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ORIGINAL ARTICLE

Effect of climate therapy at Gran Canaria on vitamin D production, blood glucose and lipids in patients with psoriasis

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Abstract

Background Climate therapy (heliotherapy) of psoriasis is an effective and natural treatment. Ultraviolet radiation (UVB) from the sun improves psoriasis and induces vitamin D_3 synthesis.

Objective The aim of the study was to investigate the effect of climate therapy on vitamin D_3 synthesis, blood glucose, lipids and vitamin B12 in psoriasis patients.

Methods Twenty Caucasian patients (6 women and 14 men; mean age, 47.2 years; range, 24–65) with moderate to severe psoriasis [mean Psoriasis Area and Severity Index (PASI) score 9.8; range, 3.8–18.8] received climate therapy at the Gran Canarias for 3 weeks. Blood samples were drawn before and after 15 days of sun exposure. In addition, the patients' individual skin UV doses based on UV measurements were estimated.

Results Sun exposure for 15 days lead to a 72.8% (± 18.0 SD) reduction in the PASI score in psoriasis patients. Although no direct correlation was observed between PASI score improvement and UVB dose, the sun exposure improved the vitamin D, lipid and carbohydrate status of the patients. The serum concentrations of 25-hydroxyvitamin D [25(OH)D] increased from 57.2 ± 14.9 nmol/L before therapy to 104.5 ± 15.8 nmol/L ($P < 0.0001$) after 15 days of sun exposure; the serum levels of 1,25-dihydroxyvitamin D [$1,25(OH)_2D$] increased from 146.5 ± 42.0 to 182.7 ± 59.1 pmol/L ($P = 0.01$); the ratio of low-density lipoprotein cholesterol and high-density lipoprotein cholesterol decreased from 2.4 to 1.9 ($P < 0.001$); and the haemoglobin A_1c (HbA_1c) levels decreased from 5.6 ± 1.7% to 5.1 ± 0.3% ($P < 0.0001$).

Conclusion Climate therapy with sun exposure had a positive effect on psoriasis, vitamin D production, lipid and carbohydrate status.

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Keywords

climate therapy, heliotherapy, psoriasis, vitamin D

Conflicts of interest

None declared.

Introduction

Ultraviolet (UV) radiation from the sun is an effective and natural treatment method for several skin diseases. Heliotherapy has been offered to psoriasis patients at low latitude for many years.[1] Artificial, or solar UVB, radiation induces local and systemic immunosuppression,[2] induces apoptosis, inhibits cellular proliferation and causes vitamin D synthesis in the skin.[3] For most people, sun exposure is the main vitamin D source, while dietary intake is of minor importance.[4] Increasing evidence indicates that cutaneous vitamin D synthesis is of importance

The study was carried out in March 2006 at Valle Marina Treatment Centre, Gran Canaria (27°N, 15°W), which is administrated by Section for Climate Therapy, Department of Rheumatology, Rikshospitalet University Hospital, Oslo, Norway.

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in the treatment of and even of the prevention of a variety of diseases, including malignancies.[5] In addition, the known therapeutic effect of UVB light therapy in the treatment of psoriasis may, at least in part, be mediated directly via UVB-caused synthesis of vitamin D.[6]

In a previous study, it was found that broadband UVB increased vitamin D synthesis in patients with psoriasis.[7] Some studies revealed that heliotherapy was more effective than UVB phototherapy in treating psoriasis.[8] The aim of this study was to investigate the effect of climate therapy on vitamin D synthesis, blood glucose and lipids, vitamin B12, C reactive protein and haemoglobin in patients with psoriasis. The hypothesis was that sun-stimulated production of vitamin D had an effect on the variables listed above in patients with psoriasis.

