Assessment of plasma 25-hydroxyvitamin D response to ultraviolet irradiation over a controlled area in young and elderly subjects

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Summary

1. The response of 25-hydroxyvitamin D [25-(OH)D] to artificial ultraviolet irradiation applied to a known area of dorsal skin was investigated in 18 subjects, small quantities of ultraviolet energy being used. Ultraviolet irradiation was administered on days, 1, 3, 5, 8, 10, 12, 15 and 17, a total of 15 min being delivered over this time. In 15 subjects plasma 25-(OH)D showed a significant increase after a total of 15 min exposure but three subjects failed to demonstrate any increment. Plasma 25-(OH)D did not increase in any subject after 2.5 min of ultraviolet irradiation (irradiation on days 1 and 3).

2. Responses were compared in young and old, in male and female and in normal and osteomalacic subjects. No significant difference in response was found between these groups.

3. When plasma volume was taken into account, it was possible to calculate the increase in nmol of plasma 25-(OH)D/cm² skin irradiated. This was 0.024 nmol/cm² with no sex difference, over the 17 days of irradiation.

4. Exposure to ultraviolet irradiation over a small area of dorsal skin led to a rapid rise of plasma 25-(OH)D in most subjects with a subsequent plateau in subjects studied for up to 15 min total exposure. This contrasted with the prolonged increase in plasma 25-(OH)D continuing over several weeks in response to whole-body ultraviolet irradiation. This may indicate that cutaneous synthesis of vitamin D is rapid but limited, and that the considerable rise in plasma 25-(OH)D during whole-body irradiation may originate from vitamin D synthesized during the first few exposures.

Key words: osteomalacia, skin, ultraviolet irradiation, vitamin D.

Abbreviation: 25-(OH)D, 25-hydroxyvitamin D.

Introduction

Vitamin D is derived from dietary sources and the action of the ultraviolet spectrum of sunlight in skin. Although the value of sunlight in healing rickets has been known for 60 years (Huldschinsky, 1919), the relative importance of sunlight and diet in contributing to vitamin D repletion in a normal environment is not yet well understood. The ability to measure 25-hydroxyvitamin D [25-(OH)D], the main circulating metabolite of vitamin D, in plasma from large numbers of subjects (Haddad & Chyu, 1971; Edelstein, Charman, Lawson & Kodicek, 1974) led to the description of seasonal variation in plasma concentrations of 25-(OH)D (Stamp & Round, 1974). A daily vitamin D intake of 12.5 nmol/day (200 i.u.) leads to little change in concentrations of plasma 25-hydroxyergocalciferol (Foskitt, Cole & Lawson, 1979) and 31.3 nmol (500 i.u.)/day leads to a small, though statistically significant, increase (Somerville, Lien & Kaye, 1977). Since the daily intake of many people in England is considerably less than this (Exton-Smith, Hodkinson & Stanton, 1966), sun-
light appears to be important in maintaining vitamin D repletion. The amount of vitamin D formed in skin in response to ultraviolet irradiation varies considerably (Bekemeier, 1959; Bekemeier & Pfennigsdorf, 1959) and some may be degraded by further exposure to ultraviolet irradiation (Bekemeier, 1966). The amount of vitamin D appearing in skin in response to ultraviolet irradiation in vitro (Bekemeier, 1958) and in vivo (Bekemeier, 1966) has been measured, but not the ability of skin to respond to ultraviolet irradiation over a long period of time. Stamp, Haddad & Twigg (1977) have compared the 25-(OH)D response in plasma after whole-body irradiation with the response seen after various oral doses of vitamin D. However, the irradiation continued only for 3 weeks and the 25-(OH)D response curve to ultraviolet irradiation suggested that the oral vitamin D equivalent might have been considerably greater if observations had been continued until the maximum response to the irradiation schedule had been reached.

In addition little attention has been paid to the effect of age on the response to ultraviolet irradiation. An increased incidence of vitamin D deficiency is found amongst the elderly population (Chalmers, Conacher, Gardner & Scott, 1967; Corless, Beer, Boucher, Gupta & Cohen, 1975) and the increase in plasma 25-(OH)D in the summer may be reduced in this age group (Lester, Skinner & Wills, 1977).

In the present paper, we have investigated in detail the response of the plasma 25-(OH)D to ultraviolet irradiation and measured the response/unit area of skin. We have compared the response in young and old people and two patients with osteomalacia, and suggest that the dermal synthesis of vitamin D is rapid, but limited.

