Moreover, UVA radiation induces delayed apoptosis, by creating singlet oxygen ( $^1O_2$ ), superoxide anion radical ( $O_2^-$ ) and other reactive oxygen species [33-35]. This may lead to membrane lipid peroxidation – phospholipids and cholesterol and mito-

chondrial cardiolipin [32]. The mechanism of UVB action consists in alternating cell membrane permeability, increasing lipid peroxidation and decreasing DNA biosynthesis and cell mitotic activity [6, 32]. In the context of numerous reports indicating concomitant occurrence of dyslipidaemia in psoriasis, attention should be paid to the potential proatherogenic potential of phototherapy.

## Aim

The study aimed to investigate the impact of phototherapy and its individual types on selected lipid profile indices, oxidation markers and antioxidant ability of plasma in middle-aged patients with psoriasis vulgaris.

## Material and methods

### Study group

The study included 60 patients with diagnosed psoriasis vulgaris, hospitalized due to disease exacerbation, aged between 18 years and 60 years (mean age 40.0  $\pm$  11.9), with mean body mass index (BMI) of 22.6  $\pm$  4.5 kg/m<sup>2</sup> (17.3-39.0). The calculated psoriasis area and severity index (PASI) score was within the range of 5.4-49.2, mean value of 22.7  $\pm$  9.3. Prior to starting the study the patients were treated with external preparations only. The type of administered phototherapy treatment was the criterion for dividing the patients into 3 groups of 20 patients each:

- group I included patients exposed to UVA radiation within the wavelength of 320-400 nm, with the Waldmann 1000 UV cabin. The patients received 10 exposures, achieving the total dose of 11.25 J/cm<sup>2</sup> (13 min 23 s), with constant intensity of 14 mW/cm<sup>2</sup>.
- group II included patients exposed to UVB radiation within the wavelength of 290-320 nm, emitted by a PSORILUX 3070 lamp. As a result, after 10 exposures the dose of energy absorbed by the patients was 0.66 J/cm<sup>2</sup>.
- group III included patients exposed to narrow band UVB radiation (311 nm). Exposures were conducted in a GP-42 cabin, equipped with 26 TL-01 torches. The average UV radiation intensity on the skin surface was 12 mW/cm<sup>2</sup>. After 10 exposures, the achieved total photobiological dose was 0.334 J/cm<sup>2</sup>.

### Studied material

Blood serum was the studied material. Samples of peripheral blood for biochemical assays were collected three times, each sample of ca. 10 ml, after a 12-h low-lipid diet period:

- 1) at the time of study initiation and qualification for phototherapy (prior to the first phototherapy exposure);
- 2) after 10 phototherapy exposures (i.e. after ca. 14 days);

3) 3 months after treatment completion (phototherapy).

Blood samples were collected from antecubital veins, in the morning and under fasting conditions. The serum obtained after centrifugation was stored at a temperature of  $-70^{\circ}\text{C}$  until assays were performed.

### Methods of biochemical assays

Levels of lipid metabolism parameters (serum total cholesterol, triglycerides, HDL and LDL cholesterol) were assayed by calorimetric methods routinely used in laboratory diagnostics, using Randox reagent kits (UK). Levels of HDL and LDL cholesterol were determined by direct methods, which are not affected by increased level of triglycerides.

Total FRAP was assayed in the serum according to the method of Benzie *et al.*, based on assessment of Fe (III) ion reduction ability exhibited by non-enzymatic antioxidants contained in the studied plasma or serum. The number of produced  $\text{Fe}^{2+}$  was then assayed in reaction with TPTZ (tripyriddy-l-s-triazine). The results of FRAP assays were given per Trolox equivalent – synthetic vitamin E analogue – used in the FRAP test as the standard substance [31].

Concentration of anti-oxLDL IgG antibodies was assayed by the ELISA method, using Biomedica Medizinprodukte GmbH & Co KG OLAB kits (Austria). Similar concentrations of oxidized low-density lipoproteins (oxLDL) were assayed by the ELISA method, using Mercodia Oxidized LDL, ELISA reagent kit (Mercodia AB, Sweden).

