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Effects of UVB Treatment on Neutrophil Function in Psoriatic Patients and Healthy Subjects

ÅSA LUNDIN,[1] GERD MICHAËLSSON,[1] PER VENGE,[2] and BERIT BERNE[1]

Departments of [1]Dermatology and [2]Clinical Chemistry, University Hospital, Uppsala, Sweden

To evaluate if whole body UVB irradiation has effects on the neutrophil function, eleven patients with mild to moderate psoriasis and 14 healthy subjects were treated with whole body UVB irradiation 3 times weekly for 4 weeks. Eight healthy untreated subjects served as controls.

After 2 weeks of treatment the individual change of phagocytosis measured by the ingestion of IgG-coated particles was related to the pre-treatment value both in the psoriatic patients and healthy subjects ($p < 0.05$ and $p < 0.001$, respectively). Similar results were obtained for ingestion of IgG-C3b-coated particles. Thus, the change in phagocytic rate seemed to be dependent on the functional activity before treatment. UV irradiation of PMNs *in vitro* did not influence the phagocytic rate.

After 4 weeks of UV treatment the healthy subjects showed a significant decrease in the rate of phagocytosis of IgG-C3b particles ($p < 0.02$) and of the serum chemokinetic activity ($p < 0.01$). In the psoriatic patients the mean chemokinetic activity in heated sera was decreased after 2 weeks ($p < 0.02$). There appeared to be no relation between improvement of psoriasis and changes in PMN function.

In an untreated group of healthy subjects no significant changes in neutrophil function were found. The results indicate that there is a change in PMN function during UVB treatment. The degree of change seems to vary, not only between individuals but possibly also between groups, e.g. healthy subjects compared with psoriatic patients.

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Å. Lundin, Department of Dermatology, University Hospital, S-751 85 Uppsala, Sweden.

In recent years the role of neutrophils in the pathogenesis of psoriasis has often been in focus. Alterations in the chemotactic (1, 2) and phagocytotic (3) functions of the polymorphonuclear cells (PMNs) have been reported to occur in psoriasis, although in some studies no changes were found (4, 5). Sera from patients with psoriasis have also been found to show alterations in chemotactic and/or chemokinetic activity (1, 6).

The healing effect of ultraviolet B (UVB) irradiation on psoriasis has long been recognized. The influence of UVB on the function of circulating neutrophils in patients with psoriasis, as well as in healthy persons, is largely unknown, however, whereas there have been several reports on neutrophil function in patients with psoriasis undergoing psoralen and UVA treatment (PUVA) (7).

The aim of this study was to determine whether repeated whole body UVB irradiation is associated with any change in the function of circulating neutrophils and in the chemokinetic and chemotactic activities in the serum in patients with psoriasis and in healthy subjects, compared with the spontaneously fluctuating neutrophil function in an untreated healthy group. In addition, an attempt was made to study the influence of UVB irradiation *in vitro* on the phagocytic capacity of normal neutrophils.

MATERIAL AND METHODS

Psoriasis patients (group I) treated with whole body UVB irradiation

This group consisted of 11 patients (3 men, 8 women) aged 22–49 years (mean 35 years) who had had psoriasis for at least 6 months. All patients had nummular or plaque psoriasis without any joint symptoms. None of them had the guttate variety. One patient, with involvement of 20 % of the skin surface, had had a thick, scaly but stable plaque psoriasis for one year. The other ten patients had experienced slow deterioration of their psoriasis during the last month; this was mainly of the nummular type but of a variable degree of severity, mainly moderate to mild. In four of these ten patients it involved 15–20 % of the skin surface, and in the remaining six patients 5 % or less of the skin surface was involved, with lesions mainly on the scalp, elbows and knees and a few thin lesions on the lower legs. None had had any systemic treatment for their skin disease and all topical treatment, except emollients, had been withdrawn for at least 2 weeks before the start of this study.

