

**Simon Vanlint**

MBBS, FRACGP, Lecturer and Assistant Dean (Students), Discipline of General Practice, University of Adelaide, South Australia. simon.vanlint@adelaide.edu.au

Michael Nugent

MBBS, BMedSci(Hons), is Senior Medical Officer, Centre for Intellectual Disability Health, Adelaide, South Australia.

Seeta Durvasula

MBBS, MPH, MPaed, DCH, is Lecturer, Centre for Developmental Disability Studies, University of Sydney, New South Wales.

Vitamin D and people with intellectual disability

Background

Vitamin D is essential for the normal functioning of a diverse range of metabolic processes, especially bone health. It is widely appreciated that the elderly are at increased risk of vitamin D insufficiency, but it is less well known that people with intellectual disability are also at increased risk.

Objective

This article summarises the issues regarding vitamin D in people with intellectual disability, making recommendations about screening, management and follow up.

Discussion

The prevalence of intellectual disability in the Australian population has been estimated at just over 1%, implying that most general practitioners will care for several people with intellectual disability. Relatively simple steps are likely to have a significant impact on the health of this vulnerable group of people.

■ **The effects of vitamin D deficiency have been recognised for centuries. However, it is a historical accident that vitamin D was classified as a vitamin, as vitamin D3 (cholecalciferol) is a fat soluble steroid hormone. Dietary vitamin D is absorbed in the small intestine and can be obtained from fish oils, liver, eggs, margarine and some dairy products. However, for most people at most times, synthesis of vitamin D in the skin is the most important source. It has been suggested that sunscreen use may interfere with vitamin D synthesis, but local data suggest that during an Australian summer sufficient sunlight is received by regular sunscreen users to produce vitamin D.¹**

Vitamin D promotes calcium and phosphate absorption by the small intestine, mineralisation of the skeleton and maintenance of extracellular calcium levels. Severe vitamin D deficiency may result in rickets or osteomalacia, rarely seen in modern Australia. However, osteoporosis and the consequent increased fracture risk can also result from vitamin D deficiency.

There are several new areas of interest in vitamin D. Vitamin D receptors are virtually ubiquitous in human tissue, and so perhaps not surprisingly, vitamin D deficiency has been linked with cancer mortality (colon, prostate, breast, ovary, pancreas), increased risk of autoimmune disease, abnormal lung function (including chronic obstructive pulmonary disease), tuberculosis, disorders of glucose metabolism, and psychiatric illness.² More tenuous associations have been proposed with detrusor instability, swallowing disorders and age related macular degeneration. Epidemiologic observational studies suggest associations, but do not prove causality, and further study is ongoing.³⁻⁵

Who is at risk?

Certain individuals are at increased risk of vitamin D deficiency, the most widely recognised being the elderly, especially those in residential care institutions.⁶ This is largely due to immobility resulting in limited sunlight exposure, and is exacerbated by the reduced ability of elderly skin to synthesise vitamin D even when exposed to sunlight.

Jenny Downs

PhD, is Senior Research Officer, Centre for Child Health Research, University of Western Australia.

Helen Leonard

MBChB, is Clinical Associate Professor, Centre for Child Health Research, University of Western Australia.

People with intellectual disability

It is less well known that people with intellectual disability (ID) are also at high risk of vitamin D deficiency, with local studies suggesting a prevalence of 50–60% in this group.⁷ It is likely that the most important factor is reduced mobility. Time constraints of carers may limit the opportunities for people with ID to be exposed to sunlight, as may concerns over the hazards of excessive exposure.

Reduced mobility is compounded by the association between anticonvulsant use and reduced vitamin D levels. Epilepsy prevalence may be as high as 26% in people with ID, and difficulties with seizure control make polypharmacy more likely.⁸ There is some evidence that the older anticonvulsants (eg. phenytoin, phenobarbitone) are more likely to be implicated, and that use of multiple anticonvulsant medications is more problematic than monotherapy.⁹

People with ID have an increased risk of falls for many reasons, eg. reduced balance and coordination associated with the underlying cause of ID such as the spasticity associated with cerebral palsy,¹⁰ and visual impairment, which affects up to 20% of people with ID.¹¹ Vitamin D deficiency has been shown to be associated with an increased risk of falls due to effects on muscle, and if prolonged and severe, can cause a true myopathy.¹²

Other conditions with a negative effect on bone health also occur in people with ID. The prevalence of coeliac disease, which causes calcium malabsorption, is increased in people with Down syndrome.¹³ Hypogonadism, which increases the risk of osteoporosis, is also more common in Down syndrome and ubiquitous in Turner syndrome.^{14,15} Women with ID may be long term users of depot progesterone only contraceptives, both for menstrual control and contraception.¹⁶ Such preparations have been associated with reduced bone density, and although it appears that this is largely reversible after cessation in short to medium term users, the effects in long term users are not clearly defined.¹⁷ It is important in all such individuals to be aware of these possible effects on bone health.