Table 1 presents standard values for the studied lipid profile indices used in the analysis, recommended in primary prevention of ischaemic heart disease, determined on the basis of the guidelines included in the ATP III report (Adult Treatment Panel III – ATP III), presented by a team of experts of the National Cholesterol Education Program (NCEP) [36, 37]. For FRAP, limit values proposed by Benzie and Strain were adopted [30].

### Statistical analysis

Assay results of the studied indices were presented using basic descriptive statistical parameters, such as: mean value, standard deviation, median, lower and upper quartile, minimum and maximum values.

Consistency of variables' distribution with a normal distribution was evaluated using Kolmogorov-Smirnov and Shapiro-Wilk tests. Comparisons between groups were made using Mann-Whitney U test and Kruskal-Wallis one-way analysis of variance, and Wilcoxon signed-rank test was used to compare associated variables within studied groups. To assess relations between studied indices with a distribution different from a standard normal distribution, Spearman's rank correlation coefficient was calculated. A level of  $p < 0.05$  was assumed as statistically significant. Calculations were made using Statistica PI software.

**Tab. 1.** Reference values adopted for the studied indices

Indices	Reference range
Total cholesterol	< 200 mg/dl*
Triglycerides	< 200 mg/dl*
HDL cholesterol	< 60 mg/dl*
LDL cholesterol	< 130 mg/dl*
FRAP	M: 354–732 $\mu\text{mol/l}^{**}$
anti-oxLDL IgG Abs	–
oxLDL	–

\*Values recommended in primary prevention of ischaemic heart disease, \*\*acc. to Benzie and Strain [30], M – males

## Results

### Impact of individual phototherapy types on lipid metabolism indices, oxidation markers and the sum of antioxidants

The first stage of the study involved performance of basic calculations concerning descriptive statistics and assessment of variables' distribution in individual study groups, depending on the applied UV wavelength range (UVA: 320-400 nm, UVB: 290-320 nm, UVB 311 nm). For most variables, the observed distribution was different from a standard distribution.

We analysed the pattern of changes induced by phototherapy, with reference to lipid metabolism indices, oxidation markers and the sum of antioxidants. Tables 2 and 3 present a summary of the results for individual phototherapy types. No significant impact of any of the phototherapy types on concentrations of the above-specified indices was observed (Kruskal-Wallis one-way analysis of variance for concentrations of cholesterol, LDL, HDL, triglycerides and FRAP, and Wilcoxon signed-rank test for concentrations of oxLDL and anti-oxLDL antibodies).

### Impact of phototherapy on lipid metabolism indices, oxidation markers and the sum of antioxidants in the entire study population

As no differences were observed between individual phototherapy types, it was assumed that the number of subjects in the groups could be insufficient to demonstrate a significant impact of the administered treatment. That is why all groups were combined into one group and further analysis was performed cumulatively ( $n = 60$ ). Table 4 presents a summary of the cumulative analysis results.

Concentrations of total cholesterol and LDL cholesterol observed in the entire group after completion of phototherapy were significantly higher; prior to starting the treatment the median value for total cholesterol was 192.4 mg/dl and 200.0 mg/dl after 14 days, and for LDL cholesterol 126.5 mg/dl prior to starting the treatment and 130.0

**Tab. 2.** Impact of phototherapy on lipid indices and the sum of antioxidants in individual study groups, depending on administered type of phototherapy, and in the group involving the entire studied population, created after combining individual study groups. The table presents median values, and lower and upper quartile (25-75%)

