



Vitamin D—how do we define deficiency and what can we do about it in New Zealand?

Robert Scragg, Jim Bartley

Although we live in a sunny climate, and the sun is the main source of our vitamin D, for some reason New Zealanders have lower vitamin D levels in their bodies than people in other comparable countries at similar latitudes.

Vitamin D status is determined by blood levels of 25-hydroxyvitamin D (25OHD). Recent findings from the 1997 adult nutrition survey show a mean 25OHD level of 50 nmol/L in New Zealanders aged ≥ 15 years, considerably lower than mean values above 70 nmol/L seen in the adult US population which lives at similar latitudes as the New Zealand population, and similar to the mean level of 50 nmol/L in the UK adult population which lives at higher latitudes (i.e. further from the equator than New Zealand).¹

Mean vitamin D levels are lower still among Māori (42 nmol/L) and Pacific people (37 nmol/L).¹ Furthermore, South Asian people are likely to have low vitamin D levels because of their darker skins, and veiling with traditional dress by many Muslim immigrants also places them at increased risk of developing vitamin D deficiency.³ Moreover, osteomalacia and rickets are conditions that have re-emerged in clinical practice.^{2,3}

Given our lower than expected vitamin D status, the two articles on vitamin D in this issue of the *Journal*, one from Christchurch which confirms the low vitamin D levels described above⁴ and one from Perth (Western Australia) which reports a significant positive association between serum 25OHD levels and leg muscle strength,³ are timely since they contain conclusions that are relevant to both clinicians and policymakers.

Although the article by Inderjeeth and colleagues reports results from a sample of women aged >60 years living in Perth,⁵ their findings most likely apply to older New Zealand women since the mean 25OHD of 52 nmol/L in the Perth sample was higher than the value of 43 nmol/L in New Zealand women aged ≥ 65 years in the 1997 nutrition survey;¹ and probably also applicable to New Zealand men aged ≥ 65 years who had a mean 25OHD level of 52 nmol/L.¹

The Perth study is consistent with a recent meta-analysis which found that vitamin D supplementation reduced the relative risk of falling by 22%, with an absolute risk reduction of 7%, so that the number needed to treat (NNT) to prevent one fall was 15, although the time period required for treatment is unclear as this was not provided by the authors of the meta-analysis.⁶

There is a well-described mechanism for this effect since vitamin D receptors have been identified in skeletal muscle, and vitamin D supplementation increases muscle strength by increasing the size and number of type II muscle fibres, so that gait and balance are improved. Thus, vitamin D, in combination with calcium, protects against hip fracture by increasing bone density and muscle strength.⁷

There is a further finding from the Perth study and this directly addresses the question of how we define vitamin D deficiency. Previous research has determined that vitamin D deficiency is defined by a serum 25OHD below 50 nmol/L.⁸ Yet, this conclusion is challenged by the observation in the Perth study showing that the association between serum 25OHD and muscle strength was strongest in women with 25OHD levels above 50 nmol/L.

This result is consistent with emerging research, from both physiology and epidemiology, that optimum vitamin D status occurs at serum 25OHD levels above 80 nmol/L. Indeed, metabolic studies have shown that the proportion of dietary calcium absorbed from the gut maximises (at just over 30%) when serum 25OHD levels are above 80 nmol/L.⁹

However, the strongest evidence comes from epidemiological studies which have shown that the risk of a range of medical conditions—including bone density, periodontal disease, colon cancer, hypertension, and lung function—is lowest in people with serum 25OHD levels above 80 nmol/L.^{10–12}

Some of this evidence comes from New Zealand studies. A large workforce diabetes survey carried out in Auckland and Tokoroa found that the risk of undiagnosed diabetes and impaired glucose tolerance was lowest at a serum 25OHD level above 83 nmol/L.¹³ An Auckland-based case control study of myocardial infarction (which reported 25OHD levels as nmol/L instead of the correct units of ng/mL) found that the lowest risk of myocardial infarction occurred at a 25OHD level above 43 ng/mL (or 107 nmol/L).¹⁴

It is still unclear whether there is a threshold at a 25OHD level of about 80 nmol/L for maximal health gains associated with increased vitamin D, or whether the relationship between 25OHD and health status continues to improve above this value; further research is being carried out to clarify this.

But either way, defining vitamin D deficiency as a 25OHD level below 50 nmol/L is clearly not supported by the current evidence; optimum health occurs at much higher levels than this. Most hospital laboratories currently define vitamin D deficiency as a serum 25OHD <50 nmol/L, and clinicians need to reinterpret this value for their patients in light of the above evidence.

Clinicians, and policymakers, will also find relevant information in the Christchurch study by Livesey and colleagues.⁴ Besides confirming the results of previous research showing low vitamin D levels in New Zealanders, this study has estimated both the amount of vitamin D synthesised from sun exposure in both summer and winter, and also the amount of oral vitamin D required to increase winter serum 25OHD levels up to optimal levels.

Their estimate—1450 IU or 2600 IU of vitamin D₃ each day is required to increase serum 25OHD to 75 nmol/L or 100 nmol/L, respectively—is way above the current recommended level of 400 IU per day for people aged 51–70 years and 600 IU for people aged >70 years, but in line with current international opinion (see reference 1 of their paper).

