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ORIGINAL ARTICLE

Prevalence of vitamin D deficiency and its determinants in Australian adults aged 25 years and older: a national, population-based study

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Abstract

Objective Vitamin D deficiency is recognized as a global public health problem, but the population-based prevalence of deficiency and its determinants in Australian adults is not known. This study evaluated the vitamin D status of Australian adults aged ≥25 years and risk factors associated with vitamin D deficiency in this population.

Design and Patients We studied a national sample of 11 247 Australian adults enrolled in the 1999/2000 Australian Diabetes, Obesity and Lifestyle (AusDiab) study drawn from 42 randomly selected districts throughout Australia.

Measurements Serum concentrations of 25-hydroxyvitamin D [25(OH)D] were measured by immunoassay. Vitamin D deficiency was defined as a concentration <50 nmol/l. Information on demographic and lifestyle factors was derived from interview-administered questionnaires.

Results The mean serum 25(OH)D concentration was 63 nmol/l (95% CI: 59–67 nmol/l). Only 4% of the population had a level <25 nmol/l, but the prevalence of vitamin D deficiency (<50 nmol/l) was 31% (22% men; 39% women); 73% had levels <75 nmol/l. The prevalence of vitamin D deficiency increased significantly with age, was greater in women, in those of non-Europid origin, in the obese and those who were physically inactive and with a higher level of education. Deficiency was also more common during winter and in people residing in southern Australia (latitude >35°S); 42% of women and 27% of men were deficient during summer–autumn, which increased to 58% and 35%, respectively, during winter–spring.

Conclusion Vitamin D deficiency is common in Australia affecting nearly one-third of adults aged ≥25 years. This indicates that strategies are needed at the population level to improve vitamin D status of Australians.

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Introduction

Vitamin D deficiency has re-emerged as a major public health problem worldwide.[1,2] Currently, there is an ongoing debate as to what the optimal serum 25(OH)D concentration is for human health, but recent guidelines suggest that concentrations <50 and <75 nmol/l represent vitamin D deficiency and insufficiency, respectively.[3] Low vitamin D status, particularly at levels below 25 nmol/l, is well recognized to have clinically adverse effects on musculoskeletal health in adults, including osteomalacia, proximal myopathy, secondary hyperparathyroidism and osteoporosis.[3,4] A recent postmortem study that performed histomorphometric analysis of iliac crest bone biopsies from individuals diagnosed without skeletal disease also showed that a large number of patients with serum 25(OH)D levels between 25 and 50 nmol/l had histologically proven osteomalacia. [5] While it is important to highlight the criteria used to define osteomalacia (>2% osteoid volume) in this study were conservative in comparison with earlier histomorphometric studies,[4] the authors concluded that a serum 25(OH)D level of at least 75 nmol/l may be required to maintain bone health. However, further clinical studies are still needed.

Over the past two decades, several national population-based epidemiological studies in the United States,[6,7] Canada,[8,9] United Kingdom, [10] and New Zealand [11] have reported that 52–77% of the populations had 25(OH)D levels <75 nmol/l and 18% to 36% had levels <50 nmol/l. In Australia, vitamin D deficiency has been identified as a problem in certain subgroups, including elderly people, dark skinned and veiled women and those living in residential care,[12] but the vitamin D status of the general population and its

correlates has never been adequately determined. The aim of this study was to assess the vitamin D status and population-based prevalence of vitamin D deficiency in a national cohort of Australian adults aged ≥25 years who participated in the Australian Diabetes, Obesity and Lifestyle study (AusDiab) in 1999/2000. This is the first nationwide epidemiological study to have evaluated the vitamin D status of Australian adults. A secondary aim was to examine determinants of vitamin D deficiency and compare the prevalence rates by age, sex, race, obesity, physical activity and other demographic factors within this population.

Materials and methods

Study design and population

The AusDiab study was a national population-based survey conducted in 1999–2000 to determine the prevalence of diabetes, obesity and other cardiovascular disease risk factors in Australian adults. A detailed description of the methodology has been described elsewhere.[13] Briefly, a representative sample of the national population was drawn from 42 randomly selected urban and nonurban areas [Census Collector Districts (CDs)] across Australia, with six CDs in each of the six states and the Northern Territory. CDs that had fewer than 100 individuals aged ≥25 years or had more than 10% of the residents as Aborigines or Torres Strait Islanders and those classified as 100% rural were excluded. All homes within each CD were approached, and usual residents aged ≥25 years were invited to attend the survey, which consisted of a short household interview followed by a biomedical examination. Of the 19 215 households contacted for the AusDiab study, 17 129 were eligible for inclusion and 20 347 noninstitutionalized adults aged ≥25 years completed a household interview. Of these, 11 247 attended a biomedical evaluation (5049 men; 6198 women) in 1999–2000. The study sites in each State and Territory were visited in the following sequence: Victoria (latitude 34–38°S; year 1999; months, May–July); Western Australia (32°S, 1999, August–September); New South Wales (33–34°S, 1999, October–December); Tasmania (41–43°S, 2000, February–April); South Australia (35–38°S, 2000 – May–July); Northern Territory (12°S, 2000, July–September) and Queensland (17–28°S, 2000, October-December). Blood specimens for analysis of serum 25(OH)D were available for 11 218 people (99·7%). The study was approved by the International Diabetes Institute ethics committee (Melbourne, Australia). Written informed consent was obtained from all individuals.