Group	Day of study	Cholesterol [mg/dl]	HDL [mg/dl]	LDL [mg/dl]	Triglycerides [mg/dl]	FRAP [μmol/l]
UVA (n = 20)	1	197.7 187.0-216.0	46.6 43.3-54.5	159.5 114.5-174.5	166.9 131.5-199.5	599.0 508.5-683.5
	14	217.5 186.0-256.5	47.8 44.2-50.1	160.0 124.0-183.5	196.5 133.5-231.0	617.0 544.0-680.0
	90	219.0 186.0-259.0	45.9 44.1-48.2	158.0 119.0-181.0	191.0 156.0-264.5	605.5 510.0-677.0
UVB (n = 20)	1	197.5 182.5-221.0	49.0 43.7-54.5	139.0 114.0-163.0	152.0 90.0-174.0	526.5 491.0-562.0
	14	202.0 187.5-233.5	46.5 43.5-57.0	152.0 115.0-160.5	149.5 98.0-187.5	513.5 487.5-573.5
	90	204.0 195.0-232.0	45.9 43.5-56.0	144.5 111.5-157.5	147.5 109.5-180.0	530.0 475.5-563.5
UVB 311 nm (n = 20)	1	166.0 154.0-203.5	46.8 44.0-50.3	99.0 92.5-134.0	143.5 130.5-191.0	579.5 495.0-633.0
	14	172.0 158.0-201.0	44.5 42.0-50.5	103.5 98.0-134.0	134.5 112.0-180.0	573.0 487.5-652.5
	90	183.5 156.0-200.0	44.8 42.3-49.3	107.0 100.5-133.5	131.0 107.5-161.5	572.5 507.5-656.5
After combining the groups (n = 60)	1	192.3 168.0-205.5	47.6 43.7-53.3	126.5 101.0-165.0	147.9 122.2-196.5	549.5 497.5-632.0
	14	200.0 171.5-233.5	46.5 43.5-51.3	130.0 104.5-170.0	156.5 120.0-198.5	567.5 505.5-652.5
	90	200.5 179.5-230.0	45.5 43.0-49.6	130.5 108.5-160.5	159.0 118.5-198.5	554.5 500.5-649.5

mg/dl after treatment completion. The observed increase of total cholesterol concentration after 90 days (to 200.5 mg/dl) was also significant compared to its baseline values. Compared to baseline values, after 90 days of treatment the observed HDL cholesterol concentration decreased significantly, from 47.6 mg/dl to 45.5 mg/dl. No significant impact of phototherapy on triglyceride concentration was observed.

Analysis of total FRAP demonstrated a significant decrease in concentrations between the 14<sup>th</sup> (567.5 μmol/l) and 90<sup>th</sup> days of the study (554.5 μmol/l).

Concentration of oxidatively modified LDLs (oxLDLs) was significantly lower directly after phototherapy completion; its value prior to starting the treatment was 65.0 U/l (28.4-196.0 U/l), and 52.6 U/l (21.0-170.0 U/l) after 14 days of phototherapy exposure. Assays for antibodies against oxLDL were performed prior to starting phototherapy and 90 days after its completion, yet no significant differences in their concentrations were observed: 662.5 mU/ml (135.0-5601.0 mU/ml) prior to and 728.0 mU/ml (182.0-5950.0 mU/ml) after treatment.

Statistical analysis of the achieved results is summarized in Tab. 5.

Correlation analysis demonstrated the existence of a significant but weak inverse relation between concentration of oxidatively modified LDL (oxLDL) and concentration of anti-oxLDL antibodies prior to starting the treatment (Tab. 6, Fig. 1). A weak correlation was also observed between body mass index (BMI) and the sum of antioxidants (FRAP) prior to treatment ( $r = 0.28$ ,  $p = 0.03$ ) and directly after phototherapy ( $r = 0.27$ ,  $p = 0.04$ ); a weak reverse correlation between oxLDL concentration and FRAP concentration was observed only after 14 days of study ( $r = -0.28$ ,  $p = 0.03$ ).

## Discussion

Skin is constantly exposed to oxidative stress induced by reactive oxygen species (ROS). A ROS are produced both by endogenous sources, resulting from enzymatic activity or activation of neutrophilic granulocytes, and due to external stimuli, such as UV radiation [20]. The

**Tab. 3.** Characteristics of the studied groups and impact of phototherapy on concentration of oxLDL and anti-oxLDL Abs in individual groups receiving phototherapy treatment and in the entire study population. The table presents median values, and lower and upper quartile (25-75%)