**Subjects and methods**

**Subjects**

Normal subjects were volunteers aged 20–45 years. All were white-skinned except one, who was an Indian/Asian female. Four subjects were female and five were male.

Elderly subjects consisted of three men (mean age 74 years) and five women (mean age 80-2 years). All were in the long-stay wards of a geriatric hospital and had cerebrovascular disease (four), treated heart failure (two), Parkinsonism (one) or post-gastrectomy osteomalacia (one). Care was taken to ensure that no enzyme-inducing hypnotic was given.

One subject with osteomalacia due to anti-convulsant drugs was also included. She was aged 14 years, and had tuberous sclerosis and epileptic fits, for which she took myosine (500 mg/day).

All subjects taking part in these studies were interviewed and the project was explained to them. Informed consent was obtained from all taking part or from their guardian.

Ethical approval was obtained for the ultraviolet irradiation studies and for the estimation of plasma volume by 129I-labelled albumin in elderly subjects only.

**Irradiation procedures**

Ultraviolet irradiation for the subjects undergoing limited body irradiation (see below) was produced from an Hanovia 7A prescription lamp. Details of the spectrum (mercury arc) are shown in Table 1, together with comparisons of the relative efficacy of each line for converting 7-dehydrocholesterol into pre-vitamin D. The lamp was placed 50 cm from the back. Whole-body irradiation was carried out with four Westinghouse FS40 lamps with a power spectrum as for FS20 lamps described by Corless, Gupta, Switala, Barragry, Boucher, Cohen & Diffey (1978).

**Limited area.** In both the limited area irradiation studies dorsal skin was irradiated, the same area being irradiated on each occasion.

(a) In the first limited area study three groups of subjects were studied. All were normal young subjects. In all groups blood was taken for 25-(OH)D estimation before ultraviolet irradiation commenced. Group 1 (three subjects) received 1 min ultraviolet irradiation on day 1 and 1-5 min on day 3. Blood was taken on day 4. Group 2 (four subjects) received 1 min ultraviolet irradiation on day 1, 1 min on day 3 and 1-5 min on day 5, blood being taken on day 6. Group 3 (four subjects) received 1 min ultraviolet irradiation on day 1, 1 min on day 3, 1-5 min on day 5 and 1-5 min on day 8. Blood was taken on day 9. In this study 600 cm² of skin was irradiated.

In the second limited area study (over 600 cm² or 900 cm² of dorsal skin) the protocol described for group 3 in the first study was extended. Nine normal subjects and eight elderly subjects (including one with osteomalacia) were studied. Thus, after day 8, 2-5 min ultraviolet irradiation was administered on day 10, 2-5 min on day 12, 2-5 min on day 15 and 2-5 min on day 17. A total of 15 min of ultraviolet exposure was given over eight exposures. Blood was taken before the first ultra-
Plasma 25-hydroxyvitamin D response to ultraviolet irradiation

TABLE 1. Power of Hanovia 7A prescription lamp

<table>
<thead>
<tr>
<th>Wavelength (nm)</th>
<th>Power at 50 cm from lamp* (μW/cm²)</th>
<th>Energy efficiency cf. 280 nm (%)</th>
<th>Relative power cf. 280 nm (μW/cm²)</th>
<th>Conversion efficiency cf. 295 nm (%)</th>
<th>Relative power cf. 295 nm (μW/cm²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>254</td>
<td>40</td>
<td>0</td>
<td>27.2</td>
<td>10.9</td>
<td>245.3</td>
</tr>
<tr>
<td>265</td>
<td>75</td>
<td>79</td>
<td>59.3</td>
<td>31.2</td>
<td>10.9</td>
</tr>
<tr>
<td>297</td>
<td>55</td>
<td>81</td>
<td>44.6</td>
<td>23.4</td>
<td>23.4</td>
</tr>
<tr>
<td>303</td>
<td>90</td>
<td>39</td>
<td>35.1</td>
<td>52.8</td>
<td>82.8</td>
</tr>
<tr>
<td>313</td>
<td>120</td>
<td>1</td>
<td>1.2</td>
<td>75.4</td>
<td>52.8</td>
</tr>
<tr>
<td>380</td>
<td>380</td>
<td>140.2</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Energy over 1 min (mJ) 22.8 8.4 14.7

* Manufacturers' data.

violet exposure and either on days 6, 9, 11, 13 and 18 (13 subjects) or on day 18 only (five subjects). Blood was taken about 18 h after exposure.