The fracture rate in people with ID appears to be higher than that of the general population. Australian studies involving both community dwelling and institutionalised participants have found it to be 2–4 times higher,^{7,15,18} consistent with the international literature.⁷

Skin synthesis of vitamin D

Where possible, it is probably best to promote adequate skin synthesis of vitamin D via sunlight exposure. *Table 1* details suggested sunlight exposure times for different geographic locations. These recommendations¹⁹ attempt to balance the need for vitamin D

Table 1. Recommended sun exposure by location and season¹⁶

	December – January	July – August
Cairns (QLD)	6–7 mins	9–12 mins
Brisbane (QLD)	6–7 mins	15–19 mins
Perth (WA)	5–6 mins	20–28 mins
Sydney (NSW)	6–8 mins	26–28 mins
Adelaide (SA)	5–7 mins	25–38 mins
Melbourne (VIC)	6–8 mins	32–52 mins
Hobart (TAS)	7–9 mins	40–47 mins
Auckland (NZ)	6–8 mins	30–47 mins
Christchurch (NZ)	6–9 mins	49–97 mins

Note: This table assumes the following: moderately fair complexion, exposure of the face and either the arms or legs, exposure before 10 am or after 2 pm (during daylight saving: before 11 am or after 3 pm)

synthesis with the known harmful effects of ultraviolet light, but as yet have not been substantiated by formal trials. It is likely that time spent outdoors, especially if combined with mobility and exercise, will have the beneficial effects of an enriched lifestyle above and beyond the simple production of vitamin D.

Testing of vitamin D levels

Given the high prevalence of vitamin D deficiency, routine testing of 25-hydroxyvitamin D (25D) levels is appropriate in most, if not all, people with ID. There is broad agreement that 25D levels should exceed 50 nmol/L,²⁰ with some suggesting that a threshold level of 60–70 nmol/L may be more appropriate.² Seasonality is important, especially in the more mobile, with levels tending to be at their lowest in late winter/early spring, and peaking in late summer/early autumn.⁶

If vitamin D deficiency is found and treated, or the dose of an established treatment is altered, 25D levels should be checked 3–4 months later. A further check 12 months later should be sufficient to guarantee adequate levels in the future, unless there is a change in health or lifestyle status. There is no published evidence base for these recommendations; they were derived by expert consensus with an understanding of the underlying physiology.

Supplementation

Where adequate sunlight exposure is not possible or has not resulted in adequate 25D levels, supplementation should be used. Vitamin D3 is preferable to vitamin D2, as it appears to be more

effective.²¹ Fracture prevention studies in the elderly suggest a dose of at least 800 IU/day in combination with adequate calcium (at least 1000 mg/day) is likely to be effective.^{22,23} For example, two tablets daily of Ostelin Vitamin D + Calcium would achieve this aim, at a cost of about \$0.30 per day. Where moderate to severe deficiency is found, higher doses may be necessary for a period of time to normalise levels rapidly. While objective evidence is limited, a regimen of 4000 IU/day for 3–4 months may be required to achieve this. Reports of vitamin D toxicity are scarce, and have not been reported with doses of 4000 IU/day.² Even 600 000 IU as an annual intramuscular 'megadose' is well tolerated.²⁴ Toxicity cannot be caused by excessive sun exposure. Hypercalcaemia is the only contraindication to vitamin D supplementation, and calcium levels should be checked before supplementation. Although the literature is not entirely consistent, it is generally held that vitamin D should be given in combination with calcium, either through a suitable diet, or as a supplement. Regrettably, vitamin D supplementation is not funded by the Pharmaceutical Benefits Scheme.

Conclusion

From being thought of as an endocrine hormone principally affecting bone, calcium and phosphate, vitamin D has become the subject of speculation and investigation in relation to a range of physiological processes and disease states. It is likely that a significant number of Australians with ID are vitamin D deficient. Greater awareness and more assertive management will have significant health and economic benefits to this group of people.

Conflict of interest: none declared.

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