Data collection

Demographic details including country of birth, location of residence and educational attainment were collected by trained interviewers using standardized questionnaires. Ethnicity was categorized into 'Europid' and 'non-Europid' based on the country of birth. The majority of participants (87·3%) were classified as Europids that included those born in Australia, Northern Europe, Canada, USA and New Zealand. The non-Europids included those born in Southern Europe (3·5%), Asia (4·0%), the Middle East (1·7%), India and Sri Lanka (1·2%), Pacific Islands (0·5%), Africa (0·6%) and South and Central America (0·3%). Aboriginal Australians and Strait Torres Islanders (0·9%) were also included as non-Europids. Participants were classified as living in an urban or rural region based upon the location of the CDs. Height, weight and BMI were measured using standard procedures. Overweight was defined as a BMI 25·0–29·9 kg/m^2 and obese as a BMI ≥30·0 kg/m^2. Smoking status was categorized into 'current smoker' versus 'ex- and non-smoker'. As reported previously,[13] total leisure-time physical activity (PA) reported for the previous week was calculated using the validated Active Australia questionnaire. Three categories were created to reflect current guidelines:[14] (i) those meeting the public health PA guidelines (≥2·5 h/week), (ii) those engaged in some PA but not meeting the public health guidelines (>0–2·49 h/week), and (iii) those reporting no activity (0 h/week). Total time spent watching television/videos in the previous 7 days was also self-reported, from which the following categories were created: <2, 2 to <4 and ≥4 h/day.[15]

Blood was drawn from participants after an overnight fast (minimum 8 h), centrifuged and transported daily to the central laboratory where an aliquot was stored at −70°C. Season of blood sampling was divided into summer (December–February), autumn (March–May), winter (June–August) and spring (September–November). The latitude of each blood collection centre was determined using the Google® GPS tool (http://maps.google.com/) and entered as a continuous variable for analysis (range 12°S to 43°S). As only 4% of the participants were located in far north Australia (latitude 12°S and 17°S), participants were categorized into three regional latitude categories corresponding to northern-central (<30°S), central (30–35°S) and southern (>35°S) Australia.

Serum 25-hydroxyvitamin D

Serum 25(OH)D was measured in the entire AusDiab population (n = 11 218 excluding 29 without any specimen available) on sera stored for 10 years at −70°C until the recent analysis. In 640 people where fasting serum specimens were not available, fluoride oxalate plasma (n = 590 from fasting plasma; n = 54 from 2 h plasma post-OGTT) was used for the analysis. As reported previously,[16] to evaluate whether fluoride oxalate plasma was comparable with fasting serum samples for the 25(OH)D analysis, we analysed both simultaneously from samples collected from 101 laboratory staff. There was excellent agreement between the two tubes: fluoride oxalate plasma 25(OH)D = 0·97 × serum 25(OH)D + 2·5, r^2 = 0·89. All the analyses for 25(OH)D were performed in the same laboratory using the Liaison®25OH vitamin D TOTAL (Liaison®25OHD) (DiaSorin Inc., Stillwater, MN, USA), a direct competitive chemiluminescent immunoassay (CLIA), with an interassay CV of 7·0% at 45 nmol/l and 6·3% at 92 nmol/l. This method in our laboratory had minimal bias against the Liquid Chromatography Mass Spectrometry (LCMS) targets set externally by the Royal College of Pathologists of Australasia & Australasian Association of Clinical Biochemistry (RCPA/AACB) Quality Assurance Program (QAP). When we compared our method directly to a LCMS method using 70 samples with serum 25(OH)D

levels ranging from 7 to 74 nmol/l, the correlation obtained was Liaison 25OHD (nmol/l) = 1·01 × LCMS 25OHD (nmol/l)–3·5 (r^2 = 0·85).

Statistical analysis

Analyses were performed using Stata Statistical Software version 10.1 (Stata Corp, College Station, TX, USA). To account for the clustering and stratification of the survey design, and to adjust for nonresponse, the data were weighted to match the age and sex distribution of the 1998 estimated residential population of Australians aged ≥25 years.[13] The weighting factor was based on the probability of selection in each cluster. Therefore, the prevalence data relate to the total 1998 Australian population aged ≥25 years.[17] Descriptive statistics were used to report the unadjusted and adjusted serum 25(OH)D concentrations by age-group categories, sex, ethnicity, season, latitude, location of residence, BMI categories, smoking status, education attainment and categories of physical activity and TV-viewing time. Mean adjusted serum 25(OH)D levels for men and women included all of the above variables as confounders. We also reported the proportion of men and women with serum 25(OH)D levels <25, <50 and <75 nmol/l. Vitamin D deficiency was defined as a level <50 nmol/l.[3] Multivariate logistic regression was used to examine independent predictors of vitamin D deficiency. For this analysis, each of the following factors were categorized as follows: age by 10-year intervals starting at 25; race into Europids or non-Europids; latitude as northern (<30°S), central (>30–35°S) and southern (>35°S) Australia; season as summer–autumn (December–May) and winter–spring (June–November); location of residence as urban or rural; BMI as <25 (normal weight), 25–30 (overweight) and >30 kg/m^2 (obese); smoking as current or never/ex-smoker; education as University/further education versus completed high/primary school or never attended school; physical activity as 0, >0 to <2·5 and ≥2·5 h/week; and TV-viewing time as <2, 2 to <4 and ≥4 h/day.

Results

As shown in Table 1, most of the participants were of Europid origin (approximately 87%) and resided in the central region of Australia (latitude 30–35°S). Approximately one-third of the men and nearly half of the women were classified as overweight or obese, and 43% of men and 53% of women were physically inactive (<2·5 h/week).

Distribution of serum 25(OH)D concentration

The mean (±SD) unadjusted serum 25(OH)D concentration for the cohort was 62·8 ± 25·4 nmol/l (median 60 nmol/l). On average, 25(OH)D levels were 9·9 nmol/l lower in women compared with men (P < 0·001) and decreased with advancing age in both genders (P for trend, < 0·001) (Table 2). Participants of non-Europid origin had 25(OH)D levels 15–20 nmol/l lower than Europids (P < 0·001). The lowest 25(OH)D levels were observed in participants living in the southern (>35°S) compared with northern (<30°S) regions of Australia (P for trend < 0·001). Levels also varied throughout the calendar year (Fig. 1), with the highest values recorded in December for men and February for women and the lowest levels observed in June for men and July for women. The mean difference in serum 25(OH)D levels between summer and winter ranged from 17 to 19 nmol/l. Mean adjusted serum 25(OH)D levels were significantly lower in those with higher BMI and lower levels of physical activity and those with a higher level of education (Table 2). There were no marked differences in serum 25(OH)D between smokers and non/ex-smokers, people living in urban compared with rural regions or those watching greater amounts of TV.

