Vitamin D insufficiency in Aboriginal Australians

Simon J Vanlint, Howard A Morris, Jonathan W Newbury and Alan J Crockett

ABSTRACT

Objective: To investigate the adequacy of vitamin D status in a South Australian Aboriginal population, and to examine the relationship between serum 25-hydroxyvitamin D (25-OHD) levels and biochemical variables of calcium and bone mineral homeostasis, as well as other factors which may influence vitamin D synthesis, storage and metabolism.

Design, setting and participants: A single-visit, observational study of 58 adults from two Aboriginal community-controlled health services in Adelaide and Yalata, South Australia. Participants were recruited between May 2008 and December 2009.

Main outcome measures: Serum levels of 25-OHD, parathyroid hormone (PTH), fasting glucose and fasting C-terminal telopeptides of type I collagen (β-CTx).

Results: Serum 25-OHD levels showed clear seasonal variation, being higher in summer ($P < 0.001$). The overall mean level was 56.8 nmol/L (SD, 22.1), which is below the recommended target level of 60 nmol/L. Serum 25-OHD levels correlated significantly with β-CTx ($P = 0.03$), but not with age, body mass index (BMI), PTH levels or levels of fasting glucose. A significant association was found between BMI and PTH levels ($P = 0.001$). A significant inverse association between serum 25-OHD levels and BMI, observed in other studies, was not found in our study.

Conclusions: Vitamin D insufficiency is highly prevalent in this population of adult Aboriginal Australians, with low mean values found in all seasons other than summer.

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Vitamin D is a steroid prohormone formed in the skin following ultraviolet B radiation. Vitamin D is hydroxylated to form 25-hydroxyvitamin D (25-OHD), the biomarker of vitamin D status. Little vitamin D is obtained through diet. Melanin filters incident ultraviolet B light, such that darker-skinned individuals synthesise less vitamin D than paler-skinned individuals,[1] and have lower 25-OHD levels.[2,3] Vitamin D is crucial in calcium homeostasis, with direct effects on intestine, bone, muscle[4] and numerous other tissues. The vitamin D receptor is virtually ubiquitous in the body, and insufficient vitamin D levels and vitamin D receptor genotype have been implicated in a wide range of health problems.[5-9]

Little is known about the vitamin D status of Aboriginal Australians. One study found that muscle pain, a potential consequence of vitamin D deficiency, is highly prevalent in Aboriginal people, and that it is often associated with low 25-OHD levels.[10] The literature regarding the vitamin D status of other dark-skinned groups, including African Americans,[2,11] Pacific Islanders and Maori,[3,12] and indigenous Canadians,[13] consistently shows that these groups have lower 25-OHD levels than comparable non-indigenous populations.

We investigated the range of 25-OHD levels in a South Australian Aboriginal population and examined the relationship between 25-OHD levels and biochemical variables of calcium and bone mineral homeostasis, as well as factors that may influence vitamin D synthesis, storage and metabolism.

METHODS

Participants were recruited between May 2008 and December 2009. Staff were asked via email if they wished to participate, and patients attending routine appointments were asked by staff if they wished to participate. Inclusion criteria were of Aboriginal or Torres Strait Islander origin and aged 18 years or over. Those who were being specifically investigated for possible vitamin D deficiency, were known to have diabetes mellitus, or were using a supplement containing vitamin D were excluded.

Fasting blood samples were collected and analysed at the Institute of Medical and Veterinary Science in Adelaide for levels of serum 25-OHD, parathyroid hormone (PTH), and fasting C-terminal telopeptides of type I collagen (β-CTx), a marker of bone resorption. Alcohol consumption and average time spent outdoors were based on participant recall of the month preceding the study visit. Skin colour was classified as pale, intermediate or dark, using the inner aspect of the upper forearm as the region of choice.

Ethics approval and consent

Ethics approval was obtained from the Aboriginal Health Council of South Australia's Health Research and Ethics Committee, and from the University of Adelaide's Human Research Ethics Committee, following discussions with community leaders at Nunkuwarrin Yunti. Participants gave written, informed consent.