Subjects and methods

Patients

The study included 20 patients [mean age, 47.2 ± 10.7 (SD) years; range, 24–65 years; 6 women and 14 men] with moderate to severe psoriasis. All patients had active plaque psoriasis Psoriasis Area and Severity Index (PASI) 9.8 (mean; range, 3.8–18.8). PASI scores were assessed by dermatologists before and after the sun exposure. The patients were without any psoriasis medication for 4 weeks before and during the study. Two patients had skin type II and 18 had skin type III according to the Fitzpatrick classification.[9]

Ethical considerations

Each patient was given written information about the aim of the study, which was approved by the Regional Committee of Medical Ethics and the Norwegian National Data Inspection Board. Declaration of Helsinki protocols was followed, and written informed consent of patients was obtained.

Norwegian health centre at Valle Marina, Gran Canaria

Norwegian psoriasis patients have been offered climate therapy since 1976. Today, this treatment option is administrated by the Section for Climatotherapy, Department of Reumatology at the Rikshospitalet University Hospital in Oslo, Norway. The patients are treated at the Norwegian Health Centre at Valle Marina, Gran Canaria, for 3 weeks during January to June and September to December. The therapy is supervised by a resident dermatologist, nurses and physiotherapists.

Measurement of vitamin D

The serum concentrations of 25-hydroxyvitamin D [25(OH)D] were measured by radioimmunoassay (DiaSorin, Stillwater, MN, USA). This assay measures both $25(OH)D_3$ and $25(OH)D_2$, and the intra- and total assay coefficients of variation (CVs) are 6% and 14%, respectively. Total assay variation includes intra- and inter-assay coefficient of variation. Inter-assay CV% = square root (total $CV\%^2$ – intra-assay $CV\%^2$). The lower limit of detection was 4 nmol/L. Functional sensitivity was 13 nmol/L.

Serum levels of $1,25(OH)_2D$ was measured by competitive radioimmunoassay (DiaSorin). Prior to the $1,25(OH)_2D$ determination, serum lipids and interfering vitamin D metabolites were removed by chromatography on a C_{18}OH column. Cross-reaction with 25(OH)D after chromatography is noted to be 0.002%. The intra- and total assay CVs for the $1,25(OH)_2D$ assay were 7% and 14%, respectively. The lower limit of detection was 12 pmol/L.

Procedures of UV exposure and UV measurement

The study was carried out for 15 days in March 2006 at Valle Marina Treatment Centre, Gran Canaria (27°N, 15°W). The patients followed a strict exposure schedule on the first day of the study, exposing first the front side of the body for 30 min, the back side for 30 min, followed by 15-min exposure on each side in the period from about 11:00 to 13:00 local time. They were allowed to stay outside after lunch, when a proper amount (2 mg/cm^2) of sunscreen with sun protection factor (SPF) of 25 (Pediatrics Photoprotector ISDN, 25B-10A-IR) was used for the whole body.[10] For the remaining days, the patients were advised to gradually increase the hours of exposure per day, and limit to use sunscreen to locations easily burned. The patients registered time spent in the sun every day each 20th minute from 9:00 to 17:00 local time, as well as the use/amount of sunscreen and type of SPF factor.

Spectral UVB (280–315 nm), UVA (315–400 nm) and CIE-weighted UV irradiances were measured every hour from 9:00 to 17:00 using two broadband instruments (Solar Light Co PMA 2100 with UVB sensor PMA 2101 and UVA sensor PMA 2110, and Gigahertz-Optik GmbH X1 1 Optometer with UVB and UVA sensors XD-9501). The CIE-action spectrum is a reference action spectrum for UV-induced erythema in Caucasian human skin valid for the UV region from 250 to 400 nm[11] The sensors were calibrated and intercompared against a spectroradiometer (Brewer#185, measurement range 286.5–365 nm, extended for UVA 365–400 nm) at Izaña, Tenerife, prior to the study (by Mr Alberto Redondas, Instituto Nacional de Meteorologia, Spain), according to internationally accepted procedures.[12,13] The overall measurement uncertainty can be estimated to ±25% and is due to uncertainty in the different instruments, temperature variations, azimuth variations and non-ideal cosine response in broadband sensors.

