The Effect of Narrowband UV-B Treatment for Psoriasis on Vitamin D Status During Wintertime in Ireland

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Objectives: To determine whether narrowband UV-B (NB–UV-B) may mediate its beneficial effect on psoriasis by increasing vitamin D levels, and to assess the effect of NB–UV-B on vitamin D status in patients with psoriasis in wintertime.

Design: A prospective controlled study from October 2008 to February 2009.

Setting: A dermatology outpatient department at a university teaching hospital.

Patients: Thirty consecutive patients with psoriasis treated with NB–UV-B and 30 control patients with psoriasis were recruited. Control patients were recruited within 1 week of treated patients to control for seasonal variation of serum 25-hydroxyvitamin D [25(OH)D] levels. One patient with photoaggravated psoriasis was withdrawn from the study.

Intervention: Narrowband UV-B was administered 3 times per week.

Main Outcome Measure: Serum 25(OH)D was measured at baseline, after 4 weeks and at completion of treatment.

Results: Levels of serum 25(OH)D increased significantly (P < .001) from a median (range) of 23 (9-46) ng/mL at baseline to 59 (32-112) ng/mL at the end of NB–UV-B treatment compared with no change in the control group. The change in serum 25(OH)D level correlated with the number of exposures of NB–UV-B (r=0.61; P < .001) and cumulative UV-B dose (r=0.47; P = .01) but not with treatment response. At the end of the study, all patients in the treatment group were vitamin D sufficient, but 75% of the control group had vitamin D insufficiency [serum 25(OH)D level <20 ng/mL]. In a multiple regression model, prior phototherapy was the sole predictor of baseline serum 25(OH)D level (r²=0.13; P = .006), whereas the number of exposures of NB–UV-B predicted change in serum 25(OH)D level (r²=0.38; P = .001).

Conclusions: Narrowband UV-B effectively increases serum 25(OH)D level while clearing psoriasis. Up to 75% of Irish patients with psoriasis were shown to be vitamin D insufficient during wintertime.

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SORIASIS IS A COMMON CHRONIC inflammatory disorder of the skin that affects 1.5% to 3.0% of the population. The epidermis plays a major role in vitamin D synthesis but is also a target tissue for activated vitamin D and its analogues. Abnormalities in vitamin D metabolism may be partly responsible partly for the pathologic proliferation and differentiation of keratinocytes in psoriasis. Narrowband (NB) UV-B has now become the standard phototherapy for psoriasis. The TL-01 lamp emits a narrow band of UV-B of 311 to 313 nm, which lies within the action spectrum responsible for cutaneous vitamin D3 production. Studies have shown that broadband (BB) and NB–UV-B affect vitamin D status while clearing psoriasis, suggesting that this could partly account for the beneficial effect of phototherapy in psoriasis.

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Most vitamin D is obtained by skin production following exposure to solar UV-B, while less than 15% is obtained from dietary sources such as oily fish and fortified foods. Cholecalciferol, also known as vitamin D3, is the form of vitamin D produced in the skin. Cutaneous synthesis of vitamin D3 is produced by the action of specific UV-B wavelengths (290-315 nm) on 7-dehydrocholesterol, a precursor in the skin, to produce pre-vitamin D3, which is rapidly converted to vitamin D3. The optimum wavelength for initiation of cutaneous vitamin D3 synthesis is 300±5 nm in vivo.
and in vitro. Solar UV-B irradiation is dependent on latitude and season, so those living in mid to high latitudes are at risk of vitamin D deficiency. Skin pigmentation also determines response to UV-B radiation, with a greater rate of vitamin D synthesis in lighter skin tones because melanin absorbs UV-B irradiation. Vitamin D is hydroxylated at the 25 position in the liver to the major circulating form, namely, 25-hydroxyvitamin D [25(OH)D], which is inert. It is then hydroxylated in the 1 position in the kidney to the active form, 1,25 dihydroxyvitamin D [1,25(OH)2D], which is vital for the endocrine functions of the vitamin D hormone.