Healthy volunteers (group II) treated with whole body UVB irradiation

This group consisted of 14 healthy subjects, seven women and seven men of ages 22–42 years (mean 32.3 years).

Reference subjects (group III)

For comparison, we also studied an untreated reference group of ten healthy volunteers (4 men, 6 women) of ages 25–41 years (mean 29.7 years) without any medical treatment. Blood samples were collected in this group once a week for up to four weeks. At the start of the study none of them had any signs of infections, but during the observation period two subjects displayed signs of slight virus infections and were excluded from further study.

UVB treatment

The subjects in groups I and II received whole body UVB irradiation three times a week for 4 weeks (12 treatments) with doses inducing slight erythema according to the routines of our clinic. All subjects had skin type 3 or 4 (8). None had been sunbathing or had used any type of artificial UV irradiation during the last 2 months before this study. In the majority of the subjects (11/14 healthy and 6/11 psoriasis patients) no such exposure had occurred in the last 7 months. The UVB source was a Waldmann 1000 cabin (Waldmann GmbH & Co, Villingen-Schwenningen, West Germany) equipped with 26 Sylvania UV6 tubes (Osram GmbH, München, West Germany) emitting 1.9–2.1 mW/cm^2 UVB and 0.7–0.8 mW/cm^2 UVA, according to measurements with a UV-meter (Waldmann type 585100) adapted for these particular tubes. The accumulated doses of UVB were 3.6 ± 0.9 J/cm^2 (corresponding to 29.1 ± 8.1 min) for the patients and 3.6 ± 1.0 J/cm^2 (corresponding to 28.7 ± 6.7 min) for the healthy volunteers (group II) . Blood samples were collected at 8–9 am, before the start of the treatment (baseline) and 20–24 hours after the 6th treatment (at 2 weeks) and 12th treatment (at 4 weeks). The UV treatment had a good effect in all the psoriasis patients, with thinning of the lesions, although only one patient with less than 5 % involvement of the skin was completely healed after 4 weeks of treatment. One man in the healthy group (II) developed severe erythema after three treatments and the UVB treatment was therefore discontinued. The second blood sample in this case was taken 48 hours after the last treatment and the data are included as 2-week values.

Subjects for UVB treatment of PMNs in vitro

PMNs from two healthy men and three healthy women at ages 15–40 years (mean 32 years) were used. None of them had had any medical treatment.

Phagocytosis assay

Leukocytes were isolated from heparinized blood as described previously (9) and resuspended in Ringerdex (Pharmacia, Sweden) with glucose (6.94 mmol/l) added. The purity of the PMN suspension was 90 ± 5 % (SD).

The particles to be phagocytized consisted of polyvinyltoluene latex particles (Coulter Electronics Ltd, Bedfordshire, England) coated with either 1) human IgG or 2) IgG + complement. The latter was achieved by incubating IgG-coated latex particles in 20 % freshly pooled normal serum, diluted in Ringerdex, at 37°C for 10 minutes followed by washing (3, 10). The main complement compound bound to such particles is C3b (11). Phagocytosis was measured by a kinetic method previously described in detail (3, 10). The initial rate of disappearance of the latex particles per minute was taken as a measure of the initial rate of phagocytosis (expressed as $minutes^{-1}$).

Phagocytosis in PMNs treated with UVB in vitro

Isolated PMNs in Ringerdex-glucose (6.94 mmol/l) (1.8 ml) were mixed with 0.2 ml of autologous (heparin) plasma in Coulter plastic cups (Sarstedt 30 ml, 3 cm radius at the top and 2×2 cm square at the bottom) and kept at 37°C. Duplicate samples from each individual were irradiated with UVB from an Osram Ultravitalux lamp (Osram GmbH, München, West Germany) placed 30 cm above the cell suspension and emitting 0.9 mW/cm^2 UVB and 0.7 mW/cm^2 UVA according to measurements with a grating (Yvon-Jobin, Longjumeau, France) and a spectrophotometer 88 XLA (Photodyne Inc, Ca, USA). The irradiation doses were 0.02 J/cm^2, roughly corresponding to 1 MED (minimal erythema dose) in man. For control, duplicate untreated samples were used. All samples were then centrifuged at $160 \times g$ for 5 min and resuspended in Ringerdex-glucose solution to a final concentration of 4×10^9/l PMNs. The phagocytosis assay was then performed as previously described (3, 10). The rate of phagocytosis was calculated from the mean of each duplicate.

