How effective is sunlight in maintaining adequate vitamin D status in Australians?

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Sources of vitamin D
Sunlight – main source

Continued UVR produces overirradiation products, so short exposures more efficient, can't become intoxicated from sunlight.
Electromagnetic Radiation

Ozone disrupts transmission of $\lambda < 290$nm
CIE (Commission Internationale de l’Éclairage) erythemal action spectrum (solid) and the vitamin D weighted action spectrum

ie UVA (λ>320nm) produce sunburn but no vitamin D
noon exposures better outside summer
Sources of vitamin D

Sun exposure to bare skin - main source

but you don’t need much time in sun – though time of day matters.

About a third of the sun exposure that would just cause faint redness, to about ¼ body surface, most days.
Sun exposure times in minutes\(^A\) at 10am or 2pm\(^B\) or 12noon for people with mod. fair skin,\(^C\) which would result in approx 1/3 MED.

<table>
<thead>
<tr>
<th>Region/Time of year</th>
<th>December-Jan(10am)</th>
<th>July-August(12md)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Northern:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cairns</td>
<td>6-7</td>
<td>7</td>
</tr>
<tr>
<td>Townsville</td>
<td>5-7</td>
<td>7</td>
</tr>
<tr>
<td>Central:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Brisbane</td>
<td>6-7</td>
<td>11</td>
</tr>
<tr>
<td>Perth</td>
<td>5-6</td>
<td>15</td>
</tr>
<tr>
<td>Southern:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sydney</td>
<td>6-8</td>
<td>16</td>
</tr>
<tr>
<td>Adelaide</td>
<td>5-7</td>
<td>19</td>
</tr>
<tr>
<td>Melbourne</td>
<td>6-8</td>
<td>25</td>
</tr>
<tr>
<td>Hobart</td>
<td>7-9</td>
<td>29</td>
</tr>
<tr>
<td>New Zealand:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Auckland</td>
<td>6-8</td>
<td>24</td>
</tr>
<tr>
<td>Christchurch</td>
<td>6-9</td>
<td>40</td>
</tr>
</tbody>
</table>

\(A\) Based on 1 MED = 200 J/m\(^2\) effective or 2 Standard Erythemaal Doses for people with type I or II (sensitive skin). Data for Australian cities from Geis et al. (ARPANSA)

\(B\) 11am or 3pm in summertime.

\(C\) Exposure times for people with highly pigmented skin would be 3-6 times x
Simpler message needed

In summer, a few minutes walk in the sun mid-morning or mid-afternoon, most days, with bare arms

In winter, walk in the sun for 20 min (Sydney*) at lunchtime, most days – walk briskly so you can roll your sleeves up

* 7 mins Cairns, 30 min Melbourne, 40 min Christchurch
Does the maintenance UV dose equal 1000IU/day?

- 16 subjects mostly skin type II
- received 1/3 minimal erythemal dose to
  face, arms, hands ~ 18 % body surface
- 4 times/week for 8 weeks

Increase in 25OHD from
48 to 61 nmol/L
increase =13 nmol/L

Cf average increase of
12nmol/L with 1000IU/d *
Vitamin D levels by season

<table>
<thead>
<tr>
<th>Season</th>
<th>Serum 25(OH)D (nmol/L) $^a$</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Summer</td>
<td>67.8 3.4 $^b$</td>
<td>24 - 160</td>
</tr>
<tr>
<td>Winter</td>
<td>58.9 3.8</td>
<td>15 - 174</td>
</tr>
</tbody>
</table>

$^a$ Mean ± standard error

$^b$ Summer significantly different from Winter (P<0.001).

Vitamin D insufficiency by season

<table>
<thead>
<tr>
<th>Serum 25(OH)D</th>
<th>Summer</th>
<th>Winter</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 50 nmol/L</td>
<td>~ 30%</td>
<td>~42%</td>
</tr>
<tr>
<td>&lt; 25 nmol/L</td>
<td>1 person</td>
<td>~ 10% (~ 7 people)</td>
</tr>
</tbody>
</table>
Length of sun exposure relates to 25OHD

During the summer months do you habitually spend hours in the sun aside from vacation?

How often were you outdoors in the last 3 months?
Body surface area exposed determines 25OHD
Causes of vitamin D deficiency (related to sun exposure)

inadequate sun exposure (older people, ill people, indoor lifestyle)
Older people make less vitamin D for a given UV dose?