A number of extrarenal sites, including the epidermis, can form the skin production of vitamin D is absent from mid-October to early February when serum 25(OH)D levels are below 20 ng/mL were considered to be vitamin D insufficient according to the recommended winter level.19,20

We assessed the UV-mediated increase in serum 25(OH)D levels in patients with psoriasis treated with NB–UV-B during wintertime compared with a control group of patients with psoriasis. Our primary objective was to determine whether NB–UV-B mediates its beneficial effect in psoriasis by improving vitamin D status. Our secondary objective was to evaluate the efficacy of NB–UV-B in preventing hypovitaminosis D in patients with psoriasis in wintertime.

**METHODS**

**STUDY DESIGN**

This was a prospective controlled study of patients with chronic plaque psoriasis treated with NB–UV-B phototherapy in our department. Patients were enrolled from late October 2008 to early February 2009, so that the effect of terrestrial sunlight on serum 25(OH)D level would be negligible. Dublin, Ireland (53°N), is at a similar latitude to Edmonton, Alberta, Canada (52°N), where Webb et al demonstrated that the skin production of vitamin D is absent from mid-October to mid-April. An equal number of patients with psoriasis not treated with phototherapy served as a control population. Thirty consecutive patients with chronic plaque psoriasis referred for NB–UV-B phototherapy in our department were enrolled. For a matched control for each participant, we selected a patient with psoriasis from the outpatient clinic who had not been treated with phototherapy in the previous 6 months. Each control patient was recruited within 1 week of his or her matched treated patient to control for seasonal variation in serum 25(OH)D levels. Exclusion criteria were as follows:

For patients treated with phototherapy:
- Systemic therapy within the previous 6 months for psoriasis
- Abnormal photosensitivity
- Previous failure of or intolerance to phototherapy

Ethical approval for the study was obtained from the ethics and medical research committee of St Vincent’s University Hospital. All patients gave informed written consent.

**PRETREATMENT ASSESSMENT**

Information on demographics and psoriasis history was recorded. A full medical history and a record of medications, daily intake of milk, use of vitamin supplements, and hours of sun exposure per week were also taken. The psoriasis area and severity index (PASI) and dermatology life quality index (DLQI) were measured.

The baseline metabolic profile included levels of serum ionized calcium, serum phosphate, serum alkaline phosphatase (ALP), serum 25(OH)D, and serum intact parathyroid hormone (PTH). Serum 25(OH)D was measured by a competitive radioimmunoassay (Immunodiagnostic Systems Limited, Tyne and Wear, England). Serum intact PTH was measured by electrochemiluminescent immunoassay on the Cobas e411 platform (Roche Diagnostics, Basel, Switzerland). Interassay and intra-assay percentage coefficients of variation are available on request.

**TREATMENT REGIMEN AND FOLLOW-UP ASSESSMENTS**

The minimal erythema dose (MED) was recorded in the phototherapy arm as previously described. Whole-body NB–UV-B was given in a Waldmann UV 5001 cabinet (Villingen-Schwenningen, Germany) incorporating forty 100-W TL-01 fluorescent lamps (Philips Co., Eindhoven, the Netherlands) (311-313 nm). Greater than 90% of body surface area (BSA) was exposed, with the exception of the genital region in men. Phototherapy was administered 3 times a week (Monday, Wednesday, and Friday) as per a standard well-established protocol in our department:

| First dose | 70% of MED |
| Effects of erythema on next dose: |
| Grade 1: Minimal perceptible erythema; same dose as previous visit; after 2 consecutive grade 1 erythemas, 10% increments |
| Grade 2: Asymptomatic well-defined erythema; postpone next exposure until resolution of erythema; 10% increments thereafter |
| Grade 3: Painful erythema persisting >24 h; postpone next exposure until resolution of erythema; next dose 50% of burning dose; 10% increments thereafter |