Locomotion of PMNs and the chemotatic and chemokinetic activities of serum

Leukocytes were isolated from heparinized blood as described previously (9) and resuspended in Gey's solution, and the final granulocyte concentration was adjusted to 1.5×10^9/l. The purity of the PMN suspension was 87 ± 5 % (SD). Serum samples were frozen and stored at –70°C. Heated serum was prepared by exposure to a temperature of 56°C for 30 min. Zymosan-activated serum from a pool of normal sera was used as chemoattractant and was prepared as previously described (6). The zymosan-activated serum was mixed with Gey's solution to a concentration of 10 %. The migration was assayed by means of the leading front technique, using a modified Boyden Chamber (12) as described elsewhere (6). The filter pore size was 3 µm (Millipore, Bedford, Mass, USA). All migration studies were performed in duplicate with three readings of each filter, and the mean was calculated from these six values.

Random migration was defined as the migration of PMNs suspended in Gey's solution and with Gey's solution below the filter.

Chemotaxis was defined as migration of PMNs in Gey's solution and with zymosan-activated pooled normal serum (10 %) below the filter.

The chemotactic activity was measured as the migration of normal PMNs from healthy blood donors in Gey's solution towards 10 % (v/v) serum from subjects in group I, II or III in Gey's solution below the filter. Pooled normal serum was used as standard, the response to which was regarded as 100 %.

The chemokinetic activity was defined as the alteration in the speed of PMN migration resulting from the presence of serum in the cell suspension with Gey's solution below the filter. The chemokinetic activity in 20 % non-heated or 10 % heated serum (56°C, 30 min) was measured. PMNs from

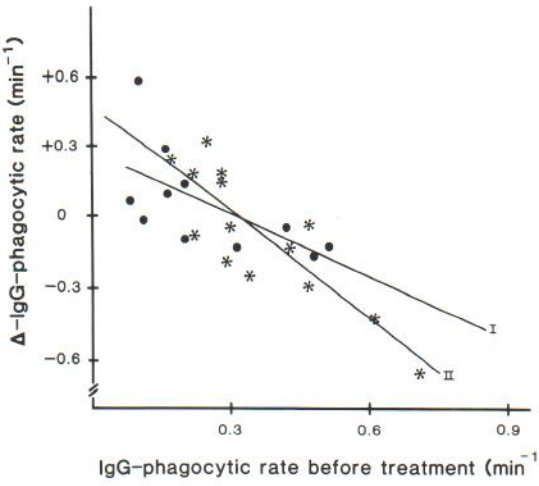

Fig. 1. The IgG phagocytosis in all UVB-treated subjects at baseline (before treatment) plotted against delta IgG phagocytosis (IgG phagocytosis after 2 weeks minus IgG phagocytosis at baseline). (•) represents UVB-treated psoriasis patients and (*) healthy UVB-treated subjects.

The lines represent the regression lines for (I) the psoriasis group ($r = -0.62$, $p < 0.05$; $y = 0.27–0.90\,x$) and (II) the healthy UVB-treated group ($r = -0.85$, $p < 0.001$; $y = 0.46–1.47\,x$).