Table 1. Descriptive characteristics of the participants

Characteristics	Males (n = 5040)	Females (n = 6178)
Age, years [mean ± SD]	47·6 ± 15·1	48·9 ± 15·7
BMI, kg/m^2 [mean ± SD]	26·9 ± 4·1	26·4 ± 5·6
Healthy weight, %	32·4	47·8
Overweight, %	48·3	30·0
Obese, %	19·4	22·2
Race/Ethnicity, %		
Europids	87·0	87·5
Non-Europids	13·0	12·5
Latitude, %		
Northern <30°S	19·3	18·9
Central 30–35°S	56·1	56·2
Southern >35°S	24·6	24·9
Season of blood collection, %		
Summer (December–February)	13·5	12·0
Autumn (March–May)	14·1	14·4
Winter (June–August)	26·3	21·1
Spring (September–November)	46·1	47·4
Location of residence, %		
Urban	56·5	57·2
Rural	43·5	42·8
Current smoker, %	19·1	13·5
University or further education, %	47·2	36·0
Physical activity		
Total activity time, h/week	5·1 ± 6·3	3·8 ± 4·8
0 h/week, %	14·3	17·4
>0 to < 2·5 h/week, %	28·2	35·9
≥2·5 h/week, %	57·5	46·7
TV-viewing time, h/week	13·5 ± 9·9	12·0 ± 9·1
<2 h/day, %	54·5	60·2
2 to <4 h/day, %	36·8	37·6
≥4 h/day %	8·7	6·2

All data were weighted to the Australian population in 1998.

Prevalence of vitamin D deficiency by risk factors

Overall, 4·1% of the entire population had a serum 25(OH)D level <25 nmol/l, with women being more than twice as likely as men to have low levels (men 2·6% *vs* women 5·6%, P < 0·001) (Table 3). While the prevalence of a level <25 nmol/l did not change with increasing age in men, it increased from 3·9% in women aged

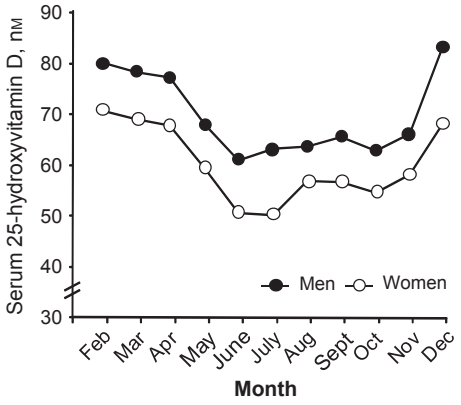

Fig. 1 Mean adjusted serum 25-hydroxyvitamin D concentrations in Australian men (n = 5040) and women (n = 6178) by month of the year. Values are means adjusted for age, ethnicity, latitude, location of residence, BMI categories, smoking status, education attainment and categories of physical activity and TV-viewing time and weighted to match the age and sex distribution of the 1998 residential population of Australian adults.

25–34 years to 12·5% in women aged ≥75 years. Vitamin D deficiency (<50 nmol/l) was found in 31% of all participants (22% men; 39% women, $P < 0{\cdot}001$), and 73% of the population had insufficient 25(OH)D levels (<75 nmol/l; men 67%; women 78%, $P < 0{\cdot}001$). As illustrated in Fig. 2, the proportion of people with serum 25(OH)D levels <50 and <75 nmol/l in both genders increased with age, with the highest rates of deficiency and insufficiency observed in men and women aged ≥75 years.

As expected, the proportion of adults with 25(OH)D levels <25, <50 and <75 nmol/l varied by ethnicity, season and latitude, with the highest rates of deficiency and insufficiency found in non-Europids, during the winter/spring months and at a latitude >35°S (Table 3 and Fig. 3). In terms of severe vitamin D deficiency, we found that 12·6–16·8% of non-Europids had a serum 25(OH)D level <25 nmol/l. In summer/autumn, the proportion of people with levels <50 nmol/l from the northern (<30°S) to southern (>35°S) regions of Australia increased from 7% to 27% in men and from 13% to 42% in women. In winter/spring, 15% and 31% of men and women living in the northern regions (<30°S) of Australia had levels <50 nmol/l, which increased to 35% in men and 58% in women for those living in southern regions (>35°S). We also found that rates of deficiency and insufficiency were greater in those with a higher level of education and those that did not meet the current PA guidelines (≥2·5 h/week) (Table 3).

Factors associated with vitamin D deficiency

In the multivariate logistic regression including all factors (Table 3), advancing age, ethnicity (non-Europids), latitude (>35°S) and season (autumn to spring for men; winter and spring for women) were all independent predictors for vitamin D deficiency (<50 nmol/l) in both men and women. In addition, lack of physical activity (<2·5 h/week), obesity (BMI >30 in men and 25–30 and >30 in women) and education (University/further education) were also found to be independent predictors for vitamin D deficiency.

Discussion

This study is the first nationwide, population-based prevalence study to have evaluated the vitamin D status of noninstitutionalized Australian adults aged 25–95 years. In this cohort of 11 218 Australian adults, we found that only 4% of the population had a level <25 nmol/l. While this level of 25(OH)D has been associated with biochemical and histological findings consistent with osteomalacia,[18] a recent study showed histological findings consistent with osteomalacia were also common in patients with levels between 25 and 50 nmol/l.[5] In our study, we found that nearly one-third (31%) of the population had a level <50 nmol/l, which has now been suggested to represent vitamin D deficiency,[3] with a higher prevalence found among women (39%) than men (22%). Furthermore, the odds for vitamin D deficiency increased with age and were greater in obese adults and those that did not meet the current PA guidelines of ≥2·5 h/week. Non-Europids and women aged ≥75 years had a four-fold increased odds of having vitamin D deficiency, after accounting for the other confounders. Based on a cut-point of 75 nmol/l, which some experts now regard as the optimal serum 25(OH)D concentration for musculoskeletal health benefits,[1,3,19] we found that nearly three-quarters (73%) of the Australian population were affected.

