# Vitamin D in Australia

## Issues and recommendations

**BACKGROUND** A significant number of Australians and people from specific groups within the community are suffering from vitamin D deficiency. It is no longer acceptable to assume that all people in Australia receive adequate vitamin D from casual exposure to sunlight.

**OBJECTIVE** This article provides information on causes, consequences, treatment and prevention of vitamin D deficiency in Australia.

**DISCUSSION** People at high risk of vitamin D deficiency include the elderly, those with skin conditions where avoidance of sunlight is required, dark skinned people (particularly women during pregnancy or if veiled) and patients with malabsorption, eg. coeliac disease. For most people, deficiency can be prevented by 5–15 minutes exposure of face and upper limbs to sunlight 4–6 times per week. If this is not possible then a vitamin D supplement of at least 400 IU* per day is recommended. In cases of established vitamin D deficiency, supplementation with 3000-5000 IU per day for at least 1 month is required to replete body stores. Increased availability of larger dose preparations of cholecalciferol would be a useful therapy in the case of severe deficiencies.

* 40 IU (international units) = 1 μg

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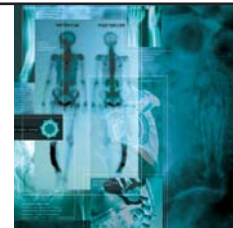
## Vitamin D and health

There is increasing recognition that a significant number of Australians and people from specific groups within the community are suffering from vitamin D deficiency. While the prevalence of vitamin D deficiency varies, it is much higher than previously thought (*Table 1*). The highest rates of hip fracture occur in women over 80 years of age,[1] many of whom are at high risk of vitamin D deficiency. Some studies suggest that mild vitamin D deficiency (insufficiency) can occur at younger ages.[2]

## Vitamin D compounds

Although a number of steroid compounds are classified as vitamin D, for practical reasons this generic term is generally applied to the following two molecules:

- cholecalciferol (vitamin D3) – formed through the action of ultraviolet light on 7-dehydrocholesterol in the skin of animals and humans to form provitamin D3 which is converted by a thermic reaction to form cholecalciferol, and
- ergocalciferol (vitamin D2) – a rarer form of vitamin D, produced by ultraviolet irradiation of the plant steroid ergosterol. This form of vitamin D is used in some supplemental products.

Vitamin D3 and vitamin D2, made in the skin and/or ingested, are transported to the liver and metabolised to 25-hydroxyvitamin D (25OHD), the major circulating form (*Figure 1*). Further hydroxylation occurs in the kidneys to form the biologically active calcitriol, also known as 1,25 dihydroxyvitamin D (1,25(OH)2D), which is transported in the blood bound to albumin and vitamin D binding protein. Only a small fraction of the 1,25(OH)2D circulates in its 'free' form to bind to a specific vitamin D receptor (VDR). The binding of 1,25(OH)2D to its receptor regulates calcium and phosphate metabolism and induces a wide array of biological responses.

**Table 1. Australian high risk population groups with low serum 25OHD levels as reported in the literature**

| Highest risk groups | | Prevalence of deficiency* |
|---|---|---|
| Residential care elderly in high level care | | 55%[34] |
| Residential care elderly in high and low level care | | 68–86%[35] |
| Residential care elderly in low level care | | 22%[34] |
| Geriatric admissions to hospital | | 67%[36] |
| Patients with hip fracture | | 61%[40] |
| | | 63%**[6] |
| Dark skinned women (particularly veiled) | | >80%**[37,38] |
| Mothers of infants with rickets (particularly dark skinned and veiled) | | 80%[39] |
| **Lower risk groups** | | |
| Community dwelling elderly | | 17%[36] |
| Women in winter | 80+ years | 22%[2] |
| | 60–79 years | 12%[2] |
| | 20–59 years | 8%[2] |

* Deficiency defined as either <25 or 28 nmol/L
** Deficiency defined as <50 nmol/L