The subject with anticonvulsant osteomalacia received an exposure to ultraviolet irradiation every Monday, Wednesday and Friday until 22 exposures had been completed. No single exposure lasted for more than 4 min and total ultraviolet exposure time was 70 min. Blood was taken the day after an exposure.

(b) Whole-body irradiation was administered to the back and front of four normal female volunteer subjects aged 20—25 years every Monday, Wednesday and Friday for 11 weeks. Blood was taken 2 days after an irradiation.

All normal subjects were irradiated in December or March to avoid coincidental ultraviolet irradiation. Some of the elderly subjects were irradiated in the summer, and during this period did not go outside the hospital ward. The amount of ultraviolet irradiation received by such subjects is very small (Challoner, Corless, Davis, Deane, Difley, Gupta & Magnus, 1976).

Plasma volume and surface area

Plasma volume was measured by 125I-labelled albumin dilution (The Radiochemical Centre, Amersham) 10 min after injection of 144 kBq of 125I-labelled albumin in the elderly subjects. In the subjects aged 45 years or less plasma volume was estimated from a nomogram connecting weight and height with blood volume. The packed cell volume was used to give the plasma volume. Surface area was calculated from a nomogram connecting height and weight (Davidson, Passmore, Brock & Truswell, 1975).

Plasma 25-(OH)D estimation

Plasma 25-(OH)D concentrations were measured by the method of Edelstein et al. (1974). 25-Hydroxyergocalciferol concentrations are about 6-5 nmol/l in normal subjects (Poskitt et al., 1979). Analysis of six replicate samples of plasma gave a mean 25-(OH)D value of 34.7 ± 0.85 nmol/l. A change in plasma 25-(OH)D concentration of more than twice this standard deviation was regarded as significant when maintained in consecutive measurements. All plasma samples from an individual subject were analysed in the same assay. An aliquot of plasma sample of known 25-(OH)D concentration was included with each batch of unknowns and the values obtained were corrected for any inter-assay variation. Since the amount of vitamin D present in the diet appears to play a minor role in maintaining plasma 25-(OH)D concentrations (Poskitt et al., 1979), no effort was made to prohibit vitamin D from the diet.

Statistical analysis employed standard parametric and non-parametric tests. Correlation was examined by the Pearson r test. When the relationship of change of plasma 25-(OH)D after ultraviolet irradiation with the initial plasma 25-(OH)D concentration was examined, it was necessary to use the formula [plasma 25-(OH)D before ultra-
violet irradiation + plasma 25-(OH)D after ultraviolet irradiation] divided by 2, instead of initial plasma 25-(OH)D, in order to avoid a spurious relationship being found (Oldham, 1968).

Results

Plasma 25-(OH)D concentrations were measured in a group of young and old people from among whom the subjects used in this study were selected. The mean values of the young and old men were 26.5 ± SD 16.0 nmol/l and 25.3 ± 14.8 nmol/l respectively and those for the young and old women were 28.5 ± 3.5 nmol/l and 20.0 ± 4.3 nmol/l respectively.

The non-osteomalacic elderly subjects irradiated in summer had a similar plasma 25-(OH)D concentration (19.5 ± 4.1 nmol/l) to a group irradiated in December (25.0 ± 1.0) and the increment of plasma 25-(OH)D in all elderly subjects was similar (11.4 ± 5.5 winter; 10.3 ± 3.9 summer). This suggested that the group in the summer were similar to the winter group and that extraneous ultraviolet irradiation was playing no part in the plasma 25-(OH)D response.

The first limited exposure study was carried out to establish the minimum exposure to ultraviolet irradiation of a known area of dorsal skin that was required to produce a significant response in plasma 25-(OH)D concentrations. No significant response was observed in any subject exposed for 2-5 min (group 1). After 3-5 min (group 2), two subjects out of four showed a small response which was not statistically significant, but after 5 min irradiation (group 3) the plasma 25-(OH)D concentrations in all subjects had increased by 10.8 ± 1.3 nmol/l (P < 0.01, paired t-test).