Statistical analysis

Statistical analysis was performed using Stata version 11 (StataCorp, College Station, Tex, USA). Non-parametric tests were used where appropriate. The precise statistical tests used are provided in the body of the text in each instance.

RESULTS

Of 58 participants (40 men, 18 women; mean age, 38.9 years) included in the study, 51 were staff or patients at Nunkuwarrin

1 Age, BMI, and levels of 25-OHD, fasting glucose, PTH and fasting β-CTx in 58 Aboriginal adults

Variable	Minimum	Maximum	Mean	SD	Reference range
Age (years)	18	67	38.9	10.3	na
BMI (kg/m^2)	17.1	55.3	29.8	7.9	19–25 (desirable)
25-OHD (nmol/L)	20	111	56.8	22.1	> 60 (desirable)
Fasting glucose (mmol/L)	3.7	11.5	5.0	1.2	3.8–5.5
PTH (pmol/L)	0.3	13	4.2	2.3	1.1–5.5
Fasting β-CTx (ng/L)	118	668	337	147.7	< 400 (desirable)

BMI = body mass index. β-CTx = C-terminal telopeptides of type 1 collagen. na = not applicable. 25-OHD = 25-hydroxyvitamin D. PTH = parathyroid hormone. ◆

2 Age, BMI, and levels of fasting glucose, PTH and fasting β-CTx in 58 Aboriginal adults, by serum 25-OHD level

25-OHD level (nmol/L)	No.	Variable	Minimum	Maximum	Mean	Median*	SD	IQR*
⩽60	36	Age (years)	18	57	36.9	—	9.1	—
		BMI (kg/m^2)	19.9	55.3	31.2	29.8	8.7	11.0
		Fasting glucose (mmol/L)	3.9	11.5	5.1	4.7	1.5	0.8
		PTH (pmol/L)	1.7	13	4.6	4.3	2.4	3.1
		Fasting β-CTx (ng/L)	144	662	363.8	362	133.7	172
61–80	16	Age (years)	24	67	41.4	—	12.2	—
		BMI (kg/m^2)	17.1	40.7	27.7	26.9	6.2	7.7
		Fasting glucose (mmol/L)	3.7	5.6	4.8	5.0	0.6	0.9
		PTH (pmol/L)	1.1	8.0	3.9	3.7	2.3	2.9
		Fasting β-CTx (ng/L)	118	668	278.5	238	152.5	195
>80	6	Age (years)	20	46	35.5	—	12.4	—
		BMI (kg/m^2)	21.6	37.3	28.1	28.0	5.6	6.6
		Fasting glucose (mmol/L)	4.1	6.2	5.0	5.0	0.7	0.4
		PTH (pmol/L)	0.3	4.2	3.0	3.6	1.5	1.7
		Fasting β-CTx (ng/L)	135	650	336.3	292.5	191.4	280

BMI = body mass index. β-CTx = C-terminal telopeptides of type 1 collagen. IQR = interquartile range. 25-OHD = 25-hydroxyvitamin D. PTH = parathyroid hormone.
* Median and IQR only given for variables that were not normally distributed. ◆

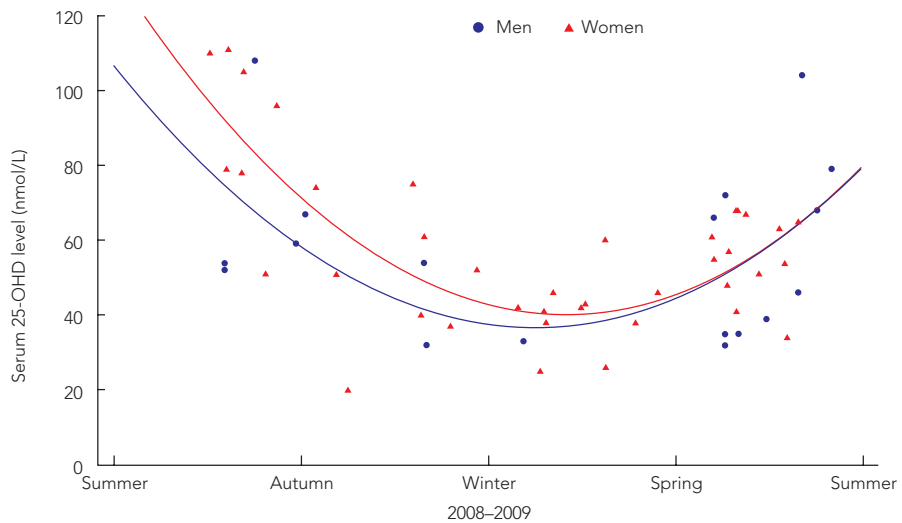
Yunti, and seven were patients from the Tullawon Health Service.