**Table 2. Causes of vitamin D deficiency**

- Reduced intake or synthesis of cholecalciferol
  - reduced sunlight exposure
  - reduced synthesis from a given UV exposure
  - low consumption of foods with high vitamin D content
- Disorders associated with abnormal gut function
  - small bowel disorders – coeliac disease (note: a common presentation for primary diagnosis of coeliac disease is the presence of osteoporosis with low vitamin D levels), sprue, inflammatory bowel disorders, infiltrative disorders such as lymphoma, granuloma, amyloid, small bowel resection
  - pancreatic insufficiency – chronic pancreatitis, cystic fibrosis
  - biliary obstruction – primary biliary cirrhosis, external biliary drainage
- Reduced synthesis or enhanced degradation of 25OHD
  - chronic hepatic disorders – hepatitis, cirrhosis

## Actions of calcitriol

- Increases absorption of calcium and phosphate from the small intestine. This is possibly the most important function of vitamin D compounds[3]
- Maintains calcium homeostasis in the extracellular fluid directly and in interaction with parathyroid hormone (PTH)
- Through its feedback mechanism, regulates its own renal production and degradation
- Facilitates skeletal mineralisation largely through enhanc-

ing acquisition of environmental mineral from the diet
- Stimulates bone resorption, particularly at high concentrations.

## Normal bone remodelling

In order to highlight the importance of vitamin D deficiency on bone turnover, it is important to understand the normal bone remodelling cycle. The remodelling process starts when osteoclasts resorb existing bone. Thereafter, a team of newly differentiated osteoblasts is recruited on the bone surface to synthesise unmineralised bone matrix called osteoid. In the presence of adequate 1,25(OH)2D and mineral, the osteoblasts mineralise the osteoid. The mineralisation process occurs in two phases – a rapid first phase taking a few days and a prolonged second phase lasting up to 6 months.

## Vitamin D deficiency and bone

Both plasma 25OHD and 1,25(OH)2D levels have been shown to decrease with age. This may occur as a result of age related factors such as reduced capacity to produce vitamin D, diminished sunlight exposure,[4] reduced intake and decline in renal function and secondary causes (*Table 2*). Vitamin D insufficiency where serum 25OHD concentrations are between 25–50 nmol/L may lead to a progressive increase in PTH secretion, high bone turnover, increased age related bone loss and an increased risk of developing osteoporosis. Low vitamin D status has been associated with reduced bone mineral density,[8] high bone turnover[5] and increased risk of hip fracture in the elderly.[6,7]

Osteomalacia, on the other hand, is the bone disease seen in true vitamin D deficiency. In more severe cases, with serum 25OHD levels usually less than 12.5 nmol/L, patients often present with bone and muscle pain, weakness and pseudofractures (ie. stress fracture where the fracture line does not breach the cortex completely). On bone histomorphometry, the classic findings of hyperosteoidosis, thickened unmineralised seams, increased osteoclast surfaces and tetracycline smearing are evident. Due to the chronicity of this disorder, progressive bone loss is often evident and osteoporosis coexists. *Table 3* classifies the stages of vitamin D deficiency.

## Vitamin D deficiency, muscle function and falls

Falls are a crucial part of the pathogenesis of osteoporotic fractures. Vitamin D deficiency has been demonstrated to be an independent predictor of falls in the elderly. The findings that 1,25(OH)2D may affect skeletal muscle function have gained much attention in recent years.

In osteomalacia, a metabolic myopathy has been

noted, consisting histologically of atrophied type 2 muscle fibres with fat infiltration, fibrosis and glycogen granules. Patients present typically with gait disturbances and difficulties arising from a chair. Abnormal motor performance, increased body sway and quadriceps weakness have been reported with serum 25OHD levels below 20–30 nmol/L in elderly men and veiled Arab women.[9,10] A randomised controlled trial demonstrated a 49% reduction in falls associated with vitamin D and calcium treatment.[11] Furthermore, polymorphisms in the VDR genotypes have been associated with reduced quadriceps muscle function.[12] These findings suggest a link between vitamin D deficiency, falls and bone fragility fractures.