Given that mean serum 25OHD levels of New Zealanders average 50 nmol/L for both children and adults, the evidence that health status improves at 25OHD levels above 50 nmol/L indicates that vitamin D needs to be promoted higher up the public health agenda, since 84% of the adult population have 25OHD levels below 80 nmol/L.¹

At the moment, non-government organisations, such as the Cancer Society of New Zealand and the Health Sponsorship Council, are driving the development of policy on sun exposure, vitamin D, and health, but with the focus firmly on (avoiding) sun exposure.

As Livesey and colleagues discuss, a second strategy—increasing vitamin D supplementation—needs to be considered in New Zealand since vitamin D synthesis from the sun during winter for people in Christchurch is estimated to be only 60 IU per day.⁴

However, a third strategy also needs to be thrown into the mix—mandatory vitamin D fortification (currently it is optional) of certain foods such as margarine and milk products, which already happens in several countries, including the US, UK, and Australia.

As research continues to emerge, the Ministry of Health soon will need to engage on the broad issue of vitamin D and health, and take ownership of it.

Competing interests: None.

Author information: Robert Scragg, Associate Professor of Epidemiology, Epidemiology & Biostatistics, School of Population Health, University of Auckland, Auckland; Jim Bartley, Otolaryngologist/Pain Consultant; The Auckland Regional Pain Service, Auckland City Hospital, Auckland

Correspondence: Associate Professor Robert Scragg, Epidemiology & Biostatistics, School of Population Health, University of Auckland, Auckland. Fax: (09) 373 7624; email: r.scragg@auckland.ac.nz

References:

1. Rockell JE, Skeaff CM, Williams SM, Green TJ. Serum 25-hydroxyvitamin D concentrations of New Zealanders aged 15 years and older. *Osteoporos Int.* 2006;17(9):1382–9.
2. Chiu G. Vitamin D deficiency among patients attending a central New Zealand rheumatology outpatient clinic. *N Z Med J.* 2005;118(1225). <http://www.nzma.org.nz/journal/118-1225/1727/>
3. Judkins A, Eagleton C. Vitamin D deficiency in pregnant New Zealand women. *N Z Med J.* 2005;118(1225). <http://www.nzma.org.nz/journal/119-1241/2144/>
4. Livesey J, Elder P, Ellis MJ, McKenzie R, Liley B, Florkowski C. Seasonal variation in vitamin D levels in the Canterbury, New Zealand population in relation to available UV radiation. *N Z Med J.* 2007;120(1262). <http://www.nzma.org.nz/journal/120-1262/2733>
5. Inderjeeth CA, Glennon D, Petta A, Soderstrom J, Boyatzis I, Tapper J. Vitamin D and muscle strength in patients with previous fractures. *N Z Med J.* 2007;120(1262). <http://www.nzma.org.nz/journal/120-1262/2730> <http://www.nzma.org.nz/journal/120-1262/2730>
6. Bischoff-Ferrari HA, Dawson-Hughes B, Willett WC, et al. Effect of Vitamin D on falls: a meta-analysis. *JAMA.* 2004;291(16):1999–2006.
7. Boonen S, Bischoff-Ferrari HA, Cooper C, et al. Addressing the musculoskeletal components of fracture risk with calcium and vitamin D: a review of the evidence. *Calcif Tissue Int.* 2006;78(5):257–70.
8. Malabanan A, Veronikis IE, Holick MF. Redefining vitamin D insufficiency. *Lancet.* 1998;351(9105):805–6.
9. Heaney RP. Serum 25-hydroxyvitamin D and parathyroid hormone exhibit threshold behavior. *J Endocrinol Invest.* 2005;28(2):180–2.
10. Bischoff-Ferrari HA, Giovannucci E, Willett WC, et al. Estimation of optimal serum concentrations of 25-hydroxyvitamin D for multiple health outcomes. *Am J Clin Nutr.* 2006;84(1):18–28.

11. Scragg R, Sowers M, Bell C. Serum 25-hydroxyvitamin D, ethnicity, and blood pressure in the Third National Health and Nutrition Examination Survey. *Am J Hypertens*. 2007;20(7):713–9.
12. Black PN, Scragg R. Relationship between serum 25-hydroxyvitamin D and pulmonary function in the third national health and nutrition examination survey. *Chest*. 2005;128(6):3792–8.
<http://www.chestjournal.org/cgi/content/full/128/6/3792?maxtoshow=&HITS=10&hits=10&RESULTFORMAT=&author1=Black&searchid=1&FIRSTINDEX=0&sortspec=relevance&resulttype=HWCIT>
13. Scragg R, Holdaway I, Singh V, et al. Serum 25-hydroxyvitamin D3 levels decreased in impaired glucose tolerance and diabetes mellitus. *Diabetes Res Clin Pract*. 1995;27(3):181–8.
14. Scragg R, Jackson R, Holdaway IM, et al. Myocardial infarction is inversely associated with plasma 25-hydroxyvitamin D3 levels: a community-based study. *Int J Epidemiol*. 1990;19(3):559–63.