Because of the wide latitude range across Australia, pooling the results and reporting year-round averages for the prevalence of vitamin D deficiency is somewhat misleading. During summer/autumn, the prevalence of deficiency (<50 nmol/l) ranged from 6% to 13% in men and women in the central to northern regions of Australia (latitude <30°S) increasing to 27% in men and 42% in women in the southern regions (>35°S). During winter/spring, however, the prevalence of deficiency doubled in men (15%) and women (31%) in the central to northern regions of Australia, increasing to 35% in men and 58% in women in the southern regions. This season and latitudinal gradient of vitamin D deficiency is not unexpected given that UV-B irradiation at a latitude of approximately 35°S or greater during the winter months is not of sufficient intensity to catalyse the cutaneous synthesis of vitamin D.[20] Indeed, these findings support data from a previous population-based sample of women aged <60 years in three locations across Australia (27°S to 43°S), which reported that 25(OH)D levels decreased, on average, by 1·0 nmol/l for every degree increase in latitude.[20]

There is ongoing debate as to the optimal serum 25(OH)D concentration for human health and disease prevention. The Institute of Medicine (IOM) recommends that a 25(OH)D level of 50 nmol/l would be sufficient for practically all persons for skeletal health benefits.[21] In contrast, the Endocrine Society Clinical Practice Guidelines recently recommend a level ≥75 nmol/l.[3] Previous population-based studies from around the world have reported the prevalence of deficiency (<50 nmol/l) to be approximately 25% in Canada,[8,9] 22% to 36% in the United States,[6,7] 45–52% in New Zealand [11] and 47–65% in Korea.[22] Because of differences in the characteristics of the cohorts studied (age, BMI, ethnicity, latitude, dietary vitamin D intake, skin type, food fortification policies) and the methodology used to assess serum 25(OH)D levels, it is difficult to compare findings across continents and countries. Nevertheless,

Table 2. Mean unadjusted and adjusted serum 25-hydroxyvitamin D concentrations (nmol/l) by age-group categories, race/ethnicity, latitude, season, location of residence, BMI categories, smoking status and categories of physical activity and TV-viewing time in Australian men and women

	Mean Serum 25-hydroxyvitamin D Concentrations (nmol/l)					
	Males			Females		
Characteristics	*n*	Unadjusted	Adjusted[†]	*n*	Unadjusted	Adjusted[†]
Australian population	5040	67·9	67·7	6178	58·0	57·9
Age categories, years						
25–34	590	73·9*	72·2*	803	66·6*	64·8*
35–44	1092	68·6	69·2	1464	59·6	59·8
45–54	1343	65·2	65·6	1541	55·7	57·0
55–64	923	63·2	64·1	1088	54·1	55·7
65–74	730	66·8	66·9	834	52·6	53·4
75+	362	62·3	59·6	448	48·1	46·3
Race/Ethnicity						
Europids	4560	70·5[a]	70·0[a]	5539	59·8[a]	59·9[a]
Non-Europids	475	50·2[b]	52·7[b]	637	45·1[b]	44·4[b]
Latitude (°S)						
Northern <30	1363	75·2*	73·8*	1720	64·6*	62·4*
Central 30–35	2147	68·2	68·4	2650	58·3	58·5
Southern >35	1530	61·6	61·6	1808	52·3	53·3
Season						
Summer (December–February)	572	86·7*	82·3*	718	73·9*	68·0*
Autumn (March–May)	1174	65·6	70·4	1386	57·1	61·0
Winter (June–August)	1721	61·9	63·3	2064	50·9	52·5
Spring (September–November)	1573	66·5	65·2	2010	58·2	57·6
Location of residence						
Urban	3143	65·3	67·8	3745	55·7	57·3
Rural	1897	71·2	67·6	2435	61·1	58·8
BMI						
Healthy weight	1483	69·8*	70·5*	2655	62·3*	61·3*
Overweight	2455	68·5	68·1	1967	56·1	57·1
Obese	1037	63·1	62·2	1419	50·6	51·8
Smoking						
Current	873	68·0	67·8	871	58·6	55·1
Ex/nonsmoker	4063	67·7	67·3	5199	58·0	58·4
Education						
University+	2101	66·7	65·6[a]	2033	58·6	56·2[a]
High/primary/no School	2939	68·9	69·7[b]	4145	57·6	58·9[b]
Physical activity, h/week						
0	779	63·3*	64·1*	1131	53·2*	55·1*
>0 to < 2·5	1313	63·8	64·4	2104	55·7	56·1
≥2·5	2895	70·9	70·3	2888	61·6	60·3
TV-viewing time, h/day						
<2	3075	67·3	67·1	4092	58·7	58·2
2 to <4	1460	68·4	68·3	1562	57·1	57·1
≥4	459	68·6	69·6	478	56·6	59·6

All serum 25-hydroxvitamin D results represent weighted means to the Australian population in 1998.
*P for trend <0·05 to <0·001.
[†]Adjusted for age, race/ethnicity, season, latitude, location of residence, BMI category, smoking, education, physical activity and TV-viewing time.
Mean values not sharing a common superscript letters are significantly different, $P < 0·05$ to <0·001.

the results from our national population-based study are in line with these results and provide compelling evidence that vitamin D deficiency, defined as a level <50 nmol/l, is a common public health problem in Australia, particularly in the southern states.

There are a number of potential factors that may have contributed to the relatively high prevalence of deficiency across Australia. Given that sun exposure is the primary determinant of vitamin D status, it is likely to be related to increased sun-consciousness. Although we did not assess common sun protection practices, including sun avoidance, use of sunscreen or protective clothing, a previous study reported that sun protection behaviours have increased in response to the longstanding 'Sun Smart'

Table 3. Prevalence of serum 25(OH)D levels <25, <50 and <75 nmol/l and adjusted odds ratios (ORs) for vitamin D deficiency (<50 nmol/l) in Australian men (n = 5040) and women (n = 6178) in multivariate logistic regression