Group	Age of subjects [years]	Duration of disease [years]	PASI	BMI [kg/m <sup>2</sup> ]	Day of study	oxLDL [U/l]	Day of study	Anti-oxLDL Ab [mU/ml]
UVA (n = 20)	45.0 28.0-51.0	24.0 7.0-28.0	21.5 12.9-28.4	29.0 23.0-30.7	1	59.4 43.2-94.5	1	566.0 336.0-1390.0
					14	51.4 45.9-70.1	90	1125.5 380.0-1682.5
UVB (n = 20)	39.0 28.5-45.0	11.5 6.0-19.0	25.2 20.4-28.9	25.3 22.0-26.5	1	68.5 38.7-95.5	1	874.0 460.5-1828.5
					14	49.6 41.5-75.0	90	728.0 485.0-1179.0
UVB 311 nm (n = 20)	38.5 31.0-53.0	9.5 1.5-15.0	20.9 13.8-30.7	25.9 24.8-31.1	1	65.2 52.4-116.0	1	584.5 360.0-1312.0
					14	62.7 48.9-90.3	90	641.0 419.0-1125.0
After combining the groups (n = 60)	39.5 28.5-50.0	12.0 5.0-21.0	22.3 15.1-30.2	26.0 23.3-30.0	1	65.0 43.4-101.2	1	662.5 388.5-1390.0
					14	52.6 43.4-74.3	90	728.0 419.0-1261.0

mechanism of UVB action consists in alternating cell membrane permeability, increasing lipid peroxidation and decreasing DNA biosynthesis and cell mitotic activity. Although UVA radiation is not directly absorbed by proteins and DNA, as it is in the case of UVB radiation, indirect effects of its action may be equally significant. UVA radiation absorbed by chromophores in the cell stimulates photo-oxidation reactions leading to production of reactive oxygen species, which in turn oxidize proteins and lipids [32].

None of the phototherapy methods used in individual groups of patients with psoriasis vulgaris produced statistically significant changes in lipid indices, compared to baseline concentrations of those parameters. No significant differences were observed in concentrations of FRAP, oxLDLs and anti-oxLDL Abs after exposure to different UV radiation wavelength ranges (UVA, UVB, UVB 311 nm) and after treatment completion. The presented data indicate that individual phototherapy methods do not differ in their impact on the studied indices.

However, within the entire studied population of patients who received phototherapy, without taking into consideration its individual types ( $n = 60$ ), there were some differences (Tab. 6). A statistically significant reduction in concentration of oxidatively modified LDL was observed after phototherapy completion. Along with a reduction of oxLDL concentration, after completion of phototherapy exposures, an increase in concentration of

the sum of antioxidants was observed. Although it was not a statistically significant increase, after 90 days there was observed a statistically significant reduction in FRAP concentration, compared to the values observed after 14 days. This was accompanied by a weak, yet statistically significant, inverse relation between FRAP concentration and oxLDLs concentration after phototherapy completion ( $r = -0.28, p = 0.03$ ). There was also observed a weak relation between BMI and activity of the sum of serum antioxidants both prior to the beginning of the study ( $r = 0.28, p < 0.05$ ), and after phototherapy cycles ( $r = 0.27, p < 0.05$ ). Concentration of anti-oxLDL Abs did not change. However, with reference to the studied lipid indices, after completion of phototherapy exposures there was a statistically significant increase in total cholesterol concentration and LDL cholesterol concentration in the entire group of patients. After 90 days, total cholesterol concentration was still significantly increased, compared to its baseline values, and furthermore there was observed a statistically significant decrease in HDL cholesterol concentration, as compared to baseline values. There was observed no significant difference in the remaining lipid profile parameters.

It appears that oxidative stress may play an important role in mechanisms related to treatment of psoriasis and adverse effects of some antipsoriatic agents. So far, only a few researchers have paid attention to this issue. The oxidative effect of anthralin used in topical treatment