With the demonstration that this system for controlled irradiation produced significant changes in plasma 25-(OH)D values, a second limited exposure study was undertaken in which 18 subjects were followed for up to 15 min of exposure time (Table 2). The results show that, in most subjects, a significant increase in plasma 25-(OH)D concentrations occurred in subjects irradiated for a total of 5 min. The variable nature of the response of these subjects is apparent from the wide SD of the values in some groups. Much of this wide variation is due to the different rates at which the subjects responded. For example, of 13 subjects studied at time intervals before 15 min of exposure time, eight showed a significant increase (see the Subjects and methods section) in plasma 25-(OH)D concentrations after 5 min irradiation, and three subjects did not show any consistent increase in plasma 25-(OH)D even after 15 min of ultraviolet irradiation (non-responders). The two subjects with osteomalacia and the Indian/Asian woman all responded to ultraviolet irradiation within 5 min of exposure time. There was no difference in changes in plasma 25-(OH)D concentrations between the age groups.

Since the changes in plasma 25-(OH)D were relatively small, it was of interest to assess the reproducibility of the response. Four subjects were re-irradiated in an identical fashion after 4 months. The change in plasma 25-(OH)D concentration on the first occasion (−2.5; 4.8; 11.9; 7.3 nmol/l) was very similar to the change on the second occasion (0.5; 9.0; 7.5; 7.5 nmol/l respectively). Thus two subjects showed almost identical responses and the subject who failed to respond on the first occasion behaved in the same fashion on re-irradiation.

### Table 2. Plasma 25-(OH)D response to ultraviolet irradiation

Mean values ± SD are given with numbers in parentheses. N.S., Not significant.

<table>
<thead>
<tr>
<th>Subject</th>
<th>Change in 25-(OH)D from zero time (nmol/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exposure time (min) . . .</td>
<td>3.5</td>
</tr>
<tr>
<td>Young female</td>
<td>7.1 ± 2.7 (4)</td>
</tr>
<tr>
<td>Elderly female</td>
<td>–3.5 (1)</td>
</tr>
<tr>
<td>Young male</td>
<td>2.1 ± 4.8 (3)</td>
</tr>
<tr>
<td>Elderly male</td>
<td>6.8 ± 5.7 (2)</td>
</tr>
<tr>
<td>All female</td>
<td>4.1 ± 5.6 (2)</td>
</tr>
<tr>
<td>All male</td>
<td>4.0 ± 5.1 (2)</td>
</tr>
<tr>
<td>P (male vs female)</td>
<td>N.S.</td>
</tr>
</tbody>
</table>
Plasma 25-hydroxyvitamin D response to ultraviolet irradiation

3.5
7.5
Cumulative exposure time (min)

FIG. 1. Change in plasma 25-(OH)D concentration from zero time/min of ultraviolet irradiation exposure time (mean ± SEM) plotted against the total ultraviolet irradiation exposure time for exposure times 3.5, 5.0, 7.5, 10.0 and 15.0 min (log, scale), for males (○—○) and females (△—△). Subjects who showed no rise in plasma 25-(OH)D concentration during ultraviolet irradiation (see the Results section) are excluded.

<table>
<thead>
<tr>
<th>No. of exposures</th>
<th>Plasma concn. of 25-(OH)D (nmol/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subject no.</td>
<td>1</td>
</tr>
<tr>
<td>0</td>
<td>27</td>
</tr>
<tr>
<td>4</td>
<td>43-8</td>
</tr>
<tr>
<td>7</td>
<td>36-8</td>
</tr>
<tr>
<td>19</td>
<td>82-0</td>
</tr>
<tr>
<td>28</td>
<td>75-0</td>
</tr>
<tr>
<td>32</td>
<td>99-0</td>
</tr>
</tbody>
</table>

Although 25-(OH)D in plasma had not changed after 2.5 min of ultraviolet irradiation exposure time (see above), plasma 25-(OH)D rose rapidly thereafter. The rate of increase was greatest over the first 5 min of exposure time and subsequently declined (Fig. 1). In the subjects who responded by 15 min the mean increase in plasma 25-(OH)D concentration/min of exposure time to ultraviolet irradiation was calculated for each total exposure time (3.5, 5.0, 7.5, 10.0, 15.0 min) and plotted against the cumulative exposure time to ultraviolet irradiation. The initial irradiation was the most effective in leading to an increase in plasma 25-(OH)D and exposure to ultraviolet irradiation for more than 5 min became progressively less efficient in increasing plasma 25-(OH)D concentrations.