	Males					Females				
		Prevalence					Prevalence			
Characteristics	n	<25 nmol/l	<50 nmol/l	<75 nmol/l	Odds Ratio (95% CI)	n	<25 nmol/l	<50 nmol/l	<75 nmol/l	Odds Ratio (95% CI)
All	5040	2·6	22·1	66·7	–	6178	5·6	39·0	78·2	–
Age categories, years										
25–34	590	2·4	16·6	59·0	1·00 (Reference)	803	3·9	26·1	64·7	1·00 (Reference)
35–44	1092	1·8	21·5	65·5	1·22 (0·82, 1·82)	1464	6·0	36·0	75·7	1·52 (1·02, 2·26)
45–54	1343	2·3	23·3	70·8	1·49 (1·07, 2·08)	1541	5·2	43·5	81·2	1·88 (1·27, 2·80)
55–64	923	5·3	28·9	72·4	1·93 (1·27, 2·94)	1088	2·6	43·7	85·5	1·84 (1·25, 2·71)
65–74	730	1·8	22·1	68·4	1·36 (0·65, 2·82)	834	7·5	46·0	88·2	2·13 (1·33, 3·42)
75+	362	2·8	27·6	73·7	2·62 (1·61, 4·27)	448	12·5	56·6	90·6	4·11 (2·38, 7·11)
Race/Ethnicity										
Europids	4560	1·1	17·7	63·3	1·00 (Reference)	5539	4·0	35·7	76·2	1·00 (Reference)
Non-Europids	475	12·6	51·2	89·7	4·68 (3·14, 6·95)	637	16·8	61·9	91·9	3·49 (2·60, 4·68)
Latitude (°S)										
Northern <30	1363	1·0	13·8	55·9	1·00 (Reference)	1720	2·0	28·1	69·9	1·00 (Reference)
Central 30–35	2147	2·3	21·0	67·0	1·35 (0·84, 2·17)	2650	5·4	37·9	78·0	1·28 (0·88, 1·86)
Southern >35	1530	4·4	31·0	74·7	2·61 (1·49, 4·58)	1808	8·7	49·7	84·9	2·16 (1·37, 3·41)
Season										
Summer (December–February)	572	0·2	5·5	39·3	1·00 (Reference)	718	0·3	18·4	57·8	1·00 (Reference)
Autumn (March–May)	1174	3·9	26·4	69·9	2·72 (1·58, 4·68)	1386	5·1	41·5	79·5	1·43 (0·93, 2·21)
Winter (June–August)	1721	3·0	28·3	75·2	4·08 (2·50, 6·66)	2064	8·9	50·2	86·9	2·69 (2·07, 3·50)
Spring (September–November)	1573	2·6	22·1	69·0	3·31 (2·21, 4·97)	2010	5·2	37·2	78·2	1·82 (1·37, 2·41)
Location of residence										
Urban	3143	3·0	25·1	70·5	1·00 (Reference)	3745	7·1	43·4	80·5	1·00 (Reference)
Rural	1897	2·0	18·2	61·9	0·95 (0·70, 1·29)	2435	3·6	33·1	75·2	0·75 (0·61, 0·92)
BMI										
Healthy weight	1483	2·1	22·5	62·5	1·00 (Reference)	2655	5·2	32·0	71·4	1·00 (Reference)
Overweight	2455	2·8	19·4	66·5	0·93 (0·67, 1·30)	1967	6·0	42·0	81·9	1·43 (1·18, 1·72)
Obese	1037	2·9	28·1	74·7	1·70 (1·13, 2·56)	1419	5·1	50·9	88·4	2·04 (1·57, 2·65)
Smoking										
Ex/nonsmoker	4063	2·6	21·9	66·9	1·00 (Reference)	5199	5·6	38·7	78·8	1·00 (Reference)
Current	873	2·6	23·6	67·5	1·21 (0·85, 1·72)	871	5·6	40·5	76·0	1·40 (0·98, 2·01)
Education										
High/primary/no School	2939	1·8	20·2	65·8	1·00 (Reference)	4145	5·1	41·0	78·7	1·00 (Reference)
University+	2101	3·4	24·2	67·8	1·56 (1·22, 1·99)	2033	6·4	35·5	77·3	1·03 (0·84, 1·27)
Physical activity, h/week										
≥2·5	2895	2·1	17·5	62·0	1·00 (Reference)	2888	4·5	31·1	73·9	1·00 (Reference)
>0 to <2·5	1313	3·5	27·8	73·2	1·74 (1·27, 2,38)	2104	6·6	44·2	81·4	1·61 (1·36, 1·90)
0	779	2·9	29·6	73·7	1·85 (1·39, 2·48)	1131	5·9	48·9	83·5	1·77 (1·50, 2·09)
TV-viewing time, h/day										
<2	3075	2·7	22·9	66·9	1·00 (Reference)	4092	6·0	37·8	77·4	1·00 (Reference)
2 to <4	1460	2·0	19·9	67·7	0·86 (0·72, 1·01)	1562	4·7	39·2	79·0	1·03 (0·79, 1·34)
≥4	459	4·4	26·3	67·2	1·15 (0·85, 1·72)	478	5·7	48·2	81·1	1·28 (0·98, 2·01)

CI, confidence interval. All prevalence data represent weighted means to the Australian population in 1998[13]. All odds ratios adjusted for all characteristics shown.

campaign in Australia, which was designed to protect against skin cancer.[23] The importance of sun exposure to serum 25(OH)D levels in Australia was also recognized in a study that found that both simulated maximum daily duration of vitamin D synthesis, representing the daily time in which UVB exceeds the threshold required to produce vitamin D, and the effective daily dose of vitamin D, which is the daily dose of UVB wavelengths relevant to the conversion of 7-dehydrocholesterol into previtamin D in the skin, were both important predictors of vitamin D status.[20] In our study, we found that low level of physical activity, which is often used as a surrogate for the amount of time spent outdoors, was also a strong determinant of vitamin D