**Tab. 4.** Descriptive statistics of assayed indices in the entire study population ( $n = 60$ )

	Median	Minimum	Maximum	Lower and upper quartile (25-75%)	Mean $\pm$ SD
Age of subjects [years]	39.5	18.0	60.0	28.5-50.0	40.0 $\pm$ 11.9
PASI*	22.3	5.4	49.2	15.1-30.2	22.7 $\pm$ 9.3
BMI*	26.0	17.3	39.0	23.3-30.0	22.6 $\pm$ 4.5
CH 1 [mg/dl]*	192.4	131.0	271.0	168.0-205.5	192.7 $\pm$ 32.3
CH 2 [mg/dl]*	200.0	136.0	316.0	171.5-233.5	203.1 $\pm$ 40.4
CH 3 [mg/dl]*	200.5	126.0	337.0	179.5-230.0	204.5 $\pm$ 41.5
TG 1 [mg/dl]	147.9	65.0	435.0	122.2-196.5	162.4 $\pm$ 66.5
TG 2 [mg/dl]	156.5	66.0	421.0	120.0-198.5	164.6 $\pm$ 67.6
TG 3 [mg/dl]	159.0	46.0	340.0	118.5-198.5	161.8 $\pm$ 64.6
HDL 1 [mg/dl]*	47.6	31.7	61.0	43.7-53.3	48.2 $\pm$ 6.5
HDL 2 [mg/dl]*	46.5	31.0	63.0	43.5-51.3	47.6 $\pm$ 6.4
HDL 3 [mg/dl]	45.5	38.0	63.7	43.0-49.6	47.3 $\pm$ 6.0
LDL 1 [mg/dl]	126.5	85.0	197.0	101.0-165.0	134.5 $\pm$ 34.4
LDL 2 [mg/dl]	130.0	80.0	201.0	104.5-170.0	137.0 $\pm$ 35.0
LDL 3 [mg/dl]*	130.5	63.0	217.0	108.5-160.5	136.3 $\pm$ 34.6
FRAP 1 [ $\mu$ mol/l]	549.5	393.0	970.0	497.5-632.0	571.4 $\pm$ 106.2
FRAP 2 [ $\mu$ mol/l]	567.5	371.0	1120.0	505.5-652.5	584.9 $\pm$ 119.3
FRAP 3 [ $\mu$ mol/l]	554.5	350.0	1113.0	500.5-649.5	573.9 $\pm$ 120.9
OxLDL 1 [U/l]	65.0	28.4	196.0	43.4-101.2	76.3 $\pm$ 38.4
OxLDL 2 [U/l]	52.6	21.0	170.0	43.35-74.3	62.4 $\pm$ 27.3
oxLDL abs 1 [mU/ml]	662.5	135.0	5601.0	388.5-1390.0	1025.3 $\pm$ 950.0
oxLDL abs 3 [mU/ml]	728.0	182.0	5950.0	419.0-1261.0	1074.7 $\pm$ 1094.8

1, 2, 3 – days of assays: 1 – prior to starting treatment, 2 – after 14 days of phototherapy exposures, 3 – after 90 days, \*distribution different from the standard normal distribution

**Tab. 5.** Impact of phototherapy on concentrations of lipid indices, FRAP, oxLDL and anti-oxLDL Abs in the entire study population ( $n = 60$ )

Studied indices	Difference between 1 <sup>st</sup> and 14 <sup>th</sup> day of study	Difference between 1 <sup>st</sup> and 90 <sup>th</sup> day of study	Difference between 14 <sup>th</sup> and 90 <sup>th</sup> day of study
Cholesterol	$\uparrow p < 0.05$	$\uparrow p < 0.05$	NS
HDL	NS	$\downarrow p < 0.05$	NS
LDL	$\uparrow p < 0.05$	NS	NS
Triglycerides	NS	NS	NS
FRAP	NS	NS	$\downarrow p < 0.05$
oxLDL	$\downarrow p < 0.05$	–	–
anti-oxLDL Abs	–	NS	–

of psoriasis has been described. The treatment uses a mechanism of action consisting in blocking keratinocyte proliferation and potentiating its immunosuppressive effect [38]. It is believed that anthralin stimulates ROS

production and generation of anthralin radicals in the skin [39]. Peus *et al.* [40] indicate that lipid peroxidation is the earliest response induced by anthralin, which leads to activation of c-Jun N-terminal kinase (JNK), a tran-