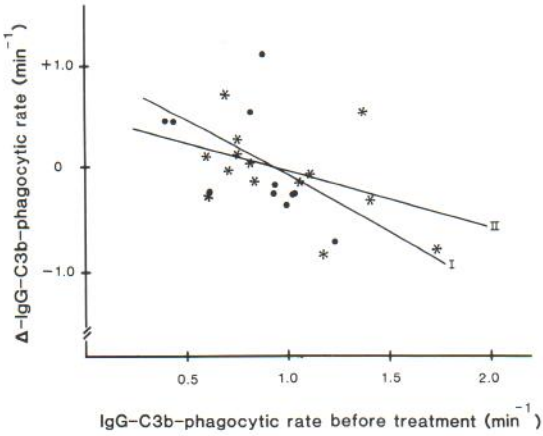

Fig. 2. IgG-C3b phagocytosis in all UVB-treated subjects at baseline plotted against delta IgG-C3b phagocytosis (IgG-C3b phagocytosis after 2 weeks minus IgG-C3b phagocytosis at baseline). (•) represents psoriasis patients and (*) healthy UVB-treated subjects.

The lines represent the regression lines for (I) the psoriasis group ($r = -0.56$, $p < 0.05$; $y = 1.01–1.17\,x$) and (II) the healthy UVB-treated group ($r = -0.47$, $p < 0.05$; $y = 0.52–0.60\,x$).

healthy blood donors were used to estimate the chemokinetic effects of sera from the subjects in groups I, II and III. Ten per cent heated normal pooled serum was used as standard, the response to which was regarded as 100 %.

Various analyses

The total numbers of leukocytes (WBCs), PMNs and mononuclear cells were counted with a Technicon H1 apparatus (Technicon instruments Co, USA). Serum concentrations of haptoglobin were determined by a routine turbidometric method at the Department of Clinical Chemistry, Uppsala, Sweden.

Estimation of the analytical error of the PMN function methods

The estimation of the analytical error of the methods is based on separate duplicate blood samples taken at the same time with the same needle and then treated separately with regard to isolation of PMNs and determination of the various functions. Healthy untreated subjects from group II were used for measurement of the analytical error of the methods.

The analytical error of the method was estimated as the coefficient of variation (CV) and expressed as CV % (Table I). The analytical errors of the phagocytosis rates and the migration were 3.7–8.6 %.

Variation in PMN function in healthy untreated subjects

The variation in PMN function was measured in healthy controls in group III with no signs of infections. The data were based on two to four blood samples from each subject taken once weekly. The average intraindividual variation was expressed as CV % (Table I). The average intraindividual variations in the phagocytosis rates and migration were 21.2–30.4 %.

Statistics

For comparisons at baseline between psoriatic patients and healthy subjects (groups I and IV), the two-sample *t*-test was used. Effects of UV treatment were assessed statistically by the matched-pair *t*-test. The direction and magnitude of the change from baseline to 2 weeks seemed to depend on the baseline level. Linear regression was used to evaluate this relationship.

RESULTS

The significant observations are summarized in Table II or shown in Figs. 1 and 2.

Table 1. *The analytical error and the average intraindividual variations PMN functions expressed as CV %*

	Analytical error		Intraindividual variation	
	n	CV %	*n*	CV %
IgG phagocytosis	4	7.9	8	24.3
IgG-C3b phagocytosis	4	3.7	8	30.4
Chemotaxis	5	8.6	8	21.2

Table 2. *PMN phagocytosis, chemotaxis and chemokinetic activities in serum in each group. Mean ($\bar{x}$), one standard deviation (SD) and number of subjects (n) in each group at baseline, after 2 weeks and after 4 weeks. Group I comprised psoriasis patients, group II healthy subjects treated with UVB, group III untreated healthy subjects*