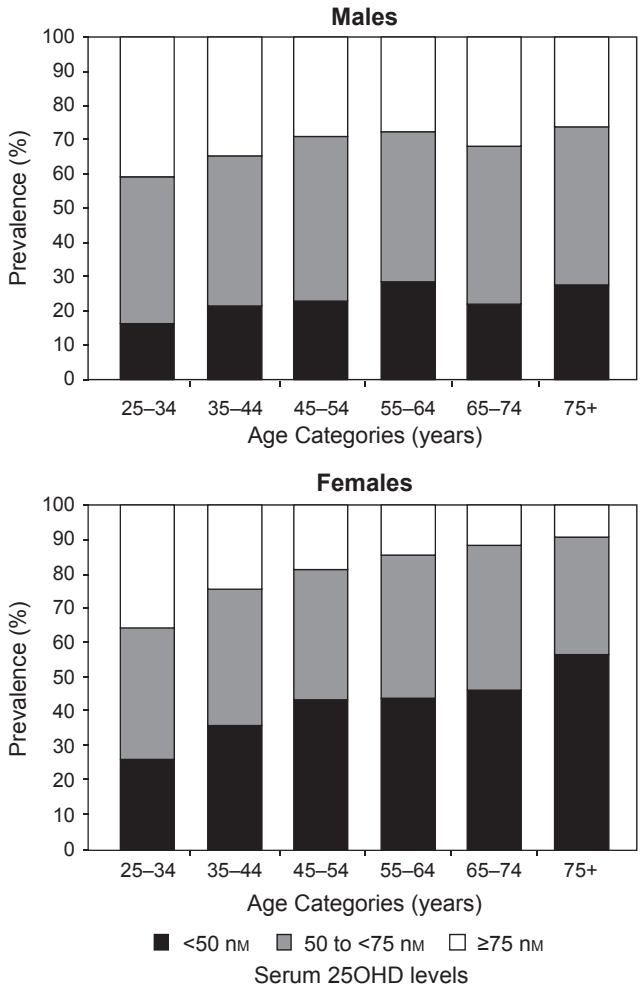

Fig. 2 Prevalence of serum 25(OH)D < 50, 50 to <75 and ≥75 nmol/l by age-group categories in Australian men (n = 5040) and women (n = 6178).

status which supports that the high prevalence of deficiency is likely due to decreased sun exposure.

Inadequate dietary vitamin D intakes may have also contributed, to a lesser extent, to the size of problem. However, it is generally understood that diet alone is insufficient to supply the required vitamin D to the body as few foods naturally contain vitamin D and margarine is the only food in Australia with mandatory fortification of vitamin D. Although we collected no data on dietary vitamin D intakes, previous research indicates that dietary intakes in Australian adults are low, varying from a mean of 1·2 to 2·6 μg/d.[12] This suggests that the majority of Australians would have an intake well below the currently recommended levels (adequate intakes) of 5, 10 and 15 μg/d for adults aged <50, 51–70 and 70 + years, respectively.[24] Furthermore, our intakes are lower than those reported in Canadian and US adults (4·3–6·2 μg/d),[25,26] which may be explained by differences in food fortification policies. In Canada, there is mandatory vitamin D fortification of milk and margarine, and in the US, more products are able to be fortified with vitamin D, and at levels higher than currently allowed in Australia.[25] Limited use of vitamin D-containing supplements, estimated to be around 8% in Australian adults aged <60 years at the time when the AusDiab study was conducted,[27,28] may also be an underlying factor contributing to the suboptimal vitamin D status in our population.

The high prevalence of vitamin D deficiency in our study may also be associated with increasing prevalence of obesity in Australia. Like many developed countries worldwide, Australia has experienced an approximate 2·5-fold increase in the prevalence of overweight and obesity over the past 20 years,[29] and adiposity is a well-known risk factor for vitamin D deficiency. The mean adjusted serum 25(OH)D level was 8·3–9·5 nmol/l lower in obese men and women compared with those of normal weight after adjusting for other confounders. This is likely caused by both decreased sun exposure from limited mobility and/or reduced outdoor physical activity [30], and the sequestration of serum 25(OH)D, a fat-soluble compound, in adipose tissue.[31] Serum vitamin D levels have been shown to be 57% lower in obese people than in those with normal weight following the same dose of UV exposure.[31]

Our multivariate analysis also showed that the odds for vitamin D deficiency increased progressively with advancing age and tended to be greater in women compared with men across all age categories. In women aged ≥75 years, the odds of vitamin D deficiency was over four-fold greater compared with women aged 25–34 years and was nearly double that of men ≥75 years. Ethnicity, as a proxy marker for darker skin pigmentation, was also a strong determinant of vitamin D deficiency as non-Europid descent was associated with a 3·5- and 4·7-fold greater odds of deficiency in women and men, respectively. These findings are consistent with previous studies[2,11] and have been attributed to decreased cutaneous production of vitamin D_3 with ageing and increased melanin, and impaired vitamin D absorption in the intestine.[32,33] The higher prevalence of vitamin D deficiency in women is also consistent with previous findings and may relate to gender differences in the time spent outdoors and/or clothing coverage.[7,8,11]

The strengths of this study include the recruitment of a large national, population-based sample of Australian adults aged from 25 to 95 years, and the assessment of 25(OH)D in one central laboratory using the same methodology. However, there are several limitations. First, this is a cross-sectional study, and thus, causality between vitamin D deficiency and its determinants cannot be determined. Second, no information was collected on the intake of vitamin D through diet or supplements, and thus, we could not evaluate their effects on vitamin D status. Third, we did not obtain data on sun exposure practices, including time spent outdoors or sunscreen use, which is important since UVB exposure is the primary source of vitamin D. Fourth, country of birth was used as a proxy measure of ethnicity. This may led to some misclassification as people who are born in the same country could have a different ethnic background. Fifth, since the prevalence of vitamin D deficiency was common, it is likely that the odds ratios overestimate the true measure of effect (e.g. relative risk).[34] Sixth, as reported previously those participants that responded to the biomedical examination (55%) tended to be slightly more educated (58·2 *vs* 51·3% with higher education), more likely to have been born in the UK (10·3 vs 8·8%) and more likely to speak English at home (96·1 *vs* 93·6%).[13] Although these differences were small, it is possible that this group was healthier and thus had higher 25(OH)D levels than nonresponders, which may have under-estimated the true