Permitted concomitant treatments:
- Aqueous cream (according to the British National Formulary [BNF])
- Hydrocortisone cream, 1%, for flexural psoriasis
- Tar pomade (BNF) for scalp psoriasis
- Irradiance measured each month using an IL1400A radiometer and calibrated sensor (calibrated annually against a reference standard at A. Coleman, Medical Physics, Guys and St Thomas’ Hospital Trust, London, England)

Participants were treated with NB–UV-B until clearance of their psoriasis. Clearance was defined as complete resolution of psoriasis or minimal residual activity of psoriasis (<1% BSA). Each control completed the study when his or her corresponding patient achieved clearance of disease. Participants were as-
sessed at baseline, at exposure 12 (4 weeks), and at clearance of psoriasis with PASI; DLQI; and levels of serum ionized calcium, serum phosphate, serum ALP, serum 25(OH)D, and serum intact PTH. Controls were assessed at baseline, 4 weeks, and when their corresponding participants reached the end point of the study. One control could not attend his final assessment at the time his corresponding participant completed the study.

### STATISTICAL ANALYSIS

Quantitative variables are expressed as median (range) unless otherwise stated. Normality was tested by D'Agostino-Pearson omnibus test. Serum 25(OH)D and serum PTH levels were log-transformed prior to analyses. Differences between groups were tested according to parametric and nonparametric tests, where appropriate. For binary variables, Fisher exact test was used to compare the 2 groups. Corrections for multiple comparisons were made according to Bonferroni or Dunn, where appropriate. Pearson or Spearman rank correlations, where appropriate, were calculated. We conducted a forward multiple regression analysis to identify determinants of phototherapy-induced change in both PASI and serum 25(OH)D level in the treatment group, and to identify determinants of baseline vitamin D status in all patients with psoriasis. P < .05 was considered significant. GraphPad Prism (version 5.01 for Windows; GraphPad Software, San Diego, California) was used for all statistical analyses except for correlation and regression analysis, which was performed using SPSS software for Windows (version 16.0; SPSS Inc, Chicago, Illinois).

### RESULTS

#### PATIENT DEMOGRAPHICS AND BASELINE ASSESSMENTS

In total, 60 patients were recruited to the study: 30 patients to be treated with NB–UV-B and 30 controls. One patient and her corresponding control were withdrawn from the study because she was found to have photoaggravated psoriasis (one of the exclusion criteria). Another control could not attend his last assessment. One control could not attend his final assessment at the time his corresponding participant completed the study. Control. This table was used to record dosimetry in the phototherapy arm in addition to the baseline demographic/psoriasis history comparison between phototherapy arm vs control arm.