## Other potential effects of low vitamin D status

The receptor for 1,25(OH)2D has been discovered in most nucleated cells of the body, which suggests that vitamin D could play an important role in a number of physiological processes. Vitamin D deficiency has been reported to result in impaired immune function, including reduced capacity to respond to agents that cause tuberculosis.[13] In addition 1,25(OH)2D has been shown to possess antiproliferative and prodifferentiating effects on several other types of cells present in the skin.[14]

## Sources of vitamin D

The main source of vitamin D for Australians is exposure to sunlight. In Australians there are seasonal variations in vitamin D status, such that serum 25OHD levels are somewhat lower at the end of winter compared to the end of summer. It has been estimated that exposure of the entire body surface to around 10–15 minutes noonday sun in summer (ie. around 1 minimal erythemal dose) is comparable to taking around 15 000 IU of vitamin D orally.[4] Less vitamin D would be synthesised at other times of the day, in winter, and in those with dark skin or the elderly.

Vitamin D3 is found in small quantities in few foods. Rich sources of vitamin D are fish, especially high fat fish such as salmon, herring and mackerel. Other sources are meat, eggs and fortified foods such as margarine. Margarine is the only food in Australia that is widely used that contains significant amounts of vitamin D through fortification. Adequate vitamin D is unlikely to be achieved through dietary means alone for most Australians with estimated daily vitamin D intake for adults between 80–120 IU. Extending fortification of the food supply would result in a modest increase in dietary vitamin D intake, taking average intakes to around 200 IU per day, and although
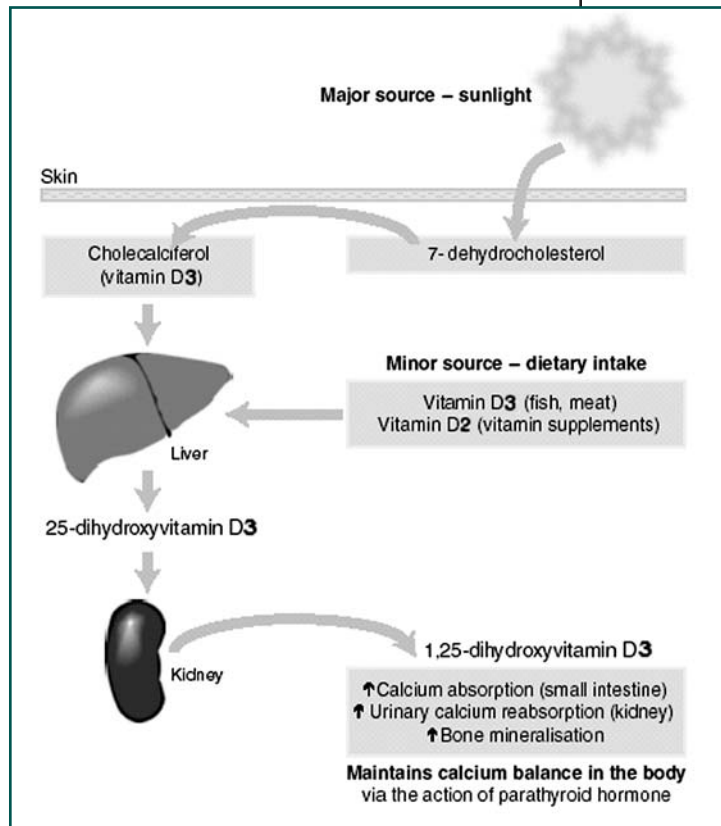


Figure 1. Absorption and metabolism of vitamin D15
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### Table 3. Stages of vitamin D deficiency

| Severity | Serum 25OHD (nmol/L) | Serum PTH (% increase) | Bone turnover |
|---|---|---|---|
| Mild (insufficiency) | 25–50 | 15 | High normal |
| Moderate | 12.5–25.0 | 15–30 | High |
| Severe | <12.5 | >30 | Osteomalacia |

this may assist in maintaining vitamin D status in low risk groups, it would not be sufficient to maintain adequate vitamin D status in high risk groups.[15] In Australia there is no current recommended dietary intake level set for vitamin D, but 200 IU has been proposed for the elderly not exposed to at least 2 hours of direct sunlight per week.[16] Recently the Food and Nutrition Board of the US Institute of Medicine proposed a daily reference intake of 200 IU (0–50 years), 400 IU (51–70 years), and 600 IU (71+ years).[17] This represents a tripling of the recommended intake for those over 70 years of age.