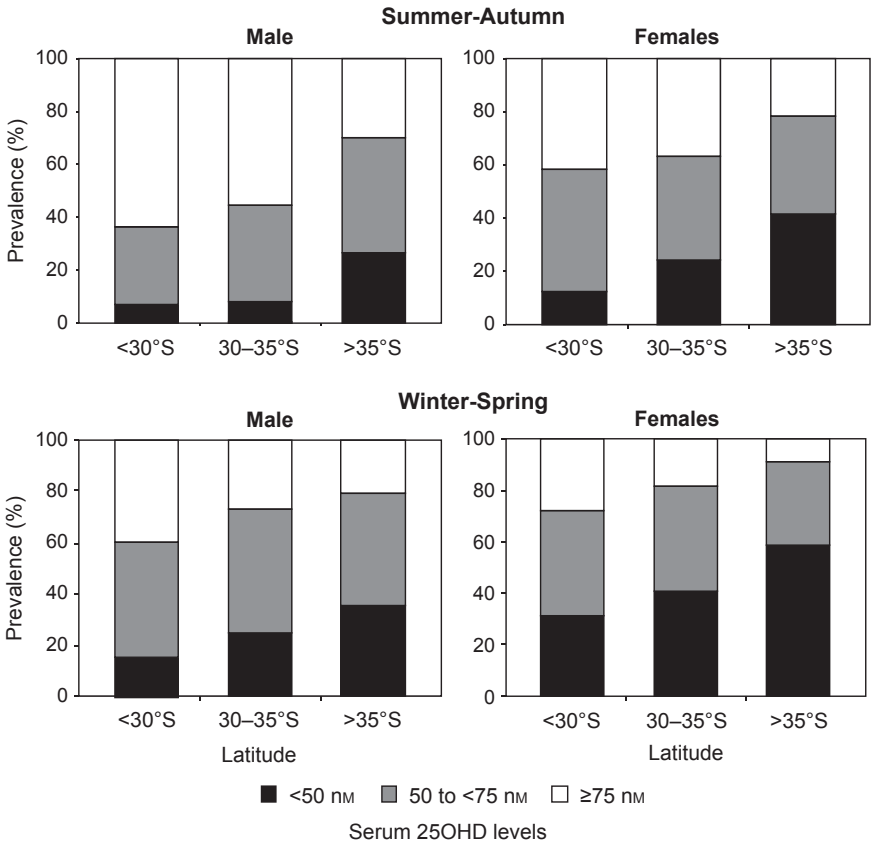

Fig. 3 Prevalence of serum 25(OH)D < 50, 50 to <75 and ≥75 nmol/l by latitude and season in Australian men (n = 5040) and women (n = 6178).

prevalence of vitamin D deficiency throughout Australia. Finally, we assessed 25(OH)D levels from the original AusDiab study conducted in 1999/2000, and thus, it is possible that there has been a secular change in vitamin D status over the past 10 years. Indeed, NHANES data demonstrate a marked decrease in mean 25(OH)D levels from 1988–1994 to 2001–2004.[6]

In conclusion, we found that nearly one-third of the Australian population had vitamin D deficiency (<50 nmol/l). When evaluated by season and latitude, it was alarming that 42% of women and 27% of men in southern Australia during summer/autumn had deficient levels, which increased to 58% and 35% in women and men, respectively, during winter/spring. Those at greatest risk for vitamin D deficiency included women, the elderly, obese, those not meeting the current physical activity guidelines of ≥2·5 h/week, and those of non-Europid descent. While it was reassuring that only 4% of the population had a level <25 nmol/l, indicative of more severe deficiency, given that emerging data have linked suboptimal vitamin D status with many chronic diseases and all-cause mortality,[32,35,36] there is a need to develop national strategies across the whole population for both safe sun exposure and adequate vitamin D intake for people who are unable to obtain sufficient sunlight to improve the vitamin D status.

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Disclosure

The authors have nothing to declare.

References

1 Dawson-Hughes, B., Heaney, R.P., Holick, M.F. *et al.* (2005) Estimates of optimal vitamin D status. *Osteoporosis International*, **16**, 713–716.

2 Mithal, A., Wahl, D.A., Bonjour, J.P. *et al.* (2009) Global vitamin D status and determinants of hypovitaminosis D. *Osteoporosis International*, **20**, 1807–1820.

3 Holick, M.F., Binkley, N.C., Bischoff-Ferrari, H.A. *et al.* (2011) Evaluation, treatment, and prevention of vitamin D deficiency: an Endocrine Society clinical practice guideline. *Journal of Clinical Endocrinology and Metabolism*, **96**, 1911–1930.

4 Lips, P. (2001) Vitamin D deficiency and secondary hyperparathyroidism in the elderly: consequences for bone loss and fractures and therapeutic implications. *Endocrine Reviews*, **22**, 477–501.

5 Priemel, M., von Domarus, C., Klatte, T.O. *et al.* (2010) Bone mineralization defects and vitamin D deficiency: histomorphometric analysis of iliac crest bone biopsies and circulating 25-hydroxyvitamin D in 675 patients. *Journal of Bone and Mineral Research*, **25**, 305–312.

6 Ginde, A.A., Liu, M.C. & Camargo Jr, C.A. (2009) Demographic differences and trends of vitamin D insufficiency in the US population, 1988–2004. *Archives of Internal Medicine*, **169**, 626–632.

7 Looker, A.C., Dawson-Hughes, B., Calvo, M.S. *et al.* (2002) Serum 25-hydroxyvitamin D status of adolescents and adults in two seasonal subpopulations from NHANES III. *Bone*, **30**, 771–777.

8 Greene-Finestone, L.S., Berger, C., de Groh, M. *et al.* (2011) 25-Hydroxyvitamin D in Canadian adults: biological, environmental, and behavioral correlates. *Osteoporosis International*, **22**, 1389–1399.

9 Langlois, K., Greene-Finestone, L., Little, J. *et al.* (2010) Vitamin D status of Canadians as measured in the 2007 to 2009 Canadian Health Measures Survey. *Health Reports*, **21**, 47–55.

10 Hypponen, E. & Power, C. (2007) Hypovitaminosis D in British adults at age 45 y: nationwide cohort study of dietary and lifestyle predictors. *American Journal of Clinical Nutrition*, **85**, 860–868.