Spectral UVB and UVA irradiances, in addition to CIE-weighted UVB and UVA irradiances, were calculated for the whole period using a radiation transfer model, libRadtran, for irradiance calculations.[14] The model was run for the following conditions: cloudless sky, albedo of 0.05, sea level and ozone values from the TOMS satellite.[15] The UV irradiances were adjusted according to the measurements taken at the treatment centre to account for the real weather situation and possible discrepancies from the model parameters, such as different albedo and aerosol amount. Combining the calculated UV irradiances

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with the sun exposure time from the patients' diaries, UV doses were estimated for each patient after 1 day and after 15 days of sun exposure.

The results are presented as spectral UVB and UVA doses, as well as CIE-weighted UV doses in Standard Erythema Dose (SED; 1 SED = 100 J/m^2 = 0.01 J/cm^2). UV doses for each patient were set equal to the ambient UV doses divided by two, since only half the body can be exposed at any time. Doses on the first day excluded exposure time when sunscreen is used, since the patients reported using approximately the proper amount of sunscreen after lunch (30 mL, SPF (sun protection factor) 25). On the remaining days, all exposure time is included, since the patients reported using small amounts of sunscreen and only on easily burned locations.

Biochemistry

The serum concentrations of calcium (Ca), ionized calcium (Ca^{++}), parathyroid hormone (PTH), plasma folate, homocysteine (HC), vitamin B12, erythrocyte haemoglobin (EHb), erythrocyte folate (EFo), erythrocyte haematocrit (EHct), C reactive protein (CRP) and micro CRP (mCRP) were obtained before the sun exposure, after 1 day and after 15 days of exposure. The serum concentrations of creatinine, glucose, apolipoprotein A1 (APO-A1), APO-B, lipoprotein A [Lp(a)], total cholesterol, high-density lipoprotein cholesterol (HDL), low-density lipoprotein cholesterol (LDL), triglyceride and haemoglobin A_1c (HbA_1c) were obtained before the sun exposure and after 15 days of exposure.

Standard blood chemistry was measured in serum/plasma using routine laboratory methods in an ISO 17025 accredited laboratory (Department of Medical Biochemistry, Rikshospitalet, Oslo, Norway).

Statistics

Data are given as mean ± SD or median (min–max) if not otherwise stated. Simple descriptive statistics and univariate correlations were performed using the statistics routines of software (Excel, Microsoft Inc., SPSS, version 15).

Student's paired *t*-test was used for comparisons of the blood test results before and after sun exposure. Associations between variables were tested by Pearson and Spearman correlation analysis. Probability values (two sided) were considered significant at values of < 0.05.

Results

Vitamin D

Sun exposure for 15 days led to a 72.8 ± 18.0% reduction in the PASI score in patients with psoriasis. Furthermore, the sun exposure increased serum concentrations of both 25(OH)D and $1,25(OH)_2D$ (Table 1; Figs 1–2).

Patients with 25(OH)D ≤ 50 nmol/L at baseline (1 woman and 5 men) increased more in 25(OH)D than patients with 25(OH)D > 50 nmol/L ($P = 0.03$). The serum concentrations of $1,25(OH)_2D$ increased after 15 days of sun exposure ($P = 0.01$, days 1–15 and $P = 0.004$, days 2–15; Table 1; Fig. 2).

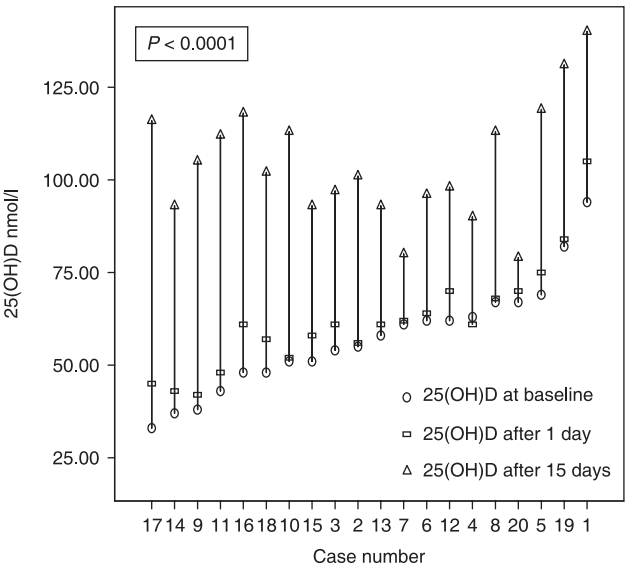

Figure 1 Changes in serum 25(OH)D for each psoriasis patient after 1 and 15 days of climate therapy. *P*-level indicates changes after vs. before.