## Sunscreens

Broad spectrum sunscreens protect the skin against UV-A (320–400 nm) and UV-B (290–320 nm) radiation.

Radiation in the UV-B range is responsible for skin carcinogenesis as well as conversion of 7-dehydrocholesterol into provitamin D3.[4] The application of sun protection factor (SPF) 8 has been reported to reduce the amount of vitamin D synthesised from a given dose of UV by more than 95%.[18] For this reason, concerns were raised that the use of sunscreens for the prevention of skin cancer may cause vitamin D deficiency, especially among the elderly. Yet sunscreens are essential to prevent skin damage if periods of sun exposure long enough to produce erythema are planned. One Australian study showed that normal SPF usage over summer did not significantly affect 25OHD concentrations.[18] For incidental sun exposures of less than 10 minutes duration, it may be better to omit sunscreen. Short exposures are more efficient at producing vitamin D. Once previtamin D3 and vitamin D3 have been formed, continued exposure to sunlight results in their degradation to relatively inert over irradiation products.[4] It therefore appears prudent to recommend exposure of hands, face and arms to 5–15 minutes of sunlight 4–6 times a week, with the greatest frequency recommended for the elderly and dark skinned populations. Data collected in New Zealand indicate that exposure to sunshine for 30 minutes a day for 1 month during Spring produced a 30% increase in serum 25OHD levels in elderly men and women.[19]

## Vitamin D supplementation

Oral preparations such as ergocalciferol (D2) and cholecalciferol (D3) are available in Australia to treat vitamin D deficiency. Twelve calcium supplements contain vitamin D, all but one contain cholecalciferol, however, the levels are quite low ranging from 32–200 IU per tablet. Halibut liver oil tablets provide 4000 IU per capsule and although these are economical (~0.08 cents/capsule) they do contain vitamin A (4000 IU) which may not be beneficial for everyone. The only pure vitamin D preparation in Australia is Ostelin 1000, which contains ergocalciferol (D2) (1000 IU at ~24 cents/capsule). Vitamin D status is assessed by measurement of blood 25OHD concentrations, not 1,25(OH)2D. Treatment with ergocalciferol does not appear to increase plasma 25OHD levels (including both 25OHD2 and 25OHD3) as effectively as supplementation with cholecalciferol.[20] It is not clear, however, how much of the apparent increased effectiveness of D3 above D2 is due to the sensitivity of the assay. The assay is more sensitive to D3 than D2.[21]

## Treatment of vitamin D deficiency

Vitamin D is stored in fat and muscle and is slowly released, particularly during winter.[22] In vitamin D deficient patients, it is necessary to replete vitamin D stores. While the daily requirement for vitamin D is 400–600 IU per day, a much larger dose is used to treat vitamin D deficient patients. As vitamin D is fat soluble with a half-life >3 weeks, large doses are needed before changes in serum 25OHD are seen. High dosages of vitamin D (3000–5000 IU per day for 6–12 weeks [Ostelin]) may be used to replete body stores. Oral doses of 10 000 IU per day over 90 days have been shown to increase serum 25OHD levels to 86 nmol/L in postmenopausal women at latitude 34°S.[23] Higher doses of 50 000–500 000 IU orally or 600 000 IU intramuscularly can effectively treat vitamin D deficiency, however, they are not currently available in Australia and there is the possibility of inducing hypercalcaemia/hypercalciuria. Serum 25OHD levels should be checked at 3–4 monthly intervals to ensure adequacy of replacement. Calcitriol, the bioactive preparation of vitamin D, is not recommended for treating patients with simple vitamin D deficiency. This agent has a narrow therapeutic window and may result in significant hypercalcaemia. Moreover, changes in serum 25OHD levels are not a reflection of the calcitriol therapy.