Less 7-dehydrocholesterol in older skin

BUT: With small doses UV, equal rises in 25OHD in young and old subjects


Adequate increases in 25OHD – 7nmol/l or 19nmol/L after 15 or 30min on open verandah in Spring Auckland daily for 28d

Reid et al Age Ageing 15:35, 1986

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**Figure 4** Circulating concentrations of vitamin D in response to a whole-body exposure to one minimal erythemal dose in healthy young and elderly subjects.

Holick et al, Lancet ii:1104, 1989
The FREEDOM Study

602 people in 51 residential care hostels mean age 86y; 71% female
Randomized to sun exposure calcium (600mg) or routine care
Adherence to sun exposure poor – attendance: 26% of available sessions
poor health, physical constraints, sense of lack of ownership of outdoor spaces
Intention to treat analysis – no effect on falls
In subjects who attended ≥ 130 sessions (>50%): falls signif reduced (0.52, CI 0.31-0.88)
balance signif better (0.43, CI 0.24-0.77)

Sambrook et al, Osteoporosis Int, 2011
Causes of vitamin D deficiency
(related to sun exposure)

inadequate sun exposure (older people, ill people, indoor lifestyle)

pigmentation (darker skin)
Dark skinned individuals (5 and 6) showed high rates of deficiency in both summer and winter.
Causes of vitamin D deficiency
(related to sun exposure)

inadequate sun exposure (older people, ill people, indoor lifestyle)
pigmentation (darker skin)
wearing covering clothing

Sunscreen use?
Sunscreens also reduce D formation under laboratory conditions

BUT – less of a problem in real life

Figure 5  Circulating concentrations of vitamin D after a single exposure to one minimal erythematous dose of simulated sunlight either with a sunscreen, with a sun protection factor of 8, or a topical placebo cream.

Matsouka et al; J Clin Endocrinol Metab, 1987
In a study of 100 office workers, sunscreen users had higher vitamin D levels than non-users*

*Sunscreen users were significantly likely to be outside often, rather than seldom (P<0.02)
Low vitamin D status in highly sun-exposed subjects.

Binkley N et al. JCEM 2007;92:2130-2135

ng/ml to nmol/l
Multiply by 2.5
Factors other than input?

• Storage

• Degradation
  • Accelerated by p450 enzyme inducers
  • Accelerated by low calcium intake

• Genetics
Balance between UV damage and production of vitamin D

• Same UV that produces vitamin D causes skin damage

• Caution for those at high risk of skin cancer

• Evidence that increasing sun exposure increases adaptation to UV
Protection against further sun exposure

• Pigmentation (tan)

• Increased cornified layer
Increased cornified layer in sun-exposed skin
Protection against further sun exposure

- Pigmentation (tan)
- Increased cornified layer
- Upregulated DNA repair pathways
- Synthesis of vitamin D and other D metabolites
Active vitamin D hormone (calcitriol) in skin

- pre-vitamin D$_3$
- vitamin D$_3$

DNA damage:
- Pyrimidine dimers
- Oxidative damage
- Nitrative damage – abasic sites

All are mutagenic /cause cancer

Process takes several hours

Overirradiation products – lumisterol, tachysterol, suprasterols

1,α,25 dihydroxylumisterol = JN

Bikle et al Biochemistry 1986
Lehmann et al JID 2001
Topical 1,25(OH)2D3 (0.1ug/cm2) reduces UVR-induced DNA damage in human subjects.

-Damian et al. Exp Derm 2010-
UV-induced squamous carcinoma incidence is significantly decreased by 1,25(OH)2D and JN*

Summary

- Vitamin D is not strictly a vitamin – main source is sunlight
- Guidelines for sun exposure seem reasonable
- Problems with older people, dark skin, modest dress, others with reduced sun
- Other factors: Physical activity, calcium intake, genetics
- No vitamin D toxicity from sunlight

What about sun damage?

- Unavoidable – so not for people with high risk of skin cancer
- There is some evidence that small amounts of DNA damage are repaired better than large amounts.
- Good evidence for photoadaptation building up over spring
- So short, frequent exposures might be the best balance

- Because vitamin D and metabolites are produced in skin with sunlight, DNA damage from UV is less than it would be otherwise.
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