### Table 1. Demographic Data, Baseline Assessment, and Dosimetry

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Treatment Group (n=29)</th>
<th>Control Group (n=29)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex ratio, male: female, No.</td>
<td>13:16</td>
<td>18:11</td>
<td>.29</td>
</tr>
<tr>
<td>Age, y</td>
<td>40 (22-64)</td>
<td>45 (21-75)</td>
<td>.10</td>
</tr>
<tr>
<td>Body weight, kg</td>
<td>74.2 (50.3-103.8)</td>
<td>79.3 (55.0-127.4)</td>
<td>.01</td>
</tr>
<tr>
<td>BMI</td>
<td>26.9 (19.5-35.9)</td>
<td>27.2 (19.5-42.8)</td>
<td>.17</td>
</tr>
<tr>
<td>Smoking, No. (%)</td>
<td>9 (31)</td>
<td>7 (24)</td>
<td>.77</td>
</tr>
<tr>
<td>Alcohol, U/wk</td>
<td>14 (0-150)</td>
<td>19 (0-70)</td>
<td>.11</td>
</tr>
<tr>
<td>Age at onset of psoriasis, y</td>
<td>17 (4-48)</td>
<td>17 (0.2-68)</td>
<td>.80</td>
</tr>
<tr>
<td>Psoriatic arthritis</td>
<td>0</td>
<td>2 (7)</td>
<td>.49</td>
</tr>
<tr>
<td>Previous phototherapy</td>
<td>20</td>
<td>21</td>
<td>&gt;.99</td>
</tr>
<tr>
<td>NB–UV-B</td>
<td>4</td>
<td>3</td>
<td>.02</td>
</tr>
<tr>
<td>PUGA</td>
<td>19</td>
<td>16</td>
<td>.59</td>
</tr>
<tr>
<td>Baseline PASI</td>
<td>7.1 (4.2-16.1)</td>
<td>3.6 (0-12.5)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Baseline DLQI</td>
<td>10.0 (1-30)</td>
<td>3.0 (0-19)</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Fitzpatrick skin type</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I</td>
<td>3</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>II</td>
<td>6</td>
<td>9</td>
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<tr>
<td>III</td>
<td>19</td>
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<tr>
<td>IV</td>
<td>1</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Time spent outdoors per week, h</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;10</td>
<td>20</td>
<td>18</td>
<td>.58</td>
</tr>
<tr>
<td>11-20</td>
<td>6</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>&gt;20</td>
<td>3</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>MED, mJ/cm²</td>
<td>390 (200-770)</td>
<td>NA b</td>
<td></td>
</tr>
<tr>
<td>First dose, mJ/cm²</td>
<td>299 (144-516)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Last dose, mJ/cm²</td>
<td>2322 (505-3833)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of exposures</td>
<td>18 (11-47)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cumulative dose, mJ/cm²</td>
<td>22 508 (5773-69 458)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: BMI, body mass index (calculated as weight in kilograms divided by height in meters squared); DLQI, dermatology life quality index; MED, minimal erythema dose; NA, not applicable; NB, narrowband; PASI, psoriasis area and severity index; PUVA, psoralen-UV-A.

aData are presented as median (range) unless otherwise indicated.

bFor MED, first dose, last dose, number of exposures, and cumulative dose, only those in the phototherapy arm were treated; hence, there is no comparison with the control arm. This table was used to record dosimetry in the phototherapy arm in addition to the baseline demographic/psoriasis history comparison between phototherapy arm vs control arm.
At baseline, the median (range) PASI in the treatment group was 7.1 (4.2-16.1), which was significantly higher than that of the control group, which had a median score of 3.6 (0-12.5) (P < .001) (Table 1). Similarly, the DLQI was higher in the treatment group at 10 (1-30) compared with 3 (0-19) in the control group (P < .01). All patients in both the treatment group and the control group had serum ionized calcium levels within the reference range (Table 2). One patient in the treatment group and 2 controls had elevated serum ALP levels; the treated patient had known primary biliary cirrhosis, 1 control had alcoholic liver disease, and the cause of elevation of ALP level in the other control was unknown. The treatment group had a higher median serum 25(OH)D level at 23 (9-46) ng/mL compared with 13 (7-33) ng/mL (P = .001). Serum PTH levels were higher in the control group compared with the treatment group (P < .001) (Table 2).

### RESPONSE TO NB–UV-B TREATMENT

The median (range) of MED was 390 (200-770) mJ/cm², with a median first dose of 299 (144-516) mJ/cm², a median final dose of 2322 (505-3833) mJ/cm², and a median cumulative dose of 22 508 (5773-69 459) mJ/cm² (Table 1). Patients were treated with a median of 18 (11-47) exposures of NB–UV-B. The median duration of study was 51 (25-118) days for patients and 52 (28-128) days for controls. Following treatment, the median PASI decreased significantly from 7.1 (4-2-16.1) at baseline to 0.5 (0-2.1) (P < .001) and the median DLQI decreased from 10 (1-30) to 1 (0-5) (P < .001), whereas the median PTH and ALP levels were unchanged throughout the study in both groups (P > .05) (Table 1). There was no correlation between change in serum 25(OH)D levels and change in PASI or change in DLQI. There was no correlation between change in serum 25(OH)D levels and change in PASI or change in DLQI.