11 Rockell, J.E., Skeaff, C.M., Williams, S.M. *et al.* (2006) Serum 25-hydroxyvitamin D concentrations of New Zealanders aged 15 years and older. *Osteoporosis International*, **17**, 1382–1389.

12 Nowson, C.A. & Margerison, C. (2002) Vitamin D intake and vitamin D status of Australians. *Medical Journal of Australia*, **177**, 149–152.

13 Dunstan, D.W., Zimmet, P.Z., Welborn, T.A. *et al.* (2002) The Australian Diabetes, Obesity and Lifestyle Study (AusDiab) – methods and response rates. *Diabetes Research and Clinical Practice*, **57**, 119–129.

14 Nelson, M.E., Rejeski, W.J., Blair, S.N. *et al.* (2007) Physical activity and public health in older adults: recommendation from the American College of Sports Medicine and the American Heart Association. *Medicine and Science in Sports and Exercise*, **39**, 1435–1445.

15 Dunstan, D.W., Salmon, J., Owen, N. *et al.* (2004) Physical activity and television viewing in relation to risk of undiagnosed abnormal glucose metabolism in adults. *Diabetes Care*, **27**, 2603–2609.

16 Gagnon, C., Lu, Z.X., Magliano, D.J. *et al.* (2011) Serum 25-Hydroxyvitamin D, Calcium Intake, and risk of type 2 diabetes after 5 years: results from a national, population-based prospective study (The Australian Diabetes, Obesity and Lifestyle Study). *Diabetes Care*, **34**, 1133–1138.

17 Australia Bureau of Statistics (1999) *Population by Age and Sex, Australian States and Territories*. Australian Bureau of Statistics, Canberra.

18 Vieth, R. (1999) Vitamin D supplementation, 25-hydroxyvitamin D concentrations, and safety. *American Journal of Clinical Nutrition*, **69**, 842–856.

19 Heaney, R.P. & Holick, M.F. (2011) Why the IOM recommendations for vitamin D are deficient. *Journal of Bone and Mineral Research*, **26**, 455–457.

20 van der Mei, I.A., Ponsonby, A.L., Engelsen, O. *et al.* (2007) The high prevalence of vitamin D insufficiency across Australian populations is only partly explained by season and latitude. *Environmental Health Perspectives*, **115**, 1132–1139.

21 Ross, A.C., Manson, J.E., Abrams, S.A. *et al.* (2011) The 2011 report on dietary reference intakes for calcium and vitamin D from the Institute of Medicine: what clinicians need to know. *Journal of Clinical Endocrinology and Metabolism*, **96**, 53–58.

22 Choi, H.S., Oh, H.J., Choi, H. *et al.* (2011) Vitamin D Insufficiency in Korea – a greater threat to younger generation: the Korea National Health and Nutrition Examination Survey (KNHANES) 2008. *Journal of Clinical Endocrinology and Metabolism*, **96**, 643–651.

23 Dobbinson, S.J., Wakefield, M.A., Jamsen, K.M. *et al.* (2008) Weekend sun protection and sunburn in Australia trends (1987–2002) and association with SunSmart television advertising. *American Journal of Preventive Medicine*, **34**, 94–101.

24 National Health and Medical Research Council (NHMRC) (2006) *Nutrient reference values for Australia and New Zealand including Recommended Dietary Intakes*. Department of Health and Ageing, Canberra. http://www.nhmrc.gov.au/_files_nhmrc/file/publications/synopses/n35.pdf.

25 Calvo, M.S., Whiting, S.J. & Barton, C.N. (2004) Vitamin D fortification in the United States and Canada: current status and data needs. *American Journal of Clinical Nutrition*, **80**, 1710S–1716S.

26 Vatanparast, H., Calvo, M.S., Green, T.J. *et al.* (2010) Despite mandatory fortification of staple foods, vitamin D intakes of Canadian children and adults are inadequate. *Journal of Steroid Biochemistry and Molecular Biology*, **121**, 301–303.

27 Pasco, J.A., Henry, M.J., Nicholson, G.C. *et al.* (2001) Vitamin D status of women in the Geelong Osteoporosis Study: association with diet and casual exposure to sunlight. *Medical Journal of Australia*, **175**, 401–405.

28 van der Mei, I.A., Ponsonby, A.L., Dwyer, T. *et al.* (2007) Vitamin D levels in people with multiple sclerosis and community controls in Tasmania, Australia. *Journal of Neurology*, **254**, 581–590.

29 Cameron, A.J., Welborn, T.A., Zimmet, P.Z. *et al.* (2003) Overweight and obesity in Australia: the 1999–2000 Australian Diabetes, Obesity and Lifestyle Study (AusDiab). *Medical Journal of Australia*, **178**, 427–432.

30 Scragg, R. & Camargo Jr, C.A. (2008) Frequency of leisure-time physical activity and serum 25-hydroxyvitamin D levels in the US population: results from the Third National Health and Nutrition Examination Survey. *American Journal of Epidemiology*, **168**, 577–586; discussion 587–591.

31 Wortsman, J., Matsuoka, L.Y., Chen, T.C. *et al.* (2000) Decreased bioavailability of vitamin D in obesity. *American Journal of Clinical Nutrition*, **72**, 690–693.

32 Holick, M.F. (2007) Vitamin D deficiency. *New England Journal of Medicine*, **357**, 266–281.
33 MacLaughlin, J. & Holick, M.F. (1985) Aging decreases the capacity of human skin to produce vitamin D3. *Journal of Clinical Investigation*, **76**, 1536–1538.
34 Zhang, J. & Yu, K.F. (1998) What's the relative risk? A method of correcting the odds ratio in cohort studies of common outcomes *JAMA*, **280**, 1690–1691.
35 Borradale, D. & Kimlin, M. (2009) Vitamin D in health and disease: an insight into traditional functions and new roles for the 'sunshine vitamin'. *Nutrition Research Reviews*, **22**, 118–136.
36 Dobnig, H., Pilz, S., Scharnagl, H. *et al.* (2008) Independent association of low serum 25-hydroxyvitamin d and 1,25-dihydroxyvitamin d levels with all-cause and cardiovascular mortality. *Archives of Internal Medicine*, **168**, 1340–1349.