		Baseline		After 2 weeks		After 4 weeks	
		n	$\bar{x}\pm$SD	*n*	$\bar{x}\pm$SD	*n*	$\bar{x}\pm$SD
IgG-C3b phagocytosis (min-1)	I.	11	0.85±0.26	11	0.87±0.45	11	0.78±0.22
	II.	14	0.98±0.35	14	0.91±0.41	13	[a] 0.81±0.26
	III.	10	0.88±0.34	7	0.79±0.21	8	0.78±0.24
Chemotaxis (μm/n)	I.	11	103.3±23.1	11	98.8±17.9	11	102.7±20.2
	II.	14	[b]84.6±20.8	14	[c]86.1±21.8	13	69.2±10.8
	III.	10	98.7±17.7	7	93.9±12.4	7	103.7±27.5
Chemokinetic activity in heated sera (%)	I.	11	103.3±10.5	11	[d]95.4± 5.5	11	100.2±6.7
	II.	14	101.0±11.1	14	101.6±11.5	12	99.9±14
	III.	9	104.8±13.5	7	98.3±13.4	7	97.3±11.6
Chemokinetic activity in unheated sera (%)	I.	11	130.1±18.6	11	123.6±19.5	11	127.4±15.9
	II.	14	137.8±24.9	14	131.4±25.2	13	[e]121.4±16.6
	III.	9	134.1±21.5	7	127.3±23.2	7	121.7±22.8

[a] $p<0.02$: compared with the mean of IgG-C3b phagocytosis at baseline in group II.
[b] $p<0.02$ and [c] $p<0.01$: compared with the mean chemotaxis after 4 weeks of treatment in group II.
[d] $p<0.02$ and [e] $p<0.01$: Compared with the baseline value in the same group.

Phagocytosis of IgG-coated latex particles (IgG phagocytosis)

Although there was no change in the group mean values during the treatment, some of the subjects exhibited large changes in the phagocytic rate after 2 weeks of treatment compared with the pretreatment value (baseline).

In the two UVB-treated groups a negative relation was observed between the baseline rate of phagocytosis of IgG-coated particles and the difference in this rate between 2 weeks and baseline (Δ-IgG phagocytosis) $r=-0.62$ ($p<0.05$) in the psoriasis group and $r=-0.85$ ($p<0.001$) in the healthy UVB-treated group (Fig. 1).

No such relation was observed in the untreated control group (III). In group III the rate of phagocytosis of IgG-coated particles at baseline ranged from 0.21–0.53 min^{-1}, and the difference between the rate of phagocytosis of IgG-coated particles after 2 weeks and that at baseline (Δ-IgG phagocytosis) ranged between –0.16 and +0.06 min^{-1}.

Before treatment, the psoriasis group (I) showed a lower mean rate of phagocytosis of IgG-coated particles than all healthy subjects at baseline ($p<0.05$). After 2 and 4 weeks there was no significant difference between groups I, II and III in the mean phagocytic rate.

Phagocytosis of IgG-C3b-coated particles (IgG-C3b phagocytosis)

As for phagocytosis of IgG-coated particles the same relation was found between the baseline rate of phagocytosis of IgG-C3b-coated particles and the degree of change after 2 weeks compared with the baseline value (Δ-IgG-C3b phagocytosis); in the psoriasis group $r=-0.56$ ($p<0.05$) and in the healthy UVB-treated group $r=-0.47$ ($p<0.05$) (Fig. 2).

No such significant relation was found in the untreated control group. In this group the baseline rate of IgG-C3b phagocytosis ranged from 0.57 to 1.0 min^{-1} and the difference between the IgG-C3b phagocytosis rate after 2 weeks an that at baseline (Δ-IgG-C3b phagocytosis) ranged from –0.18 to +0.22 min^{-1}.

In the healthy UV-treated group (II) there was a significant decrease in the mean of phagocytosis of IgG-C3b-coated particles after 4 weeks of treatment compared with the pretreatment value ($p<0.02$).

PMN locomotion

There was no significant difference in the mean random migration between the groups at 0, 2 or 4 weeks. Nor was there any change within the groups during treatment. In the healthy UVB-treated group (II) there was a significant decrease in chemotaxis after 4 weeks of treatment compared with the values at baseline ($p<0.02$) and 2 weeks ($p<0.01$). After 4 weeks of UVB treatment the mean chemotaxis in group II was significantly lower both than that in group I and group III ($p<0.001$).