VITAMIN D STATUS AND DETERMINANTS

At baseline, 10 patients in the treatment group (34%) and 20 patients in the control group (69%) had serum 25(OH)D levels in the insufficient range, below 20 ng/mL (P = .02). At the end point, all patients in the treatment group were vitamin D sufficient, but 75% of the control group were vitamin D insufficient (although the median serum vitamin D value did not change in the control group). The 2 variables with significant correlations were number of exposures and cumulative NB–UV-B dose. In the treatment group at the end of the study, 2 variables with significant correlations were number of exposures ($r = 0.61; P < .001$) and cumulative dose of NB–UV-B ($r = 0.47; P = .01$). A forward multiple regression model including all of these variables for identifying pre-

### Table 2. Psoriasis and Biochemical Indices at Baseline and During the Studya

<table>
<thead>
<tr>
<th>Index</th>
<th>Treatment Group</th>
<th>Control Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Baseline</td>
<td>Week 4</td>
</tr>
<tr>
<td>PASI</td>
<td>7.1 (4.2-16.1)</td>
<td>2.8 (0-10.6)</td>
</tr>
<tr>
<td>DLQI</td>
<td>10 (1-30)</td>
<td>2 (0-16)</td>
</tr>
<tr>
<td>Serum ionized calcium level mmol/L</td>
<td>1.28 (1.15-1.36)</td>
<td>1.27 (1.19-1.35)</td>
</tr>
<tr>
<td>ALP level, U/L</td>
<td>62 (34-242)</td>
<td>61 (37-268)</td>
</tr>
<tr>
<td>25(OH)D level, ng/mL</td>
<td>12 (7-42)</td>
<td>42 (29-78)</td>
</tr>
<tr>
<td>PTH level, pg/mL</td>
<td>30 (11-73)</td>
<td>21 (13-83)</td>
</tr>
</tbody>
</table>

Abbreviations: ALP, alkaline phosphatase; DLQI, dermatology life quality index; PASI, psoriasis area and severity index; PTH, parathyroid hormone; 25(OH)D, 25-hydroxyvitamin D.

$SI$ conversion factors: To convert ALP to microkatals per liter, multiply by 0.0167; to convert PTH to nanograms per liter, multiply by 1; to convert 25(OH)D to nanomoles per liter, multiply by 2.496.

$^a$ Data are presented as median (range).
dictors of change in serum 25(OH)D level identified the number of exposures as the only significant predictor (P = .001), accounting for 38% of the variance (Table 3).

In the combined group at baseline, serum 25(OH)D levels correlated directly with prior phototherapy (r = 0.36; P < .005), PASI (r = 0.31; P = .02), and skin type (r = 0.3; P = .02), and inversely with BMI (r = −0.27; P = .04) and serum PTH level (r = −0.53; P < .001). In a forward multiple regression model, prior phototherapy was the only variable that predicted baseline serum 25(OH)D level accounting for 13% of the variance (r = 0.36; P = .006) (Table 3).

### Table 3. Forward Multiple Regression Analysis of 3 Factors

<table>
<thead>
<tr>
<th>Variable</th>
<th>β</th>
<th>F</th>
<th>r²</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model 1 (n=27)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Last NB–UV-B dose</td>
<td>0.47</td>
<td>7.35</td>
<td>0.22</td>
<td>.01</td>
</tr>
<tr>
<td>Model 2 (n=27)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exposures, No.</td>
<td>0.61</td>
<td>15.64</td>
<td>0.38</td>
<td>.001</td>
</tr>
<tr>
<td>Model 3 (n=57)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prior phototherapy</td>
<td>0.36</td>
<td>8.13</td>
<td>0.13</td>
<td>.006</td>
</tr>
</tbody>
</table>

Abbreviations: β, standardized beta coefficient; F, F statistic for model; NB, narrowband; PASI, psoriasis area and severity index.