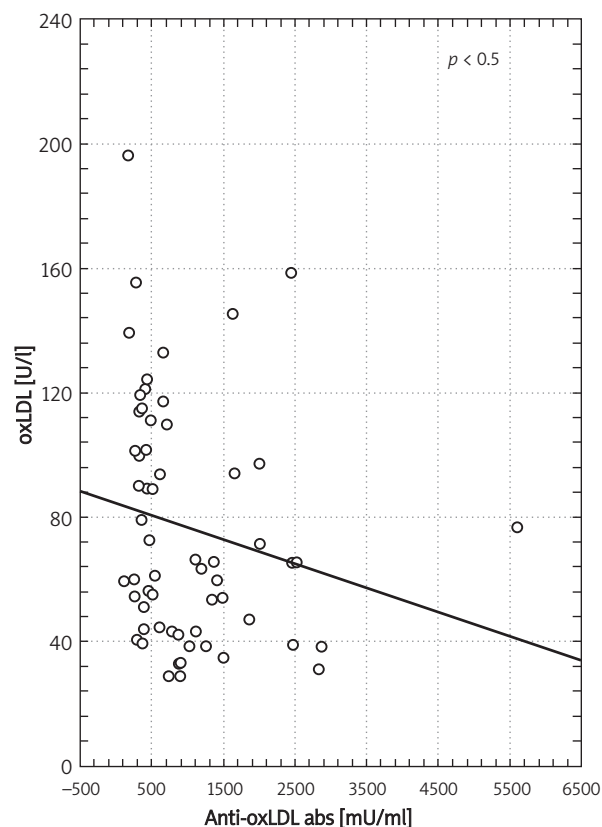
**Tab. 6.** Analysis of correlation between concentration of oxidatively modified LDLs (oxLDLs) and concentration of anti-oxLDL Abs in the serum prior to and after treatment ( $n = 60$ )

Studied relation	Spearman's rank correlation coefficient ( $r$ )	Level of significance ( $p$ )
oxLDL: anti-oxLDL Abs prior to treatment	-0.33	0.01
oxLDL: anti-oxLDL Abs after treatment (after 90 days)	0.15	NS

NS – statistically insignificant

scription factor responsible for regulation of cell proliferation and apoptosis. Unfortunately, the subject of the possible impact of phototherapy on the lipid oxidation mechanism in psoriasis vulgaris has not been broadly discussed in the literature so far. Coimbra *et al.* [41] evaluated the impact of various forms of psoriasis treatment – including external treatment and phototherapy – on lipid parameters, oxidation markers and inflammatory markers. Prior to starting treatment of psoriatic patients, proatherogenic lipid metabolism disturbances and a significant increase in CRP and oxLDL concentrations were observed, compared to the control group of healthy volunteers. After 12 weeks, oxLDL concentration in patients treated with narrow band UVB and in the PUVA group was significantly reduced, yet remained statistically higher than in the control group. Concentrations of lipid indices did not change significantly, but levels of inflammatory markers decreased. Observations made by the authors of the study indicate that treatment with narrow band UVB and PUVA is effective in reduction of inflammatory marker levels and oxidation indices, although low-grade inflammation could not be eliminated.

These results are similar to those achieved by us, albeit somewhat puzzling considering the previously described mechanism of action of individual UV wavelength ranges. They do not allow explicit confirmation of the hypothesis that the administered treatment method intensifies oxidative LDL modification processes; on the contrary, oxLDL concentration was significantly reduced (the trend to reduce concentrations of oxLDLs after phototherapy was observed in all studied groups, although the achieved differences were not always significant). After phototherapy, no significant increase of antioxidative defence nor significant stimulation of immune defence in the form of increased production of antibodies was observed. This could be due to insufficient duration of the administered phototherapy cycle, which was limited to 10 phototherapy exposure sessions. According to relevant guidelines, there should be 10-30 phototherapy exposure sessions within one phototherapy cycle [42, 43]. However, these guidelines refer only to the number of exposures required to achieve clinical improvement in the condition of patients. The duration of UV radiation exposure after which oxidation processes start to prevail remains unknown. The achieved results lead to the conclusion that a 10-exposure phototherapy cycle used in each of



**Fig. 1.** Inverse relation between concentration of oxidatively modified LDLs (oxLDLs) and concentration of anti-oxLDL antibodies prior to starting treatment

the phototherapy methods is safe and does not additionally increase risk of developing atherosclerosis in psoriatic patients.

## Conclusions

Although phototherapy treatment led to an increase in total cholesterol and LDL cholesterol concentrations in the entire studied group, no intensification of oxidation processes was observed after its completion. On the contrary, there was observed a significant reduction of oxLDLs concentration after phototherapy exposures, with no reaction of antibodies and oxidative potential, probably due to reduction of the inflammatory condition related to the therapeutic action of UV radiation.

Individual phototherapy methods do not demonstrate differences in their impact on the studied lipid indices, oxidation markers and the sum of antioxidants.

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