The rather limited response under this irradiation system contrasts with the major changes in plasma 25-(OH)D seen in subjects undergoing prolonged whole-body irradiation. The results in Table 3 show the increase of plasma 25-(OH)D in four young women exposed to ultraviolet light three times a week for up to 3 months. The change in plasma 25-(OH)D ranged from 72 to 137 nmol/l, concentrations which were much higher than were seen in the limited area irradiation experiments.

The increase in plasma 25-(OH)D concentrations after 15 min exposure was greater in females than in males (n = 15, P < 0.05, Mann–Whitney U test) (Table 2). This difference may be eliminated either by removal of the non-responding males or by taking plasma volume into account. A significant negative correlation was found between the change in concentration of plasma 25-(OH)D/cm² skin irradiated and the plasma volume (r = −0.56, P < 0.05). By taking the product of the plasma volume and the maximal increment of plasma 25-(OH)D during the 15 min exposure an estimate of total 25-(OH)D in plasma was obtained. When this was divided by the area irradiated the mean response was 0.024 ± 0.018 nmol/cm² in males.
and $0.024 \pm 0.008$ nmol/cm$^2$ in females. The frequency distribution of the sexes combined approximated to a Gaussian distribution.

The importance of assessing response in terms of total 25-(OH)D produced can be seen in Figs. 2(a) and 2(b). Fig. 2(a) shows the change in plasma 25-(OH)D concentration (nmol/l) in a subject with plasma volume of 0.94 litre and one with a plasma volume of 2.76 litre during exposure to ultraviolet irradiation. The maximum change in 25-(OH)D concentration in the smaller subject is 2.5 times that of the larger subject. This could be construed as a significant difference, but when the plasma volume and area irradiated are considered, the maximal response of the smaller subject is only 1.3 times that of the larger subject (Fig 2b).

**Discussion**

The first study of the response of plasma 25-(OH)D to irradiation of a limited area showed that the system was capable of producing significant increases in plasma 25-(OH)D concentration. Moreover, it also revealed that plasma 25-(OH)D had not increased on day 4 (after 2-5 min of ultraviolet irradiation). It was only after 3-5 min ultraviolet irradiation, when blood was taken 5 days after the first administration of ultraviolet irradiation, that plasma 25-(OH)D was increasing. This may mean that 3-5 min of ultraviolet energy over this area is required to produce a significant increase in plasma 25-(OH)D. Alternatively, there may be a delay in the release of cholecalciferol (or precholecalciferol) from skin.

The results from longer periods of exposure of the same area of skin indicate that the plasma 25-(OH)D response to ultraviolet irradiation over a limited area of skin is restricted. In many cases maximum increments of plasma 25-(OH)D are reached before 15 min of total exposure time have elapsed (Table 2). Moreover, the most rapid change in plasma 25-(OH)D follows the initial exposures (Fig. 1). The failure of plasma 25-(OH)D to continue increasing in this group could be due to a number of factors. It is possible that hepatic 25-hydroxylation could be limiting as plasma 25-(OH)D concentration rises (Bhattacharya & De Luca, 1973); or that alternative metabolism to prevent vitamin D intoxication may become important; or that vitamin D is destroyed in the skin by continued ultraviolet irradiation (Bekemeier, 1966).

It is unlikely that hepatic 25-hydroxylation is limiting. Firstly subjects undergoing whole-body irradiation demonstrated a much more rapid rise of plasma 25-(OH)D compared with the limited area subjects. Secondly, no correlation existed between prevailing plasma 25-(OH)D (see Subjects and methods section) and the change in plasma 25-(OH)D concentration in the subjects undergoing limited area irradiation. This suggests that lack of precursor (vitamin D), not inhibition of hepatic 25-hydroxylation, is the cause of the restricted rise.
The implication of this is that vitamin D synthesis in skin is limited and confined to the initial exposures. However, in the subjects undergoing whole-body irradiation, there may be some constraint on 25-(OH)D appearing in plasma. If the figure for 25-(OH)D appearance/cm² of skin (0.024 nmol/cm²) in the limited area subjects is applied to the whole-body irradiated subjects, a total of about 360 nmol of 25-(OH)D should appear in plasma, producing a rise in plasma 25-(OH)D concentration of about 175 nmol/l. Only one subject approached this value, and this occurred over 3 months. This suggests that the deficit may be constituted by cholecalciferol, and preliminary work measuring this metabolite directly suggests that this is the case (unpublished data).