Factors determining change in PASI (model 1), factors determining change in serum 25 hydroxyvitamin D [25(OH)D] level (model 2), and factors determining baseline serum 25(OH)D level (model 3).

In this study, we showed that NB–UV-B markedly increased serum 25(OH)D levels during wintertime while clearing psoriasis. The number of exposures was the sole predictor of increase in serum 25(OH)D level following NB–UV-B, whereas prior phototherapy was the sole predictor of baseline serum 25(OH)D levels in the entire group. There was no correlation between increase in serum 25(OH)D level and treatment response, as determined by the number of exposures required to clear psoriasis or by change in psoriasis measures such as PASI or DLQI. In fact, those who required a greater number of exposures to clear had a significantly higher serum 25(OH)D level, most likely produced by more prolonged exposure to NB–UV-B. We cannot conclude, therefore, that NB–UV-B mediates its therapeutic effects by increasing vitamin D levels. This suggests that the improvement in both vitamin D status and psoriasis are contemporaneous, but unrelated, consequences of NB–UV-B, or that there is another explanation for the causal relationship.

One possible explanation could be the intradermal production of 1,25(OH)₂D. By increasing the substrate supply of vitamin D, NB–UV-B therapy could indirectly enhance cutaneous production of 1,25(OH)₂D. To our knowledge, in previous studies, no relationship was found between levels of serum 25(OH)D and psoriasis severity, but in these studies a negative correlation was found between the severity of psoriasis and the basal serum level of 1,25(OH)₂D. We did not measure serum 1,25(OH)₂D levels, but, because the effect of 1,25(OH)₂D is intracrine in nature, serum levels may not reflect intradermal production.

We identified a substantial benefit of NB–UV-B on vitamin D status in patients with psoriasis. Two other studies have shown that NB–UV-B increases serum 25(OH)D levels in patients with psoriasis. In a study comparing the effect of BB–UV-B and NB–UV-B treatment on vitamin D synthesis, 26 patients were treated with BB–UV-B and 42 with NB–UV-B for 8 to 12 weeks. Serum 25(OH)D levels increased from 37.9 ± 16.9 ng/mL to 69.4 ± 19.7 ng/mL (P < .001) in patients treated with BB–UV-B compared with 34.8 ± 11.9 ng/mL to 55.3 ± 17.6 ng/mL in the NB–UV-B group (P < .001), with a significantly higher increase in the BB–UV-B group (P = .008). Thirty-nine of these patients were treated in spring (March–June) and 29 were treated in winter (November–March). There was no control group in this study, so the authors could not control for intraseasonal variation in vitamin D within the winter period. From November to March there may be considerable variation in baseline serum 25(OH)D values for a given individual. In our study, matching controls were recruited within a week of their corresponding patient to control for this fluctuation.

In the study by Osmancevic et al., patients were treated with both BB–UV-B and NB–UV-B. Both groups achieved clearance at a similar total number of exposures. In clinical practice, the number of NB–UV-B MEDs needed to clear psoriasis is less than one-third of that needed to clear psoriasis using BB–UV-B. This may indicate that the morphologic characteristics or extent of psoriasis differed between the 2 groups; for instance, the selection of the UV lamp was based on patients’ previous experience of the respective therapy. Another small study compared 25(OH)D levels in 7 patients treated with NB–UV-B for psoriasis or dermatitis to 7 patients or physicians with vitamin D deficiency treated with 50,000 IU of vitamin D per week during wintertime in Melbourne, Australia. This showed NB–UV-B to be an effective means of raising serum 25(OH)D levels.