Chemotactic and chemokinetic activity in serum

There was no significant difference in the mean chemotactic activity of serum between the groups or within the groups during treatment. In the psoriasis group the chemokinetic activity in heat-inactivated sera was lower after 2 weeks than before treatment ($p<0.02$), but no such tendency was seen after 4 weeks. In group II the chemokinetic activity in non-heated sera was decreased after 4 weeks of UV treatment compared with the pretreatment value ($p<0.01$), but not after 2 weeks.

Total number of leukocytes (WBC)

There was no difference in the total WBC count between psoriasis patients and healthy subjects before treatment. This count did not change significantly during the UV treatment (0–2, 0–4, 2–4 weeks) in the psoriasis group.

In the healthy UVB-treated group (II) the mean WBC count was decreased after 2 weeks ($5.5\pm1.0\times10^9$/l; $p<0.01$) and after 4 weeks ($5.7\pm1.0\times10^9$/l; $p<0.02$) of treatment compared with the pretreatment value ($6.5\pm1.4\times10^9$/l). There was also a decrease in the number of both PMNs ($3.2\pm0.9\times10^9$/l; $p<0.02$) and mononuclear cells ($2.3\pm0.6\times10^9$/l; $p<0.05$) after 2 weeks of treatment compared with the pretreatment value ($3.9\pm1.2\times10^9$/l and $2.6\pm0.6\times10^9$/l, respectively).

Haptoglobin

The psoriasis group did not differ significantly from the healthy subjects regarding the mean haptoglobin value. No changes in haptoglobin levels were observed in any of the groups during the observation period.

Effects of UVB irradiation on PMN phagocytosis in vitro

There were no significant changes in the mean values for phagocytosis of either IgG-coated (0.50 ± 0.12 min^{-1}) or IgG-C3b-coated (0.85 ± 0.11 min^{-1}) particles after UVB treatment of PMNs in 10 % plasma compared with the values before treatment (phagocytosis of IgG-coated particles 0.52 ± 0.35 min^{-1}, IgG-C3b-coated particles 0.91 ± 0.15 min^{-1}).

DISCUSSION

In this study we have found that regular whole body UVB treatment for four weeks is associated with changes in the function of the neutrophils. During this treatment there were significant alterations in the phagocytosis and chemotaxis of PMNs, in the number of leukocytes, and in the chemokinetic activity of serum. However, the same pattern of changes was not observed throughout the study period and there were also large interindividual variations in the degree of changes in PMN function. The treatment was given when there was minimal natural sunlight and none of the subjects were exposed to UVA irradiation in commercial solariums. The changes are therefore likely to have been induced by UVB, as the proportion of UVA received from the treatment lamps can be considered negligible with regard to its biological effects.

Considerable intraindividual fluctuations in the PMN function from one week to another in healthy untreated subjects were another finding in this study. Publications on the spontaneous biological variation in healthy persons are sparse (13). In our healthy subjects, who were followed up once a week, the average intraindividual variations in phagocytosis and chemotaxis was about 20–30 %, whereas the variation in random migration was only 10 %. As the error of analysis for phagocytosis and chemotaxis was less than 10 %, the high intraindividual variations suggest that there is a biological variation in these functions. The reason for these apparently spontaneous and often irregular fluctuations in healthy persons without clinical infections and with sampling at the same point in time in the morning—is not known, but food intake, exercise, subclinical infections and hormonal influence (13) are factors which might have some impact. The biological relevance of these *in vitro* findings is not clear, but if spontaneous fluctuations in the response to various chemotactic and phagocytic stimuli, for example, also occur *in vivo*, this might be of significance both for a spontaneous flare-up and for an improvement of neutrophil-dominated skin affections such as psoriasis.