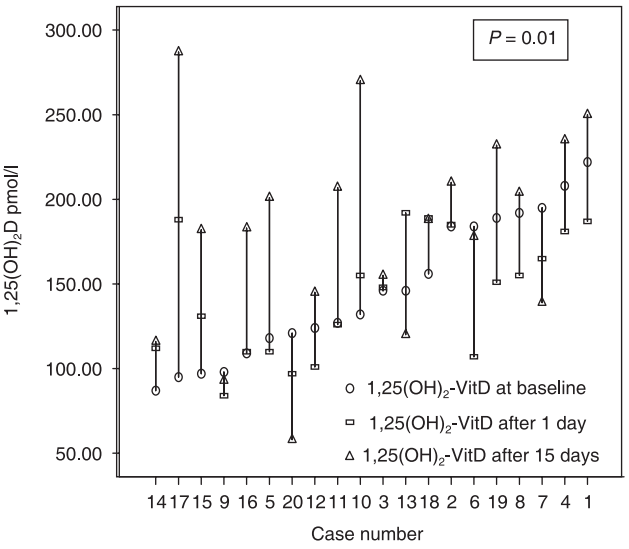

Figure 2 Changes in serum $1,25(OH)_2D$ for each psoriasis patient after 1 and 15 days of climate therapy. *P*-level indicates changes after vs. before.

Biochemistry

The serum concentrations of PTH decreased ($P = 0.04$, days 2–15; Table 2).

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Table 1 Serum 25(OH)D, 1,25(OH)2D, APO-A1, Erythrocyte Haemoglobin (EHb), Folate (EFo), HDL-cholesterol, Homocysteine (HC), urate, APO-B, vitamin B12 and HbA1c before, during and after climate therapy in 20 patients with psoriasis (means ± SD). *P*-level indicates changes between days 1 and 15

	Day 1	Day 2	Day 15	*P*-level day 15 vs. day 1
25(OH)D, nmol/L	57.2 ± 14.9	62.2 ± 14.7	104.5 ± 15.8	< 0.0001
$1,25(OH)_2D$, pmol/L	146.5 ± 42.0	143.7 ± 36.1	182.7 ± 59.1	0.01
APO A-1, g/L	1.44 ± 0.28		1.59 ± 0.33	0.001
EHb, g/L	200.30 ± 72.70	195.85 ± 66.92	244.24 ± 76.22	0.000
EFo, nmol/L	454.00 ± 166.30	433.5 ± 152.42	551.75 ± 176.80	0.000
HDL, mmol/L	1.27 ± 0.36		1.50 ± 0.50	0.001
HC, μmol/L	12.50 ± 4.08	13.35 ± 2.99	14.20 ± 3.29	0.016
Urate, μmol/L	325.05 ± 73.44		361.35 ± 90.55	0.006
APO B, g/L	0.91 ± 0.25		0.82 ± 0.23	0.001
Vitamin B12, pmol/L	325.50 ± 104.44	306.5 ± 104.33	278.25 ± 98.26	0.003
HbA1c, %	5.63 ± 1.71		5.09 ± 0.32	0.000

Table 2 BMI, systolic blood pressure (SBP), diastolic blood pressure (DBP), pulse, serum calcium, ionized calcium, PTH, folic acid, creatinine, EHct, CRP, mCRP, glucose, Lp(a), total cholesterol, LDL-cholesterol and triglyceride before, during and after climate therapy in 20 patients with psoriasis (means ± SD). P-level indicates changes between days 1 and 15