The capacity of plasma, compared with other organs, to hold vitamin D activity [as 25-(OH)D] has been repeatedly observed (Quarterman, Dal-garno, Adam, Fell & Boyne, 1964; Rosenstreicht, Rich & Volwiler, 1971).

The possibility that ultraviolet irradiation was breaking down vitamin D is also unlikely at the energy used. No destructive effect in vivo in the rat was found with 95 J/cm² (Bekemeier, 1966) and it seems unlikely that the energy acquired after 15 min exposure (0.31 J/cm²) would lead to degradation.

The quantities of vitamin D synthesized by skin are very small compared with the concentration of precursor. Up to 5 nmol of 7-dehydrocholesterol/cm² has been found in human skin (Bekemeier, 1966; D. Fraser & M. Davie, unpublished work). Although irradiation of human or animal skin in vitro produces higher values (Bekemeier, 1958, 1959; Bekemeier & Pfennigsdorf, 1959; Bekemeier, 1966), human skin subjected to ultraviolet light in vivo produced about 25 ng (0.063 nmol) of vitamin D anti-rachitic activity/cm², a figure close to the mean value for 25-(OH)D appearance/cm² (0.024 nmol) calculated from the present data. The rapid but limited response of plasma 25-(OH)D in the limited area subjects implies that there may be a small pool of 7-dehydrocholesterol, probably in the upper layers of the epidermis, that is available for immediate conversion into vitamin D in response to ultraviolet irradiation, and that this pool is only slowly replenished. In keeping with this hypothesis is the finding that irradiation of the rat yields a considerable rise of plasma 25-(OH)D without much change in epidermal 7-dehydrocholesterol (Ohata, Sakagami & Fujita, 1977; Yasumura, Okano, Mizuno & Kobayashi, 1977).

The results also suggest that the response to ultraviolet irradiation is similar in men and women, a finding confirmed by a recent study of larger numbers of male and female subjects (M. Davie & D. E. M. Lawson, unpublished work). Several surveys have suggested that plasma 25-(OH)D concentrations are lower in the elderly (Corless et al., 1975) and that the seasonal variation is diminished (Lester et al., 1977). Elderly women may be particularly at risk of getting osteomalacia (Chalmers et al., 1967) and it is of interest that, in the small numbers in the present study, elderly women, but not men, had a significantly lower plasma 25-(OH)D compared with normal young persons of the same sex. A previous study suggested that elderly subjects were capable of showing a good plasma 25-(OH)D response to ultraviolet irradiation (Corless et al., 1978). The present findings of a similar response of young and old to ultraviolet irradiation indicate that the elderly respond as well as the young, and together with the study of Corless et al. (1978) suggest that low plasma 25-(OH)D concentrations in the elderly are not due to impaired hepatic 25-hydroxylation. It is possible that low plasma 25-(OH)D concentrations in elderly subjects result from failure to go outside or possibly that their skin requires more ultraviolet power to synthesize vitamin D.

Ultraviolet irradiation also seems to contribute more to plasma 25-(OH)D than does oral vitamin D. After 15 min ultraviolet irradiation in the limited area subjects, the mean rise of plasma 25-(OH)D was almost three times the increase of 25-hydroxyergocalciferol seen in subjects who ingested 375 nmol of ergocalciferol (12.5 nmol/day) over 1 month (Poskitt et al., 1979). In another study 31.3 nmol of cholecalciferol orally/day for 2 weeks resulted in a significant, though small, rise of plasma 25-(OH)D concentrations (Somerville et al., 1977). It is, however, possible that oral vitamin D, which is well absorbed (Thompson, Lewis & Booth, 1966), is metabolized differently from that originating in skin (Fraser, 1980).

The present results confirm that the plasma 25-(OH)D response to vitamin D synthesized by ultraviolet irradiation of skin is brisk and of some magnitude. It is, however, limited and it is uncertain how long the plasma concentration of 25-(OH)D will remain elevated after cessation of ultraviolet irradiation. This point is currently being studied. There also may be a difference in the handling of vitamin D from oral or dermal sources, and the recent report of low plasma 25-(OH)D concentrations in patients without osteomalacia (Davie, Lawson & Jung, 1978) suggests that there may be...
other reserves of vitamin D which are important in maintaining vitamin D repletion.

Acknowledgments

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References