After 2 weeks of UVB treatment the individual pattern of change in phagocytic activity compared

with the value before treatment seemed to vary, and the occurrence of decreased, unchanged and increased phagocytic rates led to a finding of no significant change in mean value for the whole groups. There was a significant relationship, however, between the degree of change, after 2 weeks and the pretreatment value both in the patients with psoriasis and the healthy subjects. Thus the phagocytic capacity seems to be affected differently in different individuals depending on its level at the start of treatment. These findings suggest that there may be a receptor-mediated regulatory system for the phagocytic capacity *in vivo* whereby a certain degree of stimulation causes an up-regulation of the receptors to a given optimum and where further stimulation might lead to down-regulation of the number of receptors. Thus a certain stimulation might be able to cause different changes in phagocytosis depending on the basal level of the phagocytosis in the individual in question at the time of stimulation.

Among the patients with psoriasis the tendency to visible clinical improvement was usually noted 2 weeks after start of treatment irrespective of the initial phagocytic rate and of the change in rate between 0 and 2 weeks. Whether the changes in phagocytic activity observed during the first 2 weeks of UV treatment are of biological significance is unknown. Nor is it known how these changes are mediated. Although the material was small, in the *in vitro* experiment with UVB irradiation of isolated neutrophil granulocytes, there was no obvious change in the phagocytic rate after UVB exposure, indicating that UVB therapy affects the neutrophils indirectly. Several mediators, e.g. histamine, interleukin-1 (IL-1) and 12-hydroxy-eicosatetraenoic acid (12-HETE), that are formed during UVB exposure (14, 15) are, however, known to be able to influence the functional properties of PMNs (16–18).

After 4 weeks of UVB treatment three times weekly in healthy subjects, moderate to strong pigmentation was observed and the tendency to erythema decreased. A reduction of IgG-C3b phagocytosis, PMN chemotaxis, and chemokinetic activity of unheated serum was seen in the healthy UV-treated group as a whole. The mechanisms of these effects are not known.

Treatment with UVB seemed to induce less change in the PMN function in the psoriasis group, although the mean UVB dosages for the psoriatic patients and the controls were almost identical. The only significant alteration in the group of psoriasis patients — apart from the previously discussed effect on the rate of phagocytosis — was a reduction of the chemokinetic activity of heated sera after 2 weeks of treatment compared with the pretreatment activity. Differences in the reactivity of psoriasis PMNs compared with that of healthy subjects cannot be excluded. However, for proper evaluation of such a possibility the comparison should be based on controls and psoriatics with as similar baseline values as possible, as the changes might be related to the pretreatment values.

In this context it should be mentioned that the psoriasis group showed a significantly lower mean phagocytic rate of IgG-coated particles than the healthy controls before treatment. Their sera did not differ in chemokinetic activity. This is in contrast to our previous observations that patients with psoriasis had an increased rate of phagocytosis (3) and that their sera showed increased chemokinetic activity (6). The majority of the patients in the previous studies had more severe, more extensive and more active disease than those in the present study.

After two weeks of treatment there was a significant decrease in the number of both poly- and mononuclear leukocytes in the healthy subjects. This contrasts with the increase, mainly in PMNs, observed up to 15 hours after an isolated UVB treatment in healthy subjects (19).

The granulocyte function has been investigated in healthy persons treated with a combination of ultraviolet A (UVA) and UVB (20, 21). Csato et al. (20) noted increased PMN chemotaxis after 10 days of treatment, and Scherf et al. (21) observed increased phagocytosis of *C. albicans* after 3 weeks of treatment. However, the studies are not comparable with the present one, as the same light source, treatment intervals, methods and follow-up period are needed to make reliable comparisons between the results of different studies.

UVB induced an improvement in all patients with psoriasis regardless of the degree and type of change in the studied PMN functions during the treatment. The role played by PMN activity during the healing phase therefore seems difficult to evaluate.

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