	Day 1	Day 2	Day 15	*P*-level day 15 vs. day 1
BMI, kg/m^2	26.6 ± 4.9		26.3 ± 4.7	0.60
SBP, mmHg	134.4 ± 16.2		137.2 ± 20.9	0.63
DBP, mmHg	84.5 ± 9.7		90.9 ± 10.6	0.007
Pulse, beats/min	76.2 ± 11.4		73.2 ± 13.9	0.11
Calcium, mmol/L	2.24 ± 0.14	2.28 ± 0.17	2.27 ± 0.11	0.18
Ionized Ca^{++}, mmol/L	1.22 ± 0.04	1.21 ± 0.03	1.24 ± 0.04	0.09
PTH, nmol/L	4.40 ± 1.82	5.15 ± 2.19	4.07 ± 1.35	0.33
Folic acid, nmol/L	12.5 ± 4.2	12.2 ± 4.7	11.5 ± 4.2	0.21
Creatinine, μmol/L	67.6 ± 9.5		69.2 ± 9.2	0.08
CRP ($n = 14$), mg/L	3.52 ± 4.75	9.46 ± 8.29	10.07 ± 23.45	0.075
EHct, ml/L	42.65 ± 3.47	43.60 ± 2.68	43.10 ± 3.02	0.59
Glucose, mmol/L	4.83 ± 0.63		4.50 ± 0.60	0.054
Cholesterol, mmol/L	5.23 ± 1.06		5.09 ± 0.82	0.27
LDL, mmol/L	3.07 ± 0.94		2.86 ± 0.75	0.074
Lp(a), mg/L	316.8 ± 346.4		314.0 ± 362.2	0.065
Micro CRP ($n = 6$), mg/L	0.60 ± 0.14	0.67 ± 0.07	0.58 ± 0.26	0.59
Triglycerides, mmol/L	1.84 ± 1.02		1.49 ± 1.22	0.098

Body mass index (BMI) was unaltered during the study period (Table 2). The sun exposure improved the lipid and carbohydrate status of the patients. The LDL/HDL cholesterol ratio decreased from 2.4 to 1.9 ($P < 0.001$; Fig. 3; Table 1). The serum concentrations of APO-A1, EHb, folic acid, HDL, homocysteine and uric acid increased during 15 days of climate therapy (Table 1). The serum concentrations of APO-B, vitamin B12 and HbA_1c decreased during the sun exposure period (Table 1). The serum concentrations of calcium, creatinine, EHct, glucose, CRP, mCRP, total cholesterol, LDL, Lp(a) and triglyceride were not influenced by climate therapy (Table 2).

Diastolic blood pressure increased from 84.5 ± 9.7 to 90.9 ± 10.6 mmHg ($P = 0.007$), while systolic blood pressure and pulse were unaltered after 2 weeks of sun exposures.

Sun exposure

Daily sun exposure time per patient increased throughout the treatment period. The patients received on average 5.1 ± 2.3 SED (median = 4.0 SED; range, 2.6–10.3 SED) on the first day of exposure (Table 3). The mean dose was similar for patients with skin type II and III. The mean dose after 15 days sun exposure was 166 ± 25 SED (range, 104–210), 135 and 169 SED for the patients

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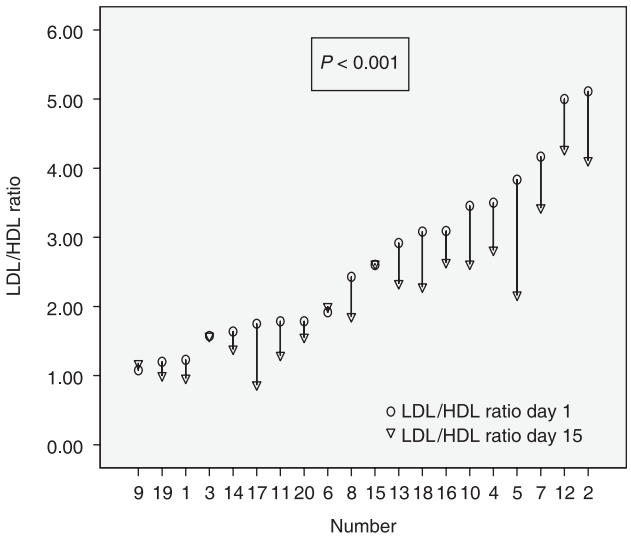

Figure 3 Changes in the ratio of LDL-cholesterol and HDL-cholesterol for each patient after 15 days of climate therapy. *P*-level indicates changes after vs. before.