The prevalence of vitamin D insufficiency in untreated patients with psoriasis during wintertime in Ireland is highlighted in this study. This is a common problem in all Irish persons in our high-latitude region that has been known for some time. In the combined group...
at baseline, serum 25(OH)D levels correlated directly with prior phototherapy, skin type, and PASI, and correlated inversely with BMI. Previous studies have shown that those with increased BMI have a lower serum 25(OH)D level.29,30 Our study suggests an inverse relationship between BMI and baseline serum 25(OH)D level. Body fat sequesters vitamin D; obese individuals exposed to a tanning bed UV light produce a lesser amount of 25(OH)D compared with nonobese individuals.30 In our study, those with more severe psoriasis, as measured by higher PASI, had a higher serum 25(OH)D level in the initial analysis, but this was not confirmed in the multiple regression model. Prior phototherapy predicted higher vitamin D levels at baseline. This was confirmed in the multiple regression model. This may be because the effect of prior phototherapy on vitamin D levels lasted longer than 6 months. Those who had seen the benefit of previous phototherapy may have also actively sought out the Irish sun during the preceding summer, resulting in marginally higher baseline vitamin D levels. Controls receiving systemic treatment may have been less likely to sunbathe because they were advised to avoid sunbathing. They may also have been less inclined to sunbathe because their disease was less severe.

At the end point of our study, all patients in the treatment group were vitamin D sufficient, showing that treatment with NB–UV-B corrects vitamin D insufficiency. Many achieved high serum 25(OH)D levels: 24 of 29 (83%) had levels above 40 ng/mL, a threshold that we have suggested previously as a desirable and safe level.31 None of the patients developed hypercalcemia, even though 20% achieved levels above 72 ng/mL. In comparison, three-quarters of the control group had vitamin D insufficiency at the end of the study, reflecting the wintertime vitamin D status of the general Irish population. This supports the findings of previous studies advocating fortification of milk with vitamin D in high-latitude European countries to diminish the wintertime fall in serum 25(OH)D levels.32,33 In addition, supplementation in Irish patients with psoriasis during the winter months is warranted; for instance, it is estimated that healthy adults need a daily supplement of vitamin D, 28 µg (1100 U), to maintain a serum 25(OH)D level higher than 20 ng/mL during wintertime.33 Our observation of a significant inverse correlation between serum 25(OH)D and serum PTH levels, as per previous studies, indicates an adverse effect of hypovitaminosis D in our patients with psoriasis during winter.31,34

In summary, this study shows that NB–UV-B effectively increases serum 25(OH)D levels while clearing psoriasis, the magnitude of increase being determined by the number of exposures. Up to 75% of Irish patients with psoriasis were shown to have vitamin D insufficiency during wintertime, highlighting the need for seasonal supplementation to prevent the deleterious effects of hypovitaminosis D in this population unless phototherapy is being administered.

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Author Contributions: All authors had full access to all of the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis. Study concept and design: Ryan, Moran, McKenna, Murray, and Kirby. Acquisition of data: Ryan, Moran, Brady, Collins, Rogers, and Kirby. Analysis and interpretation of data: Ryan, Moran, McKenna, Murray, Brady, and Kirby. Drafting of the manuscript: Ryan, McKenna, and Kirby. Critical revision of the manuscript for important intellectual content: Moran, McKenna, Murray, Brady, Collins, Rogers, and Kirby. Statistical analysis: McKenna. Administrative, technical, and material support: Ryan and Murray. Study supervision: McKenna and Kirby.

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REFERENCES


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Correction

Errors in Abstract and Table 2. In the Study by Ryan et al titled “The Effect of Narrowband UV-B Treatment for Psoriasis on Vitamin D Status During Wintertime in Ireland,” published in the August 2010 issue of the Archives (2010;146[8]:836-842), the first sentence of the “Results” section in the abstract should have read as follows: “Levels of serum 25(OH)D increased significantly (P<.001) from a median (range) of 23 (9-46) ng/mL at baseline to 31 [rather than 59, as given in the originally published article] (32-112) ng/mL at the end of NB–UV-B treatment compared with no change in the control group.” In Table 2, the final 25(OH)D level for the treatment group and the baseline 25(OH)D level for the control group were transposed and should have read 51 (32-112) and 13 (7-33) ng/mL, respectively.