## Toxicity

Vitamin D toxicity cannot be caused by prolonged exposure of the skin to UV light but may occur with excess oral intake through supplementation.[24] Vitamin D intoxication may cause anorexia, nausea, weakness, weight loss, polyuria, constipation, headache, depression, vague aches, stiffness, soft tissue calcification, nephrocalcinosis, hypertension, anaemia, hypercalcaemia, acidosis and irreversible renal and heart failure.[25] It should be noted that although vitamin D is toxic, relatively high doses are safe with no signs of toxicity at doses up to 4000 IU per day.[26] Although there is data indicating that high dose 300 000 IU injections given at intervals of at least a few months cause little evidence of toxicity,[27] such supraphysiological doses are yet to be established as safe. Cod liver oil, in addition to containing vitamin D, contains vitamin A which can also be toxic at high doses.

## Effectiveness of vitamin D supplementation

The greatest therapeutic effect of vitamin D supplementation is seen in high risk individuals who demonstrate decreases in bone densities, ranging from 0–4% in vitamin D insufficient patients to 10–40% in vitamin D deficient patients with osteomalacia.[28,29] The pivotal trial relating fracture reduction in a high risk group treated with vitamin D3 supplementation (800 IU of cholecalcif-

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**Table 4. Recommendations**

**High risk groups**
- elderly people, particularly in residential care
- people with skin cancers or skin related conditions where avoidance of sunlight is required
- dark skinned people, particularly pregnant or veiled women
- individuals presenting with osteoporosis or minimal trauma fractures
- patients with malabsorption

**Optimal blood test**
- 25OHD (Diasorin, radioimmunoassay kit)

**Good dietary sources of vitamin D**
- fatty fish, eg. mackerel and herring, margarine (fortified)

**Minimum sun exposure to prevent deficiency**
- exposure of hands, face and arms to 5–15 minutes of sunlight 4–6 times per week
- the elderly or those with dark skin require the greater frequency of exposure

**Amount of dietary vitamin D required to prevent deficiency**
- between 200 IU (<50 years) – 600 IU (>70 years) per day

**Amount of vitamin D supplementation required to reduce fracture risk in the elderly**
- between 800–1000 IU per day

**Amount of vitamin D required to treat deficiency**
- between 3000–5000 IU per day for at least 1 month

erol for 18 months) was published in 1992 and demonstrated a 41% reduction in hip fractures in elderly women in residential care establishments.[30] More recently, in a double blind randomised controlled trial, oral cholecalciferol 100 000 IU administered every 4 months for 5 years, reduced the risk of first hip, wrist, forearm or vertebral fracture in community dwelling persons by 33% compared with placebo.[31] Other studies in community dwelling individuals and lower vitamin D doses have not shown fracture reductions.[32]

It is unlikely that vitamin D supplementation has any role in vitamin D replete individuals, although the optimal level of serum 250HD is not precisely known (but may be above 60 nmol/L). Many studies have been undertaken to determine whether vitamin D therapies can prevent or treat postmenopausal osteoporosis. The varying increases in bone densities and reduction in fracture rates may be attributed to differences in pretreatment PTH and vitamin D levels. A comprehensive

meta-analysis suggests that vitamin D reduces the risk of vertebral fractures by 37% (RR: 0.63; 95% CI: 0.45–0.88, p<0.01) with no significant reduction in non-vertebral fractures (RR: 0.77; 95% CI: 0.57–1.04, p<0.09).[33] Most studies have utilised a combination of vitamin D and calcium supplementation therefore adequate dietary calcium may be required in conjunction with adequate vitamin D to reduce fracture rate.

## Conclusion

Treatment with a vitamin D supplement with or without screening should be considered for groups at greatest risk of vitamin D deficiency as listed in *Table 4*. The amount of recommended dietary vitamin D is the best estimate based on current available evidence, acknowledging that more data is required to make specific recommendations for different population groups. Accordingly the estimated amount of dietary or supplemental vitamin D required to: prevent deficiency 200–600 IU, reduce fracture risk in the elderly 800–1000 IU, and to treat deficiency 3000–5000 IU per day for at least 1 month.