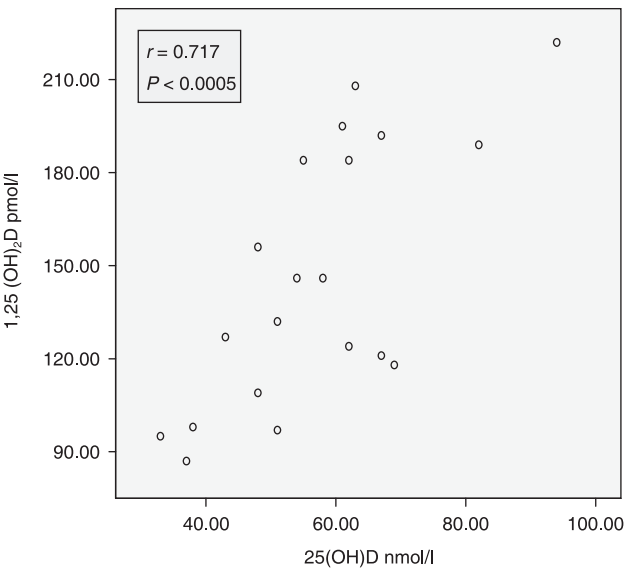

Figure 4 Correlation between levels of 25(OH)D at baseline and levels of $1,25(OH)_2D$ at baseline.

Table 3 Estimated UV doses to the patients after 1 and 15 days of sun exposure (means ± SD; note: the values correspond to the respective ambient doses divided by 2).

	UV doses after 1 day of exposure	UV doses after 15 days of exposure
UVB, J/cm^2	0.36 ± 0.16	11.5 ± 1.7
UVA, J/cm^2	14.9 ± 6.9	452 ± 70
CIE-weighted UV, SED	5.1 ± 2.3	166 ± 25

CIE, International Commission of Illumination.

with skin type II and III, respectively. The variation between minimum and maximum patient doses each day was large. The patients' sunbathing with sunscreen during the first day reported a use of approximately 30 mL of cream each on body sites easily burned. Although 14 out of the 20 patients reported erythema after the first day of sun exposure, but none reported blistering erythema.

Correlations

The reduction in PASI score was 72.8%, but there was no correlation between the improvement in PASI score and vitamin D or the UV dose. Furthermore, we did not find any correlation between changes in the single component of PASI score, including area, erythema, infiltration and desquamation, and the dose of received UVB.

Increase in 25(OH)D correlated negatively with levels of 25(OH)D at baseline ($P = 0.023$). No correlation was found between age and changes in 25(OH)D. Neither the serum concentration of 25(OH)D nor the increase of 25(OH)D correlated with BMI. There were no differences in serum levels of 25(OH)D or $1,25(OH)_2D$ between smokers ($n = 10$) and nonsmokers ($n = 10$), respectively. We did not find any correlation between the dose of UVB and increase in 25(OH)D.

Serum concentration of $1,25(OH)_2D$ at baseline correlated positively to serum concentration of 25(OH)D at baseline ($P < 0.0005$; Fig. 4) and negatively to increase of 25(OH)D ($P = 0.023$). The increase in 25(OH)D correlated to the increase in $1,25(OH)_2D$ after 15 days of climate therapy ($P < 0.0005$).

$1,25(OH)_2D$ correlated positively to PTH at start ($r = 0.544$; $P = 0.019$). Using a regressive analyse of changes in PTH and changes in 25(OH)D and 1,25(OH)D, respectively, we found a positive partial correlation between Δ PTH and Δ $1,25(OH)_2D$ ($r = 0.894$; $P = 0.02$) but no correlation between Δ PTH and Δ 25(OH)D was found.

Serum concentrations of 25(OH)D at baseline correlated positively to serum HDL at baseline ($r = 0.462$; $P = 0.04$).