Demographics and overall mean biochemical variables for our study population are shown in Box 1. Mean levels for biochemical variables, grouped according to serum 25-OHD levels, are set out in Box 2. Serum 25-OHD levels were normally distributed for the bulk of participants, with a maximum level of 111 nmol/L.

Serum 25-OHD levels showed a definite seasonal variation: highest in late summer–autumn, lowest in late winter–spring (Box 3), and significantly higher in summer than in all other months ($P < 0.001$, analysis of variance [ANOVA]) (Box 4 and Box 5).

Spearman rank correlation coefficients between the measured variables are shown in Box 6. β-CTx levels decreased with age and were inversely related to 25-OHD levels ($P = 0.03$). Body mass index (BMI) was positively related to fasting glucose ($P = 0.002$) and PTH levels ($P = 0.001$), but not related to 25-OHD levels ($P = 0.87$). Age was also positively related to fasting glucose levels ($P = 0.025$). There was a non-significant trend towards an association between 25-OHD levels and time spent outdoors ($P = 0.058$, ANOVA) (data not shown). No significant association was found between 25-OHD level and smoking status ($P = 0.91$), alcohol consumption level ($P = 0.47$) or skin colour ($P = 0.20$) (data not shown).

3 Seasonal variation of 25-dehydroxyvitamin D (25-OHD) levels in 58 Aboriginal Australian adults, by sex*



* Data collected from May 2008 to December 2009 and aggregated by season. ◆

DISCUSSION

Our findings suggest that vitamin D insufficiency (serum 25-OHD ⩽ 60 nmol/L) is highly prevalent in this population of adult Aboriginal Australians. Mean 25-OHD levels peaked in summer (84.2 nmol/L), but were below 60 nmol/L in other seasons, indicating prolonged periods of vitamin D insufficiency. The overall mean serum 25-OHD level (56.8 nmol/L) was significantly lower than the mean of 76.9 nmol/L reported for a control group of 36 Adelaide residents (mean age, 32 years).[14] Others have reported data from non-Indigenous Australians indicating that in winter and spring the proportion of the population with 25-OHD levels less than 50 nmol/L varied between 37.4% (Geelong, latitude 37 °S) and 67.3% (Hobart, 43 °S).[15] In our Aboriginal Australian population, 20 of 37 participants (54%) assessed during winter and spring in Adelaide (35 °S) had a level below 50 nmol/L. These data suggest that Aboriginal Australians are likely to have a poorer vitamin D status than non-Indigenous Australians.

The seasonal variation in 25-OHD levels and the trend towards an association between serum 25-OHD levels and time spent out-

4 Seasonal variation of serum 25-hydroxyvitamin D (25-OHD) levels in 58 Aboriginal adults

Season	*n*	Mean 25-OHD level (nmol/L)	Number			
			⩽ 25 nmol/L	26–60 nmol/L	61–80 nmol/L	> 80 nmol/L
Autumn	10	51.1	1	5	4	0
Winter	12	40.8	1	11	0	0
Spring	23	52.1	0	15	8	0
Summer	13	84.2	0	3	4	6

5 Significance of seasonal differences in 25-hydroxyvitamin D status in 58 Aboriginal adults

Index season	Comparator season	Differences of least squares means				
		Estimate	SEM	df	*t*	*P*
Autumn	Spring	–0.05	5.98	54	–0.01	0.994
Autumn	Summer	–32.41	6.64	54	–4.88	< 0.001*
Autumn	Winter	11.07	6.76	54	1.64	0.108
Spring	Summer	–32.37	5.67	54	–5.71	< 0.001*
Spring	Winter	11.11	5.81	54	1.91	0.061
Summer	Winter	43.48	6.49	54	6.70	< 0.001*

df = degrees of freedom. * Significance of correlation: $P < 0.05$. ◆

doors suggest that production of vitamin D in the skin is the principal determinant of 25-OHD levels. These findings are consistent with research findings regarding the overall Australian population[16] and African Americans.[17] It is likely that time spent outdoors, particularly if it includes weight-bearing exercise, will have health benefits in addition to those associated with increased vitamin D production.