### Acknowledgment

**SUMMARY OF IMPORTANT POINTS**

- People at high risk of vitamin D deficiency include the elderly (particularly in residential care), those with skin conditions where avoidance of sunlight is required, dark skinned people (particularly if veiled), and patients with malabsorption.
- Combined vitamin D and calcium supplementation has been shown to reduce falls and fractures in the elderly.
- Exposure of hands, face and arms to 5–15 minutes of sunlight 4–6 times a week is recommended, with the greatest frequency for those who are older and have dark skin. If this is not possible then a vitamin D supplement of at least 400 IU per day is recommended. Note: deliberate exposure to sunlight between 10 am and 3 pm in the summer months is not advised.
- In the case of established vitamin D deficiency cases, supplementation with between 3000-5000 IU per day for at least 1 month is required to replete body stores.

### References

1. Sanders KM, Seeman E, Ugoni AM, et al. Age and gender specific rate of fractures in Australia: a population based study. Osteoporos Int 1999;10:240–247.
2. Pasco JA, Henry MJ, Nicholson GC, et al. Vitamin D status of women in the Geelong Osteoporosis Study: association with diet and casual exposure to sunlight. Med J Aust 2001;175:401–405.
3. Malloy PJ, Pike JW, Feldman D. The vitamin D receptor and the syndrome of hereditary 1,25-dihydroxyvitamin D resistant rickets. Endocr Rev 1999;20:156–188.
4. Holick MF. McCollum Award Lecture, 1994. Vitamin D new horizons for the 21st century. Am J Clin Nutr 1994;60:619–630.
5. Kipen E, Helme RD, Wark JD, Flicker L. Bone density, vitamin D nutrition, and parathyroid hormone levels in women with dementia. J Am Geriatr Soc 1995;43:1088–1091.
6. Diamond T, Smerdely P, Kormas N, et al. Hip fracture in elderly men : the importance of subclinical vitamin D deficiency and hypogonadism. Med J Aust 1998;169:138–141.
7. Weatherall M. A meta-analysis of 25 hydroxyvitamin D in older people with fracture of the proximal femur. N Z Med J 2000;113:137–140.
8. Outila TA, Karkkainen MU, Lamberg-Allardt CJ. Vitamin D status affects serum parathyroid hormone concentrations during winter in female adolescents: associations with forearm bone mineral density. Am J Clin Nutr 2001;74:206–210.
9. Glerup H, Mikkelsen K, Poulsen L, et al. Commonly recommended daily intake of vitamin D is not sufficient if sunlight exposure is limited. J Intern Med 2000;247:260–268.
10. Dhesi JK, Bearne LM, Moniz C, et al. Neuromuscular and psychomotor function in elderly subjects who fall and the relationship with vitamin D status. J Bone Miner Res 2002;17:891–897.
11. Bischoff HA, Stahelin HB, Dick W, et al. Effects of vitamin D and calcium supplementation on falls: a randomised controlled trial. J Bone Miner Res 2003;18:343–351.
12. Geusens P, Vandevyver C, Vanhoof J, et al. Quadriceps and grip strength are related to vitamin D receptor genotype in elderly nonobese women. J Bone Miner Res 1997;12:2082–2088.
13. Bouillon R, Okamura WH, Norman AW. Structure function relationships in the vitamin D endocrine system. Endocr Rev 1995;16:200–257.
14. DeLuca HF, Zierold C. Mechanisms and functions of vitamin D. Nutr Rev 1998;56:S4–S10.
15. Nowson CA, Margerison C. Vitamin D intake and vitamin D status of Australians. Med J Aust 2002;177:149–152.
16. National Health and Medical Research Council. Recommended dietary intakes for use in Australia. Canberra: AGPS, 1991.
17. Food and Nutrition Board. Dietary reference intakes for Ca, P, Mg, vitamin D and F. Washington DC: National Academy Press, 1997.
18. Matsuoka LY, Ide L, Wortsman J, et al. Sunscreens suppress cutaneous vitamin $D_3$ synthesis. J Clin Endocrinol Metab 1987;64:1165–1168.