Positive correlations were found between age and changes in ionized calcium during sun exposures ($P = 0.005$).

There were no correlations between serum concentrations of 25(OH)D or $1,25(OH)_2D$ and blood pressure or pulse rate, respectively.

Discussion

Climate therapy at Gran Canaria enhanced vitamin D_3 production and improved the PASI score in patients with psoriasis. Furthermore, positive effects were seen on glucose and

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lipid metabolism. These findings support the opinion by many dermatologists, that psoriasis is not only a chronic inflammatory disease restricted to the skin, but also a systemic one.

There were, however, no dose-dependent correlation between the vitamin D metabolite levels, PASI score and the received UV dose. The serum concentrations of 25(OH)D almost doubled during 15 days of climate therapy. Patients with lower the 25(OH)D levels at the baseline responded better to sunlight which is in accordance with other studies.[4,7] All patients reached levels of 75 nmol/L after 2 weeks of sun exposures. A circulating level of 25(OH)D of > 75 nmol/L, or > 30 ng/mL, is suggested as necessary to maximize the vitamin D's beneficial effects for health.[4] Sun exposure is the major source of vitamin D for most humans.[4] Skin pigment, sunscreen use, ageing, time of day, season and latitude affect pre-vitamin D_3 synthesis.[16]

In accordance with a previous study,[7] age did not correlate to the increase in 25(OH)D. The increase in 25(OH)D was very similar to the increase in 25(OH)D during treatment with broadband UVB for 2 to 3 months.[7] During prolonged exposure to the sun, the accumulation of pre-vitamin D_3 is limited to about 10% to 15% of the original 7-dehydrocholesterol content because the pre-vitamin also photoisomerizes to two biologically inert photoproducts: lumisterol 3 and tachysterol 3.[17]

The vitamin D production is a very unique, autoregulated mechanism. The autoregulation is at two levels. Excessive sun exposure does not lead to overdose of vitamin D_3 due to conversion of pre-vitamin D_3 to inactive photoproducts (lumisterol 3 and tachisterol 3) as well as conversion of vitamin D_3 to its isomers in the skin (5,6-*trans*-vitamin D_3, supersterol I, supersterol II) which are thought to have low calcemic effect at physiological concentrations. Vitamin D_3 is synthesized in the skin and releases steadily and slowly from the skin into the circulation.[17]

Our method to measure and estimate UV doses is easier compared with using personal UV dosimeters, but is affected with more uncertainties. The predominant uncertainty is assuming that the skin dose equals half the ambient dose. This is appropriate as a first approximation, in particular for the abdomen and back during sun bathing if the patients turned to expose these sites at equal amounts. Measurements using personal dosimeters in other studies support this assumption.[18–20] For extremities, doses are reported to be higher and lower for various activities, and all vary more than during sun bathing.[18,20,21] The upper extremities probably often receive more than 50% of the ambient UV[18–21] and are therefore more susceptible to be sunburned. These are where the patients reported to have used sunscreen through the treatment period.

The few studies to assess the relation between sunlight measures and serum 25(OH)D showed weak correlations.[22] The patients reached the plateau of daily sun exposure after the first week. It might be that the vitamin D production was most prominent during the first week when the patients had experienced redness and some of them even got sunburned.

The increase of 25(OH)D during 15 days of climate therapy was significant, even though the patients used sunscreens on the body sites susceptible to be sunburned and even though the skin covered with psoriasis lesions. A sunscreen with a SPF-8 reduces the skin's production of vitamin D_3 by 95%. Clothing completely blocks all solar UVB radiation and thereby prevents vitamin D_3 production.[23]

The serum concentrations of $1,25(OH)_2D$ increased, which is in accordance with a previous study in psoriasis patients with vitamin D deficiency.[7]