Darker skin pigmentation in Aboriginal Australians probably contributes to their lower mean 25-OHD level compared with that of non-Indigenous Australians. The lack of a significant association between skin colour and serum 25-OHD in our study may reflect the small sample size. We found no significant association between serum 25-OHD levels and BMI, in contrast with other studies that have shown a significant, negative correlation between BMI and serum 25-OHD levels in populations of all pigmentation types.[18-20] It is not clear why our study population should differ from others.

The significant association between BMI and PTH levels found in our study has been reported elsewhere, although it is unknown whether elevated serum PTH levels cause obesity, or whether obesity causes elevated serum PTH levels.[21]

A significant association was found between serum 25-OHD and fasting serum β-CTx levels, but not between serum 25-OHD and PTH levels. Clinical and animal studies have shown that inadequate serum levels of 25-OHD cause a rise in serum PTH levels, providing information on critical 25-OHD levels required to suppress calciotropic hormones and bone turnover markers. In our study, the serum 25-OHD level at which the β-CTx level began to rise (the inflection point) was about 80 nmol/L, which is comparable to that reported previously.[22] A slightly lower inflection point for 25-OHD and PTH (40 nmol/L) was reported in African American women.[23] We found that β-CTx levels decreased with age. This may be a reflection of higher bone turnover in participants aged about 28 years or younger.

Important limitations of our study were the small sample size, non-random selection process, and self-reporting of events in the month leading up to the study, which is subject to recall bias. While not truly representative of the Aboriginal population as a whole, our study participants included both staff and patients, and excluded people known to have diabetes, which was felt to be a reasonable compromise given the difficulties associated with recruiting a more formally representative sample. Assuming an effect size similar to previously published work, a sample size of 202 participants would have been required to properly examine the relationship between levels of vitamin D and fasting glucose.[24,25] Our sample was therefore too small for this purpose, and was also too small to confirm any negative effect of smoking on vitamin D status.[26,27]

In summary, vitamin D insufficiency is common in this population of adult Aboriginal Australians, as indicated by the low mean serum 25-OHD level overall and low mean levels found in all seasons other than summer. Target serum 25-OHD levels above 60 nmol/L have been recommended for prevention of fractures and falls,[28] with the suggestion that higher target levels (75–80 nmol/L) may be required for preventing the proposed immunologic and metabolic consequences of vitamin D deficiency.[29] Our study adds to previous work concerning the vitamin D status of dark-skinned, populations and will aid the design of future studies with adequate statistical power.

6 Correlation matrix for 25-OHD and age, BMI, and fasting glucose, PTH and fasting β-CTx levels in 58 Aboriginal adults (Spearman rank correlation coefficient)

Variable	Age	BMI	Fasting glucose*	PTH	Fasting β-CTx
BMI	0.069				
Fasting glucose*	0.306†	0.421†			
PTH	0.129	0.428†	0.143		
Fasting β-CTx	–0.408†	–0.146	–0.041	0.183	
25-OHD	0.08	0.02	0.196	–0.141	–0.293†

BMI = body mass index. β-CTx = C-terminal telopeptides of type 1 collagen. 25-OHD = 25-hydroxyvitamin D. PTH = parathyroid hormone. * In all analyses of fasting glucose, values were excluded for two participants whose fasting glucose levels were indicative of the presence of diabetes. † Significance of correlation: $P < 0.05$. ◆

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COMPETING INTERESTS

Howard Morris has spoken at meetings sponsored by Roche Diagnostics and Abbott Diagnostics; all honoraria were paid to his institution.

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