19. Reid IR, Gallagher DJ, Bosworth J. Prophylaxis against vitamin D deficiency in the elderly by regular sunlight exposure. Age Ageing 1986;15:35–40.
20. Tjellesen L, Hummer L, Christiansen C, Rodbro P. Serum concentration of vitamin D metabolites during treatment with vitamin $D_2$ and $D_3$ in normal premenopausal women. Bone Miner 1986;1:407–413.
21. Hollis BW. Comparison of commercially available (125)I-based RIA methods for the determination of circulating 25-hydroxyvitamin D. Clin Chem 2000;46:1657–1661.
22. Mawer EB, Backhouse J, Holman CA, et al. The distribution and storage of vitamin D and its metabolites in human tissues. Clin Sci 1972;43:413–431.
23. Mastaglia S, Oliveri B, Parisi MS, Cristofari A, Mautalen C. 10 000 IU of oral vitamin D per day are required to rapidly (3 months) reach adequate 25OHD in osteoporotic women. J Bone Miner Res 2003;18(Suppl 2):S55.
24. Koutkia P, Chen TC, Holick MF. Vitamin D intoxication associated with an over-the-counter supplement. N Engl J Med 2001;345:66–67.
25. Swain R, Kaplan B. Vitamins as therapy in the 1990s. J Am Board Fam Pract 1995;8:206–216.
26. Vieth R, Chan PC, MacFarlane GD. Efficacy and safety of vitamin $D_3$ intake exceeding the lowest observed adverse level. Am J Clin Nutr 2001;73:288–294.
27. Lips P. Vitamin D deficiency and secondary hyperparathyroidism in the elderly: consequences for bone loss and fractures and therapeutic implications. Endocr Rev 2001;22:477–501.
28. Parfitt A. Osteomalacia and Related Disorders. In: Avioli L, Krane S, eds. Metabolic bone diseases and clinically related disorders. 2nd edn. Philadelphia: WB Saunders Company, 1990.
29. Mithal A, Sen I, Naik V, Ravishankar U. Spectacular recovery of bone mineral density following treatment of osteomalacia. J Bone Miner Res 2000;15(Suppl 1):S577.
30. Chapuy MC, Arlot ME, Duboeuf F, et al. Vitamin $D_3$ and calcium to prevent hip fractures in the elderly women. N Engl J Med 1992;327:1637–1642.
31. Trivedi DP, Doll R, Khaw KT. Effect of four monthly oral vitamin $D_3$ (cholecalciferol) supplementation on fractures and mortality in men and women living in the community: randomised double blind controlled trial. BMJ 2003;326:469–475.
32. Lips P, Graafmans WC, Ooms ME, et al. Vitamin D supplementation and fracture incidence in elderly persons: a randomised placebo controlled trial. Ann Intern Med 1996;124:400-406.
33. Papadimitropoulos E, Wells G, Shea B, et al. Meta-analyses of therapies for postmenopausal osteoporosis. VIII Meta-analysis of the efficacy of vitamin D treatment in preventing osteoporosis in postmenopausal women. Endocr Rev 2002;23:560-569.
34. Flicker L, Mead K, MacInnis RJ, et al. Serum vitamin D and falls in older women in residential care in Australia. J Am Geriatr Soc 2003;51:1533-1538.
35. Sambrook PN, Cameron ID, Cumming RG, et al. Vitamin D deficiency is common in frail institutionalised older people in northern Sydney. Med J Aust 2002;176:560.
36. Inderjeeth CA, Nicklason F, Al-Lahham Y, et al. R. Vitamin D deficiency and secondary hyperparathyriodism: clinical and biochemical associations in older noninstitutionalised Southern Tasmanians. Aust N Z J Med 2000;30:209-214.
37. Grover SR, Morley R. Vitamin D deficiency in veiled or dark skinned pregnant women. Med J Aust 2001;175:251-252.
38. Diamond TH, Levy S, Smith A, Day P. High bone turnover in Muslim women with vitamin D deficiency. Med J Aust 2002;177:139-141.
39. Nozza JM, Rodda CP. Vitamin D deficiency in mothers of infants with rickets. Med J Aust 2001;175:253-255.
40. Crone RP, Pasco JA, Henry MJ, Sanders KM, Nicholson GC, Kotowicz MA. Vitamin D deficiency among hip fracture patients admitted for rehabilitation. Proceedings of the ANZBMS 2002;67:96.

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