It has been postulated that the synthesis of $1,25(OH)_2D$ is tightly regulated, and that increases in 25(OH)D concentrations due to exposure to sunlight have no effect on serum $1,25(OH)_2D$ levels.[4,24] Some other studies[25,26] have shown a positive correlation between serum 25(OH)D and serum $1,25(OH)_2D$ in vitamin D–deficient patients, indicating substrate-dependent synthesis of $1,25(OH)_2D$. The finding that our patients increased in their $1,25(OH)_2D$ might be due to low levels of 25(OH)D before the heliotherapy. This is in accordance with previous reports after UVB exposure[7] and vitamin D supplementation.[27] PTH is a well-known stimulator of $1,25(OH)_2D$ synthesis. The positive correlation between $1,25(OH)_2D$ and intact PTH reflects the physiological effects of PTH on the $1,25(OH)_2D$ response to low calcium. The positive relationship between PTH and $1,25(OH)_2D$ and inverse relationship between PTH and 25(OH)D has been shown in a previous study from the same city.[28] The persistently elevated $1,25(OH)_2D$ concentration in patients with low levels of 25(OH)D might be explained by PTH-induced chronic stimulation of the renal 25(OH)D-1α-hydroxylase.[27]

We did not find any correlation between reduction in PASI score and serum concentrations of 25(OH)D in accordance with another study.[7] The known therapeutic effect of UVB light therapy in the treatment of psoriasis may, at least in part, be mediated via UVB-induced production of vitamin D that is thought to be converted to 25(OH)D and then to $1,25(OH)_2D$.[6] $1,25(OH)_2D$ and its analogs, as well as UVB phototherapy, exert antiproliferative, prodifferentiative and immune modulatory effects on keratinocytes that have a particular impact on the therapy of hyperproliferative skin diseases such as psoriasis vulgaris.[29,30] This fact is supported by findings of Vanhooke *et al.*,[31] indicating that a systemic vitamin D deficiency does not stimulate epidermal synthesis of $1,25(OH)_2D$. However, the full range of UVB and vitamin D_3 effects is not completely understood.

Vitamin B12 (cyanocobalamin) can be photolysed by UV light.[32] Thus, the decrease in serum vitamin B12 during climate therapy can be explained by its photodegradation.

Ultraviolet radiation of the UVB region readily destroys tryptophan (Trp) residues of LDL and HDL. Moreover, LDL and HDL cholesterol are natural carriers of vitamin E and carotenoids. These two antioxidants are also rapidly bleached by UVB.[33]

Increase in HDL-cholesterol and decrease in HbA_1c during climate therapy could be explained by several factors. One possible mechanisms could be the action of vitamin D that includes

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stimulation of insulin secretion and effects on insulin sensitivity.[34] The other one is that sun exposure usually implies greater outdoor physical activity, which in itself may have beneficial effects on lipids and insulin sensitivity, unrelated to serum 25(OH)D concentrations.[34] In addition, the diet might also influence on glucose and lipid metabolism. The observed associations among vitamin D, insulin and glucose metabolism in humans have not yet been confirmed by intervention studies and, hence, a causal association has not been established.[34] Although the climate therapy did not change the basal glucose levels of the patients, the HbA1c levels decreased about 10% – indicating improved insulin sensitivity leading to decreased postprandial glucose response. This suggestion is supported by Chiu[35] who reported a clear negative correlation between the plasma levels of 25(OH)D and glucose 60 to 120 min following a standard 75-g oral glucose tolerance test.

There are seasonal variations in lipid levels, with total cholesterol, LDL, triglyceride and Lp(a) highest in the winter, a time at which UVB-induced synthesis of vitamin D would be expected to be at its minimum.[36] Serum concentrations of 25(OH)D at baseline correlated positively to serum HDL at baseline in accordance with a previous published study.[37] We did not find any other correlation between vitamin D and blood lipids.

Maintained body weight speaks against any caloric reduction as an explanation for the improved metabolism.

Diastolic blood pressure increased in patients with psoriasis after 15 days of climate therapy. Some other studies have described that sun-like UV irradiation of healthy persons decreased resting pulse rate, decreased recovery pulse rate, and decreased systolic blood pressure.[38] We have no explanation for the present opposite finding, but it could be due to chance or possibly an increased level of physical activity.

Conclusion

Climate therapy with sun exposure had a positive effect on psoriasis, vitamin D production, lipid and carbohydrate status.

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